Ocular damage after blunt trauma to the eye

Its relationship to the nature of the injury

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Blunt injury to the eye results in a spectrum of damage to the intraocular structures. Many retrospective but few prospective studies have been performed on selected aspects of ocular damage after blunt injury. This review has been conducted on a prospective basis, and the total ocular damage and subsequent complications have been studied, particularly in relation to the site and nature of the injury, to see if any correlation could be found.

Review of the literature

(1) *Anterior segment damage after blunt trauma*

Blunt injuries to the eye commonly present with an hyphaema; associated damage to the ciliary body, iris, or lens may be present. Secondary bleeding, uveitis, and abnormal intraocular pressure may complicate the immediate post-injury period.

(i) The primary hyphaema results from a tear into the face of the ciliary body or iris. Treacher Collins (1892) was the first to draw attention to tears into the face of the ciliary body, between the circular and longitudinal muscle fibres, after a contusion injury to the eye. Experimentally, Kilgore (1942) demonstrated that tears of this nature were the result of blunt injury directed at the limbus. Wolff and Zimmerman (1962), in a large pathological study on eyes enucleated after blunt injury, described the frequent occurrence of cleavage into the face of the ciliary body, with damage to the major arterial circle. Tönjum (1966) confirmed by gonioscopy a high incidence of angle cleavage after a traumatic hyphaema.

(ii) The source of secondary bleeding is often uncertain; it may arise from newly formed capillaries in the area of damage, or from the original lacerated vessel. Secondary bleeding most commonly occurs in the first 5 days after injury; the incidence from various series ranges from 5 to 30 per cent. (Table I). A statistical analysis of the incidence of secondary bleeding in these series, where varying modes of treatment for the primary hyphaema were used, showed no statistically significant difference.

In one series (Thygeson and Beard, 1952), secondary bleeding was related to the severity of the injury, as judged by the incidence of associated ocular damage, but the number of cases in this series is small. Oksala (1967) graded injuries according to the size of the primary hyphaema, and found that there was a higher rate of complications when the primary hyphaema exceeded half the anterior chamber depth.

In several of these series, the visual prognosis was found to be worse after secondary bleeding had

Table I Incidence of secondary hyphaema in various published series, grouped according to method of treatment of primary hyphaema

Authors	Date	Hyphaema		Treatment of primary hyphaema
		Primary	Secondary (per cent.)	
Shea	1957	113	13	Rest in bed, patching, sedation
Gregersen	1962	200	6	Rest in bed
Spaeth and Levy	1966	85	24	Rest in bed (+/- oestrogens)
Darr and Passmore	1967	75	5	Rest in bed
		+34	32	Variable
Henry	1960	137	13	Rest in bed
		+52	12	Rest in bed + Homatropine drops
Loring	1958	56	30	Rest in bed + Homatropine drops
Oksala	1967	128	6	Rest in bed + Homatropine drops
Cole and Byron	1964	100	30	Rest in bed + Phenylephrine drops
Thygeson and Beard	1952	34	38	Variable

occurred, more so if raised intraocular pressure had developed, and especially so if surgical intervention became necessary. The inference drawn was that secondary bleeding itself adversely affected the visual outcome, although data from one series (Cole and Byron, 1964) would suggest that the extent of damage from the original injury was more significant.

(iii) Raised intraocular pressure may occur during the early post-contusion periods, or as a late problem after several years; the latter problem does not fall within the scope of this study. Blanton (1964) showed that early raised intraocular pressure occurred in 5 per cent. of hyphaemas, and was unrelated to angle cleavage.

(2) Posterior segment damage after blunt trauma

This frequently coexists with anterior segment damage, and is not apparent until the primary hyphaema has cleared. It may involve the posterior pole of the eye, or the peripheral retina. Commotio retinae, which in more severe injuries is associated with intraretinal haemorrhages, or haemorrhages into the anterior vitreous from the pars plana region, is the posterior segment counterpart of an hyphaema. In the most severe injuries, retinal or choroidal breaks, or massive anterior vitreous haemorrhage may occur.

