COMMUNICATIONS

TONOGRAPHIC STUDIES IN CAROTID OCCLUSIVE DISEASE*†‡

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IN recent years there has been a growing awareness of the part played by carotid stenosis and occlusion in causing cerebrovascular accidents (Baker, 1962). In a society whose aged population is increasing, strokes will become a growing problem. Although arteriographic techniques have been developed to a high degree of excellence, and provide the maximum amount of information with relatively little morbidity, the discomfort of the procedure and the possibility of a serious mishap (Walsh and Smith, 1952; Bull 1960), still make it desirable to search for other methods of diagnosis which are less taxing to the patient, safer, and less exacting to perform.

This need has been met in part by the development of a variety of techniques designed to give an indication of cerebral vascular pressures and blood flow. Ophthalmodynamometry is one such technique and has proved fruitful in the diagnosis of carotid stenosis in trained hands (Lawton Smith, 1964; Sisler, 1960). However, not everyone finds the technique easy to use, and other techniques are still needed either as screening procedures before angiography or to make the definitive diagnosis of occlusive disease in those subjects in whom angiography cannot be performed.

The purpose of this study has been to investigate the origin of the ocular pulse in the normal subject, and to assess its variation in patients with carotid occlusive disease. The ocular pulse wave may be magnified and studied in detail by coupling the indentation tonometer to an amplifying recorder.

In the normal subjects studied, the size of the ocular pressure wave was found to be related to the size of the systemic arterial pulse pressure, suggesting that the ocular pulse is dependent on volume changes within the ocular arteries.

In patients with carotid occlusive disease, this relationship was disturbed, and further, a distinctive pattern of tonographic responses was demonstrated.

Pulsation may be demonstrated in any distensible organ, and is due to the pulsatile quality of its blood supply. Ocular pulsation was first recognized by Weber (1850),

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who demonstrated that ocular pressure varied in a pulsatile fashion. Wegner (1930) devised a capsule for recording this pulse in man. Uemura devised the electronic ophthalmic sphygmomanometer (Uemura, Utsumi, and Horiuchi, 1952) to study the pulse in normal subjects. Utsumi (1954) used this apparatus in normal and hypertensive subjects to measure the transmission time between a point on the electrocardiogram cycle and a point on the ocular pulse wave.

Castrén and Lavikainen (1964), using an electronic tonometer to record the ocular pulse, showed that this transmission time may be lengthened on the side of a carotid occlusion.

Suzuki, Nishi, Otsuki, and Azuma (1960) and Suzuki (1962) presented evidence to show that the ocular pulse is markedly diminished in a subject with congenital luetic choroidal atrophy and that the pulse is relatively unaltered in size in the presence of retinal artery occlusion, the latter having already been shown by Thiel (1928). These observations support the present view that the uveal blood flow makes a greater contribution to the total ocular blood flow than the retinal blood flow.

Hager (1964) devised a cup-like device which overlies the orbit and records the combined ocular and orbital pulse. By observing the appearance and disappearance of the pulse produced by variations in the pressure within the cup, he used this technique to measure ophthalmic artery pressure and the volume pulse of the orbit. In the presence of carotid occlusive disease, the volume pulse is diminished in amplitude and the ophthalmic artery pressure is low. This appears to be a promising diagnostic tool. Other methods for recording such pulses are also available (Johnson, 1965).

Technique

Equipment

A Crescent electronic ocular tonometer, model 800-B, was used throughout the study. The signal was amplified by a Sanborn D.C. Coupling pre-amplifier, model 350-1300, and recorded with a Sanborn model 296 T.C. Oscillographic recording system. The second channel of this recorder was used to record the electrocardiogram of the subject, using a Sanborn model 350-3200 ECG pre-amplifier.

With this equipment the amplification of the signal can be varied by adjustment of the attenuator setting. Routine tonography is performed with an attenuator setting of $\times 10$. Attenuations of $\times 5$ or $\times 2$ were also used. This increases the amplification by twice and five times respectively.

Three different paper speeds were employed: 1.25 mm./sec., 5 mm./sec., and sometimes 25 mm./sec.

Methods

A standard procedure was developed over the course of the study. This was not used in all subjects reported here because of physical limitations such as severe hemiplegia. The present procedure is as follows:

The patient is put at ease and told the purpose of the tests. A brief ocular and general history is taken. Slit-lamp examination of the anterior segment and applanation tonometry are performed and the ocular media and fundus are then examined. In particular, signs of ischaemic ocular disease are searched for (Knox, 1965; Hedges, 1962). The patient then lies supine on a mobile stretcher and the head is supported if necessary. The tonometric record is calibrated and the electrocardiographic leads are attached. By this time the patient is relaxed and the blood pressure is then taken in each arm with a sphygmomanometer.

During tonography, particular attention is paid to keeping the tonometer in the vertical position, since tilting the instrument can alter the size of the recorded pulse wave. Also the tonometer is cleaned between the recordings for each eye whenever the records are required for comparative measurements of the pulse size. Sticking of the plunger readily reduces the size and distorts the shape of the pulse wave. To clean the tonometer, both the barrel and plunger are rinsed in a stream of distilled water and dried with lint-free absorbent tissue (Garner, 1965). A dry twist of tissue drawn partly through the barrel from either end of the tonometer is usually effective.

Proparacaine hydrochloride (Ophthaine-Squibb) was the anaesthetic used throughout the study. Excessive tears or anaesthetic solution in the conjunctival sac were wiped away before proceeding. This prevented sticking of the plunger due to the capillary action of fluid high in the barrel of the instrument.

