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The ocular pulse and intraocular pressure as a screening test for carotid artery stenosis

EDWARD S PERKINS

From the Department of Ophthalmology, University of Iowa Hospitals and Clinics, USA

SUMMARY The ocular pulse and applanation tension were measured with a recording applanation tonometer in 38 patients suspected of having internal carotid artery stenosis. Abnormalities of the ocular pulse amplitude, intraocular pressure, or combinations of these two measurements were found in 23 (82%) of 28 patients who were subsequently found to have angiographic evidence of 50% or more stenosis of one or both internal carotid arteries. Of 10 patients without angiographic evidence of carotid stenosis the ocular pulse amplitude, intraocular pressure, or both were abnormal in five, but two patients had an ocular cause for abnormality and two of the remaining three had evidence of carotid disease in the form of atheromatous plaques. These results suggest that measurement of the amplitude of the ocular pulse in addition to the intraocular pressure can predict the presence of carotid artery stenosis and indicate the need for further investigation. If a tonometer capable of measuring the pulse amplitude was used routinely in ophthalmic examinations, it would provide a useful screening test for the early diagnosis and treatment of carotid artery disease and might thereby reduce the frequency of strokes.

Carotid artery disease is an important cause of strokes, and, if the condition could be recognised before symptoms arise, surgical treatment might be effective in reducing the incidence of death and disability from strokes. It is not surprising, therefore, that many non-invasive screening tests for carotid disease have been devised to detect patients requiring angiography. Such tests, however, are applied only when the patient presents with symptoms suggestive of vascular insufficiency.

Most people over the age of 40 require glasses for near work and are likely to attend an ophthalmologist or optometrist at regular intervals for a refraction, so that a screening test which was part of a routine ophthalmic examination could be applied on a large scale with little extra effort or cost. One such test is the measurement of the ocular pulse and the comparison of the pulse amplitude in the two eyes. Ophthalmologists and many optometrists routinely measure the intraocular pressure as a screening test for glaucoma, and some tonometers can indicate the ocular pulse amplitude, though the value of this measurement is rarely recognised, and only the mean

level of pressure is normally recorded. As the amplitude of the ocular pulse reflects the volume of blood entering the eye with each cardiac pulse wave, it is dependent in part on the integrity of the arterial supply to the eye, and stenosis of the internal carotid would be expected to result in a reduced ocular pulse amplitude, and perhaps a low intraocular pressure.

The purpose of this paper is to compare the ocular pulse amplitudes and intraocular pressure with the results of carotid angiography in a series of patients suspected of having carotid artery disease.

Patients and methods

PATIENTS

The patients were derived from the Vascular Clinic of the Department of Ophthalmology and the Cardiovascular Department of the University of Iowa Hospitals and Clinics. They were suspected of having carotid artery disease and had had angiography of the internal carotid arteries (ICA). The ocular pulse studies were performed and the records analysed before the angiography results were known.

TONOMETRY AND OCULAR PULSE RECORDING

The ocular pulse amplitude was determined with a

Correspondence to Dr Edward S Perkins, C S O'Brien Library, Department of Ophthalmology, University of Iowa Hospitals and Clinics, Iowa City, Iowa 52242, USA.

Goldmann applanation tonometer modified to give continuous recording of the intraocular pressure on a Hewlett-Packard heated stylus recorder. The Goldmann prism had a small chamber 2 mm in diameter drilled into the face of the prism, and the opening was covered with a very thin mylar membrane. The chamber was filled with water through lateral openings and connected by a short length of tubing to a pressure transducer. The fluorescein rings were adjusted in the normal manner and the prism centred so that the opening of the chamber lay within the applanated area. Under these conditions the intraocular pressure was transmitted through the cornea and the membrane to the fluid in the chamber and was recorded by the pressure transducer.¹

CRITERIA OF ABNORMALITY OF THE OCULAR PULSE
The ocular pulse results from the influx of blood into the eye with each cardiac cycle, and the amplitude of the pulse wave (i.e., the change in intraocular pressure from diastole to systole) will depend on the pulse pressure and the adequacy of the blood supply to the eye, the volume of the choroidal vascular bed, and the relation between volume changes and pressure changes in the individual eye. In normal eyes the latter factor is probably the most important cause of interindividual variations. If a similar volume of blood enters the eye of normal subjects the resulting pressure change will be greater in small hypermetropic eyes than in large myopic eyes. A low pulse amplitude can therefore be due to myopia,^{2,3} and the level below which the pulse amplitude is abnormal has to be modified with respect to the refraction.

