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# Emotion recognition from facial expressions in a temporal lobe epileptic patient with ictal fear

Makiko Yamada[a,*], Toshiya Murai[b], Wataru Sato[a], Chihiro Namiki[b], Toru Miyamoto[c], Yoshitaka Ohigashi[a]

[a] Laboratory of Cognitive Science, Department of Cognitive and Behavioral Sciences, Graduate School of Human and Environmental Studies, Kyoto University, Yoshida Nihonmatsu-chou, Sakyo-ku, Kyoto 606-8501, Japan

[b] Department of Neuropsychiatry, Graduate School of Medicine, Kyoto University, Kyoto, Japan

[c] Department of Neurosurgery, Graduate School of Medicine, Kyoto University, Kyoto, Japan

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**Abstract**

Ictal fear (IF) is an affective aura observed in patients with temporal lobe epilepsy. It has been suggested that the amygdala, a region implicated in emotion processing, is involved in generating IF. Several studies have reported that the patients with IF have disturbances in emotional experience, but there has been no testing of the emotional recognition in those patients. In this report, emotion recognition from facial expressions was investigated in a patient with IF. The patient suffered from IF due to temporal lobe epilepsy, and underwent hippocampectomy surgery which completely suppressed IF. We examined the patient before and after surgery. Before surgery, the patient tended to attach enhanced fear, sadness, and anger to various facial expressions. After surgery, such biases disappeared. As an underlying mechanism of the pre-surgical skewed perception of emotional stimuli, the abnormal epileptogenic circuits involving a hypersensitive amygdala possibly due to the kindling mechanism were suggested.



*Keywords:* Ictal fear; Emotion recognition; Facial expression; Temporal lobe epilepsy; Amygdala; Kindling

## 1. Introduction

Ictal fear (IF) is a well-recognized affective aura in patients with temporal lobe epilepsy (TLE) (Gloor, Oliver, Quesney, Andermann, & Horowitz, 1982; Gupta, Jeavons, Hughes, & Covanis, 1983; Taylor & Lochery, 1987). Ictal fear is defined as “a sudden, often short, fearful affect at the beginning of, or during, an epileptic seizure, without context or any relation to a precedent causal perception or cognition (Feichtinger et al., 2001).” In most cases of TLE with IF, the epileptic discharges originated from the amygdala, the hippocampus, or its adjacent structures (Gloor, 1972; Palmini & Gloor, 1992). The involvement of the amygdala in generating IF is also supported by electrical stimulation studies (Gloor, 1972, 1992, 1997; Gloor et al., 1982) and volumetric and quantitative analyses (Cendes et al., 1994; Feichtinger et al., 2001; Van-Paesschen, King, Duncan, & Connelly, 2001).

Recent neuropsychological and neuroimaging studies have demonstrated the crucial role of the amygdala in emotion recognition, particularly in the case of negative emotions, such as fear, sadness, and anger (Adolphs, Tranel, Damasio, & Damasio 1994, 1995; Adolphs et al., 1999; Anderson, Spencer, Fulbright, & Phelps, 2000; Blair, Morris, Frith, Perrett, & Dolan, 1999; Calder, Young, Rowland, & Perrett, 1996; Young, Hellawell, Van De Wal, & Johnson, 1996). As indicated by patients with amygdala damage, failure to evaluate fear and danger would induce lowered sensitivity to situations that evoke fear in normal subjects, resulting in the reduced real-life experience of fear (Sprengelmeyer et al., 1999), in spite of a preserved ability to generate

* Corresponding author. Tel.: +81 75 753 2551; fax: +81 75 703 4545.
*E-mail address:* myamada@jinkan02.mbox.media.kyoto-u.ac.jp (M. Yamada).

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fearful expressions (Anderson & Phelps, 2000). For example, Sprengelmeyer et al. (1999) reported that a bilaterally amygdala-damaged patient showed an impaired recognition of others' fearful facial expressions and reduced levels of fear experience in everyday situations.

Previous clinical interviews reported that up to half of TLE patients have serious emotional experience disturbances (Blumer, Montouris, & Hermann, 1995), such as interictal experience of fear, anxiety, or depression (Adamec, 1990; Bear & Fedio, 1977; Dodrill & Batzel, 1986; Gloor, 1990; Hermann & Chhabria, 1980; Hermann, Dikman, Schwartz, & Karnes, 1982; Perini & Mendius, 1984; Schmitz, Robertson, & Trimble, 1999; Weil, 1956, 1959; Williams, 1956). Using a self-report questionnaire, Bear and Fedio (1977) suggested that behavioral traits of TLE patients were the result of an attachment of enhanced affective tone to certain types of information. Some studies further suggested that these interictal emotional disturbances apply more to those with IF. Hermann and Chhabria (1980) reported increased interictal fear-related behavior in two TLE patients with IF. In addition, Hermann et al. (1982) compared TLE patients with and without IF, and revealed that those with IF had more deviation in Minnesota Multiphasic Personality Inventory profiles.

