The Neurologic Examination: High-Yield Strategies

S V Khadilkar MD, DM, DNBE, FIAN, FICP,FAMS, FRCP (LONDON) Dean, Bombay Hospital, Mumbai

History

 Tell us what your phobias are and we shall tell you what you are afraid of !

Robert Benchley

History

 No poet ever interpreted nature as freely as a lawyer interprets law.

Jean Girdoux

Or a patient his symptoms!!

Examination Approach

Two types of neurologic examinations
1. Screening Examination
2. Testing Hypotheses ____t

Select high-yield tests and techniques

Examination Approach

Organization

- 1. Mental Status
- 2. Speech
- 3. Cranial Nerves
- 4. Motor
- 5. Reflexes
- 6. Sensory
- 7. Coordination
- 8. Gait

 If the patient can give a completely coherent history, then the mental status examination is probably normal

MMSE is a good screening test

Speech does not equal language: test three elements of language in each patient

Voice Box: Dysphonia Articulatory apparatus: Dysarthria Language: Dysphasia

Aphasia Testing

Fluency: Use Naming and Conversation

- Comprehension: 3 stage commands
- Repetition: "Today is a good day..."

Analysis of Aphasias



Cranial Nerve Testing

II: Pupils, Acuity, Visual Fields III, IV, VI: Extraocular Movements V: Facial Sensation **VII: Facial Strength** VIII: Hearing IX, X: Palatal Elevation and Gag XI: SCM and Trapezius Power XII: Tongue Power

3. Visual field testing is highly informative and underutilized by the non-neurologist

Screening for Visual Field Deficits

- Cooperative patient: Move finger in the center of each quadrant with patient gaze fixed
 - Test each eye by covering the opposite eye, present stimulus in all 4 quadrants
- Uncooperative patient: Use a single digit to suddenly approach each half of the visual fields; normally elicits a blink
 - Avoid using entire hand: elicits corneal reflex
 - Report as "Does/Does not blink to threat"



4.Cranial Nerves in the Brainstem have localizing value

<u>Level</u> Midbrain Pons Medulla Cranial Nerves III, IV V, VI, VII, VIII IX, X, XI, XII

Blumenfeld H. Neuroanatomy Through Clinical Cases. 2002.

 Use an appropriate screen for Upper Motor Neuron-type weakness

Upper Motor Neurons of the Pyramidal Tract



Predictable Pattern of Weakness

Distal Extensors of the UEs and Distal (Dorsi)Flexors of the LEs

Quick Screen for Upper Motor Neuron/Pyramidal Weakness

- Pronator Drift
- Fine Finger Movements/Toe Taps
- One muscle in each of four extremities

 Upper Extremities: 1st DI or finger extensors
 Lower Extremities: Extensor of big toe

• Hand grip screen tends to be insensitive!

6. Use examination to localize the weakness in the nervous system

	UMN	LMN	
Pattern of Weakness	Pyramidal	Variable	
Function/Dexterity	Slow alternate motion rate	Impairment of function is	
	ht	mostly due to weakness	
Tone	Increased COPYRIGHT	Decreased	
Tendon Reflex	Increased	Decreased, absent or normal	
Other signs	Babinski sign, other CNS signs	Atrophy (except with problem	
	(e.g. aphasia, visual field cut)	of neuromuscular junction)	

	Motor Neuron Disease	Neuropathy	NMJ	Myopathy
Weakness Pattern	Variable	Distal	Diffuse	Proximal
DTR	Increased, normal and/or decreased	Decreased or absent	Normal or decreased	Normal or decreased
Atrophy	Yes	COPHesight Sometimes	No	No
Fasciculations	Yes	Sometimes	No	No
Sensory symptoms/ signs	No	Yes	No	No

7. Use the sensory examination sparingly and logically, testing each major pathway

Sensory Testing Modalities

- Vibration (128 Hz Tuning Fork)
- Joint Position Sense/Proprioception
- Temperature
- Pinprick
- Light Touch (Not Useful)

8. Symmetry of reflexes is important, rather than absolute value

Reflex Tips

- Symmetric positioning is key
- Expose the muscle being tested
- Strike with only moderate force

