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SOMATOSENSORY EVOKED POTENTIALS AND HEADACHE: A FURTHER EXAMINATION OF THE CENTRAL THEORY

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Abstract—The central theory of headache was investigated by examining the amplitude of the somatosensory evoked potential (SSEP) in headache sufferers and headache-free controls. The P_1–N_1 amplitude was found to be greater, and to increase more rapidly with increasing stimulus intensity, for headache subjects than for controls. The N_1–P_2 amplitude was also found to be larger for headache subjects than for controls, but there was no significant difference between groups on the rate at which this component increased with stimulus intensity. When the P_1–N_1 and N_1–P_2 amplitudes were assessed in headache subjects, during and between attacks, no significant differences between conditions were observed. No significant differences between tension and migraine sufferers were observed on either component. It was concluded that the central nervous systems of headache sufferers may be more reactive to somatosensory input than those of headache-free persons and that this might be an important factor in the pathophysiology of headache.

Keywords: Headache, Somatosensory evoked potential, Pathophysiology.

INTRODUCTION

Headache has been conceptualized as a disorder of central pain control mechanisms [1–3]. Although this central theory has difficulty accounting for the localization of head pain [4], the assumption that headache sufferers may be characterized by deficient central pain regulatory systems is supported by the following findings: (1) Blood levels of the pain inhibitory substance seretonin have been observed to be lower in sufferers of daily tension headache than in headache-free controls [5, 6]. (2) CSF levels of the morphine-like substance enkephalin have been found to be lower during episodes of migraine than between attacks [7]. (3) CSF levels of a second endogenous opiate, beta-endorphin, have been observed to be lower in migraine sufferers between attacks than in headache-free controls [8–10].

Thus, it is conceivable that the pathophysiology of headache could be described by a deficiency in the central pain control system, with the locus of head pain being determined by some further segmental disturbance of pain processing in the trigeminal nerve [11] or peripheral process such as muscle contraction, vasodilatation and/or local chemical action [4].

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The hypothesis that central pain control processes may be involved in the pathophysiology of headache suggests that the nervous systems of headache sufferers may be more reactive to sensory input than those of headache-free controls. Clinically, there is some support for this view, with many migrainous patients reporting a hypersensitivity to light, noise and odours during headache attacks [4]. Furthermore, headache sufferers tested between attacks have been observed to be more sensitive to experimentally induced pain in the head and finger [12].

The amplitude of the averaged evoked potential (AEP) generated in response to sensory input represents a further avenue through which any heightened sensitivity to stimulation amongst headache sufferers might be explored. Very few studies have been carried out along these lines, and those that have been conducted have dealt almost exclusively with the response of migraineurs to visual stimulation [13–17]. These studies have yielded equivocal results. This is not surprising given the plethora of AEP recording and peak measurement techniques employed and the fact that none of these studies controlled for eye-blink antifacts, which are known to affect AEP records [18].

The most thorough examination of the visual evoked potential (VEP) in migraineurs and headache-free controls to date was conducted by Connolly *et al.* [19]. These researchers employed six different intensities of light and eliminated eye movement artefacts from recordings made at vertex and from bilateral temporal sites.

The VEP component amplitudes were identified by the method proposed by Connolly and Gruzelier [20]. The N_1 peak was defined as the most negative point in the latency range 80–180 msec after stimulus onset; P_1 was defined as the most positive peak within the first 60 msec before N_1 and P_2 as the next positive peak occurring between N_1 and 280 msec after stimulus onset.

Pooling their results across stimulus intensities, Connolly *et al.* [19] found that, for recordings made at vertex, the P_1–N_1 peak-to-peak amplitude and the N_1 peak amplitude (relative to a pre-stimulus baseline) were larger for migraineurs than for controls. Pooling the results across the temporal sites, the N_1–P_2 peak-to-peak amplitude was larger for migraineurs than for controls.

Unfortunately, Connolly *et al.* [19] did not present a between groups analysis for linear or quadratic trend across intensities. Such a procedure would have made for some evaluation of any differential rates of amplitude increase with stimulus intensity. This point is particularly important given that greater rates of amplitude increase with stimulus intensity (augmenting) have been associated with heightened sensitivity to experimental pain [21–24] and with lower concentrations of CSF endorphins [25].

Since Connolly *et al.* [19] employed photic stimulation and focused specifically on migraineurs, many of whom are known to be particularly sensitive to light when they have a headache [4, 26], their results may be interpreted in terms of some specific hypersensitivity in the visual system of migraineurs, rather than in terms of any general failure of sensory modulation.

The present study sought to extend the results obtained by Connolly *et al.* [19] by studying the amplitude of the somatosensory evoked potential (SSEP) in migraine, tension headache and control subjects, and by examining the hypothesis that the P_1–N_1 and N_1–P_2 amplitudes would show greater augmentation (that is, increase more rapidly with increments in stimulus intensity) for headache subjects than for controls.