Identification and Treatment of Pusher Syndrome

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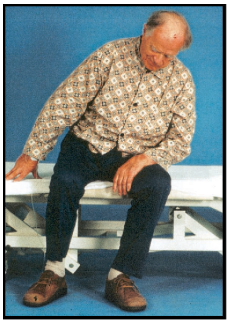
Objectives

- Define typical characteristics of pusher syndrome and identify differential diagnoses
- Understand general neuroanatomy and hypotheses for pathogenesis behind pushing behavior
- Identify two valid and reliable outcome measures to diagnose and assess change in pushing behavior
- Recognize factors that impact prognosis for functional recovery
- Identify general treatment goals and learn about intervention strategies to decrease pushing behavior and improve functional independence
- Explore some case studies and current research

Typical Presentation

- Spontaneous lateral tilt towards weak side in sitting and standing
- Abduction and extension of non-paretic extremities when in physical contact with a surface
- Resistance to any attempt at passive correction



Karnath & Broetz, 2003

Karnath & Broetz, 2003

Pusher "Syndrome"

- First described by Davies as a "syndrome" common after right hemisphere damage
  - Pushing behavior
  - Contralesional hemiplegia
  - Anosognosia
  - Neglect
  - In severe cases – ipsilesional gaze preference and rotation of neck
- Not a true "syndrome"
  - Later studies found no direct causal link (Pedersen et al)

Alternative Names

- Lateropulsion*
  - Implies postural movement in frontal plane
  - Ipsilesional vs. contralesional lateropulsion?
- Contraversive pushing*
- Ipsilateral pushing
- Listing*
- Pushing
- Right hemisphere syndrome

Common neurologic disturbances of balance and postural control

- Differential Diagnoses:
  - Listing phenomenon
  - Ipsilesional Lateropulson
  - Thalamic astasia

Listing phenomenon

- Loss of lateral balance towards hemiparetic side
- Without assistance, patient's trunk will "list" toward the affected side
- CONTRAST: Patient will attempt to hold onto something or use non-paretic hand to *prevent* a loss of balance towards paretic side

Lateropulsion

- "A tendency to fall sideways"
- Frequently seen in Wallenberg's syndrome (acute unilateral medullary brainstem infarcts)
- CONTRAST: Patient falls *towards* the side of the lesion, no active pushing or resistance to passive correction

Thalamic Astasia

- Patients are unable to stand unsupported, fall backward or to affected side when left unsupported
- During supine>sit, patients don't use trunk muscles but attempt to use hands to pull themselves up
- CONTRAST: No pushing or active resistance to correction

	Pusher Syndrome	Thalamic Astasia	Wallenberg's Syndrome	Vestibular Cortex Stroke
Direction of push or LOB	Sideways contralesional (pushing)	Backwards or to paretic side (no pushing)	Sideways ipsilesional (no pushing)	Lean and LOB contralesional side (no pushing)
Location of lesion	Posterolateral thalamus	Posterolateral thalamus	Medulla of brainstem	Posterior insula
Severity of hemiparesis	Severe	Mild to none	Mild	Mild
SVV	Intact	Intact	Impaired	Impaired
SPV	Impaired	Not stated	Not stated	Intact

Adapted from Roller 2004 Special Interest Paper

Incidence

- **10%** (Pedersen et al 1996) **to 63%** (Dannels et al 2004)
  - Differing diagnostic criteria
  - Also seeing in other types of brain damage (trauma, tumors, etc)
- More recent: 14.2% (Abe et al 2012)
- RN's and PT's often first to diagnose

Right vs. Left Lesions

- Variable reported incidences
- 65% of 23 patients with pushing behavior had right-sided lesions (Karnath 2000)
- Directional bias of pushing typically contralesional

