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Higher Level Gait Disorder

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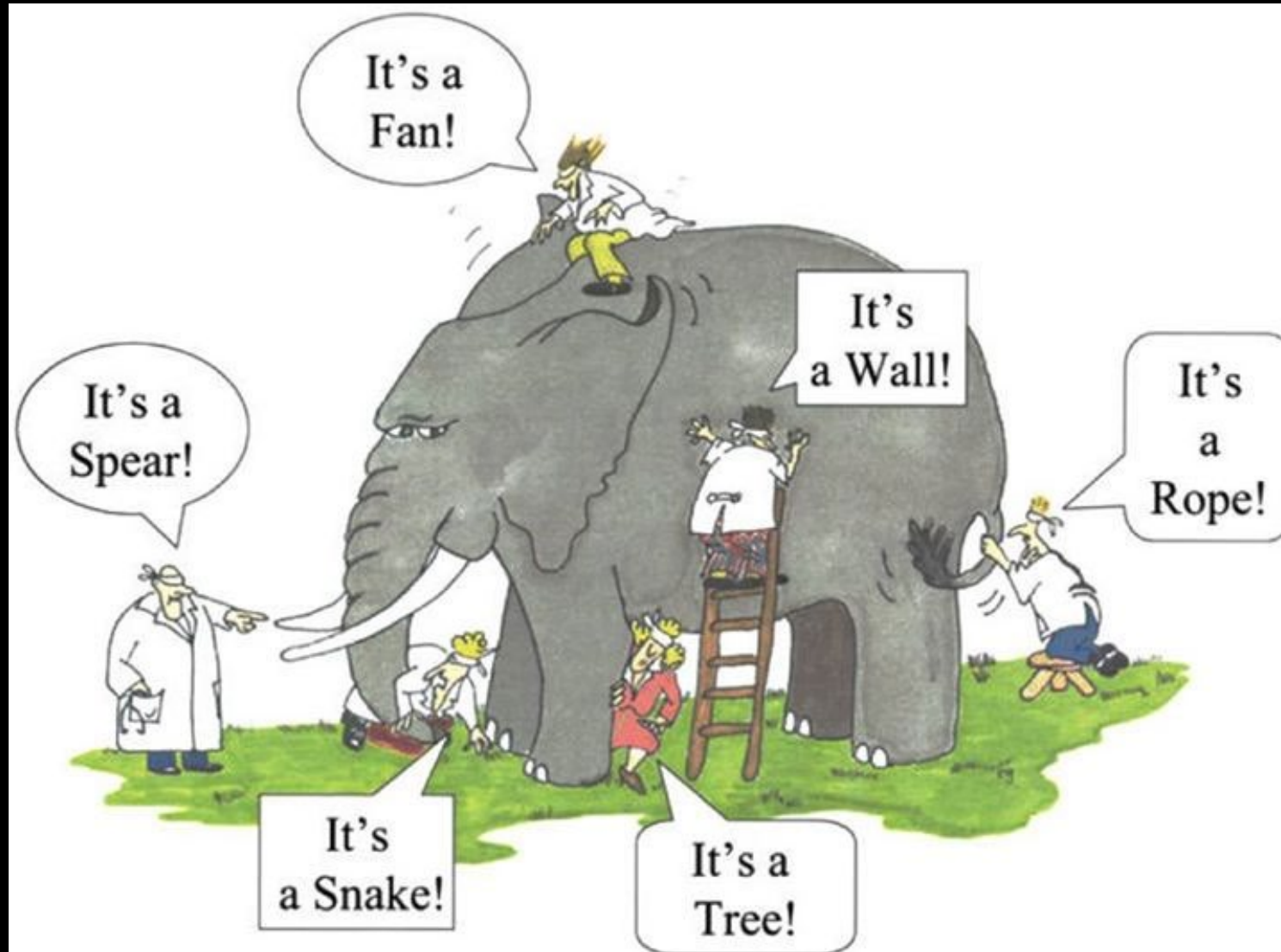
Disclosures

- No relevant disclosures

Learning Objectives

- Recognize the phenomenology of higher order gait disorders and outline management strategies

