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New Insights for a Better Understanding of the Pusher Behavior: From Clinical to Neuroimaging Features

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1. Introduction

Disorders of postural balance are common in patients with encephalic lesions. According Tyson et al. (Tyson et al., 2006), around 80% of patients experiencing their first cerebrovascular event have static or dynamic postural imbalance. Historically, the first description of postural balance dysfunction in stroke patients dates back to more than one hundred years ago. In 1909, Beevor described the occasional lack of lateral balance in stroke patients that cause them to fall towards their contralesional side due to their paresis (Beevor, 1909). Later, Brunnstrom reported the 'listing phenomenon' as a list toward the affected side that patients cope by climbing onto something with their nonparetic hand to prevent listing (Brunnstrom, 1970).

In 1968, a tendency to fall towards the lesion side and lateropulsion were described by Bjerver and coworkers in patients with Wallenberg's syndrome due to dorsolateral medullary infarction (Bjerver &Silfverskiold, 1968). These patients also presented with transient ocular tilt reaction and ipsiversive deviations of the subjective vertical, which indicate a pathological shift in the internal representation of the gravitational vector (Dieterich &Brandt, 1992; Brandt &Dieterich, 2000; Dieterich, 2007).

Another postural imbalance observed in patients with encephalic lesions is thalamic astasia. According to Masdeu and Gorelick, this disorder is characterized by the inability to maintain an unsupported upright posture even without paresis or sensory or cerebellar deficits.[8] When asked to sit up, patients with this disorder use the unaffected arm to pull themselves up (Masdeu & Gorelick, 1988). This behavior could be explained in part by a vestibular tone imbalance in the roll plane, especially since skew deviation was included as a feature of the syndrome (Brandt & Dieterich, 2000; Dieterich, 2007).

As opposed to all other syndromes and phenomena described above, the pusher behavior (PB) is characterized by actively pushing away from the nonparetic side (Davies, 1985). Moreover, patients with PB lean to the side opposite the lesion and strongly resist any attempt at passive correction of their tilted body while sitting or standing. In the most severe cases, this resistance occurs even in a supine position (Pedersen et al., 1996; Lafosse et al., 2005). Such patients report a fear of falling towards their ipsilesional side (Davies, 1985; Pedersen et al., 1996; Lafosse et al., 2005) and are not aware that their active pushing is counterproductive and makes it impossible for them to stand without assistance (D'Aquila

et al., 2004). Thus, the listing phenomenon, thalamic astasia and Wallenberg's syndrome need to be considered in the differential diagnosis of PB. Although the PB was originally described in association with neglect and anosognosia as a syndrome that is related to right encephalic lesions by the physical therapist Davies (Davies, 1985), several studies have demonstrated that it can occur in patients with lesions in both hemispheres and is distinct from those neuropsychological deficits (Pedersen et al., 1996; Karnath et al., 2000b, 2000a; Premoselli et al., 2001; Pérrenou, 2002; Bohannon, 2004; Santos-Pontelli et al., 2004). Since the definition of 'syndrome' is "a set of qualities, events or behaviors that is typical of a particular kind of problem' (Longman dictionary of Contemporary English; 1995) and the diagnostic criteria for PB are presence of the 3 behaviors observed by the examiner described above, the term 'pusher syndrome' can be considered appropriate. Alternative labels of the PB are 'contraversive pushing' (Santos-Pontelli et al., 2004; Lafosse et al., 2005; Baccini et al., 2006; Karnath &Brotz, 2007), 'ipsilateral pushing' (Pedersen et al., 1996) and 'lateropulsion' (Babyar et al., 2009). D'Aquila et al (2004) (D'Aquila et al., 2004) referred to this behavior as being synonymous with the 'listing phenomenon', but Brunnstrum's first description mentions neither the behavior of active pushing away from the nonparetic side nor the resistance to posture correction (Brunnstrom, 1970).

Since it was first described in 1985 (Davies, 1985) , interest in PB has been increasing. The aim of this review is to summarize and critically discuss several aspects of this intriguing disorder that are described in the literature.

2. Assessment

The assessment of PB has been conducted either by clinical examination according the recommendations of the physiotherapist Davies (Pedersen et al., 1996; Lafosse et al., 2005; Baccini et al., 2006) or by ordinal scales (Babyar et al., 2009). According to the systematic review by Babyar and coworkers, there are three appropriate clinical examination scales for evaluation of PB (Babyar et al., 2009): the Scale for Contraversive Pushing (SCP), the Modified Scale for Contraversive Pushing (mSCP) and the Burke Lateropulsion Scale (BLS).

Based on the Davies' criteria, Karnath et al. (Karnath et al., 2001) created the SCP that assesses three distinct aspects of postural control: A) symmetry of spontaneous posture while sitting and standing, B) the use of the ipsilesional extremities to abduct and extend the area of physical contact with the surface (arm/hand on mattress; leg/foot on floor) while sitting and standing, and C) resistance to passive correction of posture while sitting and standing. The authors made the diagnosis of PB if all three criteria were present, reaching a total score of at least 1 in each criterion (sitting plus standing in the three situations).

By analyzing the clinimetric properties of the SCP, Baccini et al. (Baccini et al., 2006) found that a cutoff score of greater than 0 in each SCP section might be more appropriate for studies aimed at investigating the prevalence of the PB or its association with other features, such as presence of neglect because this cutoff enhanced the specificity without any decrease in sensitivity or the predictive value of a negative test. Nevertheless, this cutoff criterion requires further investigation in an unselected group of acute neurologically injured patients (Karnath &Brotz, 2007). Since the original cutoff score suggested by Karnath et al. has no false-positive diagnoses (Baccini et al., 2006), it should be better used for pathophysiological studies or investigations about the neural substrates involved with the PB.

