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Clinical assessment

Peter Bodkin and Elizabeth Visser

Introduction

The practical process of clinical assessment in modern neurosurgery is slightly different to the traditional model. The ground rules of history taking and examination set down by William Osler and the other forefathers of clinical method rightly remain core to our approach, but we must also take into account the realities of the patient journey before the first encounter with a neurosurgeon. Whether by the bedside or in the outpatient setting, it would be rare nowadays for us not to have already been presented with pertinent background information—and indeed, often we will have seen high-quality imaging detailing the patient's pathology with great anatomical detail. The referring physician will most likely have provided a potential list of differential diagnoses. In other words, the patient has usually been neatly packaged well before we ever get to meet them.

Our job, however, is more than simply to supply a surgical fix to a given problem. We are not merely technicians. Although we must take into account the information given to us, we must never take it on face value. All sorts of errors in assessment and incorrect conclusions may have been made along the way. Likewise, imaging is very useful but should never be viewed outside the clinical context. An MRI scan will localize a lesion with more confidence than the best clinician, but technology cannot make us consider a diagnosis of acromegaly from a spongy handshake or common peroneal palsy from the sound of a slapping foot coming to the clinic door.

As we gather and make sense of this information there is an equally, if not more, important process at work. By putting the patient's symptoms into the context of their daily life we begin to develop a relationship with the patient, starting to establish trust and mutual understanding. The rapport built here will be the basis of how a patient measures the success or failure of our interventions.

The traditional neurological clinical assessment described in most textbooks has other subtle differences to those required of the neurosurgeon. This chapter aims to cover that broad spectrum of clinical method for the practising neurosurgeon and those in training.

The neurosurgical history

The main aim of the neurosurgical history is to gain sufficient information to estimate the anatomical location of the problem and to get an impression of the pathological process at work. In particular, the time course and severity of the symptoms will be important guides as to whether surgical intervention is required and how quickly.

As with any important task, being well prepared will provide a good first impression and will save time in the long run. Referral letters, clinical notes, imaging, and other investigations should have been carefully reviewed. Prior discussions with relevant team members may also be useful.

In the clinic, calling the patient from the waiting room yourself can be very valuable. An impression of their social support may be gained by seeing who is with the patient and how attentive they are. Anaesthetic fitness can be crudely assessed by how long it takes to get up and into the clinic room or how out of breath they might be. It is also an opportune moment to make an assessment of gait and a note of walking aids, and so on.

Thought should be given to the physical environment for the meeting. This should be arranged such that the patient and doctor are on as level a playing field as possible. One should be aware that being at a higher eye level or sitting behind a desk may have an intimidating effect and will detract from getting the most out of the encounter. When dealing with digital images, viewing platforms should already be opened with relevant images downloaded.

Introductions should be clear, giving your name and position. Significant others should be welcomed and acknowledged but it should be made clear that the patient is the focus of discussions.

How you open the consultation is important. One should start with open questions, 'So what's been the trouble?', or 'How can I help you?' The referral letter or consultation request has a tendency to emphasize the symptoms that will lure you into seeing the patient in the first place. It is wise, therefore, to avoid saying things like, 'So your doctor has asked me to see you about your facial pain?' Assumptions can be misleading and may encourage patients to tell you what you are expecting to hear.

Once you have encouraged the patient to tell their story, it may be necessary to fill in some gaps. The patient may have painted a picture but there could be large areas missing or fundamental details that are only sketchy. When it comes to the information that is going to influence your decision on treatment keep delving until you feel there is satisfactory detail for a conclusion to be drawn. In the course of this it is important to remember to find out the occupation of the patient and often handedness is pertinent. It is not enough to say a patient

is 'retired'. A retired university professor will have quite different expectations in life compared to a retired ship builder.

The history taking needs to cover time course, anatomical location of symptoms, variability, and character. One must refine questioning with the aim of localizing the lesion or getting clues as to the underlying pathological process. If a patient is not volunteering a symptom, one must consider symptoms that might be associated and ask about them directly. For example, when assessing a patient presenting with spatial disorientation due to a right parietal tumour one needs to remember to ask about problems with their visual fields in case of involvement of the optic radiation, and so on.

The most vital part of the story is often the impact that the condition is having on the patient's way of life. It may seem an unnecessary intrusion to pursue this but most patients are happy to let you know their problems and the knock-on effects on their home, work, and family life. By establishing that you are not solely interested in dealing with their particular pathological entity but rather you want to help them get back to doing the things they enjoy, you turn a medical interview into a more meaningful conversation. The patient will understand that you are treating them and not just their tumour or slipped disc.

Pick up cues. Have an ear for the incongruous. If what you hear doesn't make sense, explore it. Don't let it pass unmentioned. It is also useful to find out what has been done so far to address the problem: pain killers, physio, injections, visits to other physicians, and so on. Is the patient fit for an anaesthetic? Are there drugs that need to be stopped prior to surgery? Could an unhealthy lifestyle be contributing to the problem?

Drawing the history to a conclusion, the patient should feel that their issues have been adequately addressed. 'Is there anything else you'd like to discuss?' is a useful way of allowing any additional information to be voiced. The patient will hopefully be in a more relaxed and open frame of mind by this stage of the interview and might reveal underlying motivations and concerns. Finally, it may be useful to agree on a brief summary and have a final effort to clarify any lingering grey areas or inconsistencies.

The neurosurgical examination

Examination of the unconscious patient

The approach to the patient who has altered conscious level is obviously limited by the inability of the patient to comply with given instructions and to provide verbal feedback. We are therefore restricted to rather crude and basic bedside tests (i.e. examination of pupillary response to light and the Glasgow Coma Score). Often high-stake decisions are made on the basis of these assessments and it is therefore crucial that they are performed with utmost care, being mindful of possible confounding factors (Table 2.1).

