Neurocrit Care
https://doi.org/10.1007/s12028-022-01627-8

NEUROCRITICAL CARE SOCIETY

NEUROCRITICAL CARE THROUGH HISTORY

"Brain + Compression = Unconsciousness": On Cones, Grooves, and Cerebellar Tissue That Moves

Eelco F. M. Wijdicks*

For most of the twentieth century, conventional wisdom taught that cortical anemia was caused by acute cerebrospinal fluid obstruction, resulted in loss of consciousness. In 1950, Jefferson and Johnson [1] sneeringly said, "In this way loss of consciousness can be rationalized by those so cortically minded that they believe that not only all thinking but simple awareness of environment [is] dependent on the cortex alone." Through a number of cases, they argued that local compression of the brainstem was the mechanism of loss of consciousness and supported it by demonstrating that patients markedly improved after decompression from evacuation (they wrote [1] that one patient "looked very dead now—there were no reflexes of any sort; no corneals"). Animal experiments showing respiratory, blood pressure, and heart rate changes could not offer clues about unconsciousness because the animals were anesthetized. Neurosurgeons were the first to note that stupor did not correlate with high cerebrospinal fluid pressures; hence, it had to be local compression. Citing Jefferson and Johnson, "the equation brain + compression = unconsciousness seemed as self-evident as 2 + 2 = 4" [1].

Earlier, Sir Geoffrey Jefferson studied the anatomical relations of the tentorial hiatus in greater detail [2] and summarized it as follows:

> *If one lobe is enlarged it cannot escape overhanging the free edge. For this reason, a tumor of the temporal lobe will be the surest way of bringing it more firmly into contact with the midbrain and squeezing its inner border over the sharp edge of the falx, into a situation in which it can herniate downward into the posterior fossa.*

Jefferson coined the terms "tentorial pressure cone" and "temporal pressure cone." Although these terms introduced the word "pressure" in the dynamics of the process, Jefferson was unable to pinpoint the main variables. Pathological findings seen at autopsy included compression of cranial nerves traversing the subarachnoid space, mesencephalic hemorrhages, and kinking of the posterior cerebral arteries. Pathologists recognized that cerebellar tissue displaces toward areas of least resistance, and therefore attention focused on the foramen magnum, where the cerebellar pressure cone not only involved cerebellar tonsils but also created upward herniation through the tentorial opening.

Earlier Observations

In his famous work published in 1896, Sir Leonard Hill also noted displacement but supported ischemia as the most important mechanism [3].

> *In this process the cerebellum takes the form of two cones constituted by the cerebellar tonsils. As a result the medulla oblongata is trapped within the foramen magnum, and if the pressure is strong enough its vessels will be compressed to the point of failing to supply the organ. Hence a severe bulbar ischemia will ensue…it follows that the most unfavorable condition of compression is the local compression of the bulbar centers and pressure applied there and only there but little above the capillary pressure is sufficient to kill…the bulbar*

*Correspondence: wijde@mayo.edu
Neurocritical Care Services, Saint Marys Hospital Mayo Clinic, 200 First Street SW, Rochester, MN, USA

This article is part of the collection "Neurocritical Care Through History".

Springer

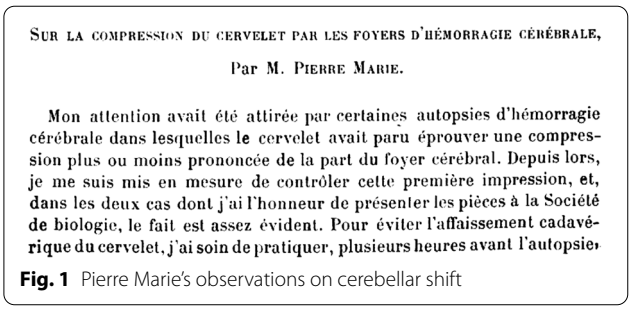
centres must be compressed to produce the major symptoms of apoplexy.

Hill understood this as "pain due to tension of the dura mater." He noted stupor; sopor; coma; clonic spasms and circuitous movements; slow heart rate; vomiting; emptying of the bladder and rectum; constriction of the pupils, first on the compressed side, followed by dilatation; and ceasing of respiration and concluded that "sudden anemia of the medulla oblongata causes spasm of an asphyxial type" [3].

