

## Ocular hypertension and posture

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**SUMMARY** Thirty-seven patients with ocular hypertension (OH) and 29 age matched controls were studied. Intraocular pressure (IOP) measurements were made in the sitting position and, immediately after changing posture, in the lying position. Sixty-three (85%) of the OH eyes and all the control eyes showed either stable IOP readings on changing posture or small elevations up to 3 mmHg. Eleven (15%) of the OH eyes showed larger elevations of IOP, between 5 and 9 mmHg when the patients changed to the lying position. We suggest that an IOP measurement in the lying position should be included in the routine evaluation of the patient with ocular hypertension.

Ocular hypertension (OH) may be defined as an IOP in excess of 21 mmHg in eyes with normal visual fields, normal angles, and without glaucomatous cupping of the optic disc.

A 15-year follow-up by Sorenson *et al.*<sup>1</sup> of OH patients showed that the great majority did not develop chronic simple glaucoma (CSG), confirming previous actuarial studies by Hollows and Graham<sup>2</sup> that a large proportion of eyes with an IOP in excess of 21 mmHg do not progress to develop CSG as defined by glaucomatous disc or field change.

The identification of susceptible OH patients who will develop CSG is an unsolved clinical problem. Hitchings and Wheeler<sup>3</sup> emphasised that the decision on which patients with OH alone require treatment must be based on factors other than the recognition of an elevated IOP. They divided these factors into high risk, predictive, and marker groups. All these parameters have been found useful but not totally reliable in recognising the OH patients most at risk. The high risk factors are found in a larger proportion of patients with CSG than normal, although they may occur in the normal population. The high risk factors are positive family history,<sup>4</sup> diabetes,<sup>5</sup> high myopia,<sup>6</sup> optic cup asymmetry,<sup>7</sup> and a cup/disc ratio  $>0.5$ .<sup>2,8</sup> An extensive review of the other possible risk factors for OH patients developing CSG has been presented by Kass *et al.*<sup>8</sup>

The normal population show a remarkable stability of IOP on changing posture.<sup>9-11</sup> IOP measurements

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in the lying position (LIOP) are the same as IOP measurements in the sitting position (SIOP). This stability may be lost in the CSG population,<sup>9-15</sup> and it has been suggested that an elevation of the LIOP in comparison with the SIOP might be a simple and useful qualitative test for CSG detection.<sup>12</sup> No study has been carried out on an OH population to this effect.

We present an evaluation of the IOP changes induced by posture in a group of patients with ocular hypertension and a group of controls and discuss their significance.

### Pilot study

Twelve patients were selected at random from the OH clinic at Moorfields Eye Hospital, High Holborn. IOP readings were taken with a Perkins hand-held tonometer in the sitting and lying positions. Two of the patients showed a marked elevation of IOP, 5 mmHg and 7 mmHg, on assuming the lying position while the remaining 10 patients showed no change.

The 2 patients who showed an elevation of the LIOP in comparison with the SIOP were also distinguished from the other OH patients by having a strong family history of glaucoma.

The other 10 OH patients had none of the recognised high risk factors for CSG. The combination of an apparently unstable IOP on changing posture in OH patients with high risk factors for CSG prompted a more detailed study into the postural response of IOP in OH patients compared to controls.

## Materials and methods

The ocular hypertensive patients were chosen from those attending the OH clinic at Moorfields Eye Hospital, High Holborn, and from patients with OH seen at St Thomas's Hospital. OH was defined as an IOP in excess of 21 mmHg in eyes with normal visual fields, normal angles, and discs without glaucomatous cupping.

Patients on systemic antihypertensive therapy were excluded, since some antihypertensive drugs (especially beta blocking agents) are known to influence the intraocular pressure. No patient was on any eye medication nor had had topical steroids for ocular disease.

