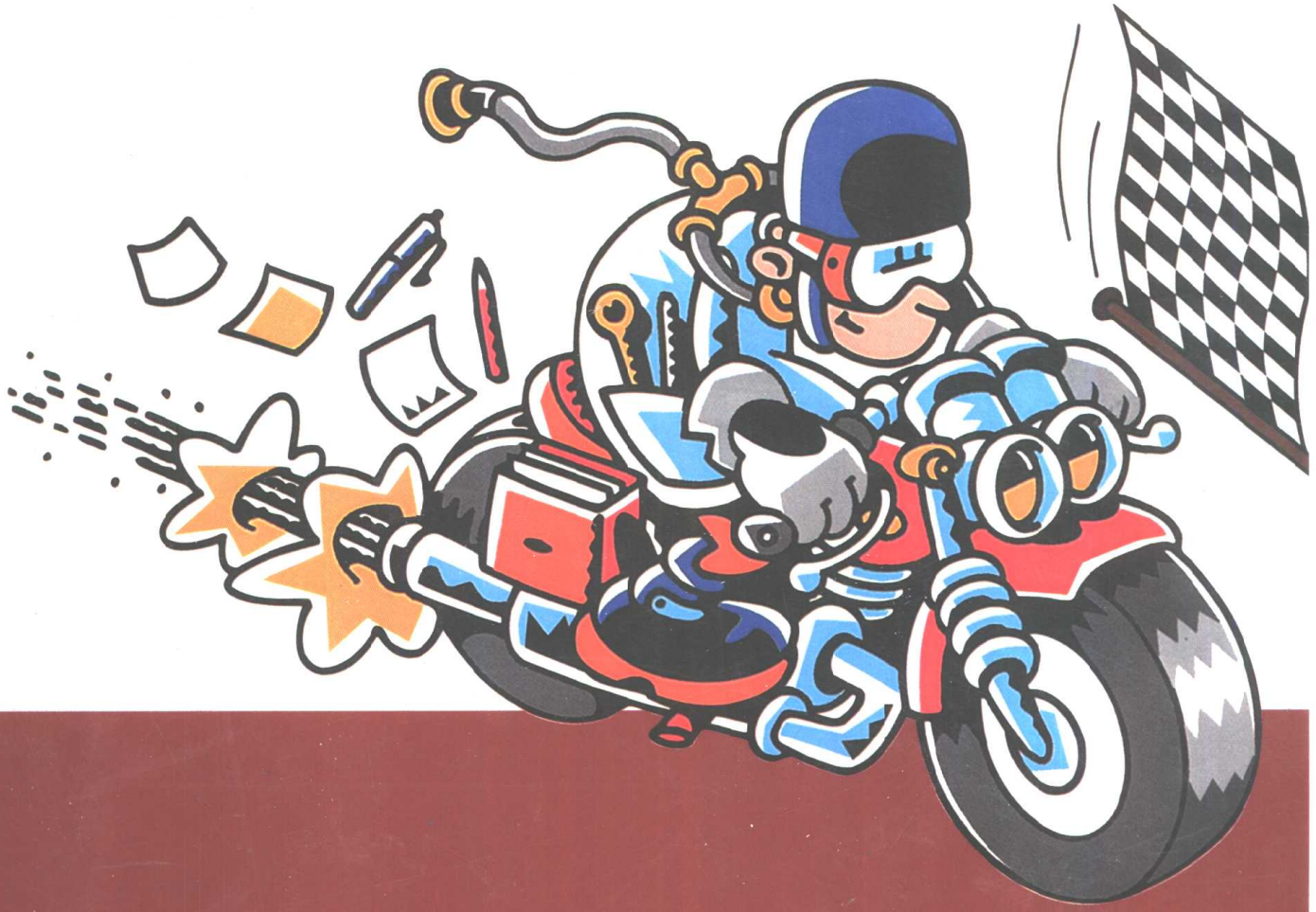
风暴式医学教程 MOSBY'S CRASH COURSE (原版英文医学教程)

