Section of Ophthalmology

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Chiasmal Compression

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Presenting Ocular Features

There are various tumours which are liable to cause chiasmal compression: mainly the chromophobe and chromophile adenomas of the pituitary gland, the craniopharyngiomas, the pinealomas and the meningiomas. The ocular effects of the pituitary adenomas and the craniopharyngiomas have been described previously in considerable detail (Wybar & Bloom 1963, Wybar 1971, Wybar & Bloom 1976). The ocular features of chiasmal compression as the result of malignant disease are discussed here in general terms only, without any attempt to bring the statistical data of the previous papers up to date. I wish, however, to include a special mention of the meningiomas because these have not been included in any of the previous communications.

It is obvious that a tumour which causes some degree of chiasmal compression must be associated with the development of a disturbance of the central or peripheral visual field of one or both eyes, and this disturbance is frequently the presenting feature of the condition. Mention will also be made of various anomalies of ocular motility which may be associated with chiasmal compression, and which are sometimes presenting features.

Visual Disturbance

An appreciation of the visual changes which occur in chiasmal compression is dependent on an intimate understanding of the disposition of the afferent visual fibres in and around the optic chiasma. The fibres in the afferent visual pathways are classified according to their origins in the retina (Fig 1), with subdivisions horizontally (upper and lower) and vertically (temporal and nasal) through the fovea, so that the visual fibres may be regarded as arising from one of four quadrants and as subserving central visual function, which is concerned with the foveal and parafoveal (macular) parts of the retina; or peripheral visual function, which is concerned with the retina from the paramacular region to the periphery. The fibres from the retina converge on the optic nerve head with a distribution which is consistent with their origins in the retina (Fig 2), but with a redistribution immediately beyond the optic nerve head (Fig 3), and this is concerned mainly with a shift of the macular fibres into the central part of the optic nerve. This distribution is maintained until the proximal part of the optic nerve, which lies adjacent to the optic chiasma, when the nasal peripheral fibres move from a medial to a slightly dorsomedial position, the temporal peripheral fibres from a lateral to a slightly ventrolateral position, and the macular fibres from a central to a somewhat dorsocentral one (Fig 4A,B,C).

The disposition of the visual fibres in the optic chiasma is shown in Fig 4A,B,C. Certain features should be emphasized which emerge as the result of the distribution of the various groups of the retinal fibres in the optic chiasma:

(1) The temporal peripheral fibres (the uncrossed

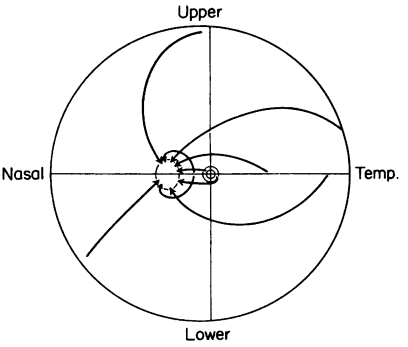

Fig 1 *Direction of visual fibres on passing from different areas of retina to optic disc. (Right eye viewed from behind)*

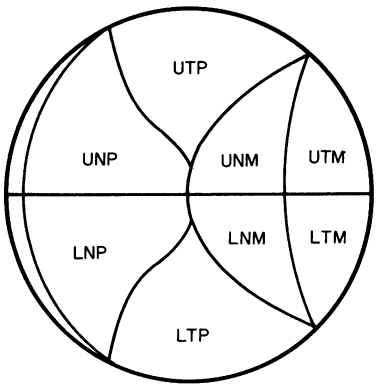

Fig 2 *Distribution of visual fibres in optic nerve head. (Right optic nerve head viewed from behind). UTP = upper temporal peripheral; LTP = lower temporal peripheral; UNP = upper nasal peripheral; LNP = lower nasal peripheral; UTM = upper temporal macular; LTM = lower temporal macular; UNM = upper nasal macular; LNM = lower nasal macular*

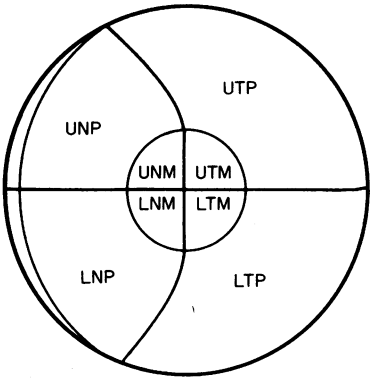

Fig 3 *Distribution of visual fibres in optic nerve. (Right optic nerve viewed from behind). UTP = upper temporal peripheral; LTP = lower temporal peripheral; UNP = upper nasal peripheral; LNP = lower nasal peripheral; UTM = upper temporal macular; LTM = lower temporal macular; UNM = upper nasal macular; LNM = lower nasal macular*

fibres) traverse the lateral parts of the optic chiasma as two widely separated compact masses, with the upper fibres lying dorsally and also to some extent medially in relation to the lower fibres (Fig 4A). This relative medial position of the upper temporal peripheral fibres is important from a clinical point of view. It is obvious that an expanding lesion which exerts its effect on the lateral part of the dorsal surface of the optic chiasma causes an involvement of the lower nasal quadrant of the visual field before the upper nasal one, but this sequence is maintained also when the lesion is impinging on the ventral surface of the chiasma because of the relatively medial position of the upper temporal peripheral fibres.

(2) The nasal peripheral fibres (the crossed fibres) (Fig 4B) traverse the optic chiasma with a considerable intermingling of the fibres from the two eyes, but with a distinct separateness of the upper fibres, which lie dorsally, and the lower fibres, which lie ventrally. The nasal peripheral fibres are particularly vulnerable to median pressure on the ventral surface of the optic chiasma from an expanding sellar lesion with involvement of the upper temporal quadrant of the visual field before the lower temporal one; this sequence is reversed in a suprasellar lesion, which impinges on the dorsal surface of the chiasma.

