No conclusions can be drawn from these few cases. There are points of interest. In the first four cases there is a marked similarity of practice. All four children rotate their heads in the interest of parallelism and in the rotated position have a fairly good, though not fully developed, fusion.

No false correspondence has developed except in one case—H. B., who tended to slip 3° .

The two little girls, *cases 6 and 7*, were too young for satisfactory synoptophore readings, but they appear to be developing along the same lines as the first four.

Case 5, with bilateral abduction paralysis, has developed a complete false correspondence and in this manner attempts to express her binocular sense.

Case 6, with the vertical paralysis, makes no attempt at binocular function, and does not posture.

My thanks are due to Miss Pugh, who very kindly worked out all the synoptophore findings.

“BILATERAL MESIAL SUPERFICIAL DEFICIENCY OF THE SCLERA”

BY

BASIL GRAVES

IN “The Eye and its Diseases,” edited by Dr. Conrad Berens (Messrs. Saunders & Co., 1936), I briefly indicated in Fig. 184, H., p. 467 (and referred in the footnote at the bottom of page 468), to a condition which sometimes conveys, at the first casual glance, a suggestion of a tenotomy of the internal rectus having been performed in the past, due to a small sharply defined area where the sclera looks thinned, appearing usually less white than the surrounding scleral tissue. One or two colleagues have asked me for details of this condition. Magnified inspection reveals what is exemplified in Fig. 1 and Fig. 2 (which is Fig. 1 enlarged) and Fig. 3.

The optical appearance of these deficiencies is characteristic. If (Fig. 4) the focused small illuminating beam is directed through the over-lying conjunctiva into the middle of the depression (*i.e.*, obliquely on to the floor, as in 1), the edge AB, as seen through the over-lying conjunctiva, is displayed as a refractile sharply defined limit to what appears to be a superficial defect of the sclera (see Fig. 3, z, which is conjectural); the sides AC and BD are less evident and the side CD is only very faintly evident.

FIG. 4.

If the beam is directed on to the floor of a same depression obliquely from the direction I_1 , the side GH is very visible, the sides EG and FH are less visible and the side EF is faintly visible. This appearance, suggesting an abrupt step-like limitation of the thin area, seems common to all the cases I have seen, though it is not necessarily displayed with equal intensity all round the margin of any given defect. (The greater visibility of the edge towards which the impinging beam is directed by "proximal illumination" is simply due to the transmitting and diffusely reflecting internal properties of the deeper laminated scleral tissue.) To summarise this paragraph, the visible definition of the bounding-edge varies relatively with the conditions of illumination; and what is, under constant conditions of illumination, interpretable as the absolute or structural definition of the bounding edge may vary in different cases and in different parts of the same case.

Case 1 is from a lady aged about 70 years. The distance M to N (Fig. 1) is 3.5 mm., and L to K 2.9 mm., M and L being estimated as at the peripheral edge of the arcus pinguiculus (arcus senilis) which is a convenient landmark for limbus measurements, representing the edge of Bowman's membrane. The measurements of the defects in this case (see the enlarged Fig. 2) were: A—B, 4 mm., C—D, 2.5 mm., E—F, 2 mm., G—H, 1.7 mm. The diagram shows samples of the conjunctival vessels running undisturbed across the scleral defects.

I should explain that I made various records of observations on the eye in the years 1921 to 1926; about 1926, on moving my abode, I destroyed most of these unpublished. I can recall having made in those years records of three such cases which I regarded as developmental defects. I cannot now give details of those cases. For many years after that I made no detailed records of cases but recently I have resumed doing so and I have observed four more cases of this defect of the sclerotic, the first being the one which I referred to briefly in Dr. Berens' book, the next being a very pronounced case of which I made no record, the next being the case of Figs. 1 and 2, and the last case being that of a lady,

