Cerebral Edema
Following Experimental Subarachnoid Hemorrhage

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SUMMARY The development of cerebral edema after experimental subarachnoid hemorrhage (SAH) was studied in cats by determining regional brain tissue water content with the microgravimetric technique as well as the drying-weighting method. SAH was induced by withdrawing needles previously pierced into one or both infracranial internal carotid arteries through a unilateral transorbital approach. Serial determinations of regional cerebral blood flow (rCBF) by labelled microspheres, and monitorings of vital signs such as intracranial pressure (ICP), blood pressure and EEG were carried out up to 24 h after SAH. Animals could be classified into three grades according to the severity of SAH. In grade I, the increase of ICP was transient and minor. In grade II, ICP increased up to 200 mm Hg with a marked reduction of rCBF below 20% of control in cerebral hemispheres. Following subsequent reduction of ICP, rCBF increased over control, indicating reactive hyperemia. Thereafter, a great reduction of rCBF was again observed. In grade III, rCBF was sustained at essentially zero flow with the presence of continuously increased ICP above 100 mm Hg. Cerebral edema was observed particularly in the parasagittal water-shed areas of all grade II animals. It is concluded that cerebral edema complicating SAH is caused by the combination of an initially induced global cerebral ischemia and the subsequent recovery of cerebral circulation. Post SAH hypertension is another factor to exacerbate the development of cerebral edema.

Stroke, Vol 13, No 3, 1982

THE PATHOPHYSIOLOGY of subarachnoid hemorrhage (SAH) has been widely studied in both its clinical and experimental aspects, being related to the cerebral circulation,1-4 neuronal function,5,6,7,8 intracranial pressure (ICP),9,10 and cerebral vasospasm.5,12 On the other hand, the development of cerebral edema in SAH has rarely been investigated.13 However, it is becoming more important to understand cerebral edema as a complication in patients with SAH, because an increasing number of patients with ruptured cerebral aneurysms are undergoing early surgery.14,15

Some difficulties are still involved in investigating cerebral edema after experimental SAH. First, to simulate human SAH due to a ruptured cerebral aneurysm, the following three conditions are required: 1: arterial wall injury; 2: acute increase of ICP; 3: a sufficient amount of subarachnoid blood clots. At the time of aneurysm surgery in the very acute stage of SAH, widely spread subarachnoid blood clots usually observed to cover the cerebral convexity may contribute to the alterations of cerebral microcirculation. In experimental SAH usually induced by cisternal blood injection or arterial puncture, such a condition cannot be easily obtained warranting a more advanced method. Secondly, changes of brain water content must be precisely assessed for the quantitative determination of brain edema. For this purpose, the recently developed gravimetric technique may be more advantageous for determining specific gravity (SG) of very small tissue fragments than the conventional drying-weighing pro-
CEREBRAL EDEMA FOLLOWING SUBARACHNOID HEMORRHAGE/Shigeno et al.

procedure. However, as already cleared in the previous study,16, 17 changes of SG cannot be considered to be equal to those of actual brain water content because of the effect of cerebral blood volume (CBV). On this point, the authors have further reassessed the accuracy of the drying-weighing procedure using very small tissue fragments.17 In the present study, the development of cerebral edema has been investigated with these technical advancements as well as measuring regional cerebral blood flow (rCBF) with the use of the labelled microsphere technique. Then the factors contributing to the development of cerebral edema have been analyzed.

Material and Methods

Production of SAH

Eleven adult mongrel cats weighing about 3 kg were anesthetized with Nembutal® (30 mg/kg) and paralyzed with gallamine (Flaxedil®). The animals were artificially ventilated via a cuffed endotracheal tube. Anesthesia was maintained with nitrous oxide-oxygen gas mixture (2:1 v/v). Following catheterization to both the femoral artery and vein, the animal was fastened onto the stereotaxic frame in a sphinx-like position. For the rCBF study with labelled microspheres, an additional catheter was introduced into the left cardiac ventricle through the femoral artery. Thereafter the left orbital contents were totally removed, and the optic foramen was enlarged by about 5 mm in diameter under the operative microscope (fig. 1). With a semicircular dural incision below the optic nerve, one or both of the infraclinoid internal carotid arteries were pierced through the arachnoid membrane with a swaged needle (Ethicon® 5-0 or 6-0). After the enlarged optic foramen had been tightly covered by a metallic plug with a guiding tube for the threads, the orbital cavity was completely covered with bone wax and acrylic resin.