神经病学

Neurology

Anish Bahra ⊙ Katia Cikurel
with Wilfred Yeo as Series Editor



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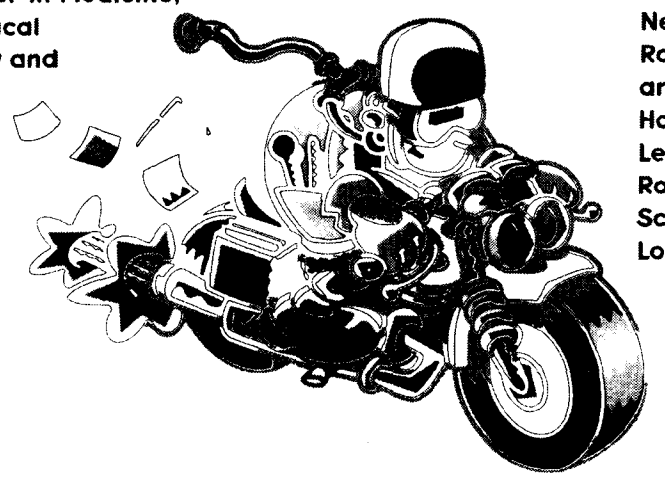

Neurology

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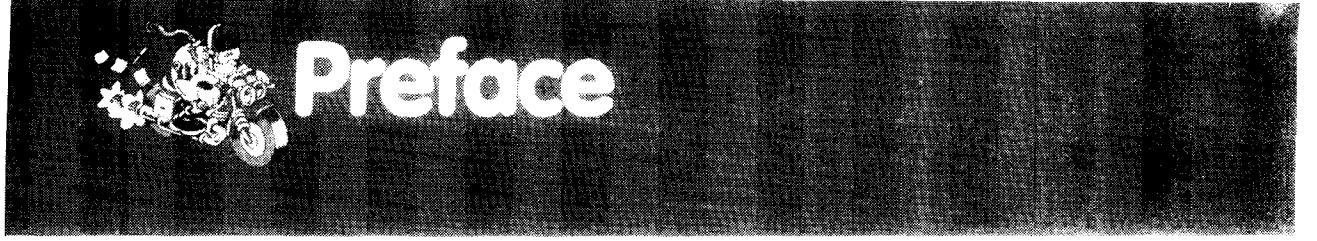


This compact book aims to provide a logical approach to understanding the fundamental complaints in neurology and addresses the most common neurological syndromes in clinical practice. It provides a useful illustrated guide to approaching a patient with a neurological problem in a concise and simple manner. We hope *Crash Course Neurology* will be valuable for both undergraduates preparing for exams and postgraduates in all fields of medicine.

Our gratitude for guidance to Jeremy Gibbs

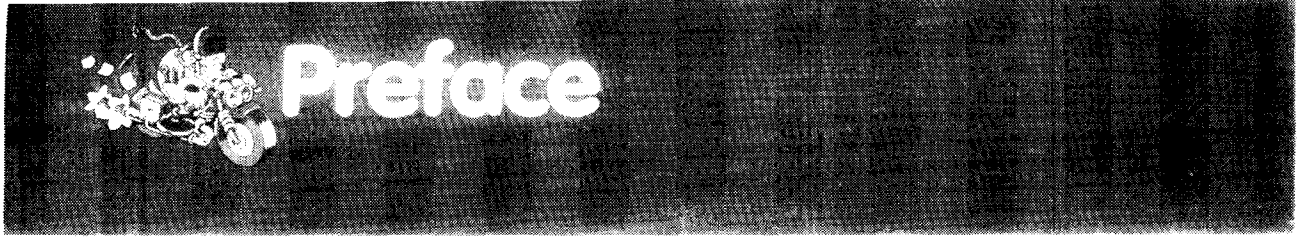
Anish Bahra
Katia Cikurel

All medical students and doctors need to acquire and maintain some basic skills in neurology. Neurological symptoms account for a high proportion of consultations in general practice, 20% of acute admissions to hospital, and many complications of trauma, critical illness, anaesthesia, and surgery. Diagnosis is primarily clinical, based on a careful history and physical examination, and any number of subsequent investigations can only supplement and never replace the process of clinical assessment.

Crash Course Neurology is a concise text presenting a lucid and systematic approach to neurological diagnosis and management. Part I of the book deals with the analysis and differential diagnosis of common presenting symptoms such as headache, dizziness, speech disturbance, and limb weakness. In Parts II and III the clinical features, investigation, and management of specific neurological disorders are discussed in more detail. The self-assessment questions and patient management problems at the end provide a useful check on the learning process and additional stimulus to effective reading.