Impairments of pupillary response may be due to damage at a number of locations along the afferent and efferent pathways. Direct trauma to the orbit may cause rupture of the pupil sphincter muscles and produce a traumatic mydriasis. Traumatic optic neuropathy may be due to direct disruption due to penetrating injury or indirectly by shearing forces in blunt head trauma. Lesions in the region of the pretectal nuclei or Edinger–Westphal nuclei in the rostral midbrain will also cause mydriasis. There are complex regulatory pathways from various sources including the ipsilateral hypothalamus that drives sympathetic pupillary tone and indeed poorly understood descending control from the cortex that results in ipsilateral or contralateral mydriasis or miosis following a seizure (Plum and Posner, 2007). Therefore, damage in many brain regions may result in pupillary abnormalities (Fig. 2.1). Compression of the parasympathetic fibres along the third cranial nerve by the herniating uncus is a common cause of pupillary abnormality in neurosurgical practice.

Table 2.1 Confounding factors in assessment of GCS

Glasgow Coma Score	Confounding factors
Eye opening (4 = spontaneous, 3 = to speech, 2 = to pain, 1 = do not open)	Orbital injuries Ecchymosis Photophobia
Verbal response (5 = oriented, 4 = confused, 3 = inappropriate words, 2 = incomprehensible sounds, 1 = no sound)	Non-native speakers Maxillofacial injury Endotracheal intubation/tracheostomy Deafness
Motor response (6 = obeys commands, 5 = localizes to pain, 4 = flexion/withdrawal to pain, 3 = abnormal flexion to pain, 2 = extension to pain, 1 = no movement)	Spinal cord injury Upper limb fractures, casts, and fixation

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The Glasgow Coma Score brings together diverse clinical features to provide a guide to global brain function. Of the three divisions (eye opening, verbal response, and motor response) the motor response is most likely to differentiate the severity of injury. Lesions above the red nucleus produce decorticate posturing (flexion of upper limbs and extension of lower, rubrospinal tract function) and those below produce decerebrate posturing (extension in upper and lower limbs, vestibulospinal tract function). Normal flexion (M4) constitutes flexion with supination; abnormal flexion (M3) constitutes flexion with pronation analogous to decorticate posturing and release of rubrospinal tract function as just described; and extension (M2) is analogous to decerebrate posturing and release of vestibulospinal tract function. It is best to apply painful stimuli to trigeminal territories in case of spinal cord injury causing peripheral numbness. Pressure over the supraorbital notch is sufficient.

Rising blood pressure, falling heart rate, and altered respiratory pattern (Cushing's triad) is a classical response to raised intracranial pressure but is usually a very late, agonal feature.

Examination of language and speech disorders

It is important to appreciate any abnormalities of speech and language as this can impact on the history taking, neurological examination, and assessment of higher function and thus alter the outcome of the consultation in general. The ability to correctly identify disorders of speech can aid in localization of neurological pathology. In order for us to communicate through speech and language, hearing, understanding, voice production, articulation, consciousness, thought, and word finding must be intact. Language is a complex

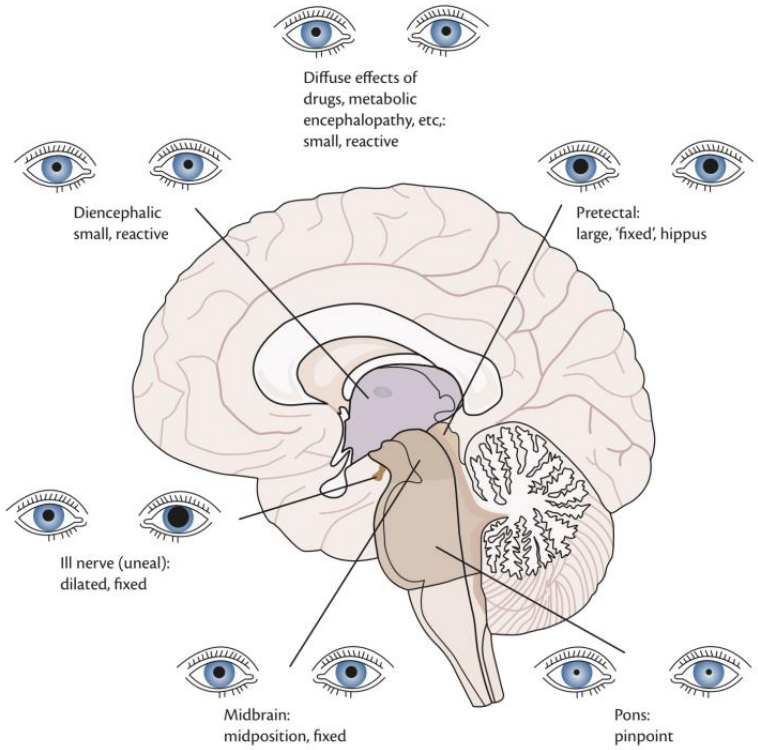

Fig. 2.1 Typical pupillary abnormalities associated with anatomical location of damage.
Reproduced with permission from Kandel et al., *Principles of Neural Science*, Fifth Edition, McGraw Hill, New York, Copyright © 2000.

interaction of combinations of sounds, writing, and meaning often linked to a cultural background.

To assess understanding start by engaging the patient in normal conversation and asking simple questions: 'What is your name? What is or was your occupation? How did you get here today?' Ensure that the patient can hear you properly and enquire as to what their first language is. Establish if the patient is left- or right-handed. As a rule of thumb, 99% of right-handed individuals have language dominance in their left hemisphere; 60% of left-handed patients will be left hemisphere dominant; 20% bilateral; and 20% right hemisphere dominant.

Now consider the different disorders of language defined here:

1. Aphasia—this is defined as a disorder of spoken language. It is divided into subcategories as follows:
   - 1.1 Non-fluent aphasia (anterior, motor, or Broca's);
   - 1.2 Fluent aphasia (posterior, sensory, or Wernicke's);
   - 1.3 Conduction aphasia;
   - 1.4 Transcortical aphasia (sensory and motor).
2. Alexia or dyslexia is a disorder of acquired reading ability.
3. Agraphia or dysgraphia is defined as disorders of written language.
4. Dysarthria is a disorder of articulation or speech production.
5. Dysphonia is defined as an abnormality of noise production by expired air over vibrating vocal cords.

