How to perform a neurological examination

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Abstract

The neurological examination is an essential part of the diagnostic process and is mistakenly thought of as difficult or esoteric by medical students and junior doctors. It should be used as a form of hypothesis testing once a differential diagnosis has been formulated from the patient's history. This contribution analyses the various components of the neurological examination so that the reader may perform with confidence. As in any other walk of life, however, practice makes perfect.

Keywords cranial nerves; gait; higher function; limbs; neurological examination; speech

There is a widespread mistaken belief that neurologists are over-reliant on 'high-tech' investigations. Some critics have argued that neurologists could easily be replaced by scanners! However, it remains true that in spite of technological advances the taking of an accurate history plays an essential role in the diagnostic process and should enable the clinician to answer the four fundamental questions: where is the lesion, what is the pathology, what treatment options are available and what is the prognosis? At this point the neurological examination can provide additional evidence that the diagnostic formulation is robust, although in many conditions, for example headache and epilepsy, there are often no abnormal signs.

The neurological examination can be broken down into five component parts: higher function and speech, cranial nerves, limbs, gait and special situations. Every patient should also have a general medical examination, paying particular attention to blood pressure, weight, urinalysis, the cardiovascular, respiratory and abdominal systems, and the presence of skin lesions (Figure 1). Remember to examine the back for kyphosis, scapular winging, etc.

Higher function and speech

Handedness should be recorded. All right-handers (and 40% of left-handers) are left hemisphere dominant. The mini-mental state exam (Table 1) is useful but rather insensitive, particularly in frontal lobe disorders.

A score of < 25 may suggest a dementing process but depressive pseudodementia and acute confusional states may cause diagnostic difficulty. The presence or absence of primitive reflexes can be useful in differentiating between dementia and pseudodementia. These include the palmo-mental (involuntary contraction of mentalis elicited by stimulation of thenar eminence) and grasp (apply distally moving deep pressure over part of the palmar surface) reflexes. The instinctive grasp reaction can be seen where progressive closure of the hand occurs on contact with the palm. A brisk pout reflex (closure of the mouth with pouting of the lips elicited by tapping around the mouth) is non-specific and can occur in many upper motor neurone lesions and may be associated with a prominent jaw jerk.

Speech may be classified as dysphasic, dysphonic or dysarthric.

Dyphasia (aphasia) – defined as impairment in the production of language – usually implies cortical dysfunction. The classification of dysphasia can be complex but can be divided into receptive and expressive components. The former sounds fluent but nonsensical, with poor comprehension (Wernicke's). Expressive dysphasia (Broca's) is often agrammatical and hesitant but comprehension is usually preserved. In this situation, asking the patient to follow one or more step commands can test comprehension. Repetition can be useful (e.g. say after me 'no ands ifs

Figure 1 Subungual fibroma in a patient with tuberous sclerosis.

Mini-mental state examination

Orientation: year, season, date, day, month	5
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 ands ifs 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

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or buts') and is usually impaired in Broca's and Wernicke's aphasia. If repetition is impaired in isolation, this suggests a lesion in the arcuate fasciculus connecting Broca's and Wernicke's areas.

Tests of frontal lobe function performed at the bedside include cognitive estimates (e.g. 'What's the length of my spine?', 'How fast does a racehorse run?') but one must take into account the educational background of the patient. Using the spouse as a control subject is often informative. Other tests, which can be applied selectively, include verbal fluency and Luria's three-step sequence. Perseveration is demonstrated by the examiner holding out his or her hand and observing that the patient will repeatedly attempt to perform a handshake.

Dyspraxia is defined as an inability to perform a complex sequence of movements where the command has been understood and in the absence of significant motor or sensory deficits. Asking the patient to copy certain hand positions or mime an action can test this. 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 may be visual, tactile or auditory. Placing a familiar object in the subject's hand while their eyes are closed may test for tactile agnosia. The pathology is usually in the contralateral parietal lobe. Visual agnosias include prosopagnosia, which implies an inability to recognize familiar faces. This is commonly associated with bilateral lesions of the parieto-occipital regions.

Memory is an example of a distributed cognitive function. Various classifications are used, including long-/short-term, episodic/semantic, retrograde/anterograde and visual/verbal. It is important to remember that digit span is not a test of memory but of alertness (patients with Korsakoff's psychosis often have preserved digit span). The duration of anterograde amnesia may be an extremely useful indicator of the severity of head injury.