Recently, more specific instructions of the SCP were published (Karnath &Brotz, 2007). The use of the nonparetic extremities to bring about the pathological lateral tilt of the body axis

was called 'variable B', and its standing assessment was described as follows: 'The examiner first observes whether the ipsilesional leg is spontaneously (already when rising from the sitting position) abducted and extended. If so, variable B is given the value 1 for standing. If abduction and extension of the nonparetic leg are not spontaneously performed, the examiner asks the patient to start walking. The examiner observes whether the patient now abducts and extends the ipsilesional leg. If so, variable B is given the value 0.5 for standing'. Because the instructions above do not consider the reaction of the arm/hand in standing position and does not include any recommendation for the examiner's performance during the assessment, we suggest the following additional instructions that we found very helpful for the SCP assessment: (1) while the patient is in the standing position, his/her paretic/plegic leg should be supported by using a knee extension split or by the examiner's stabilization (see figure 1); (2) also in the standing position, the examiner should guarantee the presence of a surface next to the patient to observe the behavior of the ipsilesional arm/hand in searching for contact with the surface and achieving extension of the elbow. Another slight but noteworthy detail that should be remembered when assessing the SCP in the sitting position is that patients should be evaluated with plantar support. Nevertheless, an additional bedside tool to detect PB is the investigation of the pusher patients' leg-to-trunk orientation (Johannsen et al., 2006a). When seated upright without contact with the ground, an ipsiversive tilt of the non-paretic leg in relation to the trunk of about 9° is observed. The inclined leg position is maintained throughout the entire tilt cycle. This reaction was not observed in non-brain-damaged subjects, in patients with acute unilateral vestibular dysfunction, or in patients with stroke without PB and vestibular dysfunction (Johannsen et al., 2006a).

The Modified Scale for Contraversive Pushing (mSCP) consists of a composite score that quantifies the PB and includes four functional conditions: (1) static sitting at the bedside with the feet on the floor; (2) static standing with a fully erect posture; (3) transferring from the bed to a chair or wheelchair (with armrests) while maintaining hip flexion; (4) transferring from the bed to a chair or wheelchair by coming to a full standing position and stepping or pivoting 90 degrees (Lagerqvist &Skargren, 2006). Each part is scored separately and the degree of pushing is evaluated on a scale from 0 to 2 points, where 0 indicates no symptoms, and 2 indicates very severe symptoms. The highest possible score is 8 and the recommended diagnostic cutoff score is 3 (Lagerqvist &Skargren, 2006). As suggested by Baccini et al. (Baccini et al., 2006), this modified version is so different from the original SCP that it should be considered a different instrument. Adding transfers and using specific scoring criteria may help examiners of patients whose PB tends to manifest with dynamic balance activities. The concurrent validity of the mSCP with Berg Balance Scale and Swedish Physiotherapy Clinical Outcome was low to moderate, and the inter-rater reliability was moderate to good. Although the mSCP seems to be practical and more sensitive for small changes in the PB's status, further studies are needed because the sample size of its only clinimetric properties' study was small, and all patients exhibited signs of PB. Moreover, sensitivity/specificity data, internal consistency and responsiveness are not available for this scale (Lagerqvist &Skargren, 2006).

The Burke Lateropulsion Scale (BLS) was first developed in 1993 and revised several times by the physiotherapist team of the Burke Rehabilitation Hospital (D'Aquila et al., 2004). This scale is rated according to the severity of resistance to passive correction of the posture or the presence of PB sensed by the examiner during supine rolling, sitting, standing, transferring and walking (0, 1=mild, 2=moderate, 3=severe). According to the authors, to

test sitting and standing, the patient is passively tilted 30° (15–20° for standing) towards his/her paretic side (contralesional tilt) and then brought back to vertical alignment. Scores are then based on any voluntary or reflex movements noted in trunks, arms or legs according to the angle from true vertical where the resistance starts. For example, the sitting scores are as follows: 0=no resistance; 1=resistance starts at 5° tilt before full vertical; 2=resistance starts at 10° tilt before full vertical; and 3 is scored if they sense true vertical between 30° and 10° (D'Aquila et al., 2004). Total scores range from 0 for those without resistance to a maximum score of 17. Patients scoring 2 or greater are considered to exhibit PB (lateropulsion).

Fig. 1. A patient with left brain damage and severe pusher behavior. Examiner stabilizes the paretic leg of the patient in order to evaluate PB sings in standing position. The absence of this stabilization makes the observation of the characteristics of the disorder significantly difficult. Also, the examiner should guarantee the presence of a surface besides the patient in standing position, in order to observe the behavior of ipsilesional arm/hand activity to search for contact with the surface and of achieving extension of the elbow.

D`Aquila et al. (2004) analyzed the concurrent validity of the BLS with Fugl-Meyer Balance score, Functional Independence Measure and length of rehabilitation stay is moderate and the inter-rater reliability is very high. However, there are no available data about sensitivity, specificity, internal consistency and responsiveness for this scale. According to the authors, one of the weaknesses of the BLS is that the assessments are subjective and can be affected by both patient and therapist comfort and familiarity with the test protocol (D'Aquila et al., 2004). It could be difficult for untrained examiners to interpret the 5 or 10-degree increments from true vertical to determine the resistance to passive correction during functional activities. Nevertheless, this is the only scale that includes PB evaluation during supine rolling and walking.

Another assessment of PB was proposed by Lafosse et al.[11] based on Davies' criteria (Lafosse et al., 2005), including (a) the presence of an asymmetrical posture or the midline of the body towards the hemiplegic side, and (b) the presence of resistance against any attempt at passive correction of any of these postures across the midline of the body towards the 'non-affected' or ipsilesional side. A patient is classified as having PB if both criteria are present. No ordinal scale is specified in this analysis. Further differentiation is used with the help of a 4-point scale that is based on the number of postures (standing, sitting and/or lying) in which contraversive pushing is present as follows: a score of 0 indicated no PB, a score of 1 indicated PB when

standing, a score of 2 indicated PB when standing and sitting and a score of 3 indicated PB when standing, sitting and lying. Measurement of inter-rater reliability revealed a percentage of agreement of 88.4% and a Kendall's coefficient of concordance of 0.83 (Lafosse et al., 2005). According to the authors, this assessment of PB is closely related to the SCP. However, it also has no available data about sensitivity, specificity, internal consistency and responsiveness.

