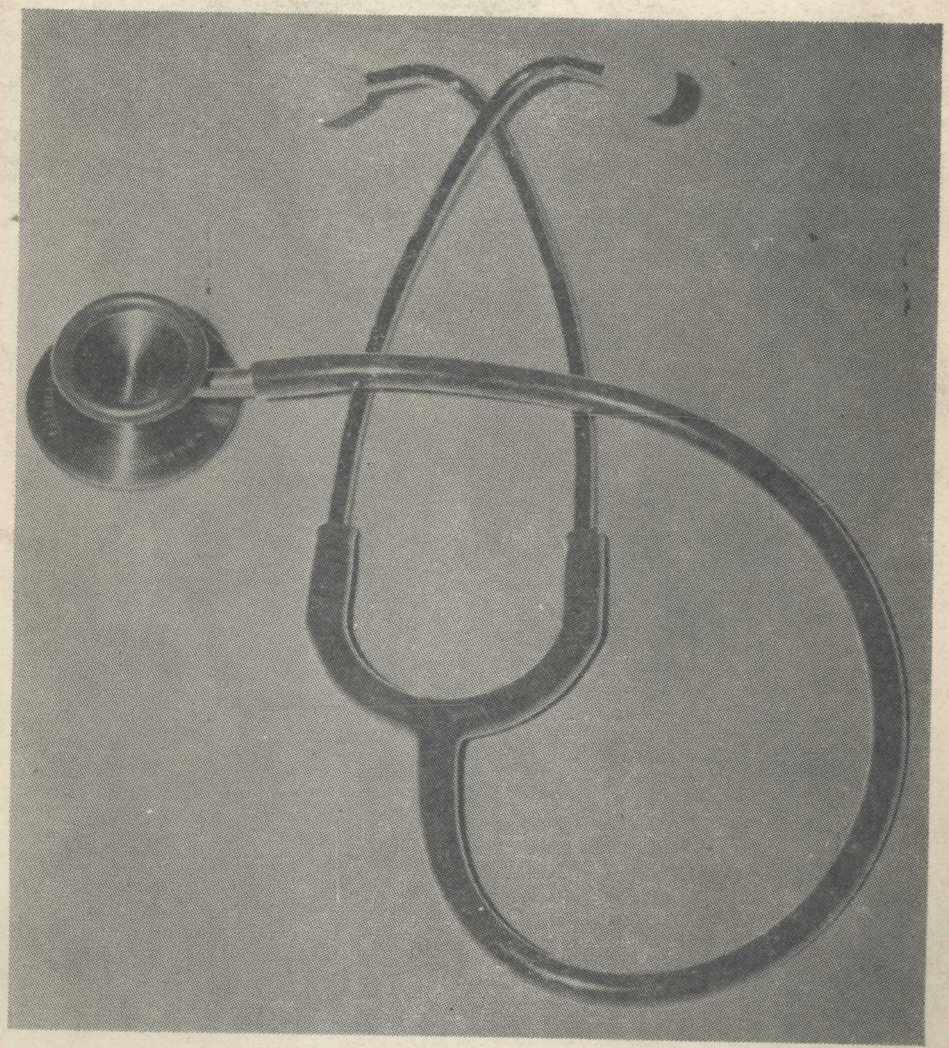
David Rubenstein
David Wayne

Lecture Notes on Clinical Medicine

Second Edition



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Contents

Preface to the second edition

In this new edition, the text has been extensively revised and much new material added. In particular we have added brief notes on diseases of travellers and on the management of drug overdosage. The section on haematology has been enlarged and a new chapter placed in Part 2. We have tried to ensure that this edition is both accurate and up to date—please tell us if you disagree.

