THE BRITISH JOURNAL OF OPHTHALMOLOGY

JUNE, 1917

COMMUNICATIONS

BITEMPORAL HEMIOPIA: THE LATER STAGES AND THE SPECIAL FEATURES OF THE SCOTOMA

With an examination of current theories of the mechanism
of production of the field defects

(Concluded from p. 294)

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Local Intoxication

In order to explain the scotomatous or neuritic type of field it was suggested by Fuchs²¹ in 1895 that toxins, present in the inflammatory œdema surrounding an actively growing tumour, acted upon the papillo-macular bundle in the chiasma. Then in connection with cases of post-nasal sinus disease, toxins are commonly believed to be the cause of the frequently associated retrobulbar neuritis, and it seems reasonable to expect that, when bitemporal hemiopia occurs in these cases, it should be produced in the same way. There is a great clinical resemblance between the field changes in retrobulbar neuritis and in scotomatous cases of bitemporal hemiopia, especially the markedly scotomatous type, of which Case III is a good example. In both conditions central defects are very prominent, while peripheral changes are usually relatively slight, and comparatively rapid variations in the size, shape, and the intensity of the scotoma are common. It seems

very probable that chiasmal or tract hemiopic or quadrantic scotomata are due to the same kind of cause as operates in many cases of retrobulbar neuritis, and, as a preliminary to examining this question as it affects the chiasma, we may shortly refer to what is known of the causation of this more familiar but equally little understood condition.

A discussion of the ætiology of retrobulbar neuritis will be found in the writings of Wilbrand and Saenger,⁶⁴ Onodi,³⁸ Birch-Hirschfeld,⁴ Schieck,⁴⁶ Augstein,¹ Langenbeck,³⁵ and others. Wilbrand and Saenger state explicitly that a central scotoma is always a sign of a *neuritic* lesion of the optic nerve path (the italics are the present writer's), and Schieck points out that retrobulbar neuritis is never primary but always dependent on some other morbid process. The cases which occur in connection with post-nasal sinus disease and with orbital tumours are of most interest in the present instance, because, as we have seen, sinus disease as well as hypophyseal tumours may cause bitemporal hemiopia.

In regard to the former variety, little or nothing is really known as to its cause, though, at the same time, the theory that the action of toxins derived from the inflamed sinuses is responsible is widely accepted. These cases are fairly common and frequently improve rapidly on opening up the sinuses, according to some writers, even if no septic condition is found. The anatomical conditions favouring this form of retrobulbar neuritis have been discussed by Onodi in his well-known book. According to Fuchs²⁰, the neuritis is caused by a direct extension of the inflammation to the orbital contents. Others, such as Wallis^{61 62}, favour a soakage of the toxins through the tissues. Wallis also observed that acute sinusitis is associated with central scotoma and chronic sinusitis with peripheral contraction of the fields. It is evident that the toxins do not act by way of the blood stream, as in this case retrobulbar neuritis would be a common accompaniment of bacterial infections of distant parts. It has been suggested that lymphatic pressure may be set up in the interior of the nerve, but, as already pointed out, this alone would not cause central scotoma. According to Groenouw²⁵, disease of the papillo-macular bundle alone does not arise through general compression of the optic nerve. Only in the intracanalicular portion of the nerve is pressure likely to develop, and the ring-scotoma which has been supposed to be specially characteristic of compression at this part occurs, as we have seen, in chiasmal lesions also, where the anatomical conditions are quite different.

It is well-known that orbital tumours may give rise to optic neuritis with central scotoma, but the mechanism of production of the nerve affection has received little attention. Birch-Hirschfeld,⁴

who is a convinced adherent of the toxic origin of retrobulbar neuritis, in his well-known paper describes a case of sphenoidal carcinoma which had broken through into the orbit and produced obstruction of the central vein of the retina. Optic neuritis and central scotoma were present, and a lesion was found in the papillo-macular bundle close behind the point of exit of the central vein. Farther backwards, where the tumour came in contact with the nerve, the conditions were normal. The microscopic characters of the lesion were similar to those found in monkeys after experimental poisoning with methyl alcohol. He suggests that the toxins are abnormal metabolic products of the tumour growth, and reach the nerve fibres along the obstructed vein. Onodi also refers to sphenoidal tumour with central scotoma, but without giving details. As Birch-Hirschfeld points out, venous congestion and oedema of the nerve cannot be the cause, as otherwise central scotoma would be a common feature of other conditions in which the nerve is affected in this way; and he believes that, in his case, it is justifiable to assume the chemical action of toxins. The same objection applies to the explanation advanced by Schieck: that the oedema interferes with proper nutrition of the nerve fibres, and, by preventing the removal, causes the accumulation, of normal toxic products of metabolism.