9. In the coordination exam, bilateral abnormalities are more likely to be benign and acute more disastrous

Cerebellar tracts run through the brainstem

Cerebellar signs with cranial nerve deficits is a brainstem lesion until proven otherwise

10. The single most useful test on the neurologic exam is having the patient ambulate

Syndrome Diagnosis (Anatomical Localization)

- 1. Pattern Recognition (syndrome)
- 2. Nine (Anatomic) syndrome patterns





Nine Syndrome Patterns

- Muscle
- Neuromuscular junction
- Peripheral nerve
- Nerve root
- Spinal cord
- Cerebellum
- Brainstem
- Sub-Cortical
- Cortical

1. Muscle – Proximal symmetric weakness <u>without</u> sensory loss

History

 Lower Ext – difficulty rising from sitting position
 Upper Ext – difficulty lifting bags, small children etc.,
 Normal sensation – may have myalgia or cramps

1. Muscle

• Exam

Proximal symmetric weakness without sensory loss

Muscles large size, no fasciculations

Tone and DTRs are normal to slightly decreased



2. Neuromuscular Junction Resembles muscle: proximal variable weakness

• History

Fatigability (waxing and waning weakness) Patient fatigues with prolonged activity (myasthenia gravis)

Patient strength improves with activity (myasthenia syndrome)

2. Neuromuscular Junction

• Exam – Eyes, eyelids, swallowing

- Fatigability of proximal muscles
- Looses strength after exercise (eg., ptosis after sustained upward gaze)
- Muscles normal size, no atrophy or fasciculations
- Normal tone and DTRs

Myasthenia: Ice Pack Test



3. Peripheral Nerve Distal Weakness

• History

LL – trips, drags feet, wears out toes of shoes

- UL-drops objects, problems with grip
 - Asymmetric weakness localized to involved nerve (compression syndromes, Mono. Multiplex, demyelinaing neuropathies)

Symmetric weakness – secondary to metabolic changes (eg., diabetes, renal etc)

Muscle atrophy, twitching or fasciculations Sensory changes - paresthesiae

3. Peripheral Nerve

• Exam **Distal symmetric weakness** Atrophy **Sensory loss** Distal symmetrical **DTRs decreased or absent** Autonomic changes **Trophic changes** – smooth shinny skin Vasomotor changes – swelling or temperature dysregulation, loss of hair or nails




Nerve Hypertrophy



4. Nerve Root Pain is the hallmark

- History sharp, stabbing, hot, electric, shooting or radiating pain
 - Resembles peripheral nerve but weakness may be proximal or distal depending on the involved nerve root
 - Lower ext L5 S1 is most common; distal
 - Upper ext C5-C6 is most common: proximal



Reflex man. Simple as counting-start from the feet

4. Nerve Root

• Exam

- Distal, asymmetric weakness
- Atrophy and fasciculations
- Tone normal or decreased
- DTR decreased or absent in involved muscles
- Sensory loss (dermatomal)
- Maneuvers that stretch the nerve root increase pain (eg., valsalva, SLR etc.,)

Distribution of Root Pain and Paraesthesiae





5. Spinal Cord Triad of Symptoms

- 1. Sensory level Pathognomonic
- 2. Distal symmetric, spastic weakness (UMN) mimics peripheral nerve
- 3. Bladder and bowel dysfunction due to autonomic fibers in spinal cord

5. Spinal Cord

• History

LL– drags toes or trips UL– drops objects or problem with grip

Symmetric – both legs or both arms and legs equally

Sensory complaint – belt, band, girdle or tightness around trunk or abdomen

Sphincter dysfunction – retention or incontinence of bladder more common than bowel

5. Spinal Cord

• Exam

Sensory level (tested with pinprick)

Weakness more common in legs than arms Urinary retention or incontinence Superficial reflexes decreased (anal wink, bulbocavernosus and cremasteric) UMN damage - distal > proximal weakness (weakness of

extensor and (anti-gravity muscles greater than flexors)



Commissural syndrome

Sensory loss with sacral sparing due to the intramedullary lesion shown on the left, involving lateral spinothalamic tracts bilaterally.

