

Understanding and Treating “Pusher Syndrome”

“Pusher syndrome” is a clinical disorder following left or right brain damage in which patients actively push away from the nonhemiparetic side, leading to a loss of postural balance. The mechanism underlying this disorder and its related anatomy has only recently been identified. Investigation of patients with severe pushing behavior has shown that perception of body posture in relation to gravity is altered. The patients experience their body as oriented “upright” when the body actually is tilted to the side of the brain lesion (to the ipsilesional side). In contrast, patients with pusher syndrome show no disturbed processing of visual and vestibular inputs determining visual vertical. These new insights have allowed the authors to suggest a new physical therapy approach for patients with pusher syndrome where the visual control of vertical upright orientation, which is undisturbed in these patients, is the central element of intervention. [Karnath H-O, Broetz D. Understanding and treating “pusher syndrome.” *Phys Ther.* 2003;83: 1119–1125.]

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A new physical therapy approach is suggested based on new insights into the nature of pushing behavior.

In 1985, Patricia Davies first described the surprising behavior of some patients with stroke who use their nonparetic extremities to push toward the paretic side.¹ When sitting or standing, these patients use their nonparetic extremities to push away from the nonparetic side, leading to a loss of lateral postural balance (Fig. 1). If not prevented, they would push themselves laterally to the point where they would fall toward the hemiparetic side. There is forceful resistance against interventions aiming to correct their tilted posture. Davies¹ termed this behavior the “pusher syndrome.” An investigation of 327 patients with acute stroke and hemiparesis revealed that the disorder was present in 10.4% of the patients.²

The purpose of this article is to summarize the recent literature related to pusher syndrome, including its clinical diagnosis, related anatomy, and a description of the mechanism found to be responsible for the disorder. Further, a new physical therapy approach is suggested based on these new insights into the nature of pushing behavior.

Pusher Syndrome—Distinctive Disorder or Catch-all for Different Expressions of Postural Instability Following Stroke?

Pushing actively with nonparetic extremities to the side contralateral to the brain lesion (Fig. 1), which is termed “contraversive pushing,” differentiates the clinical picture of pusher syndrome from the loss of equilibrium that can occur in other patients with hemiparesis. Because of their paresis, patients who do not exhibit pusher syndrome may show deficits in balance and may fall toward their paretic side.^{3,4} In contrast to patients who exhibit contraversive pushing, these patients recognize when they lose equilibrium but are unable to support themselves because of their paresis. They usually cling to something with their nonparetic hand (ie, they tend to pull, not push).

Use of the term “pusher syndrome” for a number of different postural instability symptoms that occur in patients after brain damage (for an overview, see Schädler and Kool⁵) should be avoided. The term should be used for the distinctive disorder of actively pushing away from the nonhemiparetic side as defined by Davies¹ and illustrated in Figure 1. Until recently, the pathophysio-

logical mechanism leading to pusher syndrome and the particular brain structure damaged were unknown.

Is Contraversive Pushing Caused by Hemineglect and Thus a Typical Disorder of the Right Hemisphere?

Davies¹ assumed that contraversive pushing frequently occurs when left-sided neglect is present after lesions of the right hemisphere. Similar assumptions have been put forward by other authors.^{6,7} Thus, there has been speculation about whether pushing behavior might be caused by spatial neglect or might reflect a severe right hemisphere syndrome.⁸

Davies,¹ however, also observed that pushing behavior is not almost exclusively associated with right brain damage, as is the case for patients who exhibit spatial neglect.^{9,10} Pusher syndrome frequently occurs also with lesions of the left hemisphere and is not related to neglect but rather to aphasia.¹ A study of 327 patients with acute stroke and hemiparesis who were investigated within the first couple of days after onset of stroke led to the observation that left and right hemisphere damage occurs with equal frequency in patients with contraversive pushing (left brain damage: 47%; right brain damage: 53%).² Moreover, there was no evidence for a regular co-occurrence of pathological pushing with spatial neglect, anosognosia, aphasia, or apraxia.²

