The Clinical Journal of Pain
3:63–80 © 1987 Raven Press, Ltd., New York

Ad Alessandro
il "fenomeno"
Sicuteri

Quasi-Phantom Head Pain from Functional Deafferentation

Federigo Sicuteri

Institute of Internal Medicine and Clinical Pharmacology, University of Florence, Florence, Italy

Abstract: The nociceptive system, as in any other, is not exempt from damage and consequent dysfunction. This dysfunction can depend not only on organic but also on "nonorganic" disorders. The clinical expression of both of these conditions is roughly the same, that is recurrent and/or persistent pains that exhibit the stereotypical characteristics of central pain. If the nociceptive dysfunction is partial in entity but quasi-systemic in extension (as is the case in spontaneous nonorganic pain), the pain will be projected to peripheral regions mostly rich in afferent nerves. This conceptualization of central nonorganic pain represents an approach to mysterious painful diseases such as migraine and correlated idiopathic headache (IH). IH [migraine and cluster headache (CH) included], should be assumed, according to this theory, to be the most common clinical expression of the impairment of the nociceptive homeostasis. Pain is thought to arise from irritative foci generated along the spinal cord and brainstem. These irritative foci represent the consequence of a deafferentation which is spontaneous in nature, partial in entity, diffuse in extension, and fluctuating in time. The "empty neuron" (EN) is the pathological unit of this disorder. EN in the IH area is both nociceptive as well as vegetative. Many clinical, biochemical, and pharmacological phenomena of migraine attacks are, in quality and chronology, analogous to those that arise following an abrupt discontinuation of a chronic hyperstimulation of opiate receptors, as happens in heroin addiction. This is the most valid support for the hypothesis that both vegetative and nociceptive neurons involved in headache are opioid dependent. As the result of a chronically deficient opioid system (its main physiological function is modulating the secretion of other neurons), opioid-dependent neurons will become incontinent and subsequently empty for sensitive and autonomic transmitters. Only a condition of sensitive and autonomic (opioid-dependent) empty neurons can explain the majority of signs of functional deafferentation and autonomic denervation present in most serious headache sufferers. Functional deafferentation creates a constellation of clinical, pharmacological and electrical signs, strictly similar to that of organic deafferentation. The clinical discrepancies arise from their different nature as organic deafferentation is usually total, circumscribed, and irreversible. The common pathological characteristic is the creation of irritative foci in the spinal cord and brainstem, capable of automatically emitting nociceptive signals registered by the cognitive sites of the consciousness. These can be electrically registered as "epileptoid" discharges. In the present paper the biochemical and pharmacological evidences of this conceptualization of headache are illustrated. Discussion is centered around clinical phenomena which can be explained in light of the present perspective. **Key Words:** Deafferentation—Idiopathic headache—Cluster headache—Empty neuron—Quasi-epileptic foci—Cerebrospinal fluid—Calcium entry blockers.

Address correspondence and reprint requests to Dr. F. Sicuteri at Institute of Internal Medicine and Clinical Pharmacology, University of Florence, Viale Morgagni, 85-50134 Florence, Italy.

The mechanism of migraine and other idiopathic headache (IH) phenomenon can hardly be explained considering the conventional vascular theory: (a) hypoesthesias and dysesthesias in pain-