Link between Pushing Behavior, Aphasia, and Spatial Neglect

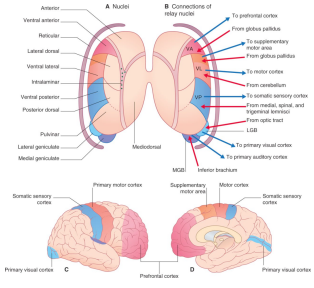
- Hemispatial neglect is NOT the *cause* of contraversive pushing, but…
  - High association with spatial neglect in patients with right hemisphere lesions
  - High association with aphasia in patients with left hemisphere lesions
- Karnath et al 2000:
  - 20% patients with right-sided lesions with pusher syndrome did not have spatial neglect
  - All patients with pusher syndrome with left-sided lesions had aphasia (none had spatial neglect)

What can cause balance and posture impairments?

- Paresis
- Sensory loss
- Vestibular dysfunction
- Disturbed sensory integration

Neuroanatomy - Thalamus

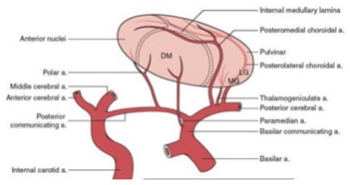
- "Relay station" – receives, interprets, and sends signals from all over the brain
- 6 functional classes of nuclei



https://www.studyblue.com/notes/note/n/thalamus-and-cortex/deck/11665047

Thalamic Lesions

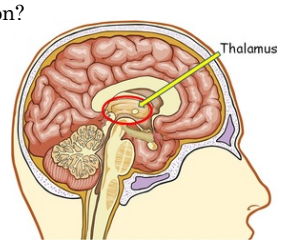
- Posterior vs. Anterior Thalamus
- Anatomical links:
  - Stroke etiology
  - Specific vascular damage within the thalamus
  - Cause of secondary impairments (paresis)?



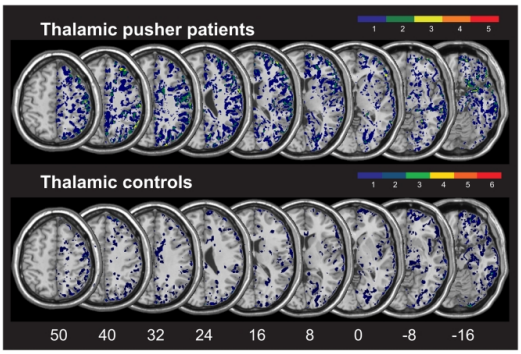
https://www.slideshare.net/danielveladuartemd/thalamic-infarction

Posterolateral thalamus

- Unilateral lesions of posterior thalamus
- Functional or metabolic abnormalities in cortical areas via diachisis?
- Vascular dysfunction?



http://brainmadesimple.com/thalamus.html



Ticini 2009

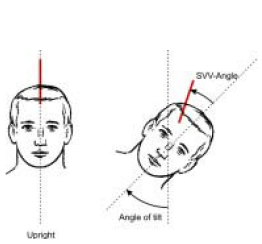
Putting it all together…

- Posterior thalamus + extra-thalamic structures are needed for intact processing of gravity and control of upright body posture
  - inferior frontal gyrus
  - middle temporal gyrus
  - precentral gyrus
  - inferior parietal lobe
  - parietal white matter
  - superior longitudinal fasciculus

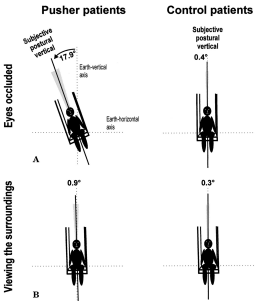
WHY DOES DAMAGE TO THESE AREAS OF THE BRAIN LEAD TO A PATIENT ACTIVELY PUSHING?