Tonographic Procedure

The three procedures are presented below in the order of performance. The 5.5 g. weight is used for pressures below 20 mm. Hg, and the 7.5 g. weight for pressures from 20–30 mm. Hg.

(1) *Base-Line Pressure Readings*.—The attenuator is set at $\times 10$, and the paper speed at 1.25 mm./sec. Pressure readings are taken in each eye, with 2 minutes between each pair of readings. The tonometry lasts about 15 sec. in each eye. The right eye is tested before the left. This provides a tonometric evaluation of intra-ocular pressure and gives an index of its stability.

(2) *Pulse Amplitudes*.—The attenuator is set at $\times 5$, and the paper speed at 5 mm./sec. The ocular pulses are recorded in each eye as before, for about 15 sec., or until a series of at least five regular pulse waves, free from artefact, are obtained. An attenuation of $\times 2$ is sometimes used.

(3) *Carotid Compression*.—The attenuator is set at $\times 10$, and the paper speed at 1.25 or 5 mm./sec.

(a) The ipsilateral compression test is performed with the tonometer on one eye while the common carotid artery on the same side is compressed low down in the neck by an assistant. Compression is maintained for 5 to 10 sec. and then quickly released. If the patient complains during the test or appears to be uncomfortable, the compression is released sooner and the nature of his symptoms ascertained.

(b) The contralateral compression test is performed with the tonometer on the same eye, while the common carotid artery on the opposite side is compressed for the same period as before.

During both tests, the assistant palpates the homolateral superficial temporal artery over the zygoma on the side of the compression in order to be certain of the effectiveness of the compression on each side. If the pulsation in the former vessel does not disappear when the carotid is compressed, then the compression is inadequate, and the test must be repeated.

Composition of the Study

Group I. Normal (Cases 1–10).—There were three males and seven females (mean age 45 years, range 13 to 68), in whom the difference in mean intra-ocular pressure between the two eyes as measured by the electronic tonometer was less than 2 mm. Hg. Two subjects had cataracts and one had heterochromic cyclitis, but none had any signs of local or systemic vascular disease.

Group II. Hypertensive (Cases 11–16).—There were two males and four females (mean age 56 years, range 46 to 67), in whom the diastolic brachial artery pressure exceeded an arbitrary 100 mm. Hg.

Group III. Miscellaneous Eye Disease (Cases 17–22).—There were six females (mean age 56 years, range 40 to 71), who did not fit into the other categories. In two the intra-ocular pressures differed by more than 2 mm. Hg, one had glaucoma, two had recently undergone eye surgery, and one was receiving thyroid hormone.

Group IV. Cerebrovascular Disease (Cases 23–33).—There were nine males and two females (mean age 57 years, range 38 to 69), in whom there was evidence of vascular disease affecting the brain, or the eyes, or both. Eight had angiographic evidence of carotid obstruction (stenosis or occlusion), eight presented with a major or minor stroke, and four complained of visual disturbance. Five showed ischaemic ocular signs of mild to severe degree. The distribution of these symptoms and signs is shown in Table I.

TABLE I
CLINICAL FINDINGS IN ELEVEN PATIENTS IN GROUP IV (Subjects 23–33)

Case No	Age (yrs)	Sex	Ocular or Cerebral Disorder	Ischaemic Eye Disease	Ophthalmodynamometry	Angiogram	Prolonged Initial Fall	Pulse Size Difference	Positive Contralateral Compression	Negative Ipsilateral Compression	Delayed Post-compression Recovery	Comment
23	63	M	L eye	L	L ↓	Diffuse R ICO L ICS	No	L ↓	—	—	—	L Glaucoma
24	53	M	L cerebrum	No	Equal	L ICO	L	L ↓	—	—	—	
25	47	M	R eye	R	R ↓	Diffuse R ICO	R	R ↓	—	—	—	Severe diabetes mellitus
*†26	69	F	L cerebrum	No	L ↓	L ICO ECO	L	L ↓	L	No	L	
*†27	54	M	L cerebrum	L	L ↓	L ICO	L	L ↓	L	No	L	
*§28	64	M	R eye R cerebrum	R	—	R CCO ICO ECO L ICO CCO	R+++ L++	L ↓	—	—	—	Marked R P.I.F. but pulse lowest on L
*29	51	F	L cerebrum	No	—	L ICO	R	R ↓	—	—	—	
*30	38	M	L cerebrum	No	L ↓	L ICO ECO CCO	L	L ↓	L	Yes	No	
*31	67	M	R cerebrum	No	R ↓	ND	No	No	Bradycardia	No	L	
*32	54	M	L cerebrum	No	Equal	ND	L	No	Bradycardia	No	No	
*33	62	M	R amaurosis fugax	R	R ↓	ND	R	No	Bradycardia	No	No	

CCO = Common carotid occlusion ICO = Internal carotid occlusion ICS = Internal carotid stenosis
ECO = External carotid occlusion ND = Not done

* See Fig. 4 † See Fig. 9 ‡ See Figs 5 and 8 § See Fig. 6

Group V. Normotensive (Cases 5–10 and 34–39).—There were twelve subjects, including six from Group I, who all had a diastolic brachial artery pressure at or below 100 mm. Hg; these were designated the normotensive group. In these cases a comparison of blood pressure and ocular pulse pressure was made.

Results

The results are presented in two sections: (A) subjects without clinical signs and symptoms of cerebrovascular disease, and (B) subjects showing evidence of cerebrovascular disease.

