The characteristic visual field defect in chronic simple glaucoma occurs as a result of damage to individual bundles of nerve fibres in the optic nerve head. Chronic simple glaucoma, while the most frequent cause, is not the only cause of nerve fibre bundle defects. The causes of such defects were divided by Harrington (1965) into those occurring at the disc, those resulting from lesion of the anterior nerve, and those due to lesions of the posterior nerve and chiasm (Table).

<table>
<thead>
<tr>
<th>Lesions at the disc</th>
<th>Lesions of the anterior nerve</th>
<th>Lesions in the posterior nerve and chiasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Juxtapapillary choroiditis</td>
<td>1. Ischaemic infarct and segmental atrophy in the optic nerve due to arterial occlusion</td>
<td>1. Meningioma at the optic foramen</td>
</tr>
<tr>
<td>3. Colobomata and pits of the optic nerve head</td>
<td>3. Cerebral arteritis</td>
<td>3. Pituitary adenoma</td>
</tr>
<tr>
<td>4. Drusen on the optic nerve</td>
<td>4. Retrobulbar neuritis</td>
<td>4. Opticochiasmatic arachnoiditis</td>
</tr>
<tr>
<td>5. Papilloedema with increased intracranial pressure</td>
<td>5. Electric shock</td>
<td></td>
</tr>
<tr>
<td>7. Papillitis</td>
<td></td>
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<tr>
<td>8. Retinal arterial plaque on the disc</td>
<td></td>
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<td>9. Papilloedema in malignant hypertension</td>
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<tr>
<td>10. Occlusion of central retinal artery</td>
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Knowledge of the visual field changes and their progression is essential so that perimetric techniques can:

1. Delineate the earliest characteristic nerve fibre bundle defects which indicate glaucomatous damage.

2. Plot the size and density of the defects, so that changes indicating progression can be recorded accurately and reproducibly.

Significance of Baring of the Blind Spot

Baring of the blind spot has been described as one of the early changes of the glaucomas (Traquair, 1931). In most normal individuals, there is a difference in the sensitivity of the retina above and below the disc. The portion of the visual field below the blind spot has a slightly lower differential threshold and is slightly more sensitive to light than the corresponding area above the blind spot. The profile of the field above the blind
The glaucomatous visual field

The glaucomatous visual field is also flatter than that below. Threshold stimuli can be used to bare the blind spot, usually above. Baring of the blind spot in many normal individuals can be produced with threshold targets (Fig. 1). Nerve fibre bundle defects also produce baring of the blind spot. While baring of the blind spot can be significant, it is not specific enough to be reliable for the early detection of a glaucomatous field defect. Baring of the blind spot to standard targets is even more likely to occur in the presence of miosis, ageing, and lens changes, factors which frequently accompany chronic glaucoma. Suprathreshold targets for the normal young eye may become threshold stimuli for the same eye at an older age when some of the changes have taken place. Baring of the blind spot to a 3/1,000 white target in a 30-year-old patient with 20/15 vision is likely to be due to a field defect, but the same finding in a 65-year-old person with 20/40 vision is much less significant. Quantitative kinetic perimetry and/or static profile perimetry are required to demonstrate the presence of a nerve fibre bundle defect in patients with baring of the blind spot.

Relative scotomata arising from and baring the blind spot, situated superiorly and not reaching the nasal horizontal meridian, can be due to a "refraction scotoma" which occurs in myopic individuals in whom the lower part of the fundus has an unusually pale appearance. The addition of concave lenses above the axial correction abolishes these scotomata (Fig. 2).

**Fig. 1** Upper baring of the blind spots in a patient with slight lens opacity, but no evidence of glaucoma. The profile perimetry 135°-315° shows a slightly lower sensitivity to light in the upper temporal part of the visual field but no evidence of a nerve fibre bundle defect.

**Fig. 2** Upper relative defect in a myopic patient which disappears with extra concave lenses. Such a defect can produce baring of the blind spot and is called "refractive scotoma"
NERVE FIBRE BUNDLE DEFECTS

Sector-shaped defects in the visual field are nerve fibre bundle defects. In chronic simple glaucoma, the superotemporal and inferotemporal parts of the nerve head seem to be most vulnerable to damage. Other parts of the nerve head may also be damaged. The nerve fibre bundle defects most frequently involve the arcuate fibres which arch above and below the fovea. These fibres end along the horizontal meridian, which extends from fixation to the nasal periphery of the field. Periophtalmic field defects along the nasal horizontal meridian should always be searched for. Sector-shaped scotomata elsewhere in the field should also be kept in mind. The size, shape, and location of the nerve fibre bundle scotoma will depend on the extent and site of the damage to nerve fibre bundles at the optic nerve head.

