POSTURAL BEHAVIOUR OF INTRAOCULAR PRESSURE

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SUMMARY

Raised intraocular pressure (IOP) is generally held responsible for causing visual loss in chronic simple glaucoma. It is therefore desirable that a safe level of IOP be maintained all the time. Elevation of IOP with change of body position has been suggested as one of the factors which result in tissue damage in low tension as well as in primary wide open angle glaucoma. Postural behaviour of IOP was therefore studied in 58 normal and 30 glaucomatous Malaysian eyes. Clinical significance and possible pathogenesis of abnormal postural response of IOP has been discussed. More application of this simple procedure is advocated.

INTRODUCTION

Knowledge of postural response of intraocular pressure (IOP) is very important for the proper management of glaucoma. Many workers have investigated the influence of body position on IOP by using different types of tonometers.[1-6] In normal individuals, an increase of 1-2mm Hg in IOP on lying down has been documented.[6-9] Most of these earlier studies have employed Schiotz indentation tonometery for the supine, and Goldman applanation tonometery for the sitting positions. The Schiotz tonometery is nevertheless difficult to correlate accurately with applanation tonometer and thus the actual measurements, by using this technique, have remained far from satisfactory. Newly developed electronic tonometers have been used in recent studies to collect reliable and accurate data.[10,11] These devices however are very expensive and are available only at large university centres.

We have measured the IOP response to postural change by using a simple, portable and easily available Perkin's applanation tonometer. This paper reports our findings in the normal and glaucomatous Malaysian eyes.

MATERIALS AND METHODS

30 normal individuals above the age of 40 years were carefully selected from the University's Ophthalmic Department. 15 patients of primary wide open angle glaucoma (WOAG) attending the Glaucoma Clinic of the University's Ophthalmology Department were also included in this study. None of the normal subjects were using any

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systemic or local eye medication. There was no evidence of any significant cardiovascular, respiratory or inflammatory eye disease in our cases. There was no positive family history of glaucoma in the control population. Subjects with high myopia and diabetes mellitus were specifically excluded. All glaucoma medication was stopped for one week before recording the intraocular pressures.

Perkin's hand-held applanation tonometer was used to measure the IOP throughout this study. The subject was asked to sit in the chair calmly. Both eyes were anaesthetised with 0.4% Oxybuprocain (novesine) eye drops and were lightly stained with flourescein dye. The IOP was recorded first in the sitting position in each eye separately. The subject was then instructed to lie down gently on the examination table. No pillows were used to support the head. After waiting for about five minutes the IOP was measured in the supine position in each eye. All the measurements were done by the same observer.

RESULTS

A total of 30 glaucomatous eyes and 58 normal control eyes were examined. The results are depicted in Tables I, II and Figures 1, 2.

Both groups were closely matched for age and sex (Table I). The majority of the cases were within the age range of 51—70 years.

The mean IOP readings in each group are shown in Table II. In normal healthy individuals, an average of 1.82 (± 0.50) mm Hg rise in IOP on assuming the supine position was observed. The comparable figure of 3.66 (± 2.8) mm Hg was found in glaucomatous patients. A difference of 1.84 mm Hg between the mean postural change of IOP in the two groups was noted.

A change in IOP of 5mm Hg or more was used to segregate a sample of 7 (23%) glaucomatous eyes which registered a rise of 5—12mm Hg in IOP on assuming the supine posture (average 8.28mm Hg). The remaining 23 (77%) glaucomatous eyes showed a rise of IOP between 0—4mm Hg with an average of 2.26mm Hg. The IOP rise in the 56 (96.6%) control eyes also varied between 0—4mm Hg with an average of 1.82mm Hg. Only two normal eyes (3.4%) showed a rise of 6mm Hg (Fig. 1). However there was a significant difference in the mean IOP rise of glaucomatous and control eyes, which registered a postural change between 0—4mm Hg. Three glaucomatous eyes in this study showed a postural change of IOP only in one eye.