The control patients were selected to match the age and sex distribution of the OH group. Myopic and diabetic patients were included in the control group. Sitting and lying blood pressures were recorded from all subjects. The patients were interviewed and examined at the same time of day, between 9 am and 11 am. Having seated the patient in a quiet room for approximately 30 minutes a full medical and ophthalmic history was taken by one doctor, who asked for evidence of the recognised high risk factors for CSG.

The initial examination consisted of a sitting and lying blood pressure reading, followed by a full ocular examination. Particular attention was given to the refraction, evidence of previous ocular disease, and the optic disc appearances.

A second doctor, with no knowledge of the previous interview and examination, measured the IOP as follows. The patient was asked to walk slowly from the interview room and sit on an examination couch. A Perkins hand-held tonometer was used to measure the SIOPI. Three readings were taken and the mean used for the study. The second eye was similarly evaluated. The patient was then asked to lie flat and the procedure was repeated immediately to measure the LIOP. A further set of LIOP readings was made on all patients after they had remained in the lying position for 15 minutes. Finally a repeat SIOPI

measurement was made after the patient regained the sitting position.

## Results

A total of 74 OH eyes and 58 control eyes were examined. No patient had systemic hypertension, nor was any patient found to have a significant difference in the blood pressure readings when measured in the sitting and lying positions.

The results are illustrated in Table 1 and Figs. 1 and 2. A change in IOP of 4 mmHg or greater on assuming the lying position was used to separate a group of 11 OH eyes which showed IOP rises of 5–9 mmHg on attaining the supine position. The remaining 63 eyes and all the control eyes showed either a small elevation up to 3 mmHg or no variation in IOP on changing posture.

The 11 OH eyes with a postural elevation of IOP between 5 and 9 mmHg belonged to 8 OH patients whose age, sex, and duration of OH since diagnosis were similar to the other OH patients. However, the high risk factors for CSG were more prevalent in these 8 OH patients, with an average of 1.12 risk factors per patient compared with 0.24 risk factors per patient in the control group and 0.18 risk factors per patient in the other OH patients (Table 1). The commonest risk factor in the OH group with raised LIOP measurements was a definite family history of glaucoma. Five of the 8 patients in this group had a family history of glaucoma, while diabetes, myopia, and optic disc asymmetry were also found. The risk factors found in the other OH patients and in the control patients were myopia and diabetes.

In every patient the LIOP readings after 15 minutes were within 1 mmHg of the readings taken immediately after changing posture as described above.

To emphasise the reproducibility of the results in this paper the readings from the patients examined in the pilot study were compared with the readings from the same patients when they were reviewed in the detailed study. In each case the 2 sets of readings

Table 1 Comparison of age, sex, IOP measurements, risk factors and longevity of OH in OH and control patients

Patients	11 OH eyes IOP elevation on lying 5 mmHg–9 mmHg	63 OH eyes IOP elevation on lying 0 mmHg–3 mmHg	58 Control eyes IOP elevation on lying 0 mmHg–3 mmHg
Average age in years	56.27	60.67	52.0
Male:female	5:3	16:16	16:13
Average SIOPI mmHg	25.54	24.04	13.79
Average LIOP mmHg	32.45	25.68	14.29
Average LIOP – SIOPI mmHg elevation on lying	6.9	1.42	0.5
Average risk factors per patient	1.12	0.18	0.24
Average longevity of OH since diagnosis (years)	6.87	7.05	Not applicable

SIOPI=sitting intraocular pressure. LIOP=lying intraocular pressure.