Further examinations of these disorders include assessment of spontaneous speech, fluency, naming, repetition, articulation, speech volume, reading, and writing, and will help to localize the pathology.

1. Assessment for aphasia

Assess spontaneous speech, fluency, and if the patient uses the wrong words (paraphasia). Ask the patient to name animals or words beginning with 'F' in a minute. This tests word finding ability. Also ask them to name familiar objects: a pen, a watch, a tie, and so on. Now ask the patient to repeat phrases. Table 2.2 summarize the findings on examination and localization.

2. To assess for alexia or dyslexia, ask the patient to read a sentence or obey a written command.
3. Agraphia or dysgraphia can be examined by asking the patient to write a sentence; this can only be assessed if there is no motor disability.
4. Dysarthria is examined by asking the patient to repeat a phrase; for example, 'red lorry, yellow lorry' requires intact lingual function and 'baby hippopotamus' requires intact labial function. Listen for slurring and rhythm of speech. Dysarthria can be described as spastic (caused by pseudobulbar palsy as in motor neuron disease), extrapyramidal (associated with Parkinsonian syndromes, often associated with dysphonia), cerebellar dysarthria (associated

Table 2.2 Assessment of aphasia (Clark, 2009; Fuller, 2013)

	Comprehension	Fluency	Naming	Repetition	Other features	Localization
Non-fluent aphasia	Intact	Non-fluent	Impaired	Impaired	Right hemiplegia, depressed	Left frontal lobe (inferior and temporal insula), Broca's area
Fluent aphasia	Impaired	Fluent	Can be intact	Impaired	Neologisms, meaningless speech, paranoid, could have a visual field defect	Posterior superior temporal lobe, Wernicke's area
Conduction aphasia	Intact	Fluent	Impaired	Impaired	Depressed, cortical sensory loss right arm	Parietal operculum/ arcuate fasciculus
Global aphasia	Impaired	Non-fluent	Impaired	Impaired	Right hemiparesis worst in arm	Peri-Sylvian, both Wernicke's and Broca's areas
Nominal aphasia		Non-fluent	Impaired			Angular gyrus
Transcortical motor aphasia	Intact	Fluent	Impaired	Intact	Halting, effortful speech	Left anterior superior frontal area
Transcortical sensory aphasia	Impaired	Fluent	Can be intact	Intact	Semantic paraphasia	Posterior temporo-occipital-parietal area
Transcortical mixed aphasia	Impaired	Non-fluent	Impaired	Intact		Both Wernicke's and Broca's areas

Data from Clarke, C; Howard, R; Rossor, M; Shorvon, S. (2009) *Neurology: A Queen Square Textbook*. Wiley-Blackwell (p. 252–4), Fuller, G. (2004) Neurological Examination Made Easy, 3e. Churchill Livingstone. (p. 17–25).

with multiple sclerosis, alcohol intoxication, or inherited ataxia), and lower motor neuron dysarthria (this is caused by lesions affecting palatal movement causing nasal speech, tongue movements causing patients to struggle with the letters 'T' and 'S' and facial movement resulting in difficulty with the letters 'B', 'P', 'M', and 'W' and often involves the lower cranial nerves).

5. Finally listen to the volume of speech; if this is reduced it is described a dysphonic speech.

The lobar examination

The examination of the functions of the individual lobes of the brain should be a familiar and fluent part of the neurosurgeon's assessment. One should be aware of the somewhat arbitrary divisions between the lobes, however, and it may well be that one should test more than one lobe if the lesion is on or near a dividing sulcus. Anatomically, Yasargil's seven lobe system is most satisfactory (frontal, central, parietal, occipital, temporal, insular, and limbic; see Ribas, 2010) but here we will use frontal, parietal, temporal, occipital, and cerebellum.

Frontal lobe

As one might expect, as it is the largest lobe, frontal examination has the most components and complexity (**Box 2.1**). Anatomically, it is useful to consider four distinct regions—the precentral gyrus, the dorsolateral cortex, the orbitofrontal cortex, and medial cortex (**Fig. 2.2**).

The assessment of the function of the primary motor cortex requires testing for upper motor neurone signs on the opposite side. The patient may adopt postures typical of pyramidal weakness (i.e. flexors stronger than extensors in the upper limbs and vice versa in the lower limbs). Pronator drift is perhaps the archetypal neurosurgical test and is extremely useful in bringing out subtle weakness. Lying anterior to the primary motor cortex lies an area known laterally as the premotor cortex and medially as the supplementary motor area (SMA). The functions of these areas are complex but act in conjunction with the primary motor cortex. The cortical area immediately anterior to the primary motor cortex (Brodmann's Area 6) comprises of the lateral premotor area on the lateral aspect and the SMA on the medial and interhemispheric aspect. The lateral premotor area has reciprocal connections with the cerebellum and is involved with refinement of movements with external sensory cues. The SMA has reciprocal connections with the basal ganglia and is involved with initiation of movements from internal sensory cues. In contrast to the primary motor cortex homunculus (leg medial, upper limb, face, and tongue lateral) the SMA homunculus is arranged horizontally (leg posterior adjacent to the paracentral lobule and primary motor area for leg, upper limb, then face and tongue more anteriorly). The SMA has roles in postural stability in walking, initiating and sequencing movements, and coordination of both sides of the body. Frontal lobe ataxia causes a characteristic magnetic gait as if stuck to the floor (Brun's apraxia). This has similarities to Parkinsonian gait, but does not have the lack of arm swing. It is part of the clinical triad of normal pressure hydrocephalus (disturbance of gait, continence, and cognition). This reflects the anatomical proximity of the micturition inhibitory area (just inferior to

