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COMMUNICATIONS

ANTERIOR CHAMBER DEPTH IN MEDICALLY-TREATED OPEN-ANGLE GLAUCOMA†*

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IN a previous communication (Romano and Jackson, 1964), the observation was reported that phakic cases of primary open-angle glaucoma undergoing treatment with strong miotics, mostly phospholine iodide, presented changes in the configuration of the anterior chamber after a variable period of therapy. These consisted of increasing bowing-forwards of the iris (physiological iris bombé) and a clinical impression of actual diminution of the depth of the anterior chamber. No accurate measurements of depth were available at the time to support this impression. It had further been observed that a correlation appeared to exist between the occurrence of these changes, particularly if they were of a severe degree, and failure of control or unsatisfactory control of the glaucoma, in the absence of peripheral anterior synechiae or of definite angle closure by contact.

The results of measurements carried out in a comparable group of patients are presented in an effort to ascertain whether this correlation can be confirmed.

Material and Methods

The study covers 33 eyes in an unselected group of twenty patients (Table I, overleaf) attending the Glaucoma Clinic, Moorfields Eye Hospital, City Road. Once included in the series, measurements of individual cases were repeated in some instances, usually to study the effect of a change of therapy or the passage of time.

The majority were phakic unoperated cases of chronic simple glaucoma on phospholine iodide therapy (26 eyes) or pilocarpine (9 eyes). One patient (2 eyes) was on pilocarpine, then on phospholine iodide.

The method employed for the measurement of the anterior chamber depth was the photographic technique of Dr. D. Maurice, Institute of Ophthalmology, London (Jones and Maurice, 1963). A photograph of the profile of the anterior chamber is taken at an angle of incidence of 45° through a camera fitted to the "900" Haag-Streit slit lamp. Illumination is by a narrow slit beam, lengthened by the interposition of a convex cylinder in the pathway of light. The negative is projected on to a special grid to correct the outline of the anterior chamber from the distortions produced by the curvature of the cornea. The true outline is plotted on squared paper. The depth can be directly read off this outline. (The volume of the anterior chamber can also be calculated from this outline and this has been done in a separate study.) In all cases the patient's age and state of refraction were noted, as these factors influence anterior chamber depth.

Arbitrary criteria were defined for "control" of the glaucoma, for the purpose of attempting a correlation between anterior chamber depth and "control". Cases were considered "uncontrolled" if the tensions remained at or above 24–25 mm. Hg (applanation), or if a carbonic anhydrase inhibitor were necessary to control the tension, or if field loss occurred or was progressive.

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TABLE I

PARTICULARS OF TWENTY PATIENTS ALL WITH CHRONIC SIMPLE GLAUCOMA EXCEPT AS STATED IN FOOTNOTES

Case No.	Age (yrs)	Refraction	Date	Eye	Treatment	Anterior Chamber Depth (mm.)	Applanation (mm. Hg)	Remarks
1	71	Low hypermetrope	Apr. 65	R	Gutt. 3 per cent. Pilocarpine three times a day	3·0	18–22	
				L	Gutt. phospholine iodide (P.I.) 0·25 per cent. twice daily + Diamox tabs	2·5	20–22	
			Aug. 65	R	P.I. 0·25 per cent. twice daily + Diamox tabs + 1 per cent. neutral adrenaline (NA) both eyes	3·0	21	Controlled without Diamox
				L		2·4	23–24	Uncontrolled, needs Diamox Field loss
2	67	+0·75 D sph.	June 64	R	P.I. 0·125 per cent. twice daily	2·7	23	
			June 65	R	,, ,, ,,	2·1	24–25	
			Aug. 65	R	,, ,, ,, + Diamox tabs	2·3	22	Uncontrolled, needs Diamox
3	69	+2 D sph.	Mar. 65	L	P.I. 0·125 per cent. twice daily + Diamox tabs	1·5	30	
			July 65	L	+ 1 per cent. NA	1·7	13	
			Sept. 65	L	,, ,, ,,	2·0	19	Uncontrolled
4	77	−3 D sph.	May 65	R	P.I. 0·125 per cent. (May, 1964 0·06 per cent.) + NA + Diamox	2·2	25–32	
			Aug. 65	R	,, ,, ,,	2·0	21	
			Oct. 65	R	,, ,, ,,	1·7	22–24	Uncontrolled Field loss
5	62	+1 to +2 D sph. both eyes	Oct. 65	R	P.I. 0·06 per cent. once a day both eyes (from July, 1964)	1·7	20	
				L		2–2·1	20	
			Sept. 65	R	,, ,, ,,	2·2–2·3	21	
				L		2·3–2·4	19	
			Jan. 66	R	,, ,, ,,		30	Uncontrolled
				L			26	
6	73	+0·5 to +1 D sph. both eyes	July 65	R	3 per cent. Pilocarpine three times daily + NA from Sept. 65 both eyes	2·4–2·5	22	
				L		2·4–2·5	20	Controlled
7	65	+1 D sph.	July 65	L	P.I. 0·06 per cent. (from Nov. 63) P.I. 0·125 per cent. + NA + Diamox (from June 64)	2·3	17–22	
			Oct. 65	L		2·2–2·3	22–23	Uncontrolled
8	78	+2·5 to +3 D sph. both eyes	Oct. 65	R	2 per cent. Pilocarpine +0·25 per cent. Eserine three times daily both eyes	2·0	21–22	Field loss, both eyes
				L		1·75	22–23	Uncontrolled

