PATTERNS OF VISUAL FAILURE WITH PITUITARY TUMOURS*†
CLINICAL AND RADIOLOGICAL CORRELATIONS
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IMPAIRMENT of vision in one or both eyes is a common sequel to a suprasellar extension of a pituitary tumour. It is widely held that this impairment takes the form of a bitemporal hemianopia in which the initial defect is a progressive loss in the periphery of the upper temporal quadrants, and moreover that this defect can be readily detected by perimetry. Yet our experience at a neurosurgical unit has suggested that many patients may seek advice repeatedly over many months or even years, from opticians, ophthalmologists, and neurologists, before the correct diagnosis is made. A major reason for this is that often the initial visual defect is not an impairment of the peripheral quadrants but a scotomatous defect in the temporal hemicentral fields.

Material
In order to ascertain the incidence and significance of the differing patterns of visual field disturbance a retrospective study has been made of fifty patients with chromophobe pituitary adenomata causing visual field defects, who were operated upon at the Guy's-Maudsley Neurosurgical Unit between 1953 and 1964. An attempt was also made to correlate these patterns with certain clinical features, such as the length of history, the presence or absence of optic atrophy, and the radiological findings. By these means we have sought to determine:

(1) The anatomical mechanisms responsible for the various patterns of visual defect;
(2) The prognosis of these various patterns after surgical operation;
(3) Criteria for earlier diagnosis.

Only patients with field defects were included, but two of them had also unilateral paresis of oculomotor muscles. The sex distribution was roughly equal (24 men, 26 women), and the age range at operation was from 24 years to 70 years (average 51). The subjective duration of visual deterioration ranged from less than a week to as long as 20 and 50 years in two patients. The average length of history, when these two last-mentioned patients are excluded, was 2 years.

Quantitative perimetric examination was performed in 49 cases before operation. One patient (Case 29) was too ill to co-operate and in fact died in coma shortly after operation, and there was one other post-operative death (Case 35). Both distance (Snellen) and reading (Jaeger) visual acuity was assessed in 43 of these 49 cases, while in the others only the reading acuity was recorded. Of the surviving patients, 45 have undergone at least one quantitative perimetric examination since the operation, whilst in the remaining three the findings on simple clinical confrontation were recorded. Although all patients had pre-operative x-ray examination of the skull, the films were

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available for study in only forty cases, the remainder having been lost or expatriated. Again, whilst 41 patients underwent pre-operative pneumo-encephalography, the films of only 36 were available for personal scrutiny. All were submitted to subtotal or intracapsular removal of the pituitary tumour via a subfrontal approach to relieve optic pathway compression as completely as possible. Postoperative radiotherapy was given in 24 cases with the aim of suppressing any residual tumour tissue.

The 48 surviving patients have since been followed up for periods of between 1 and 10 years. In the Tables the postoperative visual result has been taken as the best improvement obtained up to 3 years after operation. Five patients subsequently had recurrent visual deterioration some months or years after an apparently successful operation, none of them having had postoperative irradiation. These five were operated upon a second time and in one instance a third time, all with ultimate visual improvement. In all five the tumour was recrudescent. These cases of relapse are distinguished in certain of the figures by a symbol. Another patient (Case 50), following surgery and radiotherapy for a very large tumour, with initial dramatic visual restoration, later suffered a slight visual relapse but had no radiographic evidence of any suprasellar recurrence.

Patterns of Visual Failure

Field defects due to tumorous compression of the optic nerves, chiasm, or tract are dynamic phenomena. Although it is often possible to classify them with reasonable certainty on a single perimetric assessment, sometimes one has to take into account the manner in which the field defects regress after operation. Traquair (1938), for instance, stated that “after a successful operation the fields retrace the stages through which they originally developed”, a surmise that Falconer (1949) also confirmed.

By adopting this principle, we were able to distinguish in our fifty patients three clear groups of visual field disturbance:

1. **Bitemporal hemicentral scotomatous hemianopia (BHS)**
   24 patients (48 per cent.) showed an essentially scotomatous hemianopia (Figs 1a and 1b, overleaf). There were ten men and fourteen women; ages 24 to 67 years (average 52).

2. **Classical bitemporal hemianopia (BTH)**
   Eighteen patients (36 per cent.) showed a classical peripheral hemianopia (Figs 2a and 2b, overleaf). There were eleven men and seven women; ages 24 to 67 years (average 52).

