Clinical Neuro-ophthalmology

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SECOND EDITION

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Preface

The satisfaction which derives from clinical practice confined to a special branch of medicine is due mainly to the grasp of detail within a limited field. There is a no-man's-land between specialities which sometimes gets neglected. The functional link between the eye and the brain is close; a large part of the afferent flow to the brain comes from the retina, and normal ocular movement is a beautiful example of neural integration.

On the clinical side difficult problems arise in this borderland between neurology and ophthalmology. Disease of the choroid, particularly if adjacent to the disc, may be wrongly diagnosed as intracranial disease, and diplopia due to myasthenia may lead to the mistake of surgical operation on the ocular muscles.

The approach is clinical and the presentation selective. An extensive revision of the text has been undertaken for this edition. We are grateful to those who pointed out errors in the first edition or made constructive criticism. Some regrouping has been made in the early chapters to avoid fragmentation in the description of methods. For example, visual field examination is now collected in Chapter 5. The section on radiology has been revised extensively to take account of the major impact of computed tomography in diagnosis and management. Sophisticated equipment is not always available and the descriptions of other established methods have been retained and placed in perspective. A new section on the visual evoked potential has been incorporated. Under retinal photography the use of red-free light is discussed. The chapters on eye movement, nystagmus, the chiasma, and cortical disorders of vision have been rewritten. Chapter 14 has been revised to take account of modern views on optic neuritis, ischaemia of the optic nerve, papilloedema, and benign intracranial hypertension. Some diagrams have been replaced and summaries of anatomy added. New clinical photographs and illustrations of pathology and electrical findings are included. The number of references has doubled. They are now placed at the end of each chapter.

The book remains an account of the subject with emphasis on neurology. It is intended primarily for the ophthalmologist though offering guidance in diagnosis and management to both the trainee neurologist and radiologist. Only elementary ophthalmology is included to give perspective, provide a coherent text, and help those who are not ophthalmologists.

Edinburgh — *Bryan Ashworth*

Manchester — *Ian Isherwood*

Acknowledgements

For this edition Emanuel Rosen has again provided the fundus illustrations. Much of the illustrative material derives from the Departments of Neurology, Neurosurgery, Ophthalmology and Medical Illustration of the University of Manchester. The influence of Dr R.G.W. Ollerenshaw can be detected in many of the illustrations.

In preparing this edition we have been helped by many friends and colleagues. Dr E.H. Jellinek has read several chapters in draft, Professor C.I. Phillips commented on Chapter 13, and Dr M. Saunders read parts of the manuscript. Dr H.R.A. Townsend discussed aspects of clinical neurophysiology and provided the illustrations of the visual evoked potential. Dr G.T. Millar prepared the illustration of the electroretinogram. Dr M.C. Grayson with Mr C. Hood provided the photograph of retinal fibre atrophy. Dr A.F.J. Maloney made available the illustrations of neuropathology. Most of the new clinical illustrations for this edition have been prepared in the University Department of Medical Illustration at the Western General Hospital, Edinburgh under the guidance of Mr W. Hopkins.

Other illustrations are gratefully acknowledged. Professor W. R. Lee of Glasgow provided the microscopical preparations of the retina (Fig 14.6), Dr C. Mawdsley the clinical photograph of orbital pseudotumour (Fig. 10.6), Mr M.J. Roper-Hall the photographs of familial ptosis (Fig. 11.8) and Marcus Gunn syndrome (Fig. 11.12) which are reproduced from *Aspects of Neuro-ophthalmology* by permission of Butterworth and Co. Professor R. Warwick and H.K. Lewis and Co. kindly allowed us to reproduce as Fig. 11.3 the schematic diagram of the oculomotor nucleus from *Wolff's Anatomy of the Eye and Orbit*, 7th edition. Professor R.H. Johnson and Dr J.M.K. Spalding made available Fig. 9.4 from their textbook of *Disorders of the Autonomic Nervous System* published by Blackwell Scientific Publications. Dr Ann Harden, Dr G. Pampiglione and the Editor of the *Lancet* have allowed us to reproduce as Fig. 7.7 an illustration from their paper in the *Lancet* (1970) **1**, 806.

Finally, we are obliged to the editor and publishers of the *Bristol Medico-Chirurgical Journal*, *Journal of Neurology, Neurosurgery and Psychiatry*, and *Recent Advances in Clinical Neurology* for permission to reproduce illustrations from our own papers as Figs. 11.10, 14.3, 15.2, 15.3 and 16.2.

