RETROBULBAR HAEMODYNAMIC CHANGES STUDIED BY COLOUR DOPPLER IMAGING IN GLAUCOMA

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SUMMARY

Purpose: **To determine the effect of spontaneously elevated intraocular pressure (IOP) on the ocular circulation, and evaluate the result of IOP-lowering procedures in terms of haemodynamics.**

Methods: **Colour Doppler imaging (CDI) was employed to determine the peak systolic velocity (PSV), end-diastolic velocity (EDV) and time average maximal velocity (TAMV), as well as the Pourcelot ratio (PR) and pulsatility index (PI), of the central retinal artery (CRA), posterior ciliary arteries (PCA) and ophthalmic artery. Various CDI parameters of the eyes with elevated IOP were compared with those of the clinically healthy fellow eyes and the control eyes, separately. Also, data from CDI of glaucoma eyes obtained during the period of elevated IOP and then following IOP-lowering procedures were compared, deliberately avoiding the influence of glaucoma medications.**

Results: **Eyes ($n = 12$) with elevated IOP showed significantly decreased flow velocities of the CRA and significantly increased PR and PI of the nasal and temporal PCA, compared with the fellow and control eyes, respectively. Following IOP-lowering procedures, the PR and PI of the nasal PCA decreased significantly.**

Conclusion: **Spontaneously elevated IOP may induce haemodynamic changes in the CRA and PCA, but no significant change is identified in the ophthalmic artery. Flow velocities tend to decrease while the resistance indices tend to increase with elevated IOP. Such haemodynamic changes may reverse following normalisation of IOP.**

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The pathogenetic basis of optic nerve damage in glaucoma is still not well understood, though increased intraocular pressure (IOP) has been recognised as one of the important risk factors. While distortion of laminar architecture at the pars scleralis and injury to axonal bundles with blockade of axoplasmic flow by mechanical force of IOP may take part in the disease process, there are several lines of evidence indicating that haemodynamic change involving the optic nerve head plays an important role in the pathogenesis of optic neuropathy. Hayreh and others pointed out the importance of perfusion pressure in the optic nerve,[1] and Socci and Anderson[2] hypothesised that inhibition of autoregulation of blood flow to the optic nerve can increase susceptibility of the disc to pressure-induced ischaemia. The effect of IOP on ocular blood flow has been studied using a variety of methods such as heated thermocouple, India ink, microspheres, iodoantipyrine iodine-125, fluorescein angiography, laser Doppler velocimetry, blue field entopic phenomenon, tritiated iodoantipyrine, hydrogen clearance method and Doppler ultrasonography.[3–11] Many of these are invasive or even destructive, and are restricted to the investigation of animal models. Others are applicable to human evaluation, yet require the use of mydriatics, or may be affected by opaque optical medium. Besides, most investigations were performed under artificially elevated IOP induced by either a suction cup acting on the sclera or fluid infusion into the anterior chamber. The impact of naturally elevated IOP on human ocular circulation is still not clear, and whether a reduction of IOP in such eyes will change the ocular haemodynamics needs further investigation.

Colour Doppler imaging (CDI) facilitates the study of blood flow velocity by colour-coding the Doppler frequency shifts of ultrasound and superimposing such a colour signal on B-scan anatomical

detail. Colour-coded flow information is helpful in identifying the location of small vessels, as well as in determining the angle of the incident beam to the direction of flow, thereby allowing the measurement of flow velocity. It is a non-invasive, painless and reproducible technique that can be performed in a clinical setting, and has been used in the examination of ocular vascular system.[12,13] Recently, CDI has been applied to the study of glaucoma by some authors.[14–17] Trible *et al.*[15] have employed it to study the retrobulbar haemodynamic change after trabeculectomy, yet they could not avoid the possible influence from glaucoma medications. The present study uses CDI to determine the retrobulbar haemodynamics in glaucoma patients during the period of elevated IOP, and to evaluate the effect of IOP-lowering procedures on ocular circulation. The inclusion criteria of patient selection allowed us to avoid the possible influence from glaucoma medications.

SUBJECTS AND METHODS

Study Group

Patients who presented themselves with an IOP equal to or higher than 30 mmHg and had never been treated with glaucoma medications were considered as candidates for the CDI study. All eyes had a complete ophthalmological examination, including best-corrected visual acuity, slit-lamp biomicroscopy, tonometry, gonioscopy and fundoscopy. To avoid possibly jeopardising the patient's final visual outcome due to a short delay in management as a result of the imaging process, patients who had a large optic disc cup (>80%) or a pale disc were excluded, except for two patients who had already lost light perception with a disc of total cupping in the diseased eye. Other exclusion criteria were as follows: (1) neovascular glaucoma and glaucoma secondary to inflammation, tumour or elevated episcleral venous pressure, (2) retinal pathology, (3) a past history of orbital or ocular surgery, and (4) a past history of optic nerve disorders. CDI was performed by one experienced sonologist as soon as the diagnosis was confirmed (occasion A). Management of glaucoma was started only and immediately after the imaging process, which took about 15–20 min for each eye. Ophthalmological examinations were repeated again after normalisation of the IOP when the eyes had a clear cornea, and visual field examinations were performed weeks later. Of all the eyes that had been enrolled into the study, 12 eventually gained good control of IOP without the need for glaucoma medication following laser treatment or surgery. These patients then received another CDI examination (occasion B), which was performed at least 3 months after surgery. CDI and a complete ophthalmological examination were also performed in the clinically healthy fellow eyes.

