Evaluation of Numbness, Tingling and Weakness

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Structure of the talk

- What is numbness, what are its causes and how to localize it
- Evaluation of numbness and tingling by exam
- Evaluation of numbness and tingling by investigations
- Electrodiagnostic evaluation of numbness and tingling

Evaluating numbness, tingling and weakness by history

- Clarify what is numbness
- Then localize where it is coming from
- Identify the etiology based on localization, temporal course and associated risk factors such as family history, occupational history and constitutional history
- Use the pneumonic SMART to guide you

Pneumonic for evaluating numbness/tingling/weakness

- S→ sensory symptoms
- M→ motor symptoms
- A→ autonomic symptoms
- R→ reflexes
- T→ trauma/falls

Causes of numbness tingling and weakness

Etiology	Central Nervous System	Peripheral Nervous System
Vascular	stroke, arterial-venous malformation, claudication	peripheral vascular disease
Structural	tumor, disk	tumor, disk
Inflammatory	infection, vasculitis	neuropathy, vasculitis, infection, myositis
Genetic	myopathy, motor neuron disease	neuropathy
Immune mediated	Multiple sclerosis, myelopathy	neuropathy, neuromuscular junction disease

Causes of numbness tingling and weakness- VITAMIN CDE

- Vascular
- Infectious / Inflammatory
- Traumatic / Toxic
- Autoimmune
- Metabolic
- Iatrogenic / Idiopathic
- Neoplastic
- Congenital
- Degenerative
- Endocrine

What is numbness?

- Patient complaints of "numbness" can include a range of true sensory disturbances.
- "Tingling," "burning," or true loss of sensation can each be described simply as "numbness."
- Specific questioning, therefore, can help in identifying the etiology of the disease process.

- Numbness can result from either a disease process located in the central nervous system (brain or spinal cord) or the peripheral nervous system (nerve root, dorsal root ganglion, or nerve).
- Recognition of both the **timing and onset** of the patient's symptoms and the **distribution** of the patient's signs and symptoms are essential.

Numbness- CNS vs. PNS

Patient's Description of Sensory Loss	Likelihood of the localization
"Tingling"	Peripheral NS > Central NS
"Burning"	Peripheral NS > Central NS
"Total loss of feeling"	Central NS[1] > Peripheral NS[2]
"Poor coordination" [3]	Central NS = Peripheral NS

[1]Localization to the central nervous system would most commonly involve unilateral signs and symptoms except with spinal cord lesions where symptoms are usually bilateral. [2]Peripheral nervous system lesions can be either multifocal or, commonly, bilateral and symmetric. Early signs in a neuropathy may involve only one extremity (usually the feet) but peripheral involvement would be much less likely with all unilateral complaints. [3]Poor coordination can result from either central (cerebellar, brainstem) or peripheral (impaired proprioception, nerve, dorsal root ganglion) pathology.

Localization of neurological symptoms

	Distribution	Facial Involvement	Characteristic	Pain
Brain	Unilateral	often	sensory + motor	no
Spinal Cord	Bilateral	no	sensory + motor	possible
Nerve Root	Unilateral	no	sensory + motor	yes
Nerve	Unilateral or bilateral	possible	sensory, motor, autonomic or combinations	yes
Neuromuscular junction	often bilateral	yes, but not always	motor	no
Muscle	Bilateral	rare	motor	unlikely

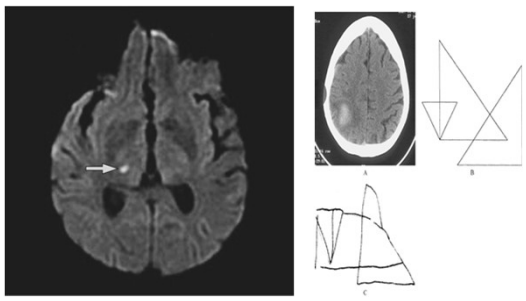
Brain-numbness/tingling/weakness

- A presenting symptom of numbness may be secondary to lesions of the parietal lobe or thalamus (ventral posteromedial nucleus).
- Thalamic lesions produce contralateral sensory loss and numbness, which may be painful.
- Caused by lacunar infarcts or hypertensive bleeds.

Brain-numbness/tingling/weakness

- Parietal lobe lesions are characterized by sensations of contralateral numbness but on examination we find loss of discriminatory sensation, possibly accompanied by neglect of that extremity.
- The patient may feel touch but may not localize it well.
- Sensory complaints should be more prominent than motor.