Nerve fibre bundle defects produce the following perimetric findings:

1. Circumscribed paracentral defects in the distribution of the nerve fibres.
2. Peripheral and central nasal steps.
3. Arcuate scotomata.
4. Sector-shaped scotomata elsewhere in the visual field.

CIRCUMSCRIBED PARACENTRAL DEFECTS

Paracentral circumscribed defects can occur in either the temporal or the nasal part of the Bjerrum area, and tend to be elongated circumferentially along the course of the nerve fibres. On the temporal side of the central field, they classically occur in the Bjerrum region between 10° and 20° from fixation in the area which constitutes the upward or downward arcuate projection of the appropriate pole of the blind spot (Fig. 3).
On the nasal side, the scotomata can come almost to fixation (Fig. 4), or alternatively they may be as much as 20° or even 30° away from fixation (Fig. 5). The Bjerrum region on the nasal side is very wide in accordance with the course of the arcuate fibres. The defects are often absolute when first discovered, or show deep relative nuclei surrounded by areas of less dense involvement. Dense nuclei are often multiple and lie along the course of nerve fibres (Fig. 6). The width of the paracentral scotoma can vary from 2° to 10° on the side, and is usually narrower on the temporal side. Paracentral scotomata are often delineated by the nasal horizontal meridian (Fig. 7, overleaf). A relative disturbance can be traced to a varying extent towards the blind spot indicating their arcuate nature (Fig. 8, overleaf).

**Nasal Steps**

All complete arcuate scotomata extend to the nasal horizontal meridian producing nasal steps. With the use of static profile perimetry, absolute or deep relative scotomata can be plotted in the course of the nerve fibre bundles which terminate in the horizontal nasal

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**Fig. 4** Paracentral scotomata with absolute nuclei. The nasal nucleus comes to within 3° of fixation.

**Fig. 5** Glaucoma field with arcuate scotoma above and a dense paracentral scotoma 15–20° from fixation below. The paracentral scotoma is probably part of a relative arcuate disturbance.
Multiple absolute scotomata along the course of the inferior Bierrum area. A relative disturbance is shown in the 225° profile (upper left) and does not appear to join the blind spot. A nasal step is evident.

Dense paracentral nuclei in relative arcuate defects with horizontal borders.
The glaucomatous visual field

FIG. 8 Arcuate scotoma with nasal step, showing the absolute nucleus to be 20–30° from fixation. The arcuate nature can be seen from the shape of the scotoma. The 315° profile shows a deep relative defect extending from 14–22°. The 225° profile shows no scotoma which indicates that the scotoma does not join the blind spot.

FIG. 9 Absolute paracentral scotoma producing a nasal step which is not present in the more peripheral isopters but reflected in the central isopter.

meridian at the site of a nasal step (Fig. 9). The shape of the nasal step and its width depend on many factors. In the periphery of the visual field, the nasal steps are often wedge-shaped (Fig. 10), whereas in the mid-periphery they tend to be more like a right angle (Fig. 11). Closer to fixation, the nasal steps assume the characteristics of an obtuse angle and this is consistent with the shape of the nerve fibre bundles reaching the horizontal nasal meridian at that point (Figs 10 and 11). Nasal steps are not necessarily found in all isopters tested. Depending on which nerve fibre bundles are damaged, they may be evident in some isopters but not in others (Fig. 11). The width of the nasal
step in degrees is also variable and no arbitrary rule, which assigns significance to a particular size of nasal step, is strictly accurate. Nasal steps are frequently found in association with arcuate or paracentral scotomata, and are only rarely (1.6 per cent.) significant isolated findings (Aulhorn and Harms, 1967). When facilities for accurate analysis of the central field do not exist, nasal steps in the periphery may appear to be the only signs of damage, but in reality they should point to more exhaustive analysis and a search of the central field. They are also useful corroborative signs when other defects are doubtful. The finding and significance of nasal steps in the central and peripheral field should not be underrated. The peripheral field is unfortunately neglected in glaucoma work.

