Transmission of Increased Intracranial Pressure

II. Within the Supratentorial Space*

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In previous experiments in which sustained increased intracranial pressure was produced by a gradually expanding extradural mass, the pressure within the extradural mass was equal to the pressure in the opposite extradural space in some animals, whereas in others it was many times the contralateral extradural pressure. This demonstrated that the dura mater can restrain significantly the expansion of an extradural mass and prevent transmission of pressure from the mass to the underlying brain.

It has also been found that a progressive failure of communication of pressure from the supratentorial to the infratentorial space occurs as cerebral tissue obstructs the tentorial incisura, and herniation of the cerebellar tonsils into the foramen magnum prevents transmission of pressure from the posterior fossa to the spinal canal. Thus, in these circumstances the brain does not transmit increased intracranial pressure.

Based on these preliminary observations experiments were designed to further investigate the transmission of increased pressure from the extradural space to the subarachnoidal space and in the opposite direction; within the supratentorial subarachnoidal space; from the subarachnoidal space to the brain; and from an intracerebral mass to the subarachnoidal space.

Materials and Methods

In these experiments 12 acute and 14 chronic monkeys were used. All of the acute monkeys also provided data for the previous report.

Communication of pressure between an intracerebral mass and the subarachnoidal space was assessed by inserting a 1 cc. balloon into the frontal lobe with care to prevent communication of the wall of the balloon with the frontal horn of the lateral ventricle. The volume of saline injected into the balloon was always less than 1.0 cc. and rarely exceeded 0.5 cc.

The relation of extradural to subarachnoidal pressure was investigated by inserting a steel bolt into the extradural space over the cerebral hemisphere. The bolt is hollowed out and needles are inserted through a diaphragm in the head of the bolt for injection of fluids into the extradural space and for recording of pressure. The bolt, which is self-tapping, is screwed into place through a trephine hole in the skull, and a gasket between the head of the bolt and the skull forms a watertight seal. Following placement of each bolt two needles are inserted through the diaphragm. Penetration of the dura mater cannot occur because the base of the shank of the bolt is left intact. One needle is attached to a Sanborn transducer and polygraph for continuous recording of the extradural pressure and injections of saline or Pantopaque are made through the other needle. In chronic animals pressures usually are recorded daily and small quantities of Pantopaque are injected in order to produce or maintain increased intracranial pressure as determined by the pressure in the extradural space contralateral to injection. This technique has been described in detail in another publication.

Results

In initial chronic experiments in which pressure was recorded from one extradural bolt it was our impression that the extradural pressure was an accurate reflection of the subarachnoidal pressure. However, when bolts for injection were inserted bilaterally it was evident that the pressure in the extradural space on the side of injection was not transmitted uniformly to the opposite side. This is illustrated in Fig. 1 where multiple prior injections of Pantopaque into the left extradural space had gradually increased the
pressure within the space to 24 mm. Hg, whereas the pressure in the right extradural space had remained essentially unchanged at 7 mm. Hg. At this time injection on the left increased the pressure to 40 mm. Hg with minimal transmission to the opposite side.

In order to determine the relation of the extradural pressure on the side opposite injection to the subarachnoidal pressure simultaneous recordings were obtained from the extradural space bilaterally and from the subarachnoidal space. In the experiment illustrated in Fig. 2, a catheter was placed in the left frontal subarachnoidal space for injection of saline, and pressures were recorded from bolts in the right frontal and left parietal extradural spaces and from the right parietal subarachnoidal space. A subarachnoidal injection of saline ① resulted in equal transmission of pressure to the opposite subarachnoidal space and peripherally into both extradural spaces. In contrast, injection into either extradural space ② and ③ resulted in incomplete communication of pressure to the subarachnoidal space. However, that pressure communicated to the subarachnoidal space was transmitted un-

diminished to the opposite extradural space.

