RETINAL ARTERY BLOOD PRESSURE MEASUREMENTS IN DIAGNOSIS AND SURGERY OF SPONTANEOUS CAROTID OCCLUSIONS

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RECENT interest in vascular surgery of atherosclerosis has been extended to the carotid system. Undoubtedly, the possibility of surgical relief of spontaneous carotid occlusions will be explored further despite the relatively few encouraging reports as yet available. The comparatively bad prognosis of the untreated lesion makes early diagnosis of the occlusions mandatory if therapy, either medical or surgical, is to be effective before important nervous function is lost. Comparative studies of retinal artery pressure can materially and safely aid the physician in diagnosis of these vascular occlusions if carefully integrated in diagnostic judgment.

While the determination of retinal artery blood pressure or ophthalmodynamometry is subject to limitations so far as absolute values are concerned, there is good evidence for its validity in comparing pressures in the two eyes. From differences in such readings one is entitled to make inferences in regard to comparative pressures in the parent internal carotid arteries.

In a normal carotid system pressure in the major intracranial branches (taken by direct measurement) is almost equal to that in the cervical internal carotid. The pressure gradient downward from large to small vessels is uniformly progressive but the over-all drop is not great. Bakay and Sweet have found after clamping the common carotid that the absolute drop in pressure throughout the carotid system on the same side is marked but the gradient of drop remains about the same as that before occlusion. It seems reasonable to assume that pressure in the ophthalmic and retinal arteries will parallel that in comparable branches of the carotid tree and will approximate the pressure in the internal carotid artery on the same side. Rand has in one case compared the retinal artery pressure readings with directly measured pressure in the internal carotid artery and found a rather close correlation.

OPHTHALMODYNAMOMETRY

The technique of ophthalmodynamometry generally used is that of Bailliart. It was described by Magitot in 1932 and elaborated by Weigelin and Müller. This relatively simple clinical procedure has been illustrated and described in detail by Thomas and Petrohelos, Svien and Hollenhorst, and by Heyman et al. The ophthalmodynamometer is a small cylinder with
spring-loaded plunger and attached rod with footplate for application of pressure to the optic globe. The plunger is calibrated in grams.

After anesthetizing the conjunctivae and dilating the pupils, the footplate is placed against the eyeball at the outer canthus and pressure is applied progressively while the operator observes the major branches of the retinal artery. The diastolic pressure reading is that which is associated with the first visible distinct pulsation of the vessels, while the systolic pressure is that which is associated with collapse of the vessels. The readings in grams can be converted to millimeters of mercury by use of a conversion table which takes into account the already existing intraocular pressure. Since the direct readings in grams under conditions of normal intraocular pressure correspond closely to those for millimeters of mercury in the conversion table and since we are concerned primarily with comparative measurements in the two eyes, we have recorded only the figures taken directly from the instrument scale.

Retinal artery pressures have been measured in healthy adults and in patients with neurological diseases not associated with apparent vascular insufficiencies. Thomas and Petrohelos reported that in their subjects the difference in diastolic pressures in the two eyes ranged from 0 to 15 per cent of the highest figure while the difference in systolic pressures ranged from 0 to 12.9 per cent. The average of the percentage differences in a larger number of subjects was 5.2 for diastolic and 3.3 for systolic pressures.

OPHTHALMODYNAMOMETRY AS AN AID IN THE DIAGNOSIS OF SPONTANEOUS OCCLUSION OF THE CAROTID ARTERIES

In analyzing the ischemic brain syndrome in 107 proved cases of spontaneous carotid occlusion, Johnson and Walker found that the locus of obstruction was at the origin of the internal carotid artery in 81 of 97 cases in which this artery alone was involved. Many of these patients were suspected of harboring an intracranial tumor. It is evident that some method of clinical examination that might provide diagnostic information relating to occlusion of the internal carotid artery, in lieu of or at least before angiography, would be quite welcome. Ophthalmodynamometry does appear to be valuable in this regard.*

Thomas and Petrohelos reviewed 19 cases of carotid occlusion from various causes. Only 4 failed to show a significantly lower pressure in the retinal artery on the side of the occlusion. The average percentage difference between the two sides for diastolic pressure was 31.7 and for systolic pressure was 24.1. Svien and Hollenhorst recently reported 4 cases of unilateral thrombosis of the internal carotid in which they found the difference in diastolic retinal artery pressures on the two sides to range from 25 to 59

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* We have repeated previously performed studies of the effect of digital compression of the common carotid artery on ipsilateral retinal artery pressures and have found that this maneuver causes an average acute drop of 50 per cent in both systolic and diastolic readings. There was considerable variation among the several subjects. Ophthalmodynamometry is of value in the management of patients undergoing therapeutic carotid ligations.
per cent. They consider a comparison of diastolic pressures more reliable than that of systolic pressure on the two sides. They noted, however, that if the diastolic pressure in both eyes is less than 20 grams, any difference in the pressure readings on the two sides is not to be considered reliable for interpretation.