The book is clearly written and logically set out, with numerous diagrams, lists, and tables to facilitate learning and revision. It is primarily designed as an introduction to neurology for students and a revision text for both finals and MRCP, but the general approach and much of the material could be usefully revisited by fully qualified doctors who encounter neurological problems during the course of their work.

Jeremy Gibbs
Faculty Advisor

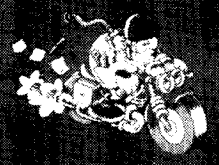


So you have an exam in medicine and you don't know where to start? The answer is easy—start with *Crash Course*. Medicine is fun to learn if you can bring it to life with patients who need their problems solving. Conventional medical textbooks are written back-to-front, starting with the diagnosis and then describing the disease. This is because medicine evolved by careful observations and descriptions of individual diseases for which, until this century, there was no treatment. Modern medicine is about problem solving, learning methods to find the right path through the differential diagnosis, and offering treatment promptly.

This series of books has been designed to help you solve common medical problems by starting with the patient and extracting the salient points in the history, examination, and investigations. Part II gives you essential information on the physical examination and investigations as seen through the eyes of practising doctors in their specialty. Once the diagnosis is made, you can refer to Part III to confirm that the diagnosis is correct and get advice regarding treatment.

Throughout the series we have included informative diagrams and hints and tips boxes to simplify your learning. The books are meant as revision tools, but are comprehensive, accurate, and well balanced and should enable you to learn each subject well. To check that you did learn something from the book (rather than just flashing it in front of your eyes!), we have added a self-assessment section in the usual format of most medical exams—multiple-choice and short-answer questions (with answers), and patient management problems for self-directed learning. Good luck!

Wilf Yeo
Series Editor (Clinical)



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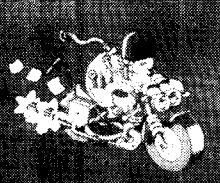

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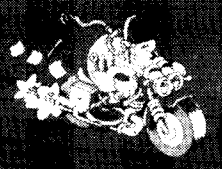

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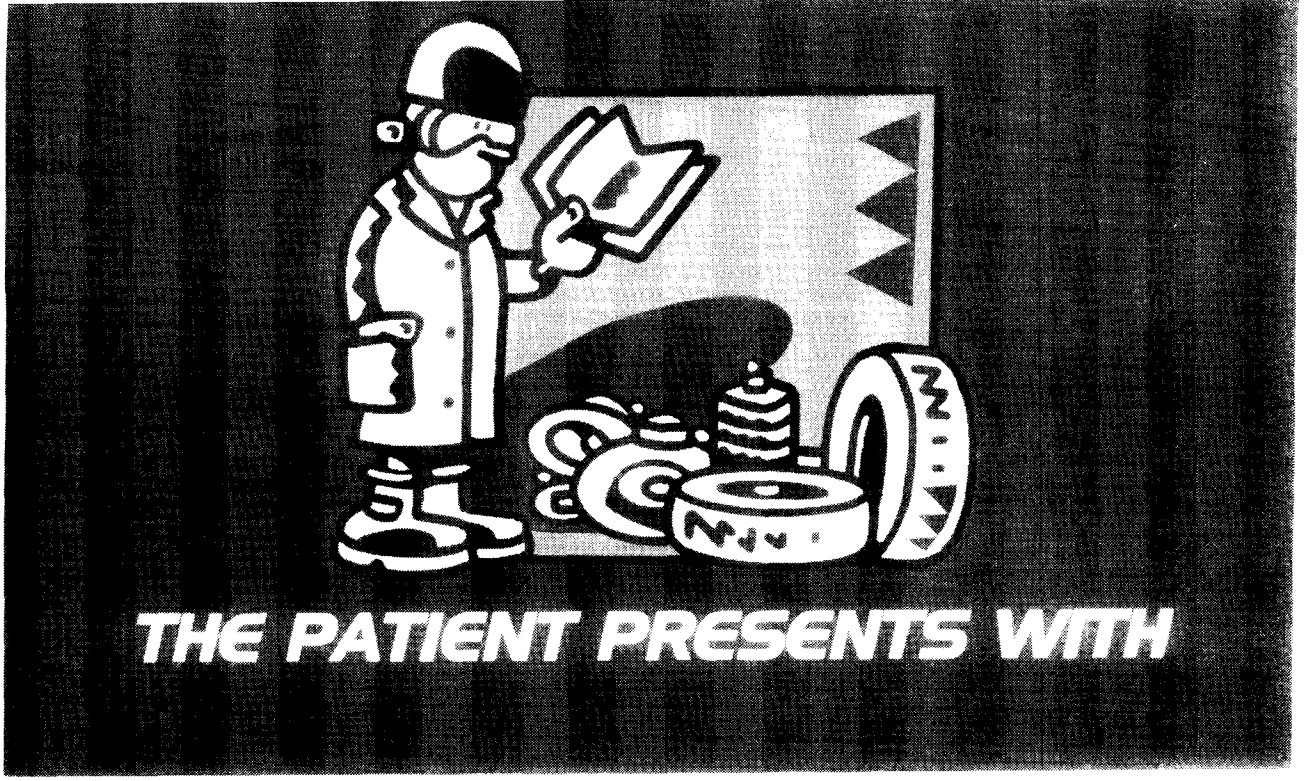

