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

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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.

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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

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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, fluidattenuated 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

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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.



Fig. 1. (a) Pre-operative coronal FLAIR-MRI shows atrophy of the left hippocampus with an increased signal intensity, indicated by an arrow. (b) Pre-operative axial FDG-PET shows an extensive hypometabolism in the left temporal area.

was revealed to be on the left side, and bilateral involvement was observed in memory function.

In order to perform a focus resection, a subdural electrode plate was implanted, and the anterior medial part of the left parahippocampal gyrus was determined as the specific epileptic focus. The patient received a selective left anterior hippocampectomy, including the parahippocampal gyrus, restricted to the epileptogenic tissue. The pathology revealed hippocampal sclerosis. A 5 months post-operative MRI is shown in Fig. 2. She has been seizure-free since surgery, and no deficits in language or memory functions have been observed after surgery (Table 1).

Subjective state of mood was assessed through the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, &



Fig. 2. Post-operative T1-weighted MRI shows a selective resection of the left anterior hippocampal area, indicated by an arrow.

Lushene, 1970) and the self-rating depression scale (SDS; Zung, 1965). The patient showed very high anxiety scores before (trait = 54, level 4; state = 54, level 5) and after surgery (trait = 51, level 4; state = 55, level 5). On the SDS, the patient scored 48 (neurosis range) before surgery and 41 (normal range) after surgery, showing a slightly reduced level of depression after surgery. Although these self-rating scales do not demonstrate a dramatic change, in clinical settings, the patient's mood and behavior clearly changed after surgery. Before surgery the patient looked depressed and timid to clinicians, whereas she looked cheerful and friendly after surgery, though she was cooperative with the examiners on both occasions. When the patient was asked to compare her mood state before and after surgery, she reported that she experienced anxiety everyday but she did not feel anxious so much after surgery.

The facial recognition tests described below were administered interictally (the last seizure occurred 2 days before the test day and she did not have any seizures on that day) 4 weeks before surgery, and 2 weeks after surgery.

3. Methods

3.1. Comparison subjects

Twelve right-handed healthy volunteers (four males; eight females) with no history of neurologic or psychiatric illness participated as the comparison group. The mean age was 25.8 years (S.D. = 3.24). All subjects gave informed consent to participate in the study.

3.2. Facial recognition task

To control for subject's basic visuoperceptual ability with facial stimuli, a short version of the Benton Facial Recognition Test (Benton, Hamsher, Varney, & Spreen, 1983) was administered. Subjects matched the faces of identical individuals among six choices, which are shown with varying views and light conditions.

3.3. Facial expression recognition task

The experimental procedure was identical to that of Adolphs et al. (1994). Six faces each of happiness, sadness, fear, anger, disgust, surprise, and three neutral faces, in total 39 face stimuli, were selected from the Picture of Facial Affect series (Ekman & Friesen, 1976). In one block, the set of 39 stimuli were presented in random order with no time limit. This was repeated six times in separate blocks. For each block, one of the six emotion terms was presented, and the patient or comparison subjects had to evaluate the facial expression with regard to the intensity of a given emotion content on scale of 0 (not at all) to 5 (very much). After rating all 39 stimuli regarding one emotion from the six, subjects were given other emotion terms to rate in a subsequent block. Thus, for each facial stimulus, subjects rated the intensity of all six basic emotions. Further details of the task have been described elsewhere (Adolphs et al., 1994).

3.4. Statistical analysis

We performed the following two analyses separately. At first, we analyzed intensity ratings of faces matching the rating category (e.g. fear ratings to fear faces). Secondly, we compared intensity ratings of faces not matching it (e.g. fear ratings to angry faces) between operations.

3.4.1. Intensity ratings of faces matching the rating category

In order to compare recognition of the six basic emotions (i.e., emotion intensities with the corresponding facial expression) between the patient and comparisons, the patient's data were converted to z scores on the basis of the comparison means and standard deviations.