**ARGUATE SCOTOMA**

An arcuate scotoma may be relative or absolute. In the temporal portion of the field it is narrower because all nerve fibre bundles converge onto the nerve head. It becomes
FIG. 12 Right field shows a nerve fibre bundle arcuate scotoma joining the blind spot. The scotoma is narrow on the temporal side, but widens nasally coming to 2° from fixation. There is additional evidence of involvement of more peripheral arcuate bundles shown by the wedge-shaped scotoma with nasal step in the peripheral field. Left eye shows an absolute arcuate “breaking through” to the periphery.

wider on the nasal side and may come almost to fixation along the nasal horizontal meridian. The scotoma can be very wide along this meridian. It is often accompanied by absolute nuclei, separate from it, situated along the nasal horizontal meridian peripherally to the arcuate scotoma. Such scotomata may be responsible for nasal steps in the peripheral isopters (Figs 7 and 12). The arcuate scotoma, bounded by its clear cut nasal horizontal border, often extends to the blind spot. It may be separated from the blind spot by an area of normal function (Fig. 8), or alternatively it may be joined to the blind spot by an area of relative impairment. Arcuate scotomata do not usually arise from the blind spot, and when they extend to the blind spot, are not densest at that point. Arcuate scotomata above and below form ring scotomata and usually show a nasal horizontal step between them. Occasionally arcuate scotomata cease abruptly at the vertical meridian, but any scotomata with vertical edges should be treated with suspicion, as they may be produced by neurological lesions involving the visual pathways.

Sector-shaped defects

There has been little interest in sector-shaped defects other than the Bjerrum scotoma which breaks through to the periphery. The reason for this is that by far the most frequent changes occur in the nasal field. The presence of sector-shaped defects, which extend towards the periphery of the visual field but not along the nasal horizontal meridian, must be borne in mind (Figs 13, 14, and 15, overleaf). This is particularly true of the
**FIG. 13** Glaucoma field, showing upper arcuate scotoma. There is an absolute sector-shaped scotoma inferiorly encroaching on the nasal and temporal field.

**FIG. 14** Glaucoma field, showing temporal sector defect with a horizontal border. The nasal field shows no defect.
progression of the one can be described in the field analyser with glaucoma where prolonged defects missed.

The nasal portions of the eye are easy to screen with the Goldmann perimeter, which is designed for the Goldmann perimeter with threshold targets. It consists of plotting the blind spot and 72 points in the central field, so that defects are likely to be picked up as abnormal responses (Fig. 16). This method was recently evaluated (Rock, Drance, and Morgan, in preparation) in people with no visual field defects and in cases of glaucoma with early and advanced visual field defects. The screening method was found to have a very high specificity (87 per cent.) and also a high sensitivity (90 per cent.). It was easy to perform and took approximately 5 minutes for each eye. The technique was then modified by us (Rock, Drance, and Morgan, 1971) to include the more peripheral nasal portions of the visual field, because peripheral nasal steps, which occur commonly, were missed. The addition of a few points in the nasal periphery did not significantly prolong the procedure and increased the sensitivity even further. The Friedmann field analyser is also suitable for glaucoma screening. The screening technique, as described for the Goldmann perimeter, is also applicable to the tangent screen providing one can make targets disappear. This can be done by having a flat wand with the target on one of its sides so that by turning the wand the target can be hidden from the patient.

**Method of screening for the presence of glaucomatous visual field defects**

Any method which is designed to screen for early visual field defects in glaucoma must have a high sensitivity, which means that it must detect as many of the visual field defects as possible, and it must have also a high specificity so that not too many false positives are produced by the screening procedure. The method must be rapid, non-tiring to the patient, and easily reproducible. Armaly (1969) has described such a screening method which involves the use of the Goldmann perimeter with threshold targets. It consists of plotting the blind spot and 72 points in the central field, so that defects are likely to be picked up as abnormal responses (Fig. 16). This method was recently evaluated (Rock, Drance, and Morgan, in preparation) in people with no visual field defects and in cases of glaucoma with early and advanced visual field defects. The screening method was found to have a very high specificity (87 per cent.) and also a high sensitivity (90 per cent.). It was easy to perform and took approximately 5 minutes for each eye. The technique was then modified by us (Rock, Drance, and Morgan, 1971) to include the more peripheral nasal portions of the visual field, because peripheral nasal steps, which occur commonly, were missed. The addition of a few points in the nasal periphery did not significantly prolong the procedure and increased the sensitivity even further. The Friedmann field analyser is also suitable for glaucoma screening. The screening technique, as described for the Goldmann perimeter, is also applicable to the tangent screen providing one can make targets disappear. This can be done by having a flat wand with the target on one of its sides so that by turning the wand the target can be hidden from the patient.