Heyman et al.\textsuperscript{12} in 16 cases of partial or complete occlusion of an internal carotid artery (verified by angiography in 14 of the cases) found the mean decrease in retinal artery pressure to be 27 per cent for the diastolic and 30 per cent for the systolic pressure on the side of the occlusion. They believe that a reduction of 25 to 30 per cent in both systolic and diastolic retinal artery pressures is diagnostic of impaired carotid circulation on the side of the lower reading. They pointed out that any conclusion to be drawn from the comparative studies of pressures in the two eyes is based on the assumption that the carotid circulation on one side is relatively normal. They believe, as we do, that both systolic and diastolic readings are important. Some of their patients with impaired carotid circulation showed a decrease in systolic readings only.

Weigelin and co-workers\textsuperscript{20} found that the diagnosis of internal carotid thrombosis could be made by the aid of dynamometry in 6 cases, 2 of which were verified by operation. In 1 case angiography did not show filling on one side and there was no corresponding pressure difference.

When it is realized that thrombosis of the cervical internal carotid is in some instances amenable to surgery and that in such cases early diagnosis and prompt efforts at alleviation of the obstruction are important, then the information provided by a comparative measurement of retinal arterial pressures on the two sides becomes of considerable significance. We are submitting our findings in 3 cases of internal carotid thrombosis. The diagnosis was confirmed by direct exploration in 2 cases and by angiography in the third. The differences in retinal artery pressures on the two sides in all 3 cases were significant and much greater than the range of differences in normal subjects. Retinal artery pressures and other data relating to these cases are given in Table 1. All measurements were made by the same observer (F.C.B.).

Case 1 (27353). R.E.B., a 65-year-old white male clerk, was admitted to the Veterans Administration Hospital, Iowa City on Nov. 20, 1956. He had been under care for arterial hypertension for 4 years but felt well until Nov. 6, 1956 when he noted loss of grip of the left hand and weakness of the arm. The following day the left arm was "useless." Later he experienced transitory numbness of the left side of the body, face and tongue. There were brief "jerking" spells beginning in the left hand and spreading to the arm, shoulder and leg with turning of the head to the right. There were no headaches or visual symptoms.

Examination. The patient was alert and oriented. The left arm hung at his side but the gait was normal. The brachial blood pressure was 220/110 mm. of Hg. The heart was moderately enlarged with a grade 2 systolic murmur. The chest was clear and abdomen was normal. The pulses in the legs were of poor amplitude. The common carotid pulses in the neck were normal. There was left facial palsy. The fundi displayed numerous superficial small hemorrhages, more on the right, and arteriolar
TABLE 1

Retinal artery pressures* in patients with thrombotic occlusion of the internal carotid artery

<table>
<thead>
<tr>
<th>Case</th>
<th>Clinical Data</th>
<th>Retinal Artery Pressure in Grams</th>
<th>Brachial Arterial Pressure, mm. of Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Presumed Normal Side</td>
<td>Occluded Side</td>
</tr>
<tr>
<td>1. R.B. Age 65 Male</td>
<td>Slowly and intermittently increasing hemiparesis on left with focal seizures on left. Paralysis increased under observation. High internal carotid occlusion established at surgery. Diabetic</td>
<td>82/26</td>
<td>42/12</td>
</tr>
<tr>
<td>2. F.A. Age 61 Male</td>
<td>Stepwise progression of right hemiparesis and dysphasia—becoming stable. Occlusion of internal carotid above bulb relieved by intimeectomy. Sudden increase in paralysis 5 hrs. after surgery. Propagating clot and possible embolism</td>
<td>86/44</td>
<td>50/32</td>
</tr>
<tr>
<td>3. F.K. Age 57 Female</td>
<td>Confusion, lethargy, slurred speech, right hemiparesis increasing in hospital. Occlusion of left internal carotid near origin found by angiography. (Readings 1 yr. after diagnosis established)</td>
<td>72/32</td>
<td>50/24</td>
</tr>
</tbody>
</table>

* Intraocular pressures were within normal limits and essentially equal in the two eyes in all cases.

sclerosis. Strength of the left arm was reduced and grip was minimal. The right arm and both legs functioned well. He had hyperreflexia on the left without Babinski sign, and hypesthesia of the left hand.