Imaging findings of thalamic stroke (left) and parietal bleed (right)



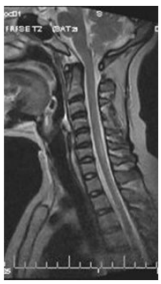
Spinal cord-numbness/tingling/weakness

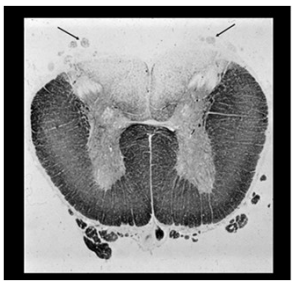
- Lesions in the spinal cord resulting only in numbness are less common than of both numbness and weakness due to the close proximity of motor and sensory neurons and their pathways.
- This is especially true of structural (herniated disk, trauma, tumor) and ischemic lesions.
- Inflammatory lesions, however, can selectively affect sensory pathways and result in profound, usually bilateral, sensory disturbance.

Spinal cord-numbness/tingling/weakness

- Sensory disturbance characterized by selective abnormality of position sensation (proprioception) suggests a selective involvement of the dorsal columns and disorders such as multiple sclerosis, vitamin B12 deficiency, and tertiary syphilis should be explored.
- Temperature, light touch and pain are mediated by the smaller fibers of the spinal thalamic pathway. These fibers are not usually selectively affected in the spinal cord.

Dorsal column involvement on MRI(left) and on microscopy (right)





Nerve root-numbness/tingling/weakness

- Dorsal Root Ganglion Dorsal (sensory) and ventral (motor) nerve roots are separate as they exit the spinal cord until their fibers combine at the level of the dorsal root ganglion.
- The most common nerve root pathology results from a herniated vertebral disk.
- This usually results in pain with radiation into the affected dermatome. Motor symptoms are uncommon due to multiple nerve root innervating a given muscle.

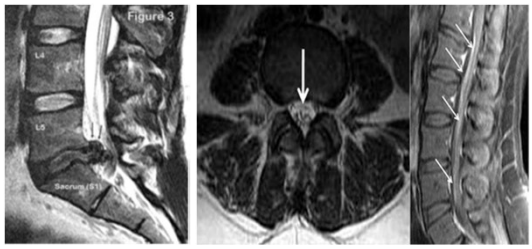
Nerve root-numbness/tingling/weakness

- The involvement of multiple nerve roots suggests-
  - inflammatory (Guillain Barré, CIDP, amyloidosis, vasculitis)
  - neoplastic (carcinomatous meningitis)
  - infectious (syphilis, Lyme)
- Selective involvement of the dorsal root ganglion cells causes a profound sensory disturbance.

Nerve root-numbness/tingling/weakness

- Cis-platinum toxicity following chemotherapy is an example.
- Paraneoplastic (anti-Hu) syndrome can result in painful, asymmetric sensory loss to all modalities with normal motor function.
- This pathology predominantly affects women and is commonly associated with concurrent small cell carcinoma

Disc herniation (left) and radiculitis (right)

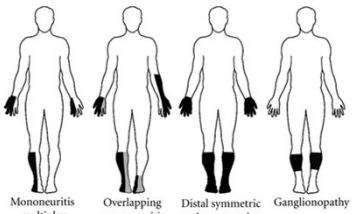


Peripheral nerve-numbness/tingling/weakness

- Sensory disturbances caused by neuropathy are common.
- More commonly, however, neuropathy presents with both motor and sensory symptoms reflecting the involvement of both fiber types in the nerve.
- Small fiber sensation includes light touch, pain and temperature while large fiber sensation includes position sensation and vibratory sensation.

	Etiology				
Primary Characteristic	**Toxic**	**Immune**	**Metabolic**	**Inherited**	**Other**
Pain	Alcohol Metals: thallium, arsenic, Meds: cis-platinum, disulfiram, nitrofurantoin, taxol	Guillain Barré syndrome, HIV, Sjögren's, Vasculitis, Cryoglobulinemia	Diabetes, Vitamin related	Fabry's disease (a-galactosidase), Hereditary Sensory Neuropathy, Amyloidosis, Porphyria	
Large & Small Fiber	Metals: thallium, mercury, Drugs: thalidomide, taxol, metronidazole, phenytoin	Paraneoplastic (anti-Hu), Anti-MAG, ant-sulfatide, Sjögren's, Cryoglobulinemia	Diabetes	Hereditary Sensory Neuropathy (AR)	
Large Fiber & Ataxia	Vitamin related, Cis-platinum, Taxol	Miller Fischer variant of Guillain Barré syndrome, CIDP, Anti-MAG syndrome[2], CALOP syndrome[3]		Friedreich's ataxia, Sensory ataxic neuropathy, Ataxia telangiectasia	Syphilis-tabes dorsalis
Small Fibers Mostly	Chronic Metronidazole, or misonidazole	HIV-Associated	Diabetes, Hypetrigly-ceridemia	Amyloidosis Hereditary Sensory Neuropathy, Tangier's Disease, Fabry's disease	Leprosy, 1° biliary, cirrhosis
Autonomic Symptoms		Guillain Barré, Paraneoplastic (anti-Hu)	Diabetes	Amyloidosis, Porphyria	MNGIE[4], #Chronic Renal or hepatic disease