Box 2.1 Scheme for frontal lobe assessment

- Ask about handedness and assess speech
- Observe behaviour—abulia, inappropriate dress, verbal dysdecorum
- Posture/gait—decorticate, 'magnetic'
- Pyramidal weakness
- Saccadic eye movements
- Primitive reflexes
- Look for urinary catheter
- Anosmia and Foster Kennedy syndrome
- Neuropsychological tests—echopraxia, perseveration, conceptualization, working memory

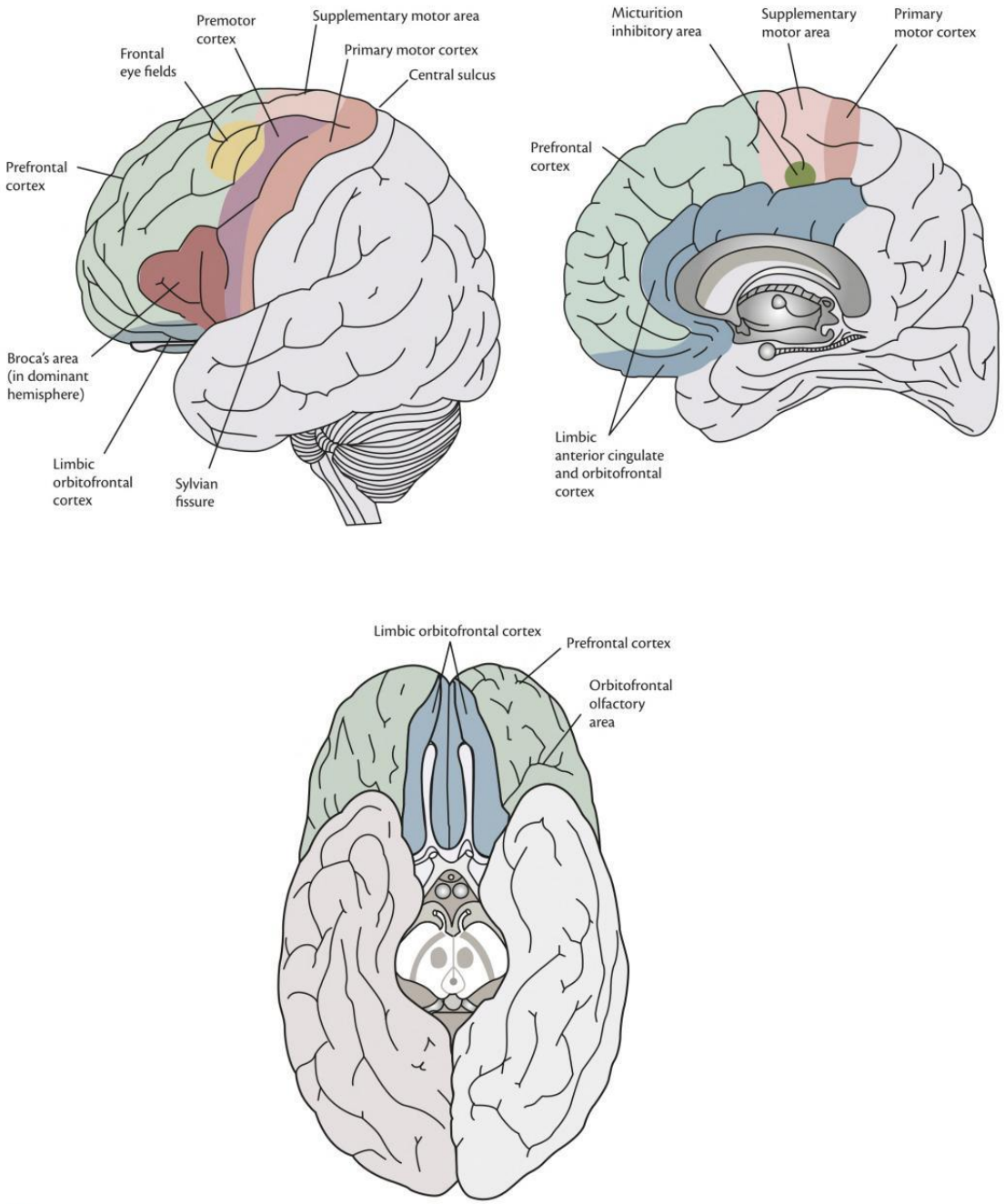

Fig. 2.2 The frontal lobe.
Neuroanatomy through Clinical Cases, 1st Edition by Blumenfeld (2002) Fig.19.11 p. 848. By permission of Oxford University Press, USA.

the SMA) and the more diffuse role of the frontal lobe in cognition. Frontal lobe dysfunction may also cause gegenhalten—the resistance to passive movement.

The frontal eye fields (FEFs, Brodmann's Area 8) lie in the posterior part of the middle frontal gyrus and adjacent precentral sulcus. A mass lesion here may result in horizontal conjugate gaze deviation

towards the side of the lesion (Prévost or Vulpian sign). Seizures will result in looking away from the lesion. Saccadic eye movement should be tested by asking the patient to look between two fixed points (e.g. a fist and a finger) while keeping the head still. Damage to the FEF may result in impaired saccades away from the lesion.

Re-emergence of primitive reflexes or 'frontal release signs' may also be demonstrated. The grasp reflex is assessed by gently passing a finger across the palm. It is most reliably done by distracting the patient by asking them to count backwards from ten. The palmomental reflex is a brief contraction of the ipsilateral mentalis muscle in response to stroking the palm. The snouting reflex is seen when the lips purse in response to tapping the upper lip or the sucking reflex when pressing an object to the lips. Glabellar tap may also cause persistent blinking, whereas in the normal individual it attenuates. It should be remembered that these reflexes may well be seen in normal individuals, particularly in older people (25% of normal adults have the palmomental reflex; see Brazis et al., 2011).