3.4.2. Intensity ratings of faces not matching the rating category

The patient's ratings of six emotion intensities across various facial expressions were compared before and after surgery, performing a 2 × 6 mixed-design ANOVA on the mean ratings separately for each of six blocks with an operation condition (pre- or post-operation) as a within-subject factor and type of facial expressions (happiness, sadness, fear, anger, surprise, or disgust) as a between-subject factor. If there was a significant interaction between the operation condition and type of facial stimuli, simple effect analyses were performed to assess the significance of the emotion intensity change over operation for each facial expression. Data analyses were performed with commercially available statistical software (SPSS version 11.0, SPSS, Chicago, IL). The significance level for all comparisons was P < 0.05.

Table 2

The patient's and comparisons' mean ratings of the degree to which facial
expressions of each emotion type looked like its corresponding emotion term
on a scale of 0 (not at all) to 5 (very much)

	Pre-operation		Post-operation		Comparisons
	Score	z	Score	z	$(\text{mean} \pm \text{S.D.})$
Happiness	4.50	-1.01	5.00	0.34	4.88 ± 0.37
Sadness	4.33	-0.07	4.67	0.33	4.39 ± 0.85
Fear	3.83	-0.51	3.33	-0.98	4.38 ± 1.07
Anger	4.17	-0.14	4.00	-0.29	4.32 ± 1.1
Surprise	4.17	-0.33	4.17	-0.33	4.49 ± 0.98
Disgust	2.67	-1.37	1.50^{*}	-2.39	4.24 ± 1.14

The patient is largely able to judge emotional meaning from faces with the exception of the expression of disgust after surgery.

* P < 0.01.

4. Results

4.1. Facial recognition task

All the comparison participants showed normal ability to process facial stimuli on BFRT (mean = 49 ± 1.8). The patient also had no difficulties in processing these stimuli before and after surgery, scoring 49 and 50 respectively.

4.2. Intensity ratings of faces matching the rating category

Regarding the mean intensity ratings of faces matching the rating category, i.e. mean ratings of, for instance, happiness intensity over six different happy faces, the patient's ratings were not different from comparison means before surgery (Table 2). After surgery, no impairments were found except for disgust, which yielded a mean rating of 1.5 out of 5 (z = -2.39, P < 0.01). Specifically, her ratings of fear, sadness, and anger were not different from comparisons before or after surgery.

4.3. Intensity ratings of faces not matching the rating category

Compared with the emotion intensity ratings before and after surgery for the patient, significant interactions between operation conditions (pre or post) and the emotional categories of facial stimuli were obtained only for sadness intensity ratings [F(5, 25) = 7.38; P = 0.000], fear intensity ratings [F(5, 25) = 7.84; P = 0.000], and anger intensity ratings [F(5, 25) = 7.84; P = 0.000]25 = 3.81; P = 0.009]. Simple effect analyses showed significant differences between before and after the operation as follows. For sadness intensity ratings (Fig. 3a), the patient perceived significantly higher sadness intensities in facial expressions of fear [F(1, 5) = 7.50; P < 0.05], anger [F(1, 5)]= 49.00; P < 0.001, surprise [F(1, 5) = 62.50; P < 0.001], and disgust [F(1, 5) = 8.45; P < 0.05] before surgery (mean: 3.00, 2.50, 1.67, 2.17, respectively) than after surgery (mean: 1.00, 1.67, 0.00, 1.00). For fear intensity ratings (Fig. 3b), the patient perceived significantly higher fear intensities toward



Fig. 3. Ratings of (a) sadness intensity, (b) fear intensity, and (c) anger intensity across all facial expressions. Data for comparisons are represented by open squares (\Box) with S.D. The patient's ratings are represented by filled squares (\blacksquare ; pre-operation) and filled triangles (\blacktriangle ; post-operation).

sad [F(1, 5) = 22.50; P < 0.01], angry [F(1, 5) = 121.00; P < 0.001], surprised [F(1, 5) = 49.00; P < 0.001], and disgusted [F(1, 5) = 7.50; P < 0.05] expressions before surgery (mean 3.67, 2.00, 1.33, 1.50) than after surgery (mean 0.67, 0.17, 0.00, 0.50). For anger intensity ratings (Fig. 3c), the patient perceived higher anger intensities toward fearful [F(1, 5) = 7.50; P < 0.05], surprised [F(1, 5) = 49.00; P < 0.001], and disgusted [F(1, 5) = 7.50; P < 0.05] expressions before surgery (mean: 1.17, 1.17, 3.33) than after surgery (mean: 0.17, 0.00, 2.33).

