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NEUROLOGIE & REHABILITATION

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S1 | 2019

Human Perception of Verticality: Lateropulsion & Retropulsion in Neurological Disorders

- Comparing and contrasting clinical outcome measures for pusher behavior
- Development of a clinical scale to assess retropulsion
- Assessment of verticality perception – tools and clinical application
- Multisensory control of posture
- Postural orientation with conflicting visual and graviceptive cues to ‘upright’
- Psychomotor disadaptation syndrome
- The effect of trunk exercises on the perception of verticality after stroke
- Brain imaging studies in pusher behavior
- Recovery from lateropulsion
- Pusher behavior visual feedback training is helpful
- Non-invasive brain stimulation to treat disorders of human verticality
- Robot assisted gait training for pusher behavior

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Dear colleagues,

It gives us the greatest pleasure to **invite you** to join us at the **Annual Conference of RIMS 'Crossing the Interface to Explore New Possibilities'**, to be held in Ljubljana, Slovenia, from **Thursday 20 June to Saturday 22 June 2019**. The Conference is organised in partnership with the **MS Centre Ljubljana**.

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Yours sincerely,
Vincent de Groot, RIMS President

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Ljubljana

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Update Neurorehabilitation 2018

Tagungsband zur Summerschool Neurorehabilitation
Alfried Krupp Wissenschaftskolleg Greifswald

Hippocampus Verlag, Bad Honnef 2018
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Ein Neurorehabilitations-Update auf 228 Seiten. Dicht gepackt und durch zahlreiche Abbildungen aufgelockert – eine gute Gelegenheit, klinische Praxis in der Neurorehabilitation im Überblick und »brandaktuell« kennenzulernen. Ein guter Start für Personen, die noch nicht lange in der Neurorehabilitation tätig sind, und ideal für alle, die ihre Erfahrungen mit dem aktuellen Stand der klinischen Wissenschaft abgleichen wollen. So multiprofessionell, wie die Neurorehabilitation ist, ist das Format der Summer School für alle Berufsgruppen des Neuroreha-Teams geeignet. Die Summer School „Neurorehabilitation" möchte mit einem kompakten Weiterbildungsformat den aktuellen Stand der klinischen Wissenschaft darstellen. Neurorehabilitative Schwerpunkte wie Beatmungsentwöhnung (Weaning), Behandlung schwerer Bewusstseinsstörungen, Dysphagie-Management, Armmotorik, Stehen und Gehen, Behandlung von Spastik, Förderung von Sprache, visueller Wahrnehmung, Kognition und Emotion wurden thematisiert, aber auch allgemeinere Aspekte wie Assessment, Behandlungsziele und Teamarbeit oder neurobiologische Grundlagen der Neurorehabilitation.

Die Summer School Neurorehabilitation ist Teil einer Fortbildungsinitiative der Weltföderation Neurorehabilitation WFNR.

SPECIAL ISSUE

Human Perception of Verticality: Lateropulsion & Retropulsion in Neurological Disorders

Papers presented at the symposium in Bad Aibling on May 09–10, 2019

SHORT REVIEWS & ARTICLES

POSTER PRESENTATIONS

Special Issue

Human Perception of Verticality: Lateropulsion & Retropulsion in Neurological Disorders

An unimpaired perception of verticality is important for human balance control. The perception of verticality is determined by visual, vestibular, and somatosensory input. Consequently, disorders impairing sensory detection, sensory pathways, or the central processing and integration of sensory signals can result in altered postural control. Further, central representation of the 3D spatial environment that uses sensory signals is relevant for orientation in space. For intact perception of upright body orientation, correct processing of somatosensory input seems to play a key role. Disorders of perceived upright body orientation in space, such as lateropulsion and retropulsion, are of high importance for neurorehabilitation. They are a negative predictor for rehabilitation success and limit the rehabilitation effectiveness and efficiency.

Over the last years, there has been increasing interest in disorders of upright body orientation. Most research focused on pusher behavior or lateropulsion, while less attention was paid to retropulsion. Lateropulsion affects body posture in the frontal plane and results in an active shift of the center of mass to either the contralesional or the ipsilesional body side depending on lesion site. By contrast, retropulsion impairs postural control in the sagittal plane leading to a backward shift of the center of mass. Common to both behaviors is that subjects seem to orient their body towards an impaired inner reference of verticality and resist passive correction of this posture. These behaviors hamper the patients' mobilization and augment the risk of falling.

Recent studies helped to gain some insight in the epidemiology, pathophysiology and treatment of disorders of upright body orientation. Nonetheless, there is still a lot to learn. Heterogeneous methods and diagnostic criteria resulted in variable data and hinder the interpretation of results. Consensus on methods and high quality studies are needed to increase the level of evidence.

This special issue of Neurologie & Rehabilitation is focused on novel findings in the field of verticality perception and related disorders. It contains articles, short reviews and poster abstracts presented at the symposium "Human Perception of Verticality: Lateropulsion & Retropulsion in Neurological Disorders". The reader gets an overview on assessments available to diagnose lateropulsion and verticality perception, and a new scale to quantify retropulsion is introduced. Further, it focuses on the role of sensory input for perception of verticality. Two experimental studies are presented investigating the influence of conflicting visual and graviceptive cues to upright body orientation and the effect of trunk exercises on verticality perception. Disorders of upright body orientation build a model to investigate the neuronal structures involved in verticality perception. Neuronal correlates associated with verticality perception and lateropulsion are reviewed in this issue, and the role of impairments in the recovery process of lateropulsion is discussed. Effective treatment of disorders of upright body orientation is essential in order to optimize the rehabilitation process of these patients. In this special issue, three promising therapeutic approaches to treat impaired perception of upright body orientation are presented: visual feedback therapy, non-invasive brain stimulation, and robot-assisted gait training. Finally, poster abstracts provide an overview on recent research and ongoing projects in the field of human verticality perception and related disorders.

Jeannine Bergmann
Klaus Jahn
Carmen Krewer
Friedemann Müller

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Comparing and contrasting clinical outcome measures for pusher behavior

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Pusher behavior (PB) is a challenging postural disorder that can manifest in individuals following stroke [8, 13]. Persons with PB have a disrupted orientation of the body in space and an altered perception of vertical, which causes them to shift their body weight across midline, push with non-paretic extremities towards the impaired side, and often strongly resist attempts from others to passively correct their tilted postural alignment [8, 20]. PB can significantly impair an individual's ability to ambulate and transition between postures [7, 15]. At present, four distinct clinical outcome measures (OMs) have been published that can be used to screen for and assess the degree of PB-related impairment present over time: the Scale for Contraversive Pushing (SCP), the modified Scale for Contraversive Pushing (M-SCP), the Burke Lateropulsion Scale (BLS), and the Four-Point Pusher Score (4PPS) [5, 9, 16, 17]. This article will compare and contrast various aspects of these tools, highlighting their individual advantages and limitations, in order to guide clinicians and researchers in choosing the scale most suitable in their respective setting.

Time of publication and place of origin vary between available tools that assess PB. The SCP appeared first in literature in 2000 while the BLS and M-SCP were respectively published a few years later [9, 12 17]. The 4PPS is the most recently published scale, first appearing in literature in 2019 [5]. Country of origin, scale components, and information about scoring are presented for side-by-side comparison in **Table 1**. Of note, the SCP first appeared in literature in English; however, the original German language version was published later in 2001 [11, 12]. The M-SCP was developed in Sweden; however, the first version of the scale that appears in literature is written in English [17]. Despite being published in a peer-review journal after the SCP, the BLS and 4PPS were initially developed for clinical use much earlier than their first appearance in literature: 11 and 20 years earlier, respectively [5, 9].

As illustrated in **Table 1**, the SCP is organized by postural components of PB (e. g. posture, extension of extremities, resistance) [12]. The BLS and M-SCP are grouped by postures and movements in which PB may manifest (e. g. standing, transfers etc.) [9, 17]. The BLS examines the widest range of functional movements, from supine rolling to walking [9]. The 4PPS groups the three postural components of PB assessed individually by the SCP to yield a single score [5]. A wide range of postures may be assessed with the 4PPS; however, testing

Table 1: Comparison of origin, components, and scoring of clinical tools that assess PB

Scale	SCP	BLS	M-SCP	4PPS
First published	2000 [9]	2004 [7]†	2006 [8]	2019 [10]‡
Country of origin	Germany	United States	Sweden	Australia
Translations (published)	English [9], German [11], Spanish [19], Swedish [17]	English, Spanish [20]	English	English
Components	1. Posture 2. Extension 3. Resistance	1. Supine rolling 2. Sitting 3. Transferring 4. Standing 5. Walking	1. Static sitting 2. Static standing 3. Sitting transfer 4. Standing/walking transfer	Concurrent assessment of posture, use of less affected limbs to push, and resistance to passive correction of posture
Scoring	Each component scored between 0 and 1 in sitting and standing Maximum component score: 2 Maximum score: 6	Each component scored 0–3; Standing sub-section scored 0–4 Maximum score: 17	Each component scored 0–2 Maximum score: 8	Scored 0–3: no pushing to severe pushing Maximum score: 3
Direction of scale	Higher score indicates stronger PB	Higher score indicates stronger PB	Higher score indicates stronger PB	Higher score indicates stronger PB
Cutoff scores indicative of PB	Original suggestion: >1 on each component); More recent suggestion: >0 each component (total score ≥ 1.75) OR >1 each component (total score ≥3), depending on aim of evaluation [12]	Original suggestion: ≥ 2; More recent suggestion: ≥ 3 [18]	Total score ≥3	>0

†Initially developed for clinical use in 1993, ‡Initially developed for clinical use in 1999
PB pusher behavior; **SCP** Scale for Contraversive Pushing; **BLS** Burke Lateropulsion Scale; **M-SCP** modified Scale for Contraversive Pushing; **4PPS** Four Point Pusher Score

Table 2: Psychometric property assessment of clinical tools that assess PB

Scale	SCP	BLS	M-SCP	4PPS
Reported psychometric properties	• construct validity (BI: r = −0.63; FM-B: r = −0.67; LM: r = −0.60) [13] • criterion validity (with expert clinician diagnosis, Cohen κ = 0.93 with > 0 cutoff for each component) [13] • cross-cultural internal consistency (Spanish: Cronbach α = 0.94) [19] • cross-cultural interrater reliability (Swedish: ICC = 0.84; [17] Spanish: ICC = 0.99) [19] • cross-cultural intrarater reliability (Spanish: ICC = 0.99) [19] • internal consistency (Cronbach α = 0.92) [15] • interrater reliability (ICC = 0.97) [15]	• concurrent validity (LOS: r = 0.60; FIM (motor): r = −0.56 to −0.58; FM-B: r = −0.57) [7] • criterion validity (with SCP) [14] • cross-cultural internal consistency (Spanish: Cronbach α = 0.91) [20] • cross-cultural interrater reliability (Spanish: ICC = 0.99) [20] • cross-cultural intrarater reliability (Spanish: ICC = 0.99) [20] • interrater reliability (ICC = 0.93) [7] • intrarater reliability (ICC = 0.94) [7] • responsiveness (SRM = 1.30 to 2.24) [16]	• concurrent validity (BBS: r = −0.49 to −0.52; S-COVS: r = −0.42 to −0.45) [8] • interrater reliability (r = 0.82 to 0.94) [8]	• concurrent validity (BLS: r = 0.95; SCP: r = 0.86; BBS: r = 0.77; CMPCS: r = −0.76; FIM (motor): r = −0.65) [10] • interrater reliability (κw = 0.97) [10] • intrarater reliability (κw = 0.97) [10]

PB Pusher Behavior; **SCP** Scale for Contraversive Pushing; **BLS** Burke Lateropulsion Scale; **M-SCP** modified Scale for Contraversive Pushing; **4PPS** Four-Point Pusher Score; **BI** Barthel Index; **FM-B** Fugl-Meyer Balance subsection; **LM** motor subsection Lindmark motor assessment; **ICC** = intraclass correlation coefficient; **LOS** length of stay; **FIM** Functional Independence Measure; **SRM** standardized response mean; **BBS** Berg Balance Scale; **S-COVS** Swedish Physiotherapy Clinical Outcome Measure; **CMPCS** Chedoke-McMaster Stroke Assessment postural control scale; **κw** weighted kappa

less supported postures (e. g. standing) is not required for persons with clearly evident PB in more supported postures (e. g. sitting) [5].

Given the multitude of postures and positions assessed in each scale and the variance in the manifestation of PB, clear and detailed guidelines for administration are important for assessors. The BLS includes the most detail in its original form. The SCP includes the least detailed guidelines. Instructions for assessors using the SCP were later published in more detailed narrative form [14]. These instructions describe the "Extension" sub-section only and would have been more effectively communicated in the form of a list or user manual in accompaniment with the original scale [12, 14]. The M-SCP has an adequate amount of detail in its original form for most sections, offers a clear definition of pushing, and does not require readers to subjectively rate PB as mild, moderate, or severe, unlike some components of the BLS. However, the standing transfer section of the M-SCP may be misinterpreted, as the item description mentions walking, yet transfers do not typically involve gait in the traditional sense [17]. The 4PPS offers succinct instruction for administration and scoring in its original form. Some wording included in score descriptions would be improved with further explanation, particularly by defining "over-activity" in the less affected side, which may be interpreted differently among scale users [5].

Regarding the psychometric properties which have been assessed thus far (per the 2017 systematic review), the BLS and SCP have been examined to a similar extent while the M-SCP appears in a single study (see **Table 2** for details) [16]. Psychometric properties of the 4PPS were assessed in a single study by Chow and colleagues that was published since the last systematic review [5]. All four scales have been shown to discriminate between those with and without PB. Most scales have moderate construct or concurrent validity when compared to various functional and balance-related measures, with the exception of the M-SCP which has low to moderate concurrent validity [1, 5, 17, 20]. All four scales have demonstrated good to excellent interrater reliability.

At present, two studies have directly compared some of these tools [4, 5]. Bergmann and colleagues directly compared two of these scales and found moderate agreement between the SCP and BLS in the diagnosis of PB [4]. When compared to the SCP, the BLS demonstrates higher sensitivity but lower specificity [4]. Chow and colleagues also compared more than one tool and examined the strength of the associations between scores on the 4PPS with the SCP and BLS [5]. While strong associations were observed between all three measures, the highest agreement about the presence or absence of PB was observed between the 4PPS and BLS [5]. The 4PPS and BLS were broadly consistent in classifying the severity of PB [5] **(Tab. 2)**.

As of 2017, certain psychometric properties had only been assessed in one OM. Internal consistency had only been examined in the SCP [2]. Responsiveness had only been assessed in the BLS [6]. Despite various origins and translations of the scales, the only cross-cultural psychometric property to have been evaluated was the reliability of the Swedish version of the SCP [10]. The fewest psychometric properties had been assessed in the M-SCP. While some studies have been limited by sample size, the methodology of published studies has otherwise been mostly sound [16]. Additional studies related to psychometric properties with larger participants pools were recommended to fill in current knowledge gaps and allow stronger recommendation for use of these instruments [16].

Along with the aforementioned manuscript from Chow and colleagues and to the best of my knowledge, three additional studies were recently published that supplement knowledge related to psychometric properties of PB OMs (details found in **Table 2**). One manuscript from Bergmann and colleagues suggests a higher BLS cutoff score of >3 to diagnose PB, after better correlations with balance impairment and verticality percep-

Table 3: Advantages and limitations associated with clinical tools that assess PB

Scale	SCP	M-SCP	BLS	4PPS
Advantages	• Most cross-cultural psychometric properties assessed in literature • Excellent interrater reliability and internal consistency • Moderate to good construct validity	• Includes clear definition of pushing • Good interrater reliability	• Includes the widest range of functional postures and movements • Includes bidirectional task assessment (e.g. transfers towards and away from hemiparetic side) • Item instructions include "expected hemiplegic response" to guide assessors • Detailed instructions for each component included in original scale form • Developed in clinical setting and adapted over time, prior to validation • Excellent reliability • Responsive to change	• Testing not required in all positions to score • Short time to administer (approximately 2 minutes) • Developed and used clinically nearly 20 years prior to validation • Succinct criteria for hierarchical grading of PB • Good to excellent agreement with the SCP and BLS, respectively
Limitations	• Least detailed instructions included in original scale; later published instructions still lack clarity • English-translated instruction for patients is not colloquial • Resistance section does not have graded scoring, potentially reducing its potential to rate severity of PB or be responsive (responsiveness not yet formally assessed)	• Single published study that examined scale psychometric properties • Low to moderate concurrent validity with balance and functional measures • Some of the instructions lack clarity (e.g. standing transfer section) • Construct validity not yet assessed	• Delineation between mild, moderate, and strong resistance is not explicit for scoring • Rating of resistance and tilt angle may require experienced users to avoid inconsistent results • Longer time to administer relative to other scales (approximately 10 minutes)	• Single published study that examined scale psychometric properties • Responsiveness not yet assessed • Some score description wording lacks clarity (e. g. "over-activity" of unaffected extremities)

PB Pusher Behavior; **SCP** Scale for Contraversive Pushing; **BLS** Burke Lateropulsion Scale; **M-SCP** modified Scale for Contraversive Pushing; **4PPS** Four-Point Pusher Score

tion were observed in the frontal plane [3]. This adjusted cutoff score improves the validity of the scale and the diagnostic agreement between the BLS and SCP (using cutoff of >0 per component) [3]. Future research using the BLS with this more valid cutoff score will yield more clinically meaningful and relevant results [3]. Additionally, two studies were recently published on cross-cultural psychometric properties of Spanish language versions of the SCP and BLS (full-texts only available in Spanish). Martín-Nieto and colleagues assessed cross-cultural validity, reliability, internal consistency, and sensitivity to change in both of these OMs [18, 19]. The Spanish language translations appear to have excellent reliability and internal consistency.

Each of these scales has unique advantages and limitations as displayed in **Table 3**. At present, the BLS appears to have the greatest advantage to limitation ratio relative to the other scales. The BLS in its original published form has detailed instructions, incorporates the most functional positions, and has been deemed reliable and valid in psychometric property assessment in more than one study. However, the BLS lacks detailed description of categorical ratings of PB (e. g. mild, moderate etc.), leaving some subjectivity for assessors, and takes up to ten minutes to administer [6]. Conversely, the 4PPS provides more succinct guidelines for these categorical ratings and takes approximately two minutes to administer [5]. Due to the absence of cross-cultural concerns, the BLS and 4PPS are the most appropriate tools for use in English-speaking locations. The SCP in its original German may make it the suitable tool for use in German-speaking regions; however, the broader array of postures and established responsiveness of the BLS make it the preferred tool to assess change or improvement over time.

The BLS remains the most recommended tool in existence to identify PB following stroke; however, the SCP may be equally suitable depending on the circumstances and expertise of the assessor, given that its psychometric properties have been examined to a similar extent. The 4PPS is most appropriate for clinical use in settings where therapists have limited time and low familiarity with any of the other existing scales. Stronger recommendations for the 4PPS and M-SCP could be made with further psychometric property assessment. Prior to selecting which tool to administer, clinicians and researchers should consider their location, language, familiarity with PB, and setting. A gold standard OM has not yet been established for identifying and quantifying the presence of PB over time.

Literatur

1. Baccini M, Paci M, Nannetti L, Biricolti C, Rinaldi LA. Scale for Contraversive Pushing: Cutoff Scores for Diagnosing "Pusher Behavior" and Construct Validity. Phys Ther 2008; 88(8): 947–55.
2. Baccini M, Paci M, Rinaldi LA. The scale for contraversive pushing: A reliability and validity study. Neurorehabil Neural Repair 2006; 20(4): 468–72.
3. Bergmann J, Krewer C, Müller F, Jahn K. A new cutoff score for the Burke Lateropulsion Scale improves validity in the classification of pusher behavior in subacute stroke patients. Gait Posture 2019; 68: 514–7.
4. Bergmann J, Krewer C, Riess K, Muller F, Koenig E, Jahn K. Inconsistent classification of pusher behaviour in stroke patients: a direct comparison of the Scale for Contraversive Pushing and the Burke Lateropulsion Scale. Clin Rehabil 2014; 28(7): 696–703.
5. Chow E, Parkinson S, Jenkin J et al. Reliability and Validity of the Four-Point Pusher Score: An Assessment Tool for Measuring Lateropulsion and Pusher Behaviour in Adults after Stroke. Physiother Can 2019; 71(1): 34–42.
6. Clark E, Hill KD, Punt TD. Responsiveness of 2 scales to evaluate lateropulsion or pusher syndrome recovery after stroke. Arch Phys Med Rehabil 2012; 93(1): 149–55.

7. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke "Pushing": Natural history and relationship to motor and functional recovery. Stroke 2004; 35(12): 2873–8.
8. Davies PM. Steps to Follow: The Comprehensive Treatment of Patients with Hemiplegia. 1st ed. Berlin, Springer 1985.
9. D'Aquila MA, Smith T, Organ D, Litchman S, Reding M. Validation of a lateropulsion scale for patients recovering from stroke. Clin Rehabil 2004; 18: 102–9.
10. Hallin U, Blomsterwall E, Svantesson U. Clinical assessment scale for contraversive pushing, interrater reliability of a Swedish version. Adv Physiother 2008; 10(4): 173–7.
11. Karnath H-O, Brötz D, Götz A. Klinik, Ursache und Therapie der Pusher-Symptomatik. Nervenarzt 2001; 72: 86–92.
12. Karnath H-O, Ferber; S, Dichgans J. The Origin of Contraversive Pushing Evidence for a Second Graviceptive System in Humans Neurology 2000;55(9): 1298–304.
13. Karnath HO, Broetz D. Understanding and Treating "Pusher Syndrome". Phys Ther 2003; 83(12): 1119–25.
14. Karnath HO, Brötz D. Instructions for the clinical Scale for Contraversive Pushing (SCP) [1]. Neurorehabil Neural Repair 2007; 21(4): 370–1.
15. Karnath HO, Johannsen L, Broetz D, Ferber S, Dichgans J. Prognosis of contraversive pushing. J Neurol 2002; 249(9): 1250–3.
16. Koter R, Regan S, Clark C et al. Clinical outcome measures for lateropulsion poststroke: An updated systematic review. J Neurol Phys Ther 2017; 41(3): 145–55.
17. Lagerqvist J, Skargren E. Pusher syndrome: Reliability, validity and sensitivity to change of a classification instrument. Adv Physiother 2006; 8: 154–60.
18. Martín-Nieto A, Martín-Casas C, Bravo-Llatas C, Moreno-Bermejo MI, Atín-Arratibel MA. Traducción y validación al castellano de la Burke Lateropulsion Scale para la valoración del comportamiento empujador. Rev Neurol 2019; 68(1): 11–7.
19. Martín-Nieto A, Atín-Arratibel M, Bravo-Llatas C, Moreno-Bermejo MI, Martín-Casas P. Spanish translation and validation of the Scale for Contraversive Pushing to measure pusher behaviour. Neurologia 2018. doi: 10.1016/j.nrl.2018.03.019.
20. Pérennou DA, Mazibrada G, Chauvineau V et al. Lateropulsion, pushing and verticality perception in hemisphere stroke: A causal relationship? Brain 2008; 131(9): 2401–13.

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Development of a clinical scale to assess retropulsion in neurological disorders

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Introduction and background

Retropulsion reflects a disturbed postural alignment in the sagittal plane, similar to lateropulsion in the frontal plane. It is characterized by a spontaneous posterior body tilt with the risk of backward falling, active backward pushing with the inability to shift the centre of mass forward, and resistance against passive correction. So far, research on this postural behavior is very limited, even though it seems quite frequent and relevant for neurorehabilitation [9, 14]. Unpublished data of a short survey among 22 therapists at our clinic showed that they are frequently confronted with retropulsion during their work and that the postural behavior hampers the therapy and negatively affects the rehabilitation process. One major problem in the investigation of retropulsion is that there was hitherto no established tool available to rate its severity. As far as we know, among existing scales for postural control, only the Backward Disequilibrium Scale qualitatively evaluates deficient postural control in the sagittal plane [10]. The Backward Disequilibrium Scale assesses the posterior position of the center of mass during five different tasks, but does not assess whether subjects actively shift their center of mass backward and whether they show resistance against passive correction. In addition, the clinimetric properties of the scale are insufficiently investigated and the scale did not make its way into clinical and research practice.

Consequently, an established clinical scale to evaluate retropulsion is urgently needed for clinical work, but also to study the epidemiology, the etiology, and the rehabilitation process of subjects with retropulsion. That's why the objective of this study was to develop a clinical scale to quantify retropulsion.

Methods

A preliminary version of the Scale for Retropulsion (Scale for Retropulsion-V0) has been set up by an interdisciplinary team at the Schoen Clinic Bad Aibling. This scale was further developed in a Delphi study.

Delphi study

The Delphi method was employed to gather opinions from experts and build consensus on the validity of the content of the Scale for Retropulsion [6]. Three rounds were intended for the Delphi study. During each round, the experts were invited to respond to specific questions in an online survey. The online tool LimeSurvey (LimeSurvey GmbH, Hamburg, Germany) was used to design and answer the surveys. The surveys included statements with instructions to indicate the level of agreement or disagreement by using a nine-point Likert scale ranging from strongly disagree (1 point) to strongly agree (9 point), and free space for the experts to explain their rating on the Likert scale and to provide comments.

After each round of the Delphi study, the agreement for each statement was quantitatively analyzed and comments were qualitatively evaluated by blinded investigators. Group results and a summary were reported after every round to the expert panel. In addition, anonymous individual feedback was provided separately to each expert [6].

Positive expert consensus was defined as a median score ≥ 7 and no disagreement (30 % of the experts rated between 1 and 3 and simultaneously 30 % between 7 and 9). Negative expert consensus was described as a median score ≤ 3 and no disagreement.

If an item achieved positive expert consensus it was no longer included in the next Delphi round for agreement evaluation. Minor revisions based on the experts' comments were still possible. The revisions were then included for comments in the next round. Items which have not reached positive consensus were considerably revised according to the experts' suggestions or excluded.

The round 1 survey included three parts. Part one asked about demographic characteristics of the experts and part two about general aspects of retropulsion. The third part focused on the Scale for Retropulsion-V0 and included 40 statements which were rated by the experts on a 9-point Likert Scale. The round 2 survey included 22 statements about the Scale for Retropulsion-V1.1 and the round 3 survey involved only one statement about the general opinion about the Scale for Retropulsion-V1.2.

Participants and recruitment

Inclusion criteria for experts in this study were 1) working in the field of neurorehabilitation and/or geriatrics for at least two years, 2) a minimum of two years of experience with topics related to postural control, and 3) regular contact with subjects who show altered postural control. Potential candidates were identified based on publications about retropulsion, backward disequilibrium, or lateropulsion. They were recruited via an email, inviting them to participate in the online Delphi study. For non-responders, an e-mail reminder was sent after three weeks, followed by another reminder after approximately five weeks. All experts gave written informed consent before the start of the first Delphi round. The experts remained anonymous throughout the Delphi process. Experts who did not answer a survey were excluded from the subsequent Delphi rounds.

Results

Expert panel

Thirteen experts were included in the panel and invited to round 1 of the Delphi study. The flow of the experts through the study is shown in **Figure 1**.

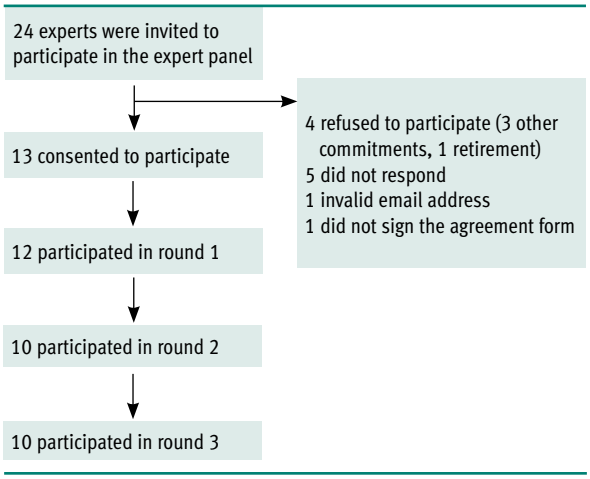

Fig. 1: Flow diagram showing the participation of experts in the Delphi study

The experts included in this study were from France (n = 4), Canada (n = 2), United States (n = 2), Australia (n = 2), Germany (n = 1), United Kingdom (n = 1) and involved different disciplines: medical doctor (3), physiotherapist (5), researcher (human movement science, biomechanics, psychologist) (3), kinesiologist (1). The overall working experience in neurorehabilitation, neurology, or/and geriatrics was on average 22.5 ± 10.0 years. Nine experts indicated experience in neurorehabilitation, 5 in neurology, 7 in geriatrics, 12 in postural control, 7 in falls, and 5 in spatial orientation.

General aspects about retropulsion

The experts rated the relevance of retropulsion high to very high for neurorehabilitation (median 7.5, IQR 2) and geriatrics (median 8, IQR 1.75). Disorders or impairments which were stated to be associated with retropulsion were neurological disorders in general, stroke, cerebellar problems, Parkinson's diseases, geriatric and older subjects, dementia and cognitive disorders, reduced alertness and attentional deficits, prolonged bed rest or long time periods in reclined positions, poor sensory acuity or perceptual deficits, risk of falling, poor balance, and ambulation problems.

While 5 experts indicated familiarity with scales or assessments for retropulsion, this was not the case for 7 experts. The former named the Backward Disequilibrium Scale, the Mini Motor Test, and the Pull test. There was disagreement between the experts about the limitation of existing scales (median 6, IQR 4). Overall, limitations of existing scales seem to be insufficient (evaluation of) clinimetric properties, lack of sensitivity, variability in the methods (pull test), lack of discriminatory power, dependency on the experience of the assessor, and absence of information for the elaboration of rehabilitation programs. There was agreement that a new scale to assess retropulsion is needed (median 7, IQR 3). The experts mentioned the following issues that should be considered in a new scale: passive and active resistance, static postures (sitting and standing) and postural transitions (sit to stand, walking, and turning), weighting of retropulsion, influence of sensory deficits, and awareness.

Delphi process

The preliminary version of the Scale for Retropulsion which has been set up by an interdisciplinary team was based on literature, existing clinical scales (e. g. Burke Lateropulsion Scale [4], Pull test [12], Backward Disequilibrium Scale [10], and the Scale for Contraversive Pushing [8] and clinical experience. The Scale for Retropulsion-Vo comprised three categories (A. Static postural control, B. Reactive postural control, and C. Resistance) and included 11 items.

The three rounds of the Delphi study were conducted over the course of 5.5 months (March 2018 to August 2018). Data collection of each of the four rounds took about four weeks. The flow diagram for the Delphi process is shown in **Figure 2.**

Round 1:
Twelve of thirteen invited experts participated in Round 1 of the Delphi process. Out of the twelve experts, two had not completed the whole questionnaire.

Results of the round 1 survey can be found in **supplement 1**. None of the statements about the Scale for

Retropulsion-Vo showed disagreement. Positive expert consensus was achieved for 37 statements while three statements did not reach consensus (statement 16, 39 a–e, and 40 a–e). Consequently, all items of subtest A "Static postural control" and subtest B "Reactive postural control" had reached positive consensus after round 1, while this was not yet achieved for subtest C "Resistance".

As a result of the experts' comments, all items were slightly revised, even though they had already reached positive consensus. The revisions of these items mainly involved clarifications of the procedures and the instructions, and adjustments of the scoring (4-level scoring for all items). The items which had not yet reached consensus were considerably revised. Based on the experts' comments, also the structure of the scale was revised and a new subtest was introduced which comprises transferring and walking.

In addition, one issue of the general instructions had not reached consensus, namely the suggested procedure if an item cannot be done to a reason other than retropulsion. Based on discussion and literature review a modified solution was proposed and an example was given at the beginning of the scale to clarify the procedure.

Round 2:

Ten of twelve invited experts participated in the round 2 survey. Results of the round 2 survey are shown in **supplement 2**. No statement showed disagreement and all statements reached positive consensus after round 2. Consequently all items of the four subtests showed consensus and no further evaluation of the items was required. Although there was an overall positive consensus, the scale was slightly revised as a result of the experts' comments. The revisions were included for comments in round 3 of the Delphi study. General satisfaction with the scale was high (median 8, IQR 1).

Round 3:

Ten of ten invited experts participated in the round 3 survey. Results of the round 2 survey can be found in **supplement 3**. The general agreement with the Scale for Retropulsion-V1.2 was very high (median 9, IQR 1).

The final version of the Scale for Retropulsion can be found in **Appendix 1**. It consists of four categories: A) static postural control, B) reactive postural control, C) resistance, and D) dynamic postural control. Each subtest is rated in a sitting (starting) position and in a standing (starting). The scale includes in total eight items which are arranged in the order of the body position to be tested (sitting and standing). Each item is scored on a 4-point rating scale (0 no retropulsion to 4 severe retropulsion). The evaluation sheet of the Scale for Retropulsion was designed in a way that the subtests and body positions (sitting and standing) can be scored separately and as a total **(Figure 3)**. The Scale for Retropulsion is designed as a bedside test, i. e. it can be tested in the patient's room and no special equipment was required. It takes about 5 to 15 minutes to complete the scale depending on the impairment level.

