the visual field. We cannot rule out glaucoma by testing a predetermined or specific area in the visual field. A complete examination of the visual field is best performed by using both static and kinetic perimetry together. This seems to constitute the best means of modern quantitative perimetry. Scatter of test points can be controlled only by careful attention to numerous details involving physical, pharmacological, physiological, and psychological factors, the “chaff” which must be differentiated from the “wheat”—the pathological patterns to be delineated in testing the visual field. The factors causing scatter also influence the reproducibility of a given visual field from one date to another, as do the refractive media, pupil size, mental status, and choice of data points.

REFERENCES

The early field defects in glaucoma

Stephen M. Drance

The origins of the depression of central isopter known as “baring of the blind spot” as an early sign of glaucoma were traced. Changes in isopter occurring with aging were reported. Baring of the blind spot could be produced in everybody with threshold targets and was therefore not a pathognomonic sign of the disease. A more rapid depression of the isopter with ocular hypertension has not yet been demonstrated but remains a possibility. The earliest changes in eyes with open angle glaucoma that could be discovered with the use of static perimetry were paracentral scotomas in the Bjerrum area separated from the blind spot, coalescing into an arcuate scotoma joining the blind spot.

Acknowledgment of the earliest stages of damage in a chronic disease process, preferably at a stage when it is still reversible, seems fundamental to an understanding and rational management of the disease. In chronic simple glaucoma, which many
ophthalmologists will diagnose only when
damage to visual function has occurred,
it is essential to know the earliest repro-
ducible disturbances and their mode of
progression in order to ensure that recog-
nition is not unnecessarily delayed and yet
treatment should not be commenced un-
necessarily early in all ocular hypertension
without an understanding of those factors
which will predict damage to the eye. It
is not certain that reversible changes oc-
cur, though we suspect they do. It is pos-
sible that the production of small nerve
fiber bundle defects may be sudden,
precipitous, and irreversible. Progression
would then be due to the fallout of more
nerve fiber bundles rather than to a more
severe disturbance of those already dam-
aged. It must be remembered that the
nerve fiber bundle defect can occur in
conditions other than glaucoma. Such
changes as a contraction of the isopter
and the consequent baring of the blind
spot are quite nonspecific. They may well
be accentuated and occur earlier in eyes
with elevated intraocular pressure and
might be reversible.

To study early changes in visual func-
tion, a prospective study of people with
all levels of ocular pressures must be
carried out over the years and many
parameters, including the field, must be
recorded. Such studies are in progress but
no concrete results are yet available. Even
for such studies it is crucial to know ex-
actly what the early stages and sequences
of change of the visual field are so that
the field techniques may be set up to give
the answers sought after. Such prospective
studies may be most rewarding in families
of patients with chronic simple glaucoma
because this raises the incidence of ocular
hypertension and might yield a higher in-
cidence of damage of the visual field.
Another way of getting at early changes
is to study those patients who have ad-
vanced damage produced by open angle
primary glaucoma in one eye and in whom the
other eye does not yet show a visual field
defect but may or may not show rises in
intraocular pressure.

Almost 100 years ago, Von Graefe\textsuperscript{1} de-
scribed the paracentral scotoma in the
central field in cases of glaucoma. The
advent of the perimeter then shifted the
emphasis from the paracentral area to the
periphery of the visual field until Bjerrum\textsuperscript{2}
and his disciple Ronne\textsuperscript{3} reverted to test-
ing of the visual field with the use of
small stimuli and a 2 meter screen. They
described the classical sequence of the
glucomatous visual field, including the
arcuate scotoma with nasal step, breaking
through to the periphery and joining the
blind spot. Traquair,\textsuperscript{4,5} whose painstaking
quantitative perimetry remains a classic,
introduced the concept of depression of
the central isopter known as "baring of the
blind spot" as the earliest change of
chronic simple glaucoma. This was fol-
lowed by paracentral scotomas with their
dense nuclei separated from the blind
spot. Traquair's concepts from which
"baring of the blind spot" were developed
and their relationship to the early diagno-

![Fig. 1. Linear regression of visual field area on
age in 134 eyes. The broken line shows the 95
per cent confidence interval for the means of any
age while the confidence interval of the individual
readings is shown by the faint solid line.](https://iovs.arvojournals.org/pdfaccess.ashx?url=/data/journals/iovs/933618/ on 11/12/2018)
Fig. 2. Visual field in a 67-year-old normal man. The I, Goldmann target was used with a 63 per cent transmission filter, baring of the blind spot occurred.