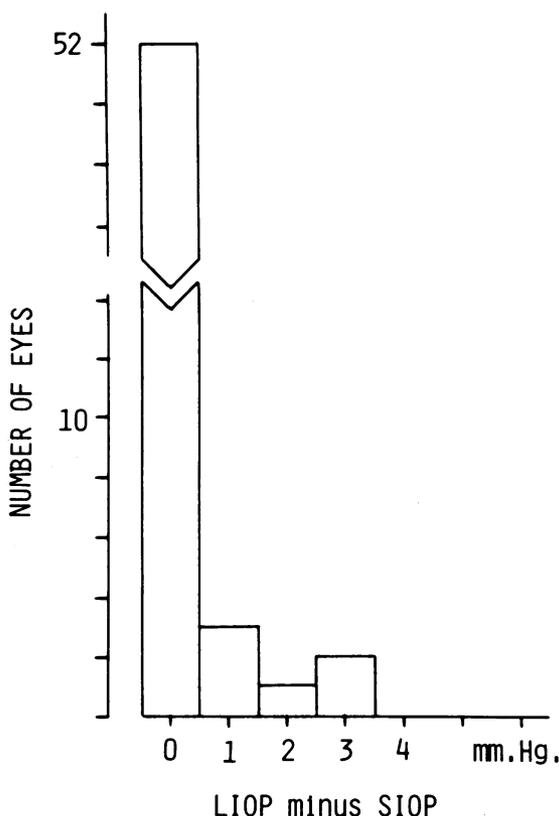


Fig. 1 Rise in IOP level on lying: 58 control eyes.

were within 1 mmHg of each other. In particular the 2 OH patients noted in the pilot study to have a marked postural elevation in LIOP, showed a similar response on the second occasion.

#### FURTHER STUDIES

Further LIOP readings were carried out with the patients remaining lying down for a period of up to 4 hours. The LIOP readings were remarkably stable, with variations confined to 1 mmHg in excess of the original LIOP. No eyes showed a fall in the LIOP reading when repeated measurements were made.

On the patient's regaining the sitting position the SIOP once more assumed a reading within 1 mmHg of the previous SIOP level.

#### Discussion

This study shows that a small proportion of an OH population has a tendency to elevation of the LIOP compared with the SIOP. This elevation persists if the patient remains in the lying position. The LIOP, once raised to a new level, remains at that level while

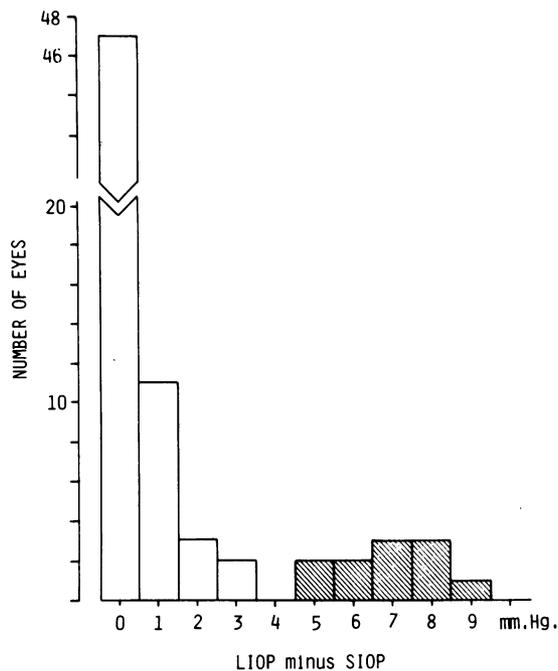


Fig. 2 Rise in IOP on lying: 74 ocular hypertensive eyes.

the lying position is maintained and does not appear to fluctuate. On the patient's regaining the sitting position the SIOP returns to the same level as the original SIOP.

The normal population do not show this elevation of LIOP, but some patients with CSG, retinal vein occlusion, systemic hypertension, and diabetes also show this anomalous elevated IOP response to a change of posture.

The discussion is directed to a brief review of the physiology of normal IOP and possible mechanisms for an apparent failure in homeostasis in patients who develop an elevated LIOP. Maintenance of the rigid scleral envelope and clear cornea are to some extent dependent on the IOP level. The IOP level also influences ocular perfusion, since perfusion pressure in the arterioles supplying the optic nerve head and retina must be in excess of the IOP to allow blood flow.

Normal IOP levels reflect a balance between aqueous production and aqueous outflow, the latter being dependent on outflow resistance. Aqueous production requires adequate perfusion of a healthy ciliary body to allow active secretion and ultrafiltration. The outflow is principally via the trabecular meshwork to collector channels leading to the episcleral veins. A small amount of aqueous also escapes via the uveoscleral outflow.

