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SUPPRESSION OF VISION IN SQUINT AND ITS ASSOCIATION WITH RETINAL CORRESPONDENCE AND AMBLYOPIA*

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Introduction

THE revival of orthoptic treatment of squint during the last seven years has brought an added interest to problems of binocular vision. Investigation of squinters with modern instruments—particularly with the illuminated amblyoscopes such as the “synoptophore”—has shown the astonishing frequency of an abnormality of binocular vision commonly called abnormal retinal correspondence, false macular vision or false projection.

For some time I have felt sure that there is some association, hitherto unexplained, between visual suppression and abnormal correspondence, and while suppression has long been considered the cause of amblyopia, no definite finality on this point has ever been reached.

The experiments described in this essay were done during the tenure of a Research Scholarship awarded by the British Medical Association, and from the findings I have attempted to reveal the

* The Report of a Research Scholarship awarded by the British Medical Association.

relationships that exist between abnormal retinal correspondence, visual suppression and amblyopia.

In addition I have drawn up a classification of the grades of normal and abnormal binocular vision. This is, I think, necessary if we would modify our ideas in the light of recent research.

I would like to thank the British Medical Association for giving me the opportunity of pursuing an interesting investigation, and for their permission to publish this work.

I also gratefully acknowledge the assistance given me by Dr. W. J. Penfold, the director of the Baker Research Institute. His great critical ability has been as much a help as his enthusiasm has been an inspiration.

Contents

	PAGE
THE NATURE OF SUPPRESSION IN SQUINT	578
THE COLOUR TEST	579
SUMMARY	585
THE MIRROR-SCREEN TEST	586
SUPPRESSION AND CONFUSION	595
SUPPRESSION IN ABNORMAL RETINAL CORRESPONDENCE	596
SUPPRESSION, RETINAL CORRESPONDENCE, AND VISUAL ACUITY ...	596
SUMMARY	598
THE GRADES OF BINOCULAR VISION	598
ABNORMAL RETINAL CORRESPONDENCE	600
NORMAL AND ABNORMAL BINOCULAR VISION	602
SUMMARY	603

The Nature of Suppression in Squint

Soon after I commenced squint-training, it became apparent to me that suppression was very marked in some cases, and was hardly present in others.

I had always been used to the idea that suppression produced defective vision, and thus expected to find suppression most marked in those cases where amblyopia was deep.

Now, while deep suppression clearly did occur in cases with defective vision, it soon became apparent that the cases of abnormal retinal correspondence showed the deepest suppression of all. And yet these patients commonly had good vision in each eye.

Clearly, then, suppression did not always produce amblyopia.

The problem was to reveal the relationships that existed between suppression, abnormal correspondence, and amblyopia.

SUPPRESSION FIELDS.

The methods I first used in the investigation of suppression were outlined in the Gifford Edmonds Essay for 1934¹. In this study they have been amplified.

Although it has been customary to say that "in squint the vision of the deviated eye is suppressed"—as if the impulses from the squinting eye were completely shut off—it has long been known that suppression is regional in extent—and not total.

In concomitant squint only one eye is "fixing," or holding the attention, and the squinting eye is directed elsewhere. Therefore, the term "squinting eye" of necessity implies that the other eye is fixing.

The areas in the field of vision of the squinting eye that are most actively suppressed are best measured by mapping what I have called a "suppression field." This is the field of vision of one eye, taken while the other eye is fixing.

It is clear that as suppression in one field only occurs when the other eye is fixing, both eyes must be open during the measurement of a suppression field. Therefore the first essential of such an experiment is a means of differentiating between the images seen by the two eyes.

THE COLOUR TEST.

One way of doing this is to place a coloured transparent celluloid shield over one eye. Objects seen by this eye will then be coloured, and objects seen by the other eye uncoloured. This method of differentiating between the images of the two eyes was described by Alfred Graefe²—and the adaptation of this method to the study of suppression was suggested to me by Dr. Ringland Anderson, and he carried out the initial experiments with me. A convenient name for this method is the "Colour Test."

I have described elsewhere¹ this method of studying suppression, and will repeat here only a slight modification of the method.

The setting up of the experiments is illustrated in Fig. 1. The patient is seated 1 metre from a Bjerrum Screen, his head supported on a Zeiss chin and forehead rest. A transparent red celluloid shield is placed over one eye. The patient looks at a small electric lamp pinned on the screen. The position of the light is so arranged that while the covered eye is directed at it, the other eye is directed at the central point of the screen. This is, in practice, effected as follows:

Suppose the patient has a convergent squint of 20° and the right eye is covered with the red glass—as in Fig. 1. The light is placed on the 20° circle of the screen—to the left of the central point. The right eye will now be directed at the light, and the left directed at the central point of the screen. To check the

FIG. 1.

To illustrate the setting up of the "Colour Test." The patient is seated 1 metre from the screen, and has a red glass over the right eye. As long as the light appears red, the right eye must be the fixing eye, and thus the left must be the squinting eye.

accuracy of this adjustment, one covers the right eye with a small card, and directs the patient to look at the central point. One then rapidly moves the card to the left eye, at the same moment directing the patient to look at the light. If the adjustment is accurate there will be no movement of the eyes when this is done, as the light will be "in line" with the right macula, and the central point of the screen "in line" with the left macula. If the adjustment is inaccurate, the eyes will be seen to move as the

card is moved from one eye to the other. The light is then adjusted until no movement is seen. (The position of the light on the screen then gives the angle of squint—measured at 1 metre).

