Ophthalmodynamometry in Internal Carotid Artery Occlusion

OLAF B. PAULSON, M.D.

SUMMARY Retinal artery pressure was measured by ophthalmodynamometry in 15 patients with occlusion of the internal carotid artery in its extracranial part. Nine of the patients had severe neurological deficit whereas the remaining six had slight or intermittent symptoms. Retinal artery pressure was reduced on the side of the internal carotid artery occlusion in all patients studied. Near-zero low diastolic retinal artery pressure on the affected side was a common finding among patients with severe deficit and was also seen in some patients with slight deficit. Its presence strongly suggests occlusion of the ipsilateral internal carotid artery.

MEASUREMENT of the retinal artery pressure by ophthalmodynamometry is commonly used in the evaluation of patients with cerebrovascular diseases. The interest has been especially to find abnormalities indicating stenotic lesions in the carotid artery in patients with transient ischemic attacks (TIAs), as such lesions might be surgically accessible.1 If pressure measurements are 20% or more lower on one side than on the other, ipsilateral carotid artery stenosis or occlusion is likely to be present. However, when a stenotic lesion of the carotid artery gives neurological symptoms this is most often due to an ulcerative plaque on which emboli are formed and later detached, and in these instances the retinal artery pressure need not be reduced. On the other hand, if the internal carotid artery is occluded in the neck, and if collateral circulation is insufficient, one might expect to find a significantly reduced pressure in the ipsilateral retinal artery as this artery is a side branch of the upper part of the internal carotid artery. If neurological symptoms are severe, a marked reduction of the retinal artery pressure could be expected. The present study was undertaken in order to evaluate these aspects of ophthalmodynamometry further.

Methods

Fifteen patients were studied. All had angiographically demonstrated occlusion of one of the internal carotid arteries in its extracranial part in the neck. Nine of the patients had severe neurological deficits (table 1) whereas the remaining six had slight or intermittent deficits (table 2). Retinal artery pressure was measured by an ordinary ophthalmodynamometer ad modum Bailliard as gram pressure which had to be applied to the ocular bulb in order to collapse the retinal arteries in the diastole and in the systole (in the tables indicated as systolic pressure/diastolic pressure). However, only diastolic values were measured in several instances as systolic readings are less accurate and more difficult, especially in patients who are somewhat unsteady as were many in the present study.

Results

The nine patients with severe neurological deficits were all studied supine. They all had a retinal artery pressure lower on the side of the occluded internal carotid artery than on the other (table 1). A characteristic finding present in five patients was a very low diastolic retinal artery pressure on the affected side of less than 8 gm (the lowest pressure which could be measured accurately with the ophthalmodynamometer used). Another patient (Case 7) had an ophthalmoscopic picture of retinal artery thrombosis. Spontaneous retinal artery collapse in the diastole was not observed. Only three of the nine patients studied had retinal artery pressure above 8 gm on the affected side.

In the group of six patients with slight neurological deficit (table 2) one (Case 11) had had a traumatic lesion of one eye and therefore could be studied only on the affected side. Otherwise all patients had lower diastolic and systolic retinal artery pressures on the side of the internal carotid artery occlusion than on the other, when studied both supine and erect. One patient (Case 12), however, had very low (< 8 gm) diastolic retinal artery pressure on both sides suggesting bilateral carotid artery disease, but angiogram was performed only on the symptomatic side. Studied in the supine position only, one of the six patients had a diastolic retinal artery pressure of less than 8 gm. However, such a low pressure could be provoked by the erect posture in two of four patients studied in this position.

Discussion

The results of the present study demonstrate reduced retinal artery pressure on the side of the occluded internal carotid artery in all patients examined. In the group of patients with severe neurological deficit near zero (< 8 gm) but not zero, low diastolic pressure was a common finding and such low pressures were also encountered in some of the patients with slight neurological deficit. The pressure in the retinal artery in cases of internal carotid artery occlusion will depend on the collateral circulation; if neurological deficit occurs, the collateral circulation must be insufficient and a very low diastolic pressure in the retinal artery can be expected. In fact, the blood flow to an organ will stop when the diastolic blood pressure and the extravascular pressure reach each other. Thus, if the "true" diastolic retinal artery pressure falls down to (or below) the intraocular pressure, i.e., the retinal artery pressure measured by ophthalmodynamometry falls to zero, then the retinal circulation stops and a picture of retinal infarction similar to that of central retinal artery thrombosis can be expected. Such an appearance of the retina in internal carotid artery occlusion has been reported, and one of the patients in the present study had signs of central retinal artery thrombosis. But this might be due to an extension of the carotid occlusion to the retinal artery.

