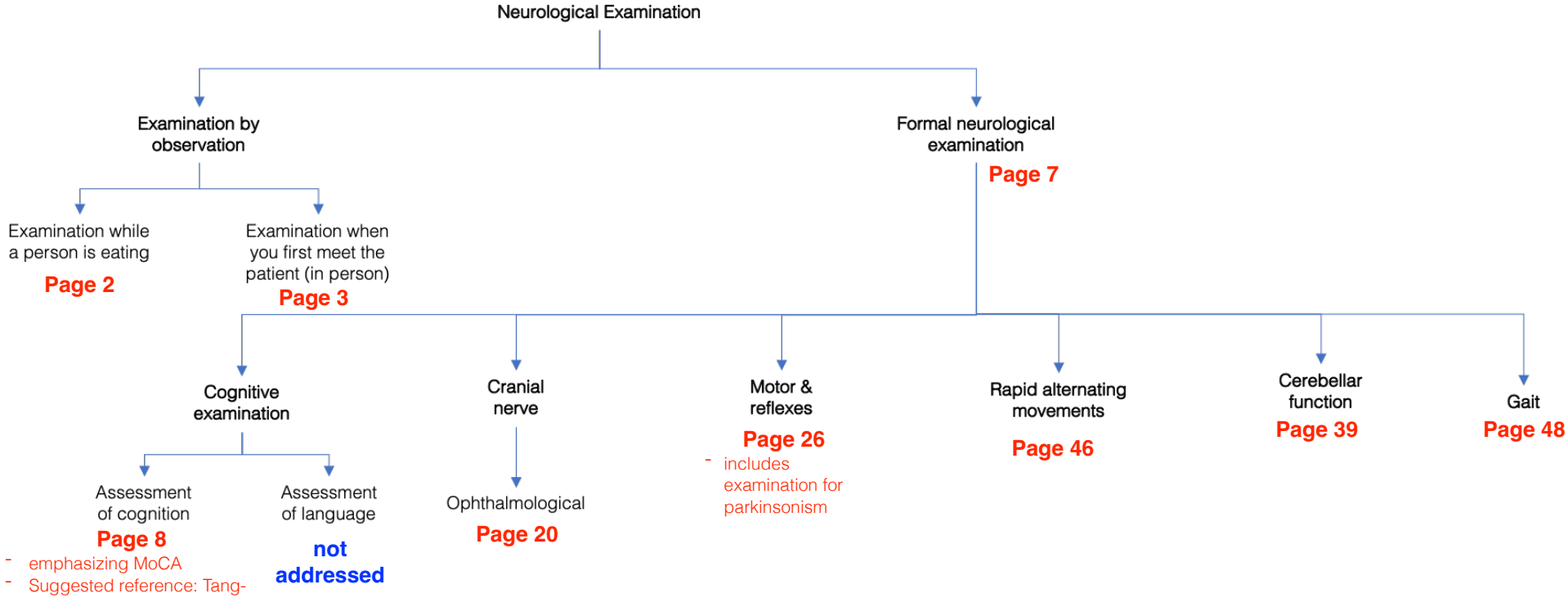
The Neurological Examination of Neurodegenerative Dementias

David F. Tang-Wai MDCM FRCPC
Professor (Neurology & Geriatric Medicine), University of Toronto
11th Canadian Conference on Dementia, Toronto ON, Saturday November 2, 2023

OUTLINE



- emphasizing MoCA
- Suggested reference: Tang-Wai DF, Freedman M. Bedside Approach to the Mental Status Assessment. Continuum (Minneap Minn). 2018 Jun;24(3, BEHAVIORAL NEUROLOGY AND PSYCHIATRY):672-703.

- includes examination for parkinsonism

DISCLAIMER

This handout emphasizes an approach and the common neurological findings among neurodegenerative dementias. It is not meant as a replacement of a more comprehensive neurological examination assessment.

What to Observe When Eating (rev 2023)

Olivia Geen MD & David F. Tang-Wai MD

Motor Features

Facial features

Features to observe include:

- Unilateral facial weakness - *e.g.* UMN or previous Bell's palsy
- Parkinsonism - *i.e.* hypomimia, chin tremor, seborrhea, sialorrhea

Eye features

Features to observe include:

- Parkinsonism - *i.e.* decreased eye-blink
- Square-wave jerks when looking at you
- Can look around the room without moving their head - if not, suggests limitation/restriction of eye movements - & indicates supranuclear palsy or cranial nerve (3 or 4 or 6) deficit

Speech & Swallowing

Features to observe include:

- Dysarthria
- Dysphagia - *NB:* subtle dysphagia presents with delayed coughing after swallowing liquids, solids, or both
- Is person eating a modified diet

Arm & Hand Mobility

Features to observe include:

- Asymmetric arm or hand movement - the side that has less movements may be weak due to either a UMN or LMN lesion; other possibility is musculoskeletal disorder
- Tremor of the hand or arm - distinguish if at rest (*i.e.* parkinsonism) or with action/movement (*i.e.* action tremor, essential tremor)
- Weakness of hand

Using utensils

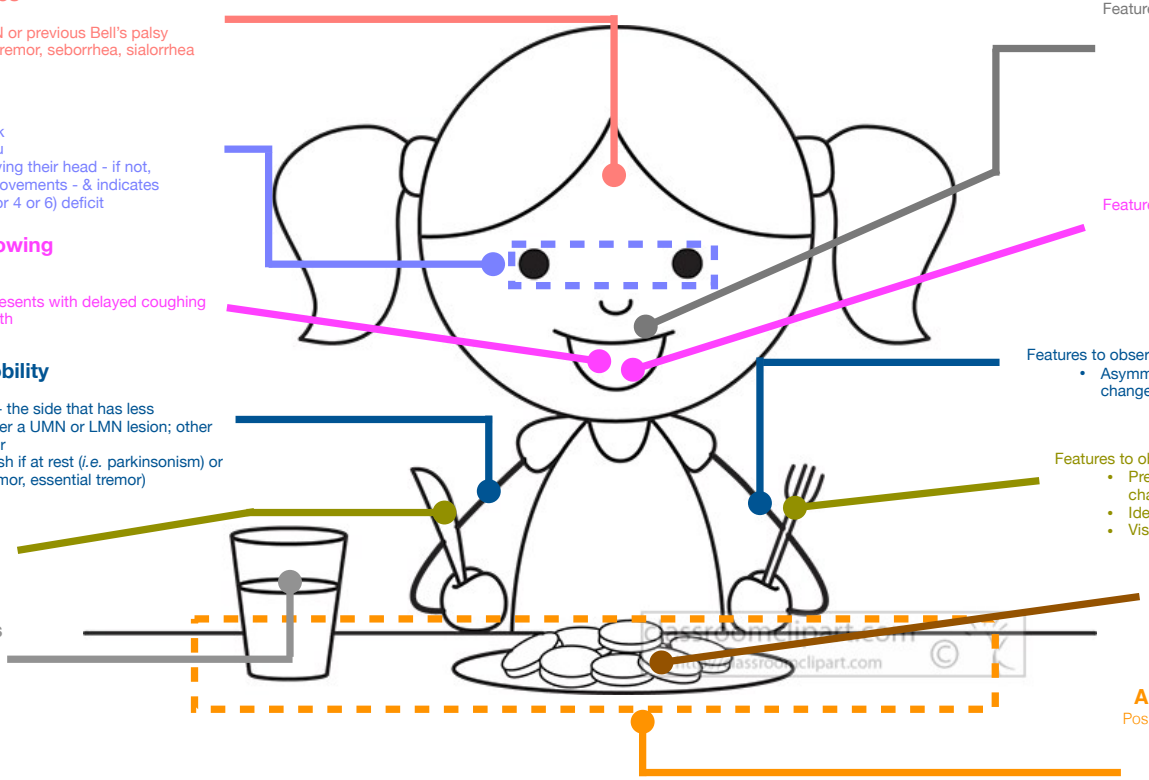
Features to observe include:

- Weakness
- Decreased dexterity

Reaching for items

Features to observe include:

- Weakness
- Ataxia
- Tremor



Cognitive/Behavioural Features

Language

Features to observe include:

- Dysphasia/aphasia
- Anomia
- Oral apraxia
- Circumlocution
- Paraphasic errors (semantic and/or phonemic)
- Fluent or non-fluent speech

Speech & Swallowing

Features to observe include:

- Putting too much food in mouth
- Pouching food

Arm & Hand Mobility

Features to observe include:

- Asymmetric arm or hand movement - due to neglect **or** change in handedness (as seen in CBS/CBD)

Using utensils

Features to observe include:

- Preferential use of their ***non-dominant*** hand - suggests change in handedness
- Ideomotor apraxia
- Visual agnosia - not recognizing utensil

Food habits

Features to observe include:

- Carbohydrate/sugary foods instead of other foods
- Hyperorality
- Poor table manners

Ability to "see" items on a plate or tray

Possible causes of impairment include:

- Poor visual acuity
- Visual field defect
- Neglect (visual or spatial)
- Simultanagnosia

WHEN TO DO

Consider when patient is:

- admitted to hospital
- has behavioural issues and not amendable to a formal examination
- anytime

What To Observe When First Meeting Your Patient - Outpatient Setting

PASSIVE OBSERVATION

Signs of Parkinsonism
- Decreased eye-blink frequency
- Hypomimia
- Procerus sign (vertical wrinkling on the forehead imparting a “staring,” reptile-like, or “astonished” look characterized by lid retraction and infrequent blinking - commonly seen in PSP)
- Resting chin tremor

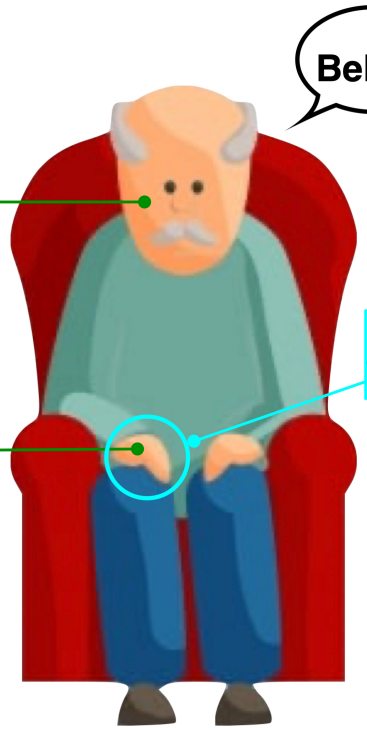
Facial Weakness
- Unilateral facial weakness (droopiness, flattening of nasolabial fold)

Signs of Parkinsonism
- Presence of rest tremor
- Decreased movements on one side (see below for differential)

Weakness
- Decreased movements on one side (see above for differential) - especially if not moving dominant side compared to non-dominant side

Neglect
- Not moving dominant side compared to non-dominant side - need to perform more exam maneuvers

ACTIVE OBSERVATION



Dysarthria
- Due to Parkinsonism (hypotonic hypokinetic dysarthria)?
- Due to weakness? Cranial neuropathy? LMN weakness? UMN weakness?

Aphasia
- Need to perform language assessment

Change in handedness
- Due to weakness?
- Due to neglect?

“Misses” your hand
- Due to visuospatial impairment (optic ataxia)?
- Due to cerebellar dysfunction?

Text with detailed explanations on next pages

The Neurological Examination of Dementia Starts in the Waiting Room - What to Observe for

Elements to Observe	Potential Observations (Abnormalities)	Potential Clinical Interpretations	What to Focus on the Neurological Examination
Saying "hello" or other introduction *from the patient*	Dysathria	• Bulbar weakness • Parkinsonism	• Cranial nerve examination with focus of CrN 5, 7, 9, 10, 11, & 12 • Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings
	Unable to answer **OR** answers inappropriately	• Aphasia	• Assessment of language (comprehension, sentence repetition, naming, reading, semantic knowledge)
Handshake	Shakes your hand with their **non-dominant** (left) hand (as most people - even left-handers - shake hands with right hand)	• Change in handedness (seen in corticobasal degeneration) • Weakness • Neglect	• Examination for Parkinsonism • Examination of cortical sensory function (tactile neglect with double simultaneous stimulation/ extinction, graphesthesia, stereognosis) • Motor examination
	Misses your hand to shake	• Visuospatial impairment (optic ataxia +/- simultanganosia) • Appendicular cerebellar ataxia	• Examination of cortical visual function (optic ataxia, ocular motor apraxia, simultanagnosia) - overlapping figures, copy figure, Trails A/B, Ishihara plates, finger nose testing) • Examination of cerebellar function
Standing from chair	Pushes themselves to standing position with their arms OR rocks themselves to standing position	• Weakness in proximal legs (hip flexors > knee extensors) • Parkinsonism	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings
	Retropulsion (takes step back into chair OR falls back into chair upon standing)	• Postural instability (commonly seen in PSP, VCI) • Weakness • Ill fitting shoes	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings • Examine shoes to see if too large

WHEN TO DO
Anytime

Elements to Observe	Potential Observations (Abnormalities)	Potential Clinical Interpretations	What to Focus on the Neurological Examination
Walk/gait from waiting room to examination room	Shuffling	• Non-specific finding - seen in multiple etiologies including VCI, parkinsonism, NPH • Shoes too big	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings • If not already asked, inquire about signs of NPH • Examine shoes to see if too large
	Asymmetric arm +/- leg movement	On the side with ***lesser*** movement: • Parkinsonism - asymmetric • Weakness	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings
Entering examination room	Freezes at doorway before entering room	• Higher-order gait dysfunction	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings • If not already asked, inquire about signs of NPH
	Bumps into doorway on one side	• Visual field cut • Visuospatial impairment • Neglect	• Cranial nerve examination - visual fields, including visual neglect • Examination of cortical sensory function (tactile neglect with double simultaneous stimulation/ extinction, graphesthesia, stereognosis) • Examination of cortical visual function (optic ataxia, ocular motor apraxia, simultanagnosia) - overlapping figures, copy figure, Trails A/B, Ishihara plates, finger nose testing)
Sitting in chair	Unable to align themselves properly to sit in chair (body is perpendicular to chair OR body is too far away from chair OR body sits on half of chair and falls)	• Body-space impairment - visuospatial impairment (right parietal dysfunction) • Cortical sensory dysfunction	• Examination of cortical visual function (optic ataxia, ocular motor apraxia, simultanagnosia) - overlapping figures, copy figure, Trails A/B, Ishihara plates, finger nose testing) • Examination of cortical sensory function (tactile neglect with double simultaneous stimulation/ extinction, graphesthesia, stereognosis)

Elements to Observe	Potential Observations (Abnormalities)	Potential Clinical Interpretations	What to Focus on the Neurological Examination
Spontaneous arm movements	Decreased spontaneous arm movements on the **dominant** side	• Parkinsonism - asymmetric • Weakness • Neglect	• Examination for parkinsonism • Complete motor examination for UMN, LMN or mixed UMN & LMN findings • Examination of cortical sensory function (tactile neglect with double simultaneous stimulation/ extinction, graphesthesia, stereognosis)
Facial features	Hypomimia/masked facies AND/OR chin tremor	• Parkinsonism	• Examination for parkinsonism
	Procerus sign (vertical forehead wrinkling around the bridge of the nose and the glabella)	• Parkinsonism - commonly seen in PSP	• Examination for parkinsonism - with attention to EOM, saccade velocity, presence of axial > appendicular rigidity
Eye movements	Looks around the room with head instead of looking around with eyes AND/OR no/limited spontaneous eye movements	• Parkinsonism - commonly seen in PSP	• Examination for parkinsonism - with attention to EOM, saccade velocity, presence of axial > appendicular rigidity
Arms and legs	Rest tremor	• Parkinsonism	• Examination for parkinsonism

The Formal Neurological Examination

Myths and Truths of the Neurological Examination

Myth	Reality
1. The neurological examination is meant to **rule out** (i) parkinsonism, (ii) stroke, (iii) other "neurological" disorders.	The neurological examination, in conjunction with the patient's history, should ***rule in*** a diagnosis.
2. The order of administration of the neurological examination is not important.	The order of the components ensures that (i) each exam is consistently administered within and between patients and (ii) provides additional information on what to look for next.
3. 4/5 power is normal in the elderly.	Ageism! 4/5 power is NEVER normal.
4. The presence of glabellar tap can diagnose parkinsonism.	It is the constellation of multiple signs that will diagnose the presence of parkinsonism.
5. A single sign on neurological examination can provide a diagnosis.	**No** single sign on neurological examination can provide a diagnosis.

Components of the Neurological Examination and the Order of Administration

Components of the Neurological Examination	
1. Mental status examination	6. Sensory
2. Speech/language	7. Coordination
3. Cranial nerves	8. Rapid alternating movements
4. Motor	9. Gait
5. Reflexes	

Cognitive Testing, Localization & Interpretation
With emphasis of interpreting the Montreal Cognitive Assessment (MoCA)

1. **Cognitive tests (*e.g.* Montreal Cognitive Assessment, Mini-Mental Status Exam, Toronto Cognitive Assessment) are comprised of subtests that examine a specific cognitive domain.**

Cognitive Domain	**MMSE** (**M**ini-**m**ental **s**tatus **e**xam)	**MoCA** (**Mo**ntreal **C**ognitive **A**ssessment)	**TorCA** (**Tor**onto **C**ognitive **A**ssessment)
Orientation	Yes	Yes	Yes
Memory - Learning/Delayed recall	Yes	Yes	Yes
Attention	Yes	Yes	Yes
Language	Yes	Yes	Yes
Visuospatial	Yes	Yes	Yes
Executive Function	Yes	Yes	Yes

Key Points
- No cognitive tests are equal as differences exist in sensitivity and specificity among different types of tests.
- Screening tests, such as MMSE or MoCA, may not detect milder changes in cognition when compared to intermediate cognitive tests or neuropsychology tests (see below)
- **It is not the total score rather the pattern of cognitive impairment that is important.**

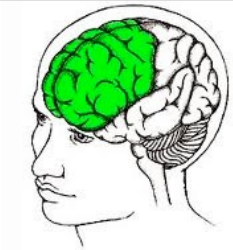
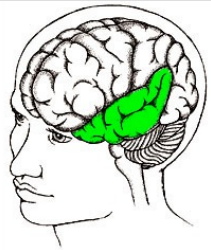
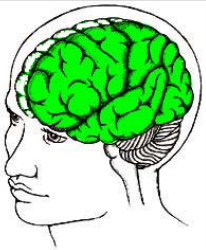
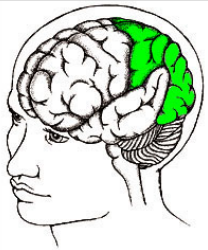
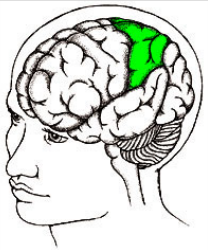
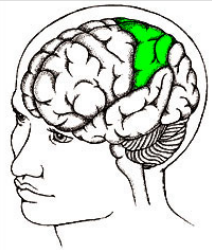
- Neuropsychological testing involves detailed assessments (specific test) of each cognitive domain

Cognitive Domain	Examples of Neuropsychological Tests
Orientation	
Memory - Learning/ Delayed recall	Logical (story) memory, California adult verbal learning test (CVLT), Free-cued recall
Attention	Reverse digit span, letter cancellation
Language	Boston Naming Test, Token Test (comprehension)
Visuospatial	Rey-O complex figure, block design
Executive Function	Wisconsin card sorting, Stroop, Trails Making Test

- Spectrum of available cognitive testing

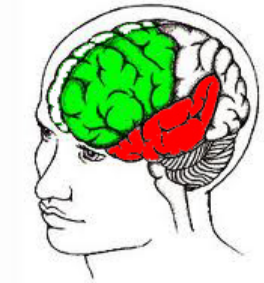
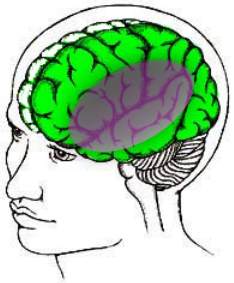
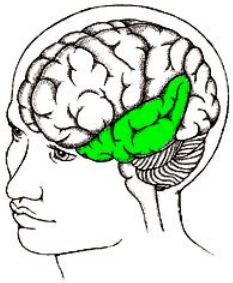
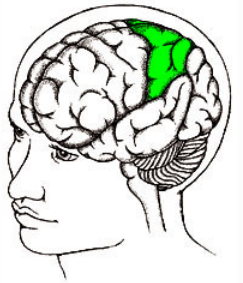
Screening Tests (5-15 minutes)	Intermediate Tests (35-60 minutes)	Detailed Tests (>60 minutes)
• ACE III (Addenbrooke's cognitive examination III) • MoCA • MMSE • SLUMS (St. Louis University Mental Status Examination) • STMS (Short Test of Mental Status, Mayo Clinic)	• TorCA (Toronto Cognitive Assessment, University of Toronto) • DCG (Depistage Cognitif de Quebec, University Laval)	• Neuropsychology testing

2. Cognitive testing should be considered as an extension of the neurological examination as each cognitive domain is localized to a specific lobe in the brain.

