

CHAPTER 8 - WEAKNESS

A. ANATOMICAL DIAGNOSIS

Neurophysiology is again the key to understanding neuroanatomy

1. The **Motor Unit**:

The reflex aspect of this consists of alpha motor neurones in the anterior horn that supply a variable number of muscle fibres and form part of the basic spinal reflex arc, whose stimulus is stretch of the muscle spindle fibre within the main muscle concerned. These afferent fibres are arranged in an annulo-spiral configuration around the central part of the spindle, whereas the spindle's contractile elements are distributed more towards its ends. The afferent fibre from each muscle spindle synapses with an alpha motor neurone in the anterior horn cell of the corresponding segment of the spinal cord. On the motor side, muscle spindles are also innervated by so-called gamma motor neurones which lie adjacent to but separate from the alpha motor neurones in the anterior horn. Gamma motor neurones are innervated from higher centres, especially the extra-pyramidal system.

The above allows us to explain a number of aspects of muscle contraction and its neurological control. Thus, when the muscle belly is stretched, the annulo-spiral fibres around the muscle spindle are also stretched, and this activates the alpha motor neurone to reflexly contract the corresponding main fibres of the muscle concerned.

Because the annulo-spiral fibres are placed centrally around the relatively non-contractile part of their muscle spindle, they can also be stretched and made to discharge by muscle spindle contraction, which fattens this central non-contractile part. In this way, not only stretch, but also muscle spindle contraction can fire off afferent nerve fibres from the muscle spindle. This explains the so-called "gamma loop", in which gamma motor neuron excitation (from higher centres) in the anterior horn leads to the following sequence: muscle spindle contraction - muscle spindle afferent fibre discharge - alpha motor neurone firing. (Figure 1c). This is an important reflex. Through it, a command signal for muscle shortening directed to the gamma motor neurone will cause reflex contraction of the main muscle itself, so that the gamma loop can act as a sort of servo-system initiating main muscle belly contraction.

In addition, because the muscle spindles lie among other ordinary muscle fibres and in parallel with them, they are relieved of tension when the surrounding muscle contracts (and become stretched when it relaxes) so that the signal transmitted from the spindles varies depending on the ratio of spindle contraction to that of surrounding muscle fibres. For example, if the spindle is contracted relatively more than the surrounding muscle, its afferent firing rate is increased to cause contraction of the surrounding muscle. If, on the other hand, the spindle is slack relative to the surrounding muscle, its firing rate decreases, and consequent inhibition of the "gamma loop" reflex produces relaxation of the surrounding muscle, thus restoring its resting state to a greater length. Hence,

spindle receptors act as detectors of discrepancies or error between length of spindles and that of surrounding muscle fibres during any state of dynamic change in muscle belly length. In this way, the gamma efferent activity helps determine the final length of the muscle fibres surrounding the spindle.

The essential point to grasp is that both muscle stretch (which lengthens the intra-fusal muscle spindle fibres) and a gamma efferent stimulation (which contracts and fattens them) stretch the centrally-placed annulo-spiral afferent fibres around the muscle spindle itself, and cause contraction of the main muscle.

There are other influences on the main alpha motor neurone, both local (inhibitory) from the so-called Renshaw cell (an internuncial neurone) at the level of the spinal reflex arc, and others arising from higher centres which (espec. cerebellum) can activate motor neurones to agonist muscles and inhibit alpha motor neurones to antagonist ones at the same time, so as to ensure co-ordinated movement.

2. The motor cortico-spinal tract

The descending cortico-spinal motor pathways.

The most important function of the pyramidal tract is control of fine, skilled finger and wrist movement, and it does this by connecting with the alpha motor neurones concerned direct. Note that some 10% of fibres do not participate in the decussation of the pyramids in the hind brain medulla, and these may be important in recovery from hemiparesis.

Important though the pyramidal tract are for movement, they is not the only descending motor pathway, nor are their functions exclusively motor. And even among cortical motor functions, not all fibres supply the alpha motor neurones direct, but only indirectly through either subcortical motor nuclei in the brain (e.g. the caudate nucleus, red nucleus etc.) or through the internuncial neurones adjacent to, and connected with, the anterior horn alpha motor neuron cells.

