How to perform a basic neurological examination

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Abstract

The neurological examination is an essential part of the diagnostic process, mistakenly viewed by medical students and junior doctors as difficult or esoteric. It is best used as a form of hypothesis testing after a differential diagnosis has been formulated from the history. This contribution analyses the components of the neurological examination; understanding these gives greater confidence when examining patients.

Keywords Cranial nerves; gait; higher function and language; limbs

Key points

- The history generates diagnostic hypotheses; clinical examination tests them
- Remember to examine the spine
- Examination of gait can be diagnostic
- Disorders of speech include dysphonia and dysarthria
- Disorders of language include expressive and receptive dysphasia
- The mini-mental state examination is insensitive to frontal lobe disorders

Introduction

Despite technological advances, an accurate history is essential for diagnosis and should enable clinicians to answer two fundamental questions:

- where is the lesion?
- what is the pathology?

The presence, and absence, of clinical signs provides evidence supporting the anatomical location of the disease and sometimes its pathology.

The neurological examination encompasses four parts: higher function and language, cranial nerves (including speech production and swallowing), limbs and gait. Patients should also undergo a general medical examination, paying particular attention to pulse, blood pressure, weight, urinalysis, cardiovascular, respiratory and abdominal systems, and skin lesions. Remember to examine the back for abnormalities such as kyphosis and scapular winging.

Higher function language

Handedness should be recorded. All right-handers (and 40% of left-handers) are left-hemisphere dominant. The mini-mental state examination (Table 1) is useful but rather insensitive, particularly with frontal lobe disorders. Scores under 25 can suggest a dementing process. Distinguishing depressive pseudodementia from neurodegenerative diseases can be difficult, but the presence of primitive reflexes can help. These include the palmo-mental (involuntary contraction of the mentalis on stimulation of the thenar eminence) and grasp (progressive closure of the hand with distally moving deep pressure over part of the palmar surface) reflexes, both signs of organic disease.

Dysphasia (aphasia) — defined as impairment of language production — usually implies cortical dysfunction. Receptive (Wernicke's) dysphasia sounds fluent but nonsensical, with poor comprehension. Expressive (Broca's) dysphasia is often agrammatical and hesitant, but comprehension is usually preserved — asking the patient to follow one or more commands tests comprehension. Repetition (e.g. 'Say after me no ifs, ands or buts') can be useful and is usually impaired in expressive and receptive aphasia. Impairment of repetition alone suggests a lesion in the arcuate fasciculus, the white matter tract connecting Broca's and Wernicke's areas.

Bedside tests of frontal lobe function include cognitive estimates (e.g. 'How fast does a racehorse run?'), but the patient's educational background must be considered; using the partner as a control is often informative. Verbal fluency is tested by asking the patient to think of words beginning with certain letters, usually F, A or S. Non-verbal tests include Luria's three-step test. This involves demonstrating to the patient five times, asking them to copy it afterwards, a sequence of three postures of the hand in contact with a flat surface — ulnar border of open hand, fist and palm. To demonstrate perseveration, hold out your hand to the patient and observe whether they attempt repeatedly to perform a handshake.

Dyspraxia is defined as inability to perform a complex sequence of movements in the absence of significant motor or sensory deficits. Ask the patient to copy hand positions or mime an action. Impairment usually implies dysfunction of the contralateral parietal lobe. Dressing and constructional dyspraxias (e.g. copying a five-point star) are seen in non-dominant parietal lobe impairment. Agnosia implies non-recognition and can be visual, tactile or auditory. Placing a familiar object in the subject's hand while their eyes are closed tests tactile agnosia. The pathology is usually in the contralateral parietal lobe. Visual agnosias include prosopagnosia, an inability to recognize familiar faces commonly associated with right temporo-occipital dysfunction.

Memory is an example of a distributed cognitive function. Various classifications are used, including long-/short-term,

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Mini-mental state examination

Time orientation: year, season, date, day, month	5
Place orientation: country, county, town, building, floor	5
Registration: name three common objects and ask the patient to repeat them	3
Attention: spell 'world' backwards	5
Recall: ask for the three objects registered above	3
Language: name two common objects	2
Language: repeat the phrase 'no ifs, ands or buts'	1
Three-stage command: take a sheet of paper in your right hand, fold it in half and put it on the desk	3
Read and obey the following: 'close your eyes'	1
Write a sentence	1
Copy a design, for example a five-point star	1
Total	**30**

Table 1

episodic/semantic, retrograde/anterograde and visual/verbal. The duration of anterograde amnesia can be a useful indicator of severity of head injury. Digit span is a test not of memory but of alertness (patients with Korsakoff's psychosis often have preserved digit span).

