TRAUMATIC OR "CONCUSSION" CHRONIC
GLAUCOMA*

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THIS paper is based upon the observation of a series of cases of monocular chronic glaucoma of traumatic origin, and it is suggested that there exists a type of chronic glaucoma hitherto inadequately recognised as a specific clinical entity, characterised by uni-ocular incidence and a history of concussion trauma. It is contended that this chronic glaucoma is one of the several possible results of concussion of the ocular bulb, and that production of this type of glaucoma is a chronic process initiated by trauma, causing a gradual and progressive blockage of the endothelium-lined spaces of the trabecular region by a sclerotic, degenerative or proliferative lesion. It is proposed to summarise and extend views expressed in previous communications on this subject in 1944 (1), 1945 (2), and 1946 (3), in other journals.

Before presenting a selection of case-histories—many of which were kindly sent to me by colleagues—and before considering what conclusions may reasonably be drawn from them, I will describe the case which first drew my attention to the possibility of uni-ocular type of chronic glaucoma.

In March 1940 I was consulted by a man aged 28 years, who told me that his right eye had been going misty for some months. On examination the right pupil was semi-dilated, and reacted only sluggishly to light, the right disc was cupped, and the intra-ocular tension was 40 mm. Hg. There was a moderate contraction of the right nasal visual field, and vision was 6/9. The anterior chamber was of normal depth. The left eye, in which vision was 6/5, appeared normal, but following orthodox procedure in cases of primary chronic glaucoma, I treated both eyes with eserine sulphate $\frac{1}{4}$ per cent. As the intra-ocular tension in the right eye under eserine, even when this drug was used in increasing frequency, never fell as low as 30 mm. Hg, and as the vision continued to appear misty to the patient, I trephined the right eye in July 1940. The tension fell to 20 mm. Hg, at which level it remained for six years.

Acting on the assumption that primary chronic glaucoma is a bilateral disease, I kept the left eye under eserine for ten months. In May 1941, after once again taking the vision, tension and visual field of the left eye, and finding no abnormality in these, nor in the appearance of the optic disc, I concluded that I might have been too ready to accept this case as one of ordinary primary glaucoma, and I did what I should have done at the outset (and what I have never since omitted to do), namely, take a careful history. I then found that eighteen years earlier, this man had fallen from a horse and injured the right side of his face. I made him ask his parents about this accident, as he was only ten at the time of its occurrence. His mother remembered it clearly, because the accident had greatly alarmed her; and she stated that his right eyelids remained swollen for many days. Evidently

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a large haematoma ensued. I took the risk of discontinuing treatment in the left eye, and for the six years during which I subsequently observed this patient, no sign or symptom of glaucoma appeared in this eye.

TABLE I

Case	Age	History	Signs	Treatment
1 Mr. P. W.	28 11/3/1940	Right eyesight had gone misty.	Right eye: Pupil sluggish and larger than left pupil; intraocular tension 40 millimetres of mercury; vision 6/9; visual field: basal contraction small with Ronne's step. Left eye: Normal in all respects.	Eserine both eyes until July, 1940, when right eye was trephined successfully, bringing the tension down to 20 millimetres of mercury. Left eye kept under eserine until May, 1941, when a history was elicited of a severe blow on the right eye from a fall 18 years previously. Eserine discontinued left eye, which was still normal in April, 1945.
2 Mr. H. M.	61 15/11/1940	Right vision falling for past twelve months. Past multiple infections of both syphilis and gonorrhoea. Was once a sailor.	Right eye: All the signs of acute congestive glaucoma. Left eye: Tension normal, but disseminated chorioiditis present. Left visual field full except for scotomata corresponding to the patches of chorioiditis.	Miotics until right eye quiet; then operation with a mechanical trephine. Tension reduced to 20 millimetres of mercury, but cataract developed later. Left eye kept under miotics for three and a half years. until June, 1944, when a history was elicited of seventeen fights, many falls (one causing a fracture of the skull), and blows on the face from bottles in tavern brawls and arguments with women. All treatment stopped. Left eye still normal in June, 1945.
3 Mr. J. W.	63 26/6/1942	Condition previously diagnosed as glaucoma by one doctor and as cataract by another. Using pilocarpine at night in the affected left eye.	Right eye: Normal in all respects. Left eye: Cupped disk, 40 millimetres tension, nasally contracted field with large Ronne's step. Left vision less than 6/60.	Eserine both eyes. In August, 1942, history elicited of injury to left eye from fall from a horse; lids had sutures inserted. Treatment stopped right eye in March, 1943. Right eye still normal in April, 1944. Not seen since.

