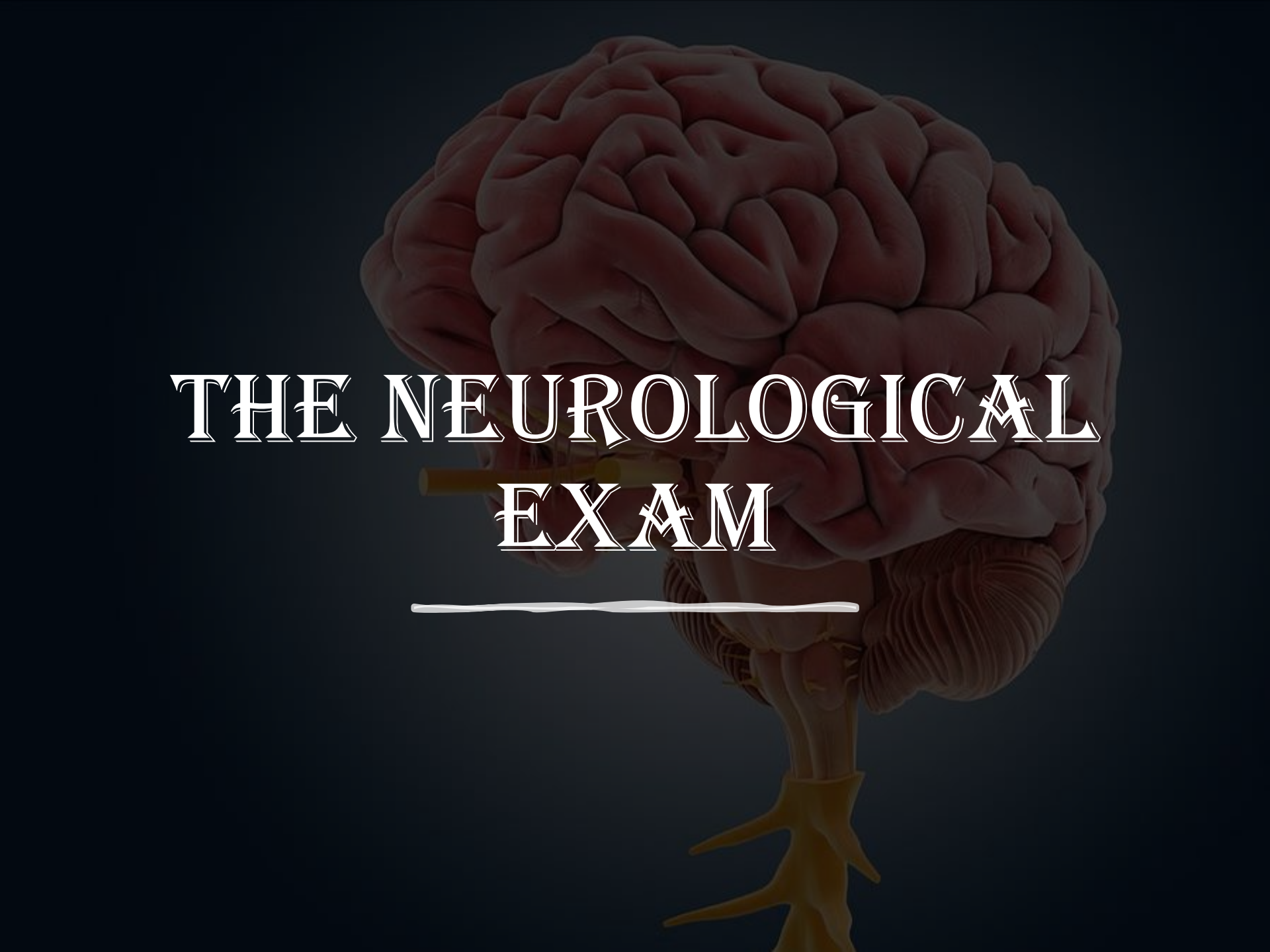
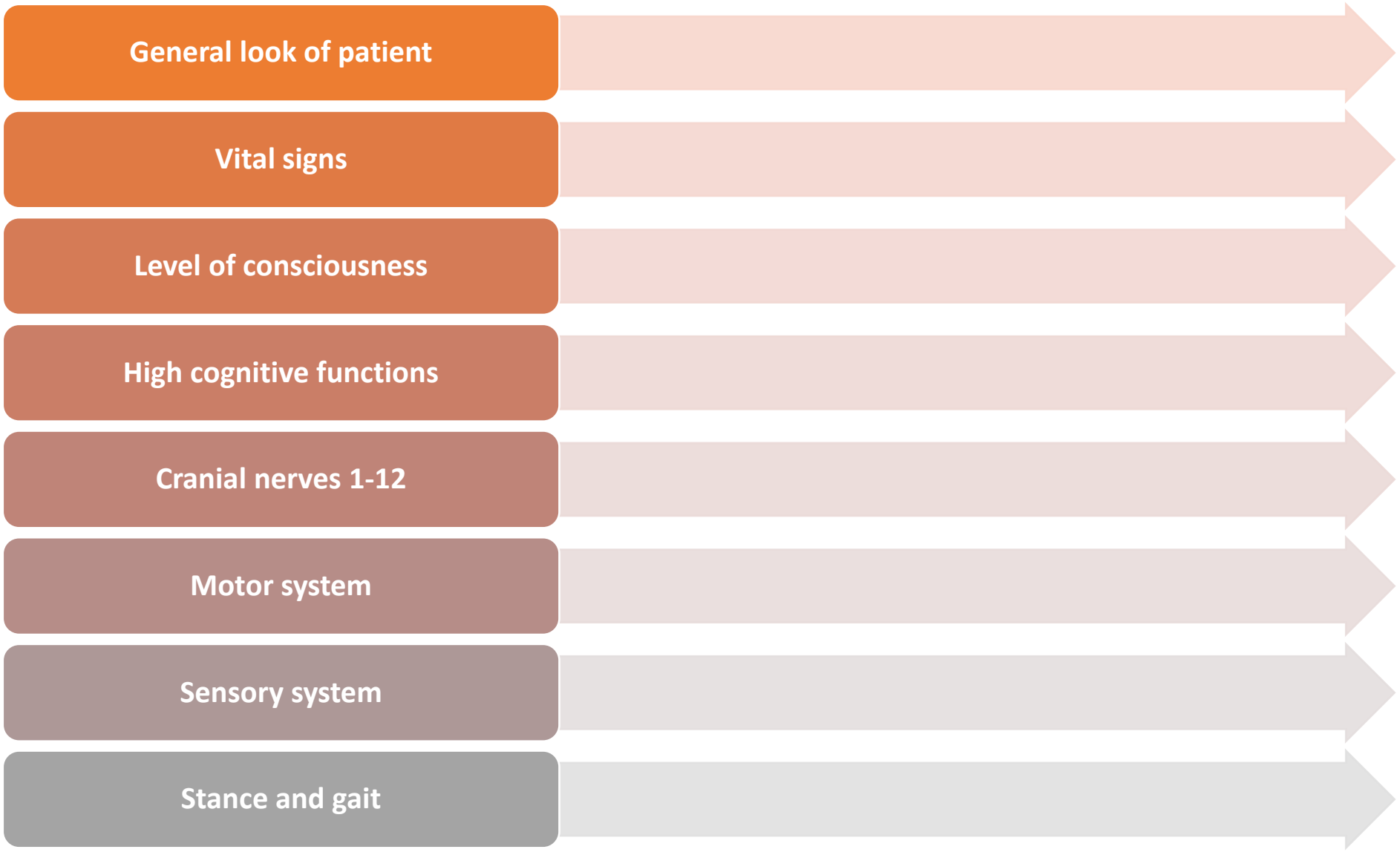


THE NEUROLOGICAL EXAM

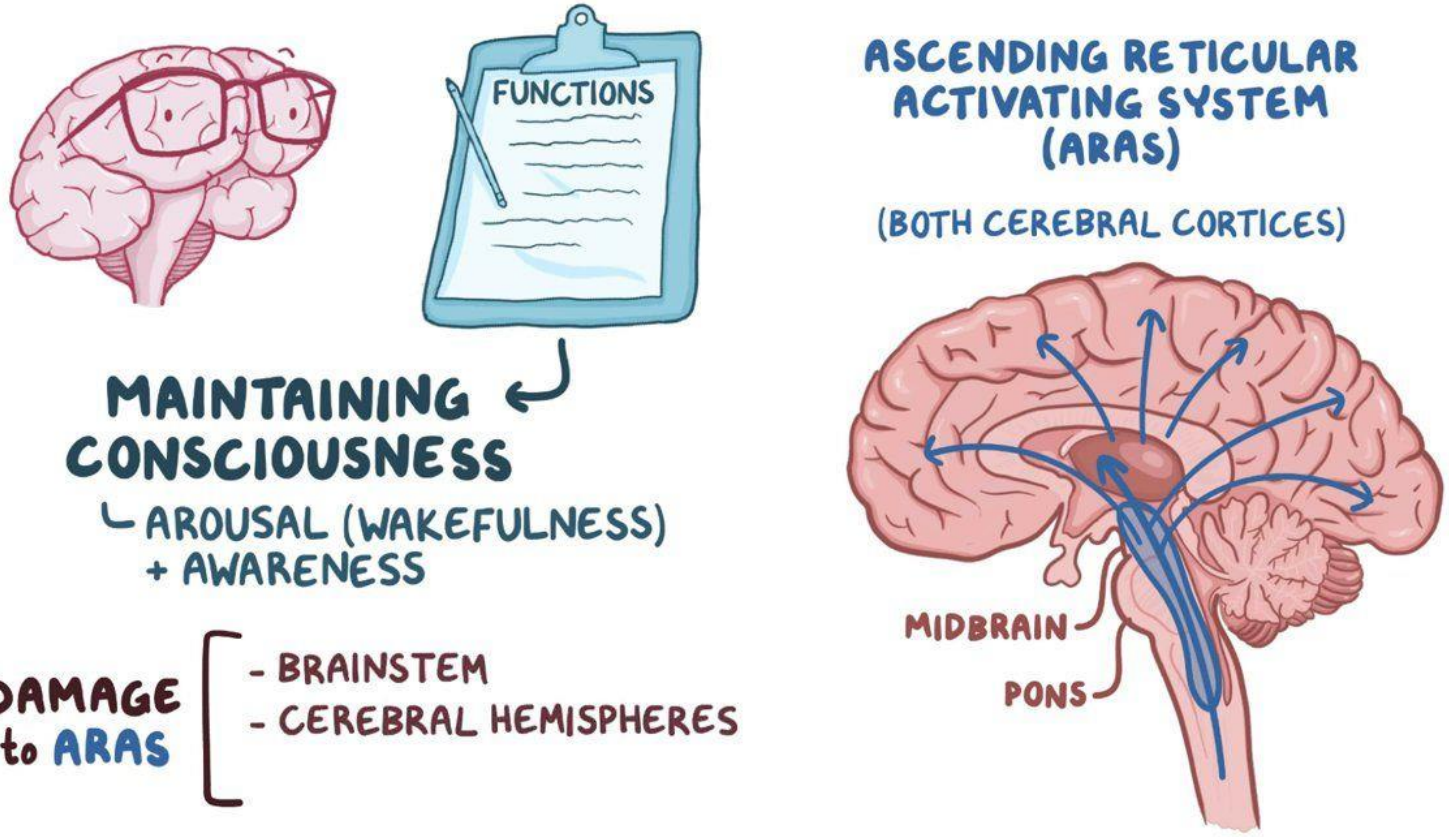
General Approach to Neurological Examination



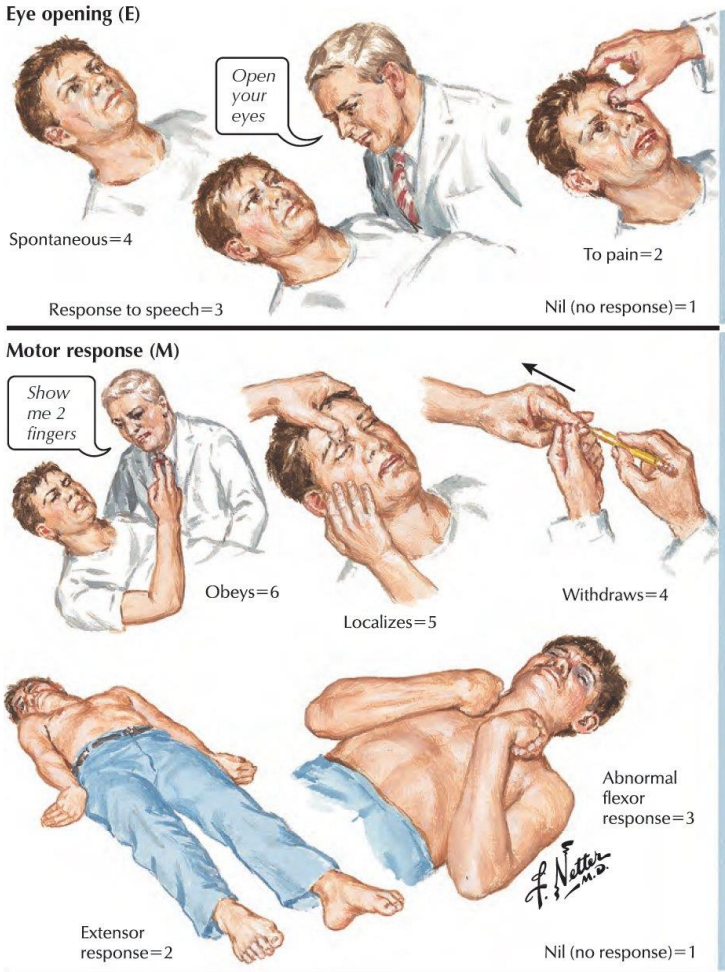
General look of the patient

Begins with your first contact with the patient and continues during history taking!

Assessment Of Consciousness Level



Glasgow Coma Scale



E	
Spontaneous	4
To speech	3
To pain	2
Nil	1

M	
Obeys	6
Localized	6
Withdraws	4
Abnormal flexion	3
Extensor response	2
Nil	6

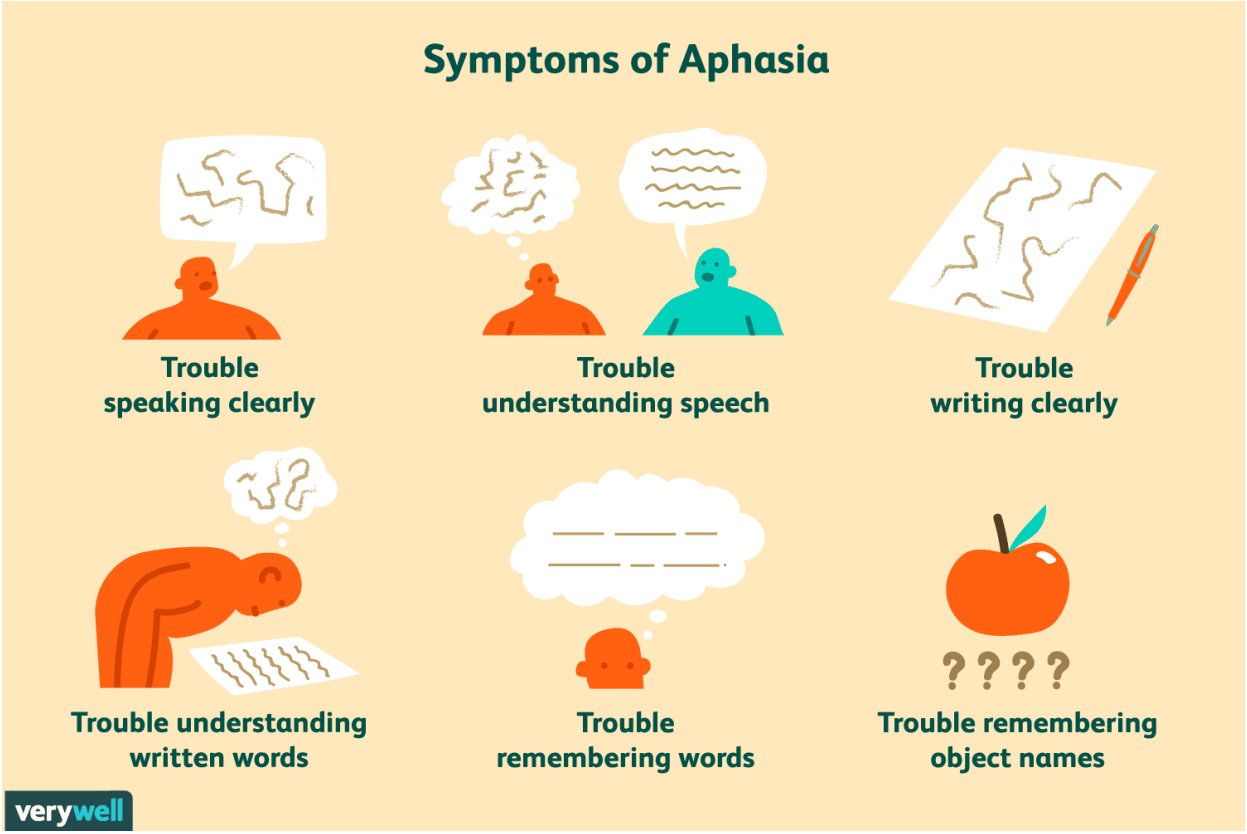
V	
Orented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1

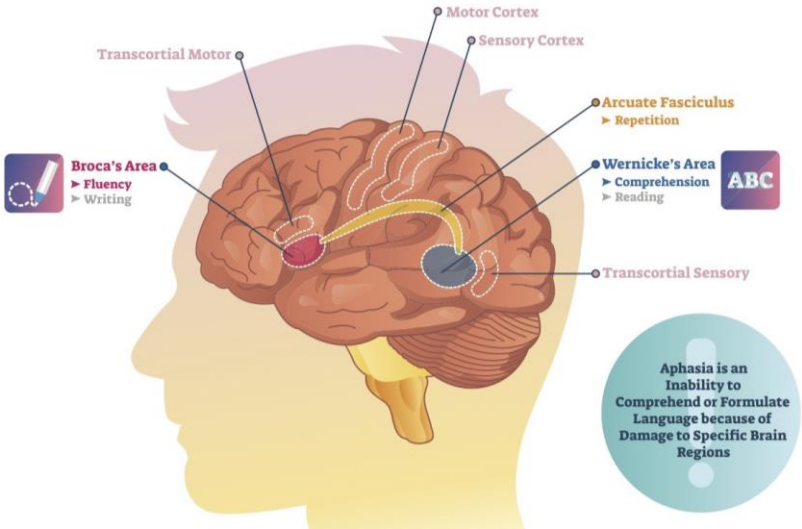
Coma score (E+M+V)=3 to 15

How to calculate a Glasgow Coma Scale (GCS) score.

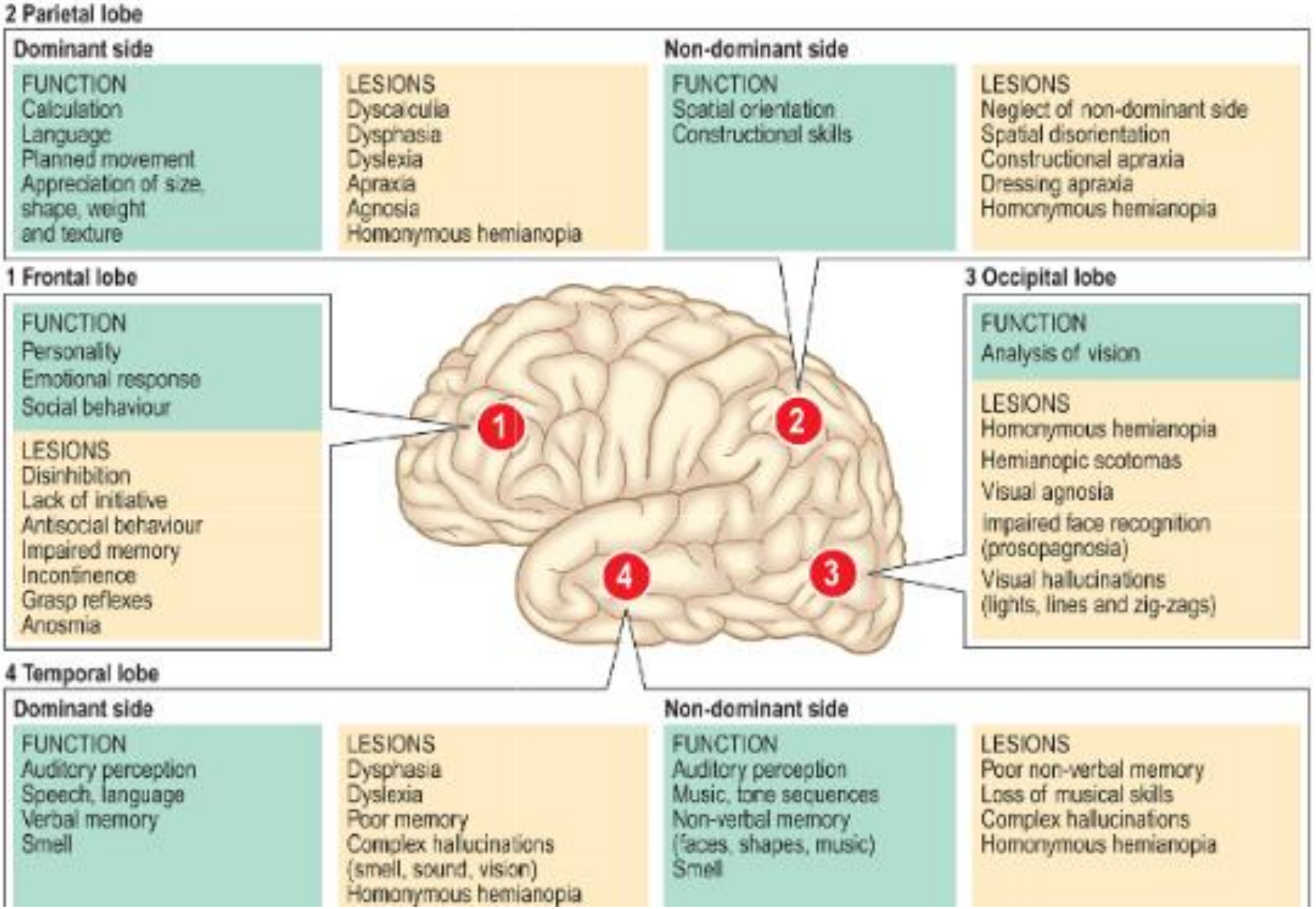
Show me how you talk!