Contents

CHAPTER 1

Introduction

Neuro-ophthalmology may be defined as the study of eye symptoms and signs in relation to disease of the nervous system. The aim of this book is to present the essential clinical aspects of the subject with due regard to scientific background and related subjects.

Space does not allow a detailed account of anatomy, physiology, pharmacology and genetics in relation to the eye. Yet these subjects form an essential foundation for the understanding of clinical problems and an attempt has been made to highlight important basic topics in relation to clinical work.

In most standard textbooks the conditions to be considered are grouped according to causative factors, or under disease headings, but this is of little help in the differential diagnosis of symptoms and signs. The first aim of any consultation is to reach a diagnosis and we have therefore adopted an analytical arrangement which is based on symptoms.

The methods of clinical investigation available to the neuro-ophthalmologist allow considerable precision in diagnosis. It is important, therefore, that they are directed towards a clear objective. Air encephalography with its attendant discomforts has now been replaced to a large extent by computerized tomography. Lumbar puncture is still required in some cases and familiarity with the technique, complications and risks is essential. As methods proliferate there is a tendency to add more tests rather than to use those which are appropriate. We have tried to give some guidance in the choice of investigations. Recent progress in retinal photography, radiology, and electrodiagnosis has increased the accuracy of clinical diagnosis and an account of these has been included.

The literature of neurology and ophthalmology is now enormous and this poses considerable problems in selection and documentation. In a volume of this size it is impossible to provide a complete bibliography. In the chapters on methods, including the section on radiology, references are limited, and often refer to the more comprehensive textbooks. In the clinical chapters the documentation is more detailed and leads to reviews, key papers and recent work. We have included recent papers in some instances to the neglect of important early contributions because the papers are themselves the source of further references and this applies particularly to topics on the borderland of the subject.

Finally, we would make a plea for closer integration between neuro-ophthalmology and other specialities. Eye signs are seldom sufficient for the diagnosis of neurological disease.

CHAPTER 2

Normal visual function

This chapter provides an outline of normal visual function with emphasis on the effect of age, the integration of various physiological mechanisms, and the disorders which may develop.

The primitive eye is a detector of light and of movement. The appreciation of colour is a refinement. In man, the other notable features are the visual field in relation to the position of the eye, binocular vision and the almost constant movement of the eye. The visual acuity also depends on the focusing mechanism which shows gradual change throughout life.

Normal vision in the human subject is therefore much more than normal visual acuity. It will be considered under the headings:

The refractive system of the eye
Macular vision and peripheral vision
The visual field
Fixation
Binocular vision
Visual pathway and cortical vision
Visual perception
Colour vision.

The refractive system of the eye

The image of an external object is brought to focus on the retina by refraction of light waves as they pass through the cornea, aqueous humour, lens and vitreous body. The cornea (about 40 dioptres) and the lens (about 17 dioptres) are powerful refracting surfaces and the lens has the additional special property of changing its shape and so

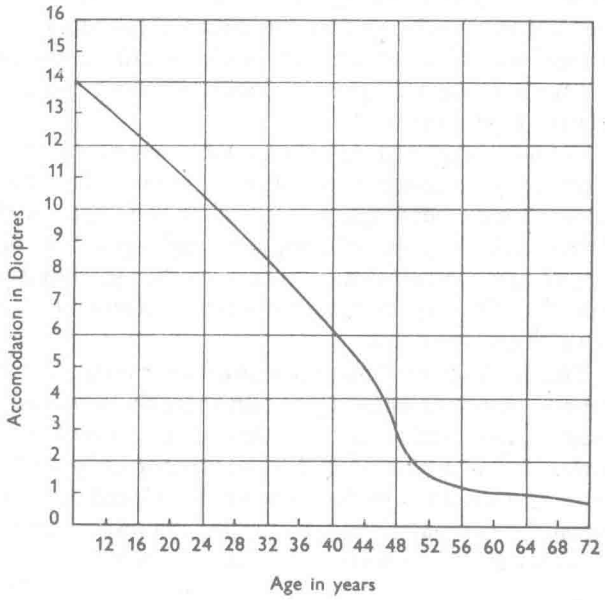

Fig. 2.1. Diagram to show the change in accommodation with age—average values (Duane). Four dioptres of accommodation with an emmetropic eye allows an object to be seen clearly at the near point (25 cm). When the range is less than this the near point is further from the eye.

altering its refractive power (*accommodation*). In early life the lens is capable of large changes in refractive power. This diminishes with age (Fig. 2.1) so that by the age of 45 years it has reached about 4 dioptres and the patient has difficulty in seeing objects held close to the eye (*presbyopia*). The loss of accommodation shows some individual variation but the diagram may be taken as representative. The other refracting media show much less change with age.