3. Incidence

Among the studies that considered the PB according to Davies' description, the incidence of this disorder ranges from 1.5 % to 63 % of patients with acute encephalic lesions (Table 1) (Pedersen et al., 1996; Danells et al., 2004; Santos-Pontelli et al., 2004; Lafosse et al., 2005; Baccini et al., 2006). Pedersen et al. (Pedersen et al., 1996) found an incidence of 5.3 % of PB in all stroke patients who were admitted in study period and 10.4 % of patients without lower extremity paresis on admission, when early death or early recovery were excluded. Danells et al. (Danells et al., 2004) found a PB incidence of 23% and 63% among 65 stroke patients with moderate to severe hemiparesis depending on the assessment cutoff. We found 1.5 % of pusher patients among all neurological inpatients of an emergency hospital (Santos-Pontelli et al., 2004), and Lafosse et al. (Lafosse et al., 2005) found an incidence of 40 % of left-brain-damaged patients and 52% of right brain damaged patients at a rehabilitation center. More recently, Baccini and coworkers (Baccini et al., 2006) compared the incidence of the PB based on 4 different criteria: 3 different cutoffs of the SCP (SCP > 0; SCP≥ 1,75; SCP ≥ 3) and a clinical examination according to Davies' recommendations that focused on careful observation of patients while lying down, sitting, standing, weight transferring and walking (table 1).

The comparison of the reported frequencies of PB is very complicated due to the differences in the timing of the first post ictal evaluation, inclusion/exclusion criteria, characteristics of the institutions where patients were investigated, etiologies included in the screening and the assessments of PB and their cutoffs.

The post ictal timing of the first identification of PB is an important aspect for incidence analysis. PB may not be observed if the assessment is done in outpatients or after several weeks because of early resolution of the behavior. On the other hand, if the assessment is conducted too early, pusher behavior can appear as a fluctuated symptom. Therefore, the screening of this behavior should be conducted as soon as clinical conditions allow and repeated afterwards during several weeks after the ictus onset.

4. Demographic and clinical characteristics

The comparison of demographic and clinical characteristics between series of pusher patients is complicated not only because of the several selection criteria discussed above but also due to the differences among the designs of the studies. Nevertheless, we summarized some demographic and clinical characteristics that have been published so far (table 2).

Pusher behavior has been found more frequently in older patients (table 2). More recently, Barbieri et al. found a correlation between age and perception of posture in healthy subjects (Barbieri et al., 2009). If the internal model of verticality is less robust in elderly people, it would be possible that this population could be more vulnerable to present PB. Though, the incidence of strokes is much greater in old than in young adults. It remains unclear the influence of the deterioration of postural control related to aging on the development of PB. Moreover, there is no investigation about the occurrence of PB in children with an acute encephalic lesion.

Author (year)	Institution characteristics	Etiologies included for screening	Time of PB evaluation (mean±SD)	Assessment	Cutoff	Incidence
Pedersen et al. (1996)	Stroke Unit (acute care, workup and rehabilitation stages)	Stroke	NA	- Lean towards the hemiplegic side in any position - Resistance of any attempt of correction	PB presence considered if 'pushing were present in any posture'.	5,3%* 10,4%**
Danells et al. (2004)	5 different acute care hospitals	Stroke	8±2 days	SCP	SCP > 0 SCP = 3	63% 23%
Santos-Pontelli et al. (2004)	Neurological Unit of an Emergency Hospital	All acute neurologic diseases	31,7 days (range=8-57 days)	SCP	SCP ≥ 3¥	1,5%
Lafosse et al. (2005)	Rehabilitation Center	Stroke	52,71±39,58	- Lean towards the hemiplegic side - Resistance of any attempt of correction (Plus a 4-point scale)	PB presence considered if both criteria were present.	40-52%
Baccini et al. (2008) [26]	2 Inpatient rehabilitation hospitals	Stroke	<=30	- Clinical examination based on Davies recommendations - SCP	- At least 2 of the authors mentioned singns were present, with one of them judge as severe - SCP > 0 - SCP≥ 1,75+ - SCP ≥ 3	- 16,2% - 61,9% - 18,1% - 10,5%

SCP= Scale for Contraversive Pushing. NA= not available. SD= Standard Deviation *All stroke patients admitted in study period. **Patients without lower extremity paresis on admission, with early death or early recovery were excluded. ¥ At least one point in each criterion. + More than 0 in each criterion.

Table 1. Dependent factors for the incidence of pusher behavior. PB= Pusher Behavior.

Author (year)	Whole Sample (pusher patients)	Male %	Age (mean±SD)	Right encepha-lic lesion %	Paresis %	Sensory deficit %	Neglect / Anosognosia	Aphasia
Pedersen et al. (1996)	327 (34)	47,1%	75±7,6	52,9%**	NA	NA	40% / 27,3%	47,1%
Karnath et al. (2000)	10 (5)	40%**	73,6±4,56**	100%	100%	100%	100%	0%
Karnath et al. (2000)	46 (23)	60,87%**	71 (39-81)▪ 68 (38-89)# Median (range)	65,2%	100%	80%▪ 62%#	80%▪ 0%#	7%▪ 100%#
Pérennou et al. (2002)	14 (3)	66,67%**	52,67±5,03**	100%**	100%**	100%**	100%	NA
Karnath et al. (2002)	23 (12)	66,6%	68,5 (38-81) Median (range)	75%**	100%	58%	67%▪ 0%#	0%▪ 100%#
Broetz et al. (2004)	8 (8)	100%	63 (51-79) Median (range)	75%	100%	71%	83%▪ 0%#	0%▪ 50%#
Danells et al. (2004)	62 (39)	59%	69 (NA)	59%	82%▲	56%	62%	NA
Santos-Pontelli et al. (2004)	530 (8)	62,5%	65,4±12,32**	75%*	87,5%▲**	50%**	75%**	25%*
Saj et al. (2005)	17 (5)	40%**	69±6,6**	100%	NA	NA	80%	NA
Karnath et al. (2005)	40 (14)	57,14%**	66,1±7,5▪ 63,9±9,7#	64,28%**	100%	89%▪ 80%#	67%▪ 0%#	0%▪ 60%°
Pontelli et al. (2005)	9 (9)	55,50%**	71,8±5,9	55,50%**	100%**	66,6%**	33,30%**	NA
Johannsen et al. (2006)	25 (15)	80%**	70 (41-88) Median (range)	86,6%**	100%	NA	73%	7%
Johannsen et al. (2006)	45 (21)	80,95%**	68±9,4▪ 67,8±8,3#	52,3%**	91%▪ 100%#	73%▪ 90%#	100%▪ 10%#	0%▪ 80%#
Johannsen et al. (2006)	25 (9)	66,67%**	69,7±13	88,88%**	100%	NA	88%	0%
Pérrenou et al (2008)	86 (6)	66,67%**	62,67±11,33**	83,30%**	NA	NA	NA	NA
Babyar et al. (2008)	72 (36)	52,77%**	74,6±9,1▪ 72,5±8,5#	58,33%**	NA	NA	0%▪** 27,77%#**	73,33%▪** 0%**
Honoré et al (2009)	18 (3)	33,3%**	66,3±6,7	100%	NA	NA	100%	NA
Ticini et al. (2009)	19 (9)	66,66%**	67,8±6,1 α 64,5±16,6 β	66,6%**	100%	NA	100%▪** 0%#**	33,3%**