Receptive (Fluent, Wernick’es) vs Expressive (Non fluent, Broca’s) Aphasia

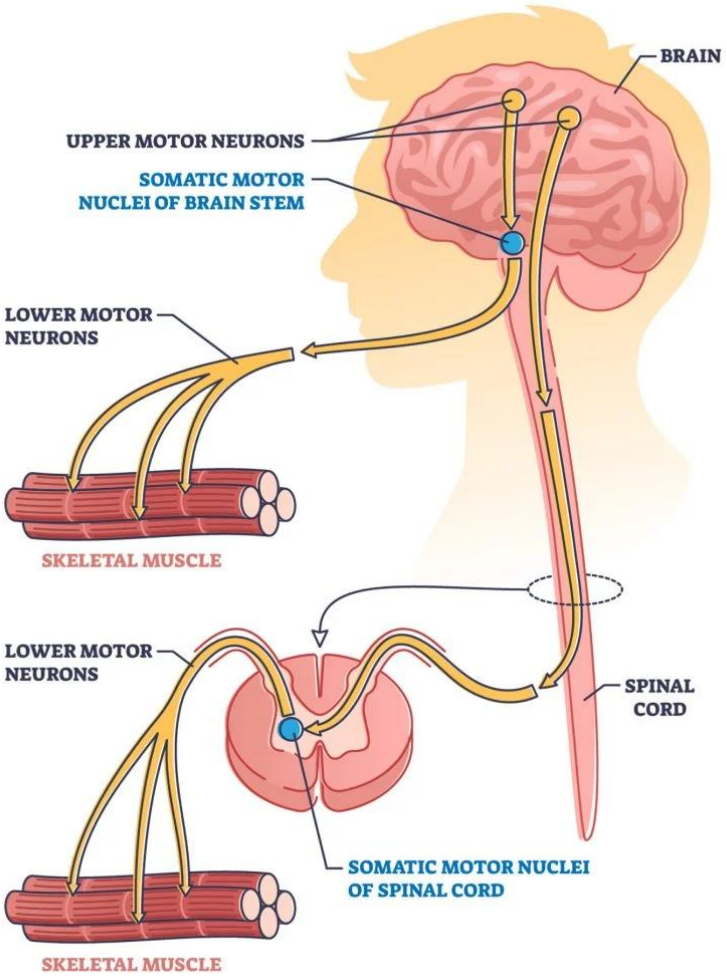


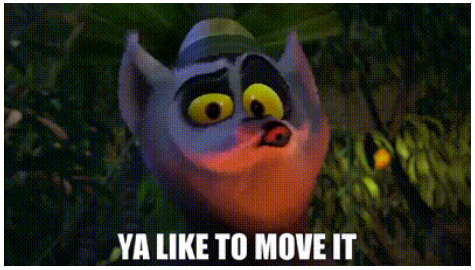


Cortical Function



The motor system





The production of complex yet smoothly coordinated movement is dependent on the integrity of much of the nervous system:

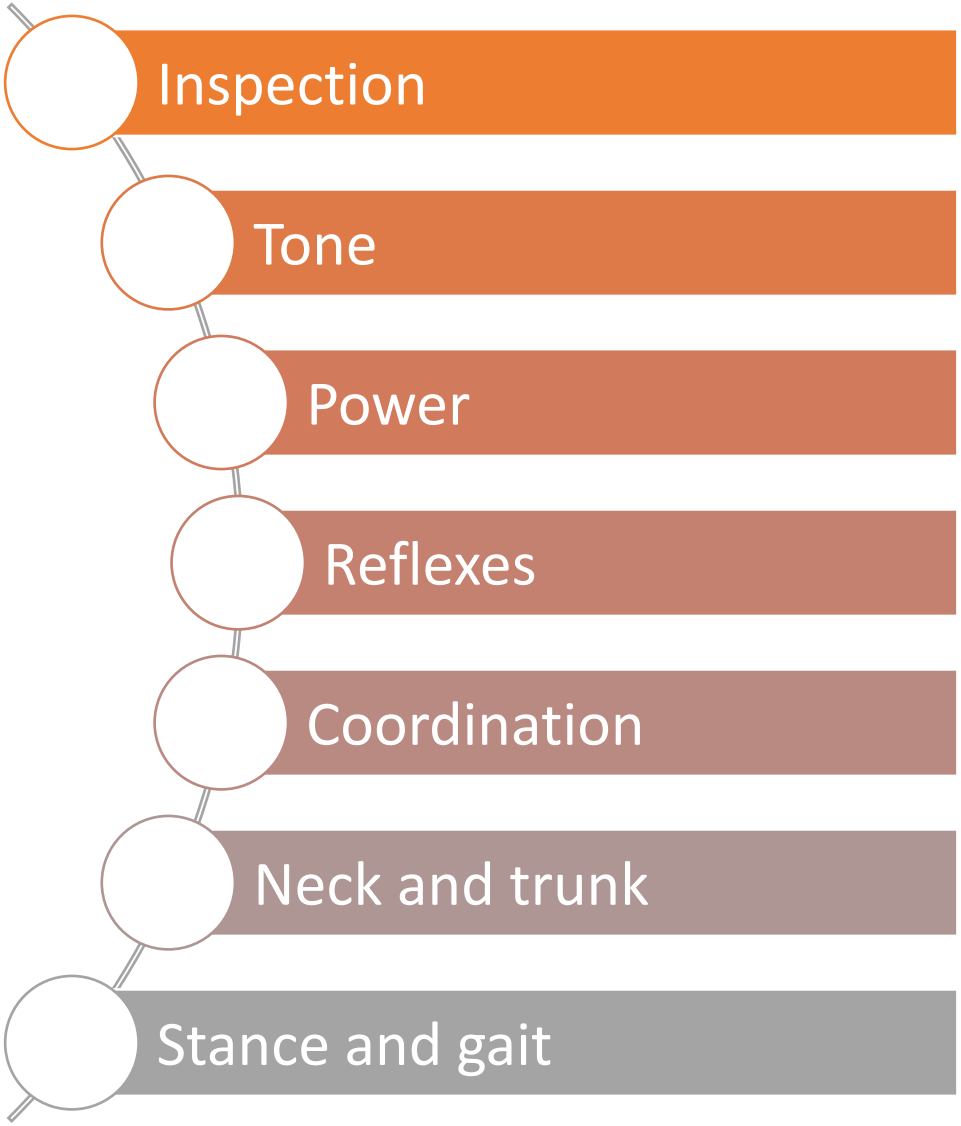
* higher centers
* upper motor neurone(UMN)
* lower motor neurone(LMN)
* neuromuscular junction
* muscle

With important input from :

* basal ganglia-extrapyramidal pathways
* cerebellum

And “ feedback” via sensory pathways, particularly conveying information about joint position

The motor system

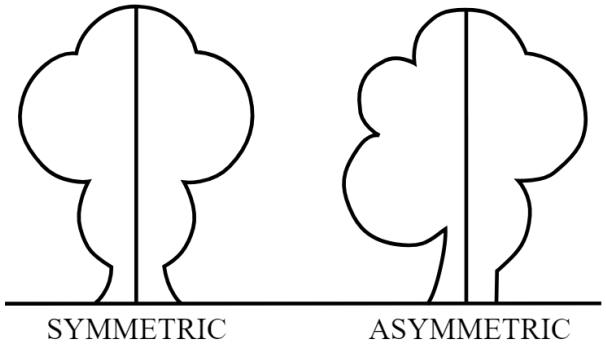


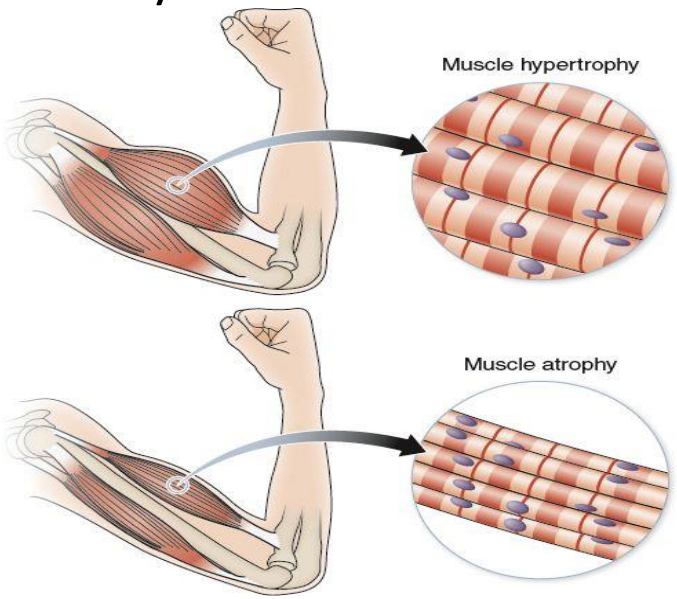
Patterns of abnormality of these aspects, along with information from observing the patient's gait and stance, and from examining for neck and trunk weakness, will generally help to localize a lesion within the motor system

Inspection and palpation of muscles

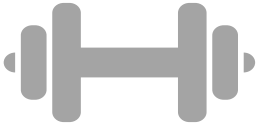
- Completely expose the patient while keeping the patient's comfort and DIGNITY.

1. Look for asymmetry, inspecting both proximally and distally.
2. Note deformities
3. Examine for wasting or hypertrophy
4. fasciculation, and involuntary movement.





Muscle Bulk



Muscle wasting

Lower motor neuron lesions

Loss of muscle bulk is typically less prominent in primary muscle disease(myopathy) than in conditions where muscles have been denervated(neurogenic wasting) as a result of LMN lesions

Wasting is not a feature of UMN lesions, though prolonged disuse may produce some atrophy

Muscle hypertrophy

Physiological muscle hypertrophy e.g. Those body builders!!!

Pseudohypertrophy may occur in muscular dystrophy but the muscles are weak.

The distribution of neurogenic wasting will depend on which LMNs have been damaged, and whether the damage has been at anterior horn cell level, or distally at the spinal roots or individual peripheral nerves

Certain patterns of wasting occur relatively commonly and these areas should be inspected routinely (figure)

Inspection alone is often sufficient to achieve some anatomical localization ; as with other areas of neurology, the examiner should look logically for a feature that discriminates between a limited set of options. A common clinical situation is of a patient presenting with wasting of the intrinsic muscles of one hand

If there is wasting of the thenar eminence (abductor pollicis brevis muscle) only, sparing the small hand muscles, it may indicate a median nerve lesion at the wrist such as carpal tunnel syndrome

If there is wasting of small hand muscles only, sparing the thenar eminence, with claw-hand deformity, it may indicate an ulnar nerve lesion at the elbow

If all hand muscles are wasted, it may indicate a combined median and ulnar nerve injury or a C8T1 radiculopathy or an anterior horn cell lesion

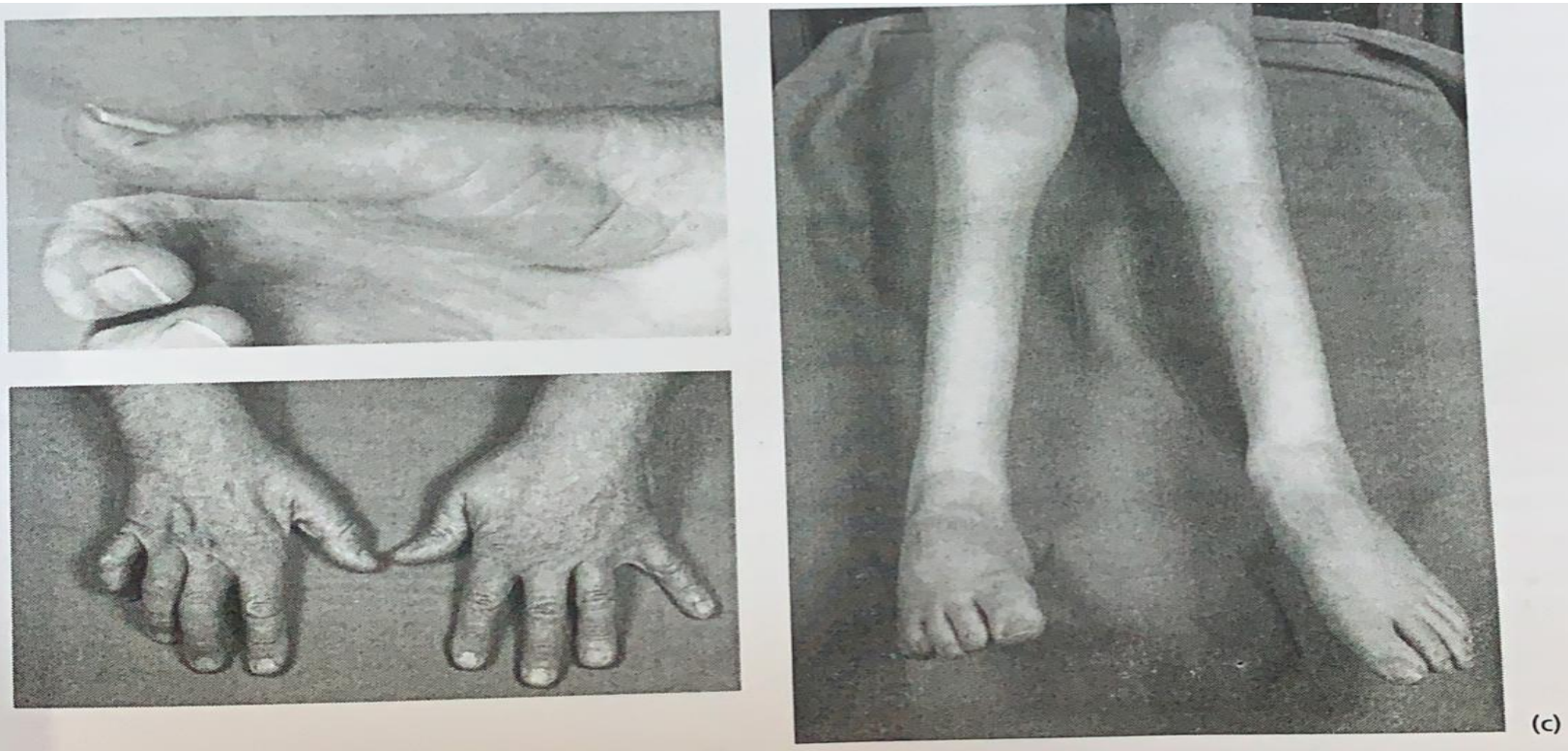


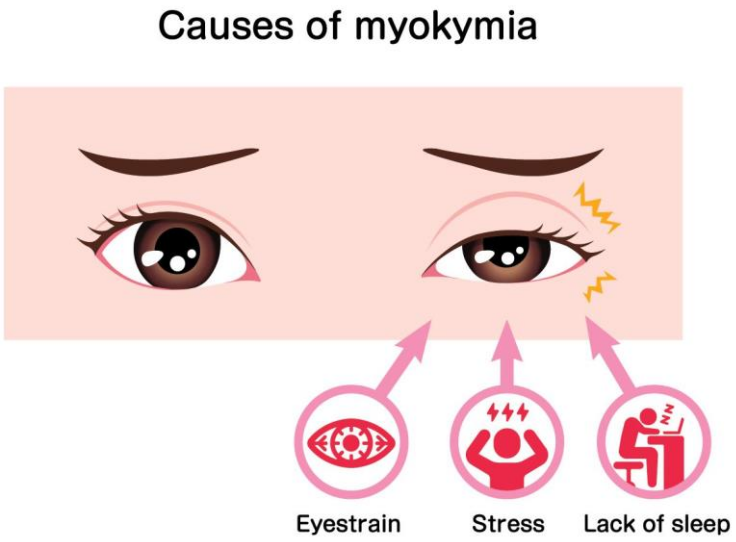
Figure 5.2 Common patterns of neurogenic wasting. (a) Atrophy of the thenar eminence. (b) Wasting of the interossei; the affected right hand (which is also clawed) may be compared with the left, which is normal. (c) Severe distal lower limb wasting. With milder degrees of wasting of tibialis anterior, an early sign is loss of the smooth contour of the shin, the anterior border of the tibia becoming more prominent. Upper and lower limbs should also be inspected for more proximal wasting (particularly the periscapular and thigh muscles).

Abnormal Movements

- Fasciculation
- Myoclonic jerks
- Tremor
- Dystonia
- Chorea
- Athetosis
- Ballism
- Tics

Fasciculation

- Irregular twitches under the skin overlying resting muscles caused by individual motor units firing spontaneously
- Occurs in LMN lesions, generally proximal and severe , especially at anterior horn cell level ,usually in wasted muscles.
- Seen, not felt.
- Physiologically is common, especially in the calves
- Myokymia: eyelid or first dorsal interosseus and is rarely pathological



Myoclonic jerks

- These are sudden shock-like contractions of one or more muscles that may be focal or diffuse and occur singly or repetitively.

- Healthy people commonly experience these when falling asleep (hypnic jerks).

- They may also occur pathologically in association with epilepsy, diffuse brain damage and dementia.

Tremor

- Tremor is an oscillatory movement about a joint or a group of joints resulting from alternating contraction and relaxation of muscles.

- Classified according to their frequency, amplitude, position, and body part affected.

INTENTION TREMOR

* INVOLUNTARY TREMORS during INTENTIONAL MOVEMENT
* MOST COMMONLY AFFECTS LIMBS (especially UPPER) and SPEECH MUSCLES

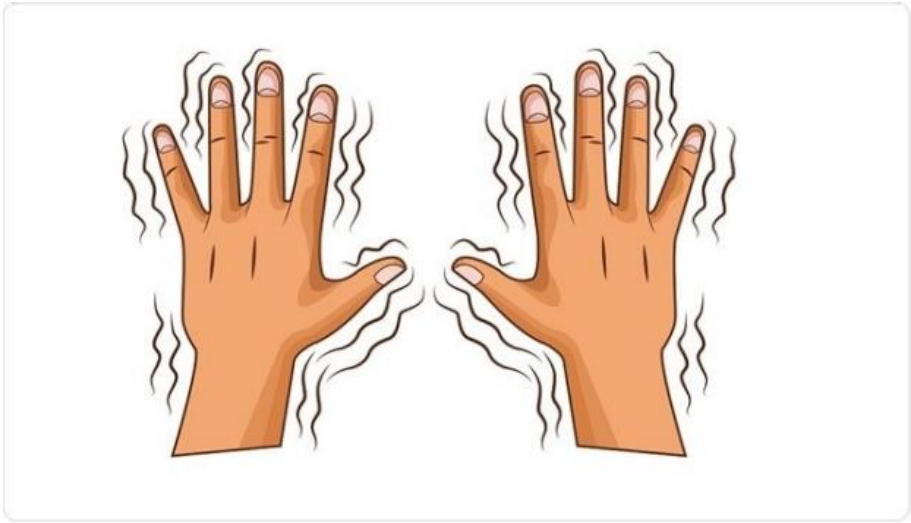
ESSENTIAL TREMOR

* CAN OCCUR without INTENTIONAL MOVEMENT
* CAN BE WORSENED with ANXIETY or INTENTIONAL MOVEMENT

PARKINSONIAN TREMOR

* MORE EVIDENT at REST
* ASSOCIATED with PARKINSON DISEASE

- Fine, fast postural tremor:
  - Physiological tremor seen with anxiety
  - Hyperthyroidism
  - Excess alcohol or caffeine intake
  - Adverse effect of β-agonist



- Essential tremor :
  - The most common pathological cause of action tremor
  - AD pattern of inheritance
  - Affecting the upper limbs and head
  - With postural and action components
  - It may be improved by alcohol

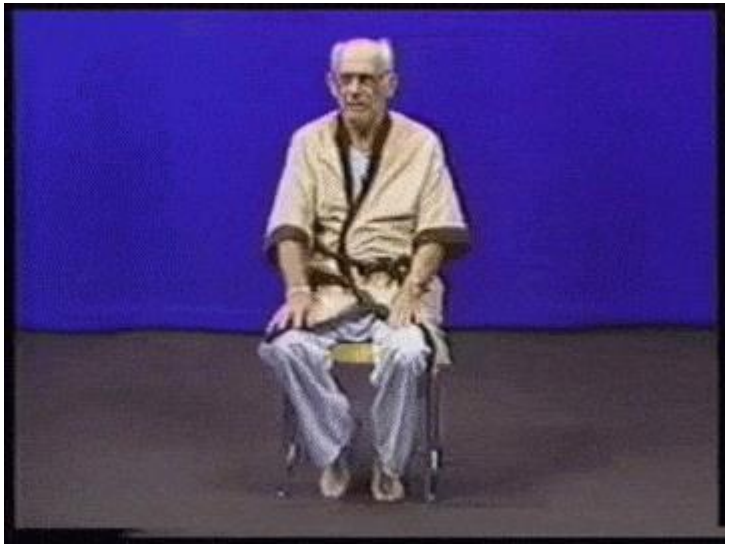
- Parkinson's disease
  - slow, coarse tremor
  - worse at rest but reduced with voluntary movement
  - Mc in the upper limbs, usually asymmetrical

- Cerebellar damage:
  - Intention tremor is absent at rest but maximal during movement
  - finger-to-nose test

- Functional tremors:
  - inconsistent
  - varying frequencies and amplitudes
  - may be associated with other signs

- Dystonia
  - caused by sustained muscle contractions, leading to twisting, repetitive movements, and sometimes tremor.
- Chorea
  - brief, random, purposeless movements which may affect various body parts, but commonly the arms.
- Athetosis
  - slower, writhing movement, more similar to dystonia than chorea.
- Ballism
  - refers to violent flinging movements sometimes affecting only one side of the body (hemiballismus).
- Tics
  - repetitive, stereotyped movements which can be briefly suppressed by the patient.


spasmodic torticolis



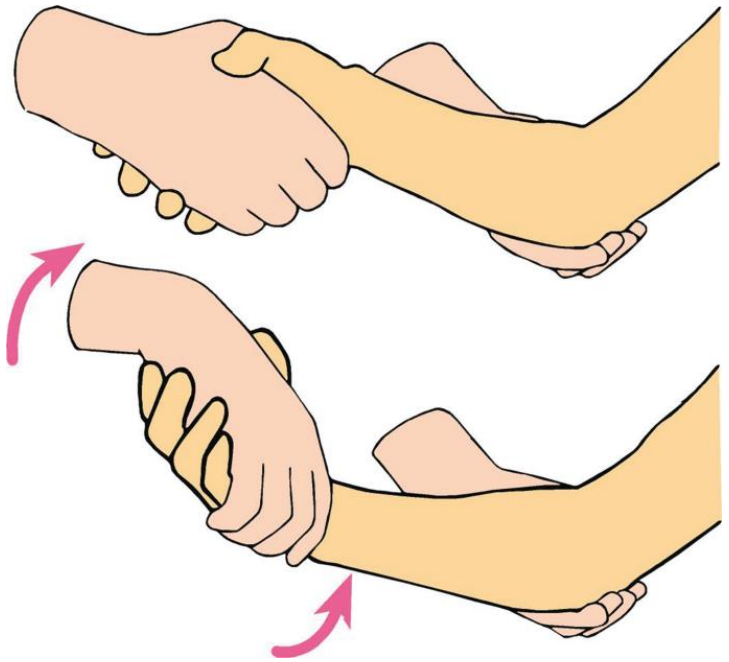
Assessment of tone

Tone is the resistance felt by the examiner when moving a joint passively.

- Ask the patient to lie supine and relax.
- Any pain or limitation?
- Passively move each joint tested through as full a range as possible, both slowly and quickly in all anatomically possible directions.
- Be unpredictable, both in direction and speed, why?

- **Upper limb**
  - **Hold the patient's hand as if shaking hands, using your other hand to support his elbow. Assess the tone at the wrist and elbow.**

- **Activation**
  - **a technique used to exaggerate subtle increase in tone and is particularly useful for assessing extrapyramidal tone increase.**
  - **Ask the patient to describe circles in the air with the contralateral limb while assessing tone. A transient increase in tone with this maneuver is normal.**



- ***Lower limb***
  - Roll the leg from side to side, then briskly lift the knee into a flexed position, observing the movement of the foot .
  - Typically the heel moves up the bed, but increased tone may cause it to lift off the bed due to failure of relaxation.

- ***Ankle clonus***
  - Support the patient's leg, with both the knee and ankle resting in 90° flexion.
  - Briskly dorsiflex and partially evert the foot, sustaining the pressure.
  - Clonus is felt as repeated beats of dorsiflexion/plantar flexion.

The phenomenon of muscle tone and many other physical signs of motor function depend on the integrity of the stretch reflex(figure)

Passive stretch of a muscle induces afferent impulses to the spinal cord, which in turn activate the motor neurone, leading to reflex contraction

As the clinical correlate of this response is normal muscle tone , it follows that interruption of the reflex arc by disease, for example by LMN damage, will lead to a reduction in tone or hypotonia- the muscle will become flaccid

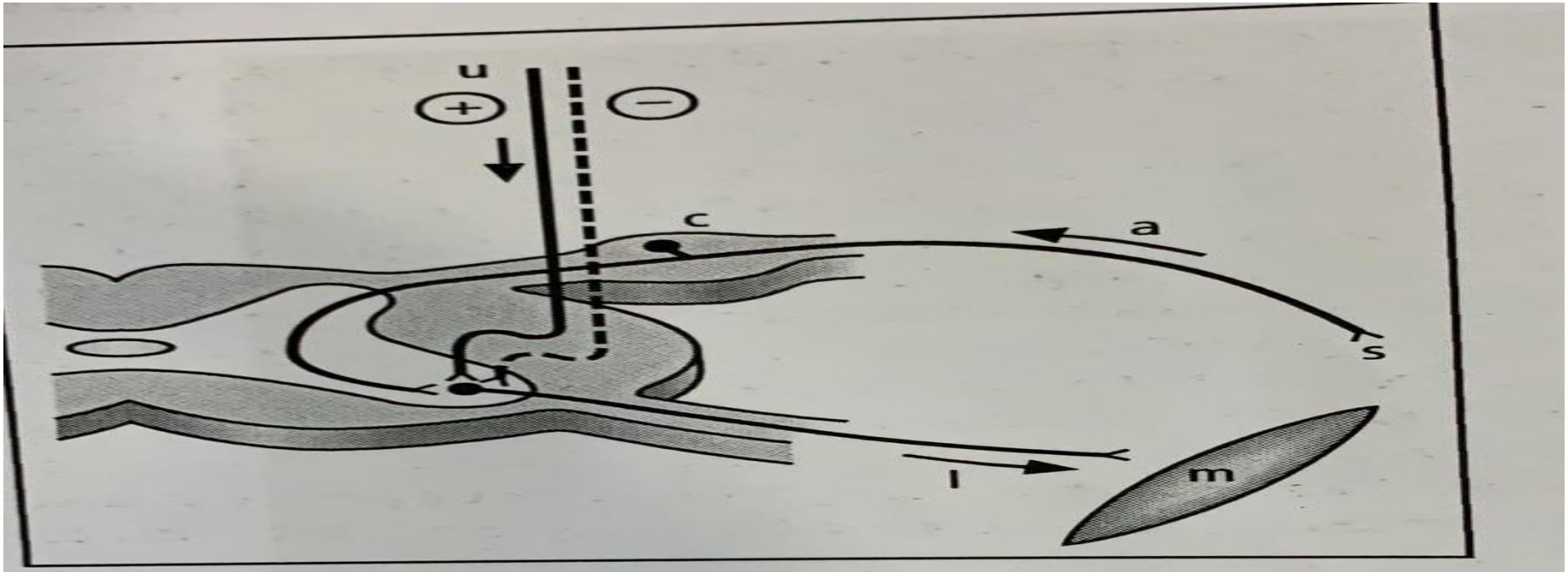


Figure 5.4 The stretch reflex. s, stretch receptor; a, afferent (sensory) neurone; c, cell body of sensory neurone in dorsal root ganglion; l, LMN originating at anterior horn cell of spinal cord; m, muscle; u, UMN; arrows indicate direction of impulse traffic; +, excitatory impulse in UMN; −, inhibitory impulse in parallel descending inhibitory pathways. Not all the components of the reflex are shown. The descending inhibitory pathways mainly act on the gamma efferents (not shown) which modulate the sensitivity of the stretch receptors.