Monitoring of Vital Signs

The following parameters were monitored: blood pressure (BP), blood gases, body temperature, ICP (i.e., cisterna magna pressure or chiasmatic cisternal pressure through the guiding tube), cortical electroencephalography (EEG), and somatosensory evoked potentials (SEP) to sciatic nerve stimulation. The animals were observed for 3 to 24 h after SAH. EEG was recorded from the right precentral area through the monopolar lead. SEP was obtained with an analysis time of 100 msec following electrical stimulation 128 times for 1 min using Nicolet® Signal Averaging System (Model 1072). After the preparatory steps the needles were withdrawn when all vital signs were within normal limits. Throughout the experiment, pO2 and pCO2 were maintained at 90 to 120 and 35 to 40 mm Hg respectively. Blood pH was frequently adjusted at around 7.4 with sodium hydrogen carbonate solution. Body temperature was maintained at about 37°C with an electrical heating blanket. One to 2 h prior to sacrifice by Nembutal®, Evans blue (5 ml of 0.5% solution) was injected to study disturbances of the blood brain barrier.

Measurement of rCBF

The microspheres used were of 15μm diameter and labelled with scandium-46, strontium-85, cerium-141 and iodine-125 purchased from the 3M® Company. Preparation of the microspheres has been previously reported.18 Four serial determinations of rCBF were carried out in 6 animals before and immediately after SAH as well as 10 to 20 min, and 3 to 6 h post SAH. After the experiment, brain tissues weighing 100 to 200 mg were rapidly dissected from 60 areas throughout the entire brain, and stored in pre-weighed plastic test tubes sealed with a cap. After wet weight determination with an accuracy of 0.0001 g (Salitarius type 2462), radioactivities were counted. Output from the γ

![Figure 1](http://stroke.ahajournals.org/) Operative procedure for the production of experimental SAH.
Determination of Brain Tissue SG and % Water Content

The percentage of brain tissue water content per wet weight was determined in the same brain tissues. In an other group of 5 animals, brain tissue SG was measured with brain tissues weighing 30 to 50 mg. The methodological details and assessment of brain water content in terms of SG have been reported elsewhere. Normal control values of regional SG and % water content of brain tissue were determined previously in each corresponding anatomical area (table).

TABLE Control Values of Regional % Water Content and Specific Gravity of Brain Tissue

<table>
<thead>
<tr>
<th>% Water content (n = 13)</th>
<th>Specific gravity (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>1. Anterior frontal grey</td>
<td>81.41</td>
</tr>
<tr>
<td>2. Anterior frontal white</td>
<td>72.54</td>
</tr>
<tr>
<td>3. Posterior frontal parasagittal grey</td>
<td>80.51</td>
</tr>
<tr>
<td>4. Posterior frontal parasagittal white</td>
<td>66.15</td>
</tr>
<tr>
<td>5. Posterior frontal sylvian grey</td>
<td>80.37</td>
</tr>
<tr>
<td>6. Posterior frontal sylvian white</td>
<td>66.28</td>
</tr>
<tr>
<td>7. Anterior parietal parasagittal grey</td>
<td>80.72</td>
</tr>
<tr>
<td>8. Anterior parietal parasagittal white</td>
<td>69.56</td>
</tr>
<tr>
<td>9. Anterior parietal sylvian grey</td>
<td>79.82</td>
</tr>
<tr>
<td>10. Anterior parietal sylvian white</td>
<td>65.95</td>
</tr>
<tr>
<td>11. Temporal grey</td>
<td>81.42</td>
</tr>
<tr>
<td>12. Temporal white</td>
<td>69.82</td>
</tr>
<tr>
<td>13. Posterior parietal parasagittal grey</td>
<td>80.71</td>
</tr>
<tr>
<td>14. Posterior parietal parasagittal white</td>
<td>69.06</td>
</tr>
<tr>
<td>15. Posterior parietal sylvian grey</td>
<td>80.33</td>
</tr>
<tr>
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<td>80.98</td>
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<td>80.71</td>
</tr>
<tr>
<td>Total white</td>
<td>68.47</td>
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</table>