In some patients near-zero low diastolic retinal artery pressure was observed only in the erect posture and not when supine. Similar observations of the influence of posture have
Table 1. Ophthalmodynamometry in Patients With Extracranial Internal Carotid Artery Occlusion Resulting in Severe Neurological Deficit of Acute Onset

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age/sex</th>
<th>Side of carotid occlusion</th>
<th>Retinal artery pressure</th>
<th>Principal clinical symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42/M</td>
<td>L</td>
<td>/20/100</td>
<td>Severe R hemiparesis and aphasia</td>
</tr>
<tr>
<td>2</td>
<td>51/M</td>
<td>R</td>
<td>/&lt;8/30</td>
<td>Severe L hemiparesis</td>
</tr>
<tr>
<td>3</td>
<td>54/F</td>
<td>L</td>
<td>/&lt;8/20</td>
<td>Severe R hemiparesis and aphasia</td>
</tr>
<tr>
<td>4</td>
<td>58/M</td>
<td>L</td>
<td>/&lt;8/28</td>
<td>Severe R hemiparesis and aphasia</td>
</tr>
<tr>
<td>5</td>
<td>60/M</td>
<td>R</td>
<td>/25<em>35</em></td>
<td>Severe L hemiparesis</td>
</tr>
<tr>
<td>6</td>
<td>65/M</td>
<td>R</td>
<td>/&lt;8/25*</td>
<td>Severe L hemiparesis</td>
</tr>
<tr>
<td>7</td>
<td>68/M</td>
<td>R</td>
<td>/ Retinal artery thrombosis</td>
<td>Moderate R hemiparesis and severe aphasia</td>
</tr>
<tr>
<td>8</td>
<td>70/M</td>
<td>R</td>
<td>39/&lt;8/27</td>
<td>Moderate L hemiparesis</td>
</tr>
<tr>
<td>9</td>
<td>88/F</td>
<td>L</td>
<td>/27/42</td>
<td>Severe R hemiparesis</td>
</tr>
</tbody>
</table>

All measurements are performed in the supine position. When studied in sitting posture instead of supine /18, /28. L = left; R = right.

been reported by another author. The change from supine to erect posture ordinarily results in a blood pressure fall in the major arteries of the head ("systemic blood pressure fall") which is only about the difference in hydrostatic pressure between the heart and the head. Thus, in the patients mentioned the relative fall of retinal artery pressure was much more pronounced than the fall of systemic blood pressure. This difference may be explained by the following mechanisms. First, the zero pressure of the retinal artery pressure measured by ophthalmodynamometry and of the systemic arterial pressure is not the same, the former being the intraocular pressure higher than the latter. Secondly, and maybe even more important, the response to a blood pressure drop will in normal tissue be a vasodilation and maintenance of blood flow, but in areas of diseased or marginally low perfused tissue this response may be abolished (lost autoregulation). Thereby an even small systemic blood pressure fall may result in a shunting of the blood flow away from the diseased toward the non-diseased tissue and thus in a marked fall of the blood pressure in the diseased tissue, e.g., in the retinal artery pressure.

Near-zero low diastolic retinal artery pressure or retinal appearance on infarction with absent pulse has been reported in only a few patients with cerebrovascular attacks with internal carotid artery occlusion, and has not been observed in the majority of such patients as well as not in patients with stenosis of the internal carotid artery. The more frequent finding of near-zero low retinal artery pressure in the present study of patients with occlusion of the internal carotid artery is probably due to the selection of the patients with very severe neurological deficit of acute onset. Near-zero low diastolic retinal artery pressure normally is not observed in stenosis of the internal carotid artery. Therefore, the finding of such a low retinal artery pressure on the affected side in a patient with an acute stroke is strongly suggestive of an occlusion of the internal carotid

Table 2. Ophthalmodynamometry in Patients With Extracranial Internal Carotid Artery Occlusion Resulting in Slight or Intermittent Neurological Deficit