Disease affecting the UMN in turn produces hypertonia or spasticity

The reason for this is not so much damage to the excitatory UMN itself but rather dysfunction of the polysynaptic pathways descending in parallel with it, which exert an inhibitory effect on the LMN and hence on the reflex arc

Loss of supraspinal inhibition unmasks the stretch reflex in a more primitive or 'undamped' form, and tone is thereby increased

The characteristic quality of hypertonia caused by UMN damage is that there is marked resistance to passive muscle stretch through part of the range of movement of a joint, but at a certain point the resistance suddenly ‘gives’(clasp- knife phenomenon)

In some patients with subtle UMN lesions,the only feature of such a lesion in the upper limbs may be a miniature version of the clasp- knife effect, elicited by supinating and pronating the forearm(supinator catch)

Tone Abnormalities

- Hypotonia
  - LMN: muscle wasting, weakness, and hyporeflexia.
  - In the early phases of cerebral or spinal shock
- Hypertonia
  1. Spasticity:
     - velocity-dependent resistance, with quick movements
     - In mild forms: 'catch' at the beginning or end of passive movement.
     - In severe cases: it limits the range of movement and may be associated with contracture.
     - In the upper limbs : on extension; in the legs: on flexion.
  2. Rigidity
     - Sustained resistance throughout the range of movement , with slow movement
     - In parkinsonism this is classically described as 'lead pipe rigidity'. In the presence of a Parkinsonian tremor there may be a regular interruption to the movement, giving it a jerky feel ('cog wheeling').
- Clonus
  - Rhythmic series of contractions evoked by a sudden stretch of the muscle and tendon.
  - Unsustained (<6 beats) clonus may be physiological.
  - When sustained, it indicates UMN damage


MakeAGIF.com


Relax
Lead-pipe rigidity
Cog-wheel rigidity
Spasticity
Little resistance
Uniform resistance
On-and-off resistance
High to low resistance
a
b
c
d

Testing movement and power

1. Any pain?
2. Assess pronator drift
3. Assess whether he can overcome gravity first
4. Then apply resistance to this movement testing across a single joint

Pronator drift

Another sign of mild UMN damage may be observed with the patient's arms outstretched, palms facing upwards and eyes shut

An affected limb will first pronate then drift downwards(pronator or pyramidal drift sign)

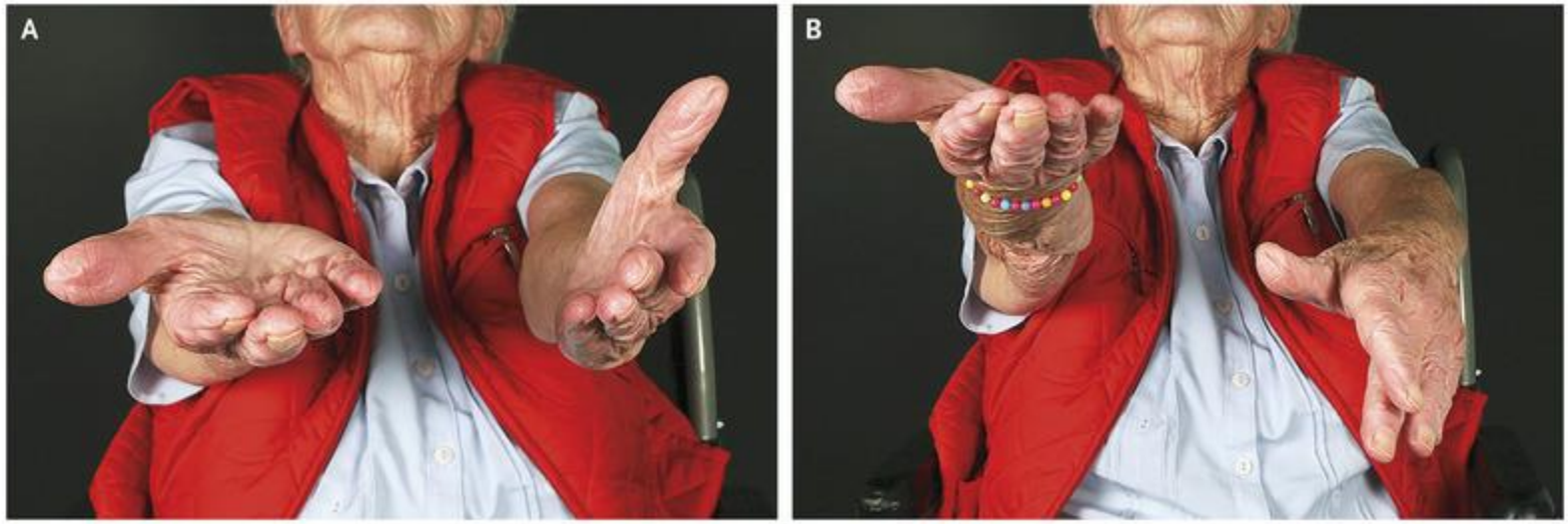
Disease of the other parts of the nervous system may also be identified by asking the patient to perform this simple maneuver

For example, a patient with loss of joint position sense in the hands may show irregular involuntary movements of the fingers when the arm is outstretched and the eyes are shut ('pseudoathetosis'), because of loss of all avenues of sensory input relating to the maintenance of this posture(deafferentation)

Pronator drift

- Observe the patient with his arms outstretched and supinated (palms up) and eyes closed for ‘pronator drift’, when one arm starts to pronate

- It is an early feature of upper motor neuron lesions, and it has good sensitivity and specificity



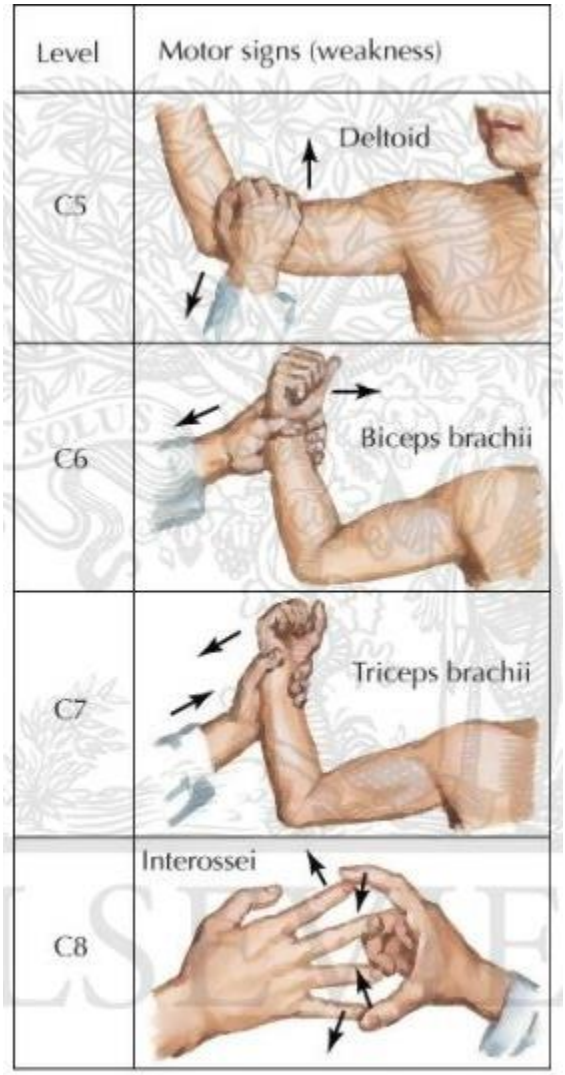
Truncal strength

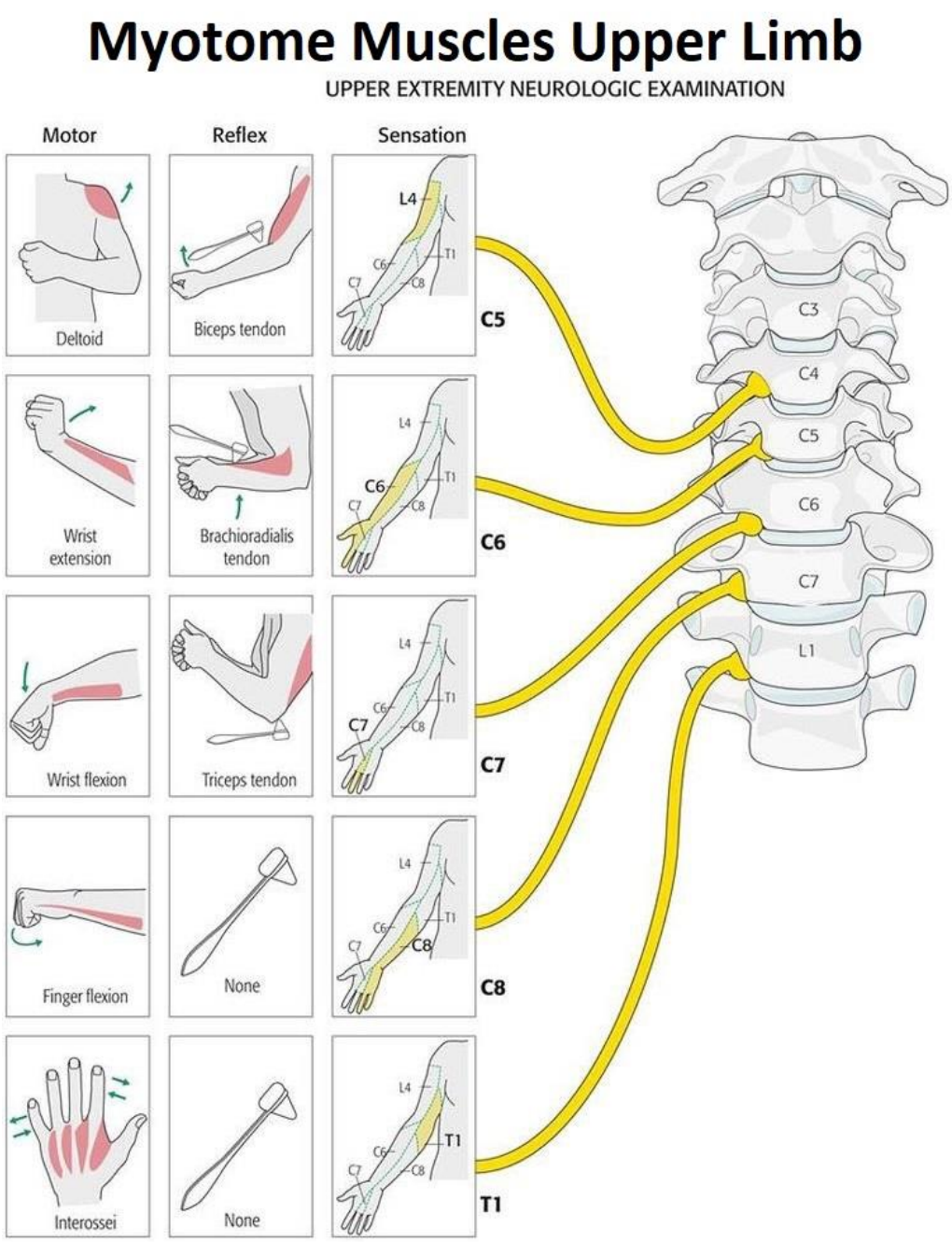
- Sit up from the lying position, or rise from a chair, without using the arms.

Commonly tested movements in upper limb

Movement	Muscle	Nerve / Root
Shoulder abduction	deltoid	Axillary C5
Elbow flexion	Biceps Brachioradialis	Musculocutaneous C5,6 Radial C6
Elbow extention	Triceps	Radial C7
Wrist extension	Extensor carbi radialis longus	Posterior interosseous nerve (radial) C6
Finger flexion	Flexor pollicis longus Flexor digitorum profundus	Anterior interosseous (median) C8 Ulnar C8
Finger extension	Extensor digitorum communis	Posterior interosseous nerve (radial) C7
Finger abduction	First dorsal interosseous	Ulnar T1
Thumb abduction	Abductor pollicis previs	Median T1

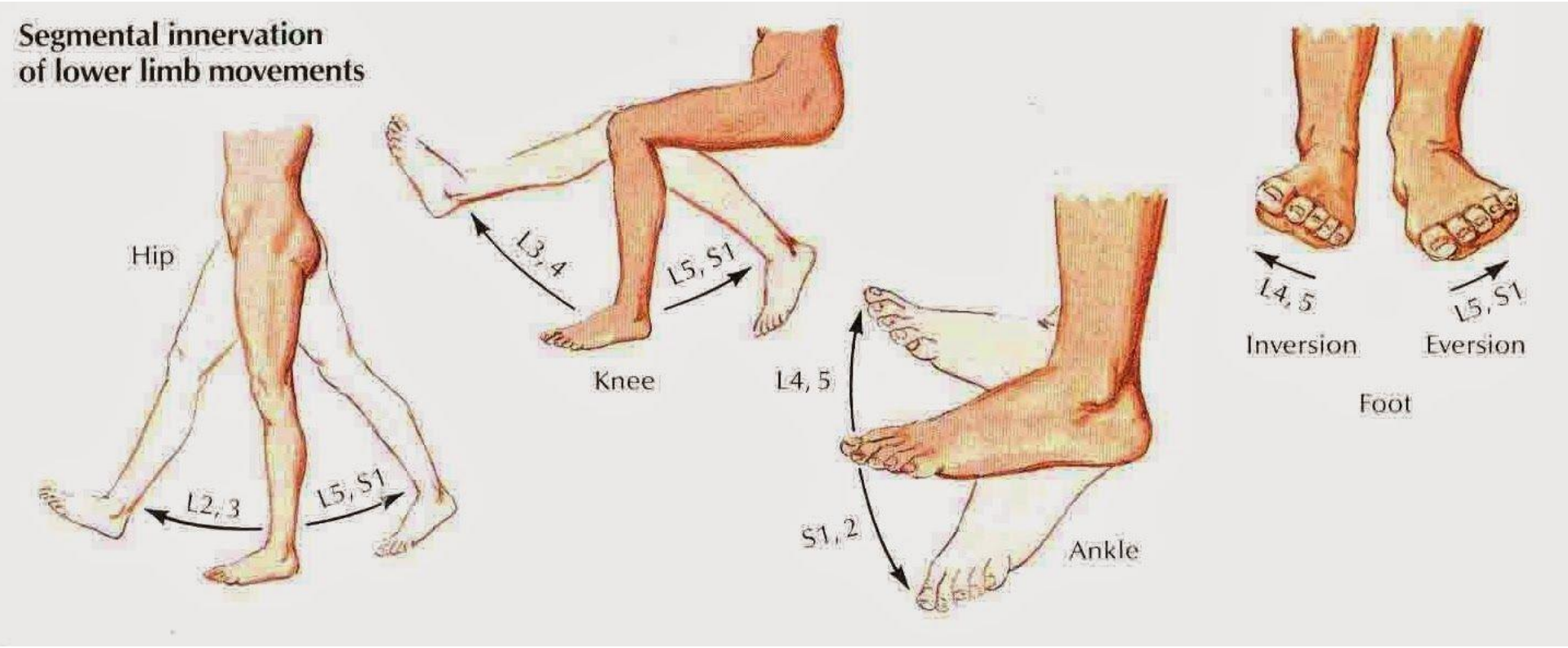
Commonly tested movements in the upper limb

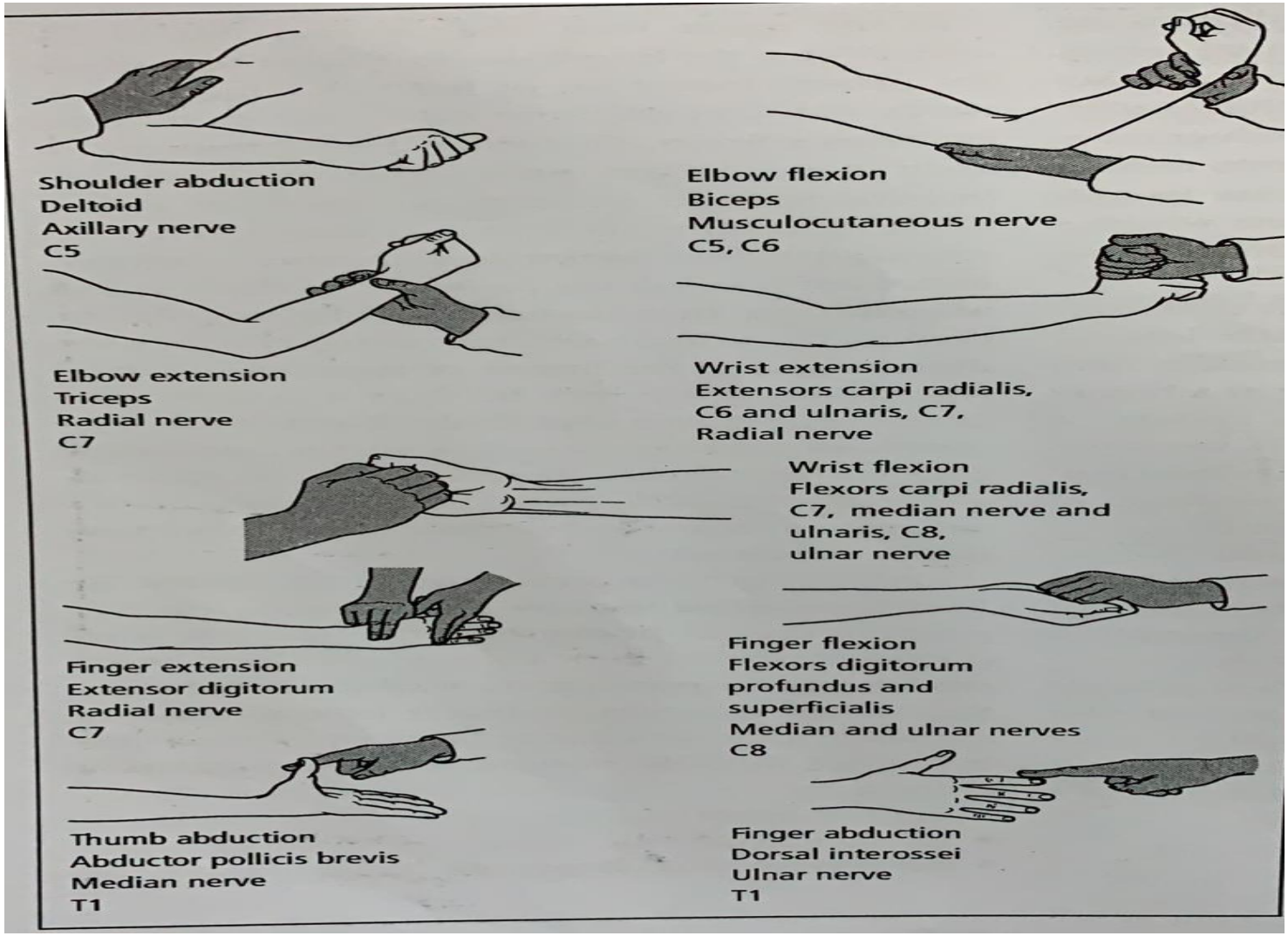




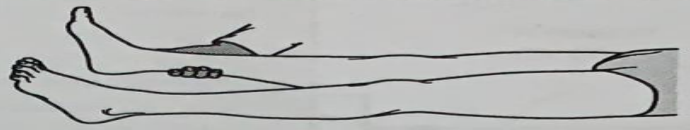
Commonly tested movements in lower limb

Movement	Muscle	Nerve / Root
Hip flexion	iliopsoas	Femoral L2- L3
Hip extension	Gluteus maximus	Sciatic L5,S1
Knee flexion	Hamstrings	Sciatic S1
Knee extention	Quadriceps Femoris	Femoral L3,L4
Ankle dorsiflexion	Tibialis anterior	Deep peroneal L4,5
Ankle plantar flexion	Gastrocnemius and soleus	Tibial nerve S1,2
Great toe extension	Extensor hallucis longus	Deep peroneal L5
Ankle eversion	Peroneus	Superficial peroneal L5,S1
Ankle inversion	tibialis posterior	Tibial nerve L4,5


Segmental innervation
of lower limb movements
Hip
L2, 3
L5, S1
L3, 4
L5, S1
Knee
L4, 5
S1, 2
Ankle
L4, 5
L5, S1
Inversion
Eversion
Foot


Shoulder abduction
Deltoid
Axillary nerve
C5
Elbow flexion
Biceps
Musculocutaneous nerve
C5, C6
Elbow extension
Triceps
Radial nerve
C7
Wrist extension
Extensors carpi radialis,
C6 and ulnaris, C7,
Radial nerve
Wrist flexion
Flexors carpi radialis,
C7, median nerve and
ulnaris, C8,
ulnar nerve
Finger extension
Extensor digitorum
Radial nerve
C7
Finger flexion
Flexors digitorum
profundus and
superficialis
Median and ulnar nerves
C8
Thumb abduction
Abductor pollicis brevis
Median nerve
T1
Finger abduction
Dorsal interossei
Ulnar nerve
T1

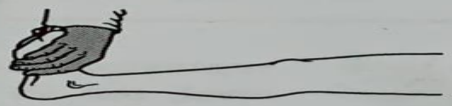
Hip flexion
Iliopsoas
Lumbar plexus and
femoral nerve
L1/L2



Hip extension
Gluteus maximus
Inferior gluteal nerve
L5, S1, S2

Knee flexion
hamstrings
Sciatic nerve
L5, S1, S2

Knee extension
Quadriceps femoris
Femoral nerve
L3, L4



Ankle dorsiflexion
Tibialis anterior
Deep peroneal nerve
L4, L5

Ankle plantar flexion
Gastrocnemius and soleus
Sciatic nerve
S1, S2

Dorsiflexion of great toe
Extensor hallucis longus
Deep peroneal nerve
L5

Ankle inversion
Tibialis posterior
Tibial nerve
L4, L5

Ankle eversion
Peronei
Superficial peroneal nerve
L5, S1

How to scale muscle power?

11.18 Medical Research Council scale for muscle power

0	No muscle contraction visible
1	Flicker of contraction but no movement
2	Joint movement when effect of gravity eliminated
3	Movement against gravity but not against examiner's resistance
4	Movement against resistance but weaker than normal
5	Normal power

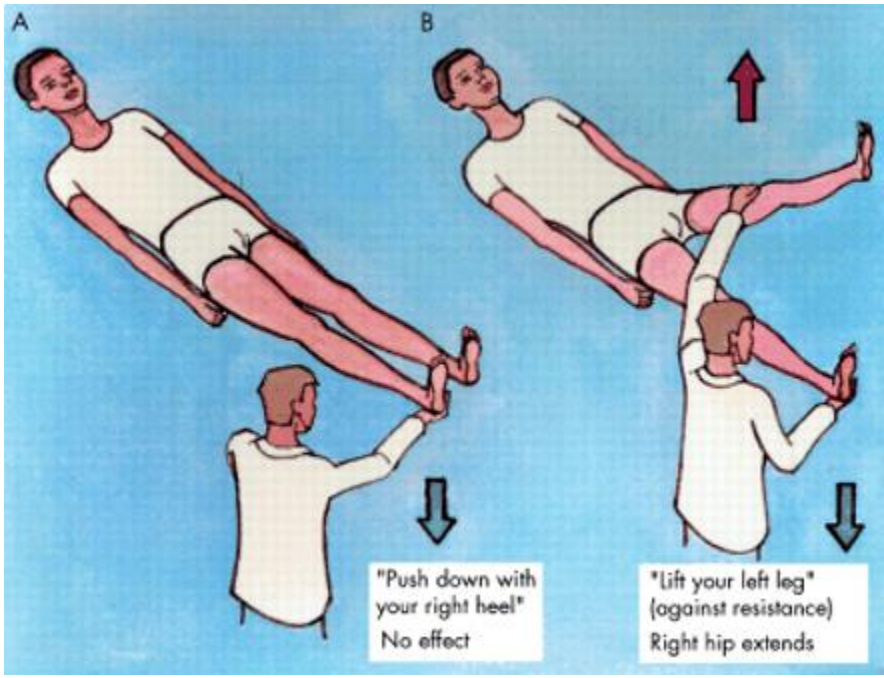
The scale is at best semiquantitative, particularly as much muscle weakness(paresis) in clinical neurology falls within 3-5 range, where it is often necessary to make further subjective subdivisions i.e 4-, 4 and 4+, denoting severe, moderate and mild weakness respectively

For most 'screening' examinations, it is sufficient to test an agonist-antagonist muscle pair at each of the major joints(Figure)

Right – and left-sided limbs should be compared at each joint because weakness is often asymmetrical and patients may therefore act as their own controls

Hints!

