LECTURE NOTES

Neurology

LIONEL GINSBERG

9th edition

WILEY-
BLACKWELL

Lecture Notes: Neurology

To my children
Amelia, Toby and Connie
and in memory of their mother
Andrea Marguerite Cobon

Lecture Notes

Neurology

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Contents

Preface to the ninth edition

The seventh edition of *Lecture Notes on Neurology* involved a complete revision of the text, an opportunity afforded by the change of author. With the eighth and ninth editions, there have been further refinements as follows:

- Selected case histories, drawn from life, are given at the end of each chapter in Part 2 of the book, so that important clinical points made in the main text can be illustrated and expanded.
- A series of sample examination questions and answers is given at the end of the volume. Desmond Kidd, Consultant Neurologist, and Thomas Solbach, Consultant Neuroradiologist, kindly provided figures to accompany some of these questions.
- Some of the figures in the main text have been replaced and a few new ones added.
- The text has been fully updated to reflect the continuing rapid advances in neurological management.

Tony Wilson and Charlie Davie, Consultant Neurologists, kindly read and commented on the case histories and examination questions, respectively. Susan Huson, Consultant Clinical Geneticist, commented on Table 18.3. Editorial staff at Wiley-Blackwell, notably Laura Murphy and Karen Moore, were patient and tolerant as always. Final thanks must again go to Sue, my wife, for her encouragement and support.

Lionel Ginsberg

Preface to the seventh edition

More than a decade has passed since the sixth edition of Dr Ivan Draper's *Lecture Notes on Neurology*. This seventh edition has been prepared with the dual aims of reflecting advances in neurology in the intervening period and changes in the undergraduate medical curriculum.

There have been dramatic developments in neurological practice in recent years, paralleling achievements in basic neuroscience research. These include new imaging techniques, which have greatly refined diagnostic accuracy and spared patients the discomfort of previous investigative approaches. Novel therapies are beginning to appear for conditions once considered untreatable. Molecular genetic research has cast new light on disease pathogenesis and should ultimately pay dividends in the treatment, as well as diagnosis, of inherited disorders.

Despite these advances, neurology remains *par excellence* a clinical discipline. Contrary to popular opinion outside the specialty, it is not a sterile and obscure diagnostic exercise. Neurological disorders are common, permeating the whole of general medicine and surgery. Their diagnosis is based on accurate history-taking and physical examination, coupled with the application of logical rules derived from knowledge of the underlying anatomy, physiology and pathology.

This volume is intended to emphasize these general principles. In line with the concept of an undergraduate 'core curriculum', it also concentrates on common diseases. The book falls naturally into two parts. The first section, 'The Neurological Approach', is concerned with history-taking and examination, where possible linked to relevant anatomy and physiology. A final chapter in this part outlines the expanding range of neurological investigations. The second section, 'Neurological Disorders', is a systematic account of the common conditions, rarities being relegated to the tables, or to a brief thumbnail sketch. There are also chapters on neurological emergencies, neurorehabilitation and the interface between neurology and other specialties. The separation of general from systematic is incomplete. For convenience, some disorders are discussed in the first part of the book and some principles appear for the first time in the second. The author also apologizes for occasions where his enthusiasm has allowed discussion of a few topics that might be considered beyond the range of a 'core curriculum'. Their inclusion may, it is hoped, be justified on grounds of continuity and interest, particularly where they reflect growing areas of neurological research and practice.

This new edition of *Lecture Notes on Neurology* should function as a portable companion during a student's neurology clinical attachment, and also senior medical clerkships. It may serve as a revision aid, and contains a grounding for early postgraduate work in general medicine.

Lionel Ginsberg

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This work would not have been completed without the secretarial assistance of Barbara Parker, who uncomplainingly coped with numerous drafts and corrections. Many colleagues read selected chapters. I am grateful to Bob Bradford, Consultant Neurosurgeon, Heather Angus-Leppan, Jeremy Gibbs, John Hodges, Gareth Llewelyn, Gordon Plant, Tony Schapira, Neil Scolding, Tom Warner and Tony Wilson, Consultant Neurologists, for their comments. The responsibility for any remaining errors is, of course, my own. Several student reviewers at Blackwell Publishing also made helpful suggestions, which have been incorporated wherever possible. The editorial staff at Blackwell Publishing, particularly Mike Stein and Andrew Robinson, have been embodiments of patience and tolerance.