Urinalysis revealed sugar, and elevated blood sugar readings confirmed the diagnosis of diabetes mellitus which was controlled with insulin and diet. Routine blood counts were normal. The blood VDRL and spinal fluid Kolmer tests were negative. Lumbar puncture disclosed a pressure of 160 mm. of water. The fluid was clear with 2 white blood cells/c.mm.; total protein was 40 mg. per cent. Roentgenograms of skull and chest were regarded as within normal limits except for left ventricular hypertrophy. Electrocardiogram showed minor ST and T wave changes.

Course. Five days after admission the left facial weakness worsened and the left upper extremity was barely moved. There were dizziness, weakness and drowsiness. The patient became bedridden. Electroencephalogram disclosed focal 4-5/sec. activity in the right temporal region. Retinal blood pressure studies done that day with brachial pressure of 172/88 mm. of Hg revealed pressures: O.D. 42/12 and O.S. 82/26 gm. With this finding of approximately 50 per cent reduction of pressures on the right, a presumptive diagnosis of occlusion of the right internal carotid artery was made.

Operation. On Nov. 26, 1956, under local anesthesia, the right cervical carotid system was exposed. There was some thickening of the wall of the common carotid artery but it and the external carotid were pulsating. The internal carotid artery
just above the bifurcation was thickened and firm with minimal pulsation and yellowish walls. The carotid bulb was opened after isolating this segment with clamps, and thickened intima, atherosclerotic plaques and a fresh clot were removed. There was no complete obstruction at this level but when the clamp was removed from the internal carotid distal to the site of incision and intimection there was no back flow of blood. Catheter suction to a distance of 6 cm. from the bifurcation did not yield further clots.

Postoperative course was that of severe cerebral infarction. He manifested left facial palsy, absence of function of the left upper extremity and paresis but good movement in the spastic left lower extremity. There was left homonymous hemianopsia. He became oriented but displayed little initiative. Carotid and temporal pulses remained palpable. The retinal pressure readings showed no tendency to return to equal levels and, 11 weeks after surgery, were: O.D. 52/26 and O.S. 115/44 gm. Brachial blood pressure was 144/70 mm. of Hg at that time.

Discussion. The clinical course suggested the diagnosis of occlusion of the right internal carotid artery. The retinal artery blood pressure on this side was approximately 50 per cent lower in both systolic and diastolic readings than that on the left. Thus, substantial weight was added to the presumptive diagnosis. Angiography was not done. Direct exploration confirmed the presence of obstruction by failure to obtain back flow into the opened and proximally occluded internal carotid artery. Subsequent studies showed no tendency for retinal artery pressures to become equal. This has been the finding of Svien and Hollenhorst in cases of spontaneous or surgical occlusion of the internal carotid artery.

Case 2 (28502). F.A., a 61-year-old white male machinist, was admitted to the Veterans Administration Hospital, Iowa City, Jan. 10, 1957. Six weeks before admission he awakened with weakness of the right hand and arm. This progressed and was soon followed by weakness of the right leg and later of the right face. He denied headaches, visual symptoms and difficulty with speech.

Examination. The patient was short, obese, moderately dysphasic but alert and cooperative. The gait was typical of hemiplegia. The fundi manifested arteriolar narrowing and a few old hemorrhages on the left. Brachial blood pressure was 160/90 mm. of Hg. The chest, heart and abdomen showed no abnormalities. The visual fields were full to confrontation. There was weakness of the right lower face. The right arm was paretic and grip was ineffective. The right leg was weak and spastic. There were hyperreflexia, Babinski sign and Hoffmann sign on this side.

Urinal and blood were within normal limits. Blood serology was negative. Lumbar puncture disclosed a pressure of 160 mm. of water. The fluid was colorless with 8 white blood cells/c.mm. Total protein was 43 mg. per cent, and spinal fluid Kolmer was negative. Roentgenograms of skull and chest were normal. Electrocardiogram showed minor abnormalities of P and T waves. Electroencephalogram revealed focal 3–4/sec. waves in the left temporal region and low voltage over this hemisphere.

