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Epilepsy & Behavior 7 (2005) 419–429
Epilepsy & Behavior
www.elsevier.com/locate/yebeh

Investigation of social and emotion information processing in temporal lobe epilepsy with ictal fear

Hazel J. Reynders [a,*], Paul Broks [b], Jon M. Dickson [a], Claire E. Lee [a], Graham Turpin [c]

[a] *Neurosciences Directorate, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF, UK*
[b] *School of Psychology, University of Plymouth, Plymouth, UK*
[c] *Department of Clinical Psychology, University of Sheffield, Sheffield, UK*

Received 30 March 2005; revised 17 July 2005; accepted 19 July 2005
Available online 19 September 2005

Abstract

This study examined whether patients with temporal lobe epilepsy (TLE) and ictal fear (IF) show emotion recognition deficits similar to those associated with amygdala damage. Three groups of patients (13 with TLE and IF, 14 with TLE and nonfear auras (non-IF), and 10 with idiopathic generalized epilepsy (IGE)) completed tests of visual and face processing, face emotion recognition and social judgment, together with measures of psychological adjustment (Hospital Anxiety and Depression Scale; SCL-90-R) and Quality of Life (QOLIE-31). All three epilepsy groups had fear recognition deficits, with relatively greater impairments in the IF group. Fear recognition deficits were associated with impaired social judgment of trustworthiness, duration of epilepsy, and a measure of quality of life. Social cognition impairments previously associated with amygdala dysfunction are also a feature of the neuropsychology of TLE, and extend the hypothesis in that they may additionally play a role in IGE.

Keywords: Temporal lobe epilepsy; Ictal fear; Emotion recognition; Social cognition

1. Introduction

The most commonly reported emotional aura in temporal lobe epilepsy (TLE) is ictal fear (IF), characterized by a profound sense of fear or foreboding [1–3] and frequently, but not invariably, accompanied by viscerosomatic symptoms and experiential phenomena [4]. IF is defined as a sudden, brief, fearful affect immediately before or during a seizure that arises out of context without a precedent causal perception or cognition [5–7]. Mesial temporal structures, including the amygdala, have been associated with seizure-related experiential phenomena, especially the sensation of fear-related experiences [8]. Further research links atrophy of the amygdala to symptoms of IF in TLE [9].

It is well established that the amygdala forms a crucial part of the neural circuitry involved in the appraisal of danger and the emotion of fear [10], and the consequences of human amygdala damage have been found to include specific impairments in the recognition of facial expressions of fear [11–13]. Functional imaging techniques have been used to demonstrate a selective response to facial expressions of fear in the normal amygdala [14,15], even when the faces are masked with neutral expressions to eliminate conscious perception of the fear stimuli [16]. Further evidence that damage to amygdala structures may reduce the ability to make appropriate judgments of negative social stimuli was found in a study in which subjects were required to make judgments of the trustworthiness and approachability of unfamiliar individuals from facial photographs [17]. Adolphs and colleagues' [17] patients with bilateral damage to the amygdala judged the unfamiliar

* Corresponding author. Fax: +44 (0)114 2268946.
E-mail address: h.reynders@sheffield.ac.uk (H.J. Reynders).

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doi:10.1016/j.yebeh.2005.07.013

individuals to be more approachable and more trustworthy than did control subjects.

A number of researchers have demonstrated an association between socioaffective disorders, such as autism [18] and schizophrenia [19,20], and abnormalities in the judgment of facial expressions of emotion. Evidence of the interconnectedness between emotional perception and affective responses comes from a different direction in a study by Birbaumer et al. [21], who found abnormal amygdala activation in subjects suffering from social phobia when they were shown a series of faces with neutral expressions.

Links between temporolimbic seizures and emotional disturbance in epilepsy have been established by a number of studies [22], and IF has been shown to be associated with abnormal affective responses [23–25]. However, evidence for a direct association between neurobiological factors and psychological distress among TLE patients remains controversial, with conflicting findings [26]. Conceptualizing the multidimensional nature of epilepsy-related psychological distress within a biopsychosocial [27,28] framework provides a basis for exploring the mechanisms underlying the emergence of psychological disturbance. Several authors have suggested that neurobiological determinants may place some epilepsy sufferers at greater risk of significant psychological disturbance, as a result of the consequent stress arising from the epilepsy condition [29,30].

As part of the brain's emotional circuitry, and also a key site of pathology in temporal lobe epilepsy, medial temporal structures, including the amygdala, are implicated but, as yet, there is no systematic account of the mechanisms through which such structures may play a role in shaping psychosocial abnormalities associated with epilepsy. Houghton et al. [31] found impairments in the ability to recognize negative emotional expressions, particularly fear, to be linked to reduced amygdala volume in patients with TLE, and Meletti et al. [32] found impaired facial emotional recognition among patients with right-sided mesial TLE. A recent single case study [33] found abnormal emotion recognition of facial expressions in a patient with IF and mesial temporal sclerosis before but not after surgery. The objective of the present study was to explore the relationship between IF, a well established neurobiological symptom of fear associated with temperolimbic seizure semiology, and the social cognition difficulties, including recognition of fear in others, that have been shown to occur among patients with structural damage to the amygdala regions. Beyond MRI evidence of mesial temporal lesions, our study was based on clinical correlates of presumed amygdala involvement in the seizure profiles of patients with IF rather than a structural indicator. A further aim was to examine relationships among patients with epilepsy between deficits in tests of social and emotion cognition and measures of psychological well-being and quality of life.

2. Patients and methods

Thirty-seven adults were recruited via the neurology services at the Royal Hallamshire Hospital in Sheffield into three groups: 13 people with temporal lobe seizures that began with a characteristic fear aura; 14 people with TLE and auras that were not fear related; and 10 patients with idiopathic generalized seizures without focal origin. Diagnosis of epilepsy was confirmed by electroencephalography and neurological investigation and, for the TLE patients, with magnetic resonance imaging (MRI). Twenty-five patients had hippocampal sclerosis. Two patients had benign mesial temporal tumors. Thirteen patients reported a psychic aura of fear that was out of context with their current affective state and was a characteristic feature of their seizures. Consistent with the literature [5–7], IF was defined as a sudden, intense feeling of fear reported as consistently occurring in the seconds before a characteristic seizure, out of context in intensity and without causal cognition. Examples of descriptions include: "funny thoughts and a feeling of panic"; "sickly, light headed feeling, stomach turns, a sense of fear but knows it is 'seizure fear' and not real fear"; "stomach churning fear." Fourteen patients reported aura phenomena without IF as defined above. Examples of descriptions include: "feeling in stomach, feeling as if holding breath"; "one side of brain asleep"; "slight nausea and queasiness"; "go dizzy for a few seconds." Patients with TLE who did not report an aura experience were excluded. General exclusion criteria included significant history of psychiatric illness, learning disability, and neurological disorder unrelated to epilepsy. The groups were matched for age, sex, and IQ. Data from 12 controls without epilepsy were provided from a previous study [34] carried out using identical tests of cognition and tests of emotion recognition. The nonepilepsy group fulfilled the same general exclusion criteria as the epilepsy groups.

2.1. Procedure

Following approval by the Sheffield Research Ethics Committee and consent to the study, participants were asked to give details of their seizures and auras. Categorization into the relevant patient group was carried out before assessment began. The initial stage of categorization was carried out via a short questionnaire. To reduce the inclusion of patients experiencing possible anticipatory anxiety, carefully conducted interviews were carried out following self-reports. Patients then underwent a range of background cognitive and perceptual measures, followed by a facial emotion recognition task and a