Moreover, by comparing the patient's pre- and postoperative intensity ratings with comparison group means, it is revealed that the observed shift of intensity ratings after surgery was attributable not to her abnormally low postsurgical ratings but to abnormally high pre-surgical ratings. The following pre-operative ratings were significantly higher than that of the comparison group: sadness intensity ratings for angry (z = 1.82, P < 0.05), surprised (z = 3.50, P < 0.001), disgusted (z = 1.78, P < 0.05) expressions; fear intensity ratings for sad (z = 3.14, P < 0.001), angry (z = 1.92, P < 0.05), disgusted (z = 2.13, P < 0.05) expressions; anger ratings for surprised expression (z = 3.90, P < 0.001). None of postoperative ratings were significantly different from comparisons.

In sum, significant pre-surgical deviations were observed in sadness, fear, and to a lesser extent, anger ratings of various facial expressions except for happiness.

5. Discussion

The present investigation demonstrated interesting findings regarding emotion processing related to IF, particularly recognition of fear from facial expressions. Previous lesion studies have suggested the crucial role of the amygdala in recognition of facial expressions. In cases of bilateral amygdala damage, deficits have been found to particularly affect recognition of fearful facial expressions, and to a lesser extent, sadness and anger (reviewed in Fine & Blair, 2000). Deficits with unilateral damage to the left or the right amygdala are more subtle; patients with right unilateral amygdala damage, as a group, were reported to show impairments in processing fearful facial expressions, but not patients with similar damage to the left hemisphere (Adolphs, Tranel, & Damasio, 2001; Anderson et al., 2000). Although there have now been investigations of emotional processing in TLE patients with unilateral temporal lobectomy, few examined it before surgery. One study examined emotion recognition in non-surgical epileptic patients with evidence of mesial temporal sclerosis (Meletti et al., 2003). These authors found that, whereas early-onset right medial temporal lobe epilepsy may contribute to a severe impairment in recognizing fearful facial expressions, neither left medial temporal lobe epilepsy nor an epilepsy focus other than the temporal lobe did.

In line with these above findings, without damage to the right amygdala, our patient was observed to possess normal abilities to recognize sad, fearful, and angry facial expressions before and after surgery, i.e. the patient's intensity rating of, for instance, fear in the facial stimuli of fear were within the normal range. However, using the more sensitive analysis applied in our study, the patient was revealed to have a skewed pattern of these negative emotion intensity ratings in judgments of other facial expressions, i.e., the patient judged, for instance, not only "sad" faces as highly sad but also "fear" faces as relatively sad. All of these deviations were directed toward the abnormally "higher" ratings, and all normalized after surgery.

A possible interpretation of these findings is as follows: in epileptic patients with IF, the volumes of the amygdala, which correlated with the hippocampal sclerosis, were very small (Cendes et al., 1994; Feichtinger et al., 2001; Van-Paesschen et al., 2001), suggesting hyperexcitable amygdala in the epileptogenic circuit for fear aura (Adamec, 1997; Racine, 1978; Rosen & Schulkin, 1998). If we presume that the same mechanism applied to in our patient before surgery, we postulate that there is a hyperexcitable left medial temporal circuit, including the amygdala. This hypersensitive amygdala may have caused the misinterpretation of the incoming emotional facial stimuli as signaling fear, sadness, or anger. This theory of hypersensitivity of the left medial temporal region is also supported by the interictal EEG findings showing epileptiform discharge in the left frontotemporal area. With the absence of seizures after resection of epileptic focus, this abnormal hypersensitivity may have normalized, which resulted in normal intensity ratings in emotional facial expressions.