**Progression of the visual field defect**

Progression of the visual field defect in chronic simple glaucoma may be due to sudden,
Stephen M. Drance

**FIG. 16** Armaly's method of screening for glaucoma on the Goldmann perimeter (Armaly, 1969a)

**FIG. 17** Multiple absolute nuclei in an arcuate scotoma. Profile 45° shows the absolute nucleus to be very narrow

**FIG. 18** Progression of field defect shown in Fig. 17, showing coalescence of absolute nuclei into a larger and wider absolute scotoma.
step-like, fresh nerve fibre bundle defects. These are most likely to occur in proximity to previous defects, as the affected part of the optic nerve head is poorly perfused and vulnerable to further changes. Fresh absolute or deep relative nuclei in the course of the same nerve fibre bundle coalesce and convert isolated paracentral scotomata into arcuate scotomata (Figs 17 and 18). Damage to adjacent nerve fibre bundles results in widening of the original field defect. Widening towards the periphery can lead to a breakthrough of the scotoma towards the peripheral isopters. Fresh scotomata can also occur in parts of the visual field previously unaffected. Such fresh visual defects may gradually lead to an upper and a lower arcuate scotoma with a characteristic ring shape which usually retains a nasal step because of the asymmetry of the bundles involved. The ring widens to involve the more peripheral areas and spreads towards the centre. Fixation is spared for a long time, and even when the upper or lower scotoma encroaches on the nasal side to within a degree or two of fixation, foveal function may remain normal with good visual acuity. When a central island of vision is the only field remnant, a nasal step can usually still be plotted. The central island of vision is often accompanied by a temporal visual field remnant which may disappear before the central vision is finally abolished (Fig. 19), but may remain permanently. A change in density of existing scotomata and conversion of relative scotomata into absolute defects, is another way of progression. This may occur as a result of a gradual deterioration of function, or of sudden step-like episodes involving fresh nerve fibres.

Static profile perimetry is most useful for recording changes in the visual field of chronic simple glaucoma. The time involved in plotting static profiles is worthwhile in this disease, because of the greater reliability of the patient's responses. Deterioration can be plotted with greater confidence and further therapy, medical or surgical, can be planned.

Isolated paracentral scotomata in the Bjerrum area can disappear with successful medical or surgical reduction in intraocular pressure (Fig. 20, overleaf). This change is slow and rather infrequent, but indicates that certain defects are reversible.

The majority of visual field defects unfortunately remain either unchanged or show progression, the latter being often related to poor medical or surgical control of the intraocular pressure. Other factors apart from the pressure level must also be considered.
Visual field defects may occur as a result of a precipitous medical reduction of systemic blood pressure (Harrington, 1959), or as a result of a reduction in blood pressure due to a haemorrhage or to myocardial infarction (Drance, Wheeler, and Pattullo, 1968). Each patient should be evaluated in terms of all the components which may impair the perfusion at the optic nerve head, and an attempt should be made to modify those that lend themselves to medical or surgical manipulation. Intraocular pressure is most readily modified, and the lower the intraocular pressure the better the perfusion of the nerve head.

Progression of a visual field defect is probably the most important indication for more intensive medical or surgical therapy, and it must therefore be properly evaluated. In many glaucoma patients changes in the contour of the isopter are due to progressive
lens opacities, which also result in a reduction of visual acuity. Changes in the isopter may therefore be a misleading sign of progression of glaucomatous damage. They may be merely due to miotic pupils or to developing opacities of the media. Static profile perimetry is well suited to an analysis of visual field progression. Careful quantitative kinetic perimetry can be used for the same purpose.

Changes in medication, which alter the refraction or pupillary diameter, must not be disregarded, and visual fields should be re-plotted so that new baselines are established. Progression should then be measured from the new baseline fields.

