

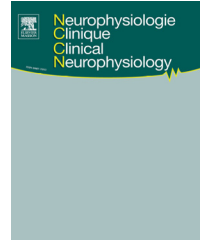


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REVIEW/MISE AU POINT

# Measuring verticality perception after stroke: Why and how?



*Mesurer la perception de la verticale après AVC, pourquoi et comment?*

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Received 12 October 2013; accepted 12 October 2013  
Available online 1 November 2013

## KEYWORDS

Postural vertical;  
Visual vertical;  
Haptical vertical;  
Lateropulsion;  
Pusher syndrome;  
Pushing behavior;  
Internal model;  
Plasticity;  
Neuromodulation;  
Rehabilitation

**Summary** About 80 papers dealing with verticality after stroke have been published in the last 20 years. Here we reviewed the reasons and findings that explain why measuring verticality perception after stroke is interesting. Research on verticality perception after stroke has contributed to improve the knowledge on brain mechanisms, which build up and update a sense of verticality. Preliminary research using modern techniques of brain imaging has shown that the posterior lateral thalamus and the parietal insular cortex are areas of interest for this internal model of verticality. How they interact and are critical remains to be investigated. From a clinical standpoint, it has now been clearly established that biases in verticality perception are frequent after a stroke, causing postural disorders. Measuring the postural vertical with the wheel paradigm has allowed elucidating the mechanisms of lateropulsion, leading or not to a pushing. Schematically, patients with a hemispheric stroke align their erect posture with an erroneous reference of verticality, tilted to the side opposite the lesion. In patients with a brainstem stroke lateropulsion is usually ipsilesional, and results rather from a pathological asymmetry of tone, through vestibulo-spinal mechanisms. These evolutions of concepts and measurement standards of verticality representation should guide the emergence of rehabilitation programs specifically dedicated to the sense of verticality after stroke. Indeed, several pilot studies using appropriate somatosensory stimulation suggest the possibility to recalibrate the internal model of verticality biased by the stroke, and to improve uprightiness. Vestibular stimulations seem to be less relevant and efficient.

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**MOTS CLÉS**

Verticale posturale ;  
 Verticale visuelle ;  
 Verticale haptique ;  
 Latéropulsion ;  
 Modèle interne ;  
 Plasticité ;  
 Rééducation

**Résumé** Environ 80 articles ont été publiés sur la notion de verticalité après l'accident vasculaire cérébral (AVC) au cours de ces 20 dernières années. Ici nous analysons cette littérature en posant les questions suivantes : pourquoi et comment évaluer la perception de la verticale après AVC ? Les recherches sur la perception de la verticale après AVC ont contribué à faire progresser les connaissances sur les mécanismes cérébraux qui sous-tendent la construction et la mise à jour d'un sens de verticalité. Les premières études ayant utilisé les techniques modernes d'analyse de l'imagerie cérébrale ont montré que le thalamus postérolatéral et le cortex pariéto-insulaire sont des zones d'intérêt pour le modèle interne de verticalité. Ces zones sont-elles critiques ? Quelles sont leurs interactions ? Ces questions demeurent ouvertes. D'un point de vue clinique, la grande fréquence des biais de perception de verticalité après AVC hémisphérique est maintenant bien établie. La mesure de la verticale posturale par le *wheel paradigm* a permis d'élucider le mécanisme de la latéropulsion et du comportement *pusher*. Schématiquement les patients avec une lésion hémisphérique alignent leur posture érigée sur une référence de verticalité erronée, inclinée du côté opposé à la latéropulsion. Chez les patients avec AVC du tronc cérébral, la latéropulsion est généralement ipsilésionnelle et résulte plutôt d'une asymétrie de tonus liée à des mécanismes vestibulo-spinaux. Ces évolutions de concept et de mesures de la représentation de la verticale devraient permettre d'émerger et guider des programmes de rééducation spécifiquement dédiés au sens de verticalité après AVC. Plusieurs études pilotes utilisant des stimulations somesthésiques appropriées suggèrent la possibilité de moduler les modèles internes de verticalité biaisés par l'AVC et, ainsi, d'améliorer transitoirement la posture érigée. Les stimulations vestibulaires semblent moins pertinentes et efficaces.

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## Introduction

The medical and scientific interest for verticality perception after stroke has incredibly increased for the last 15 years. This may be explained by at least 2 reasons. Firstly, a link has been shown between dramatic postural disorders and abnormal verticality perception. Secondly, stroke yields a model to analyze how the human brain builds up and updates a sense of verticality, and which neural bases underlie this function. In this paper, we reviewed this literature, by addressing the following questions: why and how measuring verticality perception after stroke?

## Why measuring verticality perception after stroke?

### To explain postural disorders

Postural disorders constitute a primary disability in patients who suffered a stroke, leading to a loss of autonomy and exposing patients to a risk of falling [57]. Although it has long been established that patients' perceptions of verticality can be altered after stroke, the demonstration of a link, presumably causal, between this altered perception of verticality and postural disability is recent [58]. A clinical feature in stroke is that erect sitting and/or standing posture can be compromised by 'lateropulsion', an active lateral tilt of the body (Fig. 1). Lateropulsion is usually ipsilesional in caudal brainstem strokes [19,24,34] and contralesional in rostral brain stem strokes [24,34,72] as well as in hemispheric strokes [13,20,56]. In some hemispheric strokes lateropulsion is associated with "pushing behaviour", which is characterized by patients resisting any attempt to correct their posture [20,31,40,53–55]. Strikingly, some of these patients ("pushers") push themselves away actively from

the non-paralysed side [2,32]. This behaviour is a major challenge for rehabilitation [4,31,54].

There is evidence that vestibular nuclear lesions (as seen in Wallenberg syndrome) or galvanic vestibular stimulations interfere with postural control via direct vestibulo-spinal mechanisms rather than via high-order representational mechanisms. Recent direct evidence of this has been provided by imaging [67]. Indirect evidence is given by verticality perception in subjects submitted to a vestibular stimulation and patients with a lesion of the vestibular nuclei. They usually show a tilt in their visual perception of the vertical (visual vertical [VV]) but not in the tactile perception of the vertical (haptic vertical, HV) or in the postural perception of the vertical (postural vertical [PV]) [17,27,49,50,58].

In contrast, in patients with a hemispheric stroke, lateropulsion responds to an attempt to align the body with an internal vertical reference, which is erroneously perceived to be tilted from true earth vertical [56,58]. In other words, patients with lateropulsion make a postural response in order to control their balance and so actively align their erect posture with a verticality reference tilted to the side opposite the stroke. A left hemiparetic pusher leans leftward. Pérennou et al. [58] designed the wheel paradigm to test PV in patients with severe motor and cognitive deficits. Subjects sit restrained in a drum-like framework facing along the axis of rotation (Fig. 2). They give estimates of their subjective postural vertical (PV) by signaling the point of feeling upright during slow drum rotation, which tilted them rightwards-leftwards, in complete darkness. Abnormal tilts were found in 34 of the 80 consecutive patients with a hemispheric stroke (42%), always contralesional (average  $-8.5 \pm 4.7^\circ$ , by convention a negative sign indicates a contralesional tilt). No ipsilesional tilt of PV was found. There was a continuum between normality, moderate and extreme tilts (Fig. 3), and a strong correlation

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