Through the kindness of Dr. Sym, I have recently seen a case in some ways closely resembling that of Birch-Hirschfeld. A tumour of five years' duration was present at the inner side of the right orbit of a woman of 35 years. The eye was displaced outwards and forwards, so as to be almost outside the orbit. Movement was extremely good except inwards, where it was somewhat restricted. About a year ago sight had begun to fail. There was marked optic neuritis, with great congestion of the retinal veins. Vision: fingers at 1.5 metres. The field showed enlargement of the blind spot and a central scotoma of about 5°, but was otherwise practically normal. The left eye was normal and healthy. A provisional diagnosis was made of obstruction to the central vein behind its exit from the nerve. The mobility of the eye seemed to indicate that the optic nerve was not greatly, or, at least, not tightly, stretched. An encapsuled soft tumour, about as large as a very big walnut, was removed. It was found to be in contact with the optic nerve behind the eye. Unfortunately, the result of the operation was not such as to enable any conclusions to be drawn. The eye became totally blind with subsidence of the neuritis, very small arteries, and pallor of the disc. The central artery of the retina was probably damaged at the operation.

Whatever the cause may be, it is evident that orbital tumours can cause retrobulbar neuritis, and Dr. Sym's case is interesting in so far as the tumour was purely orbital and encapsuled.

With regard to neuritis of the intracranial, as distinct from the orbital, portion of the optic nerve, we have little information. The observations of Foster Kennedy³¹ show that tumours and abscesses of the frontal lobes may sometimes produce symptoms of retrobulbar neuritis on the ipso-lateral side, with the ordinary papilloedema with good vision, characteristic of increased intracranial pressure on the contra-lateral side. He suggests that this neuritis, with central scotoma, is the result of mechanical pressure on the intracranial portion of the nerve, the macular fibres suffering on account of more fragile structure in spite of their being surrounded by grosser strands, which retain their function, although more immediately subjected to the pressure. Kennedy's cases seem to favour a purely mechanical origin of retrobulbar neuritis, although this is opposed to Groenouw's view.

If we now apply our knowledge of retrobulbar neuritis, scanty though it be, to the study of chiasmal scotomata, it becomes evident that it is unnecessary to confine ourselves to theories of causation based solely upon the mechanical action of tumours. These scotomata indicate a neuritic process, and the condition we are dealing with is, as was suggested in my previous paper, a chiasmal retrobulbar neuritis, which may be produced by many causes, of which the proximity of a tumour constitutes only one. Rönne^{22, 43} who is, as far as I am aware, the only other observer who has described this form of scotoma, attributes it to disseminated sclerosis or retrobulbar neuritis of the chiasma. An interesting and illustrative case is reported by Augstein. In a man of 38 years rapid loss of vision occurred in the left eye following severe left-sided headaches. When seen, the left field was reduced to the *upper-inner* quadrant only, while the vision and field of the right eye were normal. The sphenoidal cells were opened but found healthy. Although the Wassermann was negative, antisyphilitic remedies were actively employed, but without success. All perception of light was lost in the left eye, and a central scotoma, together with peripheral, mainly temporal, constriction appeared in the right field. After seven weeks, pallor of the temporal side of the left disc was observed. It was thought that a basal lesion in front of the chiasma might be present, possibly a rapidly growing sarcoma of the dura. The case was seen by Uhthoff and a diagnosis of retrobulbar neuritis, independent of syphilis, and with favourable prognosis, was arrived at. Recovery of vision ultimately took place. Augstein is a strong supporter of the toxic origin of the condition, and believes that some form of auto-intoxication was the causal agent. His case is quite a characteristic example of chiasmal retrobulbar neuritis, and the fields were of the same type as occurs in association with hypophyseal tumours; unfortunately the scotoma in the right field was not minutely analysed. It is certain, in my opinion, that if more exact

methods of perimetry were in general use, such cases would be more frequently detected.