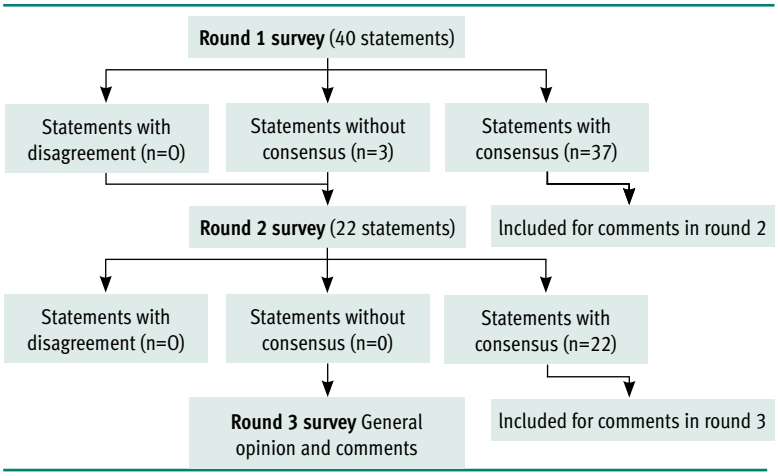

Fig. 2: Flow diagram showing the number and outcomes of statements in each Delphi round

		Sitting	Standing	Subscores
A	Static postural control	1A = _____	2A = _____	A : _____ (Max. 6)
B	Reactive postural control	1B = _____	2B = _____	B : _____ (Max. 6)
C	Resistance	1C = _____	2C = _____	C : _____ (Max. 6)
D	Dynamic postural control	1D = _____	2D = _____	D : _____ (Max. 6)
		Sitting: _____(Max. 12)	Standing: _____(Max. 12)	Total score: _____ (Max. 24)

Fig. 3: Evaluation sheet of the Scale for Retropulsion

Discussion

We developed a clinical Scale for Retropulsion for individuals with neurological disorders that was modified and approved by experts within a Delphi study. The development process which was based on experts' opinion established the content validity of the scale. The level of international and multidisciplinary expert consensus was very high for the final version of the Scale for Retropulsion.

The four components which are evaluated by means of the Scale for Retropulsion in four subtests cover the different characteristics of retropulsion which we see in neurorehabilitation: spontaneous posterior body tilt with the tendency of backward falling, insufficient reactive postural responses, an active backward pushing with resistance against passive correction, and the difficulty to shift the center of mass actively forward [2, 3, 9, 10, 11, 13, 14]. The Scale for Retropulsion assesses sitting and standing, but also posture transition from sitting to standing and walking. We suppose that retropulsion compromises different postures and activities depending on the severity of the behavior, similar to lateropulsion [1].

The Delphi process represents a structured method for gathering opinions and achieving consensus when there is lack of evidence [5]. This Delphi study involved a heterogeneous expert panel from different disciplines and professions. Heterogeneous panels appear to make better quality decisions [7]. However, the rather small number of experts might have limited the stability of the results. When building up the panel, we focused on experts with high expertise in the field of retropulsion and related disorders to keep the quality high. As retropulsion is to date a rather neglected topic, only 24 experts where invited.

So far, research on retropulsion is very limited, amongst others due to the absence of an established tool to assess the behavior. It is suggested that the prevalence of retropulsion is quite high among elderly in an inpatient or outpatient clinical setting [14]. However, up to date there are no data available about the incidence and prevalence of retropulsion as well as about its time course.The Scale for Retropulsion provides clinicians, therapists and researchers with a clinical tool to evaluate and investigate postural instability in the sagittal plane. Clinical evaluation of the scale is now required in order to determine the clinimetric properties of the scale and its implementation in clinical practice and research.

Summary

Retropulsion represents a severe disorder of postural control in the sagittal plane. It is characterized by a posterior displacement of the center of mass with respect to the base of support and active resistance to passive correction of this posture.Although retropulsion seems quite frequent and relevant for neurorehabilitation, research on this postural behavior is limited. One major problem is that there is no established tool available to assess and quantify the behavior. Thus the objective of this Delphi study was to develop a clinical scale to assess retropulsion in neurological disorders. The Delphi method was applied to gather opinions from experts and build consensus on the validity of the content of theScale for Retropulsion. Twelve experts from different countries and disciplines participated in the Delphi study. The Delphi process comprised three rounds. All items of the scale reached consensus in the second round. The final version of the Scale for Retropulsion includes four subtests: A) static postural control, B) reactive postural control, C) resistance, and D) dynamic postural control which are tested in a sitting and a standing (initial) position. High level of international, multidisciplinary overall expert agreement was achieved for the scale (median 9, IQR 1). This bed side test will help clinicians, therapists and researchers to evaluate postural instability in the sagittal plane and to study the epidemiology, the etiology, and the rehabilitation process of patients with retropulsion. The next stage is to evaluate the clinimetric properties of the scale.

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Reference

1. Babyar SR, Peterson MG, Bohannon R, Perennou D, Reding M. Clinical examination tools for lateropulsion or pusher syndrome following stroke: a systematic review of the literature. Clinical rehabilitation 2009; 23(7): 639–50.
2. Barbieri G, Gissot AS, Perennou D. Ageing of the postural vertical. Age 2010; 32(1): 51–60.
3. Cardoen S, Santens P. Posterior pusher syndrome: A report of two cases. Clinical neurology and neurosurgery 2010; 112(4): 347–9.
4. D'Aquila MA, Smith T, Organ D, Lichtman S, Reding M. Validation of a lateropulsion scale for patients recovering from stroke. Clinical rehabilitation 2004; 18(1): 102–9.
5. Hohmann E, Brand JC, Rossi MJ, Lubowitz JH. Expert Opinion Is Necessary: Delphi Panel Methodology Facilitates a Scientific Approach to Consensus. Arthroscopy: the journal of arthroscopic & related surgery: official publication of the Arthroscopy Association of North America and the International Arthroscopy Association 2018; 34(2): 349–51.
6. Jones J, Hunter D. Consensus methods for medical and health services research. Bmj 1995; 311(7001): 376–80.
7. Jorm AF. Using the Delphi expert consensus method in mental health research. Aust N Z J Psychiatry 2015; 49(10): 887–97.
8. Karnath HO, Ferber S, Dichgans J. The origin of contraversive pushing: evidence for a second graviceptive system in humans. Neurology 2000; 55(9): 1298–304.
9. Manckoundia P, Mourey F, Perennou D, Pfitzenmeyer P. Backward disequilibrium in elderly subjects. Clinical interventions in aging 2008; 3(4): 667–72.
10. Manckoundia P, Mourey F, Pfitzenmeyer P, Van Hoecke J, Perennou D. Is backward disequilibrium in the elderly caused by an abnormal perception of verticality? A pilot study. Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology 2007; 118(4): 786–93.
11. Mourey F, Manckoundia P, Martin-Arveux I, Tavernier-Vidal B, Pfitzenmeyer P. Psychomotor Disadaptation Syndrome (PDS) – A new clinical entity in geriatric patients. Geriatrics 2004; 59(5): 20–4.
12. Nonnekes J, Goselink R, Weerdesteyn V, Bloem BR. The retropulsion test: a good evaluation of postural instability in Parkinson's disease? J Parkinsons Dis 2015; 5(1): 43–7.
13. Pfitzenmeyer P, Mourey F, Tavernier B, Camus A. Psychomotor desadaptation syndrome. Archives of gerontology and geriatrics 1999; 28(3): 217–25.
14. Scheets PL, Sahrmann SA, Norton BJ, Stith JS, Crowner BE. What is backward disequilibrium and how do i treat it? A complex patient case study. Journal of neurologic physical therapy: JNPT 2015; 39(2): 119–26.

Conflict of interest:
The authors state that there is no conflict of interest.

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Appendix

Scale for Retropulsion

Note: Although the masculine form is used for better legibility in this document, it always refers to both the female gender and the male gender alike.

General instructions

The Scale for Retropulsion can be tested in the patient's room and does not require any special testing materials. The scale includes four subtests: A) Static postural control, B) Reactive postural control, C) Resistance against passive movement, and D) Dynamic postural control. All subtests are first tested in a sitting (starting) position and thereafter in a standing (starting) position. The items are arranged in the order of the body positions to be tested (sitting and standing).

The scale evaluates postural behavior in the sagittal plane (spontaneous backward body tilt with the risk of backward falling, insufficient reactive postural responses, and active backward pushing with the inability to shift the center of mass forward, as well as resistance against passive correction), but not the patient's general ability to perform a task. This is why it is permitted to assist the patient as much as needed in the completion of the tasks. The assistance can be provided by the examiner and/or another person. The examiner only rates the assistance and the postural behavior in the sagittal plane, and not the assistance needed due to motor weakness, for example. The examiner's position is defined in the description of the individual item (e. g. behind the patient in subtest B, on the paretic/more severely affected side of the body in subtests C and D). However, if this position is not feasible or not safe enough, another position can be chosen. That position is then noted in the evaluation sheet.

Each item can be tested up to two times. No feedback about uprightness or performance should be given to the patient.

The scores are noted in the evaluation sheet which is designed in a way that the subtests and the body positions (sitting and standing) can be scored separately and as a total score.

The scale is not valid if a patient cannot sit despite being given maximal assistance and for any reasons other than retropulsion. In this case, the scale cannot be performed.

If the scale can generally be performed, but testing of individual items is not possible due to reasons other than retropulsion and despite maximum assistance, the examiner marks the corresponding item and notes down the reason in the evaluation sheet.

The item is then scored as follows in the evaluation sheet:

- For an item in the sitting (starting) position (1A–1D): The subtest cannot be scored.
- For an item in the standing (starting) position (2A–2D): The item is scored 0 if the item is 0 in the sitting position in the corresponding subtest (no sign of retropulsion). The examiner gives it a score of 3 if the item is ≥1 in the sitting position in the corresponding subtest.

For example: If static postural control in the sitting position was scored 2 (sitting is possible, but there is an increased tendency to fall backward), but the task cannot be tested in a standing position due to a very pronounced paresis, this item is given a score of 3 in the standing position. If, on the contrary, static postural control in the sitting position was scored 0 (stable, no falling backward), but the task cannot be tested in a standing position for a reason other than retropulsion, the item is given a score of 0 in the standing position.

1. Sitting

Starting position for testing of the following subtests (1A–1D): Patient is sitting on the bed* (about ¾ of the femur should be supported by the sitting surface), the back is unsupported, the feet are resting on the floor, both hands are lying in the thighs, the eyes are open.
The examiner clarifies in advance that the patient has no orthopedic hip problems that could limit the hip flexion between 70° and 120° (e.g. joint prostheses, arthrosis of the hip, lower back pain).

*If a different support surface is used, please indicate here:_____

	Task	Score
1 A	**Static postural control** The patient is asked to sit for 20 seconds. Assistance by one or two persons is allowed. Only postural instability and assistance in the sagittal plane are scored. Instruction: »Please sit for 20 seconds without holding on to anything. I will assist you if necessary.« Scoring: 0 = stable, no backward body tilt and no falling backward 1 = the task can be performed, but with a slight tendency to fall due to a mild and/or intermittent backward body tilt; supervision is needed 2 = the task can be performed, but with an increased tendency to fall due to a moderate and/or constant backward body tilt; supervision and/or intermittent light manual contact is needed 3 = the task cannot be performed due to severe backward body tilt; assistance needed to prevent falling backward	1A* _____ *If 1A=3 then score 1B=3
1 B	**Reactive postural control** The examiner is behind the patient. The examiner gives a sudden firm and quick backward pull to the patient's shoulders (in case of shoulder pain, pull more medially on the shoulder girdle) with sufficient force to cause the patient to lose his balance. For patients who have a pronounced instability, a light pull might be sufficient. The patient is not informed about the direction in which he will be pulled or when. If the patient exhibits severe spontaneous instability in the sitting position (i.e. item 1A=3), this item does not need to be tested and will be scored 3. Instruction: »Please try to keep your balance. If you lose your balance after all, I will catch you before you fall.« Scoring: 0 = normal postural reaction and can immediately stabilize his balance, possibly uses his hands to stabilize 1 = delayed or altered postural reaction, but can stabilize himself independently 2 = inadequate postural reaction, needs assistance to prevent falling 3 = loses balance spontaneously or when pulled only slightly by the shoulders	1B _____
1 C	**Resistance** The examiner sits on the paretic/more severely affected side of the patient and places the hands on the patient's sternum and the upper thoracic spine. If the patient cannot be moved in this position, the examiner can also sit behind the patient (the hands are about the level of the scapula). The examiner slowly tilts the patient's trunk passively backward until it is about 30 degrees off the true vertical. Afterward, the examiner attempts to bring the patient's trunk back into a vertical position (hip flexion to 90°, loadunder the ischial tuberosities) and then forward across the vertical line. The examiner rates the patient's response to this attempt. Instruction: »I will slowly tilt you backward and then forward again. You don't have to do anything. Don't be afraid, I will hold you.« Scoring: 0 = no resistance; it is possible to shift the center of mass to a few degrees forward of the vertical line 1 = resistance is noted, but only when the vertical position is reached or afterwards 2 = resistance is noted before the vertical position is reached 3 = not possible to bring the patient into vertical position due to strong resistance; the center of mass remains shifted backward	1C _____
1D	**Dynamic postural control** The patient is asked to stand up (he may use the hands to push off). The examiner first observes whether the task can be performed without assistance. If this is not possible, the examiner can assist the patient as much as needed. In that case, the examiner stands on the patient's paretic/more severely affected side and places the hands on the patient's sternum and upper thoracic spine, and then shifts the patient's trunk forward. If the patient cannot be sufficiently supported from the side, the examiner can also assist the patient from the front (with hands under the patient's arms at the level of the scapula). If necessary, a second person can provide additional assistance, e.g. if the patient has very pronounced muscle weakness. Instruction: »Please stand up. I will help you as much as needed.« Scoring: 0 = no retropulsion; can sufficiently shift the center of mass forward in order to stand up 1 = can actively shift the center of mass forward with minimal assistance, intermittent manual contact or verbal instructions 2 =assistance is needed to shift the center of mass forward; resistive movements are noticed in the lower extremities or the trunk 3 = maximal assistance from at least one person is needed or the task cannot be performed because of the inability to shift the center of mass in the sagittal plane; resistive movements are noticed in the lower extremities and in the trunk	1D _____

2. Standing

Starting position for testing in the following subtests (2A-2D): Patient is standing with feet comfortably apart and parallel if possible.

	Task	Score
2A	**Static postural control** The patient is asked to stand for 20 seconds. Assistance by one or two persons is allowed. Only postural instability and assistance in the sagittal plane are scored. Instruction: »Please stand for 20 seconds without holding on to anything. I will assist you if necessary.« Scoring: 0 = stable, no backward body tilt and no backward falling 1= the task can be performed, but with a slight tendency to fall due to a mild and/or intermittent backward body tilt; supervision is needed 2= the task can be performed, but with an increased tendency to fall due to a moderate and/or constant backward body tilt; supervision and/or intermittent light manual contact is needed 3= the task cannot be performed due to severe backward body tilt, assistance needed to prevent falling backward	2A _____* *If 2A=3 then score 2B=3
2B	**Reactive postural control** The examiner stands behind the patient. The examiner gives a sudden firm and quick backward pull to the patient's shoulders (in case of shoulder pain, pull more medially on the shoulder girdle) with sufficient force to cause the patient to lose his balance. For patients who have a pronounced instability, a light pull might be sufficient. The patient is not informed about the direction in which he will be pulled or when. If the patient exhibits severe spontaneous instability in the standing position (i. e. item 2A=3), this item does not need to be tested and will be scored 3. Instruction: »Please try to keep your balance. If you lose your balance after all, I will catch you before you fall.« Scoring: 0 = normal postural reaction and can stabilize his balance independently, may take one or two steps or an ankle or hip reaction 1= three or more steps backward, but can stabilize himself independently 2 = inadequate postural reaction, needs assistance to prevent falling 3 = loses balance spontaneously or when pulled only slightly by the shoulders	2B _____
2C	**Resistance** Patient is standing with as much assistance as need. If possible, the entire soles of the feet are on the floor and the upper body is in an upright position. The examiner stands on the patient's paretic/more severely affected side and places the hands on the patient's sternum and the upper thoracic spine. If necessary, a second person can additionally provide assistance, e. g. in case of severe muscle weakness. If the patient cannot be moved in this position, the examiner can also stand behind the patient. The examiner slowly and passively shifts the patient's center of mass backward toward the heels. Afterward the examiner attempts to bring the patient back into a vertical position and forward toward the front of the feet. The examiner rates the patient's response to this attempt. The range of the motion is the length of the sole of the foot. Instruction: »I will slowly tilt you backward and then forward again. You don't have to do anything. Don't be afraid, I will hold you.« Scoring: 0 = no resistance, it is possible to shift the center of mass a few degrees forward of the vertical line and to bring load on to the entire foot (including the front of foot) 1 = resistance is noted, but only when the vertical position is reached or afterward (when the weight is shifted onto the forefoot) 2 = resistance is noted before the vertical position is reached 3 = not possible to bring the patient into a vertical position due to strong resistance; the center of mass remains shifted backward	2C _____
2D	**Dynamic postural control** The patient is asked to walk 4 meters. The examiner first observes whether the task can be performed without assistance. If this is not possible, the examiner can assist the patient as much as needed. In that case the examiner stands on the patient's paretic/more severely affected side and places the hands on the patient's sternum and upper thoracic spine, and then assists the patient in maintaining an upright body position and shifting the center of mass in the sagittal plane. If the patient cannot be sufficiently supported from the side, the examiner can choose different position and/or have a second person provide assistance. Instruction: »Please try to walk up to 4 meters. I will help you as much as needed.« Scoring: 0 = no retropulsion, can independently maintain a forward progression of the center of mass and bring load on to the whole foot 1= can actively maintain a forward progression of the center of mass with minimal assistance, intermittent manual contact or verbal instructions 2= assistance is needed to bring the body in a vertical position and to shift the center of mass forward; slight resistance is noticed 3 = maximal assistance from at least one person is needed or the task cannot be performed because of the inability to bring the body in a vertical position and to shift the center of mass in the sagittal plane; resistive movements are noticed	2D _____

Evaluation sheet

If an item cannot be tested for a reason other than retropulsion, mark it and note down the reason why it was not possible to do the testing.
The scoring of an item that cannot be tested for reasons other than the retropulsion and despite maximum allowed assistance is as follows:

- For an item in the sitting (starting) position (1A-1D): The subtest cannot be scored.
- For an item in the standing (starting) position (2A-2D): The item is scored 0 if the item is 0 in the sitting position in the corresponding subtest (no sign of retropulsion). The examiner gives it a score of 3 if the item is ≥ 1 in the sitting position in the corresponding subtest.

		Sitting	Standing	Subscores
A	Static postural control	1A = _____	2A = _____	A : _____ (Max. 6)
B	Reactive postural control	1B = _____	2B = _____	B : _____ (Max. 6)
C	Resistance	1C = _____	2C = _____	C : _____ (Max. 6)
D	Dynamic postural control	1D = _____	2D = _____	D : _____ (Max. 6)
		Sitting: _____(Max. 12)	Standing: _____(Max. 12)	Total score: _____ (Max. 24)

Please note the reason why an item could not be tested: _____

_____

Documentation of the examiner's position and the form and amount of assistance: _____

_____

Main direction of retropulsion: ❑ posterior ❑ posterior-left ❑ posterior-right

Supplement 1: Results round 1 Delphi study

Scale for Retropulsion – V0		Number of replies	Median	Percentile 25	Percentile 75	Disagreement	Consensus
General aspects							
1.	What is your general opinion of the Scale for Retropulsion-V0 (1=very unsatisfactory, 9=very satisfactory)?	10	7	6	7	No	Yes
2.	The Scale for Retropulsion-V0 includes all clinically important characteristics of retropulsion.	10	7	6	9	No	Yes
3.	The three subscores of the scale (A. static postural control, B. reactive postural control, and C. resistance) are appropriate.	10	9	7	9	No	Yes
4.	The order of the items arranged in the order of the tested body position is appropriate.	10	8	8	9	No	Yes
5.	The structure of the scale is clear.	10	9	8	9	No	Yes
6.	The Scale for Retropulsion-V0 has an appropriate length and duration for testing.	10	8	6	9	No	Yes
7.	The Scale for Retropulsion-V0 suits as a bed side test (difficulty, equipment, space requirements, etc.).	10	8	7	9	No	Yes
8.	The name »Scale for Retropulsion« is appropriate.	10	9	7	9	No	Yes
9.	General instructions of the scale are appropriate.	10	7	4	8	No	Yes
10.	It is appropriate to include a different number of items in the subscores (3 items in A. static postural control and B. reactive postural control, and 5 items in C. resistance).	10	8	8	9	No	Yes
11.	It is appropriate that the scoring of the subscores results in a higher weighting of the resistance subscore (score 0–2 for subscore A. static postural control and B. reactive postural control, and score 0–3 for subscore C. resistance).	10	7	5	8	No	Yes
12.	It is appropriate to score the subscores seperately and not in a total score.	10	8	7	8	No	Yes
13.	It is relevant to document a potential association with lateropulsion or pusher behavior.	10	9	8	9	No	Yes
14.	It is appropriate to give no feedback about body orientation and patient's behavior during the administration of the scale.	10	9	9	9	No	Yes
15.	It is appropriate to perform up to two trials to ensure appropriate scoring.	10	8	8	9	No	Yes
16.	It is appropriate that, if an item cannot be done to a reason other than retropulsion and despite maximum help allowed, the same score should be given as in the previous item of the same subscore.	10	5	4	7	No	No
Subscore A. Static postural control							
17.	It is relevant to include backward falling due to backward body tilt in different static postures in the Scale for Retropulsion.	10	9	8	9	No	Yes
18.	It is appropriate to assess the subscale in the following three body positions: sitting with feet on ground, sitting with feet off ground, and standing.	10	8	7	9	No	Yes
19.	The title of the subscale “static postural control” is appropriate.	10	9	6	9	No	Yes
20.	The scoring of the items (A1, A2, A3) from 0 to 2 with increasing severity is suitable.	10	9	7	9	No	Yes
21.	Item A1 – Static postural control during sitting with feet on the ground						
21.a)	Relevance		10	9	8	9	No
21.b)	Procedure		10	8	7	9	No
21.c)	Description of procedure	10	9	7	9	No	Yes
21.d)	Instruction	10	9	7	9	No	Yes
21.e)	Scoring	10	9	7	9	No	Yes
22.	Item A2 – Static postural control during sitting with feet off the ground						
22. a)	Relevance	10	8	7	9	No	Yes
22. b)	Procedure	10	9	8	9	No	Yes
22. c)	Description of procedure	10	8	7	9	No	Yes
22. d)	Instruction	10	9	8	9	No	Yes
22. e)	Scoring	10	9	8	9	No	Yes
23.	Item A3 - Static postural control during standing						
23. a)	Relevance	10	9	8	9	No	Yes
23. b)	Procedure	10	9	8	9	No	Yes
23. c)	Description of procedure	10	9	8	9	No	Yes
23. d)	Instruction	10	9	8	9	No	Yes
23. c)	Scoring	10	9	8	9	No	Yes
Subscore B – Reactive postural control							
24.	It is relevant to include reactive postural control in the Scale for Retropulsion.	10	9	6	9	No	Yes
25.	It is appropriate to assess the subscale in the following three body positions: sitting with feet on ground, sitting with feet off ground, and standing.	10	7	5	9	No	Yes
26.	The title of the subscale »reactive postural control« is appropriate.	10	9	9	9	No	Yes
27.	The scoring of the items (B1, B2, B3) from 0 to 2 with increasing severity is suitable.	10	9	8	9	No	Yes
28.	Item B1 - Reactive postural control during sitting with feet on the ground						
28. a)	Relevance	10	8	7	9	No	Yes

28. b)	Procedure	10	9	7	9	No	Yes
28. c)	Description of procedure	10	9	7	9	No	Yes
28. d)	Instruction	10	9	7	9	No	Yes
28. e)	Scoring	10	8	5	9	No	Yes
29.	Item B2 - Reactive postural contol during sitting with feet off the ground						
29. a)	Relevance	10	8	7	9	No	Yes
29. b)	Procedure	10	9	7	9	No	Yes
29. c)	Description of procedure	10	9	7	9	No	Yes
29. d)	Instruction	10	9	7	9	No	Yes
29. e)	Scoring	10	8	7	9	No	Yes
30.	Item B3 - Reactive postural control during standing						
30. a)	Relevance	10	9	8	9	No	Yes
30. b)	Procedure	10	9	8	9	No	Yes
30. c)	Description of procedure	10	9	7	9	No	Yes
30. d)	Instruction	10	9	7	9	No	Yes
30. e)	Scoring	10	8	5	9	No	Yes
Subscore C – Resistance							
31.	It is relevant to include resistance to passive correction in the Scale for Retropulsion.	10	9	8	9	No	Yes
32.	It is appropriate to assess the scubscale in the following five body positions/posture transitions: sitting with feet on ground, sitting with feet off ground, standing, transferring, and walking.	10	8	5	9	No	Yes
33.	The title of the subscale "resistance" is appropriate.	10	9	7	9	No	Yes
34.	The scoring of the items (C1, C2, C3, C4, C5) from 0 to 3 with increasing severity is suitable.	10	8	6	9	No	Yes
35.	It is relevant to note the position of the examiner.	10	8	7	9	No	Yes
36.	Item C1- Resistance during sitting with feet on the ground						
36. a)	Relevance	10	9	7	9	No	Yes
36. b)	Procedure	10	9	7	9	No	Yes
36. c)	Description of procedure	10	7	6	9	No	Yes
36. d)	Instruction	10	9	7	9	No	Yes
36. e)	Scoring	10	8	6	9	No	Yes
37.	Item C2 - Resistance during sitting with feet off the ground						
37. a)	Relevance	10	8	7	9	No	Yes
37. b)	Procedure	10	9	7	9	No	Yes
37. c)	Description of procedure	10	7	6	9	No	Yes
37. d)	Instruction	10	9	7	9	No	Yes
37. e)	Scoring	10	8	6	9	No	Yes
38.	Item C3 - Resistance during standing						
38. a)	Relevance	10	9	7	9	No	Yes
38. b)	Procedure	10	8	7	9	No	Yes
38. c)	Description of procedure	10	7	7	8	No	Yes
38. d)	Instruction	10	7	7	9	No	Yes
38. e)	Scoring	10	8	7	9	No	Yes
39.	Item C4 – Resistance during transferring from sitting to standing						
39. a)	Relevance	10	6	5	9	No	Yes
39. b)	Procedure	10	6	5	8	No	No
39. c)	Description of procedure	10	6	5	9	No	No
39. d)	Instruction	10	6	6	9	No	No
39. e)	Scoring	10	7	6	9	No	No
40.	Item C5 – Resistance during walking						
40. a)	Relevance	10	6	6	9	No	No
40. b)	Procedure	10	6	4	8	No	No
40. c)	Description of procedure	10	6	4	8	No	No
40. d)	Instruction	10	6	4	9	No	No
40. d)	40. e) Scoring	10	6	4	9	No	No

Supplement 2: Results round 2 Delphi study

Scale for Retropulsion-V1	Number of replies	Median	Percentile 25	Percentile 75	Disagreement	Consensus
General aspects						
1. General opinion of the Scale for Retropulsion-V1.	10	8	7	8	No	Yes
2. The four subscores of the Scale for Retropulsion-V1 (A) static postural control, B) reactive postural control, C) resistance), and D) dynamic postural control) are appropriate.	10	9	7	9	No	Yes
3. The structure of the scale is clear.	10	9	8	9	No	Yes
4. The order of the items, arranged in the order of these two body positions, is appropriate.	10	9	9	9	No	Yes
5. It is appropriate to assess the subscores only in one sitting position, namely in sitting with feet on the ground.	10	9	8	9	No	Yes
6. The Scale for Retropulsion-V1 has an appropriate length and duration for testing.	10	9	9	9	No	Yes
7. The descriptions of the tasks are clear.	10	8	7	9	No	Yes
8. A video is useful to clarify the implementation of the items.	10	9	9	9	No	Yes
9. It is appropriate to have a 4-level scoring for all items of the scale (i.e. the same weighting for all subscores).	10	9	8	9	No	Yes
10. It is appropriate that the subscores and body position (sitting and standing) can be scored separately and in total.	10	9	9	9	No	Yes
11. The documentation of the most prominent direction of retropulsion is appropriate.	10	8	7	9	No	Yes
12. The evaluation sheet is clear.	10	8	8	9	No	Yes
13. The procedure, if an item cannot be done to a reason other than retropulsion and despite the maximum help allowed						
13. a) ...is appropriate	10	8	7	9	No	Yes
13. b) ...is clear	9	7	6	8	No	Yes
Subscore A) Static postural control						
14. The 4-level scoring is appropriate for thissubscore.	10	9	8	9	No	Yes
Subscore B) Reactive postural control						
16. The 4-level scoring is appropriate for thissubscore.	10	9	7	9	No	Yes
Subscore C) Resistance						
18. It is appropriate to evaluate the subscore "resistance" during sitting and standing.	10	9	8	9	No	Yes
Subscore D) Dynamic postural control						
20. It is relevant to evaluate the inability to shift the COM forward during active functional tasks.	10	8	7	9	No	Yes
21. It is appropriate to assess transferring from sitting to standing and walking in this subscore.	10	8	8	9	No	Yes
22. The title of the subscore "dynamic postural control" is suitable.	10	8	5	9	No	Yes
23. The scoring of the items (D1, D2) from 0 to 3 with increasing severity is appropriate.	10	9	7	9	No	Yes
Item D1						
24. a) The description of the procedure is appropriate.	10	7	7	8	No	Yes
24. b) The instruction to the patients is appropriate.	9	8	7	9	No	Yes
24. c) The scoring is appropriate.	10	8	7	9	No	Yes
Item D2						
25. a) The item walking (ability to maintain a forward progression of the center of mass during walking) is relevant.	10	8	7	8	No	Yes
25. b) The description of the procedure is appropriate.	9	7	7	8	No	Yes
25. c) The instruction to the patients is appropriate.	9	8	7	9	No	Yes
25. d) The scoring is appropriate.	10	8	7	8	No	Yes

Supplement 3: Results round 3 Delphi study

Scale for Retropulsion-V1.1	Number of replies	Median	Percentile 25	Percentile 75	Disagreement	Consensus
Please indicate your general opinion with the Scale for Retropulsion-V1.1.	10	9	8	9	No	Yes

Assessment of verticality perception – tools and clinical application

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Introduction and background

In clinical and research settings, verticality perception is often tested by measuring the subjective visual vertical (SVV), the subjective haptic vertical (SHV), and the subjective postural vertical (SPV) [7, 10, 12–14, 20]. The following article focuses on tools and clinical applications for assessment of SVV. The internal model of verticality is based on bilateral graviceptive vestibular inputs from the otoliths and, most importantly, from the vertical semicircular canals [15]. Accordingly, measurement of SVV can be applied in *acute* unilateral peripheral and central vestibular lesions (chiefly in the brainstem and cerebellum), as well as balance disorders in acute hemispheric lesions (thalamus, temporoparietal cortex) [4, 5, 9, 18]. SVV deviations recover within two to four weeks [11, 24]. Assessment of verticality perception is used for the topographic diagnosis of lesions affecting balance control, documentation of their acuteness and recovery, and guidance of the mode of rehabilitation [8–10]. Several tools and paradigms, which are suitable for testing of verticality perception, have been described [2, 17, 20, 21, 23]. A consensus for standardization is necessary to further establish the method as a psychophysical biomarker and surrogate parameter across centers and in prospective settings [20]. The aim of this article is to summarize the current knowledge of assessment methods for testing of verticality perception and their implication in clinical routine and research.

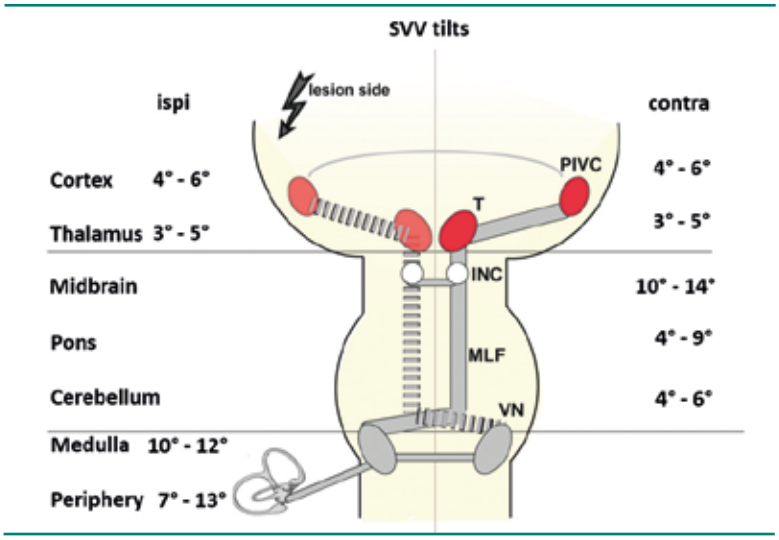

Fig. 1: Vestibular pathways and verticality perception. Schematic graviceptive pathways together with the amount (in deg) of SVV deviation for ipsi- and contralateral lesions depending on the level of acute unilateral vestibular damage. The range of the mean values was calculated on the basis of previously published studies. The five major messages are as follows. (1) In peripheral and pontomedullary brainstem lesions, SVV tilts are ipsilateral. (2) In pontomesencephalic vestibular pathway lesions up to the INC, SVV tilts are contralateral. (3) In cerebellar lesions, SVV tilts are mostly contralesional, especially if the dentate nucleus in involved. (4) In vestibular thalamic and cortical lesions, SVV tilts may be either ipsilateral or contralateral with an intraindividual consistency and an equal distribution interindividually. (5) The amount of SVV tilt is maximal in complete peripheral lesions, followed by tilt in brainstem. INC: interstitial nucleus of Cajal, MLF: medial longitudinal fascicle, PIVC: parieto-insular vestibular cortex, T: thalamus, VN: vestibular nucleus. Adapted from [15].