(3) The lower nasal peripheral fibres lie in the most ventral part of the chiasma (Fig 4B), and after crossing in the anterior region (the anterior loop) travel towards the contralateral optic nerve, with a

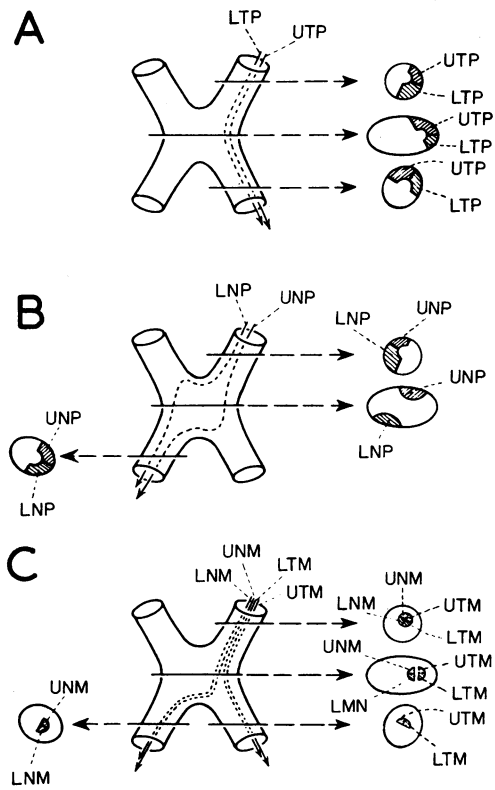

Fig 4 *Distribution of visual fibres in optic chiasma and adjacent parts of optic nerves and optic tracts. A, temporal peripheral fibres; B, nasal peripheral fibres; C, temporal and nasal macular fibres. (Viewed from above, and in cross-section (arrowed)) UTP = upper temporal peripheral; LTP = lower temporal peripheral; UNP = upper nasal peripheral; LNP = lower nasal peripheral; UTM = upper temporal macular; LTM = lower temporal macular; UNM = upper nasal macular; LNM = lower nasal macular*

few fibres actually entering the nerve, before passing backwards to the contralateral optic tract. The fibres which enter the optic nerve are the most peripheral ones, and this is achieved by a single spiral twist of all the nasal peripheral fibres as they cross the chiasma. These fibres are liable to be involved in a lesion near the junction of the optic nerve and chiasma (the anterior chiasmal angle) with the appearance of a small defect in the upper temporal peripheral part of the visual field of the contralateral eye (Fig 5). In such a case the most striking changes occur in the ipsilateral eye: frequently an upper temporal defect followed by a lower temporal one; sometimes a paracentral (junctional) scotoma (Fig 5), which lies in the upper temporal quadrant and tends to extend upwards and inwards as far as the vertical meridian and downwards into the lower temporal quadrant. It tends to be associated with a peripheral temporal defect, so that there is a general confluence of the paracentral and peripheral changes, but sometimes even when the condition is advanced there is the retention of some visual awareness in an area in the midperipheral part of the temporal field. Eventually, in a lesion of the anterior chiasmal angle, there is an involvement also of the nasal field so that the eye becomes more or less blind, but almost invariably there is the retention of a vague awareness of movement.

(4) The upper and lower temporal macular fibres from each eye traverse the lateral parts of the optic chiasma to enter the ipsilateral optic tract, but the upper and lower nasal macular fibres from each eye cross in the posterior part of the optic chiasma to enter the contralateral optic tract (Fig 4C), and median pressure in that region is liable to cause an early disruption of central visual function.

(5) A lesion of the junction of the right optic tract and the optic chiasma (the posterior chiasmal angle) may affect the crossed nasal peripheral fibres with the production of a contralateral temporal hemianopia, and subsequently an ipsilateral nasal hemianopia due to an involvement of the uncrossed temporal peripheral fibres; when such a homonymous hemianopia follows a loss of the opsilateral temporal field from an earlier median pressure on the ventral surface of the chiasma there is blindness of the ipsilateral eye (Fig 6), and it follows that the contralateral hemianopia which is caused by lesion of the posterior chiasmal angle is merely superimposed on a previous temporal hemianopia as the result of the median pressure on the chiasma.

Disorders of Ocular Motility

Chiasmal compression may be associated with a disorder of ocular motility at an early stage so that this is the presenting feature. It may take the form of

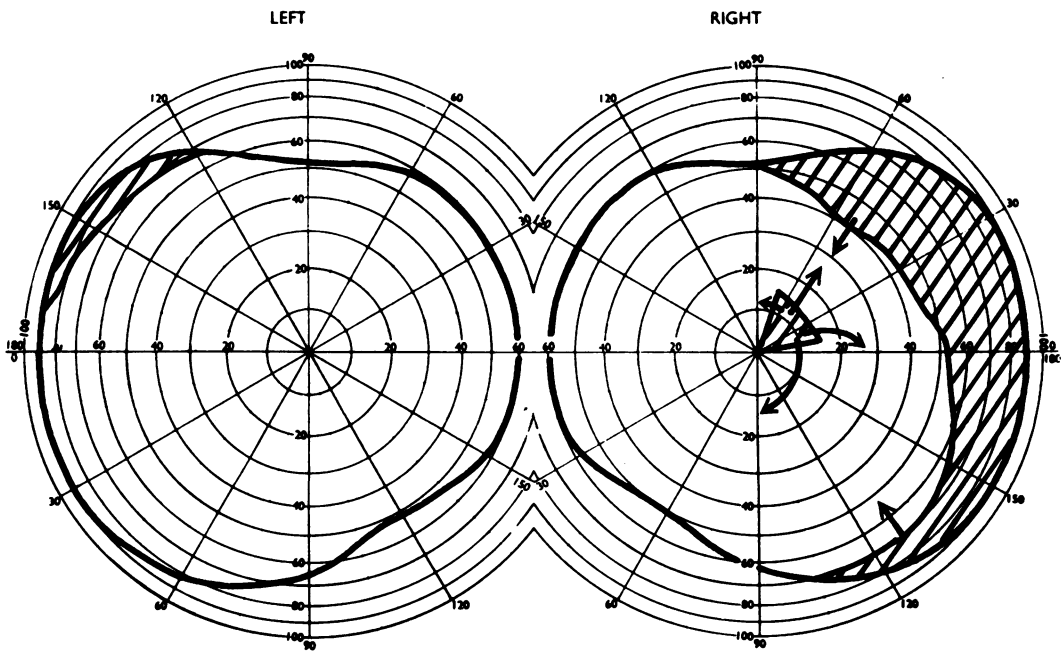

Fig 5 *Visual field charts to show pattern of changes in lesion of right anterior chiasmal angle: in ipsilateral field there is an expanding temporal peripheral defect, upper before lower, and frequently a paracentral scotoma in upper temporal quadrant which expands as indicated by arrows but limited by vertical meridian; in contralateral field there is a small upper temporal peripheral defect. (Hatching = absolute loss)*