From Fig. 1 it can be seen that 82.4% (48 eyes) of the normal control eyes showed either no rise at

TABLE I

AGE AND SEX DISTRIBUTION

Age	Normal		Open Angle Glaucoma		Total	
	M	F	M	F	M	F
41 – 50	1	2	0	2	1	4
51 – 60	4	7	4	1	8	8
61 – 70	6	4	4	2	10	6
71 – 80	4	2	0	2	4	4
Total	15	15	8	7	23	22

Note: M – male; F – female.

TABLE II

MEAN INTRAOCULAR PRESSURE MEASUREMENT (MM HG)

		Eyes	Sitting IOP	Lying IOP	Rise in IOP
Normals	Males	29	14.68 (SD ± 3.32)	16.48 (SD ± 3.69)	1.79 (SD ± 0.54)
	Females	29	13.65 (SD ± 3.8)	15.45 (SD ± 4.37)	1.86 (SD ± 0.46)
Total		58	14.14 (SD ± 3.56)	15.96 (SD ± 4.00)	1.82 (SD ± 0.50)
Glaucomatous	Males	16	30.62 (SD ± 3.44)	34.37 (SD ± 3.7)	3.75 (SD ± 2.6)
	Females	14	29.85 (SD ± 3.66)	33.42 (SD ± 4.5)	3.57 (SD ± 3.0)
Total		30	30.23 (SD ± 3.55)	33.89 (SD ± 4.1)	3.66 (SD ± 2.8)

all or only 1 to 2mm Hg elevation in IOP on changing from the erect to the supine position. The comparable figure for the glaucomatous population was found to be 60% (18 eyes).

The effect of age on postural behaviour of IOP is correlated in Fig. 2. There seems to be a tendency towards enhanced effect on postural change of IOP with advancing age in glaucoma patients.

DISCUSSION

Maintenance of the rigid scleral envelope and clear cornea are to some extent dependant on the level of IOP. This level of IOP also plays a part in the effective perfusion of the retina and optic nerve head. Immediate rise in IOP on assuming the supine posture could result from a sudden increase in the uveal blood volume which in the abnormal eye is not regulated by redirecting the uveal blood flood from within the eye.[12] The increased ciliary blood flow may also enhance aqueous production. Elevation of episcleral venous pressure in the supine position would increase the outflow resistance and thus result in rise of IOP. Krieglastein *et. al.,*[13] suggested that the postural response depends on arterial and venous vascular changes when the subject moves from an erect to a horizontal position. Weber and Price[14] claimed that postural rise in IOP results from increase in the diastolic ophthalmic arterial pressure which is transmitted to the intraocular blood vessels causing their dialatation. William *et. al.,*[15] could not directly implicate the systemic blood pressure as the cause of this postural change in IOP. Local adrenergic receptors[16] are said to be important to maintain a stable IOP despite changes of posture. Inadequacy of this local regulatory mechanism might account for the pathogenesis of elevated IOP on lying down.

Postural rise of IOP shows a wide variation depending on the methods employed to measure the IOP.[6–9, 17, 18] Although the Perkin's hand-held tonometer demands some skill to use it accurately, the detection of rise in IOP is relatively easy. For the first time in normal Malaysian eyes, the sitting IOP has been found to be 14.14 (SD ± 3.56) mm Hg. This figure is similar to the one reported for the European population. However a large sample survey is needed to find out the exact figures for the Malaysian population. No attempt was made to study the variation of normal IOP in various Malaysian ethnic groups. Such a study will be obviously necessary as a

