OCULAR HYPERTENSION: CORRELATION OF ANTERIOR CHAMBER ANGLE WIDTH AND RISK OF PROGRESSION TO GLAUCOMA

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SUMMARY

Twenty five patients with ocular hypertension (OH) and a narrow angle and 34 patients with OH and an open angle were followed for an average of six years. One eye of each patient had been randomly assigned to treatment with topical timolol. A shallow axial anterior chamber depth and a narrow angle (van Herick Grade 2 or less) was associated with the development of angle-closure glaucoma (ACG) in the narrow angle group, but the open angle group developed significantly more visual field loss. Nine patients (36%) in the narrow angle group developed ACG, and nine patients (26·5%) in the open angle group developed glaucomatous discs and field loss. The reduction in intraocular pressure due to topical timolol was equal in the narrow angle and the open angle groups, but topical timolol did not protect against the development of angle-closure glaucoma or visual field loss.

Ocular hypertension (OH) is the principal known risk factor for the development of open angle glaucoma,[1,2] and an eye with a narrow anterior chamber angle is at risk of developing angle-closure glaucoma.[3,4,5]). The decision whether or not to treat ocular hypertension is usually made after an assessment of the relative risk of the eye developing glaucomatous visual field loss. Factors such as a very high intraocular pressure (IOP), a family history of glaucoma, or diabetes mellitus are associated with a greater likelihood of progression to glaucoma, influencing the decision to treat the patient. In managing eyes with ocular hypertension and narrow angles (OHNA), the clinician must also assess the risk of the eye developing angle-closure glaucoma (ACG) and decide whether the narrow angle itself is primarily responsible for the raised pressure. If relative pupillary block is present and causing peripheral iris bombe with obstruction of the trabecular meshwork, then a peripheral iridectomy or laser iridotomy (LI) might lower the IOP as well as prevent the development of ACG.[6,7,8]

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The purpose of this study was to discover whether a narrow angle should be regarded as a specific risk factor in patients with ocular hypertension, either for the development of visual field loss or angle-closure glaucoma, and to determine whether treatment with a topical beta-blocker is of benefit. Mapstone believed OH could be a manifestation of a covert angle-closing mechanism called 'partial angle closure' which he said could occur even in eyes with open angles.[8,9,10] A further aim of this study, therefore, was to examine the outcome of two different types of OH patient—one with open angles who would not normally be considered at risk of ACG, and one with narrow angles.

This paper presents the outcome of a group of 61 ocular hypertensive patients, 26 of whom had narrow angles and shallow anterior chambers and 35 with open angles. One eye of each patient was randomly assigned to receive topical beta blocker therapy, the fellow eye serving as control, to determine whether treatment with a topical beta blocker was protective against the development of glaucoma.

MATERIALS AND METHODS

The patients selected for this study were drawn from a population of individuals referred to the Glaucoma Unit at St Pauls Eye Hospital for assessment of an incidental finding of a narrow AC angle and/or OH. The study was begun in 1982 by the late Roy Mapstone who performed a pilocarpine phenylephrine provocative test (PPPT) on all patients. If the test was positive, Mapstone believed an angle-closing mechanism had been identified, even in an eye with an apparently open angle, and the eye would undergo a peripheral iridectomy.[8,9,10] If the PPPT was negative in both eyes, and the patient had OH, the patient was invited to take part in the ocular hypertension study in which one eye was randomly assigned to treatment with topical timolol 0.5% BD, with no treatment to fellow eye which would act as a control.

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Entry criteria for the study included the following:
—an IOP of more than 21 mmHg in both eyes
—normal visual fields on Friedmann field analysis
—normal optic discs
—no history of anterior segment disease
—clear optic media
—open or narrow anterior chamber angles.
—An exclusion criterion was any contraindication to beta blocker therapy.

At entry to the study, stereo optic disc photographs were taken and the anterior chamber depth (ACD) was measured with the Haag-Streit pachymeter. The anterior chamber angle was examined by Goldmann gonioscopy and the width of the angle was graded by van Herick's method.[3] The angle was considered to be narrow if the peripheral ACD close to the limbus was one quarter or less of the thickness of the cornea (van Herick Grade 2 or less), and open if the peripheral ACD was equal to or greater than half the corneal thickness (van Herick Grade 3 or 4). If the IOP rose to more than 31 mmHg in any eye pilocarpine would be added to study eyes, and control eyes would receive topical timolol. If any sign of glaucomatous damage became evident in follow-up, that eye was to be treated accordingly. Follow-up examination for the first three years was every three months and included Goldmann applanation tonometry, optic disc assessment with the direct ophthalmoscope and slit-lamp examination of the anterior segment.