In 1899, during a session of the Société de Biologie, Pierre Marie reported on two cases of cerebral hemorrhage resulting in secondary compression of the cerebellum [4] (Fig. 1). In the first case, a putaminal hemorrhage correlated with a flattened superior face of the left cerebellar lobe and shifted the vermis. In the second case, a thalamic hemorrhage additionally caused protrusion of the cerebellar tonsils (*amygdales cérébelleuse*). Pierre Marie considered the consequences of cerebellar tonsillar descent into the occipital foramen but decided that bulbar hypoperfusion was the main mechanism. In the next year, Pierre Marie reported another two cases of raised intracranial pressure, but in the second case, which had increased intracranial pressure with hydrocephalus, he noted that cerebellar tonsils formed a cone enclosing the bulbus. In his conclusions, Pierre Marie suggested that the compression of the medulla contributed to the fatal outcome [5]. This differed markedly from his earlier opinions, in which he considered it an autopsy artifact.

In 1938, Jacques LeBeau reported herniation of the cerebellar roof upward through the tentorial hiatus in a case of cerebellar tumor [6] (Fig. 2). However, he later described the ventriculographic changes caused by tumor-associated tissue shift of the upper portion of the cerebellum in the tentorial notch [7].

Working more diligently, Arthur Ecker developed a detailed anatomical and physiologic description of increased intracranial pressure as a result of direct compression of the vein of Galen by upward push of the herniating cerebellar roof through the tentorial notch [8]. Moreover, downward herniation of the hippocampal gyrus or upward herniation of the cerebellum further compresses the veins of Rosenthal and Galen. He also noted that the basilar vein of Rosenthal was distorted as a result of dislocation or rotation of the brainstem in relation to the tentorial notch. These changes would cause "double blockage of cerebrospinal fluid: both in downward flow at the aqueduct and in upward flow at the pontile and ambient cisterns," adding to the hydrocephalus, intracranial pressure, and the congestion of the brainstem (Fig. 3) [8]. Ecker concluded that prolonged supratentorial ventricular drainage might be useless or harmful, but sectioning of the tentorium at the notch was essential in many of these cases to decompress the brainstem and nearby veins.



SUR LA COMPRESSION DU CERVELET PAR LES FOYERS D'HÉMORRAGIE CÉRÉBRALE,

Par M. PIERRE MARIE.

Mon attention avait été attirée par certaines autopsies d'hémorragie cérébrale dans lesquelles le cervelet avait paru éprouver une compression plus ou moins prononcée de la part du foyer cérébral. Depuis lors, je me suis mis en mesure de contrôler cette première impression, et, dans les deux cas dont j'ai l'honneur de présenter les pièces à la Société de biologie, le fait est assez évident. Pour éviter l'affaissement cadavérique du cervelet, j'ai soin de pratiquer, plusieurs heures avant l'autopsie,