TABLE I (*continued*)

Case No.	Age (yrs)	Refraction	Date	Eye	Treatment	A.C. Depth (mm.)	Applanation (mm. Hg)	Remarks
9	75	+2·5 to +4 D sph. both eyes	Oct. 65	R	P.I. 0·06 per cent. (from Sept. 65) + NA + Daranide both eyes	2·1	20	
				L		2·2	16	Uncontrolled
10	—	Emmetrope	June 65	R	P.I. 0·25 per cent. (from Oct. 64)	2·0	18	Field loss Uncontrolled
11	77	Emmetrope	Nov. 65	R	P.I. 0·125 per cent. once a day	2·5	21	Field loss Uncontrolled
				L	P.I. 0·125 per cent. twice daily + NA	2·4–2·5	25	
12	66	+4 D sph. both eyes	Nov. 65	R	P.I. 0·125 per cent. once a day both eyes	2·2	17–22	Controlled
				L		2·3	20–22	
13	73	−3 D sph., −4 D cyl.	Oct. 65	L	P.I. 0·125 per cent. once a day (from Nov. 64) twice daily from Jan. 65	3·0	20–22	Controlled
14 *	70	−3 D sph., −2 D cyl.	Oct. 65	R	P.I. 0·125 per cent. once a day (from Nov. 64) +NA from Jan. 65	1·7	18–23	Uncontrolled
				L	P.I. 0·06 per cent. once a day + NA (from July 65) and Diamox later		20	
15 †	67	Emmetrope	Oct. 65	R	P.I. 0·125 per cent. twice daily + NA + Diamox both eyes	2·4–2·5	28	(Operated later) Uncontrolled
				L		2·5	28	
16	75	Low hypermetrope	Oct. 65	R	P.I. 0·025 per cent. twice daily (from Aug. 65) + NA both eyes	3·0	11–16	Controlled
				L		3·1	14–18	
17	57	−6 to −9 D sph. both eyes	Oct. 65	R	2 per cent. Pilocarpine three times a day both eyes	2·5	21	Retinal detachment
				L		2·5	20	Early field loss Uncontrolled
18 ‡	59	Emmetrope	June 64	R	P.I. 0·125 per cent. twice daily 0·25 per cent. twice daily from Aug. 64	2·5	20	Controlled
19	54	Emmetrope	Dec. 65	R	2 per cent. Pilocarpine four times a day + Diamox both eyes	2·8–2·9	above 40	
				L				
			Dec. 65	R	P.I. 0·125 per cent. + NA + Diamox both eyes	2·4–2·6	30	Uncontrolled Right trephine Feb. 1966
				L		2·3–2·5	32	Uncontrolled
20	77	+0·5 D sph.	Dec. 65	R	P.I. 0·125 per cent. twice daily both eyes	2·2	20	Controlled
		+1 D sph., +2 D cyl.		L		2·2–2·3	18	

* Left eye absolute glaucoma † Right eye absolute glaucoma ‡ Capsulo-cuticular glaucoma

Results

(1) The average anterior chamber depth (using the average of individual readings for each eye) for the phakic unoperated cases of chronic simple glaucoma treated with phospholine iodide (26 eyes) was 2·32 mm. (S.D. 0·36 mm.).

(2) The average depth in the small series (six eyes) treated with pilocarpine was 2·68 mm. (S.D. 0·23 mm.).

(3) No correlation was found to exist between individual measurements of anterior chamber depth and corresponding applanation estimation of the intra-ocular pressure.

(4) Considering the cases of chronic simple glaucoma treated with phospholine iodide and using the arbitrary criteria mentioned above to define "control" of the disease, the following observations were made (Table II):

(*a*) No eye with an anterior chamber depth of less than 2·2 mm. was controlled.
(*b*) All the eyes with an anterior chamber depth greater than 2·5 mm. were controlled.
(*c*) The mean depth of the controlled eyes was 2·6 mm. (S.D. 0·32 mm.), and that of the uncontrolled eyes was 2·2 mm. (S.D. 0·29 mm.).