Acknowledgements

We are indebted to the many readers who have written to us, including those who have suggested improvements or pointed out inaccuracies. Once again, Sir Edward Wayne has read the entire text and made many useful suggestions. We are grateful to the following friends and colleagues who have read and commented on our chapters in their fields of interest:
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Some of the ECGs first appeared in Schamroth's *An Introduction to Electrocardiography* (1977, 5th edn. Blackwell Scientific Publications, Oxford) and Table 15 appeared in *Medicine* published by Medical Education International Ltd.

David Rubenstein
David Wayne
January 1980

Preface to the first edition

This book is intended primarily for the junior hospital doctor in the period between qualification and the examination for Membership of the Royal Colleges of Physicians. We think that it will also be helpful to final year medical students and to clinicians reading for higher specialist qualifications in surgery and anaesthetics.

The hospital doctor must not only acquire a large amount of factual information but also use it effectively in the clinical situation. The experienced physician has acquired some clinical perspective through practice: we hope that this book imparts some of this to the relatively inexperienced. The format and contents are designed for the examination candidate but the same approach to problems should help the hospital doctor in his everyday work.

The book as a whole is not suitable as a first reader for the undergraduate because it assumes much basic knowledge and considerable detailed information has had to be omitted. It is not intended to be a complete textbook of medicine and the information it contains must be supplemented by further reading. The contents are intended only as lecture notes and the margins of the pages are intentionally large so that the reader may easily add additional material of his own.

The book is divided into two parts: *the clinical approach* and *essential background information*. In the first part we have considered the situation which a candidate meets in the clinical part of an examination or a physician in the clinic. This part of the book thus resembles a manual on techniques of physical examination, though it is more specifically intended to help the candidate carry out an examiner's request to perform a specific examination. It has been our experience in listening to candidates' performances in examinations and hearing the examiner's subsequent assessment, that it is the failure of a candidate to examine cases systematically and his failure to behave as if he were used to doing this every day of his clinical life that leads to adverse comments.

In the second part of the book a summary of basic clinical facts is given in the conventional way. We have included most common diseases but not all, and we have tried to emphasise points which are understressed in many textbooks. Accounts are given of many conditions which are relatively rare. It is necessary for the clinician to know about these and to be on the lookout for them both in the clinic and in examinations. Supplementary reading is essential to understand their basic pathology but the information we give is prob-

ably all that need be remembered by the non-specialist reader and will provide adequate working knowledge in a clinical situation. It should not be forgotten that some rare diseases are of great importance in practice because they are treatable or preventable, e.g. infective endocarditis, hepatolenticular degeneration, attacks of acute porphyria. Some conditions are important to examination candidates because patients are ambulant and appear commonly in examinations, e.g. neurosyphilis, syringomyelia, atrial and ventricular septal defects.

We have not attempted to cover the whole of medicine but by cross-referencing between the two sections of the book and giving information in summary form we have completely omitted few subjects. Some highly specialised fields such as the treatment of leukaemia were thought unsuitable for inclusion.

A short account of psychiatry is given in the section on neurology since many patients with mental illness attend general clinics and it is hoped that readers may be warned of gaps in their knowledge of this important field. The section on dermatology is incomplete but should serve for quick revision of common skin disorders.

Wherever possible we have tried to indicate the relative frequency with which various conditions are likely to be seen in hospital practice in this country and have selected those clinical features which in our view are most commonly seen and where possible have listed them in order of importance. The frequency which which a disease is encountered by any individual physician will depend upon its prevalence in the district from which his cases are drawn and also on his known special interests. Nevertheless rare conditions are rarely seen; at least in the clinic. Examinations, however, are a 'special case'.

We have used many generally accepted abbreviations, e.g. ECG, ESR, and have included them in the index instead of supplying a glossary.

Despite our best efforts, some errors of fact may have been included. As with every book and authority, question and check everything—and please write to us if you wish.

We should like to thank all those who helped us with producing this book and in particular Sir Edward Wayne and Sir Graham Bull who have kindly allowed us to benefit from their extensive experience both in medicine and in examining for the Colleges of Physicians.