If the patient now looks at the light with the right eye, it will be seen as a red light, and as long as the light remains red, the right eye must be the fixing eye, and thus the left eye must be the squinting eye.

A 1° white test object (as supplied by Bausch and Lomb) was now moved over the screen in the usual way.

If the object appeared red, it was perceived by the fixing eye—if white, by the squinting eye. If a red and white test object were seen, it was perceived by both eyes.

A constant finding of experiments conducted on squinters by this method was for the test object to appear red over a varying area around the light (Fig. 2). As the object was moved away from the light, its "redness" gradually faded, and it finally became white. Sometimes this transition was gradual—sometimes so definite that the patient would say "red," "pink," "white" (Fig. 3). Often there was an intermediate zone of diplopia or "two spots" (Fig. 4).

FIG. 2.

Typical suppression scotomata by the Colour Test. The cross on the 25° line indicates the position of the fixation light. The test object appeared red over the areas indicated, and white beyond them.

FIG. 3.

A suppression scotoma in each field, with a definite gradation in the degree of suppression.

FIG. 4.

A suppression scotoma in each field—surrounded by a zone of diplopia.

FIG. 5.

This shows how the scotoma changes position with the direction of gaze. The patient has a convergent squint of 15° . In the right field the fixation light was placed in three different positions, 1, 2, 3, and a similar scotoma was found in each position.

This coloured area represents either a scotoma in the squinting field, an area in the fixing field which is holding the attention, or more probably an area in the binocular field where the fixing eye is completely dominant.

The area varies in position according to the direction of gaze (Fig. 5). It does not vary appreciably with the size of the stimulus nor with the intensity of illumination. (This was repeated in many cases varying the "size" of the stimulus from $1/4^\circ$ to 2° , and the illumination from 4 to 10 foot-candles.)

FIG. 6.

This shows how the scotoma is larger in the eye with worse vision. This point is also illustrated in Figs. 2, 3, 4. In Figs. 1 and 5 the visual acuity of the two eyes was the same, with scotomata of almost the same size.

The coloured area or scotoma was usually larger in the eye with worse vision (Fig. 6). There was usually an alteration in the size of the scotoma associated with an alteration of visual acuity in individual cases (Fig. 7). There appeared to be no constant relation between the size of the scotoma and the visual acuity in

FIG. 7.

The two charts, both from the same case, show how the scotoma may alter in size, with an alteration in visual acuity.

different cases, so that one could not say that a scotoma of such a size was associated with such a visual acuity.

In my first experiments conducted with a perimeter I was sometimes able to show a second scotoma in line with the squinting macula (Travers¹). In these experiments I did not see this scotoma, although I looked for it frequently.

INTERPRETATION OF COLOUR TESTS.

These experiments show, to my mind, an area in the binocular field where the fixing eye is completely dominant.

This means that a squinter, when fixing an object, uses only his fixing eye over an area, roughly circular in shape, which has the fixing macula as its centre. Corresponding in visual direction with this area is a suppression scotoma in the squinting field.

The suppression in the squinting field is deepest in the same direction as the fixing macula, and as objects move away from the macula, the suppression becomes less marked. This is shown by the colour change that occurs—the spot appearing deep red in the centre of the scotoma (fixing eye predominating entirely) then becoming paler (as the impulses from the squinting field

approach more nearly to the potency of those from the fixing eye).

As suppression becomes less marked, there is an area of diplopia where the two monocular sensations are equal in potency. Beyond this area, the test object is well away from the fixation light, and is not related to the fixation object. It is thus not "coloured"

FIG. 8.

To illustrate the relationship between the area where the visual acuity is best in the fixing eye, and suppression most marked in the squinting eye. In the upper half of the diagram we see how the right macula of the fixing eye is in the same visual direction as the eccentric point A in the squinting eye, when the right eye is fixing a distant object O. The lower part of the diagram illustrates the relationship between the variations in visual acuity of the right retina and the areas of suppression in the left retina. Above the graph illustrating the variation in suppression, is a diagrammatic representation of a typical suppression scotoma, showing a central red area surrounded by a zone of diplopia.

by the field of attention, and so appears white. This alteration in the intensity of suppression is well illustrated by a diagram (Fig. 8).

The colour test shows how *diplopia* is prevented in squint. The brain is presented with two monocular images of the fixation object, one from the fixing macula which is clearly imaged upon

the very sensitive macula. The other, distorted by axial astigmatism, is formed upon the insensitive peripheral retina. In these circumstances, as Sherrington³ has shown, fusion of the two images is impossible (quite apart from their different space projection), and so the dull image is suppressed. The colour tests merely show how this suppression is effected, and it should be noted that this type of suppression is unrelated to retinal correspondence. The colour tests obtained from patients with normal and abnormal correspondence are the same.

SUMMARY.