counter was punched on paper tape for subsequent computer processing (Wang 2200). RCBF was calculated from the following equation.

\[ rCBF = \frac{C_b \times RBF \times 100 \text{ ml/100g}}{C_r} \]

where \( C_b = \) counts per gram of brain, \( C_r = \) total counts in the reference arterial blood samples, and \( RBF = \) reference blood flow (rate of blood withdrawal). Thereafter, the brain tissues in the test tubes were refrigerated and dried in the vacuum freeze-drying device (Leybold® Gefriertrocknings-Anlage GT1,5), the caps being previously perforated. Drying was carried out for 48 h at a pressure below \( 1 \times 10^{-2} \) torr. Then the percentage of brain tissue water content per wet weight was calculated.

Determination of Brain Tissue SG and % Water Content

In the above mentioned group of animals, both rCBF and percentage of water content were simultaneously determined in the same brain tissues. In another group of 5 animals, brain tissue SG was measured with brain tissues weighing 30 to 50 mg. The methodological details and assessment of brain water content in terms of SG have been reported elsewhere. Normal control values of regional SG and % water content of brain tissue were determined previously in each corresponding anatomical area (table).

Results

Grading According to the Severity of SAH

SAH was successfully induced in all animals. Three patterns of ICP changes were observed, depending on the severity of SAH (fig. 2, fig. 3, fig. 4, fig. 5). In grade I (n = 4), following an acute rise of ICP to about 50 mm Hg, control values were reached again within ten min. Blood pressure and electrical activities did not change significantly. In grade II (n = 4), ICP increased up to 200 mm Hg with a concomitant rise of blood pressure and a total suppression of EEG and SEP. Ten to 20 min later, ICP nearly returned to control values, accompanying a partial recovery of electric-
CEREBRAL EDEMA FOLLOWING SUBARACHNOID HEMORRHAGE/Shigeno et al.

Changes of rCBF

There was a marked change of rCBF, particularly in grade II (fig. 7, 8). Immediately after SAH, rCBF decreased markedly below 20% of control blood flow in both cerebral hemispheres, while the brain stem circulation was relatively well preserved. Ten to 20 min later, hemispheric rCBF increased even above control values, paralleling the decrease in ICP and indicating reactive hyperemia. Three to 6 h after hemorrhage, a marked reduction of rCBF was observed again. Grade I corresponded a slight decrease in rCBF. In contrast, in grade III, rCBF remained nearly zero following SAH. There was a tendency towards greater rCBF reduction in the parasagittal area as compared to the sylvian region. Although the decrease of rCBF after SAH was generalized in the cerebral hemisphere, the distribution of decreased rCBF was inhomogeneous, and the reduction was greater in the cerebral hemisphere with the arterial puncture site than without puncture.
FIGURE 5. Changes of blood pressure and intracranial pressure after SAH classified into three grades. Shaded area shows cerebral perfusion pressure in a representative case.

Changes of SG and Water Content of Brain Tissue

The difference of SG from control values in each corresponding area (table) was estimated in the parasagittal cortex and subjacent white matter, as well as in the sylvian cortex and subjacent white matter (fig. 9). A marked decrease in SG was observed only in grade II animals, particularly in the parasagittal area. These animals had a moderate arterial hypertension post SAH. In grade I or III, there was even a tendency of SG to increase. The percentual increase in water content of brain tissue was also estimated in the same manner (fig. 10). A marked increase was observed only in grade II animals. When rCBF and percentual brain water content were measured simultaneously, rCBF values immediately after SAH were plotted against brain water content (fig. 11). In animals of grade II with reactive hyperemia, a significant increase in water content was observed only in areas in which rCBF had decreased below 20 ml/100g/min both in cortex and white matter. In contrast, in grade III animals with permanent decrease of rCBF below 10 ml/100g/min, there was no significant increase in water content.