3. **Atypical field defects (AFD)**
   Eight patients (16 per cent.) showed defects not classifiable in the first two groups, viz. homonymous hemianopia (3); unilateral central scotoma (2); complex hemianopic defects with large scotoma (3). There were three men and five women; ages 24 to 70 years (average 54).

The correlations which exist between each group and certain clinical and radiological characteristics will now be described.

Length of History and Mode of Progression of Symptoms

The history of the onset and course of visual failure in optic pathway compression is notoriously protean (Table I, overleaf). Some patients first notice a gross defect such as uniocular near-blindness suddenly but quite fortuitously, though it must have been present for weeks, months, or years beforehand (termed “spurious abrupt onset” in Table I). Others describe an insidious progressive deterioration. There may be intervals in which the visual decline spontaneously slows or arrests. There may be periods of rapid or urgent progression of visual failure. Sometimes a genuine abrupt onset of visual failure is due to acute chiasmal compression as in so-called “pituitary apoplexy”.

VISUAL FAILURE WITH PITUITARY TUMOURS
Fig. 1a.—Pre-operative visual fields of Case 3 on March 14, 1954.

Fig. 1b.—Postoperative visual fields of Case 3 on March 23, 1954.
VISUAL FAILURE WITH PITUITARY TUMOURS

PREOPERATIVE

Fig. 2a.—Pre-operative visual fields of Case 26 on May 3, 1951.

11 WEEKS POSTOPERATIVE

Fig. 2b.—Postoperative visual fields of Case 26 on July 27, 1951.
Table I shows that a steady progression is the commonest mode (50 per cent.) and has a comparable incidence in the three visual defect groups. Rapid or urgent progression is the next most common mode (27 per cent.), again occurring in all three visual defect groups but particularly often with scotomatous defects (8 cases out of 13).

**Table I**

**PROGRESSION OF VISUAL FAILURE**

<table>
<thead>
<tr>
<th></th>
<th>Steady Progression</th>
<th>Rapid Progression</th>
<th>Intermittent Progression</th>
<th>No Progression</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHS</td>
<td>□ □ □ □</td>
<td>□ □ □ □</td>
<td>□ □ □ □ □</td>
<td>□ □ □ □ □</td>
<td>23</td>
</tr>
<tr>
<td>BTH</td>
<td>□ □ □ □ □</td>
<td>□ □ □ □ □</td>
<td>□ □ □ □ □</td>
<td>□ □ □ □ □</td>
<td>17</td>
</tr>
<tr>
<td>AFD</td>
<td>□ □ □ □ □ □ □</td>
<td>□ □ □ □ □ □ □ □</td>
<td>□ □ □ □ □ □</td>
<td>□ □ □ □ □ □ □</td>
<td>8</td>
</tr>
<tr>
<td>Totals</td>
<td>24</td>
<td>13</td>
<td>6</td>
<td>5</td>
<td>48</td>
</tr>
</tbody>
</table>

□ Optic Atrophy absent  * Genuine abrupt onset  ○ Optic Atrophy probable  + Spurious abrupt onset  ● Optic Atrophy present

Two cases omitted because of incomplete data

**Incidence of Optic Atrophy**

Unequivocal pallor of one optic disc or both was noted in 28 of the fifty patients (56 per cent.). Fig. 3 shows that half or more of the cases in each of our three subgroups had atrophic optic discs and, equally important, an appreciable number had normal discs. Optic atrophy may be present even where the subjective duration of visual failure is short but becomes increasingly frequent the longer the history. This tendency is especially obvious in the scotomatous group, where there is a heavy loading of patients whose visual symptoms have lasted longer than either the average period for the series (2 years) or the average period for the group (16 months). Fig. 3 (opposite) also shows that optic atrophy may not be present even when visual symptoms have lasted longer than 6 months to 2 years. However, in all patients with chiasmal compression, whether the defects were peripheral or scotomatous, optic atrophy had invariably appeared after 2 years of visual symptoms. The two patients in the atypical group still without atrophy after 2 years of visual symptoms both had homonymous hemianopias due to optic tract involvement. It will also be noted from Table I that patients with optic atrophy are distributed remarkably evenly among all four modes of symptomatic progressions and all three groups of field defects.
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Radiological Findings

(1) Sellar Enlargement
The lateral skull radiographs of forty patients were scrutinized (Table II) and the degree of sellar enlargement arbitrarily assessed on a scale from nil to 4 (gross enlargement). 38 cases (95 per cent.) had sellar enlargement somewhere between these two extremes, and in eleven (27·5 per cent.) the enlargement was asymmetrical. There were no obvious differences in the distribution of sellar enlargement among the three visual defect groups.