The patient's abnormal intensity ratings were observed only in certain categories of emotion ratings (i.e., fear, sadness, anger) of various facial stimuli except for happiness. When the performances before and after surgery were compared, the differences were: higher "fear" ratings for sad, angry, surprised, or disgusted faces, higher "sadness" ratings for fearful, angry, surprised, or disgusted faces, and higher "anger" ratings for fearful, surprised, or disgusted faces before surgery. The categorical interrelationship of these findings is rather complicated. However, the pre-surgical pattern of the patient's emotional judgment, i.e., the most prominent deviations in fear and sadness ratings and a smaller deviation in anger ratings, suggests that the amygdala is involved in the recognition of multiple negative emotions, and this result is generally in accordance with the recent lesion studies that following damage to the amygdala, impaired recognition of emotions, particularly fear, and to a lesser extent sadness and anger, were reported (Fine & Blair, 2000). IF itself is the manifestation of fearful affect; however, the skewed appraisal of facial expressions was observed in fear, sadness and anger ratings. This implies that the deficit in this patient is related to the interictal functional abnormalities of the amygdala rather than the mood state of IF influencing the interictal affective evaluation.

Neuroimaging studies have demonstrated enhanced activations in the left amygdala following the presentation of fearful faces (Breiter et al., 1996; Morris et al., 1996, 1998; Phillips et al., 1997; Thomas et al., 2001; Whalen et al., 1998) and sad faces (Blair et al., 1999). These findings are again, in line with our data, and the above mentioned our "hypersensitive amygdala" hypothesis. Concerning the laterality, our result is also compatible with the left lateralized activation of these neuroimaging studies, although it should also be noted that the relative importance of the right amygdala in emotion recognition was suggested by some lesion studies (Adolphs et al., 2001; Anderson et al., 2000). It may be presumed that the emotional aspects of facial stimuli, which were preferentially processed in the amygdala, were processed by a hypersensitive left amygdala in our patient before surgery, and as a result, the patient judged various face stimuli as highly fearful, sad, or angry ones.

Finally, as an underlying biological mechanism to account for our hypothesis of a "hypersensitive amygdala", the role of kindling in limbic system was considered. Goddard (1967) and Goddard, McIntyre, and Leech (1969) reported that the application of repeated electrical stimulations to all limbic and some cortical sites in the rat brain would lead to a change in the stimulus response to the point that the stimulus produces a convulsion. In particular, kindling of the amygdala and the hippocampus in rats and cats results in the development and gradual intensification of changes in emotional behavior and EEG preceding the convulsion, producing high levels of emotionality that are fearful and defensive in nature (Adamec, 1990, 1997; Adamec & Young, 2000; Kalynchuk, 2000). This animal model of kindling fits well with human epilepsy to explain serious emotional disturbances in TLE patients, such as fear, anxiety, and depression. In our case, the effects of the kindling mechanism in the limbic system could have caused the amygdala to have a lowered threshold for the judgment of emotional stimuli, which may have resulted in attaching enhanced affective significance to emotional facial expressions.

In contrast with the above-discussed amygdala hypothesis, the effect observed in this case may be explained by an alternative interpretation. Normalization of the emotional bias with the resolution of IF was observed after the removal of the left anterior hippocampus but not the amygdala, therefore it would be more straightforward to interpret this effect as the result of a dysfunctional hippocampus. However, there are some arguments against this interpretation. At first, in addition to the amygdala, epileptic discharge of IF is also reported to originate from the hippocampus. However, only the anterior portion of the hippocampus was involved in such cases (Cook, Fish, Shorvon, Straughan, & Stevens, 1992; Feichtinger et al., 2001; Kuks, Cook, Fish, Stevens, & Shorvon, 1993; Van-Paesschen, Connelly, King, Jackson, & Duncan, 1997). Thus, the cause of the fearful affect in TLE was suspected to be the seizure discharge arising elsewhere spreading to involve the amygdala (Cendes et al., 1994; Fish, Gloor, Quesney, & Oliver, 1993; Gloor, 1972, 1992; Halgren, Walter, Cherlow, & Crandall, 1978). Secondly, stimulation of the hippocampus or the parahippocampal gyrus produced fear less often than that of the amygdala (Gloor, 1972, 1992; Gloor et al., 1982; Halgren et al., 1978). And finally, regarding the recognition of facial expressions, there is no doubt that the amygdala plays a crucial role in processing facial expressions, but the hippocampus was reported not to be involved in it in neuropsychological (Broks et al., 1998) and neuroimaging studies (Williams, Phillips, Brammer, Skerrett, Lagopoulos, & Rennie, 2001). Taking into account the above evidence, it is unlikely that the observed shift of emotional recognition in our patient after surgery can be explained by the structural abnormalities of the hippocampus alone.

