Arq Neuropsiquiatr 2007;65(2-B):536-539

PUSHING BEHAVIOR AND HEMIPARESIS

WHICH IS CRITICAL FOR FUNCTIONAL RECOVERY IN PUSHER PATIENTS ?

Case report

Taiza E.G. Santos-Pontelli[1], Octávio M. Pontes-Neto[2], José Fernando Colafêmina[3], Dráulio B. de Araújo[4], Antônio Carlos Santos[5], João P. Leite[6]

ABSTRACT - We report a sequential neuroimaging study in a 48-years-old man with a history of chronic hypertension and lacunar strokes involving the ventral lateral posterior nucleus of the thalamus. The patient developed mild hemiparesis and severe contraversive pushing behavior after an acute hemorrhage affecting the right thalamus. Following standard motor physiotherapy, the pusher behavior completely resolved 3 months after the onset and, at that time, he had a Barthel Index of 85, although mild left hemiparesis was still present. This case report illustrates that pushing behavior itself may be severely incapacitating, may occur with only mild hemiparesis and affected patients may have dramatic functional improvement (Barthel Index 0 to 85) after resolution pushing behavior without recovery of hemiparesis.

KEY WORDS: postural control, pusher syndrome, stroke, thalamic hemorrhage.

Comportamento de empurrar e hemiparesia: qual o déficit crítico para a recuperação funcional nos pacientes com a síndrome do empurrador? Relato de caso

RESUMO - Relatamos o estudo de neuroimagem seqüencial de um homem de 48 anos com história de hipertensão arterial crônica e acidentes vasculares cerebrais (AVCs) lacunares nos núcleos ventral lateral posterior do tálamo. O paciente desenvolveu hemiparesia leve e síndrome do empurrador (SE) grave após AVC hemorrágico no tálamo direito, sendo tratado com fisioterapia motora convencional. Três meses após o *ictus*, os sinais da síndrome haviam desaparecido e o paciente apresentava Índice de Barthel 85, apesar da permanência da hemiparesia leve. Este caso demonstra que a síndrome do empurrador isolada pode ser gravemente incapacitante, pode ocorrer associada a hemiparesia leve e que os pacientes com esta síndrome podem apresentar recuperação funcional importante (Índice de Barthel inicial 0 e final 85) após a resolução da SP sem alteração do grau de hemiparesia.

PALAVRAS-CHAVE: controle postural, síndrome do empurrador, AVC, hemorragia talâmica.

Pusher syndrome is a curious disorder of postural control that may affect patients with hemispheric lesions associated with hemiparesis[1]. Rather than use the unaffected arm to pull themselves up, pusher patients extend this arm and actively push away, toward the paretic side. When sitting or standing, they lean toward the hemiparetic side and resist any attempts of passive correction toward the earth-vertical upright orientation[1]. Initially, this behavior was described in patients with right hemisphere strokes and was associated with spatial neglect and anosognosia. Subsequent studies in affected patients revealed that the pushing behavior can be dissociated from both spatial neglect and anosognosia, and

[1]PhD, Physical Therapist, Department of Neurology - University of São Paulo School of Medicine, Ribeirão Preto SP, Brazil; [2]PhD student, Neurologist, Department of Neurology - University of São Paulo School of Medicine, Ribeirão Preto SP, Brazil; [3]MD, PhD, Professor of the Department of Otorinolaringology - University of São Paulo School of Medicine, Ribeirão Preto SP, Brazil; [4]PhD, Professor of the School of Physics - University of São Paulo, Ribeirão Preto SP, Brazil; [5]MD, PhD, Professor of the Department of Internal Medicine - University of São Paulo School of Medicine, Ribeirão Preto SP, Brazil; [6]MD, PhD, Professor of the Department of Neurology - University of São Paulo School of Medicine, Ribeirão Preto SP, Brazil. João P. Leite, Octávio M. Pontes-Neto, Antônio Carlos Santos, Dráulio B. de Araujo are supported by Fundação de Apoio a Pesquisa do Estado de São Paulo (FAPESP) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq).

Received 2 October 2006, received in final form 29 January 2007. Accepted 5 March 2007.

