FIELDS OF VISION IN INTRACRANIAL LESIONS*

BY

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In the first place it is necessary to state clearly what is meant by the expression "fields of vision." Until recently the examination of the field of vision meant simply the demarcation of the extent of the seeing field and the position and shape of blind areas. Modern perimetry includes, in addition to the separation of blind from seeing areas, an estimation of the functional efficiency of the whole field. The normal field is not a field having merely normal extent, but one having, in addition, normal acuteness all over its area. When defects are found the degree of visual impairment must be estimated, and also the functional value of the apparently unaffected parts.

This *quantitative* perimetry is carried out by the use of serial stimuli varying from a test object of only 1 mm. in diameter to a strong electric light, on the same principle as Snellen's test card for central vision. In this way the "characters" of field defects are elicited. These are (1) position, (2) shape, (3) size, (4) intensity, (5) uniformity, (6) margins, and (7) behaviour or course. Two other features require investigation—namely, the presence of *proportion* or *disproportion* and, in cases of homonymous hemianopia, the presence of *congruity* or *incongruity*.

The examination of the field is usually carried out by moving test objects of different sizes along the arc of a perimeter or in front of an extensive black background, the screen or campimeter. Elaborate instruments have been devised, but frequently, and perhaps more especially in cases of intracranial lesion, the simple confrontation test, carried out by the examiner moving his hand or some object in front of the patient's face, provides information of the greatest value. This test is often omitted, not improbably on account of its simplicity.

It will be seen that quantitative perimetry demands much more than the mere demarcation of boundaries between grossly differing parts of the visual field. Only when full data have been ascertained are we in a position to proceed to the interpretation of the field changes and the diagnosis of their cause.

These characters require little explanation. The *intensity* of a defect means the degree of blindness present, and the *uniformity*, the relative distribution of the blindness within its area; the *margins*, the nature of the edges, whether abrupt or sloping. The examination of these characters constitutes *analysis* of the defect, and is very important. The blind half field in hemianopia, for example, may be found on analysis to consist of two quadrants of different intensities, or the composite nature of a central scotoma may be revealed. The *behaviour* includes the mode of onset and course.

Under normal conditions the fields for a coloured—for example, a red—object and a white object of the same size preserve a certain relationship in regard to area. When this is present in pathological conditions, as, for example, in concentric contraction of the field, *proportion* is said to be present. In certain abnormal conditions the field for the red object may be unduly small in proportion to the amount of reduction for white. This is a simple example of the presence of *disproportion*.

The nerve fibres from each retina are gathered into the optic nerve, and, as you know, at the chiasma separate

into the crossed and uncrossed bundles. In the optic tract the crossed fibres from the one side join with the uncrossed fibres from the other, and before, or by the time that, the external geniculate body is reached, the fibres from corresponding areas of the homonymous halves of the two retinae have come to lie in close juxtaposition. Thus a single lesion in the visual nerve pathway before this juxtaposition has been attained might produce a defect in one field only, or, when both fields are affected, defects which, though symmetrical in position and similar in shape, are more extensive, more intense, or earlier in appearance in one field than in the other. In cases of homonymous hemianopic field change defects of this kind are *incongruous*; *congruous* homonymous hemianopic defects are simultaneous in appearance, coextensive, and equal in intensity in the two fields. It will be seen that the establishment of congruity or incongruity has a localizing value, since all hemianopias due to supragenulate lesions must be congruous.

There is, however, an exception to this rule, owing to the fact that the temporal and nasal portions of the field are not normally coextensive, the temporal field being the larger by a crescentic area about 30 degrees wide at its widest part. This area corresponds to the nasal periphery of the retina, to the inner side of the visual path, and posteriorly to the most anterior part of the occipital cortex, and the nerve fibres concerned have no mates, since there are no corresponding temporal retinal areas. It is possible, therefore, in a supragenulate hemianopia, for the temporal 30 degrees of the binocular field to be affected alone, or for this part to be spared in an otherwise congruous homonymous hemianopia. Thus this isolation of the temporal crescent, or temporal half-moon as it is sometimes called, has a localizing value of its own.