The cranial nerves

I Olfactory

Most smell bottles in outpatient departments are years out of date but still work. If unavailable ask about sense of smell. Anosmia can be a useful sign, particularly when gauging the severity of head injuries.

II Optic

Colour vision should be tested with the 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 but the former are more sensitive. Each eye should be tested in turn and a correction for refractive errors documented using either the patient's glasses or a pinhole. In papilloedema (Figure 2) due to raised intracranial pressure, visual acuities are preserved until late in the disease process. This contrasts with optic neuritis or infiltration, where acuity is often markedly impaired.

The visual fields should be tested by sitting opposite the patient. Uncooperative or aphasic patients can have their fields crudely measured by observing their reaction to menace (pretend to poke their eye out with your finger!). Test for visual inattention first and then ask the patient to close each eye in turn, comparing their field with the examiner. Subtle defects can be picked up with a red pin, which is also used to document blind spots.

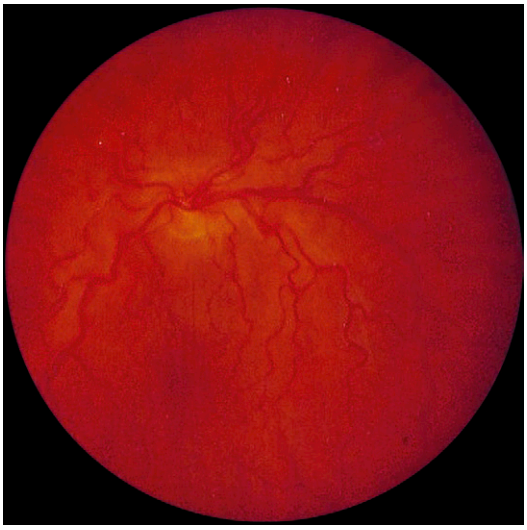

Figure 2 Papilloedema.

Monocular defects are usually caused by ocular, retinal or optic nerve pathology. Constricted fields occur in glaucoma or chronic papilloedema. Tunnel vision may arise in association with retinitis pigmentosa and should not be confused with tubular vision in hysterical patients. Central scotomas are usually caused by optic nerve or macular disease (Figure 3). Altitudinal defects (horizontal meridian) indicate retinal vascular pathology or ischaemic optic neuropathy.

Defects affecting both eyes may indicate a lesion of or behind the optic chiasm (vertical meridian). The common patterns of field loss are shown in Table 2.

The pupillary reactions to light and accommodation should be tested. If the pupils are different sizes (anisocoria) and the difference is 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 installation (may be surreptitious) of atropine or scopolamine.

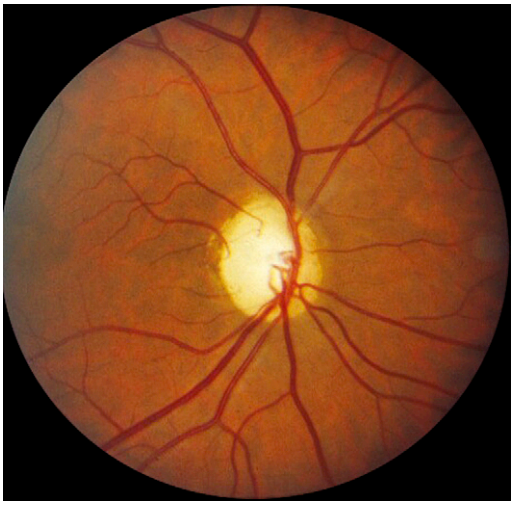

Figure 3 Optic atrophy.

The common patterns of field loss

Field defect	Site of lesion(s)	Aetiology
Homonymous hemianopia	Optic tract, optic radiation, occipital lobe	Stroke, tumour
Superior quadrantanopia	Temporal lobe	Stroke, tumour
Inferior quadrantanopia	Parietal lobe	Stroke, tumour
Bitemporal hemianopia	Optic chiasm	Pituitary adenoma, craniopharyngioma
Binasal hemianopia	Perichiasmal	Bilateral internal carotid artery aneurysms
Junctional scotoma	Junction of optic nerve and chiasm	Tumour
Bilateral scotomas	Occipital pole	Head injury

Table 2

The Argyll–Robertson pupil (light-near dissociation) is usually small, irregular and bilateral but can be mimicked by a chronic Holmes–Adie syndrome. Syphilis is the usual cause and the lesion is thought to be in the rostral midbrain.