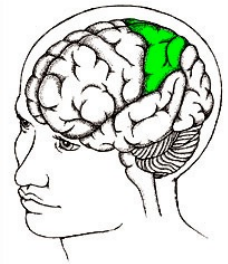
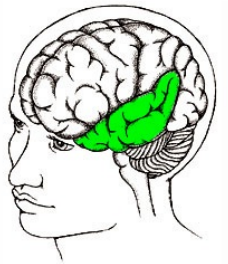
	Executive Function	Memory	Language	Visuospatial	Arithmetic	Ideomotor Praxis
Localization in the Brain	Frontal Lobe	Temporal Lobe	Left Hemisphere	Parietal & Occipital Lobes	Left parietal lobe	Left parietal
						
Function of the cognitive domain	• ability to plan, perform abstract reasoning, solve problems, focus despite distractions and shift focus when appropriate • deficit in SOAP: **S**equencing; **O**rganizing; **A**bstracting & **P**lanning	• ability to learn and recall new information	• either comprehension or expression	• comprehension and effective manipulation of nonverbal, graphic or geographic information	• simple arithmetic - adding, subtraction, multiplication, division	• the ability to perform skilled actions

Sample Tests	• Trails B
	• Digit span (reverse > forward)
	• WORLD/Serial 7's
	• Verbal **(letter)** fluency
	• Letter cancellation
		• Orientation
		• Learning
		• Delayed Recall
			• Reading
			• Naming
			• Writing
			• Repetition
			• 3-step command
			• Semantic (category) fluency
				• Cube *copy*
				• Pentagon *copy*
				• Benson complex figure *copy*
				• Overlapping figures
					• Calculations (simple arithmetic):
					  • 5 x 13=
					  • 65 - 7 =
					  • 59/2 =
					  • 29 + 11 =
						• Ask patient to show how to comb their hair, brush their teeth

3. Localization can be further refined to a specific area within a lobe of the brain

	Language (left hemisphere)			
	Fluency[1] (phonemic/letter & semantic/category)	**Repetition (sentence)**	**Naming**	**Writing**
Localization	• Letter fluency (frontal lobe) • Semantic fluency (temporal lobe)	Left Perisylvian Area (shaded purple area)	Left Temporal lobe	Left Temporal-Parietal Lobe
				

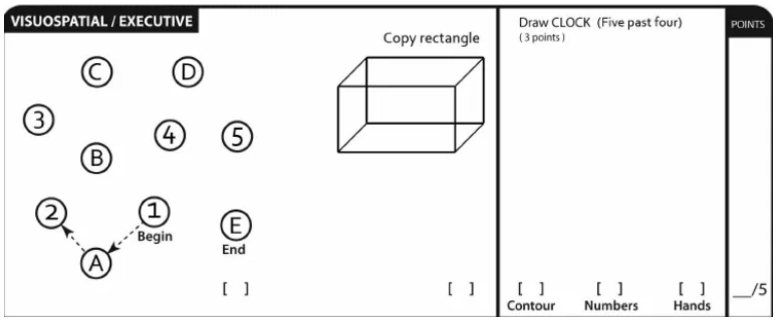
[1]Fluency in this circumstance refers to fluency in cognitive testing - not fluency in spontaneous speech

	Arithmetic, Writing Right-left discrimination (Gerstmann syndrome)	Facial Recognition
Localization		
	Left Parietal Lobe	**Right** Occipital-Temporal Lobes

4. Any cognitive test can be subdivided into the cognitive domain being tested. The MoCA is being used for illustrative purposes only.

Visuospatial

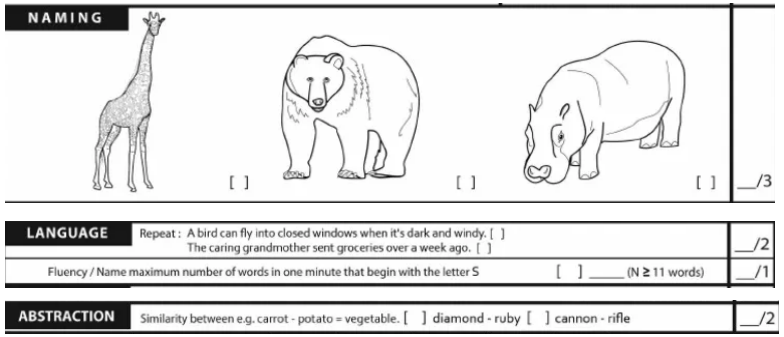
1.



It is also the only time you observe patient's ability to write/ draw

Language

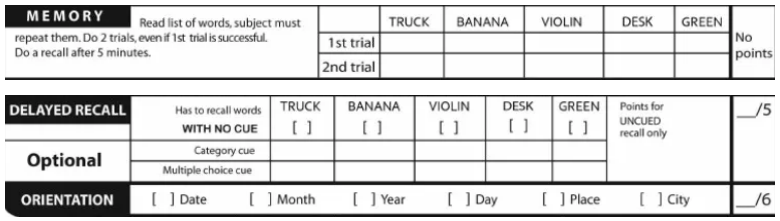
2.



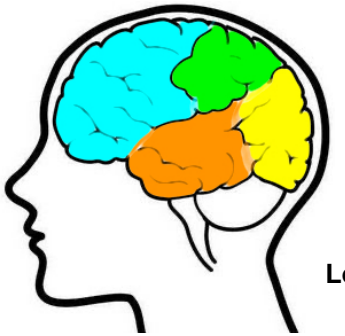
Partial assessment of a patient's language & you get to listen to how they answer the questions

Memory (Learning & Recall)

3.



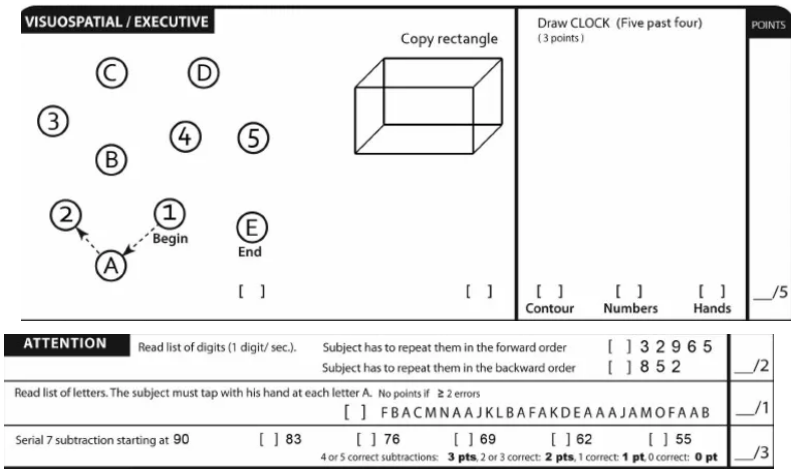
Assessment of patient's ability to learn and recall



Localization of cognitive functions in the brain

Frontal Executive/Attention

4.

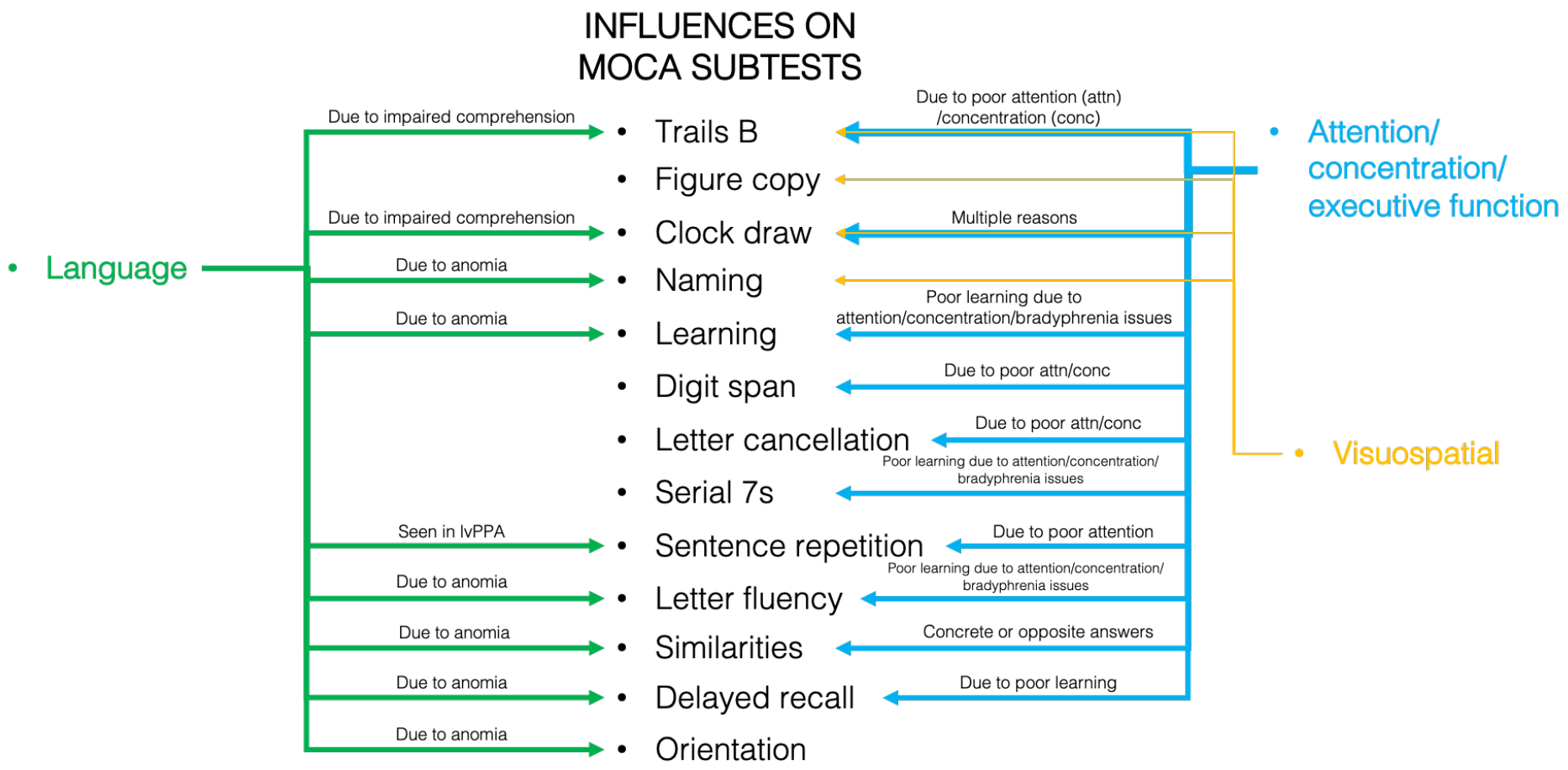


Assessment of patient's attention/concentration and ***some*** executive functions

Key Point

When looking al the results, look at which domain is **MOST** impaired to provide a clue into the patient's problem.

5. **Beware of how an impairment in a single cognitive domain can influence the entire test and can lead to an overcall of impairment.**



6. **Record the what the patient says when answering the questions as this can often provide clues into their cognitive impairment.**

Ask the question → *Record* the patient's answer → *Interpret* the patient's answer → *Analyze* the response in context with answers and performance from other questions used in the cognitive test

Need to record **WHAT** the patient says - not if answer is correct or not

Determine **HOW** the patient answers the question (*e.g.* anomia)

Determine **WHY** the patient answers the question in this manner - **is a pattern emerging?**

7. Determine what analysis of each cognitive domain can indicate to you

Memory (Learning & Recall)

3.

MEMORY	Read list of words, subject must repeat them. Do 2 trials, even if 1st trial is successful. Do a recall after 5 minutes.		TRUCK	BANANA	VIOLIN	DESK	GREEN	No points
		1st trial						
		2nd trial						

DELAYED RECALL	Has to recall words WITH NO CUE	TRUCK [ ]	BANANA [ ]	VIOLIN [ ]	DESK [ ]	GREEN [ ]	Points for UNCUED recall only	/5
Optional	Category cue							
	Multiple choice cue							
ORIENTATION	[ ] Date	[ ] Month	[ ] Year	[ ] Day	[ ] Place	[ ] City		/6

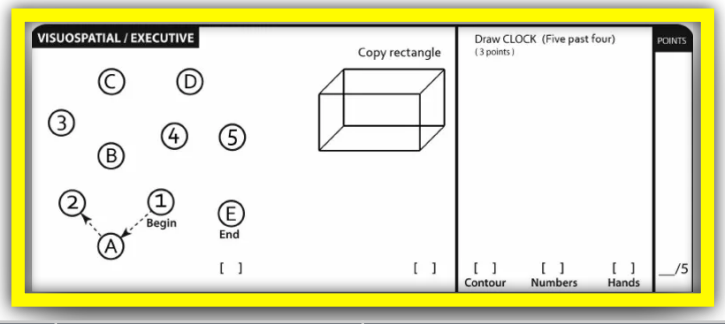
Assessment of patient's ability to learn and recall

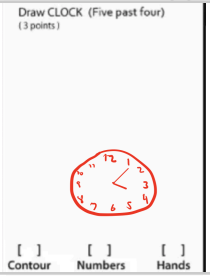
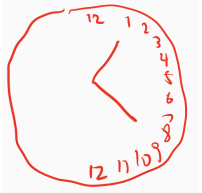
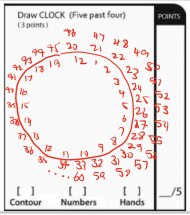
Clinical Pearls

1. Number the order of words learned as indicates learning ability
2. Observe for a pattern of poor learning with either good free recall and/or good cued/recognition recall with normal orientation to suggest a problem with learning (frontal dysfunction)
3. A person with an aphasia will perform poorly on both learning and delayed recall as the primary deficit is language/words.

MoCA Items	Normal	COGNITIVE DEFICITS		
		Amnestic		Attention/Concentration
		Mild	Moderate/Severe	
Learning *Do not forget to provide 2 (and only 2) learning trials even if the 1st trial is successful*	TRUCK BANANA VIOLIN DESK GREEN 1st trial 1 2 3 4 5 2nd trial 1 2 3 4 5	TRUCK BANANA VIOLIN DESK GREEN 1st trial 1 2 3 4 5 2nd trial 1 2 3 4 5	TRUCK BANANA VIOLIN DESK GREEN 1st trial 1 2 3 4 5 2nd trial 1 2 3 4 5	TRUCK BANANA VIOLIN DESK GREEN 1st trial 2 3 1 2nd trial 4 2 3 1
	Most learn the words in the order presented to them			Patients with poor attention/ concentration learn haphazardly and may not recall the words later
Delayed Recall (free & effect of cueing)	TRUCK BANANA VIOLIN DESK GREEN Category cue Multiple choice cue 5/5	TRUCK BANANA VIOLIN DESK GREEN Category cue Multiple choice cue 1 0/5	TRUCK BANANA VIOLIN DESK GREEN Category cue X 1 X X X Multiple choice cue X X X X X 0/5	TRUCK BANANA VIOLIN DESK GREEN Category cue 1 1 1 Multiple choice cue 0/5
	No issues with free delayed recall	Despite normal learning, recall may be better with cueing	Despite normal learning, recall is poor despite cueing	Patients may not: • recall the words later **OR** • demonstrate variable recall of words (including words not learned), **AND/ OR** • demonstrate better recall with cueing. [Retrieval problem]
Orientation	6/6	4-5/6	<3/6	6/6
	Performs well on orientation	Performs reasonably well on orientation	Performs poorly on orientation	Performs well on orientation
DISORDERS		• Amnestic MCI • Alzheimer's disease (typical, early/mild stage)	• Alzheimer's disease (typical, severe stage) - other domains will also be impaired on the MoCA	• Non-amnestic MCI • Vascular cognitive impairment • Parkinsonism (DLB, PDD, PSP) • Atypical Alzheimer's disease

Visuospatial

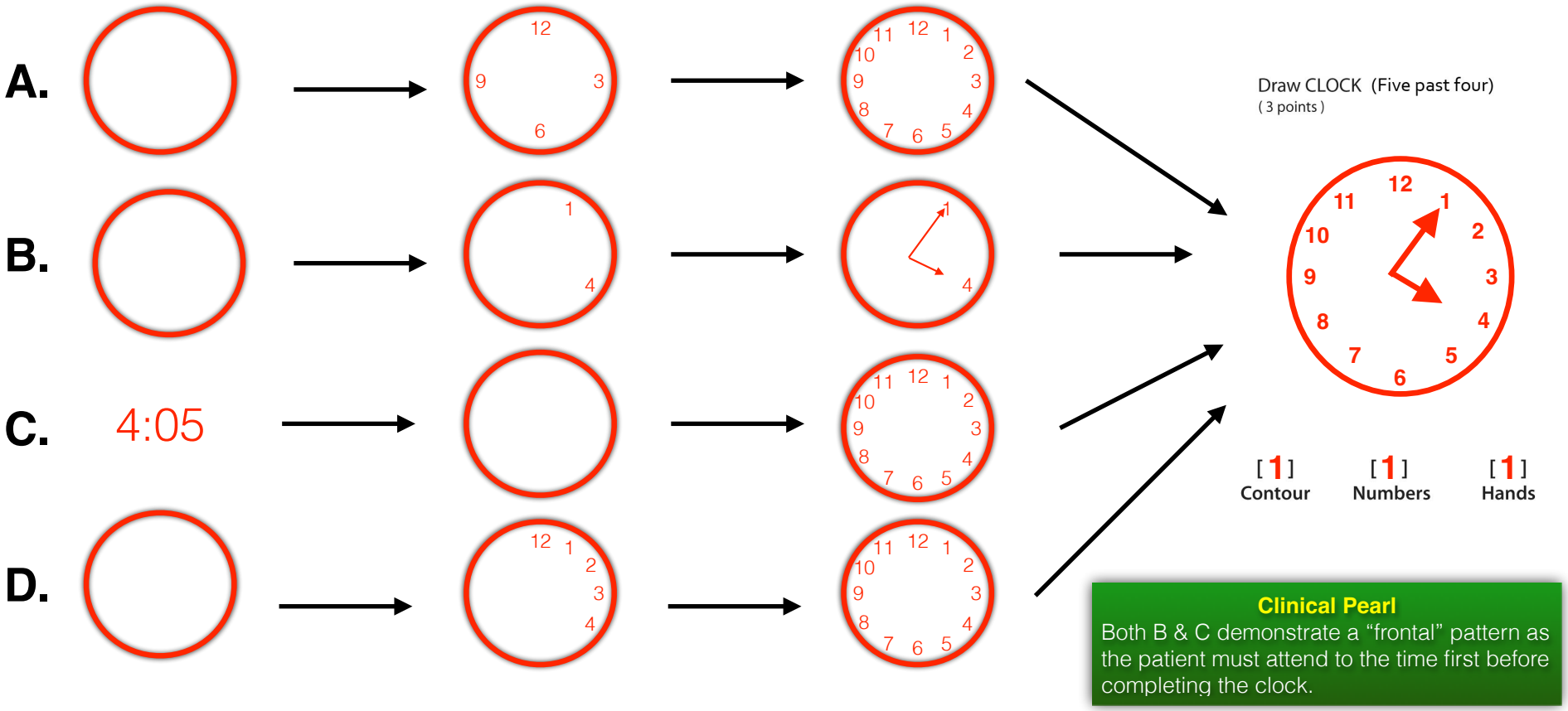


What can be observed	Sample Figure	Clinical Implications
Tremor		• Essential tremor • Parkinsonism
Micrographia		• Parkinsonism
Neglect		Parietal deficit - can be seen in: • Stroke • Neurodegenerative dementia (e.g. CBD/CBS)
Perseveration		Frontal lobe deficits - can be seen in: • bvFTD (behavioural variant frontotemporal dementia) • fAD - frontal variant Alzheimer's disease • Autoimmune encephalitis

Clinical Pearl

In addition to executive and visuospatial dysfunction, the following can be discerned from the patient's own writing/drawing. **You will still need to perform a full physical/ neurological examination to verify the deficits (e.g. Parkinsonism).**

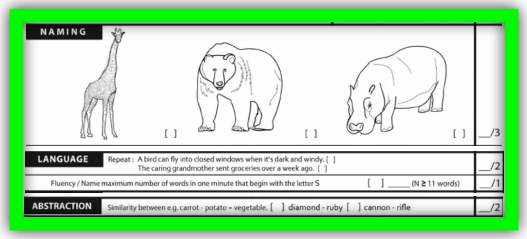
Clock Draw Observe how the patient draws the clock

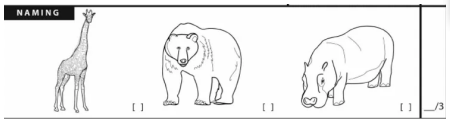


Clinical Pearl

Both B & C demonstrate a "frontal" pattern as the patient must attend to the time first before completing the clock.

Language





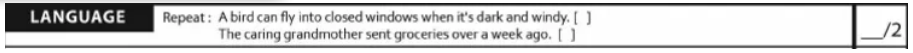
Inability to name some or all the animals

↓

Suggests an anomia

↓

Uses circumlocution to describe (*e.g. "found in Africa, has long neck, eats green stuff from trees"*)	Unable to describe or does not know what it is (but would have known previously)
↓	↓
Suggests *intact* semantic knowledge	Suggests *impaired* semantic knowledge
↓	↓
Example disorders to consider: 1. Logopenic or non-fluent primary progressive aphasia 2. Early Alzheimer's dementia	Example disorders to consider: 1. Semantic variant primary progressive aphasia 2. Advanced/severe Alzheimer's dementia



Impaired sentence repetition

↓

Due to impairment in language function	Due to impairment in attention/concentration
↓	↓
Disorders to consider: 1. Logopenic primary progressive aphasia 2. Advanced Alzheimer dementia	Disorders to consider: 1. Mood disorder 2. Parkinsonism 3. Frontal variant Alzheimer 4. Behavioual variant frontotemporal dementia

Impaired *letter* fluency

↓

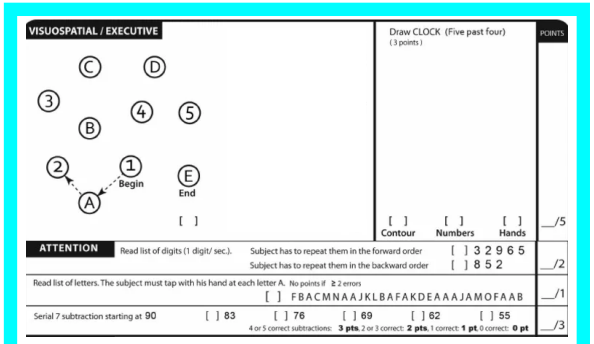
Due to impairment in language function	Due to impairment in attention/concentration
↓	↓
Disorders to consider: 1. Primary progressive aphasia 2. Advanced Alzheimer dementia	Disorders to consider: 1. Mood disorder 2. Parkinsonism 3. Frontal variant Alzheimer 4. Behavioual variant frontotemporal dementia

Clinical Pearls

1. **Sentence repetition:** To distinguish between language versus attention difficulties, recommend adding another sentence repetition test ***after*** the MoCA[2]. If normal, then suggests attention difficulties. If impaired, suggests language difficulties.
2. **Letter fluency:** Suggest adding semantic fluency ***after*** the MoCA (*e.g.* list as many animals in 1 minute). If semantic fluency is also impaired, suggests either PPA or AD. If semantic fluent is not impaired, suggests a frontal lobe dysfunction.