<table>
<thead>
<tr>
<th>Scale of Enlargement</th>
<th>Nil</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHS</td>
<td>0</td>
<td>6</td>
<td>4</td>
<td>7</td>
<td>2</td>
<td>19</td>
</tr>
<tr>
<td>BTH</td>
<td>0</td>
<td>3</td>
<td>6</td>
<td>4</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>AFD</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Totals</td>
<td>0</td>
<td>11</td>
<td>12</td>
<td>15</td>
<td>2</td>
<td>40</td>
</tr>
</tbody>
</table>

(2) Involvement of Anterior Clinoid Processes
In the same forty patients the degree of anterior clinoid destruction, rarefaction, shortening, or sharpening was arbitrarily assessed on a similar scale from nil to 4 (Table III, overleaf).

Here each symmetrically involved pair of anterior clinoid processes is denoted by a circle, but where involvement was not symmetrical each process is represented by a semicircle. It is clear that in the scotomatous group the anterior clinoid processes are relatively spared.
TABLE III
DESTRUCTION OF ANTERIOR CLINOID PROCESSES

<table>
<thead>
<tr>
<th></th>
<th>Nil</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHS</td>
<td>O O O O</td>
<td>O O O O</td>
<td>O O</td>
<td>D</td>
<td></td>
<td>19</td>
</tr>
<tr>
<td>BTH</td>
<td>D D D D</td>
<td>O O D D</td>
<td>O O D</td>
<td></td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>AFD</td>
<td>O D</td>
<td>O O D D</td>
<td>D D D</td>
<td></td>
<td></td>
<td>8</td>
</tr>
</tbody>
</table>

O Both  D Right  D Left

In the classical group, however, involvement is not only heavier but also much less symmetrical, while in no case in this group were the anterior clinoid processes completely spared.

(3) Involvement of Dorsum Sellae and Posterior Clinoid Processes

The degree of destruction, rarefaction, or fragmentation of the dorsum sellae and posterior clinoid processes was arbitrarily assessed on a similar scale from nil to 4. Table IV shows that in no patient did the dorsum sellae escape completely, while severe grades of destruction were common among all groups of field defect. Asymmetrical involvement was not often observed. The brunt of an expanding intrasellar tumour evidently tends to bear upon the posterior part of the sella turcica.

TABLE IV
DESTRUCTION OF DORSUM SELLAE AND POSTERIOR CLINOID PROCESSES

<table>
<thead>
<tr>
<th></th>
<th>Nil</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHS</td>
<td>D</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>19</td>
</tr>
<tr>
<td>BTH</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>13</td>
</tr>
<tr>
<td>AFD</td>
<td>D</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td></td>
<td>8</td>
</tr>
</tbody>
</table>

O Both  D Right  D Left

(4) Backward Tilt of the Dorsum Sellae

Backward tilt of the dorsum sellae was arbitrarily assessed on a scale from 1 (slight) to 4 (gross). Table V indicates a slight tendency for the greater degrees of backward tilt to occur in classical and atypical field defect groups. Nevertheless, just over half the patients showed only slight backward tilt.

TABLE V
BACKWARD TILT OF DORSUM SELLAE

<table>
<thead>
<tr>
<th>Scale of Tilt</th>
<th>Nil</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>BHS</td>
<td>0</td>
<td>11</td>
<td>7</td>
<td>0</td>
<td>1</td>
<td>19</td>
</tr>
<tr>
<td>BTH</td>
<td>0</td>
<td>6</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>AFD</td>
<td>0</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Totals</td>
<td>0</td>
<td>21</td>
<td>11</td>
<td>5</td>
<td>3</td>
<td>40</td>
</tr>
</tbody>
</table>
(5) Relationship of the Summit of the Tumour to the Third Ventricle and Optic Chiasm

One of us (Falconer, 1949) had gained an impression that in patients with scotomatous field defects the summit of the suprasellar tumour as judged at pre-operative air encephalography was characteristically behind the presumptive position of the optic chiasm, whereas in patients with classical field defects the tumour summit typically lay in front of the chiasm. Our present study supports this impression (Table VI). Tracings of the pre-operative air studies were made from the relevant lateral projections, including hanging-head lateral autotomographs in the majority of cases, to show the configuration of the sella turcica and front end of the third ventricle and the distribution of air in the chiasmatic and interpeduncular cisterns (Schechter and de Gutiérrez-Mahoney, 1962). The radiological extent of the suprasellar mass was then marked out on the tracing. Finally, the rostral border of the optic nerves and chiasm was represented by a line drawn from the floor of the optic recess to the superior face of the better-preserved anterior clinoid process (Walker, 1962). This last expedient was regarded as an adequate approximation to actuality in those cases (thirty in number) in which the anterior recesses of the third ventricle were still identifiable. When the front of the third ventricle was grossly displaced or deformed the probable location of optic nerves and chiasm could only be surmised (six cases). Where the summit of the tumour lay mainly in front of the line described the assessment was “Pre-chiasmal”, and where it lay behind, “Post-chiasmal”. In five instances, all of them patients with atypical field defects and very large tumours, the relationship of summit to chiasm could not be made out, and these are assessed as “Other”.