HYPOTHESIS: DISTURBANCE OF THE VISUAL-VESTIBULAR SYSTEM

- Subjective visual vertical: "earth vertical," dependent on visual and vestibular processing



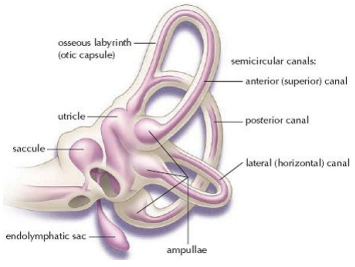
https://www.researchgate.net/figure/Principle-of-the-estimation-of-subjective-visual-vertical-with-full-head-and-trunk-tilt_fig3_228727499



https://www.physio-pedia.com/Pusher_Syndrome

HYPOTHESIS: DYSFUNCTION OF SEMICIRCULAR CANALS OF VESTIBULAR SYSTEM

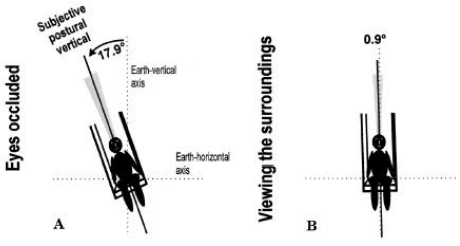
- Rotation and caloric stimulation showed that vestibular system not relevant to pushing behavior



http://dizziness.webs.com/anatomyphysiology.htm

HYPOTHESIS: SUBJECTIVE POSTURAL VERTICAL DISTURBANCE

- Subjective postural vertical: perceived upright orientation of body



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A "SECOND GRAVICEPTIVE SYSTEM"

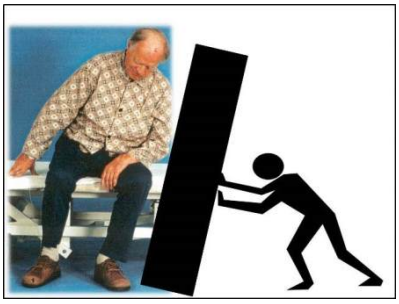
- Pusher syndrome is associated with a severe misperception of body orientation in relation to gravity
- There appears to be a **distinct neural pathway for sensing upright body posture and orientation to gravity**

Pérennou 2008

ROLE OF SOMATOSENSORY AND PROPRIOCEPTIVE INPUT?

- No significant differences in somatosensory testing
- Case study: Astronauts and divers
- Case study: Complete hemisensory loss due to right thalamic lesion
- CONCLUSION: Somatosensory input plays a minor role in perception of body posture and does not necessarily result in pushing behavior

*IPSI*VERSIVE TILTED PERCEPTION OF BODY ORIENTATION LEADS TO *CONTRA*VERSIVE PUSHING?



https://appliedstrokerehab.wordpress.com/2015/07/23/strategies-for-the-treatment-of-pusher-syndrome/

HYPOTHESES:

- Conflict between two reference systems
  - Less pushing with eyes closed
- Secondary response to unexpected experience of loss of balance
- Disturbed spontaneous postural responses

Perennou 2014

Impaired Postural reactions

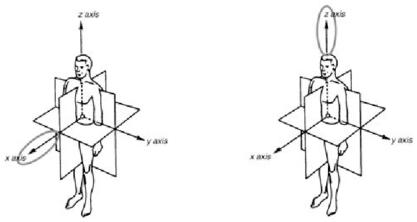
Ipsiversive trunk tilt | No trunk tilt | Contraversive trunk tilt

Johannsen 2006

Pusher Syndrome vs. Spatial Neglect

- Brain has separate but overlapping systems for perceiving and controlling body orientation in different dimensions of space

Pusher syndrome | Spatial neglect



Karnath 2006

Posterior thalamus
Insula
Postcentral gyrus

Right superior temporal complex
Insula
Temporo-parietal junction

FUN FACT!

- "**Camptocormia**" – disturbance in the sagittal (pitch) plane
  - Abnormal trunk posture with increased bending forward (pathogenesis unclear..)

Case Study: Pusher Syndrome in a medicine patient??