After December 1986, one of the authors (PKW) took over the care of the patients in this study, and at this point a review of all patients was made. This included intraocular pressure (IOP) measurement, indentation gonioscopy with the Zeiss 4-mirror goniolens to reveal peripheral anterior synechiae (PAS) of the AC angle (Figs 2a and 2b), anterior chamber depth (ACD) measurement with the Haag-Streit pachymeter, stereoscopic optic disc biomicroscopy, and visual field analysis firstly with the Hensen Screener and later with the Humphrey Visual Field Analyser, using programme 24-1 or 24-2. Progression to glaucoma was judged to have occurred on the basis of visual field loss on Humphrey Field Analysis of four or more contiguous points depressed by eight decibels or more, present on two or more consecutive fields. Humphrey Statpac analysis was performed on all fields and those fields judged unreliable by this analysis (low reliability indices) were not considered to be evidence of either normality of field or field loss. Any dissimilarity between the initial photograph of the disc and the current examination prompted further field analysis to detect field loss.

The classical signs of glaucomatous disc damage were taken to be vertical increase in cup height, increase in cup depth, pallor, and thinning or notching of the neuro-retinal rim.[11] If there was no evidence of glaucoma at this point a trial period without treatment was made with re-assessment of the IOP at regular intervals. A minimum four week wash-out period was allowed before repeat IOP check. Those eyes in which the IOP was below 25 mmHg off all treatment remained untreated, but if the IOP was more than 25 mmHg or if there were other risk factors treatment was restarted.

The presence of established PAS visible on indentation gonioscopy was taken as evidence that creeping ACG was