The characteristics of post-concussion retinal breaks leading to retinal detachment were described by Cox, Schepens, and Freeman (1966). They found, in a retrospective study, that oral tears were the commonest cause of post-traumatic retinal detachments; 30 per cent. presented within 1 month of injury, and 80 per cent. within 2 years of injury. Some experimental work by Weidenthal and Schepens (1966) and the high incidence of upper nasal tears, described in the previous paper, suggested that traction on the vitreous base was the mechanism of these tears.

Method

108 consecutive cases admitted to the Birmingham and Midland Eye Hospital after blunt injury to the eye, between December 1971, and May 1972, were studied. Patients were seen on the day of admission, and the nature of the injury was recorded, together with such features as lid lacerations, subconjunctival haemorrhage, or corneal staining, by which the site of impact was determined. The extent and subsequent duration of any hyphaema, plus any anterior or posterior segment damage, was noted. A few cases in this series were admitted primarily because of extraocular damage.

Patients with an hyphaema were treated with rest in bed and patching of the injured eye, for 4

days. Topical antibiotics were used in the presence of a corneal abrasion, but mydriatics or miotics were not used. Restless children were sedated.

On the fourth post-traumatic day, in uncomplicated cases, the patients were mobilized, and both eyes were assessed as follows:

- (1) Assessment of visual acuity.
- (2) Slit-lamp examination of the anterior segment, and applanation tonometry.
- (3) Dilatation of both eyes with cyclopentolate 0.5 per cent. drops and Phenylephrine 10 per cent. drops.
- (4) Repeat slit-lamp examination and gonioscopy.
- (5) Direct ophthalmoscopy.
- (6) Indirect ophthalmoscopy, with scleral indentation to view the peripheral retina.
- (7) Three-mirror examination in selected cases.

These procedures were carried out without any complications; in particular, dilatation and gonioscopy at this stage did not precipitate any case of secondary bleeding. In the more severe injuries, full examination was delayed a few days.

In selected cases of posterior segment damage, central and peripheral field assessments were made, using the Friedmann Visual Field Analyser, and the Goldmann perimeter respectively.

All patients were reassessed at 6 weeks, and at 6 months after injury, or more frequently when indicated.

Classification of nature and site of injury

- NATURE (1) Diffuse periorbital impact.
 (2) Sharply localized impact.
- SITE (1) Corneal.
 (2) Limbal.
 (3) Scleral.

Classification of ocular dam

A grading scheme was devised, as shown in Table II (opposite).

Results

A general analysis of the cases is shown in Table III (opposite).

Of the original 108 patients, five failed to attend follow-up, and are excluded from the analysis, as was one patient with a previous contusion injury. The age range, sex distribution, and type of injury conform to data given in other series. Injuries due to diffuse and localized impact formed two approximately equal groups.

ANTERIOR SEGMENT DAMAGE

Comparison of the grade of hyphaema with total intraocular damage (Fig. 1, opposite).

This Figure is based on 82 patients who presented within 24 hours of injury, and in whom gonioscopy was feasible; the estimation of the severity of ocular damage was based on the grading in each case of damage to the angle, to the other anterior segment structures, to the posterior pole, and to the peripheral retina.

This demonstrates that in most cases minimal damage only is incurred after blunt trauma; severe damage is most commonly found in patients in whom the primary hyphaema persists beyond the first post-traumatic day, although some patients with only microscopic bleeds had severe damage. Conversely, one-third of patients with a prolonged hyphaema had only minimal damage.

Table II Grading scheme for damage to the eye

(1) <i>Hyphaema</i>	0—nil 1—microscopic 2—macroscopic 3—macroscopic, still present on second post-traumatic day L—late presentation
(2) <i>Angle recession</i>	0—nil 1—less than 90° 2—90 to 180° 3—exceeding 180°
(3) <i>Other anterior segment damage</i>	0—nil, or traumatic mydriasis only 1—Vossius' ring; one sphincter tear 2—anterior concussion lens opacities; multiple sphincter tears; radial iris pigment epithelial defects 3—progressive cataract; lens subluxation or dislocation; iridodialysis
(4) <i>Posterior polar damage</i>	0—nil or transient macular oedema 1—macular oedema persisting beyond fourth post-traumatic day, but resolving 2—pigment scarring around the macula; small choroidal haematoma 3—macular haemorrhage; cystic degeneration of the macula; massive choroidal haematomas and choroidal ruptures; avulsion of the optic nerve
(5) <i>Peripheral retinal damage</i>	0—nil or transient commotio retinae 1—commotio retinae persisting beyond fourth post-traumatic day 2—commotio retinae plus intraretinal haemorrhages, and anterior vitreous haemorrhage from the pars plana 3—extensive vitreous haemorrhage; peripheral retinal or choroidal breaks; retinal detachment

