HYPOTONY AFTER CATARACT EXTRACTION*

BY

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TREACHER COLLINS (1917), in his classical article on the sequelae of persistent hypotony, stated that the pathological changes in the ocular tissues were "brought about either from the relaxation of tissues usually kept taut or from altered conditions in the circulation". Subsequent reports have only confirmed the accuracy of these observations. Within recent years the physiology of hypotony, especially the influence of neurogenic factors, has been investigated by many authors (Magitot, 1917, 1918; Schmidt and de Decker, 1930; Weekers, 1932; Kronfeld and Lin, 1937; Poos, 1952; Kronfeld, 1953), yet very little consideration has been given to the signs and symptoms produced by a continuing low ocular tension. In this paper the varying nature of both the symptoms and the complications of persistent hypotony will be emphasized.

Experimental studies indicate that, although the posterior part of a limbal wound heals very slowly, a well-closed cataract incision is strong enough to withstand normal intra-ocular forces by the end of nine days (Yasuna, Ojers, Frayer, and Scheie, 1954; Gliedman and Karlson, 1955). Furthermore, it has been shown that when any rise in intra-ocular pressure occurs during the first week after operation aqueous humour will seep through the wound without disrupting it (Dunnington, 1955). The clinical appearance of such drainage is a boggy conjunctival flap throughout the entire length of the incision without evidence of hyphaema. Under normal conditions the oedema of the conjunctival flap disappears and union becomes firm by the end of 10 days. However, when healing is impaired, the aqueous humour continues to leak and the globe remains hypotonic. In as much as this drainage may be constant or intermittent, complete or incomplete, the accompanying signs and symptoms are not uniform. They are determined by the completeness and constancy of the loss of the anterior chamber. It is well, therefore, to subdivide the cases into the following two types, which differ greatly in onset, course, and clinical manifestations:

(1) *Shallow or Absent Anterior Chamber with Choroidal Detachment.*—This type of wound leakage presents a well-known and characteristic clinical picture. The globe is moderately congested and very soft. The wound is flat and appears well, closed, although close inspection may reveal a slight irregularity at the site of

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one or more corneo-scleral sutures. The cornea is clear at first but becomes hazy if the condition persists. The anterior chamber is extremely shallow or absent, a difference detectable only by the corneal microscope. In the presence of a peripheral iridectomy, the pupil is either round or slightly irregular and usually moderately dilated. The vitreous is ballooned forward through the pupillary area and often touches the posterior surface of the cornea. If a complete iridectomy has been made this area of contact extends upward to the wound. Very extensive choroidal detachments are always present. This condition usually occurs spontaneously during the second post-operative week but may be preceded by a hyphaema. In these instances the blood clot, by acting as a wick, prevents closure of the wound. As we have all observed, this form of hypotony may persist from one day to several weeks. Although most of these leaking wounds close spontaneously in a few days without sequelae, persistence of the condition may lead to irreversible corneal changes and secondary glaucoma. Consequently, many measures have been advocated within recent years to insure prompt and permanent restoration of the anterior chamber. Early reformation has been facilitated by the use of a slanting incision, physiologic glue (thrombin), and tight closure with deep corneo-scleral sutures; but subsequent loss of the anterior chamber is still a frequent happening. When the anterior chamber is absent, there is great variation in the speed with which permanent corneal opacities and peripheral anterior synechiae develop. However, it does appear to be largely influenced by the completeness of the contact between the posterior surface of the cornea and the intra-ocular tissues. Chandler (1947, 1954) believes that four or five days is the maximum time an eye will tolerate an absent anterior chamber without developing undesirable sequelae. Kronfeld (1954), in an analysis of 83 cases with delayed reformation or late loss of the anterior chamber, observed no cases of glaucoma among the 66 patients in whom restoration had been completed by the sixth post-operative day, but found six cases of glaucoma among the seventeen patients in whom the anterior chamber remained absent for 9 or more days. In an attempt to evaluate the significance of prolonged absence of the anterior chamber, we reviewed the records of 800 patients upon whom we had performed cataract extractions. In this series there were eighty cases in which the anterior chamber was absent on or after the 9th post-operative day and did not reform for at least 4 days. Twelve (15 per cent.) of these eighty eyes developed glaucoma: in six of them the ocular tension has been brought under control, by miotics, surgery, or both, and good vision has been maintained. In four of the other six cases the vision remains good (20/50 or better), but for 2 or more years the ocular tension has been consistently above 35 mm. Hg Schiötz. The glaucoma became absolute in the other two cases and enucleation was necessary in one of them. The pathological diagnosis in this eye was glaucoma secondary to epithelization of the anterior chamber. In this series permanent opacification of the cornea occurred only in the two cases of absolute glaucoma. Thus it would seem that although glaucoma is to be expected when the anterior chamber remains flat for 4 or more days during the second or third post-operative week, secondary corneal opacification does not develop with the same frequency.