The prefrontal area contains a large volume of brain that has complex roles in control of behaviour. One may divide these into restraint (the restriction of behaviour to that which is socially and culturally acceptable), initiative (motivation to put thoughts into action), and order (to sequence tasks appropriately; see Blumenfeld, 2002). Simply observing and talking with the patient will give insights into this. Apparently quite contradictory behaviours may be encountered, some patients lacking any kind of 'get up and go' compared to others for whom it is difficult to stop talking, or who are overfamiliar or tactless. In general, abulia is more frequently seen in lesions of the dorsolateral convexity whereas disinhibition is often an orbitofrontal feature. There are many neuropsychological tests that are pertinent to examining the frontal lobes (**Box 2.2**).

Parietal lobe

The parietal lobe (**Fig. 2.3**) may be divided into the postcentral gyrus, posteriorly is the superior parietal lobule and inferior parietal lobule (angular and supramarginal gyrus). Processing of somatic sensations and perceptions occurs in the postcentral gyrus (monomodal); the posterior parts are polymodal assimilating inputs from somatic, visual, and other sensory modalities mostly for the control of movement especially the hand and upper limb (Kolb and Whishaw, 2009). The left inferior parietal lobule has a role in language and is considered under that heading. Disorders of the parietal lobe will impair sensation. However, as much of somatic sensation is processed in the thalamus there will not be complete numbness but rather more subtle impairments. These may be tested looking for sensory extinction, astereoagnosia, dysgraphasthesia, and two-point discrimination (**Box 2.3**).

Lesions of the dominant parietal lobe have been associated with a collection of signs known as Gerstmann's syndrome. Although rare in combination, it is still useful to have these four signs in mind for completeness of examination (**Box 2.4**). For the non-dominant parietal lobe (**Box 2.5**) there is a preponderance for there to be contralateral neglect. This may be noted from the patient's appearance with lack of grooming on one side. Other features may become apparent such as loss of geographical orientation (getting lost in familiar places), dressing apraxia, and anosognosia (lack of awareness of illness).

Temporal lobe

The temporal lobes have roles in processing auditory information (Heschl's gyrus), visual information (inferotemporal cortex), emotion (amygdala), and memory and spatial navigation (hippocampus). Its role in language is important especially in the vocalization (dominant) and perception of (non-dominant) emotion in language (emotional prosody). It should be recalled that Meyer's loop passes over the roof of the temporal horn and lesions may, therefore, cause a 'pie in the sky' quadrantanopia. Apart from simple tests of memory, speech, and visual fields there are limited bedside tests for the temporal lobe. Neuropsychologists may perform dichotic listening tests where different recordings are presented to either ear of a pair of headphones to assess selective auditory attention or may carry out advanced tests of verbal and non-verbal memory.

Occipital lobe

Vision and its interpretation is the primary function of the occipital lobe and is dealt with in that section.

Cerebellum

The cerebellum coordinates smooth, planned motor actions by analysing extensive sensory inputs from the brain and spinal cord. The midline structures (the vermis and flocculonodular lobes) control coordination of trunk and eye movement. The cerebellar hemispheres maintain control of limb movement and aid motor planning. Because cerebellar outputs remain ipsilateral or cross twice, lesions of a cerebellar hemisphere will cause an ipsilateral deficit. Ataxia is a characteristic sign of cerebellar dysfunction. Movements are clumsy due to poor coordination between agonist and antagonist muscle groups. There is imprecision of trajectory though space (dysmetria) and also in timing of movements. Truncal ataxia may make even sitting up in bed very difficult. Gait may be wide-based and staggering. Appendicular ataxia may be tested by asking the patient to repeat rapid alternating movements of the limbs (e.g. supinating/pronating one hand on the other). When abnormal this is known as dysdiadochokinesia. One can also ask the patient to outstretch their arm, then touch their nose, or to touch the examiner's finger and back to their nose. An intention tremor may be also elicited. Holmes

Box 2.2 Neuropsychological tests of frontal lobe function

Luria's three-step test
This tests motor sequencing. Tell the patient you are going to show him a series of hand movements. Without verbal prompting show a sequence of fist, edge, palm, and repeat five times. Ask the patient to do the same. The patient may demonstrate perseveration with repetition of the same movement or be quite unable to do the sequence in order.

Go/no-go test
This tests ability to shift set. Hold two fingers out palm down—'put out one finger when I do this', hold down one finger—'put out two fingers when I do this'. Do this several times. Then change the instructions. Place two fingers—'put out one finger when I do this'. Place one finger—'do nothing when I do this'. The inability to follow the second set of instructions implies a deficit.

Verbal fluency
Produce as many words beginning with a particular letter in one minute, proper nouns not allowed. Normally 12 or more.

Tower of Hanoi
A game moving discs between stacks aiming to achieve goal in as few moves as possible

Wisconsin card sorting test
This is a card matching test testing executive function.

Stroop test
Read out a list of coloured words (i.e. the word green spelt out in yellow text). Test of restraint.

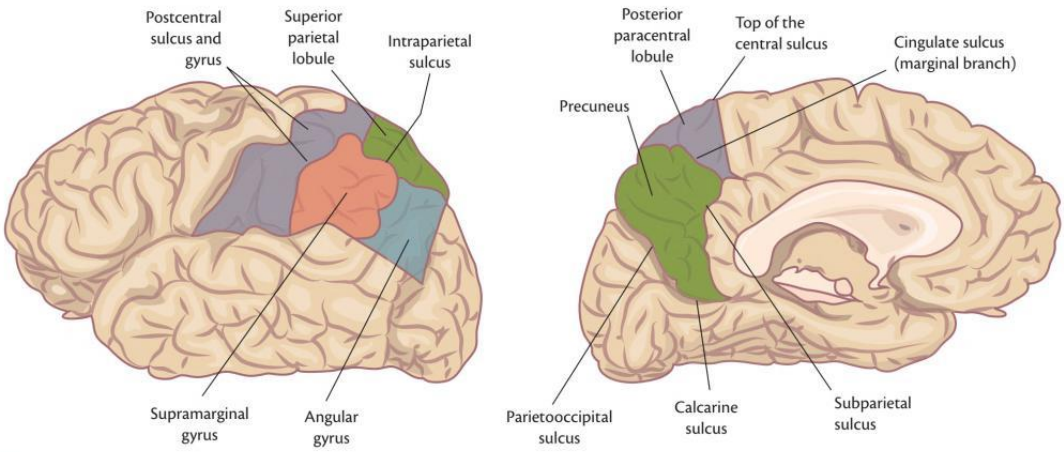

Fig. 2.3 Parietal lobe.