Table III General analysis of 102 cases included in survey (89 male and 13 female)

Age range (yrs)		Nature of injury (102)			
		Diffuse (49)		Localized (53)	
0-15	32				
16-25	38				
26-35	15	Punch or kick	18	Racquet or stick	15
36-45	7	Football	14	Steel chips, pellets	11
46-55	5	Squash ball	6	Stone	9
over 55	5	Miscellaneous	11	Miscellaneous	18

FIG. 1 Comparison of grade of hyphaema with total intraocular damage

Comparison of the grade of hyphaema with grade of angle recession (Fig. 2).

This is based on the same 82 patients as Fig. 1.

These results confirm those of previous workers (Tonjum, 1966; Mooney, 1972) that angle recession is almost invariably present after a macroscopic hyphaema. A high proportion of patients who have had a prolonged primary hyphaema show extensive angle recession.

Comparison of the grade of angle recession with other anterior segment damage (Fig. 3).

This is based on 88 patients, in whom gonioscopy was feasible.

These results show that, in the majority of cases, angle recession of less than 90° is the only permanent damage to the anterior segment; a small number of patients showed extensive angle recession, which was associated with moderate or severe damage to other anterior segment structures.

FIG. 2 Comparison of grade of hyphaema with grade of angle recession

FIG. 3 Comparison of grade angle recession with other anterior segment damage

Relationship of site of impact to site of angle recession

In 39 patients, the site of impact could be determined, and gonioscopy was possible. In 29 patients, after limbal impact, angle recession was directly related to the site of impact; seven of these had smaller *contre-coup* recessions in the opposite quadrant. In three patients, where the impact was well behind the limbus, recession was found in the opposite quadrant only. Six patients had extensive angle recession, associated with lens subluxation, after central corneal impact.

In eight of these patients, after localized limbal impact, anterior concussion lens opacities were related both to the site of impact and to the site of angle recession, suggesting that displacement of the lens, with traction on the ciliary body *via* the zonule, may be the mechanism for these tears.

The site of angle recession can be predicted from the presence of "traumatic mydriasis"; the affected segment of the pupil is atonic, and neither constricts nor dilates fully. It corresponds to the position of angle recession.

Other anterior segment damage

Although minor angle recession was often the only permanent damage to the anterior segment, in 28 per cent. of patients more extensive damage was found. This was much more common after localized impact, occurring in 41 per cent. of patients, as compared to 14 per cent., after diffuse injuries.

A qualitative difference in the nature of damage was also found:

Localized impact This resulted in limited anterior concussion lens opacities, associated with either sphincter tears or iridodialysis; after a more severe impact, progressive cataract resulting.

Diffuse impact Anterior segment damage was a feature of only very severe diffuse injuries, which resulted in lens subluxation or dislocation, associated with radial pigment epithelial defects of the iris.

*Complications of secondary hyphaema, uveitis, and abnormal intraocular pressure**(a) Secondary hyphaema*

67 patients with an hyphaema were admitted within 24 hours of injury; twelve of these patients developed secondary bleeding, an incidence of 17.9 per cent. Three patients presented late with full secondary hyphaemata.

Of these fifteen patients, only three showed associated raised intraocular pressure requiring surgical management.

The peak incidence of secondary bleeding was on the second post-traumatic day.

Table IV shows the grades for the primary hyphaema, angle recession, and other anterior segment damage for the twelve cases that developed secondary bleeding in hospital. The majority of patients had prolonged primary hyphaemas, but secondary bleeding developed in four patients who had been admitted with only microscopic bleeds. Patients showed a spectrum of damage to the anterior segment.