Frontal

Frontal Executive/Attention



Assessment of patient's attention/ concentration and *some* executive functions

1. Trails B
2. Construction of clock
3. Digit span
4. Letter cancellation
5. Serial subtractions

Clinical Pearl

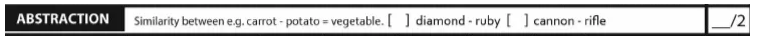
Remember **MoCA is a screening test**. If your patient has a history of executive dysfunction that is NOT evident on the MoCA, consider adding a full Trails A and Trails B, longer forward and reverse digit span (normal is 7 forward, 5 reverse).

Other potentially frontal (ie. hidden) tasks:

1. Learning

	TRUCK	BANANA	VIOLIN	DESK	GREEN
1st trial		2	3		1
2nd trial	4	2	3		1

2. Similarities



Impaired *similarities*

- Due to education and/or cultural issues
- Due to anomia
  - If patient uses circumlocution to describe similarities (*e.g.* for jewelry: found in store, expensive)
- Due to frontal/ dysexecutive difficulties
  - If patient provides opposites or concrete answer (*e.g.* for objects that measure: numbers)

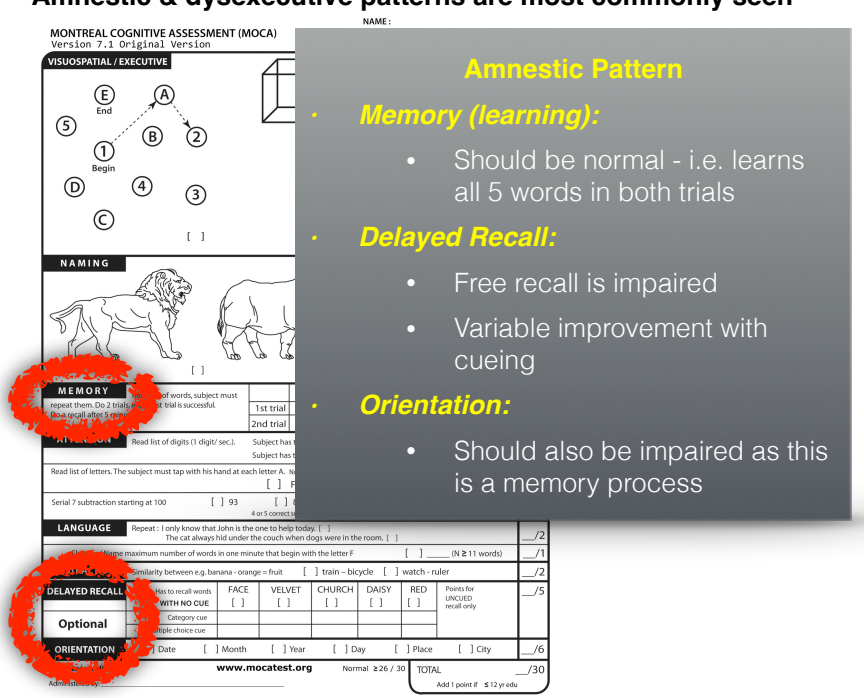
Clinical Pearl

Caveats to the interpretation of similarities

8. Determine pattern of cognitive impairment

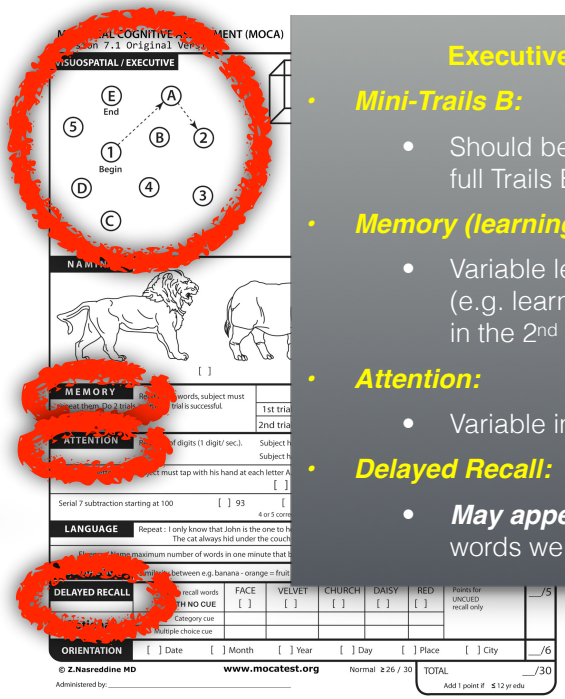
Major Deficit Seen On Testing	Pattern	Example Conditions
Orientation Delayed word recall	Amnestic	• Mild cognitive impairment (amnestic) • Alzheimer's dementia
Attention 3-step command Learning the words (if not complete)	Executive Dysfunction/Frontosubcortical	• Dementia with Lewy bodies • Parkinson's disease dementia • Vascular dementia • Normal pressure hydrocephalus
Drawing Trails (searching)	Visuospatial	• Posterior cortical atrophy • Dementia with Lewy bodies • Corticobasal degeneration
Naming Repetition Writing	Aphasia	• Primary progressive aphasia
Normal testing	Disinhibition	• Frontal/behavioural variant FTD

Amnestic & dysexecutive patterns are most commonly seen



Amnestic Pattern

- ***Memory (learning):***
  - Should be normal - i.e. learns all 5 words in both trials
- ***Delayed Recall:***
  - Free recall is impaired
  - Variable improvement with cueing
- ***Orientation:***
  - Should also be impaired as this is a memory process



Executive Dysfunction

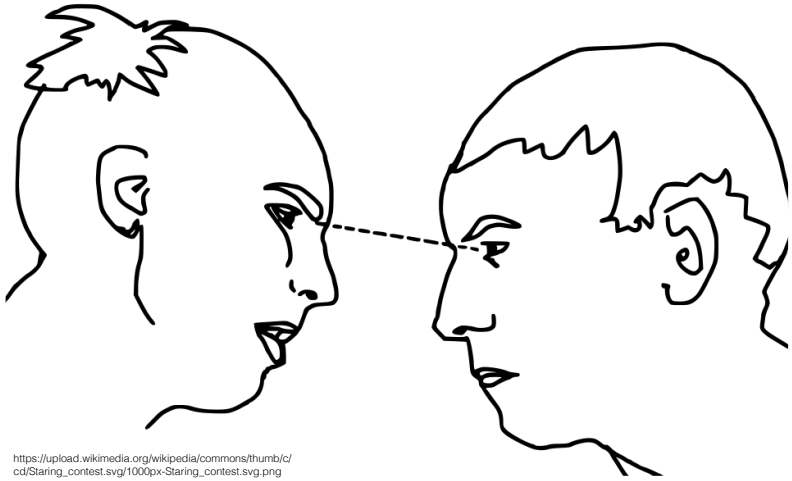
- ***Mini-Trails B:***
  - Should be impaired, if not perform full Trails B
- ***Memory (learning):***
  - Variable learning of the 5 words (e.g. learned 3 in the 1st trial and 5 in the 2nd trial)
- ***Attention:***
  - Variable impairment
- ***Delayed Recall:***
  - ***May appear to be impaired*** if words were not learned previously

9. Examine for overall patterns in the clinical case - combining history and exam

History - Initial Presentation	Cognitive Profile	Physical Exam	Cranial Nerves	Motor Examination	Reflexes	Gait	Disorder to Consider
Rapid forgetting	Amnestic	Normal (including vitals)	Normal	Normal	Normal	Normal	**Alzheimer's Disease**
Visuospatial	Visuospatial	Normal (including vitals)	• Normal • Visual field cut • Visual neglect	Normal	Normal	Normal	**Posterior Cortical Atrophy (AD)**
Anomia	Anomia Acalculia	Normal	Normal	Normal	Normal	Normal	**Logopenic progressive aphasia (AD)**
Slow, executive dysfunction, inattention	• Executive dysfunction • Slow	Signs of peripheral & cardiovascular disease	Normal	UMN pattern of weakness	Hyperreflexia Babinski sign	• Normal • Slow, decreased stride length • Hemiparetic gait	**Vascular Cognitive Impairment**
Behavioural changes (apathy or disinhibition)	• Normal • Executive dysfunction	Normal	Normal	• Normal • UMN pattern of weakness • LMN pattern of weakness	• Normal • Hyperreflexia • Babinski sign	Normal	**bv-FTD ± motor neuron disease**
Anomia - circumlocution	Anomia	Normal	Normal	• Normal • UMN pattern of weakness • LMN pattern of weakness	• Normal • Hyperreflexia • Babinski sign	Normal	**Non-Fluent Primary Progressive Aphasia (FTD) ± motor neuron disease**
• Anomia - loss of semantic knowledge • Prosopagnosia	Anomia	Normal	Normal	• Normal • UMN pattern of weakness • LMN pattern of weakness	• Normal • Hyperreflexia • Babinski sign	Normal	**Semantic Variant Primary Progressive Aphasia (FTD) ± motor neuron disease**

Observing What to See - The Neuro-ophthalmological Examination in Dementia

EXAMINATION OF EYE-BLINK FREQUENCY - IS IT REDUCED?



https://upload.wikimedia.org/wikipedia/commons/thumb/c/cd/Staring_contest.svg/1000px-Staring_contest.svg.png

CLINICAL PEARL

Blink at the same rate as your patient. If you need to blink first, the patient has a reduced eye-blink frequency. Consider any cause parkinsonian condition.

Key Points

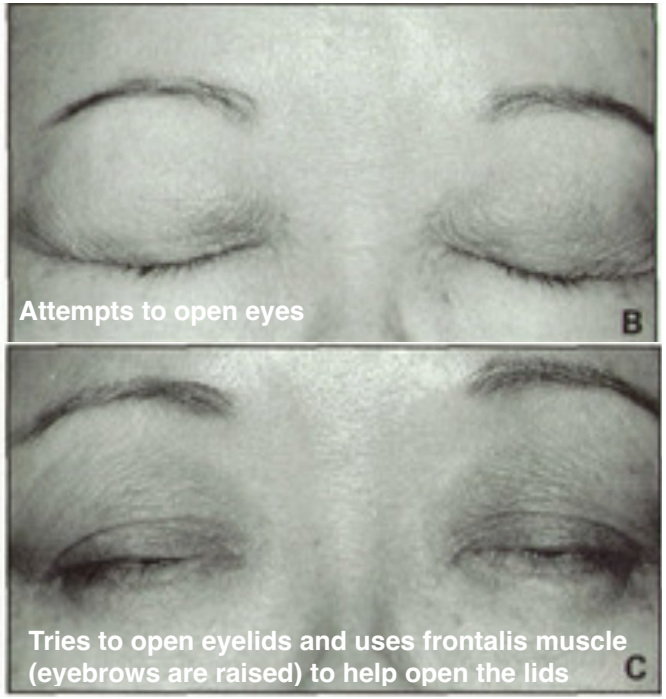
Normal:

- blink frequency is 12 to 20/minute
- eyelid opening apraxia does NOT occur

DISCLAIMER

All parts of the ophthalmological assessment are required. This handout illustrates some of the key differentiating features among common neurodegenerative causes of dementia.

EXAMINATION OF EYELIDS - IS IT IMPAIRED (EYELID OPENING APRAXIA)?



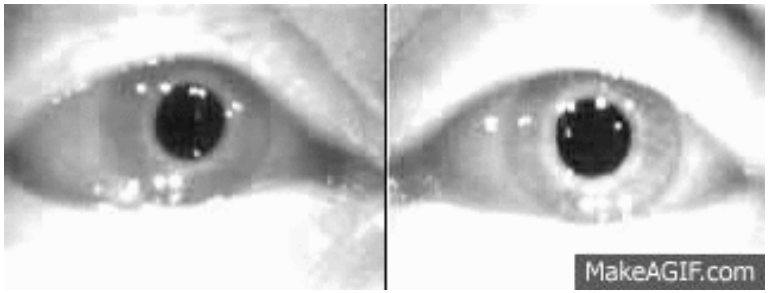
David R Jordan DR et al. *Ophthalmic Surgery, Lasers and Imaging Retina.* 2013; 21(5):331–334

- Apraxia of eyelid opening is defined as a non-motor abnormality characterized by the patient's difficulty in eyelid elevation bilaterally.
- Commonly occurs after the patient voluntarily close the eyes.
- The definition is a misnomer, given that it is very rarely pure and true apraxia.

CLINICAL PEARL

Eyelid opening apraxia is commonly seen in progressive supranuclear palsy (PSP) - thus examine for other features of PSP to support this diagnosis.

OBSERVE FOR SQUARE-WAVE JERKS

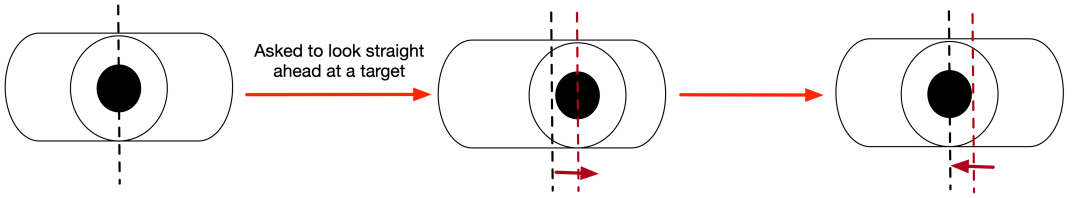


https://makeagif.com/gif/square-wave-jerk-YRJV9v

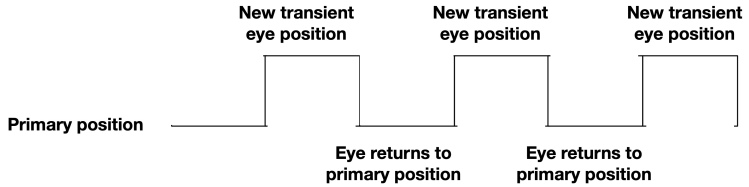
CLINICAL PEARL

Square-wave jerks is commonly seen in PSP and is one of the earliest findings.

A small saccade of 0.5 to 3° transiently takes the eye away from fixation on a stationary or moving target. After 200ms or more a similar saccade returns the eye to the target. This was named a square wave jerk after the shape it creates on ocular motor recordings. Seen in progressive supra nuclear palsy (PSP) and other disorders affecting the cerebellar-basal ganglia circuitry.

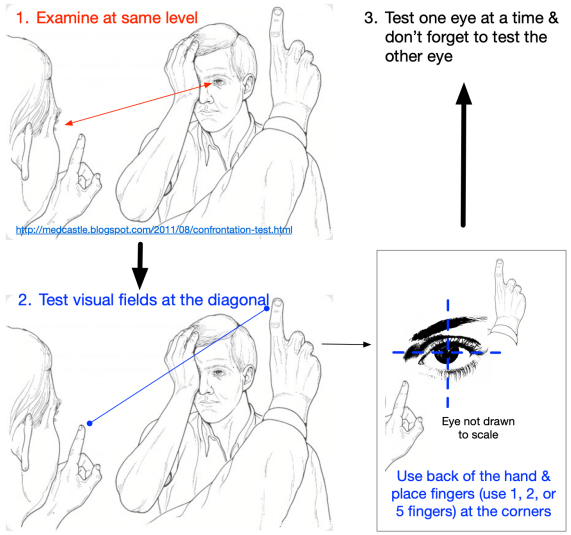


Graphical Representation of Square Wave Jerks

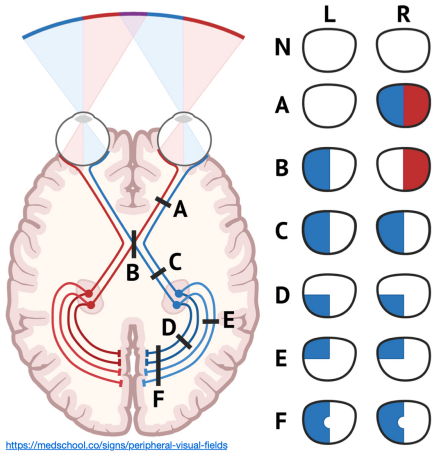


EXAMINATION OF VISUAL FIELDS

RAPID METHOD TO TEST VISUAL FIELDS



POTENTIAL FINDINGS

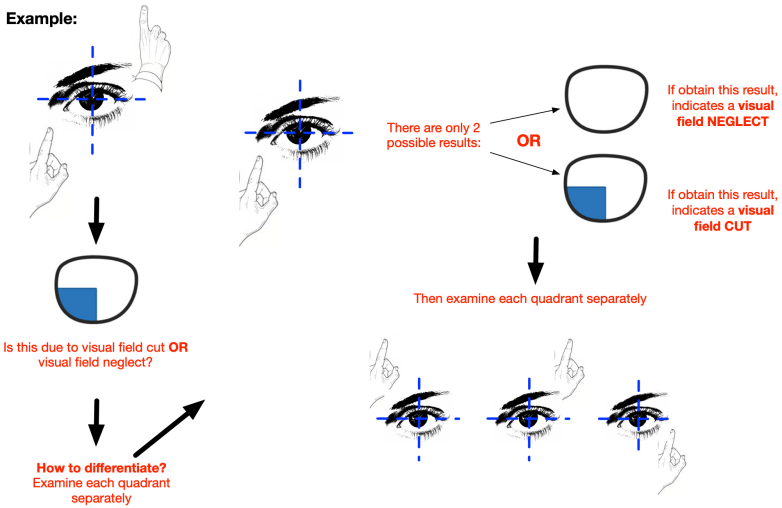


N = normal
A = unilateral anopia (unilateral optic nerve lesion *OR* ocular pathology)
B = bitemporal hemianopia (optic chiasmal compression)
C = homonymous hemianopia (contralateral optic tract lesion)
D = homonymous inferior quadrantnopia (contralateral upper optic radiation lesion)
E = homonymous superior quadrantnopia (contralateral lower optic radiation lesion)
F = homonymous hemianopia with macular sparing (contralateral occipital lobe lesion)

CAVEAT

If patient is not able to "see" a finger - this could be due to either a field cut OR visual neglect.

Example:



Visual neglect is a neuropsychological disorder of attention in which patients exhibit a lack of response to stimuli in one half of their visual field that cannot be explained by primary damage to the visual pathways (https://eyewiki.aao.org/Visual_Neglect#:~:text=Visual%20neglect%20(visual%20hemi%2Dinattention,to%20the%20visual%20geniculostriate%20pathways.).

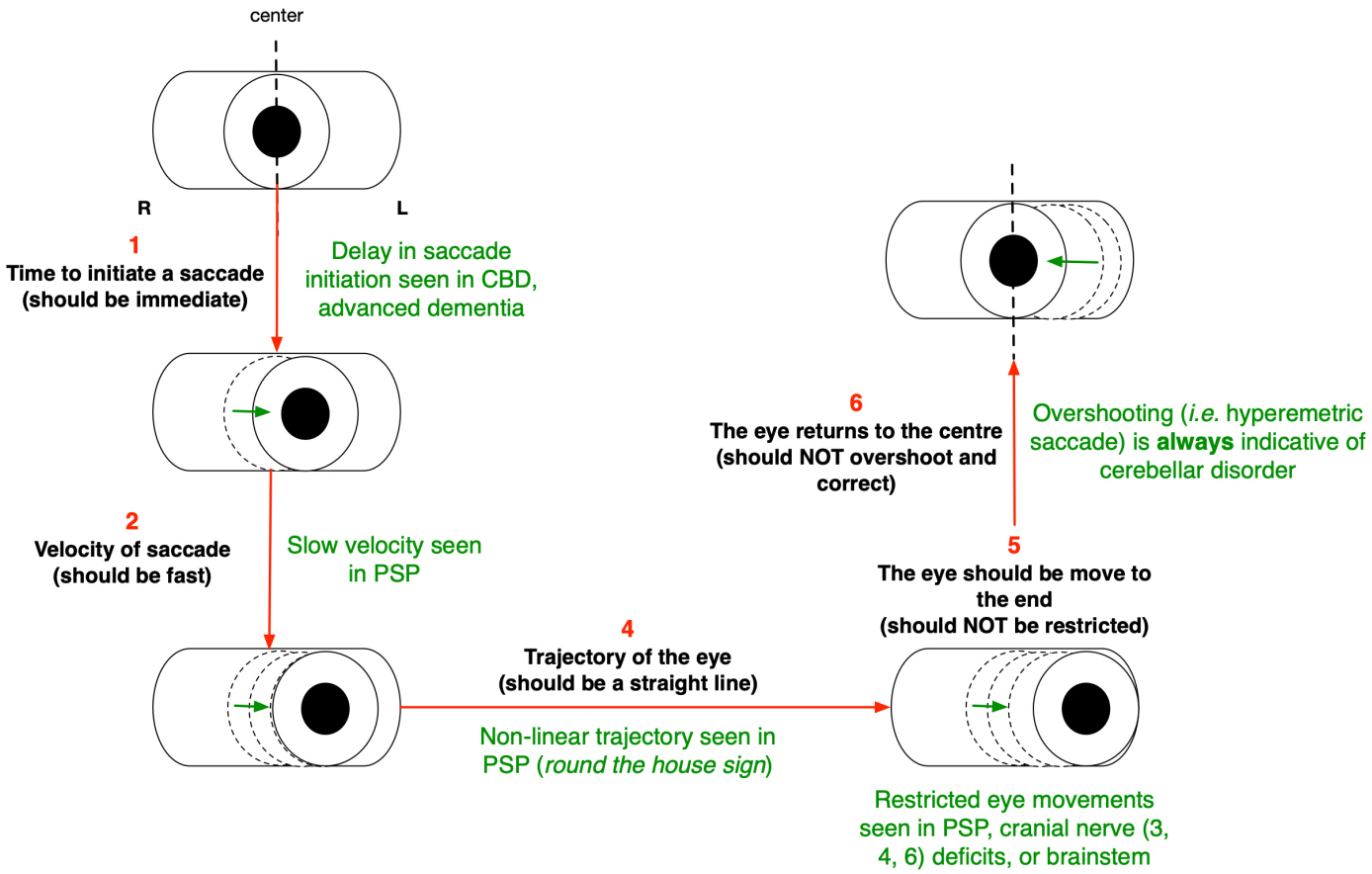
CLINICAL PEARLS

Visual field impairment can be seen in:

- stroke (visual field cut or visual neglect)
- posterior cortical atrophy (visual neglect)
- corticobasal degeneration (visual neglect)

EXAMINATION OF SACCADES - SOME COMPONENTS ARE INDICATIVE OF SPECIFIC DISORDERS

WHAT TO LOOK FOR WHEN EXAMINING SACCADES



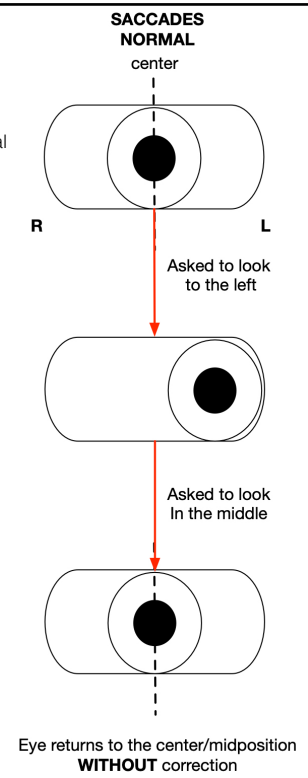
CLINICAL PEARLS

Observe the following parameters:

1. Time to initiate a saccade
2. Velocity of saccade
3. Trajectory of saccade - especially vertical
4. Restriction of saccade
5. Corrections when return to centre

Associated disorders are listed in the figure above.