The foregoing may now be briefly summarised :

1. The Colour Test for suppression is described. It demonstrates a functional scotoma in the deviated eye of squinters.
2. This scotoma surrounds a point which lies in the same visual direction as the fixing macula.
3. This phenomenon may equally well be described as a scotoma in the squinting field or an area of monocular dominance in the binocular field.
4. The scotoma alters its position with the direction of gaze.
5. It is present in whichever eye is squinting at any particular moment. When that eye holds the attention the scotoma ceases to exist and is transferred to the other eye.
6. The scotoma varies in size—is usually roughly circular in shape—and usually bears some relationship to the visual acuity of the eye in question.
7. This relationship is as follows: If the visual acuity of the two eyes is not the same, the scotoma will be larger in the eye with worse vision. However, a certain sized scotoma bears no constant relationship to any particular visual acuity in different cases.
8. The size of the scotoma is not apparently affected by an alteration in intensity of illumination of 4 to 10 foot-candles—of a variation in size of stimulus of a $1/4$ to 2 degrees viewed at 1 metre.
9. The scotoma is not uniform in density. Suppression is most marked at its centre and fades towards its periphery.
10. This type of suppression is not related to retinal correspondence.
11. An interpretation of the findings is given. The colour test shows how a squinter avoids diplopia.

THE VARIABILITY OF SUPPRESSION.

Now it is clear that suppression is a functional condition, and thus it will vary a great deal. A simple observation will serve to illustrate this point.

A.B., a boy aged five years, had a convergent squint of 20°. He had 6/6ths vision in each eye. He habitually "used" the right eye and "turned" the left. On the synoptophore his retinal correspondence was normal with good amplitude of fusion and depth perception of fairly easy stereoscopic slides. He had no diplopia.

Now, if he has no diplopia in everyday life, he must be suppressing the vision of one eye, and yet on the synoptophore no suppression can be demonstrated.

Therefore he "suppresses" in certain circumstances, and does not do so in others.

This brings us to a very important point. Suppression varies according to circumstances.

Suppression in everyday life, to avoid *diplopia*, is easy because, as explained above, the two monocular images are of such a different order of clearness. This type of suppression is easy to understand, and its mechanism has been illustrated in the colour tests.

The patient A.B., just described, does not suppress with the synoptophore because, when he is looking into the instrument, two pictures are presented simultaneously to his two maculae—not to one macula and one eccentric retinal area. Thus, with the synoptophore, two areas of similar visual acuity are stimulated at the same moment, and so suppression is much more difficult (note Sherrington's experiments³).

In the course of ordinary squint-training, the patients who are found to have the deepest forms of suppression are those with defective vision, and those with abnormal correspondence.

To study this question further I then devised a second method of mapping suppression fields. This, once again, was a way of differentiating between the images seen by the "fixing" and "squinting" eye.

THE MIRROR-SCREEN TEST.

The method consists of separating the field of one eye from the other by means of a plane mirror placed obliquely in front of one eye. It is conveniently called the "Mirror-Screen Test." It is best understood by a diagram (Fig. 9).

The patient is seated in front of the Bjerrum screen in the usual way, and a mirror is placed as indicated in front of say, the right eye. A second black screen B is arranged on the patient's right side. This screen should be large enough to cover the whole field seen in the mirror, and the patient will then project this black screen as if it were on the Bjerrum screen. A small electric light placed on the screen B, will thus appear as if it were on the Bjerrum screen.

Now it is clear that if the electric light on the screen B is fixed by the right eye, its light will strike the mirror, and be reflected on to the right macula, as in Fig. 9. This, I have described, as the light being "in line" with the right macula.

If the light is suitably arranged it can be so placed on the screen B that while it is "in line" with the right macula, the centre of the Bjerrum screen, A, will be in line with the left

FIG. 9.

To illustrate the setting up of the "Mirror-Screen Test." The patient is seated 1 metre from the screen, and a mirror is placed obliquely in front of his right eye. The left macula is directed towards the centre of the Bjerrum screen A. The fixation light on the screen B will be projected as if it were on the screen A.

The figure illustrates the manner in which all the experiments were arranged—with the fixation light on B, "in line" with one macula, and the centre of the screen A in line with the other. If the retinal correspondence is normal the fixation light will be projected as if it were at A.

macula. This arrangement is best effected by placing a small fixation object, such as a small white cross, on the centre of the Bjerrum screen, and telling the patient to look at it. The left eye is then momentarily covered with a card, and the patient told to look at the light. If a movement of the right eye occurs, the light is adjusted in position, and the procedure repeated until no movement of the eyes occurs when the patient looks from the cross to the light. This technique is quite easy to perform with a little practice, and an ocular movement of 1 degree can be easily recognised. The covering and watching of the eyes is just the

same as in the screen test for detecting muscle-balance anomalies described by Duane⁴ and White⁵.

If we refer again to Fig. 9, we may see how these experiments provide a very interesting way of studying retinal correspondence. If the apparatus is so arranged that the light on the screen B is in line with one macula and the centre of the Bjerrum screen is in line with the other, then if the patient's retinal correspondence is normal, the light will be projected as if it were on the centre of the Bjerrum screen.

Therefore, if the apparatus is set up in this way, and the light is not seen on the central point of the Bjerrum screen, the retinal correspondence is abnormal, and the angle of anomaly may be measured directly by noting the position on which the light is projected. Thus, if the light were seen as if it were say, on the 10 degree line, the angle of anomaly would be 10 degrees.

The setting up of these experiments was as follows:

The patient was arranged sitting 1 metre from the screen in the usual way, with his head supported on a Zeiss chin and forehead rest. The mirror was placed in position and the apparatus adjusted so that a small cross in the centre of the Bjerrum screen was "in line" with the left macula, and the light was moved until it was "in line" with the right macula (as described above). The patient was directed to watch the light, and the cross was then removed from the centre of the Bjerrum screen. The apparent position of the light on the Bjerrum screen was then noted—this telling one whether the retinal correspondence was normal or abnormal. A $1/4^\circ$ white test object (supplied by Bausch and Lomb) was then moved over the Bjerrum screen, and any areas where it disappeared were noted.

