

Posterior synechiae after glaucoma operations: aggravation by shallow anterior chamber and pilocarpine

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SUMMARY Posterior pupillary synechiae affect a proportion of eyes subjected to iridectomy with or without drainage operation because (1) aqueous humour bypasses the pupil; (2) traumatic iridocyclitis occurs; (3) there is immobility of the iris in the iridectomy sector; (4) in eyes with angle closure glaucoma closer apposition of the iris to the anterior lens capsule increases the tendency; (5) pilocarpine aggravates (4) both in angle closure glaucoma and open angle glaucoma and produces a small immobile pupil facilitating pupillary membrane formation (occlusio pupillae). Pilocarpine should be avoided if possible as medical treatment at any time after a drainage operation. A beta blocker is the drug of choice. To eliminate posterior synechiae over a fair number of degrees of pupil (say 30°) sector iridectomy can be done.

Iridectomy is a well established procedure in the treatment of glaucoma,¹ specifically angle-closure glaucoma (ACG).² In patients with closed-angle glaucoma (CAG) 45–70% of untreated fellow eyes will develop closed angles within 10 years of initial presentation.^{3,4} Prophylactic peripheral iridectomy—or laser iridotomy—is therefore advocated for prevention.⁵ There are some complications of surgical iridectomy.^{5–8} However, these are usually considered to be infrequent or innocuous enough, so that the operation is done in preference to the risk of acute closed-angle glaucoma.

This paper examines the prevalence and distribution of posterior synechiae, a common side effect of surgical procedures in the treatment of glaucoma—particularly of surgical iridectomy—and the role of postoperative pilocarpine as an aggravating factor, especially in eyes with shallow anterior chambers.

This series preceded our adoption of laser iridectomy for ACG, but, as far as posterior synechiae are concerned, laser iridectomised eyes behave very much like eyes which have had surgical iridectomy.^{9,10} Indeed, posterior synechiae are easier to miss after laser iridectomy, so that the surgeon must remain vigilant with this newer treatment.

Patients and methods

Retrospective analysis of 63 consecutive patients attending a glaucoma clinic who were operated on for glaucoma determined the prevalence of postoperative posterior synechiae and their association with the postoperative administration of pilocarpine. Sixty-three eyes from 63 patients (mean age 69.8, SD 10.4 years) were included in the study. There were 39 eyes from 39 patients with closed-angle glaucoma (mean age 69.0, SD 9.9 years) and 24 eyes from 24 patients with open-angle glaucoma (mean age 71.0, SD 11.1 years). There was no significant sex predilection (30 males : 33 females). Patients with 'secondary' glaucoma were excluded. All patients had had glaucoma operations during the preceding 15 years. The classification of surgical procedures is shown in Table 1. A detailed history was recorded from each patient, with particular reference to postoperative medical treatment, which information was available from the case records. The presence of posterior synechiae was assessed by (attempted) dilatation of pupils by guttae 1% tropicamide and guttae 10% phenylephrine. A diagram of the ensuing pupil size and shape was drawn, and the presence or absence of posterior synechiae noted on a clock diagram divided into 12 sectors.

Only one eye was chosen from each patient, since both eyes in one patient can be expected to behave similarly; thus each individual observation would not then be completely independent of all others. The choice was random: right eyes were chosen from patients whose years of birth ended in even numbers and left eyes from odd numbers.

Results

Comparisons were made between the prevalence of posterior synechiae in patients who had required operation for ACG/CAG (excluding acute cases) and for open-angle glaucoma (OAG). Each group was subdivided into those who had or had not required pilocarpine afterwards. Assessments were 'single masked,' and significance was assessed by the χ^2 test with Yates's correction. The results are shown in Tables 2 and 3.

Even without pilocarpine some posterior synechiae were present: 6/19 for CAG (Table 2) and 2/10 for OAG (Table 3). Although the proportion is

higher for CAG, the difference is not significant ($\chi^2=0.05$, DF=1, $p>0.05$).

The presence of posterior synechiae was highly significantly associated with postoperative pilocarpine as judged by a comparison between eyes which had received no pilocarpine at any time after operation and those which had received the miotic, in each category separately: see Table 2 for closed-angle glaucoma ($\chi^2=14.4$, DF=1, $p<0.001$), and Table 3 for open-angle glaucoma ($\chi^2=10.3$, DF=1, $p<0.001$), and both together CAG+OAG ($\chi^2=27.1$, DF=1, $p<0.001$). The type of iridectomy performed was not a significant factor in the predisposition to posterior synechiae in closed-angle glaucoma patients, there being no significant difference between the prevalence of posterior synechiae in patients who had sector iridectomy and those who had peripheral iridectomy ($p>0.05$).