Motor control.

In contrast to the fine control pyramidal tracts exert over hand and finger movements, the trunk and girdle muscles are much less directly innervated by the pyramidal tract, and indeed receive a major input via sub-corticospinal pathways arising from the brain stem. These fibres allow automatic maintenance of body posture and the control of basic trunk movements.

Another two points. First, the system of internuncial neurons lying adjacent to the alpha motor neuron is important in processing descending messages from higher up and thereby ensuring that the appropriate pattern of protagonistic motor neurones is recruited while antagonist neurones are simultaneously inhibited. Second, in all movements there is co-activation of the alpha and gamma motor neurones so that the sensitivity of the muscle spindles is maintained during contraction of the main muscle mass.

Finally, much normal motor function can be controlled at a very basic level. Postural control particularly involves proprioceptive, vestibular, cerebellar and brain stem nuclear mechanisms. Walking, in particular, can be performed at a surprisingly low level, once learned, with much of its even detailed programme in the adult eventually being built into the spinal cord and hind brain.

B CLINICAL PROBLEMS

1. Generalised Weakness

(a) First distinguish true generalised muscular weakness from the more vague symptoms of lethargy, malaise and tiredness. The latter are non-specific, and may be associated with either organic or psychogenic disturbances, with or without subjective muscular weakness.

In particular, patients with anxiety/depression and other symptomatic neuroses often have generalised lassitude, and may complain of fatigue or of feeling weak, but there is usually very little evidence for this on objective testing, and no associated CNS signs such as wasting and depression of reflexes (reflexes are usually brisk in patients with anxiety). Indeed in such patients there are not only no positive organic features in the CNS but there are usually positive features of the anxiety (symptoms of "shortness of breath", on observation often a sighing irregular hyperventilation, in severe cases even producing respiratory alkalosis, reduced ionised calcium and therefore "pins and needles" in the fingers and around the mouth; also cold clammy hands, rapid fine tremor of the outstretched hands, tachycardia, restlessness).

In some patients with neurosis, particularly where there are hysterical features, weakness may be more prominent, but the clues that it is not organic are its variable degree during physical examination, and your difficulty of relating it to any segmental innervation; in addition, so-called "conversion" hysteria is classically associated with the patient being indifferent to what appears to be a severe weakness; and in the background history there is usually evidence of some psycho-social maladjustment.

However, unless you have positive evidence of anxiety/depression/hysteria, do not lightly assume that any generalised lethargy (or for that matter "weakness" you do not understand) is psychogenic in origin. Vague symptoms may be the first presentation of real organic disease (e.g. vague weakness with muscular stiffness in polymyalgia rheumatica, multiple sclerosis).

Be particularly wary in patients who have lost weight, even without corresponding loss of muscle bulk and demonstrable muscular weakness, because though this may happen in the psychogenic illness, it is best to regard it as organic until proved otherwise. Not only this, but patients with neuroses do get real organic disease; indeed many think more often than usual (the so-called psycho-somatic or stress-induced organic disorders). "Chronic fatigue syndrome" lies somewhere in the borderland

between the psychogenic and the organic; it is probably 'functional' in some patients and post-viral in others. Some drugs also cause of lethargy/'weakness,' especially the statins

(b) Organic Causes of Generalised Weakness

Here, the weakness is objective, and is usually out of proportion to any general weight loss. Examine all muscle groups carefully to define the degree of weakness. Even when there is generalised weakness some muscle groups may be weaker than others, and this provides another example of diagnostic category overlap, because delineating the anatomical distribution of weakness may be very helpful in making our Clinical Pathological Diagnosis. For example, if the weakness falls more on the proximal muscles (history may suggest this by difficulty in climbing stairs, and examination may show difficulty in standing unaided from the squatting position), this suggests metabolic, endocrine or electrolyte disturbance (e.g. Cushing's syndrome, thyrotoxicosis, hypokalaemia, hyperkalaemia, hypercalcaemia, diabetes mellitus).

