MOTOR FUNCTION

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1. Wasting
2. Involuntary movements
3. Tone
4. Posture
5. Power
6. Coordination
7. Reflexes: tendon reflexes, cutaneous reflexes
8. Neck and trunk
9. Gait and stance

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

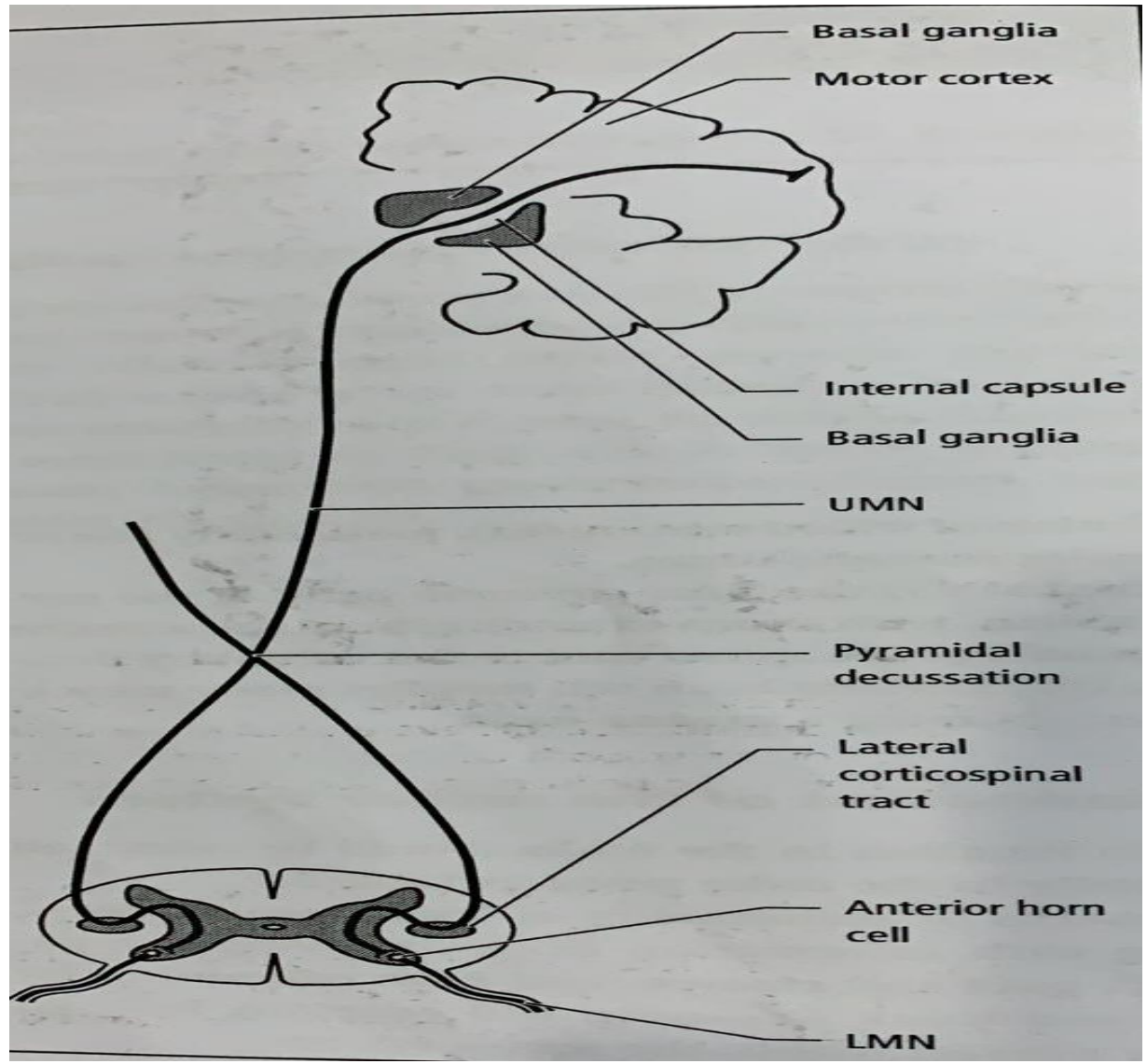
And “ feedback” via sensory pathways, particularly conveying information about joint position

UMN and LMN

There are many motor pathways descending from the cerebral cortex and brainstem

However, for the purpose of classifying disorders of voluntary movement, the UMN may be considered synonymous with neurons whose cell bodies are in the motor cortex and whose axons run in the corticospinal(pyramidal) tracts to synapse with anterior horn cells(Fig)

These neurons may be considered the anatomical substrate for the initiation of willed movements, particularly fine or complex manipulations


Basal ganglia
Motor cortex
Internal capsule
Basal ganglia
UMN
Pyramidal decussation
Lateral corticospinal tract
Anterior horn cell
LMN

The LMN is the ‘ final common path’ of the motor system, with axons extending from the anterior horn cells of the spinal cord to the voluntary muscles

One anterior horn cell supplies many muscle fibres-forming a motor unit

Examination of the motor system

Motor function in the limbs should be examined in the following order:

- Wasting
- Involuntary movements
- Tone
- Posture
- Power
- Coordination
- reflexes

Patterns of abnormality of these 7 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

1. Wasting

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

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

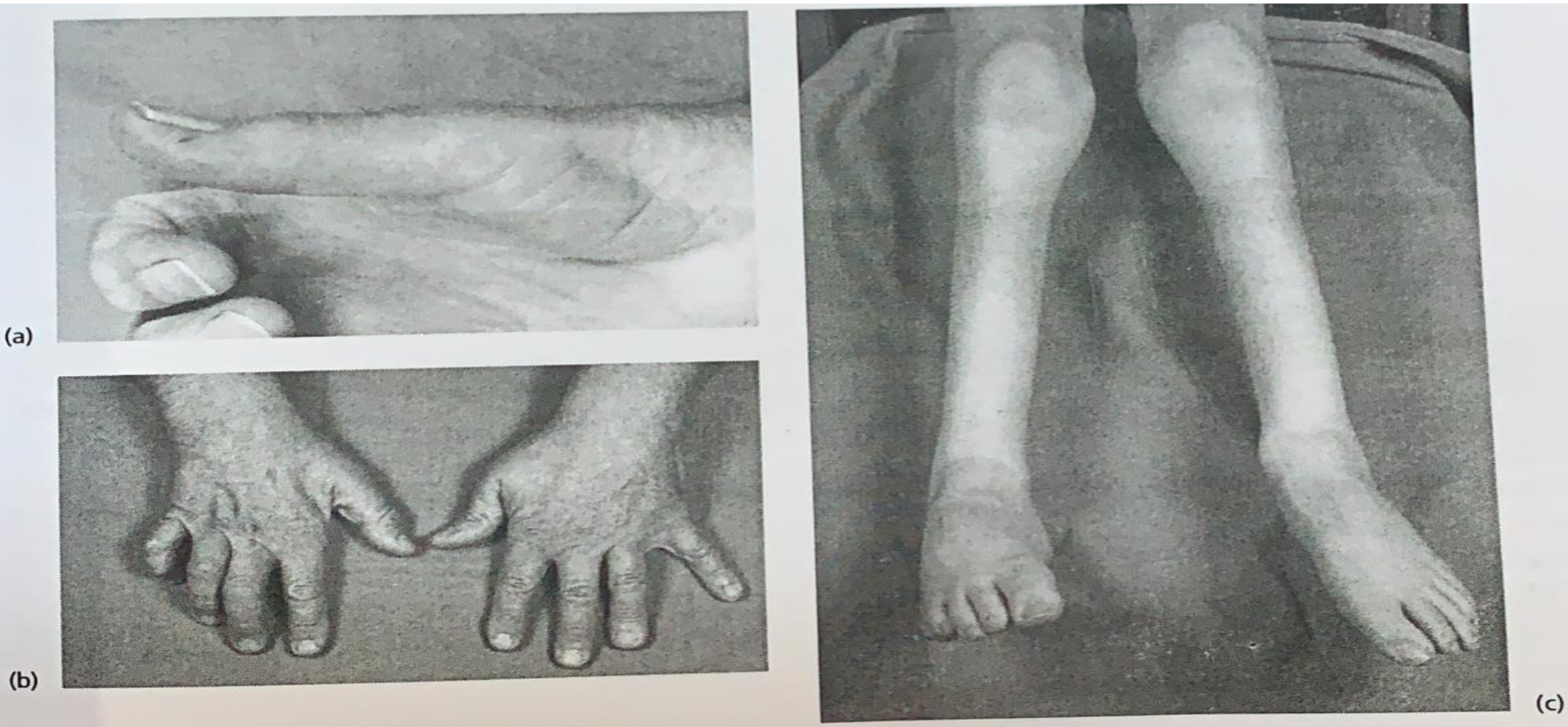
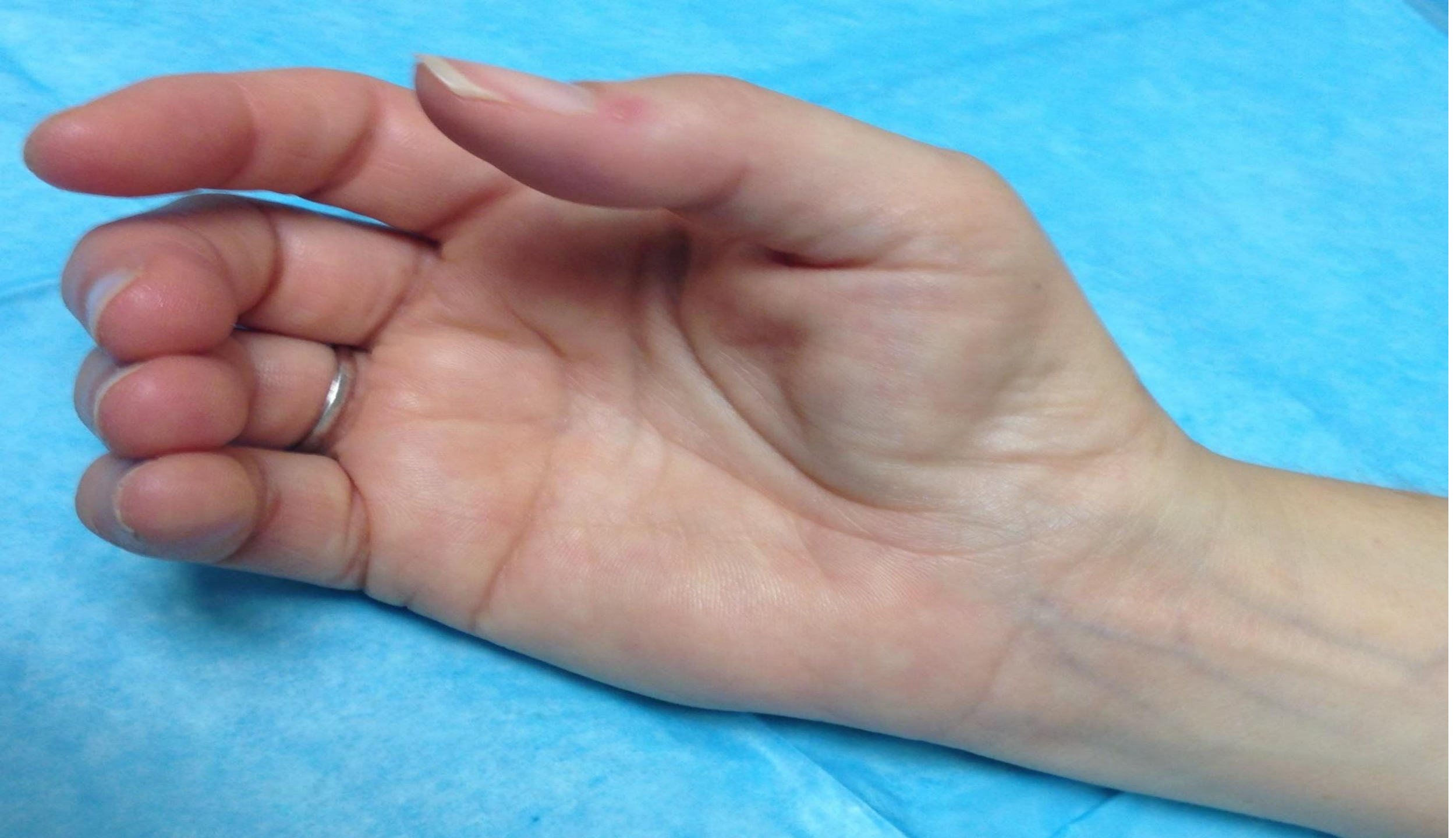
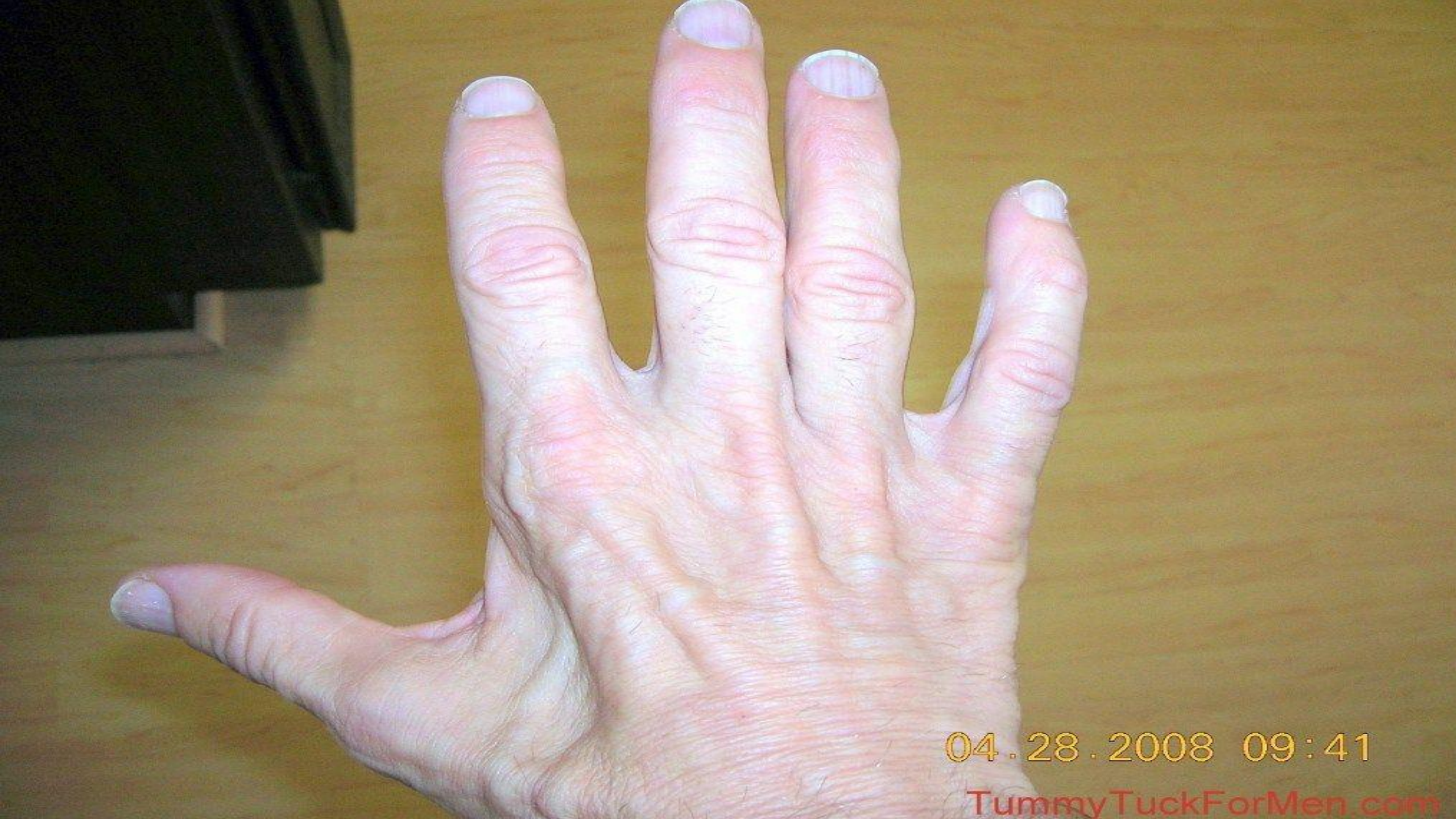