Fig. 1 shows a typical eye with ring posterior synechiae, only revealed by powerful mydriatics.

The sectors of pupillary circumference occupied by posterior synechiae are shown in Table 4 (and diagrammatically in Fig. 2). 83% of all patients had either no posterior synechiae (40%) or total posterior synechiae (43%); only 17% had an intermediate degree of posterior synechiae (95% confidence interval: 9%–25%). Of those eyes developing posterior synechiae there was involvement of the whole pupil circumference in 76% of CAG patients, which is more than the 62% found in open-angle glaucoma patients (71% of all glaucoma subjects), but this difference was not significant ($\chi^2=0.31$,

Table 1 *Glaucoma surgery in 63 eyes from 63 glaucoma patients*

	<i>Closed-angle glaucoma</i>	<i>Open-angle glaucoma</i>
Peripheral iridectomy	22	3
Trabeculectomy	5	11
Sector iridectomy	5	0
Sector iris inclusion	2	2
Scheie's operation	3	2
Trephine	2	6
Total	39	24

Table 2 *Prevalence of postoperative posterior synechiae in 39 eyes from 39 patients with closed-angle glaucoma*

	<i>Posterior synechiae absent</i>	<i>Posterior synechiae present</i>
No pilocarpine postoperatively	13	6
Pilocarpine postoperatively	1	19

$\chi^2=14.4$, DF=1, $p<0.001$.

Table 3 *Prevalence of postoperative posterior synechiae in 24 eyes from 24 patients with open-angle glaucoma*

	<i>Posterior synechiae absent</i>	<i>Posterior synechiae present</i>
No pilocarpine postoperatively	8	2
Pilocarpine postoperatively	1	13

$\chi^2=10.3$, DF=1, $p<0.001$.

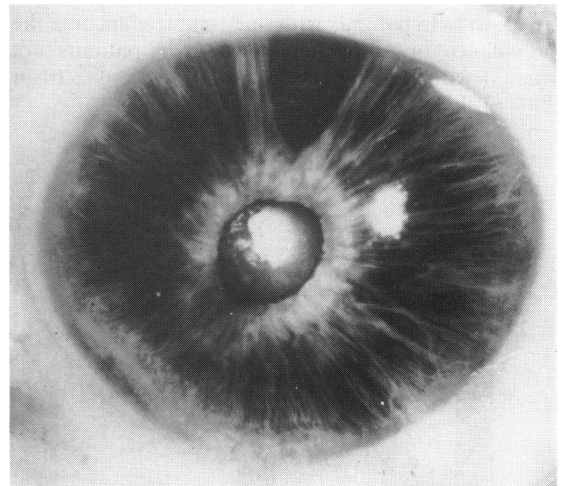


Fig. 1 *Total posterior synechiae in an eye operated on for chronic CAG—owing to the bypassing of the pupil by aqueous, plus traumatic iridocyclitis, aggravated by pilocarpine and a shallow anterior chamber—become obvious only when strong mydriatics are used.*

Table 4 Proportion of pupil circumference occupied by posterior synechiae in 63 eyes from 63 glaucoma patients

	0°	30°	60°	90°	120°	150°	180°	210°	240°	270°	300°	330°	360°
Closed-angle glaucoma (n=39)	14	2	2	1	0	0	1	0	0	0	0	0	19
Open-angle glaucoma (n=24)	11	1	2	0	0	0	2	0	0	0	0	0	8
Total (n=63)	25	3	4	1	0	0	3	0	0	0	0	0	27

DF=1, p=>0.05). Of eyes with subtotal posterior synechiae the peripheral iridectomy quadrant was always involved (other sectors might be free from posterior synechiae), but the numbers were too small for analysis.

Discussion

The prevalence of posterior synechiae in closed-angle glaucoma following peripheral iridectomy, without postoperative pilocarpine, has previously been reported to be 33% with a predilection for the iridectomy quadrant.¹¹ In the present study the prevalence of posterior synechiae in closed-angle glaucoma following a number of different surgical procedures in patients not treated with postoperative pilocarpine was remarkably similar at 32%. In ACG patients after peripheral iridectomy alone the prevalence was 3/12 or 25%. A similar tendency exists in cases of laser iridectomy, with a prevalence reported as 14%¹⁰ Almost all the points discussed here can be applied to this newer technique. Three of 22 patients developed posterior synechiae after argon laser iridectomy, prevented in 19/22 by topical steroids and mydriasis.⁹ In patients with open-angle glaucoma the prevalence of posterior synechiae in patients not treated with postoperative pilocarpine was 2/10 or 20% (Table 3).