We have considered the suggestions which have been offered as to the way in which the optic nerves become affected in cases of sinusitis or orbital tumour. The problem in regard to the chiasma, which is practically surrounded by cerebro-spinal fluid, and nowhere in contact with the dura mater, is much more difficult, and our ignorance is correspondingly greater.

Venous congestion *alone* may be rejected ; it is doubtful whether it occurs to any great extent even in tumour cases.

By direct extension the inflammation might possibly be conducted from the dura over the olivary eminence, along the pia mater, which lies between the two optic nerves, to that which invests the lower surface of the chiasma and infundibulum. Were it to spread along the optic nerve sheaths, one would expect the nerves to be affected before the chiasma was reached, with consequent masking of the clinical picture. Where the chiasma is exceptionally far forwards, so that it lies over the olivary eminence, it might conceivably become affected by direct extension from a subdural focus depending on a sinusitis.

In regard to toxins, only those arising from a neighbouring local source need be considered, although it is of course possible that toxins absorbed into the blood from a distant focus may affect the chiasma. It is a characteristic feature of the action of toxins in the circulation, which affect the visual nerve path, that they exercise a marked selectivity in favour of the extracranial portion of the papillo-macular bundle, although, of course, their action is not limited to this part. Circulating toxins would therefore by preference produce ordinary retrobulbar neuritis. The proximate causes of disseminated sclerosis and of tabes are, however, said to attack the chiasma in some cases, although here also the optic nerves themselves are usually the site of election.

In regard to soakage or diffusion of toxins from the sphenoidal sinus, the chiasma is in quite a different position from the optic nerves, on account of the surrounding fluid. This explanation is applicable to the chiasma only if we postulate specially favourable anatomical conditions. If the clinical picture of chiasmal neuritis is to be produced without being masked by that of retrobulbar neuritis—and it is quite conceivable that both conditions might occur together—the optic nerves must be protected by thick bone, by position, or in some other way, and at the same time the chiasma must be specially exposed by conditions such as a well developed sphenoidal sinus, a prominent and thin-walled olivary eminence, and unusual shortness of the intracranial portions of the optic nerves. While such circumstances would possibly enable toxins to reach the chiasma by diffusion, it seems

unlikely that they would often all occur together. A powerful factor would be the development by direct infection of a local periostitis in the neighbourhood of the olivary eminence, with the production of a swelling possibly sufficiently elevated to touch the chiasma into which toxins might then be diffused, or to which an inflammatory condition might be communicated. As veins pass from the sphenoidal sinuses to the circular sinus (Evans¹²), the occurrence of such a periostitis in some cases seems quite probable. It must not be forgotten, however, that the occurrence of these pathological anatomical conditions has never been demonstrated.

When we come to consider the case in regard to tumours, it is necessary to assume that the zone of inflammatory œdema and cell activity which surrounds tumours, especially such as exhibit active growth and metabolic changes, contains toxins which can cause a neuritic process in the chiasma. In the tissues surrounding such tumours histologists have observed cell changes of a necrobiotic nature which have been attributed to the action of toxic metabolic products of the tumour. The causation of an active cyclitis by a necrosing glioma retinae, an excellent case of which I saw some years ago, is a more familiar example of the action of these toxins. On this hypothesis, before actual pressure has developed, a neuritis may be set up in the chiasma by the tumour toxins.

We have seen that the results of operation often afford a strong argument in favour of pressure; there are, nevertheless, many cases in which it is not evident that the results have been due to relief of pressure. In two of Hirsch's cases the sella was opened but nothing removed, yet temporary benefit ensued. Throughout Hirsch's paper runs the assumption that the visual symptoms are due to pressure, and recovery of vision is held to prove considerable relief of pressure even when what was done at the operation had not led to any such expectation. The author himself expresses great surprise at the unexpectedly good results following very partial removal of the tumour which he obtained in some cases. At the same time he suggests that when a cyst has been evacuated its walls fall together, partly on account of pressure from the brain, and the remains of the tumour become confined to the sella. No doubt this is just what occurs, yet, if the brain pushes the remains of the tumour back into the sella in this way, the local pressure on the chiasma will be kept up to some extent and the relief cannot be very great. Remembering that the pressure theory depends largely upon the assumption that very slight pressure can cause well marked symptoms, the question arises whether the unexpectedly good results may not have been due, partly, at any rate, to some cause other than the relief of pressure.