NA= not available. *Data informed by the authors. **Calculated from the data available in the reference. ▪Right brain damaged patients. #Left brain damaged patients. ▲Severe hemiparesis. αThalamic brain damaged patients. βExtra-thalamic brain damaged patients.

Table 2. Overview of demographic and clinical characteristics observed on the first evaluation of pusher patients in published literature.

A possible gender influence on the incidence of PB was initially suggested (Lafosse et al., 2005). Nevertheless, analysis of several studies performed in large samples of neurologic injured patients found no persistent gender predominance (Danells et al., 2004; Santos-Pontelli et al., 2004; Lafosse et al., 2005).

Paresis of the contralesional extremities seems to be more frequent and more severe in pusher patients than in control encephalic lesioned patients (Karnath et al., 2005). On the other hand, severe PB can occur despite mild degree of hemiparesis (Santos-Pontelli et al., 2007). This observation raises an interesting question: is hemiparesis necessary for the development of the pushing behavior? We reported a patient that the resolution of the contraversive pushing did not depend on the resolution of the hemiparesis (Santos-Pontelli et al., 2007). Therefore, it is possible that hemiparesis may be more properly considered a commonly associated symptom of PB rather than an essential component of the syndrome and its damaged graviceptive circuitry. Further studies involving patients with pusher syndrome controlled for the degree of hemiparesis may be necessary to clarify the impact of PB itself on long-term prognosis after neurologic conditions.

5. Postural control

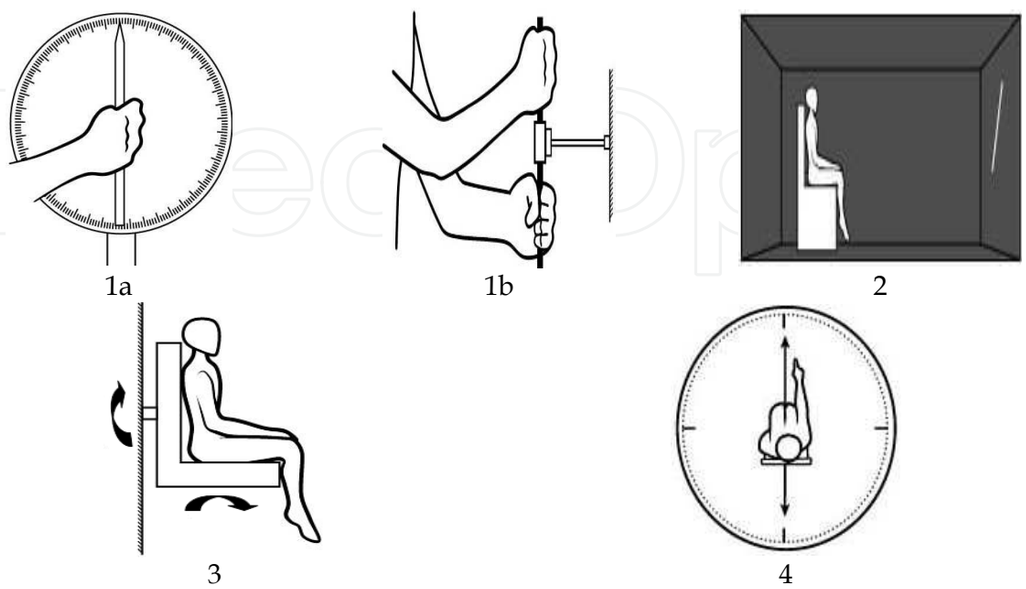
The mechanisms underlying PB have been attributed to a dysfunction of sensory (vertical) perception that leads to a postural reactive behavior (Karnath et al., 2000b; Karnath &Broetz, 2003; Saj et al., 2005; Johannsen et al., 2006c; Perennou, D. A. et al., 2008). These perceptions represent the subjective spatial perceptions, which include the haptic vertical (SHV), visual vertical (SVV), postural vertical (SPV) and the subjective straight ahead (SSA). Figure 2 shows the methodology and sensorial systems involved with these perceptions, and table 3 summarizes the available data about the subjective spatial perceptions of pusher patients published so far.

Karnath et al. found 5 patients with severe PB (SCP=6) who experience their body as being oriented "upright" when it is actually tilted about 18° towards the side of the brain lesion and with no SVV bias (Karnath et al., 2000b). According to the authors, the possible explanation for the PB is that when patients try to move their body to a subjectively 'upright' position, they became laterally unstable because their center of mass was shifted too far to the ipsilesional side and they react to this imbalance by pushing themselves to the contralesional side (Karnath et al., 2000b; Karnath, 2007).

In contrast, Pérrenou et al. recently found a contralesional bias of SPV in 6 pusher patients with an SCP score ranging from 3 to 6. Moreover, all these patients also presented with contralesional tilts in SHV and SVV (Perennou, D. A. et al., 2008). Their hypothesis was that pushing is an implicit active body postural alignment with the perceived vertical. Interestingly, Johannsen et al. demonstrated that patients with PB align their nonparetic leg upright when their trunks are actually tilted to the side opposite to the encephalic lesion (Johannsen et al., 2006a). The authors pointed out that observing the spontaneous posture of the body segments in a seated subject may be a reasonable approach to predict the subject's SPV (Johannsen et al., 2006a). However, future research is needed to verify the correlation between SPV and non-paretic leg orientation in the same sample of pusher patients.