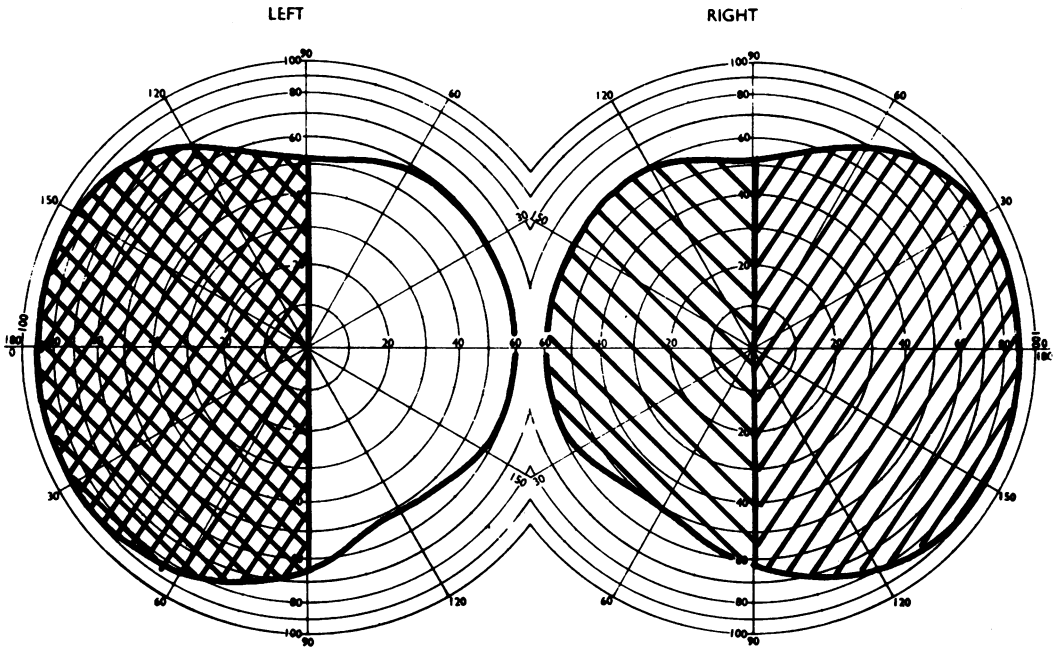

Fig 6 *Visual field charts to show changes in advanced lesion of right posterior chiasmal angle following bitemporal hemianopia due to previous median pressure on ventral surface of chiasma; in left eye a temporal hemianopia (already present) and in right eye a nasal hemianopia, so that right eye is blind. (Hatching = absolute loss; double hatching = absolute loss twice)*

a manifest squint, usually an exotropia or sometimes an esotropia, as the result of a profound defect of the central vision of the squinting eye. It can follow a paresis: rarely a III nerve paresis (when an aneurysm causes chiasmal compression this is more common); and less rarely a VI nerve paresis as the result of increased intracranial pressure, so that it tends to occur mainly in children when the tumour, usually a craniopharyngioma, is associated with hydrocephalus.

Sometimes diplopia may be experienced in the absence of a paresis – the so-called nonparetic diplopia. This is liable to occur when loss of a considerable part of the temporal visual field of each eye, with consequently a significant reduction in the binocular visual field (Fig 7), becomes superimposed on a preexisting phoria so that there is an increasing difficulty in maintaining fusion.

Nystagmus is a relatively rare feature of chiasmal compression. It may, of course, be found after a partial recovery of a VI nerve paresis when there is an exaggeration of the so-called end-point nystagmus. A pecular form of see-saw nystagmus occurs sometimes in a craniopharyngioma or pinealoma: this consists of a movement of one eye upwards with some intorsion and at that time a movement of the other downwards with some extorsion, and then a reversal of these movements (Fig 8). This is a dissociated form of nystagmus.

Meningiomas

This series comprises 21 meningiomas, seen over a nine-year period (1966–74), arising in different situations: suprasellar (13 cases), parasellar (2 cases), frontal (5 cases) and subfrontal (1 case) – but all causing chiasmal compression at some stage. There was no sex difference; 10 were male and 11 female. The average age at the time of the apparent onset was 46 years, with a wide range from 3 to 72 years, but there were only two under 33 (3 and 24 years) and only seven cases under 40, so that it is mainly a condition of early or later middle age.

There was a significant delay in reaching a diagnosis in most cases: only in four before a period of six months; in eight between 6 and 12 months, and in the others after a period of more than one year, with a delay as long as 11 years in one case. The serious nature of any such delay as regards vision is emphasized by the frequent occurrence of visual defects as the presenting feature.

Presenting features: These were mainly different forms of visual disturbance in one or both eyes (18 cases), but in one case the sudden onset of diplopia of a horizontal type, particularly on distant fixation (probably the result of a VI nerve paresis), preceded the disturbance of vision by one year, and in the other cases features of a non-ocular nature – amenorrhœa (1 case) and dizziness with fainting

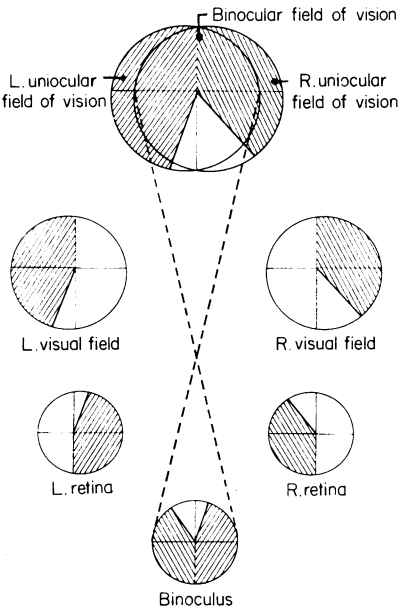

Fig 7 *Gross reduction of binocular visual field in an advanced but not complete bitemporal hemianopia; this may cause a loss of fusion when there is an underlying phoria with production of diplopia (nonparetic diplopia). (Hatching = absolute loss)*

spells and ataxia (1 case) – preceded the visual disturbance.