Figure 5.2 Common patterns of neurogenic wasting. (a) Atrophy of the thenar eminence. (b) Wasting of the interossei; the affected right hand (which is also clawed) may be compared with the left, which is normal. (c) Severe distal lower limb wasting. With milder degrees of wasting of tibialis anterior, 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).

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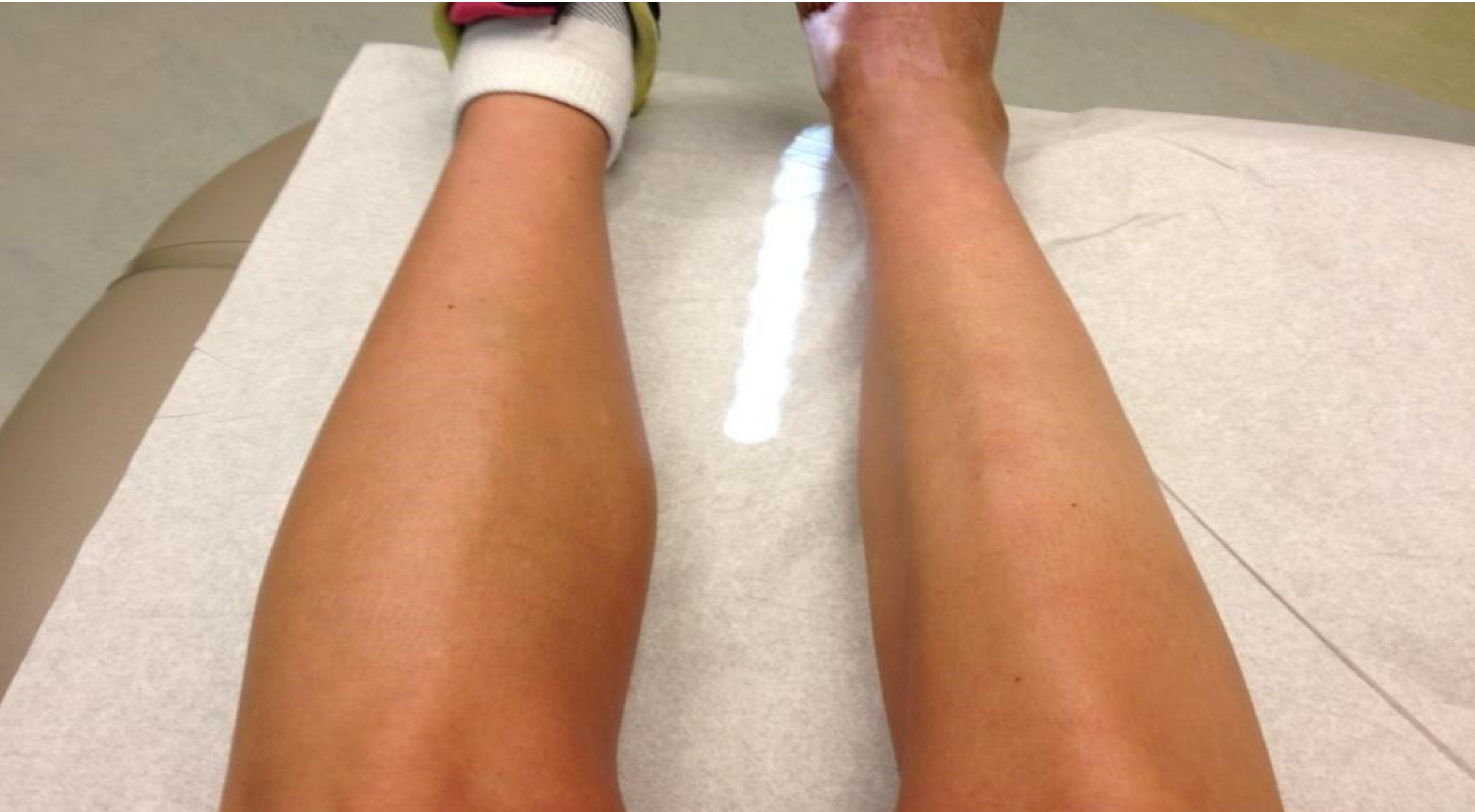