Control Group

Twenty-seven consecutive patients who had no ocular disorders other than mild lens opacity and/or refractive error (spherical equivalent <3 D and astigmatism <1.5 D) were enrolled. None are blood relatives of glaucoma patients. All had a complete ophthalmological examination. Patients who had a history of ocular surgery were excluded. All control subjects had an IOP less than 22 mmHg as measured on three separate occasions, and had a normal optic cup/disc ratio (less than 0.4) with healthy neuroretinal rim.

Colour Doppler Imaging

All examinations were performed using a CDI machine (Acuson 128XP/10, Acuson, Mountain View, CA) with a 7.0-MHz linear probe. The patient was in the supine position with eyes closed during the examination. The ultrasound transducer was applied onto the eyelid with ultrasonic jelly as an acoustic coupling agent, and care was taken to exert as little pressure as possible on the eye to avoid artifacts. We first scanned the whole orbit with grey-scale imaging in transverse and sagittal planes, then applied CDI with the colour scale set at slow flow (3 cm/s) to identify the orbital vessels.

For Doppler spectral analysis, the sample volume was adjusted to a range of 1 mm, and the best-quality curve for each vessel was chosen for analysis. The central retinal artery (CRA) was identified within the anterior optic nerve, and the area analysed was placed with its centre about 3 mm behind the surface of the disc. The temporal and nasal posterior ciliary arteries (PCA) were identified along the sides of the optic nerve and the centre of the sample volume was set within 8 mm behind the posterior sclera. To measure the ophthalmic artery flow velocity, the sample volume marker was set about 25–30 mm posterior to the globe.

The shift of Doppler frequency within the CRA, nasal and temporal PCA and ophthalmic artery was measured to determine the peak systolic velocity (PSV), end diastolic velocity (EDV) and time average maximum (TAMV) velocity. Then the Pourcelot ratio (PR) and pulsatility index (PI) were calculated by PR = (PSV − EDV)/PSV, PI = (PSV − EDV)/TAMV. Both PR and PI provide measures of downstream resistance to blood flow.

Informed consent was obtained from all patients and volunteers before examination.

Statistical Analysis

The IOP, perfusion pressure (2/3 mean blood pressure − IOP) and various CDI parameters

Table I. Characteristics of patients in the study and control groups

	Study group	Control group
Number	12	27
Age (years)	64.5 ± 7.5 (mean ± SD)	71.7 ± 5.5 (mean ± SD)
Gender		
Male	9 (75%)	24 (89%)
Female	3 (25%)	3 (11%)
Hypertension	5 (42%)	13 (48%)
Diabetes mellitus	1 (8.3%)	2 (7.4%)
Current smoker	4 (33%)	12 (44%)
Under systemic vasoactive drugs	5 (42%)	11 (41%)

measured from the right and left eyes of the control subjects were compared with each other by paired Student's *t*-test. As no statistically significant difference between right and left eyes was assured, one eye of each was chosen randomly and then grouped together as the control. A normality test had been performed before Student's *t*-test was used.

Comparisons were made for the IOP and every CDI parameter of each blood vessel. First, the study group was compared with the control group using an unpaired Student's *t*-test. Second, the diseased eyes were compared with the fellow eyes using a paired Student's *t*-test. Finally, the data from occasion A and occasion B were compared with each other using a paired Student's *t*-test for the diseased and the fellow eyes, respectively.

RESULTS

The characteristics of the patients in the study and control groups are listed in Tables I and II. The presenting IOP of the diseased eyes was 49.3 ± 13.6 mmHg (mean ± SD), which reduced significantly to 13.3 ± 3.9 mmHg after Nd:YAG laser peripheral iridotomy (LPI; 1 eye), LPI and argon gonioplasty (1 eye), trabeculectomy (7 eyes), or combined cataract and glaucoma surgery (3 eyes) (Table II). The mean IOP of the fellow eyes was 16.4 ± 3.7 mmHg (range 9–21 mmHg) on occasion A and 16.4 ± 2.2 mmHg (range 12–20 mmHg) on occasion B. Prophylactic LPI was performed in the fellow eyes of 9 cases of primary angle-closure glaucoma.