Because of the possibility of gradual transmission of pressure from an extradural mass to the brain over a prolonged period of time, the problem was also investigated in chronic preparations in which a slowly expanding extradural mass was created by periodic injections of Pantopaque. In Fig. 3A pressures were recorded daily from steel bolts inserted bilaterally in the extradural space. Injection of 1 cc. Pantopaque on the right side on day 1 increased the pressure ipsilaterally, but there was failure of significant communication of pressure to the left side.

Fig. 1. Pressures are recorded from the extradural space bilaterally. Prior injections on the left have produced a sustained elevation in the pressure in the left extradural space whereas the pressure on the right has remained normal. Another injection on the left at this time shows minimal communication to the opposite extradural space. In this and all subsequent illustrations pressures are recorded in mm. Hg.

Fig. 2. Pressures are recorded from the right cerebral subarachnoidal space and from the extradural space bilaterally. Injection of saline through another catheter in the cerebral subarachnoidal space results in complete communication of pressure outward into both extradural spaces. In ① an injection is made through a bolt into the right extradural space while recording from another needle in the bolt, the opposite extradural space, and the cerebral subarachnoidal space. Only a fraction of the pressure on the side of injection is transmitted to the subarachnoidal space, but the pressure in the subarachnoidal space is fully communicated to the opposite extradural space. Similar pressure relationships are seen in ③ with injection of the left extradural space, but with a greater degree of communication of pressure.
On the following day pressure on the left was elevated and the previous differential of 19 mm. Hg had been reduced to 5 mm. Hg. Pantopaque was again injected on the right, and at the end of 60 min. of continuous recording a differential of pressure of 18 mm. Hg persisted. A similar result was obtained on day 3, and on the 4th day a spontaneous rise in pressure had occurred with partial transmission. On day 5 the pressure in the right extradural space had been dissipated, but the intracranial pressure remained elevated with nearly complete communication between the two sides.

In another experiment (Fig. 3B) a quantity of Pantopaque insufficient to produce a significant elevation of pressure was injected into the left extradural space on the first day. Twenty-four hours later pressure on the left was increased because of the formation of an extradural exudate, and there was approximately 50 per cent transmission to the right side. During the next 4 days the pressure gradually equalized and remained essentially equal until a further injection was made on day 8. Again transmission was delayed but was complete by the following day.

The extent of immediate communication of pressure was dependent upon the rate of injection. With rapid injections the pressure in both extradural spaces rose abruptly and often equally as a “shock wave” was transmitted across the intracranial space. However, as both pressures fell a differential frequently developed, and it is this incomplete communication of pressure from the extradural space to the brain that is of primary interest.

Rarely, even with slow injections, there was full and persistent transmission of pressure, as illustrated in Fig. 4. On day 1 a 1 cc. 15-sec. injection of Pantopaque was made into the right extradural space, and pressure on the opposite side followed the injected side with a slight lag. On the following day an additional 1 cc. was injected with essentially the same pattern of response. Following the injection on day 3, the pressures rose promptly, with again a slight lag on the opposite side, and then continued to increase to 135 mm. Hg at the end of 3 min. Respirations became irregular, and 0.5 cc. Pantopaque was withdrawn from the extradural space with a fall in pressure to 38 mm. Hg at 5 min. In spite of the decrease in pressure, respiratory arrest occurred and an additional 0.2 cc. Pantopaque was removed, following which the pressure returned to normal and spontaneous breathing was restored. During this entire period the pressures in the extradural spaces remained nearly identical, demonstrating complete and almost immediate transmission throughout the supratentorial space. We have observed such marked secondary increases in intracranial pressure in numerous acute and occasional chronic experiments in which the volume of the extradural mass was gradually enlarged.
Transmission of Increased Intracranial Pressure. II

The phenomenon has been described and discussed in detail by Lundberg in patients with intracranial hypertension.