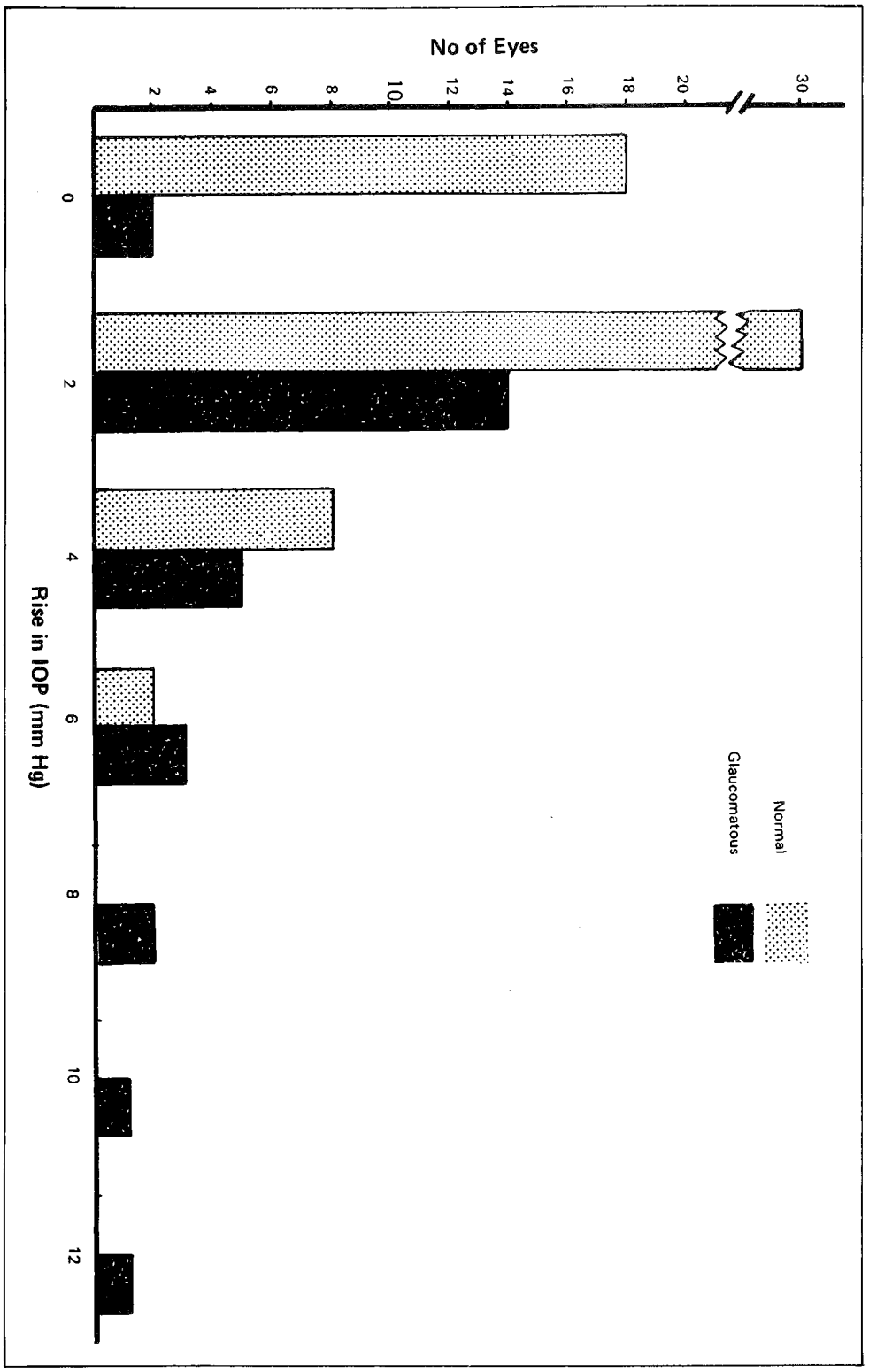

Fig. 1 Histogram of pressure differential between patients sitting and lying down.

