**FOLLOW-UP OF 54 CASES OF OCULAR CONTUSION
WITH HYPHAEMA***
**WITH SPECIAL REFERENCE TO THE APPEARANCE AND FUNCTION
OF THE FILTRATION ANGLE**

BY

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Introduction

FOLLOWING renewed interest in the occurrence of chronic unilateral glaucoma several years after contusion injury, this paper reports on 54 cases of ocular contusion with hyphaema examined on average 5·7 years after injury. Particular attention was paid to the appearance and function of the filtration angle, and in addition an assessment was made of permanent damage to the iris, lens, and posterior pole of the eye.

The pathological appearance of tears involving the trabeculae and ciliary muscle following contusion injuries has been well documented. Collins (1892) described two eyes enucleated soon after severe contusions; in addition to other damage, the ligamenta pectinata had been disrupted and there was a split between the circular and longitudinal fibres of the ciliary muscle. While the latter remained attached to the scleral spur, the circular fibres, together with the root of the iris, were displaced backwards deepening the anterior chamber angle. Parsons (1908), Lister (1924), and Lamb (1927) described similar findings.

Wolff and Zimmerman (1962), in a review of 300 eyes removed a variable time after ocular contusions, described and illustrated lesions involving the anterior chamber angle. These comprised disruption of the trabecular meshwork, iridodialysis, cyclodialysis, and lacerations into the face of the ciliary body similar to those described by Collins (1892). Following this latter injury the amount of recession of the iris root and the percentage of its circumference involved were variable; this injury was also seen to occur occasionally in combination with either iridodialysis or cyclodialysis. In many cases the circular muscle fibres had atrophied, causing the iris root and the pars plicata to appear to arise from the belly of the longitudinal muscle, and in all, the trabecular meshwork showed advanced degenerative changes, with fibrosis and hyalinization causing partial obliteration of the intertrabecular spaces and the canal of Schlemm. Definite recession of the iris root was found in 17 of the 300 eyes, an incidence of 6 per cent. In some of the older injuries a hyaline membrane similar to that described by Reese (1944) covered the inner surface of the trabeculae.

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The possible relationship between these changes and the important, but rarely recognized, chronic unilateral glaucoma developing insidiously as a late complication of ocular injury, was stressed by Wolff and Zimmerman (1962) and again by Hogan and Zimmerman (1962). They emphasized that relatively mild trauma might cause a tear into the trabeculae or ciliary body, and that it need not necessarily cause the patient either to remember the incident, or to seek the advice of an ophthalmologist.

Rodman (1963) examined sections of 120 eyes removed following traumatic dislocation of the lens; 31 had posterior dislocations, open angles, and glaucoma, and of these 30 showed traumatic damage to the chamber angles similar to that already described. He suggested that the traumatic damage to the trabecular meshwork, rather than the split in the ciliary body, was the cause of the glaucoma, and that the actual posterior dislocation of the lens was incidental to it.

The clinical association between ocular contusion and delayed chronic glaucoma was not recorded until several years after the pathology had been described.

D'Ombra (1949) detailed 3 out of 37 cases; the gonioscopic appearances were not described, but he emphasized the insidiousness of the onset of the glaucoma which, in his first case, was not discovered until eighteen years after the injury. Duke-Elder (1954a) refers to delayed post-traumatic glaucoma occurring as a result of cicatricial sclerosis of the drainage area, following rupture of the sclera, the trabeculae, or the ciliary body, but does not cite any instances.

Pettit and Keates (1963) described 8 cases with gonioscopic evidence of traumatic cleavage of the chamber angle. This evidence comprised widening and deepening of the peripheral anterior chamber, with backward displacement of the iris causing the appearance of a characteristic cleft or recess. One case seen ten years after injury had unilateral chronic glaucoma, but the remainder had normal tensions, although two showed a lowered facility of outflow as compared with the uninjured eye. Six of the 8 eyes affected had gonioscopic changes in all four quadrants.