(2) *Delayed Fistulization without Loss of Anterior Chamber.*—In contrast to the findings just described and usually attributed to post-operative hypotony, there is another symptom complex produced by fistulization of an operative wound. In

these cases the fistula is small and is the result of a localized defect in healing. This imperfection may be the final outcome of a large opening (Type 1), but is more frequently seen in eyes without a history of delayed reformation or subsequent loss of the anterior chamber. These tiny dehiscences are produced by a slough around a deep suture or by incarceration in the wound of a fragment of lens capsule or iris. Because of their minuteness they are prone to become covered with a thin layer of epithelium which ruptures from time to time. Thus the symptoms are characteristically intermittent. At first the complaints appear trivial, but as the condition progresses they assume serious proportions. The usual history is an uncomplicated operation with a normal immediate post-operative course and a good visual result. After a few weeks or months the eye becomes vaguely uncomfortable, with episodes of blurred vision and profuse lacrimation. Frequently the patient states the discomfort disappears promptly with the onset of tears but that the vision clears more slowly. Although the symptoms often occur on awakening and disappear within 30 minutes, they may take place at any time and may persist for hours. Between attacks the vision is unaffected, the eye is white, the cornea is clear, and the anterior chamber is of normal depth and free of microscopic signs of inflammation. The wound may be smooth but often contains a barely detectable localized irregularity or a small cystoid cicatrix. The pupil is either round or slightly distorted with the apex of the irregularity directed towards the site of the opening. The ocular tension is either normal or low. On palpation the eye at times becomes soft and a simultaneous shallowing of the anterior chamber is observed, but when manipulation is stopped it usually regains normal depth. The fluorescein test may or may not be positive. As weeks pass the episodes usually increase in frequency and in severity, the vision begins to fail, and the globe becomes tender and injected. Examination at this time reveals mild ciliary congestion, an anterior chamber of normal depth with a moderate number of cells, fine posterior synechiae, and clouding of the anterior part of the vitreous. Choroidal detachment is very rarely seen unless the hypotony is extreme. In these instances papilloedema and retinal oedema in the macular region are also encountered. There may be a thin membrane on the posterior surface of the cornea adjacent to the area of fistulization with the surrounding cornea showing epithelial oedema and stromal striation. However, in other cases, the only objective findings may be a mild iritis and clouding of the anterior part of the vitreous. In most untreated cases the symptoms progress and vision is eventually lost. The sequelae are opacification of the cornea or bullous keratitis, epithelization of the anterior chamber, iris bombé, secondary glaucoma, vitreous opacification, and endophthalmitis.

When the tension remains more or less normal throughout the entire course of the disease, it is difficult to demonstrate the exact site of drainage because the fluorescein test as usually applied yields negative findings. However, a positive reading can often be obtained if the real nature of the condition is suspected and the test done in the following manner:

After the eye has been anaesthetized by instillation of tetracaine hydrochloride solution (0.5 per cent.), firm pressure is made along the incision with a tightly wound cotton applicator moistened with 2 per cent. aqueous solution of fluorescein. The small amount of dye thus applied along the incision, combined with direct pressure to the globe, frequently discloses the exact location of the tiny opening; but in as much as drainage is often intermittent, the test may have to be repeated several times.

Although we have observed cases with long-standing profound hypotony in which no such irritative symptoms have been present, once they begin they do not subside spontaneously. Cases with intermittent fistulization are to be differentiated from those with hypotony produced by excessive filtration. This latter condition is a common finding after corneo-scleral trephining, and as long as the conjunctival covering of the filtering area remains intact the eye is free from symptoms. However when the thin layer of epithelium ruptures, the irritative signs and symptoms of intermittent fistulization develop. In many cases both the patient and the oculist ignore the mild attacks of lacrimation and blurred vision, until a fulminating secondary infection sets in.