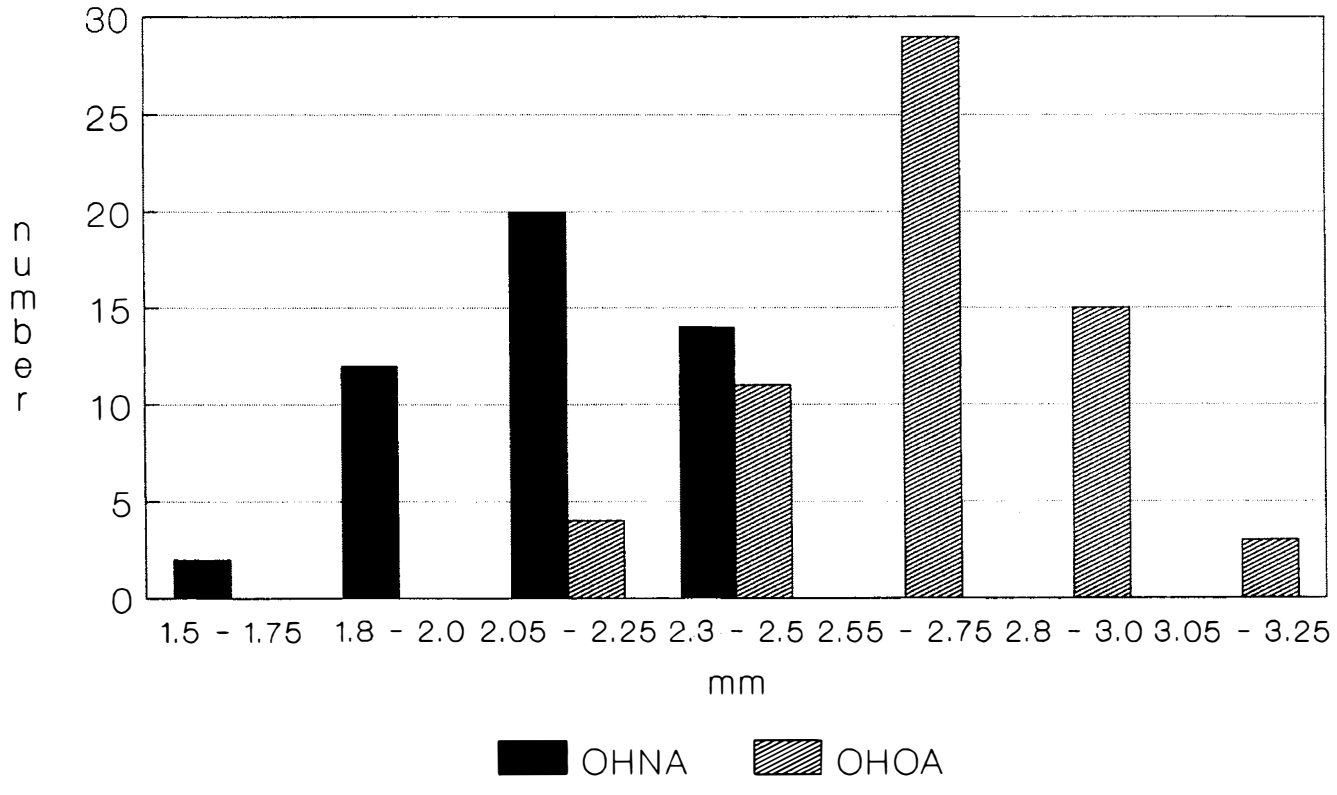


Fig. 1. *Anterior chamber depths of OHOA and OHNA eyes.*

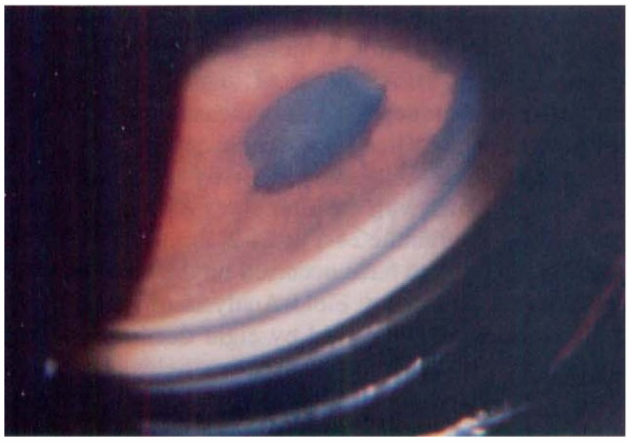


Fig. 2a. *Gonioscopic view of superior AC angle with Goldman goniolens showing how peripheral iris obscures view of angle structures.*

Fig. 2b. *Same view of same eye with indentation gonioscopy. Aqueous displaced from the central AC pushes peripheral iris posteriorly and shows extensive closure of AC angle by PAS.*

present (Figs 2, 3 and 4) and laser iridotomy (LI) was performed. Similarly eyes felt to be at very high risk of developing ACG because of occludable angles underwent LI (Fig 5). The presence of glaucoma was taken as a study end-point for that patient, and treatment manoeuvres, such as additional medical treatment or surgical treatment, were instituted as required clinically. In some patients topical treatment was discontinued if there was no evidence of progression to glaucoma, with careful follow-up of IOP.

RESULTS

Sixty one patients (122 eyes) were recruited into this study, but two patients are excluded from the analysis—one was excluded because of the development of the exfoliation syndrome, and one who completed only one year of follow-up. There were 25 patients in the OHNA group (12 with van Herick angle grade less than 2, and 13 grade 2), and 33 patients in the OH with open angles (OHOA) group—all with van Herick angle grade greater than 2. Table I shows the demographic details of the patients in both groups, with IOP, ACD and length of follow-up. The ACDs were significantly less in the OHNA group (Fig 1). The ACD measurement was not recorded in six eyes as the initial record was lost and the patient was either lost to follow-up, aphakic or deceased.

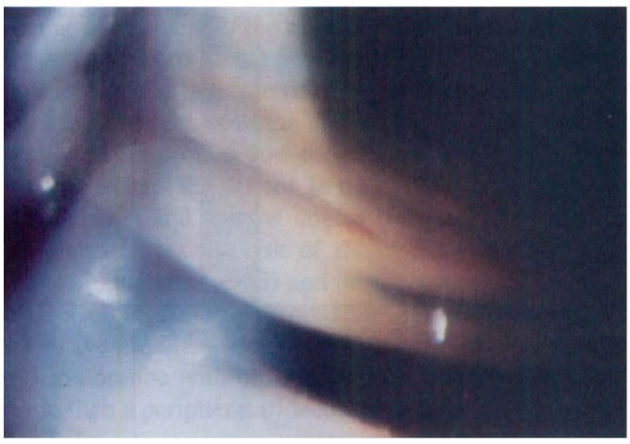


Fig. 3 *Indentation gonioscopy of the superior AC angle shows nasal angle is openable, but PAS have closed temporal half of angle.*

(A) Development of Angle-Closure Glaucoma

Of the 25 patients in the OHNA group, nine developed ACG over an average four year follow-up (Table II). One patient was diagnosed as having developed acute-on-chronic ACG with an IOP of 48 mmHg at five months into the study. This eye was a control eye and required peripheral iridectomy (PI) and later trabeculectomy for IOP control. Three patients developed symptoms of subacute ACG (SAACG) and underwent bilateral LI. Five patients developed creeping ACG shown by established bands of PAS of the superior angle of two or more clock hours, visible on indentation gonioscopy and persisting after LI. Four of these patients underwent bilateral LI, the other surgical iridectomy. The case of acute-on-chronic ACG occurred in a control eye. Where creeping ACG was predominantly uniocular, it occurred in the timolol treated eye (three of five patients). Two of the three cases of SAACG occurred while on timolol treatment, and in one case SAACG only developed when the timolol treatment

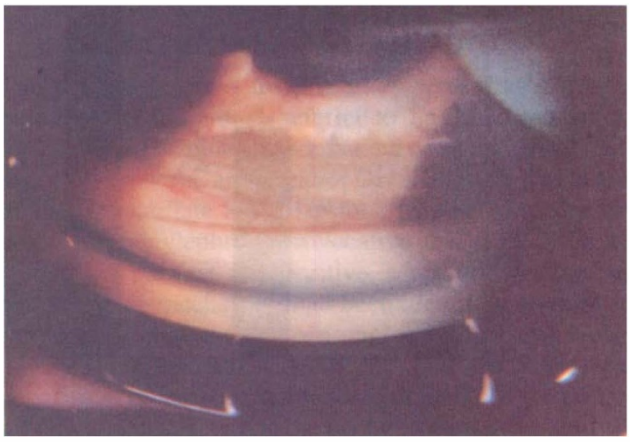


Fig. 4 *Indentation gonioscopy of superior AC angle reveals PAS of iris attaching to pigmented tabecular meshwork in temporal half of angle, while nasal half remains open. A blood vessel is seen in the recess of the open nasal portion of the superior angle.*