THE PATIENT PRESENTS WITH

1. Disorders of Higher Cerebral Function

Impairment of cognitive (intellectual) function is often a prominent manifestation of cerebral hemisphere disease. The common diffuse or multifocal pathologies in the brain—Alzheimer's or generalized vascular disease, respectively—cause the syndrome of dementia, in which several aspects of cognitive function are impaired. Single localized lesions will predictably cause more focal cognitive deficits, such as dysphasia or agnosia. For this reason it is important to be aware of the function of the different lobes and anatomical areas within the cerebral hemispheres. The four lobes—frontal, parietal, temporal, and occipital—are shown in Fig. 1.1. The cerebral cortices have specialized functions. Certain functions are attributes of either the right or left hemisphere; one hemisphere is therefore termed 'dominant' and the other 'non-dominant'. The left hemisphere is dominant in over 90% of right-handed people and in about 60% of left-handed people.

Cortical function and clinical manifestations of dysfunction of each lobe are considered below.

THE FRONTAL LOBE

Function

- The motor cortex. The primary motor cortex is concerned with motor function of the opposite side of the body; the corticospinal and corticobulbar fibres are topographically represented in Fig. 1.2.
- The supplementary motor cortex. This area is concerned with turning of the eyes and head contralaterally.
- Broca's area (dominant hemisphere). Broca's area is the motor centre for the production of speech. This is a function of the dominant hemisphere.
- The prefrontal cortex. Personality, emotional expression, initiative, and the ability to plan are governed by the anterior part of the frontal cortex.
- The cortical micturition centre. There is normally a cortical inhibition of voiding of the bladder and bowel.

The blood supply to the frontal lobe is from the anterior and middle cerebral arteries.

Lesions of the frontal lobe

Lesions of the frontal lobe give rise to:

- Contralateral mono- or hemiparesis and facial weakness of upper motor neuron type. The pattern of weakness depends on the area of cortex damaged.
- Paralysis of contralateral eye and head turning.
- Broca's expressive dysphasia. This comprises non-fluent, hesitant speech with intact comprehension. The patient knows what he or she wants to say but has difficulty finding the correct words, often producing the wrong word. The ability to repeat words is better than spontaneous speech.
- Behavioural change. Features of altered behaviour include social disinhibition, loss of initiative and interest, inability to solve problems and loss of