The contradictory findings described above may reflect a difference in the methodology and inclusion/exclusion criteria. Karnath et al. (Karnath et al., 2000b) evaluated the SPV with the

patients' legs hanging freely, while Pérennou et al. used a plantar support (Perennou, D. A. et al., 2008). Additionally, Pérrenou et al. did not screen for neglect. The influence of the presence of plantar support or neglect on the measurement of the SPV is unknown.



1. SHV: determined by manipulation of a wooden or metal rod to the earth-vertical position with the patients' eyes closed: this is essentially driven by proprioceptive afferences (Sharpe, 2003). 1a: with one hand.1b: with two hands.
2. SVV: assessed by the patients' verbal command to adjust a visible line in complete darkness. It depends only on vestibular information with the assistance of the visual cues, independent of the proprioceptors and truncal graviceptors when the subjects are positioned in alignment with Earth vertical (Anastasopoulos et al., 1997; Mittelstaedt, 1998; Trousselard et al., 2004; Lopez et al., 2011).
3. SPV: assessed with subjects seated on a tiltable chair that is capable of rotating in a particular plane and is immobilized by lateral stabilization to prevent postural reactions. The examiner asks the subjects to state, in absence of vision, when they feel their body as vertically oriented (Karnath et al., 2000b; Sharpe, 2003; Perennou, D. A. et al., 2008). The tilting velocity must be 1.5°/s to minimize semicircular canal stimulation (Sadeghi et al., 2007), and acoustic and vibration feedback should also be taken into account. This is determined essentially by interoceptive inputs (Mittelstaedt, 1998; Karnath et al., 2000b).
4. SSA: evaluated by asking the patient to point to the position they perceived as straight ahead and represents an egocentric reference framework (Richard et al., 2004; Saj et al., 2006).

Fig. 2. Methodology description and the sensorial systems involved with SHV, SVV, SPV and SSA.

The SVV (with a haptic component) and the SSA was found to be tilted to the side of the lesion in patients with neglect without PB and tilted to the contralesional side in patients with neglect and PB (Saj et al., 2005; Honore et al., 2009). Nevertheless, the SVV with no haptic influence conducted in a representative sample of pusher patients with and without neglect did not reveal a tilt of this perception[46]. Unfortunately, none of the above studies performed a systematic evaluation of the vestibular system for review see (Eggers & Zee, 2003). Although the dysfunction of the vestibular system is not assumed to be involved with PB (Perennou, D., 2005; Pontelli et al., 2005), its evaluation became imperative to dissociate

vestibular dysfunction from the vertical misperceptions of pusher patients because SVV is essentially driven by this system (Anastasopoulos et al., 1997; Mittelstaedt, 1998; Trousselard et al., 2004). Other aspects to be considered for the evaluation of verticality perception are the learning effect and the number of trials performed. Therefore, in order to state which vertical perception is disturbed in pusher patients, the studies' designs require a meticulous methodology and a large sample of pusher patients. The underlying mechanisms of PB still remain unclear.

Author (year)	Number of patients	Lesion side	Neglect	SVV Mean (SD)	SPV Mean (SD)	SHV Mean (SD)	SSA Mean (SD)
Karnath et al. (2000)	5	RBD	100%	-0,4 º (2,5°)	+17,9 º (4,7°)	NA	NA
Saj et al (2005)	4 1	RBD	100% 0%	+4,8 º (5,1°) +2,2°	NA	NA	NA
Johannsen et al. (2006)	15	13 RBD 2 LBD	73%	-3,2º (4,8°)	NA	NA	NA
Pérennou et al. (2008)	6	5RBD 1LBD	NA	-6,53º (1,86)**	-10,6º (5,85º)**	-7,48º (1,71º)**	NA
Honnoré et al. (2009)	3	RBD	100%	NA	NA	NA	-8,7º (2,4°)

SD: Standard Deviation; RBD: Right Brain Damage; LBD: Left Brain Damage; *(with haptic component)
** Mean and standard deviation calculated from the data available in the reference (Perennou, D. A. et al., 2008).

Table 3. Summarized available data about the subjective perceptions of pusher patients.

6. Prognosis and rehabilitation

There are few studies that address the resolution of PB (Karnath et al., 2002; Broetz et al., 2004; Danells et al., 2004; Santos-Pontelli et al., 2004; Lafosse et al., 2005). Until now, the PB is described as having good prognosis with a maximum recovery time of 6 months (Karnath et al., 2002). Dannels and coworkers showed that the recovery of PB is neither strongly associated with age nor with the recovery of motor control evaluated by Fugl-Meyer motor scale (Danells et al., 2004). However, patients with neglect and those who presented higher initial SCP scores had longer PB recovery times (Danells et al., 2004; Lafosse et al., 2005). Recently, Babyar and coworkers demonstrated that pusher patients following stroke have a lower Functional Independence Measure efficiency and more dependency at discharge when compared with matched controls with equal functional limitations (Babyar et al., 2009). In addition, stroke patients seem to have worse PB prognosis than patients with brain trauma (Santos-Pontelli et al., 2004); this difference in recovery time may be related to etiology, extension, or inherent resolution mechanisms of the causative lesion.

Based on the Bobath concept, Davies described several activities using manual guidance (somesthetic information) to induce the midline body position in the pusher patients (Davies, 1985). Later, Broetz and Karnath suggested a visual feedback approach for PB based on their findings in 5 patients who presented with tilted SPV with unaffected SVV, as discussed above (Broetz et al., 2004; Broetz &Karnath, 2005). According to the authors,

because the orientation perception of visual cues in pusher patients is not impaired, they can be trained to use conscious strategies to realign their body.

However, the contralesional tilts of SPV, SVV and SHV recently described in patients with PB raise the question about the utility of visual feedback treatment in all pusher patients (Pedersen et al., 1996). Some findings with healthy subjects have shown a difference in performance if the learner directs attention toward the effect of the movement (an external focus) instead of to the movement itself (an internal focus) (Wulf et al., 1998). It is possible that in pusher patients with multimodal misperception, we could induce the patient to perceive that their body position is tilted by showing the difference between the effect of the movement using their perceived (wrong) vertical reference and using the (somesthetic or verbal) reference given by the therapist. Broetz and Karnath recommended this demonstration of the ineffective result of the pathological pushing in patients with unimodal misperception (Broetz et al., 2004).

