RESECTION OF THE GREATER SUPERFICIAL PETROSAL NERVE IN THE TREATMENT OF UNILATERAL HEADACHE*

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H. G. WOLFF,24 whose opinions regarding headache certainly command respect, has stated that from the data available six basic mechanisms of headache from intracranial sources may be formulated:

Headache may result from (1) traction on the veins that pass to the venous sinuses from the surface of the brain and displacement of the great venous sinuses; (2) traction on the middle meningeal arteries; (3) traction on the large arteries at the base of the brain and their main branches; (4) distention and dilatation of intracranial arteries; (5) inflammation in or about any of the pain-sensitive structures of the head; and (6) direct pressure by tumors on the cranial and cervical nerves containing many pain afferent fibers from the head.

Of these six mechanisms, only one can conceivably be responsible for any significant proportion of cases of chronic recurring headache. That mechanism is distention and dilatation of intracranial arteries, which is the theme of this paper.

In the latter part of November 1940, 3 patients with a strikingly similar clinical picture were observed simultaneously. These patients were subject to attacks of excruciating unilateral head pain recurring once to several times a day, lasting from 30 minutes to a few hours, and frequently awakening them from sleep. The pain was deep and boring, limited to the periorbital and retro-orbital areas. It caused the patient to pace the floor, and nothing appeared to give relief. The attacks were accompanied by profuse lacrimation, nasal discharge, and nasal obstruction, all limited to the side of the headache. To the neurophysiologist these objective signs indicate excitation of the greater superficial petrosal nerve, which is the parasympathetic portion of the facial nerve. Therefore, it was presumed that the unilateral head pain accompanying these phenomena was also due to abnormal parasympathetic discharges coming over this same nerve. In other words, these patients were presumed to have a "neuralgia" of the greater superficial petrosal nerve.

In 1939 Horton, MacLean, and Craig12 described this syndrome and suggested the term "erythromelalgia of the head."

Horton11 in 1941 redescribed the condition and because of its response to histamine therapy suggested the name "histaminic cephalgia."

In searching for a physiologic explanation as to how an excitation of the

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greater superficial petrosal nerve could cause unilateral head pain, it was found that in 1932 Cobb and Finesinger\(^3\) collaborated with Chorobski and Penfield\(^1\) in a series of physiologic and anatomic studies on this nerve. From a review of the literature these authors showed that the greater superficial petrosal nerve carried secretory fibers for the lacrimal gland and secretory and vasodilator fibers for the mucous membrane of the nasal cavity. As a result of their own investigations they showed that this nerve carries somatic afferent fibers from the dura mater, internal carotid artery, and

![Diagram showing the course of the vasodilator nerve fibers for the cerebral blood vessels, according to Chorobski and Penfield.\(^1\)](image)

sphenopalatine ganglion to the ganglion cells in the geniculate ganglion. Of greatest significance in the present consideration, however, was their demonstration that the greater superficial petrosal nerve carries vasodilator fibers to the ipsilateral cerebral hemisphere (Fig. 1). In 1940 when Schumacher, Ray, and Wolff\(^7\) showed that cerebral vasodilatation causes headache, the chain of evidence was complete.

To recapitulate, in view of the above evidence it appears that periodic discharges of parasympathetic impulses over the greater superficial petrosal branch of the 7th nerve should cause unilateral lacrimation, unilateral swell-