The physiological basis of perception of verticality

Verticality perception relies on bilateral vestibular graviceptive signals, which are integrated to construct and update an internal model of verticality. SVV testing does not assess the visual contribution to verticality perception, as visual orientation cues are masked. Traditionally, verticality perception is considered to reflect vestibular inputs, which come from the otoliths and vertical semicircular canals, so-called graviceptive inputs [9]. However, based on computational prediction models of SVV adjustments in patients with unilateral vestibular lesions, it was recently proposed that the SVV deviation in the upright position is indeed predominantly caused by the effect of semicircular canal bias on the gravity estimator and not only by a utricular hair cell asymmetry [15]. The direction of SVV tilt (ipsi- versus contralesional) follows the anatomy of the ascending vestibular pathways **(Figure 1)**: peripheral vestibular and central pontomedullary brainstem lesions cause ipsilesional tilts and pontomesencephalic brainstem lesions contralesional tilts of the SVV [5, 12, 22]. In vestibular thalamic and cortical lesions, SVV tilts may be either ipsilateral or contralateral with an intraindividual consistency and an equal distribution interindividually [6, 13, 15]. Cerebellar lesions mostly induce a contralesional tilt of the SVV, especially when the dentate nucleus is involved [3]. The absolute values of SVV deviation are highest in peripher-

al and medullary as well as mesencephalic lesions, and decrease at the level of the thalamus and above. The currently favored hypothesis for this observation is the following: while the gravity direction in the peripheral and brainstem pathways is coded in sensory coordinates, thalamic and cortical neurons show a population coding and a tuning that is distributed over various directions similar to that of the head direction cell system [15].

Tools to assess verticality perception

Several protocols have been proposed for the assessment of verticality perception over the last decades [20]. The most frequently used and best validated paradigms are the following:

- In the *hemispherical dome method* patients look into a semicircular dome of approximately 60 cm diameter with their head fixed on a chin rest **(Figure 2A)**. The surface of the dome extends over the entire visual field and is covered with a random pattern of colored dots, providing no cues to gravitational orientation. A linear target, whose center is fixed on the shaft of a servomotor, is located 30 cm in front of the subject and can be rotated in the subject's frontal plane. After rotation of the target and dome to a randomized offset position, the patients are instructed to align the target with their perceived vertical by using a joystick device. The difference between the adjusted orientation and the true spatial vertical is calculated and averaged over several repetitions. The hemispherical dome method has been validated in various studies on peripheral and central vestibular lesions [9, 11, 12, 20].
- In the *bucket test* patients sit upright looking into a translucent bucket so that their visual field is covered completely by the rim of the bucket. A straight diametric dark line is drawn at the bottom of the bucket on the inside, and, a perpendicular originating from the centerpoint and a degree scale with the zero line adjusted to the dark line inside is placed on the bottom of the bucket on the outside **(Figure 2B)**. For measurement, the bucket is rotated right or left randomly by the examiner to a variable end position and then slowly rotated back to the zero degree position. Patients indicate the position at which they estimate the inside bottom line to be truly vertical by giving a stop signal. Degrees are read off on the outside scale by the examiner. Ten repetitions (clockwise and counterclockwise rotation) are performed and a mean of the deviations is calculated [23].
- In the *rod and frame test* a luminous line has to be orientated vertically in the presence of a static or dynamic visual distractor background **(Figure 2C)**. This test is thought to measure the effect of visual cues on perception of verticality [2, 16].

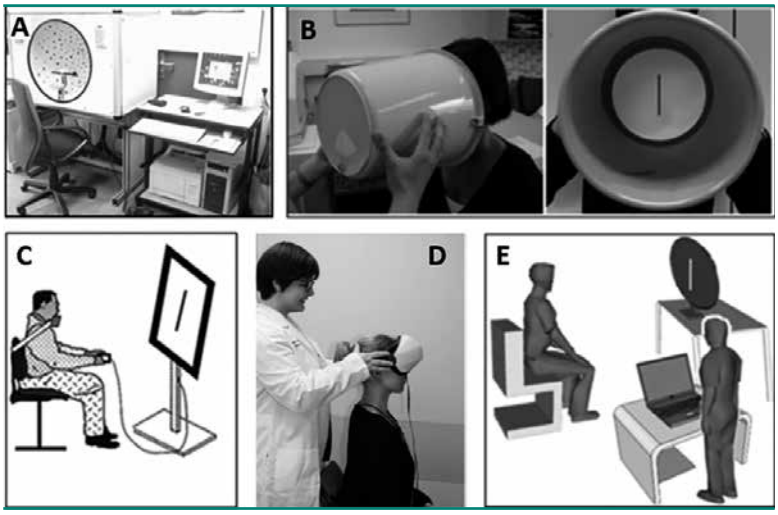

Fig. 2: Devices for assessment of verticality perception. A: hemispheric dome method [23]; B: bucket test [23]; C: rod and frame test [16]; D: virtual SVV system [17]; E: protocol with a luminous line projected on a screen [21]. Adapted from [20].

- The *virtual SVV system* is a commercial computerized system for measuring SVV (Virtual SVV, Interacoustics, Software Version 1.1.0.0). The system consists of light-occluding goggles, which display a luminous bar visible to the participant **(Figure 2D)**, and a wireless controller. The luminous bar appears in the headset tilted to the right or left at random computer-generated angles. The participant adjusts the bar's vertical position using controller buttons until the luminous bar seems to be aligned with true vertical. The subject's head position and the SVV angle relative to true vertical are recorded [16]. Whether this system measures the »graviceptive« SVV or not depends on the distance between the apparatus and the eyes. When the apparatus is too close to the eyes, the cyclorotation of the eyes influences the measurements; this results in tilt angles different from those of the above mentioned techniques [12, 13].
- Various *other protocols*, which use a mechanical luminescent rod or a luminous line projected on a wall by a beamer or presented on a computer screen, were described **(Figure 2E)** [20, 21].
- In visuo-haptic protocols, subjects orientate a metal rod with red diodes fixed on a metal disk to vertical. A potentiometer displays the angle between the true vertical and the perceived vertical. These paradigms test multisensory clues for verticality perception, rather than vestibular inputs.

Only a few studies have compared various methods in terms of the diagnostic validity. The hemispherical dome and bucket method showed a high concordance ($r = 0.89$) [23]. The virtual SVV system measured similar SVV values to the bucket test in zero degree head position, but the test-retest reliability was better [17].

Table 1: Assessment of verticality perception: modulatory factors and recommendations for standardization

Modulatory factors	Recommendation for standardization
Head/body position	• Maintenance of upright head and body position by a support system (e.g. chinrest)
Test stimulus	• Visual target with good contrast (e.g. luminous line, length of 10–100 cm) and adequate distance from the subject's eye (> 30 cm) • Initial tilt angle of 30–60° from center
Response modality	• Verbal response in case of apraxia, paresis • Verbal code answer for aphasic patients • Motor-guided response in patients without accompanying central deficits
Patient's task	• Adjustment of the visual target to perceived vertical
Number of repetitions	• Even number of repetitions • 6–10 repetitions, balanced for clockwise and counterclockwise rotations
Mode of recording	• Computer-based automatic recording in laboratory settings • Analog measures (e. g. using bucket test) in acute settings and bedside examinations
Outcome measure	• Orientation criterion: mean average deviation of SVV across repetitions • Uncertainty criterion: standard deviation of mean error or maximum difference of clockwise/counterclockwise adjustments
Standard values	• Mean average deviation of SVV: ± 2.5° • Uncertainty criterion: 3–8°

Relevant factors for reliable testing of verticality perception

- *Body position:* There is a consensus across studies that patients should be examined in a sitting position, because it is comfortable and safe. The head has to be maintained in an upright position by a chinrest, to correct for the impact of spontaneous head and body lateral inclination. This intervention is especially important in patients who are unable to sit (e. g. due to stroke), as it is well-known that a head or body tilt influences SVV measurements. Furthermore, correction of the body axis to the upright position helps to standardize testing conditions and thus facilitates comparison across patients and repetitive measurements [18, 19].
- *Test stimulus:* The visual target should have a good contrast and attract the patient's attention. Suitable stimuli are luminescent rods, lighted leads, or luminous lines presented on a computer screen. The line's length does not have an impact on the results of SVV measurement (range from 10–100 cm) [1]. The visual target should be at an adequate distance from the subject's head (> 30 cm) to avoid overlaying effects of cyclorotation of the eyes [12]. Furthermore, the initial tilt of the line, in terms of side and angle magnitude, affects SVV measurements. Most studies report a maximal angle of initial tilt in a range of 30–60° [20].
- *Response modality:* Most studies describe two principle types of response – adjustment of the target to vertical by verbal instructions to the operator or by active manipulation using a joystick, computer mouse, or visual-haptic task. No definite comparisons or recommendations have been made so far. Verbal commands may avoid a bias due to apraxia, executive function, or handedness, especially in patients with central lesions. Further limitations may emerge from cognitive capacities or attentional resources [18].
- *Patient's task:* In most protocols, the task for the patient is to adjust the visual stimulus to the perceived vertical. Only one study assessed SVV by a forced choice procedure (clockwise versus counterclockwise tilt) of two alternative stimuli presented briefly.
- *Number of repetitions:* Recommendations for trial repetition vary considerably across the literature, with most studies using at least 10 trials. A higher number of trials may help to counterbalance the effect of the initial side of the tilt and may result in more robust averaging. On the other hand, a high number of trial repetitions may add to variability because of fatigability and the limited attentional resources of some patients. A recent study in patients with subacute hemispherical stroke found that 6–10 repetitions are adequate to identify a SVV bias [20].
- *Mode of recording:* A digital method of SVV assessment allows automatic computation of data without interventions from the operator as well as fast editing of results and therefore is preferable for laboratory conditions. On the other hand, analog procedures as in the bucket test do have the advantage that the tool is portable and applicable at bedside 24 hours a day and may therefore be favorable in emergency and acute care settings [23].
- *Outcome measure:* The orientation criterion calculates a bias or constant error by averaging the deviations of SVV from true vertical across several repetitions [12, 20, 23]. As a second criterion, the standard deviation of the mean error can be used to estimate the uncertainty of verticality perception. Another measure of uncertainty may be the maximum difference of clockwise and counterclockwise adjustments or the complete range during which the patients considered the visual target as correctly aligned.
- *Standard values:* Across different studies and protocols, the range of normality differed only slightly. For the orientation criterion, a normal range of ±2.5° is defined for healthy subjects based on measurement for the hemispherical dome method, bucket test, and various computerized vertical perception tasks [20, 23]. The values do not seem to depend on age. For the SVV uncertainty criterion, two studies used a cutoff of 3–8° and described an increase of the range with age [1].

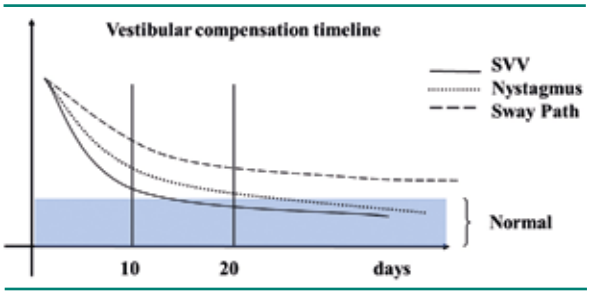

Fig. 3: Compensation of signs of vestibular imbalance after acute vestibular lesions. The subjective visual vertical (SVV) gradually normalizes within the first 2–4 weeks after an acute unilateral vestibular lesion. Provocation nystagmus and postural imbalance can persist longer. Adapted from [24].

Clinical relevance of verticality perception

Tests of verticality perception are used as diagnostic support tools especially in the acute phase of disease, assessment tools for recovery, and guidance of the mode of physical therapy in vestibular and balance disorders. The most important indications are the following:

1) Patients presenting with acute vertigo. Pathological SVV deviation is a highly sensitive indicator for an acute imbalance within the bilateral vestibular system due to unilateral damage of peripheral or central graviceptive pathways. SVV tilts from unilateral brainstem and cerebellar lesions are of topographic value to indicate the level or the side of the lesion [3, 4, 9, 12, 22].
2) Patients with balance problems due to hemispherical lesions. Patients with a biased reference of verticality should benefit from specific rehabilitation programs focused on lateropulsion and postural control and aiming to recalibrate the internal model of verticality [18].
3) Patients with ocular motor symptoms. Testing for mon- and binocular SVV can help to differentiate peripheral or central origin of symptoms. The major indications for SVV testing are summarized in **Table 2**.

Table 2: Assessment of verticality perception: clinical use and interpretation (adapted from [23])

Applications for SVV measurement	Pathological SVV deviation
1. Detection of unilateral graviceptive pathway damage:	
• acute unilateral peripheral vestibulopathy	> 90 %
• vestibular pseudoneuritis	> 90 %
• Wallenberg's syndrome (medullary lesion)	> 90 %
• internuclear ophthalmoplegia	> 90 %
• midbrain damage	> 90 %
• cerebellar lesion	> 90 %
• hemispheric lesion (frontal, insular, temporal)	35 %
2. Quantification of vestibular compensation:	
• unilateral peripheral graviceptive pathway damage • unilateral central graviceptive pathway damage (vestibular nucleus, MLF, cerebellum)	Mean normalisation time: 20–30 days
3. Topographic classification of brainstem lesions with vestibular or ocular motor symptoms:	
• vestibular nucleus	> 90 % ipsilesional
• MLF between abducens nucleus and -midbrain	> 90 % contralesional
• midbrain	>90 % contralesional
• thalamus	50 % ipsilesional/contralesional
• hemisphere	60 % contralesional
• cerebellum	75 % contralesional
4. Detection of central ocular motor damage:	
• nerve III or nerve IV	variable SVV deviations, monocular SVV deviation on the affected side

Summary

Measurement of the perception of verticality has wide implications in the diagnosis and treatment of patients with vestibular and balance problems. It is an important diagnostic tool in the acute setting, as well as a valuable surrogate marker in rehabilitation of disorders of postural control. In light of the previous literature on SVV assessment, the following recommendations can be given to establish a more consistent and standardized use of this measure in clinical and research settings:

1) Sufficient control of upright head and body position by a suitable device (e.g. chinrest), especially in patients with lateropulsion;
2) masking of visual orientation clues for verticality (e.g. edge of computer screen);
3) a motor response modality for patients without cognitive or speech impairments or paresis; a verbal response modality in case of apraxia, learning or executive dysfunctions and a verbal code answer for aphasic patients;
4) an adjustment method to align the visual stimulus to the perceived vertical;
5) an even number of trials, between 6 and 10, to account for the effect of the starting position of the line and obtain reliable SVV orientation by averaging;
6) an electronic assessment procedure with direct and automatic recording of data without operator intervention in laboratory settings and an analog procedure like the bucket test in emergency and bedside examinations; and
7) standard values for SVV adjustments of ±2.5° from true vertical.

Overall, more standardized and rigorous test protocols for assessment of perceived verticality should enhance the use of this parameter as a readout for clinical trials and for quantification of therapeutic interventions during rehabilitation in patients with vestibular and balance problems.

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References

1. Baccini M, Paci M, Del Colletto M, Ravenni M, Baldassi S. The assessment of subjective visual vertical: comparison of two psychophysical paradigms and age-related performance. Atten Percept Psychophys. 2014; 76: 112–22.
2. Bagust J. Assessment of verticality perception by a rod-and-frame test: preliminary observations on the use of a computer monitor and video eye glasses.Arch Phys Med Rehabil 2005; 86: 1062–4.
3. Baier B, Bense S, Dieterich M. Are signs of ocular tilt reaction in patients with cerebellar lesions mediated by the dentate nucleus? Brain 2008; 131: 1445–54.
4. Baier B, Suchan J, Karnath HO, Dieterich M. Neural correlates of disturbed perception of verticality. Neurology 2012; 78(10): 728-35.
5. Baier B, Thömke F, Wilting J, Heinze C, Geber C, Dieterich M. A pathway in the brainstem for roll-tilt of the subjective visual vertical: evidence from a lesion-behavior mapping study. J Neurosci 2012; 32(43): 14854–8.
6. Baier B, Conrad J, Stephan T, Kirsch V, Vogt T, Wilting J, Müller-Forell W, Dieterich M. Vestibular thalamus: Two distinct graviceptive pathways. Neurology 2016; 86(2): 134–40.
7. Bergmann J, Kreuzpointner MA, Krewer C, Bardins S, Schepermann A, Koenig E, Müller F, Jahn K. The subjective postural vertical in standing: reliability and normative data for healthy subjects. Atten Percept Psychophys 2015; 77(3): 953–60.
8. Bonan I, Hubeaux K, Gellez-Leman M, Guichard J, Vicaut E, Yelnik A. Influence of subjective visual vertical misperception on balance recovery after stroke. J NeurolNeurosurg Psychiatry 2007; 78: 49–55.
9. Brandt T, Dieterich M. Vestibular syndromes in the roll plane: topographic diagnosis from brainstem to cortex. Ann Neurol 1994; 36(3): 337–47.
10. Čakrt O, Slabý K, Kmet' J, Kolář P, Jeřábek J. Subjective visual and haptic vertical in young and elderly. J Vestib Res 2016; 25(5–6): 195–9.
11. Cnyrim CD, Rettinger N, Mansmann U, Brandt T, Strupp M. Central compensation of deviated subjective visual vertical in Wallenberg's syndrome.J NeurolNeurosurg Psychiatry 2007; 78(5): 527–8.
12. Dieterich M, Brandt T. Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. Ann Neurol 1993; 33(3): 292–9.
13. Dieterich M, Brandt T. Perception of verticality and vestibular disorders in the roll plane. Front Neurol 2019; 10: 172.
14. Frisén L. Deviations of the visual upright in three dimensions in disorders of the brainstem: a clinical exploration. Brain 2010; 133: 3541–51.
15. Glasauer S, Dieterich M, Brandt T. Neuronal network-based mathematical modeling of perceived verticality in acute unilateral vestibular lesions: from nerve to thalamus and cortex. J Neurol. 2018; 265(Suppl 1): 101–12.
16. Guerraz M, Yardley L, Bertholon P, Pollak L, Rudge P, Gresty MA, Bronstein AM. Visual vertigo: symptom assessment, spatial orientation and postural control. Brain. 2001;124:1646-56.
17. Michelson PL, McCaslin DL, Jacobson GP, Petrak M, English L, Hatton K. Assessment of Subjective Visual Vertical (SVV) Using the "Bucket Test" and the Virtual SVV System. Am J Audiol. 2018; 27(3): 249–59.
18. Pérennou D, Piscicelli C, Barbieri G, Jaeger M, Marquer A, Barra J. Measuring verticality perception after stroke: why and how? Neurophysiol Clin 2014; 44: 25–32.
19. Piscicelli C, Barra J, Davoine P, Chrispin A, Nadeau S, Pérennou D. Inter- and Intra-Rater Reliability of the Visual Vertical in Subacute Stroke. Stroke 2015. 46: 1979–83.
20. Piscicelli C, Pérennou D. Visual verticality perception after stroke: A systematic review of methodological approaches and suggestions for standardization. Ann Phys Rehabil Med 2017; 60: 208–16.
21. Tesio L, Longo S, Rota V. The subjective visual vertical: validation of a simple test.Int J Rehabil Res 2011; 34(4): 307–15.
22. Zwergal A, Cnyrim C, Arbusow V, Glaser M, Fesl G, Brandt T, Strupp M. Unilateral INO is associated with ocular tilt reaction in pontomesencephalic lesions: INO plus. Neurology. 2008; 71(8): 590–3.
23. Zwergal A, Rettinger N, Frenzel C, Dieterich M, Brandt T, Strupp M. A bucket of static vestibular function. Neurology 2009; 72(19): 1689–92.
24. Zwergal A, Schniepp R. Vestibular compensation: Clinical and scientific aspects. Nervenheilkunde 2010; 29(10): 648–53

Conflict od interest:
The authors state that there is no conflict of interest.

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Multisensory control of posture: Clinical assessment and disorders

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Introduction

Impairment of postural control affects a majority of older people. When assessed by using a simple clinical test (modified Romberg test), the prevalence of balance disorders is high and depends on age (60–69 years: 49.4%, 70–79 years: 68.7%, >80 years 84.8%; n>5000) [1]. In advanced age, it is of particular importance to differentiate causes of imbalance, as overlapping aetiologies are common [8]. **Figure 1** summarizes important domains for postural control.

For balance, sufficient sensory functions (visual, vestibular, proprioceptive), intact central networks (sensory pathways, cerebellum, thalamus, multisensory cortex areas), and the integration of automated postural reflexes in the cognitive context are essential. Impaired balance is associated with falls, high morbidity, reduced participation, and reduced quality of life. In the following, we summarize physiology of postural control and the clinical approach to assess balance. Knowledge on pathophysiology and clinical syndromes forms the basis for the development of rationale therapies.

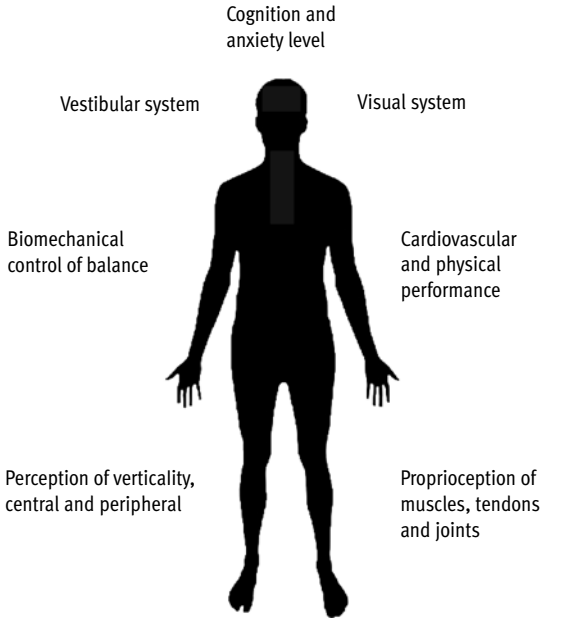

Fig. 1: Domains for Postural Control. Peripheral and central nervous systems, biomechanics, general physical performance, and cognitive aspects are all important for equilibrium

Human postural control

Postural control requires peripheral perception of sensory signals and central representation of the 3D space using gravity information and a pre-formed body scheme. Sensory information from the visual, vestibular, proprioceptive systems need to be integrated to provide equilibrium maintenance [6]. Different domains and aspects contribute to postural control **(Figure 1)**. Concerning neuronal control of stance and locomotion the so-called pattern generators in the central nervous system provide the basic repetitive motor pattern [5]. The central pattern generators in humans are networks of interneurons in the lumbar and cervical spinal cord that regulate activation of antigravity muscles and the alternated activation of agonists and antagonists in legs and arms during standing and walking. These pattern generators interact with sensory afferents from the periphery (proprioceptive) and from the head (vision, vestibular system). The supraspinal locomotor network is involved in the control of posture for new and more complicated tasks, i.e. reacting to disturbances [7]. In the human brain network for postural control, premotor and motor regions in the frontal lobe are involved in initiating and adapting the motor pattern [9]. Sensory feedback via the thalamus is needed for postural control [15]. Regions in the brainstem and cerebellum, the homologues to the locomotor regions described in quadrupedal animals, connect between supratentorial brain structures and the spinal cord pattern generators [7] **(Figure 2)**.

With ageing, the interaction between brain structures for postural control is impaired so that weighting and priority settings are altered [16]. An example for such a change with ageing is the cross-inhibition between different sensory modalities that ensures the use of the most appropriate sensory signal for a certain task (e.g.

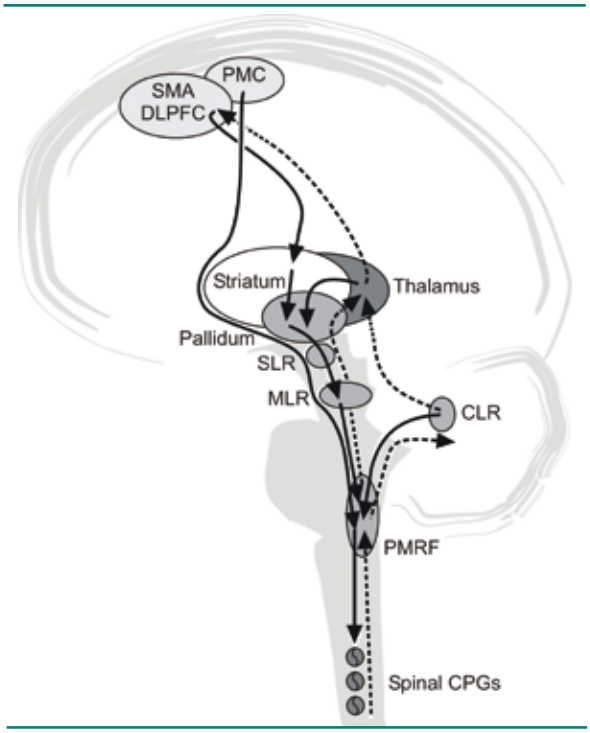

Fig. 2: Central postural and locomotor control. Frontal and prefrontal cortical areas, basal ganglia, thalamus, midline cerebellum, and brainstem tegmentum are essentially involved in the networks. Abbreviations: **CLR** cerebellar locomotor region; **CPG** central pattern generator; **DLPFC** dorso-lateral prefrontal cortex; **MLR** mesencephalic locomotor region; **PMC** primary motor cortex; **PMRF** ponto-medullary reticular formation; **SLR** subthalamic locomotor region; **SMA** supplementary motor area. Modified after [7]

vison instead of vestibular signals). In advanced age, this cross inhibition is reduced and conflicts between sensory canals contribute to balance problems. Another example for impaired network interactions to executive functions is the dual task interference with postural tasks in the elderly [4].

Assessment of postural control

Clinical assessment of balance requires observing and rating of stance with eyes open on firm ground (full sensory control), with eyes closed (vestibular and proprioceptive control), and on foam or with reduced base of support (visual and vestibular control). Vestibular control can be modified by head extension. In general, the test of equilibrium should proceed stepwise from standing with full control to standing with minimal control (eyes closed, on foam, tandem stand, head extension) to get a good view on problematic aspects. While patients with functional and psychogenic causes of imbalance are able to stand in the more difficult conditions despite the fact that standing with full control is impaired, patients with organic dysfunction become worse with increasing complexity of the task. The clinical examination of postural imbalance should always include the evaluation of sensory deficits (visual, vestibular, somatosensory). Furthermore, patients should undergo complete internal medical and neurological examinations (especially of the extremities and oculomotor function), and active and passive mobility in the large joints should be assessed [8]. For evaluation of postural reflexes, the pull-test (postural recovery after a sudden but announced backward pull), reaching tests (testing for the limits of stability), and tests including transfers from sitting to standing (timed up and go) are helpful [8].

Concerning laboratory testing, tests for perception of verticality and static/dynamic posturography (stabilometry) on a force platform are the methods of choice. Perception of verticality can be measured as subjective visual, subjective haptic, and subjective postural vertical. Ageing processes influence verticality perception during standing and sitting. With increasing age, the perceived vertical shifts backwards, i.e. it is tilted in the posterior direction and the risk of falls in the sagittal plane rises [3]. The reduced sensitivity may reflect an age-related decline of vestibular and somatosensory functions. These sensory systems are involved in creating and updating the central representation of verticality. For clinical use the subjective visual vertical can be measured with the simple bucket method [17]. For posturography, reliable normal values are of importance as simple parameters like foot position play an essential role in the comparability of data between laboratories [10].

Clinical implication

Differentiation of neurological causes of postural impairment requires multimodal assessment. For differential diagnosis it is helpful to observe balance performance during walking (dynamic postural control) and during standing (static postural control). While standing (or walking) sensory and cognitive challenges should be tested. **Figure 3** summarizes the implication of these aspects for differential diagnosis.

Among older patients the following causes for postural imbalance are common:

- sensory deficits (e.g., polyneuropathy, vestibulopathy)
- neurodegeneration (e.g., Parkinson's disease, cerebellar ataxia)
- cognitive deficits and anxiety (e.g., dementia, fear of falls).

In addition, antalgic (e.g., osteoarthritis of the knee) and paretic (e.g., after disc herniation) disorders account for a large proportion of patients in general practice, orthopaedic surgery, and neurology [12]. Important aspects in association with unsteady gait in old age also include the function of the sensory systems, cognitive reserve, and locomotor reserve [14].

Walking	Dual Task	Eyes Closed
Fast walking better Sensory deficit (neuropathy, vestibulopathy)	**Much worse** Dementia Mild cognitive impairment	**Much worse** Sensory deficit (neuropathy, vestibulopathy)
Fast walking worse Spasticity Cerebellar	**Worse** Parkinson Vascular NPH	**Worse** Cerebellar Vascular
Reduced modulation Parkinson	**Minor effect or better with DT** Functional	**Minor effect** Parkinson Spasticity

Fig. 3: Differential control of posture. During walking, postural control is most important when moving slowly (fast walking is more automated). Cognitive deficits can be unmasked with dual-task tests. Sensory deficits become most obvious when single channels are blocked (eyes closed to block vision; standing on foam to block proprioception)

The sensory function is important in the context of conscious postural control and slow walking and is less relevant in automatic stance and locomotion. The risk of falls increases with the variability of the sway pattern [8]. The concept of 'motoric cognitive risk syndrome' (MCR syndrome) has been proposed for old patients with subjective cognitive impairment and slow gait (>1 standard deviation under the age-specific mean). Persons with MCR syndrome are at risk of developing a degenerative form of dementia [13]. Dual task performance provides an insight into the functional locomotor-cognitive reserves of an old person [4]. This ability is often impaired before obvious cognitive deficits manifest [2]. Dual task paradigms also help in the differential diagnosis [11].

Summary

Imbalance, impaired gait, and falls are highly prevalent in older patients. They have a substantial impact on independence and quality of life. Aetiology is often related to sensory functions (vision, proprioception, vestibular system), motor and cognitive neurodegeneration, and fall-related psychological concerns. The assessment, therefore, should include clinical tests for these deficits.

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References

1. Agrawal Y, Carey JP, Della Santina CC et al. Disorders of balance and vestibular function in US adults: data from the National Health and Nutrition Examination Survey 2001–2004. Arch Int Med 2009; 169: 938–44.
2. Beauchet O, Annweiler C, Dubost V et al. Stops walking when talking: a predictor of falls in older adults? Eur J Neurol 2009; 16: 786–95.
3. Bergmann J, Kreuzpointner MA, Krewer C et al. The subjective postural vertical in standing: reliability and normative data for healthy subjects. Attention Perception Psychophysics 2015; 77: 953–60.
4. Bridenbaugh SA, Kressig RW. Motor cognitive dual tasking: early detection of gait impairment, fall risk and cognitive decline. Z Gerontologie Geriatrie 2015; 48: 15–21.
5. Grillner S. Neuronal networks in motion from ion channels to behaviour. An R Acad Nac Med (Madr) 2006; 123: 297–8.
6. Ivanenko Y, Gurfinkel VS. Human Postural Control. Frontiers Neuroscience 2018; 12: 171.
7. Jahn K, Deutschlander A, Stephan T et al. Supraspinal locomotor control in quadrupeds and humans. Prog Brain Res 2008; 171: 353–62.
8. Jahn K, Kressig RW, Bridenbaugh SA et al. Dizziness and unstable gait in old age: etiology, diagnosis and treatment. Dtsch Arztebl Int 2015; 112: 387–93.
9. Jahn K, Zwergal A. Imaging supraspinal locomotor control in balance disorders. Restor Neurol Neurosci 2010; 28: 105–14.
10. Krewer C, Bergmann J, Gräfrath P et al. Influence of foot position on static and dynamic standing balance in healthy young adults. Hearing Balance Communication 2019. DOI: 10.1080/21695717.2018.1507576 (in press).
11. Selge C, Schoeberl F, Zwergal A et al. Gait analysis in PSP and NPH: Dual-task conditions make the difference. Neurology 2018; 90: e1021–8.
12. Snijders AH, Van De Warrenburg BP, Giladi N et al. Neurological gait disorders in elderly people: clinical approach and classification. Lancet Neurology 2007; 6: 63–74.
13. Verghese J, Annweiler C, Ayers E et al. Motoric cognitive risk syndrome: multicountry prevalence and dementia risk. Neurology 2014; 83: 718–26.
14. Verghese J, Xue X. Predisability and gait patterns in older adults. Gait & Posture 2011; 33: 98–101.
15. Zwergal A, La Fougere C, Lorenzl S et al. Postural imbalance and falls in PSP correlate with functional pathology of the thalamus. Neurology 2011; 77: 101–9.
16. Zwergal A, Linn J, Xiong G et al. Aging of human supraspinal locomotor and postural control in fMRI. Neurobiol Aging 2012; 33: 1073–84.
17. Zwergal A, Rettinger N, Frenzel C et al. A bucket of static vestibular function. Neurology 2009; 72: 1689–92.

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Postural orientation with conflicting visual and graviceptive cues to 'upright' among individuals with and without a history of post-stroke 'pushing'

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Abstract

Purpose: This study aimed to determine how people with stroke, with and without pushing behaviour, use sensory cues to control postural orientation.

Methods: Eight people with chronic stroke (4 with history of pushing behaviour), 5 people with sub-acute stroke (1 with active pushing behaviour) and 8 similarly-aged controls with no history of stroke participated. Participants sat in a motion platform while viewing a 240-degree screen upon which a city street scene was projected. Postural orientation (shoulder and trunk angles) was measured relative to the direction of gravity during 6 trials: visual scene tilted 18-degrees left and right; motion base tilted 18-degrees left and right; and both visual scene and motion base tilted 18-degrees left and right.