Most of the radiological figures were kindly provided by Alan Valentine, Consultant Neuroradiologist. Figure 10.1 was from the Department of Clinical Neurophysiology at the Royal Free Hospital. Pathological plates were prepared by Jim McLaughlin, Consultant Neuropathologist. The Department of Medical Illustration at the Royal Free Hospital helped provide most of the clinical photographs. Several other figures were prepared by Kieran Price. Finally, I thank my wife, Sue, who encouraged and supported me, and drafted most of the line drawings.

Lionel Ginsberg

Part 1

The Neurological Approach

Chapter 1

Neurological history-taking

The diagnosis and management of diseases of the nervous system have been revolutionized in recent years by new techniques of investigation and new treatments. But neurology continues to rely as much as any other branch of medicine on the fundamental clinical skills of history-taking and physical examination.

Neurological diagnosis

The neurological diagnosis is generally separable into two parts:

- Anatomical: What is the site of the lesion in the nervous system?
- Pathological: What disease process has occurred at that site?

This division is helpful as it can reduce possible confusion caused by the many available sites for neurological disorder (Table 1.1).

The history is of paramount importance in determining both the anatomical and pathological diagnoses. Indeed, many neurological patients have no abnormal signs, or simply have physical features that confirm clinical suspicions based on the history.

Sometimes, however, particularly with complex problems, the history can only yield a 'shortlist' of potential sites of the lesion(s) and final localization must await the formal examination. This is because disease at one site in the nervous system may produce symptoms mimicking a lesion at another.

Table 1.1 Potential sites of neurological disease.

Cerebral cortex
Cerebral white matter
Basal ganglia
Cerebellum
Brainstem
Cranial nerves
Spinal cord
Spinal roots
Peripheral nerves
Neuromuscular junction
Muscle

History of presenting complaint

How can the history best be taken to provide the maximum diagnostic information? An important rule is first to allow the patient sufficient uninterrupted time to speak. Most patients can give a reasonable account of their symptoms within 2 or 3 minutes and time spent listening at this stage is not wasted.

The nature of the main complaint and its duration will usually have been established in this early part of the interview, along with three further essential pieces of information about the patient:

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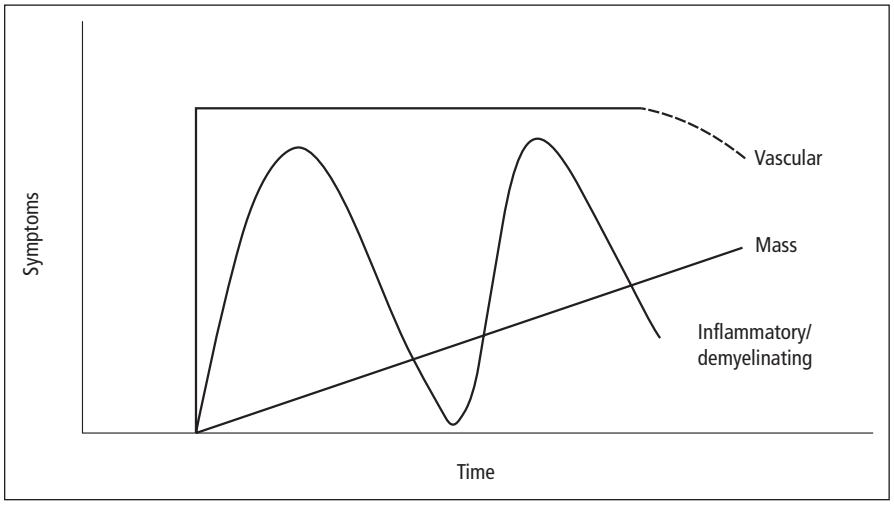

Figure 1.1 Temporal patterns associated with specific neuropathological causes. Using the example of a cerebral hemisphere lesion presenting with contralateral weakness, a rapid onset (seconds, minutes or at most hours) and static subsequent course, ultimately possibly with some improvement, suggest a vascular event (**stroke**), i.e. haemorrhage or infarction. A slowly progressive course (days, weeks or months) is more indicative of a **mass** lesion, i.e. a tumour. A relapsing and remitting pattern (with symptoms typically developing and resolving over days or weeks, then perhaps recurring with a similar time course) generally implies a **chronic inflammatory** or **demyelinating** process, of which multiple sclerosis is the prime example in the central nervous system.

- Age
  - Certain neurological disorders are associated with specific age groups.
- Occupation
  - A patient may have experienced occupational exposure to a toxin or other potential causative agent of disease.
  - Some neurological symptoms may limit the patient's ability to perform certain occupations.
- Handedness
  - To obtain information about cerebral hemisphere dominance.
  - To establish the extent to which a patient is disabled if the presenting complaint concerns the upper limbs.