Discussion

Validity of the SAH Model

Regarding the method for inducing SAH, two models have been applied. One is through injection or topical application of blood in the subarachnoid space. The other is through rupture or puncture of an intracranial artery. In both methods, one more factor should be considered: is the model applied with or without a tight intracranial cavity, or in other words, is SAH hyperbaric or isobaric? Inasmuch as most of the experimental SAH have been concerned with cerebral vasospasm, isobaric SAH through blood injection into the subarachnoid space is common. From a clinical point of view there is no occasion for isobaric SAH, indicating a significant role of increased ICP in the pathophysiology of SAH. Asano et al. also confirmed that acutely increased ICP in experimental SAH causes cerebral microcirculatory disturbances. On the other hand, arterial wall injury should also be considered, because it has been suggested that alterations of the perivascular adrenergic innervation might affect the responsiveness of the artery, particularly in connection with cerebral vasospasm. Furthermore, the recent development of early surgery for ruptured cerebral aneurysms has revealed that a significant amount of subarachnoid blood clots surround not only the basal cistern but also the subarachnoid spaces on the cerebral hemisphere, producing a state of cerebral tamponade with blood. Such blood clots situated directly above the pial arteries or arterioles are also considered to cause microcirculatory disturbances. Thus, the following three conditions are required to simulate human SAH in the experimental model; 1: arterial wall injury; 2: acute increase of ICP; 3: a sufficient amount of subarachnoid blood clots.

Following this concept, Hayakawa et al. developed some implanted devices for tearing or puncturing the middle cerebral artery of the cat through a transorbital...
approach. With this method, however, a punctured or torn middle cerebral artery frequently seems to cause an ischemic complication in the brain. Furthermore, an extensive hemorrhage cannot be expected to occur because there is little subarachnoid space around the artery. Then the internal carotid artery was punctured. Through the unilateral transorbital approach, it was even possible to puncture both infraclinoid carotid arteries, which allowed a greater hemorrhage and will allow a rebleeding model if desired. In the operative procedure, however, great care should be taken not to widely open the arachnoid membrane, otherwise the hemorrhage induced will be subdural and not subarachnoid. A device more simplified than the one used by Hayakawa et al. was developed for the maintenance of a tight intracranial cavity. Through the guiding tube for the threads, it was also possible to measure the chiasmatic cisternal pressure, whereas it was frequently blocked with blood clots after SAH. With this methodological development, ICP increased up to 200 mm Hg after SAH, which was sufficient to cause global cerebral ischemia and suppression of electrical activities. With an increase of ICP by around 50 mm Hg as reported by Hayakawa et al., enough perfusion pressure was still maintained. Initial loss of consciousness which is a common finding in patients with SAH can be explained by such a great increase of ICP. In this respect, the present model seems to be an appropriate one.

**Subsequent Course of Parameters after SAH**

Immediately after SAH, arterial pressure was transmitted directly into the subarachnoid space until hemostasis when cerebral tamponade occurred. Increases in ICP were related to extent and distribution of the hemorrhage and classified into three grades. The clinically

**Figure 6.** Gross appearance of the brain with SAH in each grade. There is a close relationship between the severity of SAH and the grading. Extravasation of Evans blue is present in the parasagittal water-shed area of the grade II animal.
accepted grading system with five grades \(^\text{21}\) cannot be easily applied to the experimental animals because of the difficulty in judging the neurological signs even in conscious animals. However, the grade I animal seems to correspond to the clinical grades I and II, the grade II animal to the clinical grades III and IV, and the grade III animal to the clinical grade V, corresponding to the severity of the illness. The most important of these is the animal of grade II, because those patients with disturbed consciousness could survive by adequate treatment. \(^\text{14}\)