Results: Participants with stroke did not appear to adjust their posture in response to visual scene tilt to a greater extent than control participants. For most conditions, chronic stroke participants with a history of pushing behaviour oriented their posture more towards the contralesional side than controls. When the motion base was tilted, sub-acute participants with no evidence of pushing behaviour oriented their posture more in the direction of motion base tilt than controls (e. g., when the motion base tilted to their ipsilesional sides, their trunks and shoulders were oriented to the ipsilesional side).

Conclusion: This study did not find evidence that people with stroke with and without a history of pushing behaviour rely more on static visual cues to control postural orientation than people without stroke.

Keywords: posture, gravity perception, visual perception, kinematics, spatial orientation, stroke

Introduction

Post-stroke pushing behaviour is characterised by postural lean to the contralesional side, despite significant weakness on that side, and resisting correction to upright [16]. To try understand the mechanisms underlying post-stroke pushing, Karnath et al. seated people with and without post-stroke pushing securely in a padded chair that could tilt left or right [10]. With eyes closed, participants were tilted in one direction, and directed the experimenter to move the chair in the opposite direction until they felt upright. The subjective postural vertical is the position relative to the direction of gravity (i. e., earth vertical) at which participants feel upright [4]. One would expect those with pushing behaviour to feel upright when aligned to the contralesional side, as this reflects clinical presentation. However, people with pushing behaviour felt upright when oriented approximately 18° to the ipsilesional side [10]. The authors speculated that the clinical presentation of pushing behaviour reflects compensation for a mismatch between perceived visual and truncal graviceptive cues to upright [10].

The unexpected finding of an ipsilesional bias in subjective postural vertical has been replicated by some [3] but not other [19] subsequent studies. Pérennou et al. observed ≥ 6° contralesional bias in subjective postural vertical among those with pushing behaviour [19]. We observed that people with chronic stroke can continue to have a contralesional bias in subjective postural verti-

cal despite resolution of obvious pushing behaviour [14]. This work [14, 19] suggests that pushing behaviour arises from misperception of body orientation relative to earth vertical, and that people with pushing align their bodies with perceived vertical (i.e., the contralesional side).

While the direction of bias is controversial, evidence suggests that people with pushing behaviour have impaired perception of vertical. The link between impaired perception and behaviour (i.e., natural postural orientation) is less clear. This study aimed to determine how people with stroke use sensory cues to control postural orientation. We seated participants in a 6-degree of freedom motion base with projected visual surround and measured participants' natural posture when the motion base was tilted left and right, and when presented with conflicting and consistent visual cues to earth vertical.

Methods

Participants

Eight participants with chronic stroke, 5 participants with sub-acute stroke and 8 similarly-aged participants with no history of stroke were recruited. Participants were excluded if they had musculoskeletal or neurological conditions (besides stroke) that affect balance, history of vestibular disorders, and/or poor corrected or uncorrected visual acuity. Participants with stroke were excluded if they had bilateral strokes. On item C of the Scale for Contraversive Pushing (SCP [10], resists correction) 4 chronic stroke participants scored ≥ 1 early in stroke recovery; these participants formed the history of pushing (HP) group. The remaining 4 chronic stroke participants had no documented history of post-stroke pushing and formed the no history of pushing (NHP) group. Four sub-acute stroke participants had no evidence of history of pushing behaviour and formed the no-active pushing group (NAP). One sub-acute stroke participant had active pushing behaviour (AP), as assessed by his treating physiotherapist; the SCP could not be assessed for this participant due to his severe postural impairment. Participant characteristics are shown in **Table 1**. The study was approved by the institution's Research Ethics Board and participants provided written informed consent.

Table 1: Participant characteristics. Values are presented for individual participants with stroke. Data for controls are means with standard deviations in parentheses

Participant/ group	Age (years)	Sex	Time post-stroke (months)	Stroke type	Stroke location	NIH-SS (score)	CMSA-leg (score)	CMSA-foot (score)	BBS (score)	SNAP (score)	Left heel touch threshold (log force)	Right heel touch threshold (log force)
Controls	64.1 (7.0)	4 M 4 F	–	–	–	0 (0)	7 (0)	7 (0)	55.9 (0.4)	0.4 (1.1)	4.38 (0.22)	4.24 (0.27)
NHP group												
NHP-1	66	F	7.3	Ischemic	Left basal ganglia	2	6	6	53	0	3.61	3.61
NHP-2	49	F	15.4	Ischemic	Right pons	2	5	5	55	0	3.61	3.84
NHP-3	62	F	17.0	Ischemic	Left internal capsule	1	7	7	56	2	4.08	4.31
NHP-4	58	M	12.2	Ischemic	Right internal capsule	1	7	7	56	7	5.07	4.31
HP group												
HP-5	80	M	44.1	Hemorrhagic	Right thalamus	1	5	4	37	0	4.93	4.93
HP-6	66	M	48.9	Ischemic	Right parietal & frontal	8	3	2	26	33	2.83	4.08
HP-7	79	F	15.6	Ischemic	Right parietal & internal capsule	4	5	5	37	60	*	*
HP-8	78	F	15.5	Ischemic	Right parietal & frontal	2	4	5	29	6	5.07	5.88
NAP group												
NAP-9	64	M	2.5	Ischemic	Right internal capsule & pons	4	5	3	41	0	4.31	4.17
NAP-10	73	F	1.2	Ischemic	Left brainstem	2	6	7	45	4	4.74	4.56
NAP-11	79	F	0.7	Ischemic	Right basal ganglia	3	4	5	29	0	5.18	4.56
NAP-12	60	M	0.6	Ischemic	Cerebellum & right medulla	1	5	5	30	0	3.84	3.61
AP participant												
AP-13	67	M	0.8	Ischemic	Left middle cerebral artery/anterior cerebral artery region	9	3	2	8	0	4.17	4.08

AP active pushing; **BBS** Berg Balance Scale; **CMSA** Chedoke-McMaster Stroke Assessment; **F** female; **HP** history of pushing; **M** male; **NIH-SS** National Institutes of Health stroke scale; **NAP** no active pushing; **NHP** no history of pushing; **SNAP** Sunnybrook Neglect Assessment Procedure; *Unable to assess (participant did not understand the instructions)

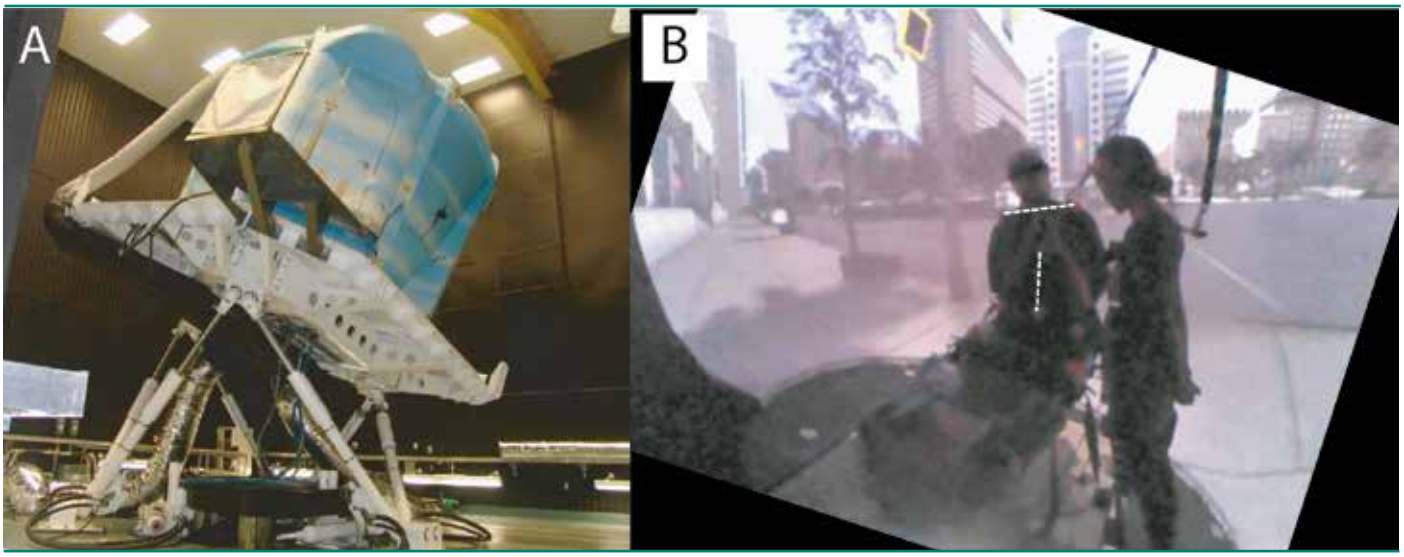

Fig. 1: Motion base and calculation of trunk and shoulder angles. Panel A shows the exterior of the 6-degree of freedom motion base. Panel B shows the view from the digital video camera within the motion base during an M-right trial (see also Figure 2). The orientation of the trunk and the shoulders are indicated by the dotted lines. Note that the image in Panel B has been rotated 18 degrees to the right; earth vertical (i.e., the direction of gravity) is up-down with respect to both images. Trunk and shoulder angles were calculated relative to earth vertical/horizontal.

Procedures

This paper presents a subset of data from a larger study; further details of study procedures not presented here can be found in companion papers [8, 14]. Cutaneous sensation at the plantar surface of the heel was assessed using Semmes Weinstein monofilaments [15].

To assess postural orientation in response to visual and gravitational stimuli, participants were seated on a plinth, placed inside a motion simulator with a 240° horizontal field-of-view projection screen **(Figure 1)**. A non-slip mat (Dycem, Bristol, United Kingdom) was placed on the seat to prevent participants from sliding. A research assistant stood beside participants (out of view) to provide instructions and physical assistance, if required. Both the research assistant and participants wore a harness attached to an overhead support as an extra safety measure. Participants' feet hung freely and they were asked to place their hands on their laps; they were otherwise free to adopt a natural posture. Participants viewed a static city street scene projected on the screen **(Figures 1 & 2)**; the scene had several cues to upright, e. g., sky in the upper portion of the scene, tall buildings [9]. Spherical markers were placed at the approximate locations of the T7 and L5 vertebrae on the back, and on the acromion processes. A digital video camera (sample frequency: 30 Hz) directly behind participants captured the position of these markers in order to calculate trunk and shoulder angles.

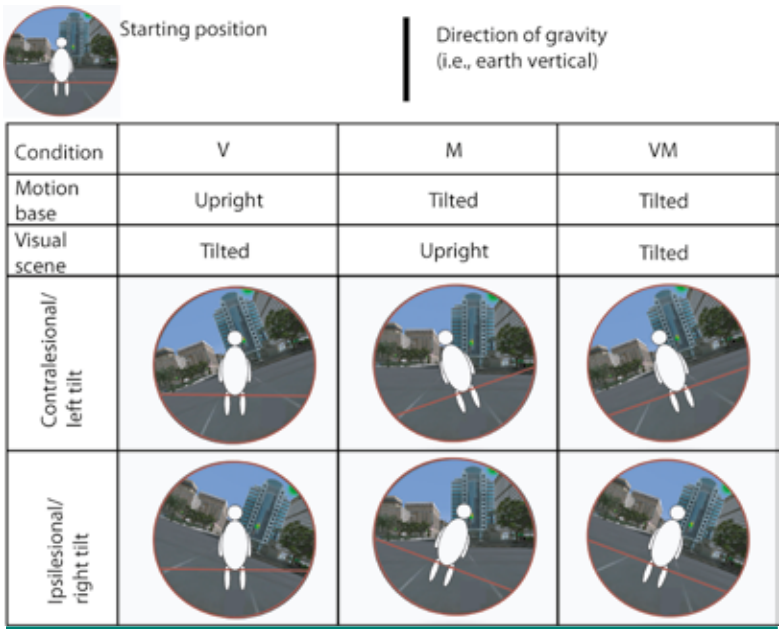

Fig. 2: Illustration of the motion and visual scene condition used in the study. The participant initially sat upright in the motion simulator (starting position). During each trial, the visual scene and/or motion base was tilted 18° to the right or left with respect to the direction of gravity (i.e., earth vertical). The final visual scene/motion base position is illustrated in the figure.

Six trials were completed in an unpredictable order **(Figure 2)**: visual scene tilted left or right (V trials);motion base tilted left or right (M trials); and both motion base and visual scene tilted left or right (VM trials). Each trial started with the motion base and visual scene oriented upright with respect to earth vertical. Participants were instructed to look straight ahead and to maintain an upright posture. The motion base and/or visual scene then tilted to the right or left at a peak angular velocity of 0.5°/s and acceleration/deceleration of 0.2°/s² until the visual scene and/or motion base was 18° from earth vertical. The motion base and/or visual scene remained static at this angle for 5–10 seconds, and then returned slowly to upright before the next trial.

Data processing

Trunk and shoulder angles were calculated for five frames at the start of each trial and when the motion base/visual scene reached the maximum angle using a custom routine implemented in Matlab (R2014a, The Mathworks, Natick, MA, USA). As the camera was placed inside the motion simulator and rotated with the motion base, angles were initially calculated relative to the motion base; the motion base angle was subtracted from the trunk/shoulder angle in order to calculate all angles relative to earth vertical/horizontal **(Figure 1)**. The sign of the angle was changed such that positive angles indicated lean to the ipsilesional side (right for controls), and negative angles indicated lean to the contralesional side (left for controls).

Statistical analyses

As there was only one AP participant, this individual was excluded from statistical analyses. Shapiro-Wilk test was used to confirm that data were normally distributed. Two-way repeated measures analyses of variance (ANOVAs) were used to examine between group responses to each condition. The dependent variables were trunk and shoulder angles, relative to earth vertical/horizontal. The two factors in the ANOVAs were group (control, NHP, HP, NAP) and condition (conflicting or consistent visual cues to vertical). The first ANOVAs compared conditions with the motion base upright with respect to earth vertical; i.e., the conditions were the start position and V trials. The second ANOVAs compared conditions with the motion base tilted; i.e., the conditions were M and VM trials. The group-by-condition interaction effect was used to determine if one group responded differently to a condition than others. In the event of significant interaction or main effects, pre-planned contrasts were used to determine if each stroke group (NHP, HP or NAP) differed from controls. ANOVAs were conducted separately by direction of visual scene/motion base tilt (ipsilesional/right or contralesional/left). Alpha was 0.05 for all analyses.

Results

Missing data

Due to technical difficulties, one NAP participant did not complete the VM contralesional trial; the M contralesional trial was also removed from the analyses for this participant.

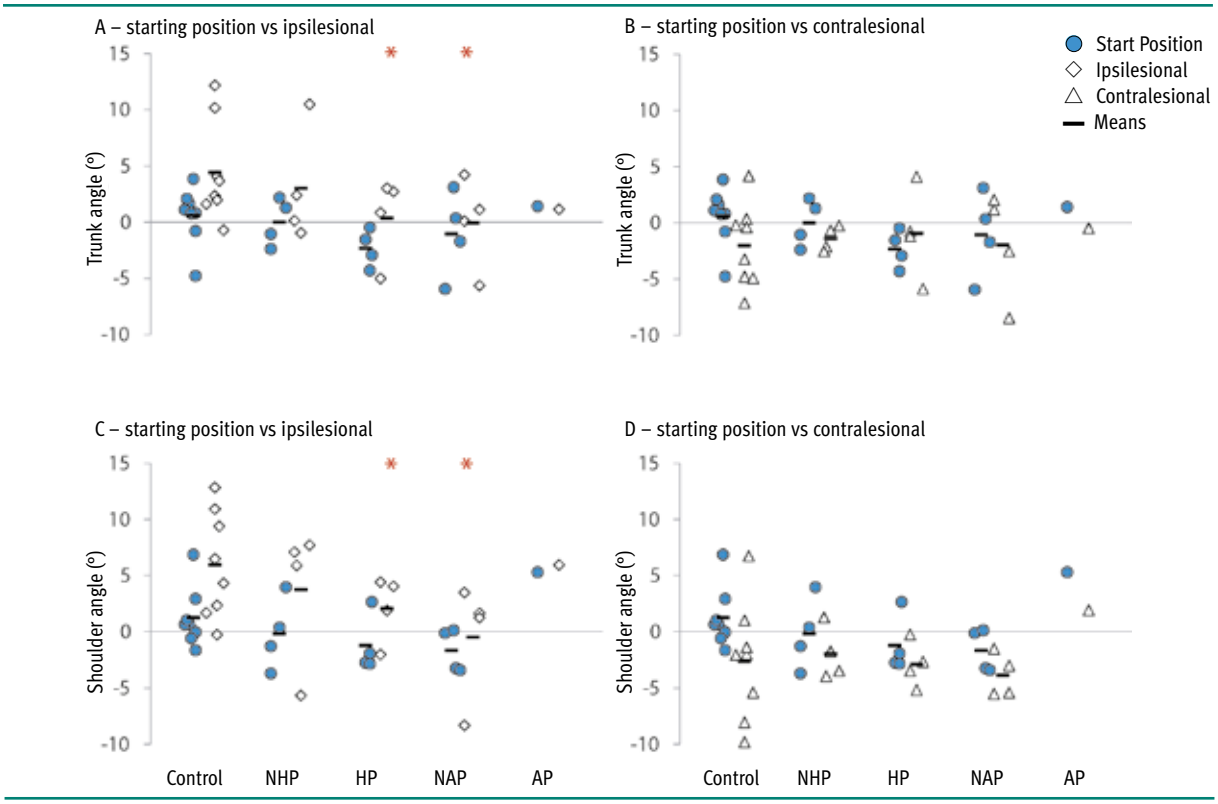

Fig. 3: Trunk and shoulder angles for trials where the motion base remained upright. Panels A and B show trunk angles, and Panels C and D show shoulder angles. Values shown are data points for individual participants, with group means indicated by the black bars. Data points are 'jittered' along the x-axis to prevent overlap of points. Angles were calculated with respect to earth vertical/horizontal, with positive angles indicating orientation to the ipsilesional/right side, whereas negative angles indicate orientation to the contralesional/left side. Data are shown for the V conditions and the starting position (see also **Figure 2**). Significant group effects are indicated with asterisks, where the groups significantly differed from the control group. There were no significant group-by-condition interactions or significant condition effects for these conditions.

Motion base upright

Figure 3 shows the results for trials where the motion base was upright; there were no significant group-by-condition interactions for trunk or shoulder angles in either direction ($F_{3,16} < 1.99$, $p > 0.15$).

For all groups, trunk and shoulder angles tended to be aligned slightly with the visual scene when it was tilted to the ipsilesional/right side; however, the condition effect was not statistically significant ($F_{1,16} < 4.48$, $p > 0.050$). There was a significant group effect for both trunk and shoulder angles ($F_{3,16} > 4.48$, $p < 0.019$) for visual scene tilt to the ipsilesional/right side. HP ($F_{1,16} > 6.49$, $p < 0.022$)and NAP ($F_{1,16} > 7.57$, $p < 0.015$) trunk and shoulder angles were oriented less to the ipsilesional/right side than controls. There were no significant group ($F_{3,16} < 2.21$, $p > 0.12$) or condition effects ($F_{1,16} < 3.17$, $p > 0.094$) for trunk or shoulder angles for visual scene tilt to the contralesional/left side.

The AP participant's trunk was oriented upright for conditions where the motion base was upright. His shoulders tended to be oriented more to his ipsilesional side, particularly in the start position and V ipsilesional trial.

Motion base tilted

Figure 4 shows the results for trials where the motion base tilted; there were no significant group-by-condition interactions for trunk or shoulder angles in the ipsilesional/right ($F_{3,16} < 0.38$, $p > 0.77$) or contralesional/left directions ($F_{3,15} < 0.41$, $p > 0.75$).

For motion base tilt to the ipsilesional/right side, there was a significant condition effect for shoulder angle ($F_{1,16} = 8.65$, $p = 0.0096$) but not trunk angle ($F_{1,16} = 3.18$, $p = 0.093$). For all groups combined, shoulder angles were oriented more to the ipsilesional/right side for VM trials (mean: 8.6°, standard deviation: 5.3°) than M trials (mean: 1.3°, standard deviation: 5.9°). For motion base tilt to the ipsilesional/right side, there were significant group effects for trunk and shoulder angles ($F_{3,16} > 8.05$, $p < 0.0018$). Specifically, NAP trunk and shoulder angles were oriented more to the ipsilesional/right side than controls ($F_{1,16} > 6.18$, $p < 0.025$), whereas HP shoulder angles were oriented less to the ipsilesional side than controls ($F_{1,16} = 10.74$, $p = 0.0047$). There was no significant difference between HP and control trunk angles ($F_{1,16} = 0.47$, $p = 0.50$).

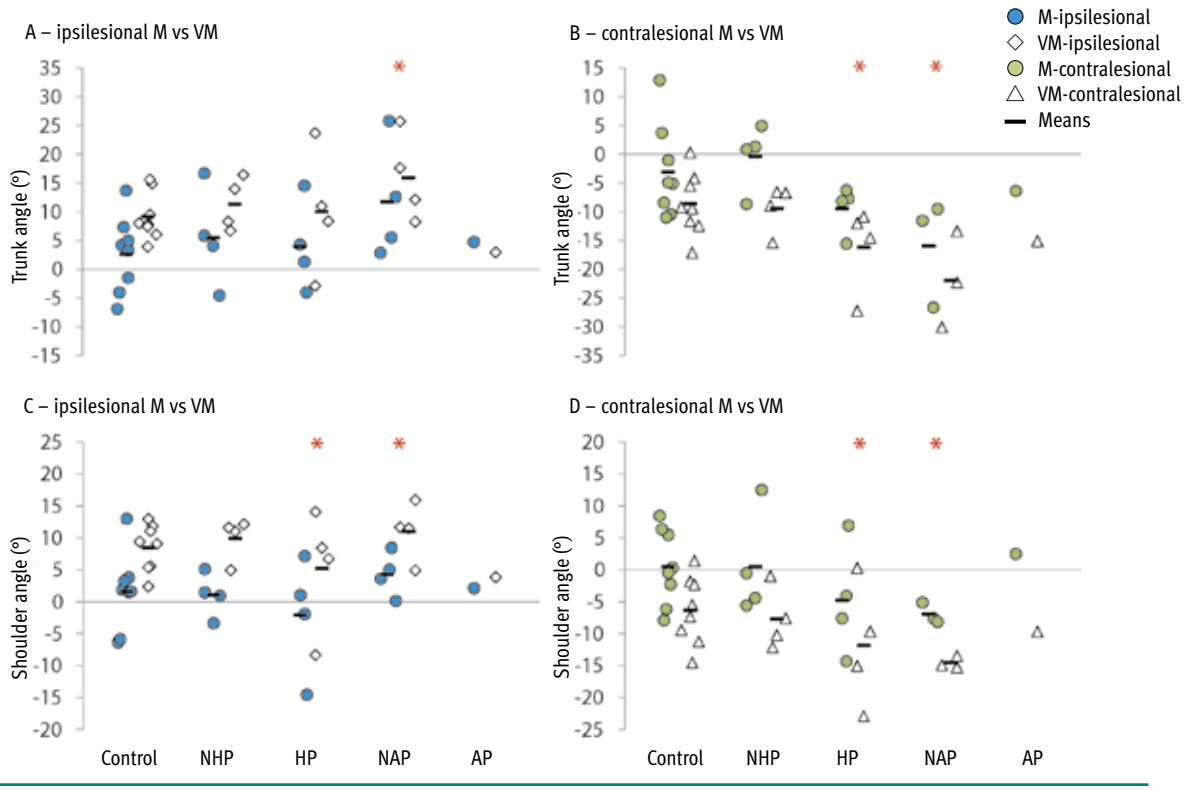

Fig. 4: Trunk and shoulder angles for trials where the motion base tilted. Panels A and B show trunk angles, and PanelsC and D shows shoulder angles. Values shown are data points for individual participants, with group means indicated by the black bars. Data points are 'jittered' along the x-axis to prevent overlap of points. Angles were calculated with respect to earth vertical/horizontal, with positive angles indicating orientation to the ipsilesional/right side, whereas negative angles indicate orientation to the contralesional/left side. The visual scene was aligned with gravity in the M trials and aligned with the motion base in the VM trials (see also Figure 2). Significant group effects are indicated with asterisks, where the groups significantly differed from the control group. There were no significant group-by-condition interaction effects for these conditions

There were significant condition effects for both trunk ($F_{1,15} = 5.20$, $p = 0.038$) and shoulder ($F_{1,15} = 6.24$, $p = 0.025$) angles for motion base tilt to the contralesional/left side. Trunk and shoulder angles were oriented more to the contralesional/left side for VM trials (trunk mean: -12.5°, standard deviation: 7.6°;shoulder mean: -9.1°, standard deviation: 6.4°) than for M trials (trunk mean: -5.9°, standard deviation: 8.6°; shoulder mean: -1.8°, standard deviation: 7.0°). For motion base tilt to the contralesional/left side, there were significant group effects for trunk and shoulder angles ($F_{3,15} > 16.40$, $p < 0.0001$). Specifically, both HP ($F_{1,15} > 19.47$, $p < 0.0006$) and NAP ($F_{1,15} > 36.23$, $p < 0.0001$) trunk and shoulder angles were oriented more to the contralesional/left side than controls.

The AP participant's shoulder and trunk angles in the M trials were similar to those of controls. In the VM ipsilesional trial his trunk was more closely aligned with earth vertical than controls, where as for the VM contralesional trial his trunk and shoulders were aligned to his contralesional side.

Discussion

This study aimed to determine how people with and without history of post-stroke pushing behaviour use visual and graviceptive cues to control seated postural orientation. Participants generally adjusted posture in an attempt to stay upright with respect to earth vertical following the motion base perturbation, in agreement with previous work [11].For M trials, where the motion base tilted with the visual scene upright, participants did not fully correct posture to earth vertical; this was likely due to limitations in spine range of motion that cannot fully compensate for this postural perturbation. The difference in trunk and shoulder angles between the VM and M conditions was approximately half of the difference in visual scene orientation between these conditions (~7°). Previous work suggests that perception of upright is approximately equally influenced by visual and graviceptive cues [7]. The current study extends these previous findings by suggesting that humans use visual and graviceptive cues approximately equally to re-orient to upright after a postural perturbation.

We did not find evidence that participants with stroke were more susceptible than controls to postural deviations from upright in the presence of conflicting visual cues. For most conditions, HP participants' posture was oriented more to the contralesional side than controls; despite resolution of obvious pushing behaviour, these individuals might still show sub-clinical signs of pushing that influence postural orientation [13, 14]. NAP participants also showed contralesional postural biases in the conditions where the motion base tilted to the contralesional side, and showed ipsilesional postural biases when the motion base tilted to the ipsilesional side. Orientation biases for NAP participants condition may reflect reduced trunk strength/control and, therefore, reduced capacity to correct postural orientation following these postural perturbations. It is possible that, due to delayed recovery for those with pushing behaviour [1, 5, 6, 17], HP participants' postures were also influenced by reduced trunk strength/control rather than perceptual impairment. Alternatively, it has been suggested that pushing behaviour occurs on a continuum rather than being strictly binary [18]. Thus, despite no clinical evidence of pushing behaviour, some NAP participants may have had sub-clinical pushing, which may account for the contralesional postural orientation for some participants.

The AP participant's postural orientation did not appear to be influenced by conflicting visual cues to upright, particularly when the motion base was upright. Our companion paper describes how this participant also did not appear to use visual cues to perceive upright [8]. In general, this participant's postural orientation followed a similar pattern to HP participants. Of note, when the motion base tilted to the ipsilesional side, the AP participant and most of the HP participants had very little ipsilesional bias, in contrast to the other groups. This postural orientation is consistent with the clinical presentation of pushing behaviour, and resistance to being passively moved to the ipsilesional side [16].

Summary

This study suggests that people with stroke with and without a history of pushing behaviour do not rely more on static visual cues to control postural orientation than people without stroke. Previous interventions focused on remediating pushing behaviour by asking participants to align their bodies with visual references to vertical (e.g., door frames) have not been as effective as interventions that stimulate somatosensation of earth vertical [2, 12]. The current findings may help to explain the results of these intervention studies.

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References

1. Babyar SR, White H, Shafi N, Reding M. Outcomes with stroke and lateropulsion: a case-matched controlled study. Neurorehabil Neural Repair 2008; 22: 415–23.
2. Bergmann J, Krewer C, Jahn K, Müller F. Robot-assisted gait training to reduce pusher behavior. Neurology 2018; 91: e1319–27.
3. Bergmann J, Krewer C, Selge C, Müller F, Jahn K. The subjective postural vertical determined in patients with pusher behavior during standing. Top Stroke Rehabil 2016; 23: 184–190.
4. Bisdorff AR, Anastasopoulos D, Bronstein AM, Gresty MA. Subjective postural vertical in peripheral and central vestibular disorders. Acta Otolaryngol 1995; 115: 68–91.
5. Clark E, Hill KD, Punt TD. Responsiveness of 2 scales to evaluate lateropulsion or pusher syndrome recovery after stroke. Arch Phys Med Rehabil 2012; 93: 149–55.
6. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke pushing: natural history and relationship to motor and functional recovery. Stroke 2004; 35: 2873–8.
7. Dyde RT, Jenkin MR, Harris LR. The subjective visual vertical and the perceptual upright. Exp Brain Res 2006; 173: 612–22.
8. Fraser LE, Mansfield A, Harris LR, Merino DM, Knorr S, Campos JL. The weighting of cues to upright following stroke with and without a history of pushing. Can J Neurol Sci 2018; 45: 405–14.
9. Harris LR, Jenkin M, Dyde RT, Jenkin H. Enhancing visual cues to orientation: suggestions for space travelers and the elderly. Prog Brain Res 2011; 191: 133–42.
10. Karnath H-O, Ferber S, Dichgans J. The origin of contraversive pushing: evidence for a second graviceptive system in humans. Neurology 2000; 55: 1298–1304.
11. Kluzik J, Peterka RJ, Horak FB. Adaptation of postural orientation to changes in surface inclination. Exp Brain Res 2007; 178: 1–17.
12. Krewer C, Rieß K, Bergmann J, Müller F, Jahn K, Koenig E. Immediate effectiveness of single-session therapeutic interventions in pusher behaviour. Gait Posture 2013; 37: 246–50.
13. Mansfield A, Danells CJ, Zettel JL, Black SE, McIlroy WE. Determinants and consequences for standing balance of spontaneous weight-bearing on the paretic side among individuals with chronic stroke. Gait Posture 2013; 38: 428–32.
14. Mansfield A, Fraser L, Rajachandrakumar R, Danells CJ, Knorr S, Campos J. Is perception of vertical impaired in individuals with chronic stroke with a history of 'pushing'? Neurosci Lett 2015; 590: 172–7.
15. Mayfield JA Sugarman JR. The use of the Semmes-Weinstein monofilament and other threshold tests for preventing foot ulceration and amputation in persons with diabetes. J Fam Pract 2000; 49: S17–29.
16. Paci M, Baccini M, Rinaldi LA. Pusher behaviour: a critical review of controversial issues. Disabil Rehabil 2009; 31: 249–58.
17. Pederson PM, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Orientation in the acute and chronic stroke patient: impact on ADL and social activities. The Copenhagen Stroke Study. Arch Phys Med Rehabil 1996; 77: 336–9.
18. Pérennou DA, Amblard B, Laassel M, Benaim C, Hérisson C, Pélissier J. Understanding the pusher behavior of some stroke patients with spatial deficits: a pilot study. Arch Phys Med Rehabil 2002; 83: 570–5.
19. Pérennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, Bronstein AM. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain 2008; 131: 2401–13.

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The authors state that there is no conflict of interest.

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Psychomotor disadaptation syndrome

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Abstract

Psychomotor disadaptation syndrome (PDS) was first described by The Dijon School of Geriatry (France) 30 years ago, and named "psychomotor regression syndrome". The initial clinical description has not changed. However, progress has been made in both the understanding of its physiopathology and in its management, hence the change in its name to PDS in the late 1990s. Since the early 2000s, another name, frontal-sub-cortical dysfunction syndrome, has been used to designate this entity.

PDS is the decompensation of postural function, gait and psychomotor automatisms linked to the impairment of posture and motor programming. This impairment is due to frontal-sub-cortical lesions. PDS is characterized by retropulsion, non-specific gait disorders, neurological signs (including akinesia, reactional hypertonia, and impaired reactive postural responses and protective reactions) and psychological disturbances (fear of standing and walking as an acute feature or slowness of cognitive processing and anhedonia as a chronic feature).

The occurrence of PDS is linked to the combination of three factors implicated in the decrease in functional reserves due to the deterioration of frontal-sub-cortical structures: ageing, chronic affections (mainly degenerative or vascular), and acute organic or functional factors which induce a reduction in cerebral blood flow.

Multidisciplinary management, including medical, motor physiotherapy and psychological approaches, is indispensable for PDS.

Keywords: psychomotor disadaptation syndrome, frontal-sub-cortical dysfunction syndrome, retropulsion, falls, elderly

Introduction

The psychomotor disadaptation syndrome (PDS), which represents a posturo-motor decompensation due to the programming disorder of posture and movement, was initially described in 1986 by the Dijon team of *Prof. Gaudet* under the name "psychomotor regression syndrome" [8]. Although clinical features of PDS have not changed since its first description, there have been some progresses in its physiopathology and management. A very close relationship between PDS and frailty criteria has been proved. Moreover, the key role of sub-cortico-frontal dysfunctions in the appearance of PDS was recognized [17, 18, 20].

Although described for more than 30 years and well known by geriatricians, there is no epidemiological study on PDS. However, its frequency seems high and can sometimes reach 75 % in acute geriatrics and geriatric rehabilitation units [17].