The importance in the treatment of the meningiomas of early recognition of the significance of a visual defect is obvious, but it is sometimes extremely difficult to detect the feature which is going to provide the diagnosis; particularly in the early stage, when any degree of optic atrophy, as seen with the ophthalmoscope, is of a very subtle nature. A careful, and if necessary repeated, scrutiny must therefore be made in any case which presents with defective vision for which there is no ready explanation. This demands an assessment of the visual fields in any doubtful case. Sometimes simply a careful history gives a worth-while clue, as in the barge-master in this series who found that he was tending to bump his barge into the bank and was experiencing increasing difficulties in negotiating locks; this was scarcely surprising, because he had a complete bitemporal hemianopia. Too often a complaint of a visual defect was explained as the result of a longstanding amblyopia, a retrobulbar neuritis or a peculiar form of macular dystrophy, on totally inadequate clinical grounds, or else it was attributed to 'nerves', an easy but irrational explanation. Sometimes the patient was supplied with new spectacles and then for a time neglected to take further advice, even when these provided only very limited, benefit.

Optic discs: In all cases at the time of the first examination there was evidence of some degree of optic atrophy, but in some this could easily have been overlooked without thorough examination, including careful comparison of the right and left optic discs. This is of importance because as a general rule both optic discs are remarkably similar in appearance in the absence of any disease process and of any marked degree of anisometropia.

In 12 cases one optic disc appeared normal, with slight temporal pallor (2), moderate pallor (2) and marked pallor (8) of the other disc. In 4 cases one disc showed slight temporal pallor with marked pallor of the other. In 2 cases there was moderate pallor of one disc and marked pallor of the other disc, and in a further 2 cases pallor was marked in both eyes. In one case there was slight temporal pallor of one optic disc, but the other could not be assessed because of extensive changes in the anterior segment of the eye as a result of a perforating injury in childhood, leading to a more or less complete loss of visual function.

Visual disturbance: In 7 cases there was blindness (no perception of light) of one eye with, in the other eye, an absence of any visual disturbance (4), a defect of the temporal peripheral part (upper more than lower) of the visual field (1 case) (Fig 9), a defect of the upper part (temporal more than nasal) of the visual field (1 case) (Fig 10), and a defect of the lower part of the visual field with some relative loss of the upper nasal part and a central scotoma (1 case). In 2 cases there was only an awareness of movement in one eye with, in the other eye, some involvement of the extreme upper temporal peripheral part (1 case), and a partial involvement of the temporal peripheral part with a paracentral scotoma in the nasal field (1 case) (Fig 11).

In 2 cases there was a general constriction of the visual field of one eye with a normal visual field in the other eye, but in one of these cases the constriction was of a relative nature. In one case there was a fairly marked involvement of the visual field of one eye except in the upper nasal region, and only a slight involvement of the upper temporal peripheral region of the other eye (Fig 12). In one case there was an involvement of the visual field of

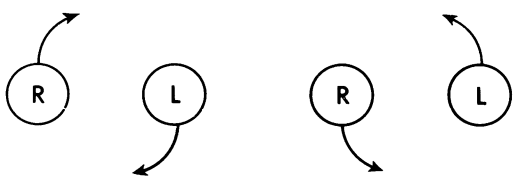

Fig 8 *Diagram to show see-saw nystagmus in chiasmal compression; right eye turns upwards with intorsion whilst left eye turns downwards with extorsion, and then right eye turns downwards with extorsion whilst left eye turns upwards with intorsion*

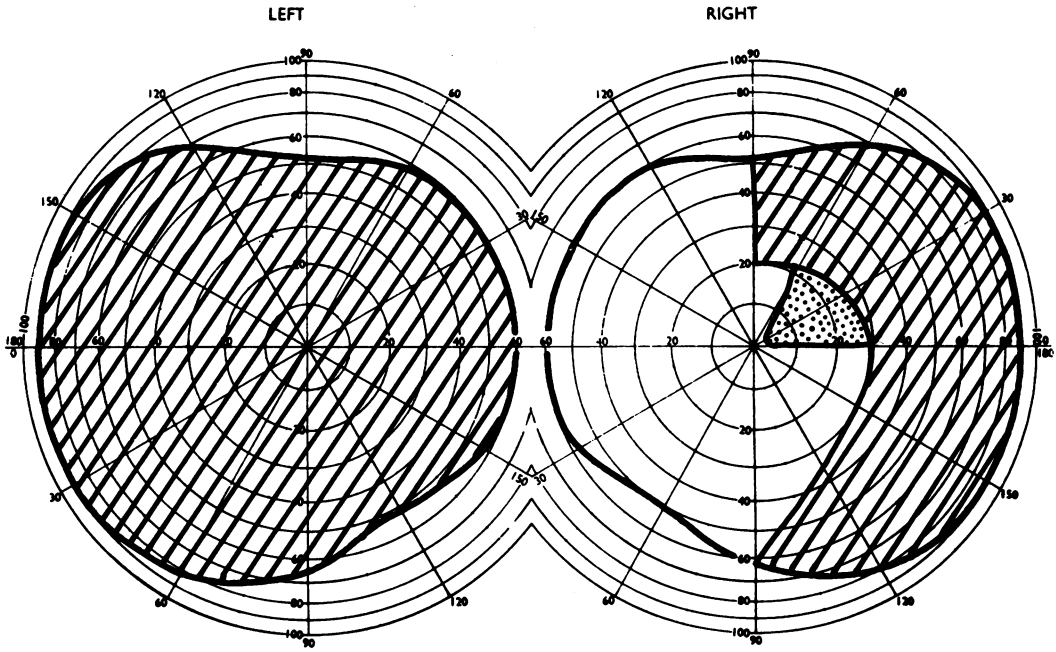

Fig 9 *Visual field charts to show blindness of left eye and partial temporal defect in right eye. (Hatching = absolute loss, dotting = relative loss)*