In the grade II animals, the initial increase of ICP, which was sufficient to cause global cerebral ischemia even in the presence of vasopressor response, was followed by reduction of ICP due to hemostasis and activation of the pressure buffering system. Thereafter, however, no marked re-elevation of ICP was observed in the follow-up period up to 24 h. This finding is contradictory to that obtained by Hayakawa et al., \(^\text{9}\) who reported a biphasic increase of ICP 6 to 12 h after SAH, indicating the development of cerebral edema. From the present results, however, ICP does not seem to increase again even in the presence of cerebral edema, which developed in grade II animals. The occurrence of acute brain swelling or vasoparalysis is also unlikely from this ICP pattern, as also suggested by Asano et al. \(^\text{1}\) In contrast to grade II, grade III animals showed a sustained elevation of ICP which was caused by continuous bleeding or acute hydrocephalus with an impaired pressure buffering system, \(^\text{11}\) and resulted in subsequent brain death.

It is of interest to note the presence of continuous systemic arterial hypertension after SAH in half of the grade II animals, which might reflect an adrenergic over-activity observed in patients with SAH as an increase of catecholamines in urine, \(^\text{22}\) plasma \(^\text{23, 24}\) and cerebrospinal fluid. \(^\text{12, 25}\)

EEG and SEP were totally suppressed when the cerebral perfusion pressure showed critical reduction below about 20 mm Hg. During the subsequent decline of ICP, however, the recovery of electrical activities was still incomplete. In patients with SAH, generalized retardation of EEG is seen during the course of the illness. \(^\text{8}\) Such a non-focal but generalized disturbance of EEG may be related to the generalized changes in cerebral circulation, as will be discussed later.

**rCBF in SAH**

This study is the first in the literature to determine rCBF in experimental SAH using the labelled micro-
CEREBRAL BLOOD FLOW

<table>
<thead>
<tr>
<th>Grade</th>
<th>Parasagittal Area</th>
<th>Sylvian Area</th>
<th>Brain Stem</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td></td>
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</tbody>
</table>

FIGURE 8. Changes of rCBF after SAH in a representative case belonging to each grade. Marked reactive hyperemia was observed in grade II. Values are mean obtained from more than three brain tissues.

CHANGES OF TISSUE SPECIFIC GRAVITY

<table>
<thead>
<tr>
<th>Grade</th>
<th>Parasagittal Cortex</th>
<th>Sylvian Cortex</th>
<th>Parasagittal White Matter</th>
<th>Sylvian White Matter</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td></td>
<td></td>
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<tr>
<td>III</td>
<td></td>
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</table>

FIGURE 9. Changes of specific gravity in five animals classified into three grades. A marked decrease in specific gravity was observed particularly in the parasagittal areas of grade II animals.

Cerebral edema following subarachnoid hemorrhage. By dissecting brain tissues weighing less than 200 mg, rCBF was obtained in 60 areas of the whole brain. Particular attention was paid to the parasagittal water-shed areas in the cerebral hemisphere, because a previous report revealed a predilection for location of the so-called no-reflow phenomenon in these areas. Furthermore, extravasation of Evans blue was mainly observed also in these areas.

As would be expected, there was a tendency towards a greater decrease of rCBF in the parasagittal area than in the sylvian area, while the difference was not significant. Though the decrease of rCBF after SAH was generalized in the cerebral hemisphere, the distribution was inhomogeneous including those brain tissues without significant reduction of rCBF. This patchy distribution of rCBF might be resulted from the possible presence of pial arterial spasm induced by contact with subarachnoid blood clots. The presence of pial arterial or arteriolar spasm has already been confirmed...
CHANGES OF REGIONAL BRAIN WATER CONTENT

Following topical application of blood to the cortical surface and by a morphological study of brain parenchymal vessels after SAH.

