HEADACHE in the frontal area is a symptom frequently associated with intracranial tumors. It occurs with many supratentorial masses and almost all tumors of the posterior fossa.\textsuperscript{14}

The mechanism of headache associated with intracranial mass lesions has been the subject of much study; in particular, the investigations of Wolff,\textsuperscript{14} Ray,\textsuperscript{12} Schumacher,\textsuperscript{13} and Kunkle\textsuperscript{8} have clarified many of the problems of pain posed by this type of pathology. Despite these and many other contributions, there remain perplexing features still hard to explain and reconcile with the known facts.

It is well known that headache may be misleading as a localizing sign of intracranial tumor, since, as pointed out by Wolff\textsuperscript{14} and others, it overlies the tumor in only about one-third of cases. Thus in many instances there is no correspondence between the mass and the headache. Of this, posterior-fossa tumors constitute a conspicuous example; for while the tumor is suboccipital, the headache commonly is frontal. Cushing,\textsuperscript{3} in reviewing his large series of cerebellar astrocytomas, stated that the headaches often were frontal rather than suboccipital and that in a few instances the frontal sinuses had been explored because of unexplained frontal headaches. He continued: "Indeed, by the time the headaches come to be localized in the suboccipital region the patient no longer refers to them as such but rather as [suboccipital] discomfort or a painful stiffness in the neck which is aggravated by any sort of effort likely to produce intracranial venous congestion."

Bailey et al.\textsuperscript{1} stated that 70 per cent of headaches were frontal in their series of tumors of the cerebellar fossa; and recently Bodechtel\textsuperscript{2} noted that when headaches are present with cerebellar tumor they are in the posterior region, the neck, and the forehead. Dandy\textsuperscript{4} also indicated the likelihood that headache with these tumors will be frontal.

Numerous other authorities have noted this referral of headache to the frontal area with expanding lesions of the posterior fossa, though occasionally it is stated that the headache in a given series of posterior-fossa tumors was in the back of the head.\textsuperscript{8}

PREVIOUS EXPLANATIONS

The mechanisms of headache in cases of brain tumor still are under study. It has been well established that increased intracranial pressure is not the basic mechanism involved. Northfield\textsuperscript{10} showed that the level of intracranial pressure as determined by spinal manometry has little to do with the presence of headache, and Wolff\textsuperscript{14} confirmed and extended these observations.

It was their opinion that the headache with mass lesions is related to shift of the intracranial contents with traction on pain-sensitive structures—an interpretation that generally is accepted. The identification of these pain-sensitive structures we owe particularly to Ray and Wolff\textsuperscript{12} who studied this point exhaustively in patients undergoing craniotomy with local anesthesia.

Although these observations and the known distribution of pain evoked by stimulation of the recognized pain-sensitive structures account for many of the peculiarities of headache of intracranial origin, in some instances one still cannot explain the discrepancy between the sites of tumor and of headache.
In the case of posterior-fossa tumors with bifrontal headache, for example, the most commonly cited explanation \(^1\)–\(^3\) is that pressure on the tentorium from below irritates the tentorial nerves. These nerves have been known to anatomists for over a century and have been the subject of careful study by Feindel and co-workers \(^5\) more recently. According to their descriptions, the tentorial nerves arise from the ophthalmic branch of the trigeminal nerve and run in a recurrent direction along the tentorial edge to distribute over the superior surface of the tentorium, the falx, and the region of the toreular. Stimulation in these regions gives rise to referred pain in the frontal and orbital regions—which is to be expected, since the tentorial nerve arises from the ophthalmic nerve.

Activation of this mechanism in the case of a posterior-fossa tumor must require a considerable degree of pressure from below to stimulate nerves that lie on the superior surface of the fairly rigid and thick tentorium. A possibility would be that branches of these nerves penetrate the tentorium to the underside and thus become accessible to stimulation. However, Ray and Wolff \(^12\) in their study of patients under local anesthesia stated:

"Slight or even moderate pressure upward [on the tentorium] usually failed to cause pain unless the margins of the venous sinuses were approached, in which case pain was usually experienced behind the homolateral ear. When pressure on the center of the tentorium was increased, pain occurred behind the ear, in the region of the forehead and eye on that side or in both regions. . . . Faradic stimulation of sufficient intensity to induce pain in the structures with well established sensitivity to pain . . . failed to cause pain when applied to points on the undersurface of the tentorium more than 5 mm. from the venous sinuses. It was found further, however, that if the intensity of the stimulus was increased sufficiently pain was experienced in the forehead and in the region of the eye on that side, suggesting that the stimulus was transmitted through the tentorium to its superior surface."

It seems clear from these observations of Ray and Wolff that a stimulus of considerable intensity is required to evoke frontal and orbital pain from the under surface of the tentorium, and furthermore, that pain behind the ear is often associated with such stimulation. Only when stimuli are applied to the upper surface of the tentorium is isolated frontal and orbital pain elicited at low-stimulus parameters.

Although in the late stages of a posterior-fossa tumor considerable distortion of the tentorium sometimes occurs, it would appear most unlikely that this mechanism is responsible for frontal pain. First, the bifrontal headaches in these cases often appear at an early stage, before there is any evidence of increase in intracranial pressure. Second, as noted previously, headache frequently is experienced without increase of pressure; and as shown by others, intracranial pressure has no relation to headache. Third, by the time there is a significant degree of pressure the headaches are no longer exclusively frontal but are replaced or accompanied by suboccipital discomfort (Cushing\(^5\)).

Thus, the current knowledge of pain mechanisms in headache does not appear to account for referral of headache to the forehead from posterior-fossa mass lesions.