David Rubenstein
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November 1975

LECTURE NOTES ON

Clinical Medicine

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Part 1: The clinical approach

Nervous system

The candidate is usually asked to examine a specific area, e.g. *'Examine the cranial nerves'*, *'Examine the lower limbs'*, *'Examine the arms'* or *'Examine the eyes'*. By far the commonest neurological disorders suitable for a clinical examination are multiple sclerosis and the results of cerebrovascular disease. Diabetes is a common disorder which fairly frequently gives rise to neurological manifestations. Carcinomatous neuropathy should always be considered when the signs are difficult to synthesise. Neurosyphilis is becoming progressively rarer—the patient will belong to the pre-penicillin era, i.e. be over 50 years old. Parkinsonism is relatively common. Motor neurone disease, myopathies, myasthenia gravis and the neurological manifestations of vitamin B_{12} deficiency are all rare in practice but more frequently seen in examinations.

In terms of examination technique the practising physician must examine case after case, both normal and abnormal, until he has developed a system which is rapid, accurate and second nature to him. An appearance of professionalism in your neurological examination may encourage the examiner to take a less unfavourable view of minor errors than he might if you appear hesitant, clumsy or imprecise.

'Examine the cranial nerves'

Many abnormalities of the cranial nerves are the results of chronic disease and patients with them are commonly seen in examination.

The commonest disorders are disseminated sclerosis (optic atrophy, nystagmus (often ataxic), cerebellar dysarthria), stroke, and Bell's palsy. The manifestation of cerebral tumour, aneurysm, syphilis, dystrophia myotonica and myasthenia gravis are seen much less frequently. It is useful to memorise diagrams of cross-sections of the brain-stem and one of the floor of the fourth ventricle (pages 5 and 6) since these may greatly improve analysis of a cranial nerve lesion. Do not spend long on the first or second cranial nerves unless there is good reason to suspect an abnormality. If the optic fundus is abnormal, the examiner is likely to ask you to look at it specifically. Eye movements must be carefully examined. Do not confuse ptosis (third nerve or sympathetic) with paresis of the orbicularis oculi (seventh nerve). Make sure you can explain clearly and concisely the difference between an upper and a lower motor neurone lesion of the seventh nerve. The corneal reflex is an essential part of the complete examination of the cranial nerves. The following approach is recommended.

Smell	*'Has there been any recent change in your sense of smell?'* If so, test formally with 'smell bottles'.
Eyes	Observe, and test when necessary, for: — *ptosis* Third nerve lesion (complete or partial ptosis) Sympathetic lesion (partial ptosis) as part of Horner's syndrome Muscle weakness. Myasthenia gravis (and rarely, dystrophia myotonica, facio-scapulo-humeral dystrophy, congenital and tabo-paresis) NB Ptosis is not due to a seventh nerve lesion. — *visual fields* to confrontation (second nerve) (page 5) — *external ocular movements* (third, fourth and sixth nerves) (page 8) and *nystagmus* (page 11) — *the fundi* (second nerve) (page 27) — *visual acuity* either formally with Snellen's charts or quickly with literature from the bedside locker.
Face (seventh nerve)	*'Screw up your eyes very tightly'*. Compare how deeply the eyelashes are buried on the two sides. Unilateral weakness is invariably due to a lower motor neurone lesion. *'Grin'*. Compare the nasolabial grooves.
Mouth	*'Clench your teeth'* (fifth nerve, motor). Feel the masseters and test the jaw jerk if indicated. The jaw jerk is obtained by placing one finger horizontally across the front of the jaw and tapping the finger with a tendon hammer with the jaw relaxed and the mouth just open. An increased jaw jerk occurs in upper motor neurone lesions of the fifth cranial nerve (pseudobulbar palsy, page 13). *'Open your mouth and keep it open'* (fifth nerve, motor: pterygoids). You should not be able to force it closed. With a unilateral lesion, the jaw deviates towards the weaker side (the weak muscle cannot keep it open). *'Say aaah'* (ninth and tenth nerves). Normally the uvula and soft palate move upwards and remain central and the posterior pharyngeal wall moves little. With a unilateral lesion the soft palate is pulled away from the weaker side (there may also be 'curtain movement' of the posterior pharyngeal wall away from the weaker side). *'Put your tongue out'* (twelfth nerve). Look for wasting, fasciculation and whether it protrudes to one side (towards the weaker side since the weaker muscle cannot push it out).
Neck (eleventh nerve)	*'Lift your head off the pillows'* or *'Put your chin on your right (or left) shoulder'* while you resist the movement. Look at, and palpate the sternomastoids. *'Shrug your shoulders'* while you push them down. Look at and palpate the bulk of trapezius.
Ears (eighth nerve	Test hearing with a wrist watch held at various distances from the ears (compare with your own) and perform Weber and Rinne tests. You should ask for an