(A) Subjects without Cerebrovascular Disease (Groups I, II, III, and V)

In the normal subject, the pressure level stabilizes rapidly as soon as the tonometer is placed on the eye. Fig. 1 shows the record from a normal subject with an attenuator setting of $\times 10$; as soon as the tonometer begins to record ocular pressure, the pulse waves can be clearly seen. The intra-ocular pressure stabilizes at or within the first pulse, and then falls gradually as seen in routine tonography.

FIG. 1.—Normal subject. Tonographic pulse record. Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 10$. The pressure stabilizes rapidly when the tonometer is placed on the eye.

With the attenuator set at $\times 5$, a convenient record is produced from which to calculate and compare pulse amplitude between the two eyes. In the normal subject there is only a small difference in size between the two pulses (Fig. 2).

FIG. 2.—Normal subject.
Upper tracing: Electrocardiogram.
Lower tracing: Tonographic pulse record.
Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 5$.

In the normal subject, carotid compression produces an abrupt fall in the intra-ocular pressure on the same side as the compression. The ocular pulse wave diminishes in size at the same time. On release of compression, the level returns very rapidly to the extrapolated baseline pressure. This point is at a pressure only slightly lower than the pressure before compression, and presumably includes a fall in intra-ocular pressure due to the expression of aqueous by the weight of the tonometer. With the return of the pressure on release of arterial compression, the pulse waves also return to their normal size. Such a normal response is shown in Fig. 3. This fall in intra-ocular pressure on the side of carotid compression will be referred to as the positive ipsilateral compression response. A negative ipsilateral compression response is designated when the intra-ocular pressure fails to fall during compression of the ipsilateral artery.

FIG. 3.—Normal subject.

Upper tracing: Electrocardiogram.

Lower tracing: Carotid compression responses.

Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 10$.

R.C.C. = Right carotid compression. L.C.C. = Left carotid compression.

On each side the eye pressure drops sharply on ipsilateral compression and returns briskly when compression is released. The eye pressure does not fall on compression of the contralateral artery.

Again, in normal subjects, in the absence of bradycardia, there is no fall in intra-ocular pressure during compression of the opposite common carotid artery. Such a response will be referred to as a negative contralateral compression response. Sometimes a transient slight rise in pressure is observed during this test. The term positive contralateral compression response will define a fall in pressure occurring during compression of the opposite carotid artery.

The bradycardia which may occur during carotid compression (seen in four patients), particularly in subjects with cardiovascular disease, is due to carotid sinus stimulation. In some it may be induced by the lightest pressure over the carotid,

insufficient to compress the artery to any great extent. Such a bradycardia may itself lower the intra-ocular pressure. It is therefore very important to avoid producing bradycardia, since a fall in pressure on contralateral carotid artery compression is an abnormal response which implies carotid vascular occlusion. It is for this reason that the carotid artery is compressed low down in the neck, to avoid stimulating the carotid sinus. The ECG is continuously recorded during the compression tests so that bradycardia may be easily identified and the test repeated if necessary. A paper speed of 5 mm./sec. makes the bradycardia easy to assess.

Analysis of Pulse Amplitudes.—During tonography the stylus of the recorder registers the degree of indentation of the cornea by the plunger of the tonometer. As the calibration of the Sanborn tonometer is linear, unit deflection on the recording chart represents unit indentation by the plunger weight. The degree of indentation is measured in "scale units", which is referred to as the "R" value. Thus, the paper of the recorder registers directly the degree of indentation in a particular eye and the pulse wave seen may be regarded as an "indentation pulse". The difference between the highest and lowest points on each wave, measured in scale units, gives the amplitude of the indentation pulse. The average of four or five pulses has been used for each subject in this study. It is worth remembering that the "R" value falls when the pressure in the eye rises; therefore, the lowest scale reading when analysing the pulse wave is a systolic value and the highest scale reading is a diastolic value.

The indentation pulse reflects pulsatile changes in intra-ocular pressure which can be calculated by referring to standard tables relating the degree of indentation produced by a plunger of a given weight to the pressure in the eye. For routine tonometric purposes, it is the pressure in the eye before the tonometer is set in place (P_0) which is required. However, in the present study, since the pressures under investigation are generated while the tonometer is on the eye, it is the intra-ocular pressure with the tonometer in place (P_1) that is needed. Therefore, to obtain the pulsatile pressure changes in the eye, the systolic and diastolic "R" values were converted into P_1 values, using the 1955 conversion tables of Friedenwald (1957).

The amplitude of the pressure pulse was then derived by subtracting the diastolic from the systolic value.

Since the indentation pulse and pressure changes are caused by pulsatile ocular blood flow, one may analyse the indentation record to obtain a volume pulse of the eye. It is possible to convert the changes in indentation by the tonometer plunger into changes in ocular volume by reference to standard tables (Friedenwald, 1957) relating indentation value and ocular volume changes. The Friedenwald table presents the change in ocular volume, ΔV , occurring when the tonometer is placed on the eye and the scale reading falls from zero to "R". If ΔV is derived for the diastolic and systolic "R" values, the difference between these two readings represents an amplitude of the volume pulse in microlitres.

In this way, indentation, pressure, and volume pulses were obtained for analysis. A sample record is given overleaf in Table II (Case 7 of Group I).