Discussion

Little attention need be directed to the complications that follow a prolonged absence of the anterior chamber because the investigations of Kronfeld (1953), Post and Harper (1953), Weekers and Delmarcelle (1953), and Dunnington (1955) have clearly demonstrated the frequency with which glaucoma occurs in these cases. An analysis of eighty cases with this complication showed that 15 per cent. of them developed a raised ocular tension. In contrast to the widespread recognition accorded this type of wound leakage, scant consideration has been given to the equally serious delayed form. The triviality of the initial signs and symptoms account for both delayed recognition and postponed therapy. If these fistulae are allowed to persist, epithelization of the anterior chamber is the most frequent complication. Delayed fistulization was demonstrable in eight of ten cases of histologically proven epithelization. In the two others, the anterior chamber was slow in reforming, and it also formed and emptied several times during the first month after operation. Another disastrous end-result of this second type of hypotony is the so-called "late infection". Five such cases were encountered in private practice and a history indicative of intermittent drainage was obtained in all of them. In three of these five patients the mild symptoms were of several years' standing. Both papilloedema and retinal oedema have been seen in cases of post-operative hypotony. In one instance the oedema of the retina was so severe that the referring oculist had made a diagnosis of detachment of the retina. In others some permanent reduction in visual acuity has resulted from long-standing oedema in the macular region. Similarly, a persistent uveitis associated with increasing opacification of the vitreous can be caused by a fistulizing wound.

Treatment

It is difficult to evaluate any treatment in Type 1 because spontaneous reformation is the rule. However, if leakage persists for more than 4 or 5 days, serious consideration should be given to removal of the corneo-scleral sutures, particularly the deeply inserted ones. The drainage track they

create is a very common cause for delayed loss of the anterior chamber. Many believe the condition can be cured by an injection of air into the anterior chamber, but Chandler (1947), Reese (1948) and Leahey (1951), have pointed out that this procedure is not sufficient. They think air injection must be combined with release of the subchoroidal fluid, and they consider the latter as the most important single measure in therapy. In our experience restoration of the anterior chamber by an injection of air has not proved satisfactory. In many cases the chamber became flat again as soon as the air absorbed, and in others the air got behind the iris and pushed it into the wound. We believe that in the type of affection which occurs during the first 2 weeks after operation, conservatism is the treatment of choice. In those rare instances where complete contact persists, surgical treatment is indicated. If no leak can be demonstrated, the procedure of Reese (1948), Leahey (1951) and Chandler (1954), should be employed; but when localized drainage can be detected, closure of the opening combined with evacuation of the subchoroidal fluid appears to be the logical treatment. The contents of the globe are reduced by withdrawing this fluid, and this causes the vitreous to recede and removes all pressure from the wound. The normal resilience of the structures enables the surfaces of the wound to fall into apposition, and the lack of tension permits them to remain in this advantageous position.

When a shallow anterior chamber is present a few weeks after cataract extraction, a discrete spot of drainage can usually be detected by the fluorescein test. In the early stages the fistula is often found at the site of a deeply placed suture. Removal of the suture and application of a mild cauterant, *e.g.* silver nitrate, is at times helpful. However, if the fistula persists or is delayed in its onset, surgical treatment is necessary. In as much as these fistulae are at times lined with epithelium, the operative procedure should include:

- (a) excision of the cystoid scar
- (b) light cauterization and curettage around the opening
- (c) closing the dehiscence with a suture
- (d) covering the opening with a fornix-based conjunctival flap.

This technique, which had been described previously (Dunnington and Regan, 1949) is neither new nor original; it is essentially the same as used by the late J. M. Wheeler (1939) for closing post-operative fistulae.

Summary

Persistent hypotony after cataract extraction is invariably the result of diffuse or localized wound leakage. When the drainage is diffuse spontaneous cure usually takes place and therapy is needed only to prevent the development of damaging adhesions. However, when the drainage is localized and persistent, the fistula must be closed. The symptoms vary with the amount of drainage. In severe cases there is complete absence of

the anterior chamber, extensive choroidal detachment, and a soft irritable eye; in mild cases the anterior chamber is present, choroidal detachment is rarely visible, ocular tension is often normal, and the irritative symptoms are slight. The subsequent courses of the two types are also very different. Persistent absence or extreme shallowness of the anterior chamber is followed by central corneal damage, extensive peripheral anterior synechiae, and secondary glaucoma. The sequelae of the second type are progressive opacification of the vitreous, increasing uveal reaction, macular oedema, epithelization of the anterior chamber, secondary glaucoma, and late infection.

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