The major limitation of the present study is that no comparison was made between TLE patients with and without IF, which makes it difficult to confirm the above-discussed mechanisms of the defect with certainty. To date, there is only one study that has examined recognition of facial expressions in non-surgical TLE patients (Meletti et al., 2003), and two studies that have examined post-operative TLE patients (Adolphs et al., 2001; Anderson et al., 2000). However, none of them examined the relationship of the performance of emotional facial recognition with types of seizures or affective symptoms. For future direction, TLE patients with and without emotional disturbances should be compared to evaluate the hypothesis discussed above.

To our knowledge, this is the first study investigating recognition of facial expressions in a patient with IF before and after surgery. Before surgery, our IF patient tended to perceive fear, sadness, or anger in various emotional expressions. Such biases reduced to the normal level after the operation, together with the resolution of IF. The possible role of abnormal epileptogenic circuits with a hypersensitive amygdala, and the role of kindling in the limbic system were suggested to underlie these emotional cognitive processes in this patient.

References

- Adamec, R. E. (1990). Dose kindling model anything clinically relevant? *Biological Psychiatry*, 27, 249–279.
- Adamec, R. (1997). Transmitter systems involved in neural plasticity underlying increased anxiety and defense — implications for understanding anxiety following traumatic stress. *Neuroscience and Biobehavioral Reviews*, 21, 755–765.
- Adamec, R., & Young, B. (2000). Neuroplasticity in specific limbic system circuits may mediate specific kindling induced changes in animal affect — implications for understanding anxiety associated with epilepsy. *Neuroscience and Biobehavioral Reviews*, 24, 705–723.
- Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature*, 372, 669–672.
- Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1995). Fear and the human amygdala. *The Journal of Neuroscience*, 15, 5879–5891.
- Adolphs, R., Tranel, D., Hamann, S. B., Young, A. W., Calder, A. J., Anderson, A., et al. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, 37, 1111–1117.
- Adolphs, R., Tranel, D., & Damasio, H. (2001). Emotion recognition from faces and prosody following temporal lobectomy. *Neuropsychology*, 15, 396–404.
- Anderson, A. K., & Phelps, E. A. (2000). Expression without recognition: contributions of the human amygdala to emotional communication. *Psychological Science*, 11, 106–111.
- Anderson, A. K., Spencer, D. D., Fulbright, R. K., & Phelps, E. A. (2000). Contribution of the anteromedial temporal lobes to the evaluation of facial emotion. *Neuropsychology*, 14, 526–536.
- Bear, D. M., & Fedio, P. (1977). Quantitative analysis of interictal behavior in temporal lobe epilepsy. Archives of Neurology, 3, 454–467.
- Benton, A. L., Hamsher, K., Varney, N. R., & Spreen, O. (1983). Contributions to neuropsychological assessment. New York: Oxford University Press.
- Blair, R. J. R., Morris, J. S., Frith, C. D., Perrett, D. I., & Dolan, R. J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, 122, 883–893.
- Blumer, D., Montouris, G., & Hermann, B. (1995). Psychiatric morbidity in seizure patients on a neurodiagnostic monitoring unit. *The Journal* of *Neuropsychiatry and Clinical Neurosciences*, 7, 445–456.
- Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., et al. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17, 875–887.
- Broks, P., Young, A. W., Maratos, E. J., Coffey, P. J., Calder, A. J., Isaac, C. L., et al. (1998). Face processing impairments after encephalitis: amygdala damage and recognition of fear. *Neuropsychologia*, 36, 59–70.

