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Postural response of intraocular pressure following traumatic hyphaema

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SUMMARY Twenty patients with previous unilateral traumatic hyphaema and 25 age-matched controls were studied. There was a progressive rise in intraocular pressure when the patient changed from the standing to the sitting position and then to the lying position in both groups. No control eye showed a rise greater than 2 mmHg when the subject changed from sitting to lying. However, 14 (70%) of the injured eyes and 12 (60%) of the fellow eyes showed an exaggerated response. We suggest that the presence of an abnormal postural response may indicate a predisposition to post-traumatic glaucoma. Our findings are compatible with a linked control of postural intraocular pressure response between the two eyes.

Abnormal postural response of the intraocular pressure has been suggested as an indicator in the detection of glaucoma[1] and also in the identification of ocular hypertensive patients at risk of developing glaucoma.[2] Traumatic hyphaema is commonly associated with damage to the anterior chamber angle,[3] and the pathological changes seen have been linked with the development of chronic secondary glaucoma.[4] We studied the abnormal postural response in an attempt to identify which patients might develop glaucoma following traumatic hyphaema.

Materials and methods

Twenty patients with unilateral traumatic hyphaema who had been admitted to Southampton Eye Hospital immediately after injury were selected from the hospital ledger. They were examined on average eight months after injury (range 5 to 36 months), and their ages were 14 to 38 years, there being 15 males and five females. The hospital routine led to topical steroids being used in most cases for the first two weeks only following discharge. No ocular therapy was being used at the time of examination. No account was taken of either the size or the duration of the hyphaema, medical illness, or drug therapy. Twenty-five control subjects were studied, all of whom were in good health and had no past ocular history. Their ages ranged from 14 to 33 years, there being 16 males and nine females. Both control subjects and patients had normal visual fields prior to investigation. The anterior chamber angle of all injured eyes was studied with a gonioscope.

All intraocular pressure measurements were taken as an average of two 10-second recordings with an Alcon applanation pneumotonograph which had been recently calibrated. The required posture was assumed for 15 minutes before each pressure measurement, and measurements were made in the following positions consecutively: sitting, lying, sitting, and standing. The right eye was always examined first.

Results

The mean intraocular pressures in the four positions are shown in Table 1. There are illustrated graphically in Fig. 1.

The intraocular pressure in the original sitting position was similar in all the eyes. The greatest postural change in intraocular pressure occurred on moving from the sitting to the lying position and is illustrated in Figs. 2, 3, and 4. No control eye had a rise greater than 2 mmHg, while 14 (70%) injured eyes and 12 (60%) fellow eyes showed a rise greater than 2 mmHg. The greatest rise was 9 mmHg in one

*Work undertaken during fourth year medical student elective.

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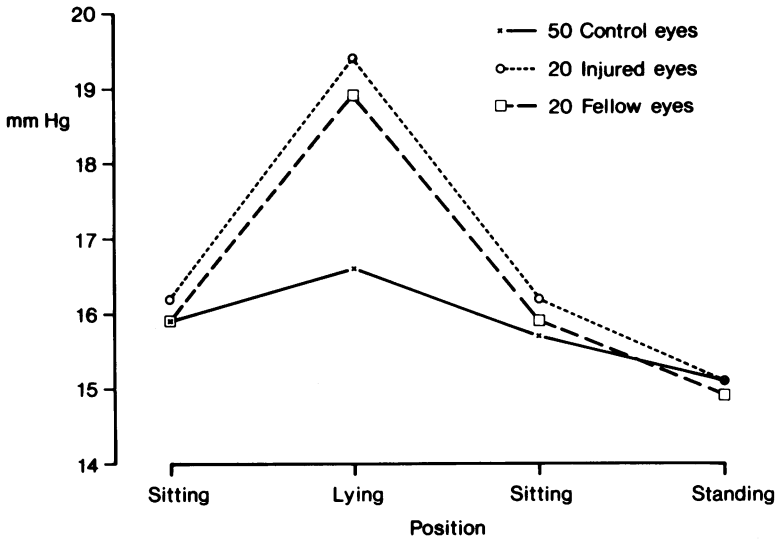

Fig. 1 *Mean intraocular pressures of control, injured, and fellow eyes in different postures.*

Table 1 *Mean (standard error) intraocular pressures in mmHg*

	Position			
	Sitting	*Lying*	*Sitting*	*Standing*
Injured eyes	16·2 (0·48)	19·4 (0·77)	16·2 (0·36)	15·1 (0·36)
Fellow eyes	15·9 (0·49)	18·9 (0·39)	15·9 (0·37)	14·9 (0·36)
Control eyes Left	15·9 (0·26)	16·7 (0·26)	15·8 (0·24)	14·9 (0·24)
Right	15·8 (0·19)	16·6 (0·19)	15·6 (0·26)	15·2 (0·21)

injured eye and 10 mmHg in one fellow eye in different patients (the contralateral eye rise being 5 mmHg and 4 mmHg respectively). Only three patients showed a rise in intraocular pressure in the fellow eye greater than the injured eye, while six injured eyes had a rise greater than the fellow eye.