In agreement with Pedersen et al,² Karnath and co-workers¹¹ found that hemispacial neglect is not the cause of contraversive pushing. In their sample of 23 patients with pusher syndrome, they found a large proportion who had left brain damage and thus aphasia but no neglect. Sixty-five percent of their patients with contraversive pushing had right-side lesions, and 35% had left-side lesions. Although contraversive pushing within the group of patients with right-side lesions was highly associated with spatial neglect (80% of these patients also had neglect), neglect did not appear to be the cause of pushing behavior. The reason for this observation was that 20% of the patients with right brain damage who exhibited contraversive pushing and 100%

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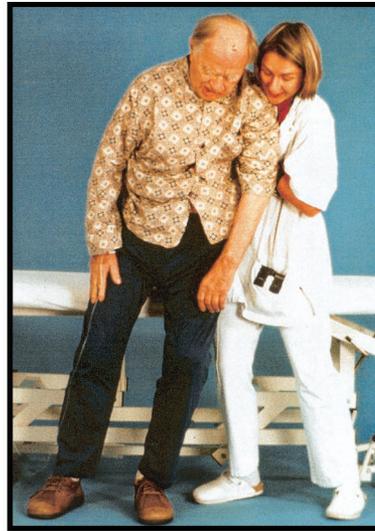
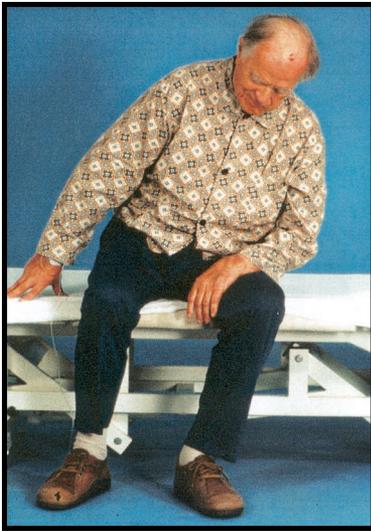


Figure 1.

A patient with right-side brain damage and pusher syndrome. The characteristic feature of the disorder is that these patients, while sitting (left) or standing (right), spread the nonparetic extremities from the body to push away actively from the nonparetic side. The result is the typical tilted body posture of these patients. If not assisted by the examiner, the patients push themselves into a lateral inclination until they fall toward the hemiparetic side.

of the patients with left brain damage who exhibited contraversive pushing showed no symptoms of spatial neglect. All of the patients had pusher syndrome due to left-sided brain lesions rather than aphasia.¹¹

We conclude that both neglect and aphasia are highly associated with pushing behavior after right-side brain damage (→ neglect) and after left-side brain damage (→ aphasia), but that both disorders neglect and aphasia cannot be the underlying cause of pusher syndrome. Symptoms such as neglect and anosognosia after right-sided lesions and aphasia after left-sided lesions frequently exist with contraversive pushing because the relevant brain structures associated with these functions lie in close proximity to each other. Neither neglect nor aphasia, however, is causally related to contraversive pushing.

What Is the Brain Structure Typically Damaged in Patients With Pusher Syndrome?

Based on the traditional assumption that pushing behavior is caused by spatial neglect, it has been assumed that the lesion location typically found in patients with spatial neglect also must be responsible for pushing behavior. Many therapists and physicians, therefore, have been taught that pusher syndrome is most common in patients with strokes in the parietal lobe of the right hemisphere.

When this assumption was studied for the first time, the data showed that the parietal cortex is not the neural correlate of pusher syndrome.¹¹ In a sample of 23

patients with severe contraversive pushing who were consecutively admitted to a neurology department, the authors identified brain lesions by magnetic resonance imaging or computed tomography. The overlap area of infarction in the patients with pusher syndrome was determined and compared with that of a sample of 23 patients with stroke admitted in the same period who did not exhibit contraversive pushing but were similar with regard to age, etiology of lesion, presence of hemiparesis, spatial neglect, and aphasia. The analysis revealed that the brain structure typically damaged in patients with pusher syndrome is the left or right posterolateral thalamus. This finding suggests that the posterolateral thalamus is involved in our control of upright body posture.