A clinician may remain complacent about the prevalence of posterior synechiae in his surgical cases unless and until he has reason to attempt to dilate the pupils with a mydriatic.

Given the validity of the proposition that posterior synechiae do not develop spontaneously in a normal eye (we present no evidence here, other than clinical impression, for that null hypothesis), it is noteworthy, though not surprising, that 8/29 eyes (Tables 1 and 2) not receiving postoperative pilocarpine developed posterior synechiae, in spite of mydriatic treatment, with or without steroids, in the immediate postoperative period. We would attribute that finding to several factors in order of importance:

- (1) All aqueous humour will take the line of least resistance through the iridectomy (peripheral or sector), so that no aqueous flow will lift the pupil as it enters the anterior chamber.
- (2) In the immediate postoperative period some generalised traumatic iridocyclitis must be present to make the whole iris sticky and synechiae-prone.
- (3) In eyes with peripheral iridectomy the remaining bridge of iris between pupil and iridectomy probably suffers ischaemic inflammation as well as a greater traumatic iridocyclitis than other areas of iris (that property would be minimal in laser iridectomy).
- (4) In eyes with peripheral iridectomy, especially large ones, the involved sector of iris will be relatively immobile, hence tending to allow posterior synechiae to form more readily because of factors 1 to 3 above (a minimal effect in laser iridectomy).

As the prevalence of posterior synechiae in ACG and OAG patients treated with postoperative pilocarpine was 95% and 93% respectively, one may reasonably conclude there was a significant association with miotics.

From first principles, and because of the slightly higher prevalence of posterior synechiae in ACG patients (32%) than in OAG patients (20%), we would add to the above list:

- (5) In ACG patients greater pressure contact between iris and lens because of the more anterior plane of the pupil in ACG eyes than in OAG eyes.

Since pilocarpine almost eliminated that 32% versus 20% discrepancy, two further aggravating factors are added:

- 6 (a) Immobility of the pupil miswed by pilocar-

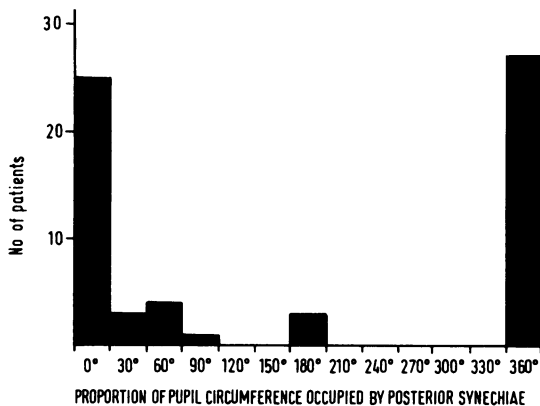


Fig. 2 Proportion of pupil circumference occupied by posterior synechiae.

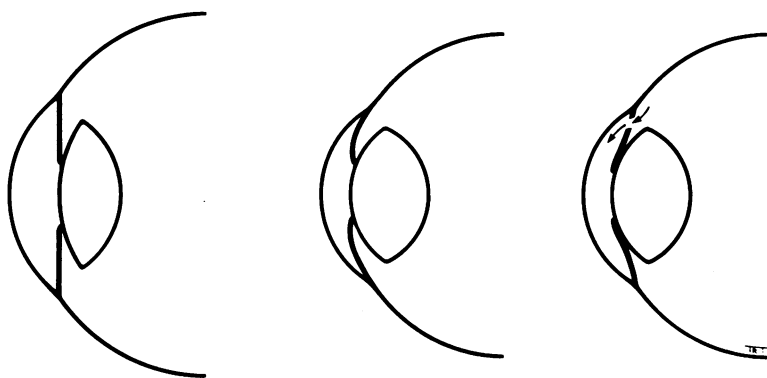


Fig. 3 *The emmetropic or myopic eye on the left has less risk of posterior synechiae because the pupil probably exerts little pressure on the lens. The iris on the right is very likely to develop wide posterior synechiae, especially if inflamed by operative traumatic iridocyclitis and/or miotic treatment, because aqueous humour will take the line of least resistance through the peripheral iridectomy, leaving a wide area of iris constantly pressing on the anterior lens surface. The central diagram shows an eye predisposed to angle-closure glaucoma, with iris bombé.*

pine; (b) even greater pressure contact between iris and lens in both ACG and OAG eyes because of pilocarpine than implied in (5) above.