Neuropathic patterns



Temporal course

- Slow progressive numbness or sensory disturbance is most commonly described as distal numbness or tingling in the toes.
- This usually progresses from the feet and then up the legs [distal to proximal progression or “dying back” pattern].
- The progression happens over many years. Most common neuropathies present this way.

Temporal course

- Sensory complaints due to radiculopathy can also evolve slowly, however they are frequently characterized by pain in the affected root distribution, which is exacerbated by activity.
- Sensory complaints from a radiculopathy or neuropathy can be episodic, especially earlier in their course.

Temporal course

- A rapidly ascending numbness and tingling should arouse the suspicion of GBS even without weakness especially with antecedent infection.
- A subacute week to few days patchy pain, numbness and tingling should give rise to suspicion of vasculitis neuropathy.
- Sensory disturbances from pathology in the brain or spinal cord usually evolve more acutely. Ischemia or inflammatory disease in the central nervous system also evolves rapidly over several days.

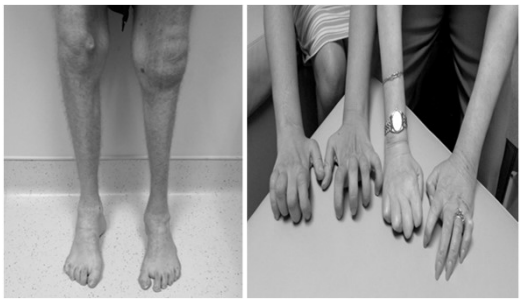
Temporal course

	ACUTE (Days)	CHRONIC (Weeks - Months)
Immune	Guillain-Barré & variants, Vasculitis	Chronic demyelinating neuropathy
Toxins	Botulism, Buckthorn, Diphtheria; Tick; Arsenic; Organophosphates; Thallium; Vacor	Heavy Metals , Environmental Chemicals
Drugs (see Table 6)	Captopril (few case reports); Gangliosides; Gold; Nitrofurantoin; Suramin; Zimeldine	Chemotherapeutic Agents
Metabolic	Porphyria	Porphyria, Diabetes
Nutritional		Vitamin toxicity or deficiency
Hereditary		Hereditary motor and sensory neuropathy (HMSN), hereditary sensory neuropathy (HSN)

Family History

- Localization to the brain or spinal cord can be aided by identification of similar disease in the family; however, the symptoms are usually not limited to sensory complaints or numbness.
- The hereditary motor and sensory neuropathies (HMSN, Charcot Marie-Tooth neuropathies) comprise a group of disorders with overlapping clinical characteristics but distinct pathology.
- History of being clumsy, recurrent falls, not being to keep up with peers need to be clarified.

Typical exam findings in CMT



Occupational History

- Incidental exposure to agents, which are toxic to nerve, may be easily missed on a routine history and review of systems.
- Contact with solvents, glues, fertilizer, oils and lubricants can result in a neuropathy indistinguishable from other causes of idiopathic or hereditary etiology.

Medication History

- Over the-counter oral preparations can result in a profound sensory predominant neuropathy even when taken at therapeutic doses.
- Patients who have neuropathy not associated with or caused by a medication may still be vulnerable to exacerbation of their symptoms by taking these neuropathic agents and should be advised to avoid these preparations

Medication History

chloramphenicol	metronidazole	phenytoin	Nitrofurantoin
cis-platinum	nitrous oxide	pyridoxine	taxol
ethambutol	nucleosides	thalidomide	
glutethimide	[didianoside (ddI)	dapsone	
hydralazine	dideoxycytosine (ddC)	disulfiram	
isoniazid	stavudine (d4T)]	disulfiram	

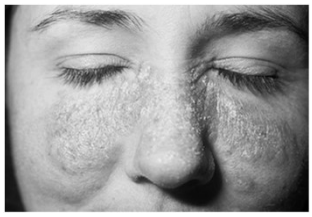
Nursing triage

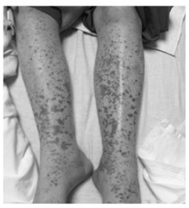
- Numbness on one side of body –could be a stroke, call 911.
- Rapidly ascending numbness/tingling starting from feet with/without recent infection- come to ER.
- Has history of neuropathy, pain worse- talk with your physicians.