There was a greater reduction of rCBF in the cerebral hemisphere with the arterial puncture site than in the hemisphere without arterial puncture, while no difference existed between both hemispheres prior to needle withdrawal. This may support the mechanical in-

**Figure 10.** Changes of brain water content in five animals. A marked increase in water content was observed in grade II animals.

**Figure 11.** Relationship between water content and rCBF immediately after SAH in a representative case from grade II and III. Each point represents a value from one brain tissue. In a grade II animal with subsequent reactive hyperemia, a marked increase in water content was observed in those brain tissues where rCBF decreased below 20 ml/100g/min. In a grade III animal with sustained no-flow, increase in water content was not evident.
fluence on perivascular adrenergic innervation, which has been suspected as a cause of cerebral vasospasm.\textsuperscript{12, 19}

When rCBF was greatly reduced in the cerebral hemisphere including thalamus and basal ganglia, brain stem circulation was relatively well preserved. Since the increase of ICP, or, in other words, the decrease of perfusion pressure is estimated to be equal between the supratentorial and infratentorial structure, it can be said that the brain stem is resistant to ischemic insult for maintenance of vegetative function of the brain.

Post-ischemic reactive hyperemia was only observed in grade II. After rCBF was restored, however, it began to decrease again, indicating post-ischemic hypoperfusion. In a clinical situation, prolonged decrease of rCBF has also been documented, particularly related to the presence of vasospasm.\textsuperscript{8, 26} This secondary decrease of rCBF can be explained by various factors such as microcirculatory disturbances, vasospasm, development of cerebral edema, and disturbed brain energy metabolism due to neuronal dysfunction.

Cerebral Edema in SAH

This is the first report dealing with both the measurement of SG and water content of brain tissue in experimental SAH. A few works on cerebral edema in SAH that ever appeared in the literature were concerned mainly with the morphological evidence of cerebral edema.\textsuperscript{27, 28} In the present study, cerebral edema could be quantitatively documented with the aid of the recently developed microgravimetric technique\textsuperscript{16} and as well as by microdetermination of regional brain water content through vacuum freeze-drying.\textsuperscript{17}

Cerebral edema developed in grade II animals in which a temporary global cerebral ischemia was followed by reactive hyperemia and subsequent post-ischemic hypoperfusion. Those animals with brain death accompanied by no-flow did not show any decrease of SG or increase of water content. Minor SAH with a slight reduction of cerebral perfusion pressure did not cause cerebral edema, either. On the contrary, in these grade I or III animals, there was even a tendency of SG to increase. This may be a result of increased cerebral blood volume, since the SG of blood is higher than that of brain tissue.\textsuperscript{16} There have been also some reports documenting the occurrence of increased cerebral blood volume in SAH related to the presence of cerebral vasospasm.\textsuperscript{3, 5}

Arterial hypertension is another factor in the development of cerebral edema. In cases where cerebral edema developed, a decrease in SG was more evident in the parasagittal water-shed areas where arterial hypertension was observed after SAH. It is well known that experimentally induced acute arterial hypertension causes extravasation of protein tracers, particularly in
the parasagittal water-shed areas. However, arterial hypertension would not necessarily be accompanied by an increase of brain water content unless hypoxia or hypercarbia were induced simultaneously. Hence, in cases of SAH, arterial hypertension is not a primary but secondary factor for the development of cerebral edema in addition to the presence of a disturbed blood-brain barrier resulting from an ischemic-anoxic insult. It is still controversial whether postischemic hyperventilation has a beneficial or detrimental effect on neuronal recovery. In a recirculation study following middle cerebral artery occlusion in the cat, Tamura et al. observed extravasation of Evans blue only in those animals with postischemic reactive hyperemia. Then, it can be postulated that postischemic reactive hyperemia accelerates the leakage of water and serum protein through the blood-brain barrier once it is damaged by an initial ischemic insult. However, the development of cerebral edema is not necessarily linked to the rearrangement of neuronal function, unless it causes cerebral herniation. Recent studies have also indicated that vasogenic edema has no influence on electrophysiological functions of the brain.