TABLE II
OBSERVATIONS ON CASES OF CHRONIC SIMPLE GLAUCOMA TREATED WITH PHOSPHOLINE IODIDE

Controlled on Phospholine Iodide	Depth of Anterior Chamber (mm.)															
	1·6	1·7	1·8	1·9	2·0	2·1	2·2	2·3	2·4	2·5	2·6	2·7	2·8	2·9	3·0	3·1
Yes							3	1		1	1		1		2	1
No	1	2			3	1	2	1	5	3						

Discussion

Törnquist and Brodén (1958) stated that the depth of the anterior chamber in (untreated) chronic simple glaucoma was slightly less than the average for the general population. However, most other authors think that it is no different, and that the relative proportions of wide, intermediate, and narrow angles are the same as in non-glaucomatous eyes.

It is now recognized that in eyes treated with miotics, particularly the "irreversible" cholinesterase inhibitors, there is a tendency for physiological pupillary block to occur, leading to some degree of bowing-forwards of the iris (iris bombé) (Barkan, 1954; Sugar, 1957; Swan, 1959; Grant, 1962).

Little reference could be found in the literature, however, to the actual depth of the anterior chamber (from corneal endothelium to lens capsule) in such cases. Rosengren (1950), working on young subjects (17 to 32 years of age), found a diminution of the anterior chamber depth of 0·16 mm. ($\pm$ 0·033 mm.) after one instillation of 2 per cent. pilocarpine. Heim (1941) described three cases in which the instillation of pilocarpine produced a decrease in anterior chamber depth of 0·31, 0·15, and 0·34 mm. respectively.

In such assessments, it must not be forgotten that glaucoma simplex is a disease of middle and old age. With age, the anterior chamber depth tends to decrease under physiological conditions, due to the increasing bulk of the crystalline lens as well as that of the ciliary

body (after age 35 this occurs principally by the laying down of connective tissue, replacing muscle tissue, and after age 50 by decomposition of amorphous hyaline substance—Kronfeld, 1962).

Rosengren (1950) indicated the average measurements in normal eyes as follows:

Age (yrs)	*Depth* (mm.)
20–29	3·63
30–39	3·46
40–49	3·34
50–59	3·24
60–69	3·16
70–79	3·10

Other estimations of average anterior chamber depth are shown in Table III.

TABLE III
ESTIMATIONS OF AVERAGE ANTERIOR CHAMBER DEPTH

Author	Date	Range of Average Anterior Chamber Depth			
		Age (yrs)	Depth (mm.)	Age (yrs)	Depth (mm.)
Törnquist	1953 (males)	19–21	3·18	64–66	2·69
Calmettes and others	1958	12–15	3·65	50–80	3·23
Rosengren	1930–31	25–30	3·57	60–65	3·12
Raeder	1922	20–40	3·46	60–80	2·90
Lindstedt	1916	15–30	3·68	31–80	3·34

Törnquist (1953) (males) from 3·18 mm. in the 19 to 21-year age group down to 2·69 mm. in the 64 to 66-year age group;

Calmettes, Déodati, Huron, and Béchac (1958) from 3·65 mm. (12–15 years) to 3·23 mm. (50–80 years);

Rosengren (1930–31) from 3·57 mm. (25–30 years) to 3·12 mm. (60–65 years);

Raeder (1922) from 3·46 mm. (20–40 years) to 2·90 mm. (60–80 years);

Lindstedt (1916) from 3·68 mm. (15–30 years) to 3·34 mm. (31–80 years);

Stenström (1946) 3·70 mm. (males) and 3·65 mm. (females).

It is also well known that hypermetropic eyes have a shallower anterior chamber and myopic eyes a deeper one than emmetropic eyes in the same age groups.

Fewer figures are available for estimates of anterior chamber depth correlated to the state of refraction of the eye, but Raeder (1922) gave the following average figures for 40-year-old subjects: hypermetropic 2·99 mm., emmetropic 3·14 mm., myopic 3·63 mm. The highest figure noted in the literature (3·97 mm.) is, naturally enough, in a myope (Zeeman, 1911), but the age of the subject was not mentioned.

Mechanisms of Decrease in Depth of the Anterior Chamber with Miotics

In the context of the present study, a decrease in the depth of the anterior chamber may be produced by one or more of the following factors:

(*i*) *Oedema or Congestion of the Ciliary Body or Iris.*—It is now well established that strong miotics and more particularly, perhaps, the cholinesterase inhibitors—eserine, di-isopropylfluorophosphate (DFP), phospholine iodide—can produce vascular congestion of the anterior segment of

the eye. Thickening of the iris would reduce the distance between its anterior surface and the endothelium of the cornea; increase in volume of the ciliary body could reduce the distance between the lens capsule and the corneal endothelium.