Alper (1963) reported on a further 27 eyes with traumatic cleavage of the chamber angle discovered on routine gonioscopy of all cases of blunt trauma. No mention of the incidence was made, but 14 were found to have chronic glaucoma and open angles; since they included the 10 eyes with 360° involvement of the angle, the likelihood of glaucoma developing appeared to increase as the angle cleavage became more extensive. In 4 of these cases the presenting symptoms were those of acute glaucoma, even though all had open angles and 3 cupping of the optic disc. These cases may be similar to one described by Greeves (1937) in which acute attacks of glaucoma recurred in an eye with an anterior chamber of normal depth, deep cupping of the disc, and a history of a severe blow twenty-two years beforehand.

From this review of the literature there appear 52 clinical cases of chronic unilateral glaucoma following trauma; of these, only 3 of the 37 collected by D'Ombra (1949) are detailed, and none of these patients had a gonioscopic examination. The remaining 15 cases all had cleavage of the chamber angle, but 20 other cases of cleavage of the chamber angle did not have glaucoma, apart from 2 with multiple peripheral anterior synechiae.

The main object in reviewing cases of ocular contusion with hyphaema was to determine the frequency of traumatic cleavage of the chamber angle, and to assess, if possible, the delayed effects of this cleavage upon the intra-ocular pressure. In

order to avoid the period of instability of tension following injury it was necessary to delay the review for at least a year; and since earlier reports all stressed the insidiousness of the onset of glaucoma, it was decided to examine cases as long after injury as possible.

Material and Methods

The surgeons whose patients are reviewed in this paper routinely admit to hospital all cases of ocular contusion with hyphaema. Treatment by rest in bed, a pad and bandage, and atropine drops is continued until the hyphaema has absorbed, when, in the absence of more serious damage, the patient is "mobilized" and allowed to go home. The in-patient records for the years 1954 to 1960 inclusive were searched for all patients who had suffered an ocular contusion with hyphaema, and those calculated to be over the age of 17 years at the time of this investigation were asked to attend for an eye examination. Circulars were sent to 140 patients and 54 agreed to attend.

Both eyes were examined and in addition to taking a history and making a routine examination, the corrected monocular accommodation, the applanation tension, and the coefficient of aqueous outflow were assessed where possible. The anterior chamber angle was examined, and its width recorded, using the classification of Gorin and Posner (1957); slit-lamp microscopy was performed both before and after dilating the pupil, and provocative tests were carried out where necessary. The accommodation was measured using the R.A.F. binocular gauge, and a Schwarzer electro-tonometer was used for tonography, the coefficient of outflow being calculated from the Friedenwald nomogram. In exactly half of the cases the injured eye was subjected to tonography before the uninjured eye. Patients over the age of 17 years were selected because they were more likely to submit to the full investigation.

Findings

Fig. 1 shows the age distribution of the patients at the time of this investigation. As would be expected, there was a preponderance of young men, there being 40 males and 14 females in the series. As a guide to the severity of the injury, the number of days of in-patient treatment is shown in Fig. 2, and the length of follow-up in years in Fig. 3.

Only twelve of the 54 eyes were found to have a diminished visual acuity attributable to the injury; the vision and cause of the decrease are shown in Table I. The vision of 8 of these 12 eyes was recorded on discharge from hospital and in 2 this was considerably better than the present vision; the further reduction was attributable in each case to lens opacities but, the precise time when they developed is unknown.

Opinions are divided as to the likelihood of recovery in function of the pupil and ciliary muscle following so-called "traumatic mydriasis" (Duke-Elder, 1954b). In

FIG. 1.—Age distribution of the 54 patients in the series.

FIG. 2.—Duration of in-patient treatment.

FIG. 3.—Length of follow-up.