- Weakness of a relatively large group of muscles >>> UMN
- Paresis of an individual and specific muscle >>> LMN
- You need only to show that the patient can achieve maximum power briefly

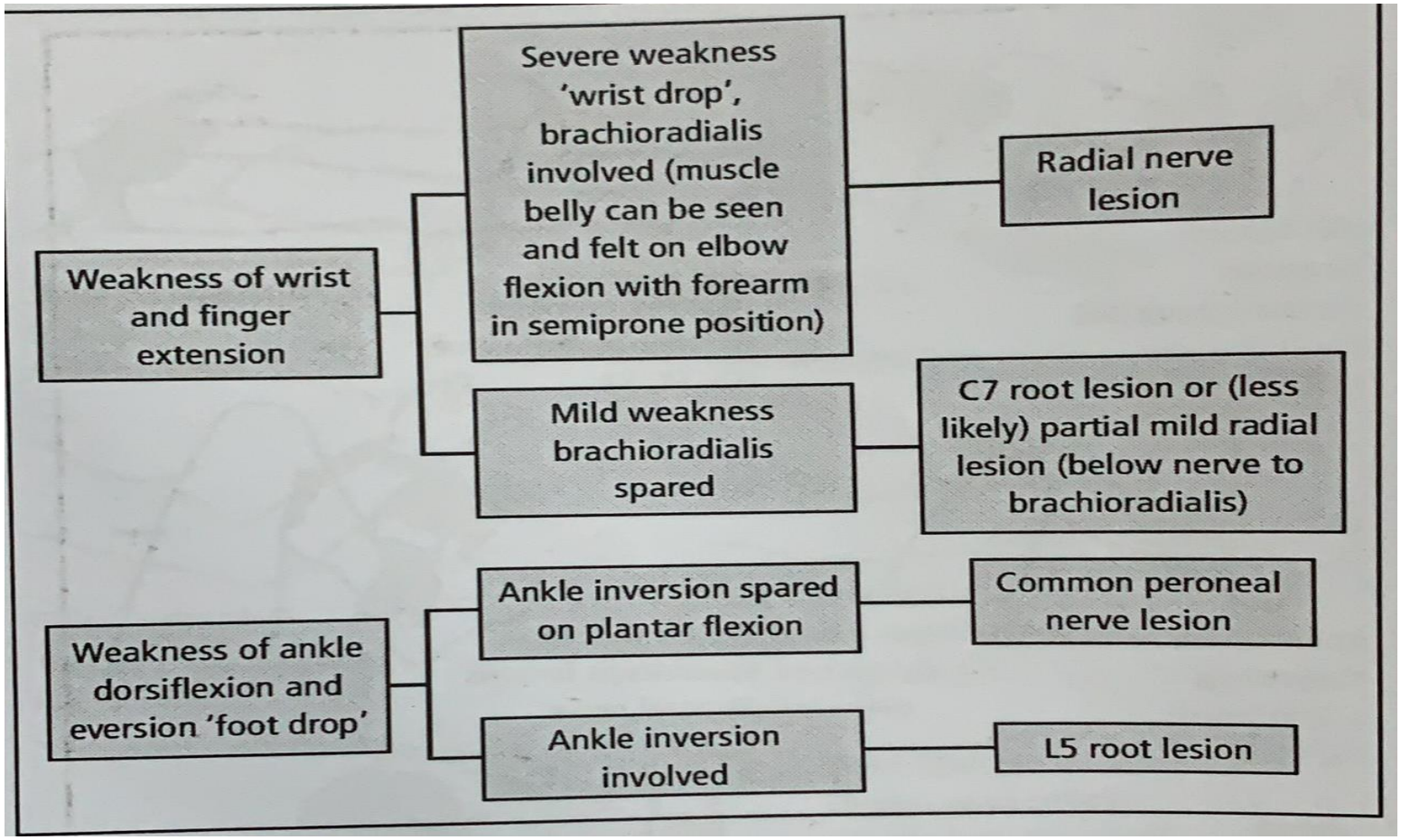


Although it is possible to assess power exhaustively in many other limb muscles, selection is required

This is entirely governed by information already available from the history, or from other parts of the examination, whereby a particular pattern of focal weakness may have suggested itself

Anatomical localization is then achieved once again by discriminating between very few options as shown for the very common clinical problems of wrist and foot drop(Figure)

Likewise,the history may have pointed to a lesion of an individual cervical spine segment(neck pain radiating down one arm), again a very common clinical situation


Weakness of wrist and finger extension
Severe weakness 'wrist drop', brachioradialis involved (muscle belly can be seen and felt on elbow flexion with forearm in semiprone position)
Radial nerve lesion
Mild weakness brachioradialis spared
C7 root lesion or (less likely) partial mild radial lesion (below nerve to brachioradialis)
Weakness of ankle dorsiflexion and eversion 'foot drop'
Ankle inversion spared on plantar flexion
Common peroneal nerve lesion
Ankle inversion involved
L5 root lesion

In this case, the aim is to detect a pattern of weakness corresponding to the muscle innervated by a single segmental nerve, its myotome

(table)

More diffuse processes affecting many nerves or muscles simultaneously, e.g. metabolic or inflammatory, may produce more generalized weakness, but specific patterns remain discernible

Thus , primary muscle disease is typically associated with proximal weakness whereas a motor polyneuropathy usually produces distal weakness

C5
Most shoulder movements, e.g. abduction
Biceps

C6
Brachioradialis
Extensor carpi radialis longus (extension and abduction at wrist)

C7
Triceps
Extensor carpi ulnaris (extension and adduction at wrist)
Finger extension

C8
Wrist flexion (and adduction)
Finger flexion

T1
Intrinsic muscles of hand

* Most of these muscles are innervated by fibres from more than one root, e.g. the 'root value' of brachioradialis is in fact C5/6 but C6 predominates.

UMN lesions are also associated with characteristic patterns of weakness

Unlike LMN lesions, these relate more to voluntary movements than individual muscles, the UMN being at a higher level of organization in the nervous system

A time-honoured term referring to UMN weakness in the limbs is the pyramidal distribution of weakness

By this is meant greater weakness of extensors than flexors in the upper limbs and of flexors than extensors in the lower limbs

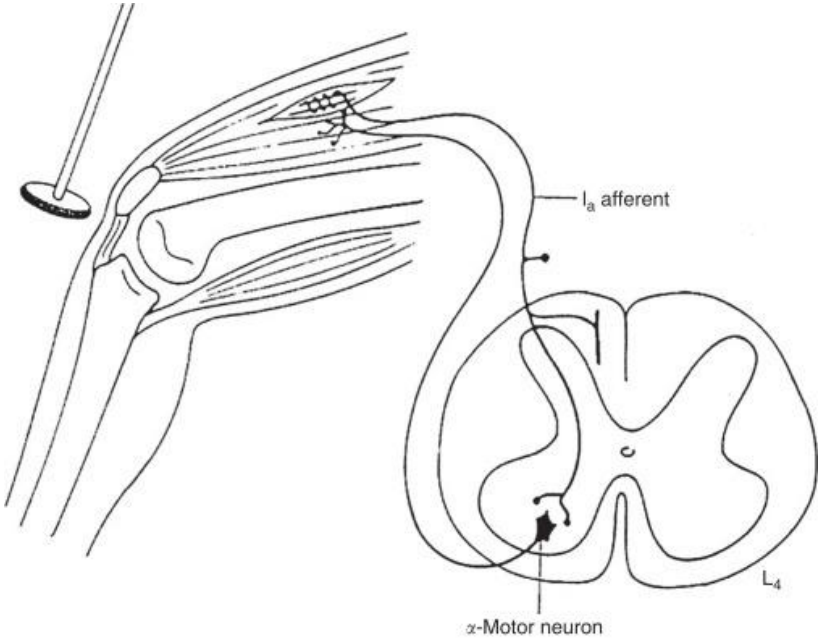
Formal objective measurement of muscle power in UMN lesions using a strain gauge(myometry) has cast doubt on this pattern

However, the description remains of clinical value, particularly as it corresponds to abnormalities of posture seen in patients with advanced UMN lesions

Thus, a patient who is hemiparetic after a vascular event in one hemisphere will typically have a flexed arm and extended leg on the opposite side of the body from the brain lesion(circumducting gait)

Examination of reflexes

- Involuntary contraction of a muscle in response to stretch.
- It is mediated by a reflex arc consisting of an afferent (sensory) >>>> efferent (motor) neuron with one synapse between (a monosynaptic reflex)
- These stretch reflex arcs are served by a particular spinal cord segment which is modified by descending upper motor neurons.



How to examine?

Position: supine on the examination couch with the limbs exposed.

Setting: relaxed and comfortable as possible, as anxiety and pain can cause an increased response.

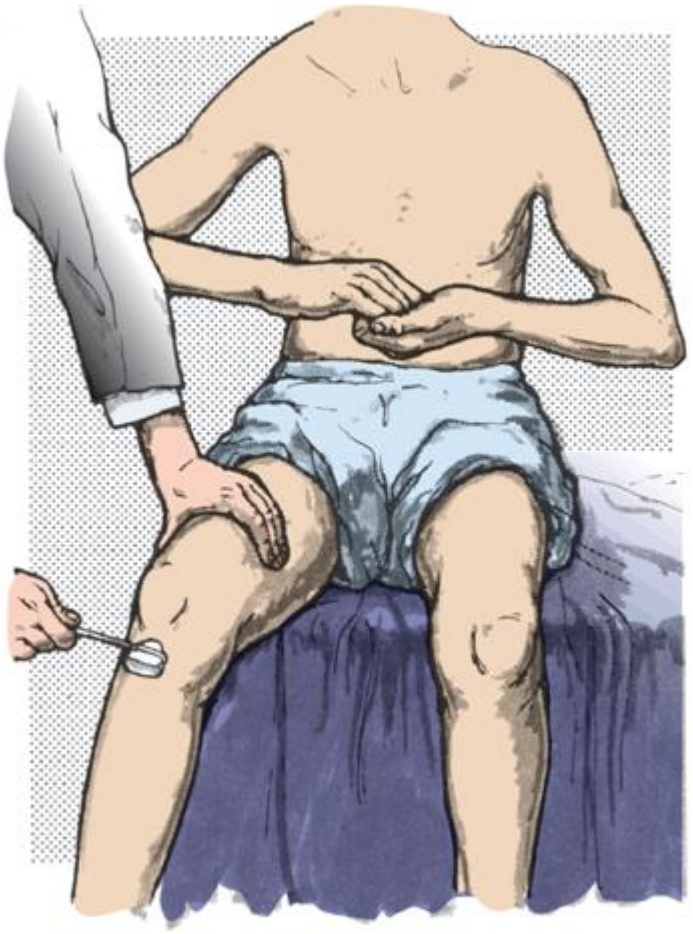
Techniques: Flex your wrist and allow the weight of the tendon hammer head to determine the strength of the blow..

Strike the tendon, not the muscle or bone

Compare each reflex with the other side; check for symmetry of response

Reinforcement

- Use whenever a reflex appears absent
- Never conclude a reflex is absent until you have used reinforcement!!!
- The patient should relax between repeated attempts
- Strike the tendon immediately after your command to the patient
- upper limb reflexes:
  - clench the teeth or to make a fist with the contralateral hand.
- Lower limb reflexes:
  - interlock the fingers and pull one hand against the other (Jendrassik maneuver)

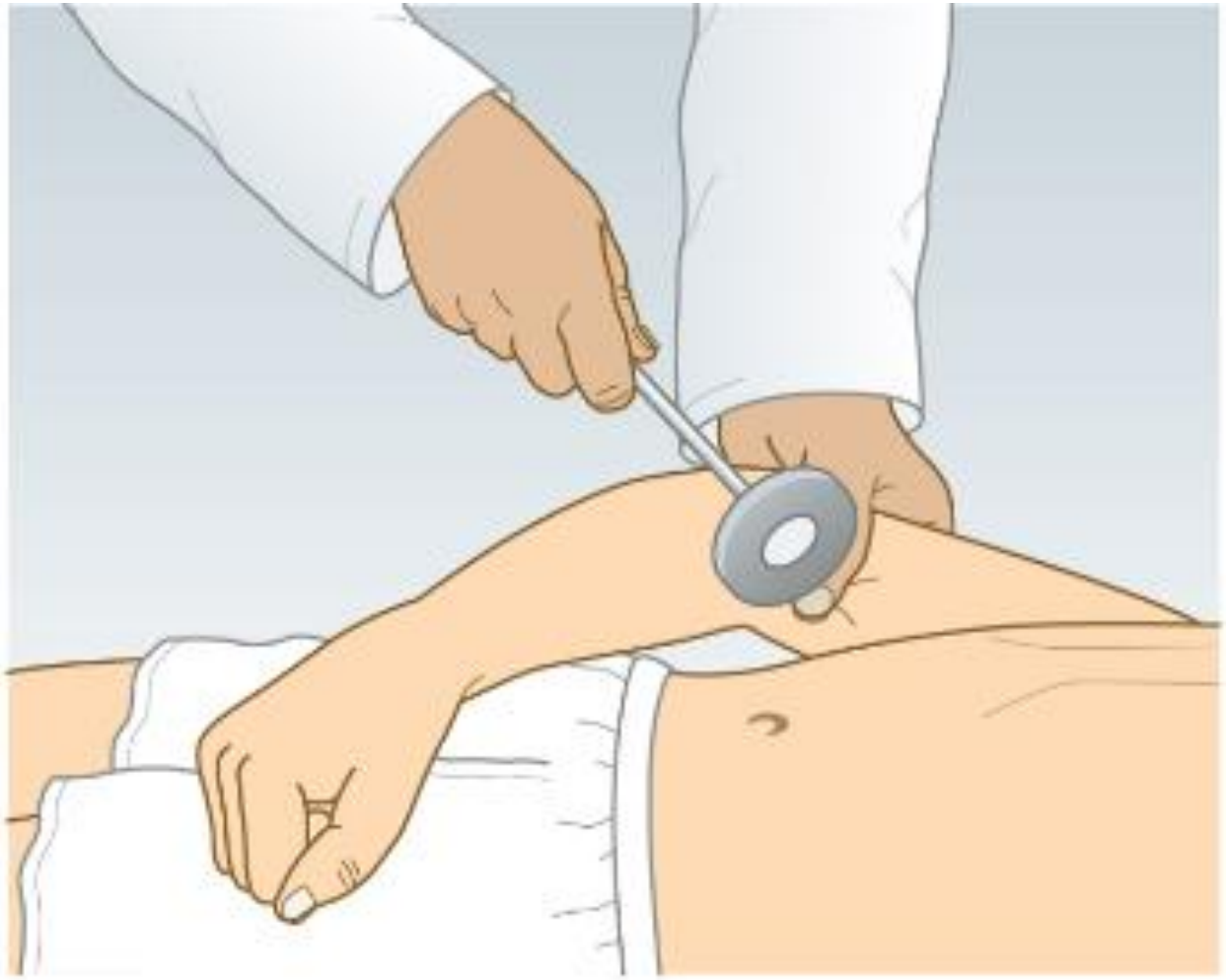


Examined DTRs

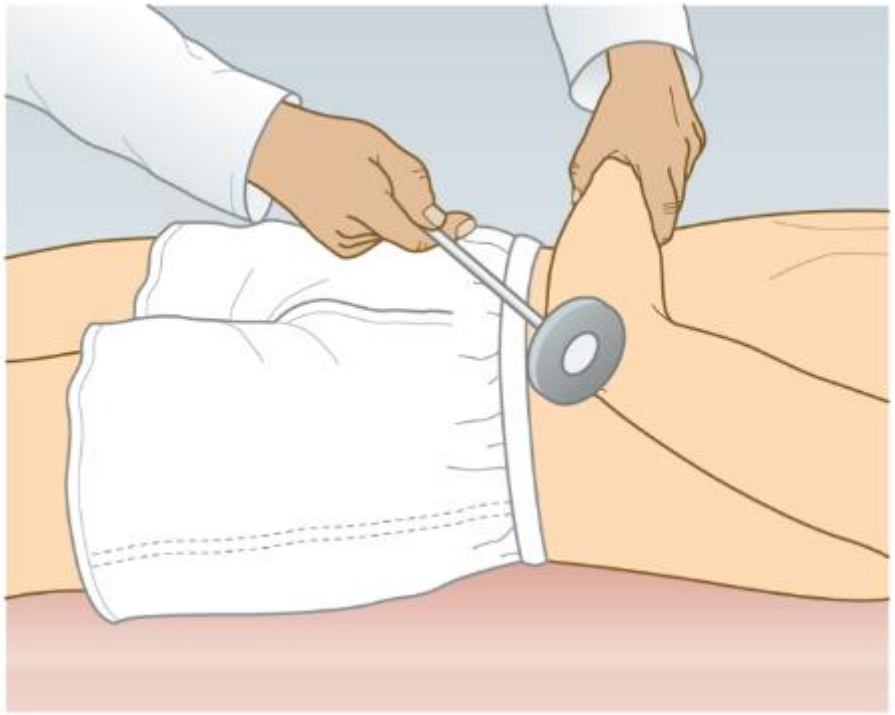
11.24 Monosynaptic (deep tendon) reflexes and root innervation

Reflex (muscle)	Nerve root
Biceps	C5
Supinator (brachioradialis)	C6
Triceps	C7
Knee (quadriceps)	L3, 4
Ankle (gastrocnemius, soleus)	S1

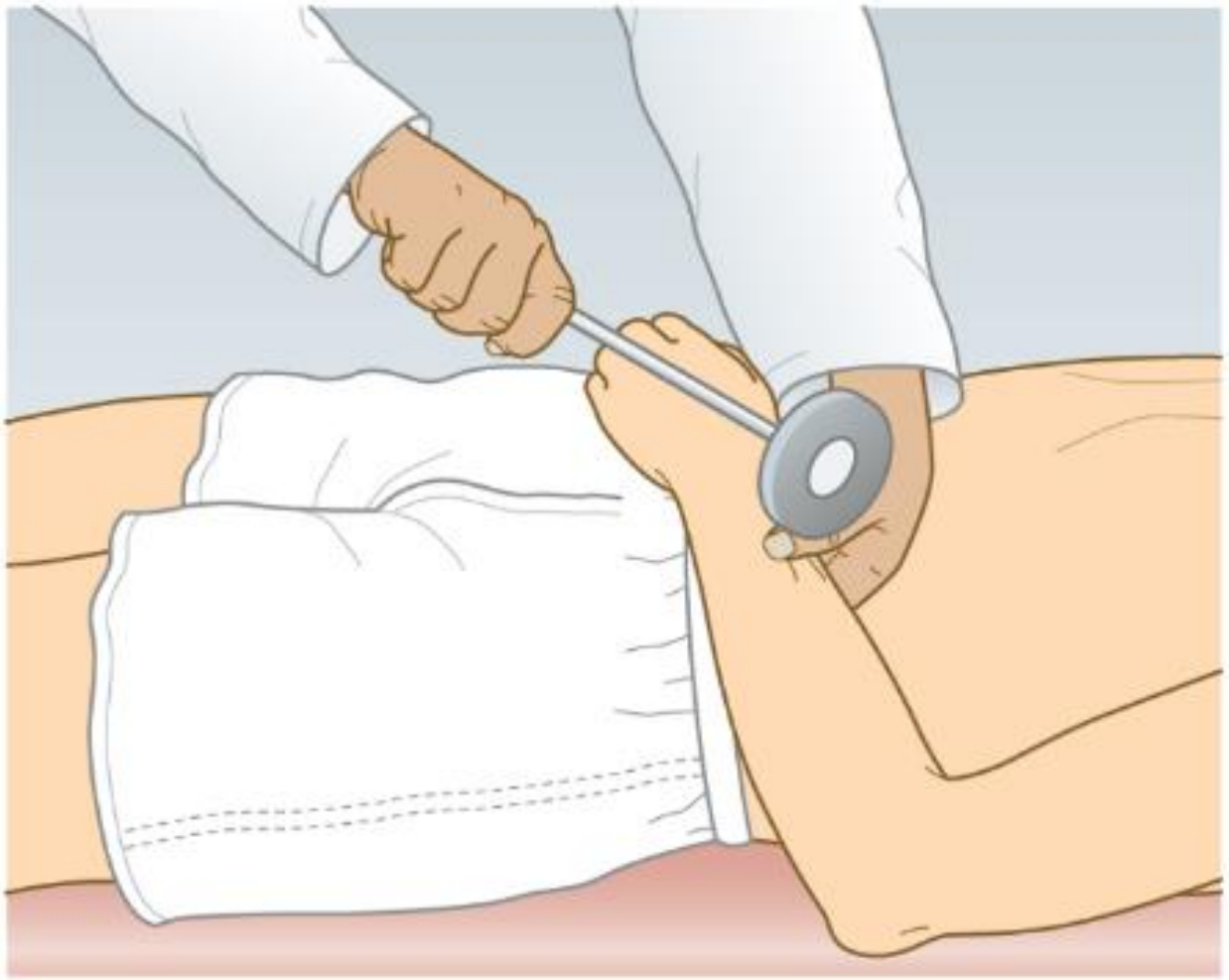
Biceps Jerk



Triceps Jerk

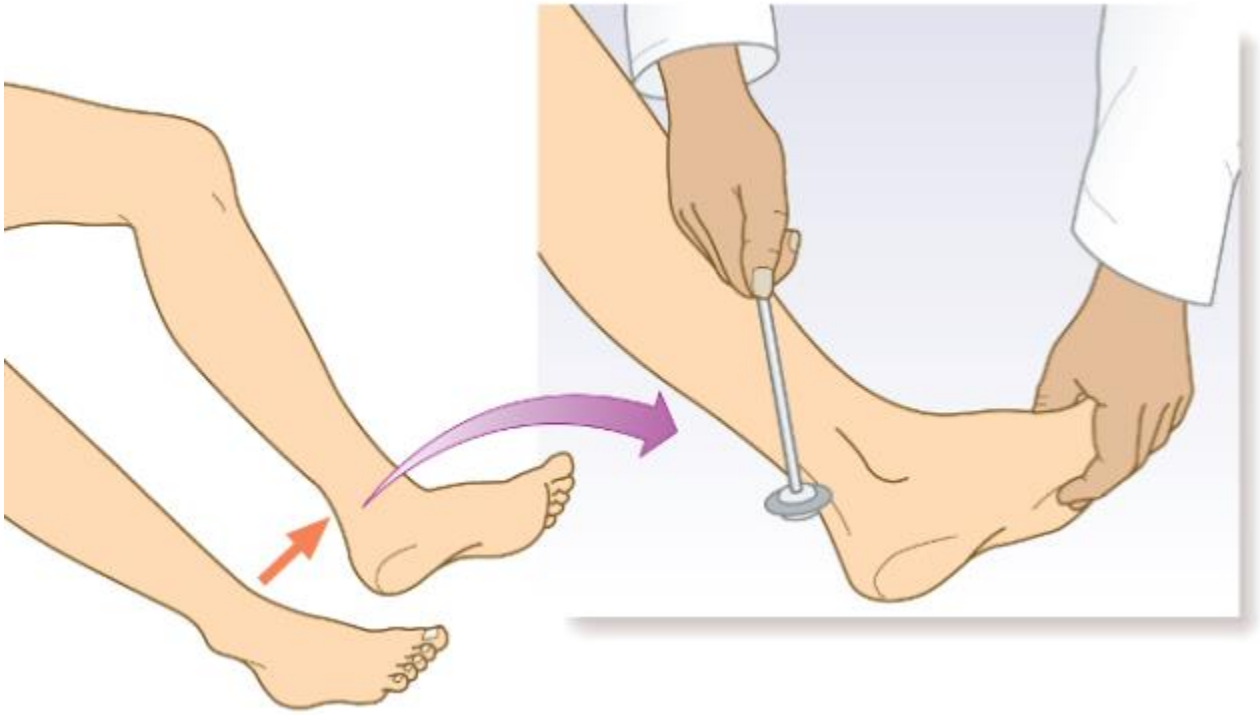


Supinator Jerk



Knee Jerk

Ankle Jerk



These are a direct method of testing the immediate action of the stretch reflex clinically

Striking the tendon of a muscle with a patellar hammer will stretch the muscle passively and induce reflex contraction

As with muscle tone, tendon reflexes may be heightened or diminished by disease

Interruption of the reflex arc, for example by LMN damage, will render the reflex depressed or absent

UMN lesions may produce brisk tendon reflexes as a result of loss of supraspinal inhibition

Recording responses

- Increased
- Normal
- Diminished
- present only with reinforcement
- Absent

The grading of tendon reflexes is usually represented symbolically as:

+++ very brisk

++ brisk

+ present

+- with reinforcement

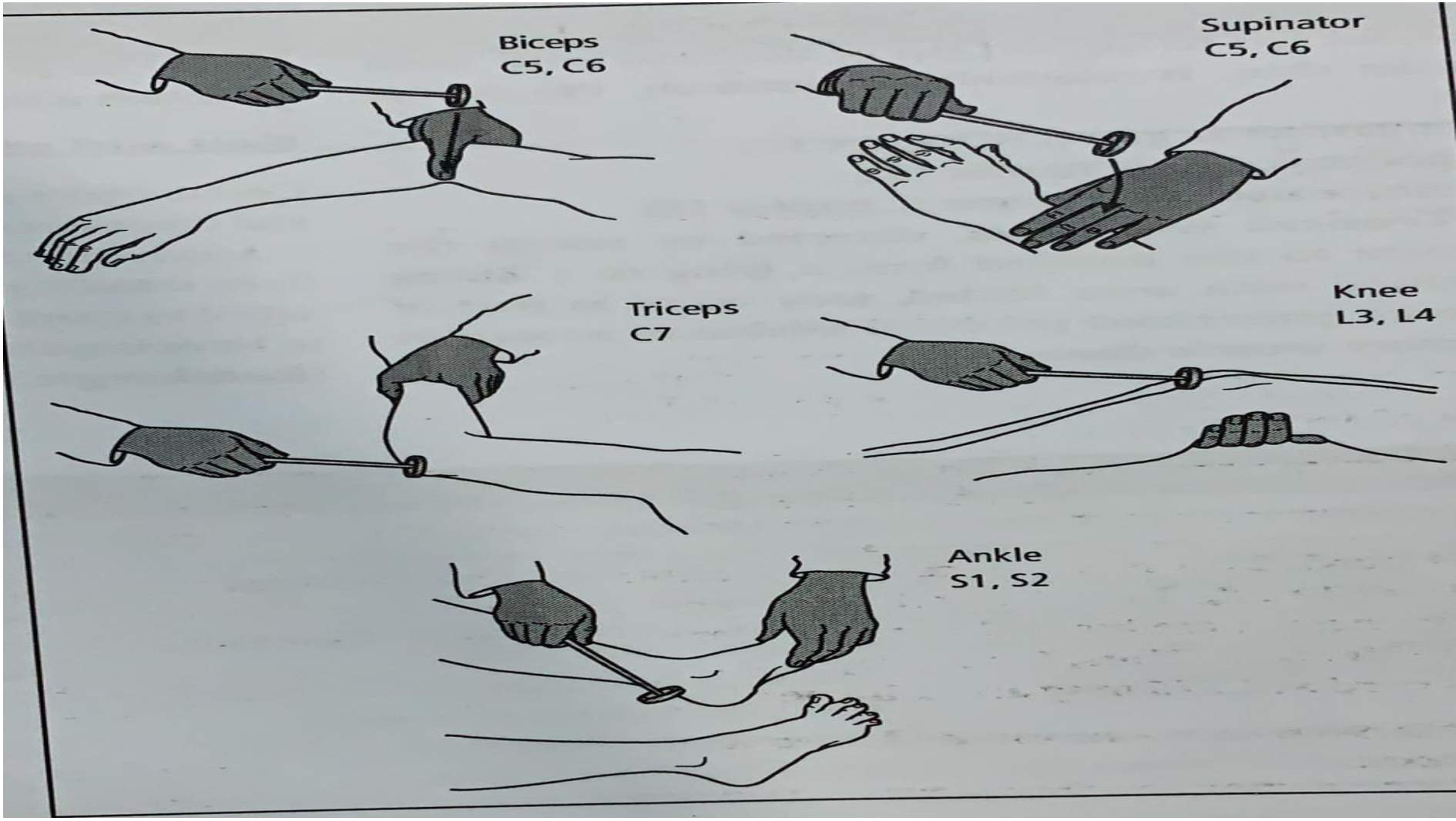
0 absent

CL clonus

The main clinical usefulness of the tendon reflexes is in localizing lesions, especially of the spinal cord.

