Increased visual after-effects in migraine following pattern adaptation extend to simultaneous tilt illusion

A. J. SHEPHERD 1,*, J. E. PALMER 2 and G. DAVIS 1

1 School of Psychology, Birkbeck College, University of London, Malet Street, London WC1E 7HX, UK
2 Department of Psychology, Lancaster University, Lancaster LA1 4YF, UK

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Abstract—Much previous research into visual processing in migraine supports a model of abnormal cortical processing, in between the headache attacks, that is characterised by hyperexcitability, heightened responsiveness, a lack of habituation and/or a lack of intra-cortical inhibition. Shepherd (2001) reported two adaptation studies that challenged this view, one using the tilt after-effect, the second using the motion after-effect. Models of cortical function in migraine based on hyperexcitability and a lack of inhibition lead to specific predictions in an adaptation study: there should have been smaller after-effects in people with migraine than in people without. Both experiments, however, revealed larger after-effects in the migraine group than in the control group. Here, these results are extended to the simultaneous tilt illusion and an identical pattern of results was obtained: there were consistently larger effects in the migraine group than in the control group. The results from the three experiments are not consistent with a lack of inhibition in migraine. The results are discussed in terms of alternative models of cortical function, including a lack of excitation and reduced central energy reserves.

Keywords: Migraine; visual adaptation; cortical hyperexcitability; inhibition; excitation.

INTRODUCTION

Migraine is a severe, recurrent headache that may be associated with visual, somatosensory or other transient neurological symptoms (migraine with aura). Despite a prevalence of approximately one in ten in the population and extensive study over the last twenty years, the aetiology and pathophysiology of this disorder remain poorly understood. Converging evidence from disparate fields supports a model of abnormal cortical processing in migraine, in between the headache attacks, that is characterised by hyperexcitability and/or a lack of intra-cortical inhibition.

*To whom correspondence should be addressed. E-mail: a.shepherd@psychology.bbk.ac.uk
For example, impaired temporal or spatial contrast sensitivity, reduced masking of central targets by striped and flickering backgrounds, and heightened pattern sensitivity in migraine — the visual illusions and the discomfort experienced when viewing certain striped or flickering patterns — have each been attributed to an underlying cortical hyperexcitability (Wilkins et al., 1984; Khalil and Legg, 1989; Marcus and Soso, 1989; Hardebo, 1991; Khalil, 1991; Wray et al., 1995; Palmer and Chronicle, 1998; McKendrick et al., 2000; Shepherd, 2000; Wilkinson and Crotogino, 2000). There is further support for some cortical abnormality from physiological, electrophysiological and biochemical studies (e.g. Passchier and Orlebeke, 1983; Kropp and Gerber, 1993; Zagami, 1994; Welch and Ramadan, 1995; Schoenen, 1996; Wang et al., 1996). Not all studies agree, however, in the details of any abnormality. Moreover, some report differences in all migraineurs (e.g. Schoenen, 1996; Wang et al., 1996), others only in migraineurs with aura (e.g. Chronicle et al., 1995) and others only in migraineurs without aura (e.g. Schoenen, 1992), fuelling the debate as to whether migraine with and without aura are one or two distinct conditions.

A person suffering from headache must have experienced at least five attacks that fulfil the International Headache Society’s (1988) criteria to have headaches compatible with a diagnosis of migraine without aura. The headache must last longer than four hours and the pain must have two of the following four characteristics: a throbbing or pulsating quality, located on one side of the head (although not necessarily always the same side), of sufficient severity to interfere with or prohibit activity, and that is exacerbated by routine physical activity such as climbing stairs. The person must, in addition, experience nausea and/or photo- and phonophobia. Finally, there should be no other causes for the headache, such as alcohol or analgesic abuse.

A person who experiences migraine with aura often shares a similar headache to one without aura, but in addition has focal neurological symptoms before or during the headache. These may include visual disturbances (ranging from simple phosphenes to more elaborate fortification spectra in which a bright white line is seen to shimmer and zigzag through one or both hemifields, leaving blindness behind), speech difficulties, impaired co-ordination, pins and needles or numbness. Aura symptoms are commonly visual, which is one reason why visual experiments are conducted for a condition characterised by headache. A second reason is that visual stimuli, such as flashing or flickering lights, bright sunshine, or high contrast striped patterns, are often reported to trigger an attack. Visual symptoms and visual triggers both indicate that the visual system is intimately involved in migraine.

The present research was motivated by a basic interest in exploring visual processing in migraine and by a practical interest in developing tests that may be useful in the clinic, either to aid diagnosis or to predict whether someone is likely to respond positively to certain medications. Previously, two experiments that explored pattern or contrast adaptation revealed larger effects in people with migraine than in people without (Shepherd, 2001). One compared the duration