Dr. João P. Leite - Campus Universitário - USP - 14049-900 Ribeirão Preto SP - Brasil. E-mail: jpleite@fmrp.usp.br

may also be a consequence of left hemisphere lesions[2-8]. Recently, pusher behavior has also been described in non-stroke conditions[8]. In addition, the resolution of symptoms in pusher patients with stroke seem to be longer than in non-stroke etiologies[8]. Patients with contraversive pushing lead to a severe tilt of perceived body verticality in the frontal plane. Although their perception of visual vertical was correct, without visual cues these patients perceive the body as oriented upright when actually tilted 18° to the nonhemiparetic, ipsilesional side[5]. In addition, contraversive pushing does not seem to be related to vestibular problems. It have been demonstrated that the dysfunction of semicircular canals and the otoliths are not associated with the intriguing postural imbalance of the patients with pusher syndrome[5,7,9]. A physical therapy approach for the pushing behavior has been suggested[10-12]. In view of the fact that the patients with pusher syndrome have unimpaired perception of visual surroundings they can be trained to compare their tilted body position to the vertical cues in the room and thus reach a vertical body position[10-12]. The posterolateral thalamus has been identified that the brain structure involved with the postural imbalance of pusher patients, though other cortical areas have also been reported[13-15]. More recently, Karnath et al. observed that paresis of the contralesional extremities was more frequent and more severe in pusher patients than in patients without the disorder[14]. There is a significant relationship between ipsilateral pushing and severity of stroke, but it is still unclear whether the delayed recovery time found in pusher patients is due to a more severe hemiparesis or to pusher behavior itself[2,6].

We report a sequential neuroimaging study of a patient that developed a severe contraversive pushing behavior and only mild hemiparesis after a thalamic hemorrhage. This study was approved by the ethics committee of our institution and the informed consent was provided by the patient.

CASE

A 48-year-old, right handed man presented to the Emergency Unit with sudden onset of left hemiparesis, dysarthria and a tonic-clonic seizure. The patient was a former smoker and had a history of mild alcohol abuse and chronic hypertension. Six years before admission, he developed acute left hemiparesis and mild dysarthria that was marginally investigated. He recovered almost completely in the following months and reassumed daily activities. Two years before admission, he developed a second event characterized by proportionate left hemiparesis. At that time, CT scans revealed mild bilateral hypodensities over the thalamus, external capsule and basal ganglia, compatible with lacunar ischemic strokes. Again, he had progressive recovery with mild motor deficits (Rankin I). After the second event, he was referred to neurological consultation at HCFMRP-USP. The patient was completely independent and oriented. No arrhythmia was found on EKG. Eight months before admission, trans-thoracic echocardiography revealed severe concentric hypertrophy of left ventricular chamber. Carotid ultrasound revealed mild atheromatosis without hemodynamic repercussion. Brain MRI (Fig 1A) six months before admission disclosed multiple foci of hyperintesities on T2-weighted sequences, compatible with bilateral multiple lacunar strokes affecting mainly the ventral posterior lateral thalamic nucleus, and diffuse alterations of deep-seated white matter compatible with microangiopathy. Figure 1C shows T2 weighted MRI two months after the stroke. Figure 1D shows T2 weighted MRI obtained five months after the thalamic hemorrhage. He had been taken anti-hypertensive medication and aspirin. On admission, he was alert, with a Glasgow Coma Scale score (GCS) of 15, a NIH Stroke Scale score of 12 and disproportionate hemiparesis (left upper limb strength grade III and left lower limb strength grade IV) without hemineglect. The CT scan of the brain showed a right thalamic hemorrhage (Fig 1B). One day after admission, due to respiratory infection, he demanded