<table>
<thead>
<tr>
<th>TABLE VI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Radiological Relationship of Suprasellar Mass to Chiasm</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>BHS</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>BTH</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>AFD</td>
</tr>
</tbody>
</table>

Denotes equivocal assessment

The location of the tumour summit was usually unequivocal (e.g. Cases 2 and 43, illustrated in Figs 4 and 5, overleaf), but in a few the decision between pre-chiasmal and post-chiasmal was difficult (e.g. Case 49, illustrated in Fig. 6, overleaf). Table VI shows that in fifteen out of eighteen cases with scotomatous defects the tumour summit was post-chiasmal, unequivocally so in thirteen of them (72 per cent.). The same Table also shows that, of ten
patients with classical defects, six had a pre-chiasmal summit, five of them unequivocally, while in the remaining four patients the tumour summit was post-chiasmal and equivocally so in two of them.

Thus, in patients with bitemporal hemicentral scotomatous hemianopia, the suprasellar mass characteristically impinges behind the optic chiasm, whereas in those with classical bitemporal hemianopia it is characteristically in front.

Quality of Visual Restoration after Treatment

The visual function before and after treatment was scored by a simple numerical method which we have termed "graded visual status" (Table VII).

<table>
<thead>
<tr>
<th>Postoperative Graded Visual Status</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>&quot;A&quot;</td>
</tr>
<tr>
<td>6-10.5</td>
<td>&quot;B&quot;</td>
</tr>
<tr>
<td>1.5-6</td>
<td>&quot;C&quot;</td>
</tr>
<tr>
<td>0</td>
<td>&quot;D&quot;</td>
</tr>
</tbody>
</table>

Complete recovery within 1 month was reckoned as "A plus" result. Category "D" contains three patients whose vision was worse after operation.

Figs 7, 8, and 9 (opposite) show the quality of visual restoration as measured by the graded visual status attained by each patient in the three basic groups of field defect. The two operative deaths are also shown but are excluded from subsequent discussion.
94 per cent. of our patients were improved visually to some degree after operation, a quarter regaining full visual function in both eyes, a half gaining useful but incomplete visual restoration, and a quarter gaining lesser degrees of recovery. Three patients surviving operation had worse vision than before.

The best results occurred in the scotomatous group of hemianopias, whose seven "A plus" results and one "A" result represent 72% of all the complete visual recoveries. The least good results were in the small group of atypical field defects, which contains no "A" results and two of the three "D" results. Both the major groups (scotomatous and classical defects) contain comparable large proportions of partial visual recoveries (63 and 66 per cent. respectively).
Discussion

(1) Incidence of Bitemporal Hemicentral Scotomatous Hemianopia

The importance or even the occurrence of scotomatous defects as a form of presentation of chiasmal compression by pituitary tumours receives little emphasis either in undergraduate texts or in publications intended for postgraduate study (Handfield-Jones and Porritt, 1957; Blackburn and Lawrie, 1958; Jennett, 1964; Houston, Joiner, and Trounce, 1966).

Chamlin, Davidoff, and Feiring (1955) found bitemporal hemianopic defects in 96 per cent. of 109 cases of proven or presumptive chromophobe adenoma, but while stating that field changes occur first in the small central isopters (e.g. 1/2000) concluded that central hemianopic defects, whether unilateral or bilateral, were very rare except with peripheral hemianopic defects, although the latter might be very inconspicuous. Traquair (1938) had earlier described the scotomatous type of hemianopia without giving a precise incidence, but indicated that it could merge with the classical type as it progresses. Clarke, Knighton, and Bebin (1963) reviewing 75 cases of chromophobe adenoma found a visual field defect in 81 per cent. and remarked that “the characteristic field defect is an asymmetrical bitemporal hemianopsia” without mentioning the occurrence of scotomatous hemianopia. Rucker (1957) found “ordinary bitemporal hemianopsia” the characteristic field defect but also described patients with bitemporal hemicentral scotomatous defects, bilateral central scotomata, and incongruous homonomous hemianopias. Clover and Lyle (1961), reporting 100 cases of proven pituitary adenoma (not all of them chromophobe adenomata), found clear-cut bitemporal hemianopic defects in 94, the peripheral fields being affected in 79 (i.e. classical bitemporal hemianopsia) and unaffected in fifteen (i.e. hemicentral scotomatous hemianopsia.)