- 61yo Female
- PMH: Budd-Chiari, polycythemia vera, hypothyroidism, Afib/Aflutter
- Recently d/c'ed from acute rehab after a prolonged hospitalization d/t ABLA s/p TIPS resulting in hemorrhagic shock
- Admitting Dx: left-sided weakness

Chart Review

- Medical:
  - Left side weakness – etiology unknown (abscess vs. hematoma vs. deconditioning?)
  - Pt was ambulating 200ft at rehab discharge but after 2 days needed assistance ambulating only 16ft
  - Neuro exam non-focal: MRI L spine pending
  - CK low, XR no fractures
- Physical Therapy:
  - No significant focal weakness
  - Mod A supine>sit
  - Mod A sitting EOB (leaning to left)
  - Active resistance to passive correction
  - Mod-Max Ax1 for sit<>stand (falling to left)

Findings:

- **Brain MRI obtained 48hrs after admission revealed an acute right parafalcine and convexity subdural hematomas in the setting of an elevated INR of 2.7**

Non-Stroke Etiology Case Studies

- 50yo male s/p MVA admitted with GCS 9 and L hemiparesis
  - Dx: right tempo-parietal contusion, laminar subdural hematomas, mild cerebral edema
- 58yo female w/ h/o right gluteus rabdomyosarcoma treated with chemo and surgery and h/o lung metastasis admitted with L hemiparesis and AMS
  - Dx: multiple hemorrhagic brain metastases with severe vasogenic edema
- 62yo male with h/o epilepsy and ETOH abuse admitted with AMS and aphasia
  - Dx: left fronto-temporal subdural hematoma with midline shift and hemorrhage in left basal ganglia and frontal cortex

Outcome Measures

- Scale for Contraversive Pushing
- Burke Lateropulsion Scale

Scale for Contraversive Pushing (SCP)

Examination Form
Clinical Scale for Contraversive Pushing (SCP)

Name..... Date of birth..... Ward.....
Examination date..... Diagnosis .....
Physician in charge..... Physical therapist in charge.....

		Sitting	Standing
(A) Spontaneous body posture			
Value 1	= severe contraversive tilt with falling to that side	☐	☐
Value 0.75	= severe contraversive tilt without falling	☐	☐
Value 0.25	= mild contraversive tilt without falling	☐	☐
Value 0	= inconspicuous	☐	☐
	Sum total (max. = 2):		
(B) Use of the non-paretic extremities (abduction & extension)			
Value 1	= performed spontaneously, already when at rest	☐	☐
Value 0.5	= performed only on changing the position (e. g. on transferring from bed to wheelchair)	☐	☐
Value 0	= inconspicuous	☐	☐
	Sum total (max. = 2):		
(C) Resistance to passive correction of tilted posture*			
Value 1	= resistance occurs	☐	☐
Value 0	= resistance does not occur	☐	☐
	Sum total (max. = 2):		

*Touch the patient at the sternum and the back. Instruction: "I will move your body sidewards. Please permit this movement".

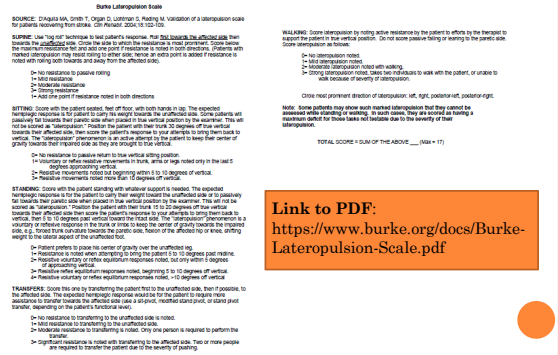
SCP Clinimetric Properties

- Criterion score was changed from >1 in each component to >0 in each component (Baccini et al)
  - Moderate agreement with clinical evaluation
  - Improved agreement with criterion change between SCP and expert clinical diagnosis
- Sensitivity: 64.7% (subscores >1), 100% with modified criterion (subscores >0)
- Specificity: 100% (subscores >1), 97.7% with modified criterion (subscores >0)