Because thrombotic occlusion of the left internal carotid artery was suspected, retinal artery pressures were determined. The readings were: O.D. 86/44; O.S. 62/26 gm. A few days later the readings were O.D. 86/52; O.S. 52/26 gm. The intraocular pressures were equal and normal. The clinical diagnosis seemed well supported. Examination the day before surgery revealed slight improvement of previous deficits. The carotid pulsations on the left as high as they could be palpated seemed less than on the right. Pulses in the temporal arteries were equal.
Operation. On Jan. 30, 1957, under local anesthesia, the left common carotid artery and its branches were exposed through an incision along the anterior border of the sternocleidomastoid muscle. The common and external carotid arteries pulsed strongly but their walls manifested atherosclerotic thickening. The carotid bifurcation was hard, irregular and not compressible. This density extended up a distance of 2–3 cm. into the internal carotid artery. No pulsation could be detected here or in the short segment of visible and relatively normal-appearing artery above. Arterial graft was considered but the difficulty in exposing the internal carotid sufficiently above the point of obstruction caused us to abandon this. An incision was made opening the common carotid artery and the bulb, and intemectomy and thrombo-endarterectomy were done in this area, removing the thick atherosclerotic lining of the bifurcation including the extensions of this into the external and internal carotid arteries. The lumen of the internal carotid artery had been completely occluded by old organized thrombus and a small recent clot. The specimen was removed in one piece (Fig. 1). A free back flow of blood was obtained from the internal carotid artery and the arterial incision was closed. When the clamps were released the entire system pulsed strongly.

Just before surgery the brachial blood pressure was 158/90 mm. of Hg. The retinal pressures were: O.D. 86/44; O.S. 50/32 mm. When the left external carotid was clamped the pressure on the left dropped to 30/14 and when the clamp was released two further immediate measurements were: 20/8, then 42/18 mm. O.S.

Course. The patient showed no immediate ill effects from the operation and retinal artery pressures in 2 hours were: O.D. 86/40 and O.S. 76/40 mm., seeming to indicate restoration of carotid flow on the left. Approximately 5 hours after surgery the patient was found to be aphasic and hemianopic on the right. Movements of right arm and leg were much reduced. The left temporal artery was pulsating. A postoperative thrombosis or embolism was suspected and the wound was opened. The common and external carotid arteries were pulsating normally. From its origin and for a distance of about 3 cm. the thin-walled internal carotid was darkened and distended by a fresh thrombus. The arterial incision was opened, clot was removed and an attempt was made to suck out propagated clot above. This was partially successful but a rather poor back flow was obtained. Stellate ganglion block was done. The next day retinal pressures were: O.D. 84/40; O.S. 46/38 mm. Brachial pressure was 130/70 mm. of Hg.

Fig. 1. Case 2. Photographs of intraluminal lesion removed in one piece (×1.4). It is a cast of the carotid bulb and proximal segments of the internal and external carotid arteries. The lumen of the external carotid is compromised by atherosclerotic changes but is patent. The internal carotid is completely occluded by an old organized thrombus at the periphery and more recent thrombus in the center.
The patient followed a typical course of severe cerebral infarction with some minor improvement in speech but none in the use of the arm. The leg could be moved weakly.

The last observation of retinal artery pressures was on Feb. 27, 1957. The readings were: O.D. 80/42; O.S. 60/30 gm.

Discussion. The clinical course and findings suggested the possibility of internal carotid occlusion on the left and several readings of retinal artery pressures on this side were significantly lower than those on the right. Palpation of the neck was not especially helpful in diagnosis. Angiography was not done.

There was complete occlusion of the left internal carotid artery and partial occlusion of the bulb and external carotid artery. Since a vessel graft seemed impossible because of difficulty in exposing the artery, intimeectomy and thrombo-endarterectomy were done. Experience in vascular surgery elsewhere suggests that intimeectomy in vessels of this size is often followed by intravascular clotting. Such was the case here, and propagating thrombus or embolus was responsible for the unfortunate outcome. The possibility of mobilizing the cervical carotid system sufficiently to anastomose common to internal carotid artery after resection of the bulb and a portion of the internal carotid artery has been demonstrated.6,18

The only observation of nearly equal retinal artery pressures in the two eyes was during the few hours following relief of arterial obstruction and before postoperative intravascular clotting occurred. The further drop in retinal artery pressure from 50/32 to 30/14 gm. on the left when the left external carotid artery was clamped deserves comment. This vessel was apparently carrying significant anastomotic blood flow to the ipsilateral ophthalmic artery. Was this flow significant in respect to the needs of the cerebral hemisphere on this side?24 It apparently did not prevent some degree of ischemia and infarction. Nor did occlusion of the external carotid artery cause any immediate increase in neurologic deficits. Miletti16 has pointed out that digital compression of the common carotid artery on the side of occlusion of the internal carotid artery does not produce the fall in retinal artery pressure that is expected in a normal system. This maneuver was not carried out here or in our other patients but it seems likely that it would have caused a further drop in pressure in this case.