These results demonstrate that the factor limiting communication of pressure between the extradural spaces is the dura mater on the side of injection, but that the pressure transmitted to the underlying subarachnoidal space is then fully communicated throughout the intracranial cavity. In order to confirm further the completeness with which pressure is transmitted throughout the supratentorial subarachnoidal space, catheters were inserted at 4 locations over the cerebral hemispheres. Each catheter was then injected with saline while recording from the other three. Fig. 5 shows complete transmission of pressure to the left frontal, left parietal, and right parietal subarachnoidal space with injection of the right frontal catheter. The other catheters were then injected in rotation with the same results.

An attempt was made to extend these observations to subacute and chronic preparations because of the possibility that failure of communication of pressure might occur with progressive obliteration of the subarachnoidal space in the basal cisterns, over the corpus callosum because of herniation of brain tissue under the falx cerebri, and over the surface of the cerebral hemispheres. In all animals in which the intracranial pressure was markedly elevated and subsequent postmortem examination showed displacement and herniation of brain tissue, the pressure was fully communicated over the surface of the cerebral hemispheres as long as it was possible to record the pressure from the subarachnoidal space. However, this does not rule out the possibility that the brain substance does not freely transmit pressure under those circumstances and that reduction in communication does occur when the subarachnoidal space is obliterated, at a time when it is no longer possible to record pressure with the present technique.

In order to investigate the possibility that the brain may not fully communicate

Fig. 4. Pressure is plotted against time. Pressure on the right is represented by circles, on the left by triangles. The open symbols indicate control pressures and the closed symbols postinjection pressures at the indicated times. The first arrow on day 3 represents withdrawal of 0.5 cc. Pantopaque and the second arrow 0.2 cc. Pantopaque from the right extradural space. Complete description is in text.

Fig. 5. Injection of saline into the right frontal subarachnoidal space demonstrates rapid and complete transmission of pressure to the remainder of the cerebral subarachnoidal space.
increased pressure developed within an expanding intracerebral mass, injections of small quantities of saline were made into an intracerebral balloon while recording bilaterally from the cerebral subarachnoidal space, and from the cisterna magna and lumbar subarachnoidal space. Care was exercised not to exceed the previously determined volume of the balloon, so that elastic tension within the wall of the balloon would not cause artifactualy high pressure.

In Fig. 6A, 0.2 cc. saline was injected into an intracerebral balloon through the side arm of a 3-way stopcock in order to record the pressure continuously during injection. As the pressure in the balloon gradually increased there was virtually no initial communication to the subarachnoidal space. At the end of injection the intracerebral pressure was 70 mm. Hg, the subarachnoidal pressure was 30 mm. Hg, and there was full communication of pressure from the subarachnoidal space to the cisterna magna and sites of lumbar recording. With repeated injections the differential of pressure decreased, but complete transmission of pressure from the intracerebral balloon to the subarachnoidal space did not occur. In contrast, Fig. 6B demonstrates immediate and complete communication of pressure from the surface of the cerebral hemisphere to the intracerebral balloon following the injection of saline into the cerebral subarachnoidal space.

Discussion

With the gradual expansion of an extradural mass, transmission of pressure from the mass to the brain is dependent upon the firmness of attachment of the dura mater to the inner table of the skull, its configuration in relation to the underlying brain, and its physical properties. An initial injection into the extradural space forces the dura mater away from the skull, and there may be immediate and complete transmission of pressure to the underlying subarachnoidal space and brain. With additional injections the pressure continues to be communicated as long as the expansion of the mass is not limited. However, if the dura mater cannot be stripped from the inner table of the skull, pressure rapidly increases within the extradural space, and communication of pressure is then dependent upon the distensibility of the dura mater.