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

To perform a fundoscopy, look at disc, vessels and retinal background. Beware of diagnosing unilateral optic atrophy where colour vision is preserved. The swinging flashlight test (Marcus–Gunn pupil or relative afferent pupillary defect) is a useful check in this situation. Accommodation should be normal in a relative afferent pupillary defect (RAPD).

III, IV, VI Oculomotor, trochlear, abducens

All external ocular muscles are supplied by cranial nerve III except the lateral rectus and superior oblique muscles, which are supplied by VI and IV respectively. If the patient complains of double vision the false image is always outermost, disappears when the affected eye is covered and is maximal in the direction of action of the affected muscle. Oculomotor palsies are usually accompanied by complete ptosis because of interruption of the fibres supplying levator palpebrae superioris. This contrasts with a Horner's syndrome where the ptosis is subtle and the pupil is constricted. A pupil-involving oculomotor palsy is usually caused by a surgical lesion, particularly a posterior communicating artery aneurysm. Lateral rectus palsies cause horizontal diplopia whereas superior oblique palsies are worse on looking infero-medially, such as when descending stairs or reading. Vertical nystagmus is far more likely to be neurological in origin than horizontal nystagmus, which can also occur in vestibular dysfunction. When testing smooth pursuit movements, always look for the jerky quality or saccadic intrusion that accompanies cerebellar and brainstem disease. Finally, asking the patient to look at alternating targets will demonstrate hypo- or hypermetria and an internuclear ophthalmoplegia.

V Trigeminal

The trigeminal nerve consists of motor and sensory components, and supplies the muscles of mastication as well as general sensation to the face via ophthalmic, maxillary and mandibular divisions. The corneal reflex has a consensual component. This is particularly useful in the presence of an ipsilateral facial nerve palsy leading to facial weakness. On mouth opening, the direction of deviation of the jaw is ipsilateral to the lesion. The jaw jerk is tested by tapping the point of the mandible with a tendon hammer; if pathologically brisk this implies pathology above midbrain level (e.g. pseudobulbar palsies).

VII Facial

Lower motor neurone palsies tend to cause complete ipilateral facial weakness, whereas because of bilateral representation the upper face is relatively preserved in upper motor neurone lesions. Ask the patient to shut their eyes tight, raise their eyebrows and smile or purse the lips. The corda tympani branch accompanies the facial nerve along some of its length and this explains why patients with Bell's palsy may complain of loss of taste from the anterior two-thirds of the tongue. This can be tested by applying various sweet/bitter/salty solutions. General sensation to the anterior two-thirds of the tongue is supplied by the trigeminal nerve, whereas the glossopharyngeal supplies taste and general sensation to the posterior third.

VIII Auditory

The eighth nerve has auditory and vestibular components. Using an auroscope, whisper into the subject's ear from 1 metre and compare both sides. Patients should hear a vibrating tuning fork more loudly when it is placed in the air (air conduction > bone conduction) adjacent to the pinna compared with resting it on the mastoid process (Rinne test). This is reversed in conductive deafness where bone conduction is better than air conduction. To conduct Weber's test, place a vibrating tuning fork in the middle of the forehead. In unilateral sensorineural hearing loss, hearing is better on the contralateral side. In unilateral conductive loss, hearing is better on the ipsilateral side. Hallpike's manoeuvre is performed by rapidly lying the patient flat with their head turned to one side. The patient should be instructed to report sensations of dizziness while the examiner observes for nystagmus. Latency and fatiguability on repeated manoeuvres suggest a vestibular lesion. The patient may feel nauseous and suffer vomiting. Note that Romberg's test (standing the patient upright with eyes closed and noting increased sway with a tendency to fall) may be positive in vestibular disease.

IX Glossopharyngeal

This nerve forms the afferent limb of the gag reflex and can be tested for by applying an orange stick to the back of the throat. However, many normal subjects show remarkable tolerance to this manoeuvre. Where dysphagia is a problem, the gag reflex is not particularly useful and a much more robust means of

assessing the likelihood of aspiration is to ask the patient to swallow a small quantity of water. Clearly in motor disorders such as motor neurone disease the presence of impaired palatal sensation should lead to a diagnostic reappraisal.