Previous studies on a presumably normal population showed that a pulse amplitude of 1.5 mmHg or less occurred in only three out of 82 patients with hypermetropia or emmetropia, and only two out of 27 myopes had amplitudes of less than 1 mmHg. The difference in pulse amplitude between the two eyes exceeded 0.5 mmHg in only 2%.

The criteria of an abnormal pulse based upon the above findings were: (1) a pulse amplitude of 1.5 mmHg or less for hypermetropic and emmetropic eyes; (2) a pulse amplitude of 1.0 mmHg or less for myopic eyes (> -1.00 D); (3) a difference between the pulse amplitudes of the two eyes of more than 0.5 mmHg.

In the same normal population an intraocular pressure of 10 mmHg or less was found in only 7.3% and a difference in pressure between the two eyes of 3.0 mmHg occurred in only 2.75%.

The criteria chosen for considering the intraocular pressure abnormal were: (1) an applanation reading of 10 mmHg or less in one or both eyes; (2) a difference of 3.0 mmHg or more between the two eyes.

Results

Thirty-eight patients who subsequently underwent carotid angiography were examined, and the results can be summarised as follows.

CASES WITH ABNORMAL OCULAR PULSE AMPLITUDES (Table 1a)

There were 26 cases with a low ocular pulse amplitude and/or an intraocular difference in amplitude of more than 0.5 mmHg. Twenty-one of these cases (81%) were found to have angiographic evidence of internal carotid artery stenosis in one or both arteries. Of the five cases with abnormal pulses but no stenosis three had atheromatous plaques in the internal carotid artery, including one patient (case 3) who had had an occlusion of the central retinal artery and one patient with amaurosis fugax (case 27). It is possible, therefore, that there was reduced flow in the arteries in these patients, though no stenosis was reported on angiography.

The two remaining cases with abnormal pulses but no stenosis consisted of one patient who was subsequently diagnosed as having low-tension glaucoma (case 14) and one patient with amaurosis fugax (case 41). Abnormally low pulse amplitudes are known to occur in some patients with low-tension glaucoma,⁴ and this may be the reason for the false positive results in this case. The remaining case with abnormal pulses but no stenosis gave a history of amaurosis fugax, which suggests an intermittent reduction of blood supply to the eye in spite of normal angiography.

CASES WITH NORMAL PULSE AMPLITUDE BUT ABNORMAL OCULAR TENSIONS (Table 1b)

There were two patients in this group who were both found to have stenosis of one or both ICAs.

CASES WITH NORMAL OCULAR PULSE AMPLITUDES AND NORMAL TENSIONS (Table 2)

There were 10 cases in this group and angiography showed no stenosis in five cases but stenosis in the other five. In three of the latter five there was a difference of 0.5 mmHg between the pulse amplitudes of the two eyes, but by the criteria used these must be included in the false negative rate of 50%.

The results in terms of positive and negative findings related to the angiographic reports are summarised in Table 3.

Discussion

Ophthalmodynamometry, ophthalmoplethysmography, photoplethysmography, and Doppler flow studies have all been used as non-invasive tests for carotid artery disease, and the results of different