Fig. 5 *Indentation gonioscopy of superior AC angle reveals peripheral iris bridging angle recess to adhere to base of pigmented band of trabecular meshwork, with early synechiae formation.*

was stopped. The average ACD in the ACG patients was 2.0 mm (range 1.8–2.2 mm). In seven patients the AC angle was considered dangerously narrow and occludable, and in some of the eyes there appeared to be the first signs of the development of PAS. All these patients (average ACD 1.94 mm) underwent bilateral LI. Nine patients (average ACD 2.3 mm) showed no sign or symptom of the development of ACG and continued follow-up with narrow though open angles. In eight of these patients, both eyes remained healthy, but one after seven years of follow-up developed unilateral central retinal vein occlusion concurrently with a rise in IOP in both eyes. Timolol treatment therefore was not shown to be protective against the development of ACG.

In the OHOA group, no patient developed primary ACG (Table II). One patient developed acute ACG due to an enlarging lens in the left eye three months after cataract extraction with lens implantation in the right eye. The ACD in the eye that developed acute ACG was 2.3 mm one year prior to the attack, but fell to 1.8 mm at the time of the attack. This eye responded to extracapsular cataract extraction and lens implantation and recovered full vision. It remained hypertensive, requiring timolol for IOP control. In one patient in the OHOA group, there was significant ACD asymmetry (2.3 and 2.6 mm respectively), and the eye with the shallower ACD was judged to be at risk of ACG, and therefore underwent LI three years into the study. In all patients other than the above two, the difference between the ACDs in the two eyes was never greater than 0.1 mm.

Table I. Presenting features of patients in the OHNA and OHOA groups.

	OHNA	OHOA	
Sample size	25	34	
Sex ratio M:F	8:17	14:20	$p>0.1$[1]
Mean age in years	63	63	$p>0.1$[2]
Mean ACD (SD)[3]	2.1 (0.21)	2.63 (0.2)	$p<0.01$[2]
Mean IOP (SD)	25.3 (2.44)	24.93 (2.43)	$p>0.1$[2]
Follow-up in months	76	75.6	$p>0.1$[2]

[1] Chi-squared analysis.
[2] T test (unpaired).
[3] SD is standard deviation.

(B) Development of visual field loss and/or optic disc damage

In the OHNA group, two patients developed glaucomatous damage—the patient with acute-on-chronic ACG, and one other patient (who had undergone bilateral LI for occludable angles) in whom one eye developed visual field loss and optic disc damage characteristic of glaucoma (Table II).

In the OHOA group, four patients developed glaucomatous visual field loss on the Humphrey Field Analyser with corresponding development of optic disc damage (Table II). Five patients in whom the visual field analysis was not reliable developed clinically detectable glaucomatous optic disc damage, one of these eyes being the eye that had undergone LI for unilateral narrow angle. Three of these five patients underwent trabeculectomy. Two other patients developed retinal vein occlusions. Glaucomatous disc damage and/or demonstrable field loss in the OHOA group was seen in the timolol-treated eyes of four patients, and in the control eyes of six patients. In one patient both study and control eyes developed glaucoma.

(C) IOP control

The IOP levels at presentation were similar in both the NA and OA groups (Table I). Throughout the period of randomisation (maximum 66 months), there was no significant difference between the mean IOP levels of the two groups (excluding the acutely elevated IOP of the eye at its presentation with acute-on-chronic ACG) (Table III). There was a significant difference in mean IOP between the treated and the untreated eyes in the two groups up to 30 months into the study (Table IV), with the timolol treated eyes in both OA and NA groups showing an equal degree of lowering of IOP. In the NA group, by the end of the study period, five patients (20%) had become normotensive off medication—four patients following bilateral YAG LI for ACG, and one patient following LI for dangerously narrow angles. In the OA group, by the end of follow-up, four patients (11.7%) had become NT off all medication and remained healthy.

Table V shows the outcome, duration of random timolol therapy and timing of occurrence of glaucoma and/or follow-up in the two groups. Figure 6 summarises the outcome of the two groups.

Table II. Outcome of OHNA and OHOA patients.

	OHNA n = 25	OHOA n = 34	Difference between proportions
Angle-closure glaucoma	9 (36%)	0 (0%)	36% $p<0.05$ (95% CI 17.2% to 54.8%)
Glaucomatous field loss or disc change	2 (8%)	9 (26.5%)	18.5% $p<0.05$ (95% CI 0.2% to 36.7%)

Table III. Difference between mean IOPs of OHOA and OHNA eyes.