X Vagus

This nerve supplies the palatal musculature. In unilateral lesions the palate is deviated away from the affected side. Ask the patient to say 'Aaaah'.

XI Accessory

The accessory nerve supplies the sternocleidomastoid (SCM) and trapezius muscles. It is tested by asking the patient to shrug their shoulders and turn their head to one side. The SCM is controlled by the ipsilateral hemisphere whereas the contralateral hemisphere supplies the trapezius. Knowledge of this anatomical arrangement is particularly useful in the assessment of functional disorders.

XII Hypoglossal

The hypoglossal nerve supplies the muscles of the tongue. On asking the patient to protrude their tongue, look for deviation from the midline. In lower motor neurone lesions the tongue is ipsilaterally wasted and deviates to the side of the lesion. Test the dexterity of tongue movements by asking the patient to rapidly alternate it from side to side. Slowness of movement without wasting implies spasticity. Fasciculations should be observed with the tongue at rest and inside the mouth.

Dysphonia is usually associated with disorders of the vocal cords and the voice has a hoarse or whispering quality. There may be impairment or alteration of a voluntary cough.

Dysarthria or impaired articulation has many non-neurological causes, such as mouth ulcers. Neurological disease affecting the cerebellum, extrapyramidal system or laryngeal musculature (upper or lower motor neurone in nature) may cause various forms of dysarthria. Cerebellar speech is described as staccato in nature and is mimicked by drinking too much alcohol. Bulbar palsies (lower motor neurone: LMN) cause a nasal twang. Pseudobulbar palsies (upper motor neurone: UMN) are guttural or growling (Donald Duck) and often associated with other features such as emotional incontinence and a brisk jaw jerk. Extrapyramidal speech (idiopathic Parkinson's disease: IPD) is quiet, monotonous and indistinct; there may be an acquired stammer. Chorea may also cause a dysarthria and the speech is explosive with repetition of phrases. Asking the patient to say 'p', 't' and 'k' tests lip, tongue and palatal dexterity, respectively.

The limbs

Remember to enquire about pain. Look for wasting or fasciculations (irregular vermiform movements or twitching of muscles). Ask the patient to hold their arms outstretched with palms facing the ceiling to observe pronator drift, which can be seen in mild pyramidal weakness. Power should be documented using the Medical Research Council (MRC) scale. Observe for postural tremor.

It is conventional to start an examination with the motor system. Examine the upper limbs first. Inspection of the muscles of the shoulder girdle should not be forgotten. Tone should be described as increased or normal. Decreased tone is a term best avoided. A spastic (pyramidal) increase in tone is best assessed by rapid flexion/extension movements at the elbow and is described as 'clasp knife', as the limb seems to suddenly give way. Extrapyramidal increases in tone can be demonstrated at the wrist by slow flexion/extension movements. Cogwheeling has a ratcheting quality whereas in 'lead pipe' rigidity the increased tone is unchanged throughout the range of passive movement. 'Gegenhalten', seen in patients with dementing disorders, describes an inability to relax where it feels as though the subject is deliberately trying to frustrate the examiner.

The muscles examined will vary according to the clinical scenario, but in the vast majority of cases eight upper limb muscle groups will suffice. These include shoulder abduction, elbow flexion and extension, wrist and finger extension, finger flexion and abduction, and a median innervated muscle (usually abductor pollicis brevis). It is worth learning the root values and nerve supply of the muscles tested.[1]

Examination of the deep tendon reflexes follows (biceps, triceps and supinator). Finger flexion jerks may indicate an upper motor neurone lesion but can also be observed in anxious patients. If asymmetrical, this latter sign is likely to have added significance (see Table 3). Hoffman's sign (flicking of the distal thumb leading to flexion of the fingers) is also suggestive of an upper motor neurone lesion.

The deep tendon reflexes are graded as 0 (absent), +/– (present with reinforcement), + (depressed), + + (normal), + + + (increased). Reinforcement can be obtained by jaw clenching or Jendrassik's manoeuvre (patient links hands and pulls). Deep tendon reflexes may also be inverted whereby the tested reflex is absent but there is spread to a lower level. This indicates a lower motor neurone lesion at the level of the reflex but an upper motor neurone lesion below.