Having heard the patient's description of the symptoms, it is usually necessary to probe the history of the presenting complaint in specific areas.

Timing of symptoms

Determining the temporal features of a patient's symptoms is essential to reach a pathological diagnosis:

- onset,
- progression,
- duration,
- recovery,
- frequency.

For example, a patient may present with weakness of one side of the body, suggesting a lesion in the contralateral cerebral hemisphere. Detailed further questioning on the timing of the symptoms may clarify the pathological nature of this lesion (Fig. 1.1).

'Discriminant' questions

If the initial history only partially solves the anatomical diagnosis, the 'shortlist' of potential sites may be reduced by asking the patient direct questions (Table 1.2).

For example, a patient presenting with numbness in both hands and both feet is likely to have a diffuse disorder of all the peripheral sensory nerves of the extremities (**sensory polyneuropathy**). But a similar '**glove-and-stocking**' sensory loss may occasionally be produced by

Table 1.2 Neurological systematic review questions: Has the patient suffered any of the following?

Pain
Headache
Facial, neck, back or limb pain
Disturbance of consciousness
Blackouts, faints, fits*
Altered sleep pattern
Cognitive and affective dysfunction
Memory, language
Depression, irritability
Cranial nerve symptoms
Loss of vision, blurred or double vision*
Hearing, sense of taste and smell
Vertigo, dizziness, giddiness*
'Bulbar' problems (swallowing, articulation of speech)
Limb symptoms
Difficulty in lifting, gripping, fine finger movements; clumsiness
Gait disorder, leg weakness or stiffness, balance problems
Loss of sensation, altered sensation, numbness*
Involuntary movements, incoordination
Sphincter disturbance
Bladder, bowel, sexual dysfunction

*If the patient uses terms like blackouts, fainting, dizziness, giddiness, double vision or numbness, it is worthwhile establishing their exact meaning, as the standard medical usage of the term may not correspond to the patient's intended meaning.

a **cervical spinal cord lesion**, mimicking a polyneuropathy.

In this instance, selecting questions from Table 1.2 likely to *discriminate* between these two anatomical diagnoses, a history of neck pain or injury will strongly favour the diagnosis of cervical cord lesion, as will the presence of sphincter dysfunction. Bladder disturbance is an early feature of spinal cord disease but only occurs in patients with a sensory polyneuropathy if there is a coexistent **autonomic** neuropathy.

Upper limit of symptoms

A useful further refinement in neurological history-taking is to check the '**upper anatomical limit**' of the symptoms. Thus, in a patient presenting with weakness of one leg the anatomical diagnostic range is wide. But specifically asking whether there are equivalent symptoms in the ipsilateral arm immediately narrows this range, the patient then being far more likely to have a **hemiparesis** caused by a lesion on the opposite side of the brain than anything else.

Negative and positive symptoms

A valid distinction may be made between 'negative' and 'positive' neurological symptoms.

Negative symptoms, or loss of particular functions, signify destructive lesions of the nervous system. Thus, a vascular event in one cerebral hemisphere will generally lead to loss of function as indicated, for example, by paralysis of the opposite side of the body.

Conversely, **positive symptoms** are those that suggest an irritative lesion, i.e. an area of abnormal excessive electrical activity in the nervous system. An irritative lesion in one cerebral hemisphere may produce repetitive involuntary (**clonic**) movements of groups of muscles on the opposite side of the body (**partial epilepsy**) rather than paralysis.

Remainder of the history

In neurology, as in other branches of medicine, valuable information, particularly about the pathological diagnosis, can be obtained by asking directly about:

- previous medical history,
- family history,
- social history,
- therapeutic history.

Considering again the patient who presents with glove-and-stocking sensory loss caused by a sensory polyneuropathy:

- **Previous medical history**: A history of diabetes mellitus would be especially relevant, this being a common cause of a sensory polyneuropathy.
- **Family history**: Some causes of a polyneuropathy are inherited.

- **Social history**: Excessive alcohol intake may lead to a sensory polyneuropathy, as may accompanying vitamin deficiencies.
- **Therapeutic history**: Many drugs may cause a polyneuropathy.

Witnesses

Many neurological patients are unable to give a complete account of their symptoms, and information must be sought from family members and other witnesses.

