The ocular pulse

Sir,—The important editorial on the ocular pulse,1 highlights the observations of the retinal arterial pulsations noted by early ophthalmologists. It was Thiel2 who first attempted to quantify the recording of the tonometric waves which had become known as the tonometric sign as early as 1879. Priestley Smith3 had clearly described the spontaneous vascular pulsations observed during tonometry. This was reinforced by the observations of Schiatt and because an important observation for those undertaking tonometry up to the time of the application method.

The earliest precise recording of the ocular pulse was that of Thiel in 1928.4 Subsequently Maurice5 used a recording tonometer, to be followed by Castren and Lavikken,1 who adapted a Muller electrotonometer. Following the work of Suzuki6 a number of groups developed a system of recording the ocular pulse using a fluid filled suction cup system. The piezoelectric system of Byrke7 seems to be the least 'invasive' and most precise. The method of Barnes and Perkins8 was developed for clinical use. In spite of the variety of approaches the amplitude and variation in pulse pressures correlate quite closely. Both in time and form the pulse wave does not correspond to a typical arterial pulse. Pulsations are seen in the dicrotic notch which is the characteristic of an arterial pulse, and when placed in time sequence in relation to the R-wave of the ECG, or the Doppler wave recruited from an adjacent orbital vessel, for example, the supra trochlear vessel, its time relationship is closer to the middle of diastole. The only report which demonstrates a dicrotic notch is that of Barnes and co-workers,9 who applied the pulse sensor, externally, through the lids, and it is believed that this was recording ophthalmic artery pressure pulsations and not an ocular pulse.

While evidence to link the ocular pulse amplitude to either C or POC is lacking, there is a significant amount of evidence from all studies that it has a direct relationship with levels of ipsilateral carotid perfusion. The consequence of this is to question the role of a topically B-blocking which does not contain intrinsic sympathomimetic activity (ISA).

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