Fig. 1 Pierre Marie's observations on cerebellar shift

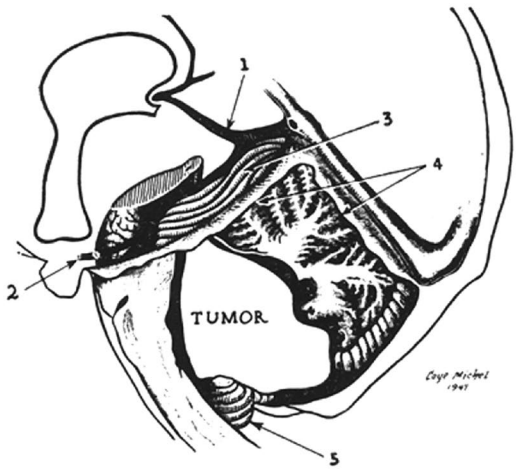
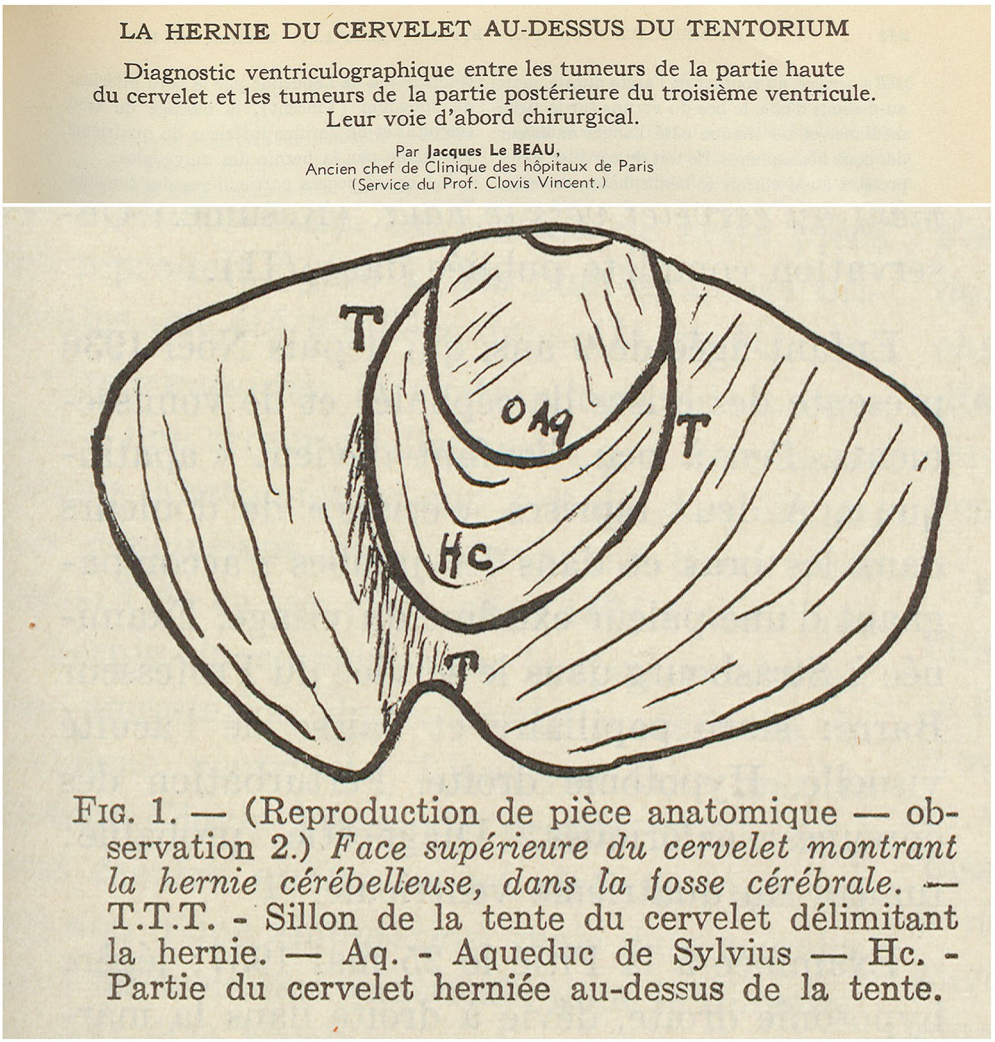


FIG. 2. Drawing based on study of necropsy, venogram and ventriculogram in Case 1, showing herniation of cerebellum (3) and brain stem upward through tentorial notch. The subtentorial structures are shown in sagittal section after removal of their left halves. The cerebral hemispheres were removed after transverse section through upper portion of midbrain before tentorium (4) or structures of posterior fossa were disturbed. Notice distortion and compression of veins of Galen (1) and Rosenthal (2). Cerebellar tonsil (5) is herniated downward into foramen magnum.

Fig. 2 LeBeau's evidence of upward herniation


LA HERNIE DU CERVELET AU-DESSUS DU TENTORIUM

Diagnostic ventriculographique entre les tumeurs de la partie haute du cervelet et les tumeurs de la partie postérieure du troisième ventricule. Leur voie d'abord chirurgical.

Par **Jacques Le BEAU**,
Ancien chef de Clinique des hôpitaux de Paris
(Service du Prof. Clovis Vincent.)