	Time into study (months)[1]					
	0	1	12	24	36	48
Treated eyes						
OHNA mean IOP	25.4	19.4	20.8	19.8	21.4	20.3
n	25	25	23	21	11	6
OHOA mean IOP	24.5	20.8	21.3	20.7	21.7	21.4
n	34	34	33	28	13	7
Difference between means	0.9	−1.4	−0.5	−0.9	−0.3	−1.1
p value [2]	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1
Non-treated eyes						
OHNA mean IOP	25.2	23.3	23.7	21.2	22.3	22.5
n	25	25	23	21	11	6
OHOA mean IOP	25.3	24	24.2	22.3	21.4	22.1
n	34	34	33	28	13	7
Difference between means	−0.1	−0.7	−0.5	−1.1	0.9	0.4
p value [2]	>0.1	>0.1	>0.1	>0.1	>0.1	>0.1

[1] No statistical significance between mean IOPs of OHNA and OHOA eyes up to 60 months follow-up, but small numbers beyond 48 months.
[2] t test of difference between unpaired means.

DISCUSSION

Recognition of a narrow anterior chamber angle during ocular examination is considered of great importance,[3,5] but determining which particular eye with a narrow angle is likely to develop angle closure is very difficult.[12] Recently, an increased lens thickness/ocular axial length of the eye has been shown to be a risk factor for the development of ACG.[13,14]

Relative pupil block, resulting from a restriction of aqueous flow from the posterior to the anterior chamber, causes forward bowing of the peripheral iris and consequent narrowing of the peripheral anterior chamber.[15] Iridotomy relieves the pressure differential between the two chambers and results in flattening of the iris contour[16] and deepening of the peripheral anterior chamber angle.[17] Because of these benefits, laser iridotomy has been advocated as a primary procedure in the treatment of eyes with chronic ACG[18,19] and in eyes with OH and narrow angles.[6] However, LI is not without complications and other studies have shown limited benefit of this treatment[20,21] especially with regard to IOP control.[6]

Perkins and Phelps[22] have shown that OH eyes with a hypermetropic refraction have a very low incidence of progression to visual field loss as compared to emmetropic or myopic eyes, although their study showed hypermetropia to be a risk factor for the development of ACG with 4.3% of 92 hypermetropic patients developing closed angle glaucoma.[22]

The present study therefore examined the relative risk of the development of angle-closure glaucoma and visual field loss in two groups of patients with OH (one with narrow angles and one with open angles) and whether treatment with topical timolol protected against the development of glaucoma. The pattern of IOP control achieved by timolol was studied in the two groups as a difference might indicate an additional benefit of timolol, an inhibitor of aqueous secretion,[23] in eyes with OHNA, by reducing pupil block force.

In the re-analysis period, two different methods of examination were employed that were not performed at the patients' entry to the study. Firstly, initial examination of the angle to determine absence of PAS was by Goldmann gonioscopy. With this method, early PAS may be hidden from view by a very convex iris, and therefore it is possible that when Zeiss indentation gonioscopy revealed PAS at a later stage, these might have been present from the outset. Secondly, as Humphrey Visual Field Analysis was used in the follow-up period after 1987 to detect glaucomatous visual field loss, and Friedmann Field Analysis was used prior to this, it is possible that some early field loss might not have been detected with the Friedmann that was later detected with the more sensitive Humphrey. However, as the Friedmann has been shown to be a sensitive instrument for the detection of early glaucomatous visual field defects,[24] it is likely that the majority of eyes judged to be ocular hypertensive on the basis of a normal optic disc appearance and a normal Friedmann visual field were indeed suffering from ocular hypertension and were not glaucomatous.

(1) Angle-closure glaucoma

Follow-up of 25 patients (50 eyes) with ocular hypertension and narrow angles over an average six years showed that nine patients developed ACG, five of these cases developing creeping ACG. In addition a further seven patients underwent Nd:YAG LI as they were judged to have occludable angles or were developing very early PAS of the superior angle. The incidence of ACG may therefore have been higher if iridotomies had not been performed on these eyes. All the patients in this study had negative PPPTs on entry to the study. The high incidence of the later development of ACG in these patients confirms the findings of a recent report showing that a negative PPPT should not be relied on for assessment of the risk of ACG.[25] A narrow angle determined by van Herick's method was closely correlated with a risk of ACG.

In patients judged to have angle width greater than two there was no case of primary ACG. Only one eye of the 34 patients in the OA group developed a gonioscopically narrow angle that was considered occludable and underwent

Table IV. Difference between mean IOPs of timolol-treated (TT) and non-timolol-treated (NT) eyes.

	Time into study (months)								
	0	1	6	12	18	24	30	36	42
TT mean IOP	24.9	20.2	20.5	21.1	20.3	20.3	20.6	21.5	20.7
n	59	59	55	56	54	49	36	24	21
NT mean IOP	25.3	25.3	22.9	24	22.7	21.8	22.8	21.8	21.1
n	59	59	55	56	54	49	36	24	21
Difference between means	−0.4	−5.1	−2.4	−2.9	−2.4	−1.5	−2.2	−0.3	−0.4
p value[1]	>.1	<.05	<.05	<.05	<.05	<.05	<.05	>.1	>.1

[1] t test of difference between paired means.