The beneficial results of simply opening the sella or sellar decompression are well known. The amount of relief of pressure

is often probably very slight, and the evidence is not convincing that the recession of symptoms is due solely to removal of mechanical pressure on the chiasma. In some cases very similar results appear to have been obtained without opening the sella at all. In Case XXIII of my series it was not absolutely certain that the sella had been opened, and yet temporary improvement in the ocular symptoms followed. In this connection an interesting case is recorded by de Kleijn.³² A woman of 41 years, complaining of defective sight, was found to have bitemporal hemiopia, together with posterior nasal sinus suppuration. Menstruation had ceased suddenly ten years before. The opening up of the sinuses was followed by an apparent great success. Headaches and vomiting disappeared entirely, and the patient became free from all symptoms except the visual defects. The exact state of the fields before and after the operation is unfortunately not stated. A diagnosis of bitemporal hemiopia due to post-nasal sinus disease was made. Three years later progressive visual disturbance and a positive X-ray picture indicated the presence of a tumour. It may, of course, be objected that the headaches were relieved by the removal of the pus from the sinuses and that this case is therefore not comparable with Case XXIII in my series, in which headaches were relieved after the opening up of healthy sinuses. In de Kleijn's case, however, the headaches returned apparently after three years, although at that time there was very little suppuration in the nasal cavities.* In any case, allowing for the psychical stimulus and the difficulty of distinguishing *post hoc* from *propter hoc*, it must be remembered that beneficial results are recognised as following the scraping out of the post-nasal sinuses in cases of retrobulbar neuritis whether definite signs of inflammation are observable or not (Fraser¹⁸). This does not seem altogether surprising as the practice of leeching or blistering the temple is common and popular in retrobulbar neuritis, and bleeding from the mesial orbital wall should, one would imagine, be still more efficient.

These considerations suggest that it is possible that operation does not always act by the removal of local pressure in the sense of removing a tumour actually pressing upon the nerve fibres, or even by the little extra room gained through sellar decompression, but that, apart from, or, at the least, in addition to these influences, the local blood-letting and devascularisation in themselves are important factors both in tumour and in inflammatory cases. While such local blood-letting may be of real benefit in sinus disease, in which a cure may result, in tumours only a temporary influence on

*Elsberg has reported a somewhat similar case. Infection of the sphenoidal sinuses with basilar meningitis developed in an acromegalic subject, followed by blindness in the right eye and a large temporal defect in the left field. After drainage of the sinus, both fields rapidly became normal.—*N. Y. State Jl. of Med.*, Nov., 1916. Ref. *Brit. Jl. Ophthalm.*, Mar., 1917, p. 181.

vision can be expected. The depletion drains away the toxins, and in the sinus cases their source also is removed with resulting cure; in the tumour cases they soon re-accumulate and the visual symptoms recur. The distortion and displacement of the chiasma with little or no visual disturbance mentioned by Cushing and Uhthoff might be accounted for by the toxin theory if we suppose that in certain cases there is a relative absence of irritating substances around the tumour, and thus neuritis is not set up. Another suggestion, which may be glanced at before passing from this part of the subject, is that a selective action is exhibited by the toxins which attack the nerve fibre bundles in a definite order and thus produce the typical field defects. There is little in favour of, and much against, such a view, and although it would be a convenient assumption on which to base the solution of some difficulties, such as the production of similar fields under very varying conditions, its serious consideration seems hardly necessary.

In a paper by Schnabel⁴⁷ a discussion of this question is recorded, the author being in favour of toxins, while others, on the whole, supported pressure. Walker⁵⁸ lends some support to the toxin theory. Uhthoff, on the other hand, does not even discuss the toxin theory in regard to the chiasma. A peculiar case recorded by Battiscombe³ is worth mentioning here. A woman developed photophobia and then extreme conjunctival chemosis. Vision was good and the fields uncontracted apparently to within a day of her death. Post-mortem.—Suppuration in the sella Turcica was found, the diaphragma sellæ forming the bulging roof of an abscess, the upper and anterior part of which was indented by the chiasma. Both toxins and pressure in this case had unusual opportunities, yet apparently no gross visual defect was produced.