Table IV *Grade of primary hyphaema, angle recession, and other anterior segment damage in twelve cases of secondary hyphaema*

Grade	0	1	2	3
Hyphaema	0	4	1	7
Recession	0	3	5	2
Anterior segment	3	3	2	4

Table V (overleaf) shows the visual outcome in the fifteen patients with secondary bleeding in relation to the anterior and posterior segment damage. Only five patients had a visual acuity of less than 6/6, because of damage incurred at the time of injury, mainly to the posterior segment. Two patients who required surgery for full secondary hyphaema with raised intraocular pressure had 6/6 vision.

(b) Abnormal intraocular pressure and uveitis

The usual response of the intraocular pressure after an injury resulting in an hyphaema, is a period of hypotony, the duration of which varies with the severity of the injury. Unless complicated by uveitis, the intraocular pressure returns to its normal level and remains stable within 1 to 2 weeks.

Fig. 4 (overleaf) shows the applanation pressure difference between the normal and the injured eye, measured on the fourth post-traumatic day, in 64 uncomplicated cases. The injured eye always showed a lower pressure than the normal eye. All these patients had normal intraocular pressures at 6 weeks and 6 months after injury.

Table V Visual acuity at 6 months in fifteen patients with secondary hyphaema; associated ocular damage; day of occurrence; size of secondary hyphaema; intraocular pressure; surgery

Visual acuity	Hyphaema (grade)	Damage		Secondary hyphaema		Raised IOP	Surgery
		Anterior segment	Posterior segment	Day	Size		
6/5	1	Nil	Peripheral commotio retinae	3rd	Small	Nil	Nil
6/5	1	Nil	Nil	1st	Small	Nil	Nil
6/5	3	Nil	Peripheral retinal haemorrhages	2nd	Small	Nil	Nil
6/5	L	Nil	Nil	3rd	Diffuse	Nil	Nil
6/6	3	Nil	Nil	2nd	Small	Nil	Nil
6/6	3	Nil	Nil	3rd & 7th	Full	Yes	Anterior chamber washout 10th day
6/6	2	Sphincter tears	Nil	2nd	Small	Nil	Nil
6/6	3	Lens subluxated	Macular oedema	2nd with recurrence	Full	Yes	Anterior chamber washout 5th day
6/6	1	Iridodialysis Anterior concussion lens opacities	Peripheral retinal haemorrhages	2nd	Small	Nil	Nil
6/6	L	Nil	Nil	4th	Diffuse	Nil	Nil
6/9	1	Nil	Retinal dialysis choroidal ruptures	3rd	Small	Nil	Nil
6/9	3	Anterior concussion lens opacities	Macular oedema	3rd	Small	Nil	Nil
6/18	L	Iridodialysis Anterior concussion lens opacities	Vitreous haemorrhage	3rd	Full	Yes	Anterior chamber washout 6th and 12th day
6/24	3	Progressive cataract	Vitreous haemorrhage	2nd with recurrence	Small	Nil	Nil
Hand movements	3	Iridodialysis Progressive cataract	Vitreous haemorrhage	2nd with recurrence	Small	Nil	Nil

FIG. 4 Difference in applanation pressure between the normal and injured eye on fourth day after injury

In the majority of cases of uncomplicated hyphaemata, the anterior chamber is clear by the fourth post-traumatic day; five patients still showed a few cells and a faint flare after small secondary bleeds or delay in absorption of the primary hyphaema.

Fifteen patients developed a marked anterior uveitis requiring treatment; in some, this was associated with an abnormal intraocular pressure. In two patients, a marked anterior uveitis developed, out of all proportion to the severity of the injury; both patients had a past history of anterior uveitis.

Table VI overleaf correlates the incidence of uveitis and abnormal pressure with anterior and posterior segment damage.

Three principal factors were involved:

(1) In the three patients with the full secondary hyphaemata requiring surgical treatment, two had subsequent uveitis for several weeks.