Of course, as with localised weakness, we must first distinguish whether the weakness is primarily muscular or neurological, and if the latter, whether it is of upper or lower motor neurone type

2. Localised Weakness

Hierarchic approach to Diagnosis

In defining Anatomy first ask BROAD questions:

(a) Is this primary neurological disease or primary muscle disease? (or even sometimes primary joint disease with secondary muscular weakness and wasting from disuse). In differentiating muscular from nervous disorders, note whether there are any sensory symptoms or signs, as these of course strongly point to a neurological disorder. On examination, always start by inspecting and palpating the limb(s) involved and watching the patient walk. Palpation of the muscles is particularly helpful in acute inflammatory conditions muscular conditions. Also, typically, hyporeflexia rather than a-reflexia is the hallmark of myopathic problems so that if the reflexes are absent, this points more to a neurological disease (of lower motor neurone type).

Practice point. In your hierarchic approach to anatomical diagnosis of muscular weakness, do not assume that you are dealing with a neurological problem until you have thought about other possibilities, particularly muscle disorders.

(b) Localising the site of weakness

If nervous system weakness, the next step is to determine whether you are dealing with an upper motor neurone lesion, or a lesion at or below the level of the anterior horn cell in the spinal cord.

(i) Upper Motor Neurone Lesions

This term is preferable to pyramidal tract lesion, because the signs traditionally described under this heading are not those of pure pyramidal tract involvement, but involve the subcortico-spinal pathways as well. We should speak of long motor tract signs in the same way. Most of these long tracts, although finishing on the alpha and gamma motor neurones in the anterior horn, are interrupted along the way by relays within the basal ganglia and brain stem nuclei, and even by spinal interneurons. It is really only the pyramidal fibres to the fingers that pass direct to the alpha neurones, and this accounts for the observation that individual movements of the fingers are most affected by lesions directly involving the cerebral cortex.

Characteristic signs of established upper motor neurone lesions are weakness (particularly involving the extensors of the upper limbs and the flexors of the lower limbs) and the development (over some weeks after an acute lesion) of spasticity (related to release of sub-cortical pathways from higher inhibition and consequent activation of the "gamma loop"), increased tendon reflexes (again due to release of the spinal reflex from higher inhibitory control by both cortical and subcortical centres), and the development of extensor plantar responses (really a withdrawal reflex present in the child but normally later inhibited by the development of descending motor control systems as the child learns to stand and walk).

Of course, having determined that the patient has an upper motor neurone lesion, we must go on to dissect hierarchically the neuro-anatomical level involved. The general principle is to look for other signs and symptoms and to see whether they point more to the cortex (i.e. other cortical signs such as dysphasia, dyspraxia, parietal neglect of the affected limb, sensory inattention); subcortical nuclei (tremor, involuntary movements, etc.); internal capsule (hemi-anaesthesia and/or hemianopia); brain stem (includes cranial nerve signs); or the spinal cord (involvement of structures near the pyramidal tracts, e.g. pain and temperature loss from involvement of the adjacent lateral spinothalamic tract).

(ii) Lower Motor Neuron Lesions i.e. lesions at or below the anterior horn cells in the spinal cord.

In most patients, the distinction between upper and lower motor neurone lesions can be made readily. Patients with lower motor neurone lesions usually have fairly marked weakness and, if it has been established for some time, associated muscle wasting from disuse. The tendon reflexes are usually depressed or absent, and the tone is flaccid.

As in the case of upper motor neuron lesions, we next need to dissect, hierarchically, the anatomical level at which the lower motor neurone unit is involved.

Anterior Horn Cell Motor Neuron Disease.

Fasciculation is characteristic of chronic motor neuron disease and arises from the spontaneous discharge of diseased alpha motor neurones, each of which supplies a whole motor neurone unit,

resulting in irregular, coarse contraction of many muscle fibres together, visible through the skin. Actually, sometimes these contractions can be quite gross, probably related to the formation of so-called "giant motor units" in chronic motor neurone disease i.e. the branching of remaining functional fibres from surviving anterior horn cell motor neurons to adjacent muscles originally supplied by now-degenerated neurons.

Acute anterior horn cell disorders include poliomyelitis, botulism, and tetanus poisoning.

Lesions Peripheral to the Lower Motor Neurone:

Most diseases in this category cause similar motor problems to motor neuron disease, except for the absence of fasciculation. Actually all denervated muscle fibres show 'denervation hypersensitivity' resulting in discharge which can be detected by EMG (fibrillation), but this discharge is due to uncoordinated contraction of individual muscle fibres rather than whole motor units, and as such is not usually visible to the naked eye.