The mean IOP of the control group was 14.7 ± 2.4 mmHg (range 11–20 mmHg), which was not significantly different from the mean IOP of the clinically healthy fellow eyes.

The mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) of the control group were 135 ± 12 mmHg and 79 ± 6 mmHg, respectively. The mean SBP and DBP of the study group was 138 ± 19 and 79 ± 10 mmHg on occasion A, and 138 ± 13 and 83 ± 8 mmHg on occasion B (Table II). There was no significant difference in SBP and DBP between the control and the study group, or between occasion A and occasion B in the study group.

The mean age of the study group was significantly younger than that of the control group ($p = 0.012$). The proportions of diabetics and hypertensives were not significanly different between the control and study group, neither were the proportions of patients who smoked and took vasoactive medicine (Table I).

Table II. Characteristics of the glaucoma patients

Patient no.	Sex	Age (years)	Diagnosis	IOP (mmHg) A/B	SBP (mmHg) A/B	DBP (mmHg) A/B	VF
1	M	68	PACG acute	59/14	165/156	94/90	MD: −3.46 dB WNL
2	M	78	POAG chronic	31/15	151/160	74/80	MS: 17.6 dB AS, NS
3	F	66	PACG chronic	38/16	158/150	89/90	MD: −7.26 dB NS
4	F	54	POAG chronic	42/18	124/134	66/80	MD: −13.48 dB AS
5	M	68	PACG chronic	39/10	110/128	70/74	MD: −13.78 dB SS, NS
6	M	68	PACG chronic	78/8	162/148	92/80	NA
7	M	67	PACG chronic	48/7	114/114	66/74	MD: −18.8 dB AS, GR
8	M	67	PACG acute	62/11	153/125	78/70	MD: −8.98 dB GR
9	M	60	PACG chronic	54/21	140/132	90/83	MS: 14.1 dB SS, NS
10	F	54	PACG chronic	38/16	136/138	74/86	MD: −20.5 dB CI
11	M	54	Angle recession glaucoma	62/11	120/136	80/95	MS: 18.4 dB SS, NS
12	M	70	PACG chronic	46/12	126/136	80/88	NA

IOP, intraocular pressure of the diseased eye; SBP, systolic blood pressure; DBP, diastolic blood pressure; VF, visual field; A, measurement on occasion A; B, measurement on occasion B; PACG, primary angle-closure glaucoma; POAG, primary open angle glaucoma; MD, mean deviation, Humphrey field analyser, H 30–2; MS, mean sensitivity, Octopus automated perimeter, G1X; WNL, within normal limits; AS, arcuate scotoma; NS, nasal step; SS, Seidel's scotoma; NA, not applicable; GR, general reduction of sensitivity.

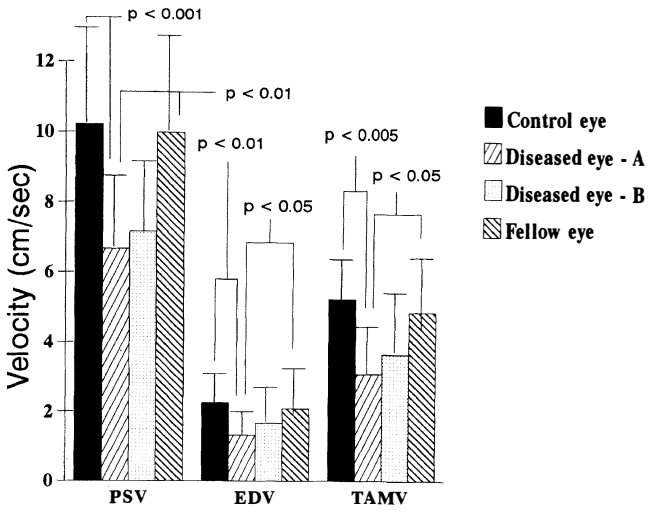


Fig. 1. *Peak systolic velocity (PSV), end-diastolic velocity (EDV) and time average maximal velocity (TAMV) of the central retinal artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

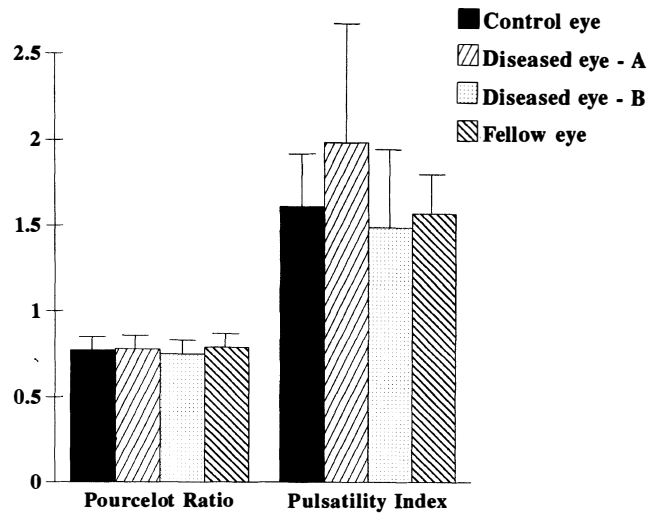


Fig. 2. *Pourcelot ratio and pulsatility index of the central retinal artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

