Carotid Occlusive Disease: Effect of Complete Occlusion of Internal Carotid Artery on Intraocular Pulse/Pressure Relation and on Ophthalmic Arterial Pressure

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SUMMARY The intraocular pressure (IOP), the intraocular pulse to pressure (pulse/pressure) relationship, and the ophthalmic arterial pressure have been measured in 20 patients with either unilateral or bilateral hemodynamically significant lesions of the internal carotid arteries (ICA) as determined from arteriography. Studies were repeated in 5 of the patients after surgical endarterectomy on the obstructed ICA. In age matched normal subjects the pulse/pressure relations were symmetrical in pairs of eyes, and the ophthalmic arterial systolic pressure was 89.0 ± 2.1 mm Hg; this was 66 ± 1% of the brachial arterial systolic pressure. In 19 of 20 patients with carotid occlusive disease in this study the IOP, pulse amplitudes and the pulse/pressure relationships differed in pairs of eyes. The ophthalmic arterial systolic pressure on the sides with 95 to 100% ICA stenosis was 49.9 ± 0.05 mm Hg, which was 33 ± 3% of the brachial arterial systolic pressure. In the remaining eyes the degree of stenosis of the ipsilateral ICA was 36.5 ± 7.9%; the corresponding eyes had an ophthalmic arterial systolic pressure of 70.1 ± 5.18 mm Hg, which was 45 ± 4% of the brachial arterial systolic pressure. Endarterectomy of the occluded arteries caused a significant increase in the ophthalmic arterial pressure on the ipsilateral side and a smaller increase in the contralateral eye; these changes were associated with a statistically significant increase in the intraocular pulse and improvement in the pulse/pressure relation.

A NON-INVASIVE clinical procedure for measurements of the intraocular pulse to pressure (pulse/pressure) relationship and of the ciliary arterial pressure has recently been described by Langham and To’mey.1 The intraocular pulse reflects the intraocular arteriolar vascular pulse caused by the bolus of blood that enters the eye with each heartbeat. Manometric studies in animals and man have shown the intraocular pulse to disappear at pressures equal to the ophthalmic arterial pressure and have shown that experimentally induced ligation of the common carotid artery caused corresponding decreases in the 2 pressures.2-4 Langham and To’mey used a scleral suction cup to induce increased pressure in the eye and a pressure sensor resting freely against the cornea to record the decrease of intraocular pulse as a function of intraocular pressure. Theoretical and manometric studies have established that the Langham pneumatic tonometer used in the procedure measures both the intraocular pressure and the pulsatile nature of the intraocular pressure with high fidelity.1-4

The procedure of Langham and To’mey does not depend on knowledge of the suction pressure to derive the intraocular pressure as is the case for the alternate procedures of suction ophthalmodynamometry1, 10 and oculopneumoplethysmography.11, 12 The system also differs from the classical procedure of compression (Baillairt) ophthalmodynamometry13, 14 and suction ophthalmodynamometry in that the pulse/pressure relation is derived from measurements based on the pulsation of the ciliary arteries. The classical technique of ophthalmodynamometry is based on visual observations of the pulsatile flow in the retinal arteries.1-14

Changes in the ophthalmic arterial pressure resulting from increasing degrees of stenosis of the internal carotid artery remain to be defined.15-18 In this study we have applied the procedure of Langham and To’mey to measure the intraocular pulse/pressure relation and the ophthalmic arterial pressure in a group of patients with either unilateral or bilateral complete or nearly complete occlusions of the internal carotid arteries, and compared the results to an age-matched group of normal subjects. Studies were repeated in 5 patients following surgical endarterectomy and the results assessed in relation to angiographic and clinical findings.

Materials and Methods

Patients ranging in age from 51 to 79 years were referred from the neurology service of The Johns Hopkins Hospital for ocular examination. These patients later had cerebral angiography, and 20 patients shown to have a stenosis of the internal carotid artery of 95% or more were studied. The results of arteriography were not known at the time the pulse/pressure studies on the eye were performed. The percentage occlusion of the internal carotid artery was based on measurement of the smallest diameter of the lumen through the stenosis compared to the diameter of the internal carotid artery free from post-stenotic dilatation. Age-matched control subjects were
randomly chosen volunteers with no history of ocular or chronic systemic disease.