Modified SCP

- 4 item scale: static sitting, static standing, sitting transfer, and standing/walking transfer
- Scoring:
  - 0 = no sign of lateropulsion
  - 8 = maximum score/severe lateropulsion
- Identifies pushing behavior in dynamic balance activities
- Different from the original scale so should be considered a new assessment tool
  - Moderate relationship between SCP and BLS

Burke Lateropulsion Scale (BLS)



Burke Lateropulsion Scale

SOURCE: D'Aquila MA, Smith T, Organ D, Lottman G, Reding M. Validation of a lateropulsion scale for patients recovering from stroke. *Clin Rehabil*. 2004;18:102-109.

SUPINE: Use "log roll" technique to test patient's response. Roll first towards the affected side then towards the unaffected side. Circle the side to which the resistance is most prominent. Score below the maximum resistance felt and add one point if resistance is noted in both directions. (Patients with marked lateropulsion may resist rolling to either side; hence an extra point is added if resistance is noted with rolling both towards and away from the affected side).

0= No resistance to passive rolling
1= Mild resistance
2= Moderate resistance
3= Strong resistance
+= Add one point if resistance noted in both directions

SITTING: Score with the patient seated, feet off floor, with both hands in lap. The expected hemiplegic response is for patient to carry his weight towards the unaffected side. Some patients will passively fall towards their paretic side when pulled in true vertical position by the examiner. This will not be scored as "lateropulsion." Position the patient with their trunk 10 degrees off true vertical towards the unaffected side. Score the patient's response to your attempts to bring them back to vertical. The "lateropulsion" phenomenon is an active attempt by the patient to keep their center of gravity towards their impaired side as they are brought to true vertical.

0= No resistance to passive return to true vertical sitting position.
1= Voluntary or reflex pushing movements in trunk, arms or legs noted only in the last 5 degrees of returning to vertical.
2= Resistive movements noted but beginning within 5 to 10 degrees of vertical.
3= Resistive movements noted more than 10 degrees off vertical.

STANDING: Score with the patient standing with whatever support is needed. The expected hemiplegic response is for the patient to carry their weight toward the unaffected side or to passively fall towards their paretic side when placed in true vertical position by the examiner. This will not be scored as "lateropulsion." Position the patient with their trunk 15 to 20 degrees off true vertical towards their affected side then score the patient's response to your attempts to bring them back to vertical. then 5 to 10 degrees past vertical toward the intact side. The "lateropulsion" phenomenon is a voluntary or reflexive response in the trunk or limbs to keep the center of gravity towards the impaired side, e.g., forced trunk curvature towards the paretic side, flexion of the affected hip or knee, shifting weight to the lateral aspect of the unaffected foot.

0= Patient prefers to place his center of gravity over the unaffected leg.
1= Resistance is noted when attempting to bring the patient 5 to 10 degrees past midline.
2= Resistive voluntary or reflex equilibrium responses noted, but only within 6 degrees of midline.
3= Resistive reflex equilibrium responses noted, beginning 5 to 10 degrees off vertical.
4= Resistive voluntary or reflex equilibrium responses noted, >10 degrees off vertical

TRANSFERS: Score this one by transferring the patient first to the unaffected side, then if possible, to the affected side. The expected hemiplegic response would be for the patient to require more assistance to transfer towards the affected side (use a sit-pivot, modified stand-pivot, or stand pivot transfer, depending on the patient's functional level).

0= No resistance to transferring to the unaffected side is noted.
1= Mild resistance to transferring to the unaffected side.
2= Moderate resistance to transferring is noted. Only one person is required to perform the transfer.
3= Significant resistance is noted with transferring to the affected side. Two or more people are required to transfer the patient due to the severity of pushing.