(*iia*) *Ciliary Muscle Contraction.*—A spasm of the circular fibres would produce a more globular lens if this structure retained enough of its plasticity—elasticity to assume an "accommodated" shape. This would reduce the distance between the endothelium and anterior lens capsule. However, all except two of the present cases were older than 60 years (and sixteen were over 65 years). Rosengren (1950) found the effect of gutt. pilocarpine 2 per cent. in the 45 to 71-year age group to be "negligible" (a decrease in depth of 0·01 ($\pm$ 0·011 mm.) after 90 minutes). This factor is not therefore likely to be of importance in the present study.

(*iib*) *Contraction of the Meridional Fibres with Possible Drawing Forwards of the Lens.*—This mechanism, as will be seen below, is considered to be of possible relevance.

(*iii*) *Pathological Increase in Size of the Lens.*—Cataract may co-exist with glaucoma which may be a causal or an aggravating factor in its evolution. It has also recently been suggested that cataract follows the use of phospholine iodide in as many as 50 per cent. of treated cases (de Roetth, 1966). In either case, intumescence may occur with decrease in anterior chamber depth.

Discussion of the Findings in this Series

(1) Testing the mean anterior chamber depth of the cases treated on phospholine iodide (2·32 mm.) against Rosengren's lowest figure of 3·1 mm. by means of the statistical "t" test shows the difference to be significant to the point of being almost conclusive:

$$\Pr\{|t_{26}| > |11{\cdot}1\} < 0{\cdot}0001$$

(2) Testing this sample against Törnquist's lowest figure of 2·69 mm.

$$\Pr\{|t_{26}| > |5{\cdot}15\} < 0{\cdot}01$$ which is significant.

(3) Submitting the comparison of the control of chronic simple glaucoma on phospholine iodide with the anterior chamber depth to statistical analysis (including the "t" test) revealed that the difference in depth between the controlled and uncontrolled cases (as defined earlier) was highly significant.

(4) The difference in anterior chamber depth between the cases treated with phospholine iodide (average 2·32 mm.) and those treated with pilocarpine (average 2·68 mm.), although appreciable, was significant only at the 10 per cent. level ($p < 0{\cdot}1$) because of the small number of cases on pilocarpine.

Remarks

The volume of the anterior chamber was found by this same method to be reduced in these cases below what is considered to be an average value, in some cases below 100 or even 60 microlitres. It is hoped to present the results of volume measurements in a future study.

Possible Significance of Anterior Chamber Depth in Treated Open-angle Glaucoma

It is believed that one of the mechanisms whereby miotics are active in reducing the intra-ocular pressure in glaucoma is a facilitation of outflow of aqueous through the trabecular meshwork, produced by a process of traction on the scleral spur by the longitudinal fibres of the ciliary muscle. This is presumed to cause the leaves of the trabeculae, attached to the spur, to be spread apart, increasing the effective "pore size" of the meshwork.

To do this, the ciliary body must presumably be firmly anchored to the sclera. If, after a period of miotic therapy, the axial depth of the anterior chamber decreases, it might

indicate a weakening of the attachment to the sclera; if this happened, the meridional fibres would no longer be able to produce so effective a pull on the scleral spur, but would instead tend to draw the whole ciliary body—and the lens—forwards. Thus control of the outflow might be made less satisfactory. Furthermore, the shallowness of the angle thus produced might lead to sub-acute angle-closure with secondary trabecular damage (mixed glaucoma).

The absence of correlation in this series between individual tension readings and depth measurements does not, however, lend support to this view, and the reason for the poorer control on phospholine iodide in the cases with shallow anterior chambers is not clear.

Summary

The results are given of measuring the anterior chamber depth in twenty cases (33 eyes) of open-angle glaucoma receiving medical treatment, using the photographic projection method of Dr. D. Maurice. The average depth for eyes on phospholine iodide (26 eyes) was 2·32 mm. (S.D. 0·36 mm.). Compared with the lowest estimates found in the literature for a similar age group, these values are significantly lower. The average depth of eyes treated with pilocarpine (6 eyes) was 2·68 mm. (S.D. 0·23 mm.); the difference from that of eyes treated with phospholine iodide, though appreciable (0·36 mm.), was significant only at the 10 per cent. level because of the small number of eyes involved.

No correlation was found between individual applanation tension readings and corresponding anterior chamber depth measurements. However, applying stated arbitrary criteria for control of glaucoma to the patients receiving phospholine iodide revealed that no eye with an anterior chamber depth less than 2·2 mm. was controlled, and that all the eyes with a depth greater than 2·5 mm. were controlled. The mean depth of the controlled eyes was 2·6 mm. (S.D. 3·2 mm.) and that of the uncontrolled eyes 2·2 mm. (S.D. 0·29 mm.). The implications of anterior chamber depth in miotic-treated open-angle glaucoma are considered, and a possible connexion with the effectiveness of this control is discussed.

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