Our series contains a high proportion of scotomatous hemianopias (48 per cent.) partly because in classifying the final preoperative field defect we have also taken into account the pattern of postoperative regression of the defect. All our patients had asymmetrical field defects.

(2) Anatomical Considerations

The interruption of chiasmal function in compression by a tumour is probably due much more to interference with the blood supply of the chiasm than to mere pressure or displacement (Traquair, 1938; Rucker, 1957). Nevertheless Rucker felt that the early visual disturbance, before mechanical factors had been overshadowed by vascular ones, had reliable localizing value. He quoted Wilbrand’s classical concept (Wilbrand, 1926) of the optic chiasm as comprising three layers. The upper layer contains mainly fibres from the upper retinal quadrants (i.e. subserving the lower visual quadrants) with non-crossing outnumbering crossing fibres. The middle layer contains crossing fibres from the upper nasal retinal quadrants in roughly equal proportion to non-crossing fibres from the lower temporal quadrants. The lower layer contains mainly crossing fibres from the lower nasal retinal quadrants (i.e. subserving the upper temporal visual quadrants) which loop forwards into the opposite optic nerve before turning backwards into the optic tract. The function of these lowest fibres is commonly the first to be interrupted by a tumour compressing the antero-inferior face of the chiasm, resulting in a peripheral bitemporal upper quadrantic hemianopia. A few non-crossing fibres from the inferior temporal retinal quadrants also run in the third or lower layer. The decussating central retinal fibres cross
in the posterior part of the chiasm. According to Traquair, scotomatous hemianopia is due to compression of these decussating fibres of the papillo-macular bundles, which he describes as forming the entire substance of the posterior edge of the chiasm. Hughes (1954) has pointed out that the point of chiasmal compression depends not only on the position of the lesion but also on whether the chiasm is pre-fixed or post-fixed. Our own material supports these views.

(3) Radiological Considerations

The size and shape of the normal sella turcica varies greatly (El Sayed Mahmoud, 1958). Although a chromophobe adenoma can exist within a very small pituitary fossa (Falconer, 1946; Jefferson, 1966), some degree of sellar enlargement is more typical. According to Lewtas (1966), asymmetrical expansion of the sella, causing a double contour, is the most common finding in lateral skull radiographs, while the classical "ballooned" sella is the least common. Table II confirms that gross sellar expansion is indeed exceptional and that minor degrees of enlargement are more usual. Asymmetry of the floor of the sella turcica was observed in eleven of our forty cases (27-5 per cent.).

Pribram and Swann (1960) found that blunting or obliteration of the optic and infundibular recesses of the third ventricle, as shown by air encephalography, was the first radiological sign of a suprasellar extension of a pituitary tumour. Lewtas (1966) found that, using conventional techniques, the front of the third ventricle was shown in only half the patients undergoing encephalography, but he greatly improved the demonstration by lateral tomography in the brow-up, over-extended position of the head. Where special apparatus permitting a vertical arc of tube swing is lacking, Lewtas advocates careful autotomography in the same head position (Schechter and de Gutiérrez-Mahoney, 1962). It is, in fact, this latter technique that has usually been employed in those of our patients who underwent air encephalography, in order to eliminate obscuration of the shape of the suprasellar extension by air in the chiasmatic and interpeduncular cisterns.

The pneumographic findings set out in Table VI and summarized earlier support Traquair’s thesis that scotomatous hemianopias are characteristic of tumorous compression of the posterior edge of the optic chiasm.

(4) Significance of Optic Atrophy

There is general agreement on the incidence of optic atrophy in tumorous chiasmal compression. The proportion in our series of 56 per cent. compares with 56 and 49 per cent. respectively given by Clover and Lyle (1961) and by Clarke and others (1963). Confusion may occur about what is meant by “optic atrophy”. In clinical practice the term is best reserved to describe excessive pallor of an optic disc as viewed through the ophthalmoscope rather than the wasted, threadlike, or ribbonlike appearance seen when an optic nerve long subjected to compression or ischaemia is exposed at operation or necropsy. In this paper we have employed the term “optic atrophy” only in the former sense.