Traditionally, the posterolateral part of the thalamus was thought to serve simply as a “relay structure” of the vestibular pathway on its way from the brain stem to the cortex. The findings of Karnath and colleagues,¹¹ however, showed that this is not the only task of the posterolateral thalamus. The ventral posterior and lateral posterior nuclei of the posterolateral thalamus rather seem to be fundamentally involved in our control of upright body posture. Patients exhibiting severe contraversive pushing showed a clear overlap of their infarctions in this portion of the thalamus.¹¹ This structure is anatomically distinct from the “vestibular cortex” identified by Brandt and co-workers¹² in the posterior insula. In addition, the clinical findings in patients with such posterior insula lesions are different. While a lesion of the human “vestibular cortex” leads to a tilt of the perceived visual vertical but not to contraversive pushing,¹² a lesion of the posterolateral thalamus in patients with pusher syndrome induces the opposite pattern. The patients with contraversive pushing show normal perception of visual vertical, but they exhibit a severe tilt of perceived body posture in relation to gravity.¹³ Thus, both graviceptive systems not only appear to use distinct anatomical structures but also seem to process afferent sensory information from peripheral input sources differently.

Future studies are needed to investigate the possible role of diaschisis.¹⁴ Lesions of those thalamic nuclei (ventral posterolateral, ventral posteromedial, and lateral posterior) that were found to be affected in patients with contraversive pushing¹¹ might lead to additional functional or metabolic abnormalities in some of the structurally intact regions of the cortex. Thalamocortical

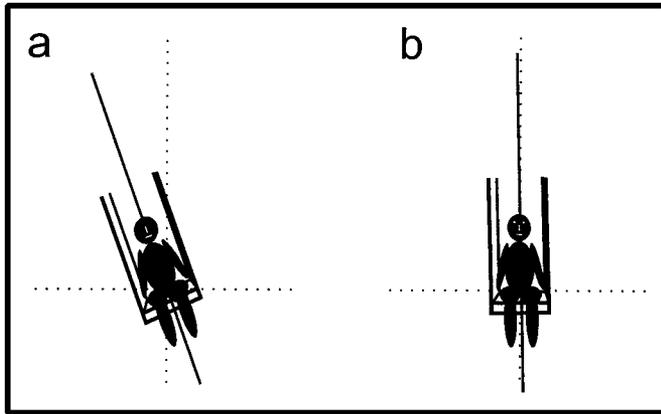


Figure 2.

Sitting on a tilting chair, patients with pusher syndrome were required to indicate when they reached “upright” body orientation.¹³ (a) With occluded eyes, the patients experienced their body as oriented “upright” when actually tilted 18 degrees to the side of the brain lesion. (b) While viewing the laboratory’s structured surroundings, the same patients could align their longitudinal body axis to earth vertical.

axons arising in the ventral posterolateral and ventral posteromedial nuclei project to the primary somatosensory cortex in the postcentral gyrus (Brodmann areas 3a, 3b, 1, and 2), to the secondary somatosensory cortex in the parietal operculum, and to the insula.¹⁵ The lateral posterior nucleus projects to the posterior parts of areas 5 and 7 of the superior and inferior parietal lobules.¹⁵ Imaging and other metabolic studies might help to assess whether additional critical substrates in the cortex are present and relevant in patients with pusher syndrome.

Which Mechanism Leads to Pusher Syndrome?

Recently, the mechanism leading to contraversive pushing has been investigated.¹³ The authors examined the ability of patients with pusher syndrome to determine upright position while their eyes were occluded. The researchers found an altered perception of the body’s orientation in relation to gravity by using a seating device that allowed tilting the patient to the right or to the left without ground contact. After a random tilt to the left or to the right of at least 35 degrees, the patients were required to indicate when they reached upright body orientation. On the average, patients with contraversive pushing experienced their body as oriented “upright” when actually tilted 18 degrees to the side of the brain lesion (Fig. 2a). Surprisingly, the same patients showed no disturbed orientation perception of the visual world (visual vertical). Thus, in contrast to their disturbed perception of upright body posture (Fig. 2a), patients with pusher syndrome could align their longitudinal body axis to earth vertical when using visual cues from the laboratory’s surroundings¹³ (Fig. 2b). In addition, without a visual surround in complete darkness, these patients could correctly determine visual vertical when sitting upright as well as when tilted in the position that is perceived as “upright.” The latter results indicate that

processing of visual and vestibular inputs for the determination of visual vertical was unaffected by the lesion. Consequently, when patients with pusher syndrome sit upright, they experience a mismatch between visual vertical, based on vestibular and visual inputs on the one side, and their perception of tilted body orientation relative to the vertical (Fig. 2).

