

Recognition of emotion with temporal lobe epilepsy and asymmetrical amygdala damage

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Abstract

Purpose. Impairments in emotion recognition occur when there is bilateral damage to the amygdala. In this study, ability to recognize auditory and visual expressions of emotion was investigated in people with asymmetrical amygdala damage (AAD) and temporal lobe epilepsy (TLE).

Methods. Recognition of five emotions was tested across three participant groups: those with right AAD and TLE, those with left AAD and TLE, and a comparison group. Four tasks were administered: recognition of emotion from facial expressions, sentences describing emotion-laden situations, nonverbal sounds, and prosody.

Results. Accuracy scores for each task and emotion were analysed, and no consistent overall effect of AAD on emotion recognition was found. However, some individual participants with AAD were significantly impaired at recognizing emotions, in both auditory and visual domains.

Conclusions. The findings indicate that a minority of individuals with AAD have impairments in emotion recognition, but no evidence of specific impairments (e.g., visual or auditory) was found.

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1. Introduction

Accurate recognition of emotions is necessary for appropriate human social interaction. The amygdala plays an important role in our ability to recognize emotions in others, and evidence from studies in nonhuman animals suggests that its functions include processing emotional memories and evaluating social cues [1,2]. Human clinical studies have shown that impairments in recognition of facial expression occur when there is bilateral damage to

the amygdala [3–5] and that recognition of fear is particularly severely affected. Because of the rarity of selective bilateral amygdala lesions, research has been limited mainly to a series of single case studies. In a larger study, nine individuals with bilateral amygdala damage were investigated [6]. Overall, this group was impaired in recognizing fear compared with a brain-injured control group, but the severity of this impairment was variable, with individual performance ranging from severely impaired to within the normal range.

The incidence of emotional [7], behavioral [8], cognitive [9], and, consequently, social difficulties is higher in those with temporal lobe epilepsy (TLE) than in the general adult population. These social difficulties may be externally

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driven in part by stigmatization and public discrimination of epilepsy [10]. However, it is hypothesized that damage to the amygdala and associated impairments in recognition of others' emotions may also contribute to social difficulties.

Research investigating the role of the amygdala raises four issues pertinent to the behavioral sequelae associated with TLE that the authors aimed to address in this study: Does less extensive damage to the amygdala result in deficits of emotion recognition? Are there functional differences between the left and right amygdala in emotion recognition? Is the amygdala's role restricted to recognition of facial expressions, or does it also process other visual and auditory signals of emotion? Does partial damage of the amygdala lead to deficits affecting recognition of specific emotions such as fear or a more diffuse problem in processing emotions in general?

Impairments in recognition of facial expressions have consistently been found when bilateral lesions in humans are studied. However, studies investigating the effects of unilateral amygdala damage differ in their findings [11,12], and evidence for the effects of less extensive amygdala damage is inconclusive. Adolphs et al. [11] tested six individuals with unilateral amygdala damage against brain-injured controls. The main finding was that unilateral damage was not sufficient to result in impaired emotion recognition. Participants with left amygdala lesions rated disgust and sadness as less intense than did those with right amygdala lesions, but all were within the normal range as derived from the control group. In contrast, Adolphs et al. [12] tested 26 post-temporal lobectomy patients with unilateral amygdala damage and observed significant impairments in emotion recognition of facial expressions for a subgroup with right temporal lobectomies. Note that the term *unilateral amygdala damage* is often taken to imply that the contralateral amygdala has no lesion. However, in studies with epilepsy populations, there is often damage to both cerebral hemispheres. In such cases, the primary focus of interest is therefore asymmetric, rather than exclusively unilateral, amygdala damage. In the present study, asymmetric damage was defined as greater than a 20% difference in amygdala volume between the two amygdala structures, as measured using volumetric magnetic resonance imaging (vMRI). In addition, all presurgical participants were due to undergo temporal lobe surgery for epilepsy on the ipsilateral side to the amygdala deemed as being significantly smaller.