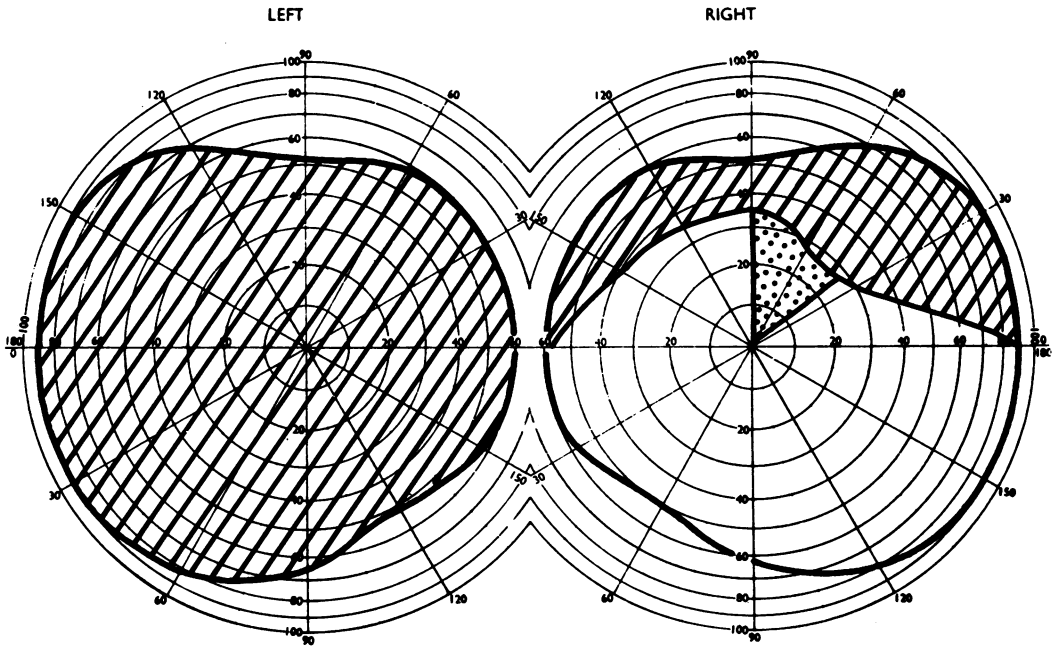

Fig 10 *Visual field charts to show blindness of left eye and partial defect of upper part of right eye. (Hatching = absolute loss, dotting = relative loss)*

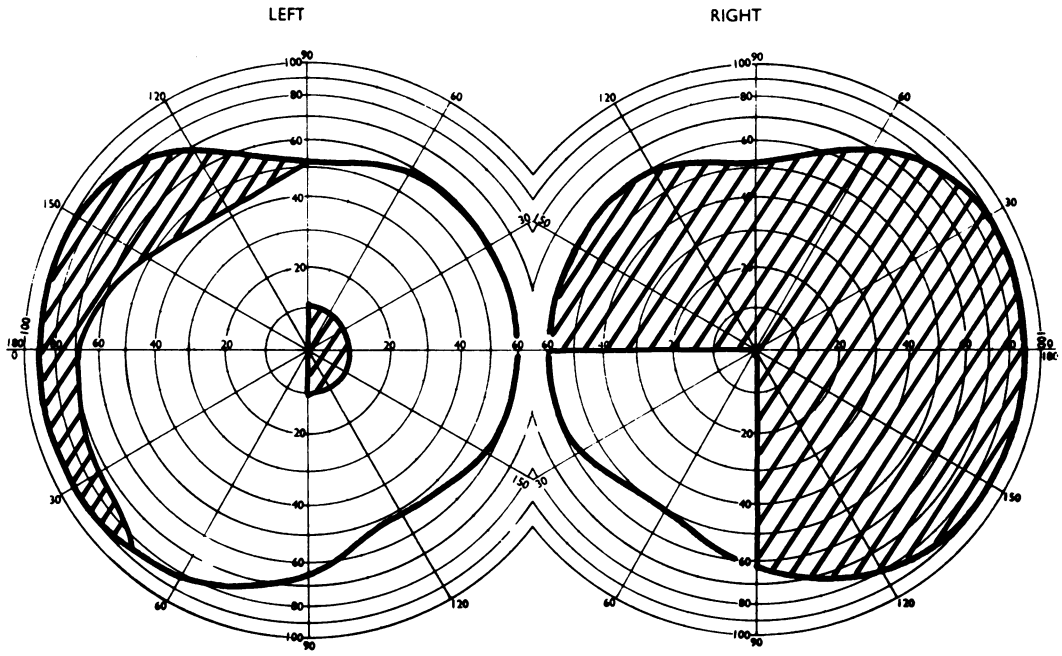

Fig 11 *Visual field charts to show retention of lower nasal field in right eye, and peripheral temporal loss with nasal paracentral scotoma in left eye. (Hatching = absolute loss)*

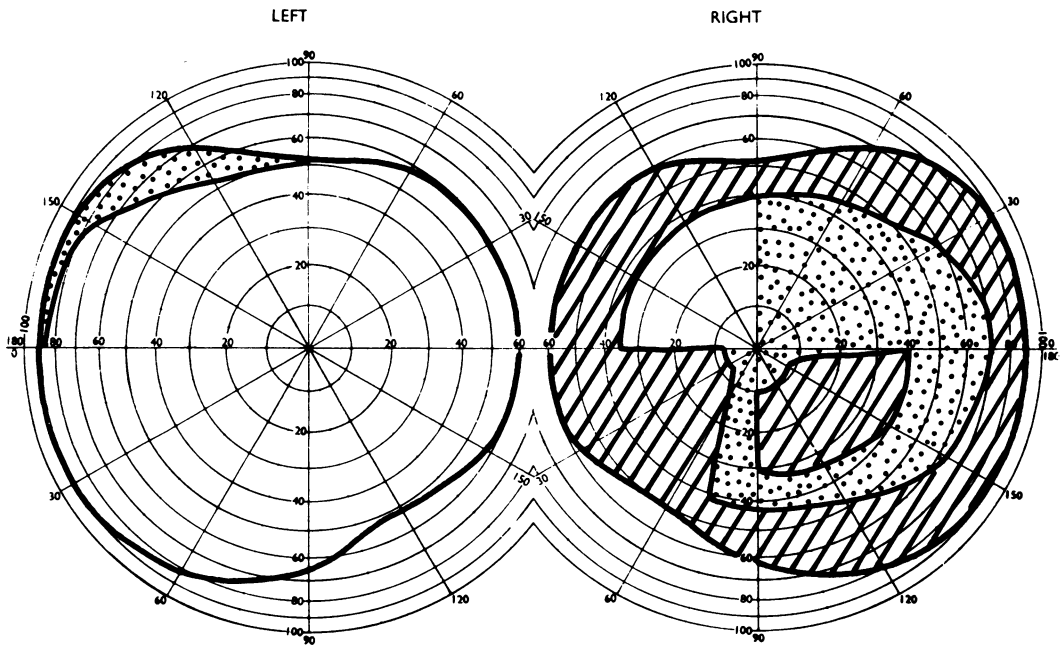

Fig 12 *Visual field charts to show marked absolute and relative loss in visual field of right eye and slight relative loss in upper temporal visual field of left eye. (Hatching = absolute loss, dotting = relative loss)*