When the test had been completed with the right eye fixing the light, the screen B and the mirror were moved to the patient's left side and the procedure repeated, the left eye now being the fixing eye and the right the squinting eye.

Apart from the blind spot there are no blind areas normally present in the 30 degree circle which the screen subtends, and any other areas of disappearance were taken to be evidence of suppression.

It will be readily appreciated that these experiments require a considerable amount of concentration and power of attention on the part of the subject, and I was not able to perform them satisfactorily on any young children. The youngest child who gave reliable information was a boy of six years. I found that the ability to recognise the normal blind spot on the Bjerrum screen was a very reliable guide to the patient's powers of attention. So, at the commencement of each experiment, an attempt was made to map out the normal blind spot. If the patient could

not appreciate this blind area, he was rejected as unsuitable. This simple test was suggested by my nurse; Miss J. Strahle.

A series of tests was first performed on three normal persons, all of whom had good vision in each eye, no squint, and stereoscopic vision.

In all these individuals the findings were the same. The fixation light was projected as if it were on the central point of the Bjerrum screen, and the spot could be clearly seen passing through the light. There was no disappearance of the spot, except over the normal blind spot. These findings were the same, whichever eye was used for fixation.

The results of these experiments are best considered under three headings:

1. Normal persons with good visual acuity in each eye, normal retinal correspondence and stereoscopic vision.

2. Squinters with normal retinal correspondence and either good or bad vision in one eye.

3. Squinters with abnormal retinal correspondence.

(N.B.—The term squinter includes those patients who had a squint but are now clinically cured.)

1. Normal persons.

With the apparatus set up in the usual way, the fixation light will be projected as if it were upon the centre of the screen and the test object will be seen to move freely through the light. It will only disappear over the normal blind spots.

2. Squinters with normal retinal correspondence.

(a) If the visual acuity of the two eyes is the same, there will probably be no suppression shown at all. However, in some cases there may be a little fluctuating suppression as shown in the following case:

Dr. B. had 5° of convergence, 6/9ths vision in each eye and stereoscopic vision. As the spot moved up to the light it faded, and occasionally vanished as it passed through. Sometimes it was the *light* that faded or disappeared as the spot moved over it.

It should be noted that this fluctuation in the intensity of the light or spot only occurred as one moved over the other. As soon as they were separated, both were seen distinctly. This type of alternating macular suppression is often observed on the synoptophore, and is only a slight accentuation of normal retinal rivalry.

(b) If the visual acuity of one eye is low, a much deeper suppression is seen. This is exemplified in the following case:

S.W. had a right convergent squint of 10° and normal retinal correspondence. Right vision was 6/60ths and left vision 6/6ths. The fixation light was projected as if it were on the centre of the screen.

With the left (good) eye fixing the light, the spot disappeared over a circular area of about 7° in diameter having the light as its centre (Fig. 10).

FIG. 10.

This shows two areas of suppression over the macular area. In the left field, the light disappeared as the spot moved over it. In the right field, the spot disappeared as it moved over the light.

With the right (defective) eye fixing the light, the *light* disappeared over a similar area. The light only disappeared as the spot moved over it, and reappeared as the spot moved away.

This disappearance was absolute. The child said it was as if the light "went out."

This suppression was so intense that the light still appeared to "go out" in the same way, even though the fixation light was made too bright to look at comfortably.

The size of this type of suppression scotoma, unlike that seen in the "colour tests," may be made to vary in individual cases.

Thus, suppose there is a 5° scotoma around the light using a $1/4^{\circ}$ white spot. If the fixation light is made brighter, the scotoma around it will usually be larger than if it is dulled. Contrariwise, if a 1° spot is used instead of a $1/4^{\circ}$ spot, the scotoma will be smaller.

Again, in cases where the visual acuity of the two eyes is nearly equal, one may obtain a reversal of the type of suppression one would first expect.

Thus, V.R. with normal retinal correspondence had right vision $6/6$ ths and left vision $6/12$ ths. With the right (good) eye fixing the light, a $1/4^{\circ}$ white spot usually vanished as it moved through the light. If, however, a 2° white spot was used, it was found that the light occasionally disappeared as the spot moved over it. This effect could be enhanced if the patient concentrated her attention on the spot, and tried to disregard the light. (The effect of attention on the predominance of one macula is well known (Helmholtz⁶).)

These experiments show well how extremely intense is the suppression that is associated with defective vision. They also show in a striking manner how a regional suppression is brought into being by the act of fixation of the other eye.

So far as I know, this interesting phenomenon has not been described before.

Thus, in the patient S.W., the visual acuity of the defective eye was sufficiently good to read fairly small print—but only as long as the good eye was closed. As soon as the impulses from the macula of the better eye came up into consciousness, the macula of the worse eye was greatly reduced in acuity. It was, indeed, virtually blinded, as the patient could not perceive light.

These experiments show that a functional macular scotoma is produced in the squinting eye by the act of fixation of the fixing eye.

Evans⁷ in a series of experiments conducted on the Lloyd stereo-campimeter—sought to correlate the amblyopia of squint with the size of a scotoma found in cases of defective vision.