Recently, Shepherd and Carr suggested that the behavioral development may be a natural adaptive response to rehabilitation methods that have the potential to increase the fear of falling and provoke defensive pushing (Shepherd &Carr, 2005). The fact that PB has been identified early after the encephalic lesion argues against this possibility. Additionally, we performed a systematic screening of PB in an acute neurological unit (Santos-Pontelli et al., 2004), and we often identified the PB while the patients were positioned sitting on the edge of the bed for the first time after the onset. Nevertheless, as pointed out by the authors, it is imperative to take the fear of falling into account and to be careful to perform the exercises without evoking fear.

Other general evidence-based methods of intervention are naturally applied for pusher patients because other neurological deficits are present. So far, several studies suggest the following: task-oriented exercises, patients' focus on the actual activity, strength and skill training, specific strategies for spatial neglect (when present), patients' awareness of their deficits, attention to the intensity of skill practice and the extent of cardiovascular stress, proper rehabilitation environment, and the use of a treadmill with and without body weight support [for review see (Carr & Shepherd, 2006)].

A consensus on neurological rehabilitation is that intervention requires specificity and that the postural balance is essential in regaining independence in the activities of daily living. Thus, exercises must be individualized, and the best therapeutic strategy for PB should be chosen based on the vertical misperception of each pusher patient as soon as possible. The absence of controlled trials that investigate the treatment of PB supports the need for further research. Moreover, we should be careful about making statements about the PB based on few samples. Multicenter researches could help PB investigative groups to perform more representative studies in order to clarify all the underlying aspects of this still largely unknown neurological disorder.

7. Neuroimaging analysis

Several brain structures have been associated with PB. In this context, Pedersen et al.(Pedersen et al., 1996) and Santos-Pontelli et al. (Santos-Pontelli et al., 2011) have indicated a wide range of findings from no visible lesion to massive hemispheric lesions on neuroimaging scans in a large sample of PB patients. In these studies, radiologists and neurologists analyzed computed tomography or magnetic resonance imaging in order to determine the type and location of the encephalic lesions. Pedersen at al. determined the size of the stroke by the largest diameter of the lesion (Pedersen et al., 1996).

Nevertheless, the location of lesion more consistently described as related to PB occurrence is the posterior thalamus (Karnath et al., 2000a; Karnath et al., 2005). Besides the usual consideration as a relay structure of vestibular pathway (Deecke et al., 1974; Buttner &Henn, 1976), the posterior thalamus is also assumed to be essentially involved in the control of upright body posture. For the lesion analysis, Karnath et al. (Karnath et al., 2000a) compared patients with PB to patients without PB but comparable demographic and clinical data. Using the Talaraich space, the central area of overlap was defined as those voxels in the MRI template that were lesioned in at least 53% or more of their series (number of PB patients=15). The center of lesion overlap was located in the ventral posterior and lateral posterior of the posterolateral thalamus.

Among 40 patients with thalamic strokes (14 pusher patients and 26 control patients), Karnath et al. (Karnath et al., 2005) found that pusher patients had lesions that typically were caused by thalamic hemorrhage. This observation seems to resemble the fact that thalamic hemorrhages predominantly affect the posterolateral part of the thalamus (Hungerbuhler et al., 1984; Kawahara et al., 1986; Kumral et al., 1995; Chung et al., 1996) and that infarctions are less frequent in the posterior thalamus vs. the anterior and paramedian thalamus (Bogousslavsky et al., 1988; Van der Werf et al., 2000). Nevertheless, their control patients presented more ischemic than hemorrhagic thalamic strokes. Using two standard protocols, the authors carried out MRI or spiral CT imaging that were fit the canonical AC-PC orientation of the MRI scans (Karnath et al., 2005). The boundary of the lesion was delineated directly on the individual MRI for every single transversal slice using MRIcro software (Roden & Brett, 2000). Both the scan and lesion shape were then transferred into stereotaxic space using the spatial normalization algorithm provided by SPM2. The MRIcro software was also used to map the lesion from transversal slices of the T1-template MRI from Montreal Neurologic Institute (MNI) space. The authors used the Talairach Z-coordinates in Talairach space by using the identical or the closest matching transversal slices of each individual. Lesion location in the thalamic stroke patients with and without PB was compared using the subtraction technique (Karnath et al., 2005). The percentage of overlapping lesions of the PB patients after subtraction of controls ranged 20%.

The PB is also observed in patients with brain lesions that spare the thalamus as postcentral gyrus (Johannsen et al., 2006b), internal capsule (Pedersen et al., 1996; Saj et al., 2005), temporal lobe (Pedersen et al., 1996; Johannsen et al., 2006b), supplementary motor area (Reding et al., 1997), superior parietal lobule (Reding et al., 1997), inferior parietal lobule (Johannsen et al., 2006b), globus pallidus (Reding et al., 1997), striatum (Saj et al., 2005), centrum semi-ovalum (Saj et al., 2005), insula (Reding et al., 1997; Johannsen et al., 2006b), isolated cerebellum (Paci &Nannetti, 2005) and isolated anterior cerebral artery territory (Karnath et al., 2008).

By analyzing neuroimaging scans of patients with and without PB with the same methodology of Karnath et al. (Karnath et al., 2005), Johannsen et al. found very small regions for pusher patients when subtracted from matched controls (Johannsen et al., 2006b). In both hemispheres, the lesion of the pusher patients centered at the insular cortex and the postcentral gyrus. However, these areas were identified with the subtraction technique where the percentage of difference between the pusher and control patients neuroimaging scans was not exclusively 100% (ranged from 81 to 100%). Although both were meticulous studies (Karnath et al., 2005; Johannsen et al., 2006b), this analysis does not exclude the same lesion location in control patients.

Recently, Ticini et al. (Ticini et al., 2009), found that the posterior thalamus itself is integral to the occurrence of PB rather than additional malperfusion in distant cortical areas by using

perfusion-weighted imaging (PWI), diffusion-weighted imaging (DWI) and T2-weighted fluid-attenuated inversion-recovery (FLAIR) imaging,. Moreover, they found no damage or malperfusion of the thalamus in patients with PB caused by extra-thalamic lesions. While DWI and FLAIR imaging reveal information about irreversibly damaged neural tissue, PWI allows the identification of structurally intact but not enough to function normally. These interesting findings indicate that the thalamic as well as the extra-thalamic brain structures previously related to the PB contribute to the network controlling upright body posture (Ticini et al., 2009).