auriscope if indicated. The commonest cause of conductive (air conduction) deafness is wax.

Facial sensation (fifth nerve)

Test the three divisions on both sides with cotton wool. Check the corneal reflexes (often the first clinical deficit in fifth nerve lesions). Ask the patient if the sensation is equally unpleasant on the two sides.

Notes

It is frequently helpful to be able to draw cross-sections of the spinal cord and brain-stem when considering lesions in those areas (figures 1–3).

Field defects

You are in principle comparing the patient's visual fields with your own. When testing his right eye, he should look straight into your left eye with his head held at arm's length. *'Keep looking at my eye and tell me when you first see my finger out of the corner of your eye.'* Then bring your finger towards the centre of the field of vision from the four main directions (right, left, up, down). It is preferable to use a white-headed hat pin if you have one. The nasal and superior fields are limited by the nose and eyebrow respectively but this is

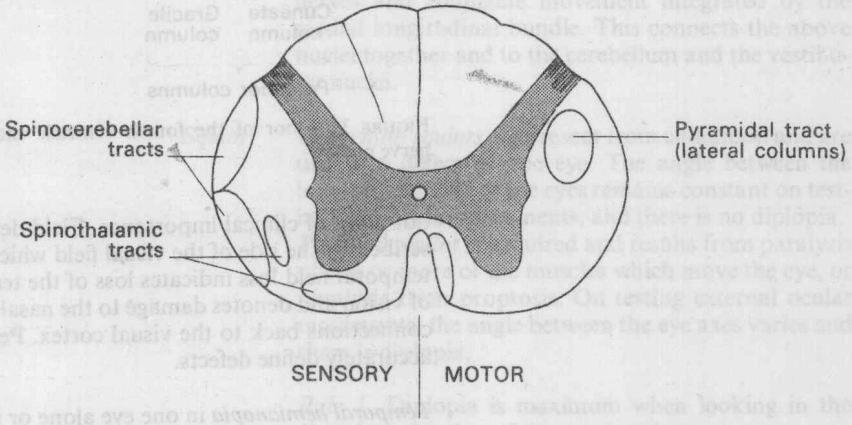

FIGURE 1 Cross-section through spinal cord.

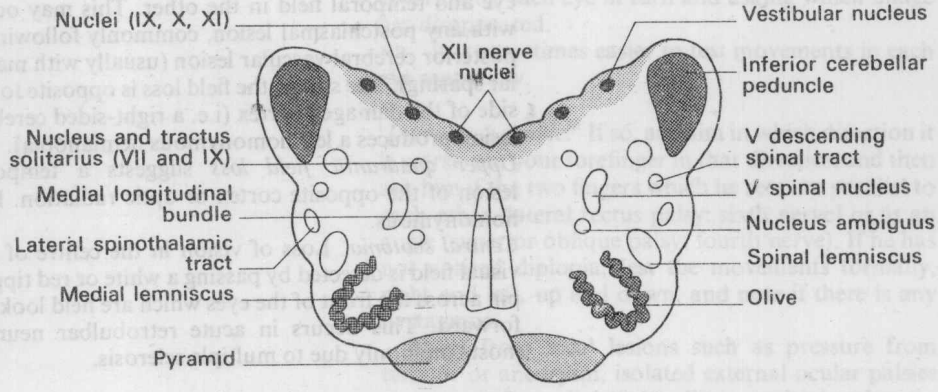

FIGURE 2 Cross-section through open medulla.