This case appeared to me of unusual interest, for although acute congestive glaucoma secondary to trauma is well known, nowhere could I find any definite record of chronic glaucoma subsequent to and caused by trauma, except from lens luxation. Moreover, the long period between the injury and the onset of symptoms seemed peculiar, to say the least, and almost made me forsake the theory of cause and effect. However, as case followed case, I began to feel that the matter was worthy of comment, and when, after my first short communication on the subject,¹ other oculists began to send me details of similar cases, I felt encouraged to follow up the trail, and have since read two papers before the Ophthalmological Society of Australia.^{2,3}

Many of my colleagues thought that I had made an observation of some value; others were doubtful that such a variety of traumata could produce so constant a lesion especially, as in many of the

cases, after so long a period of time. Therefore I have now set out the matter in some detail, for if there is indeed such a clinical entity as traumatic uni-ocular chronic glaucoma, or, as I now name it, concussion glaucoma, issues of medical and medico-legal importance are raised.

One issue of medical importance is this: if, as a result of concussion trauma to one eye, or affecting one eye, a state of chronic glaucoma is set up in that eye, there is no need to fear an onset of glaucoma in the other eye, nor any need to treat the other eye. The other and wider medical corollary is, that if so simple a factor as concussion trauma can initiate a chronic glaucomatous process, then primary simple non-inflammatory glaucoma may be in essence more truly simple in nature than the multiple theories of its genesis would suggest.

The issue of medico-legal import is, of course that of compensation, either civil or military, for uni-ocular chronic glaucoma resulting from trauma.

In reviewing this series of cases, comment will be made under four headings:

1. The nature of the lesion.
2. The nature of the causative traumata.
3. The probable aetiological mechanism.
4. General conclusions and comments.

1. THE NATURE OF THE LESION

First, in these cases we are confronted, not with the well recognised condition of traumatic acute congestive glaucoma — though a congestive state may become a terminal stage—but with a true, chronic, progressive non-inflammatory glaucoma which may incidentally exhibit the signs and symptoms of sub-acute attacks, namely rainbow haloes, attacks of pain and blurred vision.

Secondly, almost without exception, a remarkable feature of these cases is a delay in the onset of symptoms, the period of delay ranging from a few weeks to many years after the date of injury. This is not necessarily to say that the start of the glaucomatous process has been delayed; it may merely indicate the delay in discovery by the patient of the symptoms of a chronic and insidious disease. What this delay in the onset of symptoms does emphasize, is that no gross visual upset occurred at the time of injury, in contradistinction to the immediate signs and symptoms seen in cases of acute congestive glaucoma due to gross trauma.

Thirdly, the lesions in this series are uni-ocular, and remain so over long periods of time, in contradistinction to primary non-traumatic chronic glaucoma, which is almost invariably bilateral sooner or later.

2. THE NATURE OF THE CAUSATIVE TRAUMA

Analysis of the cases shows three groups of injuries. These are :—

- (a) Blows upon the eye itself.
- (b) Injuries to the skull, mostly causing either a fracture of the bone or a concussion of the brain.
- (c) Heavy falls, as from a horse or vehicle, or from a height of some feet.

Looked at as a group, these injuries can be regarded as various forms of concussion, and I would suggest that these glaucomatous conditions represent one of the several possible results of concussion of the eyeball. With other possible results everyone is familiar, *e.g.*, striation of the cornea, mydriasis, dislocation of the lens, cataract, irido-dialysis, detachment of the retina and intra-ocular haemorrhage.

Some of those lesions may produce a condition of acute congestive glaucoma. I am suggesting that, in addition to such well-known possible results of concussion trauma, another should be added, namely, the initiation of chronic glaucoma.

3. THE PROBABLE AETIOLOGICAL MECHANISM

How is it that an injury not sufficient to cause an attack of acute congestive glaucoma, could be conceived of as initiating chronic glaucoma? I believe that these cases display the effects of concussion, effects produced either by direct trauma to the cornea or by the pressure of suddenly compressed aqueous humour. Presumably the lesion is in the region through which the aqueous filters away from the anterior chamber, namely in the trabecular meshwork of the cornea adjacent to Schlemm's canal; and I interpret the lesion either as a fibrosis or as a cuticular proliferation ("glass membrane") similar to that recorded by several workers, notably Reese (1944)⁴.