When the field changes have been ascertained the next step is to interpret their meaning and to estimate their significance. Interpretation may be anatomical or pathological. By anatomical interpretation we gain information as to the site of the interference with the visual path, and by pathological interpretation we gain some guidance as to the nature of the lesion. These two forms of interpretation lead us towards the diagnosis, and, together with the behaviour of the field defects, assist us in connexion with prognosis. The same inferences based on the anatomical and pathological interpretation are often helpful in connexion with treatment—for example, in suggesting the best surgical approach to a tumour in the pituitary region. Finally, in patients who present certain symptoms suggestive of neoplasm, together with pronounced changes in the visual fields, an examination of the latter may be of use in determining their purely psychological origin.

We see, then, that perimetry may be of use in indicating the site and the nature of an intracranial lesion—that is to say, the diagnosis—and in helping us in connexion with prognosis and treatment. It is hardly necessary for me to emphasize the point that a diagnosis should never depend on perimetric findings alone, but that perimetric findings should be used as part of the clinical picture on which the diagnosis is based. Thus cases in which other symptoms are absent or minimal are those in which perimetric findings, if present, are of the greatest value. In other cases, in which the site and nature of the lesion may be evident on other grounds, the perimetric findings are always useful and sometimes valuable.

Turning first of all to the relationship of the field changes to the site of the lesion it will be noted that the long intracranial course of the visual pathway from the optic foramen to the occiput gives ample scope for the action of many forms of interference, while, in addition, some intracranial conditions not directly affecting the intracranial visual pathway produce effects on the optic nerves.

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Fortunately for the clinician the nerve fibre architecture of the visual pathway is so arranged that, on the whole, its elements retain the same relative positions throughout—that is to say, a lesion of one side of the visual pathway at any point, from the optic nerve back to the occipital cortex, will produce a defect on the opposite side of the field.

If we trace the visual pathway backwards from the optic foramen we find that different forms of field defect are produced at different levels, so that it is possible to obtain immediately from the fields a great deal of guidance as to which part of the pathway is involved. For our purposes the pathway may be regarded as consisting of a supragenicular part, including the cortex and the radiations, and a subgenicular portion, which is divided into subchiasmal, chiasmal, and suprachiasmal sections. All of these divisions give more or less characteristic field changes which enable the site of the lesion to be established.

We find also that the different parts of the visual path are liable in different degrees to different forms of lesion, so that if the site of the interference is established some guidance is already received as to the probable nature of the lesion. The establishment of the site is based upon the anatomical interpretation of the field defects. The nature of the lesion is based upon pathological interpretation. This form of interpretation depends chiefly upon the behaviour of the field change. Field changes which are rapid in onset, and progress rapidly or variably, correspond to lesions which behave in a similar way. Thus, if anatomical and pathological interpretation are taken together, it is often possible to gain from perimetry very considerable help in diagnosis.

The accompanying table shows in a general way, without going into great detail, the relationship between the different parts of the visual field and the more commonly occurring lesions and the field changes produced.

It is necessary to make special reference to one point in the anatomy of the visual pathway. It has hitherto

been believed that the visual fibres on leaving the external geniculate body pass directly backwards in a defined band until they spread out to their distribution in the calcarine area of the occipital cortex, the internal fibres passing to the anterior part of the cortex, the more external fibres to the posterior part. In recent years, however, a different view has been advocated, according to which the fibres of the radiation on leaving the geniculate body divide into two main groups—dorsal and ventral. Only the dorsal fibres pass directly backwards. The ventral bundle is said to pass downwards and forwards into the temporal lobe extending, in a fan-shaped arrangement, as far forwards as the tip of the anterior horn of the lateral ventricle, which it surrounds before turning backwards to the lower part of the visual area of the occipital cortex. This arrangement is known as Meyer's temporal loop. The evidence for this distribution is anatomical, and no clinical proof has yet been produced. If this distribution is correct a lesion of the temporal lobe, either in its upper or lower part if anteriorly situated, or in its lower part if in the posterior portion of the lobe, should produce a superior homonymous quadrantal defect in the fields; and, since the supragenicular part of the visual path is involved, the defects in the two fields should be congruous.