Another distinguishing feature from motor neuron disease is that conditions involving the nervous system peripheral to the lower motor neuron usually have an element of sensory impairment. Working anatomically from the centre to the periphery, there are a number of conditions which may be associated with lower motor neuron weakness, including, in order, spinal cord lesions, diseases of the (motor and sensory) nerve roots just outside the spinal cord (radiculopathies), and more peripheral neuropathies, either generalised (polyneuropathy), or localised i.e. picking out one or more motor nerves (single or multiple mononeuropathies).

(iii). Spinal Cord Lesions are particularly important to bear in mind when you find sensory impairment, especially when there is a mixed motor picture of both upper and lower motor neurone involvement (also if there is bowel and/or bladder disturbance). With the slightest suspicion (e.g. bilateral weakness of the legs of mixed upper and lower motor neurone type) always strip the patient completely and examine for an upper level of sensory impairment.

Such a level would not only tell you that you are dealing with a spinal cord lesion, but from your knowledge of sensory and motor dermatomes, would define its site. If you think you are dealing with weakness in the distribution of the lower sacral nerve roots, examine carefully genital and peri-anal sensation and anal tone. Generally with spinal cord lesions, you will find upper motor neurone signs below the level of the lesion. However, this will, of course, not be so with lesions of the cauda equina, i.e. with any spinal lesion below the level of the first lumbar vertebra. With spinal cord lesions in the neck, you may find lower motor neurone signs in the upper limbs and upper motor neurone signs (from lateral cord compression) in the lower limbs. Once again look for a sensory level.

Be able to work out the effects of hemisection of the cord (Brown-Sequard syndrome) which may be caused by disease in one half of the cord.

In cervical cord lesions, two conditions should be differentiated, both of which can cause lower motor neurone signs in the upper limbs and upper motor neurone signs in the lower. The first is anterior spinal artery thrombosis. This is usually of acute onset and involves more the anterior aspect of the spinal cord at the site involved, with associated pyramidal &/or anterior horn cell signs. The second is so-called cervical myelopathy resulting from chronic cervical osteoarthritis. This characteristically has a slower onset and more sensory involvement, from posterior cord compression of the dorsal columns, and compression of posterior nerve roots by narrowed intervertebral foraminae.

(iv). Radiculopathies are the next level for consideration. These may be single as in compression of one or more of the nerve roots (eg. lumbar/sciatic) by prolapsed intervertebral discs, or polyradiculopathies involving different areas of the body (seen particularly in the metabolic disorders such as diabetes mellitus). Clearly, radiculopathies will be associated with weakness and/or sensory change in a nerve root rather than a peripheral nerve distribution (the latter is "glove and stocking"). Examine the patient carefully to distinguish between these two.

(v). Peripheral neuropathies i.e. involvement of the nerves themselves peripheral to the spinal cord and nerve roots.

These can be subdivided into:

a). Generalised peripheral neuropathies (including those caused by drugs, toxic chemicals, heavy metals, alcohol; vitamin deficiencies including B1, B6, and B12; carcinoma; metabolic disorders such as diabetes mellitus, renal failure and porphyria; infective polyneuropathy, diphtheria; and infiltration by amyloid disease, sarcoidosis and leprosy). Of course, the precise diagnosis of any generalised peripheral neuropathy will depend on your associated pathological, functional and aetiological diagnoses. Also, examine for signs of autonomic neuropathy, as this gives you a good idea of how extensive the process of nerve involvement is; e.g., autonomic neuropathies are often found in association with severe diabetic peripheral neuropathy.

b). Mononeuropathies (either single or multiple). This is where one or more nerves are involved, including the cranial as well as other peripheral nerves.

Mononeuropathies may be produced by trauma, compression (e.g. crutch palsy), nerve entrapment (e.g. median nerve involvement in the carpal tunnel syndrome), or ischaemia (as in diabetes, polyarteritis). Clues to nature will come, as usual, from mode of onset and time-intensity relationships (see Pathological Diagnosis).

