Retrobulbar pressures in dysthyroid optic neuropathy

In their article entitled 'Retrobulbar pressures measured during surgical decompression of the orbit', the team from the Netherlands has produced a paper (p 1042) that is interesting as much for the questions it asks as for the answers it proposes. By direct measurement of orbital pressures in patients with Graves' orbitopathy, they have shown a raised retrobulbar pressure with dysthyroid optic neuropathy (DON) and a decrease in this pressure of 8–12 mm Hg with orbital decompression. They therefore postulate that nerve dysfunction may not be exclusively the result of direct swelling of the extraocular muscles on the optic nerve but an effect of raised retrobulbar pressure on the optic nerve. In many ways, this is an attractive concept which helps to explain other features of DON, such as the dilated superior ophthalmic vein (and associated congestion which disappears rapidly after decompression) and the dilated retrobulbar optic nerve sheath.1 This work supports the concept of an orbital compartment syndrome described by Kratky et al and Hurwitz et al.2 3 It is also useful to explain the proposal of Trokel et al4 that anterior fat excision alone may reverse DON.5 Further experiments along this line would be useful and could include evaluation of the size of the drop in orbital pressure following fat excision alone and other decompressions, as well as indirect evidence of decrease in size of the superior ophthalmic vein and retrobulbar optic nerve sheath following decompression. Another feature that may be worth assessing is whether or not pressure correlates with the number of walls decompressed at the time of surgery.

As an aside, the authors note a dramatic rise in intra-orbital pressure brought about by compression of orbital structures with instrumentation at the time of surgery, a feature that cautions care during retrobulbar procedures.

Their concept may not explain the small, tight orbit syndrome associated with optic neuropathy, which appears to result primarily from apical compression by swollen extraocular muscles in a disproportionately small orbital apex as is noted in some patients, particularly Asians. It also does not explain cases of persistent optic neuropathy following adequate release of orbital pressure. These cases appear to be the result of persistence of apical bone and require a secondary procedure to relieve the direct pressure brought about by the disproportion between soft tissue and bone.

What is harder to understand is their concept of fat protrusion into the optic canal as a cause of dysthyroid optic neuropathy. For anyone who has dissected the orbital apex, either in a cadaver or in the operating room, it is hard to imagine protrusion of fat through the very tight adherence of the annulus of Zinn to the apical optic nerve. The tissues here are absolutely intimate. This would suggest that their concept of direct orbital pressure comes into play just anterior to the annulus. Further, the dense retinacular network of the orbit described by Koornneef increases apically, which would tend to localize fat and not allow for much movement of orbital contents posteriorly.

Their line of work is most interesting and suggests that non-invasive methods of direct orbital pressure measurement would be extremely useful in defining the critical point in orbital pressure that leads to optic neuropathy. It would also help to explain differences in effect on optic nerves of acute versus chronic orbital pressure, where down regulation may be a factor.6 The fact that orbital volume can show signs of having increased with contouring of the orbital wall brought about by volume and pressure changes is another feature that suggests raised orbital pressure is of significance on a chronic basis in DON, and with Graves' orbitopathy in general.

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