The main superficial reflexes include the abdominal (upper T8/9, lower T10/11), cremasteric (L1/2) and anal (S4/5). These are absent in some upper motor neurone syndromes. The cremasteric reflex can be elicited by stroking the inner aspect of the thigh with consequent ipsilateral testicular elevation.

Assess coordination by asking the subject to perform a hand-tapping task (listen to the rhythm) and perform the finger-nose test. Past pointing or intention tremor is a hallmark of cerebellar disease and in contrast to other tremulous disorders the amplitude increases as the finger nears the target. Classical pill-rolling tremor, virtually diagnostic of idiopathic (IPD) or drug-induced Parkinsonism, is a low-frequency resting tremor that ameliorates

The main deep tendon reflexes

Reflex	Nerve	Root
Biceps	Musculocutaneous	C5/6
Supinator	Radial	C5/6
Triceps	Radial	C7
Finger flexors	Median/ulnar	C8
Knee	Femoral	L3/4
Ankle	Tibial	S1/2

Table 3

The various gait disturbances encountered in clinical practice

	Type of gait	Description	Common causes
1	Gait apraxia	Small shuffling steps Marche à petit pas	Small vessel disease Hydrocephalus
2	Parkinsonian	Shuffling Loss of arm swing	Idiopathic Parkinson's disease
3	Spastic paraparesis	Stiff walking through mud	Cord lesion Parasagittal lesion
4	Myopathic	Waddling	Muscular dystrophy
5	Foot drop	Foot slapping	Neuropathy
6	Spastic monoplegia	Exaggerated circumduction	Stroke
7	Cerebellar ataxia	Wide based 'Drunken'	Any cerebellar pathology
8	Sensory ataxia	Wide based Foot slapping Deteriorates with eye closure	Sub-acute combined degeneration of the cord

Table 4

on posture, although it can also be seen whilst the subject is walking. Essential and dystonic tremors are prominent on posture (arms outstretched, palms to floor) and tend to improve when the arm is resting.

Examination of the lower limbs should include assessment of tone by rapid, passive flexion of the subject's hip and knee. The examiner should feel for the spastic 'catch' that accompanies pyramidal disorders. Clonus is best demonstrated by rapid ankle dorsiflexion; sustained clonus of greater than four beats is considered pathological. Observe for wasting/fasciculations but also isolated lower limb tremor, which is strong evidence for IPD. Assessment of power should include hip flexion/extension, knee flexion/extension, and ankle plantar- and dorsiflexion. Elicit the knee and ankle reflexes next. Scratching an orange stick along the lateral border of the sole and then turning it medially to finish below the first metatarsal elicits the plantar response. An extensor plantar response is always pathological in any patient over the age of 12 months. Finally, assess the gait and perform Romberg's manoeuvre by asking the patient to stand with their feet slightly apart and eyes closed. This should be recorded as positive only if the patient would fall without the intervention of the examiner (be prepared!).

The sensory examination follows, but it is not necessary to spend too much time on this. Ideally, one should attempt to map out all the sensory modalities (pinprick, light touch, temperature and joint position/vibration sense) on a chart. In practice it is better to do this in a more efficient way. For spastic paraparesis find the sensory level with a pin, and for functional disorders look for complete hemisensory loss with different vibration sense either side of the midline at the sternum. Remember that loss of joint position sense and vestibular pathology may lead to a positive Romberg's sign whereas cerebellar pathology does not. Testing two-point discrimination is unlikely to be particularly useful.

The gait

The various gait disturbances encountered in clinical practice are shown in Table 4.

Special situations

The assessment of a patient in coma is covered in the next issue.[2] Patients with ventilatory failure may have diaphragmatic weakness. Abdominal paradox relies on the fact that in normal subjects inspiration when supine causes an outward expansion of the abdominal wall due to downward movement of the diaphragm. If the diaphragm is weak this movement is reversed and the anterior abdominal wall recedes. Assessment of sniff is a surrogate marker for vital capacity.

Summary

The neurological examination should be no more challenging than other aspects of patient assessment. In real life, examination findings should be analysed in the context of the history; a patient with tension headaches is far more likely to have brisk reflexes induced by anxiety rather than a lesion in the pyramidal tract. However, in many postgraduate exams (such as MRCP) the inability to perform a slick neurological examination is often the main stumbling block for the failing candidate. Although this article may provide guidance, it is no substitute for patient contact and bedside teaching. ◆

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