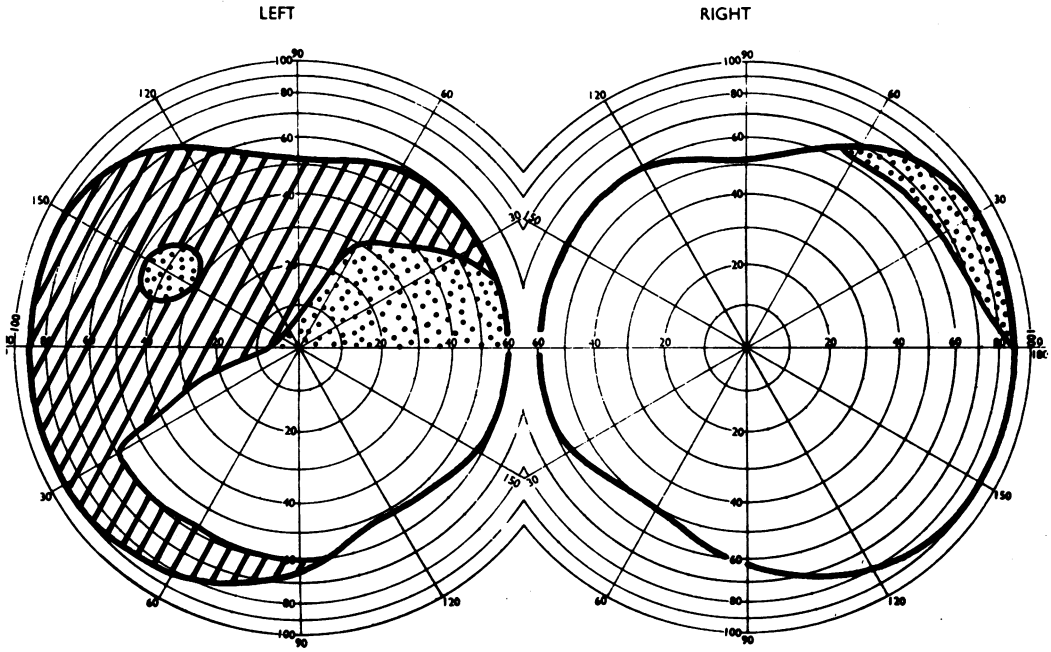

Fig 13 *Visual field charts to show marked absolute and relative loss of left visual field with sparing of lower part, particularly lower nasal, and slight relative loss in upper temporal part of right eye. (Hatching = absolute loss; dotting = relative loss)*

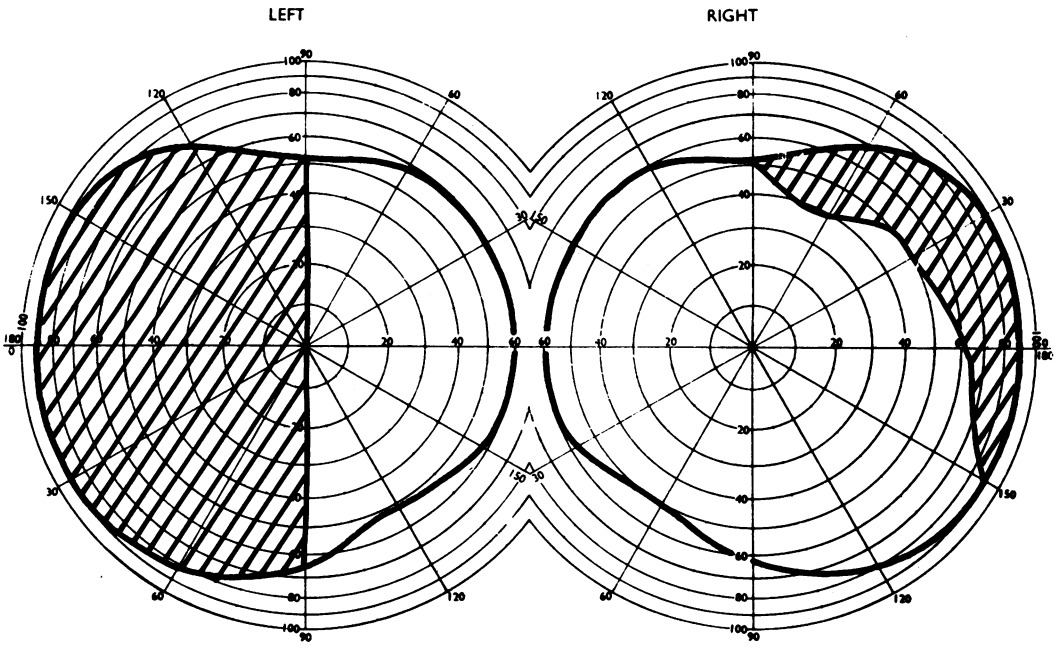

Fig 14 *Visual field charts to show complete temporal hemianopia of left eye and partial temporal hemianopia of right eye. (Hatching = absolute loss)*