Chamlin, Davidoff, and Feiring (1955) have stressed the difficulty of diagnosis of optic atrophy which may be due to observer variation, but solved the problem by terming pallor of a disc “pathological” if a field defect was also present and “physiological” if no field defect was present. They concluded that optic atrophy was at best merely a corroborative sign of chiasmal compression. Although it is impossible to be sure at what stage optic atrophy develops in chiasmal compression, it is probably not an early sign.
The prognostic significance of optic atrophy is not as gloomy as is sometimes thought. Rucker (1957) considered that, in cases with slight pallor but relatively great visual loss, improvement after surgery was to be expected, and that even in cases with distinct pallor, improvement might sometimes unexpectedly occur. Our own findings in Figs 7, 8, and 9 confirm this. Six of the eleven perfect visual recoveries were seen in patients with definite or probable optic atrophy, whilst of 21 good quality but incomplete visual recoveries, definite or probable optic atrophy was present in sixteen. In the scotomatous group optic atrophy was present in six of the eight patients who made perfect visual recoveries, whereas in the classical group none of the three patients whose vision returned to normal had optic atrophy. The three worst visual results were seen in patients with definite optic atrophy. Again, the absence of optic atrophy in a particular case does not by itself seem necessarily to imply a good visual prognosis. In our series three out of sixteen patients with poor quality postoperative visual status had no optic atrophy, although two of these were suffering from optic tract involvement rather than chiasmal compression.

Thus the presence of optic atrophy does not preclude the possibility of full and rapid recovery of vision in cases where the field defect is a bitemporal hemiscentral scotomatous hemianopia. On the other hand a perfect recovery is not to be expected if optic atrophy is present in cases of classical bitemporal hemianopia, although good partial recovery is still likely.

(5) Mode of Progression of Visual Failure

Traquair (1938) stated that the scotomatous type of field defect was characteristic of activity of the lesion and the non-s Scotomatous type of a slowly-growing or stationary condition. Table I indicates that our series of cases broadly supports this opinion. Only three out of seventeen cases (17 per cent.) of classical bitemporal hemianopia described a rapid or urgent progression of visual failure, whereas eight out of 23 cases (35 per cent.) of scotomatous hemianopia did so. Conversely, while only eleven of 23 cases (48 per cent.) of scotomatous hemianopia described either a stationary defect or an insidiously progressive one, thirteen of seventeen cases (76 per cent.) of classical hemianopia did so. However, no clear correlation exists between the mode of progression of visual failure and subsequent visual improvement.

(6) Subjective Duration of Visual Failure

When speed and completeness of visual recovery were studied, six of the seven “A plus” results followed a history of visual failure of between 6 months and 2 years. Two “A” results occurred in patients with histories of less than 1 month. Only one patient (a case of classical bitemporal hemianopia) regained full visual function after a history exceeding 2 years. All three patients with deterioration of vision after operation had histories greatly in excess of 2 years.

It would seem therefore that good results can be expected only if subjective visual failure has been of less than 2 years’ duration.

(7) Quality of Diagnosis before Definite Referral

Long delays were often noted between the patient’s first seeking advice and his final definitive referral to a neurosurgeon. These delays were due to failure to detect or to realize the significance of a visual field defect and thus the time was often taken up with multiple refractions or other ocular treatments.
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We have grouped 46 of our patients for whom data are complete into three arbitrary categories of time of recognition of visual field defect.

(i) Recognized early, i.e. visual field defect recognized less than 6 months after subjective onset of visual failure. Seventeen cases (37 per cent. of the whole series) came into this category. The best score for early recognition was in the classical hemianopic group (eight cases: 48 per cent. of all cases recognized early). In this category the average interval between the onset of visual symptoms and surgery was 3.5 months.

(ii) Recognized late, i.e. visual field defect recognized more than 6 months after subjective onset of visual failure. 25 cases (54 per cent. of the whole series) came into this category. The score was worst in the scotomatous group (seventeen cases: 68 per cent. of all cases recognized late). In this category the average interval between the onset of visual symptoms and operation was 29 months.

(iii) Not recognized, i.e. visual field defect not detected until final neurosurgical referral of patient, or if detected not appreciated as an index of optic pathway compression. Four patients came into this category. Three of them had classical hemianopia and two of them had in fact never sought ophthalmological advice.

