Tips for trainees: some practical tips on clinical examination

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The following tips are designed to help trainees or inexperienced neurologists to reach the right answer in a time-efficient manner. The tools required are easily carried. The list is personal and idiosyncratic; other experienced neurologists may feel they can improve on it. Some tips are very simple and most trainees will know them already, but experience shows that not all do.

Expert clinicians use the examination to test hypotheses and solve problems: for this to work you need to 'think on your feet' and adapt the examination to the circumstances. This includes confirming abnormalities in detail when a screening test is positive. Likewise, any unexpected abnormality on examination must be confirmed, with its relevance being determined in the context of the history and the overall clinical presentation.

COLOUR DESATURATION IS A SIMPLE SCREENING TEST FOR OLD OPTIC NEURITIS

Hold up a red object such as the top of a hatpin and cover each of the patient's eyes in turn. Ask whether the colour seems washed out or dulled in one eye compared with the other.

Clearly, this detects asymmetrical colour desaturation. Its advantages are that it is quick and reasonably sensitive. It can be included in the examination with minimal loss of time even when the chance of a positive response is quite low. Naturally, a positive response or a history suggesting that optic neuropathy is likely should provoke a more detailed assessment for evidence of optic neuritis.

Other signs of old optic neuritis include the following:

A. Pale optic discs (subtle changes may be difficult for the inexperienced).
B. Impaired colour vision when tested formally with Ishihara charts (more time-consuming).
C. Afferent pupillary defect (quick, effective and well known, but subtle changes may be difficult to see).
D. Subtle central or paracentral field defects (not easy to detect at the bedside by ordinary methods). Testing visual fields with a laser pointer can be helpful.[1]

Colour desaturation can also be a feature of partial field lesions from anterior optic pathway lesions. For example, chiasmal lesions may result in temporal field desaturation or ischaemic optic neuropathy may result in superior or inferior desaturation. The patient is asked to look at a central target with red squares to either side (or above and below for vertical field defects) and asked if one square looks washed out compared with the other (figure 1).

ARROW SIGN FOR FOURTH NERVE PALSY

The action of the superior oblique muscle is complex and results from its line of action. When the eye is adducted, the muscle pulls the eye down. When the eye is abducted, its main action is to intort the eye. In the primary position, the action is a mixture of the two.

Consequently, a lesion of the fourth (trochlear) nerve causes the eye to deviate upwards and into extorsion from the unopposed actions of remaining intact muscles. The patient will complain of diplopia with a vertical and torsional component.

A simple way to demonstrate this is to hold a long straight object (such as the handle of a tendon hammer) horizontally in front of the patient who will report images tilted at an angle. The two images will converge on the side of the trochlear palsy, forming an arrow pointing to the affected side (figure 2).

The oblique action of the fourth nerve leads to another helpful sign in patients with a complete third nerve palsy. In most such patients, the third nerve palsy

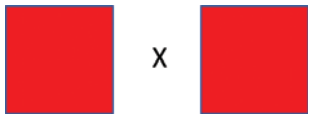

Figure 1 Target for detecting relative colour desaturation in part of the visual field.

Figure 2 The diplopia described by a patient with a trochlear nerve palsy.

is isolated (eg, being caused by microvascular disease or a posterior communicating artery aneurysm), but in some, there is pathology in the cavernous sinus or superior orbital fissure causing a fourth nerve palsy as well.

If there is a complete third nerve palsy, the eye will be abducted: ask the patient to look down. The action of superior oblique is to cause intorsion and this can easily be seen by looking at vessels on the sclera. If the eye does not intort with attempted down gaze, one can conclude that the fourth nerve is damaged, with the diagnostic implication that this is not an isolated third nerve palsy.

OBLIQUE SACCADES

Some conditions cause selective slowing of vertical saccades with preservation of horizontal saccades (eg, Niemann-Pick type C or the early stages of progressive supranuclear palsy), while in other conditions, horizontal saccades are slowed (most commonly in the adducting eye in internuclear ophthalmoplegia). Experienced neurologists can usually judge whether saccadic speed is normal but inexperienced examiners may find this more difficult.

One way to make differences in vertical and horizontal speed more apparent to the inexperienced is to ask the patient to perform an oblique saccade: they are asked to look from a high target on the left to a low target on the right for example. If vertical saccades are slowed, the horizontal movement will be completed first and the eyes will follow a curved path, rightwards first and then downwards. If there is slowing of adduction of the left eye but other movements are normal, the right eye will travel in a straight line and the left eye will follow a curve going downwards before it goes rightwards (figures 3 and 4).

Many will find the abnormal shape of the path of the eye easier to detect than the slowed speed.

Figure 3 Oblique saccades. Path of eye with slow downward saccade.