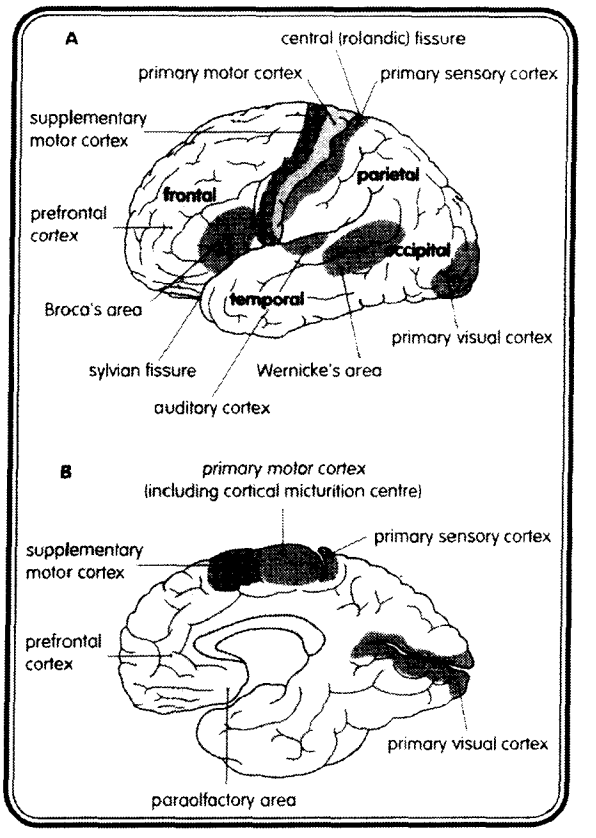

Fig. 1.1 *Functional regions of the cerebral cortex. (A) Lateral* left hemisphere. (B) Medial right hemisphere.

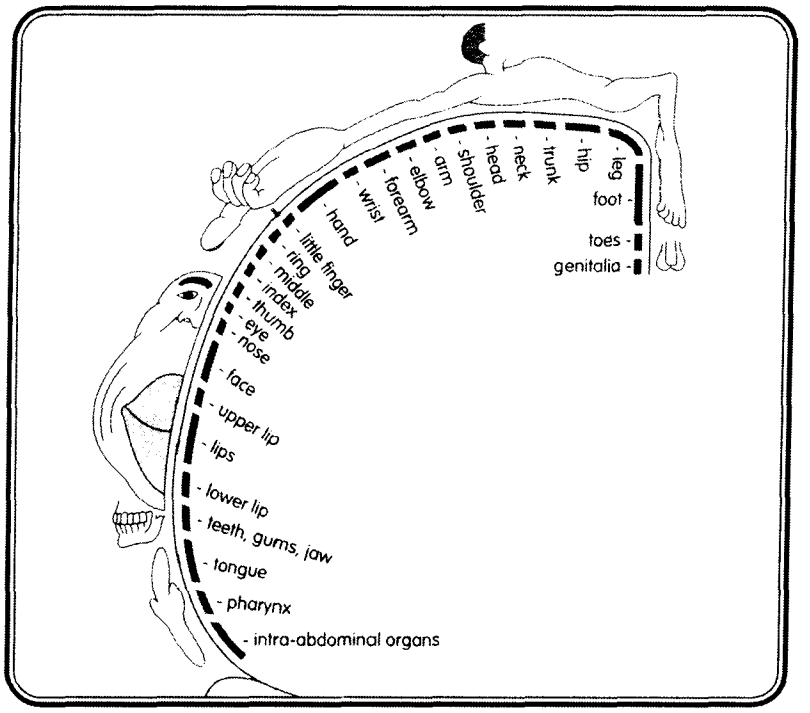

Fig. 1.2 Topographical distribution of the sensorimotor pathways.

abstract thought, and impaired concentration and attention without intellectual or memory decline. This is more common in bilateral lesions. Severe bilateral pathology may result in akinetic mutism, in which the patient is not paralysed and has the ability to speak but lies still and silent.

- Elicitation of primitive (grasping and sucking) reflexes. These reflexes originate from the parietal cortex and are usually inhibited by the prefrontal cortex.
- Apraxia of gait. This is the inability to walk normally despite preservation of motor and sensory function.
- Incontinence of urine and/or faeces. This results from loss of cortical inhibition. There is no desire to micturate. Milder symptoms are frequency and urgency of micturition.

Focal seizures arising from the frontal cortex give rise to clonic movements of the contralateral lower face, arm, and leg, and congugate deviation of the head and eyes toward the convulsing side, i.e. away from the side of the lesion.

By virtue of their proximity, lesions of the frontal lobe may be accompanied by disturbance of the olfactory and visual pathways.