- Calder, A. J., Young, A. W., Rowland, D., & Perrett, D. I. (1996). Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cognitive Neuropsychology*, 13, 699–745.
- Cendes, F., Andermann, F., Gloor, P., Gambardella, A., Lopes-Cendes, I., Watson, C., et al. (1994). Relationship between atrophy of the amygdala and ictal fear in temporal lobe epilepsy. *Brain*, 117, 739–746.
- Cook, M. J., Fish, D. R., Shorvon, S. D., Straughan, K., & Stevens, J. M. (1992). Hippocampal volumetric and morphometirc studies in frontal and temporal lobe epilepsy. *Brain*, 115, 1001–1015.
- Dodrill, C. B., & Batzel, L. W. (1986). Interictal behavioral features of patients with epilepsy. *Epilepsia*, 27(Suppl. 2), 64–76.
- Ekman, P., & Friesen, W. (1976). *Pictures of facial affect*. Palo Alto, CA: Consulting Psychologists Press.
- Feichtinger, M., Pauli, E., Schäfer, I., Eberhardt, K. W., Tomandl, B., Huk, J., et al. (2001). Ictal fear in temporal lobe epilepsy: surgical outcome and focal hippocampal changes revealed by proton magnetic resonance spectroscopy imaging. *Archives of Neurology*, 58, 771–777.
- Fine, C., & Blair, R. J. R. (2000). The cognitive and emotional effects of amygdala damage. *Neurocase*, 6, 435–450.
- Fish, D. R., Gloor, P., Quesney, F. L., & Oliver, A. (1993). Clinical responses to electrical brain stimulation of the temporal and frontal lobes in patients with epilepsy: pathophysiological implications. *Brain*, *116*, 397–414.
- Gloor, P. (1972). Temporal lobe epilepsy: its possible contribution to the understanding of the functional significance of the amygdala and its interaction with neocortical-temporal mechanisms. New York, NY: Plenum Publishing Corp.
- Gloor, P. (1990). Experiential phenomena of temporal lobe epilepsy: facts and hypotheses. *Brain*, *113*, 1673–1694.
- Gloor, P. (1992). Role of the amygdala in temporal lobe epilepsy. New York, NY: John Wiley & Sons Inc.
- Gloor, P. (1997). The temporal lobe and limbic system. New York: Oxford University Press.
- Gloor, P., Oliver, A., Quesney, L. F., Andermann, F., & Horowitz, S. (1982). The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Annals of Neurology*, 12, 129–144.
- Goddard, G. V. (1967). Development of epileptic seizures through brain stimulation at low intensity. *Nature*, 214, 1020–1021.
- Goddard, G. V., McIntyre, D. C., & Leech, C. K. (1969). A permanent change in brain function resulting from daily electrical stimulation. *Experimental Neurology*, 25, 295–330.
- Gupta, A. K., Jeavons, P. M., Hughes, R. C., & Covanis, A. (1983). Aura in temporal lobe epilepsy: clinical and electroencephalographic correlation. *Journal of Neurology, Neurosurgery, and Psychiatry*, 46, 1079–1083.
- Halgren, E., Walter, R. D., Cherlow, D. G., & Crandall, P. H. (1978). Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain*, 101, 83–117.
- Hermann, B. P., & Chhabria, S. (1980). Interictal psychopathology in patients with ictal fear. Archives of Neurology, 37, 667–668.
- Hermann, B. P., Dikman, S., Schwartz, M. S., & Karnes, W. E. (1982). Interictal psychopathology in patients with ictal fear: a quantitative investigation. *Neurology*, 32, 7–11.
- Kalynchuk, L. E. (2000). Long-term amygdala kindling in rats as a model for the study of interictal emotionality in temporal lobe epilepsy. *Neuroscience and Biobehavioral Reviews*, 24, 691–704.
- Kuks, J. B., Cook, M. J., Fish, D. R., Stevens, J. M., & Shorvon, S. D. (1993). Hippocampal sclerosis in epilepsy and childhood febrile seizures. *Lancet*, 342, 1391–1394.
- Meletti, S., Benuzzi, F., Rubboli, G., Cantalupo, G., Stanzani Maserati, M., Nichelli, P., et al. (2003). Impaired facial emotion recognition in early-onset right mesial temporal lobe epilepsy. *Neurology*, 60, 426–431.
- Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., et al. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, 383, 812–815.