Higher Level Gait Disorder



Definitions

- HLGD is a category of gait disorders
 - Not just a single gait pattern
- HLGD is not a single pathological or etiological entity but may arise in a variety of neurodegenerative, vascular, neoplastic, and other conditions
- Locomotor **and** balance difficulties that cannot be explained by peripheral motor, sensory, pyramidal, cerebellar, or basal ganglia lesions

Contributions of John Hughlings Jackson to Neurology

A Brief Introduction to His Teachings

F. M. R. WALSH

Arch Neurol. 1961;5(2):119-131. doi:10.1001/archneur.1961.00450140001001

- Anterior Horn Cells - lower level
- Basal Ganglia and Motor Cortex - middle level
- Prefrontal Cortex - higher level

Higher-Level Gait Disorders: An Open Frontier

John G. Nutt, MD*

Department of Neurology, Oregon Health and Science University, Portland, Oregon, USA

Movement Disorders, Vol. 28, No. 11, 2013

H I G H E R - L E V E L G A I T D I S O R D E R S : A N O P E N F R O N T I E R

TABLE 1. Jacksonian classification of gait disorders

Level	Anatomy	Function	Example
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Motor	Motor neuron, muscle	Force	Motor neuropathy
Sensory	Sensory nerve, vestibular nerve, vision	Orientation in environment	Sensory neuropathy

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Motor	Corticospinal, cerebellum, basal ganglia	Refine force	Myelopathy with spasticity
Sensory	?		

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Sensory	?		
Higher	Cortex, basal ganglia	Collate and interpret sensory information; select and modify motor programs	Frontal lobe vascular disease

Clinical Features

- HLGD
 - Postural instability
 - Stepping dysrhythmicity
 - Recurrent falls
 - Progressive immobility
- Gait and balance patterns that cannot be attributed to the classic motor or sensory deficits
- Frequent concomitant cognitive impairment
- Rapid progression with loss of independence

Spectrum

- Can be signs of lower/middle-level dysfunction but that the lower/middle-level dysfunction is insufficient to explain the gait disorder
- Lower/Middle-level dysfunction may evolve into HLGD
 - Parkinsonism>>Balance difficulties and Freezing of Gait
- Differing trajectories within patients
 - Differences in the pattern of underlying pathology

Examination

- Classic neurological examination less relevant (e.g. power, reflexes, cerebellar, etc..)
- More of a functional assessment
 - Sitting and standing posture
 - Rising from the seated position
 - Walking and Turning
 - Tandem walking
 - Response to postural perturbation

OPINION

10.1038/nrneurol.2017.178

Neurological disorders of gait, balance and posture: a sign-based approach

Jorik Nonnekes, Rianne J. M. Goselink, Evžen Růžička, Alfonso Fasano, John G. Nutt and Bastiaan R. Bloem

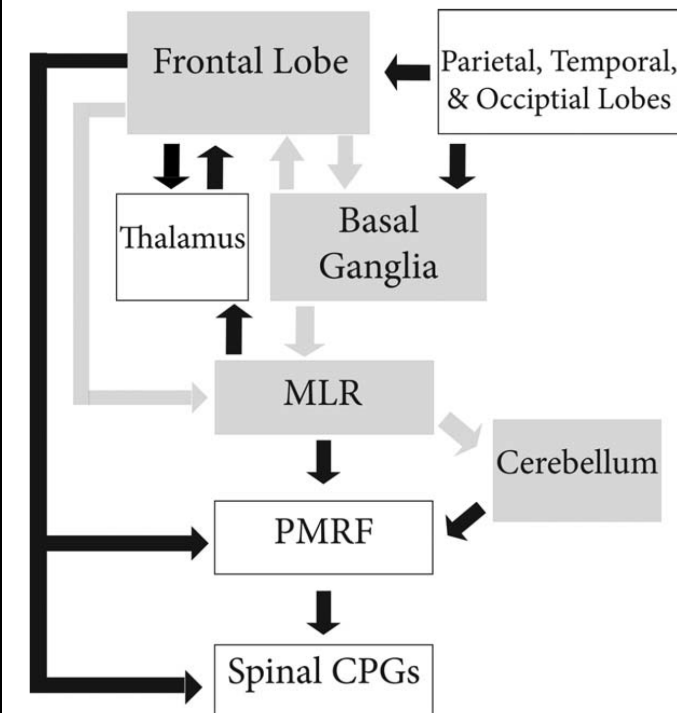
- Abnormalities observed during sitting, rising from a chair, and quiet standing**
- Abnormalities observed during the walking component of the clinical assessment**
- Additional tests that can be used to elicit informative gait and balance features**