A witness account is especially valuable for patients reporting transient alterations in their state of consciousness. By their very nature, such attacks may prevent the patient from recalling the details of all that occurred. In the acute setting of an unconscious patient in the hospital casualty department, obtaining a history from anyone accompanying the patient is essential.

The need for witnesses also applies to those presenting with progressive **cognitive** impairment in adult life (**dementia**). Indeed, corroboration of such symptoms by a close family member lends weight to this diagnosis. A patient who reports problems of memory and intellect unnoticed by family or colleagues at work may be experiencing the consequences of anxiety or depression ('**pseudodementia**') rather than 'organic' dementia, i.e. associated with a recognized macroscopic or microscopic change in brain structure.

History and examination

In neurology, separating the history and examination is artificial in practice, in the sense that the examination really begins before and during the formal history-taking. Much may be learnt from initial impressions of a patient's:

- gait,
- facial expression,
- handshake,
- speech.

The neurological examination must also be performed in the context of the general physical examination. This applies particularly to the cardiovascular and musculoskeletal systems. The following features are important in assessing vascular disease of the nervous system:

- pulse – rate and rhythm,
- blood pressure,
- murmurs and bruits – cardiac, carotid, cranial or spinal.

In the musculoskeletal system, it is important to examine for skull, spine and joint deformity.

The various components of the neurological examination should be 'screened' in each patient:

- level of consciousness,
- cognitive function,
- speech,
- cranial nerves,
- neck and trunk,
- limbs – motor and sensory examination,
- gait.

The detail required for each part will be dictated by the history. Thus, in many standard outpatient consultations, level of consciousness and cognitive function are screened merely by assessing the patient's ability to give a coherent history. However, in the emergency setting of an unconscious patient in the casualty department, or a confused patient on a general medical ward, these aspects require much more detailed assessment. The remaining chapters of this section outline these parts of the neurological examination in the context of relevant anatomy and physiology.

Key points

- Neurological diagnosis is best divided into two steps: site of lesion (anatomical diagnosis) and disease process (pathological diagnosis).
- The time course of a patient's symptoms provides clues to the pathological diagnosis.
- Neurological symptoms may be negative (loss of function) or positive.
- History from witnesses is essential for patients presenting with disturbances of consciousness, or with cognitive impairment.
- A full neurological examination is time-consuming and potentially exhausting for patient and doctor; selection of the components requiring detailed assessment is determined by the history.

Chapter 2

Consciousness

Consciousness is an individual's awareness of self and surroundings. This definition is narrow and incomplete but useful in the clinical context of acute disturbances of consciousness. Pathophysiologically, normal consciousness depends on the sensory input into the brain and the intrinsic activity of the reticular activating system, the ascending reticular formation in the brainstem and its rostral connections, which maintain the cerebral cortex in an alert state.

In clinical practice, previous attempts to classify the severity of an alteration in level of consciousness, using imprecise terms such as stupor, semicoma, have been superseded by the **Glasgow Coma Scale** (Table 2.1).This more objective approach has become universally accepted as valuable in assessing a patient's initial condition and subsequent response to treatment and time.

Causes of altered level of consciousness

The normal function of the reticular activating system may be disturbed by focal structural lesions of the brain or by more diffuse processes:

- Structural
  - infratentorial (directly involving the brainstem) (e.g. trauma, infarction, haemorrhage, tumour, demyelination),
  - supratentorial (compressing the brainstem) (similar pathological causes, particularly affecting the right cerebral hemisphere);
- Diffuse
  - decreased availability of substances required for normal brain metabolism (hypoxia, hypoglycaemia),
  - other metabolic disorders (e.g. renal and liver failure, hypothermia, vitamin deficiencies),
  - epilepsy (interfering with the normal electrical activity of the brainstem),
  - inflammation of the brain or its coverings (encephalitis, meningitis),
  - drugs and toxins (opiates, antidepressants, hypnotics, alcohol).

Management of the unconscious patient

The emergency management of the unconscious patient consists first of protecting respiratory and circulatory function with standard life support techniques.

Airway – remove any obstruction, use oropharyngeal airway or endotracheal tube if necessary.

Breathing – give oxygen, ventilate if respiratory movements are inadequate.

Circulation – check pulse and blood pressure, gain intravenous access and replace any blood loss.

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Table 2.1 Glasgow Coma Scale.