The second issue regarding the possibility of functional differences between the left and right amygdala structures also presents with mixed results. For example, Morris et al. [13] used positron emission tomography to demonstrate that facial expressions of fear resulted in greater activation of the left amygdala than expressions of happiness. This activation of the left amygdala increased when the intensity of the fear expression increased. An individual with impaired recognition of

emotion linked to bilateral amygdala damage, D.R. [4,5], had greater damage to the left amygdala than the right amygdala. However, Adolphs et al. [12] found that damage to the right amygdala, but not the left amygdala, was associated with impairments in emotion recognition of facial expressions.

The evidence of the amygdala's role in processing both visual and auditory correlates of emotion recognition is also mixed. Researchers investigating emotion recognition and the amygdala have generally used facial expressions [14] as the target stimuli. When the stimuli used to measure emotion recognition have been expanded to include auditory correlates, results have differed. Scott et al. [15] showed that D.R., a person with bilateral amygdala damage, was impaired in recognition of facial expressions of fear and anger, and had impairments in auditory recognition of fear and anger. Sprengelmeyer et al. [16] investigated an individual, D.M., with bilateral amygdala damage, a left thalamic lesion, and known impairments in recognition of facial expressions of fear. The findings were that D.M. also had a specific deficit in recognizing fear in the domains of body posture and emotional sounds. In contrast, Anderson and Phelps [17] found no difficulties with auditory prosody when they assessed another individual with bilateral amygdala damage and suggested that auditory processing is reliant on amygdalostratial interactions rather than the amygdala alone. Likewise, Adolphs et al. [12] reported no significant differences in auditory prosody recognition when they compared participants with left amygdala damage, participants with right amygdala damage, and a comparison group.

With respect to the debate as to whether the amygdala is particularly concerned with certain emotions or has a more global function in processing all emotions, the majority of investigators report impairments in recognizing negative emotions, particularly fear. However, Rolls [18] argues that this does not imply that the amygdala is involved only in processing these emotions. In his opinion, the finding that negative emotions are impaired may be associated with methodology and the intensity of the stimuli. This theory is consistent with the observation that happy faces in the regularly used Ekman and Freisen series [14] are generally easier to recognize than faces displaying negative emotions, but it would run into problems if findings of impaired recognition of specific emotions (such as fear), regardless of the domain of presentation (faces, voices, etc.), proved to be the norm.

This brief review of the literature provides a clear reason to investigate these four issues further, and, to the authors' knowledge, this is the first study to do so. We used a group design and supplementary analyses of individual cases, testing multiple correlates of auditory and visual emotion recognition and making use of volumetric brain imaging to accurately assess the effects of asymmetrical lesions in the amygdala.

2. Methods

2.1. Participants

All participants gave informed consent. The local medical ethics committee and the local university ethics committee approved the study. For each participant, demographic, psychological, and sociocultural information, including age, sex, ethnicity, employment, current mood (anxiety and depression), intelligence quotient, and neuromedical history, was collected. Participants were adults with an estimated premorbid intelligence quotient (IQ score) above 80. Potential participants with a significant history of neurological disorders other than epilepsy in the AAD groups (e.g., traumatic brain injury, organic brain injury, degenerative neurological changes), a psychiatric disorder, uncorrected hearing or visual impairments, and motor difficulties subjectively affecting their dominant hand were excluded. Participants who reported having a seizure during the day prior to testing were also excluded.

All groups had fewer males than females (total: males = 19, females = 27), but there was no significant difference between groups in ratio of male to female participants ($\chi^2 = 0.846$, $P = 0.655$). No statistically significant differences between groups were detected with respect to premorbid IQ score, as measured by the National Adult Reading Test—Revised [19] ($F(2,45) = 1.96$, $P > 0.1$), anxiety ($F < 1$), depression ($F(2,45) = 2.75$, $P > 0.05$) measured using the Hospital Anxiety and Depression Scale [20], or face recognition ($df = 2, 45$, $P > 0.1$) tested with the Benton Facial Recognition Test—Short Form [21]. This was used to check whether impairments in facial emotion recognition were related to inability to simply perceive a face rather than recognize the expression in individuals who were impaired.