Technique



Ask the patient to:

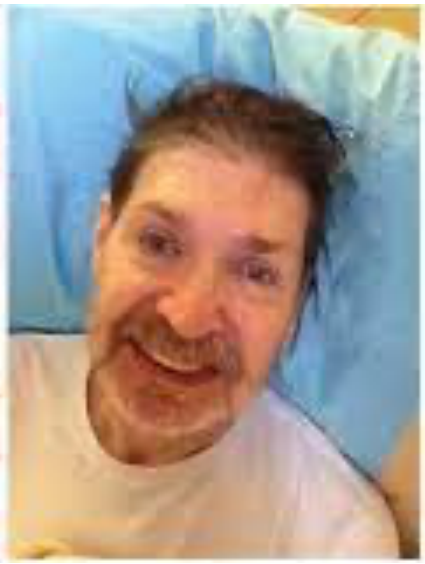
HORIZONTAL SACCADES

1. between your nose and a target off the left side and then back to you nose (or other midline target);
2. then from your nose to a target to the right side and back to your nose (or other midline target);

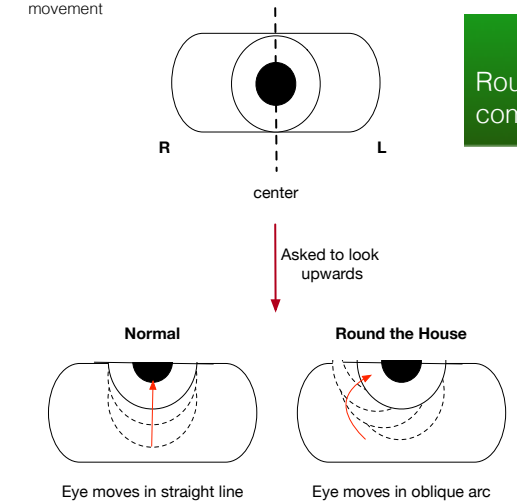
VERTICAL SACCADES

1. then from your nose to a target that is above their head in the midline and back to your nose (or other midline target);
2. then from your nose to a target that is below their head in the midline (or other midline target)

Saccade trajectory

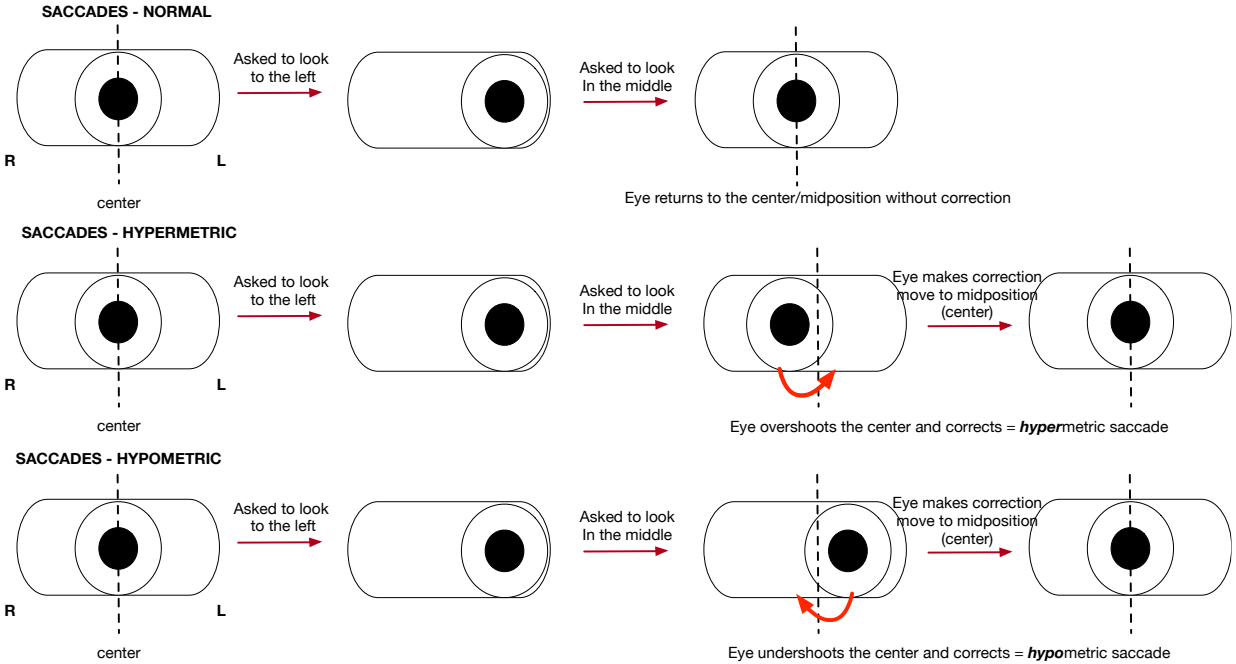


ROUND THE HOUSE SIGN:
The inability to produce vertical saccades along the midline. Instead, the eyes move vertically in a lateral or oblique arc to accomplish the movement



Clinical Pearl
Round the house sign is commonly seen in PSP.

Saccade accuracy



Clinical Pearls

1. **Hypo**metric saccades can be normal if there are no other neurological signs
2. **Hyper**metric is **always abnormal and indicates a cerebellar dysfunction**

NEURO-OPHTHALMOLOGICAL FINDINGS AMONG THE VARIOUS NEURODEGENERATIVE DEMENTIAS

	Eyelids				Pupils, Light Reflex & Visual Acuity	Colour Vision & Contrast Sensitivity	Visual Fields	Complex Visual Dysfunction		Eyes in Primary Position (Ocular fixation)
Abnormality	Decreased eyeblink frequency	"Apraxia" of eyelid opening	Blepharo-spasm	Lid retraction	Minimal change	Decreased contrast sensitivity & decreased colour vision	• Hemifield loss • Visual neglect	Simultan-agnosia	Impaired figure copying	Square-wave jerks
Disorders to consider	• PSP • CBD • MSA • PDD • DLB	• PSP • MSA • CBD		PSP		• AD (esp PCA) • CBD (if presents as PCA)	• AD (esp PCA) • CBD (if presents as PCA)	• AD (esp PCA) • CBD (if presents as PCA)	• AD • CBD • DLB	• PSP • CBD

	Eye Movements								
	Pursuits		Saccades						OKN
Abnormality	Saccadic (choppy) smooth pursuit	Impaired	Inability to initiate	Delayed initiation	Slow velocity	Limited range (restricted movement)	Hypometric	Hypermetric	Impaired
Disorders to consider	Any neurodegen-erative condition	• PSP • CBD	• CBD (if very delayed initiation • AD (oculomotor apraxia)	CBD	• PSP • CBD	• PSP (vertical followed by horizontal) • CBD	• Normal • Any neurodegenerative condition	MSA	PSP

LEGEND

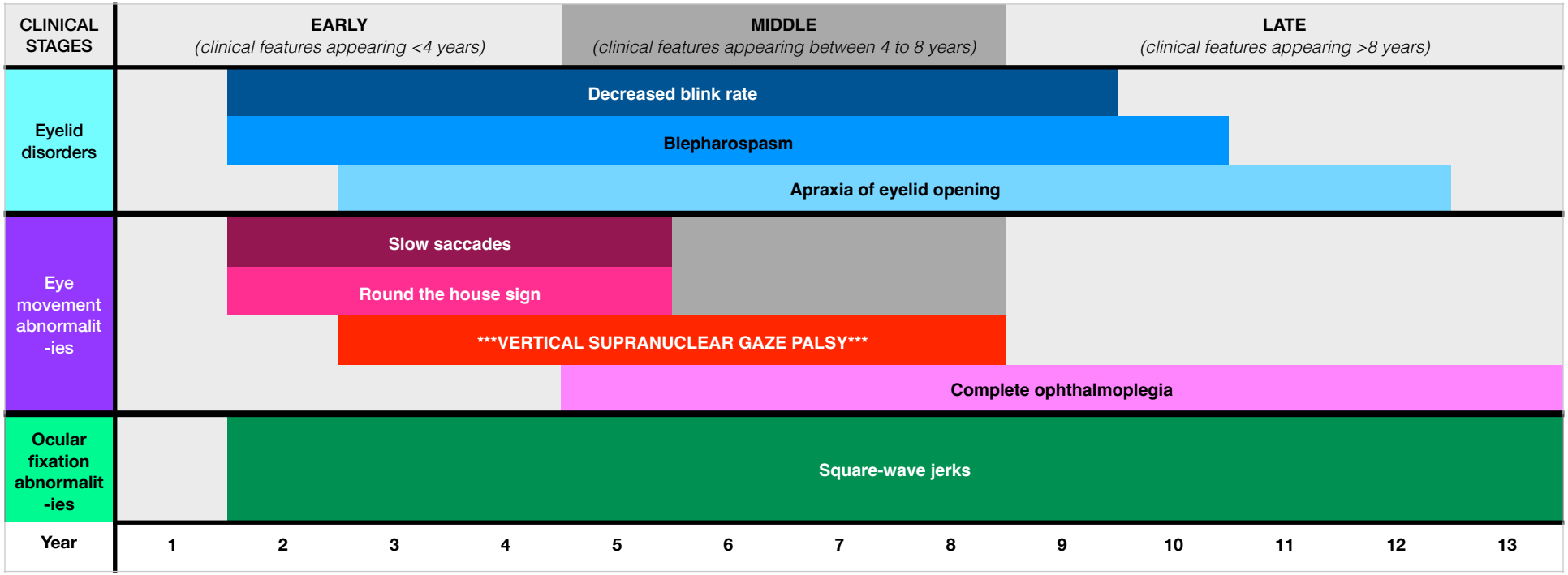
AD = Alzheimer's disease
CBD = corticobasal degeneration
DLB = dementia with Lewy bodies
MSA = multiple system atrophy
OKN = optokinetic nystagmus (ie the vertical lines strip maneuver)
PCA = posterior cortical atrophy
PDD = Parkinson disease dementia
PSP = progressive supranuclear palsy

CLINICAL PEARL
Jung I & Kim JS 2019
Abnormal eye movements may follow or precede the motor symptoms of movement disorders.

Characteristics of ocular motor abnormalities in PD-related disorders (adapted from Jung I & Kim JS 2019)

Disorder	Saccadic intrusions (square wave jerks)	Horizontal saccades	Vertical Saccades	Smooth pursuit (degrees of impairment)	Blepharospasm or eyelid apraxia
Parkinson Disease (PD)	+	Hypometric	Hypometric	Mild	Very rare
Progressive supranuclear palsy syndrome (PSP-S)	++	Slowed, late in disorder	Slowed, early in disorder	Severe	Common
Corticobasal syndrome (CBS)	+	Delayed	Impaired, late in disorder	Mild	Common
Multiple system atrophy (MSA)	+	Hypometric	Hypometric	Moderate	Rare

Evolution of Ocular Changes in PSP in Chronological Order (adapted from Phokaewvarangkul O & Bhidayasiri R. Translational Neurodegeneration



Motor Examination In Dementia

Includes examination for parkinsonism

General principles of the motor examination:

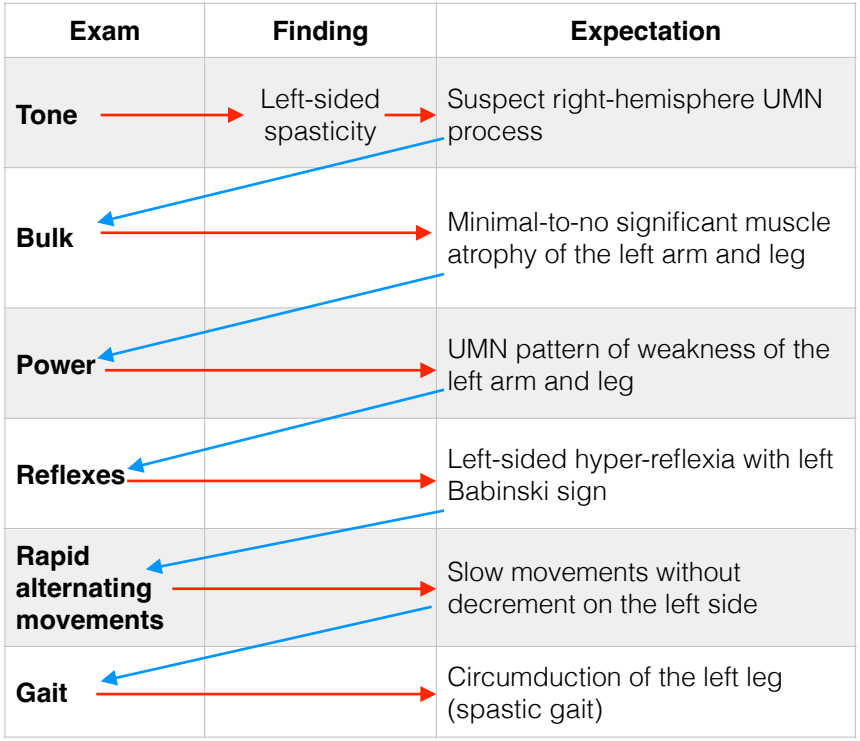
1. Always compare 1 side to another & never perform the maneuver simultaneously
2. Always test 1 system at a time (tone, then power, then reflexes, then sensory etc)
    a. For example, tone in the arms then legs - followed by power in the arms then the legs.
    b. Do not test tone in the arm then power in the arm, then tone/power in the other arm, then tone/power in the one leg followed by the other
3. Be systematic & consistent from patient to patient and within a patient - follow the same process and do not skip around
4. Anticipate findings based on the history - what to look for and what should not be present
5. Observe for anything that does not look like what you have seen before
6. It's OK NOT to know/label the finding, just be able to describe in detail what you observe

Importance of the order of the motor examination & how it informs/predicts the rest of the examination

Inspection →	Muscle Tone →	Power →	Reflexes
Muscle bulk - usually atrophy in dementia cases	Spasticity present - if so, indicates an upper motor neuron (UMN) lesion is present	Pattern of weakness important - does it follow a UMN or LMN or mixed pattern	Asymmetric reflexes are most helpful - just need to determine which side is the pathologic one
Fasciculations present - if so, indicates lower motor neuron (LMN) disease is present (e.g. ALS)	Rigidity - if so, indicates an extrapyramidal process (ie parkinsonism)		
Features of Parkinsonism - resting tremor, bradykinesia	Paratonia - not specific for any disorder		
	Hypotonia - rare to see in dementia unless acute stroke or cerebellar dysfunction		

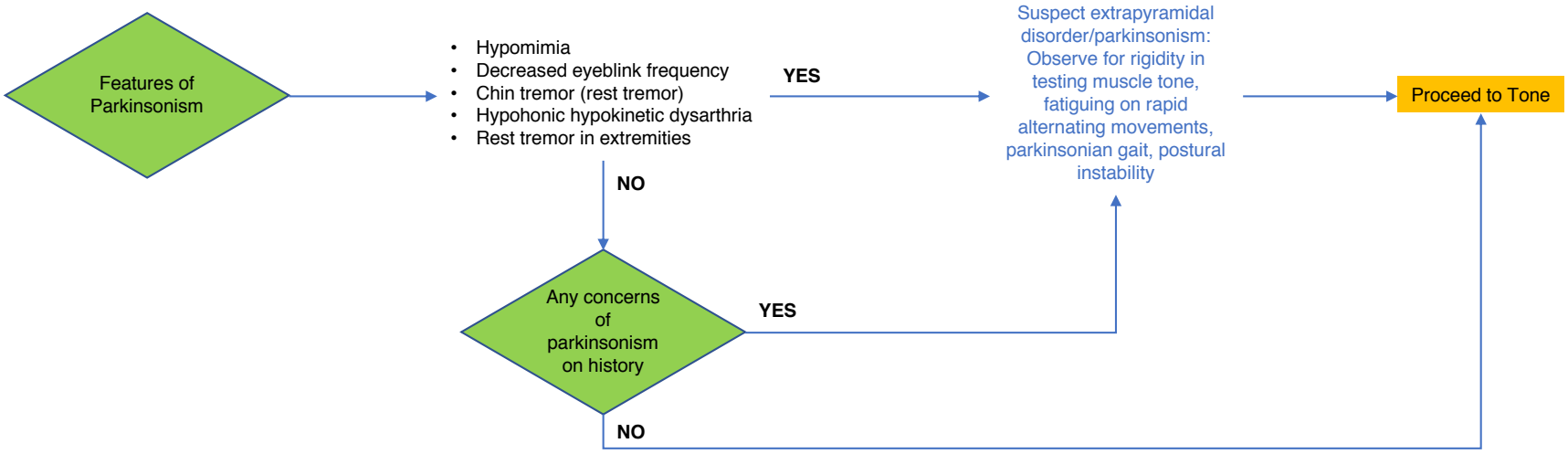
Illustrative example:

Red lines indicate clinical rationale
Blue lines indicate next step in the neurological exam



[1]**UMN pattern of weakness** = weakness of extensors in the arm & flexors in the leg

On inspection - can also observe for signs of parkinsonism (see page 3)

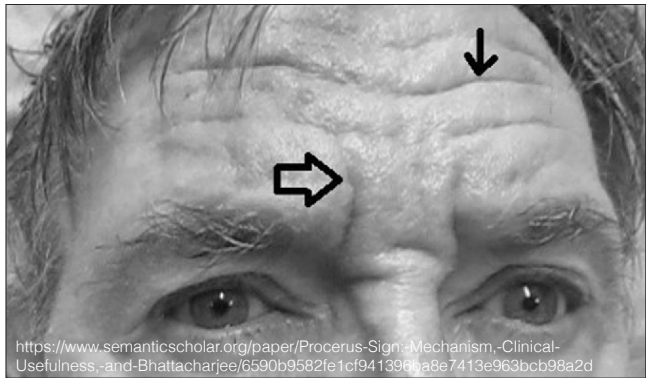


seborrheic dermatitis (possible)

- due to increased production of host sebum as well as the presence and increased reproduction of Malassezia yeasts

hypomimia (masked facies)

decreased eye-blink frequency



Procerus sign

- Vertical wrinkling on the forehead imparting a "staring," reptile-like, or "astonished" look characterized by lid retraction and infrequent blinking
- **Commonly seen in PSP**

Rest tremor in Parkinsonism

Parkinsonian Disorder	Rest Tremor
1. Parkinson's Disease (PD)	**YES** **Approximately 20% of iPD do not have rest tremor.*
2. Parkinson's Disease with Dementia (PDD)	**YES**
2. Dementia with Lewy bodies (DLB)	**NO**
3. Corticobasal degeneration (CBD)	**NO**
4. Progressive supranuclear palsy (PSP)	**NO** *****Few cases of rest tremor reported in PSP

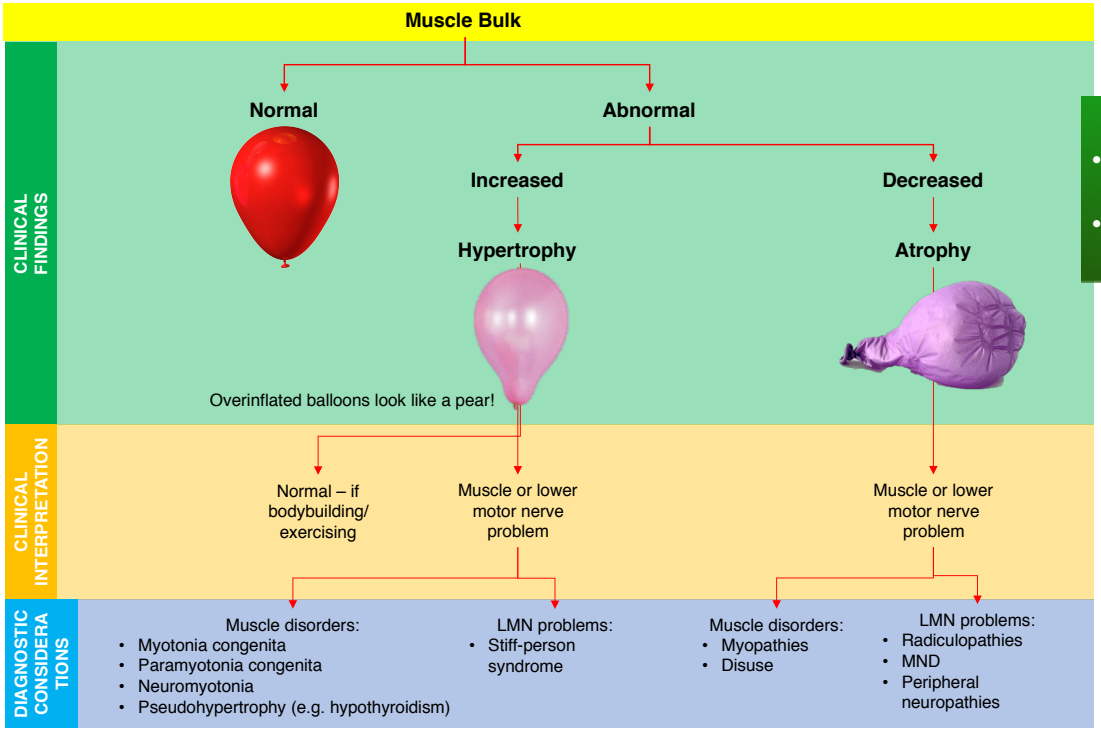
Why is this important?