WALKING: Score lateropulsion by noting active resistance by the patient to efforts by the therapist to support the patient in true vertical position. Do not score passive falling or waning to the paretic side. Score lateropulsion as follows:

0= No lateropulsion noted.
1= Mild lateropulsion noted.
2= Moderate lateropulsion noted with walking.
3= Strong lateropulsion noted. Takes two individuals to walk with the patient, or unable to walk because of severity of lateropulsion.

Circle most prominent direction of lateropulsion: left, right, posterior-left, posterior-right.

Note: Some patients may show such marked lateropulsion that they cannot be assessed while standing or walking. In such cases, they are scored as having a maximum deficit for those tasks not testable due to the severity of their lateropulsion.

TOTAL SCORE = SUM OF THE ABOVE ___ (MAX = 17)

Link to PDF:
https://www.burke.org/docs/Burke-Lateropulsion-Scale.pdf

SITTING: Score with the patient seated, feet off floor, with both hands in lap. The expected hemiplegic response is for patient to carry his weight towards the unaffected side. Some patients will passively fall towards their paretic side when placed in true vertical position by the examiner. This will not be scored as "lateropulsion." Position the patient with their trunk 30 degrees off true vertical towards their affected side, then score the patient's response to your attempts to bring them back to vertical. The "lateropulsion" phenomenon is an active attempt by the patient to keep their center of gravity towards their impaired side as they are brought to true vertical.

0 = No resistance to passive return to true vertical sitting position.
1 = Voluntary or reflex resistive movements in trunk, arms, or legs noted only in the last 5 degrees approaching vertical
2 = Resistive movements noted but beginning within 5 to 10 degrees of vertical.
3 = Resistive movements noted more than 10 degrees off vertical

Comparing Outcome Measures

- SCP and BLS are both reliable and valid measures
- BLS is more responsive to small changes

Functional Prognosis

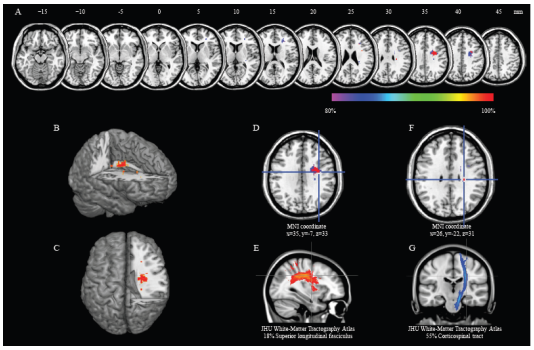
- Karnath et al 2002 (N=12)
  - Symptoms nearly resolved after 6months post-stroke
- Danells et al 2004 (N=65)
  - By 6 weeks, 62% of pushing symptoms resolved
  - By 3 months, 79% of pushing symptoms resolved
  - Longer hospital LOS (89 vs. 57 days) for patients with pushing behavior vs. those without pushing
  - Used SCP cut-off >0

Prognosis: Recent Research

- Babyar et al 2008 (case-matched controlled study)
  - FIM efficiency and d/c FIM scores worse in Pusher Syndrome group
  - Pusher syndrome + R CVA required more dependent d/c living situation
- Abe et al 2012 (N=1660)
  - 156 (9.4%) had pusher behavior
  - Patients with right brain damage had significantly slower recovery vs. those with left brain damage
  - Helpful for discharge planning/goal setting

Prognosis: Recent Research

- Babyar et al 2015 (N 169, BLS score of 2 or greater)
  - Motor deficits only: 90.5% reached BLS 0 or 1 at D/C
  - Motor + visual-spatial deficits: 59% reached BLS 0 or 1 at D/C
  - Motor + proprioceptive + visual-spatial deficits : 37% reached BLS 0 or 1 at D/C
- Babyar et al 2016 (N=1671)
  - Indicators of delayed recovery:
    - L brain damage: older age, worse RLE Motricity Index score on admission
    - R brain damage: older age, greater limb placement error on admission, lower FIM cog score
    - Spatial neglect and gender did not impact recovery



Abe et al 2017

Persistent Pushing?