YAG LI. The patient who developed acute glaucoma secondary to lens enlargement already had a cataract removed from the other eye, demonstrating the importance of an increased lens size in the aetiology of ACG.[13,14] Similarly, ACD estimation was equally effective in identifying the eyes at risk of ACG, confirming Alsbirk's findings[26] that the risk of ACG increases as ACD decreases. Alsbirk showed that with an AC depth less than 2 mm, the prevalence of ACG is 18% for men and 24% for women. A recent review of patients attending the Glaucoma Unit at St Paul's who had suffered acute or subacute ACG found the mean AC depths to be 1.7 and 1.9 mm respectively.[25] Figure 1 shows that one third of the eyes in the OHNA group had ACDs in this region and were therefore at risk of ACG.

Indentation gonioscopy is essential in the management of patients with NA and raised IOP as it will reveal whether or not PAS are closing off the anterior chamber angle (Illustration). McGalliard and Wishart in their study of patients with narrow angle glaucoma showed LI was most effective in patients with a history of subacute ACG, and reduced the IOP in 69% of eyes with well-established PAS, whereas eyes without obvious PAS did not benefit from LI.[20] In the present study, 55.5% of eyes that developed ACG became normotensive after LI, while only 14% of OH eyes that were thought to be at risk of ACG and underwent LI became normotensive.

This study has shown that OH eyes with NAs had a high incidence of development of angle-closure glaucoma—36%. This is higher than the prevalence of ACG in the general population described by Alsbirk,[26] and it may indicate that some of the OHNA eyes were already suffering from ACG at entry to the study. Such patients warrant close follow-up and indentation gonioscopy to detect PAS of the angle. In view of the effectiveness and safety of YAG LI, patients with OH and NA should be considered for YAG LI especially if ACD is less than 2.2 mm. Unless there are obvious symptoms or signs of ACG, however, LI is unlikely to result in lowering of the IOP.

Topical timolol administration was not shown to prevent the development of ACG, as an equal number of study and control eyes developed this complication.

Table V. The number of patients, the average duration of random timolol treatment and the average years of follow-up/average years at which status changed from ocular hypertension in OHNA and OHOA Groups.

Outcome	n	Duration of random timolol (years)	Mean follow-up (years)[1]
OHNA group			
OHNA	9	3.5	6
ACG	9	2.6	2.6
Occludable	7	4.6	4.6
OHOA group			
OHOA	21	3.4	6.4
OA glaucoma	9	2.3	4
Normotensive	4	2.7	6.5

[1] Follow-up in years, or follow-up until change of status.

(2) Control of IOP

The mean levels of IOP in the OHNA and OHOA groups were similar up to 66 months into the study (Table III), although sample size is small beyond 30 months, and both the OHNA and OHOA groups showed a similar drop of IOP in the eyes receiving timolol as compared to the non-timolol-treated eyes. This finding would seem to indicate that the aetiology of the raised pressure in the two groups was similar in that reduction of aqueous secretion by timolol produced a similar effect.

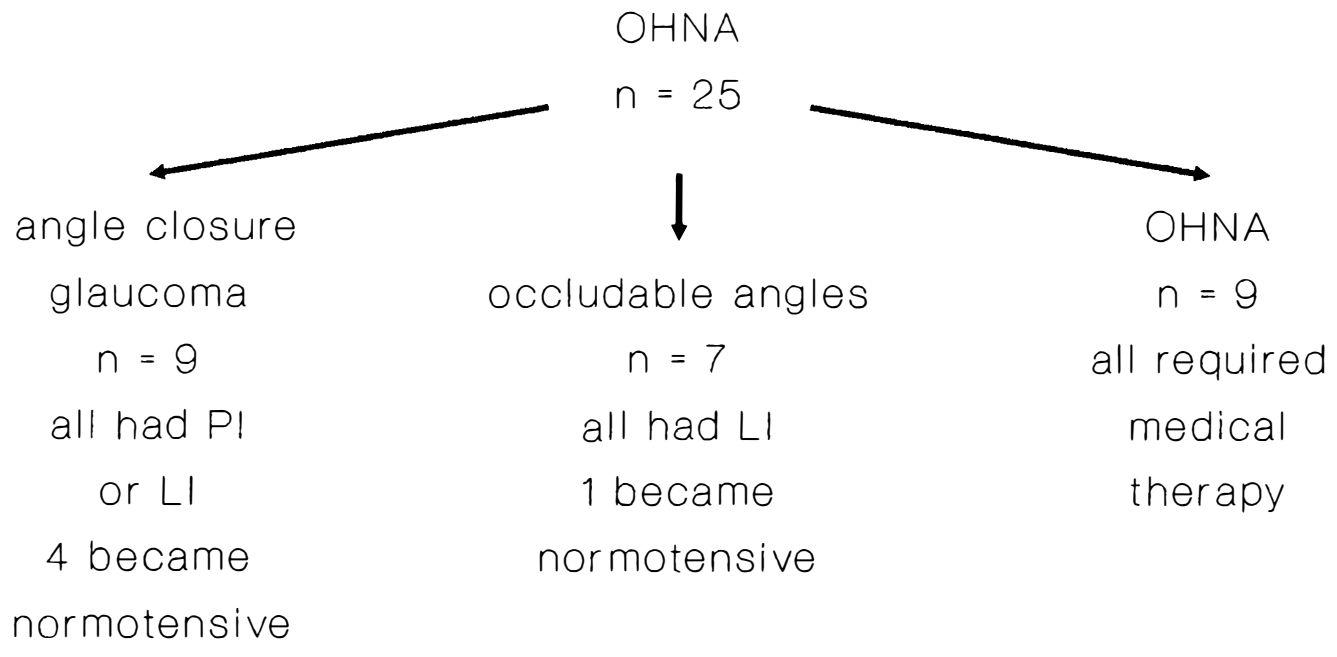
Twelve per cent of patients in the OHOA group, after a one month wash-out period, remained normotensive after discontinuation of timolol therapy. Other long term follow-up studies have shown that raised IOP may spontaneously revert to normal.[27,28,29] Although five of the 25 NA patients became normotensive by the end of the study, four of these patients had undergone LI as they had developed ACG.