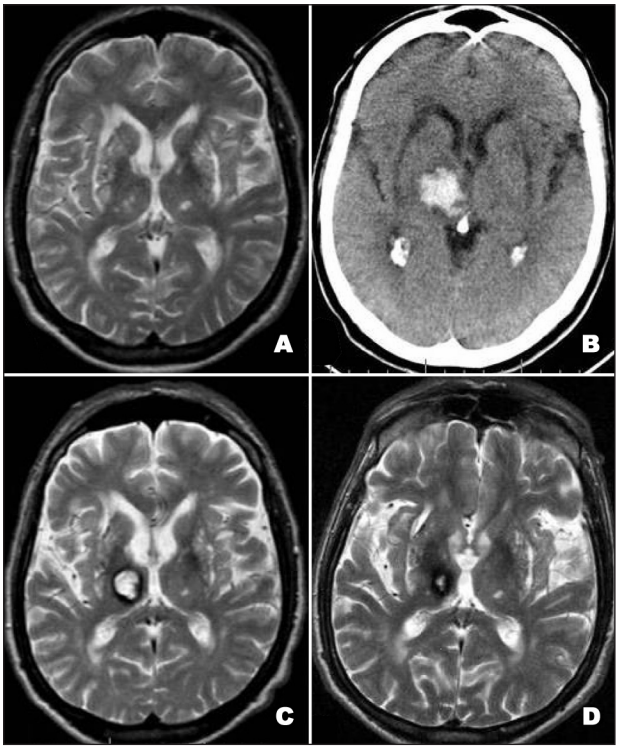

Fig 1. Sequential images (presented in radiological space) of the patient before, during and after pusher behavior. (A) MRI T2-weigheted axial image, obtained six months before the hemorrhagic stroke, with thalamic and putaminal lacunar infarctions bilaterally. (B) Non-enhanced CT image obtained right after thalamic hemorrhage. (C) T2-weighted MRI two months after the stroke. (D) T2-weighted MRI obtained 5 months after the thalamic hemorrhage. The pusher behavior started after the thalamic hemorrhage and disappeared after three months.

orotracheal intubation, ventilatory and intensive care support. At the Intensive Care Unit the patient had standard respiratory and motor physical therapy for 20 to 40 minutes, twice a day, 7 days a week. The motor physical therapy consisted in passive stretch exercises, passive and, as it became possible, assisted active and active motion exercises of trunk, upper and lower limbs.

After 17 days, he was transferred to the neurological unit, when a severe contraversive pushing behavior to the left side was detected using the Scale for Contraversive Pushing[4,10] (SCP=6). This scale assesses three distinct aspects of postural control: 1) symmetry of spontaneous posture while sitting and standing, 2) the use of the arm and/or the leg to extend the area of physical contact to the ground while sitting and standing, and 3) resistance to passive correction of posture while sitting and standing. A patient is scored as having contraversive pushing if all three criteria are present, reaching a total score of at least 1 in each criterion (sitting plus standing in the 3 situations)[4,10]. Activities of daily living (ADL) function was assessed by the Barthel Index (BI), which evaluates 10 different abilities (feeding, transfer, mobility, dressing, stairs, toilet use, bathing, grooming, bladder and bowel status) and ranges a total score from 0 to 100 points[16]. At that time the patient had left upper limb strength grade III, left lower limb strength grade IV and a Barthel Index zero.

The inpatient rehabilitation at the neurological unit consisted in standard motor (kinesiotherapy) and respiratory physiotherapy for 30 to 50 minutes, once a day, 5 days a week. Additional seated balance exercises were included in the motor rehabilitation. Because of the strong pushing behavior the balance exercises were assisted by two physical therapists and consisted in stimulation of trunk posture control. He was discharged from hospital after 18 days with a traqueostomy, a GCS score 15, a BI zero, with grade IV strength on both upper and lower left limbs, and SCP 6.

Due to social and financial problems, he had standard outpatient physiotherapy only once a week besides the additional daily exercises and stimulations that the family was oriented to carry out. The home exercise program included first and foremost seated trunk balance. Assisted stretch and active exercises with upper and lower limbs were also oriented to do at home. Since the pushing behavior was not so strong that he could stand safely with non-specialized assistance, the family was oriented to assist the patient to perform the balance training in the stand position. The indicators of whether the patient actually participated in the home program were obtained by requesting the patient and the family to perform the exercises they do at home and by asking how often they actually could have done.

Only the bowel and bladder status of all the activities of daily living evaluated by the BI recovered before the complete resolution of the contraversive pushing. The pusher behavior completely resolved 3 months after the onset (SCP=0) and, at that time, he had a BI 85, although mild left hemiparesis was still present (grade IV). Since the pusher syndrome was not present and with the same degree of hemiparesis, the activities of daily living items "feeding", "grooming", "toilet use", "bathing" and "transfer" improved and were scored as completely independent. In addition, for dressing he needed help but could do about half unaided and for walking and climbing stairs he also needed the help of one person.

DISCUSSION

The patient reported had severe contraversive pushing evaluated 17 days after the stroke onset. His activities of daily living (ADL) were severely affected while he had the pusher syndrome, reflected by a BI of zero. His ADL function largely improved after the complete resolution of the pushing behavior, despite maintenance of the same degree of hemiparesis. Therefore, BI improvement on this case appears to be exclusively due to resolution of pusher behavior. According to Hsieh et al., there is strong evidence of the predictive value of trunk control on comprehensive ADL function in stroke patients[17]. Unfortunately, there are few data regarding the relationship between ADL function, hemiparesis and the pusher syndrome. Pedersen et al. also reported a severe impairment of the ADL in pusher patients with stroke but they were not able to find any independent influence of their postural imbalance on their percentage of gain in the BI at the end of the rehabilitation[2]. We believe that this lack of influence may be explained by the absence of a meticulous and sequential evaluation of hemiparesis, and by the heterogeneity of their series. Recently, Dannels et al. identified in their series that the recovery of pushing behavior was not 'strongly associated' with the recovery of motor control[6]. Further studies involving patients with pusher syndrome controlled for the degree of hemiparesis may be necessary to clarify the impact of pusher behavior itself on long term prognosis after acute neurologic conditions.