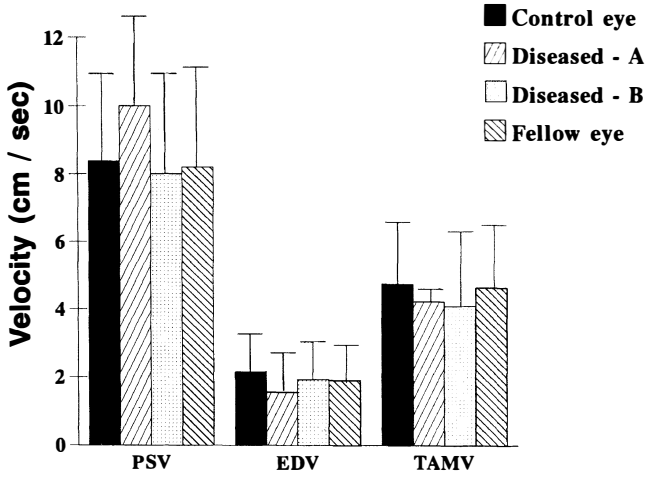


Fig. 3. *Peak systolic velocity (PSV), end-diastolic velocity (EDV) and time average maximal velocity (TAMV) of the temporal posterior ciliary artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

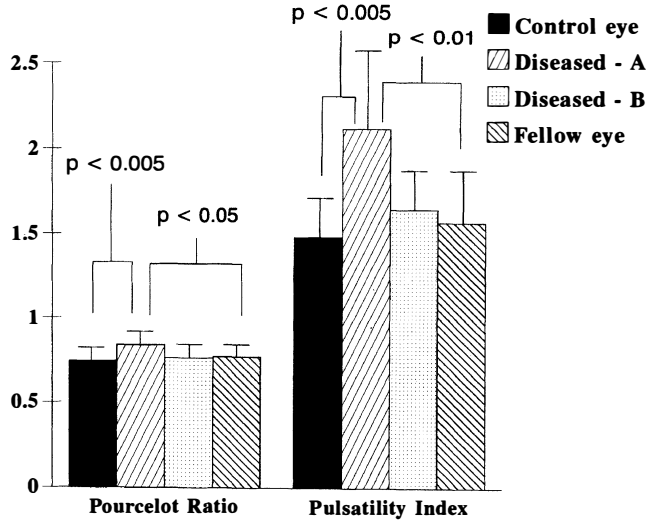


Fig. 4. *Pourcelot ratio and pulsatility index of the temporal posterior ciliary artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

Colour Doppler Imaging Findings

Comparison Between the Diseased Eyes with Elevated IOP and the Control Eyes. The eyes with elevated IOP showed decreased PSV ($p<0.001$), EDV ($p<0.01$) and TAMV ($p<0.005$) in the CRA (Figs. 1, 2). The nasal and temporal PCA showed significantly increased PR (both $p<0.005$) and PI (both $p<0.005$) in eyes with elevated IOP (Figs. 3–6). The ophthalmic artery showed no significant difference in any CDI parameter between these two groups (Figs. 7, 8).

Comparison Between the Diseased Eyes with Elevated IOP and the Clinically Healthy Fellow Eyes. The eyes with elevated IOP showed decreased PSV ($p<0.01$), EDV ($p<0.05$) and TAMV ($p<0.05$) in the CRA (Figs. 1, 2). The diseased eyes also showed an increase in PR (both $p<0.05$) and PI ($p<0.01, p<0.05$, respectively) of the nasal and temporal PCA. The PSV of the nasal PCA increased in eyes with elevated IOP ($p = 0.0469$), but the p value did not reach significance when the Bonferroni correction was used (Figs. 3–6). Comparison of each parameter of the ophthalmic artery found no significant difference between these two groups (Figs. 7, 8).

Comparison of the Results from the Diseased Eyes on Occasions A and B. The PR and PI of the nasal PCA were significantly reduced (both $p<0.05$) on occasion