rebound test demonstrates overshooting. For example, with arms outstretched and eyes closed, the examiner pushes one arm downward. On release the patient's arm shoots up higher than originally placed. There is ipsilateral hypotonia and a pendular knee jerk may be found. The cerebellum has a role in articulation and when damaged will cause dysarthria with laboured, slurred speech. Mutism is a well-recognized problem following surgical resections in the midline, well documented in children following medulloblastoma resection. The anatomical basis is from disruption of a dentato-rubro-thalamo-cortical pathway such that bilateral dentate nucleus injury causes mutism. This explains why dominant hemisphere SMA injury, bilateral thalamotomies and mesencephalic strokes (red nucleus) can result in the same syndrome. Cerebellar mutism was originally, mistakenly, attributed to approaches that split the vermis given the close proximity of the dentate nuclei to the midline.

Nystagmus may also be present with the fast phase towards the abnormal side. Vertical nystagmus (e.g. downbeat nystagmus may be seen in Chiari malformation). Head tilt may occur in children with posterior fossa lesions. Cognitive-affective symptoms are increasingly being recognized in cerebellar disorders.

Box 2.3 Examination of parietal lobe function (either hemisphere)

Sensory extinction
Ask patient to hold out arms with eyes shut. Touch either one or both sides of the corresponding part of the body and ask where he has been touched. Extinction occurs when the patient says that only one side is being touched when in fact it is both.

Stereognosis
With the patient's eyes closed, place a familiar object in their hand and ask them to identify it. Coins of different denominations may be used.

Graphesthesia
Ask the patient to identify the number or letter that you trace on their palm. It should be agreed which way is up before starting.

Two-point discrimination
Using callipers or a bent paper clip, ask the patient if they can feel one or two points. On the fingertips one should be able to recognize two separate points to about 2–4 mm apart, on the palm 8–15 mm.

Visual fields
Examine for homonymous inferior quadrantanopia.

Constructional apraxia
Ask patient to copy a 3D drawing.

Box 2.4 Parietal lobe examination—dominant hemisphere (Gerstmann's syndrome)

Dyscalculia
Ask the patient to subtract 7 from 100 and continue subtracting 7 sequentially.

Agraphia
Ask the patient to write a simple sentence.

Finger anomia and left-right disorientation
These two can be examined together by crossing your hands and asking, 'Which finger am I wiggling?' (Fig. 2.4) or alternatively asking the patient to touch their right ear with their left ring finger. Remember to also assess speech (see earlier).

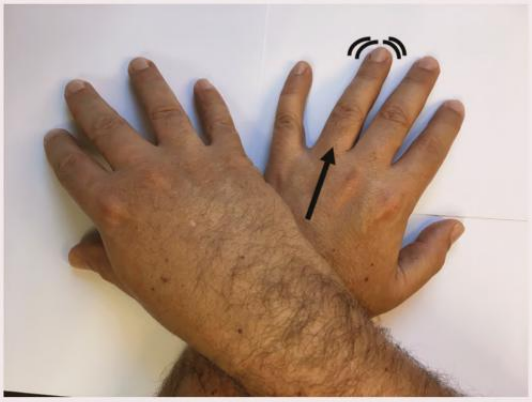

Fig. 2.4 Testing finger anomia and left-right disorientation: 'Which finger am I wiggling?' Correct answer: 'Your left ring finger.'

Box 2.5 Parietal lobe examination—non-dominant hemisphere

Unilateral spatial neglect
Ask the patient to mark the middle of a horizontal line. Displacement of the centre towards the side of the brain lesion (generally right hemisphere) indicates neglect. Target cancellation tests can also be used. This is where a patient is asked to circle every 'a' on a page or something similar. Only one side of the page will be attended to.

Crossed response test
Ask patient to move the limb opposite the one that is touched (motor neglect).

Dressing apraxia
Ask the patient to take off a jumper or other item of clothing. Turn it inside out and ask the patient to put it back on the right way.

Paper cutting
Ask patient to use a pair of scissors to cut out a shape from a piece of paper.

The visual system examination

This section will detail the examination of the eye and cranial nerves II, III, IV, and VI. A thorough history is necessary to understand the patient's range of visual symptoms. These may include drooping eyelids, blurred vision, double vision, 'seeing things', visual loss (transient or persistent, partial, or complete), abnormal movements of the visualized world, eye pain, headache, or orbital pain. In this section we will provide an examination example that avoids missing the most important eye signs.

The neuro-ophthalmological examination should include:

1. *Visual acuity*
   Optic nerve, chiasmal, optic tract lesions, and ocular pathology can all influence visual acuity. This is assessed with corrected vision or refractive vision, like the patient's own glasses or a pin hole. The standard is the Snellen chart that measures vision at a distance of 6 metres. This is expressed as a fraction (distance in metres from chart/distance in metres at which letters should be seen). If the acuity is reduced but is correctable with refraction; this is due to an ocular defect. If the acuity is not correctable, then it signifies a problem in the visual pathway. If the patient is unable to see the largest print, the chart can be brought closer or assessed by finger counting, perception of hand movements, or perception of light.
2. *Colour vision*
   This is most useful for assessing the optic nerve function. It is assessed by using Ishihara plates and scored in each eye individually by the number of plates correctly identified. The speed of identifying the plate should also be considered when comparing the eyes. Remember that 8% of males and 0.5% of females may have X-linked recessive congenital colour deficiency or dyschromatopsia. In these patients the loss of colour vision will be bilateral with normal visual acuity and fields. A cruder method of assessing colour vision is to ask the patient to look at a coloured target like a red cap and in the affected eye it may seem faded or 'washed out'.
3. *Visual field testing*
   Visual fields are tested to direct confrontation and each eye is assessed individually. The patient should fixate on the examiner's nose and cover each eye in turn. This test can detect hemianopia, quadrantanopia, altitudinal, and central field defects. With the eye covered ask the patient if all the parts of the examiner's face are clear, or if there are parts that are blurred or missing. Ask the patient to count fingers in each quadrant.