The experiments just described suggest that Evans' scotoma was a suppression scotoma. Although there may also have been an absolute scotoma present, the *binocular* nature of his experiments prevented proof of this. In his cases, a transitory and functional scotoma was measured, which may have had no existence as soon as the eye in question was fixing. He measured a scotoma which probably only existed while the eye in question was "squinting." He then attempted to correlate the size of this scotoma with the reduced visual acuity, measured of necessity when the eye was fixing.

3. Patients with abnormal retinal correspondence.

With these patients, the fixation light of the "mirror-screen" will not be projected upon the central point of the Bjerrum screen,

FIG. 11.

This shows two large suppression scotomata in the temporal fields surrounding the position where the light is projected. The patient had a convergent squint of 40° with an angle of anomaly of 25°. The scotoma in the left field is separated from the normal blind spot, but in the right field includes it.

but a varying distance away. As mentioned above, the position of the light gives a direct measurement of the angle of anomaly.

With these patients a much larger area of suppression was usually observed (Fig. 11).

Using the $1/4^\circ$ spot, the spot was found to vanish over a varying area around the light. This area varied in size from about 2° in diameter to well over 20° in diameter.

It was found, as a general rule, that the larger the angle of anomaly, the larger the area of suppression.

FIG. 12.

This shows suppression scotomata in the nasal fields. The patient had a divergent squint of 30° with an angle of anomaly of 25° . This field was taken before operation. A field taken 20 minutes after the bandages had been removed after operation was exactly the same, although the angle of squint was 0° .

These fields are from the same patient as in Fig. 12. Fig. 13 was taken one week after operation. The angle of squint was 2° of convergence. Angle of anomaly was 12° and the suppression scotomata were about 4° in diameter.

In Fig. 14—six weeks later—the angle of squint was 2° of divergence. Angle of anomaly was 10° and the scotomata about $2\frac{1}{4}^\circ$ in diameter.

The probable conclusion of this important finding is that the suppression scotoma would become smaller and smaller and finally vanish when the angle of anomaly was reduced to zero and correspondence became normal again. This change in the size of the scotoma, associated with an alteration in correspondence after operation is typically seen in Figs. 12, 13, 14.

Another example of this condition is seen in patients whose angle of anomaly is different according to which eye is fixing. A marked case of this variety is illustrated in Fig. 15. The same phenomenon, only much less, is seen in Fig. 16.

FIG. 15.

This patient previously had 40° of convergence. The field was taken two months after operation. There was then 2° of convergence. The angle of anomaly was 2° with the right eye fixing, and 15° with the left eye fixing.

FIG. 16.

This patient had a divergent squint with an angle of anomaly of 22° with the left eye fixing, and 26° with the right eye fixing. There was a bilateral weakness of the superior recti, and overacting inferior obliques. Note that the suppression scotoma in the left field is slightly larger than that in the right, presumably because in the former the angle of anomaly is larger.

If the squint is convergent, the scotoma is in the temporal field, if divergent, in the nasal field. There is often a vertical component in the angle of anomaly if the squint itself has a vertical component (as in the cases complicated by weak superior recti and overacting inferior obliques (Fig. 16).

Using the mirror-screen, a typical suppression scotoma could always be found in cases of abnormal correspondence. It appeared to be immaterial whether they could or could not superimpose monocular images on the synoptophore (Fig. 17). This, I think,

FIG. 17.

This patient had a convergent squint of 20° . With the synoptophore the butterfly appeared in the net at 10° . The mirror-screen shows a bilateral suppression scotoma surrounding the normal blind spots.

proves that there is no essential difference in the various groups of abnormal correspondence described by Miss Mayou⁸, and justifies one in saying that every case which has not got normal correspondence has, in fact, abnormal correspondence. This point will be referred to later.

INTERPRETATION OF MIRROR-SCREEN EXPERIMENTS.

Before considering the interpretation of these experiments, let us look for a moment at a stereoscopic slide that illustrates very well some of the phenomena of normal retinal rivalry. This is the slide (Fig. 18).

If an attempt is made to combine the two parts of this picture in a stereoscope, a very striking fluctuating type of picture is seen. This slide illustrates normal retinal rivalry. The variations in the

FIG. 18.

This stereogram illustrates some of the features of retinal rivalry.

composite picture seen are due to alternating macular suppression. As the gaze moves over the picture, first one and then the other set of lines is seen. The *fluctuating* nature is very striking.

Now in persons whose binocular vision is almost normal, there may be a slight accentuation of this alternating macular suppression (as in the case of Dr. B. described above).

In persons with normal retinal correspondence and amblyopia, however, we see a marked accentuation of the suppression. But the suppression is still a result of binocular vision, and is only seen in the defective eye as a result of the fixation of the better eye.

If the retinal correspondence is abnormal, we see a much larger area of suppression in the squinting field. In these cases, however, the suppression is not over the macula, *but over the area where the fixation light is projected.*

REASON FOR SUPPRESSION, AND DESCRIPTION OF CONFUSION.

When an eye deviates there are two phenomena that must be prevented if the patient is to have comfort. These are :

1. Diplopia.

This is easily prevented, as described above, and the suppression of the peripheral retina which it effects is illustrated in the "Colour Test."

2. Confusion.

This phenomenon is not usually considered at all, and I can find no mention of it in the usual text-books. It is of extreme importance in squint. The term "confusion" was originally used by van Kries⁹.