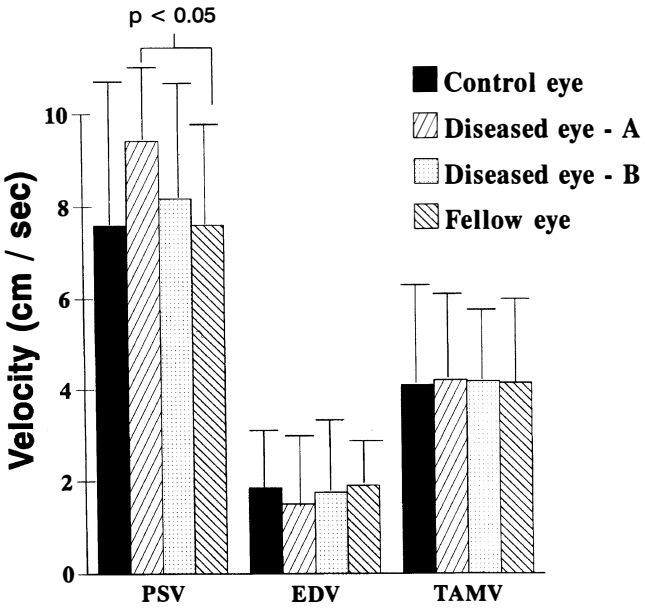


Fig. 5. *Peak systolic velocity (PSV), end-diastolic velocity (EDV) and time average maximal velocity (TAMV) of the nasal posterior ciliary artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

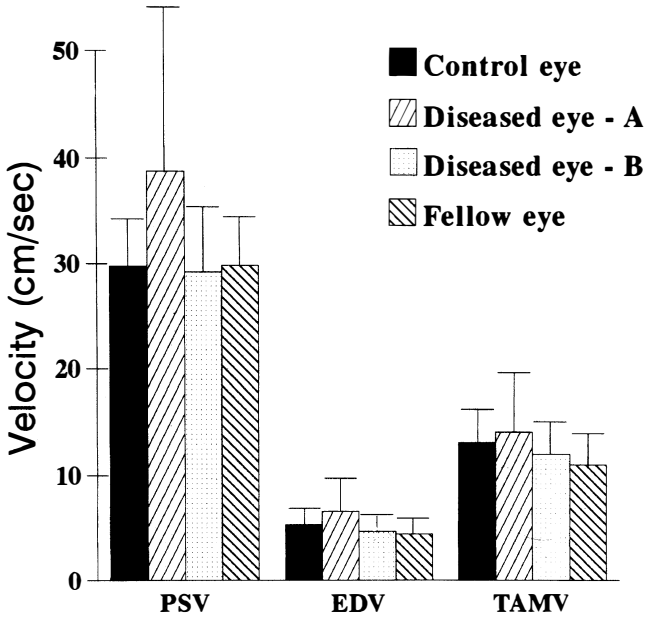


Fig. 7 *Peak systolic velocity (PSV), end-diastolic velocity (EDV) and time average maximal velocity (TAMV) of the ophthalmic artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

B. The resistance indices of the temporal PCA tended to reduce on occasion B, but the difference was not of statistical significance (PR: $p = 0.066$, PI: $p = 0.193$) (Figs. 3–6). None of the parameters in the CRA and ophthalmic artery showed a significant difference between occasion A and occasion B (Figs. 1, 2, 7, 8).

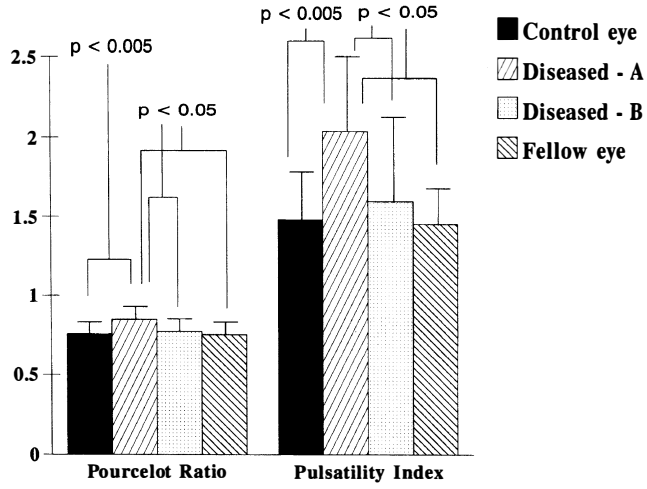


Fig. 6. *Pourcelot ratio and pulsatility index of the nasal posterior ciliary artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

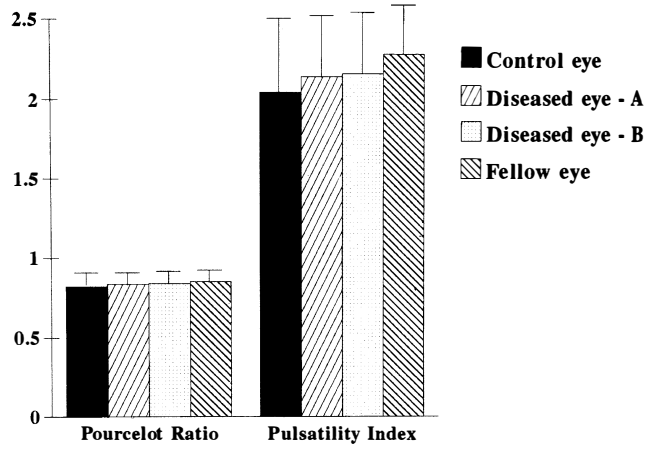


Fig. 8. *Pourcelot ratio and pulsatility index of the ophthalmic artery of the diseased eyes (A, occasion A; B, occasion B), clinically healthy fellow eyes and control eyes.*

No significant difference in CDI parameters was identified between the fellow eyes and the control eyes, or between occasions A and B in the fellow eyes.