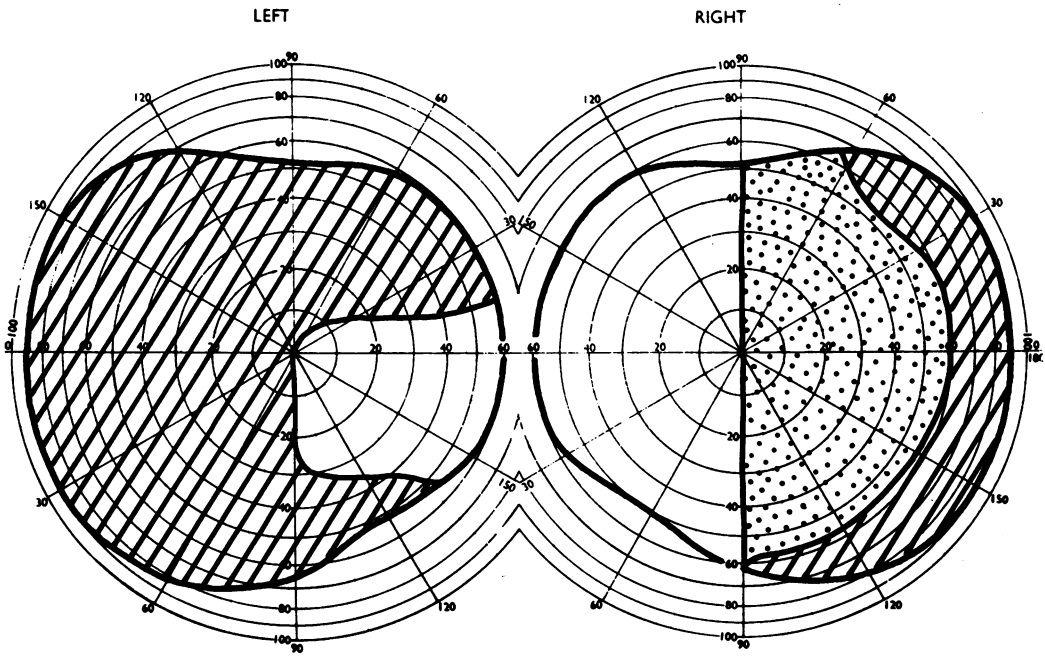

Fig 15 *Visual field charts to show bitemporal hemianopia (relative to some extent in right eye) with partial involvement of nasal field in left eye. (Hatching = absolute loss; dotting = relative loss)*

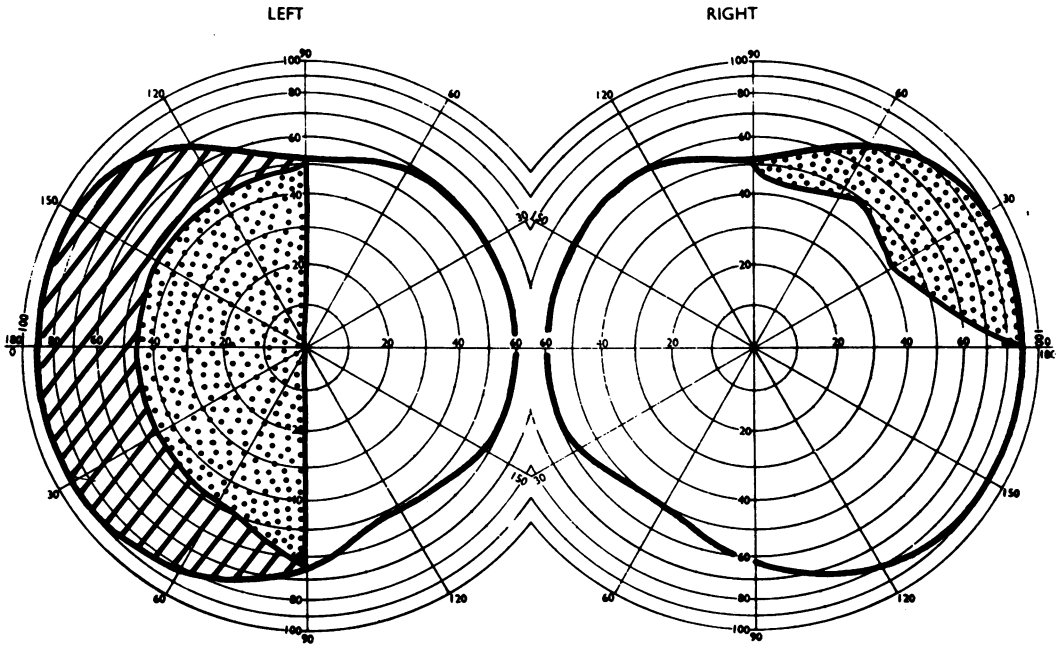

Fig 16 *Visual field charts to show relative peripheral loss in upper temporal part of right eye, and absolute and relative temporal hemianopia of left eye. (Hatching = absolute loss; dotting = relative loss)*

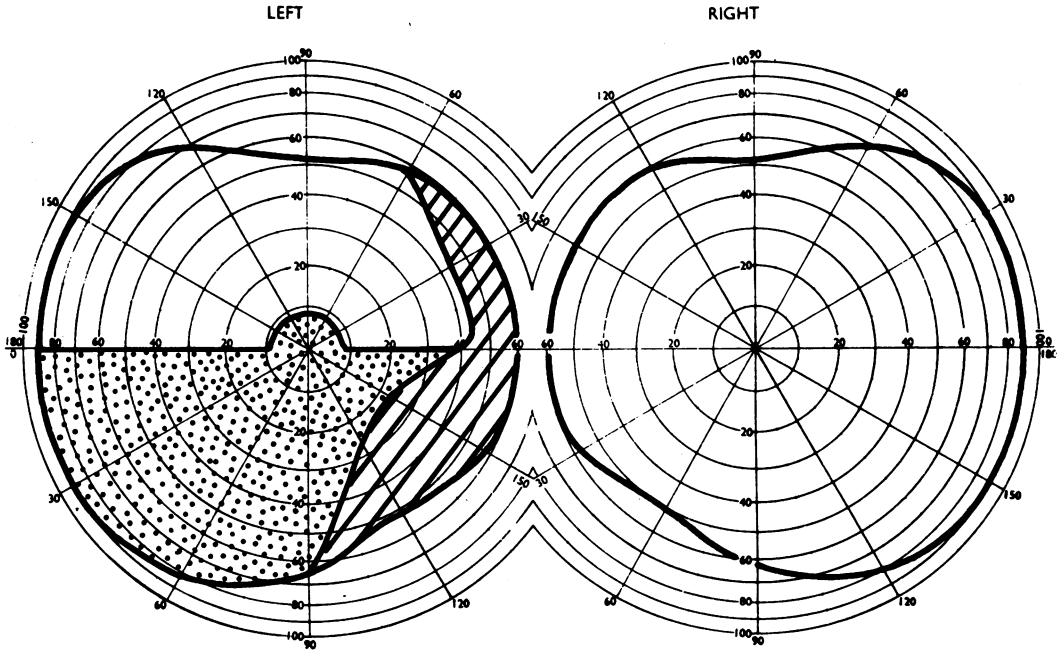

Fig 17 *Visual field charts to show relative and absolute loss of lower half of left visual field with relative central scotoma and with some absolute loss of peripheral part of nasal field; right visual field normal. (Hatching = absolute loss; dotting = relative loss)*