Multiple mononeuropathies (also called mononeuritis multiplex) is where a number of peripheral nerves are involved usually asymmetrically). The usual underlying factor is ischaemia of different nerves, and the condition occurs most characteristically in polyarteritis nodosa and diabetes mellitus. The nerves involved may be peripheral or cranial.

(vi). Motor End-Plate Disease

This is the most peripheral level of the nerve which can cause motor weakness. The characteristic disease here is myasthenia gravis, which is an auto-immune disease where auto-antibodies produced block the acetyl choline receptor on the muscle. Because it involves the motor end-plate, there is no sensory impairment. It is characterised not only by the usual signs of a peripheral motor lesion, including weakness, but also by fatiguability (i.e. weakness deteriorating as the day goes by, or as the movements are repeated on examination; ocular muscles and eyelids tend to be particularly involved).

Difficulties in anatomical localisation:

A particular difficulty is the differential diagnosis of polyradiculopathies from peripheral neuropathies but, as mentioned, careful mapping of the sensory and muscular involvement can usually separate the two. Mixed pictures can also cause difficulty e.g. in diabetes where polyradiculopathy may be associated with peripheral neuropathy of either the generalised, or localised mononeuropathy, type. Vitamin B12 deficiency classically causes subacute combined lateral (upper motor neurone) and posterior (position sense, vibration sense) column degeneration in the spinal cord; however, it is not infrequently also associated with a generalised peripheral neuropathy and some lower motor neurone weakness as well, so in practice this, too, can present difficulty in diagnosis.

Local Muscle Disease: Bear this in mind, particularly in cases where there are no sensory symptoms.

B. CLINICAL PATHOLOGICAL DIAGNOSIS

If the weakness is of very acute onset think of compression, trauma, or infarction/ischaemia as well as acute severe toxic poisoning (including botulism, tetanus, strychnine). If less acute, think of inflammatory problems, particularly if there is an associated fever (as in infective polyneuritis). Look for muscle tenderness as a sign of myositis. If more chronic without fever or weight loss, think of metabolic causes, including vitamin deficiencies (especially B12) as well as electrolyte and endocrine causes (see above). In cases of chronic weakness with weight loss, think of underlying neoplasia (e.g. cancer of the lung - which may also be associated with peripheral neuropathy and/or polymyositis). If very chronic, think of the degenerative processes, e.g. old poliomyelitis, hereditary disorders, myopathies, muscular dystrophies, etc. (check family history).

One important point to remember - if there is weight loss and no loss of appetite, think of thyrotoxicosis, diabetes mellitus, and the malabsorption/ maldigestion syndromes.

C. FUNCTIONAL DIAGNOSIS

Already made, because we have to know this before we can make an Anatomical diagnosis. But in looking at function, watch the patient walk; in bilateral upper motor neurone lesions, for example, there may be a "scissor" gait due to bilateral adductor spasm.

One particular trap is where the patient presents with apparently classical intermittent claudication on history but where examination shows normal peripheral lower limb pulses. That sometimes occurs in true ischaemic claudication, but to confirm this take the patient for a walk and show that the pulses disappear when he develops pain. If not, you may be dealing with so-called pseudo-claudication, a condition which may occur, for poorly understood reasons, in lower spinal canal lesions (spinal canal stenosis) compressing the cauda equina.

D. AETIOLOGICAL DIAGNOSIS

There are several points worth making. In general, toxic (endogenous metabolites or exogenous poisons, drugs, heavy metals etc.) produce neurological effects that are fairly symmetrical bilaterally, e.g. the symmetrical polyneuropathy of arsenical poisoning. At least that is true of uncomplicated cases. The corollary is that if you have a patient with distinctly unilateral weakness, there is little reason to suspect toxic drugs, poisons, chemicals etc. as the underlying aetiological cause. Of course, there are always exceptions, e.g. patients with diabetes mellitus may sometimes present with single nerve lesions (due to the superimposed complication of ischaemia of the vasa vasorum to that nerve).