The difference in pulse amplitude between the right and left eye was calculated for each subject in Groups I, II, and III, and expressed as a percentage of the highest

TABLE II
 SAMPLE RECORD OF OCULAR PULSE VALUES IN A NORMAL SUBJECT (GROUP I, CASE 7)
 WITH TONOMETER IN PLACE
 INDENTATION, PRESSURE, AND VOLUME PULSES ARE SHOWN, WITH THE PERCENTAGE DIFFERENCE
 BETWEEN THE TWO EYES IN EACH CASE

Pulse Values	Scale Reading "R"		Intra-ocular Pressure P _t (mm. Hg)		Volume Change ΔV (μ l.)	
	Right	Left	Right	Left	Right	Left
Systolic Value	6.4	6.2	28.1	28.5	22.5	21.8
Diastolic Value	7.0	6.8	27.1	27.4	24.6	23.9
Pulse Difference	0.6	0.6	1.0	1.1	2.1	2.1
Percentage Difference	0		9		0	

of the two pulse amplitudes (Table III). This small series shows that in the normal subjects the variation in size of pulse between the two eyes is least for the indentation and volume pulses, and most for the pressure pulse. Thus the difference in pulse size is less than 25 per cent. for the indentation and volume pulses, and above 25 per cent. for the pressure pulses in Groups I and II. The range of variation was greater in Group III.

TABLE III
 PERCENTAGE DIFFERENCE IN PULSE SIZE BETWEEN THE TWO EYES FOR GROUPS I, II, and III
 (Difference expressed as percentage of higher reading)

Group	No. of Cases		Pulse		
			Indentation	Pressure	Volume
I	10 (1-10)	Mean Range	7 0-25	20 13-38	5 0-19
II	6 (11-16)	Mean Range	6 0-17	21 8-45	6 0-23
III	6 (17-22)	Mean Range	15 0-29	19 3-50	16 0-29

Systemic Blood Pressure and Ocular Pulse.—At the inception of these studies it was hoped that the ocular pulse might provide a measure of ocular blood flow. Such a prospect has not been realized, but some insight has been gained into the factors generating the ocular pulse.

To test an impression that pulse size was related to systemic vascular pulse pressure, the ocular pulse pressure for each eye in Group V was plotted against the left brachial artery pulse pressure. The results are plotted in Fig. 4 (opposite). A regression line was fitted to the data which showed a positive correlation which was highly

FIG. 4.—Left brachial artery pulse pressure plotted against ocular pulse pressure for each eye in twelve normotensive (Group V) subjects (5–10 and 34–39); a regression line is plotted for the points. A similar plot for the eye shows the lower ocular pulse pressure only in eight subjects (26–33) with cerebrovascular disease.

The number of each subject is shown (see Text and Table I and B).

significant ($P < 0.001$). No statistical relationship was found with these small numbers between ocular pulse pressure and mean brachial artery pressure, and there was no relationship between mean brachial artery pressure and pulse pressure on the one hand and ocular volume pulse on the other.

(B) Cerebrovascular Disease (Group IV)

Although every patient in the study did not undergo all the tests devised, sufficient information has been obtained to suggest that carotid occlusive disease may be characterized tonometrically by some or all of the five features discussed below:

(1) *Prolonged Initial Fall in Intra-ocular Pressure.*—The term “prolonged initial fall” refers to a failure of the intra-ocular pressure to stabilize as soon as the tonometer is placed on the eye. Instead, the pressure falls rapidly from the starting point and stabilizes only after a few seconds have elapsed. During the fall, the pulse waves either fail to appear, or are only just visible, but when the pressure stabilizes, the pulses are seen. An example is shown in Fig. 5 (overleaf).

This response is seen in the eye on the side of a carotid occlusive lesion. In one patient with gross bilateral occlusive disease (Fig. 6, overleaf), the response was seen in both eyes, but was much more marked on one side. Of the eight patients tested who had proven carotid occlusion, seven showed this response. Of the three patients in whom carotid occlusion was not proven, two showed this response.

(2) *Small Pulse Amplitude.*—The percentage difference in pulse size between the two Groups I and II and Group IV is shown in Table IV (overleaf). Indentation and volume pulses only are presented, since these showed the least difference between the two eyes in normal subjects.

FIG. 5.—Subject 27, with complete occlusion of left internal carotid artery.
Upper tracing: Prolonged initial fall in ocular pressure in left eye. The pulse on the left side is smaller.
 Weight 5.5 g.; paper speed 5 mm./sec.; attenuation $\times 5$.
Lower tracing: Electrocardiogram.

FIG. 6.—Subject 28, with bilateral occlusive disease. Prolonged bilateral initial fall in ocular pressure, greater on the right.
 Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 10$.

TABLE IV
 PERCENTAGE DIFFERENCE IN PULSE SIZE BETWEEN THE TWO EYES FOR NORMAL + HYPERTENSIVES (GROUPS I AND II) AND CEREBROVASCULAR DISEASE (GROUP IV)

Group	No. of Cases		Pulse	
			Indentation	Volume
I and II	10 + 6 (1-16)	Mean Range	7 0-25	5 0-23
IV	11 (23-33)	Mean Range	37 0-67	34 0-67

Eight of the eleven patients showed a difference greater than 25 per cent., whereas all the normal subjects and hypertensive patients showed a difference less than 25 per cent. The eight patients all had arteriographic confirmation of occlusion, and in seven the lower pulse was on the side of the occlusion. The eighth subject showed a low pulse in the right eye, opposite to a left carotid occlusion demonstrated some months before, but had undergone a left carotid endarterectomy before the pulse study was performed. Therefore it is quite reasonable to suppose that the operation served to increase the ocular pulse size on the

side of the lesion, and that the presence of unrecognized carotid disease on the opposite side accounted for the small pulse. Unfortunately no arteriogram had been performed on the side showing the lower pulse. These results are shown in Fig. 7.