Generally, a conflict between 2 reference systems is either resolved by suppressing one of them, or both, or by a compromise (eg, by weighted summation). However, neither happens in the present case.¹³ Under normal bedside conditions, the patients with contraversive pushing do not align their body with the visual vertical, with their perceived postural vertical, or with an intermediate posture. They instead move the body in the opposite direction. Karnath et al¹³ speculated that the patients, by pushing their longitudinal body axis toward the contralesional side, might be trying to compensate for the mismatch between visual vertical and the tilted orientation of body verticality. The clinical observation that patients with contraversive pushing diminish their pushing behavior when visual input is excluded (with eyes closed) supports this notion. Accordingly, contraversive pushing did not occur when the patients with contraversive pushing were tilted to the ipsilesional side (the perceived “upright” position) and structured visual input was excluded.¹³ Thus, the therapist’s attempt to correct the patients’ body posture toward the upright position (undertaken with eyes open) seems to contradict the patients’ effort to “compensate” the mismatch between visual vertical and the tilted orientation of perceived body verticality, and it induces the feeling of lateral instability and their fear of falling and provokes their active resisting against such attempts.¹³

Alternatively, it is possible that the pushing behavior is a secondary response to the patients’ unexpected experience that they lose lateral balance when trying to get up and sit upright. The experiment of Karnath et al¹³ revealed that the patients’ perceived “upright” orientation was tilted about 18 degrees toward the ipsilesional side. Thus, when patients try to get up and orient the body to what they perceive to be “upright,” they become laterally instable because the center of mass is shifted too far to the ipsilesional side. Pushing the body to the opposite (contralesional) side might be the ensuing reaction to this experience.¹³ Therefore, no pushing occurs when patients sit immobilized by lateral stabilization in the cushioned safety of the experimental chair that was used in the study.¹³

In the future, researchers may want to further investigate these possible interpretations. Nevertheless, the study of Karnath et al,¹³ clearly showed for the first time that contraversive pushing is due to a severe misperception of

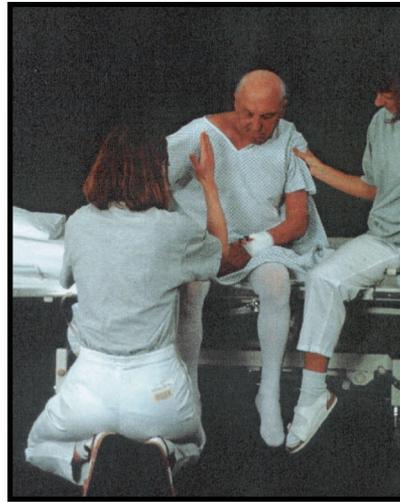
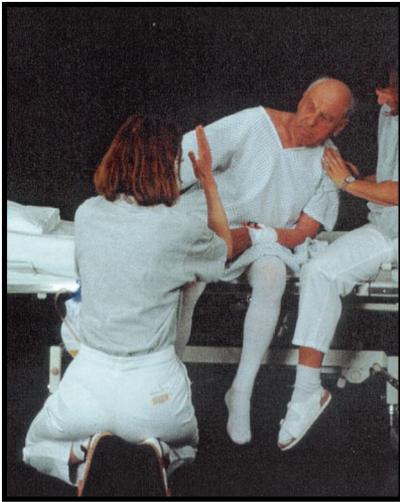


Figure 3. Patient with left-sided hemiparesis and pusher syndrome. Because orientation perception of the visual surround is not impaired in these patients,¹³ they realize they are not in an erect position by looking at their structured surroundings and can align their body with these earth-vertical structures. In addition, the therapist may give visual aids (eg, use of an arm) to demonstrate the earth-vertical, upright orientation (left). With little help, the patient is now able to orient his body upright (right).

body orientation in relation to gravity. Moreover, the data suggest that, for the purpose of rehabilitation, the preserved ability to align the body axis to earth vertical with the help of visual cues might be helpful. Although patients with pusher syndrome are not spontaneously able to use the visual input to control upright body posture, this might become possible when training procedures apply this ability as part of conscious strategies to control posture in these patients.