(2) Four patients rapidly developed a mature cataract, and all were associated with marked uveitis. One patient had a transient elevation of pressure at 2 weeks, which was controlled medically, but another had severe uveitis and persistently raised pressure, which was difficult to control even after lens extraction. This was the only patient in this group with dense vitreous haemorrhage.

(3) Seven patients had uveitis associated with marked vitreous haemorrhage. Two of these persisted in hypotony: the remainder developed raised intraocular pressure 2 weeks after injury, which was usually transient.

Six patients with extensive angle recession, but without progressive lens changes or vitreous haemorrhage, did not develop uveitis or abnormal intraocular pressure.

POSTERIOR SEGMENT DAMAGE

Some permanent damage to the posterior segment occurred in 30 per cent. of patients.

Damage to the posterior pole of the eye

Thirty patients showed commotio retinae affecting the macular region. In most cases recovery of visual acuity occurred within 2 weeks of injury, but twelve patients were left with some permanent macular damage.

Table VII (overleaf) shows the relationship of macular damage to the nature of the injury, the visual acuity 6 months after injury, and the grade of hyphaema, plus the anterior segment damage.

Severe macular oedema most commonly followed localized impact, and moderate or severe anterior segment damage was present in over two-thirds of patients.

Serial assessment of visual acuity and central field analysis showed that maximum recovery took up to 6 months to occur. Central field analysis was helpful in the visual prognosis, as areas of absolute scotomata (unless due to choroidal haematomata) showed no improvement with time, whereas cases with relative defects tended to improve considerably with time, although full recovery only occurred in the mildly affected. One

Table VI *Relationship of uveitis and ocular hypertension to anterior and posterior segment damage in fifteen patients*

<i>Duration of uveitis (wks)</i>	<i>Raised IOP</i>	<i>Secondary hyphaema</i>	<i>Damage</i>	
			<i>Anterior segment</i>	<i>Posterior segment</i>
2	Associated with secondary hyphaema	Yes	Nil	Nil
6	Associated with secondary hyphaema	Yes	Iridodialysis Iris atrophy	Dense vitreous haemorrhage
8	Associated with secondary hyphaema	Yes	Lens subluxation	Nil
4	Nil	Nil	Progressive cataract	Peripheral retinal haemorrhages
9 mths	Severe 9 mths	Yes	Progressive cataract	Dense vitreous haemorrhage
3	Transient	Nil	Progressive cataract	Nil
5	Nil	Nil	Progressive cataract	Nil
3	Transient	Nil	Lens subluxated	Peripheral retinal haemorrhages
6	Transient	Nil	Lens subluxated	Vitreous haemorrhage Retinal detachment
10	Hypotony Phthisis	Nil	Lens subluxated	Avulsion of optic nerve
12	Hypotony	Yes	Cyclodialysis	Vitreous haemorrhage
18 mths Continuous	Severe Continuous	Nil	Lens subluxated	Vitreous haemorrhage Retinal detachment
Continuous	Pupil block glaucoma at 2 weeks	Nil	Lens dislocated	Dense vitreous haemorrhage
4 (past history of anterior uveitis)	Nil	Nil	Nil	Nil
6 (past history of anterior uveitis)	Nil	Nil	Nil	Nil

Table VII Macular damage related to visual acuity 6 months after injury, nature of injury, grade of hyphaema, and anterior segment damage

Macular damage	Visual acuity	Hyphaema (grade)	Injury	Anterior segment damage
Macular oedema Pigment scarring	6/6	3	Localized limbal	Lens subluxated
Macular oedema Pigment scarring	6/9	3	Localized corneal	Anterior concussion lens opacities
Choroidal rupture nasal to macula	6/9	2	Localized corneal	Nil
Macular oedema Pigment scarring	6/9	3	Localized limbal	Iridodialysis Anterior concussion lens opacities
Macular oedema Pigment scarring	6/12	1	Localized limbal	Anterior concussion lens opacities
Pigment scarring Cystic macular degeneration	6/18	3	Localized limbal	Progressive cataract
Cystic macular degeneration	6/18	3	Localized corneal	Progressive cataract
Macular haemorrhage Pigment scarring	6/18	3	Localized scleral	Lens subluxated Vitreous herniation
Macular haemorrhage Cystic degeneration	6/18	L	Diffuse	Nil
Massive choroidal haematoma Choroidal ruptures	6/18	L	Diffuse	Nil
Macular oedema Severe pigment scarring	6/60	2	Localized scleral	Nil
Avulsion of optic nerve	No perception of light	3	Diffuse	Lens subluxated

patient had an absolute defect corresponding to the macula, and failed to improve beyond 6/60 vision; the remainder recovered vision of 6/6 to 6/18.