As usual, delve into the patient's background in this category, checking for example the family history which may not only tell you that the condition is familial, but support your suspicion of a muscular dystrophy as the basis for the Anatomico-Pathological diagnosis. With any acute ischaemic lesion, try to define its cause, not only whether embolic or thrombotic etc. but, if embolic, from whence that embolus has arisen. Equally, the anatomical distribution of muscle weakness may point to particular directions in relation to underlying cause; (thus, proximal myopathy suggests a metabolic, electrolyte or endocrine disturbance - see above).

MECHANISMS IN DISEASE: MCQs

MULTIPLE CHOICE QUESTIONS

1. Mechanisms in Disease:

A patient presents to your ward with a diagnostic label of "chronic progressive spinal anterior horn cell neuronitis". Which of the following would be characteristic if this diagnosis is correct?

1. Diminished pain and temperature sensation in the corresponding spinal dermatomes.
2. Onset over a period of several days.
3. Hyper-reflexia and spasticity in the affected muscle distribution.
4. Inflammatory changes in the muscle spindles.
5. Fasciculation of the muscles concerned.
6. Muscle tenderness.

Answers in Final chapter section

CLINICAL PROBLEM SOLVING

2. Clinical Problem-Solving:

A 38-year-old (1) industrial chemist (2) presents with a 4-week history (3) of weakness in the right leg, first noticed on a Friday afternoon as a tendency to drag his right foot, so tending to trip him up on walking upstairs (4). He has also noticed a tingling sensation over the dorsum of his right foot (6). Completely well otherwise (6), but on direct questioning recalls some thirst, polydipsia and polyuria, and associated nocturia in the last six weeks or so (7). No specific symptoms related to other organ systems. He has had no previous illnesses and the family history is unremarkable. In the social history, he is happily married with two children, and takes only "occasional" alcohol. Not on any drug therapy. Non-smoker (8).

On general examination, he looks a little thin (9), and his skin seems somewhat dry (10). Afebrile (11). The cardiovascular, respiratory and gastro-intestinal systems are all normal. In the central nervous system the only abnormality is in the right leg where he has a weakness of dorsiflexion of the right ankle and of the related right anterior tibial muscles (12). There is also weakness in dorsiflexion of the right toes, particularly the great toe (13). In addition, there is some weakness of eversion of the right foot when dorsiflexed (14). There is a blunting of all modalities of sensation over the medial 3/4 of the dorsum of the right foot though not including sensation between the first and second right toes (16), and no sensory involvement of the right calf (17). No muscular tenderness (17) but slight wasting of the anterior tibial and peroneal muscles (18). Knee and ankle reflexes normal (19). Plantar reflexes are both downgoing, and the tone in the affected leg is completely flaccid (20). No abnormal

movements or muscle fasciculations (21); however, when asked to walk, he has a noticeable right foot drop, and seems to exaggerate right knee flexion to compensate for this; correspondingly weak R. plantar flexion (22). Urine examination shows SG 1.025, no protein or red cells, but glucose ++++ (23).

Drawn up your columns and **solve the problem in all four diagnostic categories**. Then draw an overall conclusion and write a diagnostic dissertation.

Then **answer the following MCQ questions**:

WHICH OF THE FOLLOWING STATEMENTS IS/ARE LIKELY TO BE CORRECT?

1. This man's problem is most likely to be due to myopathy (primary muscle disorder).
2. If perchance the weakness has a neurological basis, it is probably below the level of the lower motor neurone in the anterior horn.
3. It would be possible to account for all of the clinical features by compression of the 5th right lumbar nerve root (L5 radiculopathy).
4. It is unlikely that exposure to industrial chemicals at work could explain this picture.
5. The glycosuria is probably of benign origin (renal glycosuria).
6. Weight loss in this case is probably partly related to dehydration.
7. The polyuria and nocturia suggest a primary disorder of renal function.