Such an hypothesis, envisaging as it does a chronic lesion, be it fibrotic or degenerative or proliferative, offers an explanation of the remoteness in the time of the history of injury in so many cases. It would also explain the cases due to indirect trauma, such trauma being of a nature to cause a concussion shock to the eye.

4. GENERAL CONCLUSIONS AND COMMENTS

These cases present three factors in common :

- (a) A history of concussion trauma.
- (b) Glaucoma in one eye.
- (c) A non-glaucomatous other eye.

It would appear, then, if these cases are in truth traumatic in origin, that some types of concussion-shock can upset the intra-ocular pressure stabilisation, either by the mechanism I have postulated or in some other way. Similarly, some cases of bilateral chronic glaucoma may be of traumatic origin. However that may be, these cases offer evidence that chronic glaucoma can be caused by trauma, and that such cases show not a static or temporary rise in the intra-ocular pressure, but rather a progressive chronic glaucoma non-inflammatory in type, though some cases display terminal inflammation.

A search of ophthalmic literature only reveals brief and scattered allusions to the possibility of such a lesion. In most accounts and discussions on glaucoma the existence of traumatic chronic glaucoma is not even considered and it would seem that this clinical entity has been missed. If so, why? The omission is explained by the way in which glaucoma has traditionally been presented to us in the text-books. These books divide *primary* glaucoma into two broad groups—namely acute congestive and chronic, but *secondary* glaucoma receives no such grouping. Instead of that we find a long list of causes, among which trauma is mentioned only as a cause of the acute congestive type. A classification of secondary glaucoma into the same two fundamental groups, acute and chronic, might be worth while, for there are other causes of *secondary* chronic glaucoma besides trauma, e.g., chronic uveitis, intumescence and subluxation of the lens, Morgagnian cataract, intra-ocular tumour, epidemic dropsy and hydrophthalmia.

Duke-Elder⁵ has a chapter in his Text Book of Ophthalmology on glaucoma secondary to trauma, but it is evident that what is envisaged is a congestive lesion. He states: "The primary mechanism is undoubtedly an upset of the local nervous control of the circulation, any disturbance of which is generalised over the entire uveal tract by axon-reflexes. Such a disturbance produces a vascular instability with dilation of the capillaries, engorgement of the circulation, stasis and oedema, a mechanism, indeed, comparable to that which occurs in inflammatory glaucoma."

Posner and Schlossman⁶ (1948) give a list of *nineteen* eye conditions associated with primary glaucoma, *forty* general diseases

associated with primary glaucoma, and *twenty-four* causes of secondary glaucoma, the twenty-fourth heading being "Unknown Aetiology." Nowhere in these long lists is concussion trauma mentioned, nor indeed any trauma except perforating wounds, traumatic cataract and dislocation of the lens, and even these three applied to glaucoma as a whole and not specifically, if at all, to chronic simple glaucoma.

The only unmistakable reference to the possible rôle of trauma in the production of a chronic glaucoma, that I have been able to find, is by Reese⁴ (1944). In his summing-up Reese states: "This (cuticular product) may occur as a primary disease with no apparent provocation. It may also occur as a result of inflammation in the anterior chamber or as the result of trauma to the eye." He does not say what kind of trauma, nor does he give any detail of traumatic cases beyond stating that a history of trauma was regarded as a significant factor in six of his twenty-six cases. Reese omitted sixteen other eyeballs with deep-chamber, wide-angle glaucoma, in which the glaucoma was obviously due to obstruction of the trabeculae from sclerosis and not to the formation of a "glass membrane." Here, then, were forty-two cases of glaucoma from trabecular obstruction of one form or another, some of them with a history of trauma.

Conclusion

1. We should not too hastily diagnose every case of chronic glaucoma in one eye as a primary and therefore eventually bilateral disease, for, upon taking a careful case-history, we may find it to be a lesion affecting only one eye, and in origin secondary to concussion trauma.

2. Probably some cases of bilateral chronic glaucoma may have their origin in the shock of a concussion trauma.

The three tabulated cases are selected from a group of 37 observed by myself or by colleagues.

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