A confusion is usually produced by impulses from the *macula* of a deviated eye rising up into consciousness. The symptoms are best illustrated by a typical case :

J.W. commenced to squint suddenly when 8 years old. The squint was at first paretic and then became clinically concomitant. The diplopia soon passed away, as the vision of the deviated eye was suppressed. Occasionally, however, particularly when the child was running, a "confusion" was noticed. In these circumstances, the rapid movement of images over the deviated macula overcame the habitual suppression, and these images came up into consciousness. They were interpreted by the patient as if they came from the "straight ahead" position (*i.e.*, the "macula" direction). Thus the patient said that as she was running, the fence "suddenly swung round in front of her, and she felt as if she were going to run into it."

Confusions are occasionally noticed after corrective operations for squint. Often a statement that the patient "sees things double" will, on careful investigation, prove to be a confusion.

Another common manifestation of a confusion is the subjective blur that patients with a recent macular lesion will notice "getting in front of them" when they are reading. They are unable to suppress the blurred image from the injured macula.

Confusion is prevented by macular suppression—if correspondence is normal, and by suppression of the corresponding eccentric retinal area, if correspondence is abnormal.

There is thus an essential unity in the purpose of suppression in squint—whether the correspondence is normal or abnormal.

For in normal correspondence the suppressed area is the squinting macula, and in abnormal correspondence, the suppressed area is the eccentrically placed portion of retina which corresponds in projection to the fixing macula.

REASON FOR DEEP SUPPRESSION IN ABNORMAL RETINAL CORRESPONDENCE.

This gives us the clue to the reason for the deep suppression clinically observed in abnormal correspondence cases.

The suppressed area in *normal* correspondence is an area of normally high visual acuity (the squinting macula). Therefore, one expects and, indeed, finds very little suppression—unless the visual acuity of the squinting macula is low.

The suppressed area in *abnormal* correspondence is an area, always of low visual acuity (the visual acuity of the retina a few degrees away from the macula is very low). Therefore, one expects, and does in fact find, deep suppression.

This explanation of the deep suppression found in abnormal correspondence is confirmed by the different sized scotomata seen with varying angles of anomaly. The suppression scotoma is large when the angle of anomaly is large (because the suppressed area is in the peripheral retina where the visual acuity is very low). When the angle of anomaly is small, the scotoma is small (because the suppressed area is nearer the squinting macula, and the visual acuity is higher). If the angle of anomaly falls to zero (as correspondence becomes normal) there will be no suppression—unless, of course, the visual acuity of one macula is low. (Compare Figs. 12, 13, 14, 15).

RELATIONSHIP BETWEEN SUPPRESSION, RETINAL CORRESPONDENCE AND VISUAL ACUITY.

The Mirror-Screen experiments have shown how a regional suppression in one eye is produced by the act of fixation of the other eye.

Thus in squint, the act of fixation with the better eye produces an act of suppression in the deviated eye.

If retinal correspondence is normal, this suppression is over the squinting macula. If retinal correspondence is abnormal, it is over an eccentric part of the retina.

Now consider the effect of these suppression scotomata upon the developing vision of a child.

When correspondence is normal, every act of fixation will produce a scotoma over the *macula* of the deviated eye. One would expect the visual acuity of such an eye to become worse.

But, if correspondence is abnormal, fixation will not produce a scotoma over the deviated macula, but over an eccentric part

	Number of Cases	Average Age of Onset of Squint	Percentage of Cases having Equal Vision
Normal Correspondence	148	3.6 YRS.	48 %
Abnormal Correspondence	132	2.0 YRS.	64 %

FIG. 19.

of the retina. Therefore, one would not expect the visual acuity of such an eye to be impaired.

Thus, one would expect squinters with normal correspondence to have worse vision than those with abnormal correspondence.

To test this theory I analysed a series of cases of squint. They were first divided into two main groups—normal and abnormal correspondence. Then, each group was classified under the following headings:

1. Age of onset of squint.
2. Visual acuity, with glasses—equal or unequal.

The visual acuity was taken when the patient first came for consultation—not after any treatment for amblyopia had been given. Snellen's test types were used for testing visual acuity, and the Bishop-Harman E. test if they were illiterate. "Equal" means that the patient could read letters of the same line with either eye. Thus 6/9ths and 6/9ths partly were taken as equal. All these cases were examined under atropine, and their fundi and media were normal.

The results are best seen in a table (Fig. 19).

The average age of onset of the groups is 3.6 years in the normal correspondence and 2.0 years in the abnormal correspondence cases.

Now, it is well known that the earlier in life a squint commences, the more likely is amblyopia to develop.

Yet you have here a group of cases whose age of onset is 2.0 years with considerably better (16 per cent. better) vision than a group whose average age of onset is 3.6 years.

Almost certainly the reason for this striking difference is that abnormal correspondence stops macular suppression, and thus prevents amblyopia.

It has long been wondered why some cases of squint develop amblyopia, and others do not. This theory gives an adequate explanation for many of these hitherto puzzling cases.

SUMMARY.