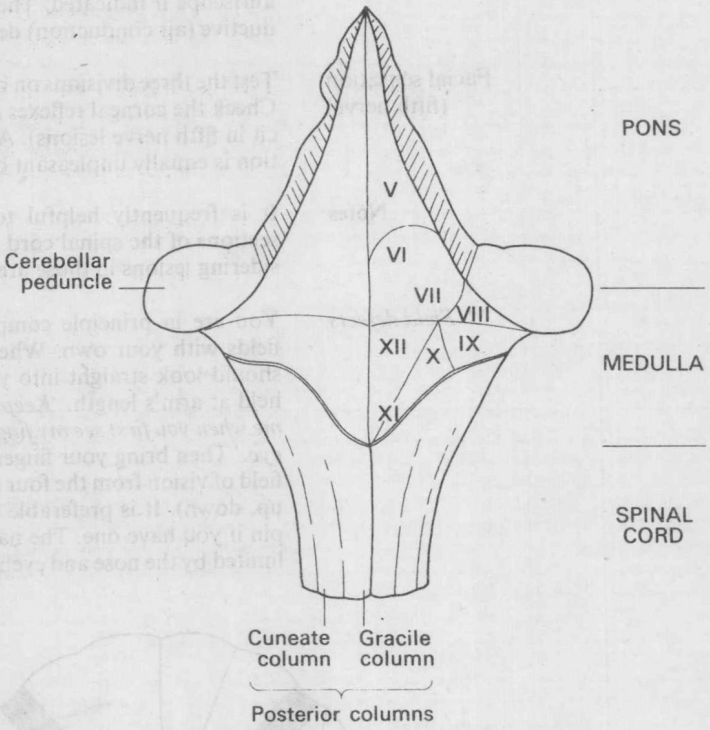

FIGURE 3 Floor of the fourth ventricle showing cranial nerve nuclei.

not often of clinical importance. Field defects are described by the side of the visual field which is lost, i.e. temporal field loss indicates loss of the temporal field of vision and denotes damage to the nasal retina or its connections back to the visual cortex. Perimetry will accurately define defects.

Temporal hemianopia in one eye alone or in both eyes (bitemporal hemianopia) suggests a chiasmal compression usually from a pituitary tumour.
Homonymous hemianopia i.e. loss of nasal field in one eye and temporal field in the other. This may occur with any postchiasmal lesion, commonly following a posterior cerebral vascular lesion (usually with macular sparing). The side of the field loss is opposite to the side of the damaged cortex (i.e. a right-sided cerebral lesion produces a left homonymous hemianopia).
Upper quadrantic field loss suggests a temporal lesion of the opposite cortex or optic radiation. It is homonymous.
Central scotoma. Loss of vision in the centre of the visual field is detected by passing a white or red tipped pin across the front of the eyes which are held looking forward. This occurs in acute retrobulbar neuritis most commonly due to multiple sclerosis.

Blindness A history of transient blindness, total or partial (with specific field defects usually in one eye) is not uncom-

mon in migraine. It may also follow vertebral arteriography and carotid transient ischaemic attacks.

Sudden blindness also occurs with
— retinal detachment
— acute glaucoma
— vitreous haemorrhage in diabetes
— temporal arteritis and retinal artery or vein obstruction
— fractures of the skull
— raised intracranial pressure

NB The light reflex is absent except in cortical blindness.