DISCUSSION

This study demonstrated that eyes with spontaneously elevated IOP showed significantly decreased blood velocities in the CRA and significantly increased vascular resistance indices in the nasal and temporal PCA. Following IOP-lowering procedures, the resistance indices of the nasal PCA decreased significantly. The blood velocity in the CRA increased and the resistance indices of the temporal PCA decreased with a reduction in IOP, but the change did not reach statistical significance.

Previous studies have demonstrated marked stagnation of fluorescein dye in the retinal and choroidal

vasculature during induced acute ocular hypertension in normal subjects.[7,18] Spaeth[19] has shown a significant correlation between levels of IOP and circulation time in glaucoma suspects and patients with primary open-angle glaucoma. By means of Doppler ultrasonography, Guthoff *et al.*[20] have found that the systolic, mid-diastolic and end-diastolic velocities in the CRA progressively diminish in response to artificially elevated IOP. It is well recognised that human retinal circulation can autoregulate to maintain constant blood flow in spite of changes in perfusion pressure.[8,9,21] Yet, by using the blue field entopic phenomenon and laser Doppler velocimetry, autoregulation is shown to be fully effective only if IOP is not elevated above 30 mmHg.[8,9] Furthermore, there is evidence suggesting an impairment in the autoregulation of the retinal blood flow in glaucoma.[9] Therefore, our findings of a significant decrease in blood velocity in the CRA of eyes with spontaneously elevated IOP as compared with the control and the fellow eyes, respectively, are in accordance with the results of previous studies and may implicate a decreased blood flow in retinal circulation with elevated IOP.

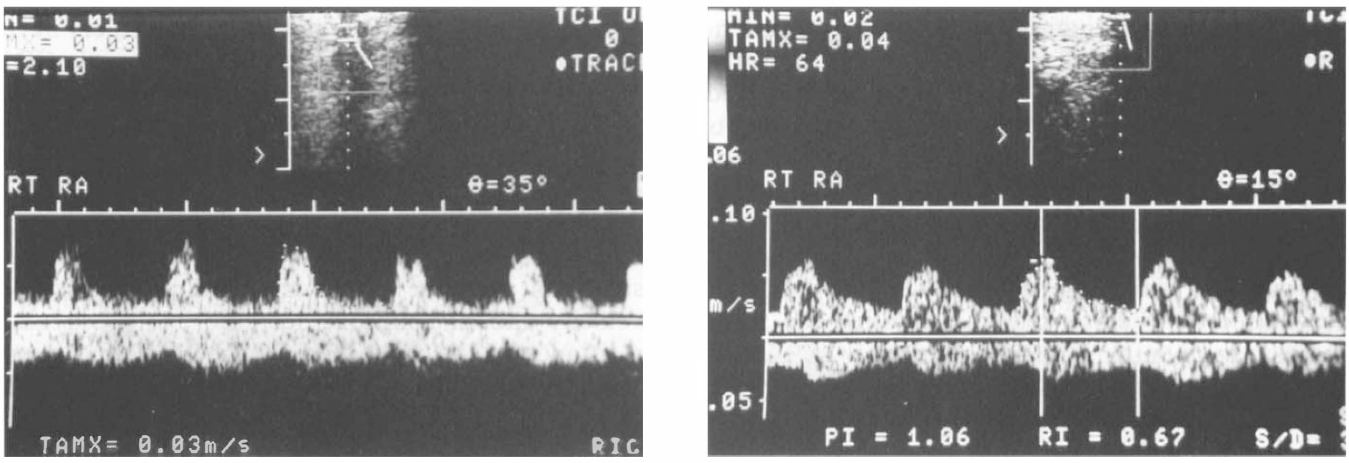
Decreased blood velocity could result from decreased blood pressure, increased cross-sectional area of a vessel, increased vascular bed resistance, or increased blood viscosity. Though there are reports concerning increased blood viscosity in glaucoma,[22,23] increased viscosity could not have been a significant factor in the present study since blood velocity in the fellow eyes did not differ much from that in the control. The CRA may have dilated in response to the relative ischaemia of the retina, yet the thick collagenous adventitia surrounding the artery in the optic nerve reduces the chance of vasodilatation. Could decreased blood pressure proximal to our measuring point be responsible for the change in velocity? It would be difficult to explain and also seems unlikely. The most reasonable explanation would be an increased downstream impedance with decreased blood velocity that accompanied an elevated IOP. Increased resistance in the vascular bed usually reduces diastolic blood flow more markedly than the systolic component, which is expressed as increased vascular resistance indices. We did find higher PR and PI of the CRA in eyes with elevated IOP than in the control and fellow eyes, but the difference was not of statistical significance (BMDP/Solo power analysis for eyes with high IOP vs the control: PR, power = 0.058; PI, power = 0.217). If a larger number of cases were studied, a significant difference might be identified.