In the normal or emmetropic eye the optical system allows the image to be brought to a sharp focus on the retina. If the image is formed in front of the retina the patient is *myopic* and if behind the retina he is *hypermetropic*. The hypermetropic subject is able to compensate for the defect by means of accommodation in early life but this becomes less effective as age advances and the power of accommodation diminishes.

The eye of the newborn infant is relatively hypermetropic. This diminishes in the first few years and emmetropia may be reached at 6 months. The infant may be able to perform imitative movements at two weeks, recognise a face at one month, and carry out smooth tracking after two months.

When growth ceases the refraction stabilises. Presbyopia usually becomes apparent in the fifth decade. In old age, changes in the lens cause hypermetropia. If the refraction differs in two perpendicular planes through the visual axis the eye is said to show *astigmatism*. Minor degrees of astigmatism may cause marked impairment of visual acuity and distortion of the image.

Macular vision and peripheral vision

The macula is formed only of cone receptors and is a more sensitive area of visual discrimination than the peripheral retina in conditions of good illumination. Disease of the macula or the nerve fibres which pass from it therefore produces a marked impairment of visual acuity. One factor of crucial importance in determining the resolving power of the eye is the distance of separation of the receptors in the macular area. If the refractive system of the eye is normal but the macula is not functioning the visual acuity is substantially reduced.

The peripheral retina contains both rods and cones. The rods are responsive in conditions of poor illumination (scotopic vision) and the capacity for *dark adaptation* is a measure of their function.

The visual field

The uniocular field is limited by the area of the retina but the projection is influenced by the position of the eye in the orbit and other anatomical factors such as prominence of the frontal ridge and the nose. The methods of assessing the field will be discussed on p. 22 and at this point only the general features will be mentioned.

The blind spot, corresponding to the optic disc, is located in the temporal field of each eye about 17 degrees out from the axis. Normally, the individual is unaware of the blind spot and interprets images as if it did not exist. It is sometimes a matter of considerable difficulty to get a patient to recognise his own blind spot using the Bjerrum screen. This is partly because the eye does not remain in a fixed position under ordinary conditions. Accurate determination of the blind spot requires that the patient can maintain fixation. If the patient cannot bring himself to do this the field findings are unreliable. If there is macular disease or other disturbance giving a central scotoma fixation becomes difficult and again the plotting of fields is unreliable.

It is possible for a patient with normal visual acuity to have a gross defect of the visual field. The extreme situation is seen in tubular vision which implies a gross concentric constriction of the field and occurs in retinal disease (retinitis pigmentosa) and cortical disturbance of vision.

Fixation

Fixation is the process by which an image is aligned on the macula and held in position there by movement of the eye. This is acquired within a few weeks of birth and convergence may be demonstrated within a fortnight of birth.

Binocular vision

The combination of two eyes with overlapping fields allows vision in three dimensions and the judgement of depth. Until recently it was believed that stereoscopic

vision was added to the system after clear images had been formed at the maculae and the eye movements coordinated. This concept has been transformed by experimental studies in man, the monkey and the cat. The new observations include the effect of covering one eye, the influence of visual environment, interhemispheric connections, and the use of radioactive isotopes in tracing neurone pathways [1–4].

The normal condition of *orthophoria* develops early in life and human binocular fusion can be demonstrated two months after birth. In the normal it soon stabilises but can be arrested by covering one eye for 3 or 4 days during development. There is a reserve of function in relation to fusion which can be assessed by placing a prism in front of one eye and increasing the strength until double vision is produced. This *verging power* has considerable range in the muscles producing horizontal movement but only one or two degrees for the vertical muscles. Impairment of this reserve of fusional capacity is *heterophoria* or *latent strabismus*.

In 2 to 5 per cent of human subjects there is significant impairment of binocular fusion and strabismus is found. It can be demonstrated by asking the patient to look at an object and then covering one eye. The occluded eye deviates (see Fig. 4.1).