- Morris, J. S., Friston, K. J., Büchel, C., Frith, C. D., Young, A. W., Calder, A. J., et al. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain*, 121, 47–57.
- Palmini, A., & Gloor, P. (1992). The localizing value of auras in partial seizures: a prospective and retrospective study. *Neurology*, 42, 801–808.
- Perini, G. J., & Mendius, R. (1984). Depression and anxiety in complex partial seizures. *The Journal of Nervous and Mental Disease*, 172, 287–290.
- Phillips, M. L., Young, A. W., Senior, C., Brammer, M., Andrew, C., Calder, A. J., et al. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature*, 389, 495–498.
- Racine, R. J. (1978). Kindling: the first decade. Neurosurgery, 3, 234-252.
- Rosen, J. B., & Schulkin, J. (1998). From normal fear to pathological anxiety. *Psychological Review*, 105, 325–350.
- Schmitz, F. B., Robertson, M. M., & Trimble, M. R. (1999). Depression and schizophrenia in epilepsy: social and biological risk factors. *Epilepsy Research*, 35, 59–68.
- Schmolck, H., & Squire, L. R. (2001). Impaired perception of facial emotions following bilateral damage to the anterior temporal lobe. *Neuropsychology*, 15, 30–38.
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). STAI manual. Palo Alto, CA: Consulting Psychologists Press.
- Sprengelmeyer, R., Young, A. W., Schroeder, U., Grossenbacher, P. G., Federlein, J., Buttner, T., et al. (1999). Knowing no fear. Proc. R. Soc. Lond. B: Biol. Sci., 266, 2451–2456.
- Taylor, D. C., & Lochery, M. (1987). Temporal lobe epilepsy: origin and significance of simple and complex auras. *Journal of Neurology*, *Neurosurgery, and Psychiatry*, 50, 673–681.

- Thomas, K. M., Drevets, W. C., Whalen, P. J., Eccard, C. H., Dahl, R. E., Ryan, N. D., et al. (2001). Amygdala response to facial expressions in children and adults. *Biological Psychiatry*, 49(4), 309–316.
- Van-Paesschen, W., Connelly, A., King, M. D., Jackson, G. D., & Duncan, J. S. (1997). The spectrum of hippocampal sclerosis: a quantitative magnetic resonance imaging study. *Annals of Neurology*, 41, 41– 51.
- Van-Paesschen, W., King, M. D., Duncan, J. S., & Connelly, A. (2001). The amygdala and temporal lobe simple partial seizures: a prospective and quantitative MRI study. *Epilepsia*, 42(7), 857–862.
- Weil, A. A. (1956). Ictal depression and anxiety in temporal lobe disorders. American Journal of Psychiatry, 113, 149–157.
- Weil, A. A. (1959). Ictal emotions occurring in temporal lobe dysfunction. Archives of Neurology, 1, 87–97.
- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *The Journal of Neuroscience*, 18, 411–418.
- Williams, D. (1956). The structure of emotions reflected in epileptic experiences. Brain, 79(1), 29–67.
- Williams, L. M., Phillips, M. L., Brammer, M. J., Skerrett, D., Lagopoulos, J., Rennie, C., et al. (2001). Arousal dissociates amygdala and hippocampal fear responses: evidence from simultaneous fMRI and skin conductance recording. *NeuroImage*, 14, 1070–1079.
- Young, A. W., Hellawell, D. J., Van De Wal, C., & Johnson, M. (1996). Facial expression processing after amygdalotomy. *Neuropsychologia*, 34, 31–39.
- Zung, W. W. (1965). A self-rating depression scale. Archives of General Psychiatry, 12, 63–70.