THE PARIETAL LOBE

Function

- The sensory cortex. The parietal cortex receives afferent projections via the thalamus from the somatosensory pathways. The fibres are represented topographically as for the motor pathways.
- Language (dominant hemisphere). Pathways within the arcuate fasciculus connecting Broca's area (frontal) with Wernicke's area (posterior temporal) pass through the inferior parietal region.
- Use of numbers, e.g. calculation (dominant hemisphere).
- Integration of somatosensory, visual, and auditory information (mainly non-dominant). This allows awareness of the body and its surroundings, appropriate movement of the body, and constructional ability.
- Visual pathways. The upper part of the optic radiation (subserving the lower quadrant of the contralateral visual field) passes deep within the parietal lobe and may be affected in lesions of the deeper white matter.

The blood supply to the parietal lobe is from the middle cerebral artery.

Lesions of the parietal lobe

Lesions of the parietal lobe give rise to the following:

- Discriminative sensory impairment of the opposite side of the face and limbs. There is impairment of position sense and two-point discrimination, and inability to recognize objects by form and texture (astereognosis) or figures drawn on the hand (agraphaesthesia). Pain, temperature, touch, and vibration are intact; however, their localization when applied to the body may be impaired.
- Wernicke's receptive dysphasia. This arises from inferior parietal and superior temporal lesions (Wernicke's area and above). There is impaired comprehension of speech and written language without difficulty with expression. The speech is fluent but words are replaced with partly correct words and an incorrect word related to the word intended (paraphrasia) or newly created meaningless words (neologisms). Thus the speech does not make sense but the patient has poor insight into the problem.

Syndromes of the dominant hemisphere

Syndromes of the dominant hemisphere include:

- Gerstmann's syndrome. This consists of confusion of the right and left sides of the body, inability to distinguish the fingers of the hands (finger agnosia), and impairment of calculation (dyscalculia) and writing (dysgraphia). Difficulty with reading (dyslexia) may also occur; this is a function of the dominant parieto-occipital cortex.
- Bilateral ideomotor and ideational apraxia. This is the inability to carry out a task on request or by imitation, with normal comprehension and without disturbance of motor or sensory function.

Syndromes of the non-dominant hemisphere

Syndromes of the non-dominant hemisphere include:

- Constructional apraxia (visuospatial dysfunction). There is difficulty in drawing simple objects (e.g. a house) and with construction (e.g. using building blocks).
- Dressing apraxia. There is difficulty with putting on clothes.
- Contralateral sensory inattention. There is neglect of the opposite of the body; this may be motor, sensory, or visual, e.g. a hemiplegic patient may ignore the paralysed side or there may be denial of the hemiplegia. Sensory and visual neglect are discussed in Chapter 14.
- Visual disturbances. If the deeper fibres of the parietal lobe are involved, a contralateral homonymous inferior quadrantanopia and ipsilateral loss of optikokinetic nystagmus may arise. Smooth pursuit eye movements may become 'broken'.

Focal seizures of the parietal cortex manifest as sensory symptoms of the contralateral side of the body. Descriptions of various sensations may be given (e.g. 'pins and needles', tingling) and the symptoms often 'march' from the lips and extremities to adjacent areas of the body.

THE TEMPORAL LOBE

Function

- Wernicke's area (dominant hemisphere). This area is concerned with comprehension of written and spoken language.
- The auditory and vestibular cortex. The primary auditory cortex receives fibres arranged in order of frequency of tone. The auditory pathways from each ear project to both auditory cortices. The dominant temporal lobe is important for the comprehension of spoken words, and the non-dominant for the appreciation of sounds and music. Vestibular fibres terminate just posterior to the auditory cortex.
- The limbic lobe. The olfactory and gustatory cortices lie in the medial temporal lobe. The limbic system is important in memory, learning, and emotion.
- Visual pathways. The fibres of the lower part of the optic radiation (subserving the upper quadrant of the contralateral visual field) pass deep through the white matter of the temporal lobe.

The blood supply to the temporal lobe is from the posterior cerebral (medial part of the lobe) and middle cerebral (lateral part) arteries.

Lesions of the temporal lobe

Lesions of the temporal lobe give rise to:

- Cortical deafness. This will only occur with bilateral lesions of the primary auditory cortices. The patient may be unaware of the deficit. Auditory hallucinations may occur in temporal lobe epilepsy.

- Auditory agnosia. This is the inability to recognize sounds, e.g. ringing of a bell, whistling of a kettle, a melody. It occurs in lesions of the non-dominant hemisphere.
- Wernicke's receptive dysphasia (temporo-parietal region)—see p. 5.