A PROPOSED MECHANISM

Observation of response to stimulation in 4 patients undergoing upper cervical dorsal rhizotomy because of hemianial-pain syndromes led to consideration of a hitherto undescribed mechanism as a possible explanation for the pain.

These patients were operated on under local anesthesia and all were cooperative. Cervical laminectomy was performed and the upper three cervical roots were exposed; during the procedure each individual rootlet composing each dorsal root was stimulated separately. Initially a Grass stimulator was used, but subsequently gentle pressure or traction with bayonet forceps was found to be equally effective.

It was noted that when the dorsal rootlets of C2 were stimulated extension of pain to the frontal and orbital regions did not occur, though on one occasion a fine filament associated with this root did produce pain in the
FRONTAL HEADACHE WITH POSTERIOR-FOSSA TUMORS

Fig. 1. First cervical dorsal root at operation. A nerve hook (G) has been passed underneath the C1 dorsal rootlets (A, B, C) and the rudimentary dorsal root ganglion (H). The spinal accessory nerve (D) is seen running upward and passes beneath the dorsal rootlets of C1. The upper C2 dorsal rootlets (F) are seen out of the plane of focus, crossing the dentate ligament (E) in the lower angle. In this instance the C1 rootlets were better developed than usual.

orbit. Hunter and Mayfield7 had observed this occasionally in some of their operative cases. However, for reasons that will be detailed elsewhere, a more careful search was made for nerve rootlets whose stimulation would produce orbital pain; in this way the fine filaments composing the dorsal root of C1 were stimulated and consistently found to produce such pain.

The first time these fibers were stimulated I was at a loss to account for them, since with others I believed that the first cervical dorsal root did not exist in man. But this root was found in each of the 8 succeeding cases. Most probably it is overlooked, for the rootlets, which usually number three, were sometimes little thicker than arachnoidal strands in the present group of patients. Stimulation, however, quickly removes any doubt as to their nature; if they are followed peripherally a small dorsal-root ganglion may be seen at the level of the dural root sleeve. Gray8 and Poirier and Charpy11 stated that this dorsal root is absent in only 8 per cent of cases; Larsell9 has stated that the C1 dorsal root is smaller than the ventral root, and in rare cases may be rudimentary or entirely absent. Fig. 1 shows the C1 root from one of the cases referred to in this report as photographed at operation.

When stimulated the uppermost rootlet of C1 evoked pain in the orbit; the mid rootlet, pain in the frontal area; and the lowest, pain in the vertex. These observations were repeated with identical response each time on the same patient and confirmed with minor variations in 2 subsequent patients; in a fourth patient orbital and suboccipital pain was elicited by stimulation of C1 but I was unable to elicit frontal pain.

Since the C1 rootlet plays no role in the innervation of the orbital structures, it appeared that the mechanism of referral of pain could only be central. The most logical association would seem to be with the descending tract and nucleus of the trigeminal nerve. A study of this problem in cats, using the Nauta Gyaxie technic for degenerating axons,* and a neurophysiologic study using microelectrodes† (to be reported shortly) indicate that convergence of cervical and trigeminal afferents at this level can be shown in the experimental animal.

Thus there is a pain-sensitive structure located at the rim of the posterior fossa whose stimulation evokes orbitofrontal pain. It seems reasonable to conclude that if these rootlets are involved by displacement of the contents of the posterior fossa, such referred pain could result.

Cerebellar Tumors. Displacements in tumors of the cerebellum are too well known to require more than mention; the tumor cannot develop far before the cerebellar tonsil begins

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*Kerr, F. W. L. Structural relations of the spinal tract of the trigeminal nerve to the upper cervical roots and the solitary nucleus in the cat. *Exp. Neurol.* (In press.)

†Kerr, F. W. L., and Olafson, R. A. Trigeminal and cervical volleys: convergence on single units in the spinal gray at C-1 and C-2. *Arch. Neurol.*, Chicago. (In press.)
to slide through the foramen magnum. The tonsil normally is separated from the C1 root by a distance of approximately 5 mm., and in this early stage there may not be any increase in intracranial pressure. But the C1 rootlets lie across the tonsil’s path of displacement as it shifts downward and necessarily they will be stretched, either by direct engagement with the tonsil or indirectly by the wedge action of the tonsil between the dura mater and the cervical cord (Fig. 2). An additional possibility is that the medulla and the tissues about the cervicomedullary junction are displaced downward to some extent also, thus stretching the transversely running root. The root itself has little latitude for displacement, anchored as it is to the dural sheath peripherally and the cord centrally.

Since at operation the cerebellar tonsils may be found as far down as the C2 root level or lower in advanced cases, the preceding course of events does not appear unreasonable.

**Supratentorial Mass Lesions.** It seems unlikely that this mechanism can be implicated in frontal headache during the early stages of tumors above the tentorium. Although cerebellar tonsillar herniation does occur with tumors in the supratentorial region, with them it is a late event. In the advanced cases in which increased pressure is present, the associated tonsillar herniation perhaps could account for the occurrence of frontal headache of late onset by the mechanism above discussed. It seems more likely, however, that tentorial herniation of the hippocampal gyrus is responsible for referral of headache to the frontal area. Such herniation would involve the tentorial nerves in their course along the tentorial notch, pressure being transmitted directly to them.

**SUMMARY**

The frontal headache accompanying tumor of the posterior fossa may be a response to traction on the first cervical dorsal root by downward displacement of structures about the cervicomedullary junction.

Clinical observations and stimulation of pain-sensitive structures and also experimental work on the cat indicate that the proposed mechanism of spread of pain is compatible with the microscopic anatomy and neurophysiology of the cervicomedullary structures.

**REFERENCES**

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