Anatomical predisposing factors in the pathogenesis of posterior synechiae are illustrated in Fig. 3.

A further disastrous consequence of the small miosed pupil is the ease with which a membrane may fill it (occlusio pupillae), resulting in loss of vision. We have not tried optical iridectomy alone in such cases, because they are impossible to differentiate clinically from cataract.

We also suspect that the posterior synechiae in iridectomised ACG eyes will be more extensive radially than in OAG eyes, but evidence for that would be difficult to obtain.

It is our clinical impression that, once posterior synechiae have started, it is difficult to prevent their spreading to involve the whole pupil, since immobility of the pupil immediately adjacent to an established synechia must facilitate the advance. Even frequent movement of the free areas of pupil by maximal atropine or other anticholinergic plus sympathomimetic will mitigate that fixity only to a limited extent. Table 4 and Fig. 2 (as well as Fig. 1) we would interpret as strongly supporting that 'all-or-none' phenomenon. 40% of eyes show no posterior synechiae, and 43% show total posterior synechiae, whereas only 17% show an intermediate involvement (with a 95% confidence interval of 9%–25%).

Seclusio pupillae (100% posterior pupillary synechiae) can readily occur in eyes which have had iridectomy, because no aqueous will pass under and lift the pupil, preferring the line of least resistance through the iridectomy. The presence of the iridectomy allows the eye to avoid iris bombé of course.

DELETERIOUS EFFECT OF POSTERIOR SYNECHIAE
Do posterior synechiae matter? We have already mentioned above the tendency for a membrane to form across the miosed pupil. We would also expect

posterior synechiae to become predisposed in the course of time to cataract formation because of damage to the lens capsule. By analogy there is some support for that from the generally accepted view that a penetrating injury or iridocyclitis tends to be complicated by cataract, at least in the long term. We will not present some statistics which did show a lower visual acuity in eyes with total posterior synechiae (i.e., peripheral iridectomy) than in others (most with sector iridectomy), because the surgeons were different in the two groups.

In all five eyes which suffered flat chamber for several days immediately after drainage operations (these eyes have not been included in the series reported here), the visual acuity was eventually reduced to counting fingers or hand movements. It would not be justifiable to blame the total posterior synechiae entirely for the very poor vision due to eventual cataract.

Apart from the risk of occlusio pupillae and cataract, fixity of the pupil in response to light and the near reflex will probably be little handicap.

PREVENTION

Even if the possible deleterious effects mentioned in the preceding paragraph are denied, it would nevertheless be reasonable to claim that, if posterior synechiae can be prevented by safe treatment, that should be done. Steroids or non-steroidal anti-inflammatory drugs for several weeks before and after operation probably have a place, in spite of the added risk of infection when the former are used. In the immediate postoperative period we aim to keep the pupil semidilated *and* moving, with careful monitoring especially to avoid overdilatation in ACG. For a blue iris or easily dilated pupil (for example, in a younger person) tropicamide 1% each evening supplemented by adrenaline 1:1000 or phenylephrine 5% each morning (lest systemic absorption of the adrenergic at night produce

insomnia) for 7–10 days will usually suffice. In dark brown or very immobile irises homatropine or even atropine each evening or twice daily, plus phenylephrine 10% each morning, may be necessary.

A closely similar situation arises with posterior synechiae in iridocyclitis, although aqueous is passing through the pupil in these eyes. In cases of iridocyclitis we apply maximum effort to avoid any posterior synechiae, or to eliminate them entirely if at all possible, by means of subconjunctival glucocorticoid and mydricine plus maximum mydriatics (atropine and phenylephrine) at the patient's first visit. Repeat maximal mydriasis often succeeds in breaking down residual synechiae on the 2nd or 3rd day following subconjunctival steroids, unless of course the iridocyclitis is long-standing. If some residual posterior synechiae remain, we prescribe atropine and phenylephrine 10% (the latter several times daily), at least while there is active iridocyclitis and for some weeks afterwards to impose maximum movement on sectors of free pupil, which minimises the spread from foci of fixed posterior synechiae.