There is insufficient evidence that toxic substances are present and active in the immediate neighbourhood of tumours, though their existence may not unreasonably be inferred. Like the pressure theory, the toxin theory is merely a working hypothesis for the purpose of explaining clinical observations. None of these explanations covers all cases, nor has any one of them been proved. Each appears to be specially applicable to a certain set of cases. It is reasonable to suppose that both toxic and mechanical causes act, the latter mainly in the later stages.

Course of the field defect

The peculiar circular course of the defect in the fields, clockwise in the right and counter-clockwise in the left, is best explained by the arrangement of the nerve fibres in the chiasma. The researches of Henschen, Uhthoff, Wilbrand, and others are discussed by Wilbrand and Saenger,⁶⁴ who state that the crossed fibres spread out in broad loops over the whole chiasma extending to its sides and

angles. At the front of the chiasma they lie dorso-medially, at the back ventro-medially, and the lower layers of the posterior part consist of crossed fibres only. Of the uncrossed fibres some bend inwards in loops almost to the centre of the chiasma and others lie along its outer side (*see* Fig. 28). They are surrounded by and mixed with crossed fibres. The papillo-macular bundles form a broad band extending nearly to the sides of the chiasma, the crossed fibres being central, the uncrossed more lateral.

The crossed central fibres lie near the dorsum of the chiasma at first, but incline more ventrally as they pass backwards. Dean and Usher¹⁰ found in monkeys that the macular fibres do not begin to cross until they are some distance within the chiasma, and that they begin to cross near the dorsal surface, becoming more ventral posteriorly, but never reach the lower surface of the chiasma. Thus the papillo-macular bundles practically form a miniature chiasma within the chiasma and rather towards its posterior part. The experiments of Parsons³⁹ and others have shown that in monkeys the retinal fibres retain the same relative positions in the optic nerves as in the eye, but become twisted over inwards at the chiasma so that the inner or crossed fibres come to lie more ventrally and the outer or uncrossed fibres assume a more dorsal position.

FIG. 28.

Suggested allocation of fibres.

Modified from Dejerine¹¹.

- A Crossed fibres from lower-inner retinal quadrant.
- B Crossed fibres from upper-inner retinal quadrant.
- C Uncrossed fibres from upper-outer retinal quadrant.
- D Uncrossed fibres from lower-outer retinal quadrant

According to this arrangement, the fibres from the upper-inner retinal quadrant (field quadrant 2) will be those which lie relatively dorsally and anteriorly in the chiasma (B) soon passing across and looping into the end of the opposite optic nerve, and thence along the opposite side of the chiasma to the tract. The fibres from the lower-inner retinal quadrant (field quadrant 1) will more rapidly assume a ventral position and will be those which lie along their own side of the chiasma (A) looping into the tract of the same side, thence crossing relatively posteriorly and ventrally to the opposite tract. Hence these fibres are the first to be affected by any interference coming from below. The uncrossed fibres from the upper-outer retinal quadrant (field quadrant 3) will be those which are looped farthest inwards (C) reaching almost to the centre of the chiasma, where they are mixed with the crossed fibres. Thus they may be affected before all the crossed fibres are blocked. Lastly, the uncrossed fibres from the lower-outer retinal quadrant (field quadrant 4) are probably those which lie toward the outer side of the chiasma (D) most remote from interference from an approximately central ventral lesion. The frequent presence of the temporal island in the fields, usually mainly if not entirely in the second quadrant, indicates the functional survival of some of the crossed fibres from the upper-inner retinal quadrant or from the junction of that and the lower-inner retinal quadrant. Their position is very possibly amongst those which cross most anteriorly and run down the sides of the chiasma among the uncrossed fibres. Such an arrangement would account for the field changes. A lesion affecting the lower and posterior part of the chiasma, where there are only crossed fibres, and spreading upwards, forwards, and laterally, would affect the field quadrants in the typical order and by attacking the inmost loops of the uncrossed fibres, while some crossed fibres were still spared, would give rise to the temporal island. Although this view of the development of the visual defects may be thought somewhat speculative, it is interesting that the peculiar looped course of the fibres should lend itself so aptly to the explanation of clinical facts of which the discoverers of this arrangement were unaware. The development and progress of the defects of the central area of the field indicate that the papillo-macular fibres closely follow the arrangement of the more peripheral fibres in regard to crossing and even to looping. The spreading out of the fibres into thin layers in the chiasma receives strong support from the clinical behaviour of the fields. Even in the early stages, in the non-scotomatous as well as in the scotomatous form of field change, the first signs of defect can be demonstrated in the central part of the field, a fact which would almost justify the inference, had objective evidence not been already to hand, that the nerve fibres are spread out in the looped arrangement described.