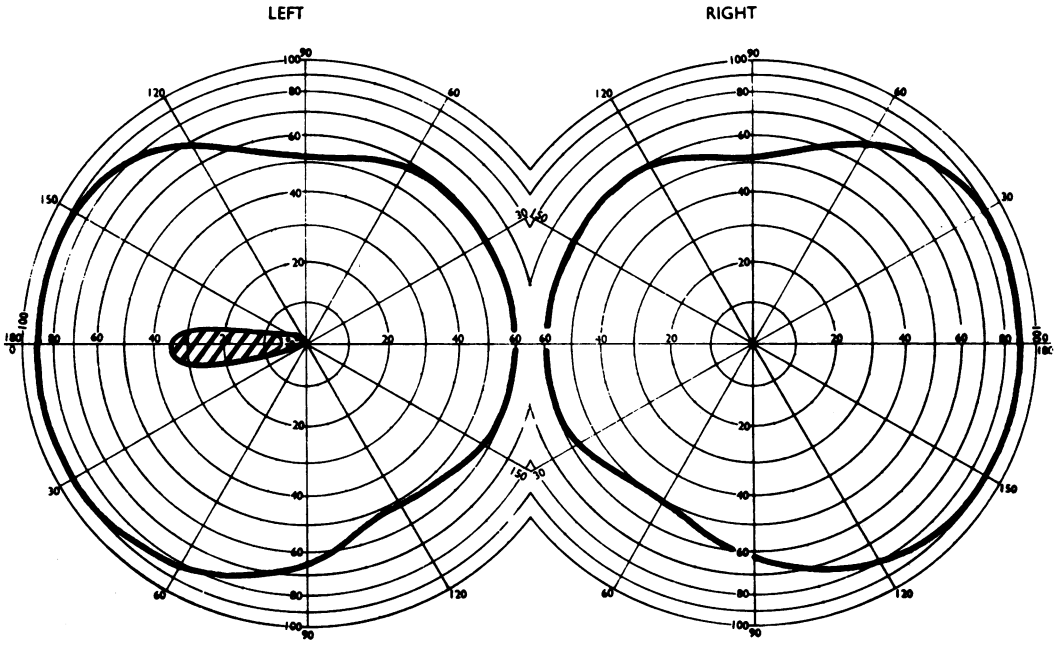

Fig 18 *Visual field charts to show centrocæcal scotoma (absolute and relative) in left visual field; right visual field normal. (Hatching = absolute loss; dotting = relative loss)*

one eye except in the lower nasal region and to some extent in the lower temporal region, and only a slight involvement of the upper temporal periphery in the other eye (Fig 13); this chart shows the retention of a small area of relative sparing in the upper temporal quadrant in the left eye.

In 2 cases there was a bitemporal hemianopia which was complete in one eye and partial in the other (Fig 14), and in one of these patients (Fig 15) there was an involvement also of the nasal field (upper more than lower) in the eye with the complete loss of the temporal part of the field. In 2 cases there was a temporal hemianopia in one eye with a partial loss of the upper temporal peripheral part of the visual field of the other eye (Fig 16). In one case there was a partial loss of the temporal part of the peripheral visual field with a paracentral scotoma in the temporal region in one eye, and an involvement of the upper part of the visual field (temporal more than nasal) with a paracentral scotoma in the temporal region in the other eye. In one case (Fig 17) there was a relative loss of the lower half of the visual field of one eye with a partial central scotoma, and an absence of any visual disturbance in the other eye. In one case there was a scotoma in the centrocæcal region of one eye and an absence of any visual disturbance in the other eye (Fig 18). In one case there was a partial defect of the temporal part of the visual field (upper more than lower) in one eye but the other eye could not be assessed because it was nearly blind as a result of an injury in childhood.

Ocular motility disorders: In 3 cases an exotropia developed because of the blindness of one eye due to complete optic atrophy; in another case there was an exotropia but this was not related to the meningioma, occurring in an eye which was almost blind as the result of a childhood injury.

In one case a horizontal form of diplopia occurred one year before the recognition of the meningioma, but this only persisted for four weeks, so that it may not have been related to the meningioma; although over a period of observation of several years no other abnormality was found to account for the diplopia.

In 2 cases there was a form of nonparetic diplopia, so that an exophoria which had previously been controlled adequately by the fusional vergence mechanism failed to remain controlled because of the reduction in the binocular field, as discussed earlier.

In one case there was a loss of elevation and abduction of one eye, but there was also a well marked forwards and downwards proptosis of this eye, so that a considerable part of the restricted movement may have been of a mechanical nature; the lesion was an extensive basal meningioma which arose from the sphenoidal ridge with involvement of the cavernous sinus.

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Surgical Management of Chiasmal Compression

There are many different lesions which may cause chiasmal compression: among the more uncommon conditions are metastatic carcinoma, optic nerve glioma and chordoma. The common compressing lesions are: pituitary adenomas; suprasellar meningiomas; craniopharygiomas; vascular lesions – aneurysms.

Pituitary Adenomas
These are by far the commonest tumours causing chiasmal compression, and of these the chromophobe adenoma is by far the most common. Eosinophil adenomas occur much less frequently and basophil adnomas virtually never produce compression of the optic chiasm.

Chromophobe adenomas: These produce typically a bitemporal hemianopia, with visual impairment and optic atrophy. The involvement of the eyes may be asymmetrical and in some cases only one eye may be involved. On occasions the field involvement is quite atypical: a binasal inferior quadrant field defect was present in one patient.

In addition to the field defect, these patients usually have some endocrine disturbance. Amenorrhœa in women may precede by many years the onset of visual disturbance, and impotence may be present in men. The skin is fine, pale and waxy, and the hair fine, thin, and of scanty growth. In some patients there is deficient suprarenal function giving weakness, apathy and tiredness, and deficient thyroid function giving myxœdema, both due to impaired function of the pituitary. More recently it has been shown that these tumours may secrete an excess of prolactin, which is possibly responsible for the amenorrhœa and impotence, and this finding has led to the earlier recognition of some tumours before signs of chiasmal compression have occurred. The course is usually steadily progressive, but sudden