Fig. 3. Right: static profiles along 3 meridians showing 2 absolute paracentral scotomas. There is a relative scotoma 2° from fixation in the 45° meridian. Left: kinetic plot shows the scotoma and nasal step. The small superior and relative scotoma could not be plotted kinetically.
sis of chronic simple glaucoma should be analyzed. In order to set the scene, I would like to quote from his very exact writings: (1) "The forms of glaucoma referred to as subacute, acute congestive, or inflammatory are to be regarded as exacerbations in simple glaucoma from which they differ only in violence but not in essential nature." (2) "The most usual symptom of which the patient complains is the presence of recurrent dimness of vision in one eye. This may last for a few hours or a day but disappears spontaneously. These symptoms indicate exacerbations of pressure and may be present for many years before a subacute or acute attack of glaucoma occurs or before cupping of the disc or change in the field of vision develop." These two statements indicate that, as angle closure and chronic simple glaucoma were not differentiated, the bulk of his patients had angle closure glaucoma and that the proof of an eye having early glaucoma was the subsequent occurrence of an acute attack.

He then stated that "since glaucoma is a bilateral disease, we search for the early stages in the apparently healthy eye of a patient who has undoubted glaucoma in one eye," and divided the eyes in which no obvious visual field defects were present into 5 groups: (1) "the suspected eye," which was in every way normal and healthy but was suspect because of definite glaucoma in the other eye; (2) eyes with no field defects in whom a history of halos and ocular discomforts and other suggestive features were present but with a normal optic nervehead and normal intraocular pressure; (3) eyes with cupping or pallor of the optic nerve found during routine examination; (4) increased pressure alone on routine examination; and (5) various combinations of the above. This group of people was then investigated with the smallest visual angles and the depression of the central field or baring of the blind spot was elucidated. He stated, "it (baring of the blind spot) is the earliest field change I have found in cases of suspected glaucoma. It is interesting to note that, although baring of the blind spot is undoubtedly the precursor of the arcuate scotoma, the arcuate scotoma does not appear to go out of the baring but seems to arise independently as a small curved scotoma on or about the 15° circle, a little distance from the blind spot. The baring of the blind spot may be present in the upper part of the field with an early arcuate scotoma in the lower part." Traquair therefore examined mostly second eyes of patients who had established angle closure glaucoma in the first eye or who had prodromal symptoms of angle closure glaucoma in an eye which had not yet been damaged. One of the main pieces of evidence for baring of the
blind spot being a precursor of the classical field defects of chronic simple glaucoma was the fact that many of Traquair's patients developed acute or subacute attacks of glaucoma within two or three years. Traquair himself stated that classical arcuate scotoma often occurred in the opposite part of the field from that which bared the blind spot.

The aging process, with changes in pupil size, changes in the clarity of media, narrowing of the palpebral aperture, and possibly some changes in the neuroretinal mechanism, leads to a diminution of the entire visual field with advancing years. In addition, the slope of the field around the blind spot is flattest. Small visual angles become threshold stimuli for many older normal individuals and it is characteristic of examinations with threshold targets that classical baring of the blind spot, more often above than below, develops as part of the change in the isopter (Figs. 1 and 2). We were able to establish baring of the blind spot in almost every body, young or old, by choosing threshold targets. Standard targets, such as the 1/1,000 or 1/2,000 or the 2/1,000 or 2/2,000 or Goldmann targets I1 or I2, will be threshold for some individuals with aging and must be expected in them to bare the blind spot at that time. This type of visual field defect can therefore not be considered as an entity indicating early damage from chronic simple glaucoma.