**Pathogenesis of visual field defects in glaucoma**

The nerve fibre bundle defects characteristically found in glaucoma occur as a result of interference with the perfusion of blood at the optic nerve head (Harrington, 1959; Drance and others, 1968). The blood supply to the nerve head comes predominantly from the posterior ciliary arteries, through the circle of Zinn, and through the peripapillary choroid. Fluorescein angiography has suggested that the ciliary supply to the choroid and optic nerve head is segmental. The perfusion of the nerve head depends on a balance between the intraocular pressure and the intravascular pressure of the small blood vessels on one hand, and the patency and calibre of the arterioles and capillaries supplying this area on the other. Artificial elevations of the intraocular pressure have more effect on the peripapillary choroidal circulation than on the retinal vessels. A chronic interference with perfusion of the disc probably leads to enlargement of the optic cup at the expense of the neuro-retinal rim and atrophy which accompanies field loss. Cupping does not necessarily indicate a slow or chronic interference with the blood supply as it may be present before the vascular derangement. Acute interference with the circulation leads to ischaemic optic neuropathy evidenced by oedema, small haemorrhages on the optic disc, and loss of nerve fibre bundles, but often without an increase in the size of the optic cup. The acute events may be due to emboli, arteritis, arteriosclerosis, severe anaemia after blood loss, and probably many other events. Such acute episodes may be superimposed on chronic perfusion changes of the nerve. Multiple causes leading to progression of the visual field can be traced in many patients.

I am indebted to Mrs. M. Fairclough, Miss J. Bryett, and Miss C. Wheeler for the excellence of the charts of the visual fields.

**COMMENTARY**

**SEIDEL SCOTOMA**

A Seidel scotoma is defined as an arcuate scotoma which arises out of the blind spot, either inferiorly or superiorly, and tapers off. It is only present to very large targets. The Seidel scotoma, as originally defined, fits those scotomata found with acute rises in intraocular pressure and steroid-induced glaucoma. These scotomata are often reversible. In chronic simple glaucoma, however, the Seidel scotoma is rare. Traquair originally wrote that he had never seen it. If a Seidel scotoma is discovered, there are usually many other changes in the central visual field. Applying perimetric techniques to find nerve fibre bundle scotomata which involve moving targets out from the blind spot would miss about 50 to 70 per cent. of the nerve fibre bundle defects.

**PERIPHERAL CHANGES IN THE VISUAL FIELDS IN SIMPLE GLAUCOMA**

Peripheral nasal steps can occur without any detectable central field loss. In one series this is said to occur only in 1-6 per cent. of cases. If the disc is suggestive of glaucoma and no field defect is found in a very careful analysis of the central field, then a peripheral field defect is likely to be sector-shaped on the nasal side, but occasionally a temporal sector defect may be present.
AGE CHANGES IN PERIMETRY

In a patient with 6/6 vision, the 1/1 object on the Goldmann perimeter disappears completely in the 70's and the 1/2 object disappears in the 80's provided there are no lens opacities to accelerate the process. The 0/1 disappears much earlier. With the 1/2 it was found that there was much variation between people and the mean isopter was around 28°. In some it reached 40° but in others it was only just outside the blind spot. In persons aged between 60 and 69, with dilated pupils and 6/6 vision, the mean isopter to the 1/2 was only just outside the blind spot but, as this was a mean reading, there were many with the isopter inside the blind spot so that under certain circumstances baring of the blind spot occurred in this age group without glaucomatous change. Baring of the blind spot occurs sometimes below but usually above the blind spot. Refractive errors must always be corrected when investigating the central field, particularly when using hemispherical perimeters.

Value of tonometry and tonography in the diagnosis of glaucoma

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The value of a technique in the diagnosis of disease depends on its ability to give a measure of the disease process. Tonometry and tonography attempt to give this measure in glaucoma by a number notation. We shall confine our attention to chronic simple glaucoma since it is in this disease that both tonometry and tonography have been most widely used together. The great disadvantage of both techniques is that they measure a feature related perhaps only remotely to the disease process. Furthermore, while tonometry at least measures a typical physical sign of glaucoma, tonography purports to measure a factor which causes only one of the physical signs of the disease, namely raised intraocular pressure.

The choice between their relative merits, therefore, is a difficult one. Tonometry, the direct measure of intraocular pressure, is notoriously variable in the disease, and at one time the reading may be representative, while at another it may have a value indistinguishable from that found in a normal eye. Tonography, on the other hand, as a measure of outflow through the trabecular meshwork, estimates a quantity which shows wide variation between normal subjects and is not constant if determined on the same patient on several occasions. Both techniques, when related to chronic simple glaucoma in a group of patients, thus show about an equal correlation with the disease process (Fisher, Carpenter, and Wheeler, 1970). This fact throws considerable doubt on the value of a single outflow facility measurement in diagnosing early cases.

Apart from their relationship to the disease, the accuracy of measurement in both tonography and tonometry is of paramount importance if either is to be of value. Tono-