An elevation of LIOP as seen in this study and in other groups of patients<sup>9-13</sup> requires mechanisms to account for an immediate rise in IOP which is subsequently sustained at that level. The immediate rise in LIOP could be explained by a sudden increase in the uveal blood volume, which in the abnormal eye is not compensated by vascular channels opening to redirect the uveal blood from within the scleral envelope. Occasionally<sup>11</sup> uveal vascular tumours are associated with an elevated LIOP. The sustained elevation of IOP might be due to an increase in aqueous production or an increase in outflow resistance. Uveal engorgement may increase the perfusion of the ciliary body allowing an increase in aqueous production. Elevation of episcleral venous pressure on lying down would increase outflow resistance and cause a rise in LIOP.

There are contrasting studies on the relationship between facility of outflow and an elevation of the LIOP. Williams and Peart<sup>16</sup> found no correlation between tonography results and postural IOP changes. Inghima,<sup>10</sup> however, found an increase in the LIOP measurements when the facility of outflow was below 0.2 in a group of CSG patients.

The role of the systemic blood pressure has been examined by Williams *et al.*<sup>14</sup> They found no correlation between levels of systemic blood pressure or postural systemic blood pressure changes and an elevated LIOP. They concluded that while some systemic hypertensive patients show elevated LIOP measurements the systemic blood pressure could not be directly implicated to explain the IOP changes.

Langham<sup>15</sup> noted an increase in the ophthalmic artery pressure in patients with an elevation of LIOP. However, ophthalmodynamometry itself may alter IOP by extrinsic pressure, and it is difficult to assess the influence of ophthalmic artery pressure on the IOP.

The unilateral elevation of LIOP in 4 OH patients in this study is interesting. This phenomenon is also found in cases of unilateral CSG<sup>10</sup> and may suggest that the mechanism for failure of IOP control may be locally within either the eye or orbit. However, Williams and Peart<sup>13</sup> found a bilateral elevation of LIOP in patients with unilateral retinal venous occlusion.

Local vasomotor dysfunction may be important in the pathogenesis of elevated LIOP, and it is interesting that patients with Horner's syndrome (Brazier J, unpublished) following cervical sympathectomy show changes in IOP control. The efficacy of beta blocking agents in the eye to treat CSG suggests that local adrenergic receptors<sup>17</sup> are important in IOP control and may contribute to maintaining a stable IOP despite changes of posture.

The quantitative aspect of the IOP rise requires

discussion. Measured by different methods, the postural changes of IOP show a wide variation in the actual rise in IOP recorded.<sup>9-13</sup> In this study the IOP rise varied from 0 to 9 mmHg by the Perkins hand-held tonometer. Eleven eyes showed elevations of IOP in excess of 5 mmHg. The Perkins instrument requires a reasonable degree of skill to use accurately, but with it the detection of a rise in LIOP is relatively easy.

The possible value of LIOP measurements is enhanced by evidence of the reproducibility of the elevated LIOP response in OH patients seen on more than one occasion. Twelve of the OH patients were seen twice—in the pilot study and in the detailed study. The results were very similar in these patients, as discussed above.

The reproducible results combined with the ease of performing LIOP measurements with the Perkins tonometer indicate that this method of assessment might be added to the general evaluation of the OH patient. A marked elevation (>4 mmHg) of the LIOP in comparison with the SIOP may be a further helpful factor in attempting to predict which OH patients will develop CSG. A follow-up study on the cases evaluated here is in progress to this effect.

#### CONCLUSION

It is likely that large rises of LIOP are associated with a complex failure of homeostasis. In an ocular hypertensive patient such a tendency might indicate that there is a greater chance of the patient developing chronic simple glaucoma, especially if there are other high risk factors present. We suggest that measurements of IOP in both sitting and lying positions should be included in the evaluation of patients with ocular hypertension.

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