	Score
Eye opening	
Spontaneously	4
To speech	3
To pain	2
None	1
Best verbal response	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Best motor response	
Obeying commands	6
Localizing pain	5
Withdraws (normal flexion)	4
Flexes abnormally (spastic flexion)	3
Extending to pain	2
None	1
TOTAL	3–15

Any immediately reversible cause should be treated:

- Is the patient hypoglycaemic?
  - Administer 50 mL 50% intravenous dextrose.
- Is there evidence of drug overdosage?
  - Administer appropriate antidote: naloxone for opiates; flumazenil for benzodiazepines.

Other treatable causes should be identified from any history available from witnesses or relatives and from the physical examination (looking particularly for evidence of injury, infection, epilepsy (Chapter 10) and raised intracranial pressure (Chapter 13)). After these emergency measures are completed, detailed neurological examination of the eyes (Chapter 4) and limbs may help localize the site of brain damage. The presence of focal neurological signs is more in keeping with structural rather than diffuse metabolic or toxic causes.

Brain death and its differential diagnosis

In some patients, irreversible brain damage may have occurred with permanent destruction of brainstem function and hence death of the patient, yet cardiovascular function may remain stable and respiration be maintained by artificial ventilatory support. In these circumstances, formal criteria of **brainstem death** are used to decide whether cardiorespiratory support should be withdrawn (Table 2.2). This has become an important ethical and practical concern, particularly with the advent of transplantation surgery.

An even more difficult ethical situation arises when patients have preserved brainstem function yet widespread severe brain damage. In one such circumstance, the **vegetative state**, individuals are unaware of self and environment yet able to breathe spontaneously, with a stable circulation and cycles of eye closure and opening resembling sleep and waking. This state may be permanent.

Equally distressing for relatives and carers, and infinitely more so for the patient, is the converse situation where the function of the reticular activating system is preserved despite extensive brainstem damage. The patient is alert but paralysed, able to communicate only by means of blinking and vertical eye movements (**locked-in syndrome**).

Transient disturbance of consciousness

Patients with transient episodes of altered consciousness constitute a common diagnostic problem in neurological outpatient practice. The main differential diagnosis is between **epilepsy** (Chapter 10) and **syncope**.

Syncope is loss of consciousness caused by a transient reduction in blood flow to the brain for which there are many causes:

- cardiac arrhythmias,
- prolonged standing, especially in warm surroundings,
- psychogenic factors, e.g. simple faint in squeamish individuals exposed to needles and other medical procedures,
- other causes of excessive reflex vagal stimulation, e.g. **micturition syncope**, **cough syncope**.

Table 2.2 Criteria for brainstem death.

Preconditions
Central nervous system depressant drugs must not be contributing to the clinical state
The patient must be on a ventilator due to inadequate spontaneous respiration; effects of neuromuscular blocking agents must be excluded
Hypothermia and severe metabolic disorders must not be possible primary causes of the patient's condition
The cause of the patient's condition must be established and must be compatible with irreversible brain damage
Tests
No pupillary response to light
Absent corneal reflexes (Chapter 4)
Absent vestibulo-ocular reflex (see Fig. 4.7)
No gag or response to tracheal suction
No motor response in cranial nerve territory to painful stimulus, e.g. supraorbital pressure
No respiratory movements when patient is disconnected from ventilator (Pao_2 is maintained by passing O_2 at 6 L/min down endotracheal tube; $Paco_2$ should be allowed to rise above 6.65 kPa, 50 mmHg)
Notes
Tests should be carried out by two doctors, both with appropriate expertise and one, preferably both, of consultant status
The tests should be repeated at an interval; death is certified at the time of the second set of tests, assuming no evidence of brainstem function is detected
The EEG (Chapter 8) is of no value in diagnosing brain death

Typically, patients may have a warning before losing consciousness and falling, with lightheadedness, nausea, blurred or tunnel vision, pallor and sweating. Once the patient is in a supine position, with the head at the same level as the heart, recovery is usually rapid (1–2 minutes or less) provided there is no continuing cardiac dysrhythmia. If falling is impeded, brain hypoxia may be prolonged and convulsive movements may occur.

Other important differential diagnoses for epilepsy and syncope are:

- Hypoglycaemia
  - Warning symptoms include anxiety, tremor, unsteadiness, sweating and hunger. Loss of consciousness may be prolonged (1 hour or more) and convulsions may occur.
- Drop attacks
  - In middle-aged and elderly women, falls without warning and without clear-cut loss of consciousness. Though usually of no sinister significance, injuries may occur because of the lack of warning.
- Psychogenic attacks
  - Either in stressful situations or as attention-seeking behaviour. These attacks may be associated with **hyperventilation** with tingling in the extremities, and may sometimes be reproduced by voluntary hyperventilation.