The fact that in chronic simple glaucoma or in ocular hypertension there may be a more rapid change in the isopter and retinal sensitivity could be true, but has not as yet been established.

Taking our cue from Traquair we carried out a study, choosing a group of people in whom one eye showed the advanced changes of open angle glaucoma, with an atrophic nervehead and a very advanced classical glaucomatous visual field defect in the presence of a normal angle, in whom the other eye was apparently not damaged to a 1 mm. or 2/1,000 white target. Static and kinetic perimetry was performed with the use of the Tubingen perimeter to plot the photopic visual field.
thresholds at 1° intervals along the oblique meridians. The entire central fields were searched diligently for evidence of other scotomas and the kinetic isopters were plotted at the end. In those patients in whom the badly damaged eye had elevated intraocular pressures, one could assume that any changes found by the more sophisticated techniques which eluded preliminary discovery with the tangent screen are likely to be early manifestations of chronic simple glaucoma. The classical changes in the second eye were found to be small absolute paracentral scotomas with their long axis usually directed in the line of the arcuate nerve.
fibers surrounded by a zone of relative scotoma and separated from the blind spot either by a completely normal field or a very much less disturbed area of visual functions (Figs. 3, 4, and 5). These paracentral scotomas often come to within 2 to 3° fixation on the nasal side and were usually further away from fixation on the temporal side. They occurred in the classical Bjerrum area although they did not form a complete scotoma at this stage. Some patients had a similar type of defect in the same locations but the defect was only relative. These relative defects were more difficult to interpret and their significance and progress are being evaluated at this time. Such scotomas can occur in people who do not have any evidence of glaucoma in either eye; we have noted them to correspond to cotton wool exudates seen after severe gastrointestinal hemorrhage and systemic hypertension. We have not found the classical Seidel scotoma, which is a scotoma arising from the blind spot, following a slightly curved course and being widest at the blind spot and then tapering out in a pointed way away from the blind spot. Traquair stated, "I have never been able to establish the presence of defects of this kind, even by serial testing."

Traquair believed that the true arcuate scotoma arose quite quickly and was often quite a large defect when first noticed. "It seems extremely difficult to detect its first appearance and to trace its early development, though I have observed many cases of glaucoma and never been able to follow its growth step by step and for this particular reason, I believe it develops rapidly." It is our impression at this time that most of the paracentral absolute scotomas in the Bjerrum region do in fact develop quickly as one would expect from the fact that they are vascular in origin (Fig. 6).

We have, however, many recorded sequential fields in which relative nuclei became gradually denser and ultimately absolute side by side with the occurrence...
of fresh defects (Fig. 7). If the occurrence of maximal defects were the only sequence occurring in glaucoma then the chances of finding reversible and predictive signs in the visual field would be small and unlikely, but the early incomplete evidence, such as we have at this time, suggests that established defects do undergo change; this gives us hope that by employing more sophisticated physiological parameters of visual function such as spatial, temporal summation, size of receptive field, and their change with states of adaptation may lead to a reliable predictor during a reversible stage. This is being investigated at this time in our laboratories and clinics.

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The Bjerrum area in ocular hypertension

R. M. H. Pinkerton

Circular static perimetry was carried out on preselected points in the Bjerrum area on normal eyes and eyes with ocular hypertension. It was found that in the normal subject there was a decline in sensitivity with age, most particularly marked in the upper field. Eyes with ocular hypertension showed more reduction in sensitivity than normal eyes in the same age group. It is postulated that in ocular hypertension there is premature aging of the field in the Bjerrum area.

We have yet to establish a definition of the glaucomatous state and lacking this we are even less able to define the conditions variously called ocular hypertension, preglaucoma, or glaucoma suspect. For the purposes of this study, ocular hypertension was defined as follows: tension: 21 to 25 mm. Hg, with a 4 to 5 mm. rise on water drinking; tonography: C values 0.12 to 0.20 before or after water drinking, and Po/C values 100 to 150; optic disc: no ophthalmoscopically visible abnormality; visual fields: no abnormality detected on kinetic Goldmann perimetry; family history: no known family history of glaucoma.