capsule. These changes can be noted in the micro-photographs. At no place could the marked thinning of the capsule or the felt-work appearance of the tissue described by Vogt be made out. The subcapsular epithelium appears normal but in one place it is duplicated. No debris of detached capsule could be recognised in the angle of the anterior chamber. Vogt³ described five cases which he had the opportunity of examining anatomically. In these he described the separation of the anterior fibres as being somewhat similar to the normal passage of the zonule from the lens capsule, except that it is more axial and in the opposite direction. Open feltwork appearance, peeling and degeneration were the apparently progressive changes noted by him.

The changes now described and illustrated are essentially the same as those recorded by Vogt and our case is of some interest as being the first to be described pathologically from this country.

The microphotographs were lent to Mr. Harrison Butler and figure in his Presidential Address at the Congress of the Ophthalmological Society of the United Kingdom on April 20, 1939.⁴

We express our thanks to the President and Council of the Ophthalmological Society of the United Kingdom for the use of the blocks of the microphotographs.

REFERENCES

1. GARROW, ALEXANDER.—*Brit. Jl. of Ophthal.*, Vol. XXII, pp. 216-217, 1938.
2. ——— *Ibid.*, p. 222.
3. VOGT, A.—*Spaltlampienmikroskopie*, Berlin, 1930.
4. BUTLER, T. HARRISON.—*Trans. Ophthal. Soc. U.K.*, Vol. LIX, Part 1, pp. 37-38, 1930.

SOME CASES OF TRAUMATIC MYOPIA*

BY

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AFTER direct trauma to the eye, one not infrequently notes the development of myopia with or without actual damage to the lens itself.

According to the intensity of the injury one can distinguish four different types:—

1. Those due to spasm of the ciliary muscle associated either with spasm of the sphincter pupillae, or with traumatic mydriasis.

* Read at the meeting of the British Medical Association in Aberdeen, 1939.

In these cases 1-4 dioptries of myopia may appear which last a few days as a rule and then disappear without treatment or under atropine.

2. Those due to partial rupture of the fibres of the suspensory ligament of the lens associated with iridodonesis. This may be responsible for 5-6 dioptries of myopia which is often permanent, and does not disappear under atropine.

3. Those due to changes in the lens itself. This is often variable in amount and may be associated with astigmatism.

4. Those due to more serious damage to the anterior or posterior coats of the eye, causing presumably some axial lengthening.

My first case illustrating the first group is remarkable for the long duration of the ciliary spasm. The patient, a man of 27 years, was injured on August 29, 1937. He had a blow on the right side of the face—the eye was closed and very bruised and the nose was deflected towards the left. He was unconscious for thirty-six hours. A day or two later he complained of diplopia on looking to the left. His vision had been tested at the Royal Naval Hospital just before the accident and was 6/6 in each eye. In December, 1937, vision was—right eye $6/18 \bar{c} -1.5 \text{ sph.} = 6/6$. Left eye normal.

I saw him in May, 1938, when I found a very slight paresis of the left external rectus and vision in the right eye $6/24 \bar{c} \begin{smallmatrix} -2.0 \\ -0.5 \end{smallmatrix} \rightarrow 180^\circ = 6/6$, left eye normal. There was no irido-

donesis, no damage to the lens and A.C. seemed normal in depth, pupils equal and active. I saw him again in July, 1938, when vision in the right eye was $<6/60 \bar{c} \begin{smallmatrix} -3.0 \\ -0.5 \end{smallmatrix} \rightarrow 180^\circ = 6/6$. I then ordered

atropine twice daily and a week later vision was 6/6 with no H.M. In this case the myopia gradually increased over a period of nearly a year and I feel there must have been some subconscious functional element which continued after the primary spasm following the injury, as the only source of continued irritation was the diplopia which remained present and in fact is still present, and was probably due to some intracranial haemorrhage affecting the nerve supply to the external rectus of the opposite eye.

A second case is somewhat similar. A young man was struck in the left eye and three days later when I saw him there was some ciliary injection, a slightly dilated pupil, no hyphaema, no iridodonesis and A.C. was of normal depth. With -2.5 sph. $V=6/6$. The condition remained very nearly as above for five weeks and then after a game of squash the myopia suddenly began to disappear. Two days after the game vision was $6/6 \bar{c} -0.5 \text{ sphere}$, and six weeks later 6/6 with no H.M. In this case there was myopia due

to ciliary spasm which needed some outside stimulus to bring it back to normal functioning.