Sleep disorders

Sleep is a normal state of altered consciousness dependent on the intrinsic rhythmicity of the reticular activating system (sleep–wake cycle). In contrast to pathological unconsciousness, a sleeping person is easily roused. Certain disorders are characterized by excessive daytime sleep, as follows.

Narcolepsy

This rare disorder consists of a tetrad of clinical features:

- **Daytime sleep attacks** (narcolepsy), typically lasting 10–20 minutes, from which the patient awakes refreshed. These episodes are irresistible and may occur under inappropriate circumstances, e.g. during conversations, meals, driving.

- **Cataplexy**: episodes of loss of postural control and limb weakness with preserved consciousness, often provoked by emotional events, e.g. laughter.
- **Sleep paralysis**: inability to move while falling asleep or waking.
- **Hypnagogic hallucinations**: frightening visual hallucinations on falling asleep.

The cause of the disease is poorly understood, though there may be a genetic basis and an association with the HLA (human leukocyte antigen; major histocompatibility) complex. Narcolepsy may be treated with amphetamines, but in view of the addictive properties of these drugs, care must be taken to reach the correct diagnosis. An alternative newer drug is modafinil. Clomipramine relieves cataplexy but has no effect on narcoleptic attacks. Other antidepressants are also effective against cataplexy.

Obstructive sleep apnoea

An under-recognized cause of excessive daytime somnolence arises in patients with partial upper airways obstruction, where further narrowing or collapse during sleep results in nocturnal apnoeic attacks. These patients generally have a disrupted night's sleep with heavy snoring. They wake unrefreshed and are sleepy during the day, but daytime sleep episodes are also not refreshing. Features of the narcolepsy complex are absent. There may be a history of otolaryngological disease and sometimes an association with obesity and excess alcohol intake. The diagnosis may be reached by recording upper airways obstruction and apnoeic episodes despite continuing respiratory effort using overnight **pulse oximetry** at home or more detailed studies in a sleep laboratory. Detailed sleep studies may be required to exclude the rarer syndrome of **central sleep apnoea**, where apnoeic episodes occur without continuing respiratory effort. Patients with obstructive sleep apnoea may be successfully treated using a device at home to maintain nasal **continuous positive airways pressure** (CPAP) at night and hence prevent collapse of soft tissues which may obstruct the upper airways during sleep. Some patients require surgery to remove excess soft tissue from the upper airways, e.g. nasal polyps, deviated nasal septum, tonsillar hypertrophy.

Key points

- The Glasgow Coma Scale provides an objective measure of a patient's level of consciousness
- Consciousness may be altered as a result of focal structural lesions of the brain, or by more diffuse processes (metabolic, inflammatory, epileptic or toxic)
- The emergency management of an unconscious patient first entails attention to airway, breathing and circulation
- Brainstem death is diagnosed using strict clinical criteria
- The main differential diagnosis in patients presenting with transient disturbances of consciousness lies between epilepsy and syncope

Chapter 3

Cognitive function

Higher brain function may be subclassified into:

- **Distributed functions**, which do not localize to a particular brain region but instead require the concerted action of multiple parts on both sides of the brain, e.g.
  - attention and concentration,
  - memory,
  - higher-order executive function,
  - social conduct and personality;
- **Localized functions**, which are dependent on the normal structure and function of a particular part of one cerebral hemisphere (Fig. 3.1).

Distributed cognitive function

Attention and concentration

Anatomy

The maintenance of normal attention is dependent on the same anatomical basis as that of consciousness, i.e. the reticular activating system which projects to the thalamus and then to the cerebral cortex diffusely.

Examination

Clinical tests of attention and concentration include:

- **Orientation** in time and place – Can the patient state the time of day, day of the week, the correct month and year, and the name of the building where they are?
- **Digit span** – ability to repeat a list of digits forwards and backwards.
- '**Serial sevens**' – ability to subtract seven repeatedly starting from 100, or, failing this, to count backwards from 20 or recite the months of the year backwards.

Clinical aspects

The syndrome most associated with impaired attention and concentration is the **acute confusional state**, nowadays usually called **delirium**, or sometimes **acute organic brain syndrome**, a very common management problem in general medicine, particularly in the elderly. Other features of this state include:

- muddled thinking and hence speech,
- visual hallucinations,
- disturbed sleep–wake cycle, the patient often being awake and indeed more confused at night,
- memory impairment – with an inability to register new material,
- mood changes.

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