The severity of contraversive pushing in our patient, despite mild degree of hemiparesis, raises another interesting question: is hemiparesis necessary for the development of the pushing behavior? The thalamus is considered a relay center subserving both sensory and motor mechanisms[18]. The lesion or edema on the posterior limb of internal capsule or on the ventral part of the ventral lateral nucleus of the thalamus is likely responsible for the mild motor weakness after thalamic stroke[19]. In contrast, the thalamic structures involved with the pusher syndrome are the ventral posterior and lateral posterior nuclei of the posterolateral thalamus[13,14]. In the present case report, we found that the resolution of the contraversive pushing does not depend on the reso-

lution of the hemiparesis. Therefore, we believe that hemiparesis may be more properly considered a commonly associated symptom of pusher behavior rather than an essential component of the syndrome and its damaged graviceptive circuitry. Although strength is important to postural and motor control, these findings indicate that the focus on hemiparesis specifically for the resolution of contraversive pushing is inadequate.

REFERENCES

1. Davies PM. Steps to follow: a guide to the treatment of adult hemiplegia. New York: Springer, 1985.
2. 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. Arch Phys Med Rehabil 1996;77:25-28.
3. Perennou DA, Amblard B, Leblond C, Pelissier J. Biased postural vertical in humans with hemispheric cerebral lesions. Neurosci Lett 1998;252:75-78.
4. Karnath HO, Brotz D, Gotz A. [Clinical symptoms, origin, and therapy of the "pusher syndrome"]. Nervenarzt 2001;72:86-92.
5. Karnath HO, Ferber S, Dichgans J. The origin of contraversive pushing: evidence for a second graviceptive system in humans. Neurology 2000;55:1298-1304.
6. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke "pushing": natural history and relationship to motor and functional recovery. Stroke 2004;35:2873-2878.
7. Pontelli TE, Pontes-Neto OM, Colafemina JF, Araujo DB, Santos AC, Leite JP. Posture control in pusher syndrome: influence of lateral semicircular canals. Rev Bras Otorrinolaringol (Engl Ed) 2005;71:448-452.
8. Santos-Pontelli TE, Pontes-Neto OM, Colafemina JF, Araujo DB, Santos AC, Leite JP. Contraversive pushing in non-stroke patients. J Neurol 2004;251:1324-1328.
9. Johannsen L, Fruhmann Berger M, Karnath HO. Subjective visual vertical (SVV) determined in a representative sample of 15 patients with pusher syndrome. J Neurol 2006; 253: 1367-1369.
10. Karnath HO, Broetz D. Understanding and treating "pusher syndrome". Phys Ther 2003;83:1119-1125.
11. Broetz D, Johannsen L, Karnath HO. Time course of 'pusher syndrome' under visual feedback treatment. Physiother Res Int 2004;9:138-143.
12. Broetz D, Karnath HO. New aspects for the physiotherapy of pushing behaviour. NeuroRehabilitation 2005;20:133-138.
13. Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. Proc Natl Acad Sci U S A 2000;97:13931-13936.
14. Karnath HO, Johannsen L, Broetz D, Kuker W. Posterior thalamic hemorrhage induces "pusher syndrome". Neurology 2005;64:1014-1019.
15. Johannsen L, Broetz D, Naegele T, Karnath HO. "Pusher syndrome" following cortical lesions that spare the thalamus. J Neurol 2006; 253:455-463.
16. Mahoney FI, Barthel DW. Functional evaluation: the Barthel Index. Md State Med J 1965;14:61-65.
17. Hsieh CL, Sheu CF, Hsueh IP, Wang CH. Trunk control as an early predictor of comprehensive activities of daily living function in stroke patients. Stroke 2002;33:2626-2630.
18. Schmahmann JD. Vascular syndromes of the thalamus. Stroke 2003;34:2264-2278.
19. Herrero MT, Barcia C, Navarro JM. Functional anatomy of thalamus and basal ganglia. Childs Nerv Syst 2002;18:386-404.