The cranial nerves

Olfactory

If smell bottles are unavailable, ask about sense of smell. Anosmia can be a useful sign, particularly when gauging the severity of head injuries. Patients may also report loss of taste when eating.

Optic

Consider five things when testing the optic nerve: colour vision, acuity, visual fields, pupil response and ophthalmoscopic findings.

Colour vision testing uses Ishihara charts. Acquired loss of colour vision associated with loss of visual acuity implies optic nerve dysfunction. The Snellen and Jensen charts have overlapping functions; the former is more sensitive. Test each eye in turn, documenting correction for refractive errors using the patient's glasses or a pinhole. In papilloedema (Figure 1) caused by raised intracranial pressure, visual *acuity* is preserved until late. This contrasts with optic neuritis or infiltration, where acuity is often markedly impaired.

When testing the *visual fields*, sit opposite the patient. With uncooperative or aphasic patients, the fields can be crudely measured by observing the reaction to menace. Test for visual inattention first; then ask the patient to close each eye in turn, comparing their field with your own. Subtle defects can be picked up with a red pin, which is also used to document blind spots.

Monocular visual field defects are usually caused by ocular, retinal or optic nerve pathology. Constricted fields occur in glaucoma and chronic papilloedema. Tunnel vision (loss of peripheral but retention of central vision) can arise with retinitis pigmentosa and should not be confused with tubular vision (visual field restriction that remains constant even when tested at a distance) in patients with a functional disorder. Central

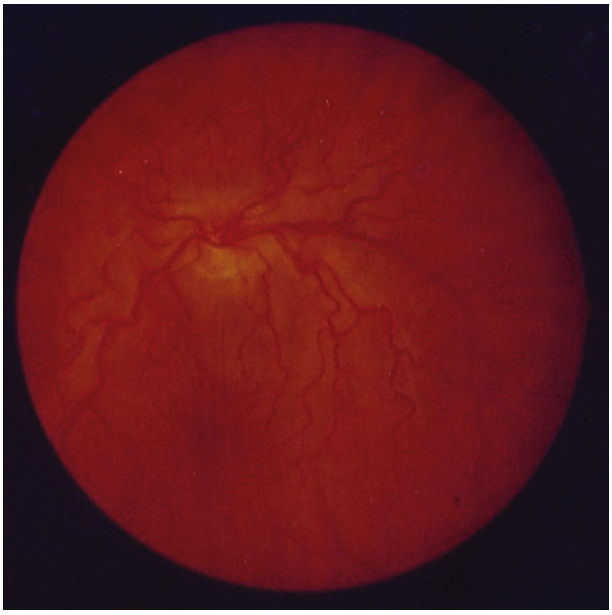

Figure 1 Papilloedema.

scotomas are usually caused by optic nerve or macular disease. Altitudinal defects (respecting the horizontal meridian) usually indicate ischaemia of the retina or optic nerve. Defects affecting both eyes may indicate a lesion of or behind the optic chiasm; they usually respect the vertical meridian. Common patterns of field loss are described in Table 2.

Test the *pupillary reactions* to light and accommodation. If the pupils are different sizes (anisocoria), with the difference accentuated in dim light, this suggests a sympathetic defect. There are four main causes of a unilaterally dilated pupil: oculomotor palsy, tonic (Adie's) pupil (light-near dissociation), iris damage (pupil usually irregular) and instillation of mydriatics (sometimes surreptitious). Argyll Robertson pupils (light-near dissociation) are usually small, irregular and bilateral; the usual cause is syphilis, and the lesion is thought to be in the rostral midbrain. It can be mimicked by a chronic Adie's pupil.

Horner's syndrome is caused by interruption of sympathetic fibres. The pupil is small but reacts normally to light and accommodation. The main clinical features are miosis and mild ptosis. Upside-down ptosis (lower lid elevation), apparent enophthalmos and transient conjunctival hyperaemia can also occur. Iris heterochromia is more common in congenital Horner's syndrome. The causative lesion may be in the brain, spinal cord, brachial plexus or sympathetic chain. Episodic anisocoria can occur in seizures, migraine and cluster headache.

To perform *fundoscopy*, look at each disc, vessels and retinal background. Beware of diagnosing unilateral optic atrophy (Figure 2) if colour vision is preserved or there is no relative afferent pupillary defect; this is detected by swinging a light between the pupils and detecting pupil dilatation on the diseased side.

Oculomotor, trochlear, abducens

All the extraocular muscles are supplied by cranial nerve III except the lateral rectus and superior oblique, which are supplied by VI and IV, respectively. If the patient complains of double