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- Anterior (*Frontal*)
- Posterior (Parietotemporo-Occipital)
- May have pathophysiological implications



Anterior (Frontal)

- Phenotype
 - Characterized by freezing of gait (FOG), start and turn hesitation, small steps, and disequilibrium
- Pathophysiology
 - Dysfunction in the cortical-basal ganglia loop
- *Unlike* Lower/Middle Dysfunction
 - Performance varies depending upon environment, mood, fatigue and other variables

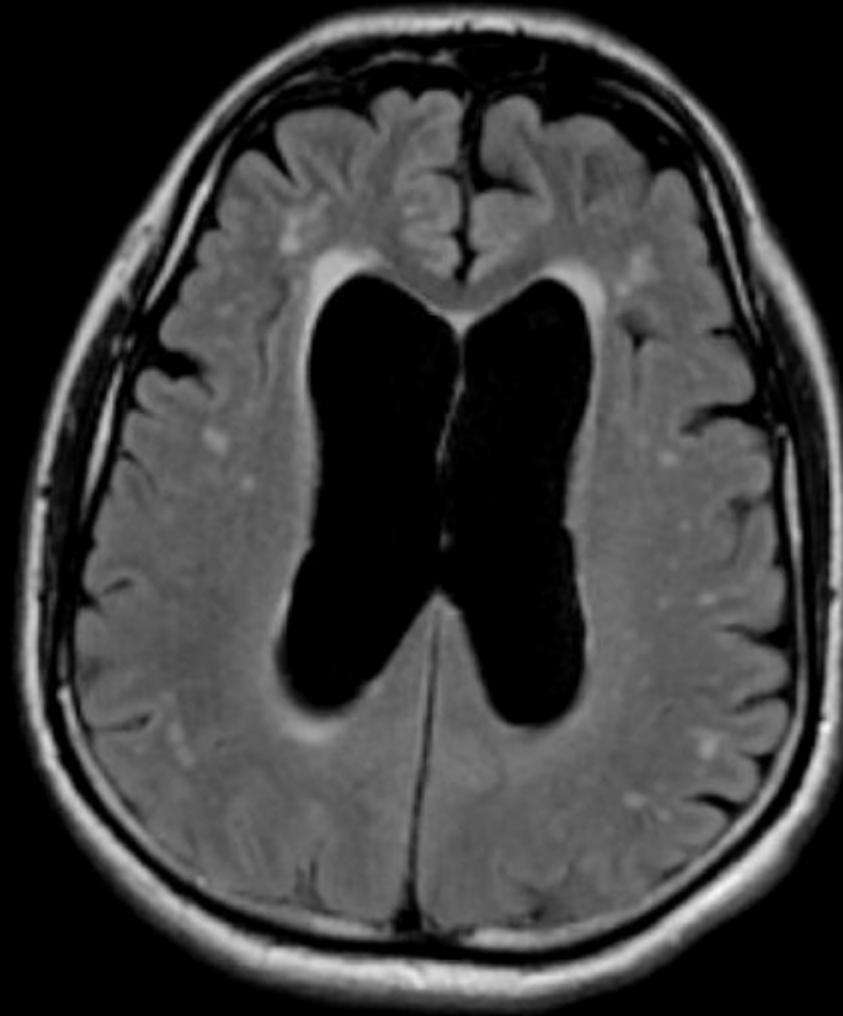
Anterior (Frontal) HLGD

- Various parkinsonian syndromes
- Multi-infarct and subcortical small-vessel disease
- Normal Pressure Hydrocephalus
- Other frontal lobe pathologies