This arises because the reflexes have 'root values' , i.e. the relevant afferent and efferent nerves are located in particular spinal segments

(figure)


Biceps
C5, C6
Supinator
C5, C6
Triceps
C7
Knee
L3, L4
Ankle
S1, S2

Thus,for example, a lesion of the spinal cord at C5/6 may abolish the biceps and the supinator reflexes, because of LMN damage at that level, but all reflexes below (triceps downwards) will be brisk, because of UMN damage and hence loss of supraspinal inhibition of those segments- a ‘reflex level’

Tendon reflexes may possess qualities indicative of disease processes other than those directly affecting the motor neurones, e.g. the slow-relaxing reflex of hypothyroidism and the pendular reflex of cerebellar disease

Cutaneous reflexes

The cutaneous reflexes most often of value clinically are the plantar and superficial abdominal responses

These depend on afferent nerves concerned with pain sensation (nociception)

The normal response in adults to a stroke along the skin of the lateral border of the foot with an orange stick is plantar flexion of the toes

('downgoing' plantar response)

In normal infants, there is a more primitive version of this flexor withdrawal reflex, with dorsiflexion of the great toe and abduction (fanning) of the other toes('upgoing plantar response).

It is this version which reappears in adult life in the context of UMN damage(positive Babinski reflex)

The superficial abdominal responses are elicited by a swift stroke with an orange stick horizontally across the skin of each abdominal quadrant

Normally there is reflex contraction of the underlying abdominal muscles, but this may be lost in UMN lesions(e.g. loss of the abdominal responses may be an early sign of multiple sclerosis)

The superficial abdominal responses may also be absent in obese patients, in those with abdominal scars and after repeated pregnancy

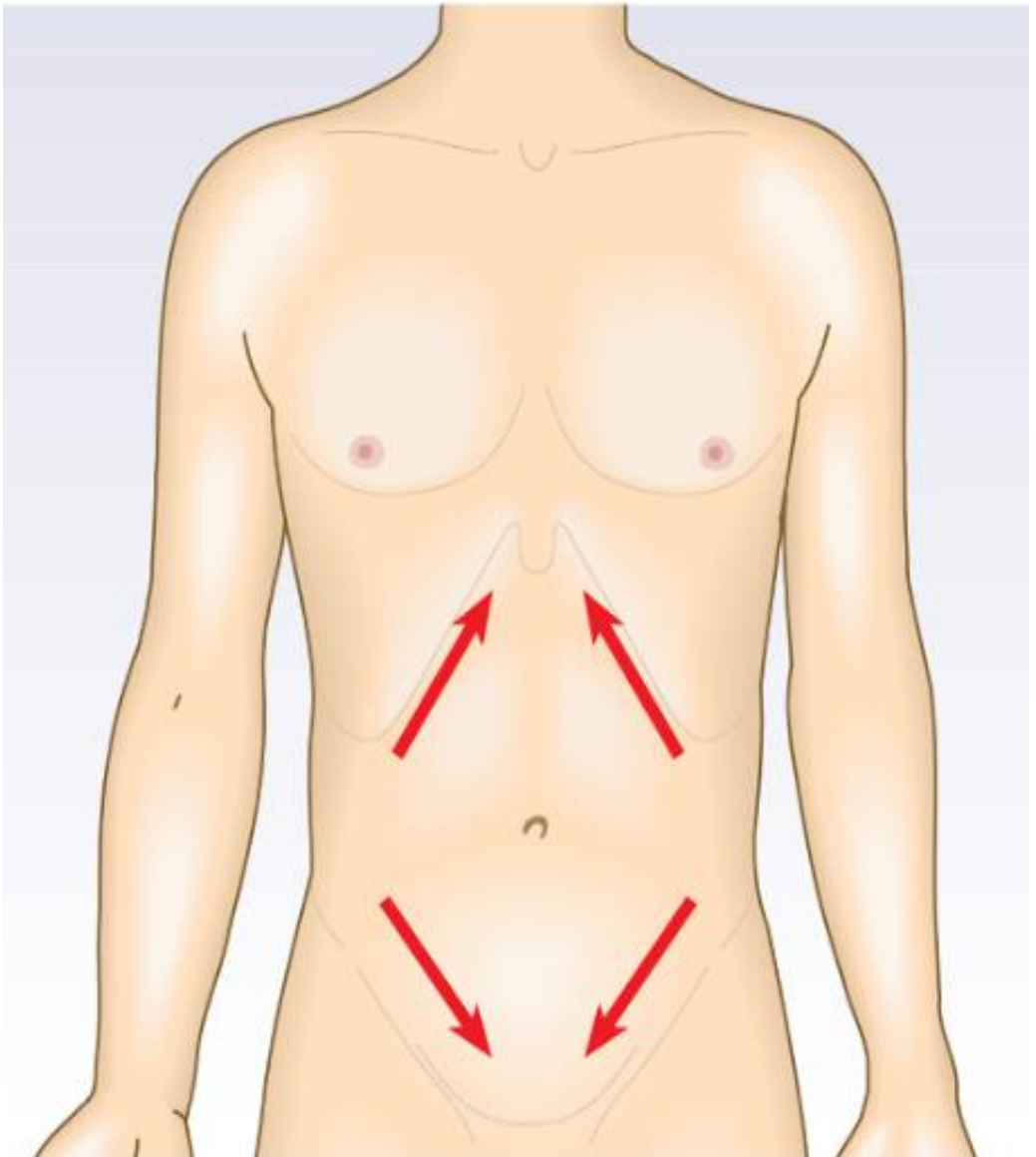
Variation in response

- Hyper-reflexia >>> UMN!!!!
- Diminished or absent jerks
  - LMN Lesions
  - In healthy elderly people the ankle jerks may be reduced or lost
  - Isolated loss of a reflex suggests a mononeuropathy or radiculopathy

- In cerebellar disease
  - the reflexes may be pendular
  - muscle contraction and relaxation tend to be slow
  - these are not sensitive or specific cerebellar signs.

Abdominal reflexes (T8–12)

- Position: Supine and relaxed.
- Technique: With an orange stick and briskly, but lightly, stroke the upper and lower quadrants of the abdomen in a medial direction
- Normally, contraction of the underlying muscle, with the umbilicus moving laterally and up or down depending upon the quadrant tested.



Plantar response

- Run a blunt object along the lateral border of the sole of the foot toward the little toe

- Watch both the first movement of the great toe and the other leg flexor muscles.

- The normal response is flexion of the great toe with flexion of the other toes.

Abnormal findings

- A true Babinski sign:
  - involves activation of the extensor hallucis longus tendon (not movement of the entire foot, a common ‘withdrawal’ response to an unpleasant stimulus)
  - coincides with contraction of other leg flexor muscles
  - is reproducible.

- This is a sign of UMN lesion
- Fanning of the toes is normal and not pathological.

Testing coordination

- Performing complex movements smoothly and efficiently
- Depends upon intact sensory and motor function and an intact cerebellum.

- In general, cerebellar midline structures, e.g. vermis, influence body equilibrium, while each hemisphere controls ipsilateral coordination.

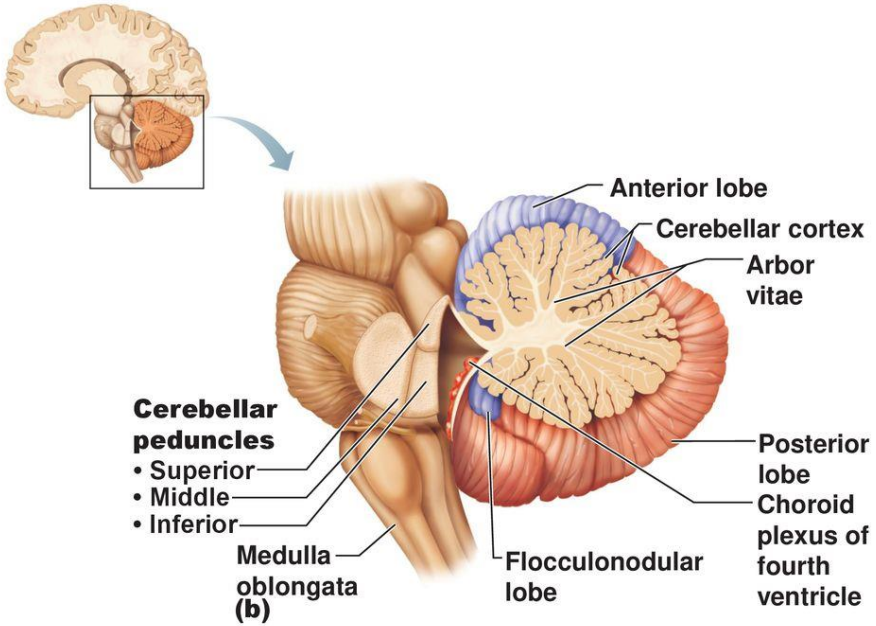


Figure 12.17b

Limb coordination

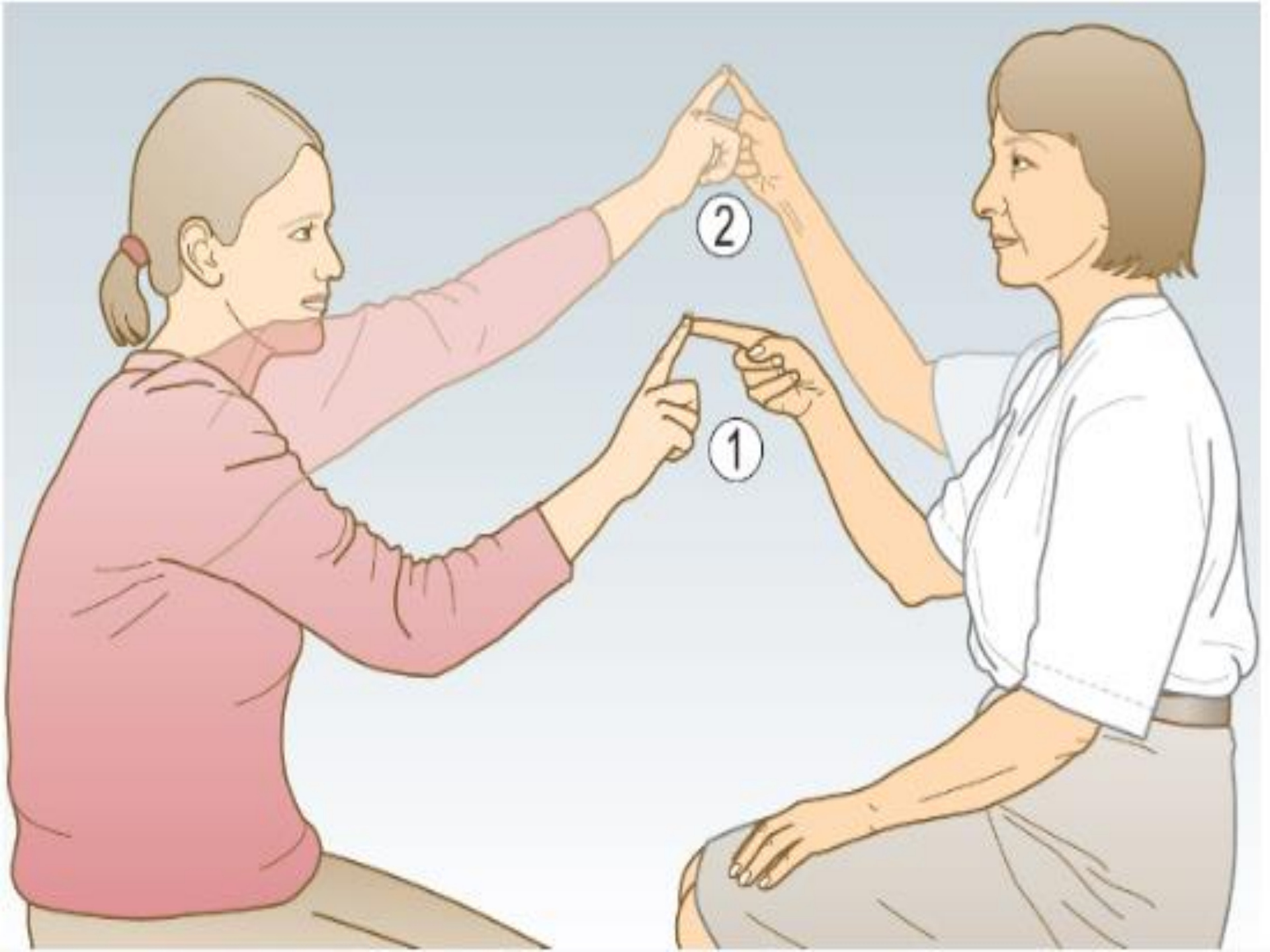
- Upper limbs:
  - Finger-to-nose test
  - Rapid alternating movements
  - Rebound phenomenon (rarely useful)

- Lower limbs:
  - Heel-to-shin test

Finger-to-nose test

- Ask the patient to touch his nose with the tip of his index finger and then touch your fingertip.
- Hold your finger just within the patient's arm's reach
- Ask him to repeat the movement between nose and target finger as quickly as possible.
- Make the test more sensitive by changing the position of your target finger.
- Move your finger just as the patient's finger is about to leave his nose, otherwise you will induce a false-positive finger-to-nose ataxia.
- Some patients are so ataxic that they may injure their eye/face with this test. If so, use your two hands as the targets


1
2


2
1

Abnormal findings

- Weakness may produce false-positive finger-to-nose test, so demonstrate that power is normal first.

- Dysmetria or past-pointing:
  - tendency to fall short or overshoot the examiner's finger

- Intention tremor:
  - In more severe cases there may be a tremor of the finger as it approaches the target finger and the patient's own nose

- Dys-synergia:
  - The movement may be slow, disjointed, and clumsy

Rapid alternating movements

- First method:
  - Demonstrate repeatedly patting the palm of your hand with the palm and back of your opposite hand as quickly and regularly as possible.
  - Ask the patient to copy your actions.
  - Repeat with the opposite hand.

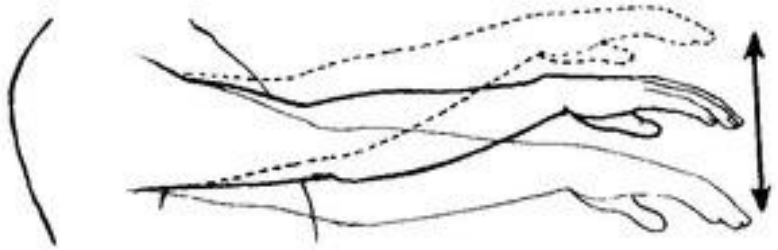
Abnormal findings: Dysdiadochokinesis

- Impairment of rapid alternating movements
- Evident as slowness, disorganization, and irregularity of movement

Rebound phenomenon

- Ask the patient to stretch his arms out and maintain this position.
- Push the patient's wrist quickly downward and observe the returning movement.
- Normally: Arm return to the original position
- Abnormally: The displaced outstretched arm may fly up past the original position

Arm bounce



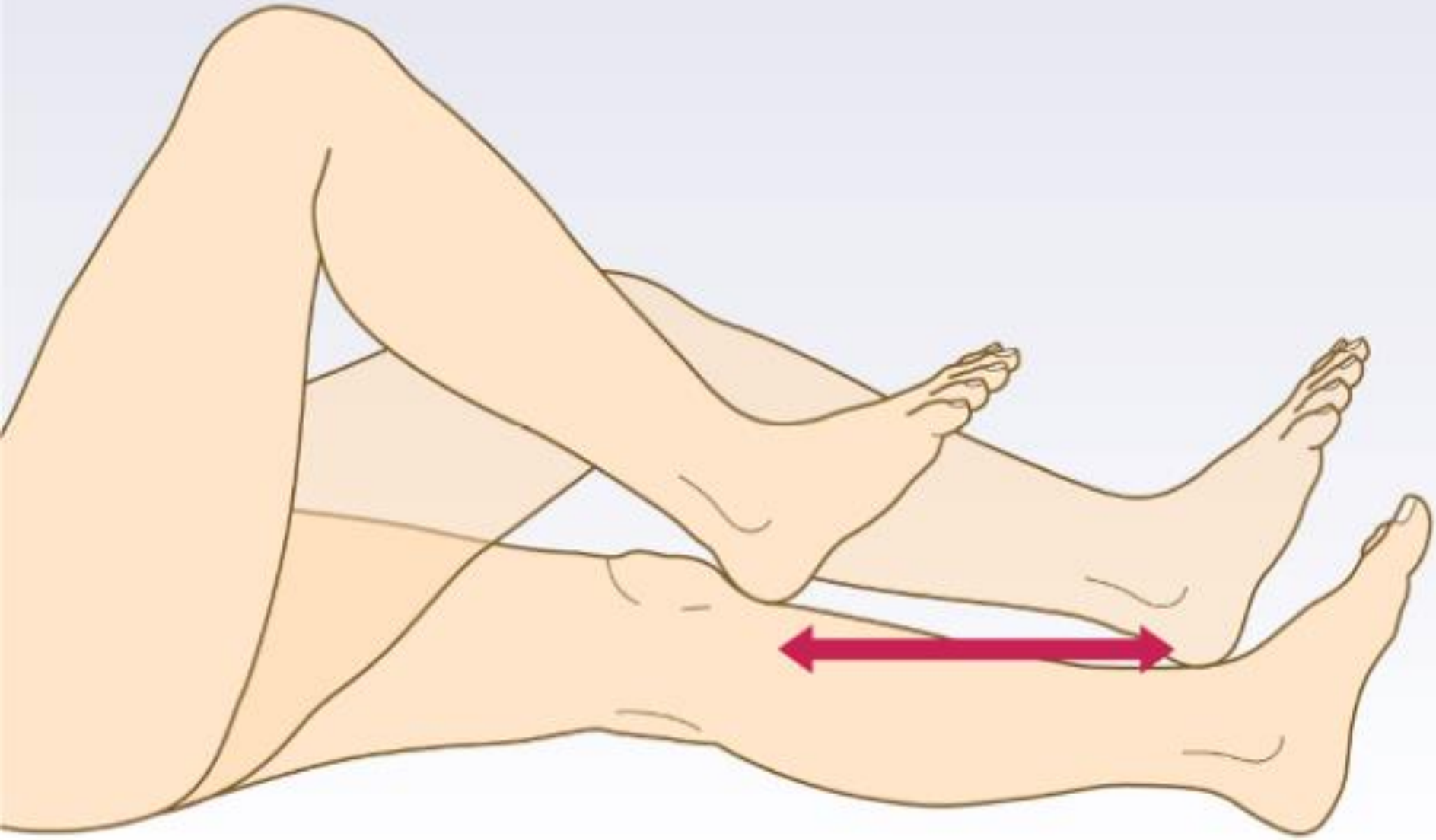
Downward pressure and sudden release of the patient's outstretched arm causes excessive swinging.

Rebound phenomenon

Ask the patient to flex elbow against resistance. Sudden release may cause the hand to strike the face due to delay in triceps contraction.

Heel-to-shin test

- With the patient lying supine, ask him to place his heel on his opposite knee, and then slide his heel up and down the shin between the knee and ankle
- Same as finger-to-nose test
- Abnormal: if the heel wavers away from the line of the shin.
- Weakness may produce a false-positive heel-to-shin test, so demonstrate that power is normal first.



These tests of limb ataxia provide only a partial picture of cerebellar function

Much may also be learnt from assessment of muscle tone, which may be reduced in cerebellar disease, from the reflexes and from examining gait, speech and eye movements

Neck and trunk

Neck flexion is achieved by simultaneous contraction of both sternomastoid muscles,innervated by the spinal accessory nerves

Weakness of neck extension , such that the patient has to support his or her head with hand under chin, is relatively uncommon, but occurs in:

- Myasthenia gravis
- Polymyositis
- Motor neurone disease

Truncal weakness, detected by asking the patient to rise unaided from a lying to a sitting position with arms folded, may occur as part of a more generalized proximal weakness, as seen in primary muscle disease

Truncal ataxia is particularly associated with damage to cerebellar midline(vermis) structures

It may be so severe that the patient is unable to maintain a stable sitting posture unsupported

Gait and stance

Certain gaits are associated with specific neurological disorders(table)

Much may also be learnt from observing the patient standing unaided

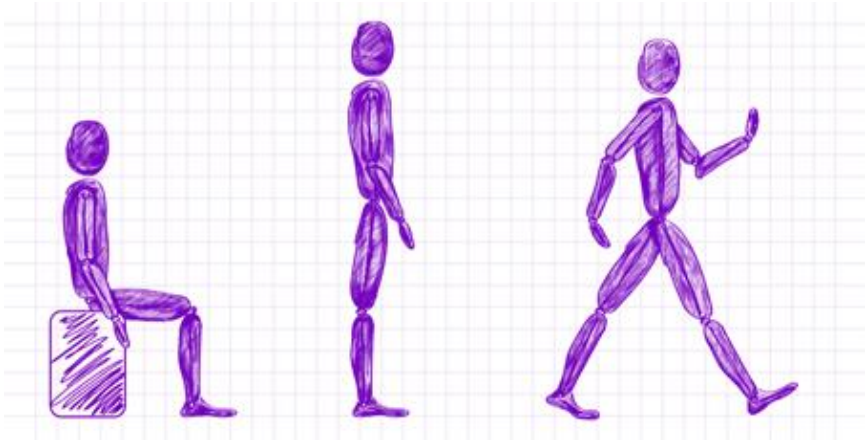
A patient who falls when asked to stand to ‘attention’ with eyes shut is likely to have impaired joint position sense at the ankles (Romberg’s sign)

Key points are shown in the table

Stance and gait

- Stance:
  - Narrow base while eyes are open and closed

- Gait:
  - slapping sound of a foot drop gait?
  - tiptoes, then on the heels.
  - Tandem gait



IS IT WHEN OPENING AND CLOSING EYES? CEREBELLAR!

IS IT MORE WHEN EYES ARE CLOSED???? SENSORY!

Abnormal Gait

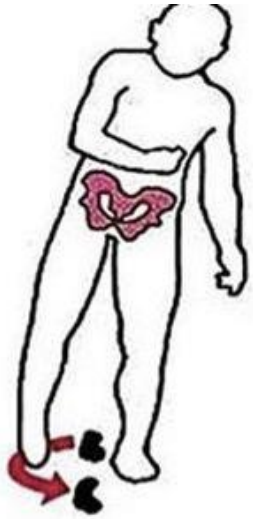
- Hemiplegic gait.
- Scissors-like gait.
- Ataxic gait.
- Foot drop.
- Parkinsonian gait
- Waddling gait.
- Bizarre gaits.

Abnormal Gait

Parkinsonian gait

Paraspastic gait

Spastic–atactic gait



gait with circumduction in a patient with right hemiparesis

Trendelenburg gait

Duchenne gait

Quadriceps weakness

Steppage gait with foot drop

Table 5.3 Neurological gait disorders.