Table 1a Cases with abnormal ocular pulse amplitudes

Case no.	Mean pulse amplitude mmHg	Difference in amplitude mmHg	Tensions mmHg	Difference in tensions	Angiographic findings	Other findings
3	OD 1.5 OS 1.5	0.0	OD 26 OS 17	9.0	Carotid plaques but no stenosis	OD central retinal artery occlusion, doppler abnormal
5	OD 0.5 OS 0.7	0.2	OD 18 OS 21	3.0	Total occlusion R ICA Almost total occlusion L ICA	
6	OD 0.25 OS 9.5	0.25	not recorded		Atheroma R ICA, no stenosis 75% stenosis L ICA	
7	OD <0.5 OS <0.5	0.0	OD 11 OS 11	0.0	Severe ulcerated plaques R ICA Total occlusion L ICA	
8	OD <0.5 OS 0.0	0.25	OD 9 OS 10	1.0	Complete occlusion both ICAs	
9	OD 0.5 OS 0.75	0.25	OD 11 OS 9	1.0	Severe stenosis both ICAs	
12	OD 2.25 OS 1.25	1.0	OD 10 OS 6	4.0	Total occlusion L ICA	
14	OD 0.75 OS 0.75	0.0	OD 15 OS 20	5.0	Minimal irregularity L ICA but no stenosis	History of amaurosis fugax; diagnosed low tension glaucoma
17	OD 0.75 OS 1.5	0.75	OD 28 OS 12	16.0	Complete occlusion R ICA 90% occlusion L ICA	Severe peripheral vascular disease; central retinal artery occlusion OD
18	OD 2.0 OS 0.75	1.25	OD 14 OS 20	6.0	Bilateral occlusion ICAs	
19	OD 0.5 OS 0.25	0.25	OD 16 OS 16	0.0	Minor irregularity R ICA Complete occlusion L ICA	Central retinal artery occlusion OS
20	OD 1.75 OS 1.25	0.25	OD 16 OS 16	0.0	Partial occlusion R ICA Complete occlusion L ICA	
21	OD 0.75 OS 0.75	0.0	OD 11 OS 8	3.0	Plaques R ICA Narrowing L common carotid and severe disease L ICA	
22	OD 0.75 OS 0.5	0.25	OD 13 OS 11	2.0	Plaques both ICAs but no stenosis	
25	OD 3.5 OS 2.0	1.5	OD 15 OS 13	2.0	Occlusion of L ICA	
26	OD 2.25 OS 1.25	1.0	OD 9 OS 9	0.0	Irregularity R bifurcation 95% stenosis L ICA	
27	OD 7.5 OS 6.0	1.5	OD 20 OS 19	1.0	Ulcerated plaque R carotid at junction Ulcerated plaques L ICA	Amaurosis fugax OS
30	OD 2.0 OS 4.0	2.0	OD 18 OS 22	4.0	90% occlusion R ICA 50% occlusion L ICA	
31	OD 3.0 OS 2.25	0.75	OD 12 OS 10	2.0	Total occlusion L ICA	
33	OD 6.5 OS 4.75	1.75	OD 20 OS 20	0.0	Minimal irregularity R ICA Tight stenosis origin L ICA	Hollenhorst plaques OS
34	OD 2.0 OS 3.0	1.0	OD 38 OS 22	16.0	Stenosis R ICA	Open-angle glaucoma
36	OD 1.5 OS 1.5	0.0	OD 9 OS 9	0.0	Less than 50% stenosis both ICAs	
37	OD 1.0 OS 1.0	0.0	OD 10 OS 10	0.0	No report R ICA 90% stenosis L ICA	
39	OD 4.0		Not recorded		Plaque R common carotid. Total occlusion R ICA on repeat angiography	
41	OD 3.5 OS 4.5	1.0	OD 14 OS 14	0.0	No evidence R carotid disease	Amaurosis fugax OD
42	OD 5.0 OS 3.0	2.0	OD 18 OS 12	6.0	No report R Total occlusion L ICA	

Table 1b *Cases with normal pulse amplitudes but abnormal tensions*

Case no.	Mean pulse amplitude mmHg	Difference in amplitude mmHg	Tensions mmHg	Difference in tensions	Angiographic findings	Other findings
1	OD 2.5 OS 2.25	0.25	OD 7.0 OS 5.0	2.0	R ICA within normal L ICA occlusion	Venous stasis retinopathy OS
32	OD 3.5 OS 4.0	0.5	OD 12 OS 16	4.0	90% stenosis R ICA 100% stenosis L ICA	

tests have been reviewed by many authors.⁵⁻⁷ All these methods require special apparatus and do not form part of a routine ophthalmic examination. They are all capable of correctly diagnosing carotid artery occlusion (particularly if it is unilateral) in a high percentage of cases.

The results of the present study suggest that abnormalities of the ocular pulse and intraocular pressure have a diagnostic value similar to that of other non-invasive tests. Eighty-two percent of cases were correctly diagnosed as having stenosis of one or both ICAs, and these results confirm and amplify previous studies on the ocular pulse.⁸⁻¹⁰ False positive results were found in five out of 10 patients without angiographic evidence of stenosis of the internal carotid, but two patients had ocular disease to account for the abnormal ocular pulse and two of the remaining three patients had angiographic evidence of severe atheromatous disease of the ICA and could therefore be considered as true positive results.

However, a further patient with atheromatous disease of the carotids had normal pulse amplitudes and appplanation tensions.