TABLE I

CAUSES OF DECREASED VISUAL ACUITY ATTRIBUTABLE TO TRAUMA

Causes of decreased Visual Acuity	Visual Acuity								
	6/9	6/12	6/18	6/24	6/36	6/60	C.F.	H.M.	P.L.
Lens opacity	1						1	2	1
Chorio-retinal damage	2	1	1		1				
Lens opacity and chorio-retinal damage	1								1
									Total
									5
									5
									2

this series 33 eyes had abnormal pupillary reactions on admission to hospital, but at follow-up these had recovered in all except 9; in these, the reactions, though present, were still diminished to light and for near, both direct and consensual. Six of these eyes also had lacerations of the pupillary margin, as did a further 4 with otherwise normal pupils. It was impossible to assess the accommodation of the most severely injured eyes, as well as that of the patients over 40; of the remaining 37, however, only 2 showed any marked defect of accommodation, one associated with a sluggish pupil, and the other with a normal pupil.

The commonest sign of previous trauma was the presence of discrete rounded clumps of dark pigment, adherent either to the surface of the iris or to the structures of the angle. Twenty-eight, or 52 per cent., showed this sign, and in 19 this was visible without the aid of a gonioscope.

Ten cases of traumatic cleavage of the chamber angle were discovered, and in these the gonioscopic findings were definite. In the affected areas recession of the angle was marked, and the junctional zones between the normal and abnormal parts of the angle further emphasized this recession. Each had medium or wide-open angles, and apart from scattered uveal pigment showed no other abnormality of the angle; 8, however, had traumatic damage to either the iris, lens, or posterior pole of the eye, and 6 had subnormal vision. Details of these, together with those of cases with coefficients of outflow of less than 0.15, are shown in Table II; and apart from one case with peripheral anterior synechiae (Case 25, Table II), no structural abnormality of the remaining angles was noted.

TABLE II

EYES WITH TRAUMATIC CLEAVAGE OF THE ANTERIOR
CHAMBER ANGLE OR A COEFFICIENT OF OUTFLOW LESS THAN 0.15

Case No.	Age (yrs)	Injured Eye						Uninjured Eye	
		Visual Acuity	Causes of decreased Visual Acuity	Persistent Pupillary Abnormality	Traumatic Cleavage of Chamber Angle	Aplanation Tension	Coefficient of Outflow	Aplanation Tension	Coefficient of Outflow
9*	19	P.L.	Lens opacity and chorio-retinal damage	Notched and sluggish	100°	16	0.10	13	0.40
19	50	H.M.	Lens opacity	Notched and sluggish	360°	13	0.43	14	0.30
24	23	6/6		Notched	40°	15	0.24	16	0.21
28	24	6/9	Chorio-retinal damage		180°	24	0.24	19	0.17
34	63	6/6			90°	14	0.18	13	0.27
39	53	C.F.	Lens opacity, no view of the fundus	Notched and sluggish	90°	25	0.34	20	0.60
41	20	6/18	Chorio-retinal damage	Sluggish	180°	15	0.45	15	0.70
42	21	6/6			180°	12	0.54	10	0.70
44	39	6/6			100°	16	0.26	16	0.50
46	23	6/12	Lens opacity		180°	18	0.23	16	0.25
6†	18	6/6		Notched		15	0.12	15	0.24
20†	18	6/9	Lens opacity and chorio-retinal damage			15	0.09	15	0.26
25‡	77	6/6				25	0.08	20	0.24

* Did not attend for water drinking test. † Negative water drinking test. ‡ Multiple peripheral anterior synechiae.

No abnormally high intra-ocular tensions were recorded, although the average coefficient of outflow of the 50 eyes with a history of trauma was slightly lower than that of the 50 eyes without trauma. Fig. 4 shows individual readings for the 50 eyes in each category, and in addition the occurrence and extent of traumatic cleavage of the chamber angle; it is emphasized that the eyes of individual patients are not paired in this figure.

The eyes of individual patients are considered in Fig. 5, which shows the difference between the coefficients of outflow of the injured and uninjured eyes of the same patient. The injured eye had a lower outflow in the 33 cases above the line, and those with traumatic cleavage of the chamber angle are again indicated.

In Table III the incidence of damage to the iris, lens, and posterior pole of the eye is recorded, together with similar figures from the review by Henry (1960) of 204 case records of patients who had suffered ocular contusion with hyphaema.