There was a significant difference between the average response in a sitting position and that in a standing position (observed $F'_{129}=31{\cdot}69$, $p<0{\cdot}01$). This pattern of response did not differ between groups or between eyes (observed $F'_{129}=1{\cdot}02$, $p>0{\cdot}05$ and observed $F'_{172}=0{\cdot}68$, $p>0{\cdot}05$ respectively).

There was a significant difference between the average response in a sitting or standing position and that in a lying position (average sitting/standing= 15·62, lying=17·76; observed $F'_{129}=218{\cdot}93$, $p<0{\cdot}01$). This pattern of response was significantly different between groups (observed $F'_{129}=66{\cdot}35$, $p<0{\cdot}01$). There was a much more pronounced rise in pressure in patients than in controls.

The pattern of response between eyes was not significantly different between groups (observed F'_{172}

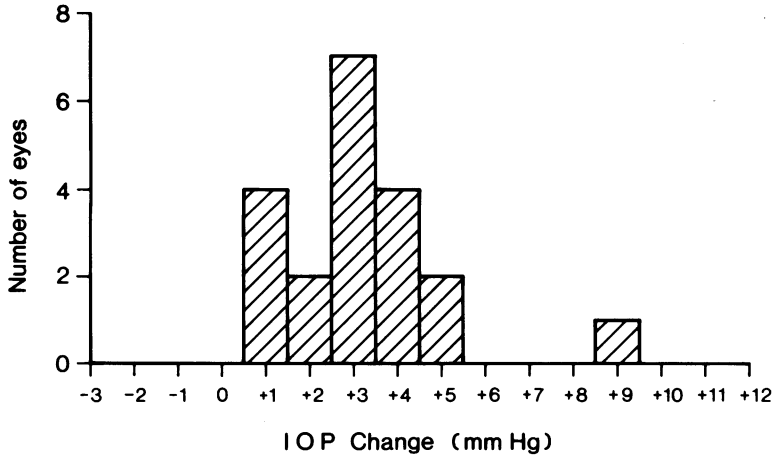

Fig. 2 *Injured eyes (20): IOPs on change from sitting to lying.*

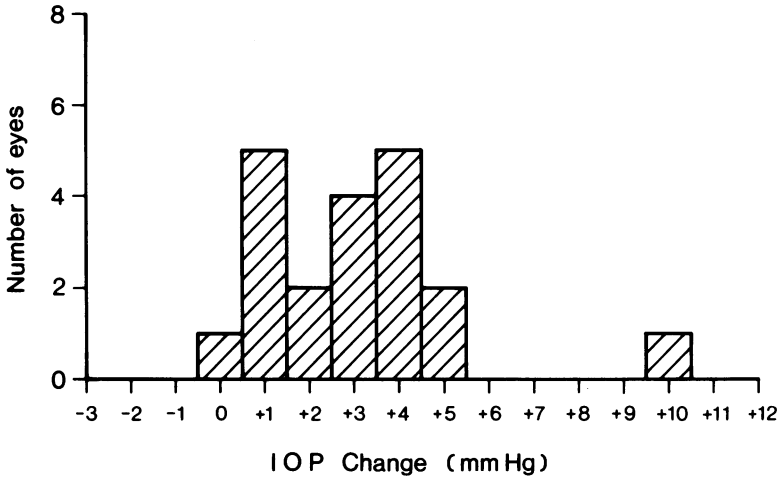

Fig. 3 *Fellow eyes (20): IOPs on change from sitting to lying.*

=0·64, p>0·05). The rise in pressure was binocular, but the magnitude of this rise depended on group membership, being greater in patients than in controls.

Discussion

The usefulness of the pneumotonometer in measuring postural changes in intraocular pressure is well documented,[5-8] and our intraocular pressure values in the sitting position correspond well with previously reported figures.