Retropulsion, postural abnormalities and atypical gait impairment appeared to be main clinical characteristics of PDS in frail elderly persons. In PDS as in pusher syndrome there is a postural perturbation with a misrepresentation of verticality but in PDS the deviation is observed in anterior-posterior direction and we cannot find a link to a specific disease as in post stroke patients.

Pathophysiological hypotheses of psychomotor disadaptation syndrome

The levels involved in posture maintenance include neuro-sensory afferences, central processing and storage of information as well as motor effectors.

In PDS, the decompensation of motor functions seems mainly due to subcortico-frontal impairments. Indeed, subcortical lesions, such as leukoaraiosis, vascular abnormalities of the white matter [17], and enlargement of the ventricles, are more frequently found, and with greater severity, in cerebral imaging of patients with PDS [18]. However, a correlation between white matter abnormalities and posture and gait disorders on the one hand [21], and executive behavior disorders on the other hand [3] has been decribed in the literature.

These alterations of subcortical-frontal circuits, which represent the common denominator of a priori different situations described below, cause a disturbance of the postural and motor programming.

Indeed, the subcortical structures, in particular the white matter, are relay zones of the sensory informa-

tion addressed to the data processing centers. Thus, the quality of the neurosensory messages will be deteriorated in case of alteration of the white matter with as consequence, the central treatment, which allows the activating of an adequate postural or motor response, in based on wrong neurosensory information. The result is non-adapted postural or motor responses to the situation. Finally, the subcortical lesions found during PDS are related to ischemia and chronic hypoxia [1, 5, 6, 9].

The physiopathology of the PDS corresponds to the decompensation theory of major geriatric syndromes, involving three cumulative components (1+2+3), aging, chronic pathologies and acute factors [2]. The leukoaraiosis prevalence increases with aging and reaches 90%, after 80 years [24].

Among the chronic affections or situations (predisposing factors) altering the subcortico-frontal structures, two groups of diseases particularly predispose to PDS. First, there are degenerative pathologies, mainly parkinsonian syndromes including idiopathic Parkinson's disease (PD), multisystemic degeneration, Steele-Richardson's disease and Lewy body disease [19]. Other causes are subcortical vascular injuries secondary to hypertension, atrial fibrillation and diabetes that entail leukoaraiosis, deficiencies or stroke [12]. In addition, other situations, such as normal pressure hydrocephalus or chronic depression support PDS. All these chronic pathologies are risk factors of PDS. However, the latter will occur only in the presence of at least one acute factor.

Acute or precipitating factors, leading to the appearance of symptoms of PDS through clinical decompensation, are functional or organic. Functional factors included falls, non-use or the bed rest [11]. Organic factors are fever, dehydration, some metabolic disorders, hypotension including orthostatic hypotension, and causes entailing hypoxia or decrease in cardiac output [11]. Finally, some drugs (benzodiazepines, antipsychotics and central analgesics) can induce PDS.

Fig. 1: Backward disequilibrium characterized by a posterior trunk tilt in sitting position

Fig. 2: Backward disequilibrium characterized by a posterior trunk tilt in standing position

Psychomotor Disadaptation Syndrome – clinical signs

The PDS combines postural disturbances, non-specific gait disorders, neurological abnormalities and psycho-behavioural disturbances [14, 19 20].

Postural disorders

Retropulsion is the major sign of PDS. It corresponds to a backward projection of the trunk in the seated position **(Fig. 1)**, a loss of the anterior projection of the trunk with a position of the feet forward during sit-to-stand and a tendency to the fall back in upright position **(Fig. 2)**. This results in a projection of the center of mass behind the support polygon, resulting in insecurity during walking and inefficiency of basic activities (raising from a seat, …). Often the patients with PDS use the knee flexion as a means to counterbalance the backward disequilibrium.

Motor abnormalities

Motor abnormalities include gait disorders and neurological signs.

Gait disturbances are not specific of PDS and reflect postural instability. They can be found in any pathological situation affecting balance. Among them, there is "freezing", reflecting the sub-cortico-frontal dysfunction, a gait with small slipped steps with no unwinding the foot on the ground and an increase in bipodal support time.

Neurological signs in PDS can be divided into two groups:

- That of symptoms related to global subcortical damage. Among them are axial akinesia and oppositional hypertonia, variable in time and according to the traction exerted on the limb.
- That of the signs reflecting the alteration of the postural automatisms, among them a reduction or even a loss of postural adaptation reactions and/or parachute reactions [7].

Psychological and behavioral signs

In the post-fall syndrome, initially described by Murphy and Isaacs [15], the psychological signs reflect functional inhibiting of motor automatisms. Fear of falling, which is strongly linked to low self-confidence, is usually associated with PDS. This results in a major anxiety and a phobia to the idea to achieve any postural or motor action as sitting to standing and walking. Several behavioral

disorders such as loss of spontaneity, diminished speed in mental functions and lack of motivation are sometimes noted. The chronic form of PDS is characterized by a resignation with executive or conation disorders, such as bradyphrenia, indifference, apathy, abulia and demotivation [18].

Evolution, prognosis, care and follow-up of elderly with Psychomotor Disadaptation Syndrome

PDS is a geriatric emergency given the risk of a cascade, often dramatic for aged subjects, related to the delay of both diagnostics and the care of acute decompensation factors that can cause serious consequences both functional and vital. Indeed, compared to elderly persons of similar age free of this syndrome, patients suffering from PDS are more at risk of falling.

The care program of patients showing PDS has a twofold objective and consists on the one hand in acting on the factors involved in its physiopathology and on the other hand in correcting its symptoms. This program must be started early, adapted to the patient and achieved by a multiprofessional team with medical care, physiotherapy, occupational therapy, psychological as well as nurse and caregiver components. Although not consistently ensuring functional recovery, appropriate care significantly improves the prognosis for people with PDS. Functional recovery is linked to predisposition and motivation of the patient. After this acute phase, the follow-up is based on this same multidisciplinary dynamic.

The medical assessment consists in the research of all the chronic affections that can lead to subcortico-frontal signs on the one hand and on the other hand of acute factors that require quick care.

Global motor rehabilitation by physiotherapists, trained in the management of frail elderly patients, must also be early and integrate the life goals and motivation of the patient. The primary objective is the relearning of the motor skills necessary for the achievement of basic acts of everyday life. The correction of the retropulsion is underlying motor rehabilitation [10]. The main goal of physical therapy is to facilitate a forward weight shift in standing up from a seated position and in standing to sitting particularly and to reactivate postural control. Moreover, learning to get up from the ground is a key element of motor rehabilitation [16]. The follow-up of people with PDS will be facilitated by Mini Motor Test (MMT) **(Table 1)** [7, 13]. This is a 20-item test that assesses balance and mobility, including transfers from lying and sitting position.

The occupational therapist acts on the maintenance and recovery of postural and motor patterns. Environmental modification and the use of appropriate assistive technology devices contribute to improve the functional capacities of the elderly subjects.

Table 1: Mini Motor Test (MMT)
Cover each item 1 if the answer is "yes" or 0 if the answer is "no"

In bed		**1**	**0**
1	Able to roll onto one side	I__I	I__I
2	Able to rise from lying to sitting position	I__I	I__I
The sitting position			
3	No retropulsion of the trunk	I__I	I__I
4	Able to bend trunk forward	I__I	I__I
5	Able to rise from a chair	I__I	I__I
The standing position			
6	Possible	I__I	I__I
7	Without assistance (material or human)	I__I	I__I
8	Able to stand on two legs with closed eyes	I__I	I__I
9	Able to stand on one leg	I__I	I__I
10	No retropulsion	I__I	I__I
11	Reactive postural responses	I__I	I__I
12	Protective reactions of upper limbs	I__I	I__I
13	Stepping reactions forwards	I__I	I__I
14	Stepping reactions backwards	I__I	I__I
Gait			
15	Possible	I__I	I__I
16	Without assistance (material or human)	I__I	I__I
17	Normal heel-strike	I__I	I__I
18	No knee flexion	I__I	I__I
19	No retropulsion	I__I	I__I
20	Harmonious turn round	I__I	I__I
		Total: .. / 20	
For the two questions below answer yes or no			
• Did the patient have one or more falls in the previous 6 months?			
• Can the patient rise from the floor?			

Support by the psychologist helps to control the fear of standing position and walking but also against bradyphrenia and demotivation [10].

The involvement of the paramedical team, with expertise in the care of older patients, is done through both standard and specific care. It consists of the regular psychic and motor stimulation of the patients.

Prevention of PDS

To prevent the occurrence of PDS, it is essential to identify markers of subcortico-frontal frailty. Their presence must lead to a medical and motor monitoring of the elderly person, the objective being the best stabilization of chronic situations predisposing to PDS. Prevention by physiotherapy concerns elderly people at home as well as those hospitalized.

Conclusion

The number of very old and frail people will continue to grow, hence there will be an increase of PDS patients. Faced with the many situations and pathologies contributing to to the occurrence of PDS, it is essential to best adapt the care programs of this syndrome, which depend on the objectives and expected functional prognosis specific to each patient. Prevalence studies and a developing of the knowledge of the pathophysiology of PDS are thus crucial, as well as the assessment of its therapeutic management by research studies with large patient samples.

Acknowledgements

With this article, we wish to pay a heartfelt tribute to *Prof. Pierre Pfitzenmeyer*, one of the first descriptors of PDS, who unfortunately left us more than seven years ago.

References

1. Bakker SL, de Leeuw FE, de Groot JC, Hofman A, Koudstaal PJ, Breteler MM. Cerebral vasomotor reactivity and cerebral white matter lesions in the elderly. Neurology 1999; 52: 578–83.
2. Bouchon JP. 1+2+3 ou comment tenter d'être efficace en gériatrie? Rev Prat 1984; 34: 888–92.
3. de Groot JC, de Leeuw FE, Oudkerk M, van Gijn J, Hofman A, Jolles J et al. Cerebral white matter lesions and cognitive function: the Rotterdam Scan Study. Ann Neurol 2000; 47: 145–51.
4. de Leeuw FE, de Groot JC, Achten E, Oudkerk M, Ramos LM, Heijboer R et al. Prevalence of cerebral white matter lesions in elderly people: a population based magnetic resonance imaging study: the Rotterdam scan study. J Neurol Neurosurg Psychiatry 2001; 70: 9–14.
5. Fernando MS, Simpson JE, Matthews F, Brayne C, Lewis CE, Barber R et al. White matter lesions in an unselected cohort of the elderly: molecular pathology suggests origin from chronic hypoperfusion injury. Stroke 2006; 37: 1348–9.
6. Fu JH, Lu CZ, Hong Z, Dong Q, Ding D, Wong KS. Relationship between cerebral vasomotor reactivity and white matter lesions in elderly subjects without large artery occlusive disease. J Neuroimaging 2006; 16: 120–5.
7. Gaudet M, Mazoyer B, Tavernier-Vidal B, Pfitzenmeyer F, Mourey F. Etude des réactions d'adaptation posturale du sujet âgé chuteur et non chuteur. Rev Geriatr 1990; 15: 5–9.
8. Gaudet M, Tavernier B, Mourey F, Tavernier C, Richard D. Le syndrome de régression psychomotrice du vieillard. Med Hyg 1986; 44: 1332–6.
9. Liao D, Cooper L, Cai J, Toole J, Bryan N, Burke G et al. The prevalence and severity of the white matter lesions, their relationship with age, ethnicity, gender, and cardiovascular disease risk factors: the ARIC Study. Neuroepidemiology 1997; 16: 149–62.
10. Manckoundia P, Gerbault N, Mourey F, d'Athis Ph, Nourdin C, Monin MP et al. Mutidisciplinary management in geriatric day hospital is beneficial for elderly fallers: a prospective study of 28 cases. Arch Gerontol Geriatr 2007; 44: 61–70.
11. Manckoundia P, Mourey F, Tavernier-Vidal B, Pfitzenmeyer P. Syndrome de désadaptation psychomotrice. Rev Med Interne 2007; 28: 79–85.
12. Manckoundia P, Ntari Soungui E, Tavernier-Vidal B, Mourey F. Syndrome de désadaptation psychomotrice. Geriatr Psychol Neuropsychiatr Vieil 2014; 12: 94–100.
13. Mourey F, Camus A, d'Athis Ph, Blanchon MA, Martin-Hunyadi C, de Rekeneire N, et al. Mini motor test: a clinical test for rehabilitation of patients showing psychomotor disadaptation syndrome (PDS). Arch Gerontol Geriatr 2005; 40: 201–11.
14. Mourey F, Manckoundia P, Martin Arveux I, Tavernier-Vidal B, Pfitzenmeyer P. Psychomotor disadaptation syndrome. A new clinical entity in geriatric patients. Geriatrics 2004; 59: 20–4.
15. Murphy J, Isaacs B. The post-fall syndrome. A study of 36 elderly patients. Gerontology 1982; 28: 265–70.
16. Passeron A, Perreira Rocha E, Dommane L, Capron L. Apprentissage de la technique pour se relever du sol chez des patients âgés hospitalisés. Étude prospective en médecine interne. Presse Med 2005; 34: 1623–8.
17. Pfitzenmeyer P, Manckoundia P, Rouaud O, Foll-Garcia MB, Giroud M, Mourey F. Actualités sur le syndrome de dysfonctionnement sous-cortico-frontal chez les patients gériatriques. Rev Geriatr 2008; 33: 703–12.
18. Pfitzenmeyer P, Martin-Hunyadi C, Mourey F, d'Athis P, Baudouin N, Mischis-Troussard C. Cardiovascular characteristics and cerebral CT findings in elderly subjects with psychomotor disadaptation syndrome. Aging Clin Exp Res 2002; 14: 100–7.
19. Pfitzenmeyer P, Mourey F, Manckoundia P, Tavernier-Vidal B. La désadaptation psychomotrice. Rev Geriatr 2003; 28: 597–600.
20. Pfitzenmeyer P, Mourey F, Tavernier B, Camus A. Psychomotor disadaptation syndrome. Arch Gerontol Geriatr 1999; 28: 217–25.
21. Whitman GT, DiPatre PL, Lopez IA, Liu F, Noori NE, Vinters HV, et al. Neuropathology in older people with disequilibrium of unknown cause. Neurology 1999; 53: 375–82.

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The authors state that there is no conflict of interest. The corresponding author assures that the topic was presented independently and product unbiased. There are no connections to a company that manufactures or sells a named product or a competing product.

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The effect of trunk exercises on the perception of verticality after stroke: A pilot study

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Abstract

Ten to 40 % of stroke patients exhibit severe balance impairments known as pusher behaviour. Patients with pusher behaviour often have a contralesional pelvic tilt suggesting an over activity within trunk muscles. As pusher behaviour is also highly correlated with misperception of verticality, the question arises whether trunk exercises can improve perception of verticality after stroke.

This pilot study is an assessor blinded, randomized, controlled trial with a control group receiving conventional treatment with additional passive mobilizations of the hemiplegic upper limb while supine. The experimental group received conventional treatment with additional trunk exercises with an emphasis on trunk muscle strength, coordination, symmetry, axial extension and selective movements of the trunk. To assess perception of verticality, the subjective visual (SVV) and subjective postural vertical (SPV) test were administered together with the Trunk Impairment Scale to measure trunk performance.

In total, 39 patients participated in this study of which 6 patients showed pusher behaviour. Trunk exercises showed no interaction effect on the SVV and SPV in the non-pusher group.The interaction effect in the pusher group could not be analysed because of the small amount of patients showing pusher behaviour. However, all pushers improved significantly over time on the SPV.

Yet, the experimental group improved significantly more on the TIS than the control group. This indicates that the trunk training was effective, even though it did not affect verticality perception. As a result, it seems that no general relationship between trunk instability and verticality perception can be observed in stroke patients. However, this could be different for patients with pusher behaviour which justifies further research within this subject.

Keywords: stroke, trunk training, perception of verticality, pusher behaviour

Introduction

Postural control in humans requires a complex interaction of musculoskeletal and neural processes [17]. One of these neural processes is the higher-level processing of sensory information into a body-centred representation in the gravitational environment. The orientation of the body relative to the gravitational vector is crucial to ensure postural control. However, perception of the physical vertical is often impaired after brain lesions [9]. This is of high importance since verticality perception has been shown to be highly related to balance control after stroke [2].

Moreover, balance control is often affected after stroke. Yet, a significant amount of stroke patients exhibit severe balance impairments referred to as pusher behaviour. The incidence of this pusher behaviour among the stroke population is reported as being between 10 % and 40 %, however, the wide range of varying definitions and scales used for assessment makes comparison between studies challenging [3, 5, 12]. These patients actively push away from the non-hemiplegic side and resist any attempt at passive correction of this posture towards or over the mid-line of the body to the non-hemiplegic side. Consequently, when sitting, they lean to the contralesionalside. At the pelvic level, the orientation of the pelvis is more tilted to the contralesional side in pushers than in non-pushers [13]. This could lead to the assumptions that the non-hemiplegic side shows a certain over-activity especially as pusher behaviour is an active compensation of the feel of imbalance when sitting upright. Evidence suggests that a misperception of verticality is one of the causal mechanisms underlying the pusher behaviour [7, 16].

As verticality misperceptions can lead to abnormal postural responses, tilted body postures can also influence perception of verticality [10, 14]. Indeed, head-on-body position has a specific effect on verticality perception, which is called the A- or E-effect [11]. This means that overall motor control of muscles responsible for axial extension against gravity will be crucial to enhance accurate verticality estimation.

As there seems to be a mutual link between perception of verticality and trunk performance, the question raises whether trunk exercises can have a positive effect on verticality perception. Especially since trunk exercises after stroke have a positive effect both on trunk performance as well as on measures of stand-

ing balance and gait [15]. This is probably the result of the supportive and stabilizing role of the trunk during stance and sitting. As trunk exercises mainly focus on symmetry, selective movements, coordination, and proprioception of the trunk it can be hypothesized that trunk exercises could have a beneficial effect on perception of verticality after stroke. Therefore, this study aims to investigate whether trunk exercises will improve verticality perception in both pushers as non-pushers after stroke.

Methods

Patients

All patients with a history of first stroke attending a rehabilitation program at the rehabilitation hospital Revarte (Antwerp, Belgium) were eligible for inclusion. Exclusion criteria were an age of 85 and older, more than 4 months post-onset, acute low back pain, and orthopaedic and neurological disorders that could influence postural control. Furthermore, patients suffering from communication disorders that interfered with the protocol were excluded. Patients suffering from brainstem, cerebellar as well as multiple lesions were excluded. After confirming the definite diagnosis of stroke based on CT and/or MRI scans, patients' age, days post-stroke, side and location of the lesion were collected. Participants signed an informed consent before participation. Approval was given by the ethics committee.

Design

This pilot study was an assessor-blinded randomized, controlled trial. Patients in the experimental and control groups received the conventional multidisciplinary stroke rehabilitation program provided by the rehabilitation hospital. The conventional treatment program is patient-specific and consists mainly of physiotherapy, occupational therapy, and nursing care. Therapists combine elements from different neurological treatment concepts but the main emphasis is on the neurodevelopmental treatment concept and on motor relearning strategies.

In addition to conventional therapy, patients received training to improve tru'ncal function (experimental group) or exercises for the upper limb (control group). Both groups received 16 hours of additional training over 8 weeks (30 minutes four times a week). This amount of additional therapy is based on a meta-analysis about augmented exercises training to improve ADLs or gait [8].

Intervention

The additional trainingfor the experimental group focused on trunk muscle strength, coordination, symmetry, axial extension and selective movements of the trunk. Patients of the control group received passive mobilization of the upper limb while supine. A small group of trained therapists delivered the same protocol in order to reduce variability in treatment. All therapists were randomly assigned to a patient. The conventional therapist for that patient was blinded for the experimental intervention. Progression was based on the patients' level of performance. (see **appendix 1**)

Outcome measures

Subjective visual vertical (SVV)

The DifraVertitest type D107201 (Difra, Welkenraedt, Belgium) was used for SVV assessment. The device has an accuracy of 0.1°. A laser bar was projected vertically at a distance of 2.5 m on an opposing wall with the centre of rotation of the laser bar on an altitude of 1.5 m. The device was calibrated in this position, approximating the average altitude of the participants' eyeline when seated. The patients are seated in front of the device on a fixed chair without any arm- or backrests. The room was darkened and a five minutes of waiting period was given allowing the subject to adjust to the darkness. Both researcher and participant obtained a remote control to rotate the laser bar either clockwise (right) or counter clockwise (left). The researcher's remote control showed

Appendix 1: Additional trunk exercises

	Supine position	Sitting position
Selective muscle strengthening 20'	• Lifting pelvis in crook lying with both feet supported (bridging) • Lifting pelvis in crook lying and place it consequently left and right of midline. • Lifting shoulder girdle symmetrically and asymmetrically from table in crook lying	• Anterior and posterior tilt of the pelvis • Selective lengthening and shortening of one side of the trunk • Lateral pelvic tilt without losing balance • Rotation of the upper and lower part of the trunk • Rotation of the upper trunk with external resistance to both sides
Functional tasks 10'	• Rolling to affected and non-affected side initiated from shoulder girdle or pelvis	• Reaching within and out of arm reach • Shuffling forward and backward on hard surface • Sitting on unstable platform
Treatment variables	With or without feet support, dual task training, number of repetitions and the amount of visual feedback	

a display with the amount of deviation in relation to the earth's gravitational vector. The researcher made the laser bar invisible and rotated it in a specific angle in relation to the earth vertical. Subsequently, the line was shown after which the patient had to place the line in upright position again with his non-hemiplegic hand using the remote control. The amount of deviation of each starting roll position was different for each trial. A specified order was followed: first the line was placed in 20° counter clockwise, 10° clockwise, 5° counter clockwise and 0° according to the earth vertical, followed by 5° clockwise, 10° counter clockwise and finally 20° clockwise. The patient was asked to hold the head in normal upright position. The clockwise rotation is represented by a positive number and the counter clockwise rotation as a negative number.

Subjective Postural Vertical (SPV)

On the back of the tilting chair, a Mitutoyo digital protractor pro 3600 (Belgium) was mounted. This allowed measurement of the chair tilt in relation to the earth vertical with an accuracy of 0.01°. Patients had no armrests but were strapped to the chair on the waist by a safety belt to prevent the patient sliding from the chair when tilted. The feet were freely hanging without feet support. Both the researcher and patient were given a remote control to rotate the chair clockwise (right) and counter clockwise (left). Movements were restricted in the frontal plane. Before the assessment started, the patient was blindfolded, depriving the subjects of visual information when readjusting the chair to earth vertical. The subject had to place the chair in upright position again by placing the seating surface of the chair horizontal. The same protocol concerning starting roll positions was used in both SVV as well as in the SPV assessment.

Besides these tests, the Scale of Contraversive Pushing (SCP) [6] and the Trunk Impairment Scale (TIS) [18] were administered. The SCP is a scale that measures lateropulsion. In this study the modified cut-off has been used to increase reliability and validity [1]. The TIS is a measure to assess static and dynamic sitting balance and coordination of the trunk. Visuospatial neglect was recorded based on the patients' medical record using a report provided by the neuropsychologist of the rehabilitation hospital. No standardized visuospatial neglect assessment was performed within this pilot study.

Statistics

Descriptive data analysis was performed for the collected variables of the participants. To examine the effect of our randomisation procedure, differences between all variables for the experimental and control groups were evaluated by means of independent t-test or chi-square test for continuous or dichotomous data, respectively. Parametric statistics were used to analyse the normally distributed data. To analyse the results, a general linear repeated measures model was used. The pre-treatment and post treatment results were entered as within-subjects variable 'time', the experimental and control groups were included as between subjects factor 'condition'. Probability values for the variable 'time' would indicate whether there is a significant change between pre-treatment and post treatment assessment. A significant interaction of 'time x condition' would mean that the change between pre-treatment and post treatment evaluation is significantly different between both groups. Since we are especially interested in the magnitude of the error, absolute values were used for SVV and SPV analyses.

Results

In this pilot study, 39 subjects were tested of which 6 showed pusher behaviour. Descriptives are reported in **table 1**.

Pushers and non-pushers were analysed separately. However, due to the low number of pusher patients and a non-equal division between experimental and control group, an interaction effect was only performed for non-pusher patients. Results of the repeated measures analyses are shown in **table 2**.

In the non-pusher group, all patients improved over time on the SVV ($p = 0.02$) but no interaction effect could be observed ($p = 0.38$). For the SPV, patients did not improve over time ($p = 0.42$) and no interaction effect ($p = 0.75$) occurred.

In the pusher group, only one patient was randomly allocated to the control group and showed a large deviation on the SVV. The SPV assessment was not possible in this patient because of safety issues. In the experimental group, 5 pusher patients did not improve over time on the SVV ($p = 0.82$) but they did on the SPV ($p = 0.03$). These results should be treated with caution because of the amount of patients. Therefore, no interaction effect has been analysed.

Table 1: Descriptives

	Experimental group (N = 23)	Control group (N = 16)	P-value
Age	61.94 (13.83)	61.07 (9.01)	0.828
Gender (Women/men)	14/9	7/9	0.563*
Time post-stroke (days)	38.72 (15.09)	32.07 (25.98)	0.366
Paretic side (left/right)	13/10	6/10	0.166*
Type stroke (ischemic/Hemorrhage)	18/5	12/4	0.461*
Visuospatialneglect (Yes/no)	13/23	10/16	0.549*
Pushers/non-pushers	5/18	1/15	AS**

* Chi-square test, **Analysed separately

Table 2: Outcome measures in the experimental and control group

	Pretreatment		Posttreatment		p-value	
Outcome measures non-pusher	Experimental (N = 18)	Control (N = 16)	Experimental (N = 18)	Control (N = 16)	Time	Time*condition
Subjective Visual Vertical Test	3.73 ± 2.71	5.15 ± 5.51	2.55 ± 1.95	4.60 ± 4.53	0.02	0.38
Subjective Postural Vertical Test	1.58 ± 1.43	2.80 ± 2.70	1.79 ± 1.40	2.90 ± 2.30	0.42	0.75
Trunk Impairment Scale	10.06 ± 3.69	10.40 ± 4.42	18.78 ± 3.53	13.27 ± 3.79	< 0.001	< 0.001
Outcomemeasures pusher	Experimental (N = 5)	Control (N = 1)	Experimental (N = 5)	Control (N = 1)	Time	Time*condition
Subjective Visual Vertical Test	4.95 ± 4.60	15.66 ± 12.43	4.53 ± 4.00	15.66 ± 12.40	0.82	NDA*
Subjective Postural Vertical Test	5.44 ± 9.01	NDA*	2.15 ± 1.71	NDA*	0.03	NDA*
Trunk Impairment Scale	8.00 ± 2.51	0.00	16.4 ± 3.74	2.00	< 0.001	NDA*

Values are presented as mean ± standard deviation. P values are the result of the general linear repeated measures model.
Significance level p=0.05; *No data available

Discussion

In the current sample of stroke patients, additional trunk exercises had no effect on the perception of verticality. Since perception of verticality is influenced by body position and all patients improved significantly on the Trunk Impairment Scale, this outcome is rather unexpected. In addition, the chair we use for SPV assessment does not support the trunk of the patient, which means that trunk performance is probably more important to reset the chair vertically with a freely moving trunk. Although we expected an higher impact of our truncal exercises, two arguments can support our findings.

At first, in the non-pusher group, the deviations on both the SVV and SPV are small to moderate, leaving less room to improve by the trunk rehabilitation [4]. Moreover, in SVV assessment, both the experimental and control group significantly improved over time, even without trunk rehabilitation. Secondly, although trunk exercises can enhance sensory input through the trunk and neck sensors, impaired perception of verticality is not associated to the input but to the processing level. This could elucidate the fact that perception of verticality emanates from the construction of an egocentric reference frame in the gravitational environment, which is the result of higher-order processing of multiple sensory input.

In the pusher group, although only a few patients were included, mean scores were higher compared to the non-pusher group. Especially in the pusher patient from the control group, SVV measurement was divergent with a lot of variation between trials compared to the other patients. In this patient, SPV assessment was impossible because of safety reasons as the patient tilted the chair more than the starting roll position of 20°, which would induce a fall. Interestingly, this patient exhibited severe trunk impairments scoring 0 on the TIS at inclusion and did not significantly improve during the 8 weeks of conventional treatment (TIS score 2). In the pusher group, a significant time effect could be observed in the SPV assessment in contrast with the non-pusher group. This suggests that trunk performance could be a valid rehabilitation approach to realign the patients with the midline and improve their verticality perception especially at the level of the trunk. Although this treatment is rather based on a symptomatic approach, trunk exercises could enhance visuospatial processing in the brain and could have an influence on other attentional deficits such as neglect, often associated with pusher behaviour [13]. These theories are especially hypothetical as further studies need to implement more samples to further elaborate on these findings of this pilot study. However, it should be highlighted that the experimental group improved significantly more on the TIS than the control group. This indicates that the trunk training was effective, even though it did not affect verticality perception. As a result, it seems that no general relationship between trunk instability and verticality perception can be observed in stroke patients. However, this might indeed be different for patients with pusher behaviour.

In the present study, the impact of visuospatial neglect has been poorly investigated. As visuospatial neglect and pusher behaviour are closely linked, the presence of visuospatial neglect could have a significant impact on the results of this study. Therefore, more variables, such as visuospatial neglect, should be included in further studies to have a better understanding of sensorimotor training on perception of verticality.

Conclusion

Trunk exercises showed no additional effect in the improvement of verticality perception after stroke in both pusher as well as non-pusher patients. The results of this pilot study, however, raises new questions justifying more research on the interaction between trunk performance, pusher behaviour, and perception of verticality after stroke.

References

1. Baccini M, Paci M, Rinaldi LA. The scale for contraversive pushing: A reliability and validity study. Neurorehabil Neural Repair 2006; 20(4): 468–72.
2. Bonan IV, Hubeaux K, Gellez-Leman MC, Guichard JP, Vicaut E, Yelnik AP. Influence of subjective visual vertical misperception on balance recovery after stroke. J Neurol Neurosurg Psychiatry 2007; 78(1): 49–55.
3. Clark E, Hill KD, Punt TD. Responsiveness of 2 scales to evaluate lateropulsion or pusher syndrome recovery after stroke. Arch Phys Med Rehabil 2012; 93(1): 149–55.
4. Conceicao LB, Baggio JAO, Mazin SC, Edwards DJ, Santos TEG. Normative data for human postural vertical: A systematic review and meta-analysis. PLoS One 2018; 13(9): e0204122.
5. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke "pushing": natural history and relationship to motor and functional recovery. Stroke 2004; 35(12): 2873-8.
6. Karnath HO, Brotz D. Instructions for the Clinical Scale for Contraversive Pushing (SCP). Neurorehabil Neural Repair 2007; 21(4): 370–1; author reply 1.
7. Karnath HO, Ferber S, Dichgans J. The origin of contraversive pushing: evidence for a second graviceptive system in humans. Neurology 2000; 55(9): 1298–304.
8. Kwakkel G, van Peppen R, Wagenaar RC, Wood Dauphinee S, Richards C, Ashburn A et al. Effects of augmented exercise therapy time after stroke: a meta-analysis. Stroke 2004; 35(11): 2529–39.
9. Lafosse C, Kerckhofs E, Vereeck L, Troch M, Van Hoydonck G, Moeremans M, et al. Postural abnormalities and contraversive pushing following right hemisphere brain damage. Neuropsychol Rehabil 2007; 17(3): 374–96.
10. Mittelstaedt H. Somatic graviception. Biol Psychol 1996; 42(1–2): 53–74.
11. Müller GE. Über das aubertsche phänomen. Z Sinnesphysiol 1916: 109–246.
12. Pedersen PM, Wandel A, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Ipsilateral pushing in stroke: incidence, relation to neuropsychological symptoms, and impact on rehabilitation. The Copenhagen Stroke Study. Arch Phys Med Rehabil 1996; 77(1): 25–8.
13. Perennou DA, Amblard B, Laassel el M, Benaim C, Herisson C, Pelissier J. Understanding the pusher behavior of some stroke patients with spatial deficits: a pilot study. Arch Phys Med Rehabil 2002; 83(4): 570–5.
14. Saeys W, Vereeck L, Bedeer A, Lafosse C, Truijen S, Wuyts FL, et al. Suppression of the E-effect during the subjective visual and postural vertical test in healthy subjects. Eur J Appl Physiol 2010; 109(2): 297–305.
15. Saeys W, Vereeck L, Truijen S, Lafosse C, Wuyts FP, Heyning PV. Randomized controlled trial of truncal exercises early after stroke to improve balance and mobility. Neurorehabil Neural Repair 2012; 26(3): 231–8.
16. Saj A, Honore J, Coello Y, Rousseaux M. The visual vertical in the pusher syndrome: influence of hemispace and body position. J Neurol 2005; 252(8): 885–91.
17. Shumway-Cook A WM. Motor control: Translating research into clinical practice. Philadelphia: Wolters Kluwer Health/ Lippincott Williams & Wilkins 2010.
18. Verheyden G, Nieuwboer A, Mertin J, Preger R, Kiekens C, De Weerdt W. The Trunk Impairment Scale: a new tool to measure motor impairment of the trunk after stroke. Clin Rehabil 2004; 18(3): 326–34.

Conflict of interest:
The authors state that there is no conflict of interest.