Frontal Subtypes

- Predominantly *locomotor* subtype
 - Start and turn hesitation and FOG
 - PAGF or primary progressive FOG
- Predominantly *disequilibrium* subtype
 - Balance disorder
 - Progressive Supranuclear Palsy
 - Frontal lesions
- Often merge over time

Normal Pressure Hydrocephalus



Impact of Alzheimer's Disease Co-Pathology on Shunt Outcomes in Idiopathic Normal Pressure Hydrocephalus: A Systematic Review and Meta-Analysis

Movement Disorders, 2026 DOI: 10.1002/mds.70333

Ameya Patwardhan, MD,^{1,2*} Mojtaba Sharafkhah, MD,^{3,4} Michael Borrie, MD,¹ and Jaspreet Bhangu, MD¹

- Pre-shunt AD co-pathology prevalence
– 23.9% to 67.6%

Prevalence and clinical impact of alpha-synuclein pathology in idiopathic normal pressure hydrocephalus: Insights from RT-QuIC assay

Parkinsonism and Related Disorders 136 (2025) 107857

Je kook Yu^{a,v}, Hye Joung Choi^{a,v}, Dongwoo Kim^c, Pham Hong Ngoc^{a,d}, In Hee Kwak^{a,b},
Huu Dat Nguyen^{a,d}, Trung Nguyen Thanh^{a,d}, Suk Jun Song^{a,d}, Jeongjae Lee^{a,b},
Hyeo-il Ma^{a,b}, Young Eun Kim^{a,b,d,*}

doi.org/10.1016/j.parkreldis.2025.107857

- RT-QuIC α -synuclein pathology
– 11/33 patients (33.3 %) tested positive

Posterior HLGD

- Abnormalities in the spatial realm
 - Disturbed sense of postural vertical
- Pusher syndrome
 - Parietal and Thalamic strokes
 - T2 hyperintense lesions in white matter of parietal and occipital lobes
- Postural vertical
 - May be 20 degrees off vertical
 - Patient pushing toward the lesioned side

Research article

Open Access

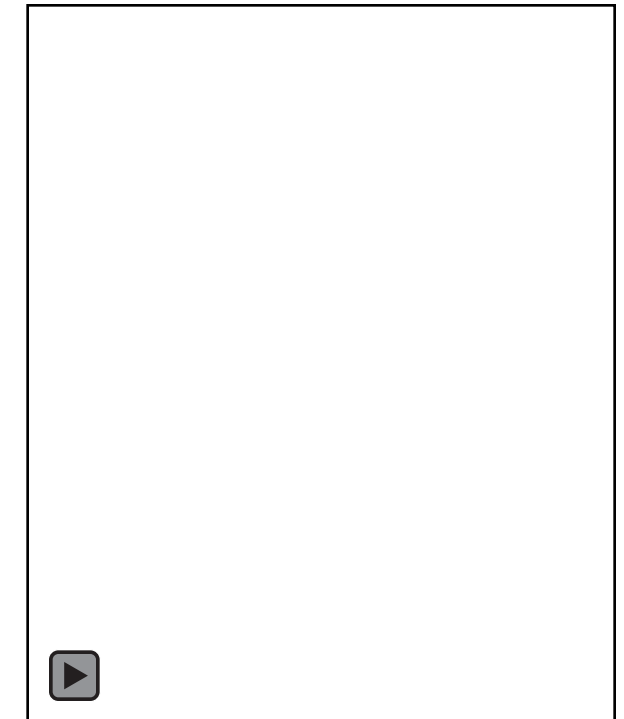
Leg orientation as a clinical sign for pusher syndrome

Leif Johannsen*^{1,2}, Doris Broetz¹ and Hans-Otto Karnath¹

Ipsiversive trunk tilt

No trunk tilt

Contraversive trunk tilt



<https://youtu.be/MIl-mkFneE4>

ORIGINAL RESEARCH ARTICLE

Effect of Rivastigmine on Mobility of Patients with Higher-Level Gait Disorder: A Pilot Exploratory Study