- **Case 1**: 77yo male with h/o HTN, admitted with L hemiparesis with NIH score of 20, found to have R MCA ischemic stroke.
  - D/C'ed from hospital after 10days (SCP 6)
  - Re-evaluated after 318 days, SCP still 6, Barthel Index 0
  - Pt died of PNA shortly after re-evaluation
- **Case 2**: 74yo male, h/o ETOH abuse admitted with R hemiparesis and aphasia, found to have L ACA and MCA ischemic strokes
  - D/C'ed after 20days (SCP score of 6)
  - Re-evaluated after 763 days, still severe PB, Barthel Index score of 0
- **Case 3**: 65yo male with R MCA aneurysm s/p surgical clipping
  - Pt referred to outpatient stroke clinic 1.6 years after initial admission
  - Severe PB when re-evaluated 729 days, Barthel Index score of 10
  - Pt died a few weeks after this evaluation

WHY?

- All patients were referred to public rehabilitation centers, but poor adherence to rehab programs
  - Restricted access to PT (average less than 1x/week)
  - Pt's spent majority of day in bed
- Comments from article:
  - Poor socioeconomic status limited resources
  - Early rehab and continuum of care reduces disability within the first year
  - Is duration of PB related to time spent in a vertical position?

Treatment Goals

- Realize the disturbed perception of erect body position
- Visually explore the surroundings and the body's relation to the surroundings. Ensure the patient sees whether he or she is oriented upright (suggest the PT uses visual aids that give feedback), utilize vertical structures in a room
- Learn the movements necessary to reach a vertical body position
- Maintain the vertical body position while performing other activities

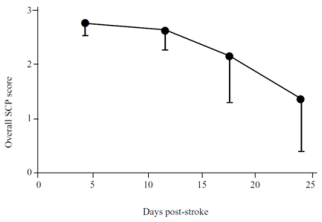
Karnath & Broetz 2003

Intervention

- Broetz 2004 (case report)
  - Realize contraversive tilt
  - Explore visual surroundings
  - Reach/transfer to non-paretic side
  - Add in dual tasking
- Shepherd and Carr 2005
  - Visual vertical cues
  - Reaching to paretic side
  - Focus on sit<>stand first
  - Try BWSTT

Visual Feedback Training – Case Report

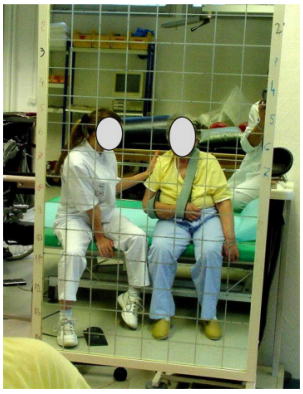
- Timeframe: 3.5weeks
- 30min, 6days/week
- Results:
  - Day 4: no patients could sit unsupported
  - Day18: 8/8 patients could stand while being supported by PT
  - Day 24: 6/8 patients could sit unsupported



Broetz et al 2004

Sitting Balance

- **First goal: achieve midline in static sitting**
- Sit on a firm stable surface (mat table)
- Feet should be supported on ground
- Use mirror for visual feedback
  - Can add vertical tape line
  - Could also put tape on pt's shirt
  - Utilize other "vertical" references in environment
- Can utilize a physical barrier on non-paretic side (wall, second person, exercise ball, etc)
- Tactile cues to the ischial tuberosity for weight shift to the unaffected side
  - Sidelying on elbow/forearm of non-paretic arm