2. Involuntary movements

Fasciculations are brief , irregular twitching movements visible through the skin and occurring within a muscle belly

They are insufficiently powerful to achieve movement around the joint served by the muscle, except sometimes in the hand

They indicate an LMN lesion, generally proximal and severe , especially at anterior horn cell level

Some benign fasciculations, particularly in the calf muscles, are of no pathological significance

Other involuntary movements are of greater amplitude and often signify disease of the extrapyramidal system

3. Tone

Muscle tone may be defined clinically as the resistance detected by the examiner on passive movement of a patient's joints, hence passive stretch of the muscles

Some resistance is observed in normal individuals, but it may be increased or decreased by disease(hyper- and hypotonia, respectively)

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

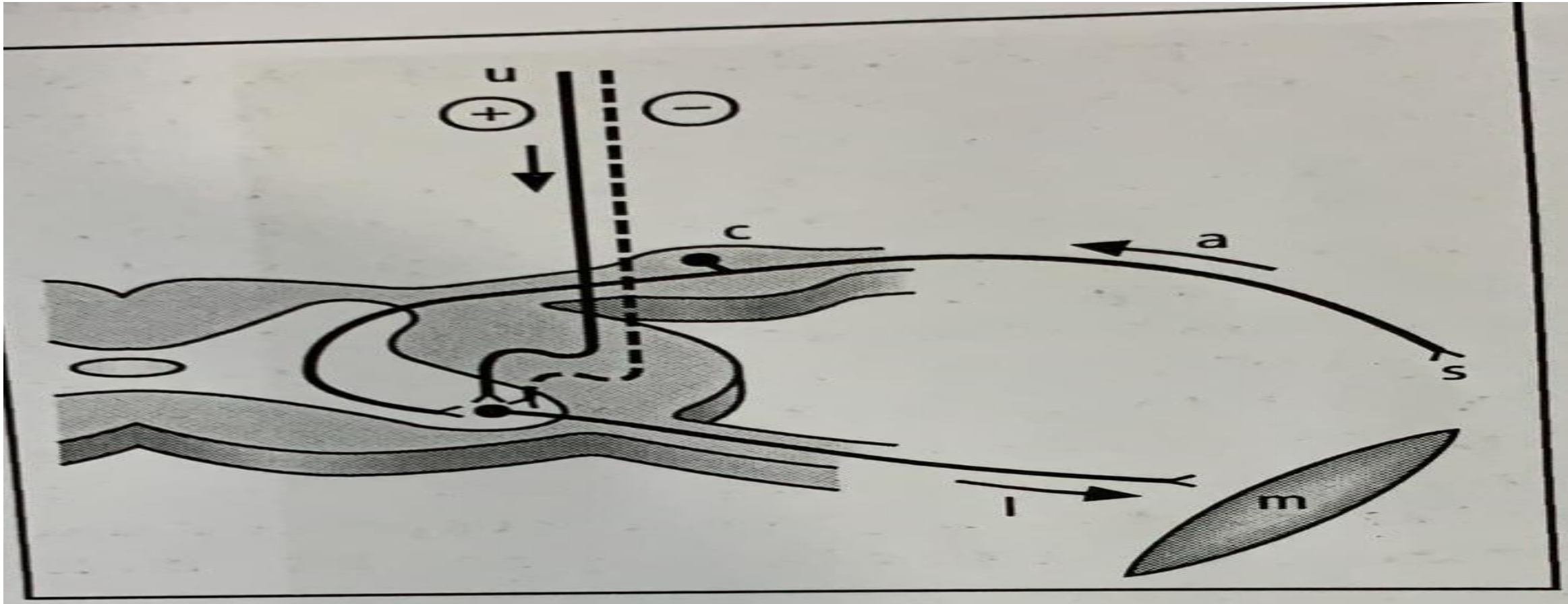


Figure 5.4 The stretch reflex. s, stretch receptor; a, afferent (sensory) neurone; c, cell body of sensory neurone in dorsal root ganglion; l, LMN originating at anterior horn cell of spinal cord; m, muscle; u, UMN; arrows indicate direction of impulse traffic; +, excitatory impulse in UMN; —, inhibitory 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)

4. Posture

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)

5. Power

Power is assessed clinically by grading the patient's ability to contract a muscle voluntarily against gravity and against resistance provided by the examiner

The Medical Research Council scale is used most commonly in the UK:

0 no contraction

1 flicker or trace of contraction

2 active movement with gravity eliminated

3 active movement against gravity

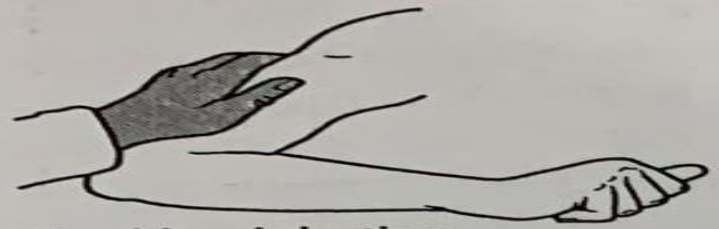
4 active movement against gravity and resistance

5 normal power

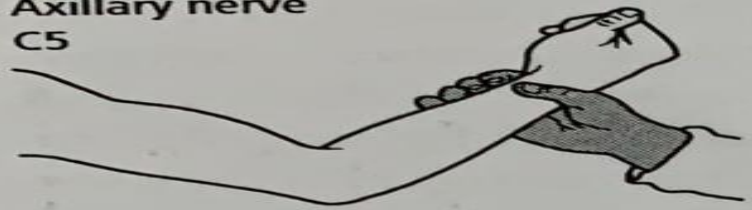
The scale is at best semiquantitative, particularly as much muscle weakness(paresis) in clinical neurology falls within 3-5 range, where it is often necessary to make further subjective subdivisions i.e 4-, 4 and 4+, denoting severe, moderate and mild weakness respectively

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



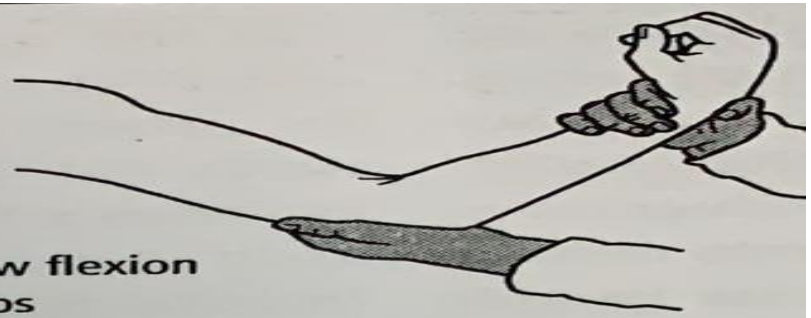
Shoulder abduction
Deltoid
Axillary nerve
C5



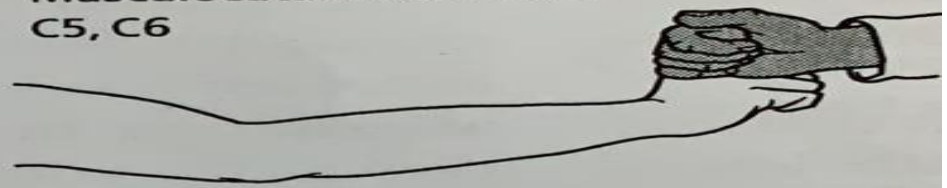
Elbow extension
Triceps
Radial nerve
C7

Finger extension
Extensor digitorum
Radial nerve
C7

Thumb abduction
Abductor pollicis brevis
Median nerve
T1



Elbow flexion
Biceps
Musculocutaneous nerve
C5, C6



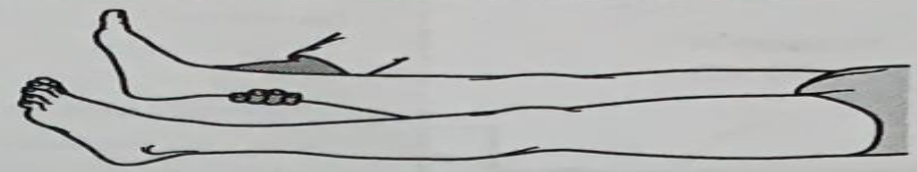
Wrist extension
Extensors carpi radialis,
C6 and ulnaris, C7,
Radial nerve

Wrist flexion
Flexors carpi radialis,
C7, median nerve and
ulnaris, C8,
ulnar nerve

Finger flexion
Flexors digitorum
profundus and
superficialis
Median and ulnar nerves
C8

Finger abduction
Dorsal interossei
Ulnar nerve
T1

Hip flexion
Iliopsoas
Lumbar plexus and
femoral nerve
L1/L2



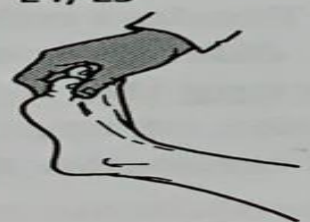
Hip extension
Gluteus maximus
Inferior gluteal nerve
L5, S1, S2