From what has been reviewed above, we can make the following summary: (1) the amygdala is involved in IF; (2) the amygdala plays a crucial role in processing specific facial expressions, particularly negative ones; (3) epileptogenic abnormalities in medial temporal structures are associated with altered affective tone beyond the ictal event. Therefore, from points (1) and (2), one would hypothesize that patients with IF, who are suspected to possess epileptogenic abnormalities in the neural substrates that mediate emotion recognition (i.e., amygdala), may have certain types of bias in emotion recognition interictally. Moreover, the same hypothesis would also follow from point (3); one may speculate that biased emotion recognition would underlie the disturbed emotional experience in the subgroup of TLE patients including those with IF, though it might not be specific to patients with IF.

As mentioned above, lesion studies provided evidence of impaired emotion recognition following bilateral amygdala damage. However, the selectivity of deficits remains unclear, such as whether the amygdala damage leads to a disproportionate impairment in recognizing fear (Anderson & Phelps, 2000; Broks et al., 1998; Calder et al., 1996; Sprengelmeyer et al., 1999), or a broader impairment in recognizing multiple negative emotions (Schmolck & Squire, 2001). By studying patients with IF interictally and comparing their performance after surgery, we may further clarify the selectivity of deficits in which the amygdala is involved, and what the underlying mechanisms may be.

In the present study, we examined an intractable TLE patient with IF before selective resection of the epileptic focus and after surgery, when seizures including IF, were suppressed. We adopted the method of facial expression intensity ratings developed by Adolphs et al. (1994). Our results revealed that the patient had skewed appraisal of facial expressions before surgery, and that this tendency disappeared after surgery when IF was completely suppressed.

## 2. Case report

A 25-year-old right-handed woman suffered from intractable TLE. Her simple partial seizures, which began at the age of 9, were manifested with initial feeling of intense fear, which sometimes progressed to complex partial seizures with motionless staring and oral automatism once or twice a month. Her seizures recurred at least once a week, sometimes 10 times a day, under maximally tolerable antiepileptic drugs; therefore, she was admitted for surgical treatment. She received a high school education and had no other history of neurologic or psychiatric illness.

At the pre-operative neurological examination, fluid-attenuated inversion-recovery (FLAIR) magnetic resonance imaging (MRI) showed left hippocampal atrophy, together with an increased signal intensity area in the hippocampus (Fig. 1a). F-18 fluorodeoxyglucose positron emission tomography (FDG-PET) revealed a left temporal hypometabolism of glucose (Fig. 1b). The patient underwent continuous scalp video-electroencephalography (EEG) monitoring. Interictal EEG revealed epileptiform discharges from the left frontotemporal area. Ictal EEG was clearly lateralized to the left frontotemporal area, showing a spread of ictal discharges from the left frontotemporal to the bilateral temporal areas.

Neuropsychological evaluations were performed presurgically (Table 1). An intelligence test (Wechsler Adults Intelligence Scale-Revised; WAIS-R) revealed her to be below average both for verbal and performance IQ. The Wechsler Memory Scale-Revised (WMS-R) revealed her verbal memory was more severely impaired than visual memory. An intracarotid amytal test was performed in order to determine language and memory dominances. Language dominance

Table 1
Shown are the patient's neuropsychological profiles 3 weeks before surgery and 2 weeks after surgery

| Neuropsychological tests | Pre-operation | Post-operation |
|---|---|---|
| MMSE | 30 | 30 |
| RCPM | 21 | 35 |
| WAIS-R | | Not performed |
| Verbal IQ | 82 | – |
| Performance IQ | 84 | – |
| Full IQ | 81 | – |
| WMS-R | | |
| Verbal | 78 | 102 |
| Visual | 111 | 116 |
| General | 85 | 107 |
| Attention | 87 | 97 |
| Delayed recall | 84 | 104 |

MMSE = mini-mental state examination; RCPM = Raven's colored progressive matrix; WAIS-R = Wechsler adult intelligence scale-revised; WMS-R = Wechsler memory scale-revised.