

ARTERIOVENOUS FISTULA BETWEEN THE MIDDLE MENINGEAL ARTERY AND THE GREATER PETROSAL SINUS*

CASE REPORT

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THE ADDITION OF THE middle meningeal artery to the anatomically named blood vessels of the body rather completes the list as sites for the development of traumatic arteriovenous fistulas. A similar experience to the case reported in this communication has not been found in the medical literature. In the combined statistics of the vascular centers in this country from World War II, 814 aneurysms and fistulas were totaled, but the middle meningeal artery was not implicated in this compilation. The fistula reported in this patient occurred between the middle meningeal artery and the greater petrosal dural sinus. Symptomatically it had its appearance one month after the patient had suffered a fracture of the skull. The commonness of fractures involving the base of the skull would intimate that a middle meningeal fistula has occurred before and will likely happen again. The diagnosis in this case was suggested by the radiologic changes brought out in the base view of the patient's skull radiograms (Fig. 1). In retrospect, had not a basic surgical principle in the treatment of arteriovenous fistulas been initially violated, the second operation might have been unnecessary.

CASE REPORT

History—Examination. On January 10, 1949, a 24-year-old woman reported for her initial neurosurgical examination. In August, 1943, she was thrown from a motor scooter and suffered a skull fracture. Three days later she recovered conscious orientation and full cognizance of her physical discomforts. She described no neurologic handicaps until 30 days later, when she became aware of a "buzzing noise" in her left ear. This was synchronous with her pulse beat and had been audible to more than one examining physician. Aside from

increasing nervousness and left-sided headaches, no new symptomatology developed during the ensuing 6 years. The bruit continued all through these years. The neurologic and physical examination of the above date netted no abnormal objective evidences. On auscultation there was a continuous bruit, "high pitched with a systolic accentuation" heard through the stethoscope and better heard with the ear directly against the zygomatic temporal area.

Angiography, External Carotid Ligation. On February 22, 1949, direct angiography of the left external and internal carotid arteries was performed. The visualization of the internal vessel in its arterial, capillary and venous phases depicted no suspicious deformities. The external carotid outline was that of a hypertrophied serpentine middle meningeal artery in that portion which traverses the lateral posterior floor of the middle fossa of the skull. The drainage from this arterial enlargement was into a pathologically hypertrophied sinus. This latter structure was anatomically that of the greater petrosal sinus (Fig. 2). Following the arterial injections, the external carotid artery was doubly ligated. The only influence on the bruit from this ligation was that subjectively there was "some alteration in the quality of the buzzing noise in the ear." The bruit was objectively audible but the pitch of the sound was higher.

Intracranial Ligation Middle Meningeal Artery. On March 1, 1949, this vessel was exposed transtemporally. Aside from increased tortuosity, the diameter of the extradural artery was some 3 times its usual appearance. The vessel seemed relatively void of blood and there were neither palpable nor visible pulsations. There was an accompanying enlarged venous sinus visible from the zygomatic exposure of the artery to within a millimeter or so of the foramen spinosum. This venous companion seemed to disappear within the dura in the direction of the innermost tip of the petrosal ridge. The middle meningeal artery was dissected out of an enlarged foramen spinosum. After this opening was completely plugged with cotton the artery was coagulated and severed some 3 mm. from its spinosal entrance on to the dura. The dural venous mate of the middle meningeal artery

was then thoroughly coagulated lateralward for a distance of 3.5 cm.

The bruit following this ligation was objectively absent for the period of the 9 days postoperative hospitalization. The patient, however, re-

of the skull from the foramen rotunda anteriorly, to the petrosal ridge posteriorly, and to the mid-level of the Gasserian ganglion mesialward. From the zygomatic level this dissected dura was circumcised. The greater petrosal sinus was sectioned in

