Transmission of Increased Intracranial Pressure

I. Within the Craniospinal Axis*

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During the latter part of the 19th century a widespread interest was manifested in the problem of cerebral compression and the etiology of the neurological deficits produced by an expanding intracranial mass. As methods were developed for recording intracranial and intraspinal pressure and for the experimental production of acute intracranial hypertension, it was noted by some investigators that increased pressure was not transmitted consistently from the intracranial to the intraspinal space. Hill maintained that rapid injections of fluid into the supratentorial subarachnoidal space forced the cerebral hemisphere downward into the tentorial incisura and the cerebellum into the foramen magnum with complete obstruction of the subarachnoidal space at both levels. On the other hand, Eyster argued that this could not occur because of the nearly incompressible fluid in the spinal canal, which he believed had no means of escape. Cushing stated that severe effects of compression could occur locally with little or no transmission to remote areas of the brain.

More recently Kahn produced experimental increased intracranial pressure by perfusion of distilled water into the common carotid artery and found that a differential of pressure developed between the supratentorial space and the posterior fossa. Meyers also found such a differential with injection of Ringer’s lactate into the lateral ventricle, but the gradients of pressure illustrated in these papers were relatively small.

Clinical interest in the transmission of increased intracranial pressure developed as a result of the demonstration of the importance of transtentorial herniation as a cause of rapid neurological deterioration and death in patients with a space-occupying supratentorial mass. This has been recently re-emphasized in a review by Finney and Walker. They found evidence of transtentorial herniation in 55.4 per cent of an unselected series of autopsied brain tumors, including an incidence of 88 per cent in glioblastomas of the cerebral hemisphere. In 23 per cent of supratentorial tumors herniations of both tentorial incisura and foramen magnum were present.

There have been few clinical studies in which simultaneous pressures were recorded above and below the tentorium in patients with intracranial pathology. Smyth and Henderson found a lower lumbar pressure in 8 of 33 patients with intracranial space-occupying lesions, most of which were tumors, but the maximum difference in pressure was 100 mm. H$_2$O. These findings and a statement by Evans: “We now know as a result of many observations that there is a close correspondence between the ventricular and lumbar pressures under almost all circumstances” contrast with the high incidence of transtentorial herniation demonstrated post mortem in patients with brain tumor.

The present investigation was undertaken in an attempt to produce experimental herniations of the tentorial incisura and foramen magnum and assess the physical factors responsible for their development. The monkey was selected as the experimental animal because of its fibrous tentorium in contrast to the bony tentorium present in the dog and cat. Also, the anatomy of the tentorial incisura in the monkey as well as
the manner of attachment of the tentorium to the petrous ridges and anterior and posterior clinoids are quite similar to man.

Materials and Methods

The data from these experiments were obtained from 28 rhesus monkeys. Each animal was anesthetized with approximately 60 mg./kg. of pentobarbital sodium and tracheal intubation or tracheostomy was performed. Small holes were made in the skull with an electric drill and the dura mater was punctured with a hypodermic needle. Polyethylene catheters with a lumen 1 mm. in diameter were then inserted into the subarachnoidal space. In most experiments 3 or 4 catheters were inserted over the cerebral hemispheres for recording of pressure and injections of saline. A midline incision was made in the posterior fossa and the atlanto-occipital membrane was exposed. After puncturing the membrane with a needle a polyethylene catheter was inserted into the cisterna magna and directed upward into the cerebellopontine angle. By using small flexible catheters it was possible to avoid damage to underlying brain tissue with consequent hemorrhage, and leakage of cerebrospinal fluid around the catheters was reduced. However, some leakage always occurred so that the opening pressure, in the position of lateral decubitus used for recording, was usually 0–3 mm. Hg. Needles were placed in the lumbar subarachnoidal space for recording of pressure and injection of fluid, and catheters were inserted in the femoral artery for recording systemic blood pressure and in the femoral vein for administration of fluids and drugs.

The catheter from each site of recording was led to a Sanborn transducer, then to either a 4- or 8-channel Sanborn polygraph. Advantage was taken of the mechanism of "zero suppression" of the polygraph whereby a large range of pressure may be covered without change in attenuation. This accounts for the difference in base-line figures present in many of the illustrations. All animals were artificially ventilated in order to prevent any effects of respiratory alterations on the phenomena of pressure recorded.

The supratentorial pressure was increased by injection of saline into a 5 cc. balloon inserted in the extradural space over the cerebral hemisphere or by injection of saline through one of the catheters into the subarachnoidal space. A 2 cc. balloon was placed in the infratentorial extradural or subdural space in order to create a mass in the posterior fossa. Injections of saline were also made into the catheter in the posterior fossa and into one of the lumbar needles. Occasionally, in order to produce sustained intracranial hypertension for brief periods, saline was infused into the subarachnoidal space from a reservoir placed at measured distances above the animal's head.

An electroencephalogram was recorded from six needle electrodes inserted into the scalp over the frontal, temporal, and posterior parietal areas bilaterally. Increasing the intracranial pressure led to awakening and artifact of movement in lightly anesthetized animals, so that additional pentobarbital was administered intravenously in increments sufficient to prevent movement.

Results

Since communication of pressure from the supratentorial space to the posterior fossa is dependent upon patency of the basal cisterns surrounding the brain stem in the tentorial incisura, an early manifestation of obstruction of the incisura is the development of a differential of pressure between the supratentorial and infratentorial spaces.* In these experiments transtentorial obstruction was said to be present whenever the intratentorial pressure fell below the supratentorial pressure during supratentorial injection of fluid.

The development of a progressive obstruction of the incisura is illustrated in Fig. 1. Several small injections, to a volume of 2.0 cc., had been made into an extradural balloon prior to injection in 1 where there is still full communication of pressure to the cisterna magna and lumbar subarachnoidal space. In 2 the peak of the pressure in the cisterna magna fell below the supratentorial pressure, and in 3 a 15 mm. Hg differential had developed. In 4 the increase of pressure with injection was followed by a secondary rise in intracranial pressure, and with the subsequent injection obstruction of the tentorial incisura was virtually complete. Injection of saline into the lumbar subarachnoidal space or cisterna magna invariably reduced the transtentorial obstruction (Fig. 1 5), and this occurred irrespective of the height of the supratentorial pressure. In one preparation the supratentorial pressure was raised to 250 mm. Hg and main-

* The term obstruction is used in preference to herniation since herniation cannot be diagnosed on the basis of a differential of pressure alone.