   Peripheral fields are best assessed with a white hat pin, whereas central fields and blind spots should be tested with a red hat pin. The latter is assessed against the examiner's blind spot by moving the pin. Visual field defects are mapped out according to the patient's description. Abnormalities are illustrated as follows (see Fig. 2.5) and can localize the defect in the visual pathway All patients with a suspected visual field

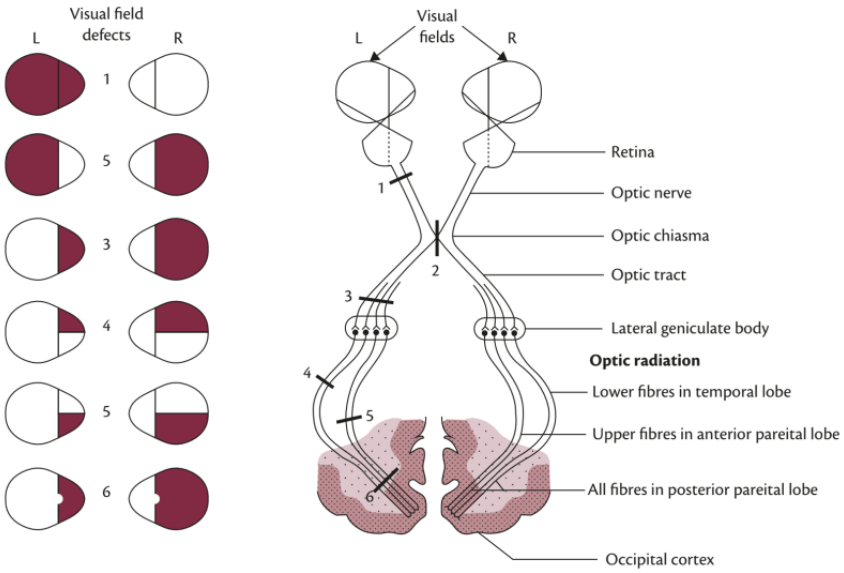

Fig. 2.5 Assessment of visual fields and localization of defects.
From: *Macleod's Clinical Examination*, Ninth edition figure 7.8 page 211, J. Munro and C.R.W. Edwards.

Box 2.6 Parinaud's syndrome

- Impairment of upgaze
- Large irregular pupils that do not react to light but can accommodate (light-near dissociation)
- Eyelid abnormalities—lid retraction or ptosis
- Impaired convergence
- Nystagmus retractorius

defect should have formal perimetry for accurate localization and monitoring. Macular sparing with ischaemic occipital lobe lesions relates to the dual blood supply to the occipital pole from both the middle cerebral artery as well as the posterior cerebral artery.

4. *Pupils*

The pupillary light reaction pathways include the optic nerve (afferent) and the parasympathetic component of the third cranial nerve (efferent). Accommodation arises from the frontal lobes (afferent) and the parasympathetic component of cranial nerve III (efferent).

Inspect first for anisocoria, and then ask the patient to fixate on a distant target and then test the direct and indirect light reflexes, the pupillary reactions to accommodation and finally perform the swinging light test.

4.1 *Size*

The examination of the pupils should first be conducted in room light, if the anisocoria is greater in light than in dark, the parasympathetic system is abnormal, and the larger pupil is abnormal. A cranial nerve III paresis, Adie's pupil, or damaged iris sphincter are examples of the parasympathetic pathway. Then examine for anisocoria in dim light, if this is greater in dim light, a sympathetic dysfunction is present and the smaller pupil is abnormal. Horner's syndrome is a manifestation of a sympathetic disorder. If the anisocoria is the same under both conditions, it is not indicative of a neurological problem.

Conditions that impair light response but do not affect accommodation (light-near dissociation) include Parinaud's syndrome (Box 2.6), neuro-syphilis, diabetes mellitus, Adie's pupil, bilateral optic neuropathy, and aberrant regeneration of cranial nerve III.

4.2 *Light response (direct and indirect)*

Shine a bright light in each eye, and observe the pupils for speed and magnitude of constriction (see Fig. 2.6). If the pupil does not react normally to light, examine the response by viewing a near target. Again, check for speed and magnitude of constriction to a near target and the speed of dilatation when looking at a distant target.

4.3 *Swinging light test*

To perform this test the lights should be dimmed while the patient should fixate on a target in the distance. Swing the light from eye to eye for about a second at a time. Look for the initial movement in each pupil (normally constriction). To diagnose a relative afferent pupil defect (RAPD), one pupil should consistently dilate rather than constrict. The presence of a RAPD is indicative of optic neuropathy (Table 2.3).

5. *Ophthalmoscopy*

For the purposes of this chapter we advise that ophthalmoscopy should be performed on all patients to examine for papilloedema that can suggest raised intracranial pressure and optic pallor suggestive of optic atrophy. Systematically examine the optic disc, the bloods vessels, and then the retinal background.