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Brain imaging studies in pusher behavior: a narrative review

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Introduction

Some patients with neurological disorders actively push away from the non-paretic side of their body, moving their weight across the midline of the body toward the paretic side. This behavior impairs their postural balance to such a degree that they are often unable to neither sit nor stand. Typically these patients resist any attempt to transfer weight to their non-paretic body side. This symptom is called pusher behavior (PB) [5]). From longitudinal studies we know that PB hampers and prolongs the rehabilitation process. Patients with PB need about 4 weeks longer to reach the same functional outcome level as stroke patients without PB [10] or are only half as efficient and effective in their rehabilitation outcome [9].

There is some evidence that the underlying mechanism of PB is based on a disturbed internal reference frame for the representation of postural verticality. Patients with PB experienced their body as oriented upright when tilted to the ipsilesional side during a test of the subjective postural vertical in sitting [7] or standing [4]. In standing position the severity of PB also correlates with the degree of the impairment of the inner verticality reference [4]. Evidence, however, also exists showing the opposite, i.e., patients experienced themselves as being upright when tilted to the contralesional body side [11]. In contrast to their disturbed perception of upright body posture, orientation perception of the visual world is described as being nearly unaffected [7]. These findings led to the hypothetical assumption of a neural pathway in humans for sensing the orientation of gravity and controlling upright body posture, separate from the one for orientation perception of the visual world [6]. Consequently, lesion of these pathways should lead to PB. This narrative literature review aimed at reviewing previous research on lesion analysis in patients with PB. The objectives of the present review were 1) to identify the imaging methods which have been used so far, 2) to collect information on the diagnostic tools applied to detect patients with and without PB within those studies, 3) to discover which patients were included in these studies with respect to the time since brain injury and, 4) to summarize the findings on brain regions that induce PB.

Methods

We searched the literature for publications mainly addressing the topic of lesion analysis in patients with PB. This topic should be either highlighted in the title, the objectives, or in the results section of the publication. Original articles, letters to the editor, short reports etc. were included, but not abstracts with an insufficient description of the methods used to analyze the images. An important aspect for our review was that the findings of the imaging studies could be clearly attributed to PB or a specific characteristic of PB. So we were looking for research where the methodological decision has been made to compare the brain lesions of patients with PB either to a control group of patients showing no PB, or where a comparison was made between different subpopulations of PB.

Results

Seven studies were included in this narrative review [1, 2, 3, 6, 7, 8, 14]. Patients' characteristics of each study, especially the diagnostic tool with its cut-off criterion, and the accompanying image analysis methods are highlighted in **table 1**.

Karnath et al. [7] investigated 23 patients with PB and 23 matched control patients to investigate the neural representation of PB. They showed that in contrast to controls, the center of lesion overlap in PB patients was located in the ventral posterior and lateral posterior nuclei of the posterolateral thalamus. Laterally and dorsally it extended into the posterior crus of the internal capsule, dorsally also slightly into the corpus of the caudate nucleus.

Karnath et al. [8] analyzed 40 patients with thalamic strokes. Patients were divided into groups with and without PB. A subtraction method was used for analysis which highlights regions that are both frequently damaged in patients with PB as well as typically spared in patients without PB. Using this subtraction method, the authors found that lesion size in patients with PB was significantly larger after a left brain damage (LBD) and tended to be larger in right brain damage (RBD). In iden-

Table 1: Imaging studies on PB: patients' characteristics and image analysis methods

Reference	Participants				Etiology	Diagnostic tool for PB	Time since brain lesion in PB patients		Imaging method/ analysis
	Patients with PB		Controls without PB				phase	[days]	
Karnath, Ferber, Dichgans (2000) [7]	23	8 LBD, 15 RBD	23	8 LBD, 15 RBD	stroke (no diffuse or bilateral brain lesions, no tumors)	SCP (≥1)	acute	n.r.	CT, MRI; software: MRIcron; patients with PB contrasted with patients without PB per hemisphere; center of overlap for voxels that were lesioned in 8 or more patients
Karnath, Johannsen, Broetz, Küker (2005) [8]	14	5 LBD, 9 RBD	26	12 LBD, 14 RBD	thalamic stroke (no diffuse or bilateral brain lesions, no tumors)	SCP (≥1)	acute	LBD: 9.4 (3.8); RBD: 6.2 (2.5)	CT, MRI; software: MRIcron; patients with PB contrasted with patients without PB per hemisphere using subtraction method
Johannsen, Broetz, Naegele, Karnath (2006) [6]	21	10 LBD, 11 RBD	24	12 LBD, 12 RBD	cortical stroke sparing the thalamus (no diffuse or bilateral brain lesions, no tumors)	SCP (≥1)	acute	LBD: 6.5 (5.4); RBD: 5.7 (4.2)	CT, MRI; software: MRIcron; patients with PB contrasted with patients without PB per hemisphere using subtraction method; subtraction method
Ticini, Klose, Nägele, Karnath (2009) [14]	9	5 thalamic (3 LBD, 2 RBD) 4 extra-thalamic (4 RBD)	10	6 thalamic (1 LBD, 5 RBD); 4 extra-thalamic (4 RBD]	stroke (without hemodynamically relevant extracranial stenosis in the internal carotid arteries)	SCP (≥1)	acute/ subacute	Thalamic lesion: 9.6 (6, range 4–18); extra-thalamic lesion: 3.5 (4.7, range 1–10)	MRI (DWI, FLAIR); PWI analysis
Baier, Dietrich (2012) [2]	0	–	19	–	cerebellar stroke	SCP (≥1)	acute	4.0 (2.2)	MRI, no analysis necessary (no patients with PB found)
Baier, Jansen, Müller-Forell, Fechir, Müller, Dieterich (2012) [3]	23	7 LBD, 16 RBD	43	21 LBD, 22 RBD	stroke (no diffuse brain damage or tumor)	SCP (≥1)	acute	LBD: 5 (2); RBD: 7 (3)	MRI (DWI, FLAIR); software: MRIcron; VLBM analyses
Abe, Kondo, Kochiyama, Oouchida, Fujiwara, Izumi (2017) [1]	9 RBD	group 1: recovery from PB (n=3); group 2: no-recovery from PB (n=6)	–	–	stroke	SCP(> 0)	acute/ subacute	24 days after stroke	MRI (DWI, T2WI); software: MRIcron; center of overlap analyses; subtraction method

CT computed tomography; **DWI** diffusion-weighted imaging; **FLAIR** fluid-attenuated inversion-recovery; **LBD** left brain damage; **MRI** magnetic resonance imaging; **n.r.** not reported; **PB** pusher behavior; **PWI** perfusion-weighted imaging; **RBD** right brain damage; **SCP** Scale for Contraversive Pushing; **T2WI** T2-weighted imaging; TBI traumatic brain injury; **VLBM** voxel-wise lesion-behavior mapping. **SCP (≥1)** criterion for the diagnosis of PB means that each tested category had to be ≥1, or greater than 0 when labeled SCP (>0). Values present the number of patients in the specific category. Only the column 'Time since brain lesion in PB patients [days]' presents means and standard deviations in parenthesis.

tifying the thalamic structures as being relevant for PB, a clear separation of lesion overlap between patients with and without PB was found. In both hemispheres, the anatomic structures that were damaged more frequently in the patient groups with PB than in controls centered on the posterior thalamus. In contrast, regions specifically affected more frequently in the control patients than in the pusher patients centered on the anterior thalamus.

Johannsen et al. [6] evaluated 45 patients with and without PB following left or right sided cortical lesions sparing the thalamus. Only very small regions at the left posterior insula and superior temporal gyrus, the left inferior parietal lobule, and the right postcentral gyrus were found to be specific for patients with PB when subtracted from the matched controls.

Using perfusion-weighted imaging (PWI), Ticini et al. [14] investigated the functioning of the structurally intact cortical tissue in patients strokes showing or not showing PB. While diffusion-weighted imaging reveals information about irreversible damaged neural tissue, PWI measures the amount and latency of blood flow reaching different regions of the brain. PWI allows the identification of structurally intact but abnormally perfused brain tissue. Nineteen patients were investigated, 9 with and 10 without PB. Groups were split into patients with thalamic and extra-thalamic brain lesions. In the group of patients with and without PB who suffered from

a thalamic stroke, only a few voxels in single patients were found to be malperfused though structurally intact. Thus, the authors concluded that the patients with PB following thalamic lesions did not show a systematic involvement of dysfunctional brain areas in addition to the ones found to be structurally damaged. In patients with extra-thalamic brain lesions, the subtraction images revealed perfusion deficits in the structurally intact inferior frontal gyrus, the middle temporal gyrus, the precentral gyrus, the inferior parietal lobule, and the parietal white matter. Further, small parts of the callosal body, the temporal white matter, and the superior longitudinal fasciculus were affected. These results showed that in patients with PB having extra-thalamic lesions, the thalamus was neither structurally damaged nor malperfused.

A group of patients with a cerebellar stroke was analyzed by Baier und Dietrich [2]. No patients with PB were found. This finding suggests that lesions in the cerebellum do not cause PB.

Baier et al. [3] performed a voxel-wise lesion-behavior mapping (VLBM) statistical approach which has the potential advantage of allowing lesions to be related to behavioral performance rather than pre-categorizing the patients into dichotomous groups. In this study, Baier et al. [3] performed a VLBM analysis with the score of the clinical Scale for Contraversive Pushing (SCP) as the dependent variable and controlled this analysis for the factors lesion size and neglect. The analysis did not reveal significant voxels for LBD and RBD patients. However, there was as a tendency for lesions located at the posterior insular cortex, the superior temporal gyrus and the operculum as well as the white matter to be the structures possibly associated with the extent of PB. In patients with a LBD, there was a trend towards an association between lesions of the anterior insular cortex, the operculum, the internal capsule, and slightly the lateral thalamus with PB. These findings confirm in part the lesion studies that applied a lesion subtraction technique and described the insular cortex as well as parts of the temporal lobe as key areas. However, in this study no association was found between PB and the posterior thalamus. Notably, the number of patients with a thalamic stroke was very small. Only 5 patients out of a sample of 23 patients with PB had thalamic strokes.

Abe et al. [1] investigated nine patients with PB after a RBD. These nine patients were divided into either a recovery or a no-recovery group, as defined by a SCP score of higher or lower than 1.75 points (no PB) 24 days after the stroke, respectively. Data provided evidence for the association between the delay of recovery and frontal white matter lesions. These regions corresponded to the cortico-spinal tract and superior longitudinal fasciculus.

Not included in this overview was an imaging study by Reding et al. [12] because it is only available as a scientific abstract with insufficient description of the imaging analysis method. Likewise not included were for example two studies where the brain analysis method was not specific enough [10, 13]. Pedersen et al. [10] divided the entire brain into only eight different large sections and calculated the frequency of their involvement in the individual computed tomography lesions. Santos-Pontelli et al. [13] have been the first who investigated brain images in patients showing PB in the chronic phase up to 789 days after brain injury. Patients were assessed until the complete resolution of PB signs. Although they analyzed a control group for comparison, this study was not included in this literature overview as a frequency analysis was limited to a small number of regions of interest, i. e., the thalamus, insula, postcentral gyrus, and posterior parietal region. Both procedures do not allow for a high resolution of PB relevant lesion location. Although Perennou et al. [11] analyzed images of patients with PB, their study was also not suitable for this overview. The main topic of their publication was on disturbed verticality perception in general, but the subgroup of PB patients was not analyzed nor described separately.

Summary

In summary, seven studies have been found to fulfill our search criteria and were included in this narrative review. To contrast patients with PB from control patients or to contrast subgroups of PB, the images were analyzed with the software MRIcron in the five studies [1, 3, 6, 7, 8] focusing on lesion. In one study a perfusion-weighted imaging analysis was performed [14], and in one study no specific analysis was necessary as no patients with PB were found in a sample with cerebellar lesions [2]. Using MRIcron, the center of lesion overlap was calculated separately for the particular groups, and subsequently a subtraction method was applied, and in only one study a voxel-wise lesion-behavior mapping was performed [3].

The diagnostic tool used in all seven studies was the SCP. All but one study [1] used the SCP in which a diagnosis of PB was made when a patient received a total score of 1 in each subcategory. So with respect to the diagnosis of PB, there was high homogeneity across the studies.

When summarizing the findings for the parameter time since injury, five studies included patients in an acute phase after their stroke [2, 3, 6, 7, 8] and two included patients in an acute/very early subacute phase (PB diagnosis at day 24 after the stroke or shorter) [1, 14]. Thus, there is still lack of knowledge about the neural substrates that lead to long-term persistent PB.

The brain areas found to be associated with PB were the ventral posterior and lateral posterior nuclei of the posterolateral thalamus, the internal capsule, the caudate nucleus, or the posterior thalamus when thalamic strokes were analyzed. In patients without

thalamic involvement very small regions of the left posterior insula and superior temporal gyrus, the left inferior parietal lobule, and the right postcentral gyrus were PB relevant regions. Not significantly but with a tendency involved in PB, lesions located at the posterior insular cortex, the superior temporal gyrus, the operculum, and the white matter were found in the VLBM. For patients with LBD the anterior insular cortex, the operculum, the internal capsule, and slightly the lateral thalamus were associated with PB. The cerebellum was reported to have no relevant impact on the behavior. Frontal white matter lesions involving the cortico-spinal tract and superior longitudinal fasciculus were found to be relevant for patients who kept the PB for at least 24 days after their stroke. No systematic involvement of dysfunctional brain areas in addition to the ones found to be structurally damaged could be located in patients after a thalamic stroke. In the group of patients with extra-thalamic lesions, thalamus was neither structurally damaged nor malperfused whereas deficits in the structurally intact inferior frontal gyrus, the middle temporal gyrus, the precentral gyrus, the inferior parietal lobule, and the parietal white matter were revealed.

If there is a neural pathway or network in humans for sensing the orientation of gravity and controlling upright body posture, all the above stated brain areas are probably relevant components.

References

1. Abe H. Delay in Pusher Syndrome Recovery is Related to Frontal White Matter Lesions. Int J Neurol Neurother 2017; 4(1).
2. Baier B, Dieterich M. Pusher syndrome in patients with cerebellar infarctions? J Neurol 2012; 259(7): 1468–9.
3. Baier B, Janzen J, Muller-Forell W, Fechir M, Muller N, Dieterich M. Pusher syndrome: its cortical correlate. J Neurol 2012; 259(2): 277–83.
4. Bergmann J, Krewer C, Selge C, Muller F, Jahn K. The Subjective Postural Vertical Determined in Patients with Pusher Behavior During Standing. Top Stroke Rehabil 2016; 23(3): 184–90.
5. Davies P. Steps to follow: a guide to the treatment of adult hemiplegia. New York: Springer; 1985.
6. Johannsen L, Broetz D, Naegele T, Karnath HO. "Pusher syndrome" following cortical lesions that spare the thalamus. J Neurol 2006; 253(4): 455–63.
7. Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. Proc Natl Acad Sci U S A. 2000; 97(25): 13931–6.
8. Karnath HO, Johannsen L, Broetz D, Kuker W. Posterior thalamic hemorrhage induces "pusher syndrome". Neurology 2005; 64(6): 1014–9.
9. Krewer C, Luther M, Muller F, Koenig E. Time course and influence of pusher behavior on outcome in a rehabilitation setting: a prospective cohort study. Top Stroke Rehabil 2013; 20(4): 331–9.
10. Pedersen PM, Wandel A, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Ipsilateral pushing in stroke: incidence, relation to neuropsychological symptoms, and impact on rehabilitation. The Copenhagen Stroke Study. Arch Phys Med Rehabil 1996; 77(1): 25–8.
11. Perennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, et al. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain 2008; 131(Pt 9): 2401–13.
12. Reding M, David A, Volpe B. 2-44-07 Neuroimaging study of the pusher syndrome post stroke. J Neurol Sci 1997; 150: S129.
13. Santos-Pontelli TE, Pontes-Neto OM, Araujo DB, Santos AC, Leite JP. Neuroimaging in stroke and non-stroke pusher patients. Arq Neuropsiquiatr 2011; 69(6): 914–9.
14. Ticini LF, Klose U, Nagele T, Karnath HO. Perfusion imaging in Pusher syndrome to investigate the neural substrates involved in controlling upright body position. PLoS One 2009; 4(5): e5737.

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Recovery from lateropulsion: The role of lesion side and impairments

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Background

Recovery from lateropulsion after stroke, also known as "pusher syndrome", [5] requires protracted rehabilitation [6] because patients must relearn how to maintain a vertical upright posture prior to mastering activities of daily living and locomotion. This relearning process occurs using a central nervous system that has been impaired by stroke. The impact of stroke deficits on recovery from lateropulsion must be considered when predicting the length of stay for inpatient rehabilitation in order maximize recovery. Studies reporting this impact serve as clinical evidence to support prolonging the length of stay for a patient with lateropulsion.

Objectives

The objective of this review is to describe a series of studies that demonstrate the impact of stroke deficits on recovery from lateropulsion after stroke. These reports support the original supposition of Davies [5] that patients with left and right brain lesions will have different stroke presentations and different ability to recover from lateropulsion.

Review

D'Aquila et al. [4] developed the Burke Lateropulsion Scale (BLS) which allows clinicians to rate the degree of pushing toward the side of weakness after stroke, as well as the resistance met when the clinician tries to return the patient to a vertically aligned position. Davies' original description of pusher syndrome [5] formed the basis for the Burke Lateropulsion Scale. This 18-point scale (0–17) rates the patient in supine rolling, transferring, sitting, standing and walking with 0 indicating no lateropulsion and 17 indicating severe lateropulsion. A cut-off score of '2' on the Burke Lateropulsion Scale indicates that a patient has lateropulsion [4]. The studies presented below use a BLS score within 1 week of admission to the rehabilitation unit of '2' to delineate that patients have lateropulsion. **Figure 1** outlines salient questions and findings.

Babyar, White, Shafi, and Reding [3] conducted a retrospective chart review of patients with unilateral ischemic stroke. Patients with lateropulsion (BLS ≥ 2) were matched to patients without it (BLS = 0 or 1). Matching criteria were: side of stroke, location of stroke (cortical, subcortical, mixed cortical and subcortical), sex, age (± 5 y), admission motor subscale score for the Functional Independence Measure (FIM; ± 6), and interval post-stroke to inpatient rehabilitation admission (± 7 d). The sample included 36 pairs of patients with a mean age of 75 y (SD = 8.7): 21 with right brain damage; 15 with left brain damage. Paired statistical analyses were performed using the aggregate data as well as separate analyses for patients with left or right brain lesions. Patients with lateropulsion had lower upper extremity Fugl-Meyer Assessment of Sensorimotor Recovery after Stroke (Fugl-Meyer) scores for the aggregate ($p < .05$) but not when right and left brain damage pairs were separately analyzed. In contrast, lower extremity Fugl-Meyer scores were statistically different in the aggregate but only for patients with right brain damage in the lesion side analyses. The admission 6-minute walk test showed a similar pattern whereby patients with right brain damage appeared to drive the statistical difference for aggregate data. The patients with lateropulsion and right brain lesions had mean lower extremity Fugl-Meyer scores of 8.1 (SD = 8.8) compared to matches without lateropulsion (Mean = 16.7, SD = 8.3) at admission. This weakness plus the lateropulsion led to mean admission 6-minute walk distances of 19 ft (SD = 37) in contrast to 73 ft (SD = 178) for patients with right brain damage without lateropulsion. Such extreme differences did not exist for patients with left brain lesions [3].

This work demonstrated that patients with lateropulsion had significant stroke deficits, especially if they had right brain damage [3]. The impact of these deficits on recovery from lateropulsion was examined with a sample of patients admitted to inpatient rehabilitation. Rather than look at the severity of the stroke deficits, Babyar, Peterson and Reding retrospectively examined if the number and combination of deficits had an impact on recovery [2]. Deficits were determined through clinical examination at admission to inpatient rehabilitation using standard tests and measures. They were categorized as: visual (hemianopsia or hemispatial visual neglect); proprioceptive (score on the limb placement error test [7, 8]); and, motor (Fugl-Meyer Motor Scores < 34 for the lower extremity or < 66 for the upper extremity).

One hundred sixty-nine patients fell into the following categories: Motor only (1 deficit); Motor + Proprioceptive or Motor + Visual (2 deficits) or Motor + Visual + Proprioceptive (3 deficits). Data were again analyzed for the aggregate as well as separately for left and right brain lesions. Time course of recovery from lateropulsion was judged as the number of days in inpatient rehabilitation until the BLS score moved from a score of 2 or above to a score below 2. Kaplan-Meier survival analysis and log rank tests (Mantel-Cox) examined if the groups had similar time to recovery from lateropulsion ($p < .05$). Patients with 3 deficits had a greater percentage of cases who were still exhibiting lateropulsion as late as 30 days after admission to inpatient rehabilitation. Once again, this larger percentage of cases with 3 deficits who had not yet recovered from lateropulsion was only observed for patients with right brain lesions [2]. The patients with left brain lesions in the 1,2 and 3-deficit groups had similar patterns of recovery from lateropulsion. This study still leaves the open question about how the severity of stroke deficits impacts recovery from lateropulsion.

A patient with lateropulsion undergoing inpatient rehabilitation must overcome their stroke deficits within a framework of allowable days for insurance coverage. Clinicians must advocate for more allowable days in cases where more severe deficits or greater lateropulsion are present. A goal for therapy is to decrease lateropulsion in functional positions. In order to examine the impact of stroke deficits on recovery from lateropulsion, Babyar, Peterson and Reding [1] performed a retrospective case-control study with a final sample of 134 patients with lateropulsion. They dichotomized the sample into those that recovered from lateropulsion (discharge BLS < 2) during their inpatient rehabilitation admission and those that did not (discharge BLS ≥ 2). Forty-nine percent of patients with left brain lesions and 58% with right brain lesions did not recovery from lateropulsion prior to discharge. This dichotomization allowed logistic regression with lateropulsion recovery status (R+ vs R-) as the dependent variable. Based on our prior work, separate logistic regressions were performed for patients with left and right brain lesions. Chi-square tests confirmed that recovery groups were similar in terms of distributions of sex, general lesion location, and incidence of visual-spatial neglect. Independent variables were: age, sex, admission limb placement error score, upper and lower extremity admission Motricity Indices, and cognitive FIM scores. Forward and backward logistic regression confirmed which variables would remain in the model (probability to enter = .05; probability to remove = .10). Mann-Whitney U-tests comparing recovery groups with left brain lesions showed that those who did or did not recover from lateropulsion had similar stroke deficits; admission and discharge BLS scores were worse for the non-recovery group. Logistic regression for patients with left brain lesions showed a relationship of age and right

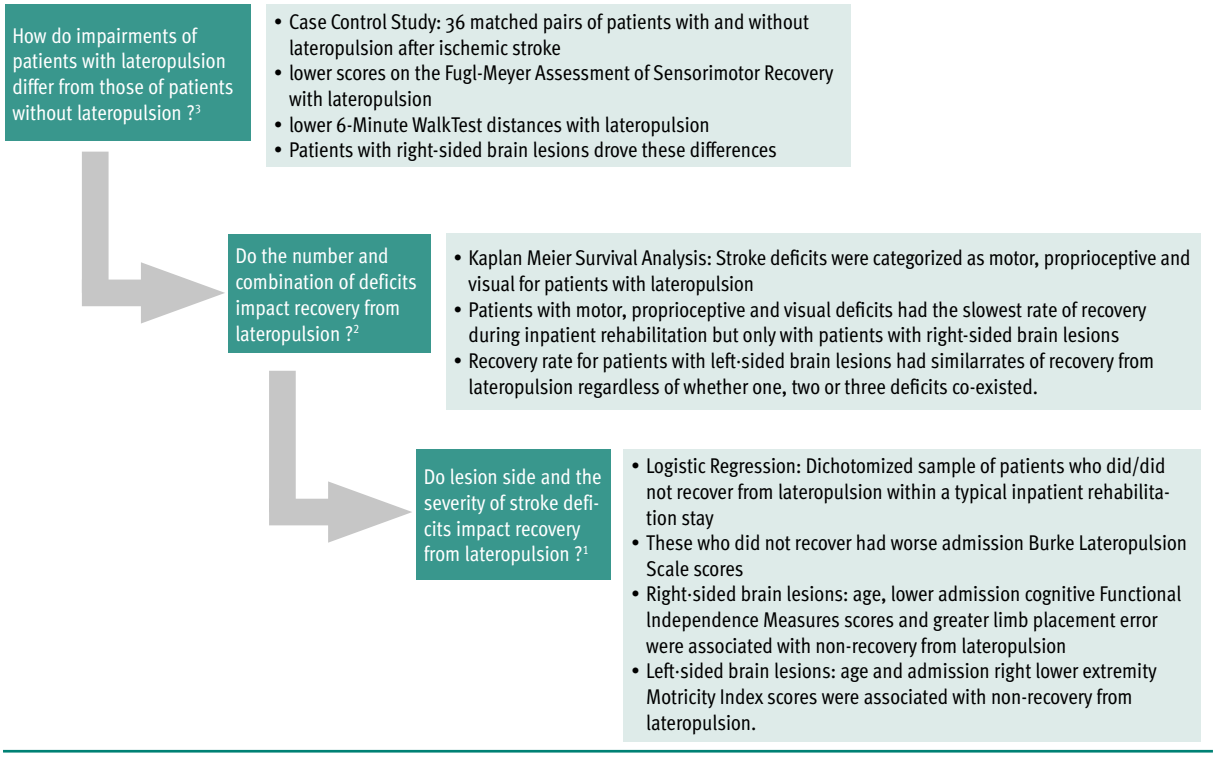

Fig. 1: Questions Asked by the Research Teams and Major Findings[1–3] (Summary of the research questions posed by the research teams 1–3 along with general study design and major findings. One notes that patients with right-sided brain lesions had stronger influences on the outcomes despite similar stroke characteristics to patients with left-sided brain lesions)

lower extremity Motricity Index score at admission to whether or not one recovered from lateropulsion during an inpatient rehabilitation hospitalization. The recovery group comparison for patients with right brain lesions yielded different findings. In addition to worse scores on admission and discharge BLS, patients with right brain lesions who did not recover from lateropulsion had lower admission cognitive and motor FIM scores, and greater deficits on admission limb placement error tests, (Mann-Whitney U-tests). Logistic regression showed a relationship of ability to recover from lateropulsion during inpatient admission to age, admission cognitive FIM score and limb placement error in cases of right brain lesion [1].

Our work highlights some important considerations for treating patients with lateropulsion. First, lesion location has important implications. Patients with left brain lesions fare better in their ability to recover from lateropulsion, especially if they have fewer [2] and less severe deficits [1]. Patients with right brain lesions who have sensory, motor and visual problems paired with lateropulsion need protracted rehabilitation [2]. If their limb placement error and admission cognitive FIM scores are low, planning for delayed discharge is vital [1]. The second treatment consideration is that patients with lateropulsion may have stroke deficits that limit their ability to relearn or reset their egocentric reference system for vertical upright orientation. They may be getting faulty information about limb position or have visual deficits that limit their view of the real world environment. Adding in cognitive issues makes patients with lateropulsion a subset of patients who may profit from additional resources to supplement standard stroke care. These resources may be in the form of sensory integration therapy, sensory or motor electrical stimulation for the trunk, neck or extremities, or non-invasive transcranial brain stimulation during a postural retraining session. The relationship of age to delayed recovery from lateropulson [1] deserves some mention. Age was part of the logistic regressions predicting recovery vs non-recovery from lateropulsion with an episode of care in inpatient rehabilitation, regardless of lesion side. Further exploration is required to determine if advanced age limits new learning or exercise endurance or other factors related to recovery from lateropulsion. Lastly, more experience in the upright position appears important for all patients with lateropulsion. Even if they need more physical support from therapists or from body weight support systems, lateropulsion will decrease during the course of inpatient rehabilitation.

Summary

This link between severity of stroke impairments and delayed recovery from lateropulsion or simply the number of deficits, without considering their severity, demonstrates that these patients lack some fundamental tools to help them re-learn vertical upright posture. This delay will impact restoration of their ability to perform activities of daily living, therefore, their inpatient rehabilitation should be longer than that for patients without lateropulsion. Physical and occupational therapists will need to focus on ameliorating the deficits as well as working on postural control, therefore, increased time in therapy and creative augmented therapy should factor into patient scheduling in cases of lateropulsion.

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References

1. Babyar SR, Peterson MGE, Reding M. Case-control study of impairments associated with recovery from "pusher syndrome" after stroke: logistic regression analyses. J Stroke Cerebrovasc Dis 2017; 26: 25–33.
2. Babyar SR, Peterson MGE, Reding M. Time to recovery from lateropulsion dependent on key stroke deficits: A retrospective analysis. Neurorehabil Neural Repair 2015; 29: 207–13.
3. Babyar SR, White H, Shafi N, Reding M. Outcomes with stroke and lateropulsion: A case-matched controlled study. Neurorehabil Neural Repair 2008; 22: 415–23.
4. D'Aquila MA, Smith T, Organ D, Lichtman S, Reding M. Validation of a lateropulsion scale for patients recovering from stroke. Clin Rehabil 2004; 18:102–9.
5. Davies PM. Out of Line (The Pusher Syndrome). In: Steps to Follow: A Guide to the Treatment of Adult Hemiplegia. Berlin: Springer 1985; 266–84.
6. Pedersen PM, Wandel A, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Ipsilateral pushing in stroke: incidence, relation to neuropsychological symptoms, and impact on rehabilitation. The Copenhagen Stroke Study. Arch Phys Med Rehabil 1996; 77: 25–8.
7. Reding M, Potes E. Rehabilitation outcome following initial unilateral hemispheric stroke. Stroke 1988; 19: 1354–8.
8. Yarnell PR, Boutell Friedman B. Left "hemi" ADL learning and outcome: limiting factors. Neurorehabil Neural Repair 1987; 1: 125–30.

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Pusher behavior
Visual feedback training is helpful

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Pusher behavior is a distinctive clinical disorder and differs from other disturbances of body posture e. g. listing phenomenon or hip pain. Patients push themselves powerful with the help of their non paralyzed extremities to the side of the hemiparesis [3, 5]. They do not pull but push, therefor the naming "pusher behavior" seems to fit better than the naming "lateropulsion". It is also no "syndrome" which is characterized by the combined occurence of different symptoms like pushing behavior and neglect [3]. Premise for the identification of patients with a defined problem was the development of a measure instrument to detect those patients – the Clinical Scale for Contraversive Pushing (SCP) [5]. The scale bases on the observation of body posture during sitting and standing: a) Spontaneous body posture b) Abduction & extension of the non-paretic extremities to increase pushing behavior c) Resistance against correction. A later found forth criterion is an abnormal equilibrium reaction of the non paretic leg during sitting [4].

With the help of the structured diagnosis it was possible to further examine patients with a unique problem. It was found that the pusher behavior characteristically occurs after thalamic brain lesions and is based on a disturbed perception of body posture in relation to gravity [5, 6]. The patients experience their body as oriented vertical when actually tilted to the side but show undisturbed processing of visual inputs determining visual vertical. This knowledge led to the development of a new physiotherapeutic approach for patients with pusher behavior named "visual feedback training" [2]. The visual recognition of vertical structures in the surrounding, which is undisturbed in these patients, is the central treatment element. The therapy should be planned in a structured manner. Patients

1. learn to realize their disturbed perception of erect body position,
2. visually explore their surroundings and own body,
3. learn the movements necessary to reach a vertical body position, and
4. learn to maintain the vertical body position while performing other activities.

Other aspects of disease like hemiparesis should be treated in separate treatment sessions. Patients should train walking in the same intensity like other patients with hemiparesis without pusher behavior. With a therapy bed at the non paralyzed side as a cue to lean on instead of pushing away, it is possible to help affected patients to train walking. The effectiveness of this approach has been shown [1]. Because of the self-determined character of the approach, patients are able to do the training of verticality during sitting even outside of the therapy setting with a lot of repetitions over the whole day.

References

1. Brötz D, Johannsen L, Karnath HO. Time course of "pusher syndrome" under visual feedback treatment. Physiother Res Int 2004; 9: 138–43.
2. Brötz D, Karnath HO. New aspects for the physiotherapy of pushing behaviour. NeuroRehabilitation 2005; 20: 133–8.
3. Davies PM. Steps to follow. A guide to the treatment of adult hemiplegia. Springer, New York 1985, 2000.
4. Johannsen L, Brötz D, Karnath HO. Leg orientation as a clinical sign for pusher syndrome. BMC Neurology 2006; 6: 30.
5. Karnath H-O, Ferber S, Dichgans J. The origin of contraversive pushing: Evidence for a second graviceptive system in humans. Neurology 2000; 55: 1298–304.
6. Karnath HO, Johannsen L, Brötz D, Küker W. Posterior thalamic hemorrhage induces "pusher syndrome". Neurology 2005; 64: 1014–9.

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Non-invasive brain stimulation to treat disorders of human verticality

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Abstract

Brain lesions frequently cause verticality misperception and result in poor postural control and functionality. Although the mechanism of verticality misperception remains incompletely understood, available evidence indicates that a cortical hub at the temporo-parietal junction is implicated. A relatively recent tool for investigating and influencing cortical activity is non-invasive electromagnetic brain stimulation (NIBS), that could plausibly be used to understand and treat disorders of verticality. NIBS can target the temporo-parietal junction, or other cortical areas related to verticality perception, such as parietal cortex, superior and middle temporal gyri, and the post-central gyrus. Here we present an overview of the elementary concepts of NIBS and studies aiming to investigate the underlying mechanisms, and develop treatment strategies for verticality misperception using NIBS. Top-down neuromodulatory effects on verticality perception have been demonstrated, using repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), with promising clinical impact. We further discuss the rationale and necessary features for developing future rTMS and tDCS protocols to treat disorders of human verticality.