2.1.1. Epilepsy groups

Twenty-eight adults with intractable TLE and clearly defined AAD were recruited from epilepsy surgery programs at a center for neurology and neurosurgery in the United Kingdom. Thirteen participants with right AAD (median age = 33, interquartile range = 31–42) and fifteen with left AAD (median age = 41, interquartile range = 31–46) were recruited; the criterion for AAD was at least a 20% difference between amygdala volumes [22; Howard, personal communication], and all presurgical participants were due to undergo temporal lobe surgery on the ipsilateral side to the amygdala deemed as smaller. All but one of these participants (a postsurgical patient) underwent vMRI. Volume estimation of the amygdala was performed using the Cavalieri method of modern design stereology in conjunction with point counting [23] on high-resolution T1-weighted images (obtained using a 1.5-T Signa whole-body MRI system (GE Medical Systems, Milwaukee, WI, USA); spoiled gradient echo (SPGR) pulse sequence: TE = 9 ms, TR = 34 ms, flip angle = 30°; 124 coronal images, FOV of 20 cm, slice thickness of 1.6 mm). This method has been described in detail previously [22]. Amygdala volume estimation was achieved by sampling a series of equally spaced magnetic resonance (MR) images in a direction parallel to the long axis of the hippocampus, beginning with a random starting position. Each image was overlain with a test system comprising a regular array of test points, and the number of points lying within the transect through the amygdala was recorded. The gray matter of the amygdala is well demarcated from surrounding white matter on T1-weighted MR images, and the posteriormost extent is demarcated from the adjoining anterior hippocampus by the white matter of the alveus. Separation between test points on the square grid used for point counting was 0.234 cm (i.e., 3 pixels), and slice interval was 0.156 cm (i.e., every second MR section). Unbiased estimates of transect area were obtained by multiplying the total number of points recorded by the area corresponding to each test point (i.e., $0.234 \times 0.156 = 0.0365 \text{ cm}^2$). An unbiased estimate of amygdala volume was obtained as the sum of the estimated areas of the structure transects on consecutive systematic sections multiplied by the distance between sections. Up to 150 points were recorded on 7 to 12 systematic random sections. Sectioning and point counting intensities were optimized to achieve a coefficient of error on the Cavalieri

volume estimates of between 3 and 5% [23]. Repeatability and reproducibility coefficients (ICC) of amygdala volume estimation were greater than 0.8.

All participants with epilepsy in the present study had mesial TLE as determined in routine multimodal presurgical investigations. In particular, seizure semiology and invasive ictal foramen ovale recordings with video telemetry suggested the presence of medial TLE, and patients with lateral/neocortical TLE were excluded. With these routine presurgical investigations, it is difficult to distinguish an amygdala seizure origin from a hippocampal onset; however, this was not the goal of the present study. Amygdala pathology was indicated by volume reduction on MRI, a hallmark of neuronal atrophy, and not the focus of seizure onset. Reported results would not have been significantly different if lateralization of epilepsy was considered, as all patients except one had reduced volume of the amygdala ipsilateral to the side of seizure onset. Five of twelve (one participant had no hippocampus volumes available) participants with right AAD and 11 of 14 (one participant had no right sided volume available) participants with left AAD had a 20% change in hippocampal volume asymmetry consistent with amygdala volume asymmetry. One participant with right AAD had a dissociation with hippocampal volume asymmetry (reduced right amygdala and left hippocampal volume) and a contralateral (left) seizure onset. Both groups contained approximately equal numbers of participants who were pre- and postsurgery (postsurgical right AAD = 6, postsurgical left AAD = 7). Although vMRI data were available only for amygdala volumes presurgery, surgical intervention served only to enhance the hemispheric difference between amygdala volumes, and so it was appropriate to include postsurgical participants whose amygdala volumes were significantly asymmetrical presurgically. The mean age of the participants with AAD was 38 (range = 21–53). The mean age at diagnosis was 10 (range = 1–29), with mean duration of epilepsy at time of assessment of 28 years (range = 3–38).