Our results show a progressive rise in the intraocular pressure from standing to sitting to lying in contrast to those of Langham,[6] who showed no difference between sitting and standing values. The patients in our study showed an abnormal postural response in the injured eye and a remarkably similar response in the fellow eye. As regards the postural change from sitting to lying (which produces the largest response), 12 patients (60%) showed a rise in both eyes greater than 2 mmHg, while a further two patients had a rise greater than 2 mmHg in the injured eye only. This marked bilaterality of abnormal postural response contrasts with the bilateral abnormal response in only three out of 37 ocular hypertensive patients studied by Leonard *et al.*,[2] while Inglima[1] described four patients with unilateral glaucoma who showed an abnormal response in the affected eye only.

Anderson and Grant[9] commented on the similarity of postural intraocular pressure response in the right and left eye of patients referred with possible glaucoma. Our study confirms this in both patients and controls, irrespective of whether the response was normal or abnormal. Our results also agree with previous reports[9,10] that the abnormal postural response is not related to the level of intraocular pressure in the original sitting position.

An abnormal postural intraocular pressure response has been repeatedly demonstrated in patients with open angle glaucoma[5,6,9,11] and more recently in conditions having a well-established association with glaucoma, these being ocular hypertension,[2] retinal vein occlusion[8,10] and this study of eyes with traumatic hyphaema. Only this study and those of Williams and Peart[8,10] show that a bilateral abnormal response between an affected eye and its fellow is a frequent finding. Williams and Peart[10] considered that the explanation for this finding was a pre-existing abnormality in both eyes tending to a predisposition to retinal vein occlusion. However, we would argue from our findings that an alternative explanation is that an abnormality in one eye (whether due to central retinal vein occlusion or trauma) resets the mechanism that controls the intraocular pressure of the two eyes when the posture is changed. Welsh[12] too showed how injury to one eye, in the form of laser trabeculoplasty, affects the intraocular pressure in the contralateral eye—both intraocular pressures falling after treatment.

Characteristics of the postural response which have been demonstrated by other workers are rapidity of change to a new level of intraocular pressure[6] and the sustained maintenance of this level.[2,6] Our results also showed reproducibility, as the second reading in the sitting position corresponds well with the previous reading in this position (see Fig. 1).

Leonard *et al.*[2] suggested that local vasomotor function may be important in the pathogenesis of raised intraocular pressure when lying and that there is a complex failure of homoeostasis when the postural response is abnormal. The homoeostatic

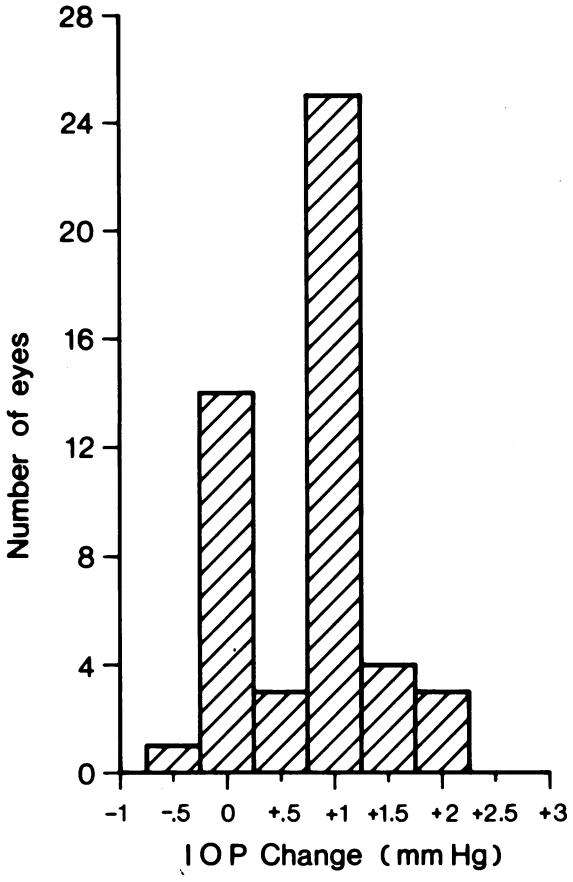

Fig. 4 *Control eyes (50): IOPs on change from sitting to lying.*

mechanism is unknown, but we conclude that a neuronal control with linkage between the two eyes is most likely to account for a bilateral, rapid, sustained, reproducible response.

Further studies are needed to assess binocular aspects of intraocular pressure control in pursuit of possible forms of linkage.

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