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

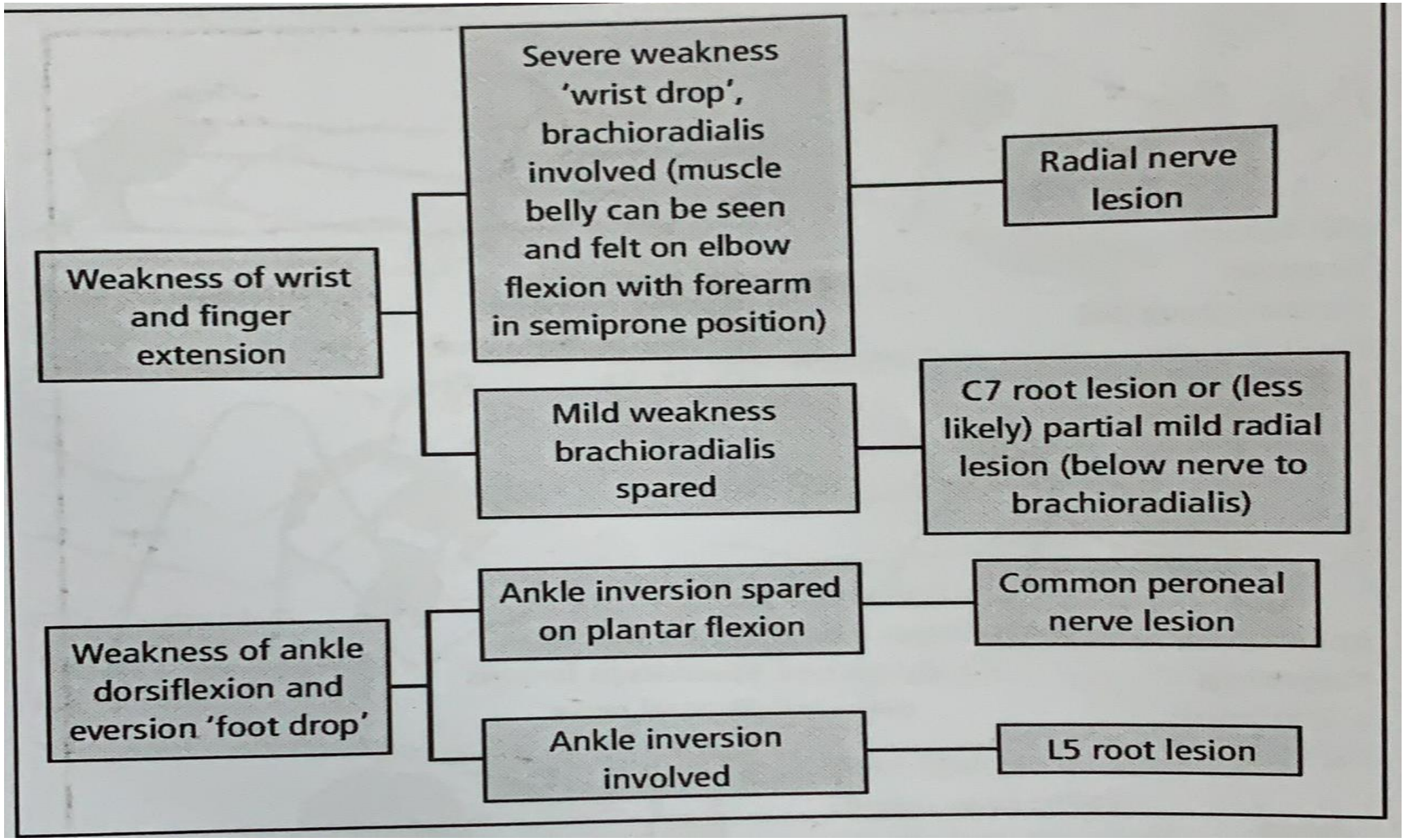
Ankle eversion
Peronei
Superficial peroneal nerve
L5, S1

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)

6. Coordination

Lack of coordination , or ataxia, is often considered synonymous with cerebellar disease

But, as previously stated, coordinated movements requires the normal action of all components of the nervous system and of parts of the sensory system, particularly joint position sense

Thus, loss of position sense may lead to a sensory ataxia. In the hand, this may have as damaging an effect on useful movement as severe muscle weakness

Formal tests of coordination in the limbs may, however, provide localizing information on cerebellar disease, the lesion generally being in the cerebellar hemisphere on the same side as the abnormal sign

In the upper limb, the cardinal test of coordination is the finger-nose-finger test, where the patient moves his or her index finger backwards and forwards from his or her nose to the examiner's finger

Cerebellar disease leads to inaccuracy in this test (past-pointing) because of the inability to judge distances(dysmetria)

As the finger approaches the target,it may oscillate increasingly wildly (intention tremor)

An alternative test is to ask the patient to perform rapid alternating movements(e.g. by tapping the dorsum of one hand with the palmar and then the dorsal aspect of the fingers of the opposite hand repeatedly), which may be jerky and inaccurate in cerebellar disease

(dysdiadochokinesis)

Dysmetria may also be assessed by gently tapping the patient's outstretched hand .Rather than immediately returning to the initial position, the patient's arm may overshoot and oscillate a few times

(cerebellar rebound)

In the lower limbs, ataxia may be detected in the heel-knee-shin test, the patient being asked to place one heel on the opposite knee then slide it accurately down the shin