Flexner et al. investigated extensively the relationship between the volume of dislocated spinal fluid and its pressure, as measured by manometers attached to a needle in the cisterna magna of dogs, and from this information they calculated a coefficient of elasticity for the contents of the cranial and spinal spaces. However, Pollock and Boshes demonstrated that this is almost entirely a measure of the elasticity of the intracranial blood vessels and not the meninges.*

* In physics, elasticity is that property of a body which allows it to resist deformation and to recover its original shape and size when the forces causing the deformation are removed. Thus, steel is a very elastic material. In contrast, in popular parlance an elastic substance is one that is easily deformed, and readily returns to its prior state, such as an elastic band. In order to avoid confusion in terminology, the word distensibility is used here to mean the extent to which the
In order to investigate the stretching properties of dura mater, two strips, approximately 3 by 2 cm. in diameter and .015 cm. thick, were obtained from a monkey immediately after sacrifice, and were suspended in Ringer's solution with weights of 50 and 200 gm. A 3-in. length of wire, measuring .2 mm. in diameter, was attached to the clamp holding each weight and served as a pointer. Each pointer was reflected in a glass mirror with a millimeter ruler between the pointer and the mirror. With this design readings were accurate to approximately .2 mm. Daily measurements of the length of the dural strips were made for 7 days. There was no measurable change in length with the 50-gm. weight and only .2 mm. of stretch occurred with 200 gm. By determining the cross-sectional area attached to the weight (width\times thickness) it was determined that the forces exerted on the dura mater by the 50- and 200-gm. weights were 1530 and 3250 mm. Hg respectively. Because these pressures are so far in excess of any encountered in vivo we believe that it is safe to assume that the dura mater is essentially nondistensible, in spite of the obvious limitations of an in vitro experiment.

Thus, if the dura mater does not stretch within the range of pressure investigated in these experiments, the volume of the extradural mass can expand only by a dissection of the dura mater from the inner table of the skull. If it remains firmly attached to the skull as the mass continues to expand, a large differential of pressure is created between the extradural and subarachnoidal spaces. As the pressure increases, gradual stripping of dura mater from the bone at the periphery of the mass may then occur and allow an increase in volume and a decrease in pressure within the mass.

Two theoretical configurations of an expanding extracerebral mass are illustrated in Fig. 7. On the right the mass is concave with respect to the underlying brain, and as it expands as the result of stripping of dura mater from the skull, the mass may retain its concave shape as it advances against the brain. With such a concave extradural collection of fluid and a taut, nondistensible dura mater, the pressure within the extradural space will be a function of the rates of accumulation of fluid and dissection of dura mater from the bones; that is, the volume of the space and the quantity of fluid within the space. On the other hand, the pressure in the underlying subarachnoidal space will be independent of the pressure in the extradural space and will be determined instead by the rate of expansion of the mass and, in turn, the ability of the volume of the intracranial contents to compensate for the volume in the extradural mass.

In contrast, if the shape of the extradural mass is convex in relation to the underlying brain (Fig. 7, left) and the dura mater is easily stripped from the skull, the extradural pressure will be fully transmitted to the underlying brain. This occurs because the only resistance the dura mater now offers to communication of pressure from the extradural space to the brain is ascribable to its rigidity, which is virtually nil compared to its nondistensibility.

That dissection of the dura mater may occur slowly is illustrated in Fig. 8. This animal received 4 daily extradural injections of Pantopaque, totaling 3.8 cc., and the antero-

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**Fig. 7.** A sketch of two possible configurations of an extradural mass in relation to the underlying brain. Description in text.
posterior roentgenogram of the skull on the left showed accumulation of the Pantopaque over the superior surface of the hemisphere. Two days after the last injection a repeated film demonstrated a thin film of Pantopaque extending toward the temporal fossa, and 7 days following the last injection the mass extended over the entire convexity of the hemisphere.

If it is assumed that the arachnoid is also relatively nondistensible, particularly with the addition of a membranous layer, as in subdural hematoma, it appears quite likely that a gradient of pressure could be created between a subdural collection of fluid and the underlying brain. Expansion of the mass might also occur with either of the configurations illustrated in Fig. 7.