The foregoing may now be briefly summarised:

1. The variability of suppression is mentioned.
2. A new method of mapping a suppression field is described. This is called the Mirror-Screen Test.
3. This method measures suppression when the two maculae are stimulated at the same moment.
4. The tests show that a regional suppression in one eye is produced by the act of fixation of the other eye.
5. Suppression in the squinting eye always occurs over the area that corresponds in projection to the fixing macula.
6. Thus, suppression is over the squinting macula if retinal correspondence is normal, and over an eccentric area if retinal correspondence is abnormal.
7. Suppression varies inversely with the visual acuity of the suppressed area. Thus, it is least if the suppressed area is the macula—with high visual acuity—and more marked if the visual acuity of the macula is lowered by amblyopia. The largest suppression scotomata are seen in cases of abnormal retinal correspondence with a large angle of anomaly where the suppressed area is in the peripheral part of the retina.
8. An interpretation of the experiments is given.
9. The phenomenon of confusion is described.
10. An explanation of the depth of suppression in abnormal correspondence is given.
11. The relationships between amblyopia, suppression and retinal correspondence are revealed.
12. It is shown that abnormal correspondence prevents the development of amblyopia.

The Grades of Binocular Vision

WORTH'S CLASSIFICATION.

C. Worth¹⁰, the pioneer of squint-training in Great Britain, classified binocular vision into three grades:

1. Simultaneous macular perception.
2. True fusion with some amplitude.
3. Sense of perspective—(perception of depth).

Worth did not describe any abnormal forms of binocular vision, and the only probable reference in his book to abnormal retinal correspondence is contained in the statement that "essentially alternating squinters have no binocular vision." Worth's investigations were done with the amblyoscope, which has now been superseded by the synoptophore and similar machines. These are essentially illuminated amblyoscopes. The illumination is very important as it produces a bright reflection of light on the corneae of the patient who is looking into the instrument. The position of the "corneal reflexes" allows one to check up on the patient's observations.

INVESTIGATION OF THE STATE OF BINOCULAR VISION.

The investigation of binocular vision with a synoptophore is conducted as follows:

The patient is seated at a table looking into the eye-pieces of the instrument. Two slides of the "simultaneous perception" variety—such as the "butterfly" and the "net" are placed in the lanterns.

The instrument is adjusted so that there is no movement of the eyes when the patient looks first at the centre of the net and then

FIG. 20.

This shows a patient looking into the synoptophore. The instrument is set at the angle of squint, and in this position the image of the butterfly is falling on the right macula, and that of the net on the left macula. If the two maculae are corresponding points, the butterfly will be seen by the patient as if it were inside the net.

at the butterfly. The instrument is now set at the true angle of squint, because the image of the butterfly will be falling on one macula and that of the net upon the other (as in Fig. 20).

Now, if retinal correspondence is normal, the two maculae are corresponding points, and thus the patient will see the butterfly and net as if they were superimposed—*i.e.*, the butterfly in the net.

Contrariwise, if the instrument is set up in this way, and the butterfly appears to be outside the net, the retinal correspondence must be abnormal.

If the retinal correspondence is normal, then one may proceed to investigate fusion with fusion slides—eliciting the amplitude of fusion. The final stage is the “stereoscopic vision” (a more accurate term than sense of perspective). This is conveniently tested with graded stereoscopic slides.

In this regard it is worth while noting that perception of depth is not dependent solely upon binocular vision (Duane¹¹)—and thus such tests as Hering’s drop test do not give proof of that which one is really seeking in squinters—*i.e.*, the ability to combine two slightly dissimilar images and so produce an *illusion* of depth.

ABNORMAL RETINAL CORRESPONDENCE.

If abnormal retinal correspondence is present, it will be found that the patient will either be able to “put the butterfly in the net” at some angle other than the true angle of squint or not be able to put the butterfly in at any angle. Of the two, the latter is the usual state of affairs. Some authors (Mayou⁸) have differentiated between these two types of case as if they were different varieties. Thus, the former group were said to have “true abnormal retinal correspondence,” and the latter group “total suppression.” Miss Mayou says of this latter group “it is presumed that the area of suppression is so large that he is unable to perceive the images at the same time without alternation.”

The term “total suppression” is very misleading, as the suppression is not total, but regional. In addition, the mirror-screen tests have shown that these two groups are essentially the same, and that all these cases may be accurately described as having an abnormal retinal correspondence.

The essential values one must know before assessing retinal correspondence are:

1. The angle of squint.
2. The angle of the patient’s “binocular projection.” (I have used this term to describe the angle between the space projection of the visual line of the fixing eye and the space projection of a

line passing through a point a known angular distance away in the squinting eye.)

These two values should be the same, and any difference between the two indicates an abnormal correspondence. The angular difference is called the angle of anomaly (Bielschowsky¹²).

MEASUREMENT OF ANGLE OF ANOMALY.

One may measure the angle of anomaly in a variety of ways.

1. By the synoptophore.

If the patient can put the butterfly in the net at some angle other than his true angle of squint, one obtains a direct measurement. Thus, suppose his angle were 40° and the butterfly were seen "in the net" at 30° , the angle of anomaly would be 10° .

2. By the mirror-screen.

As described above, one obtains a direct measurement of the angle of anomaly by this method.

3. By the Maddox-rod.

One uses the Maddox-rod in the usual way. Thus, suppose the angle of squint were 30° and the light and streak subtended an angle of 15° at the eye, the angle of anomaly would be 15° . Muegge¹³ used this method, and differentiated between those cases where the angle of squint and angle of anomaly were equal (which he called harmonious) and those whose angle of anomaly was less than the angle of squint (called unharmonious).

4. By the diplopia method.

The angle of squint may be 20° , but when diplopia images are induced, the images may only subtend an angle of 10° (angle of anomaly of 10°).