The mean age of the study group was significantly smaller than that of the control group. Some authors have demonstrated that flow velocities decrease and resistance indices increase as a function of age in the ophthalmic artery, CRA and PCA;[20,24] others have shown little correlation between age and CDI parameters in the orbital vessels.[25,26] In this study, it seems unlikely that age is a factor that causes decreased flow velocities in the CRA of eyes with elevated IOP.

Trible *et al.*[15] have determined the colour Doppler haemodynamic changes after trabeculectomy. A significant increase in the mean and end-diastolic velocity and a significant decrease in the vascular resistance of the CRA and PCA were found postoperatively. In our study, the blood velocities and the resistance indices of the CRA showed similar changes with the reduction of IOP, but the difference was not of statistical significance (power for PSV: 0.065, EDV: 0.114, TAMV: 0.078, PR: 0.169, PI: 0.439). It is of note that in the study by Trible *et al.*[15] there were patients who achieved good IOP reduction without a significant change in velocity or resistance index. We found that the flow velocity waveform improved significantly following a reduction in the IOP in patients with good neuroretinal rim (Fig. 9). On the contrary, the velocity waveform did not improve significantly after surgery in patients with advanced disc damage (Fig. 10). Patients with varying degrees of nerve damage were included in this study, and this may be the reason why we found little difference in various CDI parameters in the CRA between occasion A and occasion B. In the study by Grunwald *et al.*[9] 67% of glaucoma patients described a difference in baseline leucocyte speed between eyes, 85% of them observing a slower leucocyte speed in the eye with more advanced glaucomatous damage. Furthermore, Sponsel *et al.*[27] have quantitatively confirmed the association between asymmetric leucocyte velocity and visual function loss in glaucoma patients. It is likely that blood velocity of the retinal circulation is slower in eyes with more advanced glaucomatous damage than in eyes with mild or moderate damage, even when the IOP is well controlled.

Hayreh[1] has demonstrated the importance of the ciliary circulation to the optic nerve, and its susceptibility to a raised IOP. In the present study, the nasal and temporal PCA showed significantly increased PR and PI in eyes with elevated IOP. Most of the blood flow from the PCA drains into the choroid. Compared with the retinal vasculature, the choroidal vessels are larger in diameter than the retinal vessels of similar thickness, and the intracapillary pressure is lower because the ciliary arteries open into a capillary bed of very large volume.[28] Both factors make the choroidal vasculature more vulnerable to compression by elevated IOP, and its flow more easily retarded by a rise in IOP.[28,29]

One concern about the PCA is that their vascular courses are tortuous, which makes it difficult to angle



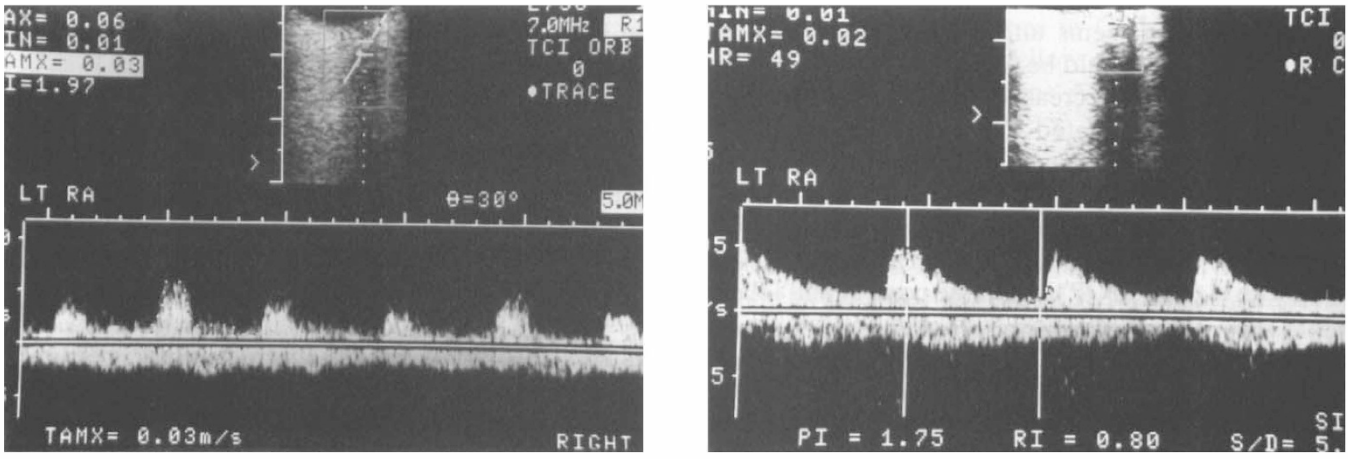
(a) (b)