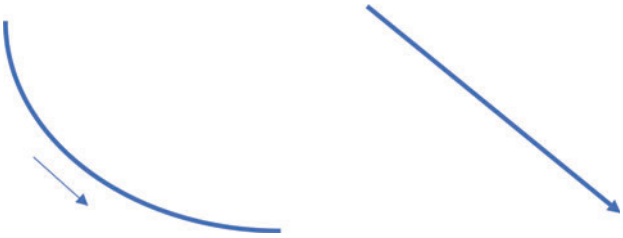

Figure 4 Oblique saccades. Paths of eyes with internuclear ophthalmoplegia.

MOTOR EXAMINATION: TARGET DIAGNOSTICALLY INFORMATIVE MUSCLES

The neurological examination is a powerful means of testing hypotheses. In the motor examination, there is often the question as to the source of localised weakness: intelligent selection of which muscles to test in detail separates the expert from the tyro.

C5 versus axillary nerve: infraspinatus

In patients with weakness around the shoulder girdle, the differential diagnosis often lies between nerve root, plexus or peripheral nerve lesion. Deltoid is supplied by C5 via the axillary nerve. Supraspinatus is supplied by C5 via the suprascapular nerve but its action, shoulder abduction, largely overlaps that of deltoid. Infraspinatus is also supplied by C5 via the suprascapular nerve and has a unique action producing external rotation, which is easily tested. Thus,

A. An axillary nerve lesion affects deltoid.
B. A suprascapular nerve lesion affects infraspinatus and supraspinatus.
C. A severe C5 root lesion affects deltoid and infraspinatus and supraspinatus.

Radial nerve versus cortical stroke: finger extensors at metacarpo-phalangeal joints compared with extensors at interphalangeal joints

Metacarpo-phalangeal extension is supplied by the radial nerve; interphalangeal extension (lumbricals) is supplied by median and ulnar nerves. The interossei are supplied by ulnar nerve but may be difficult to activate in the presence of wrist drop.

Weakness of metacarpo-phalangeal joint extension with preserved strength of interphalangeal joint extension strongly suggests radial nerve dysfunction: more proximal lesions do not produce this pattern.

The examination of possible 'Saturday night palsy' requires precise technique: stabilising by gripping just proximal to the joint to be tested and resisting the attempted extension just distal to the joint. The principle is when testing strength, test one joint at a time.

L5 root lesion versus peroneal nerve palsy: hip abduction weakness

Ankle dorsiflexion and eversion should be weak in both L5 root lesion and peroneal nerve palsy, and ankle plantar flexion in neither.

It is said that ankle inversion should be preserved in a peroneal nerve lesion (true) and may be weak with L5 root lesion: while this weakness with an L5 lesion may occur, it is not universal and is a sign that I feel is overemphasised.

Strength of hip abduction can be very useful. This is often weak with an L5 lesion and is not affected by peroneal nerve palsy. This can be assessed directly, but also a tendency for the hip to drift into adduction when flexed against resistance may also draw one's attention to it.

HAVE A STRATEGY FOR OBTAINING REFLEXES IN PATIENTS WHO 'WON'T RELAX'

We are all familiar with the frustration of trying to obtain reflexes in a patient who seems incapable of relaxing. This occurs particularly in the elderly, when paratonia or frontal rigidity may be contributing. Some unconventional techniques can help in this situation. These have been described in more detail elsewhere, together with evidence of their effectiveness.[2]

Superior patellar strike for knee jerk

See figure 5. The response is easily felt and is usually visible as well.

Plantar strike method for ankle jerk

See figure 6. This is my preferred initial method in all patients.

Achilles strike elevated for ankle jerk

The Achilles strike with leg elevated method requires you to sit beside the patient (who is reclining in bed) on the edge of their bed, facing their feet; you then elevate the patient's leg with the knee flexed and tuck the knee between your arm and torso. Strike the Achilles tendon with the foot held slightly dorsiflexed (figure 7). This requires some practice in positioning and is more complex than the plantar strike method. I therefore suggest beginning with the plantar strike method and then, if necessary, moving on to use the Achilles strike with leg elevated method.

Figure 5 Superior patellar strike method for knee jerk.

Figure 6 Plantar strike method for ankle jerk.

Figure 7 Achilles strike elevated method for ankle jerk.

GRADING THE PLANTAR RESPONSE AND BING'S SIGN

We are used to thinking of the plantar response as being either flexor or extensor (Babinski sign), but just as tendon reflexes may range from absent through various degrees of briskness, the plantar reflex may show grades of abnormality. If a patient clearly has a flexor response on one side and an equivocal response on the other, this may indicate a subtle upper motor neurone abnormality.