(3) Visual field loss

The link between raised IOP in ocular hypertension and the development of glaucomatous optic disc damage and visual field loss has been the subject of intensive study. Studies of patients randomly assigned to treatment with topical medication or no treatment have led to conflicting conclusions. Some studies have shown that lowering IOP decreases the chance of conversion to glaucoma,[30,31] but others have shown no benefit.[2,32,33,34]

In the present study, no definite protective effect of timolol was demonstrated against the development of visual field loss. Open angle glaucoma occurred in four timolol treated eyes, and in six control eyes in the OHOA group.

Mapstone described a covert angle-closing mechanism (partial angle closure) as being one possible cause of elevated IOP.[8,9,10] However, he used the PPPT to diagnose this condition and long term follow-up studies have shown



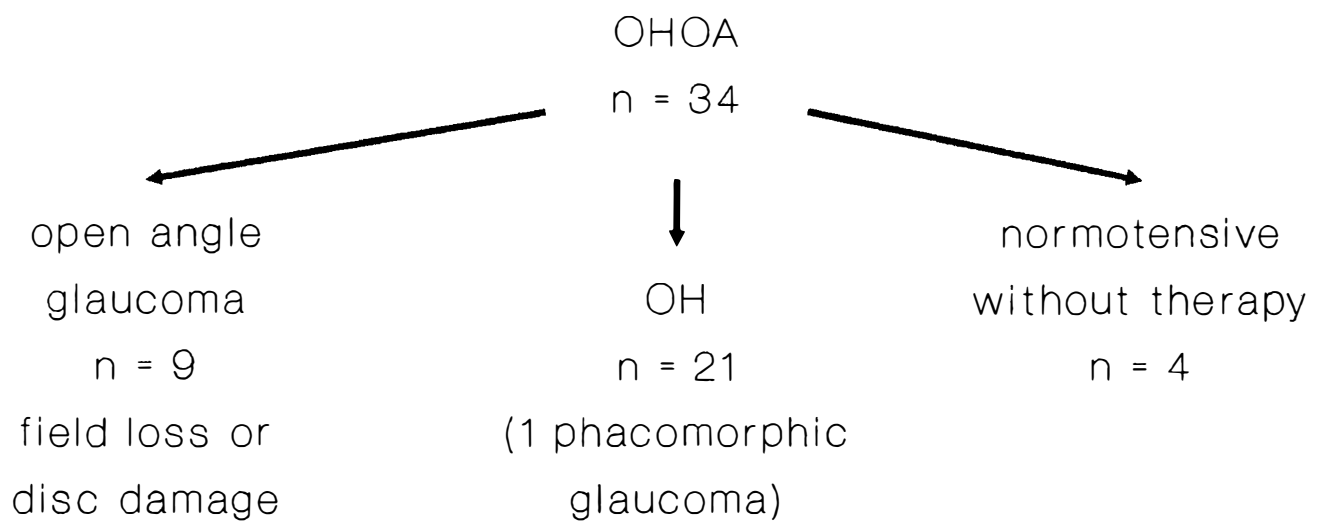


Fig. 6 *Outcome of open angle and narrow angle ocular hypertensives.*

that the PPPT is a test with poor specificity and sensitivity.[25,35] In the present study all eyes had a negative PPPT, yet there was a high incidence of the development of ACG. Appositional closure of the angle by the iris may have been responsible for the raised IOP in some of the OHNA patients, but where ACG developed, it was identifiable as such and there was a low incidence of visual field loss in this group. Perkins and Phelps[22] showed that patients with a predominantly hypermetropic refraction are at low risk for the development of visual field loss. Our results support this finding, in that only two patients in our OHNA group (8%) developed optic disc damage and visual field loss, one of these patients being the acute-on-chronic ACG patient. In the OA group with deeper ACs, nine patients (26.5%) developed glaucomatous damage, a significantly greater proportion than in the NA group (Table II). In the narrow angle group two (10%) of the 25 patients developed glaucomatous disc damage, but none of the nine patients who did not require LI developed glaucomatous field loss.