It is noteworthy that six of the seven “A plus” results, already referred to, occurred in patients whose visual field defects (all scotomatous) had been recognized “late”. The three worst results were all in patients whose field defects were either recognized late or not recognized.

(8) Errors in Early Diagnosis and Treatment

Clover and Lyle (1961), in their review of 100 cases of proven pituitary tumour, found a record of the initial visual diagnosis in only thirty instances. Fourteen cases were diagnosed as “refractive error”. The other diagnoses included myopia, astigmatism, amblyopia, tobacco amblyopia, optic neuritis, glaucoma, “haemorrhage behind the eye”, migraine, and optic atrophy. Two patients were simply “reassured”.

Of our 49 patients whose dominant symptom had been deterioration of vision, 45 at some stage had sought advice from an optician or an ophthalmologist. No treatment was given to 26, and furthermore nine of them were not even advised to seek a second opinion. In one patient the initial diagnoses included “ocular imbalance” and “optic nerve haemorrhage”, whilst in another the diagnosis was “senile vascular disturbances”.

Nine of our patients underwent one or more refractions over periods averaging 20 months; one had eight consecutive pairs of spectacles during the year of his visual deterioration; he had well-preserved visual acuity and normal optic discs but a bitemporal hemi-central scotomatous hemianopia, and following pituitary surgery made a rapid and complete visual recovery.

One patient, domiciled in France, was given vitamin injections for 6 years before his gross visual failure and bilateral optic atrophy were recognized as being due to optic pathway compression. He had a large subfrontal extension of his pituitary tumour and his visual status unfortunately became worse after operation.

Another patient, having suffered from failing vision and headache for 2 years, underwent bilateral trephining for supposed glaucoma, but another 4 years elapsed before his field defect was recognized and his pituitary adenoma removed.

These facts suggest that correct early diagnosis in patients with optic chiasmal compression by a pituitary tumour is the exception rather than the rule, particularly if the visual failure takes the common form of a bitemporal hemi-central scotomatous hemianopia.
Problems of Early Diagnosis

Several ophthalmologists, neurologists, neurosurgeons have already drawn attention to the problems of early diagnosis of visual failure due to pituitary tumours. Clover and Lyle (1961), for instance, stressed that signs such as bitemporal hemianopia and optic atrophy may precede symptoms, and that the patient himself is often unaware of an advanced defect. They drew attention to "non-paretic diplopia" as an early symptom of gross bitemporal hemianopia due to loss or reduction of normal overlap of the two fields. They thought that, in a patient with a predominantly unilateral scotomatous hemianopia, the early complaint is of defective uniocular vision, but that patients with scotomatous defects involving the fixation point frequently complain of binocular visual defect (non-paretic diplopia).

Rucker (1957), an ophthalmologist, stressed that whenever a reduction of acuity could not be readily explained either by the patient's refractive error or by ophthalmoscopic examination, the visual fields should be carefully charted. Chamlin and others (1955) stressed the importance of tangent (Bjerrum) screen testing at 2 metres using white test objects. They routinely charted the 1/2000 isoptre on the screen as well as the 2/330 on the perimeter, and in their view the visual field examination is the most important single ophthalmological criterion for pituitary tumours. Walsh and Gass (1960) amplified their views by advocating also the use of red test objects. Jefferson (1966) emphasized the importance of tangent screen testing.

Quantitative perimetry, however, is a time-consuming test requiring patience and skill. In order to sort out those cases in which it should be performed, we advocate that as a simple clinical expedient every patient suspected of chiasmal or optic pathway compression should have the peripheral fields tested by simple confrontation using the examiner's fingers, and also the central fields by a white-headed pin. Those who show a suspected defect can then be submitted to more detailed testing on a Bjerrum screen.

Recently Buchanan and Gloster (1965) have developed an automatic recording apparatus (Globuck screen) for the rapid assessment of the central visual fields. It consists of a tangent screen used at 1 metre distance with a central red fixation light, 2 mm. diameter, surrounded at 5° intervals up to 25° by five circles of some 74 white lights, each 1 mm. diameter. All the lights are flashed in random order and the position of any light not perceived by the patient is marked on a scotoma chart by pressing a button. The test can be performed by a technician in a few minutes. Although it may not be as accurate as the Bjerrum screen in the hands of an experienced perimetrist, it should pick up the scotomatous defects which we have described.

