Pressure and posture

The paper by Yamabayashi and others in this month’s issue explores once again the relationship between intraocular pressure (IOP), blood pressure (BP), and posture.

According to Jain and Marmion, the postural IOP rise in normal individuals seems to be slightly less than 10% of the sitting IOP. Jain and Marmion also give a useful league table of rises recorded by other authors between 1963 and 1976. The average rise appears to be about 3.22 (higher than that recorded by Jain and Marmion) with a range between 1.40 and 5.00.

In 1978 Williams and Peart, studying cases of branch and central retinal venous obstruction (their term), discovered that whereas the cases had rises of this order (actually 3-10 mm Hg for tributary and 1-8 for central veins) the normal controls only had rises of 0-60 mm Hg, considerably less than those collected by other authors. Both the tributary and central vein cases had moderate vascular hypertension compared with the normal controls. It is of interest that in the cases where the rises were of approximately similar magnitude in each eye regardless of which had the venous obstruction, in fact in some the rise was greater on the unaffected side. This suggests that a systemic factor was responsible for the enhanced rise rather than a local event in the eye, such as the retinal venous obstruction.

It would be natural to assume that the difference was attributable directly to the difference in BP between the two groups but the authors seem to have rejected this, preferring the possible explanation for the difference between normals and controls as being due to a local ocular abnormality in both the affected and the fellow eye. Co-existing glaucoma was the obvious possibility because at the time it was widely believed that pre-existing glaucoma and retinal venous occlusions were associated. However, the association of glaucoma with venous occlusion is now known not to be as straightforward as was once believed and indeed Cole and colleagues showed that the principal medical irregularities which might reasonably be expected to be associated with retinal venous accidents (hypertension and hyperlipidaemia) were just as common in venous occlusion cases with glaucoma as in those without.

One has to ask the question, therefore, why did Williams and Peart reject the BP differences as explaining the postural IOP differences? One reason seems to have been inability to correlate postural IOP changes with postural BP changes (as distinct from the very significant differences in sitting BP between the groups). Another reason is the presence of similar anomalies in a further study of diabetics’ where like differences were found and were thought not to be correlated with hypertension (although it has to be said that eight of the 14 diabetic patients were hypertensive).

The current paper by Yamabayashi and colleagues is not entirely comparable with the previous papers of Williams and colleagues since the earlier papers deal with differences between normals and patients with vascular hypertension whereas the current paper is concerned with possible differences between normals and ocular hypertensives and low-tension glaucomas. The Yamabayashi paper is interesting in that very little difference was found between the various groups, the normals having rises of the same magnitude (4.4 mm Hg) as the ocular hypertensives or low tension glaucomas. There was admittedly a further rise in the ocular hypertensives after 30 minutes but it was of relatively low magnitude (1.60 mm Hg).

The chief difference between the present paper and those of Williams and colleagues is in the results for the normals. It is not easy to understand why one group of workers should have found such a small rise in normals (0.60 mm Hg repeated in two separate studies) compared with the substantial rise in normals reported in the present paper, but it is perhaps rather characteristic of this particular type of study. There have been a considerable number of fairly similar studies linking posture and IOP, most of which are provided as references in the current paper, but having looked at most of them one is left with rather a vague impression as to how to interpret the results. Apart from the discrepancy between the controls just quoted there is another discrepancy concerning low tension glaucoma in which Yamabayashi et al point out that their results, in which such cases have similar rises to ocular hypertensives, are at variance with previous work where low tension glaucomas seemed most susceptible to postural pressure elevation.

One is left with the impression that although the postural changes are of interest and could lead eventually to a better understanding of the mechanisms of glaucoma, there is at least no real consensus as to exactly what happens to the ocular dynamics on lying down. It seems most likely to me that the venous pressure is something which should perhaps be studied more closely. What happens to it in vascular hypertension for example? Does the venous pressure behave in the same way in vascular hypertensives and controls on lying down, and how can the venous pressure be monitored? One is uncomfortably conscious, as an ordinary ophthalmologist, of a marked personal inadequacy in understanding the niceties of vascular dynamics, quite apart from ocular dynamics, and one looks forward to more collaborative work between ophthalmologists and general physicians in this difficult area.

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