FIG. 1. — (Reproduction de pièce anatomique — observation 2.) *Face supérieure du cervelet montrant la hernie cérébelleuse, dans la fosse cérébrale.* — T.T.T. - Sillon de la tente du cervelet délimitant la hernie. — Aq. - Aqueduc de Sylvius — Hc. - Partie du cervelet herniée au-dessus de la tente.

Fig. 3 Ecker's observations on upward herniation

Questioning the Pathoclinical Correlate

Gordon Klintworth was the first to question the meaning of cerebellar grooves and to look for another explanation for the superior cerebellar grooves, which for pathologists, were considered pathognomonic for cerebellar herniation [9]. He noted that narrow dural folds on the inferior surface of the tentorium cerebelli could produce grooves when in contact with the superior surface of the cerebellum in brains free from overt diseases.

Cuneo and colleagues claimed there were recognizable clinical signs, but this attempt at a full description had to wait until 1979 [10]. Their observations claimed a trajectory of clinical findings related to tissue shift. The initial presentation was coma, small but reactive pupils, absent corneal reflexes, absent oculocephalic responses, and bilateral signs of pyramidal tract dysfunction referring to acute brainstem compression. The subsequent appearance of bilaterally fixed and dilated pupils indicated compression of the midbrain. "In our two patients, the progression from equal reactive pupils to anisocoria and then to midposition, fixed pupils indicated evolving upward herniation. In each case, pathologic evidence of upward transtentorial herniation with midbrain distortion and displacement was present" [10].

Not everyone agreed that an upward herniation syndrome could be defined, and most notably, C. Miller Fisher wrote [11], "The claim is being made that the aperture cerebellar herniation is an incidental late byproduct, a harmless telltale of increased posterior fossa content on the diffuse elevated pressure and not a special instrument for adding to the damage by throttling the brain stem". Fisher noted that extreme cerebellar herniation can be asymptomatic

and described an autopsy case of a 3- to 4-cm cerebellar herniation with hemorrhagic necrotic tips, proving its presence of several days. In a pathological study, Fisher found cerebellar hemorrhage with posterior fossa compression and respiratory failure but no pressure cone in 17 of 18 patients. Fisher wrote, "Displacement of cerebellar tissue into the foramen magnum may even afford perhaps some relief from crowding rather than being harmful." He also questioned whether urgent decompression could rescue these patients from respiratory arrest. "Our concern is how often we see respiratory arrest before loss of brainstem reflexes. Acute respiratory failure causing death in this condition may be rare" [11].

There is no documentation of neurosurgeons trying decompression, except for Harvey Cushing, who made one desperate attempt and, according to Shelton et al., never repeated it [12]. Cushing believed that increased intracranial tension led to tonsillar herniation and medullary compression, causing respiratory failure. In 1909, while removing a supratentorial tumor, he ineffectively attempted emergency decompression of the medulla oblongata in the hope of relieving possible bulbar ischemia due to compression. He removed the posterior margin of the foramen magnum and arch of the atlas and axis when herniation was extensive. In another article that year [13], Cushing warned about the danger of lumbar puncture: "If the brain after such an accident be removed from the cranial chamber soon after death, and particularly if hardened in situ, it will show the imprint of the foraminal ring about the protrusion which has been tightly jammed into the opening."

As a side note indicative of their perspicacity, Jefferson and Johnson postulated an affected center in the brainstem, which also implied that these neurons were involved in awareness. They hypothesized that the compression localized to an area in the mesencephalon extending down over the pons to the upper medulla oblongata and postulated a "waking" center but did not think it was "a group of master cells functioning as a nuclear mass" (Fig. 4) [1]. This prescient idea predated Horace Magoun's presentation of the ascending reticular activating system in his Salmon Lecture at the New York Academy of Medicine [14, 15] by nearly a decade.

Fast forward to today, we have not resolved the enigma of these clinical syndromes of cerebellar herniation. But we know surgery may be urgent [16]—or not [17, 18]. Research in swollen and moving cerebellar tissue caused us to question our knowledge of mechanisms and, incidentally, highlighted the upper brainstem as an important structure in understanding unconsciousness.