- Most of the Parkinsonian disorders do NOT have rest tremor but all have rigidity

Examination Technique

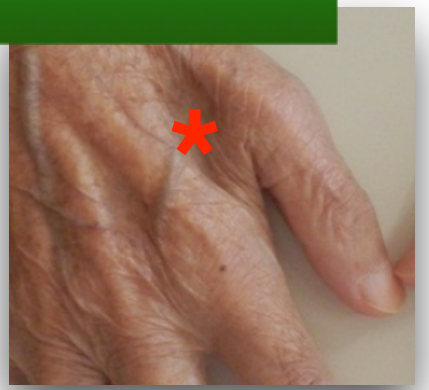
1. Observe for a rest tremor while also observing for muscle bulk and muscle atrophy in the following areas:
    a. Chin
    b. Fingers
    c. Hands and/or feet
    d. Limbs (arm and/or leg)
2. If suspect a rest tremor, ask the patient to perform serial 7s or state the months of the year backwards (cognitive distraction) and observe for rest tremor in the locations above

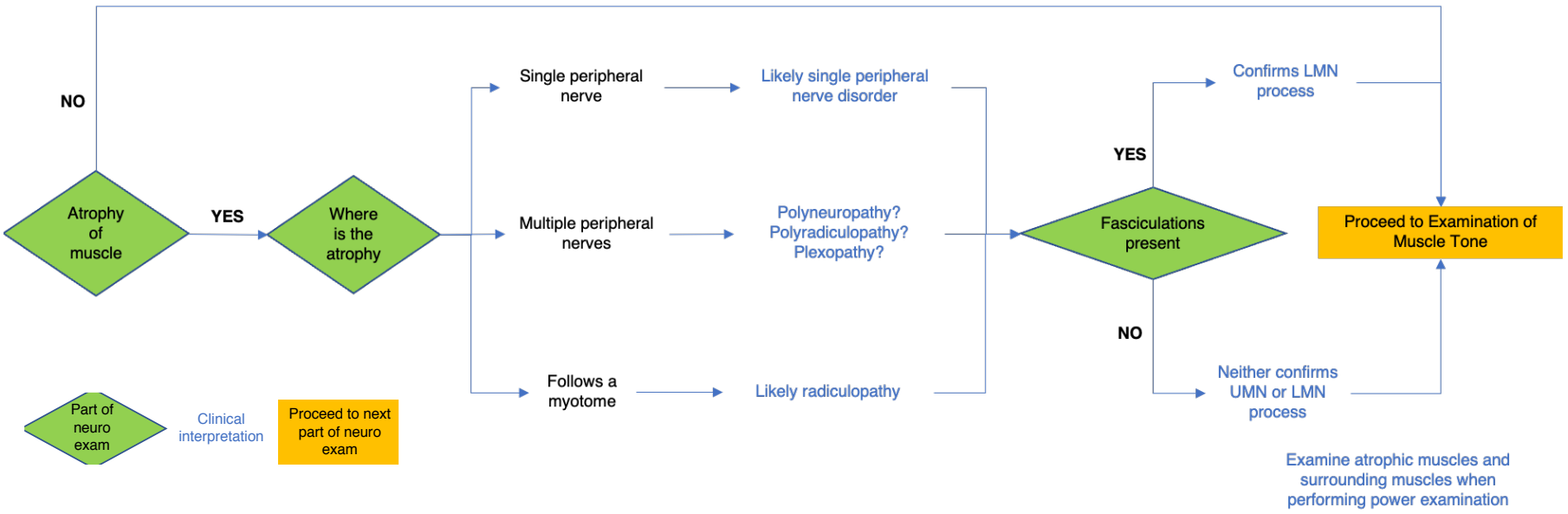
Muscle bulk - assessing for muscle atrophy



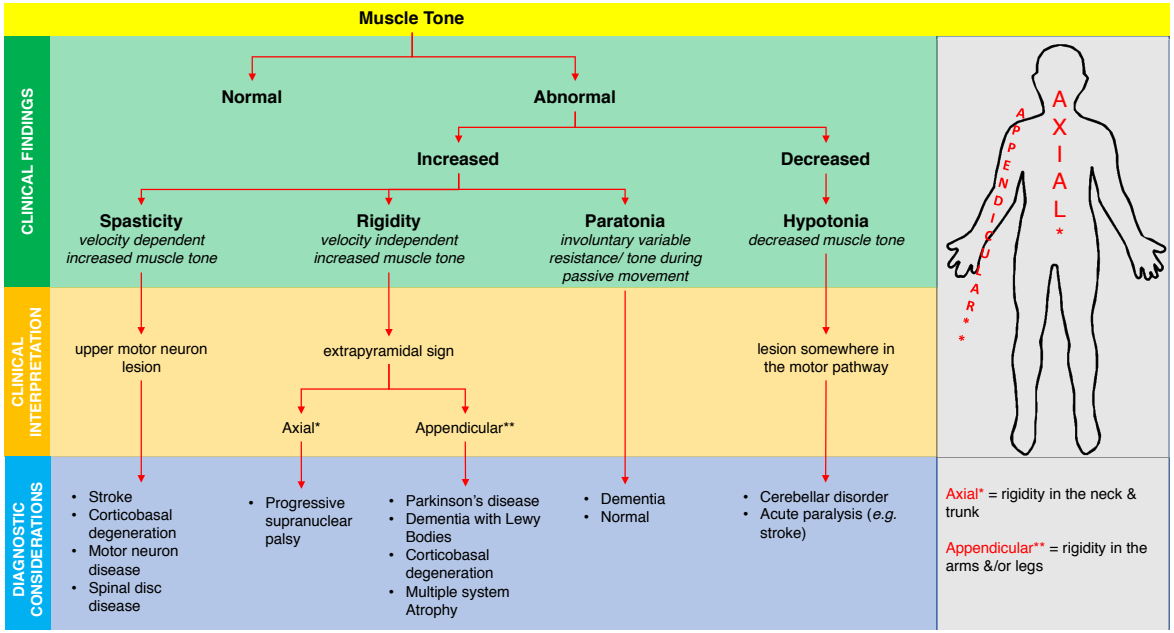
Clinical Pearl

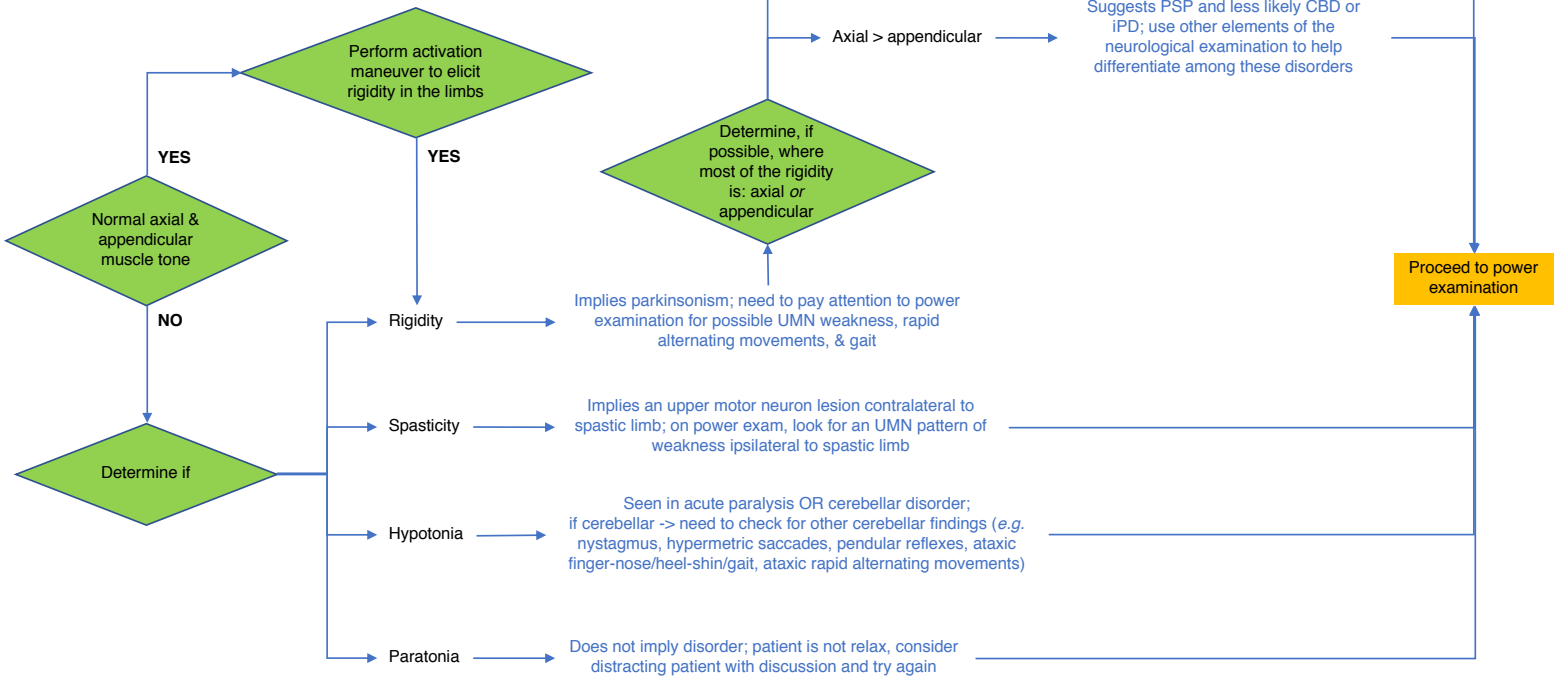
- **Normal** muscle bulk - muscle appears convex/ bulging
- **Atrophic*** muscle - muscle appears concave/ scalloped



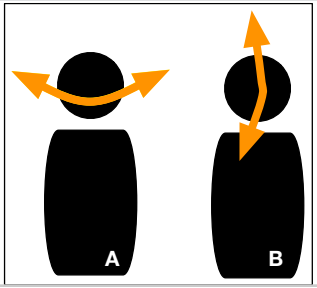
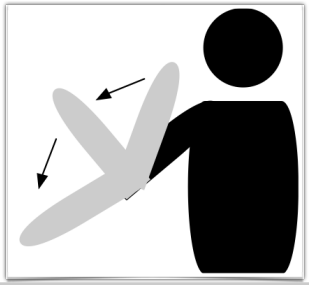
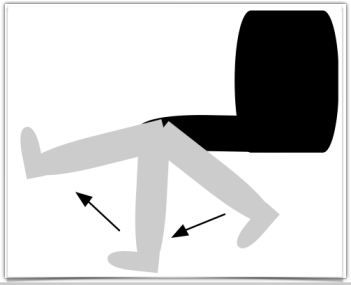


Muscle tone - in dementia, assessing for spasticity, rigidity, both spasticity and rigidity

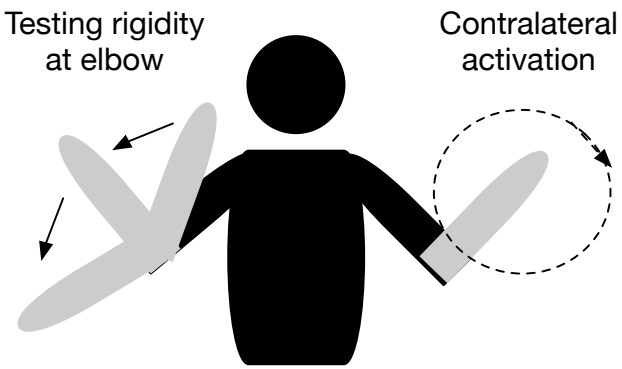




Examination of Muscle Tone

	Head	Arm	Leg
Figures			
General Principles	1. ***Slowly*** move the head so as NOT to cause harm as patients often have cervical disc disease, arthritis	1. Ensure to test over the entire range of movement of the joint (*i.e.* from full extension to full flexion) **[pitfall]** 2. First move the limb slowly to assess tone "at rest" - *i.e.*: is it normal, hypotonic, rigid, or paratonic 3. Then move the limb, as instructed in #1, as quickly as possible to determine if spasticity is present **[pitfall]** 4. Then test the opposite limb for comparison and the other limbs. **[pitfall]**	
Maneuvers	Test: • Side-to-side head movement (**A**) • Flexion-extension of head (**B**) **DO NOT TEST FOR SPASTICITY HERE**	Test: • Flexion-extension at elbow • Pronation-supination at forearm • Circular movement at wrist for cogwheeling rigidity	Test: • Flexion-extension at knee *(if sitting position)* • Quick lift of the leg proximal to knee joint *(if lying position)*
Clinical rationale	• PSP (progressive supranuclear palsy) often has axial more than appendicular rigidity.	• Rigidity implies extrapyramidal disorder/parkinsonism. Need to look for other signs of parkinsonism on examination. • Spasticity implies an upper motor neuron (UMN) lesion somewhere. Need to look for UMN pattern of weakness.	
PITFALLS	Often not assessed	· **Improper instructions** • With paratonia - imitation behaviour of the examiner (DO NOT SHAKE YOUR LIMBS!) · **Improper technique** • Trying to imitate the neurologist's "rotating" movements - end up rotating the limb at the elbow • Leg rolling is insufficient • Reinforcement not performed if suspect parkinsonism · **Relying on false signs** • "Looking" for cogwheeling and only at the wrist and not looking for "rigidity"	

What to do if you suspect Parkinsonism but your patient does not exhibit rigidity? ACTIVATE IT!

	Example of Activation (figure)
Activation procedures - having the patient perform a simultaneous motor task with the arm contralateral to the one being tested - increase rigidity in the tested arm in people with PD	
Types of contralateral movements: • Opening-closing a hand • Heel tapping • Circular movements of the arm	

CLINICAL PEARL

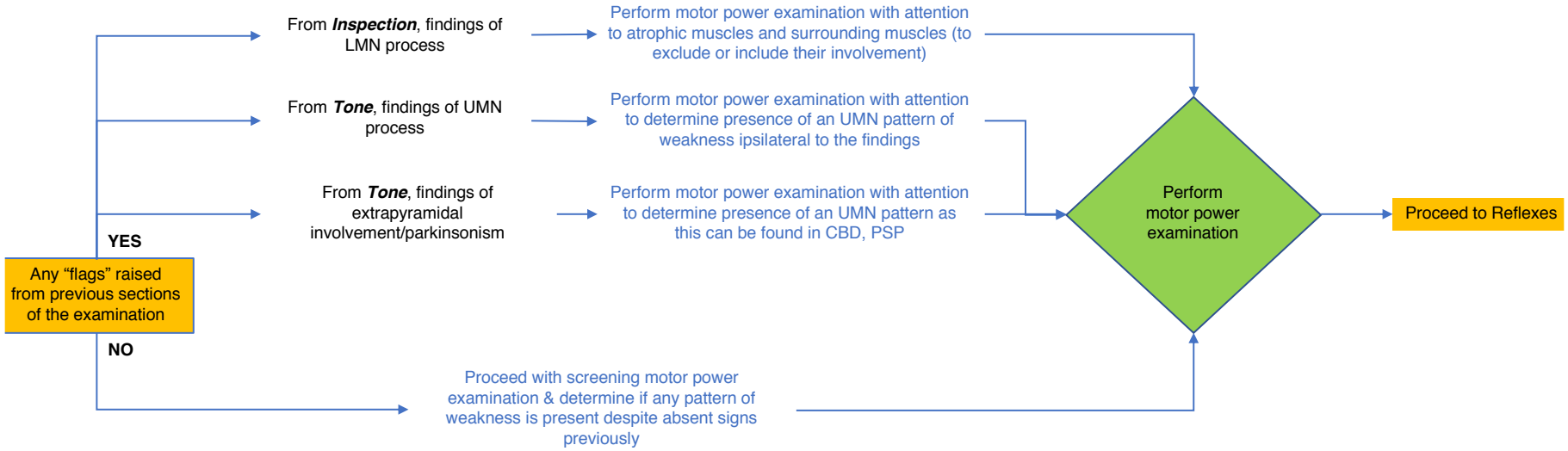
Rely on rigidity and not cogwheeling rigidity to determine parkinsonism & do NOT forget to examine axial rigidity

- Presence of cogwheeling is due to an underlying presence of rest tremor. Other than Parkinson's disease, the other parkinsonian disorders rarely - if ever - has rest tremor.
- All parkinsonian disorders however have a degree of rigidity.

Muscle tone findings among the dementias

<table>
<tr><th>AD</th><th>VCI</th><th>FTD</th><th>PSP</th><th>CBD</th><th>DLB</th><th>PDD</th></tr>
<tr><td colspan="3">Normal</td><td></td><td></td><td></td><td></td></tr>
<tr><td></td><td>Appendicular rigidity
If basal ganglia circuitry involved; consider vascular parkinsonism</td><td></td><td>Axial > appendicular rigidity</td><td colspan="3">Appendicular > axial rigidity
*In CBD, very asymmetric rigidity is usually present (compared to PDD and DLB)</td></tr>
<tr><td></td><td>Spastic
If corticospinal tract involved</td><td>Spastic
Suggests UMN involvement and probability of motor neuron disease</td><td colspan="2">Spastic (rare)
Suggests additional UMN involvement</td><td></td><td></td></tr>
</table>

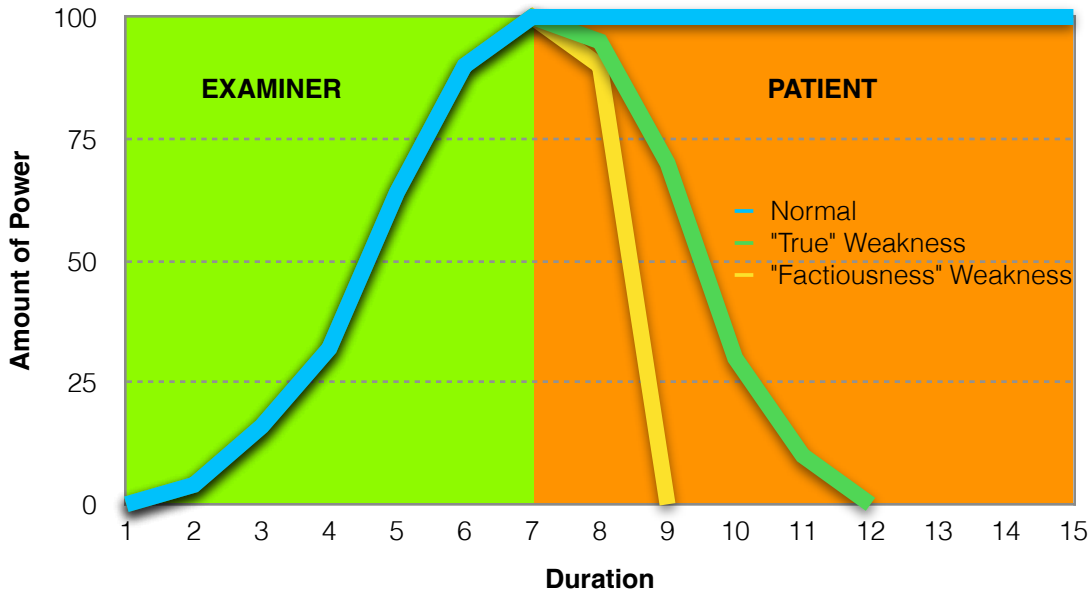
Power Exam - Determining strength and pattern of weakness



Technique:

1. There is no such thing as examining too *few* muscles - the more muscles tested, the more accurate you can be
2. Be systematic
    a. start in the arm and then followed by leg
    b. always compare one side and then the other - NEVER both at the same time
3. Properly position and support the patient's limb to maximize results (and not have interference from other muscle groups)
    a. isolating the joint/muscle
    b. most, if not all, muscles are tested at 90° angles
4. Gradually increase your power to your maximum (blue line on figure to the right) - or else will under call potential weakness
5. Grade the power (see next page)
6. Interpret the pattern of weakness (see next page)

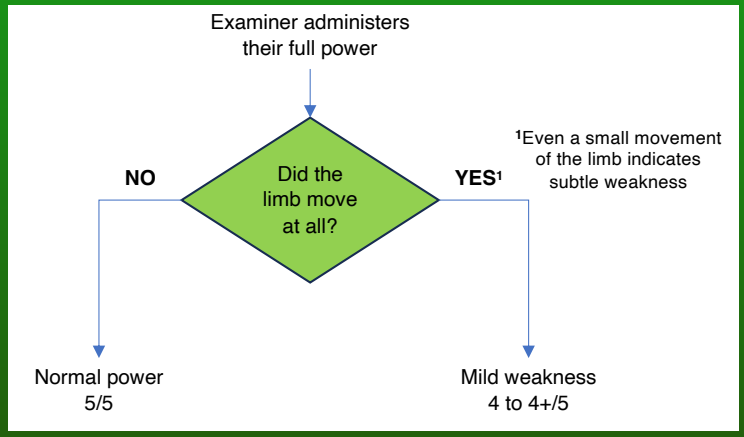
Examiner's "Feel" during Assessment of Power/Strength



Grading Muscle Power	
Medical Research Council	
Grading Scale	**Description**
0	No muscle activation
1	Trace muscle activation, such as a twitch, without achieving full range of motion
2	Muscle activation with gravity eliminated, achieving full range of motion
3	Muscle activation against gravity, full range of motion
4	Muscle activation against some resistance, full range of motion
5	Muscle activation against examiner's **full resistance**, full range of motion

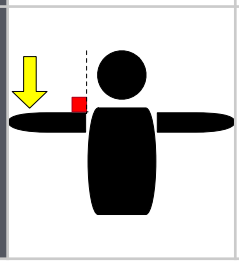
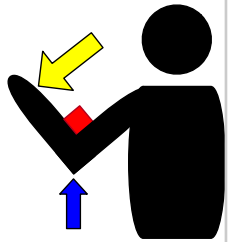
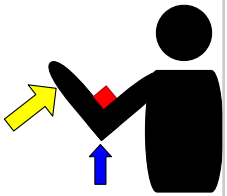
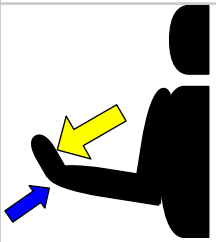
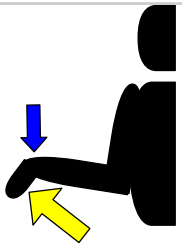
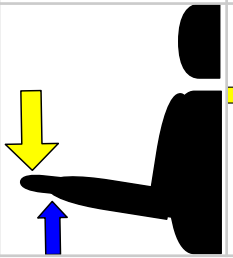
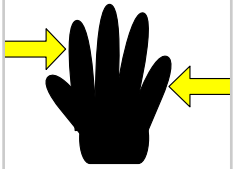
CLINICAL PEARL