Note that with auditory agnosia and Wernicke's receptive dysphasia the function of hearing is normal.

- Vestibular dysfunction. Vestibular dysfunction from a lesion of the vestibular cortex is uncommon, but vertigo may occur as part of the aura of temporal lobe seizures.
- Olfactory and gustatory hallucinations. Olfactory hallucinations and, less commonly, gustatory hallucinations may arise from lesions within the medial temporal lobe, particularly during seizures.
- Learning difficulties. Learning difficulties with auditory information occur in dominant hemisphere lesions; learning difficulties with visual information occur in non-dominant hemisphere lesions.
- Memory impairment. This occurs with lesions of the medial temporal lobe involving the hippocampus and parahippocampal gyrus. Bilateral damage results in marked impairment of retention of new information.
- Emotional disturbances. Emotional disturbances from damage to the limbic system may include aggression and rage, apathy, and hypersexuality.
- Visual disturbances. A lesion involving the deeper fibres within the temporal lobe will cause a contralateral superior homonymous quadrantanopia. Complex visual hallucinations can occur in temporal lobe seizures.

Temporal lobe seizures begin with a prodrome of auditory, olfactory, gustatory, or visual hallucinations, a sensation of anxiety or fear, and often a rising epigastric sensation. There may be disturbances of memory, with feelings of familiarity (déjà vu) or unfamiliarity (jamais vu). Behavioural changes may occur; aggression and hypersexuality are reported but are uncommon.

Dysphasia

Dysphasia is disorder of spoken and written language; it occurs with damage of the frontal, parietal, or temporal cortices. Broca's expressive and Wernicke's receptive dysphasias occur with damage of the dominant hemisphere and have been discussed (see p. 3 and p. 5). The cortical areas subserving these functions are linked by the arcuate fasciculus, which runs in the subcortical white matter. This enables the comprehension of language with subsequent production of speech in response.

- Conduction dysphasia occurs with damage to the arcuate fasciculus. The speech is fluent but 'jargon', with paraphrasia and neologisms as in Wernicke's dysphasia. However, comprehension of language is intact, the patient is aware of the problem, and repetition is markedly impaired.
- Global dysphasia occurs with lesions of both Broca's and Wernicke's areas. There is a combination of non-fluent speech and impaired comprehension of language.
- Nominal dysphasia is an inability to name objects and arises from a lesion of the dominant parieto-temporal cortex. It may occur during recovery from the aforementioned dysphasias.

THE OCCIPITAL LOBE

Function

The function of the occipital cortex (Fig. 1.3) is the perception of vision and recognition of whatever is visualized. The blood supply is from the posterior cerebral artery, but the occipital poles have additional supply from a branch of the middle cerebral artery.

Lesions of the occipital lobe

Lesions of the occipital lobe give rise to:

- Contralateral homonymous hemianopic field defect. If this arises from a lesion of the posterior cerebral artery, there will be sparing of the macular area. A lesion of the occipital pole will affect the macular fibres only and result in a contralateral homonymous hemianopic macular field defect.
- Cortical blindness. Bilateral occipital lesions render the patient blind, with retention of the pupillary reflexes. The patient may deny the blindness (Anton's syndrome).
- Visual agnosia. Lesions of the visual association cortices cause impaiment of recognition of faces and objects.
- Visual illusions. Objects may appear larger (macropsia) or smaller (micropsia); there may be disturbances of shape, colour, and number. This is more common with lesions of the non-dominant hemisphere.

Visual hallucinations in seizures of the primary visual cortex are unformed (flashes of light and geometric shapes); those due to seizure activity from the visual association cortex or its connections with the temporal cortex, are formed (objects, people).

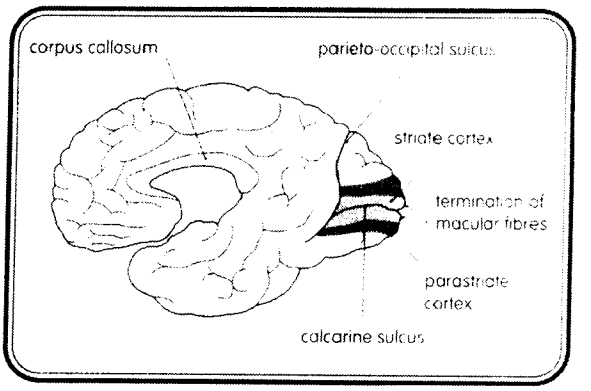

Fig. 1.3 The occipital lobe. The striate (primary visual) and parastriate (visual association) cortices are shown, and termination of the macular fibres at the poles.