Thus, it is more than mere ward-round theatricality to see if a plantar response can be 'coaxed into extension'. In a patient with an equivocal (in fact mildly abnormal) reflex, a smooth, slow stimulus a little more medially on the sole than usual tends to produce a flexor response while a brisker more lateral stimulus tends to produce an extensor response.[3]

There are many methods for stimulating the extensor response of the toe, mostly named eponymously 100–150 years ago.[4 5] Most add little to the routine examination but do demonstrate the wide sensory range of the reflex. However, one such method is helpful when the patient appears to be very ticklish or otherwise sensitive, such that an extensor movement of the toe is thought possibly to be a withdrawal response of no clinical importance. In this situation, the (so-called) Bing sign[6] is useful: the dorsum of the great toe is pricked several times with a disposable pin (like testing for pain sensation). If the toe extends as if to impale itself on the pin, it is genuinely extensor: a withdrawal response in this case would be down away from the pin. The apparently self-destructive behaviour of the toe in this sign has resulted in it being given various flippant or humorous names which are memorable but inappropriate for a scientific journal.

MEDIAN STRIP/CHRISTMAS TREE SENSORY LOSS ON THE TRUNK IN NEUROPATHY

The sensory examination is a powerful tool for testing hypotheses but of very little value as an unfocussed data-gathering exercise. Hence, my first rule of the neurological examination is 'never do a sensory examination until you know what you will find, or at least what you are looking for'. The distribution of sensory loss is the key to localisation and so the edges of any area of loss should be explored intelligently.

An example of a sensory sign that will never be found unless sought explicitly is the loss of sensation on the trunk in length-dependent sensory neuropathies. All medical students know that such patients will have glove and stocking sensory loss. But few know that there is often a similar phenomenon on the trunk. One would predict that sensory loss in the most distal areas supplied by thoracic and abdominal nerves would involve a median strip down the front of the trunk.

In fact, this finding is quite common in patients with significant sensory neuropathy, if one bothers to look for it.[7] It takes only a moment to test pain or temperature in the midline of the abdomen and move the stimulus laterally to detect a change in perception. The area of loss is often broader over the lower abdomen and narrower high on the chest, producing a 'Christmas tree' shape.

This Christmas tree shape can be a trap when a patient is examined for a spinal sensory level. If you run the stimulus up a vertical line on the anterior trunk, you will cross the diagonal edge of the Christmas tree shape and, unless you delineate the area of loss carefully, you may erroneously conclude that there is a spinal sensory level.

DIRECTION OF SCRATCH AND TEXTURE SENSE

Dorsal column sensation is typically examined by joint-position sense and vibration sense. These are of limited value on the trunk, and so there is a need for a sign of dorsal column function that can help to produce evidence of a sensory level in the case of thoracic cord lesions.

The direction of scratch test is useful for this.[8] It is simple and quick and requires no special tools. A stimulus is moved a few centimetres up, down or across the area to be tested and the patient is asked to report the direction. The stimulus can be the end of a tendon hammer or a finger.

This test also helps in assessing large sensory fibre function in the limb in neuropathies when other methods are technically difficult (eg, arthritis limiting joint-position sense testing or oedema making vibration sense testing difficult).

Another useful test to assess large fibre/dorsal column/parietal sensory function in the hand is texture sense. This is also quick and simple. The patient closes his/her eyes and the examiner takes the patient's index finger and touches the pulp against the skin of the examiner's hand or his/her shirt or coat sleeve. The patient is given a series of these stimuli in succession and is asked to identify 'skin' or 'sleeve'.

This can particularly help in patients with parietal sensory dysfunction (from a stroke, for example) and is less cumbersome and requires less concentration from the patient than graphaesthesia or stereognosis.

Key points

- These tips are designed to help trainees or inexperienced neurologists to reach the right answer in a time-efficient manner.
- It is best to use the examination to test hypotheses and solve problems.
- For this to work you need to 'think on your feet' and adapt the examination to the circumstances.

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REFERENCES

1 Stark R. Clinical testing of visual fields using a laser pointer and a wall. *Pract Neurol* 2013;13:258–9.
2 Ziff M, Stark RJ. How to assess tendon reflexes of the lower limb in the elderly. *J Neurol Sci* 2017;372:196–200.
3 Khadilkar SV, Chheda AH. A systematic and quantitative evaluation of plantar stimulation: the effect of type, pattern, force of stimulation in eliciting an accurate plantar response. *Clin Neurol Neurosurg* 2018;168:83–90.
4 Ambesh P, Paliwal VK, Shetty V, *et al*. The Babinski sign: a comprehensive review. *J Neurol Sci* 2017;372:477–81.
5 Boes CJ. The history of examination of reflexes. *J Neurol* 2014;261:2264–74.
6 Davis AP, Wettstein A, Swanson PD, *et al*. The search for the elusive Bing sign. *Neurology* 2018;91:354–6.
7 Said G, Baudoin D, Toyooka K. Sensory loss, pains, motor deficit and axonal regeneration in length-dependent diabetic polyneuropathy. *J Neurol* 2008;255:1693–702.
8 Hankey GJ, Edis RH. The utility of testing tactile perception of direction of scratch as a sensitive clinical sign of posterior column dysfunction in spinal cord disorders. *J Neurol Neurosurg Psychiatry* 1989;52:395–8.