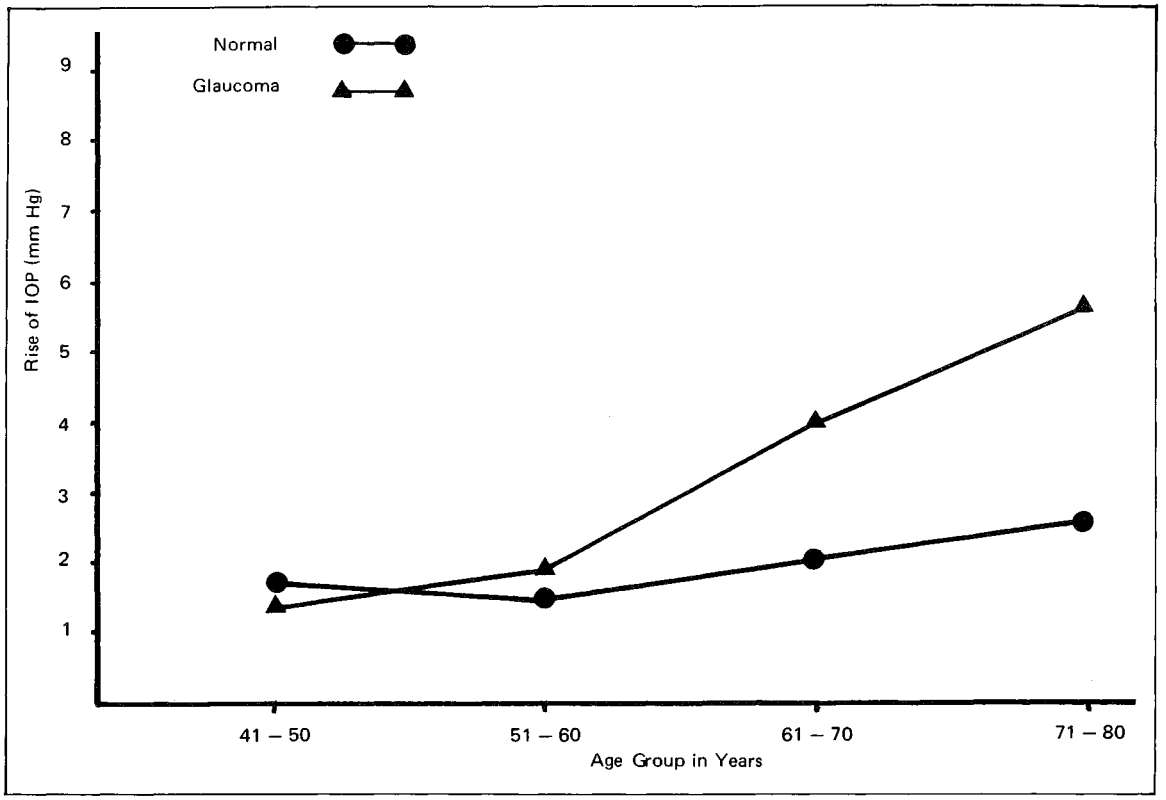

Fig. 2 Correlation of postural rise of intraocular pressure and age of the subjects.

baseline parameter for any further work on disease affecting the intraocular pressure, in this country.

Postural response of IOP in normal Malaysian eyes was found to be no different from that recorded in other studies.[6,12] 7 (23%) glaucomatous eyes showed a considerable rise in supine IOP, ranging from 5–12mm Hg. This study indicates that a small proportion of glaucomatous population has the tendency to elevation of IOP in the horizontal compared with the erect posture. Such a postural change in IOP has been reported to persist and remain constant, about five minutes after assuming the recumbent position.[12] This anomalous elevated IOP response to postural change has also been seen in some cases of retinal vein occlusion,[19] diabetes [15] and hypertension.[15]

Some workers have observed that rise in IOP when a patient becomes supine is higher in glaucomatous eyes.[6,7,20] This study has also confirmed this. Considerable rise of IOP in one eye alone was observed in three glaucomatous eyes in the present study. This phenomenon has been reported in unilateral chronic simple glaucoma[18] and ocular hypertension.[12] It seems to suggest an abnormality of pressure regulation locally within the eye.

A significant postural change of IOP has been recently reported in patients with low tension glaucoma.[11] It has been suggested that damage to the optic nerve in these cases might be occuring when the patient is actually asleep in the supine position. Greater postural rise of IOP in a given eye might be a helpful factor in the diagnosis of glaucoma. This method of assessment should be added to the general evaluation of IOP behaviour in glaucoma suspects. This should become particularly relevant when dealing with cases of low tension glaucoma or ocular hypertension.