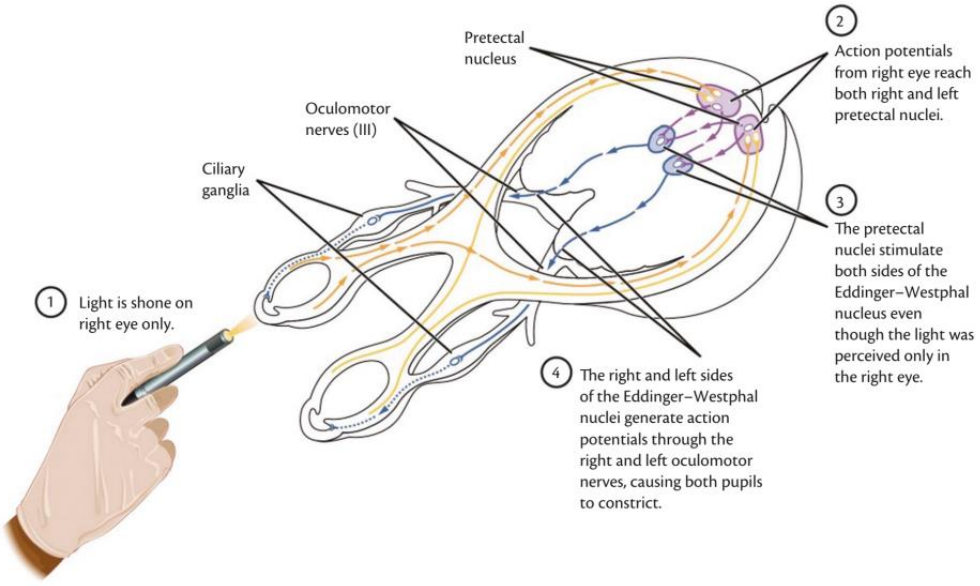

Fig. 2.6 The light reflex.

Authored by: OpenStax College. Provided by: Rice University. Project: Anatomy & Physiology. http://cnx.org/content/col11496/latest/

Table 2.3 Relative afferent pupil defect (Pane, 2007)

Normal pupil		
Light stimulus	**Right pupil**	**Left pupil**
None	•	•
Left	•	•
Right	•	•
Left relative afferent pupil defect		
Light stimulus	**Right pupil**	**Left pupil**
None	•	•
Left	•	•
Right	•	•

Table 2.4 summarizes some findings that can aid in the localization of lesions that present with visual symptoms using the techniques described so far.

6. *Eye movements*

6.1 *Fixation, nystagmus, and saccades*

Disorders of fixation include nystagmus and saccadic intrusions. The examination will stem from a history of involuntary eye movements or oscillopsia. Congenital fixation disorders are usually asymptomatic and incidentally noticed.

Nystagmus is the rhythmic oscillation of the eyes. Start the examination with eyes in the primary position, then different positions of gaze. Note the fast phase of the nystagmus and the direction or if it is pendular. Also note if the nystagmus is similar in both eyes and if there is a latent component or increased nystagmus with one eye covered. Different types of nystagmus can aid in localization of pathology as summarized in Table 2.5.

Assessing how rapidly and accurately patients can fixate on an eccentric target by testing saccades can expose a subtle internuclear ophthalmoplegia or sixth cranial nerve palsy. It is tested by asking the patient to look at two different objects on either side of the patient's head. The patient is asked to alternate gaze between the objects. Note saccadic initiation, velocity, and accuracy. This can be either too small (hypometric) or too large (hypermetric).

6.2 *Eye movements and cranial nerves III, IV, and VI*

Eye movements include saccades controlled by the frontal lobe, pursuit (the slow movement that facilitate fixation on a moving object) that is controlled by the occipital lobe, the vestibulo-ocular reflex (that allows compensation of eye position for movement of the body or head) controlled by the cerebellar vestibular nuclei and convergence (fixating on an object close to the face) that is controlled by the midbrain. Input from the different control centres have to be integrated to allow synchronous eye movement. The medial longitudinal fasciculus (MLF) in the midbrain runs between the nuclei of cranial nerves III and IV in the midbrain and VI in the pons. The eye muscles are the lateral rectus controlled by cranial nerve VI, the superior oblique controlled by cranial nerve IV, and all the rest are controlled by cranial nerve III.

6.2.1 *Cranial nerve III palsy (oculomotor nerve)*

A complete palsy is a triad of ptosis, a large and unreactive pupil, and the eye position of 'down and out' (Fig. 2.7). Pupil sparing implies that the deeper nerve fibres have been affected most likely from ischaemia. The parasympathetic fibres lie circumferentially on the outside of the nerve and are more commonly affected by compressive causes, classically a posterior communicating artery aneurysm.

Internuclear ophthalmoplegia is the result of a lesion of the MLF. The patient has dysconjugate eye movements; there is incomplete adduction of one eye and jerky nystagmus of the other eye on abduction when testing lateral gaze. It is described as left-sided when there is failure of left adduction when looking to the right (Fig. 2.8). The horizontal conjugate gaze centre resides in the paramedian pontine reticular formation at the level of the fifth cranial nerve nucleus. On an attempt to move the eyes in a horizontal direction, the abducting eye is able to move (VI) but the signal to the third nerve is unable to pass through the MLF to the third nerve is nucleus and there is a failure of adduction. The resulting diplopia may underlie the nystagmus in the abducting eye as there is an attempt to overcome this. The vertical gaze centre resides in the rostral interstitial nucleus of the MLF (riMLF) at the level of the third nerve/superior colliculus.

Table 2.4 Localization of lesions in the visual system (see Beck and Smith, 1988)

	Optic nerve	Optic chiasm	Optic tract	Temporal lobe	Parietal lobe	Occipital lobe
Visual acuity	Normal or reduced	Normal or reduced	Normal or reduced	Normal	Normal	Normal
Colour vision	Normal or reduced	Normal or reduced	Normal or reduced	Normal	Normal	Normal
Visual field	Central scotoma	Bitemporal	Homonymous incongruous	Homonymous superior	Homonymous inferior or complete	Homonymous exquisitely congruous
Relative afferent pupil defect	Present	Present or absent	Present or absent	Absent	Absent	Absent
Disc pallor	Present or absent	Present or absent	Present or absent	Absent	Absent	Absent