The intraocular pressure and the intraocular pulse were recorded with either the Alcon Pneumatonograph (Alcon Laboratories, Fort Worth, Texas) or the Digilab Oculocerebrovasculometer (OCVM) (Digilab, Inc., Cambridge, Massachusetts). The former instrument has a single recording channel and the intraocular pressure and the pulse amplitude are read from the record (see figs. 1, 2). The Digilab OCVM instrument has 2 recording channels; the first channel gives a continuous amplified recording of the intraocular pulse which remains centered on the recording chart as the intraocular pressure is increased; the intraocular pressure is recorded digitally on a second channel and the print out is given directly above the corresponding pulse. The pressure sensors are identical for both instruments. The intraocular pressure readings from both systems have to be corrected for curvilinearity of the calibration curve at intraocular pressures exceeding 50 mm Hg. This calibration table has been described. Standardization of the instruments was made daily as described by the manufacturer. The IOP of the subjects was increased using a scleral suction cup connected via polyethylene tubing to a motorized driven syringe (The Langham Pressure Cup System, Digilab Laboratories, Cambridge, Massachusetts). The motorized syringe produced a maximal vacuum of 600 mm Hg and was controlled for increasing and decreasing pressure by a 2 pedal foot control.

**Measurement of Ocular Pulse-Pressure Relationship**

IOPs were recorded on patients in both the seated and supine positions prior to application of the scleral cup. Between 5 and 10 complete pulsations were recorded, and after taking a first set of readings on both eyes the readings were repeated. To apply the cup to the left eye, the patient was asked to look to the right; the cup was placed temporal to the limbus and a negative pressure of 50 mm Hg applied. The patient then observed a fixation light placed at 6 ft. The tonometer was applied perpendicularly to the apex of the cornea and a minimum of 5 pulses were recorded. The recordings were repeated as the IOP was increased using 50 mm Hg increments of negative pressure until the pulse was almost, or completely suppressed. At each pressure increment the ability of the patient to see the fixation light with the tested eye was checked. Once the final recording was made the negative pressure was decreased gradually to zero (during approximately 5 sec) and the cup was lifted from the eye. (The negative pressure was never released suddenly because this could induce conjunctival petechiae.) Finally, a reading of IOP and pulse was

![Diagram of Brachial Blood Pressure 110/70 mm Hg](image_url)

**Figure 1.** The influence of increasing IOP on the pulse amplitude of a normal adult subject. The pulse disappeared at an IOP of approximately 67 mm Hg in both eyes. The brachial arterial pressure was 110/70 mm Hg.

![Diagram of Brachial Pressure 150/90 mm Hg](image_url)

**Figure 2.** The influence of increasing intraocular pressure (IOP) on the pulse amplitude (PA) of a patient with carotid occlusive disease. The stenosis of the right and left internal carotid arteries based on arteriography was 100% and 50%, respectively. The first reading on each eye represents the IOP and the ocular pulse of the undisturbed eye. Note that the pulse in the right eye disappeared at an increased IOP below 40 mm Hg whereas the pulse in the left eye persisted until an IOP of about 60 mm Hg. The brachial arterial pressures were 125/70 mm Hg in the 2 arms.
taken on both eyes. At this time the pulse amplitude and the pulse rate of the untreated eye were approximately equal to the initial values. In the treated eye the tonographic effect of the induced increase of IOP caused the final IOP to be less than its initial value.

The pulse amplitude was defined as the pressure differences between the lowest and highest peaks of the ocular pulse waveform and was calculated from a minimum of 5 complete pulsations. These values and the corresponding IOPs were used to plot the non-linear pulse/pressure relation, and a line of best fit through the individual points was drawn by eye. The point of intersection of this curve with the abscissa (IOP) was read as the ophthalmic arterial systolic pressure. All results are expressed as the arithmetic mean ± the standard error of the mean. The number of experiments is shown in parentheses.