FIG. 7.—Percentage difference in pulse volume between the two eyes. Ten normotensive and six hypertensive subjects (Groups I and II) are compared with eleven subjects with cerebrovascular disease (Group IV). The three subjects in Group IV whose values fell within the range for Groups I and II did not have angiography to demonstrate carotid occlusion.

A low ocular pulse on one side thus appears to demonstrate the presence of unilateral carotid occlusive disease. However, in cases of bilateral occlusive disease, it would be expected that the ocular pulse would be low on both sides, and the difference might then be less than 25 per cent., in which case the abnormality would be missed. Some absolute index of pulse size is needed. To obtain this, the lowest ocular pulse in eight cerebrovascular disease subjects from Group IV with known blood pressures was plotted against the brachial artery pulse pressure in that subject, and compared with a similar plot for Groups I and II. In each case, the point fell below the regression line for Groups I and II, some far below (see Fig. 4). This presumably reflects a reduction in arterial pulse pressure above the carotid occlusion.

(3) *Negative Ipsilateral Compression Response.*—This was found in one patient only. The absence of an ipsilateral compression response is abnormal, and implies that the ipsilateral common carotid makes an insignificant contribution to ophthalmic artery pressure on the same side.

A 38-year-old Negro male (Case 30, Table I) was brought into the Johns Hopkins Hospital emergency room, confused, dysphasic, and unable to walk. He soon developed a right hemiplegia and complete aphasia. He had an 18 months' history of atypical Menière's syndrome. A left carotid arteriogram showed a complete block of the left common carotid, and operation demonstrated thrombus extending up into the bifurcation. One week later he was still dysphasic and hemiplegic. Ophthalmodynamometry at this time showed a diastolic reading 18 units lower on the left than on the right. Pulse studies showed a prolonged initial fall, a low pulse on the affected side and a positive contralateral and negative ipsilateral compression response. The left superficial temporal artery later became impalpable.

The interpretation of a negative ipsilateral compression response is vitiated by the fact that when the external or common carotid systems are obstructed, the superficial temporal artery will receive its blood supply from other sources. Then the common carotid compression will not be associated with extinction of the superficial temporal pulse on the same side. Thus the best means of confirming the adequacy of common carotid compression is lost, and it only remains for the assistant to judge whether compression has been satisfactory.

(4) *Positive Contralateral Compression Response.*—A negative contralateral compression response is normal, and a positive response implies that a substantial contribution to ophthalmic artery flow is derived from the opposite carotid system. This might occur with occlusion of the internal carotid or common plus internal carotid artery. This test has

been performed in only three subjects with proven carotid occlusion. In each of these, a positive contralateral compression response was obtained (Fig. 8), as in the following example.

A 54-year-old Caucasian male (Case 27, Table I) presented in March, 1965, with a mild dysphasia and right-sided sensory symptoms. He was found to have a right homonymous hemianopia. Ophthalmodynamometry showed a diastolic pressure of 35 units in the right eye, the pressure reading being too low to measure in the left. The arteries at the left disc were readily collapsed by light finger pressure on the globe. The veins were engorged and there was no venous pulsation at the disc. There was a small blot haemorrhage in the upper temporal fundus. A gamma scan demonstrated a circumscribed lesion in the left occipital pole, which was suggestive of a neoplasm. On the basis of the ophthalmodynamometry and the fundus appearance, pulse studies were performed. These showed prolonged initial fall and decreased pulse size on the left, a positive contralateral compression response, and delayed post-compression recovery. These results suggested the presence of a left internal carotid obstruction at least. Arteriography confirmed this; the internal carotid artery was blocked from its origin to the posterior end of the carotid syphon. The external carotid system filled rapidly and showed numerous collateral channels.

The patient was regarded as having a left internal carotid occlusion of long standing, with a more recent obstruction of the left posterior cerebral artery causing an occipital pole infarct.

FIG. 8.—Subject 27, with left internal carotid occlusion, showing positive contralateral compression response. The tonometer is on the left eye.

Upper tracing: Left-hand: Fall in pressure on compression of right carotid artery.

Right-hand: Fall in pressure on compression of left carotid artery.

There is delayed recovery of ocular pressure on release of arterial compression, shown best in the left-hand record.

Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 10$.

Lower tracing: Electrocardiogram.

FIG. 9.—Subject 26, with left internal and external carotid occlusion. Delayed post-compression recovery of ocular pressure. The tonometer is on the left eye. The ocular pressure falls after right carotid artery compression (positive contralateral compression response). After release of compression, the ocular pressure rises to a lower level than the previous baseline.

Weight 5.5 g.; paper speed 1.25 mm./sec.; attenuation $\times 10$.

(5) Delayed Post-compression Recovery of Ocular Pressure.—In three subjects with carotid occlusion, when the tonometer was placed on the eye on the side of the lesion and the pressure in the eye was lowered by carotid compression, it was found that the recovery of pressure on release of compression was delayed, so that the new pressure remained at a lower level than that expected by extrapolation of the base-line pressure (Fig. 9). It did not matter whether the fall in pressure was induced by ipsilateral or contralateral artery compression. A possible mechanism for this response is considered in the discussion.

Discussion

The ocular pulse is caused by vascular pulsation within the eye. The increase in

volume of these vessels occurring with each arterial systole causes a rise, and the fall in vascular volume in diastole causes a fall in intra-ocular pressure. This cycle of events produces the ocular pulse wave.

A variety of factors may alter the size and form of the pulse recorded by the indentation tonometer, such as cardiac output, heart rate, arterial pulse pressure, the elasticity of arteries inside and outside the eye, intra-ocular pressure, ocular rigidity, and finally, the weight, inertia, and friction of the tonometer.