Fig. 9. *A case of secondary glaucoma with angle recession and an optic cup/disc ratio of 0.4. The presenting IOP was 62 mmHg (occasion A), which reduced to 11 mmHg after trabeculectomy (occasion B). (a) Flow velocity waveform of the central retinal artery on occasion A. (b) Velocity waveform of the same artery on occasion B.*

the ultrasound beam within them accurately. Since the resistance indices are a ratio of velocities and are angle independent, they are supposed to be more reliable parameters than flow velocities within the PCA. With the current imaging technology of CDI it would be impossible to distinguish among the ciliary arteries, thus casting doubt on the reproducibility of the results. Some authors have pointed out that CDI measures in the PCA are more variable than those in the ophthalmic artery and CRA, suggesting that current methods for assessing these vessels may not be sufficiently reliable.[30–32]

Following a dramatic reduction in IOP, the resistance indices decreased in the nasal and temporal PCA, but only the difference in the nasal PCA attained statistical significance. This finding might imply that the temporal PCA are more vulnerable to compression by a rise in IOP than the nasal PCA, and that permanent change may occur in ciliary circulation. Whether the increased resistance to pressure damage of the nasal PCA is associated with the clinical observation of a residual temporal island in eyes with advanced glaucomatous optic neuropathy needs further investigation. Prior study has shown greater absence of ink filling in the capillaries of the temporal optic nerve than in those of the nasal side with raised IOP.[4] Rosen and Boyd[28] have demonstrated that the delay in circumpapillary choroidal perfusion might exist even at very low IOP in eyes with deep cupping. In Laatikainen and Mantyla's study[33] fluorescein filling defects of the peripapillary choroid or disc were not clearly influenced by a decreased IOP level, indicating the presence of permanent anatomical change in the peripapillary choroidal vasculature.

Both the Pourcelot ratio and the pulsatility index



(a) (b)

Fig. 10. *A case of primary angle-closure glaucoma with total cupping. The presenting IOP was 78 mmHg (occasion A), which reduced to 11 mmHg after trabeculectomy (occasion B). (a) Flow velocity waveform of the central retinal artery on occasion A. (b) Velocity waveform of the same artery on occasion B.*

have been widely used in the clinical setting to quantify the relative pulsatility of the velocity waveform. Adamson *et al.*[34] found an excellent linear correlation between downstream microvascular resistance and these two indices, respectively. Opinions differ concerning which is the better index. There is evidence that PI may not be normally distributed, which would invalidate some statistical tests,[35] and Thompson *et al.* concluded that it did not show any benefit over the PR. On the other hand, Pearce *et al.*[36] claimed that PI was preferable because PR cannot be used when diastolic velocity is equal to or less than zero. In general, PR is suitable for a low-resistance vascular bed, such as the ocular circulation. PI, which is for a higher resistance system, was also measured in this study because initially we were not sure of the impact of elevated IOP on ocular vascular resistance. Since the results showed that both PR and PI gave similar information, PR alone may serve well in further studies of this kind.

In the present study, eyes with varying aetiologies and degrees of glaucomatous optic neuropathy were included because our aim was to investigate haemodynamic changes associated with spontaneously elevated IOP and the reversibility of such changes following IOP-lowering procedures. The influence of glaucoma medications was deliberately avoided, so only patients who were free from medications were included in the study group. Unlike most studies in which circulation was evaluated in eyes with artificially elevated IOP or in eyes with chronic open angle glaucoma, there were 9 cases of primary angle-closure glaucoma (3/4) in our report. Primary open angle and angle-closure glaucoma differ in the mechanism that causes elevated IOP, yet they may share something in common as to the effects of IOP on ocular haemodynamics, with a final common pathway leading to nerve damage. We are aware that eyes with acute elevation in IOP may respond differently from eyes with long-term elevation, but the limited number of eyes in the study group prevented us from making a comparison.

In conclusion, we have demonstrated the haemodynamic changes associated with spontaneously elevated IOP by the use of CDI, i.e. decreased flow velocities in the CRA and increased resistance indices of the PCA. After the IOP had been reduced, the resistance indices of the nasal PCA decreased significantly. Whether a reduction in IOP may reverse the haemodynamic changes associated with ocular hypertension might be related to the degree of optic nerve damage, as well as the level of IOP obtained. Further improvement in CDI technology would be desirable to facilitate our understanding of the optic nerve circulation in glaucoma.

Key words: Circulation, Colour Doppler imaging, Glaucoma, Haemodynamics, Intraocular pressure, Ultrasound.

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