Results

Representative recordings of the intraocular pressure (IOP) and the intraocular pulse in pairs of eyes of a normal subject in the same age group as the patients with occlusive disease is shown in Fig. 1. The IOP and pulse in the undisturbed eyes were equal and this close symmetry persisted as the intraocular pressure was increased stepwise by equal increments of the applied suction pressure. The pulse/pressure relations in the group of age matched normal subjects were not significantly different from the results on young adult subjects. In all individual subjects a close symmetry in the pulse/pressure relations was found in pairs of eyes, and typical examples are included in figure 3. A comparison of results on normal subjects below and above 30 yr of age are summarized in Table 1.

Typical recordings of the IOP and pulse with induced increase of IOP in a patient with carotid occlusive disease is shown in figure 2. On the side with complete occlusion of the ICA the ocular pulse amplitude decreased rapidly with increased IOP and was completely suppressed at 40 mm Hg. At this pressure the patient noted dimming and loss of vision which returned immediately upon our decreasing the IOP. On the side with 50% stenosis of the internal carotid artery, the ocular pulse was present at IOPs of 40 and 50 mm Hg but was extinguished at 60 mm Hg; in this eye full vision was sustained at IOPs of 40 and 50 mm Hg. The pulse/pressure relations in 19 of the 20 patients with carotid occlusive disease were asymmetric in pairs of eyes.

Table 2 summarizes the ocular findings compared to the arterial angiographic findings in the 20 patients with carotid occlusive disease. Asymmetry in either the IOP or both IOP and pulse was found in all pairs of eyes, and in 12 of the 40 eyes the IOPs were abnormally low (10 mm Hg or less). The ophthalmic arterial pressure (OAP) on the side with 95 to 100% internal carotid stenosis was 49.9 ± 4.05 (25) mm Hg, and the corresponding brachial arterial systolic pressure (BrAP) was 155.5 ± 6.13 (20) mm Hg. In 17 of the 20 patients the ophthalmic arterial pressures were significantly different in pairs of eyes. In the remaining 3 patients the differences in the ophthalmic arterial pressures in pairs of eyes were 3, 8, and 3 mm Hg, and in each patient the results of arteriography indicated a similar degree of stenosis in both internal carotid arteries. The difference in the ophthalmic pressures in pairs of eyes for all 20 patients was 24.1 ± 3.03 (20) mm Hg.

In 25 individual eyes ipsilateral to an ICA stenosis of 95 to 100% the ratio OAP/BrAP was 0.33 ± 0.03

![Figure 3](http://stroke.ahajournals.org/)

**Figure 3.** The pulse/pressure relationship in the eyes of a patient with carotid occlusive disease (broken line) compared to a typical pulse/pressure relationship in eyes of a normal subject (continuous line). The open and closed circles represent the right and left eyes, respectively. In the patient with arterial occlusive disease, arteriography indicated 90% and 70% stenoses of the right and left internal carotid arteries, respectively; the brachial arterial pressure in both arms was 135/100 mm Hg. The brachial blood pressure of the normal subject was 135/95 mm Hg.