Surgical removal of the anterior part of the temporal lobe should produce a permanent, congruous, superior, homonymous quadrantal defect on the opposite side. It has been shown, however, by Cushing, that tumours of the temporal lobe produce incongruous homonymous hemianopic field changes which may disappear after operation. We are here faced with two alternatives. Either supragenicular lesions may produce incongruous field defects, a view which, if proved, would greatly disturb if not totally upset the present basis of the anatomical interpretation of homonymous hemianopia, or we must believe that these temporal lobe lesions affect a part of the visual pathway where interference is already known to produce incongruous field defects. Since the optic tract lies immediately beneath the temporal lobe

The Visual Pathway and Intracranial Lesions

				Field Defect	Usual Lesions
Extracranial	...	...	Optic nerve	Enlargement of blind spot. Concentric contraction. Binasal hemianopia.	Papilloedema due to increased intracranial pressure
Intracranial	Subgenicularate	Subchiasmal...	Optic nerve	Defects unilateral. Central scotoma. Hemianopic and quadrantic defects.	Tumour or abscess of frontal lobe (Foster Kennedy syndrome). Local tumour. Disseminated sclerosis. Trauma.
			Chiasmal	Anterior angle...	Quadrantic temporal scotoma (junction scotoma). Unilateral temporal hemianopia, passing into bitemporal hemianopia, or blindness of one eye with contralateral temporal hemianopia.
		Body		Bitemporal hemianopia.	Local tumour. Disseminated sclerosis. Syphilis.
		Posterior angle		Unilateral hemianopia passing into homonymous hemianopia.	Local tumour.
	Supragenicularate	Suprachiasmal	Optic tract	Homonymous hemianopia. Sparing of central area uncommon. Quadrant defects common. Defects frequently incongruous.	Local tumour. Temporal lobe tumour or abscess
			Optic radiation	Homonymous hemianopia. Sparing of central area common. Upper quadrant defects rare. Peripheral defects due to internal lesions. Central defects due to external lesions. Defects may be ill defined with sloping edges. All defects congruous.	Haemorrhage (?) Thrombosis. Tumour or abscess. Injury.
		Occipital cortex		Homonymous hemianopia. Sparing of central area common. Upper quadrant defects rare. Homonymous hemianopic scotomata. Double homonymous hemianopia with retention of central field. Peripheral defects due to anterior lesions. Central defects due to posterior lesions. Defects sharp-edged and well defined. All defects congruous.	Thrombosis. Tumour. Injury.

it seems justifiable to suppose that this is the part of the visual pathway which is affected, as the incongruity of the field defects would, on this assumption, offer no difficulty.

It is further evident that if cases can be found—and with the progress of intracranial surgery undoubtedly they will occur—in which the anterior part of the temporal lobe has been surgically removed, cutting through a large part of the temporal loop, definite and permanent upper quadrantal hemianopic field defects should be produced.

I have been able to obtain notes of two cases of this kind; one I saw in association with Mr. Dott, who will refer to it himself, the other was described by Kravitz. In this case the entire temporal and part of the frontal lobes on the right side were removed. Four months later an incomplete, irregular, incongruous, left homonymous hemianopia was found, which affected the lower as well as the upper left quadrants. Since the dorsal fibres of the radiation do not pass through the temporal lobe in Meyer's loop, the field, after such an operation, should exhibit a fairly clear-cut upper homonymous quadrant defect, and since the fibres were completely cut through the defects should be congruous, whether the fibres from corresponding retinal areas lie side by side at this level or not. No such field defect was present, and Dr. Kravitz, in a private letter, has informed me that he also doubts the existence of Meyer's temporal loop, at any rate as a portion of the visual pathway directly conveying visual impulses from the geniculate body to the occipital visual cortex.

In a recent study of the field changes produced by occipital tumours Horrax and Putnam showed that relatively congruous homonymous hemianopia with sparing of the central area was the characteristic feature. An upper quadrantal homonymous defect was not found in their series of occipital lobe tumours. The field changes in temporal lobe tumours differ greatly from these in the frequency of incongruity and of upper quadrantal defects, and the relative rarity of sparing of the central area.

