Infratentorial Tumors and the Dural Venous Sinuses

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It has been shown that septic, traumatic, and congenital lesions involving the major dural venous sinuses are accompanied by intracranial hypertension and hydrocephalus. It is more intricate and difficult to explain the mechanics of intracranial hypertension and hydrocephalus in intracranial tumors on the basis of hemodynamics in the dural venous sinuses. With a few rare exceptions, no satisfactory explanation for the mechanics of intracranial hypertension in intracranial tumors can be found.

However, Swift observed that a large tumor in the frontal lobe produced papilledema very late. In contrast, a small tumor in the cerebellum produced papilledema and symptoms of intracranial hypertension very early. After astute clinical observations and pathologic studies, Swift arrived at plausible mechanical explanations for these phenomena. Thus he was the first to describe in detail the mechanical development of intracranial hypertension and papilledema as a result of direct and indirect pressure of brain tumors on the dural venous sinuses.

In 1928, it occurred to Gardner that a tumor of the cerebellopontine angle might cause compression of the lateral sinus. To demonstrate this effect he carried out the Tobey-Queckenstedt test in a series of 5 cases suspected of posterior fossa lesions. In 3 cases the lack of response was attributed to obliteration of the lumen of the lateral sinus by pressure of a cerebellopontine angle tumor. No mention was made of the degree of intracranial hypertension or hydrocephalus or the results of postoperative jugular compression of these cases.

Russell considered an alternative mechanism to obstruction of fluid pathways in the production of hydrocephalus. He felt that obstruction of the lateral sinus by a mass in the neighborhood might sufficiently impair the absorption of cerebrospinal fluid to cause some degree of hydrocephalus. In one case a meningioma astride the right lateral sinus was associated with an appreciable degree of hydrocephalus. There was no actual occlusion of the lumen of the aqueduct and it might be argued that the hydrocephalus followed interference with the circulation of the cerebrospinal fluid in the basal cisterns.

Lindblom found a wide occipital emissary vein opening in 10% of intracranial tumors. He ascribed this to obstruction of drainage of venous blood toward the sinuses, secondary to compression of the sinuses. Taversa and Chynn agreed that the sign indicated chronic increased intracranial pressure. Chynn also stated that the widened occipital emissary channel decreased in size after removal of lesions which caused intracranial pressure.

Recently Kruyff and Munn described lateral displacement of the tuberculum jugulare as a significant sign of an expanding posterior fossa lesion. They thought that an impairment of venous blood flow through the sigmoid sinus and jugular foramen caused by-passing of venous blood, thus overloading and enlarging the emissary veins.

There is, therefore, sufficient indirect evidence of pressure application by tumors to the lateral and sigmoid sinuses, causing impairment of venous drainage via the normal and major channels of drainage. Until dural sinus venography was applied to this problem, there was no other direct method by which the hemodynamics could be studied. This technique has been applied to infratentorial tumors with results which may clarify the problem of increased intracranial pressure associated with these lesions.

Batson studied all the venous channels and emissary veins draining the adult skull and found that the venous outflow of the brain is by way of the internal jugular veins, by way of the anterior anastomoses with the orbit and pterygoid plexuses, by way of emissaries though the cranium and, especially im-

Received for publication February 21, 1966.

* Editor's Note: This manuscript was found among the papers of the author after his recent death and was forwarded to the Journal by his wife. Communications regarding this paper should be addressed to: Dr. Alexander Slepian, 959 Lafayette Avenue, Niagara Falls, N.Y.
The occipital sinus, the basilar sinus, the mastoid veins and the posterior condylar veins all drain into the network of the vertebral veins. This network begins with the dural sinuses and extends the entire length of the spinal column. The total cross-sectional area of the system, as it leaves the skull, has not been estimated. From corrosion preparations it appears greater than the combined area of the two jugular veins. There are also several unpredictable communications of the cerebral circulation with the vessels of the scalp by way of emissary veins.

The significance of all the plexuses is that they appear to operate under normal physiological conditions as in coughing, sneezing, straining, and jugular compression. In a certain number of individuals they are capable of taking over the entire drainage of blood after acute occlusion of the dural sinuses or jugular veins. Should these collateral channels of drainage become inadequate because of abnormal occlusion of the major dural venous sinuses, the intrasinus venous pressure will be increased. Compensation for increased intrasinus venous pressure occurs when the collateral channels are adequate in number and caliber and capable of diverting the blood into adequate exit channels.

We tested the deductions made by Swift and others in 4 cases of posterior fossa tumor, evaluated by dural sinus venography. These cases illustrate the preoperative venous drainage of the cranium affected by indirect and extrinsic pressures of brain tumors on the dural venous sinuses. Postoperative films in these cases illustrate venous drainage of the cranium after restoration of normal channels of drainage by removal of the tumor and thereby the indirect pressure on the dural venous sinuses. At the same time Batson’s anatomical observations on the collateral channels during major obstruction of the dural venous sinuses were re-evaluated. In each case we have reported only the material pertinent to the subject.

Case 1. A 4-year-old girl had the signs and symptoms of a posterior fossa tumor. The cracked-pot sound of MacEwen was present. There was no papilledema. X-ray of the skull showed suture separation. The cranial bones were thin and neither imprint of the lateral sinus was seen on the occipital view. The jugular tubercles were displaced laterally and the occipital emissary vein was not enlarged. The ventriculograms showed a mass lesion in the 4th ventricle.

Comment. It was apparent that the medulloblastoma exerted indirect pressure in the posterior fossa sufficient to impair the venous drainage via the lateral and sigmoid sinuses. The drainage of blood via the dominant right sigmoid sinus and the stenotic left sinus system was virtually abolished. Probably the tributaries to these sinuses, the superior and inferior petrosal sinuses, were also rendered useless. The collateral channels of drainage visible in this case were the diploic, scalp, external occipital and condylar veins, and the vertebral plexuses. These findings implied that there was considerable pressure on the dural sinuses before surgery, not only from the posterior fossa lesion but also from the distended ventricles in the supratentorial compartment. After relief of indirect pressure on the dural venous sinuses by removal of the tumor, the dominant right dural system and the stenotic left dural sinus system were clearly visible. The caliber of the dural sinuses at this time was increased in comparison to their preoperative state. The collateral channels of drainage were not visible, probably because there was no need for them following the relief of the intracranial pressure on the dural sinuses (Figs. 1 and 2).

Case 2. A 7-month-old infant showed signs and symptoms of progressive hydrocephalus. The head circumference was 18\frac{1}{2} inches. The suture lines were palpably separated and the anterior fontanelle was tense. The scalp veins were distended. X-rays of the skull showed thin cranial bones with separated sutures and an open, bulging anterior fontanelle. Neither the occipital emissary vein nor the jugular tubercles were identified. Cerebrospinal fluid pressure was over 600 mm. The spinal fluid proteins were 139 mg. A ventriculogram showed marked hydrocephalus and obstruction in the upper end of the aqueduct. The preoperative serial sinograms were shown in Fig. 3.

Operation. Suboccipital craniectomy showed a cystic astrocytoma (grade 3) of the vermis with complete obstruction of the 4th ventricle and the aqueduct.

Postoperative Course. Seven weeks after surgery, shortly after regression began, the serial sinograms were repeated and are shown in Fig. 4. The intrasinal pressure was 220 mm, while the intraventricular pressure was 240 to 250 mm.