### Table 1

**Summary of Results on 2 Groups of Normal Subjects.** IOP Represents Intraocular Pressure, PA is the Pulse Amplitude, OAP is the Ophthalmic Arterial Systolic Pressure, BrAP is the Brachial Arterial Systolic Pressure. Results on Patients Less than 30 Years of Age are Taken from Langham and To'omey.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Age &lt; 30 yr</th>
<th>Age &gt; 30 yr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age</td>
<td>24.3 ± 1.25 (10)</td>
<td>55.85 ± 3.55 (13)</td>
</tr>
<tr>
<td>IOP (O.D.) mm Hg</td>
<td>16.0 ± 0.54 (10)</td>
<td>16.8 ± 0.63 (13)</td>
</tr>
<tr>
<td>IOP (O.S.) mm Hg</td>
<td>16.3 ± 0.56 (10)</td>
<td>17.2 ± 0.61 (13)</td>
</tr>
<tr>
<td>P.A. (O.D.) mm Hg</td>
<td>2.15 ± 0.15 (10)</td>
<td>2.2 ± 0.12 (13)</td>
</tr>
<tr>
<td>P.A. (O.S.) mm Hg</td>
<td>2.15 ± 0.15 (10)</td>
<td>2.2 ± 0.14 (13)</td>
</tr>
<tr>
<td>O.A.P. (O.D.) mm Hg</td>
<td>80.8 ± 2.3 (10)</td>
<td>86.4 ± 2.8 (13)</td>
</tr>
<tr>
<td>O.A.P. (O.S.) mm Hg</td>
<td>82.8 ± 2.6 (10)</td>
<td>89.7 ± 3.2 (13)</td>
</tr>
<tr>
<td>Br.A.P. (syst) mm Hg</td>
<td>128 ± 8.6 (10)</td>
<td>134 ± 6.6 (13)</td>
</tr>
<tr>
<td>Br.A.P. (diast) mm Hg</td>
<td>77.2 ± 5.2 (10)</td>
<td>79.4 ± 8.0 (13)</td>
</tr>
<tr>
<td>Ratio O.A.P./Br.A.P.</td>
<td>0.67 ± 0.01 (20)</td>
<td>0.67 ± 0.01 (26)</td>
</tr>
</tbody>
</table>
Clinical descriptions of 2 of these patients which are OAP/BrAP in pairs of eyes is summarized in table 3, history of basilar vertebral insufficiency. She complained of weakness in the left upper extremity present for 2 weeks. The patient had been hospitalized 1 and 3 months previously after experiencing "blackout" spells lasting 15 and 20 min. With the patient supine, the intracocular pressures were 9 (O.D.) and 11 (O.S.) mm Hg and the corresponding pulse amplitudes were 0.8 (O.D.) and 1.0 (O.S.) mm Hg. On increasing the intracocular pressure the pulse disappeared at 25 mm Hg in the right eye and at 45 mm Hg in the left eye; the left and right brachial blood pressures were 140/80 mm Hg. The results indicated a marked arterial stenosis on the right side and a lesser but still severe stenosis in the ipsilateral ICAs.

Effect of Endarterectomy

Five patients had corrective surgery on the stenotic ICA, and the effect of the operation on the ratio of OAP/BrAP in pairs of eyes is summarized in table 3, and a representative record of the intraocular pulses prior to and following surgery is shown in figure 5. Clinical descriptions of 2 of these patients which are representative of the 5, are as follows:

Patient 1. A 63-year-old white female had a past history of basilar vertebral insufficiency. She complained of weakness in the left upper extremity present for 2 weeks. The patient had been hospitalized 1 and 3 months previously after experiencing "blackout" spells lasting 15 and 20 min. With the patient supine,
arterial stenosis on the left side. Cerebral angiograms revealed 95% stenosis of the right ICA at its origin. The right carotid siphon and ophthalmic artery filled by retrograde flow. The left carotid arteriogram showed a 60 to 70% narrowing of the lumen of the ICA at the bifurcation of the common carotid artery. Endarterectomy was performed on the right ICA. The ocular examination was repeated 5 months following surgery. In the supine position the intraocular pressures were 16 mm Hg in both eyes and the pulse amplitudes were 1.5 (O.D.) and 1.7 (O.S.) mm Hg. With increasing intraocular pressure, the pulse amplitude disappeared at 64 mm Hg in the right eye and at 58 mm Hg in the left eye; the brachial blood pressures were 155/100 mm Hg. Visual fields were normal in both eyes. The patient improved clinically. Carotid arteriography made 2 years following surgery showed a narrowing of 30% in the right ICA. The left ICA appeared unchanged.

