BRITISH JOURNAL OF OPHTHALMOLOGY

Editorial

The vascular factor in low tension glaucoma: alchemists' gold?

The search for evidence of the elusive vascular factor in the pathogenesis of low tension glaucoma continues. In this issue Usui and Iwata report their investigation of alteration in finger blood flow induced by temperature change. They used the same laser-Doppler flow technique as others,^{1,2} but have not confirmed the reduced blood flow characteristics identified in these two papers. What are we to make of it? The differences may reflect the different racial characteristics of the subjects in the various studies but much other work on low tension glaucoma produces conflicting evidence, and it is more likely we are trying to produce results with investigative tools which are too crude for the job. Even the definition of low tension glaucoma (or normal pressure glaucoma) is imprecise. It usually depends on the absence of a raised intraocular pressure and the exclusion of other causes of optic nerve or retinal disease. This negative description cannot be satisfactory. Was there raised pressure in the past or are there continuing undetected fluctuations?³ The difficulty of definition applies equally to primary open angle glaucoma with raised pressure the prevalence of which may vary from 0.4% to 11.2% depending on the diagnostic criteria.⁴

Tests other than finger blood flow have been used in the attempt to identify a systemic vascular abnormality specific to low tension glaucoma. Study of blood coagulation and rheology has proved fruitless.⁵ The same authors however claim that multivariate analysis of finger blood flow and haematological factors can distinguish separate groups in both low pressure and high pressure glaucoma.⁶ Those patients in whom highest intraocular pressure correlated with severity of field loss had vasospastic finger blood flow measurements and those with no correlation between highest pressure and field loss had disturbed coagulation and biochemical factors. This study depended on impenetrable statistical analysis and even if mathematically sound its biological and clinical significance remains doubtful as the authors themselves point out. The postulated association of low tension glaucoma with carotid stenosis has not been confirmed.⁷ An association between migraine and low tension glaucoma has been found by some workers⁸ but not by others.⁹ Low blood pressure has been identified as a risk factor for progressive visual field loss in elderly glaucoma patients^{10,11} and in low tension glaucoma.¹⁰ Yet others find the opposite with higher levels of blood pressure carrying the risk.¹²

Attempts to measure optic nerve blood flow have fared no better yielding only unsatisfactory and contradictory results.¹³ However the optic nerve head in low tension

glaucoma may be more vulnerable to damage at a lower level of intraocular pressure. There is some evidence to support this view¹⁴ but further studies are required. More convincing support for the concept of low tension glaucoma as a distinct entity comes from visual function studies which suggest that the pattern of field defect varies from high pressure¹⁵⁻¹⁷ glaucoma. Differences in colour vision loss have also been described.¹⁸

The theory that low tension glaucoma has a specific vascular aetiology remains unproved but there seems to be something in it. We must not let this concept divert attention from the overwhelming evidence now available that raised intraocular pressure, overt, concealed, constant, or intermittent is the greatest risk factor for field loss in primary open angle glaucoma.

J L JAY

- Drance SM, Douglas GR, Wijsman K, Shulzer M, Britton RJ. Response of blood flow to warm and cold in normal and low tension glaucoma patients. *Am J Ophthalmol* 1988; 105: 35-9.
- Gasser P, Flammer J. Blood cell velocity in the nailfold capillaries of patients with normal tension and high tension glaucoma. *Am J Ophthalmol* 1991; 111: 585-8.
- Zeimer RC, Wilensky JT, Gieser DK, Viana MAG. Association between intraocular pressure peaks and progression of visual field loss. *Ophthalmology* 1991; 98: 64-9.
- Kahn HA, Milton RC. Alternative definitions of open angle glaucoma. Effect on prevalence and associations in the Framingham eye study. *Arch Ophthalmol* 1980; 98: 2172-7.
- Carter CJ, Brooks DE, Doyle DL, Drance SM. Investigations into a vascular aetiology for low tension glaucoma. *Ophthalmology* 1990; 97: 49-55.
- Schulzer M, Drance SM, Carter SJ, Brooks DE, Douglas GR, Lau W. Biostatistical evidence for two distinct chronic open angle glaucoma populations. *Br J Ophthalmol* 1990; 74: 196-200.
- Pillunat LE, Stodtmeister R. Inzidenz des neiderdruckglaukoms bei hemodynamisch relevanter karotisstenose. *Spektrum Augenheilkd* 1988; 2: 24-7.
- Phelps D, Corbett JJ. Migraine and low tension glaucoma. A case control study. *Invest Ophthalmol Vis Sci* 1985; 26: 1105-8.
- Usui T, Iwata K, Shirakashi M, Abe H. Prevalence of migraine in low tension glaucoma and primary open angle glaucoma. *Br J Ophthalmol* 1991; 75: 224-6.
- Perasalo R, Raitta C. Low blood pressure. A risk factor for nerve fibre loss in institutionalised glaucoma patients. *Acta Ophthalmol (Kbl)* 1990; 68: 65-7.
- Richler M, Werner EB, Thomas D. Risk factors for progression of visual field defects in medically treated patients with glaucoma. *Can J Ophthalmol* 1982; 17: 245-8.
- Rouhiainen HJ, Terasvirta ME. Haemodynamic variables in progressive and non progressive low tension glaucoma. *Acta Ophthalmol (Kbl)* 1990; 68: 34-6.
- Glazer LC. Methods for determination of optic nerve blood flow. *Yale J Biol Med* 1988; 61: 51-60.
- Pillunat LE, Stodtmeister R, Wilmanns I. Pressure compliance of the optic nerve head in low tension glaucoma. *Br J Ophthalmol* 1987; 71: 181-7.
- King D, Drance SM, Douglas G, Schulzer M, Wijsman K. Comparison of visual field defects in normal tension glaucoma and high tension glaucoma. *Am J Ophthalmol* 1986; 101: 204-7.
- Glicklich RE, Steinmann WC, Spaeth GL. Visual field change in low tension glaucoma over a five year follow up. *Ophthalmology* 1989; 96: 316-20.
- Chauhan BC, Drance SM, Douglas GR, Johnson CA. Visual field damage in normal tension and high tension glaucoma. *Am J Ophthalmol* 1989; 108: 636-42.
- Yamazaki Y, Lakowski R, Drance SM. A comparison of the blue colour mechanism in high and low tension glaucoma. *Ophthalmology* 1989; 96: 12-5.