Two diffuse injuries resulted in severe damage to the posterior pole; one patient was blind because of avulsion of the optic nerve, and the second had an extensive choroidal haematoma because of multiple choroidal ruptures.

Peripheral retinal damage

Excluding commotio retinae alone, which resolved without trace in a few days, 37 patients

had changes resulting in some permanent scarring to the peripheral retina. In nine of these, peripheral retinal or choroidal breaks were present. Only one-third of these 37 patients had significant anterior segment damage.

In 37 patients, the site of peripheral commotio retinae or more severe damage was directly related to the site of impact. Damage to the peripheral retina occurred equally after both diffuse and localized impact, but the more severe forms followed localized impact, which was usually situated behind the limbus.

27 patients showed peripheral commotio retinae associated with varying degrees of intraretinal and anterior vitreous haemorrhage. Absorption of the haemorrhages occurred over several weeks, leaving fine pigment scarring of the retina, and small anterior vitreous opacities. Hyperpigmentation of the affected vitreous base was common; two patients with quite marked anterior vitreous haemorrhage were subsequently found to have linear pigmented excavations of the posterior border of the vitreous base. These changes resulted from traction damage on the vitreous base at the time of the injury.

Three patients developed atrophic retinal holes in areas of pigment scarring, but these did not give rise to retinal detachment during the period of study (now 18 months).

Marked anterior vitreous haemorrhage initially obscured the view in seven patients; all except two cleared sufficiently to view the peripheral retina, and two of these were found to have peripheral retinal breaks associated with retinal detachment.

Table VIII (opposite) shows the type of peripheral retinal or choroidal damage, and the occurrence of vitreous haemorrhage, in relation to pre-existing retinal changes, the occurrence of retinal detachment, the type and timing of surgery, and the visual acuity 6 months after injury.

Table IX (overleaf) shows the relationship between the site and nature of peripheral retinal damage, and the site and nature of the impact, plus the grade of hyphaema, and the incidence of anterior segment damage.

In this small series, all large round holes and extensive retinal dialyses led to progressive retinal elevation within the first 2 to 3 weeks of injury. With the exception of one case, which was considered inoperable because of multiple fishmouthing tears extending from the equator to the posterior pole, with a total retinal detachment, uveitis, and glaucoma, all cases of retinal detachment were treated surgically, with cryotherapy, and either local indentation or encirclement with a strap and buckle. In all patients, surgery resulted in successful reapposition of the retina. In one of these patients, the detachment had extended to involve the macula; surgery was delayed until 8 weeks after injury, because of severe anterior segment damage and vitreous haemorrhage accompanied by uveitis and raised intraocular pressure; macular damage was already present. All other patients were operated on from 2 to 5 weeks after injury. One patient with a very small retinal dialysis and very localized retinal elevation was not treated surgically; no progression has occurred over the last 12 months.

The site of damage in all cases was directly related to the site of impact. Four of the patients had no macroscopic hyphaema, and the incidence of anterior segment damage was low, reflecting the purely scleral nature of the impact in all cases.

This is contrary to the mechanism of indirect traction *via* the vitreous base proposed by Schepens (1969), but two of the patients showed more extensive changes, suggesting that traction *via* the vitreous base may have a secondary role in the production of retinal tears. One patient had a dialysis at the site of impact, and an additional dialysis in the opposite quadrant; the second patient had an extensive dialysis exceeding half the circumference of the globe, the apex of which corresponded to the site of impact.