TABLE III
INCIDENCE OF DAMAGE TO IRIS, LENS,
AND POSTERIOR POLE OF THE EYE,
AND OF LOWERED VISUAL ACUITY

	Henry (1960) (per cent.)	Britten (1965) (per cent.)
Traumatic Cataract	11	15
Chorio-retinal Damage	14	18
Final Visual Acuity of 6/12 or less	17	15
Vitreous Haemorrhage following Accident	10	15
Secondary Hyphaema following Accident	17	11

FIG. 4.—Outflow coefficient of 50 normal eyes (upper readings), and of 50 fellow, but unpaired, eyes approximately five years after ocular contusion injuries. The occurrence and extent of traumatic cleavage of the chamber angle is also indicated.

FIG. 5.—Differences in outflow coefficients of 50 pairs of eyes approximately five years after one eye sustained a contusion injury; in those above the line the injured eye had the lower coefficient of outflow. Those with traumatic cleavage of the chamber angle are indicated.

Discussion

Little is known of the incidence and effect of traumatic cleavage of the anterior chamber angle; but delayed glaucoma following contusion injuries has been related either to the occurrence and extent of this lesion or to microscopical damage to the trabeculae.

In this series the incidence of traumatic cleavage was 20 per cent., and the coefficient of outflow was lower in the traumatized eye than in the normal eye in 33 out of 50 patients (Fig. 5); this figure is just statistically significant ($2 \times \text{S.E.} = 13.4$ per

cent.). This, together with the lower average coefficient of the 50 injured eyes (see Fig. 4), suggests that there is a tendency for contusion injuries to lower the coefficient of outflow, although too much attention should not be paid to a mathematical finding in a series of 50 cases.

Figs 4 and 5 also show that cases of traumatic cleavage are distributed over the full range of readings, and that there is no tendency for them to constitute the majority of the eyes with the lower coefficients of outflow; similarly, they are not congregated amongst those showing the bigger differences in outflow between the injured and the uninjured eye. The suggested relationship between traumatic cleavage and a lowered outflow is therefore not borne out; but it still remains possible that microscopical damage to the trabeculae, not occurring *pari passu* with traumatic cleavage, is responsible. It has not been possible to demonstrate this, since eyes that have sustained a relatively minor contusion are rarely available for histological study; and although Weidenthal (1964) succeeded in reproducing these contusion injuries in the Rhesus monkey, the force required was also sufficient to rupture the globe.

No cases of glaucoma were found in this series, but those with a lowered outflow following injury must now be more liable to glaucoma than beforehand. The cause of the long delay before the onset of some cases of glaucoma is difficult to explain in terms of sclerosis of the drainage area continuing over a period of several years. It is, perhaps, more reasonable to suggest that the injury reduces the outflow initially, and that the added fall in outflow of both eyes with age (Miller, 1961) is finally responsible for the glaucoma. If both eyes were initially similar, the injury might determine that this eye was the first to develop glaucoma, the uninjured eye, on occasions, not becoming affected during a normal span of life.

Although it is realized that chronic simple glaucoma can remain unilateral for a long time; it is suggested that all patients with unilateral glaucoma should be questioned about previous injury, and that signs of previous trauma, including scattered uveal pigment and cleavage of the chamber angle, should be sought. The absence of a positive history of injury is not sufficient, since relatively severe injuries are frequently forgotten; in this series over a quarter of the patients could not remember which eye had been affected, after only five years had elapsed.

Concerning the other findings, the low incidence of persistent pupillary abnormality and the frequent recovery of the power of accommodation are notable, as is the similarity between the incidence of persistent ocular damage in this and Henry's (1960) series.

Summary

The incidence of traumatic cleavage of the chamber angle in a follow-up of 54 cases of ocular contusion with hyphaema was 20 per cent. A tendency for contusion injuries to lower the coefficient of outflow was noted, although this was not related to the occurrence of traumatic cleavage of the chamber angle; instead it is suggested that this might result from microscopical damage to the trabeculae. A high incidence of recovery of function following traumatic mydriasis was found.

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