This leads on to a consideration of the National Health Service (Supplementary Ophthalmic Services) (Nos 1 and 2) Regulations, 1961. More than 80 per cent. of the sight-testing in this country is done by ophthalmic opticians, and the testing of visual fields is not normally part of a refraction. Even so, some of our cases were tested by ophthalmological surgeons who missed the presence of a field defect. It is expected that an optician or ophthalmic surgeon testing sight under the Supplementary Ophthalmic Services will refer a patient back to his own doctor if he believes that some cause other than a refractive error is present.

The key to the problem of better early diagnosis is embodied in Lyle's dictum that "few ophthalmologists are sufficiently 'field-conscious.'" Visual field testing should be mandatory whenever a patient presents with failing vision and there is no obvious intra-ocular cause.
ILLUSTRATIVE CASE REPORT

CASE 3, A WOMAN AGED 38 YEARS AT OPERATION, was a housewife with normal and regular menstrual patterns, but childless and for about 5 years without libido. She began to suffer frontal headache and ocular discomfort, and in July, 1953, first noticed mistiness of vision. An ophthalmic surgeon in November, 1953, found her visual acuity in both eyes to be 6/6, the optic discs normal, and the peripheral fields full. Skull X rays however showed asymmetrical sellar enlargement suggestive of a pituitary tumour. A neurologist then reported that he could find no signs of organic disease of the nervous system and regarded the skull X-ray appearances as being within normal limits. He advised her to “neglect the eye symptoms”.

However, she continued to complain of misty vision and of dark areas in the right temporal field, together with increasing headache. In January, 1954, another ophthalmic surgeon (Mr. R. Pitts Crick) found her visual acuity to be 6/12 and demonstrated a central scotoma in each eye. She was referred with a diagnosis of chiasmal compression.

Examination.—She was an alert woman whose uncorrected visual acuity was 6/18, J2 in the left eye and 6/6, J2 in the right. The optic discs were normal. There was a large central temporal scotoma in the left field encroaching on the inferior nasal macular field, whilst in the right eye the blind spot was enlarged and the upper temporal central isopters were indented. The peripheral fields showed slight bitemporal constriction (Fig. 1a).

X-ray examination of the skull confirmed the slight sellar enlargement and also showed backward tilting of the dorsum sellae. Air encephalography showed blunting of the recesses of the front end of the third ventricle.

Operation.—On 15 March, 1954, a large rounded suprasellar mass was found, its summit lying just behind the chiasm, and containing a cavity occupied by 5 ml. dark altered blood. The walls of the cavity were smooth and collagenous with islands of brownish tumour tissue. The cyst wall was partially removed and showed tissue consistent with chromophobe adenoma.

Result.—The patient recovered uneventfully from the operation, her vision returning to normal within one week (Fig. 1b). At her last postal follow-up in December, 1965, she remained symptom-free.

Summary

Clinical and radiological findings have been reviewed in fifty patients with proved and surgically-treated chromophobe pituitary adenoma whose first or dominant symptom was visual deterioration due to optic pathway compression.

The commonest basic field defect was a bitemporal hemicentral scotomatous hemianopia (24 cases; 48 per cent.). Late recognition was common. Optic atrophy was present in over half the cases. The sella turcica was always enlarged but the anterior clinoid processes were relatively spared. The radiological summit of the tumour (as demonstrated by air encephalography) was usually behind the optic chiasm, consistent with predominant involvement of the decussating papillomacular fibres. Eight patients (one third) made complete visual recoveries, seven of them within one month of operation, and these complete recoveries included patients with optic atrophy and long histories. One patient was not improved by operation. The remainder had varying degrees of improvement short of full restoration.

The classical bitemporal hemianopia affecting peripheral fields was seen in eighteen cases (36 per cent.). Recognition tended to be earlier. Optic atrophy was present in over half the cases. The anterior clinoid processes typically showed asymmetrical destruction. The tumour summit was characteristically in front of the chiasm rather than behind it. Three patients, none of whom had optic atrophy, made full visual recoveries. Two with large tumours died postoperatively.
The remaining eight patients had miscellaneous field defects indicating optic nerve, optic tract, or composite optic pathway involvement. Optic atrophy was present in five cases. The tumour was often very large and bore no characteristic relation to the optic chiasm. No patient in this group regained full visual function, and two were worse after operation.

An important factor in the frequent failure of recognition of chiasmal compression in its early stages is that the field defect more commonly evolves as an asymmetrical bitemporal hemicentral scotomatous hemianopia which is overlooked if only the peripheral fields are examined. Patients with visual failure not obviously due to a local ocular cause should have detailed quantitative perimetric assessment. A quick preliminary expedient for "screening" the central fields of such patients is simple confrontation with a white-headed pin.

REFERENCES