The technique involved in recording pulses with the tonometer is simple in the normal subject, but in dysphasic and hemiplegic subjects full co-operation may be difficult to achieve. A recently hemiplegic patient, for instance, may reflexly close the lids on the non-paralysed side when the tonometer approaches the eye. However, satisfactory records can be obtained in most circumstances with perseverance. The tonometer provides a convenient rather than ideal instrument for recording the ocular pulse wave. It raises the intra-ocular pressure, its moving parts are far from frictionless, and its inertia doubtless modifies the wave form obtained from the eye. Nevertheless, the tonometer has certain advantages over the ophthalmodynamometer in the diagnosis of vascular disorder. Thus, for tonometry, mydriasis and clear media are unnecessary to obtain good results; even patients with poor vision can maintain fixation for long enough to obtain a good record. Also, it is felt by the authors that the tonometer has special advantages in that a record of intra-ocular pressure is obtained from the moment the tonometer is placed on the eye, and therefore while the ocular pressure is rising to its new value. The importance of this will be elaborated later.

An attempt to correlate ocular pulse pressure with brachial artery pulse pressure might seem unjustified, but is made valid by the observations of Hager (1964), who has shown that ophthalmic artery pressure in man is equal to, or only 5 to 10 mm. Hg lower than, the brachial artery pressure.

It has long been known that ocular pulsation has a vascular cause, but it has not been clearly stated which intra-ocular vessels are responsible. It is thought that the uveal circulation makes a greater contribution than the retinal circulation and it is evident that volume changes within these vessels are the immediate cause of the pulsatile changes in the eye. But whether arteries, veins, or capillaries make different or equal contributions is not known.

The excellent positive correlation found in normal subjects in this study between intra-ocular pulse pressure and systemic arterial pulse pressure suggests that pulsation in the ocular arteries is chiefly responsible for the form taken by the ocular pulse wave, and that veins and capillaries play a minor part. Indeed, it may be that expressible blood in this low-pressure portion of the vascular bed buffers the effects of volume change in the ocular arteries and reduces the size of the ocular pulsation.

Fig. 4 shows data for eight subjects from Group IV (with cerebrovascular disease), for whom blood pressure records were available, plotted with similar data for Group V (normotensive). To bring out any change in the ratio of ocular pulse pressure to systemic arterial pulse pressure in the disease group, only one eye was plotted for each subject, that with the lower ocular pulse pressure.

In six of the eight subjects, the plotted pressure was on the same side as a proven (four) or suspected (two) occlusion. Of the other two, Case 31 showed a slightly

lower ocular pulse on the side opposite the occlusion. In Case 29 the lowest pulse was opposite a known carotid occlusion (which had been treated by endarterectomy). It is of interest that the latter subject showed a ratio of ocular pulse pressure to systemic arterial pulse pressure which was nearer that of the normotensive group. The others showed a fall in this ratio.

A fall in the ratio of ocular pulse pressure to systemic arterial pulse pressure, in the presence of a homolateral carotid occlusion, could be explained by a reduced compliance of the intra-ocular arteries in the presence of generalized atherosclerotic disease. It could also be explained by a failure to transmit arterial pressure from the great vessels in the neck to the vessels of the eye. The latter explanation is more acceptable in the present context and suggests that the arterial pulse pressure falls distal to an internal carotid artery block, and that this lowered arterial pulsatility produces a lowered ocular pulsation on the side of the lesion.

Another factor which might lower the pulse size is an increase in the capillary and venous blood volume due to ischaemia. This would serve to damp the effect of arterial volume changes.

The low ocular pulse size in these subjects does not in itself indicate a reduction of blood flow through the eye. This is in keeping with the findings of workers in the field of peripheral vascular occlusive disease. Wakim, Slaughter, and Clagett (1948), studying the femoral artery pressure and flow in subjects with varying degrees of coarctation of the aorta, showed that, while the arterial pulse pressure beyond the level of the block is reduced, the blood flow may remain essentially normal. Hillestad (1962), in a comparative study of blood flow and oscillometry in intermittent claudication, showed that the size of the volume pulse of the limb in this situation may be unrelated to the blood flow through the part.

It is thus not surprising that only half the patients with a reduced pulse size showed signs of ocular ischaemia.

The prolonged initial fall in pressure seen in patients with carotid occlusive disease was not seen in normal subjects in the present study. Its salient features are a rapid fall in intra-ocular pressure when the tonometer is placed on the eye, and a lack of ocular pulsation during the fall. This response is best explained by vascular changes occurring in the eye when the tonometer is put in place.

A reduction in pressure in the arteries perfusing the eye, as produced by carotid compression, lowers both the ocular pressure and pulsation (Fig. 5). In the prolonged initial fall response, both these events occur in the short period before the ocular pressure stabilizes and pulsation resumes. A hypothesis may be put forward that the prolonged initial fall represents a cessation of arterial flow through the eye in the first few seconds after the tonometer is placed on the eye. This concept is acceptable if two points are borne in mind:

- (1) The ocular pressure is raised when the tonometer is placed on the eye.
- (2) The ophthalmic artery pressure may be very low on the side of a carotid artery occlusion. Tindall, Dukes, Cupp, and David (1960) found that the ophthalmic artery pressure after carotid ligation might fall by 3.3 to 59.4 per cent. of the pre-ligation level. Thus, if the mean pre-occlusion ophthalmic artery pressure were 100 mm. Hg, it might fall to 40 mm. Hg after occlusion, and pressures might be lower in the small vessels of the eye, say, in the order of 30 mm. Hg.