Spastic paraparesis (UMN lesions, both legs)	Scissoring, 'wading through mud'
Spastic hemiparesis (UMN lesion, one side of body)	Leg is rigid and circumducts (describes a semicircle rotating at hip)
Bilateral foot drop (LMN lesions, both legs)	Steppage – legs lifted high to avoid scraping toe
Cerebellar lesion	Wide-based gait, staggering, unable to walk heel–toe
Parkinsonism	Stooping posture, rigid shuffling gait, 'festinant', no arm swing
Proximal myopathy	Waddling

Key points

	LMN	UMN
Wasting	Present (neurogenic wasting)	Disuse atrophy only
Fasciculations	May be present	Absent
Tone	Normal or decreased (flaccidity)	Increased (spasticity)
Posture	–	Drift of outstretched arm (eyes shut)
Power	Focal weakness, e.g. distribution of individual nerves or roots	Movement-based Pyramidal distribution
Tendon reflexes	Depressed or absent	Brisk
Clonus	Absent	May be present
Plantar response	Downgoing or absent	Upgoing (positive Babinski)
Superficial abdominal responses	Present	May be absent
Gait	May be high-stepping	Spastic, scissoring, circumduction

11.12 Features of motor neurone lesions

	Upper motor neurone lesion	Lower motor neurone lesion
Inspection	Usually normal (wasting in longstanding lesions)	Wasting, fasciculation
Tone	Increased with clonus	Normal or decreased, no clonus
Weakness	Preferentially affects extensors in arms, flexors in leg	Usually more focal, in distribution of nerve root or peripheral nerve
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor	Flexor

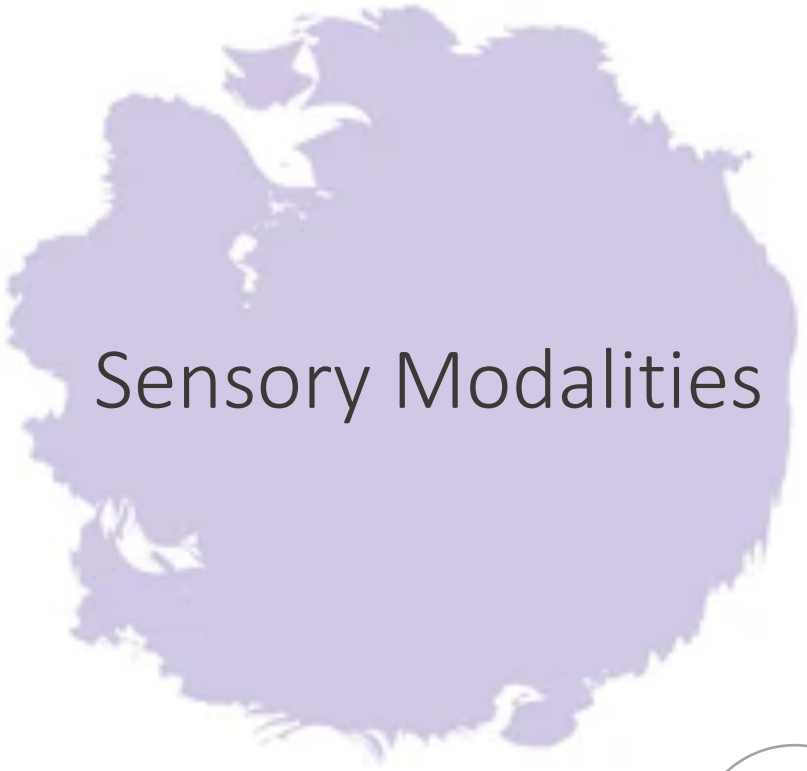

IM SO TIRED
ME


Take a rest

The sensory system

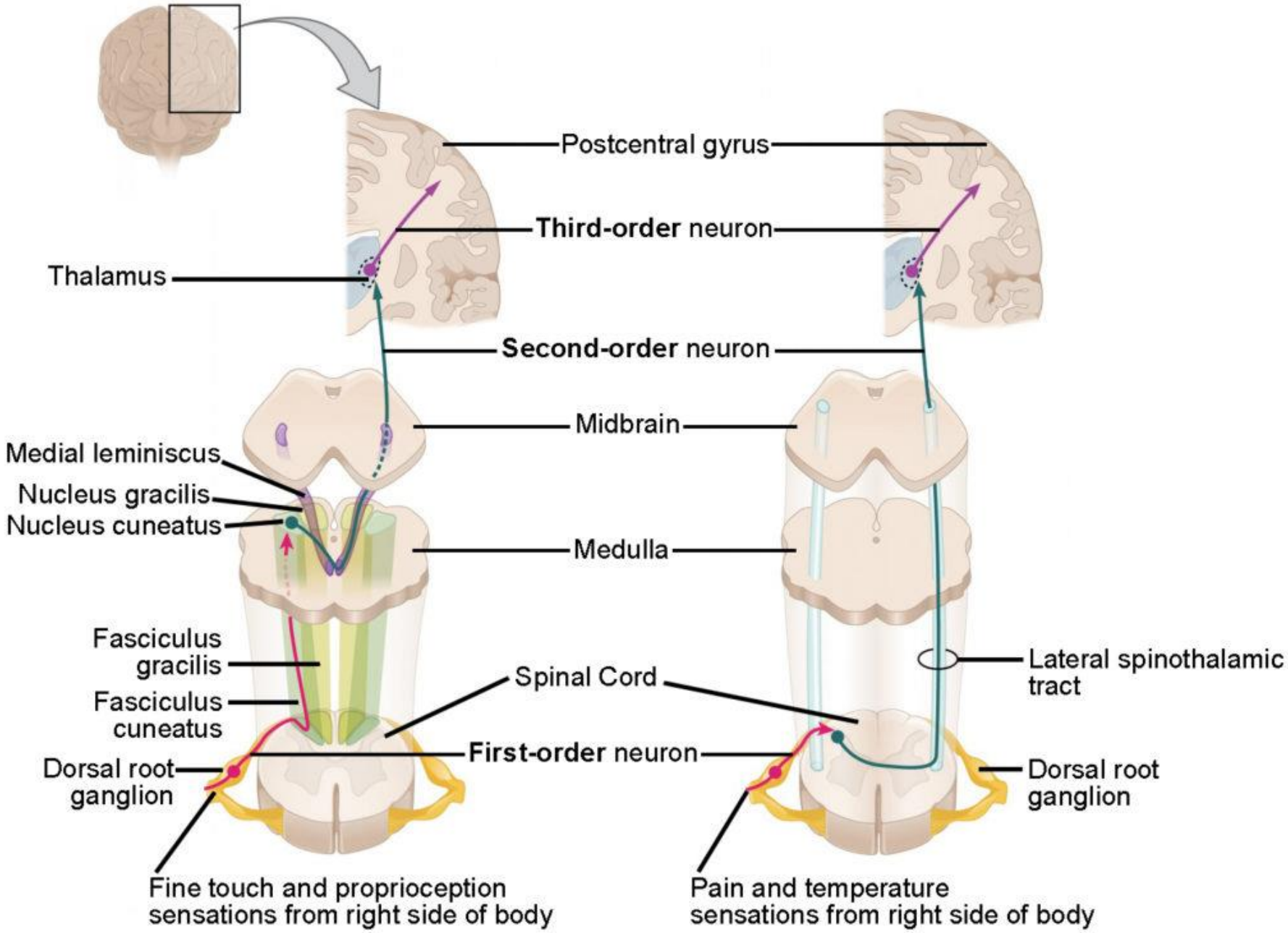
A small tip!

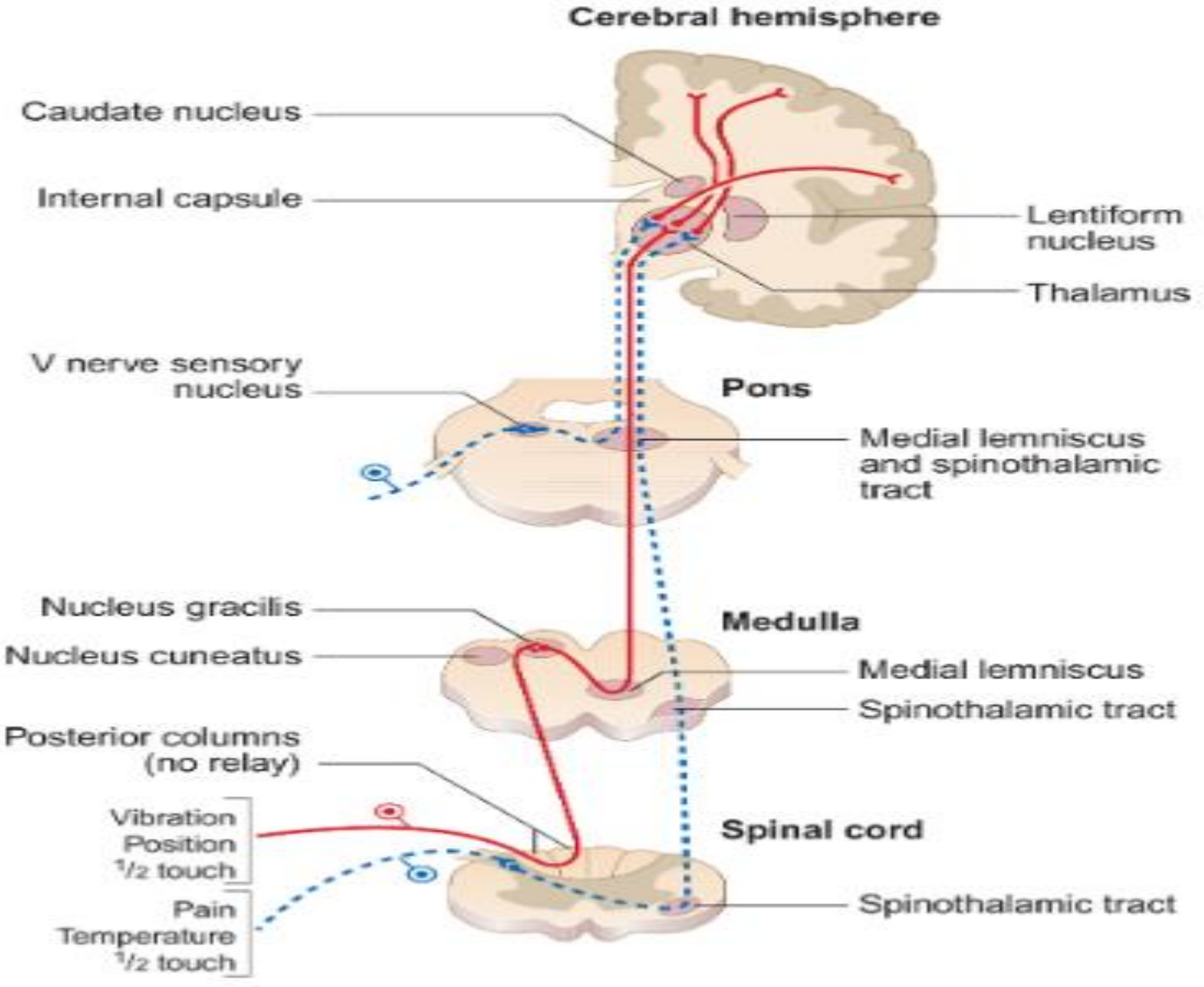
Detailed examination of sensation is time-consuming and unnecessary unless the patient demonstrates sensory symptoms, or you suspect a specific pathology



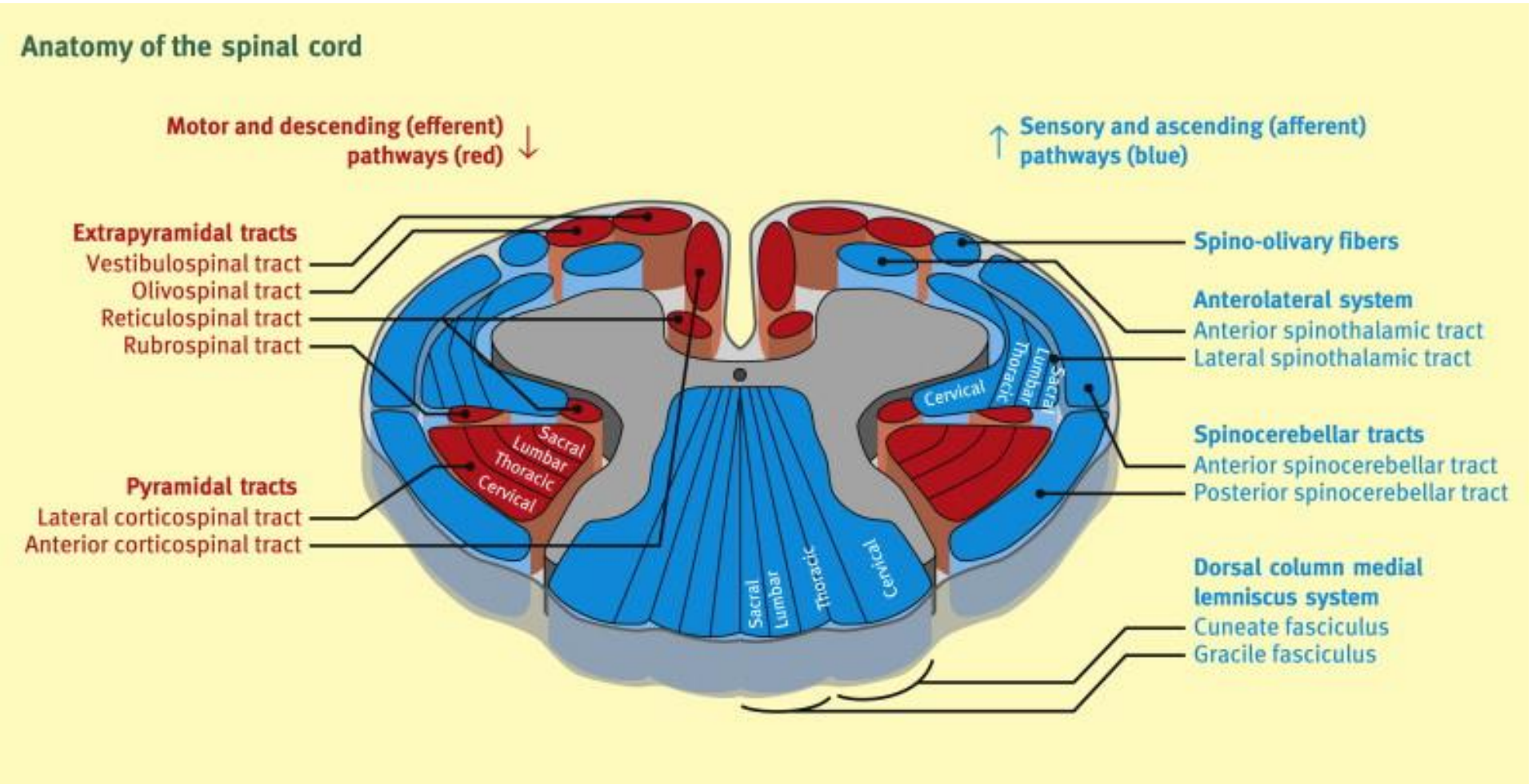
- Light Touch
- Superficial pain
- Temperature
- Vibration
- Proprioception

Let’s review some anatomy!


Postcentral gyrus
Third-order neuron
Thalamus
Second-order neuron
Midbrain
Medial leminiscus
Nucleus gracilis
Nucleus cuneatus
Medulla
Fasciculus gracilis
Fasciculus cuneatus
Spinal Cord
First-order neuron
Dorsal root ganglion
Fine touch and proprioception sensations from right side of body
Lateral spinothalamic tract
Dorsal root ganglion
Pain and temperature sensations from right side of body
Dorsal column system
Spinothalamic tract


Cerebral hemisphere
Caudate nucleus
Internal capsule
Lentiform nucleus
Thalamus
V nerve sensory nucleus
Pons
Medial lemniscus and spinothalamic tract
Nucleus gracilis
Nucleus cuneatus
Medulla
Medial lemniscus
Spinothalamic tract
Posterior columns (no relay)
Vibration
Position
1/2 touch
Spinal cord
Pain
Temperature
1/2 touch
Spinothalamic tract

Anatomy of the spinal cord



So, Let's Recap on the Diagrams

- Proprioception and vibration sensation:
  - Conveyed in large, myelinated fast-conducting fibers in the peripheral nerves and in the posterior columns of the spinal cord
  - The posterior column remains ipsilateral from the point of entry up to the medulla.
- Pain and temperature sensation
  - Carried by small, slow-conducting fibers of the peripheral nerves and the spinothalamic tract of the spinal cord.
  - Most pain and temperature fibers cross to the contralateral spinothalamic tract within one or two segments of entry to the spinal cord.
- All sensory fibers relay in the ***thalamus*** before sending information to the sensory cortex in the ***parietal lobe***

Symptoms and definitions

Paraesthesia	Tingling, or pins and needles Spontaneous or provoked Not unduly unpleasant or painful
Dysaesthesia	Unpleasant paraesthesia
Hypoaesthesia	Reduced sensation to a normal stimulus
Analgesia	Numbness or loss of sensation
Hyperaesthesia	Increased sensitivity to a stimulus
Allodynia	Painful sensation resulting from a non-painful stimulus
Hyperalgesia	Increased sensitivity to a painful stimulus

Light Touch

- While the patient looks away or closes his eyes, use a wisp of cotton wool (or lightly apply your finger) and ask the patient to say yes to each touch.
- Time the stimuli irregularly and make a dabbing rather than a stroking or tickling stimulus.
- Compare each side for symmetry.

Superficial pain

- Use a fresh neurological pin
- Explain and demonstrate that the ability to feel a sharp pinprick is being tested.
- Map out the boundaries of any area of reduced, absent or increased sensation and compare with.
- Move from reduced to higher sensibility: i.e. from hypoaesthesia to normal, or normal to hyperaesthesia.

Temperature

- Touch the patient with a cold metallic object, e.g. tuning fork, and ask if it feels cold.

- More sensitive assessment requires tubes of hot and cold water at controlled temperatures but is seldom performed.

Vibration

- Teach your patient what to fell?
  - Place a vibrating 128 Hz tuning fork over the sternum.
  - Ask the patient, ‘Do you feel it buzzing?’

- Lower limb:
  - Place it on the tip of the great toe
  - If sensation is impaired, place the fork on the interphalangeal joint and progress proximally, to the medial malleolus, tibial tuberosity and anterior iliac spine, depending upon the response.

Vibration

- The upper limb:
  - Start at the distal interphalangeal joint of the forefinger, and if sensation is impaired, proceed proximally.

- If in doubt as to the accuracy of the response, ask the patient to close his eyes and to report when you stop the fork vibrating with your fingers.

Proprioception

- With the patient's eyes open, demonstrate the procedure.
  - Hold the distal phalanx of the patient's great toe at the sides.
  - Tell the patient you are going to move his toe up or down, demonstrating as you do so
- Ask the patient to close his eyes and to identify the direction of small movements in random order.
- Test both great toes (or middle fingers). If impaired, move to more proximal joints in each limb.

The sensory modalities (Identifying the lesion level)

- Peripheral nerve and dorsal root
- Spinal cord
- Intracranial

>> Interpretation of sensory signs requires knowledge of the relevant anatomy of sensory nerves and dermatomes

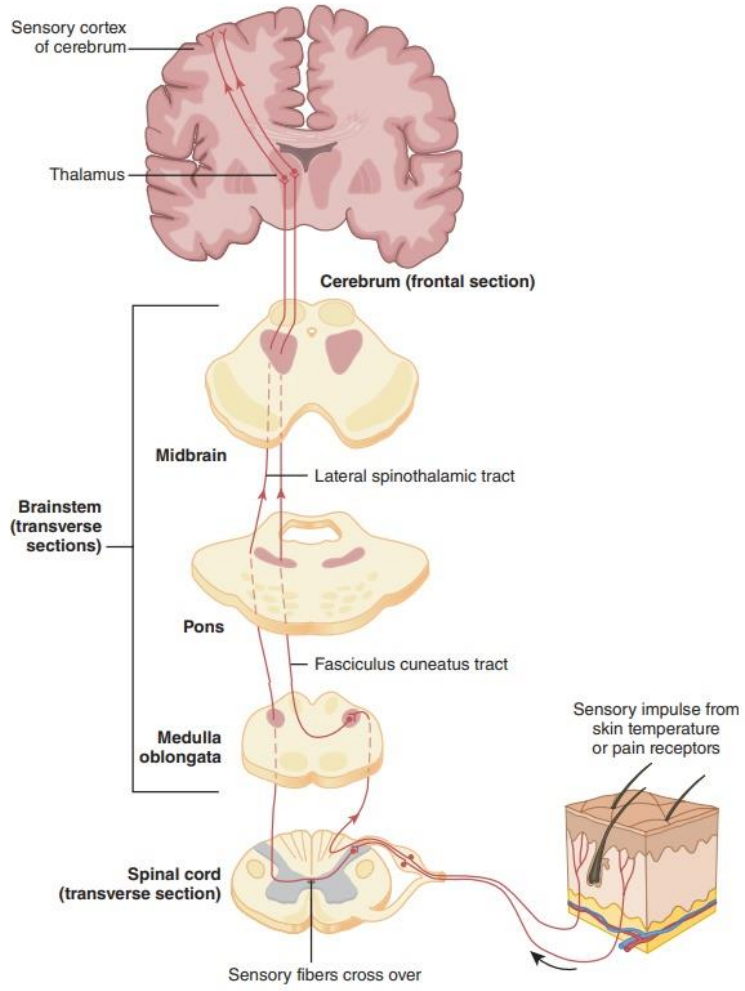
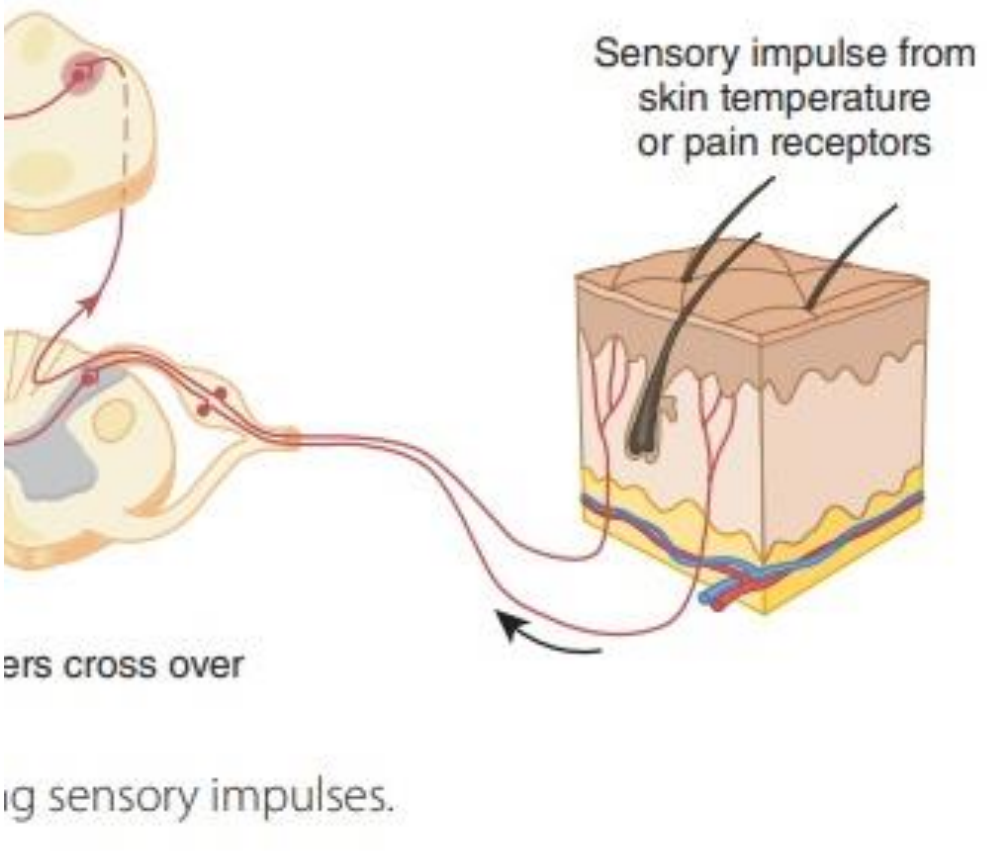


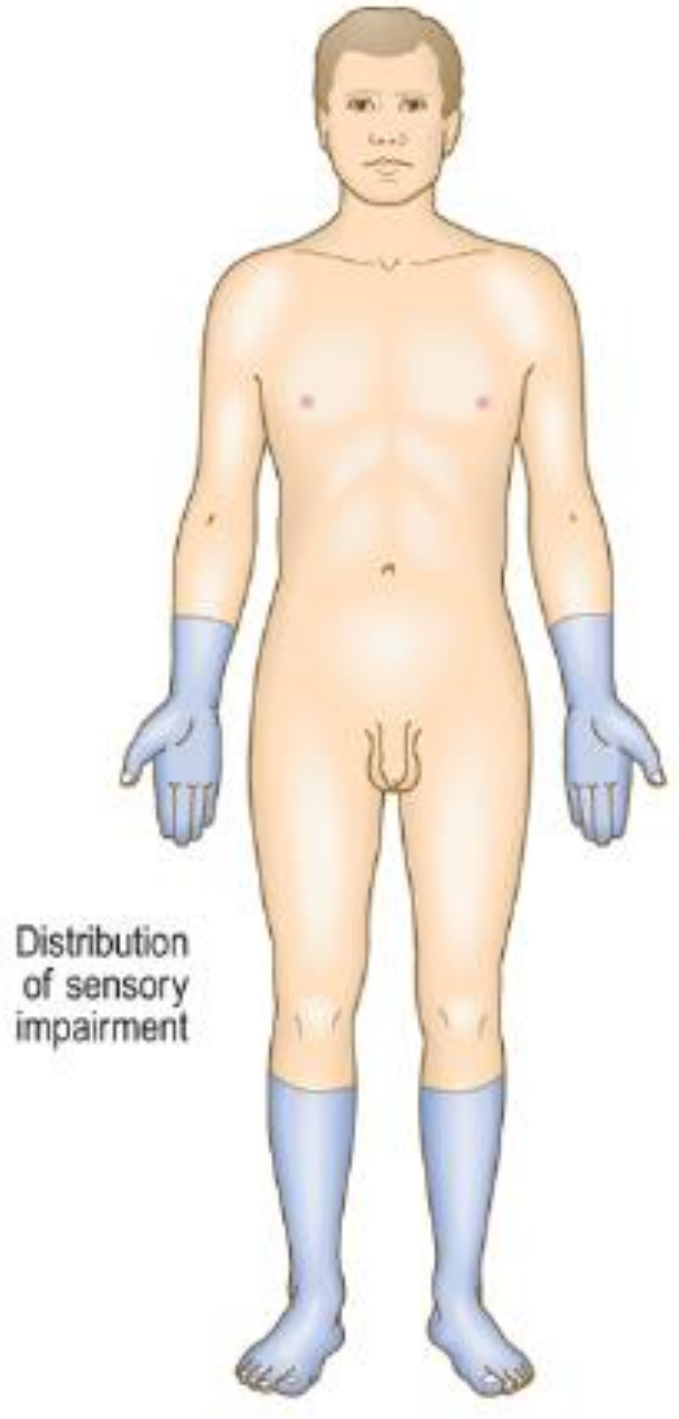
FIGURE 12-16 The path of ascending sensory impulses.