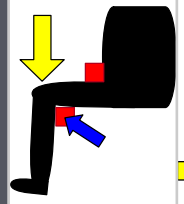
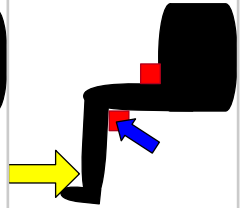
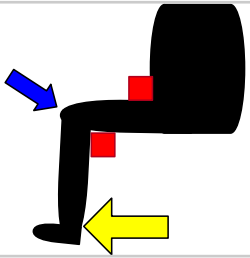
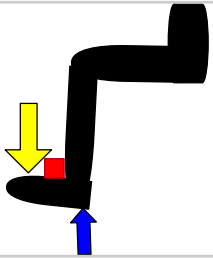
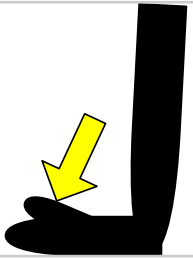
How to differentiate 5/5 from 4+/5 power and not to normalize power



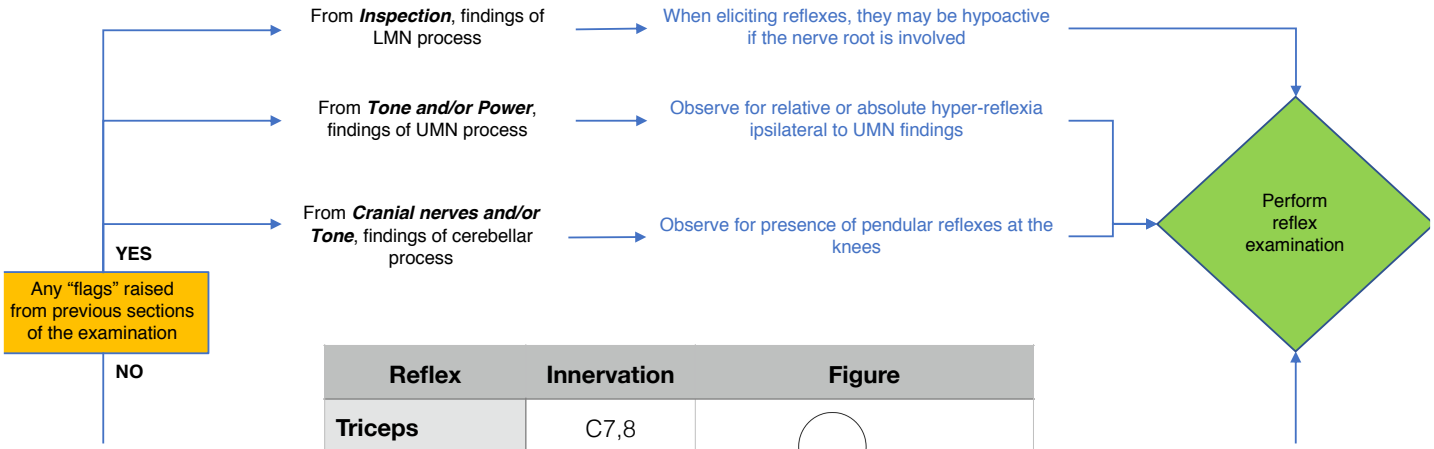
Positioning of limbs (patient) and hands (examiner) for the power examination
Technique: Arms (*Muscles affected in an upper motor neuron (UMN) lesion (pyramidal or UMN pattern of weakness)

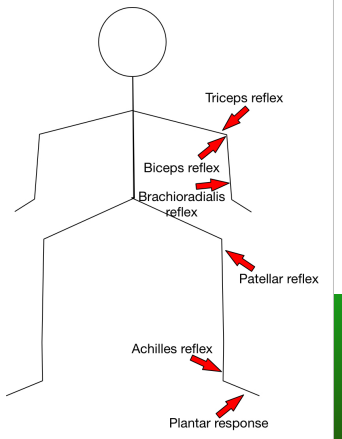
LEGEND			
Symbol			
Meaning	Direction of force	Where to stabilize joint	Indication of 90°

	*Deltoids**	**Biceps**	*Triceps**	*Wrist Extensors**	**Wrist Flexors**	*Digit Extensors**	**Interossei**
Muscle Action	Shoulder abduction	Arm/elbow flexion	Arm/elbow extension	Wrist extension	Wrist flexion	Finger extension	Finger abduction
Figure							

	*Iliopsoas**	**Quadraceps**	*Internal Hamstring**	*Anterior Tibialis*	**Gastrocnemius**	*Extensor Hallucis Longus**
Muscle Action	Hip flexion	Knee extension	Knee flexion	Foot extension	Foot flexion	Large toe extension
Figure						

Reflexes

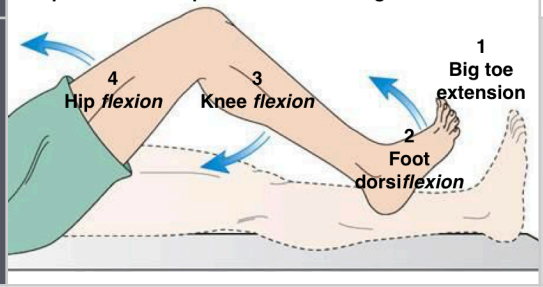


Reflex	Innervation	Figure
Triceps	C7,8	
Biceps	C5,6	
Brachioradialis	C5,6	
Patellar	L3,4	
Achilles	S1,2	

CLINICAL PEARLS

1. Observe for reflex asymmetry
2. Observe for ***relative*** hyper- or hypo-reflexia - not absolute hyper-/hyporeflexia

Distinguishing Triple Flexion Response versus Withdrawal

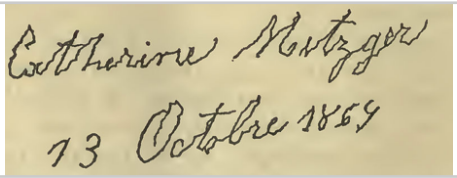
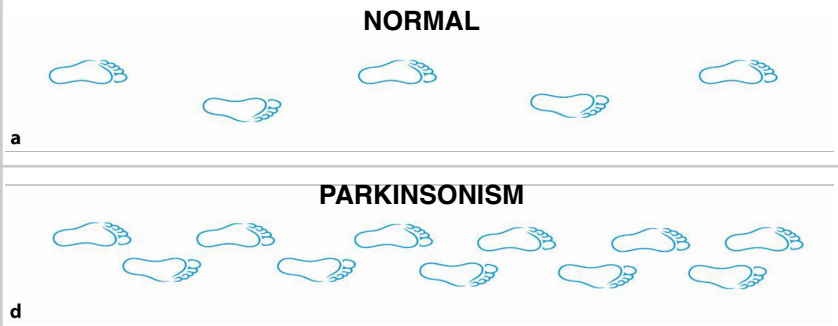
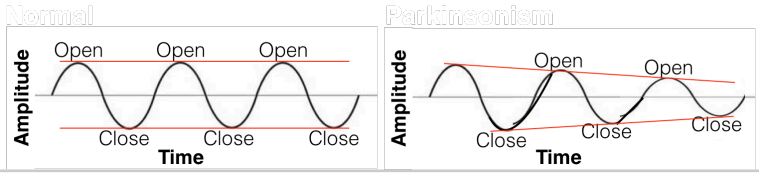
	Figure of Triple Flexion (True Babinski Sign)	Triple-Flexion Response *or* "True" Babinski Sign	Withdrawal
Description	**Triple Flexion Response = Babinski Sign = UMN Lesion**	They superficially look the same	
Differences		1. Extension of the big toe 2. Response lasts **LONGER** than the duration of stimulus 3. Occurs with the mildest of stimulus	1. **NO** extension of the big toe 2. Response lasts for the duration of the stimulus 3. Should **not** occur with the mildest of stimuli

Common Patterns of Weakness & Reflex Changes Seen in Some Neurodegenerative Dementias

	Muscle Bulk	Muscle Tone	Muscle Pattern of Weakness	Reflexes	Associated Dementias *(not a comprehensive list)*
Normal	Normal	Normal	None	Normal or symmetrical	• AD (Alzheimer disease) • VCI (vascular cognitive impairment)
Upper motor neuron (UMN)/ Pyramidal	Normal to minimal atrophy	Spastic	• **Arm:** extensors weaker than flexors • **Leg:** flexor weaker than extensors	Increased ± present Babinski sign	• VCI • FTD-MND (PLS) • PSP, CBD (rare) • Structural (*eg.* Myelopathy, tumour)
Lower motor neuron (LMN)	Marked atrophy ± fasciculations	Normal to hypotonia	Dependent on the affected nerve	Decreased (if nerve involved in reflex affected)	• Structural (e.g. disc causing a radiculopathy) • Diabetes (VCI) • SMA (spinal muscular atrophy)
Mixed UMN + LMN	Marked atrophy ± fasciculations	Spastic	Mix of UMN and LMN pattern of weakness	Increased or decreased (if muscle too weak) ± Babinski sign	• FTD-ALS

Key Point
Up to 15% of people diagnosed with FTD go on to develop MND. Therefore need to follow them over time.

Other Parkinsonism Signs to Observe During the Examination

Where Can Observe For Fatiguing	Description
1. Speech	Starts "normal" but volume becomes softer, loss of diction, then mumbling/incomprehensible
2. Writing	
3. Gait **Progressive decline in stride length in parkinsonism*	
4. Rapid alternating movements	

Why is this important?

- Commonly seen in parkinsonism
- In Parkinsonism, to maintain a rate there is a reduction the amplitude of movements (***fatiguing/motor decrement***).
- Eventually, the movement will slow down further and stop (freeze)

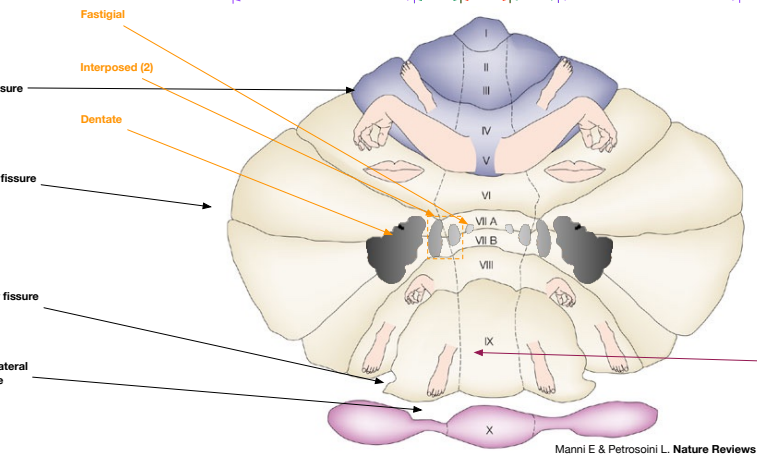
Examination Technique - Rapid Alternating Movements

1. Always perform 1 side then the other (if not, the "slower" side can entrain to the faster side and the fatiguing will be missed)
2. Always have the patient perform ***the movement as fast as possible WITH the largest amplitude/ opening*** (as will need to observe for fatiguing)
3. Can demonstrate the movement but DO NOT continue with the task while the patient is being tested - observe for about 10 repetitions
4. Where to test:
   a. Pronation-supination of hands
   b. Finger opening
   c. Hand opening
   d. Foot tapping
   e. Heel tapping

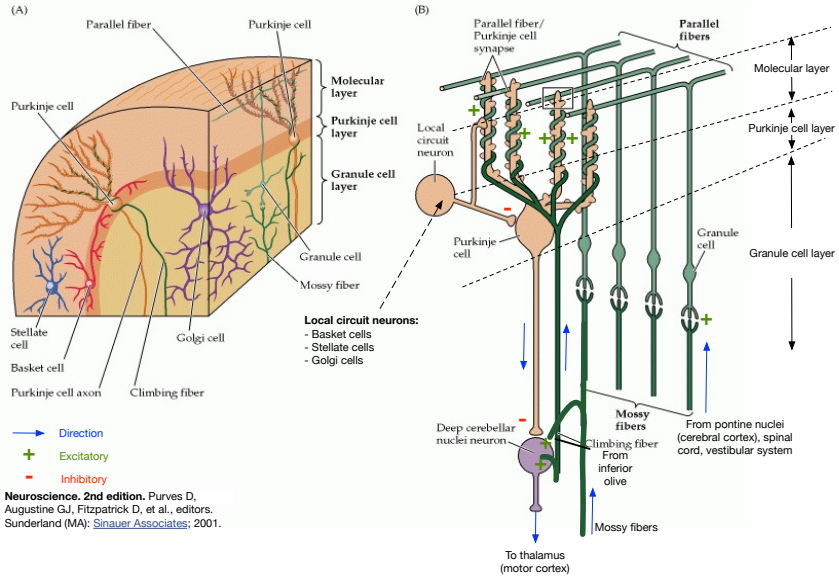
The Straight Forward Examination of the Cerebellum

BASIC NEUROANATOMY → **FUNCTIONAL NEUROANATOMY**

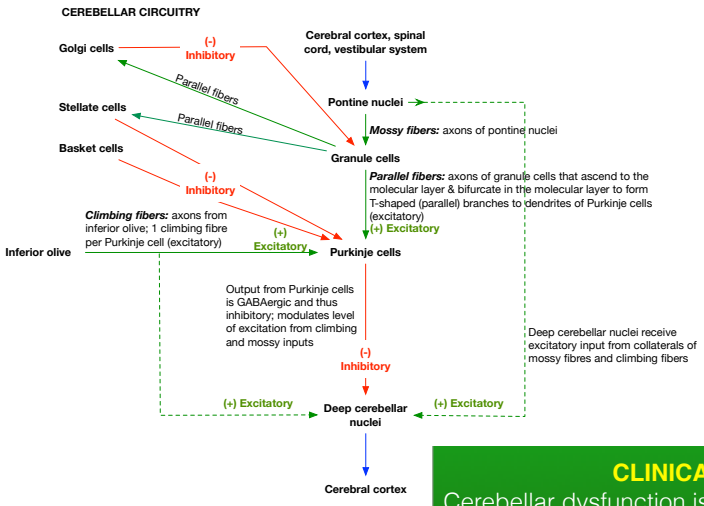
LOBES	FISSURES	DEEP NUCLEI	SECTIONS	FUNCTIONS	DISORDERS	DETAILS
Anterior lobe	Primary fissure	Fastigial, Interposed (2), Dentate	Cerebellar hemisphere (left) – Para-vermis – Vermis – Para-vermis – Cerebellar hemisphere (right)	**Postural stability (*with rest of vermis & paravermis*)**	**Truncal ataxia**	**ROSTRAL VERMIS SYNDROME** (anterior lobe) 1. Wide-based stance & titubating gait 2. Ataxia of gait, with proportionally little ataxia on heel-to-shin maneuver with patient lying down 3. Normal or slightly impaired arm coordination 4. Infrequent presence of hypotonia, nystagmus, and dysarthria
Posterior lobe	Horizontal fissure Secondary fissure			**Motor** Fine motor movements **Cognition** ***Left*** hemisphere - visuospatial function - executive function ***Right*** hemisphere - language function - executive function	Executive dysfunction, visuospatial disorganization, impaired visuospatial memory, mild anomia, agrammatism, dysprosody	**HEMISPHERIC SYNDROME** 1. Incoordination of ipsilateral appendicular movements, particular if they require fine motor coordination, esp. speech and finger movements
	Posterior lateral fissure			**Posterior Vermis** Affect & behaviour	Disinhibition/ inappropriate behaviour, apathy/blunted affect	**CAUDAL VERMIS SYNDROME** (flocculonodular lobe & posterior lobe) 1. Axial disequilibrium & staggering gait 2. Little or no limb ataxia 3. Sometimes spontaneous nystagmus & rotated postures of the head
Flocculanodular lobe				**Balance/equilibrium & eye-movements**	**Nystagmus**	



Manni E & Petrosoini L. **Nature Reviews Neuroscience**. 2004; **5**: 241–249.



Neuroscience. 2nd edition. Purves D, Augustine GJ, Fitzpatrick D, et al., editors. Sunderland (MA): Sinauer Associates; 2001.

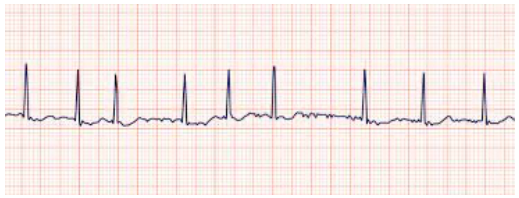
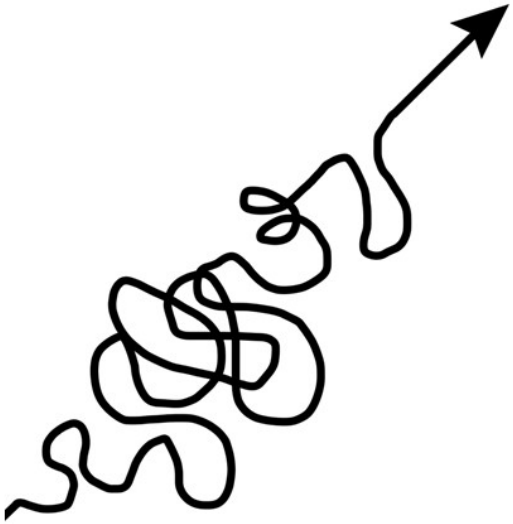


CLINICAL PEARLS

Cerebellar dysfunction is not often seen in neurodegenerative dementias with the exception of:

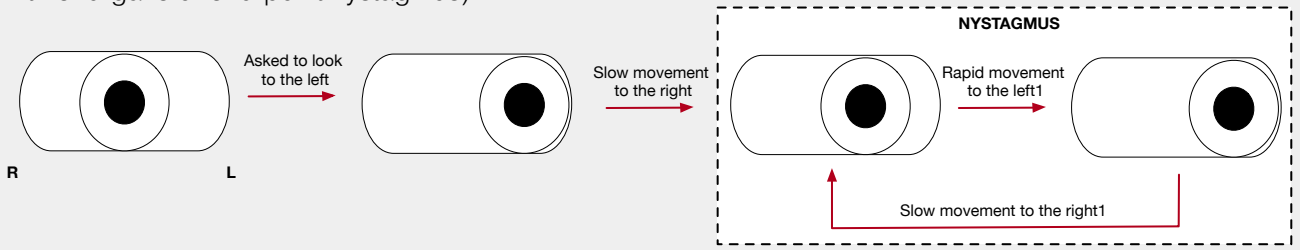
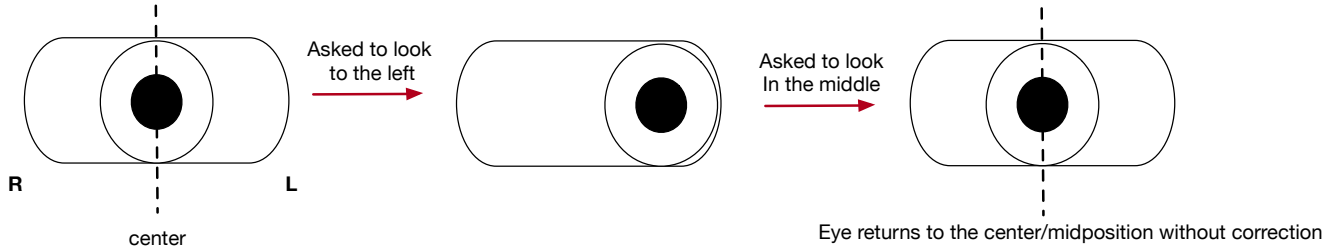
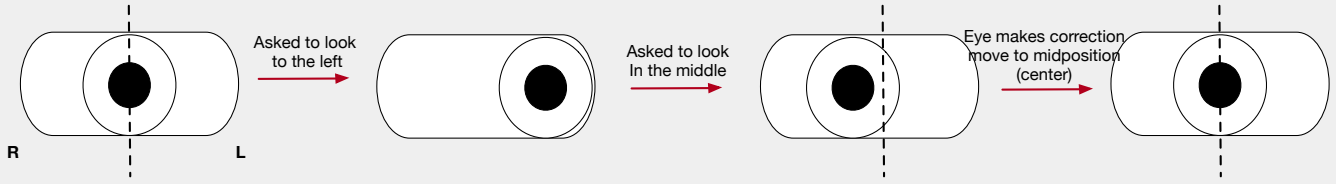
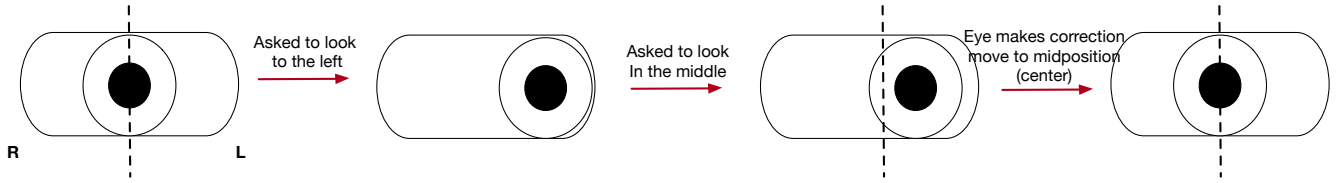
- VCI - if a stroke affected the cerebellum or cerebellar pathways
- PSP - if cerebellar presentation of PSP
- CJD - if cerebellar presentation
- MSA-C - but this is not a dementia

The 3Ts of Cerebellar Function

	***T*iming**	***T*rajectory**	***T*arget**
Normal Function	Involved in the regular/specific timing of movements	Involved in a straight/linear trajectory	Involved in precise targeting when reaching for an item
What to observe when cerebellar dysfunction is present	Observe for the ***atrial fibrillation*** (irregularly irregular) pattern	Observe for a non-linear (not a straight line) path between 2 targets	Observe for missing the target (overshoot or undershoot) and require a correction to reach it
			
	Rapid alternating movements will have irregularly irregular spaced timing, force and amplitudes		Appreciated with saccades, finger-nose, and heel-shin testing

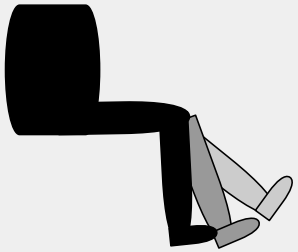
Head-to-Toe Approach of All of the Cerebellar Functions

Level of Nervous System	Specific Area	Signs & Symptoms of Dysfunction	Timing	Trajectory	Target
BRAIN	Cognition (Cerebellar cognitive affective syndrome)	Frontal cognitive & behavioural impairment	Slow, apathy	• Disinhibited to apathy • Dysexecutive	N/A
	Eyes	Impaired saccades & development of nystagmus	N/A	N/A	• Hypermetric (overshoot) • Hypometric (undershoot)
	Speech	"Ataxic," "Explosive" or "scanning" dysarthria	• Decreased motor coordination for accurate articulation • imprecise consonant production • distorted vowel production • prolonged phonemes. • Slow rate	Excessive loudness variation	Equal and excessive stress is placed on all syllables
MOTOR SYSTEM	Motor Tone	Hypotonia	N/A	N/A	N/A
	Reflexes	Pendular reflexes (only seen at the triceps jerk & knee jerk)	N/A	N/A	Due to hypotonia where the limbs swings back & forth several times before stopping (like a pendulum), instead of the normal 1-3 swings
COORDINATION & GAIT	Coordination	Clumsiness, difficulty with reaching	N/A	Ataxic	• Pass-pointing or under-pointing • Terminal tremor
	Stance & Gait	Broad-based ataxic	Irregularly irregular	Ataxic	Wherever

BRAIN	Technique/Finding
Cognition & affect	Perform a history, mental status examination, and cognitive assessment
Eyes - nystagmus	Examine the extraocular movements for nystagmus towards the end of the eye movement (so as not to confuse with end-gaze or end-point nystagmus)  1 Speed in any direction but nystagmus usually has a fast and slow phase 2 Nystagmus can be vertical, horizontal, torsional (twist), variable, pendular, seesaw
Eyes - saccades (normal)	 Eye returns to the center/midposition without correction
Eyes - saccades (hypermetric)	 Eye overshoots the center and corrects = ***hyper***metric saccade
Eyes - saccades (hypometric)	 Eye undershoots the center and corrects = ***hypo***metric saccade
Speech	Listen for an irregularly irregular speech with variable/explosive articulation &volume of a word/phoneme and variable timing of words within a sentence. Think bad Shakespeare.