When a tonometer is placed on the eye, the intra-ocular pressure rises to a new level (from P_0 to P_1). If the initial pressure were 20 mm. Hg, it would rise to about 33 mm. Hg with a 5.5 g. weight on the eye. This intra-ocular pressure would exceed the pressure in the intra-ocular vessels, producing closure and cessation of flow. This would be followed by a fall in intra-ocular pressure due to a fall in ocular blood volume. With this fall in pressure the factor causing vessel closure would be diminished so that the vessels could open again and flow be resumed. This would be associated with a stabilization of pressure and a resumption of ocular pulsation. In this way, the events seen in the prolonged initial fall response could be explained exactly.

The above hypothesis requires that intra-ocular pressure exceed arterial pressure for arterial flow to cease. It is possible that the arterial vessels may be caused to collapse when the pressure in them is greater than that in the eye, particularly if the phenomenon of "critical closure" of vessels occurs in the eye. This concept, introduced by Burton (1965), suggests that muscular vessels whose walls exhibit active tension or tone will collapse when the transmural distending pressure (the intravascular, minus the tissue pressure) falls below a certain critical value, termed the critical closing pressure. This implies that the vessels will close while their intra-vascular pressure is higher than the tissue pressure; the higher the muscle tone, the higher the critical closing pressure. Once closed, the vessels may open at the same or a higher transmural pressure, the critical opening pressure. Critical closure is thought to occur in the arterioles.

In the eye, the tissue pressure is the intra-ocular pressure (Fig. 10 *a* and *b*). Considering the situation in the eye on the side of a carotid occlusion, the intra-vascular pressure in the uveal and retinal arteries is low, the intra-ocular pressure not much altered. The net result is that the transmural pressure for these vessels is below normal (Fig. 10*c*). When the tonometer is placed on the eye, the sudden rise in tissue pressure will produce an equally sudden fall in the transmural pressure, which was already low (Fig. 10*d*). The possibility then exists that the critical closing pressure will be reached and the vessels will close. With the ensuing fall in

FIG. 10.—Total height of columns represents hypothetical intra-ocular arteriolar pressures.

Normal

- (a) Tonometer off the eye: I.O.P. 20 mm. Hg, arteriolar pressure 80 mm. Hg, transmural pressure 60 mm. Hg.
(b) Tonometer on the eye: (5.5 g. wt.) I.O.P. rises to 33 mm. Hg, arteriolar pressure unchanged, transmural pressure 47 mm. Hg.

Side of Occlusion

- (c) Tonometer off the eye: I.O.P. 20 mm. Hg, arteriolar pressure 63 mm. Hg, transmural pressure 43 mm. Hg.
(d) Tonometer on the eye: I.O.P. rises to 33 mm. Hg, arteriolar pressure 63 mm. Hg, transmural pressure falls to 30 mm. Hg. This is the critical closing pressure and therefore the vessels close.
(e) Tonometer on the eye: With vessel closure, the I.O.P. begins to fall until, at a pressure of 25 mm. Hg, the transmural pressure exceeds the critical opening pressure so that the vessels open and flow resumes. This would be speeded by a rise in arterial pressure.

intra-ocular pressure (Fig. 10e), the transmural pressure rises, so that the vessels are allowed to re-open once the critical opening pressure is reached. The argument here is the same as before, but would permit the sequence of events seen in the prolonged initial fall in intra-ocular pressure to occur at a higher level of intra-ocular arterial pressure.

In this regard, the record of Case 27 (Fig. 5) is particularly interesting. When the tonometer was placed on the eye on the side of the carotid occlusion, the ocular pressure first fell rapidly and then a double hump appeared in the pressure record before ocular pulsation finally began and the pressure stabilized. Although this has the appearance of a poor record, the response was in fact reproducible on three separate occasions, the patient's eyes were fixing well, and the tonometer was applied accurately. In terms of the hypothesis put forward, this would be regarded as an oscillation of ocular pressure caused by opening and closing of vessels whose transmural pressures are very close to their critical closing pressures.

The responses to carotid compression are straightforward. In the normal subject each internal carotid is responsible for supplying the homolateral eye with blood. Compression of the opposite carotid does not greatly influence this situation, and therefore the intra-ocular pressure in an eye contralateral to the carotid compression does not fall, whereas the pressure in the ipsilateral eye falls abruptly. After occlusion of an internal carotid, the opposite carotid system takes over a proportion of the blood supply to the eye on the side of the lesion. In this case, compression of the contralateral artery deprives the tested eye of a proportion of its arterial supply, so that there is a fall in intra-ocular pressure on the side of the lesion. This is the basis of the positive contralateral compression response.

The negative ipsilateral compression response is explained because an occluded carotid system does not fill the homolateral ophthalmic artery with blood. Therefore compression cannot interfere with blood supply. This may be caused by an internal plus an external occlusion.

The cause of delayed post-compression recovery can only be speculated upon. It has already been stated that raising the intra-ocular pressure by placing the tonometer on the eye serves to lower the transmural pressure of the intra-ocular vessels. Further, the transmural pressure will be much lower on the side of a carotid occlusion than on the opposite side. When carotid compression is performed, the arterial pressure falls still further and with it the transmural pressure. On the side of the lesion, this might lead to critical closure of vessels which might not occur on the side with healthy vessels. Thus, with release of the carotid artery compression and the rise in the ophthalmic artery pressure, the rise would be slower on the side of the lesion for various reasons. The critical opening pressure must be reached before the vessels will open and blood flow will resume, and the critical opening pressure is not necessarily the same as the critical closing pressure, but may in fact be higher. Also, the rise in ophthalmic artery pressure may be slowed down by an increased resistance in the diseased vessels on the side of the lesion.