General Physical Exam

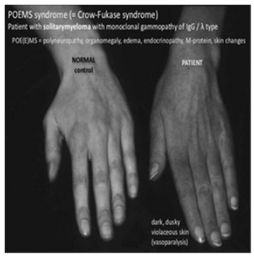
Clinical Sign	May suggest:
Rash	Lupus
Funduscopic examination	Diabetes, Vasculitis
Adenopathy	Infection, Cancer
Weight loss	Diabetes, Cancer, Endocrinopathy
Bony or Cutaneous abnormalities	Inherited neuropathy, Endocrinopathy
Organomegaly	"POEMS" syndrome[1]

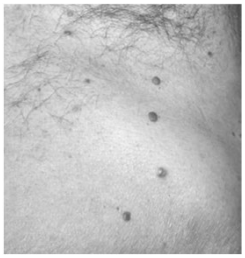
Lupus rash (left) vasculitic rash (right)



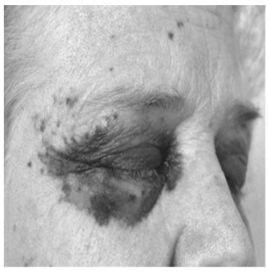


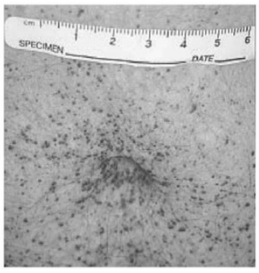
Dusky skin changes (Left) and Cherry angioma-like vascular lesions (right)



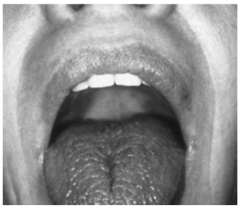


Amyloid rash (Left) and angiokeratoma in Fabry disease (right)

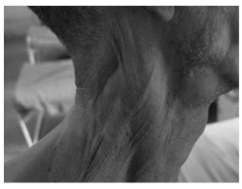


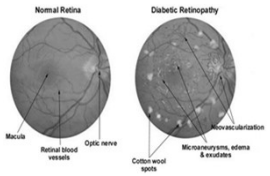


Dry mouth Sjogren's (left) and dry skin hypothyroidism (right)



Nerve thickening in Leprosy (left) and Fundus changes in Diabetes (right)

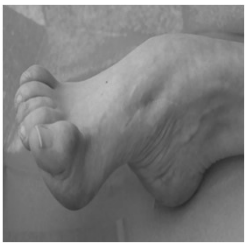
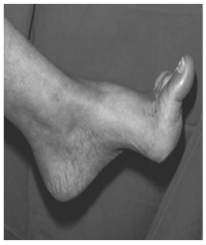




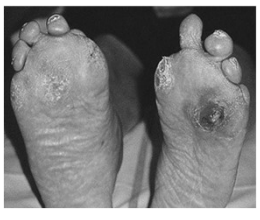
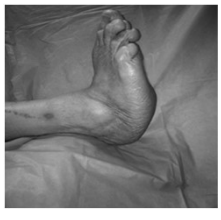
Examination of the feet

- Critical in the diagnosis of neuropathy
- Look for high arch, hammer toes
- Look for Charcot feet
- Look for pressure ulcers
- Look for color and nail changes

High arch with hammer toes (left) and hair loss with dry skin(right)

Pressure ulcer (left) Charcot feet (right)

Neurological Exam-Reflexes

Lesion	Reflexes
Brain/spinal cord	Brisk (hemi body with brain, b/l with spinal cord)
Roots	Reduced/absent (affected root only)
Large nerves	Reduced/absent (b/l and symmetric)
Small nerves	Normal

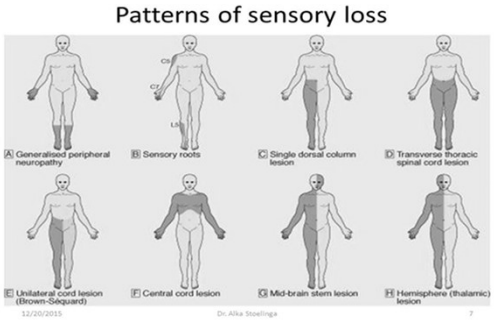
Sensory Exam

- Differences between patient perceptions and their ability to relate subtle differences in pinprick or temperature can make this the most challenging part of the physical examination.
- In general, absolute differences in sensation are not as important for localization or diagnosis as are the relative differences in perceived sensation.
- The most useful information from the sensory examination results, therefore, from the distribution of the deficit (symmetric vs. asymmetric) and the type of the sensory loss.