The discrepancy of a pathologically tilted *postural* vertical concurrent with an unimpaired perception of the *visual* vertical shows that patients with contraversive pushing manifest a selective disturbance of control of upright body posture.¹³ Although they are no longer able to determine when their body is oriented in an erect position, they have no problems correctly determining the orientation of the visual world around them. Patients with lesions of the vestibular system behave exactly the opposite. They show visual-vestibular dysfunction with a perceptual tilt of the visual vertical but have no problems orienting their body to an earth-vertical, upright position.^{12,16,17}

These dissociations provide evidence for a separate pathway in humans for sensing the orientation relative to gravity that is apart from the well-known pathway for orientation perception of the visual world. For this reason, Karnath et al¹¹ posited that the brain structure typically damaged in patients with pusher syndrome—the posterolateral thalamus—might constitute the neu-

ral representation of this second graviceptive system in humans.

Diagnosis of Pushing Behavior

Our daily clinical experience leads us to suggest 3 variables important in the examination of patients with contraversive pushing: (1) spontaneous body posture, (2) increase of pushing force by spreading of the nonparetic extremities from the body, and (3) resistance to passive correction of posture. We determine these variables with the patient both sitting (feet with ground contact) and standing. The examiner sits or stands on the paretic side of the patient to prevent falling.

Spontaneous Body Posture

The most striking feature of patients with contraversive pushing is their spontaneous posture while sitting and standing. Their longitudinal body axis is tilted toward the paretic side. This

behavior is best observed without prior instructions, right after changing position (eg, from a supine position to sitting at the bedside). To quantify pathological body posture, we differentiate among 3 intensities (see Appendix): severe contraversive tilt with falling to the side contralateral to the brain lesion, severe contraversive tilt without falling, and mild contraversive tilt without falling. A tilted longitudinal body axis must occur regularly, not just occasionally, due to the normal insecurity in balancing when patients become hemiparetic after stroke.

Abduction and Extension of the Nonparetic Extremities

Another feature of contraversive pushing is the use of the nonparetic extremities to bring about the pathological lateral tilt of the body axis. With the patient sitting on the bedside, we observed that the ipsilesional hand is abducted from the body searching for contact with the surface and the elbow is extended (Fig. 1). In our experience, if the feet have ground contact, the ipsilesional leg will be abducted, and the knee and hip joints will be extended as well. To quantify this characteristic feature, we use visual assessment of abduction and extension of the extremities (see Appendix), depending on whether the movements occur spontaneously even at rest or only on changing position (eg, on moving the patient from the wheelchair to the bed or on standing up after sitting).

Resistance to Passive Correction of Tilted Posture

Evaluating a patient's behavior on being corrected by the investigator to an upright position is the third

diagnostic feature for determining the presence of contraversive pushing. It is known that any attempt by the examiner to move the tilted body axis to an upright position by shifting the weight toward the nonparetic side elicits active resistance from the patient. The patient increases the force in the already extended nonparetic extremity. During our clinical examination (see Appendix), we evaluate the occurrence or nonoccurrence of active resistance to being interventionally corrected.

The Appendix summarizes the 3 variables (ie, spontaneous body posture, increase of pushing force by spreading of the nonparetic extremities from the body, and resistance to passive correction of posture) in the form of a scale, published as the so-called “Clinical Scale for Contraversive Pushing (SCP).”^{13,18} The authors^{13,18} intended the scale to aid clinicians in diagnosing the presence of pushing behavior and determining its severity. The weighted values that were tentatively assigned to each finding of the examination in the Appendix are still in the process of being validated. For a firm diagnosis of contraversive pushing, we suggest a value of 1 or more (summed over the results for sitting and standing; maximum=2 per variable) for each of the 3 variables. However, further investigation of the scale is needed; lower or higher values might turn out to be more adequate for a firm diagnosis.

