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## *Editorial*

# The ocular pulse

The early ophthalmoscopists overcame the deficiencies of their instrument by employing highly tuned powers of observation inspired by the knowledge that they trod where no man had trod before. To us more pedestrian 20th century ophthalmic observers they seem to have recorded signs which are difficult to identify even with our modern halogen powered equipment. Among the smaller signs detected was the spontaneous pulsation of the retinal arteries. Duke-Elder, in summarising the work of these early pioneers, noted that both Donders (1852) and Becker (1872) drew our attention to these pulsations.[1]

The amplitude of this arterial pulse is dampened by the lack of distensibility of the ocular coats. Unless there was an area of weakness in the coats of the eye, such as a posterior staphyloma or a nearly perforated corneal ulcer,[1] the arterial pulse was transformed into a volume pulse of such low amplitude that it was invisible to even the 19th century ophthalmologists.

The widespread adoption of applanation tonometry, by allowing the routine observation of pulsating mires at the slit-lamp, highlighted the existence of such a volume pulse and its variations in physiological and pathological states. It is well recognised that a reduction in movement of the mires may occur in myopia, ocular disease, or carotid insufficiency.

Quantification of this ocular pulse became possible when Perkins modified an applanation prism by replacing the contact surface with a distensible film. This film allowed continuous transmission of the ocular pulse to a pressure transducer, which in turn generated a pulse trace.[2] He was able to define the normal pulse amplitude as >1·5 mmHg and showed that an interocular difference in pulse amplitude rarely exceeded 0·5 mmHg. Using these data he identified carotid insufficiency from dissimilar or reduced pulse amplitudes.[2] He also noted that his device could provide a reasonably accurate IOP measurement as well.

Langham adapted the pneumotonometer for a similar purpose, using it to provide a wave form of the ocular pulse. By analysing this wave form, its amplitude, and the heart rate he was able to derive values for the pulsatile component of blood flow.[3] It needs to be remembered that non-pulsatile blood flow, as revealed by Doppler ultrasound studies of the ophthalmic artery,[4] has not been estimated by this technique.

The versatility of the pneumotonometer, allowing measurements of the IOP to be made while the subject varied his posture, has allowed Trew and Smith to re-evaluate the relationship of changes in IOP to changes in posture, as well as assess pulsatile ocular blood flow. In two papers in this issue the authors confirm that, while the assumption of a supine posture is followed by an increase in IOP in both normal persons and glaucoma patients, the rise was far greater in the latter group. They also report that this IOP change was associated with a fall in pulsatile ocular blood flow, which was not improved despite a fall in IOP with topical timolol therapy.

As the increase in IOP seen on adopting the supine position is greatest in glaucoma patients, it could be suggested that a compromised ocular circulation would be put at further risk by any additional increase in IOP, as well as by a possible fall in the systemic blood pressure in the sleeping, treated hypertensive patient. Indeed that idea has been put forward as a major cause of progressive visual loss in low tension glaucoma. The authors suggest that these pressure changes are associated with a reduction in pulsatile ocular flow in patients with primary open-angle glaucoma on adopting the supine position and that this reduction is not reversed despite lowering of IOP with topical β blockers.

What is the significance of these findings for the practising ophthalmologist? Firstly, glaucoma patients have an exaggeration in the posture induced IOP increase, which may be associated with postural hypotension in the treated hypertensive patient, and this might reduce ocular flow during sleep. However, it has yet to be demonstrated that glaucoma patients are more liable to have suffered episodes of visual loss during sleep (in contrast to the timing of episodes of ischaemic optic neuropathy). There is therefore no proved need to suppress this IOP increase, though, perhaps by a nightly acetazolamide tablet it may help the occasional patient.

Secondly, what is the relevance of pulsatile ocular flow to the circulation at the optic nerve in glaucomatous eyes? It must be remembered that >90% of ocular blood flow is taken by the choroidal circulation and that the optic nerve circulation accounts for a minute fraction. Any recorded changes can reflect changes only in the former. This casts doubt on attempts to relate total flow changes to optic nerve disease. Unless it can be shown that changes in the former mirror or induce changes in the latter, interpretation of treatment-induced modifications in flow must be treated with caution.

R HITCHINGS

1 Duke-Elder S. The development form and function of the visual apparatus. *Textbook of ophthalmology*. St Louis, Mosby, 1944; 1: 398–9.
2 Perkins ES. The ocular pulse and intraocular pressure as a screening test for carotid artery stenosis. *Br J Ophthalmol* 1985; 69: 676–80.
3 Langham ME, Farell MA, O'Brien V, Silver DM, Schilder P. Blood flow in the human eye. *Acta Ophthalmol (KGh)* 1989; 67 (suppl 191): 9–12.
4 Canning C, Restori M. Doppler ultrasound studies of the ophthalmic artery. *Eye* 1988; 2: 92–5.