We avoid pilocarpine if at all possible when medical treatment is required to control pressure at any time postoperatively, even long after the operation, because of the very high risk of total posterior synechiae.

In this situation we regard a topical beta blocker as the first line of defence, and it is usually adequate, because it has no effect on the pupil. Timolol's effectiveness after operation on CAG is already established.¹² (We also regard a beta blocker as a valuable adjuvant to weak pilocarpine for a fellow ACG eye awaiting iridectomy, because the reduced production and flow of aqueous humour will reduce the height of the iris bombé. The same consideration will apply to those occasional patients who unfortunately refuse both surgical and laser iridectomy.) Occasionally pilocarpine does have to be added to a beta blocker, but we are always very reluctant to do that.

A PLACE FOR SECTOR IRIDECTOMY

If we suspect that an eye will need medical treatment, especially pilocarpine, after operation for glaucoma, we perform a sector iridectomy. Indeed, the need for medical supplementation to a drainage operation is frequent enough, especially in the long term, for us to favour sector iridectomy in most drainage procedures. Rather similarly, in ACG eyes which we judge may not respond completely to peripheral iridectomy, surgical or laser, but which probably do not need a drainage procedure, we do a sector

iridectomy. In the latter case our rationale is two-fold: in the operation of sector iridectomy the iris has to be pulled well out of the eye to allow the pupil to present, so that the breaking down of the freshest goniosynechiae inferiorly is facilitated, unlike the situation in the less disturbing peripheral iridectomy.^{12–16} A more important rationale applies to both groups, whether or not pilocarpine is used after operation: even if a membrane does spread inwards into the pupil area from the edge of a pupil immobilised by total posterior synechiae, in the sector where the pupil margin is missing that complication cannot occur. The British climate makes the glare through a sector iridectomy more tolerable than in sunnier situations.

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References

- 1 Graefe A von. Über die Iridectomie bei Glaucom und den glaucomatosen Process. *Graefes Arch Clin Exp Ophthalmol* 1857; **3**: 456–555.
- 2 Murphy MB, Spaeth GL. Iridectomy in primary angle-closure glaucoma. *Arch Ophthalmol* 1974; **91**: 114–22.
- 3 Mapstone R. Glaucoma. In: Davidson SI, ed. *Recent advances in ophthalmology*. 6th ed. Edinburgh: Churchill Livingstone, 1983: 23.
- 4 Duke-Elder S. *System of ophthalmology*. Kimpton: London, 1969; **11**: 621.
- 5 Douglas WHS, Strachan IM. Surgical safety of prophylactic peripheral iridectomy. *Br J Ophthalmol* 1967; **51**: 459–62.
- 6 Ghoshal TK, Blaxter PL. Results of peripheral iridectomy in closed-angle glaucoma. *Br J Ophthalmol* 1969; **53**: 110–5.
- 7 Krupin T, Mitchell B, Johnson MF, Becker B. The long-term effects of iridectomy for primary acute angle-closure glaucoma. *Am J Ophthalmol* 1978; **86**: 506–9.
- 8 Williams DJ, Gills JP, Hall GA. Results of 233 peripheral iridectomies for narrow-angle glaucoma. *Am J Ophthalmol* 1968; **65**: 548–52.
- 9 Abraham RK, Miller GL. Out-patient argon laser iridectomy for angle-closure glaucoma: a two year study. *Ophthalmology* 1975; **79**: 529–38.
- 10 Abraham RK. Procedure for out-patient argon laser iridectomies for angle-closure glaucoma. *Int Ophthalmol Clin* 1976; **16**: 1–14.
- 11 Phillips CI, Snow JT. Peripheral iridectomy in angle-closure glaucoma—a common complication. *Br J Ophthalmol* 1967; **51**: 733–8.
- 12 Phillips CI. Timolol in operated closed-angle glaucoma. *Br J Ophthalmol* 1980; **64**: 240–6.
- 13 Phillips CI. Sector iridectomy and timolol in closed-angle glaucoma. *Res Clin Forums* 1981; **3**: 43–7.
- 14 Phillips CI. Closed-angle glaucoma: significance of sectoral variations in angle depth. *Br J Ophthalmol* 1956; **40**: 136–43.
- 15 Foulds WS, Phillips CI. Some observations on chronic closed-angle glaucoma. *Br J Ophthalmol* 1957; **41**: 208–13.
- 16 Phillips CI. Aetiology of angle-closure glaucoma. *Br J Ophthalmol* 1972; **56**: 248–53.

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