Prognosis of the Disorder

At the time of admission to the hospital following the stroke, patients with contraversive pushing have a more severely impaired level of consciousness and ability to walk, paresis of the upper and lower extremities, and lower initial function in activities of daily living than patients with hemiparesis but without contraversive pushing.² However, in contrast to other neuropsychological deficits such as aphasia or spatial neglect, we found that contraversive pushing is a disorder that can be well compensated for by the brain. Only 6 months after a stroke, pathological pushing behavior is rarely still evident.¹⁹ Pusher syndrome thus has a good prognosis¹⁹ and does not seem to negatively influence the outcome of rehabilitation. However, we also know that patients with contraversive pushing take 3.6 weeks (ie, 63%) longer than patients without pusher syndrome to reach the same functional outcome level.² Thus, physical therapy for contraversive pushing should aim to shorten this period. Patients with contraversive pushing should become independent of help from other people in less time and should be discharged from inpatient care earlier.

Suggestion for a New Strategy for Treating Pusher Syndrome

From the recent finding that patients with contraversive pushing have impaired perception of the body’s orien-

tation in relation to gravity,¹³ it follows that pathological pushing should not be treated in a horizontal position, but in an earth-vertical position (ie, while the patient is sitting, standing, or walking). Moreover, because perception of the visual surround turned out to be unimpaired in patients with contraversive pushing,¹³ they can see that they are not in an erect position by looking at their structured surroundings (Fig. 2b). The patients appear to be unable, however, to spontaneously make use of this preserved ability; they have to be trained to do so.

Because the patients *feel* erect when they *see* that they are tilted, and vice versa,¹³ we believe the first goal of physical therapy should be to demonstrate this, showing the patients that visual information corresponds to reality. While sitting or standing, the patients should be asked to *see* whether they are oriented upright. We also provided an experience that showed patients that it is beneficial to use visual aids (eg, the therapist’s arm as shown in Fig. 3) to give patients feedback about their body orientation. It is our observation that the experience of not falling after attaining the corrected position, combined with seeing that they are upright, increases the patients’ confidence and lowers both the presence and the extent of the reaction to abduct and extend the nonparetic extremities to push toward the paretic side.

In our clinical experience, the intervention plan that is most effective is the one that is designed in such a way that the patients learn the following in sequential order:

- Realize the disturbed perception of erect body position.
- Visually explore the surroundings and the body’s relation to the surroundings. Ensure that the patient *sees* whether he or she is oriented upright. We suggest that the physical therapist use visual aids that give feedback about body orientation (eg, the therapist’s arm as shown in Fig. 3) and work in a room containing many vertical structures, such as door frames, windows, pillars, and so on.
- Learn the movements necessary to reach a vertical body position.
- Maintain the vertical body position while performing other activities.

In our day-by-day clinical management of patients with pusher syndrome, we see that this procedure produces successful results. However, research is needed involving controlled studies of this new approach to examine the effects of the intervention and whether it shortens the time for inpatient care and accelerates independence in daily living.

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Appendix.

Clinical Assessment Scale for Contraversive Pushing (SCP)^{13,18}

Examination Form		
Clinical Scale for Contraversive Pushing (SCP)		
Name _____	Date of birth _____	Ward _____
Examination date _____		Diagnosis _____
Physician in charge _____		Physical therapist in charge _____
(A) Spontaneous body posture		
Value 1 = severe contraversive tilt with falling to that side	Sitting <input type="checkbox"/>	Standing <input type="checkbox"/>
Value 0.75 = severe contraversive tilt without falling	<input type="checkbox"/>	<input type="checkbox"/>
Value 0.25 = mild contraversive tilt without falling	<input type="checkbox"/>	<input type="checkbox"/>
Value 0 = inconspicuous	<input type="checkbox"/>	<input type="checkbox"/>
	Sum total (max=2): _____	
(B) Abduction and extension of the nonparetic extremities		
Value 1 = performed spontaneously, already when at rest	<input type="checkbox"/>	<input type="checkbox"/>
Value 0.5 = performed only on changing the position (eg, on transferring from bed to wheelchair)	<input type="checkbox"/>	<input type="checkbox"/>
Value 0 = inconspicuous	<input type="checkbox"/>	<input type="checkbox"/>
	Sum total (max=2): _____	
(C) Resistance to passive correction of tilted posture^a		
Value 1 = resistance occurs	<input type="checkbox"/>	<input type="checkbox"/>
Value 0 = resistance does not occur	<input type="checkbox"/>	<input type="checkbox"/>
	Sum total (max=2): _____	

^aTouch the patient at the sternum and the back. Instruction: “I will move your body sideways. Please permit this movement.”