Cerebrovascular Responses to Increased Intracranial Pressure*

THOMAS R. HEDGES, M.D., AND JAMES D. WEINSTEIN, B.S.
With the Assistance of NEAL KASSELL and SHERMAN STEIN

Neuro-Ophthalmologic Laboratory, Department of Neurosurgery, Pennsylvania Hospital, Philadelphia, Pennsylvania, and The South Jersey Medical Research Foundation, Camden, New Jersey

We have succeeded in making direct measurements of orbital vascular pressure in the rhesus monkey. To our knowledge this has not been done before and we believe that the direct measurements from the orbital ophthalmic artery can be utilized to monitor terminal carotid vascular responses. The results of the utilization of this technique have included certain aspects of the cerebrovascular responses to increased intracranial pressure and these will be discussed in this paper. Our results also will be correlated with previous work done in the investigation of increased intracranial pressure and resulting cerebrovascular responses.

Method

Rhesus monkeys, approximately 8 to 10 lbs. in weight, were used in all experiments. They were anesthetized with sodium pentobarbital given intravenously, 1 gr. per 5 lbs. All pressures were taken through polyethylene catheters attached to Sanborn transducers, amplifiers, and a 4-channel oscillographic recording system. In this series of experiments recordings were made from the ophthalmic artery, sagittal sinus, intracranial subarachnoid space over the temporoparietal area, jugular vein and femoral artery.

The frontal branch of the ophthalmic artery is approached through the soft tissues over the supraorbital notch by dissecting behind a frontal flap of skin to the bony notch. Gentle dissection enables one to isolate the artery from its associated vein and nerves as these structures leave the notch (Fig. 1). A 6-0 silk suture is placed beneath the artery for subsequent traction. The artery then is entered with a 30-gauge needle, and a finely drawn polyethylene tubing is inserted through this opening into the vessel for recording.

The pressure from the subarachnoid space and sagittal sinus is measured by inserting a catheter through an opening in the dura mater made with a 20-gauge needle. Larger tubing was used for catheterization of the jugular vein and femoral artery.

To record and change the cerebrospinal-fluid pressure simultaneously, 2 holes were made, 1 over each temporoparietal area. Physiological saline was injected into the subarachnoid space to elevate cerebrospinal-fluid pressure. Fluid was injected in varying amounts sufficient to maintain pressures at the desired levels in the different experiments.

Results

Ophthalmic Artery. The ophthalmic artery (Fig. 2) initially showed slowing with increased amplitude (vagal effect) as the intracranial pressure began to rise. This was followed immediately by a profound fall in ophthalmic arterial pressure as the intracranial pressure reached the height of the blood pressure. At this point, pressure of the ophthalmic artery fell from its initial mean level of approximately 100 to 40. We believe that this dramatic fall in pressure occurred because the ophthalmic artery became cuffed off intracranially where it was exposed in the subdural space to the high elevation of intracranial pressure. This cuffing effect continued until an arterial pressor response (Cushing phenomenon)* became evident.* It is then that an increased pulse pressure and a rise in

* The Cushing phenomenon is the increase in systemic arterial pressure and increased pulse pressure which occur as intracranial pressure is elevated. It was found that the intracranial pressure does not necessarily have to exceed the systemic or ophthalmic arterial pressure to induce a Cushing response, the response often occurring with the cerebrospinal-fluid pressure at or about the mean systemic blood pressure.

Received for publication August 7, 1963.
Revision received December 4, 1963.
* Aided by grants from The National Society for the Prevention of Blindness, The National Council to Combat Blindness (NSF 197 C5), and John A. Hartford Foundation.
ophthalmic arterial pressure occurred, with bradycardia evident.

The cuffing effect seen in the ophthalmic artery is notably absent in the pressure of the femoral artery (Fig. 2). The femoral recording shows only a very slight drop in pressure as the intracranial pressure is raised. This transient slowing and hypotension (vagal effect) in the femoral artery is followed in several seconds by the Cushing phenomenon as manifested by increase in pulse pressure and increase in blood pressure.

The ophthalmic arterial pressure remains elevated with a similar reflection of bradycardia and increase in pulse pressure until the cerebrospinal-fluid pressure begins to drop off. The ophthalmic and femoral arterial pressures then fall together with the pressures of ophthalmic vein and cerebrospinal fluid. It is of interest to note that simultaneous depressions seen on all of the tracings in Fig. 2 are a reflection of the Hering-Traube waves as an alternate depression and restoration of medullary blood flow in the face of increased intracranial pressure.

**Sagittal Sinus.** A noteworthy and repeatedly demonstrable response in the superior sagittal sinus is shown in Fig. 3. With the elevation of intracranial pressure to levels below the ophthalmic arterial pressure, the pressure of the superior sagittal sinus drops. As soon as the intracranial pressure starts to drop, the pressure of the superior sagittal sinus rises to its original level and then slightly above it. We feel that this drop in pressure is again the result of a cuffing effect, but this time on the cerebral veins where they are exposed to the increased pressure of subarachnoid fluid. Thus, a transient cuffing off of inflow from the cerebral veins is reflected in a drop in pressure which is rectified only when cerebral blood flow is re-established.

The pressure of the sagittal sinus drops slightly each time intracranial pressure is elevated even to levels of subarterial pressure. Kety et al. have shown that a decrease in cerebral blood flow occurs whenever the cerebrospinal-fluid pressure is above 40 mm.