Tanya Gurevich · Yacov Balash · Doron Merims ·
Chava Peretz · Talia Herman · Jeffrey M. Hausdorff ·
Nir Giladi

- 15 non-demented HLGD (79 y)
- Open label rivastigmine for 12 weeks (mean 5mg/day)
- Assessed weeks 0, 12 and 16 after a 4-week washout period

Effect of Rivastigmine on Mobility of Patients with Higher-Level Gait Disorder: A Pilot Exploratory Study

Table 1 Effects of rivastigmine on cognitive characteristics and gait parameters in 15 patients with higher-level gait disorder

	Baseline, week 0 (<i>n</i> = 15)	After treatment, week 12 (<i>n</i> = 15)	Washout after treatment, week 16 (<i>n</i> = 15)	Pillai's trace test
Mean rivastigmine dose (mg/day)	0	5.1 ± 2.3	0	
MMSE	28.3 ± 1.4	28.13 ± 1.1	28.4 ± 1.4	NS
Mindstreams global cognitive score	90.43 ± 7.1	91.52 ± 7.5	93.47 ± 9.8	NS
Memory subscale	85.75 ± 9.6	88.97 ± 6.6	93.98 ± 13.1	$F(6,724) = 0.508^a$ $p = 0.010$
Anxiety subscale	37.46 ± 7.6	34.26 ± 8.1	38.53 ± 10.0	NS
Executive function subscale	90.10 ± 8.5	90.56 ± 8.4	92.72 ± 8.7	NS
Visuospatial subscale	86.49 ± 11.0	86.99 ± 15.8	86.6 ± 12.7	NS
Attention subscale	92.48 ± 14.9	96.29 ± 12.7	98.19 ± 12.8	NS
ABC (fear of falling) scale	68.3 ± 12.6	69.7 ± 16.0	65.7 ± 17.8	NS
STAI (Spielberger Anxiety Inventory)	37.5 ± 7.6	34.3 ± 8.1	38.5 ± 10	$F(7,792) = 0.545$ $p = 0.006$
Geriatric Depression Scale	9.4 ± 5.7	9.07 ± 5.3	10.26 ± 5.8	NS
Timed Up and Go test (s)	14.1 ± 3.8	13.1 ± 2.4	13.5 ± 2.5	$F(4,863) = 0.448$ $p = 0.028$
Gait speed (m/s)	0.86 ± 0.8	0.90 ± 0.1	0.90 ± 0.2	NS
Stride-time variability (%)	3.65 ± 1.3	3.29 ± 1.0	3.36 ± 1.3	NS

Rivastigmine for gait stability in patients with Parkinson's disease (ReSPonD): a randomised, double-blind, placebo-controlled, phase 2 trial

Lancet Neurol 2016; 15: 249-58

Emily J Henderson, Stephen R Lord, Matthew A Brodie, Daisy M Gaunt, Andrew D Lawrence, Jacqueline C T Close, A L Whone, Y Ben-Shlomo**

- Rivastigmine (3-12mg) over 32 weeks
 - Improved step time variability
 - Reduced falls per month
 - Improved gait speed whilst dual-tasking
 - Reduced freezing during the last month of trial

CHIEF PD (CHolinesterase Inhibitor to prEvent Falls in Parkinson's Disease) (CHIEF PD)

Conventional physiotherapy and treadmill re-training for higher-level gait disorders in cerebrovascular disease

Age and Ageing 2000; **29**: 311–318

RICHARD LISTON, JANE MICKELBOROUGH, BARBARA HARRIS, ALISON WYNN HANN, RAYMOND C. TALLIS

- Single-blind crossover study (HLGD n = 18)
 - 4-week baseline period, 4 weeks of treadmill re-training and 4 weeks of conventional physiotherapy
 - Gait ignition failure and turning; postural alignment and enhanced balance reactions; other components of cerebral multi-infarct state disordered gait
 - No significant difference between interventions but significant improvements

Summary

- HLGD is a category of gait disorders
 - Not just a single pathology
 - Multiple aetiologies
- Network disorder
- Physiotherapy
- Greater understanding will enhance future therapies