It is quite possible that when the neurologic status of the patient with carotid occlusion is stable or slowly improving he is no longer a candidate for surgery. The few gratifying results so far reported have resulted from relief of obstruction during the period of vacillating function of the involved hemisphere.

Case 3 (S.U.I. 56–1130). F.K., a 57-year-old white female, was admitted to the State University of Iowa Hospitals Jan. 24, 1956. Complete history was not available. Disorientation had been noted by her physician 3 weeks before. The blood and

* The authors wish to express their appreciation to George Perret, M.D. and Arthur Eisenbrey, M.D. of the Neurosurgical Service, State University of Iowa Hospitals for permission to include this case.
spinal fluid Kolmer were found to be positive with elevation of gold curve. Penicillin in total dosage of 12,000,000 units was given.

**Examination.** The patient was lethargic and disoriented. Speech was slurred. Simple commands were carried out. Brachial blood pressure was 116/65 mm. of Hg. The chest was clear, and there was a grade 1 systolic murmur. Peripheral pulses were of good quality and carotid pulses were present. There was right hemiparesis, more evident in face and arm, with hyperactive reflexes and Babinski sign on this side. Retinal vessels were sclerotic. Visual field determination could not be accomplished.

Routine laboratory studies showed a mild hypochromic anemia. Blood and spinal fluid Kolmer were positive. Lumbar puncture revealed a pressure of 110 mm. of water. The fluid was colorless. There were 5 white blood cells/c.mm. and total protein was 61 mg. per cent. Fasting blood sugar and blood urea nitrogen levels were normal. Roentgenograms of skull and chest were within normal limits. Electroencephalogram revealed focal slow activity in the left frontotemporal region.

**Course.** Confusion and lethargy continued. There was persistent headache in the left temporal region. Thrombosis of the internal carotid artery was considered as was neurolues and cerebral tumor. Determination of retinal artery pressures was attempted but the patient could not cooperate. The hemiparesis progressed and ventriculography done on Feb. 3, 1956 revealed slight ventricular dilatation. On February 16 bilateral percutaneous carotid angiography was done. There was obstruction of the left internal carotid artery just beyond its origin. Injection of dye on the right side resulted in bilateral filling of middle and anterior cerebral arteries. Unfortunately there was a subsequent marked increase of the hemiparesis and nearly complete aphasia. Stellate ganglion blocks were done. The right arm remained flaccid but the patient eventually could walk. Speech was unintelligible. No gross visual field defect was evident.

At her last visit on Feb. 20, 1957, the patient was ambulatory with hemiparetic gait. Dysphasia was marked. The right arm was flaccid and hypesthetic.

Ocular pressure was normal and equal on the two sides. The retinal artery pressures were: O.D. 72/32; O.S. 50/24 gm.

**Discussion.** A question that might have been raised in this case is whether the demonstrated occlusion of the left internal carotid artery was responsible for the signs and symptoms. This point has been raised before. It seems quite likely that occlusion of the internal carotid artery causes a sufficient deficit in blood pressure and blood flow to endanger function if occlusive disease exists in the intrinsic blood vessels of the brain or in the other arteries supplying the brain or if collateral circulation is defective. The vascular deficiency might also unfavorably influence the course of a coexisting disease. Diminished blood flow and pressure based on lowered cardiac output and systemic circulatory changes would be much more serious so far as nutrition and oxygenation of the brain is concerned if a major vascular path to the brain were obstructed.

The case is more immediately in point in demonstrating a significantly lower retinal artery pressure on the side of the carotid obstruction one year after onset of disability and discovery of the occlusion. It also calls attention to the necessity for cooperation of the patient in measuring retinal artery pressures.
Ventriculography might well have been indicated even though a difference in retinal artery pressures could have been demonstrated. However, many case reports would suggest that patients who demonstrate a more typical syndrome of vascular insufficiency could be spared air studies if the clinician had knowledge of lowered retinal artery pressure on the involved side.

The case emphasizes the hazard of angiography in the patent carotid when the other is obstructed. It seems quite possible that the hemisphere in which blood pressure, and presumably blood flow, is deficient is more vulnerable to the deleterious effects of this procedure. Knowledge of significant difference in retinal artery pressures in the two eyes should make the surgeon aware that the carotid system ipsilateral to the lower pressure may be partially or completely occluded.