These findings appear to me to afford considerable support to the view that it is the optic tract and not the optic radiation which is affected in temporal lobe tumours.

Turning now to the pathological interpretation of field defects, we have seen that anatomical interpretation has helped us considerably in this direction. A few examples will suffice. In disseminated sclerosis of the chiasma the predominant central position of the defects, their rapidity of appearance, variability, and spontaneous disappearance, agree with the symptomatology of disseminated sclerosis in general.

The slow, relentless development of hemianopic defects due to tumour pressure, the sudden or rapid appearance with possible spontaneous resolution of similar defects due to haemorrhage and thrombosis, are closely related to the development of the corresponding lesions. The presence of disproportion indicates activity of the lesion and progress either in the direction of increase or of resolution. The nature of the fields may be helpful in determining the best surgical approach, and the effects of surgical or radiological treatment may be gauged by repeated perimetric examinations.

The inconsistent and contradictory findings in hysterical and neurasthenic cases are sufficiently characteristic to indicate the true state of affairs.

The foregoing sketch is intended to illustrate the general principles on which the use of perimetry in connexion with intracranial lesions is based. Time does not permit of discussion in fuller detail.

SUMMARY

In the study of intracranial lesions perimetry is of value:

1. In locating the site of the lesion.
2. In assisting the determination of the nature of the lesion.
3. In affording guidance as to prognosis and treatment.
4. In throwing light upon the anatomy of the visual nerve path.

BIBLIOGRAPHY

- Meyer: "The Connexions of the Occipital Lobes and the Present Status of the Cerebral Visual Affections," *Trans. Assoc. Amer. Phys.*, 1907, xxii, 7.
- Traquair: *An Introduction to Clinical Perimetry*, second edition. London, Kimpton, 1931.
- Idem: "The Course of the Geniculo-calcarine Visual Path in Relation to the Temporal Lobe," *Brit. Journ. Ophthalm.*, 1922, p. 251.
- Kravitz: "The Value of Quadrant Field Defects in the Localization of Temporal Lobe Tumours," *Amer. Journ. Ophthalm.*, 1931, xiv, 781.
- Horrax and Putnam: "Distortions of the Visual Fields in Cases of Brain Tumour," *Brain*, 1932, lv, 499.

A NOTE ON VITAMINS IN PRACTICAL EXPERIENCE

BY

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In my paper to the Section of Diseases of Children at the Dublin Meeting of the British Medical Association,* dealing with the practical significance of vitamins in day-to-day clinical experience, I have confined my attention largely to the anti-rickets vitamin D, which, of course, in this country, is the factor of pre-eminent importance. A list of conclusions is, however, included relating to vitamins A, B, and C and certain other practical issues; and it is the purpose of the present brief notes to summarize the evidence on which these conclusions are based.

VITAMIN A NOT A GENERAL ANTI-INFECTIVE AGENT

Week by week advertisers' announcements are sent to us all by post, or appear in the journals, which rather convey the impression that vitamin A builds up the general resistance to infection and is of value in the treatment of toxæmias and infectious diseases of all kinds. This is claiming far too much. Vitamin A is anti-infective in only a limited way. As I have shown, in collaboration with J. R. M. Innes,¹ the local infections to which vitamin A deficiency gives rise are of a quite special type, being caused by structural breakdown of membranes. There is no change in the general immunity. These scattered, localized infections are restricted in origin to the abnormal membrane, and are set up by chance micro-organisms which would normally prove to be non-pathogenic. There is no reason to suppose that vitamin A could exert any direct bactericidal or similar effect when given in definite cases of infectious diseases and septicaemias caused by the invasion of specific virulent micro-organisms. Dr. Griffith and I showed,² for example, that vitamin A had no effect on the spread of experimental tuberculous disease. Again, our colleague Dr. Thomas Moore³ has found that in human beings dead from the most varied types of infectious diseases there may be abundant reserves of vitamin A in the body, so that little could have been gained by its administration in such cases, and it is obviously no longer possible to

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