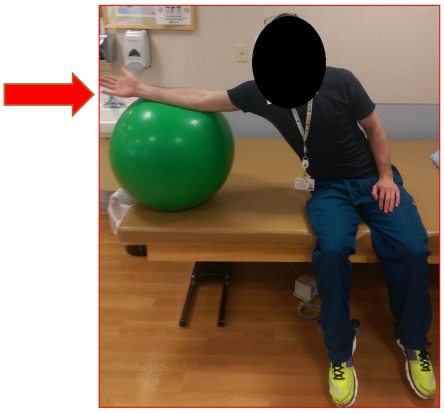


Perennou 2013

http://www.neuropt.org/docs/default-source/csm2017handouts/pusher-syndrome---neuroscience-evidence-assessment-amp-treatment.pdf?sfvrsn=2

Sitting Balance

- **Goal: prevent pushing**
- PT sitting on paretic side
- Do NOT push/pull patient to midline
- Non-paretic arm placement
  - Supinate and externally rotate pushing hand
  - Rest arm on exercise ball
  - Verbal/tactile cues to relax shoulder
  - Second person sits on non-paretic side – hand on their shoulder or leg
- Place pushing hand up
- Watch positioning of leg
  - Block with your foot or give verbal cues



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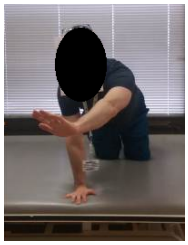
Sitting Balance – Dynamic

- **Next goal: move in and out of midline**
- Patient should be actively moving (don't passively correct)
- Dynamic reaching
  - Can start with sliding hand on mat (increase distance; raise height of object)
  - Facilitate trunk for return to midline
  - Reaching towards unaffected side (may be active-assisted)
  - Also want to reach to paretic side (promote UE WB'ing)
    - Can work on visual scanning (pt may have visual neglect)
  - Incorporate A-P movements as well for midline orientation
- Quadruped
  - Add unilateral reaching
- Tall kneeling
  - Add bilateral reaching, trunk rotation

http://www.neuropt.org/docs/default-source/csm2017handouts/pusher-syndrome---neuroscience-evidence-assessment-amp-treatment.pdf?sfvrsn=2



https://appliedstrokerehab.wordpress.com/2015/07/23/strategies-for-the-treatment-of-pusher-syndrome/

Wheelchair Positioning /Mobility

- Need appropriate seating system!
  - Supportive cushion and backrest for midline
  - Pelvic positioning belt for safety
- If pt has visual neglect
  - Bright tape if visual neglect present
  - Allow errors with mobility to practice scanning

Transfer Training

- Elevate EOM
- Elevated mat on non-paretic side for sit<>stand from wheelchair
  - Cue weight shift
  - Keep elevated height to prevent leaning/heavy weight-bearing

http://www.neuropt.org/docs/default-source/csm2017handouts/pusher-syndrome---neuroscience-evidence-assessment-amp-treatment.pdf?sfvrsn=2

Standing

- Practice weight shifting with elevated mat on non-paretic side (used as visual/tactile cue)
  - Can also use a flat wall or corner
- Add in reaching tasks or pre-gait activities
  - Reach for objects (varying heights)
  - Stepping forward/backward
  - Step up's
  - Visual scanning

http://www.neuropt.org/docs/default-source/csm2017handouts/pusher-syndrome---neuroscience-evidence-assessment-amp-treatment.pdf?sfvrsn=2

https://appliedstrokerehab.wordpress.com/2015/07/23/strategies-for-the-treatment-of-pusher-syndrome/

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Robotic-Assisted Gait Training

Kim 2017

Single Session Results??

- Krewer et al (2013)
  - Gait-assisted training had significant effect on BLS
  - Forced control of upright position + massed practice = immediate reduction in pushing
- GVS (Galvanic Vestibular Stimulation) did not produce significant improvements
  - Not effective because not a vestibular problem

TENS

- Applied to contralesional side of neck while performing balance activities
- Improved upright orientation with TENS on patients with pushing behavior + spatial neglect + somatosensory loss
- Theory: "reactivating damaged neural circuits involved in somaesthetic graviception"

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