Keywords: Transcranial magnetic stimulation, transcranial direct current stimulation, temporo-parietal junction, verticality, postural control, lateropulsion

Human functional movement typically requires maintaining the body in a correct orientation within the limits of stability. The capacity to identify, judge, and interpret orientation of the body and environment in relation to *Earth vertical* is necessary to preserve whole body postural control, as well as accurate arm-hand function, and involves a complex interaction of sensory, motor and cognitive systems. This capacity can be dissociated into three main classifications of verticality perception: (1) subjective visual vertical (SVV), (2) subjective postural vertical (SPV), and (3) subjective haptic vertical (SHV).

The occurrence of verticality misperception after neurological and neuro-otological lesions is widely known. Nearly 10% of a general stroke population [21] and up to 63% of a population with moderate to severe stroke [8] exhibit contraversive lateropulsion ('pushing behavior'), a syndrome caused in part by verticality *mis*perception. Patients with lateropulsion maintain a tilted body position, actively push themselves towards the contralesional side and resist any attempt of passive correction. Moreover, around 90% of patients with vestibular neuritis [17], 94% of patients with unilateral brainstem lesions [9], and 40 to 57% of patients after stroke (regardless the presence of lateropulsion) experience one or more types of verticality misperception, associated with worse postural control and functionality [4, 22, 23, 30, 38]. Therefore, the investigation of verticality misperception and potential treatments form an important area of research with clinical significance and impact.

Scientific advancement in developing potential interventional studies for patients after neurological damage (that also applies to other diseases) is primarily based on observational studies that provide information about the underlying cause, diagnosis, lesion location, range of severity, prognosis and phenomena that influence the behavior. Although several observational studies on verticality misperception and lateropulsion have been conducted, most include small sample size and many features of this disabling deficit remain unclear, for example; 1) the relationship between the type and severity of verticality misperception with recovery from postural imbalance, 2) which sensory and cognitive systems are relevant for the recovery of each type of postural imbalance related to verticality misperception, 3) the microstructural and functional neuroimaging data that can identify the specific areas, connectivity and plasticity of the neural network related to verticality perception, and 4) dominance of each node of this neural network in relation to lesion location and susceptibility to different types of intervention.

In this context, the knowledge of verticality misperception can be advanced using innovative tools for investigating or influencing brain activity such as non-invasive electromagnetic brain stimulation (NIBS). NIBS methods have potential to contribute to all phases of neurological care, i. e., diagnosis, prognosis and therapeutic intervention, including Transcranial Magnetic Stimulation (TMS) and Transcranial Electric Stimulation

(TES), which are established modalities [5, 19]. TMS uses electromagnetic coils to produce a brief electric current and neuronal depolarization of a focal cortical area. The application of single pulse TMS over the primary motor cortex can stimulate the cortical-spinal tract, and surface electromyography can be used to measure the corresponding muscle response [12]. The electromyographic response in relation to the stimulus intensity provides information about the corticospinal excitability. By using a paired-pulse TMS procedure, it is possible to analyze intra-cortical (single coil, same location) and inter-hemispheric interaction (two coils, different but connected locations). Paired-pulse TMS at the same location provides information about cortical interaction elicited by two single pulses with different intensities that, depending on each stimulus intensity and interstimulus interval, indicates net excitatory or facilitatory activity (for review read [10]). The application of repetitive pulses of TMS (rTMS) can increase or decrease the excitability of the stimulated area and its respective neural network, depending on the protocol [25]. Typically, high frequencies (above 5Hz) can increase excitability, and low frequencies (1Hz or less) can decrease excitability. More complex protocols like quadripulse and theta-burst may result in greater and longer-lasting neuromodulatory effects in relation to traditional rTMS procedures [15, 25]. TES methods can include a neuromodulatory strategy that involves weak electrical current passed transcranially (scalp, skull, CSF, dura) to the brain tissue delivered via scalp-surface electrodes, and modulates the neuronal firing rate (without overt synaptic activity). The effects of TES on neuronal excitability depend on the current flow direction. In general, transcranial direct current stimulation (tDCS) induces an increase in neuronal excitability under the anode electrode (orthogonal current flow from the electrode to the brain tissue) and a decrease under the cathode electrode (orthogonal current flow from brain tissue to the electrode) [24]. Therefore, the anatomical characteristics of the cortical region influence the effects of this NIBS modality. Conventional tDCS conditions require sponge electrodes that modulate a large area of the brain. Using multiple "smaller" gel based electrodes, high-definition tDCS (HD-tDCS) can optimize the focality of tDCS by maximizing the current density along the desired directional field in the region of interest. The most common HD-tDCS arrangement is one center electrode surrounded by four returns where the central electrode indicates the polarity of the stimulation. The HD-tDCS also enables the concurrent stimulation of different areas (for review read [11]).

The physiological concept that cortical excitability changes and follows the neuroplastic processes after brain lesions have enhanced the range of possibilities for the investigation and development of treatments for several neurological conditions. Previous studies indicate that motor cortical excitability can predict the functional recovery of stroke patients in the acute phase [32, 34]. Worse functional prognosis has been related to excessive inhibition of the lesioned hemisphere by the non-lesioned homologue area [20, 33], especially after subcortical lesions [35]. These studies have been part of the rationale for the dichotomized usage of non-invasive transcranial neuromodulation techniques over the past years. Most recently, this concept has been challenged by the results of a systematic review [6] and a longitudinal observational study in 22 patients after stroke that found normal interhemispheric inhibition in the acute/subacute period suggesting that interhemispheric imbalance is not necessarily related to a poor recovery, but a result of the underlying recovery processes [37]. The existence of a wide variability of lesions and their respective neuroplastic mechanisms after brain damage among patients requires the development of patient-centered therapeutic strategies with specific type and timing for intervention [7]. The comprehension of verticality perception is far behind the current knowledge about motor function. Future studies should provide information about the inter-hemispheric interaction between the areas related to the verticality perception, and the relationship of the motor cortical excitability with verticality misperception to guide the rationale for neuromodulatory patient-centered protocols for verticality misperception.

The application of TMS or TES for verticality perception modulation is possible due to accessibility of cortical areas of its neural network that can be targeted by these interventions. Neuroimaging description and lesion-behaviour mapping studies have indicated parietal cortex, superior and middle temporal gyrus, temporo-parietal junction, post central gyri, inferior frontal gyrus, occipital cortex, insula, and thalamus as critical areas related to verticality perception, with dominance of the right hemisphere for SVV [4, 5, 9, 19, 22, 23, 30, 38]. Focus on the temporo-parietal junction (TPJ) is rational, since it is a hub for multisensory and cognitive processing and lesion of this area is associated with lateropulsion.

The neuromodulatory effect of TMS and TES can be observed following direct target stimulation, or by remote effects or distant areas of its neural network with or without direct axonal projections [27, 31]. Also, the induction of electric current of the cortex can disturb the neural processing of the stimulated area and induce a transient disruption of the related function in healthy subjects [39]. Based on these principles, some studies have applied low frequency (inhibitory) rTMS protocols over the right TPJ and induced transient misperception of upright in healthy subjects using SVV paradigm with the head tilted (A-effect), and Gabor patch visual detection task [13, 16].

Recently, we applied conventional bipolar bilateral tDCS over the temporo-parietal region in healthy subjects to test if the inter-hemispheric imbalance of these brain

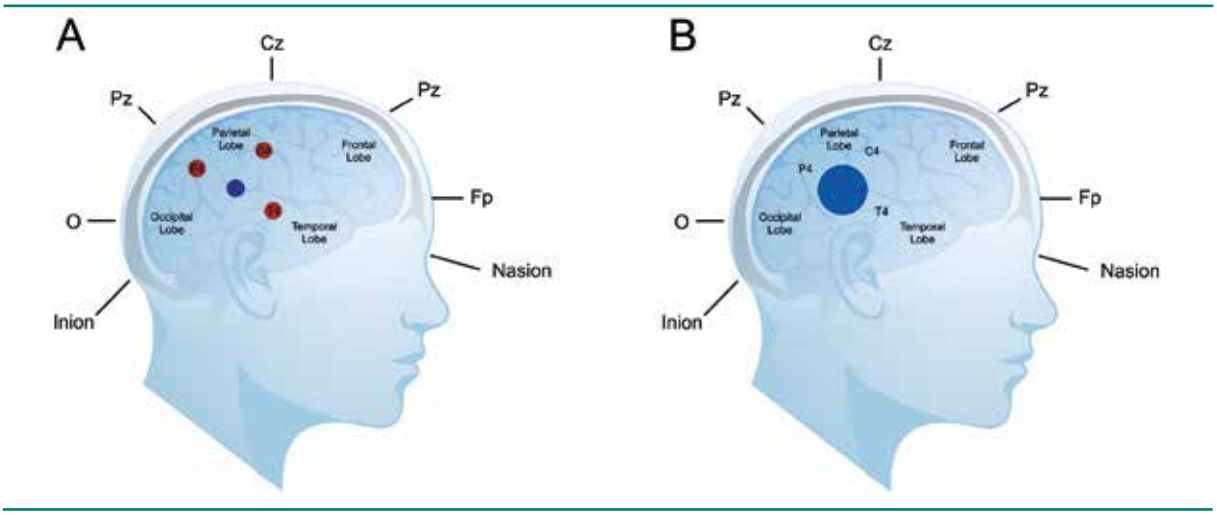

Fig. 1: Schematic representation (not in exact proportion) demonstrating the position of the electrodes for two different transcranial direct current stimulation (tDCS) approaches to target temporo-parietal junction, that can impact human perception of verticality; **A.** unilateral high-definition tDCS (HD-tDCS$_{3x1}$); **B.** bipolar bilateral conventional tDCS (right hemisphere shown).

areas could influence the visual vertical perception [29] **(Figure 1B).** The results indicated a polarity dependent tilt of SVV away from the cathode. The effect magnitude was greater with the HD-tDCS$_{3x1}$ protocol (a central cathode, surrounded by an array of three anodes for unilateral stimulation) over the right TPJ that induced a tilt of the SVV away from the cathode [28] **(Figure 1A).** The cathode-centered HD-tDCS$_{3x1}$ also induced an effect on standing weight bearing asymmetry of healthy subjects with a shift toward the side of HD-tDCS$_{3x1}$ stimulation [28]. Taken together, the effects induced by HD-tDCS$_{3x1}$ transiently reproduced in healthy subjects the perception and postural tilts observed in patients with lateropulsion and indicate that the TPJ is an important node for verticality perception and posture [3, 4, 14]. Moreover, the evidence that the right hemisphere is dominant for verticality perception was confirmed by the observation of greater effects on SVV when the HD-tDCS$_{3x1}$ protocol (at 2 mA for 20 minutes) was applied over the right than the left TPJ (unpublished data). The effects of HD-tDCS over the left hemisphere were also significant and give credence to the hypothesis that patients with verticality misperception after both left or right hemispheric lesions may respond to treatment using this protocol. Pilot studies using conventional bipolar bilateral tDCS [2] and HD-tDCS [1] over the temporo-parietal cortex have indicated change on seated postural oscillations in patients with lateropulsion. Although the small sample size of these studies prevented us from having strong conclusions, they provided evidence for the development of future clinical trials.

The effects of tDCS, HD-tDCS and rTMS applied over TPJ indicate a top-down influence on verticality perception and high-level process of modulation. Galvanic vestibular stimulation, another type of non-invasive transcranial stimulation, can produce a bottom-up influence on verticality misperception [1, 18, 26, 36], and could be considered separate to the present discussion. The optimal NIBS protocol for human verticality manipulation will need to be systematically evaluated examining the factors such as the targeted brain region, phase of recovery and timing within the session to apply the intervention, number of sessions, interval between sessions, type and amount of activity during the session. For rTMS protocols, the additional definition of the frequency of the pulses, number of pulses, time between the pulses, stimulus intensity, type of coil is needed. For tDCS protocols, the type, size, shape, number and position of the electrodes, the duration of stimulation and current intensity will be anticipated to influence the effects [5]. Other models of TES such as pulsed current, alternating current, and random noise stimulation are also promising alternatives for the treatment of verticality misperception and may induce different effects. Furthermore, the consequences of each neuromodulatory strategy on the neural and vascular function, and their relation to the recovery of verticality misperception, are also open questions in this field.

TMS/TES is a safe and effective way to modulate cortical activity. How this can be harnessed to build on our current data for effects of verticality such that clinical populations can benefit, is an exciting area of exploration, and will need careful and detailed experimental approaches. This includes understanding when treatment approaches are not effective, as well as reproducibility studies for effective treatments, both of which are scarce in this field, but necessary to gain traction clinically.

References

1. Babyar S, Santos T, "Will-Lemos" T, Mazin S, Edwards D, Reding M. Sinusoidal Transcranial Direct Current Versus Galvanic Vestibular Stimulation for Treatment of Lateropulsion Poststroke. J Stroke Cerebrovasc Dis 2018; 27(12): 3621–5.
2. Babyar S, Santos-Pontelli T, Will-Lemos T, Mazin S, Bikson M, Tru-

ong DQ, Edwards D, Reding M. Center of Pressure Speed Changes with tDCS Versus GVS in Patients with Lateropulsion after Stroke. Brain Stimul 2016; 9(5): 796–8.
3. Baier B, Suchan J, Karnath HO, Dieterich M. Neural correlates of disturbed perception of verticality. Neurology 2012; 78(10): 728–35.
4. Barra J, Oujamaa L, Chauvineau V, Rougier P, Pérennou D. Asymmetric standing posture after stroke is related to a biased egocentric coordinate system. Neurology 2009; 72(18): 1582–7.
5. Bikson M, Brunoni AR, Charvet LE, Clark VP, Cohen LG, Deng ZD, Dmochowski J, Edwards DJ, Frohlich F, Kappenman ES, Lim KO, Loo C, Mantovani A, McMullen DP, Parra LC, Pearson M, Richardson JD, Rumsey JM, Sehatpour P, Sommers D, Unal G, Wassermann EM, Woods AJ, Lisanby SH. Rigor and reproducibility in research with transcranial electrical stimulation: An NIMH-sponsored workshop. Brain Stimul 2018; 11 (3): 465–80.
6. Boddington LJ, Reynolds JNJ. Targeting interhemispheric inhibition with neuromodulation to enhance stroke rehabilitation. Brain Stimul 2017; 10(2): 214–22.
7. Buchwald A, Calhoun H, Rimikis S, Lowe MS, Wellner R, Edwards DJ. Using tDCS to facilitate motor learning in speech production: The role of timing. Cortex 2019; 111: 274–85.
8. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke "pushing": natural history and relationship to motor and functional recovery. Stroke 2004; 35(12): 2873–8.
9. Dieterich M, Brandt T. Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. Ann Neurol 1993; 33(3): 292–9.
10. Di Pino G, Pellegrino G, Assenza G, Capone F, Ferreri F, Formica D, Ranieri F, Tombini M, Ziemann U, Rothwell JC, Di Lazzaro V. Modulation of brain plasticity in stroke: a novel model for neurorehabilitation. Nat Rev Neurol 2014; 10(10): 597–608.
11. Edwards D, Cortes M, Datta A, Minhas P, Wassermann EM, Bikson M. Physiological and modeling evidence for focal transcranial electrical brain stimulation in humans: a basis for high-definition tDCS. Neuroimage 2013; 74: 266–75.
12. Edwards MJ, Talelli P, Rothwell JC. Clinical applications of transcranial magnetic stimulation in patients with movement disorders. Lancet Neurol 2008; 7(9): 827–40.
13. Fiori F, Candidi M, Acciarino A, David N, Aglioti SM. The right temporoparietal junction plays a causal role in maintaining the internal representation of verticality. J Neurophysiol 2015; 114(5): 2983–90.
14. Genthon N, Rougier P, Gissot AS, Froger J, Pélissier J, Pérennou D. Contribution of each lower limb to upright standing in stroke patients. Stroke 2008; 39(6): 1793–9.
15. Huang YZ, Rothwell JC, Chen RS, Lu CS, Chuang WL. The theoretical model of theta burst form of repetitive transcranial magnetic stimulation. Clin Neurophysiol 2011; 122(5): 1011–8.
16. Kheradmand A, Lasker A, Zee DS. Transcranial magnetic stimulation (TMS) of the supramarginal gyrus: a window to perception of upright. Cereb Cortex 2015; 25(3): 765–71.
17. Kim HA, Hong JH, Lee H, Yi HA, Lee SR, Lee SY, Jang BC, Ahn BH, Baloh RW. Otolith dysfunction in vestibular neuritis: recovery pattern and a predictor of symptom recovery. Neurology 2008; 70(6): 449–53.
18. Krewer C, Rieß K, Bergmann J, Müller F, Jahn K, Koenig E. Immediate effectiveness of single-session therapeutic interventions in pusher behaviour. Gait Posture 2013; 37(2): 246–50.
19. Lefaucheur JP, André-Obadia N, Antal A, Ayache SS, Baeken C, Benninger DH, Cantello RM, Cincotta M, de Carvalho M, De Ridder D, Devanne H, Di Lazzaro V, Filipović SR, Hummel FC, Jääskeläinen SK, Kimiskidis VK, Koch G, Langguth B, Nyffeler T, Oliviero A, Padberg F, Poulet E, Rossi S, Rossini PM, Rothwell JC, Schönfeldt-Lecuona C, Siebner HR, Slotema CW, Stagg CJ, Valls-Sole J, Ziemann U, Paulus W, Garcia-Larrea L. Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS). Clin Neurophysiol 2014; 125(11): 2150–206.
20. Pascual-Leone A, Amedi A, Fregni F, Merabet LB. The plastic human brain cortex. Annu Rev Neurosci 2005; 28: 377–401.
21. Pedersen PM, Wandel A, Jørgensen HS, Nakayama H, Raaschou HO, Olsen TS. Ipsilateral pushing in stroke: incidence, relation to neuropsychological symptoms, and impact on rehabilitation. The Copenhagen Stroke Study. Arch Phys Med Rehabil 1996; 77(1): 25–8.
22. Pérennou D. Postural disorders and spatial neglect in stroke patients: a strong association. Restor Neurol Neurosci 2006; 24(4–6): 319–34.
23. Pérennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, Bronstein AM. Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? Brain 2008; 131(Pt 9): 2401–13.
24. Rawji V, Ciocca M, Zacharia A, Soares D, Truong D, Bikson M, Rothwell J, Bestmann S. tDCS changes in motor excitability are specific to orientation of current flow. Brain Stimul 2018; 11(2): 289–98.
25. Rossi S, Hallett M, Rossini PM, Pascual-Leone A, Group S. o. T. C. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. Clin Neurophysiol 2009; 120(12): 2008–39.
26. Saj A, Honoré J, Rousseaux M. Perception of the vertical in patients with right hemispheric lesion: effect of galvanic vestibular stimulation. Neuropsychologia 2006; 44(8): 1509–12.
27. Sale MV, Mattingley JB, Zalesky A, Cocchi L. Imaging human brain networks to improve the clinical efficacy of non-invasive brain stimulation. Neurosci Biobehav Rev 2015; 57: 187–98.
28. Santos TEG, Favoretto DB, Toostani IG, Nascimento DC, Rimoli BP, Bergonzoni E, Lemos TW, Truong DQ, Delbem ACB, Makkiabadi B, Moraes R, Louzada F, Bikson M, Leite JP, Edwards DJ. Manipulation of Human Verticality Using High-Definition Transcranial Direct Current Stimulation. Front Neurol 2018; 9: 825.
29. Santos-Pontelli TE, Rimoli BP, Favoretto DB, Mazin SC, Truong, DQ, Leite JP, Pontes-Neto OM, Babyar SR, Reding M, Bikson M, Edwards DJ. Polarity-Dependent Misperception of Subjective Visual Vertical during and after Transcranial Direct Current Stimulation (tDCS). PLoS One 2016; 11(3): e0152331.
30. Santos-Pontelli TEG, Pontes-Neto OM, de Araujo DB, Santos AC, Leite JP. Persistent pusher behavior after a stroke. Clinics 2011; 66(12): 2169–71.
31. Siebner HR, Hartwigsen G, Kassuba T, Rothwell JC. How does transcranial magnetic stimulation modify neuronal activity in the brain? Implications for studies of cognition. Cortex 2009; 45(9): 1035–42.
32. Simis M, Di Lazzaro V, Kirton A, Pennisi G, Bella R, Kim YH, Takeuchi N, Khedr EM, Rogers LM, Harvey R, Koganemaru S, Turman B, Tarlacı S, Gagliardi RJ, Fregni F. Neurophysiological measurements of affected and unaffected motor cortex from a cross-sectional, multi-center individual stroke patient data analysis study. Neurophysiol Clin 2016; 46(1): 53–61.
33. Stinear CM, Barber PA, Petoe M, Anwar S, Byblow WD. The PREP algorithm predicts potential for upper limb recovery after stroke. Brain 2012; 135 (Pt8): 2527–35.
34. Stinear CM, Petoe MA, Byblow WD. Primary Motor Cortex Excitability During Recovery After Stroke: Implications for Neuromodulation. Brain Stimul 2015.
35. Thickbroom GW, Cortes M, Rykman A, Volpe BT, Fregni F, Krebs HI, Pascual-Leone A. Edwards DJ. Stroke subtype and motor impairment influence contralesional excitability. Neurology 2015,85 (6): 517–20.
36. Volkening K, Bergmann J, Keller I, Wuehr M, Müller, F, Jahn, K. Verticality perception during and after galvanic vestibular stimulation. Neurosci Lett 2014; 581: 75–9.
37. Xu J, Branscheidt M, Schambra H, Steiner L, Widmer M, Diedrichsen J, Goldsmith J, Lindquist M, Kitago T, Luft AR, Krakauer JW, Celnik PA, Group SS. Rethinking interhemispheric imbalance as a target for stroke neurorehabilitation. Ann Neurol 2019.
38. Yelnik AP, Lebreton FO, Bonan IV, Colle FM, Meurin FA, Guichard JP, Vicaut E. Perception of verticality after recent cerebral hemispheric stroke. Stroke 2002; 33(9): 2247–53.
39. Zmigrod S. The role of the parietal cortex in multisensory and response integration: evidence from transcranial direct current stimulation (tDCS). Multisens Res 2014; 27(2): 161–72.

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Robot assisted gait training for pusher behavior

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Introduction: Pusher Behavior

Since Patricia Davies introduced the term pusher syndrome in 1985, it has been known as a behavior that usually affects severely disabled stroke patients with hemiplegia. As indicated by the alternatively used wording lateropulsion it is characterized by the disturbance of graviception leading to a behavior of actively pushing away from the non-hemiparetic side. Therapists are severely challenged by this behavior since it increases the difficulty of relearning sitting and standing balance as well as gait. It has been shown to slow down the rehabilitation process by many weeks [8]. Although it eventually recovers in many patients, it still persists in a major proportion of patients after months (e. g. 21 % after 3 months in a study by Danells 2004 [2]).

Physiotherapists have developed several strategies for dealing with the pusher behavior, mainly trying to give the patient back a sense of midline verticality, e. g. by using external vertical cues like standing with the patient besides a wall. Only recently, there is an increasing body of research to empirically compare treatment strategies.

Technological Advances

After the introduction of treadmill training into neurorehabilitation for spinal cord injury [10] and stroke patients [5], there has been increasing interest in advancing technology to support gait training in non-ambulatory patients. While the results of large trials were not able to show convincing superiority of treadmill training, there are advanced gait trainers that support the patient otherwise unable to walk. Construction principles are either end-effector devices, mainly consisting of actuated footplates and a body weight support and so called exoskeletons, that control hip and knee joint movements in combination with body weight support in a stationary device like the Lokomat® (Hocoma). Newer mobile exoskeletons allow for free movements of the patient with trunk stabilization by the device and postural assistance by a therapist [4]. Due to the higher degree of controlling the patient and securing balance, patients can start exercising in an exoskeleton very early after a stroke.

Treatment of pusher behavior with RAGT

In non-ambulatory patients gait training is extremely challenging and only a few steps will be performed by the patient with intensive therapist support during a session. In contrast, in robot assisted gait training (RAGT), patients are secured in a perfect vertical alignment and can easily walk for 15 or 20 minutes. Early observations by physiotherapists had shown that pusher patients were better able to assume a vertical balance after a session with the gait trainer Lokomat®.

In a first study we compared three different therapeutic interventions for pusher behavior [9]. Physiotherapy with visual feedback about body orientation using external vertical structures was compared with galvanic vestibular stimulation and a single session of RAGT with the Lokomat®. Fourteen patients with ischemic or hemorrhagic stroke participated in the study in a cross-over design, each patient receiving one treatment of each of the three interventions in pseudo-random order on separate days. The therapeutic effect was measured by applying the Burke Lateropulsion Scale (BLS) immediately before and after each intervention. The results showed a clear advantage of RAGT compared to the two alternative treatments with a significant improvement only for the RAGT. However, this study did not address long-term therapeutic outcomes.

Randomized controlled trials of RAGT

As of today there are two RCTs that addressed the question, whether RAGT effectively improves long-term pusher behavior in patients in a neurorehabilitation center. Our own study [1] applied two weeks of RAGT compared to the same amount (5 sessions per week at 60 minutes) of non-robotic physiotherapy that applied the standard physiotherapeutic principles for treatment of pusher behavior. The intervention period lasted 2 weeks with a follow-up of 2 more weeks. Pusher behavior was assessed with the BLS and the SCP (Scale for Contraversive Pushing, [7]) at 3 time points. Of 38 patients randomized, 15 participants in each group could be included in the analysis. RAGT caused a significantly larger reduction of pusher behavior

than physiotherapy as measured by both scales at post test. At follow-up the difference was still significant for the SCP. Overall, pusher behavior was not any more diagnosed after the intervention for 6/15 patients in the RAGT group compared to 1/15 in the physiotherapy group. This result further improved until follow-up (9/15 compared to 5/15 patients without pusher behavior).

In a similar design with 18 patients in a RAGT group compared to 18 patients in a conventional physiotherapy group Yun et al [11] yielded a similar result with more extended periods. Participants received 15 treatments over 3 weeks and the follow-up period lasted 4 weeks. Both groups improved their pusher behavior as measured in the BLS during treatment, and even more during follow-up. As secondary outcome the Berg Balance Scale was used, which evaluates balance functions in different situations and movements challenging posture. The Berg Balance Sclae showed a similar different therapeutic effect with superiority of RAGT.

Overall, both studies support the idea, that RAGT is not only effective in short lasting improvement of pusher behavior but also leads to a better and long lasting treatment effect. Both studies also confirm that eventually pusher behavior will be improved in both groups. Since the additional disability by the pusher behavior is clearly seen in the Berg Balance score, all studies underline the importance of better and faster therapy results in this special problem.

Summary and Outlook

Advantageous short term and long term effects of RAGT to improve pusher behavior could be shown in several studies, however the precise mechanism needs further analysis. One explanation of the superiority of RAGT is the significantly larger amount of actual steps during a therapy session, although that effect has not always been so convincing in gait studies without specific impairments (e. g. our own study, [6]). The exoskeleton Lokomat® is characterized as allowing the patient exercise with bearing part of his body weight on his non-paretic as well as the paretic leg without any risk of falling. In addition, the patient is tightly fixated to the trunk control of the device, so that an unchallenged vertical posturing is possible. It will be interesting to compare this rigid exoskeleton to a much more liberal ambulatory exoskeleton as the EKSO™ (Ekso Bionics). These newer devices induce and allow the patient to walk with weight shifting while walking without maintaining a rigid vertical posturing. Newer software allows for preparatory exercises of weight shifting in a Pre-Gait Stepping mode in the EKSO™ with securing the patient by the therapist holding the handle of the exoskeleton. Acoustic feedback can reinforce regaining a sense of verticality. However at the same time, the absolute rigid vertical as in the Lokomat® is not fed back.

References

1. Bergmann J, Krewer C, Jahn K, Müller F. Robot-assisted gait training to reduce pusher behavior: A randomized controlled trial. Neurology 2018; 91: e1319–27.
2. Danells CJ, Black SE, Gladstone DJ, McIlroyWE. Poststroke pushing: Natural history and relationship to motor and functional recovery. Stroke 2004; 35: 2873–8.
3. Davies P. Steps to follow: a guide to the treatment of adult hemiplegia. New York: Springer 1985.
4. Dohle C, Müller F, Stephan KM. Technical Developments for Rehabilitation of Mobility. Neurology International Open 2017; 1: E211–6.
5. Hesse S, Bertelt C, Jahnke MT et al. Treadmill training with partial body weight support as compared to physiotherapy in nonambulatory hemiparetic patients. Stroke 1995; 26: 976–81.
6. Husemann B, Müller F, Krewer C, Heller S, Koenig E. Effects of locomotion training with assistance of a robot-driven gait orthosis in hemiparetic patients after stroke: a randomized controlled pilot study. Stroke 2007; 38(2): 349–54.
7. Karnath HO, Ferber S, Dichgans J. The origin of contraversive pushing: evidence for a second graviceptive system in humans. Neurology. 2000; 55(9): 1298–304.
8. Krewer C, Luther M, Muller F, Koenig E. Time course and influence of pusher behavior on outcome in a rehabilitation setting: a prospective cohort study. Top Stroke Rehabil 2013; 20(4): 331–9.
9. Krewer C, Riess K, Bergmann J, Müller F, Jahn K, Koenig E. Immediate effectiveness of single-session therapeutic interventions in pusher behaviour. GaitPosture 2013b; 37(2): 246–50.
10. Wernig A, Müller S. Laufband locomotion with body weight support improved walking in persons with spinal cord injuries. Paraplegia 1992; 30: 229–38.
11. Yun N, Joo MC, Kim SC, Kim MS. Robot-assisted gait training effectively improved lateropulsion in subacute stroke patients: a single-blinded randomized controlled trial. Eur J Phys Rehabil Med 2018; 54: 827–36.

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POSTER PRESENTATIONS

01

The four-point pusher score (4PPS): an alternative simple screening tool for lateropulsion and pusher behaviour

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Introduction: Lateropulsion, also called pusher-behaviour, is an aberration of postural control seen in some people after stroke. The physiotherapist Patricia Davies, first to extensively describe this behaviour, described the "severe form" as one where, the patient had difficulty in all positions correcting posture both to and past midline (p 266) [6]. The 4PPS was developed in Melbourne in 2000 and refined through use over 14 years. It takes approximately two minutes to administer in addition to usual assessments and is designed to assess lateropulsion across the spectrum of severities and stroke types, explicitly examining past midline [3]. Mild lateropulsion is considered to only affect standing or walking, while severe lateropulsion affects sitting, standing and walking [3]. As previous validation studies of iterations of the 4PPS were not published in peer-reviewed journals, the authors undertook this study to examine the reliability and validity of this measure [3].
Method: Stroke survivors with a non-spinal stroke were invited to participate within 48 hours of admission to an inpatient rehabilitation ward in a public tertiary hospital. The 4PPS was assessed on each patient four times on the same day (≥20 minutes apart), three by one therapist and once by an additional therapist. Intra-rater and inter-rater reliability were determined using the weighted κ statistic. Concurrent validity was assessed by examining associations of the 4PPS with the more widely used lateropulsion measures, the Burke Lateropulsion Scale (BLS) [7] and the Scale for Contraversive Pushing (SCP) [5]. Differences between indicators of pushing based on SCP ≥0/2 for A, B and C [2], BLS ≥2/17 [1] and 4PPS >0/3 were tested using McNemar's test. Associations for scores of pushing used SCP>0/6 [2, 5] and BLS >0/17 [4]. Associations with functional scales including FIM motor subscale (FIM.MSS) and the Berg balance scale (BBS) were also assessed.
Results: 85 (62 unilateral cerebral) stroke survivor participants, median 13 (IQR 9–21) days post stroke completed this study. The weighted κ statistic for 4PPS intra- and interrater reliability was 0.97 (p<0.001). Lateropulsion was indicated in 46/85 patients in one or more scales. Indicators of lateropulsion between 4PPS and BLS scales are not significantly different (43/46, $p=1.000$) but are significantly different between 4PPS and SCP (35/46, $p=0.002$). 4PPS scores were significantly associated with BLS ($r_s=0.95$), and SCP ($r_s=0.96$) scores, as well as between 4PPS and FIM.MSS ($r_s=-0.64$), BBS ($r_s=-0.77$) and Chedoke-McMaster Stroke Assessment postural control scale (CMSA.PC) ($r_s=-0.76$). For the 62 unilateral cerebral strokes, lateropulsion indicators are not significantly different between 4PPS and BLS (34/34, p=1.000), or 4PPS and SCP (30/34, p=0.125), with stronger correlations compared to the total sample for FIM.MSS ($r_s=-0.73$), BBS($r_s=-0.86$) and CMSA.PC ($r_s=-0.79$; all association $p<0.001$).
Conclusion: The 4PPS is a time efficient reliable and valid scale to assess for lateropulsion in an in-patient rehabilitation setting. The time efficiency of scale application relative to other scales, and the meaningful hierarchy of the scoring system, suggests the 4PPS is a useful alternative simple screening tool for lateropulsion and pusher behaviour.

1. Babyar SR, Peterson MGE, Reding M. Time to Recovery From Lateropulsion Dependent on Key Stroke Deficits: A Retrospective Analysis. Neurorehabilitation and Neural Repair 2015; 29(3): 207–13.
2. Baccini M, Paci M, Nannetti L, Biricolti C, Rinaldi LA. Scale for Contraversive Pushing: Cutoff Scores for Diagnosing 'Pusher Behavior' and Construct Validity. Physical Therapy 2008; 88(8) :947–55.
3. Chow E, Parkinson S, Jenkin J et al. Reliability and Validity of the Four Point Pusher Score: An Assessment Tool for Measuring Lateropulsion and Pusher Behaviour in Adults Following Stroke. Physiotherapy Canada 2019; 71(1): 34–42.
4. D'Aquila MA, Smith T, Organ D, Lichtman S, Reding M. Validation of a lateropulsion scale for patients recovering from stroke. Clin Rehabil 2004; 18(1): 102–9.
5. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke "Pushing": Natural History and Relationship to Motor and Functional Recovery. Stroke 2004; 35(12): 2873–8.
6. Davies PM. Steps to follow: a guide to the treatment of adult hemiplagia: based on the concept of K. and B. Bobath. Berlin: Springer-Verlag 1985.
7. Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. Proc Natl Acad Sci U S A 2000; 97(25): 13931–6.