Peripheral nerve and dorsal root

- Many diseases affect peripheral nerves, generally resulting in peripheral neuropathies or polyneuropathies
- Peripheral neuropathies tend to affect the lower limbs first (length-dependent).
- Symptoms affecting the upper limbs first suggest a demyelinating rather than axonal neuropathy or a disease process in the spinal cord.

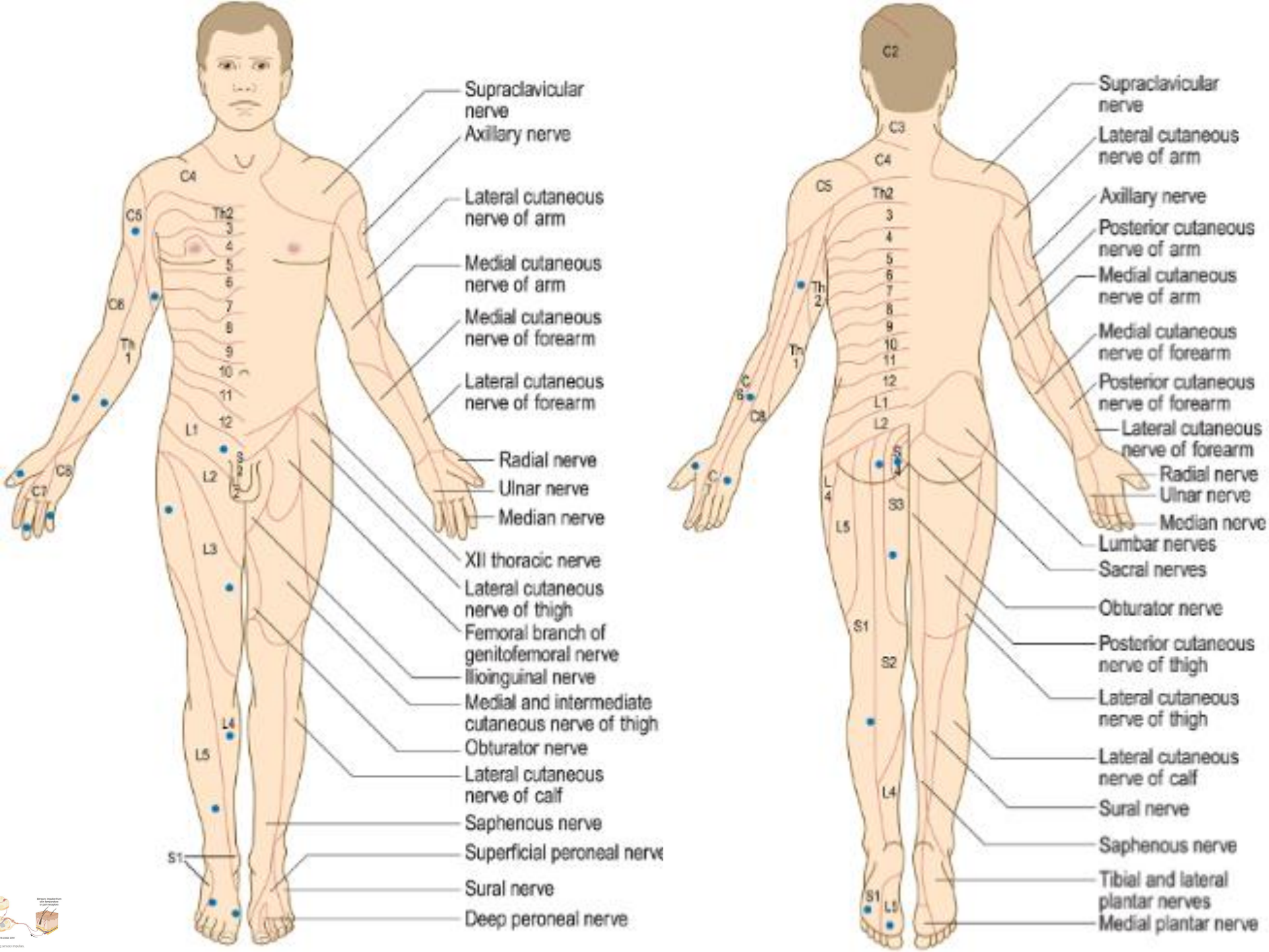


Gloves and Stockings (Diabetic Neuropathy)



Loss of Proprioception (Pseudoathetosis)

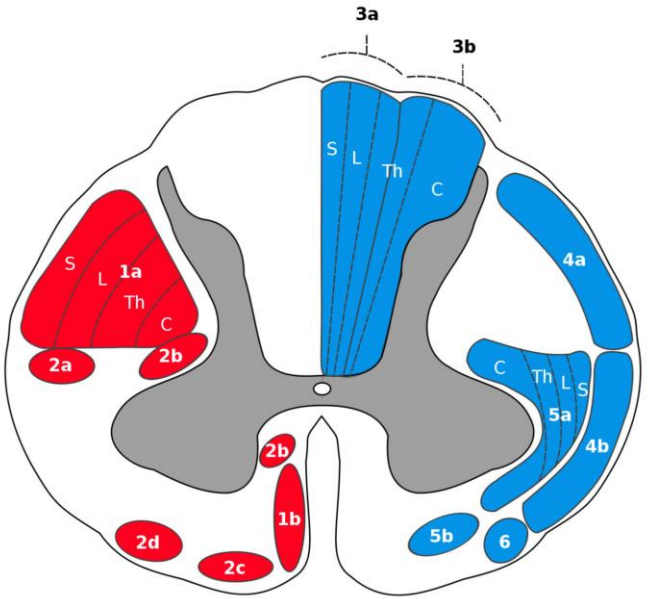
- May be demonstrated by asking the patient to close his eyes and hold his hands outstretched: the fingers will make involuntary, slow wandering movements, mimicking athetosis.


Supraclavicular nerve
Axillary nerve
Lateral cutaneous nerve of arm
Medial cutaneous nerve of arm
Medial cutaneous nerve of forearm
Lateral cutaneous nerve of forearm
Radial nerve
Ulnar nerve
Median nerve
XII thoracic nerve
Lateral cutaneous nerve of thigh
Femoral branch of genitofemoral nerve
Ilioinguinal nerve
Medial and intermediate cutaneous nerve of thigh
Obturator nerve
Lateral cutaneous nerve of calf
Saphenous nerve
Superficial peroneal nerve
Sural nerve
Deep peroneal nerve
C4
C5
Th2
3
4
5
6
7
8
9
10
11
12
L1
L2
L3
L4
L5
C6
Th 1
C7
C8
S1
C2
C3
C4
C5
Th2
3
4
5
6
7
8
9
10
11
12
L1
L2
L3
L4
L5
S1
S2
S3
S4
S5
Th 2
Th 1
C 6
C8
C
L 4
Supraclavicular nerve
Lateral cutaneous nerve of arm
Axillary nerve
Posterior cutaneous nerve of arm
Medial cutaneous nerve of arm
Medial cutaneous nerve of forearm
Posterior cutaneous nerve of forearm
Lateral cutaneous nerve of forearm
Radial nerve
Ulnar nerve
Median nerve
Lumbar nerves
Sacral nerves
Obturator nerve
Posterior cutaneous nerve of thigh
Lateral cutaneous nerve of thigh
Lateral cutaneous nerve of calf
Sural nerve
Saphenous nerve
Tibial and lateral plantar nerves
Medial plantar nerve

Spinal cord

- Traumatic and compressive spinal cord lesions cause loss or impairment of sensation in a dermatomal distribution below the level of the lesion.

- A zone of hyperaesthesia may be found immediately above the level of sensory loss.



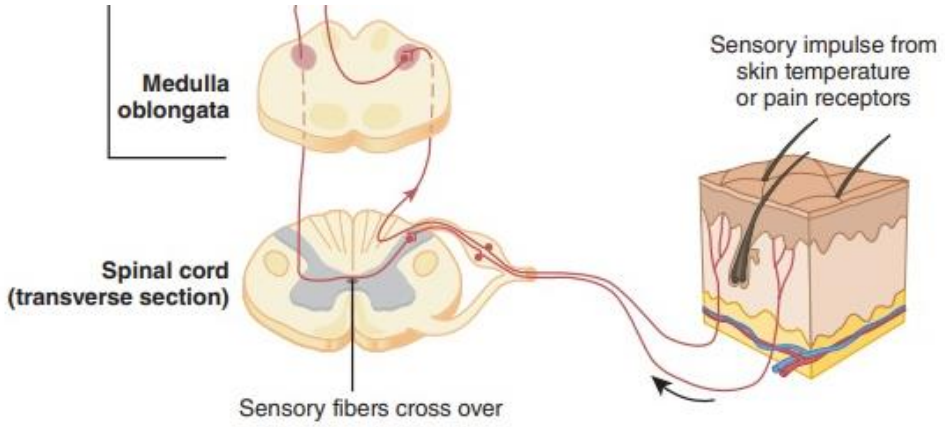


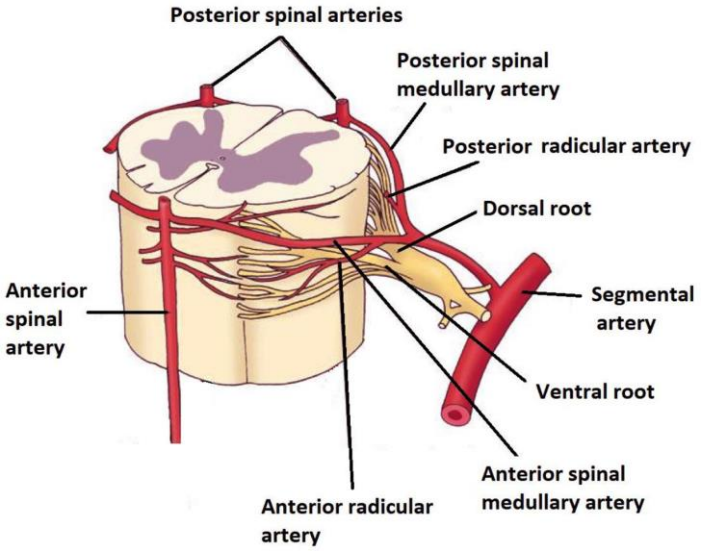
FIGURE 12-16 The path of ascending sensory impulses.

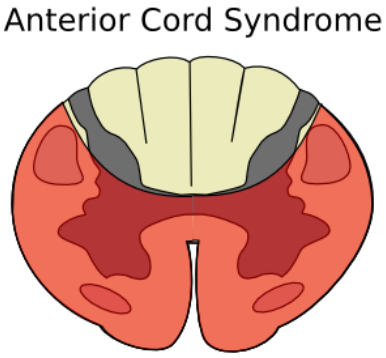
Spinal cord lesions

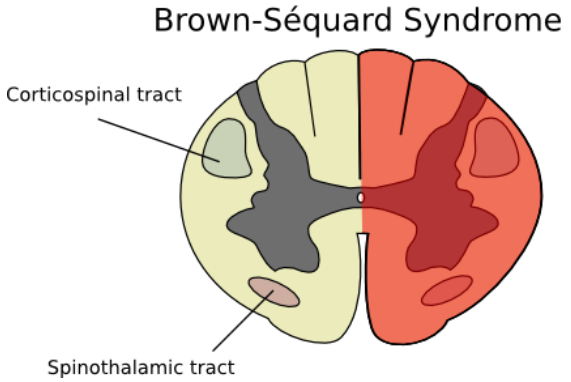
- Anterior spinal artery syndrome:
  - Results in loss of spinothalamic sensation and motor function, with sparing of dorsal column sensation.

- A similar dissociated pattern of pain and temperature loss and sparing of dorsal column sensation occurs in syringomyelia.

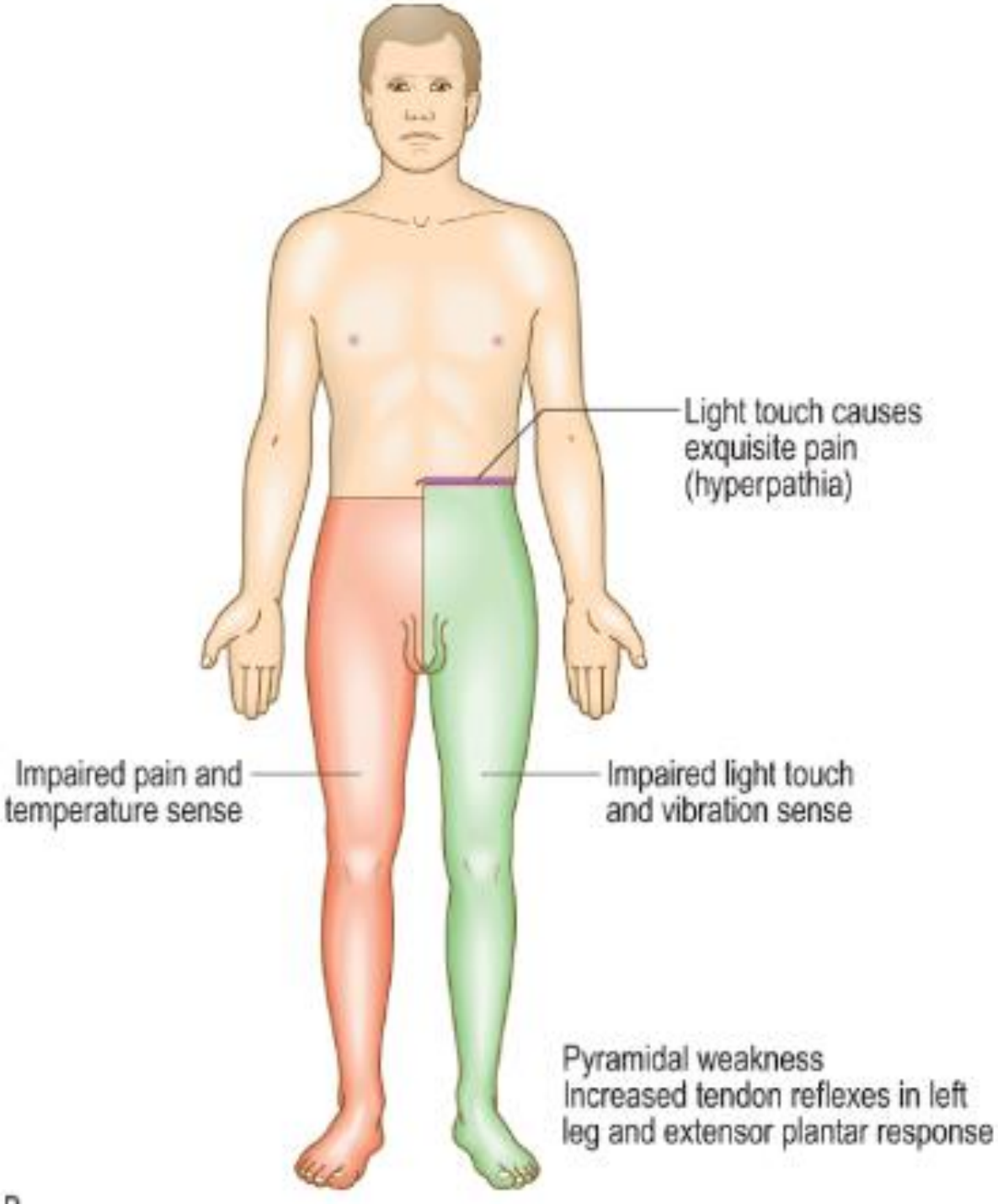
- Brown-Séquard syndrome
  - When one-half of the spinal cord is damaged.
  - This is characterized by ipsilateral motor weakness and loss of vibration and joint position sense, with contralateral loss of pain and temperature







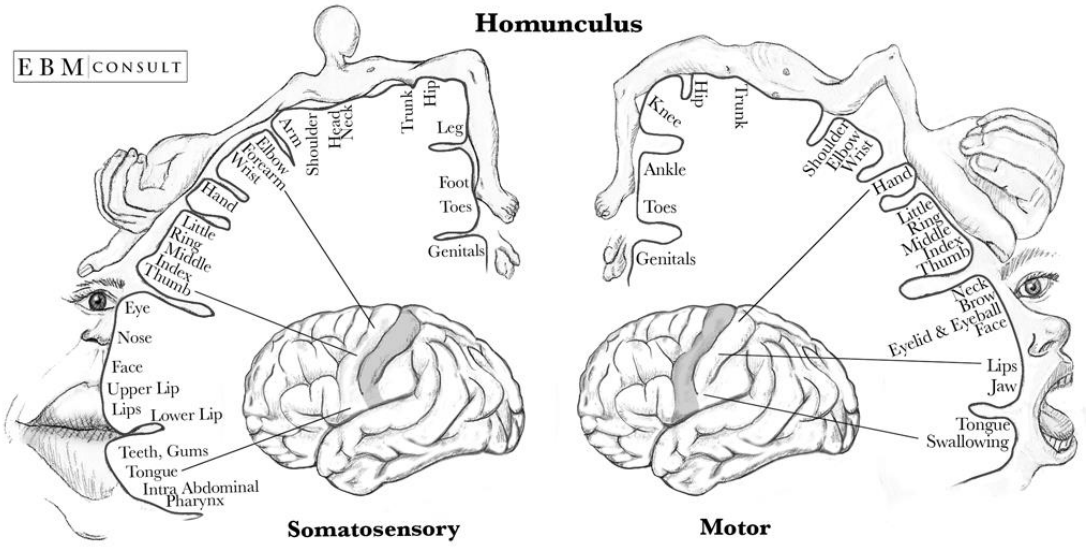
Brown-Séquard syndrome

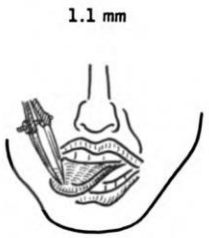


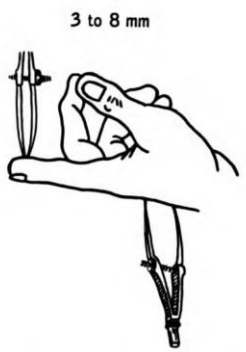
Intracranial

- Thalamic lesions may cause a patchy sensory impairment on the opposite side with unpleasant, poorly localized pain, often of a burning quality

- Cortical parietal lobe lesions typically cause sensory inattention but may also affect joint position sense, two-point discrimination, stereognosis (tactile recognition) and localization of point touch.

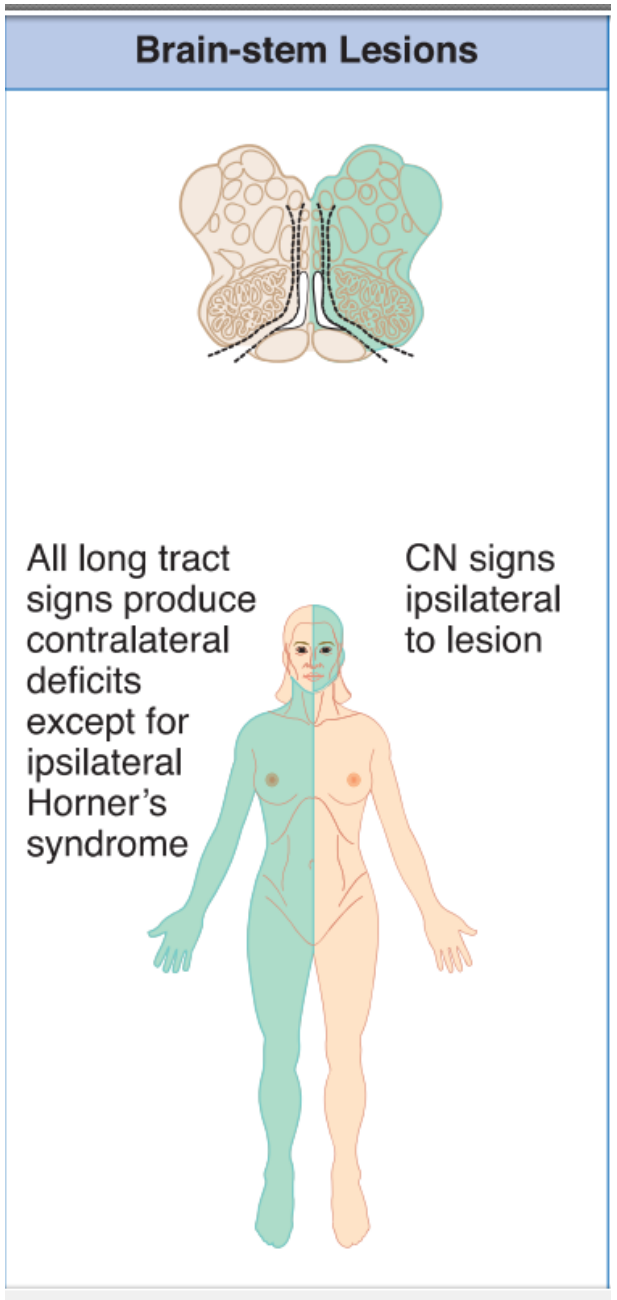






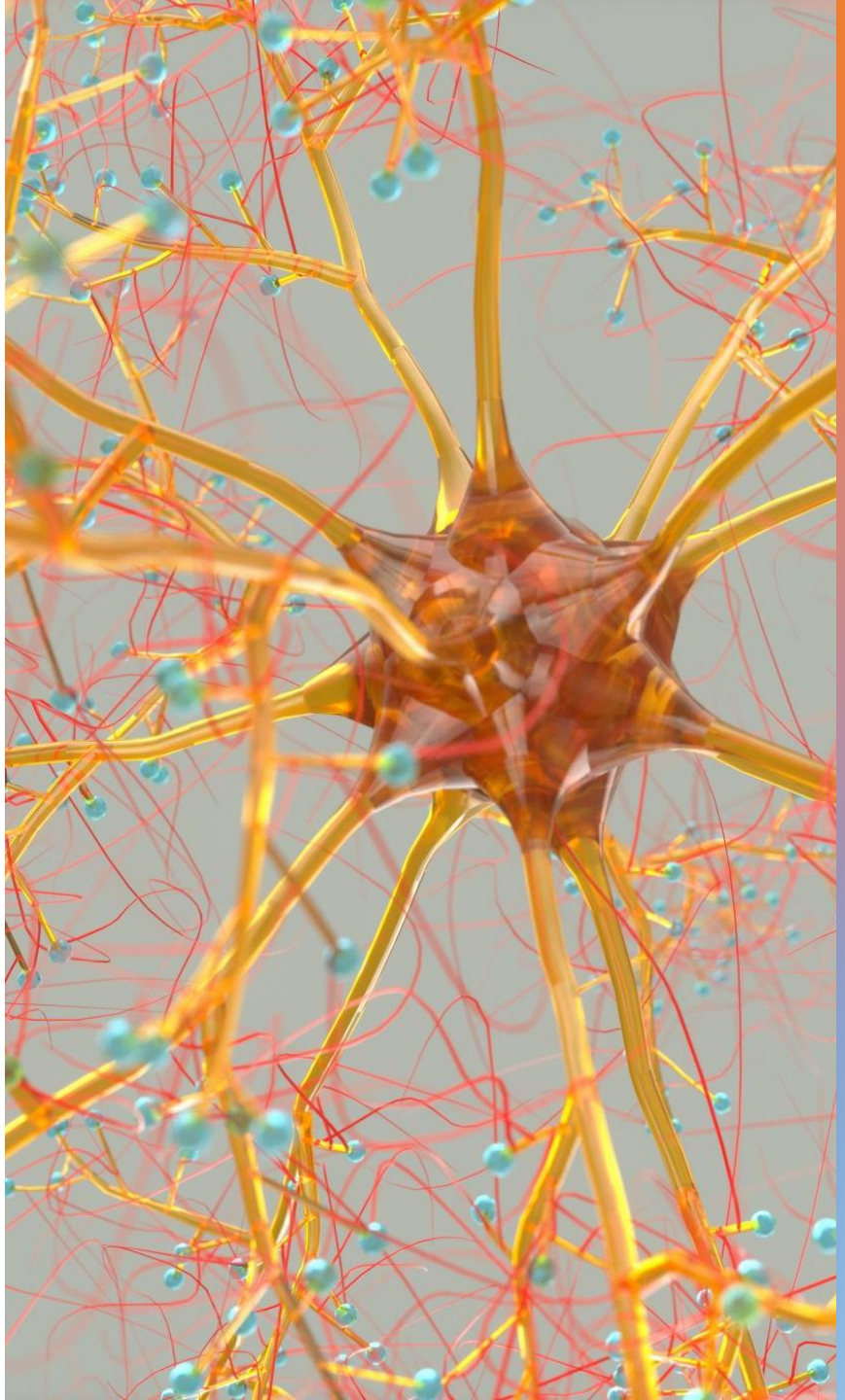
Brain stem lesions

- Lower brainstem lesions may cause ipsilateral numbness on one side of the face (V nerve nucleus) and contralateral body numbness (spinothalamic tract).