Cases of injury associated with rupture of the suspensory ligament and iridodonesis are not uncommon. There are probably two factors in the production of myopia.

1. The more anterior position of the lens itself.
2. The fact that the lens in these circumstances becomes more globular.

In congenital dislocation of the lenses the refraction is usually very highly myopic due to the second of these factors.

The case was that of a patient, aged 17 years, who was struck in the eye with a football. I did not see him until six weeks after the accident, but there had been a hyphaema at the time. On examination there was an irregular pupil with two marked radial iris tears. There was marked iridodonesis and the anterior chamber was shallower to the temporal side. It would seem as if nearly half the suspensory ligament had been ruptured. The refraction was $6/12$ with -3.5 sph. and this could not be improved although there were no lens opacities and the fundus was normal. The visual acuity had not altered appreciably nine months later.

My next case occurred in an eye which had suffered from trauma many years previously, but the exact causation of the myopia seems rather obscure.

The patient, when aged 8 years, was hit in the right eye by a slug from a catapult. I saw him first when he was 22. He had $6/6$ vision in that eye without correction. There was an iridodialysis, but no hernia of vitreous or iridodonesis, and the lens was normal and fundus healthy. When he was 30 he began to get attacks of misty vision and I found when he came to see me that the tension was raised, the cornea was slightly steamy and that, although best vision was only $6/60$, he improved on one occasion with a -2.25 sph. and on another with a -1.5 sph. to about $6/24$. When on pilocarpine with normal tension in between the attacks, the myopia had disappeared. I trephined the eye and the tension and myopia have not recurred though vision is poor as he has lost most of his nasal field.

An interesting feature in this case was that after the operation he had four huge choroidal detachments and in between the lower two was a retinal detachment. All these gradually subsided though the fundus over the area occupied by the retinal detachment shows the scattered pigment dots which are not present in the rest of the fundus.

The myopia here may have been due to swelling of the vitreous and anterior displacement of the lens or to some alteration in the refractive power of the media due to the oedema.

Traumatic changes in the lens leading to opacities are quite common and there is no doubt that these may be localised and may disappear without leaving any permanent opacity. This is certainly true of the typical posterior subcapsular fan shaped opacity.

A man, aged 36 years, was struck by a tennis ball in the right eye two weeks before I saw him. On examination the right eye $\bar{c} \begin{smallmatrix} -1.25 \\ -1.5 \end{smallmatrix} \downarrow_{30^\circ} = 6/5$ partly. Left eye 6/5 unaided. The A.C. was a

little shallow—iridodonesis. In the lower nasal part of the lens there was a definite sector-like area with dot opacities. Three weeks later the refraction was $\begin{smallmatrix} -1.0 \\ -0.5 \end{smallmatrix} \downarrow_{60^\circ \swarrow} = 6/5$ partly. Three weeks

later vision with -0.5 sph. = 6/5 and the lens was definitely clearer—in fact it was difficult to detect any opacities which could be focused at all. I did not see him again. In this case there was obviously a localised swelling of the lens which produced myopia and astigmatism, both of which subsided when the oedema cleared.

A more striking case was recorded in the *American Journal of Ophthalmology*, of a man, aged 33 years, who was struck in the eye with an iron pin with considerable force. There was a corneal abrasion, traumatic mydriasis, but no haemorrhage or iridodonesis. The tension was slightly minus.

Four days after the accident vision with $\begin{smallmatrix} -3.5 \\ -4.0 \end{smallmatrix} \downarrow_{105^\circ} = 6/24$. Ten days later with $\begin{smallmatrix} -.75 \\ -4.0 \end{smallmatrix} \downarrow_{90^\circ} = 6/12$. At this stage some lens opacities

could be seen up and in, in the posterior lens cortex. Four weeks later with -1.5 D.cyl. 180° vision was 6/12. Two months later with -1.5 sph. vision was 6/9. Some fine radiating lines could be seen in the lens two years later.

The fourth group—myopia associated with damage to the coats of the eye is not common, but one does see cases of birth injury with a long oblique scar in the cornea associated with a considerable degree of myopia and myopic astigmatism and I think the rupture in these cases even though it has healed, must be responsible for some increase in corneal curve or elongation of the eyeball. Theoretically a similar condition could occur from changes in the posterior coats from a severe blow with scleral ruptures, but I have not come across such a case.