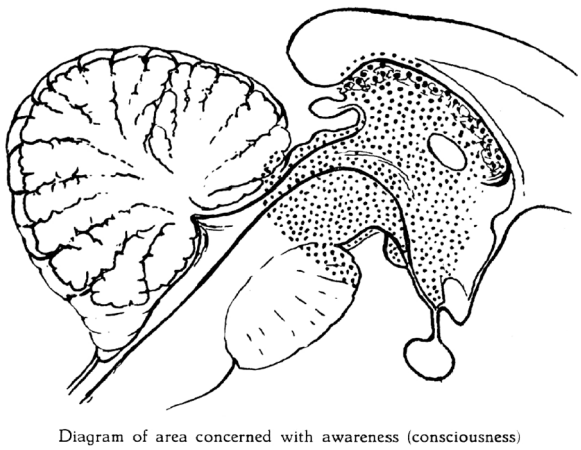


Fig. 4 Jefferson's theory of a "waking center."

Source of Support
No extramural funding supported this effort.

Conflicts of interest
There are no conflicts of interest.

Publisher's Note

Received: 30 September 2022 Accepted: 4 October 2022
Published online: 17 November 2022

References

1. Jefferson G, Johnson RT. The cause of loss of consciousness in posterior fossa compressions. Folia Psychiatr Neurol Neurochir Neerl. 1950;53(2):306–19.
2. Jefferson G. The tentorial pressure cone. J Nerv Mental Dis. 1938;40:857–76.
3. Hill L. The physiology and pathology of the cerebral circulation an experimental research. London: J & A Churchill; 1896.
4. Marie, P. Sur la compression du Cervelet par les foyers d'hemorrhagie cerebrale. Comples rendus des seances et memoires de la Societe de Biologie et de ses Filiales 1899.

5. Marie PD. l'engagement des amygdales cerebelleuses a l'interieur du trou occipital dans les cas ou la pression intracranienne se troube augmentee. Revue Neurol. 1900;8:252–3.
6. Lebeau, J., L'oedème du cerveau. Son role dans l'evolution des tumeurs et des abces intra-craniens. (Thesis), 1938, J. Recht.
7. Lebeau, J. La hernie du cervelet au-dessus du tentorium. Diagnostic ventriculographique entre les tumeurs de la partie haute du cervelet et les tumeurs de la partie postérieure du troisième ventricule. Leur vole d'abord chirurgical. Union Med du Canada 1944;73:243–52.
8. Ecker A. Upward transtentorial herniation of the brain stem and cerebellum due to tumor of the posterior fossa with special note on tumors of the acoustic nerve. J Neurosurg. 1948;5(1):51–61.
9. Klintworth GK. Superior cerebellar grooves in relation to upward transtentorial herniation. Arch Pathol. 1968;86(5):568–9.
10. Cuneo RA, Caronna JJ, Pitts L, Townsend J, Winestock DP. Upward transtentorial herniation: seven cases and a literature review. Arch Neurol. 1979;36(10):618–23.
11. Fisher CM. Brain herniation: a revision of classical concepts. Can J Neurol Sci. 1995;22(2):83–91.
12. Shelton BA, O'Hara E, Tubbs RS, et al. Emergency suboccipital decompression for respiratory arrest during supratentorial surgery: the untold story of a surgeon's courage in times of despair. J Neurosurg. 2009;110(2):391–4.
13. Cushing H. Some principles of cerebral surgery. JAMA. 1909;52(3):184–95.
14. Wijdicks EFM. The ascending reticular activating system. Neurocrit Care. 2019;31(2):419–22.
15. Magoun HW. The waking brain. 1st ed. Springfield: Charles C Thomas; 1958. p. 119.
16. Wang Y, Binkley MM, Qiao M, et al. Rate of infarct-edema growth on CT predicts need for surgical intervention and clinical outcome in patients with cerebellar infarction. Neurocrit Care. 2022;36(3):1011–21.
17. Wijdicks EF, Maus TP, Piepgras DG. Cerebellar swelling and massive brain stem distortion: spontaneous resolution documented by MRI. J Neurol Neurosurg Psychiatry. 1998;65(3):400–1.
18. Khan M, Polyzoidis KS, Adegbite AB, McQueen JD. Massive cerebellar infarction: "conservative" management. Stroke. 1983;14(5):745–51.