When the condition is found in the early years of life attention is usually directed to the correction of any refractive error and patching of the eye with better vision. This is combined with orthoptic exercises and in more severe cases surgical transposition of the insertion of appropriate external ocular muscles. The cosmetic result is good but the stereoscopic vision is often not restored and this hardly ever happens when treatment is attempted after the fifth year of life. Recent work has stimulated a revision of these methods and there may be a place for intensive controlled stimulation of the amblyopic eye for short periods [5].

ELECTRICAL RECORDING

Much of the new work is based on microelectrode recording from the cortex or lateral geniculate body (LGB) in animals. The number of cells is large but it is possible to isolate cell units. The cell unit can be identified by exploring the receptor field within which stimulation is effective in producing an electrical discharge. The stimulus may be a flash of light, specific wave band of the visible spectrum, change in intensity, or a pattern of movement.

The rate of cell discharge is increased by stimulation and it is found that cell units respond only to a specific stimulus. Particular interest arises in relation to line orientation. Response from a given cell may only be demonstrated by movement at a given angle [6].

In the LGB each cell is influenced by several hundred retinal ganglion cells. In general, a cell in this region responds to stimulation of one or other eye but not both.

In the cortex most cells respond to stimulation of either eye and a slight disparity between the two eyes might be an important factor. Cells which are responsive to binocular depth have been found in area 18 of the macaque [7].

It is clear that the cortex does not receive all impulses generated in the retina. There is a process of excitation and inhibition along the visual pathway. Inhibitory fibres pass forwards from cortex to LGB and so prevent many afferent impulses from reaching the cortex.

OCCLUSION OF ONE EYE

Studies of eye closure in kittens show some parallel with the problem of squint in the human. Occlusion of one eye, even for a brief period of time, may, if carried out at a sensitive stage of development, have permanent effects on cortical responses and the pattern of visual function [6]. Some recovery occurs after a short period of occlusion. The effect of covering one eye depends on the other being open and binocular occlusion at any stage has little effect. When the sensitive period of development has passed occlusion has no influence on vision.

VISUAL ENVIRONMENT

The immature visual system of the kitten is permanently influenced by the environment. For example, kittens exposed only to vertical stripes become unresponsive to horizontal lines [8] and those reared in an environment of spots do not respond to line stimulation [9].

INTERHEMISPHERIC CONNECTIONS

Evidence from both anatomical and physiological studies shows that fibres of the corpus callosum include connections between the occipital lobes. It might be expected that stereopsis depends on these fibres and this is supported by observations in human subjects in whom the corpus callosum has been divided. This is occasionally performed as a treatment for intractable epilepsy. A detailed study of one patient showed that the depth of an object in the midline could not be appreciated although peripheral stereopsis was normal [10]. Judgement of depth in the midline is still possible after antero-posterior transection of the optic chiasm [11].

Fibre tracts. The pathway of fibre tracts has been studied in the past by showing degeneration after transection. By using radioactive isotope tracers it is now possible to utilise axonal flow and identify substances in the visual pathway. For example labelled aminoacids may be injected into the eye and detected in the appropriate layers of the LGB. Similiarly, horseradish peroxidase introduced into the cortex can be used to trace the pathway to the LGB and show the effects of monocular occlusion.

Visual pathway and cortical vision

The fibre pathways passing from the retina to the occipital cortex show a constant anatomical arrangement and synapse in the lateral geniculate body. Fibres from the nasal half of the retina cross in the chiasma and the proportion which cross is about half of those making up the optic nerve. A point-to-point relationship has been shown between the retina and calcarine cortex. It is now generally agreed that no single receptor point in the retina is bilaterally represented in the cortex. The clinical applications of these findings are discussed in the chapters on visual failure.

Experimental work in the cat has shown that some cells in the cortex are sensitive to a narrow band of spatial frequency [12]. The visual cortex can therefore be regarded as a spatial frequency analyser. Spatial frequency can be defined by using a grating of alternate light and dark bars and stating the number of bars per unit of visual angle. The response can be recorded from the retina, LGB and cortical cells. In the cat it can be shown that the cellular response at all these sites increases as the contrast of the grating increases. For some cortical cells there is a linear relationship between response amplitude and the logarithm of grating contrast [13]. On this basis the visual system could be considered as a Fourier analyser. In other words any spatial distribution of luminance is the sum of sinusoidal components which are defined in terms of amplitude, frequency and phase. Synthesis of these components in the human cortex has been shown by applying different sinusoidal waves to each eye of a subject [14].