Patient 2. A 53-year-old white male had been in good health until 3 months before examination when he first experienced dizziness and unsteadiness during manual work. This was followed by numbness and weakness in the left arm. Vision was normal. In the supine position, the intraocular pressures were 11 (O.D.) and 14 (O.S.) mm Hg and the corresponding pulse amplitudes 1.0 (O.D.) and 1.5 (O.S.) mm Hg. The intraocular pulse disappeared at 27 mm Hg (O.D.) and at 52 mm Hg (O.S.). The brachial blood pressures were 135/80 (RA) and 130/85 (LA) mm Hg. The subject noted visual loss at these pressures. These results indicated a severe arterial stenosis on the right side and a lesser but significant stenosis of the internal carotid artery on the left side. Visual fields were normal. Carotid arteriograms revealed 95% stenosis of the right ICA and an atherosclerotic plaque in the left ICA near the origin, causing a 40% stenosis. Right IC endarterectomy was performed. Ocular examination 5 months after surgery showed the intraocular pressures to be 16 mm Hg in both eyes; the pulse amplitudes were 1.0 (O.D.) and 1.2 (O.S.) mm Hg; the brachial arterial pressures were 140/90 mm Hg. The intraocular pulse disappeared at 64 mm Hg in the right eye and at 61 mm Hg in the left eye. These results indicate a significant improvement in the ophthalmic arterial pressure on the operated side. At this time, cerebral angiography indicated a 40% stenosis of the right common carotid artery below the bifurcation. The

Figure 4. The relationship between percentage occlusion of the internal carotid artery (evaluated from arteriography), and the ratio of ophthalmic arterial pressure to brachial systolic arterial pressure. The filled-in circles are results from the patients with occlusive disease, and the open circles are results from normal subjects.

Figure 5. Comparison of intraocular pulse in the 2 eyes of a patient 2 months before (upper records) and after right internal carotid endarterectomy (lower records). Arteriography revealed occlusions of 90% and 100% of the right and left internal carotid arteries, respectively, prior to surgery. Arteriography 2 months after unilateral surgery revealed occlusion of 0% and 100% in the same arteries (patient 4, table 3).
plaque, near the origin of the left ICA, was essentially unchanged. Left endarterectomy was performed at this time and the recovery was uneventful.

Discussion

The normal subjects in this study did not have arteriography and it may be questioned whether it was justified to consider this group as having patent arteries. The symmetry of all the measured parameters in pairs of eyes, including IOP, pulse amplitude, pulse/pressure curves, and the ophthalmic arterial pressures, make it highly probable that these patients were free of significant carotid occlusive disease.

In the present group of patients, stenosis or complete occlusion of one or both internal carotid arteries was characterized by a marked decrease of the ophthalmic arterial pressure as determined by the Langham pneumatic technique and an abnormally low value of the ratio OAP/BrAP. In the eyes ipsilateral to either a complete occlusion or to a high grade stenosis this ratio ranged from 0.12 to 0.41 with a mean value of 0.25 ± 0.02 (14). The corresponding ratios in normal subjects ranged from 0.54 to 0.86 with a mean of 0.67 ± 0.01 (46) which is significantly higher than found in the patients with severe stenosis (p < 0.001).

A well defined difference of patients with a complete occlusion or medium grade stenosis of the internal carotid artery versus normal subjects was also seen in the form and symmetry of the pulse/pressure relation and in the symmetry in the IOP in pairs of undisturbed eyes. The ocular pulse/pressure curves in normal subjects of younger and older age groups were similar and agreed closely with those found in young adults in a previous study. The normal pulse/pressure relation has a sigmoid shape in which the initial phase is characterized by rapid decrease of pulse amplitude when the IOP is experimentally increased 10 to 15 mm Hg above its normal mean value; a second rapid decrease of ocular pulse amplitude to zero occurs when the IOP approaches within 10 to 15 mm Hg of its normal mean value; a second rapid decrease of ocular pulse amplitude to zero occurs when the IOP is experimentally increased 10 to 15 mm Hg above its normal mean value; a second rapid decrease of ocular pulse amplitude to zero occurs when the IOP approaches within 10 to 15 mm Hg of its normal mean value.