Table VIII *Correlation of peripheral retinal or choroidal damage and vitreous haemorrhage with presence of pre-existing retinal changes, occurrence of retinal detachment, surgery, and visual acuity 6 months after injury*

<i>Peripheral damage</i>	<i>Vitreous haemorrhage</i>	<i>Pre-existing changes</i>	<i>Retinal detachment</i>	<i>Surgery</i>	<i>Visual acuity</i>
Area of choroidal ruptures	Minimal	Nil	Nil	Nil	6/5
Area of choroidal ruptures	Minimal	Nil	Nil	Nil	6/6
Extensive area of choroidal ruptures Dialysis 3-4° ^c	Moderate	Nil	Nil	Yes Local 3 wks	6/9
Two dialyses: 7-9° ^c and 12-3° ^c	Minimal	Nil	Yes	Yes Local 5 wks Strap 8 wks	6/6
Dialysis 6-9° ^c Vitreous base detachment	Nil	"White sine pressure", both eyes	Yes	Yes Local 5 wks	6/12 (amb)
Giant dialysis exceeding half circumference	Minimal	"White sine pressure", other eye	Yes	Yes Strap - 10/7	6/9
Very small dialysis	Nil	Cystoid and lattice degeneration, both eyes	Nil	Nil	6/6
Large round hole Choroidal ruptures (macular damage)	Moderate	Nil	Yes	Yes Local 5 wks	6/36
Multiple retinal tears	Marked	Cystoid degeneration in fellow eye	Yes	Nil (inoperable)	Perception of light

Discussion

It has been shown that the extent of intraocular damage can often be predicted from the nature of the injury and site of impact. Diffuse injuries, in which the brunt of the force is taken by the orbital margin, usually result in mild ocular damage. Fourteen of the patients in this series had signs of extraocular damage after a diffuse injury. In contrast, localized impact results in significant ocular damage in a high proportion of cases. The degree of damage depends on the force with which the object hits the eye; the greatest damage was found after injury with small high-speed objects, such as lead pellets. The site of impact determines the nature of the ensuing damage; where this is over some part of the anterior

Table IX Relationship between site and nature of peripheral retinal damage, site and nature of impact, grade of hyphaema, and anterior segment damage

Damage		Impact		Grade of hyphaema	Anterior segment damage
Site	Nature	Site	Nature		
Nasal periphery	Choroidal ruptures	Nasal sclera	Lead pellet	2	Nil
Temporal periphery	Choroidal ruptures	Temporal sclera	Lead pellet	0	Nil
Upper temporal periphery	Choroidal ruptures Dialysis	Temporal sclera	Cardboard tube	1	Nil
Lower temporal and upper nasal periphery	2 dialyses	Lower temporal sclera	Stick	1	Nil
Lower temporal periphery	Dialysis	Lower temporal sclera	Punch	2	Nil
Nasal periphery	Giant dialysis 180° +	Upper nasal limbus	Racquet	3	Anterior concussion lens opacities
Lower temporal periphery	Small dialysis	Lower temporal limbus	Racquet	1	Nil
Lower temporal periphery	Large round hole Choroidal ruptures	Lower temporal sclera	Arrow	3	Lens subluxation Vitreous herniation
Temporal equator	Irregular retinal tears	Diffuse	Chair back	L	Lens subluxation

segment, damage to the iris, ciliary body, and lens may result. When the impact is scleral, the anterior segment is often not involved, but peripheral retinal damage over the site of impact may result. *Contre-coup* macular damage is also common after localized impact, and is often associated with anterior segment damage.

When the duration of the primary hyphaema is prolonged, a higher incidence of moderate or severe anterior segment damage is found, but this fact has only limited prognostic value, as there are many exceptions.

Angle recession is a feature of all macroscopic hyphaemata; in extensive angle recession, other anterior segment damage is common. The relationship between the site of impact

and the position of angle recession and anterior concussion lens opacities suggests a traction mechanism for these tears into the face of the ciliary body, as described.

Too much emphasis has been placed in the past on the importance of secondary bleeding in relation to the visual outcome. Although secondary bleeding may occur in any patient with an hyphaema (even after a microscopic bleed), the problem is usually more serious, and often recurrent, in patients with severe ocular damage. In this series, the visual outcome has been shown to depend on ocular damage sustained at the time of injury; secondary bleeding did not appear to have a direct influence on the visual outcome.