Finally, **compare your solution with graphic** (centered with space bar) and author's diagnostic dissertation in the **next section**

PROBLEM SOLUTION-1

Weakness Diagnostic Problem			
Where?	What?	How?	Why?
	<p>3. 4/52 = <div style="border: 1px solid black; padding: 2px; display: inline-block;">Sub-acute</div></p> <p>6. Prev. well = ? No underlying chronic illness.</p> <p>9. Thin = ? weight loss - ? related to 10.</p> <p>11. Afebrile = probably not inflammatory.</p>	<p>4. R. foot drop - ? R. lateral popliteal nerve - ? R. radiculopathy involving L4, L5, nerve roots.</p> <p>5. Paraesthesia dorsum R. foot. = ? R. lateral popliteal nerve - ? R. L5 nerve root.</p> <p>10. Skin dry = ? body fluid depletion.</p> <p>12. Weak dorsiflexors R. ankle, = ? R. lateral popliteal nerve - ? R. L4 nerve root.</p> <p>13. Weak dorsiflexors R. great toe etc. = ? R. lateral popliteal nerve - ? R. L5 nerve root.</p> <p>14. Weak R. foot eversion = ? R. lateral popliteal nerve - ? R. S1 nerve root.</p> <p>15,16. Sensⁿ. over med. 3/4 R. foot = more likely <div style="border: 1px solid black; padding: 2px; display: inline-block;">R. lateral pop. nerve (below lateral cutaneous branch)</div> than L5 root, espec. in view of</p> <p>16. = no sensⁿ. over lower R. leg.</p> <p>17. No muscle tenderness i.e. no evidence that this is a muscle (inflammatory) disorder.</p> <p>18. Wasting of ant tibial and peroneal muscles = <u>R. lower motor neurone</u> involvement (of some weeks' duration).</p> <p>Distribⁿ. of weakness suggests - <u>R. lateral popliteal nerve</u> , or - R. L4, L5, S1 nerve roots.</p> <p>19. Knee jerk normal, therefore unlikely to involve nerve roots L3, L4. Ankle jerk normal, therefore unlikely to involve S1 nerve root.</p> <p>20. R. leg tone flaccid, plantar reflex ↓ = <u>R. lower motor nerve lesion</u> .</p> <p>21. No muscle fasciculations = no evid. of anterior horn cell dis.</p> <p>22. Gait = <div style="border: 1px solid black; padding: 2px; display: inline-block;">R. foot drop.</div></p>	<p>1. 38 yr. old male.</p> <p>2. Industrial chemist. ? exposure to toxins.</p> <p>7. Thirst, polyuria polydypsia = ? Diabetes mellitus (or insipidus).</p> <p>8. No other evident background features.</p> <p>23. Heavy glycosuria = <div style="border: 1px solid black; padding: 2px; display: inline-block;">Diabetes mellitus.</div></p>
<div style="border: 1px solid black; padding: 2px; display: inline-block;">R. lat. popliteal nerve palsy.</div>	←	→	

PROBLEM SOLUTION-2

Weakness Diagnostic Problem (continued).

Where?	What?	How?	Why?
R. lat. popliteal nerve — below origin of lateral cutaneous branch.	Sub-acute process Not inflammatory ? Acute (ischaemic) onset in view of aetiology (more history needed).	Occam's razor principle suggests R. lateral popliteal nerve mononeuropathy , rather than the more wide-spread L4, 5, S1 radiculopathy; - no L4, S1 sensory involvement; - R. knee jerk (L4), R. ankle jerk (S1) present.	Diabetes mellitus - ischaemic neuropathy. Chemicals not likely to be relevant to a unilateral nerve lesion).

Final Diagnosis

Anatomic Diag.	Pathological Diagnosis	Functional Diagnosis	Aetiological Diagnosis
R. lat. popliteal nerve (below lateral cutan. branch).	Sub-acute, but ? initial ischaemic cause.	R. foot drop and altered gait, R. foot sensory impairment.	Diabetes mellitus (giving rise to ischaemic neuropathy).

PROBLEM SOLUTION-COMMENT

The graphic solution refers to the **lateral popliteal nerve**. This is old terminology.

The nerve is now called the **superficial branch of the peroneal nerve**, which is a branch of the common peroneal nerve.

The lateral cutaneous nerve of the calf arises from the common peroneal nerve, above the branching of that nerve into superficial and deep branches; this accounts for the sparing of sensation of the lateral calf in this man.

The deep peroneal nerve, among other things, supplies sensation to the small area between the 1st and 2nd toes. This explains the sparing of sensation of this area in the present case.

MCQ ANSWERS

A. Mechanisms in Disease

1 & 5 correct. Others false

B. Clinical Problem solving.

2, 4, & 6 correct. All others false.