Conclusion

A working hypothesis explaining the development of cerebral edema after SAH can be proposed as in the figure 12. Since hypervolemic hypertension has recently been proposed for the treatment of ischemic pathology in patients with SAH, it should be born in mind that postischemic overperfusion might accelerate the development of cerebral edema as demonstrated by our results.

References

Ocular Pneumoplethysmography and Ophthalmodynamometry in the Diagnosis of Central Retinal Artery Occlusion

DAVID O. WIEBERS, M.D., W. NEATH FOLGER, M.D., AND BRIAN R. YOUNGE, M.D.

SUMMARY Ocular pneumoplethysmography and ophthalmodynamometry measure ophthalmic arterial system pressures to assess noninvasively the hemodynamics of the carotid system. A previously unreported circumstance in which these tests complement one another is central retinal artery occlusion. Typically, the ipsilateral retinal artery pressure, measured by ophthalmodynamometry, is greatly decreased or is zero, whereas the ophthalmic systolic pressure, measured by ocular pneumoplethysmography, is normal.

CENTRAL RETINAL ARTERY OCCLUSION usually presents as an acute, painless, unilateral loss of vision. Ophthalmoscopically, the optic disk and the retina become pale, the retinal vessels narrow, and a macular cherry red spot is often visualized. Central retinal artery occlusion may be confused with venous occlusions, branch artery occlusions, and ischemic optic neuropathy, despite the usual differences in temporal profile, clinical course, and ophthalmoscopic manifestations. Furthermore, any of these phenomena may occur in various combinations. Ocular pneumoplethysmography and ophthalmodynamometry, in combination, provide a safe and rapid means of confirming the diagnosis of central retinal artery occlusion and may provide clues as to the cause of the condition.

Report of Cases

Case 1

A 59-year-old man with a long history of hypertension experienced an episode of left amaurosis fugax 1 month before admission. The episode lasted 4½ minutes and resolved completely. Two more such episodes occurred the same day, prompting hospitalization and treatment with intravenously administered heparin. Results of four-vessel cerebral angiography were normal. The patient was dismissed on warfarin therapy.

Two weeks before admission, he had another episode of sudden onset of complete visual loss of the left eye, without improvement. An ophthalmologist in his home community diagnosed ischemic optic neuropathy. Treatment with inhalation of 95% oxygen and prednisone was given, but no improvement occurred. A left temporal artery biopsy specimen was normal, as was the sedimentation rate. He was referred to the Mayo Clinic for further evaluation.

Ophthalmologic examination showed a visual acuity of 14/21 on the right and light perception only on the left. Intraocular tensions were 16 mm Hg bilaterally. There was a left-sided afferent pupillary reflex defect. Ophthalmoscopic examination of the left eye showed a pale retina with thready vessels and edema of the macular area with an associated cherry red spot. Ophthalmodynamometry revealed retinal artery pressures of 130/54 mm Hg on the right and 12/0 mm Hg on the left. Ocular pneumoplethysmography revealed ophthalmic systolic pressures of 110 mm Hg bilaterally, with brachial systolic pressures of 154 mm Hg on the right and 148 mm Hg on the left. Results of a peri-orbital Doppler study were normal. General examination revealed a blood pressure of 190/110 mm Hg but was otherwise normal, as was a cardiac sector scan.

Case 2

A 71-year-old man reported the sudden onset of decreased vision in his right eye 2 weeks before admission. He could only see gross hand movements. By the following day, he was blind in the right eye. An ophthalmologist in his home community made a diagnosis of central retinal artery occlusion and referred the patient to the Mayo Clinic for further evaluation. The patient’s medical history had been unremarkable, with the exception of long-standing mild hypertension treated with a diuretic (Dyazide).

On admission, visual acuity was nonexistent on the right and 14/21 on the left. Ocular tensions were 11 mm Hg on the right and 15 mm Hg on the left. There was a right afferent pupillary reflex defect. Ophthal-
Cerebral edema following experimental subarachnoid hemorrhage.
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Stroke. 1982;13:368-379
doi: 10.1161/01.STR.13.3.368

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