Contralateral compression of the cervical carotid artery is often practiced during angiography. It is potentially dangerous when an obstruction already exists in the carotid to be injected. This inadvertence could be avoided with knowledge of retinal artery pressures pre-operatively. The performance of angiography only on the side of suspected occlusion or brain disease may result in failure to recognize bilateral occlusion of the internal carotid arteries, but this condition is quite uncommon. Diminished carotid and retinal artery blood pressure may be contemporaneous with independent disease of the brain.

COMMENT

We do not intend to imply that a presumably occluded cervical carotid system should always be explored directly before percutaneous angiography.* However, when a patient presents symptoms appropriate to internal carotid thrombosis and a significant lowering of retinal artery pressure on the side of the lesion, direct exploration without preliminary percutaneous angiography may prove to be the best judgment. This has been suggested in effect by Schneider and Lemmen in the management of traumatic internal carotid thrombosis. They believe that in this circumstance open angiography can be performed more reliably than can percutaneous angiography. In addition, it gives opportunity for direct inspection of the area of trauma. Much the same argument could be given for direct exploration in the cases of spontaneous carotid occlusion. Open angiography could be done if an occlusive lesion were not disclosed at operation. Boldrey et al.3 recommended open angiography in their report of 24 cases of angiographically demonstrated internal carotid thrombosis. The open method permits inspection of the vessel walls and assures proper placement of the needle. Since time is of importance in these cases and since most carotid occlusions are segmental and near the bifurcation the argument would seem to be supported. However, symptom-producing obstructions may not be complete and information as to this matter is of importance in preparing for operation. If occlusion is complete, the surgeon may take his time in arterial resection and anastomos-

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* The use of anticoagulants in syndromes of impending cerebral infarction is not within the scope of this study.
sis, intimecтомy or vessel graft without fear that while so doing he is increasing cerebral ischemia. This is not the case in partial obstructions. Here some expedient in surgery, artificial arterial by-pass and hypothermia may be in order. Retinal artery pressures could not be relied upon to distinguish partial from complete occlusion although angiography could do so. However, if digital compression of the common carotid artery caused a further decrease in an already lowered ipsilateral retinal pressure then a partial occlusion might be suspected (see discussion of Case 2). A significant residual flow in a partially obstructed carotid system could be detected at exploration. If the operation were performed under local anesthesia the patient's ability to tolerate complete occlusion could then be tested. The Mata's test done pre-operatively might give this same information and perhaps should cautiously be done as a routine.

It is possible that abnormalities of arterial supply of the brain, as well as intracranial masses that interfere with circulation, may be a source of error in interpretation of the meaning of differences in readings of retinal artery pressure. A finding of unilateral abnormally elevated retinal artery pressure has been recorded in a case of intracranial tumor, the latter being on the side opposite the higher pressures. In cranial arteritis the opthalmic artery may be severely involved with or without evidence of cerebral ischemia. Retinal artery pressures might be quite low in this condition with no corresponding reduction in carotid flow.

**SUMMARY**

An approximation of retinal artery blood pressure can be made by the technique of ophthalmodynamometry. The measurements so obtained directly reflect the pressure in the internal carotid artery and are of definite value in comparing the pressures in these arteries on the two sides.

Comparative studies of retinal artery pressure in 3 cases of proven occlusion of the internal carotid artery revealed that pressures on the side of occlusion were lower than those on the contralateral side by 30 to 49 per cent in systolic and 25 to 54 per cent in diastolic readings. Two of the 3 patients were subjected to surgery without preliminary angiography and in these the measurements of retinal artery pressure proved to have given substantial and valid support to the clinical diagnosis.

In the 2 cases in which the carotid system was explored, the obstruction in one was above the limits of cervical approach to the vessel. In the other satisfactory relief of obstruction was provided by thrombo-endarterectomy. Evidence of re-establishment of circulation by direct inspection was accompanied by an increase in retinal artery blood pressure on the same side. Early intravascular clotting and possible embolism vitiated the immediate success and disability was increased.

Surgery of vascular occlusion of the internal carotid artery in the neck is still in an investigative stage. Advances in vascular surgery generally are likely to promote considerable effort toward an attack on the diseased carotid system. Determination of retinal artery pressures is a simple, safe
and valuable aid to diagnosis of occlusive vascular disease of the carotid system. It promises, as well, to be of value in directing the best use of angiography.

REFERENCES