Senile changes and *glaucoma* account for about two-thirds of blindness in this country. Diabetes is the major non-ocular (systemic) cause (7–10%) due chiefly to vitreous haemorrhage and cataract. Trachoma is a common cause on a world-wide basis. *Hysterical blindness* is uncommon and should never be confidently assumed.

The blindness of *temporal arteritis* is preventable if steroid therapy is started in time.

Eye movements

These are controlled by the third, fourth and sixth nerves and conjugate movement integrated by the medial longitudinal bundle. This connects the above nuclei together and to the cerebellum and the vestibular nuclei.

Squint

Congenital squints are present from childhood and are due to a defect of one eye. The angle between the longitudinal axes of the eyes remains constant on testing extraocular movements, and there is no diplopia.

Paralytic squint is acquired and results from paralysis of one or more of the muscles which move the eye, or paralysis from proptosis. On testing external ocular movements, the angle between the eye axes varies and there is diplopia.

Rule 1. Diplopia is maximum when looking in the direction of action of the paralysed muscle.

Rule 2. The image furthest from the midline arises from the 'paralysed' eye. This may be determined by covering up each eye in turn and asking which image has disappeared.

NB It is sometimes easier to test movements in each eye separately.

'*Do you see double?*' If so, ask him in which direction it is worst, put your forefinger in that direction and then ask him if the two fingers which he sees are parallel to each other (lateral rectus palsy: sixth nerve) or at an angle (superior oblique palsy: fourth nerve). If he has not noticed diplopia, test the movements formally, right and left, up and down, and note if there is any nystagmus.

Apart from local lesions such as pressure from tumour or aneurysm, isolated external ocular palsies may result from diabetes mellitus, multiple sclerosis,

polyarteritis, sarcoidosis, syphilis and meningitis (usually tuberculous or pneumococcal).

Lateral rectus palsy (sixth nerve)

This produces failure of lateral movement with convergent strabismus. It is the commonest external ocular palsy. The diplopia is maximal on looking to the affected side. The images are parallel and separated horizontally. The outermost image comes from the affected eye and disappears when that eye is covered. The palsy is produced as a false localising sign in raised intracranial pressure, by direct involvement with tumour, aneurysm, or rarely with acoustic neuroma (page 107).

Superior oblique palsy (fourth nerve)

This type is rare. Palsy produces diplopia maximal on downward gaze. The two images are then at an angle to each other when the palsied eye is adducted and one above the other when the eye is adducted. The diplopia is therefore noticed most on reading or descending stairs.

Third nerve palsy

It may not present with diplopia because there is complete ptosis. When the lid is lifted the eye is seen to be 'down and out' (divergent strabismus) and there is severe (angulated) diplopia. The pupil may be dilated. It occurs with space-occupying lesions, brain-stem vascular lesions (Weber's syndrome) and aneurysm of the posterior communicating artery.

The muscles themselves are involved in myasthenia gravis and in the ophthalmoplegia of thyrotoxicosis.

Pupillary reflexes

The balance between parasympathetic (constrictor) and sympathetic (dilator) tone controls pupil size.

Constriction of the pupil in response to light is relayed via the optic nerve, optic tract, lateral geniculate nuclei, the Edinger–Westphal nucleus of the third nerve and the ciliary ganglion. The cortex is not involved.

Constriction of the pupil with accommodation. Convergence originates within the cortex and is relayed to the pupil via the third nerve nuclei. The optic nerve and tract and the lateral geniculate nucleus are not involved.

Therefore:

— if the direct light reflex is absent and the convergence reflex is present, a local lesion in the brain-stem or ciliary ganglion is implied, e.g. Argyll Robertson pupil

— if the convergence reflex is absent and the light reflex is present, a lesion of the cerebral cortex is implied, e.g. cortical blindness

Examination of the pupillary reflexes should be performed in subdued light. The pupil should be positively inspected for irregularity. A torch is flashed twice at each eye (once for direct and once for consensual responses), preferably from the side so that the patient does not focus on it (and hence have an accommodation-convergence reflex).