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

7. Reflexes

A) Tendon reflexes

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

Sometimes a reflex that initially appears absent may be obtained by asking the patient to clench his or her teeth(for upper limb reflexes) or to interlock the fingers of the right hand with those of the left then try to pull the hands apart(for lower limb reflexes, Jendrassik's manoeuver), at the same time as the examiner strikes the tendon

This phenomenon of reinforcement is due to such manoeuvers increasing the sensitivity of the stretch receptors throughout the body

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

Clonus is a physical sign most often elicited at the ankle where sudden but maintained dorsiflexion by the examiner(with the patient's knee also partially flexed) produces rhythmical repetitive alternating plantar flexion and dorsiflexion

This is also due to loss of supraspinal inhibition, the sharp muscle stretching, leading to oscillation within the circuit of the reflex arc

Clonus may be sustained or may persist for only a few ‘beats’

It may be present in normal individuals, particularly if symmetrical in duration

It is of pathological significance if asymmetrical or if there is sustained symmetrical ankle clonus in the presence of other UMN signs

Clonus at sites other than the ankles (knees, fingers) is also generally pathological

The grading of tendon reflexes is usually represented symbolically as:

+++ very brisk

++ brisk

+ present

+- with reinforcement

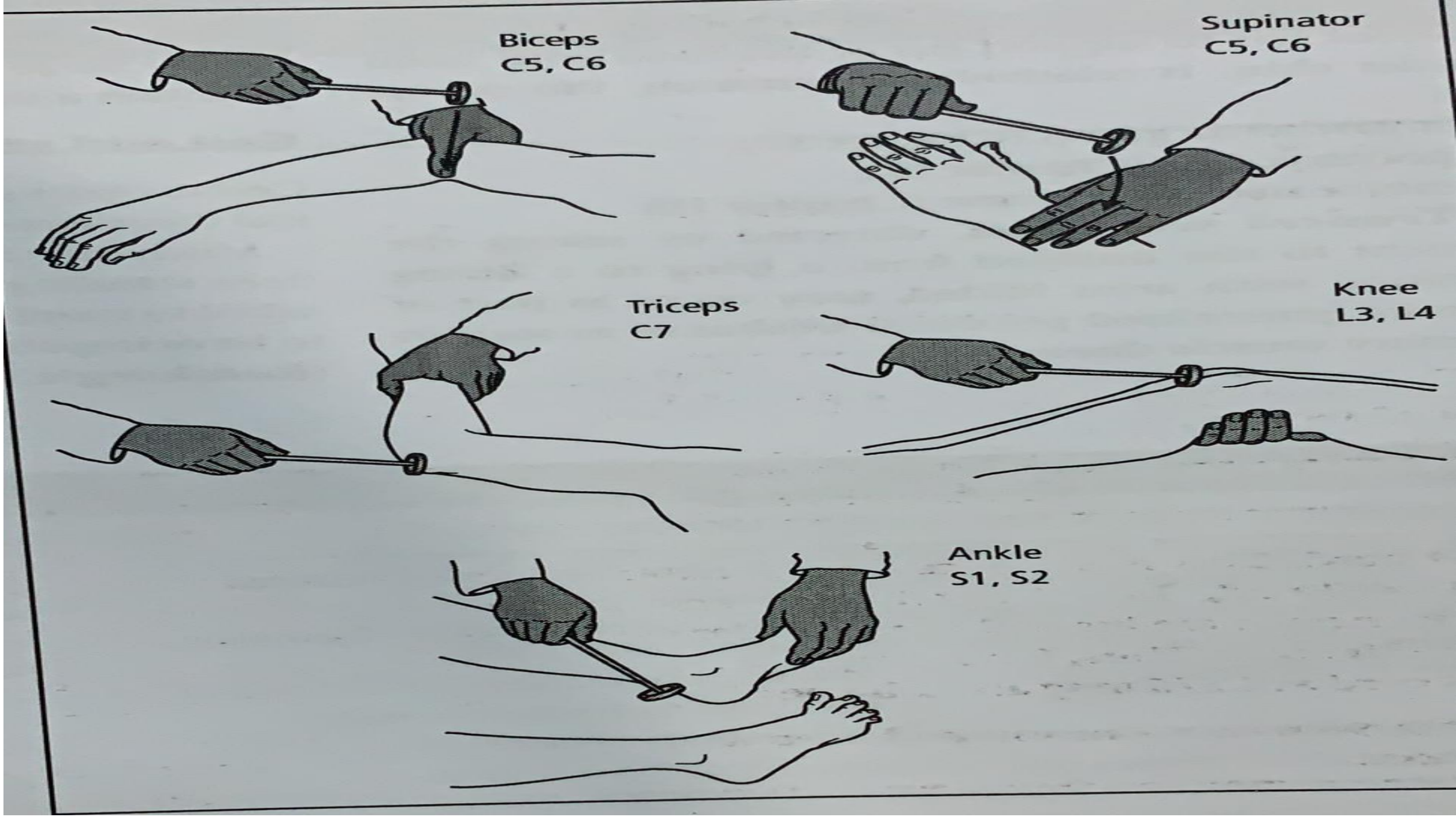
0 absent

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

B) 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

8. 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

9. 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

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