Most recently, the relationship between neuroimaging data of stroke and non-stroke PB patients and the severity and prognosis of PB was analyzed (Santos-Pontelli et al., 2011). In order to measure the hemorrhage stroke volume (HSV) in patients with hemorrhagic stroke it was used the ABC/2 method (Zazulia et al., 1999) on CT scans of the acute stroke stage. A positive correlation of the National Institute of Health Stroke Scale (NIHSS) score with HSV in hemorrhagic stroke PB patients was found. In spite of this fact, neither the NIHSS score nor HSV were related with the severity or recovery time of PB. Conversely, previous studies showed that the hemorrhagic volume is highly associated with functional and neurologic deficits (NINDS ICH Workshop Participants, 2005). These data and the fact that the NIHSS score is a good neurologic outcome predictor (Wilde et al, 2010; Wityk et al., 1994; Aslanyan et al., 2004) indicate that the PB evolution and severity may be independent from other neurologic deficits such as those measured by the NIHSS. However, more research is needed to confirm this observation.

The fact that all the pusher patients described in literature had an acute event raises the question that the velocity of lesion´s onset may be essential for PB occurrence. In fact, PB also has been reported in patients with other acute brain lesions other than stroke, but not in patients with chronic neurodegenerative disorders (Santos-Pontelli et al., 2004). These observations may indicate that the related alteration of postural control observed in PB may be a consequence of any acute encephalic lesion that lead to a dysfunction in the neural network which processes the input for vertical perception. Figure 3 and 4 show examples of neuroimaging scans of stroke and non-stroke patients with PB.

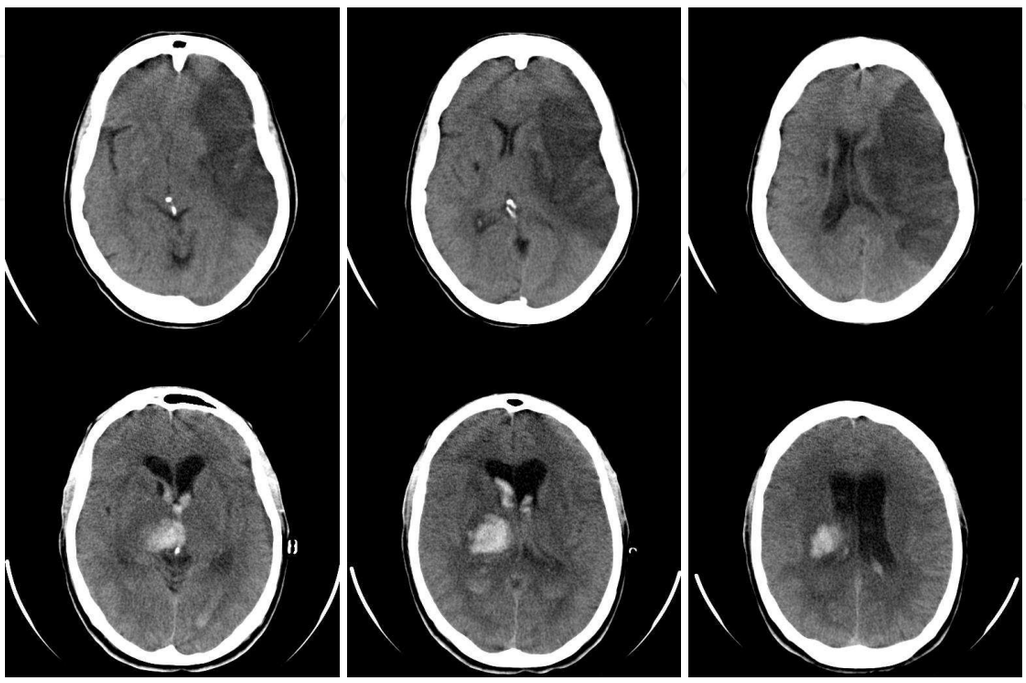
8. Clinical implications of neuroimaging findings

The analysis of the clinical implications of neuroimaging findings requires an important discussion about some limitations of the neuroimaging methods in order to critically interpret the results of the several PB studies.

The localization of human brain functions by studying the correlation between a behavioral disorder and the region of brain lesion has an historical and huge contribution to the understanding of brain function. Nevertheless, as well as all the neuroimaging techniques, the 'lesion method' has some noteworthy limitations.

Roden and Karnath pointed out that the lesion method usually assumes that after a focal lesion, the intact regions of the brain continue to function in the same manner as before the lesion (Roden & Karnath, 2004). However, with tasks controlled by spread and changeable circuits, the brain start to adapt rapidly following the lesion. This rearrangement is helpful for recovery, but makes it difficult to infer the original function of the healthy brain. Also, the design of the brain, its blood supply and the surrounding skull mean that some areas of the brain are injured more often than others what implicate that the locations of brain damage are not randomly distributed in the brain. Roden and Karnath highlighted that this

makes it difficult to interpret lesion overlay plots (Roden & Karnath, 2004). Moreover, if we test patients in the acute stage of their disease, we will not be able to accurately identify all of the brain regions that are damaged. However, if we wait for these initial issues to resolve, the issues associated with brain plasticity will become more evident.