Gratings are of some clinical value in the assessment of visual function (p. 13).

Visual perception

Perception may be defined as the process by which information is isolated and identified from environment or background noise. Visual perception is primarily concerned with light and colour. Other basic considerations are brightness and movement. Spatial features are secondary. In the processes of perception eye movement is closely associated. Other factors are vertical and horizontal orientation, depth and scanning movement [15, 16].

Some disorders of perception due to organic disease are discussed but the reader is referred to Heaton [17] and Davidoff [18] for further information.

Colour vision

Theories of colour vision are outside the scope of this book. Any colour can be formed by the compounding of the three basic colours—red, blue and green (or yellow). White light is formed of these three basic colours as can be shown when a prism is placed in a beam of light and forms a spectrum. This trichromatic system forms a basis for classification of disorders of colour appreciation. Failure to appreciate colour may be congenital, or acquired as a consequence of disease.

Congenital disorder of colour vision is rare in women but affects about 8% of men. The majority are *anomalous*

trichromats and have the same general characteristics as normal trichromats but with minor differences. About 2·6% are *dichromats* and can match any colour with a mixture of two primary colours. A very small number of *monochromats* exist and they can match a colour with any other colour by adjustment of intensity alone. Monochromats can be divided into *rod* monochromats who lack functioning cones and have poor vision and *cone* monochromats whose vision is normal apart from the colour defect.

In disease of the retina there may be a disturbance of colour appreciation and this is usually in the yellow-blue or violet-blue-green area. Disease of the conductive layers of the retina or optic nerve may lead to impairment of red-green appreciation.

The clinical testing of colour vision is often unreliable and detailed investigation tedious. Pseudoisochromatic plates such as those of Ishihara are often used for screening but are not entirely satisfactory for classification of patients with neurological disease and may give false positives [19].

The Farnsworth–Munsell 100 Hue test is useful for comparative or serial studies. The patient is given 85 coloured caps placed in four groups and asked to arrange them in a series. Each eye is tested separately and without limitation of time. Normal subjects show some deterioration after 55 years of age [20]. Comparison of the two eyes [21] and modification of the test [22] have improved the accuracy and shortened the procedure. The Farnsworth dichotomous colour test panel D15 is an abbreviated version of the test. Despite these improvements the method does not always clarify red-green defects and gives no indication of the degree of anomaly.

A new method of colorimetry is based on the chromaticity circle of Newton and provided by illumination of red, blue and green filters balanced with a neutral density filter. This gives a white centre to a coloured circle. The area of white is recorded with a mechanical device. The test can be performed in less than five minutes and compares favourably with other methods [23].

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CHAPTER 3

The clinical history

GENERAL

There is a tendency to underrate the value of the medical history but it often indicates the nature of the problem and frequently leads to a provisional diagnosis. In a difficult case it may provide the clue which cannot be obtained in any other way.

The preliminary information to be noted must include the full name, address, age, marital status, and occupation of the patient and name and address of the doctor. This is often entered by a clerical assistant and it is important that the details are accurate.

The medical history is best obtained at first hand from the patient. It may be supplemented by relatives or others. The environment of the consultation should be quiet and free from interruption by telephone and other people passing in and out of the room. Above all, the taking of a satisfactory history requires a relaxed attitude combined with intense concentration on what is being said. The details are obtained more easily in complete privacy but a nervous person may feel more comfortable if accompanied by a relative or friend. Parents will expect to be present when a child is seen but must not be allowed to interrupt the most important source of information. The presence of relatives avoids the need to go over the story again and it is always worth asking at the end if they have anything to add. It may also be helpful to see a relative alone and ask about the patient's history and habits.

SYMPTOMS

The first step is to find out what is the main symptom. *What does he feel wrong?* If there is more than one symptom it is helpful to try to place them in order of importance to the patient. The next step is to trace each main symptom from the time of onset and note the course. At this stage it is worth allowing the patient to give his own account even if it does not follow an exact sequence of time or logic. There is a tendency to relate not only the facts but the interpretation placed upon them by himself and others. This is sometimes irritating to the doctor but does provide a glimpse of the patient's attitude towards his illness. If the flow of history is stopped then many facts may go unmentioned, but that is not to say that a garrulous individual should be tolerated indefinitely. In trying to assess the information provided by the patient it is useful to distinguish information volunteered from that revealed by questioning. It must, of course, be realised that information which the first doctor obtained by questioning may be volunteered to a second doctor. The patient must be given time to describe his symptoms but valuable additional data may be obtained by direct questions and leading questions.