5. By the measurement of the projection of after images.

Tschermak¹⁴ used this method, and devised an apparatus called the Kongruenz Apparat. This was later modified by Chavasse¹⁵. Hering¹⁶ described a similar method in 1863.

Now the angle of anomaly will be found to vary very greatly according to the method used for measuring it, and some persons may be found to have normal correspondence when measured by the synoptophore and yet appear to have an abnormal correspondence when measured by other methods. (For example, a girl who had a squint of 20° , had normal correspondence with the synoptophore and yet her diplopia images were only 8° apart—suggesting an angle of anomaly of 12°).

Because of these seeming variations in the measurements, I was of the opinion that abnormal correspondence was not a fixed relationship, as is normal correspondence (Travers¹⁷).

But, if one reflects, one will see that this is an unwarrantable assumption, as the methods used for measuring the angle of anomaly are not comparable.

Thus, by the synoptophore and mirror-screen one measures the angle between the projection lines of the *two maculae* (points of similar visual acuity).

By the Maddox-rod and diplopia tests one measures the angle between the projection line of one macula and an eccentric point in the other eye (one is here comparing the projection of a point of high with one of low visual acuity).

Therefore one should not draw conclusions regarding the *instability* of anomaly angles from these tests.

Tests with the mirror-screen show that the angle of anomaly in an individual case does not vary from week to week.

Retinal correspondence is of clinical importance only in relation to fusion—that is, fusion of *macular* images. Therefore I think that abnormal correspondence should only be diagnosed by a method which involves measurement of the projection lines of *both maculae*. The synoptophore and mirror-screen are two such methods.

Scheme of Binocular Vision

It is clear that, with recent research in squint, some alteration of Worth's classical grades of binocular vision is needed. It is simpler to give a different classification than to amplify his groups.

FIRST STAGE OF BINOCULAR VISION.

The first stage of binocular vision is clearly "simultaneous perception." Reverting to the testing of binocular vision with a synoptophore, if the patient can see the butterfly at the same time as he sees the net, he has "simultaneous perception." This term is accurate, and makes no reference to the correspondence.

FIRST GRADE OF NORMAL BINOCULAR VISION.

There are various authors (Mayou⁸, Lyle and Jackson¹⁸) who describe sub-groups of the first grade of binocular vision. Thus there is simultaneous macular perception (using a slide which subtends an angle of 8° at the nodal point) and simultaneous foveal perception (using a slide subtending an angle of 3°). These two groups designate simply a difference in the extent of suppression, and once again there is no real difference between them.

NORMAL AND ABNORMAL BINOCULAR VISION.

After simultaneous perception has been reached, binocular vision may develop normally or abnormally. The first requisite of normal binocular vision is normal correspondence. After this comes normal fusion, and then stereoscopic vision.

If the patient has abnormal correspondence, it should be noted that he still has simultaneous macular perception—because the two monocular images are seen simultaneously, although not superimposed. Therefore, it is inaccurate to say that simultaneous macular perception is the first stage of normal binocular vision, as is so commonly done.

Abnormal retinal correspondence does not provide the patient with a type of binocular vision that allows one macular image to combine with the eccentrically placed image in the other eye, and therefore any reports of grades of abnormal correspondence simply indicate grades of suppression. (It has been freely suggested that abnormal correspondence does imply a useful though inferior type of binocular vision (Lyle and Jackson¹⁸ and Pugh¹⁹).

The following is a scheme of binocular vision :

Simultaneous Perception.

<i>Normal Development.</i>	<i>Abnormal Development.</i>
Normal correspondence.	Abnormal correspondence.
True fusion.	
Stereoscopic vision.	

SUMMARY.

The foregoing may be now summarised :

1. Worth's classical grades of binocular vision are shown to be inadequate.
2. The methods of assessing the state of binocular vision are briefly described.
3. To determine if correspondence is normal or abnormal one must know two values—the angle of squint and the “angle of binocular projection.”
4. Various methods of estimating the angle of anomaly are mentioned.
5. It is shown that these methods are not comparable, one with the other.
6. It is suggested that tests such as the synoptophore method or the mirror-screen—which measure the binocular projection lines of the *two maculae*, are the best methods of diagnosing abnormal correspondence and measuring the angle of anomaly.
7. It is suggested that there is no real difference in the various types of abnormal retinal correspondence described.
8. It is suggested that the various grades of the first stage of normal binocular vision described only indicate different degrees of suppression.
9. A scheme of the development and grades of binocular vision is given.

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A NEW TECHNIQUE FOR THE APPLICATION OF RADON SEEDS TO THE SCLERA IN THE TREATMENT OF GLIOMA RETINAE

BY

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THE value of radiotherapy in the treatment of glioma retinae has become established. A number of cases have been reported in which this neoplasm has proved to be very radiosensitive and when of a moderate size it has disappeared and there have been no recurrences in periods up to 9 years after irradiation; some useful vision has been retained and the child's life saved.

Foster Moore at St. Bartholomew's Hospital used at first interstitial irradiation. He incised the sclera over the centre of the neoplasm using an angular knife with two cutting edges and the exact width of this instrument equalled the diameter of a radon seed. One side of the blade was painted with sterile gentian violet solution to mark the site of the scleral incision after the knife had been withdrawn. The point of the knife was passed through the sclera and choroid and entered the base of the neoplasm. On