02

A virtual reality based therapy for improving the postural control of patients with critical-illness-polyneuropathy/ -myopathy – a pilot study

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Introduction: Critical-Illness-Polyneuropathy and -myopathy are the most common reason for neuromuscular weakness of patients on the intensive care unit. They are a major cause of failure to wean from the ventilator and are accompanied by a prolonged length of stay. Until now there is no evidence-based treatment. Early mobilization and rehabilitation are supposed to show favorable effects on the functional outcome of critical ill patients. More research is necessary to develop new rehabilitation methods. One opportunity is the virtual reality based therapy with the MindMotionGo, which was investigated in this pilot study.
Methods: The duration of this monocentric, prospective interventional case study was scheduled for six weeks. Patients participated in an intervention of two weeks with the MindMotionGo and were investigated two weeks before and after the therapy. The MindMotionGo is a Kinect based mobile neurorehabilitation system which involves a variety of gaming engaging activities. The intervention was done three times a week for 30 minutes. Outcomes were on the one hand the feasibility and safety of the therapy and on the other hand the functional outcome. The primary outcome parameter was the Berg-Balance-Scale. Secondary parameters were the posturography, the Trunk Impairment Scale, manual muscle testing and the EQ-5D-5L.
Results: Two patients completed the intervention phase with the MindMotionGo, but only one patient finished the follow-up period. The intervention was feasible and safe for the patients with CIP/CIM. There were no adverse events. Only a few technical problems hindered the therapy. There was an improvement in the Berg-Balance-Scale and the other outcome parameters in both patients.

Discussion: Caused by the low number of patients, the validity of this study is limited. Nevertheless, the therapy could be seen as feasible and safe for patients with CIP/CIM. For future studies the inclusion and exclusion criteria and the acceptance of a virtual reality based therapy for patients who are critically ill should be discussed. Further research is necessary to strengthen the findings of this pilot study.

03

Interactive sonification for balance training in neurological rehabilitation

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Background: A growing body of research suggests that auditory information has a profound effect on the motor system. Neuroimaging research has shown an acoustic sensitivity of the vestibular apparatus [6] and a broadly distributed neuroanatomic network connecting auditory and motor systems [4].
In the event of impaired intrinsic feedback after stroke, augmented external feedback is frequently used to support the recovery of motor skills in therapy. This form of feedback – if provided in parallel (as opposed to later) with the exercise – can accelerate motor learning by providing additional movement information and thus reducing the error rate [5].
However, while the interdependence of action and perception is generally well studied (mostly from studies in the visual field), the auditory feedback channel remained largely unconsidered [3]. The assumption that the long-term effect of feedback can vary based on the sensory modality in which it is presented is relatively new [2].
Interactive sonification is a type of acoustic feedback that uses non-speech audio to convey information within a human-computer interface. Acoustic parameters (i. e. loudness, pitch, timbre, harmony, and rhythm) can provide otherwise unavailable biomechanical information [1, 5].
External real-time auditory feedback has been frequently applied in upper-limb rehabilitation post-stroke [4]. However, the potential of interactive sonification to (re)train postural stability post-stroke remains largely untapped.
Experiments: A series of tests was conducted both with healthy and with neurologically impaired participants for proof of concept and to determine strategies that are useful for balance training in neurological rehabilitation.
In a preliminary evaluation, 20 apparently healthy participants (M = 43, SD = 13.78, f/m = 7/13) were guided through a balance training that involved acoustic target detection and musical sonification. The goal of this study was 1) to test and evaluate different sonification models and balance exercises with a certified balance training system, and 2) to control for factors associated with the outcomes (e. g., musical education).
Generally, participants preferred movement on the X-axis (mediolateral). However, a musical background of the participants was associated with an increased usage of the Y-axis (which was represented acoustically by pitch and melody). A background of musical training was also related to better overall exercise scores. Simple functional sounds have shown to be most effective for localizing targets (i. e. to guide and refine the activity), but musical sound models were perceived significantly more pleasurable.
In a subsequent feasibility study with stroke patients at the Schön Klinik Bad Aibling, two women (age 72 and 76) and two men (59 and 63) with Berg Balance Scale scores ranging from 22 to 42, were each guided through four 30-minute sessions of sonified exercises to train standing balance with a certified medical device. The patients were given visual and/or auditory feedback over headphones according to their training progress (adjusted level of difficulty).
Based on a questionnaire (Intrinsic Motivation Inventory), the training was perceived as very challenging, both physically and cognitively. High scores on the subscales for interest/pleasure and value/benefits suggest a general feasibility and high acceptance of the intervention. Analysis of the video recordings of all sessions and the raw data of the sensors confirmed that all of the patients were able to follow the acoustic guidance – in the absence of visual feedback – and change their body's center of gravity accordingly (especially towards the end of the study).
Conclusions: Interactive sonification appears to be a well-received and feasible approach to incorporate into neurological balance training.
Findings from other studies show that during similar sound-making experiences, strong neurological auditory-motor associations are developed, providing support for the use of real-time auditory feedback to enhance sensorimotor representations and facilitate movement (re)acquisition [4].
Further clinical research should be carried out to evaluate this approach with larger group samples and with refined auditory feedback, including spatial hearing, rhythmical cueing and dynamic difficulty adjustment of the software.

1. Dubus G, Bresin R. A systematic review of mapping strategies for the sonification of physical quantities. PloS one 2013; 8 (12): e82491.
2. Dyer JF, Stapleton P, Rodger MWM. Sonification as Concurrent Augmented Feedback for Motor Skill Learning and the Importance of Mapping Design. The Open Psychology Journal 2015; 8(1): 192–202.
3. Kennel C, Streese L, Pizzera A, Justen C, Hohmann T et al. Auditory reafferences. The influence of real-time feedback on movement control. Frontiers in psychology 2015; 6: 69.
4. Schaffert N, Janzen TB, Mattes K, Thaut MH. A Review on the Relationship Between Sound and Movement in Sports and Rehabilitation. Frontiers in psychology 2019; 10: 244.
5. Sigrist R, Rauter G, Riener R, Wolf P. Augmented visual, auditory, haptic, and multimodal feedback in motor learning: a review. Psychonomic bulletin & review 2013; 20(1): 21–53.
6. Todd NPM, Paillard AC, Kluk K, Whittle E, Colebatch JG. Vestibular receptors contribute to cortical auditory evoked potentials. Hearing research 2014; 309: 63–74.

04

Gait stability in patients with mild Parkinson's Disease

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Introduction: Parkinson's disease [PD] is the fastest growing neuro-degenerative disorder [3] and near-falls or falls are a considerable issue for patients [2]. Tripping and postural instability seem to be common causes [4]. Importantly, fall-related activity avoidance already affects patients who did not previously fall [5] and may promote inactivity and physical degeneration.
Objective: To determine whether the margins of stability (MOS) and foot clearance measures distinguish patients with mild PD from controls and correlate with falls or the fear of falling.
Methods: Gait of 12 patients with PD (age: 69 ± 12 y.; Hoehn & Yahr stage 1–3) and 14 controls (age: 61 ± 13 y.) was compared at self-selected speed using 3D motion capturing (Simi Motion, Germany). All patients were evaluated on medication. The margins of stability (MOS) in anterior, posterior and lateral directions were calculated. They relate the extrapolated centre-of-

mass (xCOM) to the boundaries of the base of support. During stance, the minimal anterior and lateral values were extracted. During swing, the minimal posterior MOS was extracted. Moreover, at contra-lateral footstrike, the posterior margins were extracted. Concerning tripping, a toe clearance integral over the swing phase was calculated. All analysis was done for the more affected leg. Complementary assessments included the Falls Efficacy Scale (FES-I) and reports about falls during the last 12 months.

Results: Patients with PD reported more falls (P = 0.040) and tended to express a greater fear (P = 0.093). They walked slower (-17.8 %, P = 0.003) but stepwidth was similar (P = 0.451). During stance, the xCOM was closer to its supporting base in anterior direction (P = 0.021). The lateral MOS were not significantly different (P = 0.47). In addition, the posterior MOS during swing were similar (P = 0.980). Yet, when the opposite foot contacted the ground, smaller (less stable) posterior MOS were found (P = 0.02). The anterior-posterior MOS during stance correlated with speed and the group difference ceased when statistically adjusting for that. The toe clearance integral was significantly lower in patients (-21.7 %, P = 0.030). Although it significantly correlated with the walking speed (r = 0.70. P < 0.001), it showed a further negative partial-correlation with the FES-I score (r = -0.61, P < 0.05). No significant correlations were found for falls. Interestingly, lateral and anterior-posterior MOS measures were not interrelated in patients (P < 0.20).

Discussion & Conclusion: Despite their relatively mild disease stage and ongoing drug treatment, patients faced difficulties with gait related postural control and ground clearance. It had been previously shown that faster walking patients with longer steps lift their feet more [1], but beginning hypokinesia may raise extra concerns about stumbling independent from walking slow. Notable, drug-administration may primarily correct for lateral instability as MOS in both planes seem uncorrelated and, in particular, the instance of contra-lateral footstrike may present a critical phase for loosing backward stability which may biomechanically be related to limited propulsion [6].

1. Alcock et al. J Biomech 2018; 11(71): 30–6.
2. Allen et al. Parkinsons Dis 2013; 906274.
3. Dorsey et al. JAMA Neurol 2018; 75(1): 9–10.
4. Gazibara et al. Clin Neurol Neurosurg 2017; 161: 51–5.
5. Kader et al. BMC Neurol 2016; 2(16): 84.
6. Sofuwa et al. Arch Phys Med 2005; 86(5): 1007–13.

05

Retropulsion and disturbed verticality perception in neurological patients

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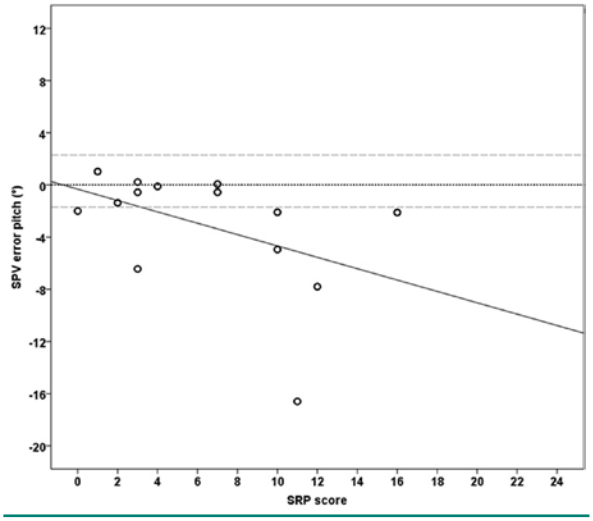
Objective: Patients with retropulsion show a posterior shift of their center of mass relative to the base of support in sitting and/or standing, a tendency to fall backwards and active resistance to passive correction. This behavior has been observed in different neurological disorders [2]. However, there is insufficient research about retropulsion, and knowledge about the pathophysiological mechanisms is missing. Similar to lateropulsion in the roll plane [1], we hypothesize that retropulsion is associated with a disturbed inner reference of verticality in the pitch plane. Therefore, the objective of this study was to investigate the relationship between retropulsion and the subjective postural vertical (SPV) as well as the subjective spontaneous body position.



05 Fig. 1

Methods: The recently developed Scale for Retropulsion (SRP) was used to quantify retropulsion. The SRP was assessed together with some questions in order to determine the patient's subjective perception of the spontaneous body position while sitting and standing with eyes open. The perception of the spontaneous body position was compared with the SRP scores. The SPV was assessed during standing by using the Spacecurl. The SPV error and the SPV range for the roll and pitch planes were correlated with the SRP scores.

Results: Data from 20 patients with different neurological diagnoses were collected (mean age 67 ± 14 years, 8 female). The results showed a significant correlation between the SRP and the SPV error in the pitch plane (r = -0.568; p = 0.034) (see **Figure 1**). No significant correlation was found between the SRP and the SPV error in the roll plane (r = -0.120; p = 0.71). Neither correlated the SPR score with the SPV ranges (p > 0.64). 5 patients showed an erroneous perception of their spontaneous standing position. 2 of these 5 patients had a posterior tilt of their SPV.

Discussion: There is a relationship between retropulsion and an impaired verticality perception in the pitch plane: the SPV shifted backward with higher scores on the SRP, i. e. increasing retropulsion. This indicates that patients with retropulsion align their body with a backward tilted inner reference of verticality. One quarter of the patients had an impaired perception of their spontaneous body position while standing. Larger data sets are required to investigate the effects of different diagnosis on retropulsion and perceived upright body orientation.

1. Bergmann J, Krewer C, Selge Ch, Müller F, Jahn K. The subjective postural vertical determined in patients with pusher behavior during standing. Top Stroke Rehabil 2016; 23: 184–90.
2. Scheets PL, Sahrmann SA, Norton BJ, Stith JS, Crowner BE. What is backward disequilibrium and how do i treat it? A complex patient case study. J Neurol Phys Ther 2015; 39: 119–26.

06

Research protocol: Should there be a push for change to service delivery for patients with lateropulsion?

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Background: Lateropulsion is a condition with negative implications for recovery, including a greater cost of care as a result of the extended inpatient length of stay required to achieve similar outcomes to stroke survivors without lateropulsion.

Primary Objective:

1. To determine the association of lateropulsion severity after stroke, as measured by the Four-Point Pusher Score (4PPS), with:
   a. Functional change during inpatient rehabilitation, at Osborne Park Hospital Stroke Rehabilitation Unit (OPH SRU), as measured by the Functional Independence Measure (FIM) and Modified Barthel Index (MBI);
   b. Length of stay (LOS) (days) in inpatient rehabilitation; and
   c. Discharge destination (categorical scale) after inpatient rehabilitation.

Secondary Objectives:

2. After accounting for post-stroke disability, as measured by Modified Rankin Scale (MRS) – to determine how inpatient LOS at OPH SRU for patients with lateropulsion compares with average LOS predicted in the Australian National Sub-Acute and Non-Acute Patient Classification (AN-SNAP) funding model in each AN-SNAP class for stroke rehabilitation.
3. After accounting for post-stroke disability (MRS) – to determine the relationship between lateropulsion severity (4PPS) on admission to rehabilitation and lateropulsion severity (4PPS) on discharge.
4. After accounting for post-stroke disability (MRS) – to determine the relationship between 4PPS on admission to rehabilitation and overall FIM change and FIM efficiency (FIM change/LOS) during rehabilitation.

Methods: The OPH SRU provides stroke rehabilitation services to patients in the North Metropolitan Area of Perth, Western Australia, accommodating approximately 100 admissions per year. A comprehensive database containing outcomes for consecutive admissions to the OPH SRU will be used to address the above aims in a retrospective observational study.

Participants: All consecutive admissions to the OPH SRU (2005–2018) are included in the database. This represents data from >1000 admissions, including >450 patients with lateropulsion. Patients are >65 years of age, have a diagnosis of acute stroke confirmed by brain imaging and review by a stroke physician or neurologist and have been transferred for rehabilitation. Patients with bilateral stroke, lateral medullary syndrome and those who were non-ambulant prior to the stroke will be excluded.

Statistical methods: A linear mixed model regression will be used to address the above objectives.

Significance of this Research: This study will explore the associations between lateropulsion and inpatient rehabilitation costs and identify how this is reflected in the Australian rehabilitation funding model. Understanding these associations will drive local policy development to adequately fund rehabilitation for these patients. Changes to service delivery have the potential to optimise distribution of rehabilitation resources in Western Australia, to reduce disability and carer burden, and to help relieve the financial burden to the health system in the cost of long-term care.

This study will provide novel information regarding the role of lateropulsion in stroke recovery and rehabilitation. Importantly, this study will use existing data containing outcomes for >1000 patients, reporting one of the largest cohorts of stroke survivors with lateropulsion (n >450) to date. The results of this study will have direct implications for service delivery and cost reduction at local, state and national levels.

07

Deviations of the postural vertical in two dimensions in peripheral and central vestibular disorders

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Background: Deviations of the subjective visual vertical (SVV) in the roll (frontal) plane occur commonly in disorders of the brainstem or acute unilateral peripheral vestibular failure and have been extensively explored. In contrast, little is known about deviations of verticality perception in other planes. Moreover, the SVV is a poor predictor of postural impairment in stroke patients.

Methods: The present prospective study focused on deviations of the subjective postural vertical (SPV) in the pitch (sagittal) and roll plane in 183 patients with a wide variety of disorders affecting postural control. Patients were categorized in those with focal lesions of the neuronal vestibular chain and those with non-focal disorders. In the focal lesion group patients were assigned to one of the following groups: acute unilateral and chronic bilateral peripheral vestibular failure, acute brainstem lesion, acute thalamic lesion, acute cerebellar lesion, and acute lesion of the multisensory vestibular cortex. In the non-focal group the subgroups were: cerebellar syndrome, obstructive hydrocephalus, normal pressure hydrocephalus (NPH), Parkinson's disease, and progressive supranuclear palsy (PSP). Moreover, patients with polyneuropathy and somatoform vertigo were measured. The test task was to indicate that a vertical body position was reached after being tilted in different directions with eyes occluded standing in a Spacecurl® device.

Results: 50 % of the patients returned abnormal results: 28 % in the pitch plane (normal range -1.7° to 2.3°), 13 % in the roll plane (normal range -1.6° to 1.2°) and 9 % in the pitch and roll plane. Backward (negative) deviations were the most common abnormalities, particularly in NPH, PSP and acute cortical lesions. In cortical lesions they were often combined with deviations in the roll plane. Deviations in roll (right or left) were especially found in acute cerebellar lesions.

Conclusions: The overall goal of developing methods for diagnosing patients with impaired postural control is of high clinical relevance. For the first time we were able to assess verticality deviations in roll and pitch plans during standing. We hypothesise that the SPV and the SVV represent two separate mechanisms for postural control: The SPV reflects the control of body orientation with respect to gravity and is egocentric. The SVV represents stabilization of vertical body orientation and is allocentric.

08

Immediate effectiveness of single-session robot-assisted gait training in pusher behavior

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After hemispheric stroke, some patients with hemiparesis exhibit a so-called pusher behavior, i.e., they actively push away from the non-paretic side and lean towards the hemiparetic side, due to a tilted vertical reference in the coronal plane. This leads to a postural imbalance to such a degree that they often are unable to sit nor stand unaided and exhibit resistance to passive correction. Pusher behavior thus substantially hampers the rehabilitation of these patients. In general, forced control of the upright position during a repetitive motion seems to be an effective method for reducing the pushing behavior in stroke patients (Krewer et al. 2013; Bergmann et al. 2018). This observer-blinded single case study investigated the immediate effects of two robot-assisted gait training methods on pusher behavior: the driven-gait orthosis Lokomat® and the wearable exoskeleton EKSO GT™.

The sequence of the interventions (Lokomat® and EKSO GT™; each 60 min) was randomly assigned and the time interval between the two interventions was two days. The therapist was free to use the full range of therapy options that the device offers and performed a minimum of 20-minute walk. The Scale for Contraversive Pushing (SCP) and the Burke Lateropulsion Scale (BLS) were measured immediately before and after the single-session intervention.

This first patient of a larger study population was randomly assigned to Lokomat® therapy first. He showed improvements in the BLS from total score of 8 to 6 and in the SCP from a total score of 3 to 2.5 points. The session in the EKSO GT™ didn´t show any changes neither in the SCP nor in the BLS. However, he started from an improved baseline compared to the first intervention (SCP 2.5 points and BLS 5 points). The findings confirm that machine-supported gait training with the Lokomat® has a positive and immediate effect on pusher behavior. The therapy in the EKSO GT™ did not result in any additional improvement.

Although both interventions involved exoskeleton devices and had been performed earlier in this patient to the same extent (each, 4 sessions within 3 weeks before testing), the first intervention had a clear positive effect on the reduction of pusher behavior. Furthermore, it can be assumed that the training with the Lokomat® showed a positive aftereffect, because the pre-test scoring in the second intervention was substantially improved compared to the pre- but also post-tests in the first intervention. In contrast to the EKSO GT™, the Lokomat® includes a body weight support system, a treadmill, and implements a rather fixed trunk position during walking. In the EKSO GT®, the patient initially has to shift his body weight to trigger the step. The finding of this study is in accordance with the assumption that better intervention effects are based on conflicting sensory information, leading to constantly challenging sensory re-weighting processes (Krewer et. al 2013).

In summary, this single case study confirmed the effectiveness of Lokomat® for reducing pusher behavior and further showed that robot-assisted gait training with a wearable robotic exoskeleton had no immediate (additional) effect on pusher behavior in stroke patients. The body weight system or guided weight shifting might explain the different effects of the two devices. The finding might also be explained by the test sequence and this research question will be further investigated in a larger population.

09

Training of repetitive weight shifting and orientation in space by using the Spacecurl® in patients with pusher behavior

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Background: The pusher behavior is a complex disorder of postural control which is associated with a change in the subjective postural vertical (SPV). Patients with pusher behavior orient their body towards an impaired inner reference of verticality in the roll plane. As a result, they actively push themselves to the paralyzed side and show resistance to passive correction.

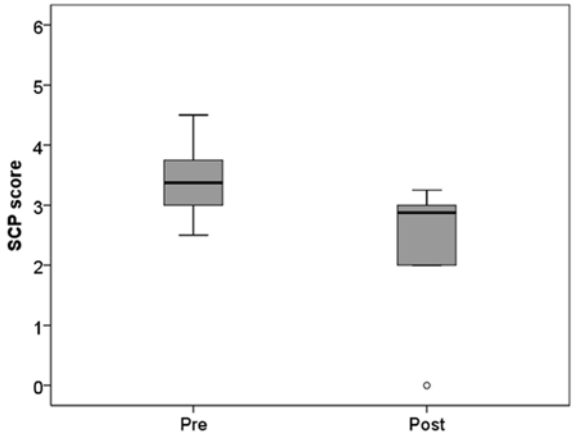
Studies have shown that robotic gait training is an effective method to reduce this behavior [1]. It might be the repetitive reciprocal movement in a vertical body position that has a positive effect on pusher behavior.

We recently described a novel therapeutic approach in a patient with pusher behavior using Spacecurl® therapy device [2]. Therapy with the Spacecurl® allows training of repetitive weight shift which is important for walking. The training can be done under safe standing conditions. In the single case study we found positive effects on pusher behavior and the SPV. The aim of this study is to further investigate the effectiveness of training with the Spacecurl® in patients with pusher behavior.

Methods: Patients with pusher behavior after stroke (Burke Lateropulsion Scale (BLS) ≥2) were included in the study. Patients trained three sessions (30 minutes each) in the Spacecurl® during one week. This training was given add on to their normal therapy in the rehabilitation hospital (robot-assisted gait training was excluded). Before the first and after the last training session in the Spacecurl® the BLS, the Scale for Contraversive Pushing (SCP) and the SPV during standing were assessed.

The Spacecurl® is a training device which consists of three cardanic-mounted rings. They are moved by body weight shifting and allow movements in the 3D space. The standing position is secured by a pelvic ring and knee pads which ensure knee-extension.

Results: So far nine patients have been included in the study. Three of them were excluded from the analysis (two had a BLS score <2 until the start of the study and one patient was not able to do the training due to strongly bent trunk position). Six data sets were analyzed (median BLS score X, range 3–8). Patients significantly improved their pusher behavior on the SCP (p=0.027, mean 1 (Q1-Q2 1-1)) **(Fig. 1)**. The improvement on the BLS did not reach significance (p= 0.068).



09 Figure 1

The SPV was initially tilted backward (mean 2.8°) and ipsilesionally (mean 1.5°) and slightly (but not significantly) approached the vertical after the intervention to -1.8° in the sagittal plane and to 0.5° in the frontal plane.
Conclusion: Training with the Spacecurl® is feasible for patients with mild to moderate pusher behavior. The device allowed patients (even if they were not able to stand unassisted) to train active weight shifting in an upright standing position for up to 20 minutes. The training had a positive effect on the standing equilibrium in the sense of reduced lateral pushing. Only three training sessions with the Spacecurl® resulted in a significant reduction of pusher behavior. Longer intervention periods might be needed for a lasting effect on the SPV, i.e. a recalibration of the disturbed inner reference of verticality.

1. Bergmann J, Krewer C, Jahn K, Müller F. Robot-assisted gait training to reduce pusher behavior: A randomized controlled trial. Neurology 2018; 91: e1319–e1327.
2. Jahn K, Müller F, Koenig E, Krewer C, Tillmann S, Bergmann J (2017). Rehabilitation of verticality perception using a new training method. Journal of Neurology 2017; 264: S26–S27.

10

Postural control and risk of fall in neurological patients with retropulsion

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Introduction: Retropulsion is characterised by a deficient postural control in the sagittal plane. Patients with retropulsion actively push themselves backward with the inability to shift the centre of mass forward. They resist passive correction and have an increased risk of backward falling. Retropulsion has been observed in several neurological patients [1]. However, up to now, there is only very little research available on the behaviour. One reason might be the lack of an established diagnostic tool. The Backward Disequilibrium Scale (BDS) is the only tool which assesses postural imbalance in the sagittal plane. This scale was developed for elderly and is insufficiently evaluated. It did not make its way into clinical practice. That's why a new Scale for Retropulsion was recently designed. The objective of this study was to investigate the postural control and the fall risk in neurological patients with retropulsion by correlating retropulsion and clinical measures for postural control.
Methods: In this study the newly developed Scale for Retropulsion (SRP) was performed in patients with different neurological disorders. Moreover, postural control and the fall risk were assessed by using the Backward Disequilibrium Scale (BDS), the Berg Balance Scale (BBS), and posturographic measurements (body sway and limits of stability).
Results: Twenty subjects with neurological disorders (acute and chronic stroke, Parkinson's disease, brain-tumour, tetraplegia and peripheral lesion) were included in this study (mean age 67 ± 13.89 years, 8 females). The median SRP score was 7.5 (range 0–17). The SRP significantly correlated with the BBS (rSp = -0.877, $p < 0.01$). This means, patients with higher SRP-scores, i.e. higher retropulsion, showed a generally reduced postural performance and higher fall risk. In addition, the SRP positively correlated with the BDS (rSp = 0.765, $p < 0.01$). This indicates that the two scales assess similar constructs, but the SRP additionally assesses characteristics such as reactive postural control and resistance which are not determined by the BDS. Regarding the posturographic measures, the SRP correlated only with the velocity of the COP in the anterior-posterior direction (rSp=0.599, p=0.034).
Conclusion: The results of this study suggest that retropulsion is associated with generally reduced postural control and high risk of fall. Even though the SRP addresses postural control specifically in the sagittal plane, it shows some correlation with the more generic BBS, but rarely with posturographic measures. The SRP assesses relevant aspects such as reactive postural control and resistance which are not detected by the BDS. The SRP seems promising to quantify disturbed postural control in the sagittal plane in neurological disorders. However, its clinimetric properties need further investigation. In addition, further studies are needed to investigate retropulsion and its impact to the rehabilitation. More knowledge about retropulsion will help to create concepts for a specific therapy and to prevent falls.

1. Manckoundia P, Mourey F, Pérennou D, Pfitzenmeyer P. Backward disequilibrium in elderly subjects, Clin Interv Aging 2008; 3: 667–72.

11

Egocentric processing in the roll plane and dorsal parietal cortex: a TMS-ERP study of the subjective visual vertical

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The intraparietal sulcus within the dorsal right posterior parietal cortex is associated with spatial orientation and attention in relation to egocentric reference frames, such as left or right hemifield. It remains unclear whether it plays a causal role in the human in the roll plane (i.e. when visual stimuli are tilted clockwise or anticlockwise) which is an important aspect of egocentric visual processing with clinical relevance in vestibular disorders. The subjective visual vertical (SVV) task measures the deviation between an individual's subjective vertical perception and the veridical vertical, involves the integration of visual, and vestibular information, and relies on a distributed network of multisensory regions that shows right lateralization and inter-areal inhibition. This study used combined TMS-EEG to investigate the role of the human dorsal parietal cortex in verticality perception using the SVV task in darkness. Participants were sorted according to their baseline bias at this task i.e. those with either a slight counterclockwise versus clockwise bias when judging a line to be truly vertical. Right parietal TMS facilitated verticality perception, reducing the difference between groups. ERPs suggested that the behavioral TMS effect occurred through normalizing individual SVV biases, evident frontally and late in the trial, and which was abolished after right parietal TMS. Effects were site and task specific, shown with a homologous left hemisphere control, and a landmark task performed on the same stimuli. These results support a right lateralization of visual-vestibular cognition and a distinct representation of the roll plane for egocentric processing in dorsal parietal cortex.

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Increased susceptibility to visually induced biases in verticality perception with age

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It has been proposed that the Central Nervous System (CNS) constructs percepts of verticality as a vector sum [5]; by combining the sensory information from the visual system and the body's inertial sensors with the prior knowledge that 'up' is usually aligned with the spine (the idiotropic vector). In subsequent work, this concept has been re-interpreted as an instance of the CNS performing Bayesian inference (e. g., [3]). Provided that some assumptions are met, this means that verticality percepts are constructed as a weighted average of sensory estimates and prior knowledge, with weights proportional to the inverse of the associated variances (e. g., [4]). Although these studies provided some support for the tenability of this framework, there are also findings that it does not readily account for: first, the weightings attributed to different sensors differ between experimental tasks, which implies that the variances of sensory outputs depend on the task that the information is used for, and second, it has also been observed that participants discarded visual cues entirely, and aligned their judgments of verticality either with inertial or idiotropic information in a dichotomous fashion [1].

To account for such observations, we recently developed a Causal Inference model of verticality perception [2]. This model accounts for different perceptual strategies by taking into account judgments on the causality of information provided by sources. Put simply, the model states that there are coexisting intermediate percepts, one that reflects integration, which is based on the Bayesian model, and one that reflects segregation, where a single sensory system dominates perception. These intermediate percepts are weighted according to the likelihood of the associated causal structure (i. e., common cause – integration; separate causes – segregation), and subsequently summed to result in a percept. Our previous study provided evidence that the CNS indeed includes judgments of causality in the construction of verticality percepts.

In the work we present here, we investigate how this process is affected by aging. In an experiment, we placed participants on a motion simulator with an 'Alternative Reality' system that could be used to independently manipulate visual and physical tilt. We presented participants from a group of younger and older participants with various combinations of visual and inertial roll-tilt stimuli with a range of discrepancies, and tasked them to adjust their orientation until they believed to be upright. The data show that visual stimuli induce larger biases in older than in younger participants. These results are consistent with decline of vestibular acuity observed in aging individuals, but also suggest that there are age-related changes in tolerances for discrepancies.

1. de Winkel KN, Clément G, Groen EL, Werkhoven PJ. The perception of verticality in lunar and martian gravity conditions. Neurosci letters 2012; 529; 7–11.
2. de Winkel KN, Katliar M, Diers D, Bülthoff HH. Causal inference in the perception of verticality. Sci Rep 2018; 8(1): 1–12.
3. Dyde RT, Jenkin MR, Harris LR. The subjective visual vertical and the perceptual upright. Exp Brain Res 2006; 173: 612–22.
4. Ernst MO, Bülthoff HH. Merging the senses into a robust percept. Trends in cognitive sciences 2004; 8: 162–9.
5. Mittelstaedt H. A new solution to the problem of the subjective vertical. Naturwissenschaften 1983; 70: 272–81.

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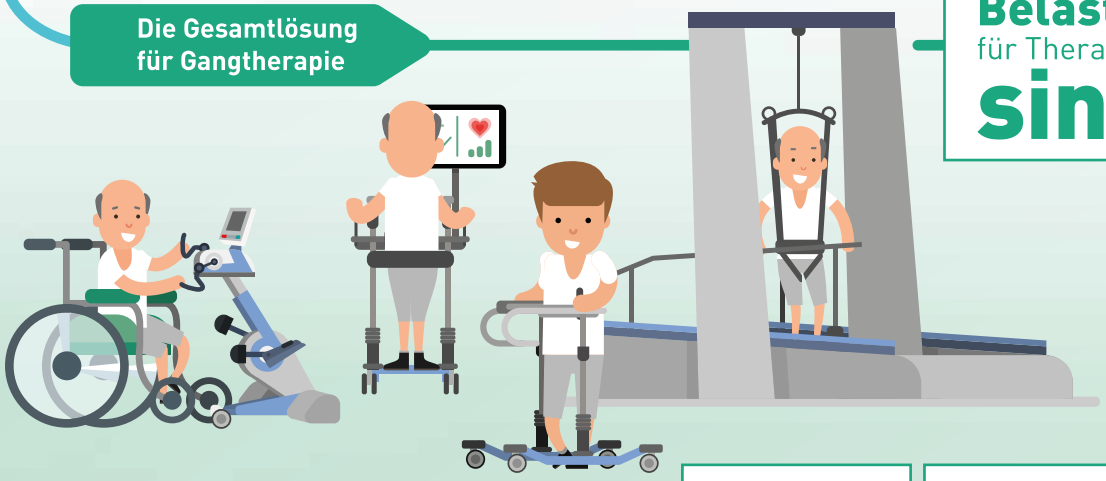

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