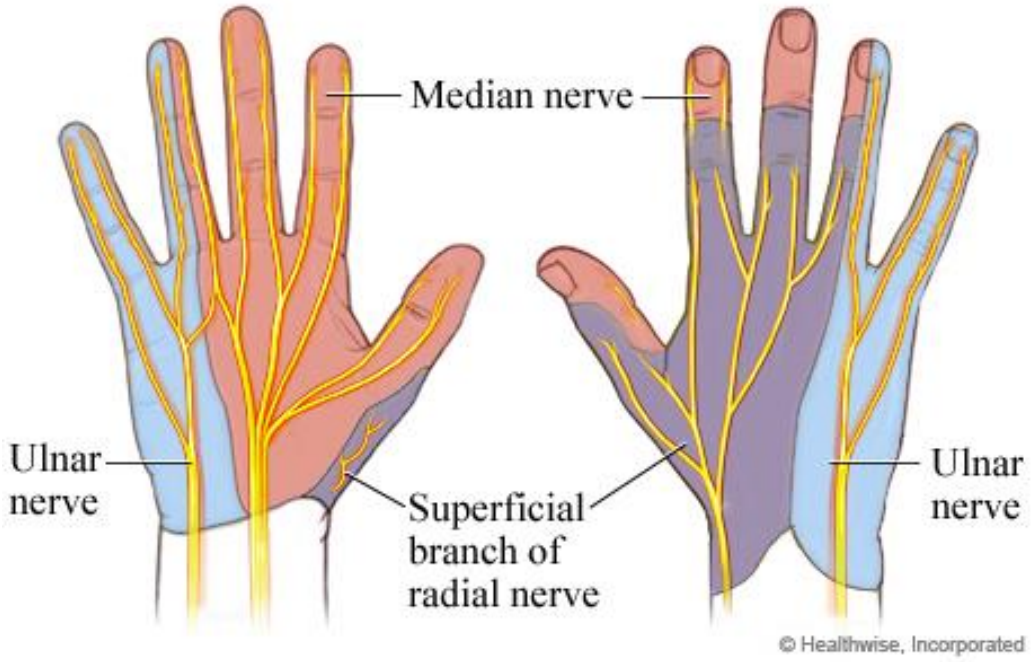
Peripheral nerves Examination

Your Famous Rock, Paper, Scissors!



Sensory examination of the hand

- Test for altered sensation over the hand involving:
  - The thumb
  - Index and middle fingers
  - The lateral half of the ring



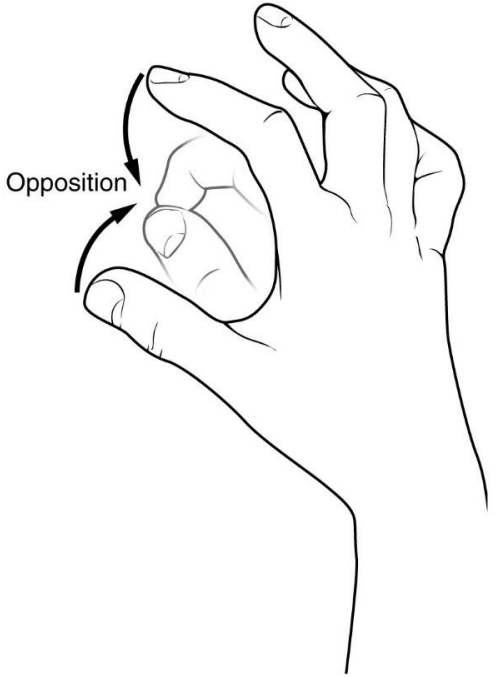
Median

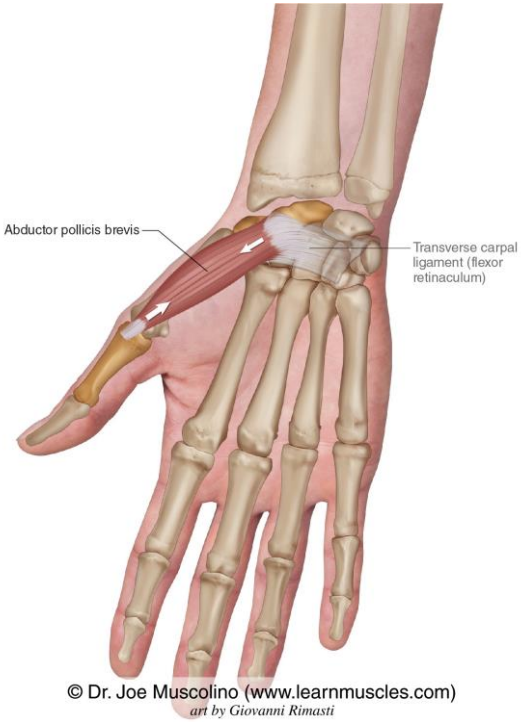
Ulnar

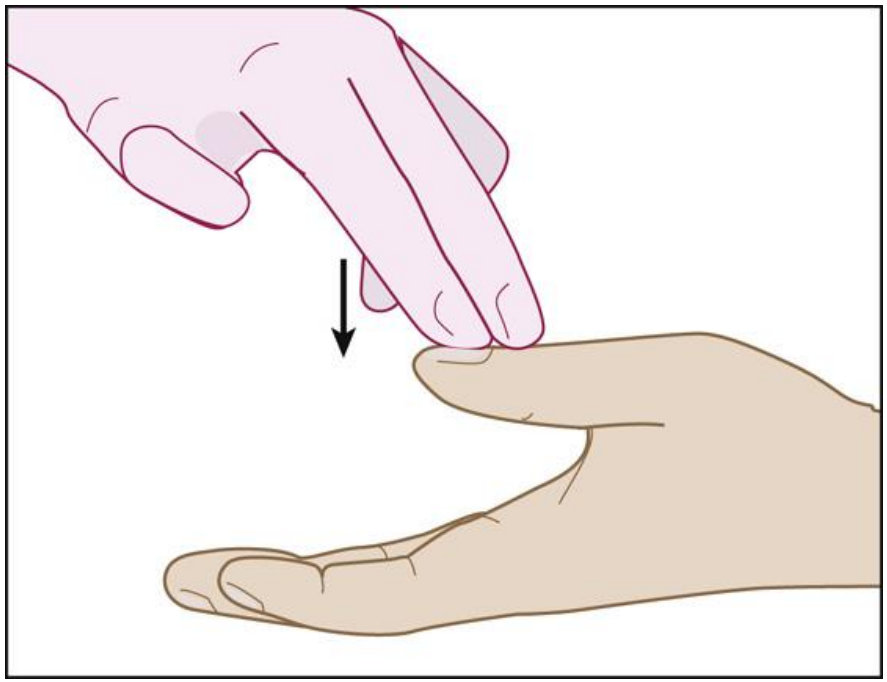
Radial

Median nerve

- Wasting of the thenar eminence
- Testing thumb abduction (abductor pollicis brevis): patient's palm up on a flat surface. Ask him to move the thumb vertically against your resistance
- Testing opposition (opponens pollicis): Ask him to touch the thumb and ring finger together while you attempt to pull them apart

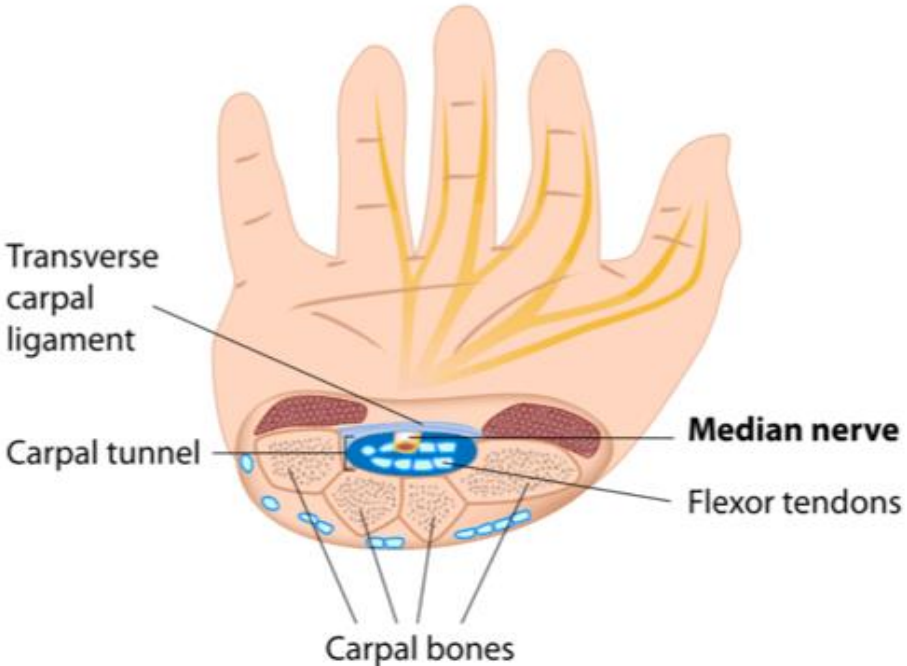






Carpal Tunnel Syndrome (CTS)

- It is the most common entrapment neuropathy
- This may be compressed as it passes between the flexor retinaculum and the carpal bones at the wrist
- Initially produces sensory symptoms

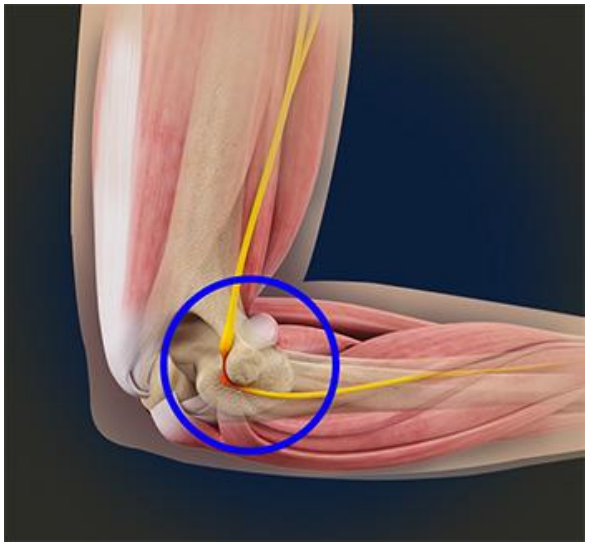


11.30 Common features of carpal tunnel syndrome

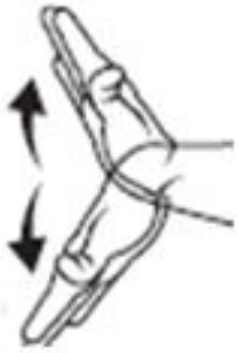
- More common in women
- Unpleasant tingling in the hand
- May not observe anatomical boundaries, radiating up the arm to the shoulder
- Weakness uncommon, but affects thumb abduction if occurs
- Symptoms commonly occur at night, wakening patient from sleep
- The patient may hang the hand and arm out of the bed for relief
- Thenar muscle wasting (in longstanding cases)
- Associated with pregnancy, diabetes and hypothyroidism

Ulnar nerve

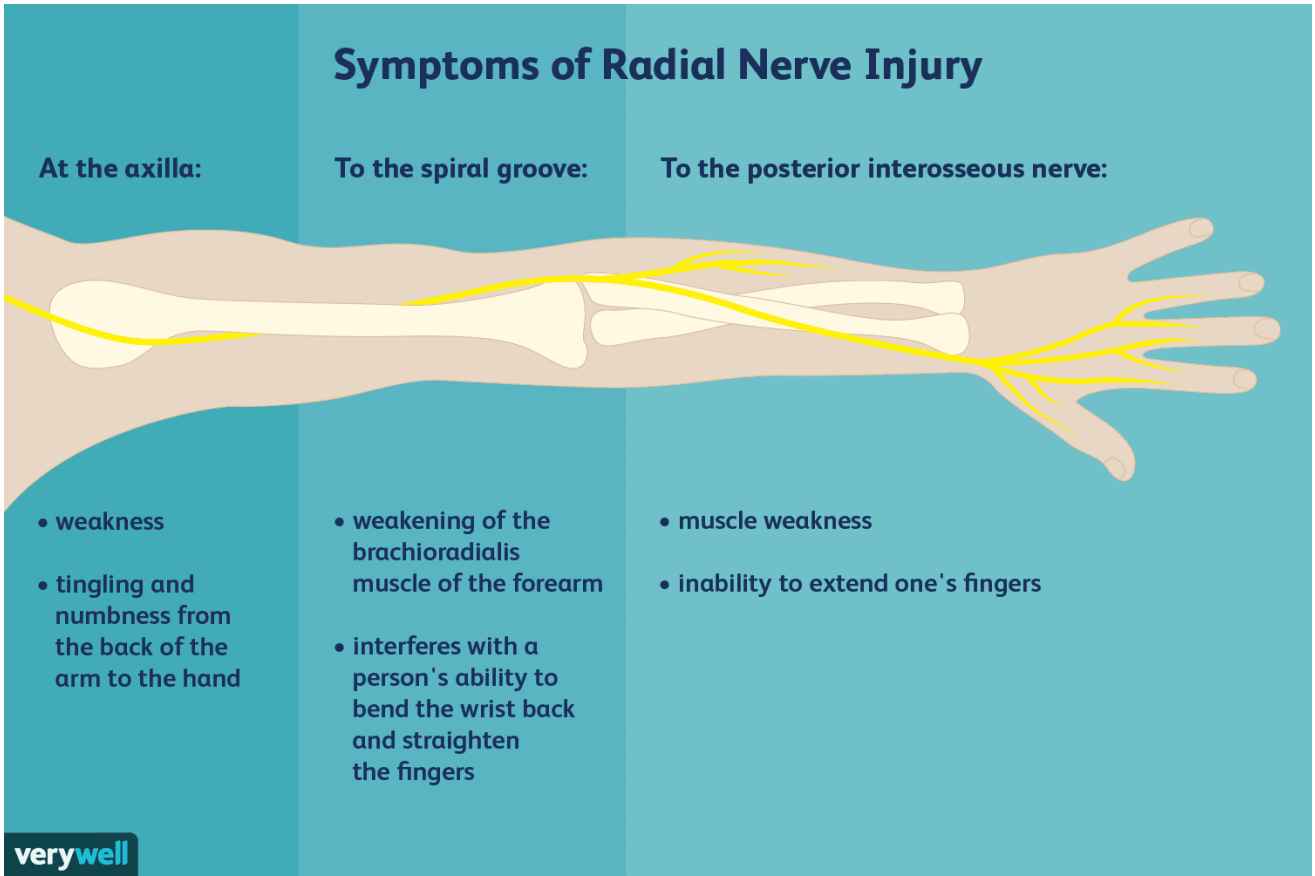
- Look for wasting of interossei (dorsal guttering).
- Fingers abduction: Patient's fingers on a flat surface and ask him to spread the fingers against resistance from your fingers.
- Fingers adduction: Placing a card between the patient's fingers and pulling it out using your own fingers.
- Examine the elbow (the commonest place of entrapment)
- Note any scars or other signs of trauma.
- feel for the nerve in the ulnar groove.



Radial nerve



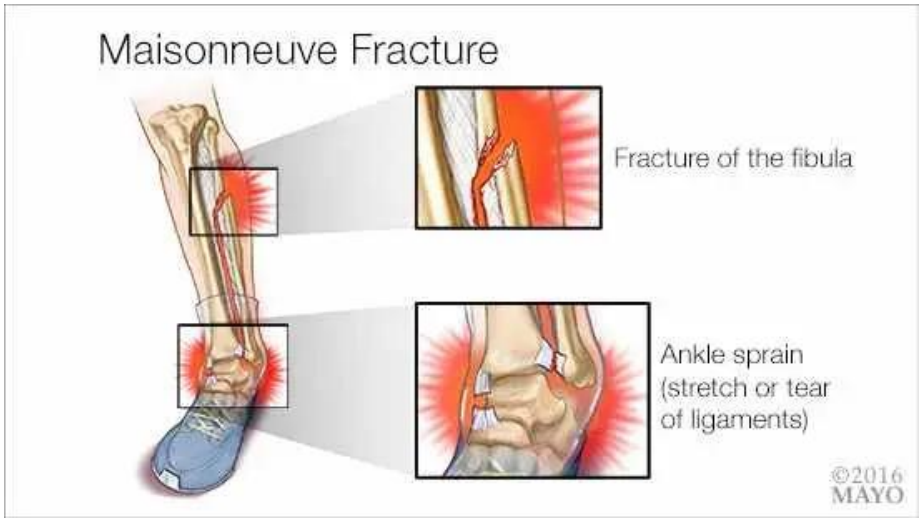
- Test for weakness of arm and forearm extensors (triceps, wrist, and fingers)
- Look for sensory loss over the dorsum of the hand
- loss of triceps tendon jerk.

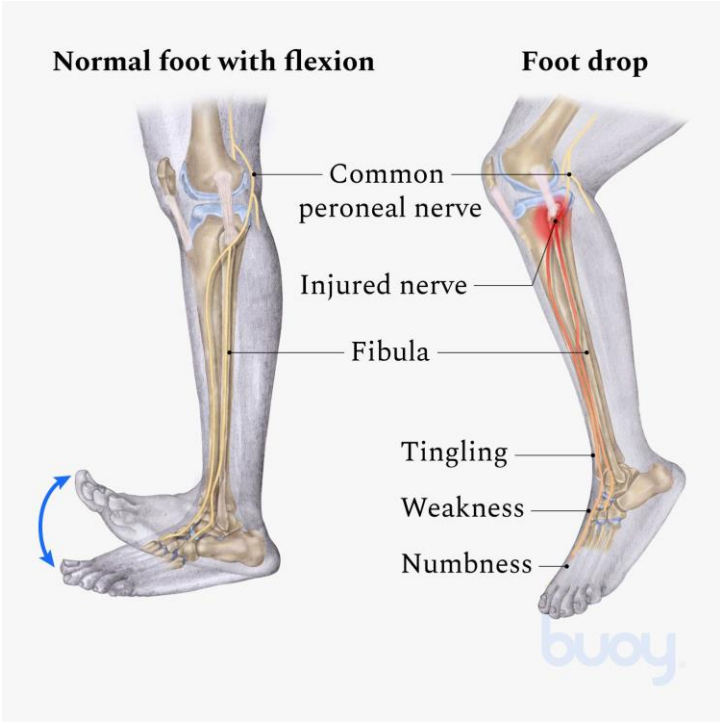


Common Peroneal Nerve

- This typically presents with foot drop.

- It may be damaged in fibular head fractures, or compressed particularly in immobile patients, or as a result of repetitive kneeling or squatting.

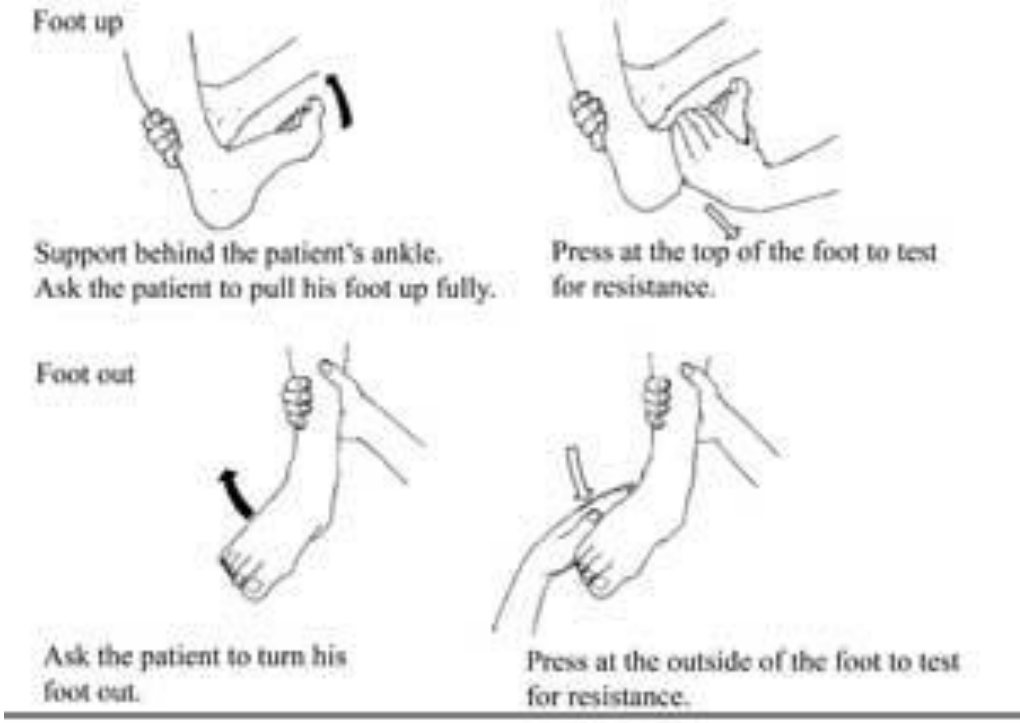




Examination

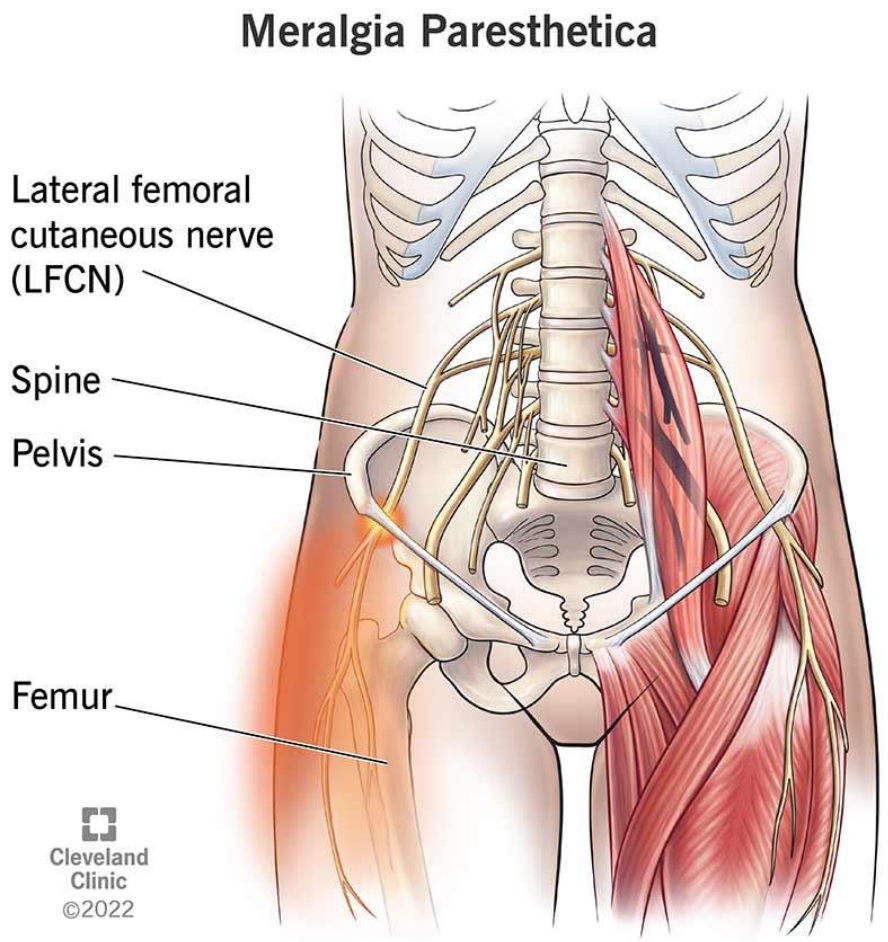
- Test for weakness of ankle dorsiflexion and eversion. Inversion will be preserved.

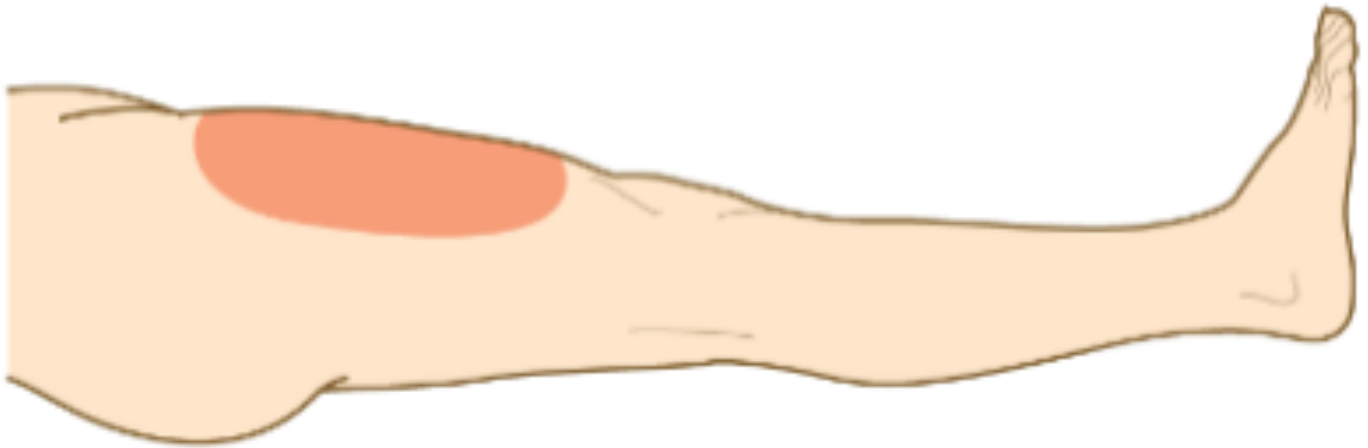
- Test for sensory loss over the dorsum of the foot



Lateral cutaneous nerve of thigh

- This purely sensory nerve may be compressed as it passes under the inguinal ligament, producing paraesthesia in the lateral thigh (meralgia paraesthetica





you make me so proud