CONCLUSIONS

A narrow anterior chamber angle is a specific risk factor for the development of ACG in eyes with OH, but not a

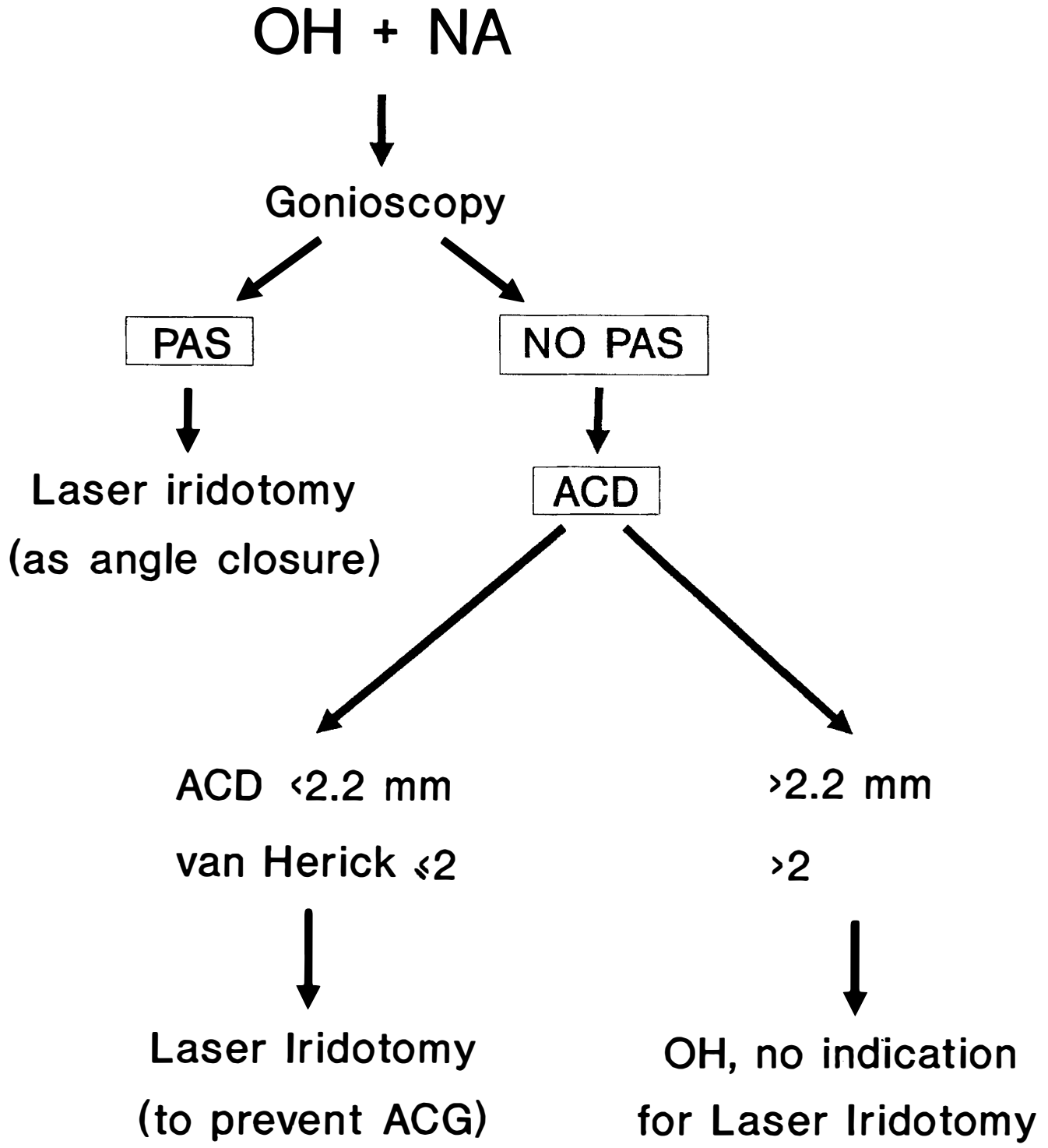


Fig. 7 *Suggested management scheme for eyes with ocular hypertension (OH) and narrow angles (NA) to identify angle closure glaucoma (ACG) and evaluate its risk of occurrence.*

risk factor for the development of glaucomatous visual field loss.

Measurement of the ACD and grading of the angle width with van Herick's method will help identify eyes at risk of ACG. OH eyes with ACD less than 2.2 and van Herick Grade 2 or less should be considered for Nd:YAG LI, as shown in the accompanying flow diagram (Fig 7).

Treatment with topical timolol was not shown to have a protective effect against the development of ACG or visual field loss in eyes with OH.

We wish to thank Mrs. C. Owen for her help with technical and administrative tasks.

Key words: angle-closure glaucoma, anterior chamber angle width, ocular hypertension.

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