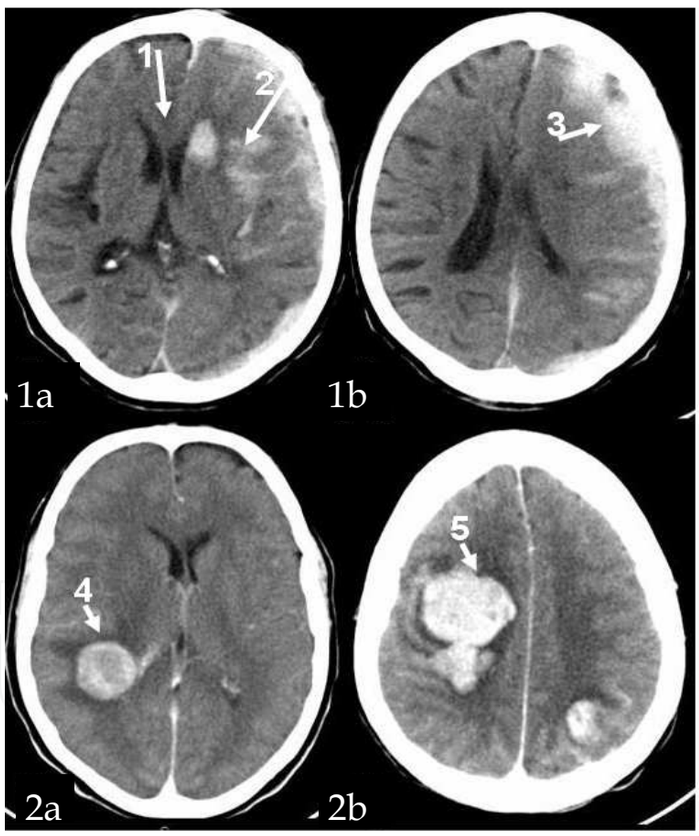


1: Ischemic stroke of the left M1 segment of the middle cerebral artery and significant midline shift (midline shift of the septum pellucidum = 8mm; interthalamic adhesion = 6mm; pineal = 7mm).
2: Right thalamic hemorrhagic stroke with intraventricular hemorrhage and midline shift (midline shift of the septum pellucidum = 2mm; interthalamic adhesion = 5mm; pineal = 9mm).

Fig. 3. CT scans of PB patients in the acute stage.

Although lesion data do not provide the precision of fMRI activation foci, they can tell us which areas are necessary for controlling a cognitive function (Roden & Karnath, 2004). According to Roden and Karnath, simple overlay plots for patients who have a disorder can be inaccurate due to the fact that the regions that they highlight might reflect increased vulnerability of certain regions to injury (as discussed above), rather than any direct involvement with the disorder of interest. A control group of neurological patients who do not exhibit the deficit of interest is, therefore, fundamental for valid anatomical conclusions (Roden & Karnath, 2004). Each technique on its own has only limited explanatory power. However, the strengths and weaknesses of these tools are complementary.
In neuroimaging studies, it is a common practice to spatially normalize subject brains to a standard coordinate system in order to reduce intersubject variability, enable intersubject image averaging, and facilitate the reporting of reduced results in the form of stereotactic coordinates. Numerous registration methods exist, and the two most established are based on the Talairach atlas (Talairach & Tournoux, 1988) and the Montreal Neurological Institute (MNI) templates (Evans et al., 1993; Collins et al., 1994; Laird et al, 2010). The Talairach cannot reflect an excellent representation of the neuroanatomy for the general population atlas because it was created based on the postmortem brain of single subject. In order to

allow better representation of average neuro-anatomy, the MNI created an average brain template based on the MRI scans from several hundred individuals (Evans et al., 1993;Collins et al., 1994). However, the Talairach coordinate system is still the standard reference system used by the neuroimaging community and it is a common practice to report the results in terms of Talairach coordinates even when different brain templates have been used to analyze imaging data. Nevertheless, there is no simple way to transform multiple subject data from the MNI space to the Talairach space. It is actually possible that the coordinate location in MNI space of two subjects would map to different points of Talairach space (Chau & MacIntosh, 2005). The discrepancy becomes a problem when the data are analyzed in the MNI space but the results are reported using the Talairach space (Brett et al., 2002; Chau & MacIntosh, 2005; Laird et al, 2010). Certainly, there is no perfect solution to the conversion problem. According to Laird et al. (Laird et al, 2010), authors should be encouraged to make a clearer distinction between the basic coordinate system as defined by Talairach and Tournoux (1998) and the reference template corresponding to a standard brain that was used during spatial normalization.



1: Scans from a PB patient with traumatic brain injury. Note the left subdural haematoma and mass effect with midline shift and multiple areas of contusion over the left hemisphere.
2: Scans from a PB patient with multiple hemorrhagic metastasis from a pelvis rabdomiosarcoma. The larger lesions were located in the right frontal and parietal lobes causing a mild falx displacement. (from Santos-Pontelli et al., 2005)

Fig. 4. CT scans of patients showing different etiologies for PB.

In this context, the PB neuroimaging studies greatly advanced our understanding of this interesting behavior. Although without a major precision, the qualitative analysis can be hepful to identify a patient that has a tendency to develop PB by the analysis of his/her neuroimage scan., specially in patients with thalamic lesions. In addition, the knowledge that several lesion locations can elicit PB reinforces the concept that this behavior can be accompanied by several neurologic deficits and all the neurologic condition can be critical for the functional prognosis of PB.

As discussed by Roden and Karnath (Roden & Karnath, 2004), the strength of cognitive neuroscience comes from using convergent tools to investigate the same theoretical question. Although there are neuroimaging studies regarding the PB, it remains an issue of future studies to investigate several aspects of PB using brain activation techniques (funcional magnetic resonance, single-photon emission computed tomography, positron emission tomography, magnetoencephalography, event related potential) and transcranial magnetic stimulation techniques in order to better understand this intriguing behavior.

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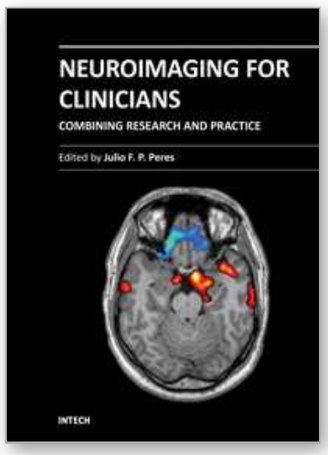

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Neuroimaging for clinicians sourced 19 chapters from some of the world's top brain-imaging researchers and clinicians to provide a timely review of the state of the art in neuroimaging, covering radiology, neurology, psychiatry, psychology, and geriatrics. Contributors from China, Brazil, France, Germany, Italy, Japan, Macedonia, Poland, Spain, South Africa, and the United States of America have collaborated enthusiastically and efficiently to create this reader-friendly but comprehensive work covering the diagnosis, pathophysiology, and effective treatment of several common health conditions, with many explanatory figures, tables and boxes to enhance legibility and make the book clinically useful. Countless hours have gone into writing these chapters, and our profound appreciation is in order for their consistent advice on the use of neuroimaging in diagnostic work-ups for conditions such as acute stroke, cell biology, ciliopathies, cognitive integration, dementia and other amnestic disorders, Post-Traumatic Stress Disorder, and many more

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