In patients with either a complete occlusion or a high grade stenosis of the internal carotid arteries investigated in this study, the decrease in ophthalmic arterial pressure resulted in a compression of the ocular pulse/pressure curve and a loss of the plateau; thus, the pulse amplitude decreased proportionately with increased IOP. Marked asymmetry of the shape of the pulse/pressure curves in pairs of eyes was present in 19 of the 20 patients with carotid occlusive disease. The results on the remaining patient differed from the other 19 patients. This patient had extracranial vascular disease associated, perhaps, with fibromuscular hyperplasia in both arteries. She was obese but had been in good health until she suddenly developed severe right side headache and a left hemiparesis. Arteriography revealed a marked narrowing of both internal carotid arteries 1.5 cm distal to the origin with the common carotid arteries, with 95 to 100% stenosis in both arteries. There was reconstitution of both carotid arteries in the region of the carotid siphon which would explain the normal intraocular pressure, the symmetry of the ocular pulses, and the normal form and symmetry of the pulse/pressure curves.

To comprehend fully the circulatory information revealed by the pulse/pressure relation it is pertinent to consider the anatomic and physiologic parameters determining the relation. Both the steady state intraocular pressure and the intraocular pressure pulse are derived from the ciliary arterial circulation which originates from the ophthalmic artery. This system includes the long and short posterior ciliaries and the anterior ciliary arteries, which supply nine-tenths of the total ocular blood flow. The steady state intraocular pressure is itself dependent on formation of aqueous humor which is proportional to the vascular perfusion of the ciliary body. The retinal artery also originates from the ophthalmic artery but it provides only one-tenth of the total ocular blood flow, and does not contribute significantly to the intraocular pulse amplitude.

The results of ocular studies made on patients before and after unilateral endarterectomy revealed clinically significant improvement in the ophthalmic arterial pressure and the pulse/pressure relation in both the eye on the operated side and to a lesser degree in the opposite eye. These improvements were consistent with the sustained clinical recovery from the neural deficit.

Acknowledgment

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References

Transient Ischemic Attacks and Strokes with Recovery Prognosis and Investigation


SUMMARY This study analyzes 234 patients who recovered from an initial ischemic episode. The object was to see if the duration of the first episode influenced the chance of finding a treatable lesion or the chance of a further episode. The initial episodes varied from less than 5 minutes to longer than 3 weeks. There seemed to be no fundamental difference between transient ischemic attacks (TIAs) (less than 24 hours) and strokes which recover. However, 51% of those whose initial episode lasted less than 5 minutes had a subsequent stroke compared to 28% of those with an initial episode of more than 24 hours duration. Thirty percent of the former group who had angiograms had an operable lesion against 10% in the latter group.

It seems that angiography has sufficiently high yield to be warranted in all patients where the initial attack lasted less than 30 minutes. In those with longer attacks the yield from angiography was much lower and non-invasive techniques should be considered in these patients, where available, prior to consideration for angiography.

Investigation should be based on the degree of functional recovery and not on the arbitrary time division which normally divides TIAs and strokes.

Bruits were the most reliable clinical indicators of stenosis. However the presence of intermittent claudication, hypertension and age over 50 were all more common in those with carotid stenosis.

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There have been many studies concerned with the natural history, investigation, and management of transient ischemic attacks (TIAs). These were comprehensively reviewed by Brust in 1977.1 Transient ischemic attacks are defined as focal neurological deficits of vascular origin, rapid in onset, with complete recovery in less than 24 hours.2 While the majority of authors have accepted the definition of a TIA as lasting up to 24 hours, some have taken a shorter duration. Acheson and Hutchinson,3 for example, considered TIAs to be any event lasting up to 1 hour while others have taken periods as short as 30 minutes.4 Not all patients whose signs or symptoms last longer than 24 hours are left with a permanent deficit. The Stroke Program at the National Institutes of Health, Bethesda, Maryland, recommends that those who recover within 21 days be considered to have had reversible ischemic neurological deficits (RIND).5

The clinical features of TIAs are, by definition, completely reversible and because of this have not usually been considered to be associated with any structural damage. However, there is evidence that this may not always be the case. For instance, while the CT scan is usually normal, it is known that infarcts are sometimes seen after what was believed clinically to be a TIA.6 Cerebral blood flow studies in TIA may remain focally abnormal for as long as 90 days after the last clinical event.7

The present study sought to determine the significance of the duration of the first ischemic disturbance with regard to its cause and the prognosis for further episodes. The question asked was: Is there any

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