This study is small and a much larger series of normal and diseased subjects will be required before norms can be established for the parameters discussed. However, it appears that the electronic tonometer may yield varied information about the

carotid vascular system and the blood supply of the eye. These may be summarized as follows:

- (1) The prolonged initial fall and delayed post-compression recovery of intra-ocular pressure are thought to be dependent upon a low mean ophthalmic artery pressure. This could result from local arterial disease or from obstruction of the internal or common carotid artery.
- (2) Lowered pulse size is thought to reflect reduced pulsatility in the arteries supplying the eye, and presumably could result from the same disease processes responsible for prolonged fall and delayed rise.
- (3) The response to carotid compression gives information about the presence of carotid occlusive disease, and indicates the side affected.

Prolonged initial fall and delayed post-compression recovery may be expected to occur independently of a reduction in pulse size, since improved collateral blood supply after a vascular occlusion might be expected to raise the mean arterial pressure in the ophthalmic artery without increasing the pulse pressure.

Summary

The use of the electronic tonometer in recording and magnifying the ocular pulse wave is described. Normal subjects and subjects with carotid occlusive disease were studied. A characteristic series of responses seen in the latter group is thought to be of diagnostic value. The nature and origin of the pulse in health and its modifications by disease are discussed.

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APPENDIX

Original data for Groups I and V and Group IV are presented in Tables A and B.

The reason for converting records into indentation, volume, and pressure pulses in this study is that the relationships between these parameters are complex, so that the pulses may differ in varying circumstances. Thus the relationship between the "R" values and the corresponding P_t values (based on Friedenwald's 1955 Tables) is such that when two eyes have different pressures, one high and one low, then the eye with the lower pressure will

TABLE A
DATA FOR GROUPS I AND V WITH 5.5 G. TONOMETER
(Group I comprises Subjects 1-10, and Group V Subjects 5-10 and 34-39)

Case No.	Eye	Systolic and Diastolic Indentation Values		Blood Pressure
		R_s	R_d	
1	R	6.5	6.9	—
	L	5.8	6.2	
2	R	6.6	6.9	—
	L	6.7	7.0	
3	R	14.1	15.5	—
	L	12.5	13.9	
4	R	6.0	6.4	—
	L	5.7	6.2	
5	R	6.4	6.8	114/86
	L	6.7	7.0	
6	R	5.8	6.2	120/78
	L	5.1	5.5	
7	R	6.4	7.0	120/80
	L	6.2	6.8	
8	R	6.1	6.7	104/54
	L	6.2	6.8	
9	R	6.1	6.8	126/76
	L	6.3	7.0	
10	R	6.1	6.4	132/82
	L	6.4	6.7	
34	R	6.0	6.5	106/72
	L	7.4	7.8	
35	R	7.2	8.3	164/96
	L	4.6	5.9	
36	R	5.7	6.3	134/78
	L	6.5	7.2	
37	R	5.0	5.7	160/86
	L	4.8	5.3	
38	R	6.3	6.9	130/92
	L	6.0	6.6	
39	R	6.0	6.6	140/96
	L	6.6	7.2	

TABLE B
DATA FOR GROUP IV (Subjects 23-33)

Case No.	Eye	Tonometer Weight (g.)	Systolic and Diastolic Indentation Values		Blood Pressure
			R _s	R _d	
23	R L	7.5	10.1 4.0	11.4 4.6	—
24	R L	5.5	5.0 6.8	5.5 7.0	—
25	R L	7.5	5.4 8.4	5.7 8.8	—
26*†	R L	5.5	5.7 7.5	6.3 7.9	158/80
27*‡	R L	5.5	8.0 10.7	8.3 10.9	127/77
28*§	R L	5.5	6.6 8.2	6.8 8.8	134/82
29*	R L	5.5	8.2 7.7	8.5 8.2	110/78
30*	R L	5.5	9.3 8.0	9.5 8.1	132/90
31*	R L	5.5	7.8 6.4	8.3 6.9	140/70
32*	R L	5.5	5.9 6.1	6.4 6.5	190/110
33*	R L	5.5	6.3 5.4	6.8 5.9	162/77

* See Fig. 4 † See Fig. 9 ‡ See Figs 5 and 8 § See Fig. 6

show a greater change in "R" value for the same pressure change in the two eyes. Thus the indentation pulse would be higher in the eye with the low pressure, though the pressure pulses are identical. Similarly, the shape of the pressure-volume relationship for the human eye is such that equal volume change in the two eyes will cause a larger pressure rise in the eye with the higher pressure. Here then, the pressure pulses would differ in size though the same volume would pass into each eye.

Choice of pressure-volume curve for calculations was also difficult. Accurate pressure-volume curves are available for both the living and the enucleated human eye (Langham and Maumenee, 1964; Eisenlohr, Langham, and Maumenee, 1962; Friedenwald, 1957). The difference in shape between these two curves is thought to be due to the presence of expressible blood in the vascular bed of the living but not of the enucleated eye. Since the ocular pulse is caused by phasic variations in the volume of the vascular bed itself, it seems reasonable to assume that compression of portions of the vascular bed occurring with each arterial systole will be less than if the fluid were delivered into the anterior chamber, which is the basis for the construction of the pressure-volume curves. This being so, the somewhat arbitrary assumption was made that the curve required for ocular pulse analysis would be nearest that of the enucleated eye. Thus the Friedenwald curves were used.