It has been shown that during the first few days after injury resulting in an hyphaema, the intraocular pressure is low. Patients who develop secondary bleeding and raised intraocular pressure must therefore have severe impairment of outflow from the presence of solid clot in the angle, and an attempt to control the raised pressure by medical treatment, when aqueous production is already low, is unlikely to succeed; it only increases the likelihood of developing extensive peripheral anterior synechiae. Surgical treatment is therefore advocated as soon as a full secondary hyphaema develops in association with raised intraocular pressure.

Ocular hypertension also developed in association with uveitis; all these patients had severe intraocular damage. This was related to progressive cataract formation or dense vitreous haemorrhage. Ocular hypertension developed towards the second week after injury, after the initial period of hypotony. Angle recession was not related to the development of either uveitis or ocular hypertension during this early post-traumatic period.

Macular damage may result in permanent visual impairment, and it is often difficult to predict recovery from the clinical appearance; the use of the Visual Field Analyser was found to be helpful in prognosis.

After blunt injury approximately 10 per cent. of cases show peripheral retinal or choroidal breaks; the two may co-exist and are present from the time of injury. A small percentage of patients with chorio-retinal scarring, after peripheral commotio retinae with intraretinal haemorrhage, subsequently develop atrophic retinal holes in the area of scarring. The significance of these holes in relation to late retinal detachment is difficult to predict.

In this series, all large retinal breaks proceeded to slow retinal elevation immediately after injury, but two patients have recently been seen with large retinal tears which started lifting but which had flattened again within 2 weeks with pigment demarcation, without any treatment.

I should like to emphasize that retinal breaks occurred after localized scleral impact, and that anterior segment involvement was often minimal. After blunt trauma all patients should have a peripheral retinal examination, with indirect ophthalmoscopy and scleral indentation. The best time for this is about one week after injury.

Summary

108 patients have been studied after blunt trauma to the eye; the resulting ocular damage has been related to the site and nature of the impact. The factors involved in such complications as secondary hyphaema, uveitis and abnormal intraocular pressure, and retinal damage have been discussed.

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References

- BLANTON, F. (1964) *Arch. Ophthalm. (Chicago)*, **72**, 39
- COLE, J. G., and BYRON, H. M. (1964) *Ibid.*, **71**, 35
- COLLINS, E. TREACHER (1892) *Trans. ophthalm. Soc. U.K.*, **12**, 180
- COX, M. S., SCHEPENS, C. L., and FREEMAN, H. M. (1966) *Arch. Ophthalm. (Chicago)*, **76**, 678
- DARR, J. L., and PASSMORE, J. W. (1967) *Amer. J. Ophthalm.*, **63**, 134
- GREGERSEN, E. (1962) *Acta ophthalm. (Kbh.)*, **40**, 192
- HENRY, M. M. (1960) *Amer. J. Ophthalm.*, **49**, 1298
- KILGORE, G. L. (1942) *Trans. Amer. ophthalm. Soc.*, **40**, 516
- LORING, M. J. (1958) *Amer. J. Ophthalm.*, **46**, 873
- MOONEY, D. (1972) *Brit. J. Ophthalm.*, **56**, 418
- OKSALA, A. (1967) *Ibid.*, **51**, 315
- SCHEPENS, C. L. (1969) In "Symposium on Retina and Retinal Surgery", *Trans. New Orleans Acad Ophthalm.*, p. 302. Mosby, St. Louis
- SHEA, M. (1957) *Canad. med. Ass. J.*, **76**, 466
- SPAETH, G. L., and LEVY, P. M. (1966) *Amer. J. Ophthalm.*, **62**, 1098
- THYGESON, P., and BEARD, C. (1952) *Ibid.*, **65**, 977
- TÖNJUM, A. M. (1966) *Acta ophthalm. (Kbh.)*, **44**, 650
- WEIDENTHAL, D. T., and SCHEPENS, C. L. (1966) *Amer. J. Ophthalm.*, **62**, 465
- WOLFF, S. M., and ZIMMERMAN, L. E. (1962) *Ibid.*, **54**, 547