Peripheral neuropathy: **Resources:** Doctor's given review article & notes.

"the review article I gave is more than enough." - Dr. Mohammed

Taking history from a patient with peripheral neuropathy: They usually present with numbness and weakness, you should analyze the symptoms.

History of presenting illness:

1. **Nature of the symptoms?**
   - **Sensory:** tingling, paresthesia, tightness around the feet like they're wearing socks, numbness (loss of sensation), burning pain.
   - **Motor (most likely weakness):** by history you should know the following:
     - **Proximal or distal? Distal** -> dragging of the foot when walking, tripping & falling, **Proximal** -> climbing stairs difficulties, washing hair, grabbing things from the top shelf, lifting things up, rising from a chair
     - **Symmetrical or asymmetrical?** Symmetric only in one part.
   - **Autonomic symptoms** (you must ask)**:** Orthostatic lightheadedness (when you stand up do you feel dizzy?), impotence, excessive sweating, erectile dysfunction, dryness, dry mouth, dry eyes.
2. **Onset: acute** (recent), **subacute** (few weeks), **chronic** (long time ago)? This is very important to generate you DDx.
3. **Duration.**
4. **Progression.** Stable or slowly progressive or quickly progressive?

Past medical history:

- **Diabetes:** which is very common. (Most common causes of neuropathies is diabetes then idiopathic and hereditary), بس انتبهوا احيانا حتى ما يكون سبب البرفال نوروباثي هو الديابيتيز
- **Thyroid diseases.**
- **Renal failure.**
- **Malignancy.** (chemotherapy can cause neuropathies - CA patients are also malnourished which can also cause peripheral neuropathy).
- **Connective tissue diseases: SLE, RA (can cause vasculitis & nerve infraction which lead to neuropathy, .. etc**
- **Lumbar or disc disease.**
- **Orthopedic procedure.**

Family history: very important, hereditary neuropathy is common. Strong family hx makes your life easy!

- **Deformity in the feet? High arched feet?** احيانا مايعرف المريض اذا كان عندهم بالعائلة فلو تشوفون أي دفورمتي اسألوه ما إذا كان فيه احد بالعائلة عنده نفس المشكلة أو اسألوهم بشكل عام ما إذا كانت فيه دفورمتيز بالعائلة
- **Walking difficulties.**

Social history: exposure to multiple toxins can cause neuropathies so don't forget this point !

- **Vitamin abuse: B6.**
- **Herbal medications.**
- **Toxins (Alcohol is the most common one)**
- **Their job also.**

So if an OSCE station comes to you asking: Take a history from a patient with peripheral neuropathy.. do you know how to ask & what to ask? EASY !

Examining a patient with peripheral neuropathy: Aim: localization

- **Motor \ sensory?**
  - **Sensory:** Compare both sides, go distal to proximal, Examine dermatomes: L4 – L5 – S1, Examination of the foot nerves: sural nerve (lateral side of the foot + sole), superficial peroneal (Area of L5: , saphenous nerve (area of L4: Medial side of the leg & medial malleolus).
    - i. **Pain & temperature.**
    - ii. **Vibration & proprioception.**
    - iii. **Light touch.**
    - iv. **Heavy touch.**
    - v. **Cold sensation.**
- **Proximal \ distal?**
- **Symmetric \ asymmetric?**
- **Systemic feature.**
- **Autonomic changes**
- **Peripheral pulses: if diabetic they might have ischemia.**

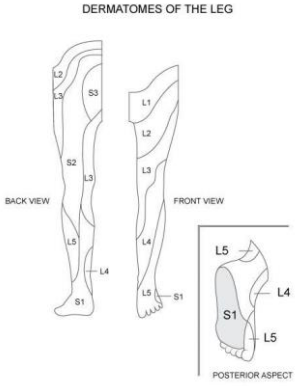

TABLE 1: DERMATOMES	
C1	Top of head
C2	Face
C3	Lateral neck
C4	Lower neck and top of shoulder
C5	Shoulder to base of thumb outside of arm
C6	Front of shoulder, down arm, into thumb and back of hand
C7	Back of shoulder, down back of arm, into back of hand
C8	Little finger and wrist
T1	Inside elbow down to wrist
L1	Back and over trochanter
L2	Back and anterior thigh to knee
L3	Back, upper buttocks, anterior thigh and knee, inner lower leg
L4	Inner buttocks, outer thigh inside of leg, and dorsum of foot and big toe
L5	Buttock, back, side of thigh, lateral leg, dorsum of foot, inner half of sole of foot, and second and third toes
S1, S2	Buttock, posterior thigh and lower leg
S3	Groin and inner thigh to the knee
S4	Genitals

الدكتور ركز على اللي بالأحمر

Approach to a patient with peripheral neuropathy:

The whole idea of this lecture is to apply the 3-6-10-step clinical approach to neuropathy: 3 goals, 6 key questions, 10 phenotypic patterns.

3 goals:

1. To determine the **location** of the lesion.
2. To know the **cause** of the lesion.
3. To determine whether the **therapy** is possible?