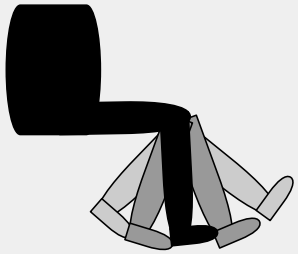
MOTOR SYSTEM	Technique/Finding
Bulk	Normal muscle bulk in pure cerebellar disorders
Tone	Hypotonia in pure cerebellar disorders (like overcooked spaghetti)
Power	Normal power in pure cerebellar disorders
Reflexes	Observe for pendular reflexes at either the patellar or triceps tendon

Normal
Knee Jerk/Patella Reflex

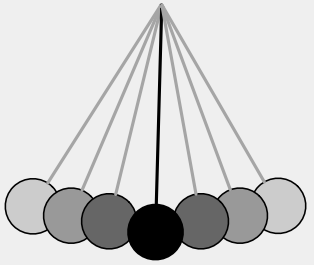


Initial swing
1 Return to primary position

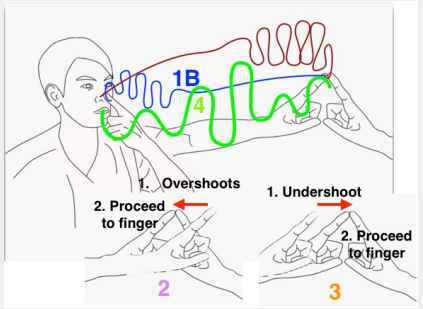
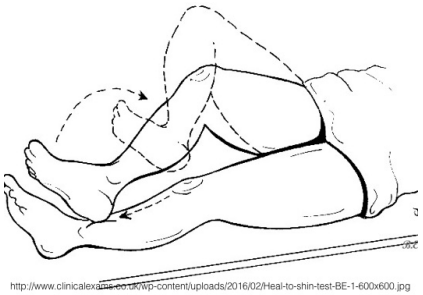
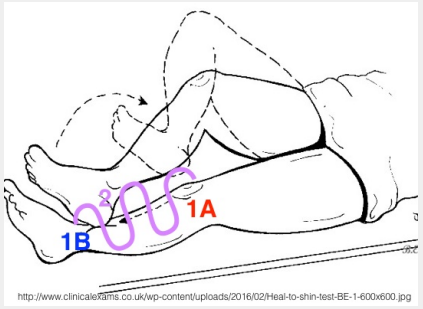
Pendular
Knee Jerk/Patella Reflex



Pendulum



Initial swing
1 Return & passes primary position
2
3 Continues to swing
4

COORDINATION	Technique/Finding	
Finger-nose (normal)	1. Need to stand in front of the patient (**1**) to observe easier ataxia, terminal tremor 2. Person's arm MUST be fully outstretched (**2**) 3. Need NOT ask the person to go as quickly as possible (it does not make a difference) 4. Need NOT move your (examiner's) finger as it does not make a difference - if true cerebellar lesion, it would be abnormal with any examiner's position	
Finger-nose (impaired)	**Target** 1. Terminal tremor at the examiner's finger (**1A**) and/or nose (**1B**) 2. Past pointing (overshoot) & correction (**2**) 3. Undershoot & correct (**3**) **Trajectory** 1. Ataxic trajectory (**4**) ***Pitfalls*** *Other causes of tremor (e.g. essential tremor) may also give the appearance of a "terminal" tremor and/or give the appearance of an impaired finger-nose task. To determine if the finger-nose difficulty is due to a cerebellar process, there should/ought be other cerebellar findings on examination.*	
Heel-shin (normal)	Technique 1. Need to observe ALL of the movements, especially the initial placement of the heel onto the knee 2. Person cannot use their arms/hand to position the leg appropriately 3. Observe a few trials	
Heel-shin (abnormal)	**Target** 1. Inability to place the heel correctly on either the knee (**1A**) or ankle (**1B**) (ie - looks "wavy," similar to the terminal tremor in finger-nose) **Trajectory** 1. Not a straight line along the shin - ie an ataxic trajectory (**2**) ***Pitfalls*** 1. *Proximal leg weakness (hip flexors) can imitate "ataxia" not due to a cerebellar process but due to inability to hold the limb against gravity* 2. *Trying to make the person perform the maneuver correctly. Often this is a finding!*	

Cerebellar Tremor vs Mimickers

Features	Cerebellar Tremor	Essential Tremor	Parkinsonian Tremor
Tremor Description	• Usually kinetic tremor (ie present with any voluntary movement) • Tremor worsens when reaches the target (terminal tremor) • Postural tremor may be present • Rare rest tremor present (aka Holmes' tremor) • Oscillations are of variable amplitude and perpendicular to the direction of the movement	• Increases/present with posture holding or action	• At rest, increases with walking • Decreases with posture holiding or action
Frequency	<5 Hz	5-12 Hz	3-6 Hz
Distribution	Variable	Symmetrical (mostly)	Asymmetrical
Body parts	Usually head and upper half of body	Head Voice Hands	Hands Legs
Writing	Tremulous	Tremulous	Micrographia
Course	Stable or progressive	Stable or progressive	Progressive
Family History	Less common (e.g. spinocerebellar ataxia)	Often	Less common (1%)
Other Neurological Signs	• Dysmetria • Dyssnergia (disturbance of muscular coordination) • Hypotonia • Pendular reflexes	None	• (TRAP) • Rigidity • Akinesia/Bradykinesia • Postural instability
Substances that Improve Tremor	None	• Alcohol • Propranolol • Primidone	• Levodopa • Anticholinergics
Surgical Treatment	Thalamic VIM DBS	Thalamic VIM DBS Thalamotomy	STN or GPi DBS

RAPID ALTERNATING MOVEMENTS

Rapid alternating movements - cerebellar dysfunction

RAPID ALTERNATING MOVEMENTS	Technique/Finding	
Hand tapping & Finger tapping	**Technique** Person must tap their hand (hand tapping) or index finger to thumb (finger tapping) as quickly AND as big (amplitude) as possible **Observe for** 1. Variability in timing from tap-to-tap (ie: atrial fibrillation for cerebellar dysfunction) 2. Variability in amplitude (trajectory) from tap-to-tap 3. Variability in target (not tapping at the same place) from tap-to-tap **Pitfalls** 1. Slow tapping without change in amplitude is likely due to weakness, either from an upper motor neuron lesion or lower motor neuron. 2. Tapping that has a progressive reduction in amplitude accompanied by slowing of speed is likely due to an extrapyramidal cause, such as Parkinson's disease. 3. To determine if abnormal finger or hand tapping is due to a cerebellar process, there should be variability in the amplitude and timing, as well as the presence of other cerebellar findings.	

Rapid alternating movements in Parkinsonism - UPDRS Instructions and Grading

Instructions

1. Always perform 1 side then the other (if not, the "slower" side can entrain to the faster side and the fatiguing will be missed)
2. Always have the patient perform the movement as fast as possible WITH the largest amplitude/opening (as will need to observe for fatiguing/ decrement)
3. Can demonstrate the movement but DO NOT continue with the task while the patient is being tested - observe for at least 10 repetitions
4. Where to test:
   a. Pronation-supination of hands
   b. Finger opening
   c. Hand opening
   d. Foot tapping
   e. Heel tapping
5. Rate the speed, amplitude, hesitations, halts & decrementing amplitude

	UPDRS Grading Scale				
Any of the following *(what to observe/ measure)*	**0 Normal**	**1 Slight**	**2 Mild**	**3 Moderate**	**4 Severe**
Rhythm	No problems	the regular rhythm is broken with 1 to 2 interruptions or hesitations of the movement	3 to 5 interruptions during the movements	more than 5 interruptions during the movement or at least one longer arrest (freeze) in ongoing movement	Cannot or can only barely perform the task because of slowing, interruptions or decrements.
Speed/slowing		slight slowing	mild slowing	moderate slowing	
Amplitude		the amplitude decrements near the **end** of the sequence.	amplitude decrements **midway** in the sequence	amplitude decrements starting after the 1st supination-pronation sequence	

Rapid Alternating Movements Findings due to Other Disorders

	Normal	Parkinsonism	Weakness	Cerebellar Dysfunction
Amplitude	Maintains large amplitude of movements without reduction	Progressive reduction/ decrement	Reduced but without reduction	Variable, erratic*
Rate	"Fast" & steady rate	Progressive slowing	Slow but steady rate	Variable, erratic*
Interruptions/Breakdown of movements	None	Can be present	None	None

Standing and Gait Examination - It's Not Easy

Examination of Standing (Schenkman M et al. Whole-body movements during rising to standing from sitting. Physical Therapy. 1990; 70(10): 638-648)

NORMAL SEQUENCE TO STAND FROM SEATED POSITION

START
Seated position

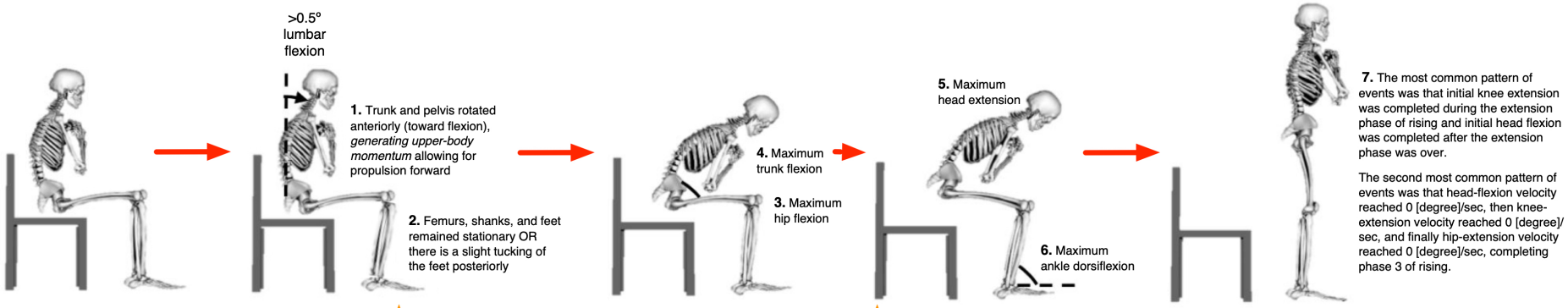
PHASE 1
Flexion-momentum phase
begins with initiation of the movement and ends just before the buttocks were lifted from the seat of the chair (lift-off)

PHASE 2
Momentum transfer phase
begins as the buttocks are lifted from the seat of the chair and ends when maximum ankle dorsiflexion is achieved

Momentum transfer occurrs when the momentum of the upper body developed in the flexion-momentum phase is transferred to the total body and contributes to total-body upward and anterior movement. During phase 2, the centre of mass (CoM) traveled anteriorly and upward. The whole-body CoM reached its maximal anterior point shortly after maximum dorsiflexion occurred.

PHASE 3
Extension phase
initiated just after maximal ankle dorsiflexion and is completed when the hip first ceases to extend

Usually, when the hip ceases to extend, it begins small rotations between flexion and extension as stabilization is achieved. There is a prolonged period of deceleration as the hip reaches the end of extension.



CLINICAL PROBLEMS LEADING TO INABILITY TO STAND CORRECTLY

Loss of ability to generate initial momentum for phase I? If the patient pulls his or her body forward using the arms during phase I, is he or she unable to generate adequate momentum with the trunk and hip flexor musculature, or is the patient attempting to increase the upper-body momentum above what would normally be used in order to compensate for lower extremity dysfunction?

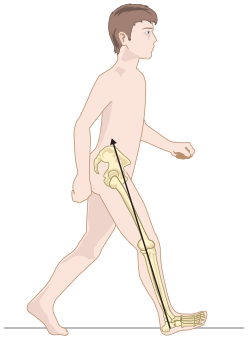
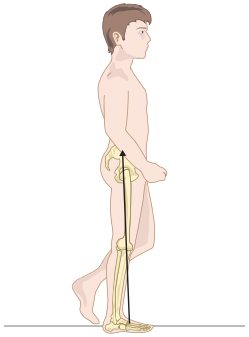
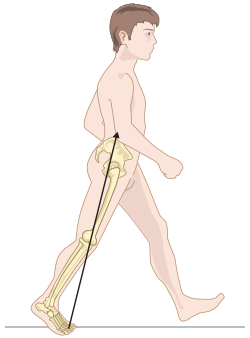
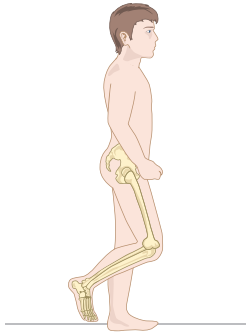
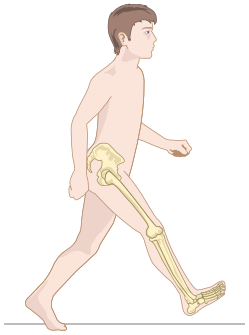
The patient must have adequate strength and coordination to generate sufficient upper-body velocity, and hence momentum, prior to lift-off from the chair seat. He or she must be able to use eccentric contractions to control trunk and hip musculature in order to slow the body's forward progression once lift-off occurs. Otherwise, the patient may fall forward during the momentum-transfer phase, which is one of dynamic stability. Finally, lower extremity joint integrity and strength must be adequate for the extension component of rising, which requires good concentric muscle control.

If a patient does not use a momentum-transfer strategy, is it because he or she has inadequate eccentric control of trunk and hip extensor musculature for dynamic stability in phase II? Are there other balance impairments that preclude the patient from remaining in a dynamically stable situation for phase II? These are only a few examples of the types of questions clinicians might ask in attempting to interpret the strategies patients use as they rise to a standing position.

	Standing	Stability After Standing
What to observe	Does the person push themselves to standing position with their arms **OR** rocks themselves to standing position?	Does the person demonstrate retropulsion (takes step back into chair **OR** falls back into chair upon standing) **OR** falls forward ?
What to consider	Suggests Parkinsonism **OR** weakness in proximal leg muscles **OR** vascular disease **OR** NPH (not exhaustive list)	Suggests postural instability (has multiple causes, including PSP, vascular disease, NPH)

Examination of Gait

Mechanics of Gait (Nonnekes J et al. JAMA Neurol. 2018;75(6):751-758)

Phase 1 Loading response	Phase 2 Midstance	Phase 3 Terminal stance	Phase 4 Early swing	Phase 5 Terminal swing
 Loading response	 Midstance	 Terminal stance	 Early swing	 Terminal swing
~60% of gait cycle			~40% of gait cycle	
• Starts when the foot strikes the floor (initial contact) and body weight is being transferred to the stance leg • In a normal gait pattern, the heel strikes the floor first with the ankle in a neutral position. • At heel strike, the knee is extended, but during the loading response, it flexes for shock absorption while the ankle shows plantar flexion.	• Starts when the stance leg has full foot contact and the contralateral leg has started the swing phase. • The body progresses over the stance leg in a coordinated fashion such that optimal stability and energy conservation are preserved. • Dorsiflexion occurs at the ankle while the knee and hip extend.	• Starts when the ankle undergoes plantar flexion again and the heel rises from the floor while the knee and hip are still extended. • The foot unwinds over the heads of the metatarsal bones, and concentric contraction of the calf musculature provides a push-off that generates most of the energy during gait. • The last part of terminal stance is characterized by knee and hip flexion while body weight is gradually transferred to the contralateral side.	• Starts when the foot is lifted from the floor as a result of a coordinated hip and knee flexion and ankle dorsiflexion. • Soon thereafter, the knee starts to extend again while the ankle maintains in a neutral position.	• Starts when the swing leg has passed the contralateral stance leg. • The hip reaches maximal flexion and the knee full extension to subserve optimal step length and adequate foot positioning for the next loading response.

Gait Parameters to Assess (Beristain X, Chapter 8: Approach to the patient with gait disturbance and recurrent falls in Practical Neurology, J Biller (editor), Wolters Kluwer; Philadelphia, 2017)

Stance phase	When the foot is on the floor
Swing phase	When the foot is in the air
Stance time	The time the foot is on the floor
Swing time	The time the foot is in the air
Cadence	Number of steps per minute
Step length	Distance advanced by one foot compared to the position of the other
Stride length	The sum of 2 consecutive step lengths or the distance advanced by 1 foot compared to its previous position
Step time	Time between heel strike of 1 foot to heel strike of the other foot
Gait cycle	The time between 2 consecutive heel strikes of the same foot
Stride time	Time for a full gait cycle
Average gait velocity	Stride length divided by stride time

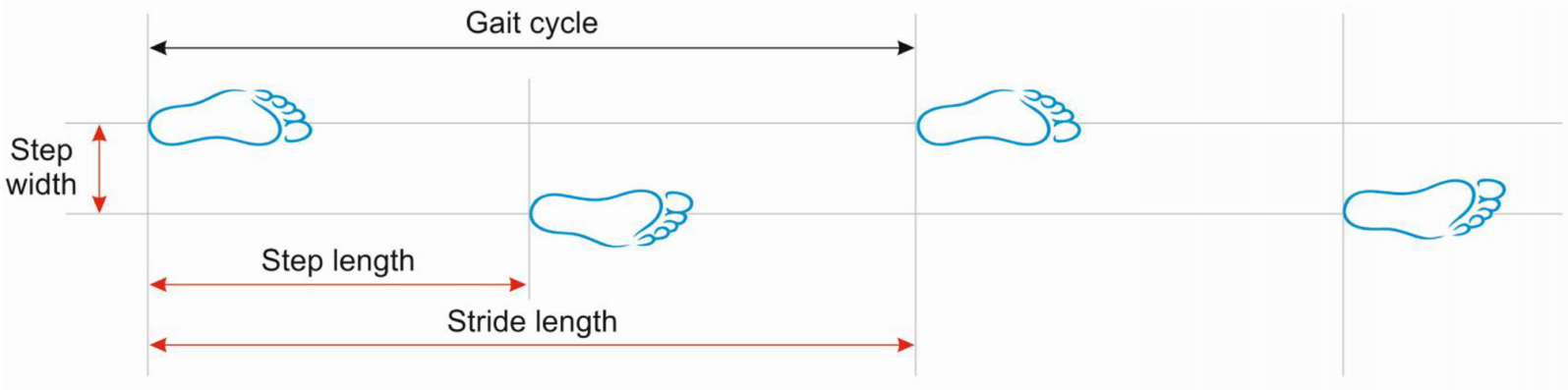


Figure from Pirker W & Katzenschlager R Gait disorders in adults and the elderly: a clinical guide. Wien Klin Wochenschr. 2017; 129(3): 81–95.