We directly investigated the relationship between structural pathology of the amygdala and recognition of emotion, irrespective of the location of the presumed epileptogenic zone. The most important factor is that all the participants with epilepsy studied formed a homogenous group of patients with medial TLE who showed evidence of left or right amygdala atrophy, which has been reported in patients with medial TLE [24,25]. The median hemispheric difference in amygdala volumes for the left AAD group was 32.95 (interquartile range = 25.5–38.3), and the median difference for the right AAD group was 25.7 (interquartile range = 21.7–30.6). This represented a statistically significant difference (one-tailed $U = 38$, $P < 0.05$).

2.1.2. Comparison group

Eighteen individuals were recruited from a hospital pain management program at the same center. This comparison group was chosen because the individuals had socioeconomic and cultural backgrounds similar to those of the participants with epilepsy.

2.2. Procedure

Four computer-generated tasks were used to test visual nonverbal, visual verbal, auditory nonverbal, and auditory verbal recognition of emotion. The stimuli administered to test these domains were: facial expressions [14], sentences describing emotion-provoking situations developed specifically for this study, nonverbal sounds (screaming, laughing, etc.) [15], and emotional prosody (digits spoken with differing emotional intonations; Calder, unpublished), respectively. Recognition of five basic emotions was tested for each stimulus type. These emotions were happiness, sadness, anger, fear, and disgust. Recognition of a sixth emotion, surprise, from the original Ekman and Friesen series was not investigated as studies have queried whether this has the same status as the five other basic emotions; for example, one can be pleasantly or unpleasantly surprised, and surprise is less well supported as a universal emotion in cross-cultural studies [26]. Administration of the emotion recognition stimuli was counterbalanced across the four tasks. A Dell Latitude CPt laptop computer with external speakers was used to present the visual and auditory stimuli, which were installed using Superlab experimental laboratory software [27]. Participants registered

each answer by pressing one of five emotion-labeled buttons on a button box integrated with the computer programs. The four tasks were designed to be comparable in the number and presentation of stimuli for each emotion. The presentation of stimuli was randomized within each task, and the task order was block randomized. The number of correct responses for each emotion in each task was recorded.

2.2.1. Administration

Prior to administration of each task, participants were given written information presented on the monitor advising them of the format of the task and instructions on how to complete it. Each participant was asked to decide which of the five emotions each stimulus best represented. To further reduce cognitive demand, participants were also reminded of the five emotions during each trial by having the emotions written across the bottom of the screen in the same order that they appeared on the button box. The written instruction “get ready” appeared briefly between presentations. Prior to the 50 trials for each task (10 trials for each of the five emotions), participants were given 10 practice examples. Participants were not given feedback on performance during the study.

2.3. Statistical analysis

To address the four issues of the effects of asymmetrical amygdala damage—lateralization of amygdala function, the amygdala’s role in emotion recognition of auditory and visual stimuli, and the specific emotions processed by the amygdala—the data were analyzed in two ways. First, the accuracy scores for the three groups were compared in each task and for each emotion. Then, the accuracy scores of individuals from the two groups with amygdala damage were compared with the scores of the comparison group by calculating z scores for each participant. A repeated-measures analysis of variance (ANOVA) was performed to test the significance of any differences between the three groups with respect to task and emotion. This ANOVA had one between-group factor of participant Group (left AAD, right AAD, comparison group), and two within-group factors of Task (recognition of emotion from faces, sentences, sounds, or prosody) and Emotion (fear, anger, disgust, sadness, or happiness). Individual differences were investigated further by calculating z scores initially for total score in each task and then by analyzing the profile of scores across emotions and domains in those individuals whose performance in each task was significantly ($P < 0.01$) below the mean of all participants.