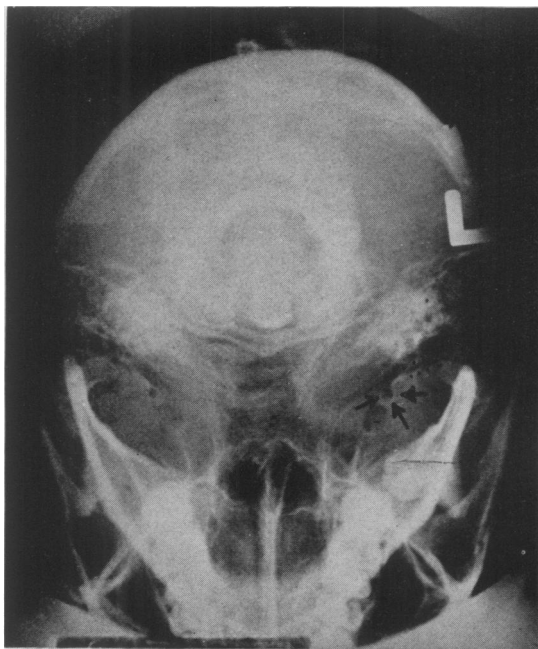


FIG. 1

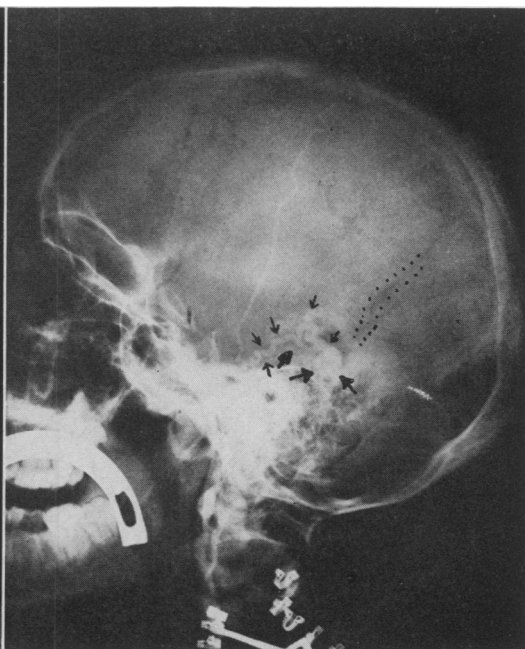


FIG. 2

FIG. 1.—The extension of the parietal-occipital skull fracture into the skull base is outlined by the inserted dots. The hypertrophy of the foramen spinosum (arrows) is obvious when compared with the normal opening on the right side.

FIG. 2.—The skull fracture was still radiologically much in evidence six years after the accident (dots). The tortuosity of the hypertrophied artery is shown; also the venous fistulous expansion, as outlined on external carotid angiography (arrows).

ported that in the quiet of the night she “thought on several occasions” the buzzing noise had been reduced to a transient momentary “purr.”

One month after leaving the hospital the patient reported a “return of the noise in the left ear—different in character” and heard “mostly at night when lying on the left side.” By October, 1949, the bruit was objectively present, having a continuous swirling sound and a sharp systolic squeak.

Resection of Dura, Intradural Artery and Greater Petrosal Dural Sinus: July 5, 1950. Two days previously the left internal carotid arterial visualization had been repeated, again with perfectly normal arterial and venous depictions of this system. The dura was stripped from the floor

the beginning of the dural sacrifice and it carried arterial blood. Until an intradural middle meningeal vessel was clipped at a point just above the foramen ovale this venous sinus persisted in containing arterial blood. When the sacrificed dura was subsequently dissected, the intradural artery, which paralleled the former extradural meningeal vessel for some 3 cm., was found to be as large as a normal mid-meningeal artery.

There developed a left Bell's palsy following the secondary operation. This was attributed to the dissection and sectioning of the greater superficial petrosal nerve. On September 6, 1950, facial symmetry, for emotional and volitional function, had been attained. The “buzzing noise in the ear” had not been heard since July 5, 1950. On November

20, 1950, the patient reported a continued freedom from her bruit, but thought that on holding her head "in a strained, extremely rotated position to the right" she could detect some abnormal sound in her left ear.

DISCUSSION

The enlargement of the foramen spinosum (Fig. 1) depicted on stereoscopic radiologic visualization of the basilar foramina of the skull implicated the middle meningeal artery as the possible site for the arteriovenous fistula in this case. The secondary operation established the superior petrosal sinus as the venous component of this fistula.

Had the author remembered the consistency of the presence of an intradural artery accompanying the first part of the

extradural middle meningeal vessel, resection of the entire lesion at the first operation might have been performed. Had the intradural artery been severed initially, the dural venous contributor likely would have necessitated its deposition for the control of further hemorrhage. From this, a more complete extirpation of the fistula would have resulted. Thus a basic surgical principle in the treatment of arteriovenous fistulas was violated for "ligation without excision usually results in a recurrence, since the fistula will be re-established rapidly through collateral vessels."¹

BIBLIOGRAPHY

- ¹ Elkin, Daniel C.: *Arteriovenous Aneurysm. Mod. Concepts Cardiovascular Dis. pamphlet, Am. Heart Assoc., 12: 1943.*

DISCUSSION.—DR. DANIEL C. ELKIN, Emory University, Georgia: I hesitate to rise, since Dr. Fincher quoted me in the last paragraph of his paper. I can assure you it is true of any arteriovenous fistula—unless you excise it, it is apt to recur. You may think there are no collateral vessels, but the tiniest vessels will become collaterals. No new vessels form, but these vessels become collaterals.

DR. EDGAR F. FINCHER, Emory University, Georgia (closing): I did not care to embarrass the Professor, but feeling that the middle meningeal artery was one textbook vessel which was a terminal structure, it was his suggestion that section of this vessel intracranially would probably cure the fistula. We only proved his early observations that total extirpation of an arteriovenous fistula is necessary if one is to have a cure.