aged about 60 years, illustrated in Fig. 3 (as "Case 2"). The distance from S to Y is 3.6 mm., and from V to X is 3.4 mm. P—Q is 2.7 mm., R—S, 1.7 mm., T—U, 1.26 mm., and V—W, 0.74 mm. This patient came to me saying that she had been given my name by a doctor in another part of the country and that she "wanted her eyes tested for glasses." I dealt with the matter and after I had finished she very casually introduced a remark about "a dark spot on the inner side of the left eye" which she had noticed only for the past six months. On inspection I found it to be a case of this characteristic scleral defect (Fig. 3, PQRS). I looked for the same condition in the right eye also and did not find it. The patient then casually asked if it "could be anything growing." She bore a family-name of old historic interest and was of a calm, intrepid and self-reliant demeanour. I have heard this question asked relatively often in recent years (three people since January, 1937, have asked me if their sty, or chalazion, is the beginning of cancer), and the remark, particularly in the tone in which it was interjected by this patient, struck me as unimportant; I replied that the condition was nothing more than a developmental defect which had been there since pre-natal days and that it is one which is usually bilateral. The patient replied that in her case it was bilateral, so I looked again and on more careful inspection found a small and faintly evident defect, TUVW, in the right eye also. She replied that the "dark areas" were more noticeable at some times than at others, but that neither of them had been remarked by her or her friends until six months previously. I explained to her that the thinning of the outer coat of the eye had certainly been there always and that if the areas had become evident to her only lately it was perhaps because some overlying fatty tissue might have become thinner or more dilute; and that the variation in appearance might also depend on the degree of vascular congestion of the dark ciliary body beneath the thin floor of each defect. I made a record of the case before the patient left, as I had already decided that I would report the condition some day. About a week later the doctor communicated with me and told me that before she came to me he had given her my name in response to her request for the name of another eye specialist to whom to go for a second opinion because she had just been to someone else who had told her that she had a sarcoma growing in the left eye and that the eye must be enucleated forthwith; and she had arranged with her doctor that neither he nor she should tell me about this previous opinion. She acted the part well and gave me no inkling that I was not the first person she had seen. I was informed that the disclosure of the opinion I gave was not received with tranquility and I now think it worth reporting these cases. Even if this condition occurred, as is doubtless possible, in one

eye only instead of in both, the very definite appearance on careful inspection of the defect—superficial and *in intaglio*—is not such as to suggest that it is caused by an eroding eruption from beneath. I am not aware of this condition having been reported before. A full title might justifiably be suggested as “developmental bilateral mesial superficial intagliated deficiency of the sclera.”

DIVERGENT STRABISMUS*

BY

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THE literature on the subject of convergent concomitant strabismus is redundant, but that on divergent concomitant strabismus is comparatively scarce.

Historical

Various causes and types of this condition have been described.

Beginning in the 18th century, Troxler and de la Hire thought that strabismus was due to retinal incongruity, *i.e.*, that in one eye the most sensitive part of the retina lay, not on the optic axis, but to one side of it. At this time, of course, the angle gamma was not appreciated, and it was thought that normally the optic axis and the sight axis were coincident.

In the same century, Buffon, who himself had a divergent squint, commented on the frequent presence of anisometropia, and gave it as the most common cause. He said that “the weaker eye” interfered with the binocular vision, and so it turned away. He also said that if the near point was very close, as in the case of myopes, it was tiring to converge sufficiently, and therefore one eye deviated to preserve clarity of vision.

Buffon noticed that in cases of anisometropia the vision in the squinting eye was improved on occlusion of the good eye.

In 1820 Johannes Müller in his book on the “Physiology of Sight” wrote at great length on the causes of strabismus.

In cases of divergent squint he gave as the causes:—

(a) *Anisometropia*. In this he quotes Buffon’s ideas, and says that a squinting eye develops suppression and so the sight in that eye fails.

* An abbreviated version of this paper was read at the Ophthalmological Section of the B.M.A. Congress at Oxford, July 24, 1936.