Sensory loss patterns

Modality	Fiber type (Periphery)	Tract (Central)
Light touch	small fiber	Spinothalamic
Temperature	small fiber	Spinothalamic
Pinprick	small fiber	Spinothalamic
Two-point discrimination	small fiber	Spinothalamic (parietal)
Proprioception	large fiber	Dorsal columns
Vibration	both small and large fiber	

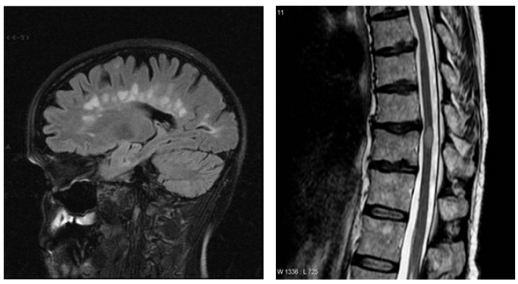
Sensory loss based on localization



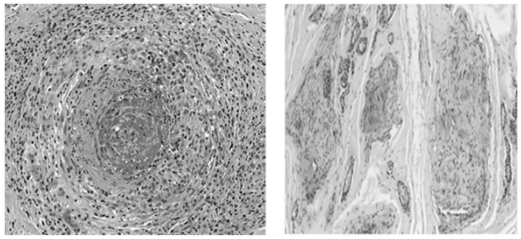
Lab tests for neuropathy

Localization	Lab tests	Radiology	Other
Brain	Hb-A1c, lipid profile, ESR, ANA, CBC, electrolytes, RPR	MRI with contrast Transcranial Doppler, Carotid Doppler	EKG, ECHO, possible Holter
Spinal cord	Routine lab screen evaluating underlying illness, B12	MRI with contrast	LP with opening pressure, protein, chemistry and cytology
Nerve Root	Not usually indicated	CT or MRI of the suspected area. Myelogram may be indicated	Nerve conduction and electromyography including F and H wave evaluation
Nerve	Hb-A1c , ESR, ANA, CBC, electrolytes, RPR, SPEP, immunofixation, B12 As indicated: ANCA, cryoglobulins	Not usually indicated	Nerve conduction and electromyography Possible nerve & muscle biopsy as indicated

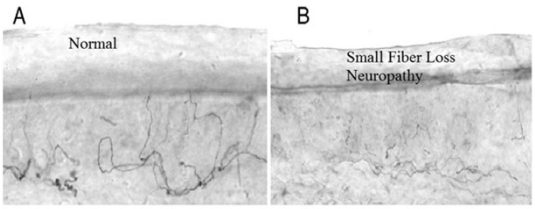
Dawson's finger in MS(left) and transverse myelitis (right)



Vasculitis on nerve biopsy (left) and amyloid deposition (right)

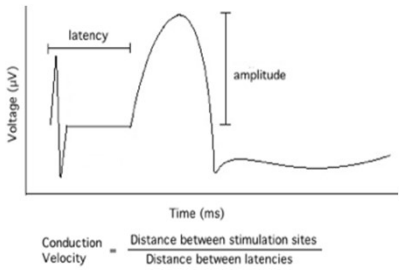


Punch biopsy for diagnosing small fiber neuropathy



Pediatrics. 2013 Mar 11 in press. Evidence of Small-Fiber Polyneuropathy in Unexplained, Juvenile-Onset, WidespreadPain Syndromes. Oaklander AL, Klein MM.

Nerve conduction study



$$\text{Conduction Velocity} = \frac{\text{Distance between stimulation sites}}{\text{Distance between latencies}}$$

Electrodiagnostic findings

Nerve conduction findings	Localization
Normal	Brain, spinal cord, small fiber nerve
Prolonged latency, slowed conduction	Demyelinating peripheral nerve lesion
Small amplitude	Axonal peripheral nerve lesion
Normal/small amplitude/prolonged F-wave latency/H wave latency	Root involvement

F-wave prolongation in lesions affecting the root

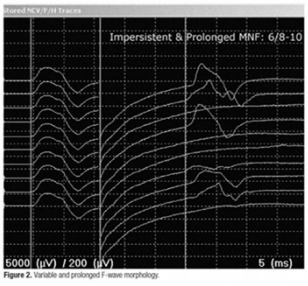


Figure 2. Variable and prolonged F-wave morphology.