It is equally important to know the postural change of IOP while treating patients with established wide open angle glaucoma. As most of our glaucoma patients are old, they conceivably spend a considerable time lying supine while sleeping or otherwise. This is particularly significant in ill and infirm bed-ridden patients. It seems reasonable to recommend that while evaluating therapeutic efficacy of antiglaucoma drugs, their effect on postural behaviour of IOP should also be studied. In the light of the present and other recent studies,[11,12] the level of supine IOP is perhaps equally important if not more, as the level of sitting IOP in relation to its harmful effects on the eye especially the optic nerve head. It can therefore be argued that this level of IOP should also be taken into consideration while assessing any medical or surgical regimen for the control of open angle glaucoma.

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REFERENCES

[1] Linner E, Rickenbach C, Wernes H. Comparative measurements of the pressure in the aqueous veins and the conjunctival veins using different methods. *Acta Ophthalmol (KBH)* 1950; 28 : 469–478.

[2] Weekers R, Lavergne G, Gongnard L. The origin of functional deficiencies in the hypertensive eye. *Doc Ophthalmol* 1959; 13 : 251–260.

[3] Armaly M F, Salamoun S G. Schiotz and applanation tonometery. *Arch Ophtal* 1963; 28 : 603–609.

[4] Langham M E, McCarthy F. A rapid pneumatic applanation tonometer: comparative findings and evaluation. *Arch Ophthal* 1968; 79 : 389–399.

[5] Hetland – Eriksen J. On tonometery 6. Comparative tonometery with Goldman applanation tonometer and the Schiotz tonometer in the lying position. *Acta Ophthalmol (KBH)* 1966; 44 : 522–538.

[6] Galin M A, McIvor J W, Magruder G B. Influence of position on introcular pressure. *Am J Ophthalmol.* 1963; 53 : 720–723.

[7] Anderson D, Grant W. The influence of position on intraocular pressure. *Invest Ophthalmol Visual Sci* 1973, 12 : 204–212.

[8] Tarkkanan A, Leikola J Postural variation of the IOP as measured with the Mackay Marg tonometer. *Acta Ophthalmol (KBH)* 1967; 45 : 569–575.

[9] Tsamparlakis J, Casey T A, Howell W, Edridge E. Dependence of IOP on induced hypotension and posture during surgical anaesthesia. *Trans. Ophthalmol Soc U K* 1980; 100 : 521–526.

[10] Jain M R, Marmion V J. A clinical evaluation of the applanation pneumatonograph. *Br J Ophthalmol* 1976; 60 : 107–110.

[11] Tsukahara S, Sasaki T. Postural change of IOP in normal persons and in patients with primary wide open angle glaucoma and low tension glaucoma. *Br J Ophthalmol* 1984; 68 : 389–392.

[12] Leonard I J K, Kerr Muir M G, Kirby G R, Hitching R A. Ocular hypertension and posture. *Br J Ophthalmol* 1984; 67 : 362–366.

[13] Krieglstein G K, Waller W K, Leydecker W. The vascular basis of the postural influence on the IOP. *Albrecht von Gradfe Arch Klin Ophthalmol* 1978; 106 : 99–106.

[14] Weber A K, Price K. Pressure differential of IOP measured between supine and sitting position. *Ann Ophthalmol* 1981; 13 : 323–326.

[15] Williams B I, Peart W S, Letley E. Abnormal intraocular pressure control in systemic hypertension and diabetes mellitus. *Br J Ophthalmol* 1980; 64 : 845 – 851.

[16] Langham M E. Role of adrenergic mechanisms in development and therapy of open angle glaucoma. *Proc R Soc Med* 1971; 04 : 622–628.

[17] Langley D, Swanljung H. Ocular tension in glaucoma simplex. *Br J Ophthalmol* 1951; 55 : 445–451.

[18] Inglima R. Effect of patient position of applanometer readings. *Ear Nose Throat J* 1966; 45 : 64–69.

[19] William B I, Peart W S. Effect of posture on the intraocular pressure of patients with retinal vein obstruction. *Br J Ophthalmol* 1978; 62 : 688–693.

[20] Hetland – Eriksen J. On tonometery 5. The pressure of glaucomatous eyes measured in the sitting and the lying positions by means of the Goldman applanation tonometer. *Acta Ophtahlmol (KBH)* 1966; 44 : 515–521.