The degree of stenosis of the ICA which is reflected in an abnormal ocular pulse cannot be assessed exactly from the present series, as the stenosis was not always quantified in the angiography report. All the patients described as having 75 to 100% occlusion were correctly diagnosed. In three patients with normal ocular pulses the angiographic findings were described as 'occlusion,' 'focal stenosis,' and '50 to 60% stenosis' respectively. It is probably necessary to have at least 50% stenosis to reduce the ocular pulse significantly, a figure which agrees with experimental findings.¹¹

The criteria used for abnormality of the ocular pulse were a difference of more than 0.5 mmHg between the two eyes and/or a pulse amplitude less than 1.5 mmHg for hypermetropes and emmetropes, and 1.0 mmHg for myopes. A difference in pulse

Table 2 *Cases with normal ocular pulses and normal tension*

Case no.	Mean pulse amplitude mmHg	Difference in amplitude mmHg	Tensions mmHg	Difference in tensions	Angiographic findings	Other findings
2	OD 2.0 OS 2.25	0.25	OD 12 OS 12	0.0	Focal stenosis R ICA No stenosis L ICA	
4	OD 2.5 OS 2.5	0.0	OD 15 OS 15	0.0	Ulcerated plaques both ICAs; no stenosis	
10	OD 3.0 OS 3.0	0.0	OD 20 OS 20	0.0	50 to 60% stenosis R ICA	
13	OD 3.0 OS 3.0	0.0	OD 18 OS 18	0.0	Internal fold R ICA, no stenosis; plaques L ICA, no stenosis	
15	OD 3.0 OS 3.5	0.5	OD 15 OS 13	2.0	Plaques bifurcation and R ICA; extensive plaques L ICA	
23	OD 3.5 OS 3.0	0.5	OD 14 OS 14	0.0	Plaques both carotids but no stenosis	Cerebral infarcts
24	OD 2.0 OS 2.5	0.5	OD 14 OS 14	0.0	Total occlusion R ICA; atheroma carotid syphon L	
28	OD 4.5 OS 4.0	0.5	OD 18 OS 16	2.0	Stenotic area R ICA; significant stenosis L ICA	
29	OD 2.25 OS 2.0	0.25	OD 14 OS 14	0.0	No stenosis ICAs	Branch artery occlusion OS
40	OD 5.2 OS 4.7	0.5	OD 14 OS 14	0.0	Complete occlusion L ICA	

Table 3 Summary of results in 38 patients

	No. of cases	Percent
Carotid stenosis correctly diagnosed	23	82.14
False negatives	5	17.86
No stenosis correctly diagnosed	5	50.0
False positives	5*	50.0

*Two patients had ocular causes for abnormality.

amplitude of 0.5 mmHg is suggestive of carotid artery disease but was found in 14% of an apparently normal population,² and this criterion is likely to lead to more false positive results than an abnormally low amplitude.

In the absence of ocular disease or high myopia an ocular pulse amplitude less than 1.5 mmHg or a difference of more than 0.5 mmHg between the two eyes and/or applanation reading of 10 mmHg or less is an indication of carotid artery disease and warrants further investigation.

The importance of these findings is not simply that the ocular pulse provides another non-invasive test of carotid artery blood flow but that measurement of the ocular pulse could be applied with tonometry as a presymptomatic screening method during a routine ophthalmic examination in the same way as tonometry is used as a screening method for glaucoma. It does demand, however, that the tonometer used is capable of measuring the amplitude of the ocular pulse. Apart from the instrument used in this study (which is not in production), there is only one type of instrument specifically designed to provide a recording of the ocular pulse. The Applanation Pneumotonograph (Alcon Surgical) and the Model 30 R/T Pneuma-Tonometer Tonographer (Digilab) both have facilities for recording the ocular pulse. The latter manufacturer also produces an oculocerebrovasculometer which includes a suction cup, so that the ocular pulse can be measured at various intraocular pressures.¹¹ Any recording Schiøtz-type indentation tonometer can also be used to detect differences in pulse amplitudes between the two eyes, but the absolute value of pulse amplitude is more susceptible to errors induced by the weight of the instrument and the indentation of the plunger, which combine to raise the intraocular pressure of the eye being measured.

Applanation tonometry with the Goldmann prism is widely accepted as the standard of reference for tonometry and combines ease of use with high accuracy. Unfortunately it does not produce a record-

ing of the pressure or ocular pulse, and the modification used in the present study was developed for this reason. It is possible to estimate the pulse amplitude by setting the semicircles of fluorescein so that they coincide first at their maximum excursion and then at their minimum excursion and noting the reading on the drum at each position. This method is less reliable than measurement from a recording, as the drum is calibrated in steps of 0.2 g (2 mmHg), so that small differences are difficult to measure accurately.

Carotid stenosis is a serious condition and is amenable to surgical treatment, so that it is a suitable candidate for presymptomatic detection if suitable screening methods were widely available. The measurement of the ocular pulse by tonometry would seem to offer a screening method easily applied during a routine ophthalmic examination. The ideal instrument for this would be an applanation tonometer with a recording system or an electrical output which could drive a small microprocessor with a visual display which could present the mean pressure and average a number of pulses to give a mean pulse amplitude.

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