Questions are certain to arise out of what has been said if details of timing and duration are to be cleared up. The symptoms may suggest a possible diagnosis and then supplementary questions may be asked with a view to confirming or ruling it out. This is largely a matter of experience and is learned in the first instance by hearing an experienced clinician at work and then developing a personal approach based on that of others. Perhaps the most important consideration is that every question should have a purpose (although the reason will not always be apparent). It is better to avoid leading questions at first but when used with discrimination they may be of the greatest help in diagnosis.

When the sequence of events leading to the consultation has been unravelled it is often clear why the patient has appeared at that particular time but sometimes it is not. The question *why has this patient come now* is always an important one to answer and particularly when the symptoms are of long duration. The answer frequently sheds light on the circumstance of the individual. He may have been pressed into seeking advice by a relative, an acquaintance, or his employer. He may be seeking reassurance about some long-standing disability before undertaking marriage or some other commitment. He may have a relative with serious disease who has similar symptoms or have some other reason for fearing the outcome of his own condition. It is not always possible to establish the reason, perhaps because of diffidence and particularly in the presence of an audience. A few patients are referred because of some finding during a routine examination.

When the immediate history has been obtained an enquiry about previous symptoms of a similar kind and *previous serious illnesses* and operations should be made. The *family history* may be significant and it is worth asking about the parents and siblings of the patient in all medical problems. When the findings suggest a possible genetic basis it may be necessary to probe more deeply into the family history. There is a tendency among members of families affected with hereditary disease to suppress information, perhaps in the reasonable hope that another cause will be found. The examination of relatives frequently reveals unsuspected findings in cases of this type. The social history is helpful and the details of the patient's work (which may be very different from what is suggested by the title of his job).

The number of supplementary questions which might be asked is legion and there is not much to be gained by using lists of questions; it is better to be guided by what has been said. It must be admitted that all the facts may not come to light at the first consultation but it is always worth trying to obtain a complete account at the beginning. Failure to do so may well undermine confidence at a later stage when some point of fundamental importance is revealed. The history provides an excellent opportunity to establish rapport with the patient and gain his confidence. The basic essentials are an interest in what is being said and a genuine desire to help.

An illustration of the value of the history may be helpful. A man of 25 years came with the complaint of double vision. Some three weeks previously he had woken in the morning to find that he saw two images, side by side, of every object in the room and that this was more marked on looking to the left. He had also found that covering up one eye relieved the diplopia which had remained unchanged since the onset. Discussion of the past history revealed that five years ago there was an episode of blurred vision in the right eye which lasted about six weeks. This was called conjunctivitis at the time but there was no redness or discharge at the onset, the eye was painful at first and the vision became more blurred over the course of a few days, after 3 weeks it began to improve and after 6 weeks it was normal.

A history of this type at once raised the possibility of multiple sclerosis, beginning with acute optic neuritis and followed by a brain stem lesion giving diplopia. The presence of other physical signs might support this diagnosis but even in the absence of these suspicion must be strong. It might therefore be better to wait for a few weeks to see if there was improvement. Remission of the diplopia would be another point in support of the diagnosis. There would then be no purpose served by elaborate neuro-radiological investigation.

The extent to which the patient's symptoms can be analysed will depend on his own perception and intelligence but is facilitated in clinical work by familiarity with the condition under consideration. In this respect the specialist has a considerable advantage but he must resist the temptation to modify the symptoms so that they can be fitted into a rigid diagnostic group. A few of the commoner symptoms will be discussed briefly.

Double vision (diplopia) is a common symptom but should not be accepted without detailed questioning. A useful distinction is made between uniocular diplopia, due to disorder of the eye, and binocular diplopia, which is due to disordered ocular movement and relieved by covering one eye. Other points include duration, relationship of the images in space, direction of maximal separation, fluctuation and provoking factors. The demonstration of impaired ocular movement may help to confirm the nature of the diplopia. On the other hand diplopia should not be discounted because the range of eye movement appears full. The analysis of diplopia is discussed in the next chapter.

Blurred vision must be distinguished from diplopia. The main points to establish are its duration, fluctuation, whether the whole field is affected, whether the image is distorted and any effect on colour vision.