Let us now briefly review the subject from the clinical aspect. In the earlier stages the primary lesion, sinusitis, syphilis, tumour, or whatever it may be, sets up a disturbance in the chiasma, the symptoms of which are closely related to those of retrobulbar neuritis. The intensity of this disturbance varies with the nature of the cause, and the more active the condition is, the more likely are centro-cæcal scotomata to appear. When the activity of the primary lesion is slight, scotomata are absent, as in chronic acromegaly. As the primary condition may be of any intermediate grade of activity, there is no hard and fast line between the scotomatous and the non-scotomatous types of field change.

Cases without tumour usually have an inflammatory basis, and scotomata are nearly always a prominent feature. Recovery frequently occurs in a comparatively short time, any permanent loss of sight being often much less than was present when the condition was at its worst. The typical changes occur in the field of vision and have a strong resemblance to those of retrobulbar neuritis, rapid and extreme alterations being of common occurrence. The cause is usually supposed to be a local action of toxins on the chiasmal fibres, but may be a limited participation by the chiasma in the inflammatory process.

Tumour cases may be roughly divided into two chief groups: the chronic and the progressive. As a type of the former we may take chronic acromegaly which, as is well-known, is compatible with long life and useful vision. The tumour is intrasellar and grows very slowly: the characteristic field defects develop gradually and without scotoma. Often progress ceases and a stationary condition is maintained for many years, so that the development of more than the early or middle stages of bitemporal hemiopia may never occur.

Owing to inadequate observation the fields in these cases are often described as normal (see Fig. 27). The sella Turcica may be only slightly altered and becomes deepened rather than flattened.

In the progressive group the tumour grows more actively. The sella Turcica becomes flattened and the clinoids eroded in contradistinction to the deepening with elongation of the posterior clinoids which takes place in the former group. The case-histories are shorter; death often occurs in a few years, although there may be considerable variation in this respect (Frankl-Hochwart).¹⁷ If acromegaly is present it assumes a more rapidly progressive character, the "acute malignant acromegaly" of Sternberg⁵⁰ and Gubler.²⁷ Recovery may sometimes occur, as in Case I, presumably owing to the spontaneous rupture of a cyst. In the visual fields centro-cæcal scotomata are always present, and the defects resemble those occurring in retrobulbar neuritis and in chiasmal neuritis due to sinus disease. The progressive nature of the condition is usually well shown by the presence of disproportionality

(Rönne)⁴⁴ in the fields, that is to say, the isopters mapped out by a moderately large white test-object (5 mm. to 10 mm.) may show relatively very little alteration in comparison with those found by a coloured or small white test. The cause of the field changes in tumour cases is attributed to pressure, traction, or local intoxication by toxins formed in or around the tumour, especially in tumours of the progressive group.

Up to a certain point the production of similar fields in inflammatory and tumour cases may be explained on the theory of chiasmal retrobulbar neuritis set up by toxins. In tumour cases, especially in the progressive group, it is obvious that after some time, varying in different instances, the growth will become large enough to exert, in addition, a purely mechanical influence. The chiasma now becomes pressed upwards and often forwards, and its connections become expanded, thinned, stretched, and compressed in various ways already alluded to. Thus in the later stages, after the space below the chiasma has been filled up, actual pressure or traction may come into play and, influenced by various circumstances, may disturb and modify the typical nature of the field changes. Thus we find cases progressing normally for some time and then suddenly changing from bitemporal to homonymous hemiopia. Such a change appears to be often followed by a temporary or possibly permanent cessation of progress of the growth, as occurred in Cases I and XV and in a case recorded by Würdemann and Barnes.⁶⁵ A very similar case is mentioned by de Kleijn³² as being recorded by Erdheim. These changes may be due to the shifting of the incidence of the interference with the nerve paths by the bursting of cysts, or the giving way of dura mater or bone which had hitherto confined the tumour. The typical nature of the field changes becomes swamped, as it were, by the new influences introduced by the intracranial extension of the growth, so that the regular progress of the defect from one quadrant to another may be greatly modified or even completely altered. These considerations may help to explain the differences found by various writers as to which part of the field is retained longest.