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age/sex</th>
<th>Side of carotid occlusion</th>
<th>Posture</th>
<th>Retinal artery pressure</th>
<th>Principal clinical symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>41/M</td>
<td>R</td>
<td>Supine</td>
<td>/22/34</td>
<td>L paresthesia and slight hemiparesis. Pulse synchronous headache, maximal over L ear. Murmur over L carotid artery and L part of forehead, maximal over the orbit. Angiographical examination had revealed occlusion of the R internal carotid artery in the neck and a severe stenosis in the siphon of the L one. During some months several attacks of R-sided clumsiness and weakness. Normal neurological examination.</td>
</tr>
<tr>
<td>11</td>
<td>56/M</td>
<td>L</td>
<td>Supine</td>
<td>47/25/28</td>
<td>Blind</td>
</tr>
<tr>
<td>12</td>
<td>59/M</td>
<td>R</td>
<td>Supine</td>
<td>30/&lt;8/46/&lt;8</td>
<td>Aphasia, dementia and slight weakness and hyperreflexia in L arm. Probably bilateral carotid artery disease; however, angigram performed only on R side.</td>
</tr>
<tr>
<td>13</td>
<td>60/M</td>
<td>L</td>
<td>Supine</td>
<td>77/37/110/47</td>
<td>During half a year clumsiness of R hand. Examination revealed a slight R hemiparesis.</td>
</tr>
<tr>
<td>14</td>
<td>63/M</td>
<td>L</td>
<td>Supine</td>
<td>42/20/113/51</td>
<td>Slight R hemiparesis of a few months' duration.</td>
</tr>
<tr>
<td>15</td>
<td>67/M</td>
<td>R</td>
<td>Supine</td>
<td>80/46/100/54</td>
<td>History of 3-4 attacks of L-sided weakness lasting 5-30 min. Normal neurological examination.</td>
</tr>
</tbody>
</table>

L = left; R = right.
artery in the neck. Obviously the pressure is not always so low and will depend on the collateral circulation which may be much better to the eye than to the brain. On the contrary, one might expect to find a near-zero low diastolic retinal artery pressure in some patients with a stroke without carotid artery occlusion but with a retinal artery stenosis or thrombosis (near-zero low pressure is not the ordinary finding in this disease) or with a unilateral glaucoma with a markedly increased intraocular pressure. These situations are, however, undoubtedly exceptional.

The clinical consistency of ophthalmodynamometry is symptomatic carotid artery occlusion and can be summarized as follows: Retinal artery pressure is practically always lower on the side of the occlusion than on the other. The diastolic retinal artery pressure is often near zero, low on the side of the occlusion. The finding of a near-zero low diastolic retinal artery pressure nearly always indicates an ipsilateral occlusion of the internal carotid artery.

**References**


**Effect of Intracarotid Prostaglandin E$_1$ on Regional Cerebral Blood Flow in Man**

**JES OLESEN, M.D.**

**SUMMARY** The effect of prostaglandin E$_1$ on regional cerebral blood flow (rCBF) was studied with the intra-arterial $^{133}$Xe method in ten awake patients under local anesthesia. Measurements were taken from 16 areas of a hemisphere in seven patients, from 35 areas of a hemisphere in two patients and from 256 areas of a hemisphere in one patient. The prostaglandin was dissolved from the crystalline state without the aid of alcohol. It was given intracarotidly as a constant infusion at a rate of 5 ng per kilogram per minute for five minutes before the measurement, and continued during the measurement. In every patient a mild increase in blood flow during the prostaglandin infusion was seen. The flow increase took place in all parts of the hemisphere. It averaged 11.2% (p < 0.01). During the infusion, the skin supplied by the internal carotid artery and the conjunctiva on the infused side became red and sometimes swollen. A slight pressure was noted by most patients, but none had pain. No side effects of the infusion were noted.

**THE PROSTAGLANDINS** are a large family of naturally occurring substances with a variety of biological actions. In a number of organs they are vasoactive. Prostaglandin E$_1$ is known to markedly increase blood flow to human skin and muscle.¹

The effect of prostaglandin E$_1$ on cerebral blood flow (CBF) and pial vessel diameter has been studied previously in animals. Published results, however, have been contradictory.²⁻⁴ There is rapidly growing interest in the widespread and important physiological actions of prostaglandins. Recently, it has been suggested that prostaglandins liberated from the lungs may play a part in the pathogenesis of migraine attacks.⁵ To possibly resolve the above-mentioned discrepancies and to gain knowledge of the prostaglandin action in humans, it was decided to study the effect of continuous infusion of prostaglandin E$_1$ on the regional cerebral blood flow (rCBF) in awake unanesthetized man.

**Methods**

Ten patients were studied. All patients were admitted to the neurological department for investigation of neurological symptoms. Clinical data are reported in table 1. In all patients diagnostic arteriography was indicated, and in conjunction with this the CBF measurements were performed. Informed consent was obtained. The patients were studied in the morning, fasting and without premedication. After local anesthesia, a catheter was percutaneously placed in the internal carotid artery by means of the Seldinger technique. rCBF was measured with the intracarotid $^{133}$Xe injection technique as described elsewhere.⁶ Ten minutes after the resting state study, intracarotid infusion of prostaglandin E$_1$ was started, and maintained for five minutes prior to the CBF determination. The

**References**

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