My Quick Bedside Evaluation of Gait

Quick Assessment of Gait - 9 things to observe

1 Erect posture

2 *Present & symmetrical* arm movements

3 Stable & levelled iliac crest while walking

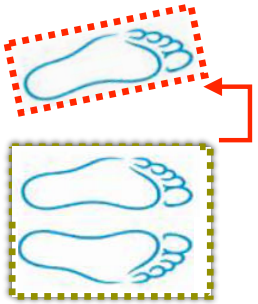
4 **Narrow-based gait** - *defined as 0-0.5 inches apart between ankles as they pass each other during walking*

5 **Size of shoes**

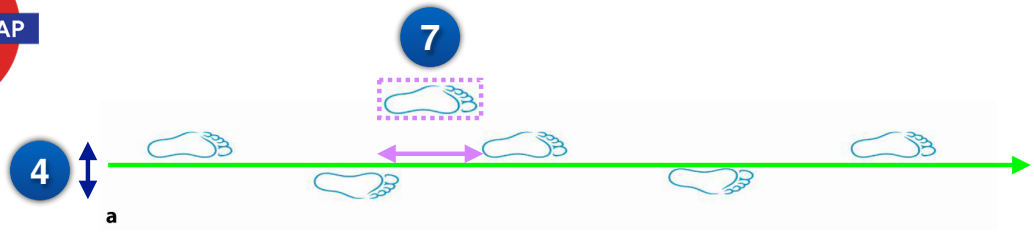
6 **Alignment of feet**

External rotation of left (eg.) foot

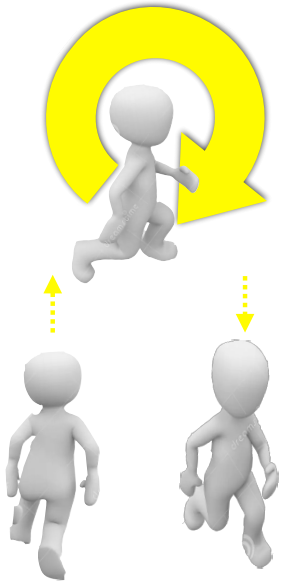
Normal (straight) alignment of foot



7 **Normal stride length** - *approximately defined as the distance of the patient's own foot between the heel of the advancing leg and the toe of the back leg*



9 **Quick turnaround** - *spin using 1-2 steps without retropulsion, hesitation, side-stepping*



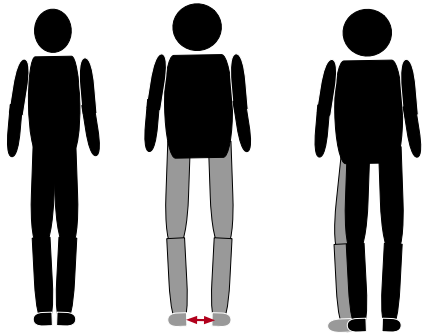
Classification of Abnormal Gait Patterns

Level of Gait Disorder	Causes at the Anatomical Level	Balance & Gait Pattern
Higher	**Subcortical**	Cautious, parkinsonian, ataxic, spastic, "magnetic," gait ignition failure, disequilibrium
	Cortex	
Middle	**Spinal cord**	Spastic
	Brainstem	Ataxia, spasticity
	Cerebellum	Cerebellar ataxia
	Thalamus	Astasia/ataxia
	Basal ganglia	Parkinsonian, dystonic, choreic
Lower	**Muscle**	Waddling, steppage, Trendelenberg
	Neuromuscular junction	Waddling
	Peripheral nerve Proprioception, vestibular, vision	Sensory ataxia/ vestibular disequilibrium/ visual disequilibrium
	Skeleton	Antalgic, compensatory for deformities

Not All Wide-Based Gait Ataxia is Due to Cerebellar Disease (Thompson PD & Nutt JG. Chapter 24: Gait Disorders in Bradley's Neurology in Clinical Practice 7th Edition, Elsevier: London, 2016)-1

Feature	Stance	Trunk Posture	Postural Reflexes	Initiation of Gait	Steps	Speed	Heel-to-Toe	Turning Corners	Romberg Test	Heel-to-Shin Test	Falls
Cerebellar	Wide-based	Leans forward	Variable	Normal	Staggering, lurching	Normal to slow	Unable	Veers away	Variable	Usually abnormal	Uncommon
Sensory		Stooped	Intact		High-steppage		Variable	Minimal effect	Positive, increased unsteadiness	Variable	Yes
Frontal Lobe		Upright	Impaired	Start hesitation	Short, shuffling	Very slow	Unable	Freezing, shuffling	Variable	Normal	Very common

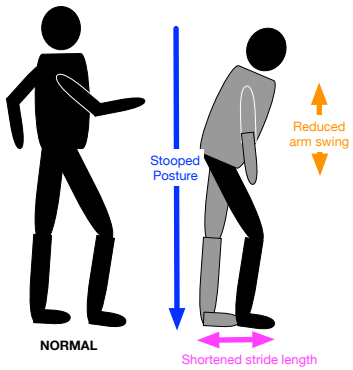
Common Findings Gait Assessments



NORMAL
Narrow base = <0.5 inches between medial aspect of ankles while walking

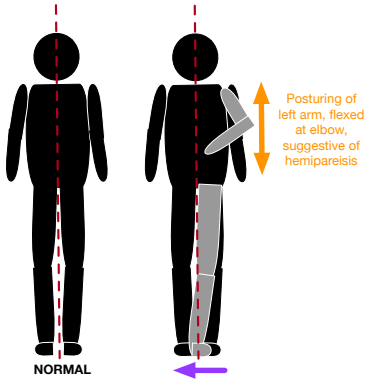
Wide base = >0.5 inches between medial aspect of ankles while walking

In this example, the right leg is externally rotated, suggestive of leg weakness or arthritis



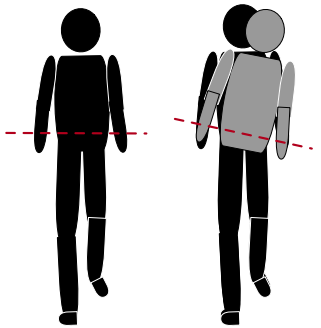
NORMAL

Shortened stride length



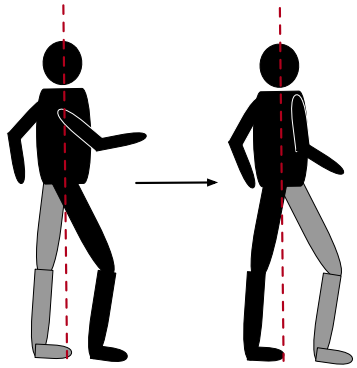
NORMAL

In this examples, the left leg abnormally crosses the midline (dotted red line) in front of right leg, suggestive of spastic (UMN) gait

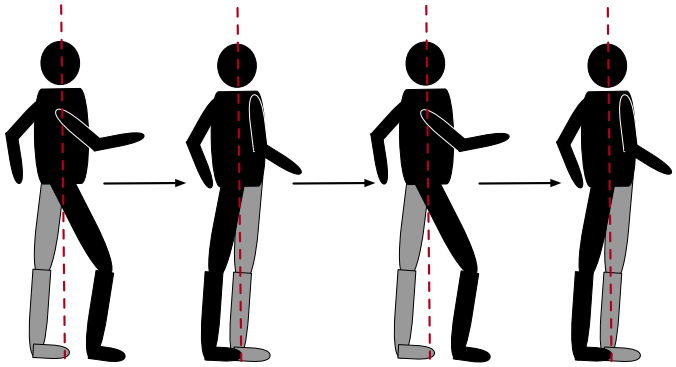


NORMAL
Pelvis (dotted red line) is level when walking

Trendelenberg Gait
Drop of pelvis when lifting leg (in this example, left) opposite to weak gluteus muscle (in this example, right)



NORMAL
In normal gait, the leg always is placed ahead of the other at the end of each consecutive swing phase. Red dotted line represents the midline.



LEG WEAKNESS
In this example, the normal right leg (black) can be place ahead of the left leg (grey). The left leg does not move "ahead" of the right leg and only comes close to the midline, given the appearance that the left leg is dragging (due to weakness, arthritis, or pain).

ABNORMAL GAIT DISORDERS

(adapted from Rubino FA Neurologist. 2002 Jul;8(4):254-62; Beristain X, Chapter 8: Approach to the patient with gait disturbance and recurrent falls in Practical Neurology, J Biller (editor), Wolters Kluwer; Philadelphia, 2017; Pirker W & Katzenschlager R. Gait disorders in adults and the elderly: a clinical guide. Wien Klin Wochenschr. 2017; 129(3): 81–95.)

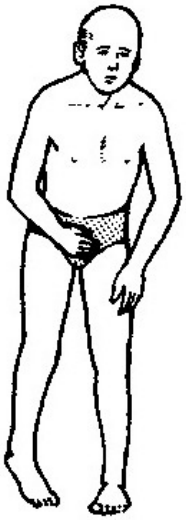
LOWER LEVEL DISORDER / Skeletal, sensory and lower motor gait disorders
Gait disturbance is usually self-limited or compensated by intact central mechanisms when only 1 of the major afferent systems is affected.

CAUSES	GAIT DESCRIPTION	ASSOCIATED NEUROLOGICAL SYMPTOMS	DISORDERS
Visual loss			
• Acute distortion of visual perception	**VISUAL ATAXIA** • Broad base gait with tentative steps		• Cataract surgery without lens replacement
Proprioceptive loss			
• Large-fiber sensorimotor neuropathies	• Broad-based & insecure stance and gait • Shortened step length • Slower gait and more cautious compared to cerebellar ataxia • Feet are sometimes lifted high and gait may have a stomping quality • Patients use visual control to compensate for the loss of proprioception.	Pseudoathetosis of the fingers>toes	Demyelinating neuropathies (eg CIDP)
• Posterior root ganglionopathies			• Paraneoplastic syndromes
Lesions of the posterior root or posterior root entry zone of the spinal cord			• Tabes dorsalis - syphillis • Friederich ataxia
Lesions of the posterior columns of the spinal cord or lemniscal pathways			• compressive myelopathies • multiple sclerosis • subacute combined degeneration - vitamin B12 deficiency
Peripheral Vestibular Lesions			
• ACUTE lesion	**VESTIBULAR ATAXIA** • Base of support is widened • Unsteadiness, usually lean or fall toward side of lesion	• Vertigo • Blurred or double vision • Nausea • Vomiting	NOTE: CHRONIC insidious, slowly progressive unilateral lesion lesion (eg. acoustic neuroma) usually do not have vertigo or gait disturbance but have tinnitus and unilateral hearing loss.
• Bilateral lesions	• Trouble with equilibrium	• No vertigo • Oscillopsia	Ototoxic drugs

LOWER LEVEL DISORDER / Skeletal, sensory and lower motor gait disorders
Gait disturbance is usually self-limited or compensated by intact central mechanisms when only 1 of the major afferent systems is affected.-1

Peripheral nerve disorders			
• Foot drop from weakness or severe deafferentation 	**STEPPAGE GAIT** • Dragging of the foot or fett with walking OR • Compensate by lifting one foot or both feet as hish as possible, with excessive flexion of the hips and knees at every step. • The toe hits the floor before the heel or ball of the foot. • With sensory loss, the heel ends to strike the ground heavily with a characteristic slapping sound.	• Bilateral weakness of the muscles innervated by the peroneal nerves • Sensory loss	• Sensory ataxias • Acquired and hereditary peripheral neuropathies • Compressive peroneal neuropathies • L4-L5 radiculopathies
• Lumbrosacral radiculopathies	• Usually lean away from the involved side, and when weight is put on the painful side, they limp. • In lumbar spinal stenosis, may present with neurogenic claudication.		
Myopathies			
• Proximal muscle weakness • Bilateral hip dislocations (less common) 	**WADDLING GAIT** • Broad-based, short-stepped gait with prounounced lumbar lordosis and exaggerated pelvic rotation • Hips oscillate up and down with every step (ie excessive drop of the hip and trunk tilting to the side opposite the foot place placement) to help shift the weight of the body and cause the characteristic waddling	• Hip (particularly gluteus medius) and shoulder girdle muscle weakness	• Progressive muscular muscular dystrophy NOTE: Adults with acquired myopathies (eg inflammatory myopathies or other diseases that lead to bilateral [eg LEMS] or unilateral [eg diabetic proximal neuropathy] proximal weakness) usually do not have difficulty with gait, but have difficulty going from a sitting to a standing position, going up stairs, or squatting and standing.

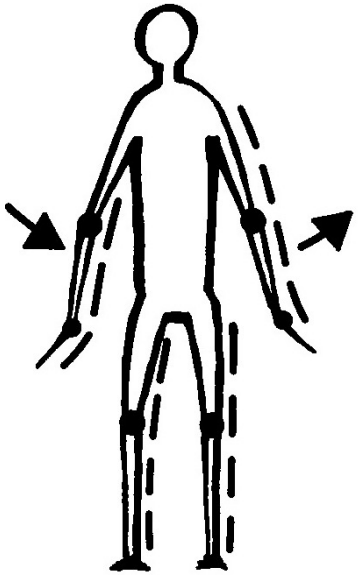
MIDDLE LEVEL GAIT DISORDER / Simpler gait disorders of central origin - Pyramidal, cerebellar, basal ganglia motor system dysfunction)

CAUSES	GAIT DESCRIPTION	ASSOCIATED NEUROLOGICAL SYMPTOMS	DISORDERS
Pyramidal/corticospinal tract lesions 	**SPASTIC GAIT** • Base of support is narrow • In bilateral lesions, the legs tend to cross in front of each other (thighs hyperadduct) in a pattern, called "scissors gait." • Leg is externally rotated at the hip. • Knee is extended and stiff • Foot is plantar flexed and inverted ("spastic foot drop")-- patient tends to scrape the floor with the outer edge of the foot (reduced toe clearance) • Circumduction occurs when the spastic leg swings outward and forward. • Slow turns In unilateral lesions -- spastic hemiparetic gait • In addition to above, the arm is flexed at the elbow and writs, adducted at the shoulder, and usually immobile across the chest or abdomen as the patient walks.	• Myelopathy • Mild standing imbalance • Urinary bladder urgency & frequency • Often absent neck pain and radiculopathy • Numb, paraparetic and clumsy hands with atrophy in the small muscles • If C5-C6. Depressed brachioradialis jerk but brisk finger flexor response is elicited when percussing the brachioradialis tendon (inverted radial reflex)	BILATERAL LESIONS • Cervical spondylosis -- commonly C5-C6 • Parasagittal meningioma • Birth injury (cerebral palsy) • Primary and metastatic spinal cord tumour • Subacute combined degeneration of the spinal cord • Syringomyelia • Multiple sclerosis • Motor neuron disease UNILATERAL LESIONS • Stroke (ischemic or hemorrhage)

MIDDLE LEVEL GAIT DISORDER / Simpler gait disorders of central origin - Pyramidal, cerebellar, basal ganglia motor system dysfunction)-1

Basal ganglia 	**PARKINSONIAN GAIT** • Slow or difficulty arising from a sitting position • Flexed posture, diminished arm swing, and a rigid small-step shuffling gait; tendency for knees to be flexed • Difficulty with initiation of movement and turns • Turning is accomplished with multiple unsteady steps, with the body turning as a single unit (en bloc) • Festination occurs while the patient walks and the upper portion of the body gets ahead of the lower part and the steps become smaller and more rapid	• Unilateral or bilateral rest tremor of the arms • Bradykinesia • Autonomic neuropathy • Ideomotor apraxia • Dementia	• Parkinson's disease • Multiple system atrophy • Progressive supranuclear palsy • Dementia with Lewy bodies • Corticobasal degeneration • Neuroleptic medications
	CHOREIC, HEMIBALLISTIC & DYSTONIC GAITS • Abnormal choreic (lesions of the anterior putamen), hemiballistic (lesions of the subthalamic nucleus), or dystonic (lenticular lesions) movements are superimposed to the normal gait		• Infarcts • Huntington's disease • Sydenham chorea • Wilson disease • Acquired hepatolenticular degeneration • Lupus • Neuroacanthocytosis • Polycythemia vera • Neuroleptic medications • Genetic dystonia • Dopa-responsive dystonia

MIDDLE LEVEL GAIT DISORDER / Simpler gait disorders of central origin - Pyramidal, cerebellar, basal ganglia motor system dysfunction)-1-1

Cerebellar disorder 	**CEREBELLAR ATAXIC GAIT** • Due to lesions of the midline or vermis of the cerebellum • Unsteady broad base gait, staggering from side to side • Unable to do tandem gait and have difficulty stopping and turning • May be rhythmic swaying of the trunk or head or both (titubation)	• Lesions of the flocculonodular lobe (vestibulocerebellum) affect equilibrium. Causes: • Truncal imbalance • Tremor of the head and neck • Falling in all directions • Nystagmus (esp downbeat nystagmus)	• Primary and secondary neoplasms • Toxins (eg. alcohol, phenytoin) • Vitamin E deficiency • Hypothyroidism • Paraneoplastic syndromes • Hypoxia • Hypoglycemia
	ALCOHOLIC GAIT • Due anterior cerebellum involvement • Severe gait and heel-shin ataxia without nystagmus, dysarthria, or arm dysmetria • Slow and halting gait with irregular steps and superimposed lurching • Gait abnormalities are accentuated at the initiation of gait, on turning, and with changes in gait speed,	• Anterior cerebellum is responsible for the coordination of proprioceptive, vestibular, and visual information.	

HIGHER LEVEL DISORDER / Complex Gait Disorders of Central Origin - Dysfunction of highest sensory motor systems

CAUSES	GAIT DESCRIPTION	ASSOCIATED NEUROLOGICAL SYMPTOMS	DISORDERS
Most common gait disorder of the elderly	**CAUTIOUS GAIT** • Normal or mildly widened base, shortened stride, slowness of walking, and turning en bloc - likened to walking on ice or on a deck of a rolling ship • Center of gravity remains within the limits of the base of support.		• Getting old • Compensation for arthritis, pain, sensory or vestibular impairment • Fear of falling
Usually due to lesions that affect the corticobasal ganglionic-thalamocortical loop. Also, lesions affecting the premotor area, projections to the origins of the tectoreticulospinal and vestibulospinal tracts Causes dysequilibrium by impairment in supporting and postural reflexes, impairment in control of proximal and axial muscles and locomotion	**SUBCORTICAL DYSEQUILIBIRUM, FRONTAL DYSEQUILIBRIUM, ISOLATED GAIT IGNITION FAILURE, FRONTAL GAIT DISORDER** • All are closely related -- from a practical clinical standpoint, can be considered as one disorder • Cannot start walking because of hesitation, and there are motor blocks or freezing spells, especially when attempting to turn. • Walk upright with good arm swings and normal base • Steps initially are short and shuffling and then increase in length as the walk continues.	• Mild dementia with slowness and paucity of thought but correct answers • Emotional lability or flat affect • Urinary frequency, uregncy and incontninence • Palmar and plantar grasping reflexes, paratonia and Babinski signs	• Frontal lobe lesions (infarcts, hemorrhages, neoplasms, hydrocephalus, degeneration)
	PRIMARY PROGRESSIVE FREEZING GAIT DISORDER • Anatomic localization is unknown • Seen mainly in older men • Restricted to the legs • Start hesitation, motor blocks or freezing spells, and recurrent falls with walking. • Patient walks as if the feet were glued to the floor, while the upper part of the body is normally mobile • Progression may lead to total inability to walk		

Step Sequence of Some Gait Disorders (from Pirker W & Katzenschlager R. Gait disorders in adults and the elderly: a clinical guide. Wien Klin Wochenschr. 2017; 129(3): 81–95.)

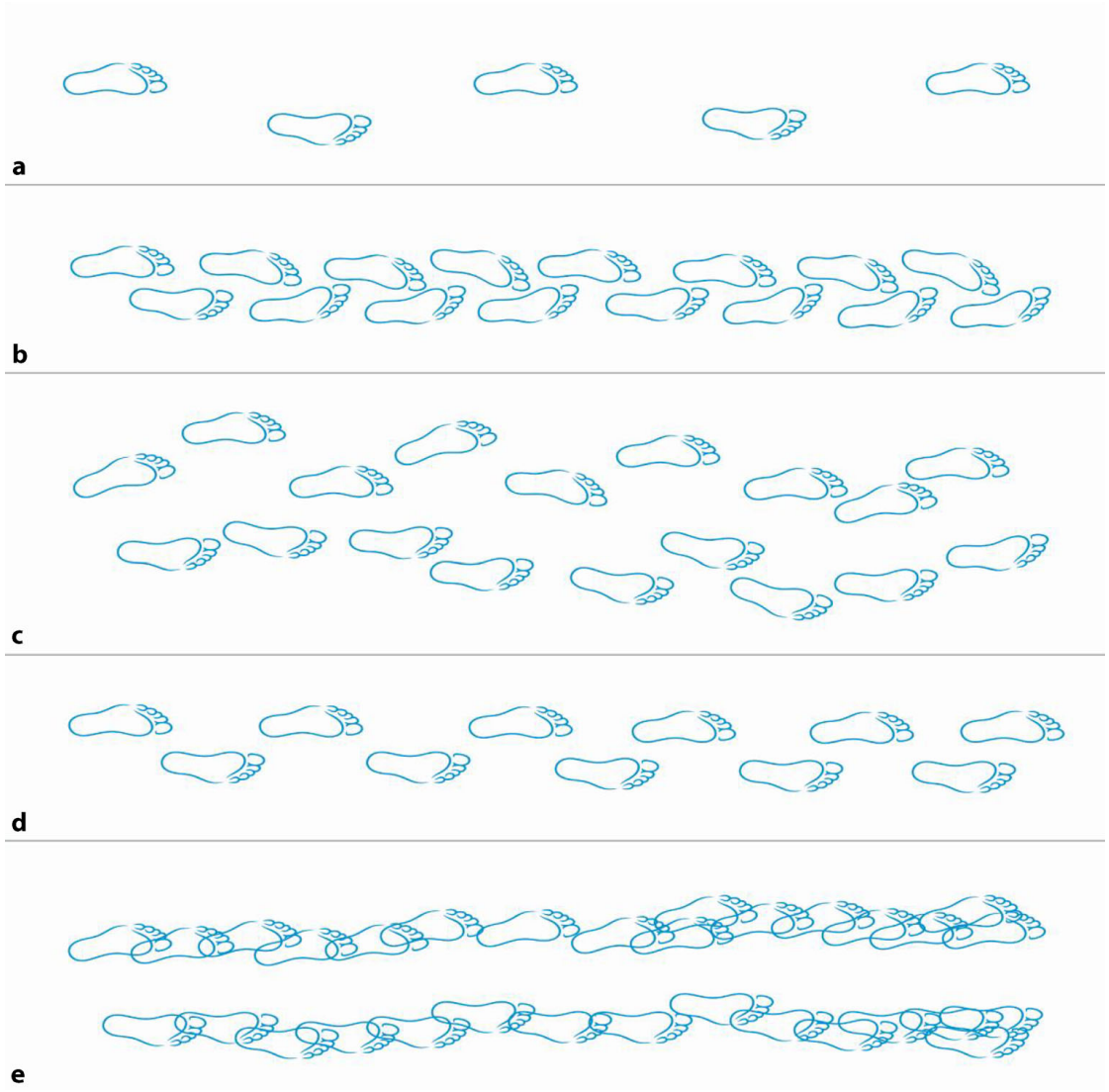


Fig. 3 Graphic representation of the step sequence in classical gait disorders. **a** normal gait, **b** spastic paraparetic gait, **c** cerebellar ataxic gait, **d** parkinsonian gait and **e** frontal gait. Note narrow step width and inwards rotation in paraspastic gait, broadened base and marked irregularity in cerebellar gait, shortened and mildly irregular step length in parkinsonian gait and broad-based, short-stepped, irregular walking in frontal gait disorder