POSTERIOR VIEW



Brown-Sequard Syndrome

6. Brainstem – Ipsilateral cranial nerve and contralateral long tract signs (essentially the spinal cord with embedded cranial nerves)

• History

- Long tracts (hemiparesis or hemisensory loss)
- Cranial nerves (the 6 Ds)
 - Diplopia
 - Dysarthria
 - Dysphagia
 - **Dizziness**
 - Deafness
 - Decreases strength or sensation over the face (crossed signs may be bilateral)

6. Brainstem

• Exam

<u>Cranial nerves</u> <u>Ipsilateral</u> -ptosis, pupillary abnormality, extraocular paralysis, diplopia, nystagmus, decreased corneal and blink reflexes, facial weakness or numbness, deafness, vertigo, dysarthria, dysphagia, weakness or deviation of the palate, decreased gag reflex, weakness of neck, shoulders or tongue

Long tracts – Contralateral distal extensor (UMN) hemiparesis, increased DTRs, spasticity, Babinski, loss of some and possibly all modalities



Distribution of pain and temperature sensation loss characteristic of lesions at the posterior fossa level.

Sensory Pathways



7. Cerebellum - In-coordination, clumsiness, intention tremor (smooths and refines voluntary movements)

• History

- Clumsiness in lower ext. –staggers, drunken walk
- Clumsiness in upper ext. difficulty with targeting movements (such as lighting cigarettes, keys in car ignition) and intention tremor
- Brainstem symptoms are common with cerebellar disease and vice versa

7. Cerebellum



LL - Gait (staggering, wide based, ataxic, difficulty with tandem walking, Heel-shin, or tracing patterns on floor with toe

UL – Intention tremor, difficulty targeting movements (such as finger-nose, heel shin) difficulty with rapid alternating movements (dysdiadochokinesis)

8. Sub-cortical verses9. Cortical

- History generally diagnosed by
 - 1. Specific cortical defects
 - 2. Pattern of motor and sensory defects
 - 3. The type of sensory defects
 - 4. Presence of visual field defects

Sub-cortical v Cortical

1. Specific Cortical Defects

Language (dominant hemisphere)

Speech – aphasia Writing – agraphia Reading – alexia Comprehension (eg., apraxia)

(agnosia)

Visual-spatial (Non-dominant hemisphere) Denial or neglect of physical signs and symptoms

Sub-cortical v Cortical

2. Patterns of motor & sensory defects (homunculus)

Cortical lesions - complete paralysis or sensory loss of face and arm (spares legs)

Subcortical lesions – complete paralysis or sensory loss of face, arm, trunk and legs

Sub-cortical v Cortical

3. Type of sensory defect (most primary sensory modalities reach consciousness in the thalamus and do not require the cortex for their perception)

Cortical lesions – patients can still feel pain, touch, vibration and position but have impaired higher sensory processing, ie., graphesthesia or astereognosis)

Subcortical defect – patient complains of significant numbness

Sub-Cortical v Cortical

4. Visual field defects (fibers run subcortically)

Cortical – no visual field defect unless occipital lobe involved (cortical blindness-Anton's syndrome)

Sub-cortical has visual field defects

Sub-cortical v Cortical Exam

- 1. Cortical aphasia, visual-spatial dysfunction or seizures
- 2. Motor UMN weakness Cortical - Face and arm

Sub-cortical - Face, arm, trunk and leg



Suprathalamic syndrome



Thalamic syndrome

Sub-cortical v Cortical Exam

3. Sensory

Cortical – impaired higher sensory processing, (eg.,graphesthesia or astereognosis) with relatively normal sensation

Sub-cortical – decrease primary sensory modalities, (eg., pinprick and touch etc.,)

4. Visual

Cortical – no defect unless occipital lobe

Sub-cortical – visual field defects

Conclusions

- Neurological examination can be tailored to a given situation
- Use of High yield strategies reduces the time required for examination
- Pattern recognition increases yield

Follow the masters of clinical neurology!!

 I am seldom interested in what he [Pound] says, but only in the way he says it.

T S Eliot

Thank you for your attention



History

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Aphasia Chart

Transcort MotorBadGoodGoodTranscort Sens.GoodBadGoodTranscort MixedBadBadGood

The "High-Yield" Neurologic Examination: Top Ten Suggestions for a Better Neurologic Examination



Two Localizations of Coma

• Step 1

• Step 2

"Fixed" Pupils and Coma