With regard to communication of pressure within the brain von Bergmann1 was apparently the first to conclude from experimental studies that the brain does not transmit pressure equally in all directions. Cushing2 stated that the pressure exerted by an intracranial foreign body “is not transmitted equally throughout the cerebral chamber, and in consequence the circulatory embarrassment in corresponding degrees is unevenly felt.” Failure of complete transmission of pressure from an intracerebral mass to the overlying subarachnoidal space has been demonstrated in the present experiments by injections into an intracerebral balloon. With small volumes of the balloon the intracerebral pressure was elevated to several times the subarachnoidal pressure over the corresponding cerebral hemisphere, and communication of pressure to the subarachnoidal space occurred gradually as the intracerebral mass was enlarged with additional injections. In contrast, transmission of pressure from the subarachnoidal space through the brain to the intracerebral balloon was invariably complete and virtually instantaneous.

If the brain were completely nondistensible the creation of an intracerebral mass would be impossible. If it were freely distensible and still enclosed in a rigid container an increase in volume anywhere within the brain would result in an increase in pressure transmitted equally and virtually instantaneously throughout the intracranial space. No attempt has been made to define experimentally the relative distensibility of brain, but it is apparent that it is partially distensible and that this is a possible explanation for the failure of communication from an intracerebral mass to the subarachnoidal space. However, another explanation is afforded by the fact that blood can be expressed readily from the brain because of the low pressure in the dural sinuses. Thus, increased intracerebral pressure could be dissipated in this manner, and the pressure at any given time would then be a function of the volume of the material added and the volume of blood expressed from the intra-
cranial cavity. When an intracerebral balloon is inflated pressure in the brain immediately surrounding the balloon is increased, probably because the local blood volume cannot be reduced rapidly enough to accommodate the volume of the balloon. As the pressure is transmitted toward the cerebral surface it should be dissipated by the progressively larger volume of blood vessels available to absorb the pressure, but this hypothesis requires further experimental evaluation.

In any study of the pathophysiology of an expanding intracranial mass both the volume of and pressure within the mass must be considered. Under certain circumstances pressure may be entirely a function of the rate of expansion of the mass and the ability of the intracranial contents to compensate for it, but this is true only if the compartment or structure containing the lesion does not resist its expansion. If expansion is limited, by the dura mater or surrounding brain for example, pressure within the mass will increase out of proportion to its volume. In the case of a lesion within the brain this may lead to functional derangement of nervous tissue in its vicinity, resulting in neurologic signs above and beyond those created by the destructive effect of the lesion.

Frequently, in patients with obviously long-standing intracranial space-occupying pathology neurologic signs and symptoms develop acutely and death will ensue if the offending lesion is not removed quickly. It is likely that this usually is caused by the volume of the intracranial contents having compensated maximally, and with a slight further increase in the size of the lesion intracranial pressure rises rapidly. However, the present investigation provides two other possible explanations. In the case of an extradural, and perhaps a subdural, collection of fluid, a change of the configuration of the mass from concave to convex in relation to the brain could lead to rapid expansion of the mass and transmission of increased pressure to the brain. Also, with an expanding intracerebral mass dissipation of increased pressure is probably dependent on a gradient of pressure between the capillaries and veins surrounding the mass and the dural sinuses. If this theory is correct an increase in systemic venous pressure transmitted to the sinus and thence to the intracerebral capillaries and veins would lead to a rapid transmission of pressure from the mass throughout the brain and the sudden development of neurologic signs.

**Summary**

A study has been made of the transmission of increased intracranial pressure among various structures and compartments within the supratentorial space.

Transmission of increased pressure from the extradural space to the subarachnoidal space varies greatly and is dependent on the volume of the mass and the configuration of the nondistensible dura mater in relation to the underlying brain.

Increased pressure is transmitted rapidly and completely throughout the subarachnoidal space over the cerebral hemispheres.

Communication of increased pressure from an intracerebral mass to the cerebral subarachnoidal space is limited by the distensibility of the brain and the fact that the capillary venous bed can absorb the pressure by an increased flow of blood toward the dural sinuses.

In contrast, rapid and complete transmission of increased pressure occurs from the subarachnoidal space to the brain.

**References**