6 Questions:

Easily defined clinical patterns of involvement are used to identify patients in need of neurologic consultation, whenever you're confronted with a case of peripheral neuropathy it is very necessary to ask yourself these 6 key questions.

1. **What systems are involved?**
   - **A. Motor:** Anterior horn cell, motor root, peripheral nerves (only motor fibers).
   - **B. Sensory:** Peripheral nerves (only sensory fibers), dorsal root ganglia (conditions that inflict dorsal root ganglion can affect reflexes) , sensory root, dorsal column (central not peripheral).
   - **C. Autonomic:** peripheral nerves**.**
   - **D. Combinations.**

2. **What is the distribution of weakness?**
   - **A. Only distal versus proximal and distal.**
   - **B. Focal/asymmetric versus symmetric:**
     a. Focal \ asymmetric: AHC motor neuron disease, multiple root, plexus, compressive.
     b. Symmetric: Acute & symmetric think about inflammatory diseases CIPD or GBS.

3. **What is the nature of the sensory involvement?**
   - **A. Severe pain/burning or stabbing**
   - **B. Severe proprioceptive loss.** Can also be seen with central spinal cord lesion (dorsal column involvement).

   ✓ Sensory can be divided into small and large fibers (with same 1st order neuron (dorsal root ganglion), and different pathways):
     a. Small fibers: Pain & temperature.
     b. Large: Vibration & joint position.

4. **Is there evidence of upper motor neuron involvement?** Think about ALS (WITHOUT SENSORY LOSS), Myelopathies, **B12 deficiency** (which can cause myelopathy & neuropathy).
   - **A. Without sensory loss.**
   - **B. With sensory loss.**

	Upper motor neuron lesion	Lower motor neuron lesion
Inspection	Normal	Wasting, fasciculation
Tone	Increased with clonus	Normal or decreased, no clonus
Pattern of weakness	Preferentially affects extensors in arms, flexors in leg. Hemiparesis, paraparesis or tetraparesis.	Typically, focal, in distribution of nerve root or peripheral nerve, with associated sensory changes
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor (Babinski sign)	Flexor

5. **What is the temporal evolution?**
   - **A. Acute** (days to 4 weeks)
   - **B. Subacute** (4–8 weeks)

     ✓ **Acute or subacute think about GBS**. (also include vasculitis, and diabetic lumbosacral radiculoplexopathy)
   - **C. Chronic** (>8 weeks): CIPD
   - **D. Is the course monophasic, progressive, or relapsing?** Relapsing course can be present in CIPD.
   - **E. Preceding events, drugs, toxins**

6. **Is there evidence for a hereditary neuropathy?**
   - **A. Family history of neuropathy.**
   - **B. Skeletal deformities.**
   - **C. Lack of sensory symptoms** Very bad sensory loss that they don't mention it & found on examination.

   In patients with a chronic, very slowly progressive distal weakness over many years, with very little in the way of sensory symptoms, the clinician should pay particular attention to the family history and inquire about foot deformities in immediate relatives!

   - Patients with hereditary neuropathy often will present with significant foot drop, with no sensory symptoms, but significant vibration loss in the toes.
   - On examining the patient, the clinician must look carefully at the **feet for arch and toe abnormalities (high or flat arches, hammer toes), and look at the spine for scoliosis.** In suspicious cases, it may be necessary to perform both neurologic and electrophysiologic studies on family members

10 Phenotypic patterns:

الجدول جدا مهم، كل الكلام اللي بالصفحات الماضية كان يهدف الى اننا نعرف المريض يتبع أي باترن؟ بناء على الباترن بنكون مجموعة من الدفرنشيلز

One can classify neuropathic disorders into several patterns based on sensory and motor involvement and the distribution of signs. Each syndrome has a limited differential diagnosis. A final diagnosis is arrived at by using other clues such as the temporal course, presence of other disease states, family history, and information from laboratory studies.

Pattern 1: Symmetric proximal and distal weakness with sensory loss

Consider:

- Inflammatory demyelinating polyneuropathy (GBS and CIDP)

Pattern 2: Symmetric distal sensory loss with or without distal weakness

Consider:

- Cryptogenic sensory polyneuropathy (CSPN)
- Metabolic disorders
- Drugs, toxins
- Hereditary (CMT (Charcot-Marie-Tooth), amyloidosis, and others)

Pattern 3: Asymmetric distal weakness with sensory loss

1. Multiple nerves, consider:
    a. Vasculitis (sudden with pain)
    b. HNPP (Hereditary neuropathy with liability to pressure palsies).
    c. MADSAM neuropathy
    d. Infectious (leprosy, Lyme, sarcoid, HIV)
2. Single nerves/regions, consider:
    a. Compressive mononeuropathy and radiculopathy

Pattern 4: Asymmetric proximal and distal weakness with sensory loss

Consider:

- Polyradiculopathy or plexopathy due to diabetes mellitus, meningeal carcinomatosis or
- lymphomatosis, sarcoidosis, amyloidosis, Lyme, idiopathic, hereditary (HNPP, familial)

Pattern 5: Asymmetric distal weakness without sensory loss

Consider:

1. With upper motor neuron findings:
    a. Motor neuron disease/ALS (Amyotrophic lateral sclerosis) /PLS
2. Without upper motor neuron findings:
    a. PMA (Progressive muscular atrophy):
        i. BAD (brachial amyotrophic diplegia): a slow pure lower motor neuron variant restricted to the arms.
        ii. LAD (Leg amyotrophic diplegia): restricted pure lower motor neuron restricted to the legs.
    b. Multifocal motor neuropath (MMN).
    c. MAMA
    d. Juvenile monomelic amyotrophy

Pattern 6: Symmetric sensory loss and distal areflexia with upper motor neuron findings

Consider:

- B12 deficiency and other causes of combined system degeneration with peripheral neuropathy
- Copper deficiency (including zinc toxicity)
- Inherited disorders (adrenomyeloneuropathy, metachromatic leukodystrophy, Friedreich ataxia)

Pattern 7: Symmetric weakness without sensory loss[a]

Consider:

1. Proximal and distal weakness:
    a. SMA (spinal muscular atrophy).
2. Distal weakness:
    a. Hereditary motor neuropathy

Pattern 8: Focal midline proximal symmetric weakness[a]

Consider:

- Neck extensor weakness (head drop): ALS
- Bulbar weakness (prominent tongue and pharyngeal weakness , dysarthria & dysphagia) : ALS, PLS (Primary lateral sclerosis).

Pattern 9: Asymmetric proprioceptive sensory loss without weakness

Consider:

- Sensory neuronopathy (ganglionopathy).
- CISP (Critical illness polyneuropathy)

Pattern 10: Autonomic symptoms and signs

Consider:

- Neuropathies associated with autonomic dysfunction.

a Overlaps with myopathies and neuromuscular junction disorders.

إذا مره مره ماعندكم وقت ركزوا على هالباترنز، أكثر الأشياء اللي ركز عليهم الدكتور:

1. Proximal weakness + Distal weakness + Symmetric + Sensory symptoms = **inflammatory** (GBS (acute presentation) or CIPD).
2. Proximal weakness + Distal weakness + Asymmetric + Sensory symptoms = **Radiculopathy** (nerve roots supply multiple proximal and distal muscles
    a. C7 nerve root supplies which muscle in the arm? Triceps, extensor digitorum communis (weakness → finger drop).
3. Distal weakness + Symmetric + Sensory symptoms = **Metabolic** (vitamin deficiency), **Diabetic neuropathy**, **Idiopathic**.
    a. length dependent, numbness, and شوية weakness.
4. Distal weakness + Asymmetric + Sensory symptoms = **vasculitis with nerve infarct** (that's why it's asymmetric), **compressive**
5. Upper motor neuron signs? **Cord involvement** (Myelopathy: deficiency or compressive) or motor neuron disease such as **ALS** that gives proximal & distal weakness.
6. Autonomic symptoms = **Autonomic neuropathy**.
7. Severe proprioceptive loss = **dorsal root ganglia** or **spinal cord** (dorsal column).

Entrapment syndrome: الدكتور خلال الشرح شرح كيسز ومنها وحدة فيها النار نوروباثي فمروا هالجزئية

- Focal compression or entrapment is the usual cause of a mononeuropathy.
- Symptoms and signs of entrapment neuropathy are listed in Box 26.102. Entrapment neuropathies may affect anyone, but diabetes, excess alcohol or toxins, or genetic syndromes may be predisposing causes. Unless axonal loss has occurred, entrapment neuropathies will recover, provided the primary cause is removed, either by avoiding the precipitation of activity or by surgical decompression.

26.102 Symptoms and signs in common entrapment neuropathies

Nerve	Symptoms	Muscle weakness/ muscle-wasting	Area of sensory loss
Median (at wrist) (carpal tunnel syndrome)	Pain and paraesthesia on palmar aspect of hands and fingers, waking the patient from sleep. Pain may extend to arm and shoulder	Abductor pollicis brevis	Lateral palm and thumb, index, middle and lateral half 4th finger
Ulnar (at elbow)	Paraesthesia on medial border of hand, wasting and weakness of hand muscles	All small hand muscles, excluding abductor pollicis brevis	Medial palm and little finger, and medial half 4th finger
Radial	Weakness of extension of wrist and fingers, often precipitated by sleeping in abnormal posture, e.g. arm over back of chair	Wrist and finger extensors, supinator	Dorsum of thumb
Common peroneal	Foot drop, trauma to head of fibula	Dorsiflexion and eversion of foot	Nil or dorsum of foot
Lateral cutaneous nerve of the thigh (meralgia paraesthetica)	Tingling and dysaesthesia on lateral border of thigh	Nil	Lateral border of thigh

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Best of luck!

Done by: Shahad Alenezi