SUMMARY

Focal damage to the cerebral hemispheres usually results from vascular events (infarction or haemorrhage), tumours, trauma, or localized inflammatory lesions (e.g. abscess, tuberculoma). Generalized or multifocal cerebral dysfunction results most often from degenerative dementias (Alzeimer's disease, Pick's disease), multiple infarcts, demyelination, or diffuse infections (encephalitis, meningitis).

Fig. 1.4. summarizes the symptoms that may arise from focal lesions of the cerebral hemisphere.

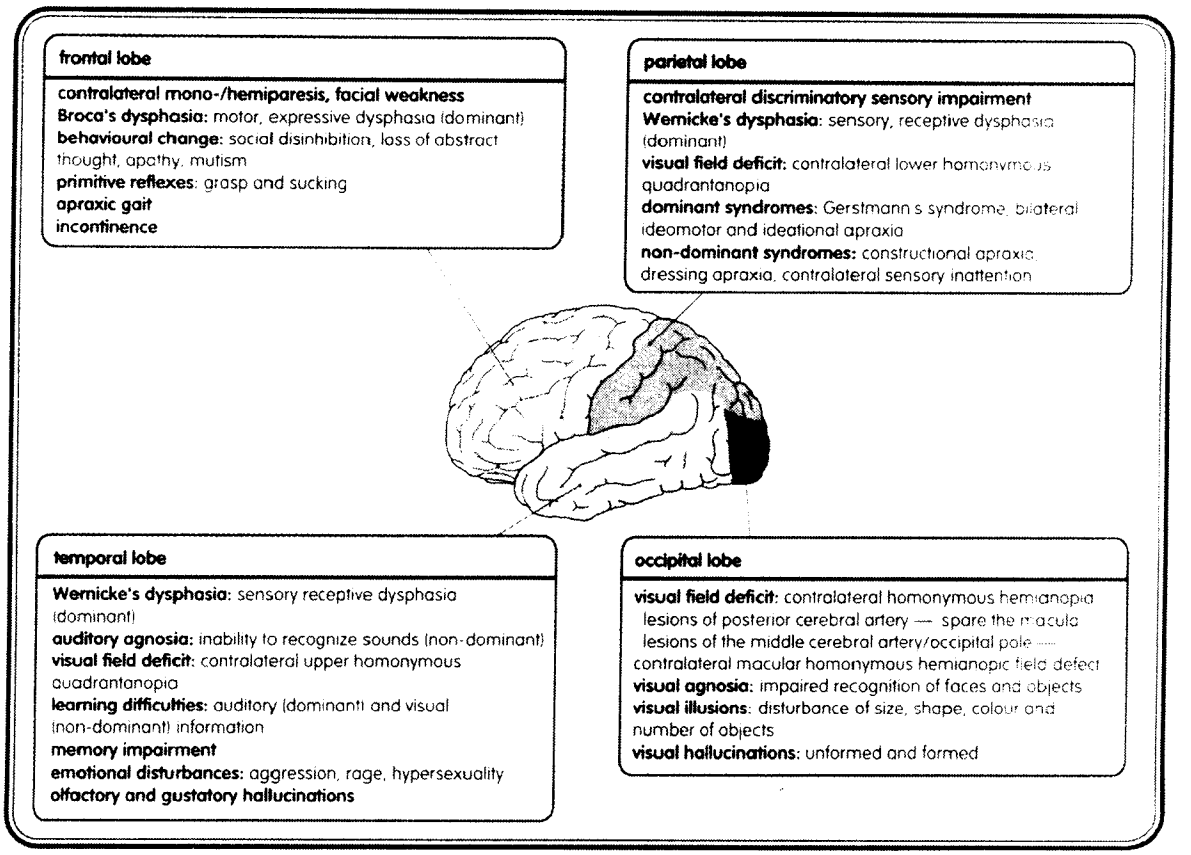

Fig. 1.4 Summary of localization of symptoms arising from focal lesions of the cerebral hemispheres.