We may, therefore, distinguish between the typical or regular and normal course of the field changes and the actual or modified course, the latter being the normal course as changed by later influences. Often the normal course persists to the end, the field terminating in the upper nasal quadrant. In other cases the relatively strongest portion of the nasal field is found to be at one time in the upper nasal quadrant, although the field may ultimately terminate otherwise. This relative superiority of the upper nasal quadrant will probably be found in the great majority of advanced cases if properly looked for. In order to detect this feature, the nasal field must be frequently examined at all stages with a declining series of test objects, so

that the typical changes may be discovered before they undergo any modifications.

In the present state of our knowledge there are naturally many exceptions to any rule of this nature, and one must agree with Walker and Cushing when they say that "there are many elements which enter into the chiasmal distortion which may affect the character of the fields and which are not thoroughly understood."

One other point remains to be noticed. In none of my cases has the remaining area of the upper-inner quadrant been followed to the vanishing point. The exact way in which the last fibres of the outer part of the uncrossed bundle become affected is a detail of only relative interest in the present instance, especially as at this stage, the regularity of the process is apt to be lost.

The difficulty of procuring material of the necessary kind, and at the particular stages which must be investigated, has made it impossible to bring forward histo-pathological evidence in regard to the cause of the field defects. In this series of cases only one post-mortem has occurred and the material was unfortunately not preserved. In most cases death only occurs after the stage at which an investigation of the parts would show what was taking place. It is therefore necessary to express on some points only qualified opinions at the present time.

The following conclusions, some of which are only tentative, may be enumerated:—

1. The perimetric defects in bitemporal hemiopia follow a typical or normal course of development. Commencing in the upper-outer quadrant the field is involved in a circular manner, the loss proceeding clockwise in the right field and counter-clockwise in the left, so that the upper nasal quadrant remains longest. This is the typical course and occurs in the majority of cases but, naturally, not in all.

2. The central defect or scotoma behaves in the same way, developing like a little field within the field.

3. This type of field defect is due to interference with the chiasmal fibres, but is largely independent of the exact nature of the ultimate cause; it occurs in bitemporal hemiopias from a variety of causes.

4. The immediate cause is very probably a chiasmal neuritis, a lesion comparable to that which, acting in the optic nerve, produces the symptoms of retrobulbar neuritis.

5. The cause of this chiasmal neuritis is not definitely known. In all probability it is set up in some way by pressure in many cases. The access to the chiasmal fibres of irritating toxic substances derived from the causal lesion may be the cause; and the presence of these substances may in some cases indirectly arise from pressure. In some cases the chiasma may directly

participate in an inflammatory process. The relation of the infundibulum to the chiasma is very probably of importance.

6. In tumour cases, and probably in some cases without tumour, mechanical pressure effects also act, mainly in the later stages when the tumour has reached a relatively large size.

7. In the later stages the normal type of progress of the field changes may be "swamped" by pressure effects and greatly altered.

8. These observations provide evidence from the clinical side in support of the looped arrangement of the fibres in the chiasma.

9. They also indicate that the papillo-macular bundle forms a little chiasma within the chiasma and that its fibres are similarly arranged.

My thanks are due to Professor Gulland, Dr. George Mackay, Dr. Sym, and Dr. J. V. Paterson, who very kindly handed over many of the cases to me for examination.

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(Figure 26 is adapted from an illustration in Hirsch's paper.)

THE TONOMETER OF SCHIÖTZ*

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SCHIÖTZ' tonometer is known to us all, and it is unnecessary to describe the instrument; but as to its method of application, there are one or two points to which I wish to draw attention.

Devised by Prof. Schiötz, of Christiania, in 1905, and first used in this country in 1911, there are now eleven years during which

*Read at a Meeting of the Midland Ophthalmological Society on Feb. 6th, 1917.