Pain is a difficult symptom to describe and assess. The main points to establish are its localisation, radiation, character (aching, burning, stabbing, throbbing), and some idea of its severity. It is also helpful to enquire about provoking and aggravating factors and any measure found to give relief. A throbbing quality may suggest a vascular basis as in migraine and arteritis. Stabbing pain in the distribution of a nerve root suggests irritation or a compressive lesion. The headache of raised intracranial pressure is typically present on waking and settles after an hour or two. Persistent and localised pain may suggest disease of bone or of a nerve root. Chronic pain without signs of disease may be a manifestation of anxiety or depression and is often unresponsive to analgesics.

Blackouts. This is often used to describe attacks with loss of consciousness but sometimes refers to loss of vision. It is therefore important to establish what is meant by the patient. If consciousness was lost it is helpful to obtain an account from a witness but the patient will be able to report the circumstances, onset and symptoms after coming round. The main point in diagnosis of attacks with loss of consciousness is the distinction between syncope and epilepsy. In syncope there is warning of the attack. The patient may feel ill beforehand and the environment may be contributory (unpleasant sight, hot room, etc.) whereas the epileptic attack is usually spontaneous and abrupt in onset.

Dizziness. This word is freely used by patients but impossible to define. A subgroup which can be more clearly described is vertigo. *Vertigo* implies an hallucination of movement with a sense of rotation of objects relative to the patient or of the patient in relation to the surroundings. It is frequently accompanied by nausea or vomiting and sometimes by loss of consciousness. Vertigo usually indicates a disturbance of the labyrinth or its central connections.

Hallucinations of vision may have a basis in disease of the eye or visual pathway and are sometimes part of the migraine attack. The possibility of infection or toxaemia (including alcohol) must be considered (p. 260).

Oscillopsia is a sensation of wobble and is often accompanied by nystagmus. It may be due to disorder of the labyrinth or its connections with the cerebellum and brain stem. Shimmering vision is characteristic of the migraine attack.

Transient visual loss is discussed on pp. 219 and 252.

CHAPTER 4

Methods of clinical examination

Although it is customary to start the consultation by taking the clinical history the examination really begins as soon as the patient is seen.

GENERAL APPEARANCE AND GAIT

The *general appearance* of the patient will indicate whether he looks ill or is in good general condition. The facial expression may reveal the immobility of *Parkinson's disease* or the pink, dry, and indurated skin of *hypothyroidism*. The contour of the face and limbs may suggest *acromegaly* and lead to a careful assessment of the visual fields for evidence of chiasmal compression. In *hypopituitarism* the skin of the face appears smooth, pale, hairless and soft to the touch. In *muscle disease* and *myasthenia* the face lacks expression.

The *gait* is significant in diagnosis and it is better if the patient is unaware that he is being observed. In disease of the basal ganglia the patient ceases to swing the arm at an early stage. Later, the posture tends towards universal flexion and the patient takes short, rapid, steps—the festinant gait—and 'runs after his centre of gravity'. Hypokinesia, rigidity and tremor may complete the clinical picture of Parkinsonism.

If muscle tone is increased the gait appears stiff (spastic). This may be unilateral or bilateral and must be distinguished from painful movement due to arthritis. Patients with spastic weakness often develop foot drop and scrape the shoe along the ground when walking. If this is marked the patient tends to swing the leg outwards (circumduction).

Sensory loss in the lower limbs may cause the patient to develop a high stepping gait and this is seen in peripheral neuropathy, tabes dorsalis and other conditions which cause sensory ataxia.

The gait in myopathy is also characteristic. The pelvis tilts forwards and the patient tends to throw the shoulders backwards and struts and waddles as he walks. He also has difficulty in rising from a chair and may overcome this by pressing the hands on the thighs and 'walking up himself'.

In cerebellar disease the gait is ataxic, the patient tending to stagger and reel like a drunken man. The disability may be considerable although there is no weakness or sensory loss.

Bizarre gaits are met with in hysteria and the features are more marked in front of an audience. The patient is unlikely to fall however extreme the posture unless she makes a deliberate dive to the ground. The patient who behaves in an hysterical manner may have organic disease, sometimes at an early stage of evolution.

General observations of this kind will often suggest the direction of further enquiry and the clinical history is also of great importance in this respect.

The neuro-ophthalmological examination will be described. For details of the neurological examination reference must be made to other works.

EXTERNAL EXAMINATION OF THE EYES

Note is taken of the general contour of the face and any weakness or asymmetry.