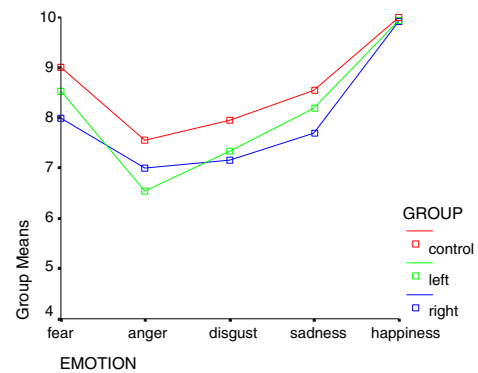
3. Results

3.1. Group data

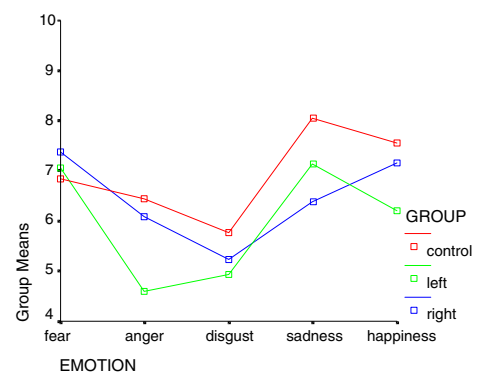
Although the two groups with amygdala damage tended to be less accurate at recognizing emotions than the comparison group, there was no significant difference between the three groups with respect to accuracy scores by emotion in each task (ANOVA; faces: $F(2, 3) = 1.39$, $P > 0.1$; prosody: $F(2, 3) = 1.51$, $P > 0.1$; sentences: $F(2, 3) = 2.26$, $P > 0.1$; sounds: $F(2, 3) = 3.7$, $P > 0.1$) (Fig. 1).

Significant main effects of Task ($F(3, 41) = 67.76$, $P < 0.001$) and Emotion ($F(4, 40) = 22.77$, $P < 0.001$) were found. Specifically, prosody scores were significantly lower than scores in other tasks, suggesting that individuals found this task more difficult than others, independent of their amygdala status. All three groups made the most mistakes recognizing anger and were most accurate in recognizing happiness. A significant Task \times Emotion interaction ($F(19, 25) = 11.47$, $P < 0.001$) reflected the fact that

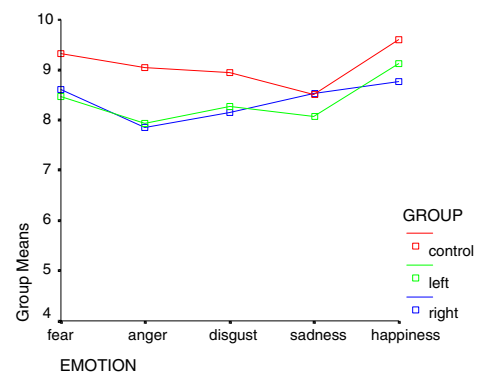
Comparison of accuracy group means for faces



Comparison of accuracy group means for prosody



Comparison of accuracy group means for sentences



Comparison of accuracy group means for sounds



Fig. 1. Comparison between groups of the mean accuracy scores for each emotion in the four tasks.

differences between overall recognition rates for the different emotions were less evident in the sentence task than in the other tasks.

The main purpose of the ANOVA, however, was to look for effects of participant group. Therefore, the results are in accordance with previous findings that asymmetrical damage to the amygdala is not consistently associated with impairments in emotion recognition [11]. With respect to functional differences between the left and right amygdala in emotion recognition, no significant differences were found in performance between the two groups, suggesting no lateralization of function in processing specific emotions or discriminating auditory and visual correlates of expressions of emotion.

3.2. Individual performance profiles

ANOVA identifies consistent effects in data, and inspection of the data revealed large standard deviations and skewed distributions of scores in the two AAD groups, indicating that some individuals with amygdala lesions had considerable difficulty with at least one of the tasks. An analysis of deficits at the individual case level was therefore conducted, to determine whether consistent patterns of impairment emerged. This was done first by identifying all individual participants in the AAD groups whose overall score on any one of the four emotion recognition tests used (faces, sentences, sounds, prosody) reflected substantial impairment. Within this subgroup of individuals whose overall performance was impaired on one or more of the tests, we then looked at the profile of ability to recognize the five basic emotions in each test. Individuals from the AAD groups with overall scores reflecting substantial impairment were identified by expressing each individual participant's overall performance on each of the four tests as a z -score difference from the comparison group mean, and choosing a conservative cutoff of $P < 0.01$ to distinguish impaired cases from nonimpaired cases. This cutoff point was chosen to reduce the probability of type 2 errors arising from the number of z scores calculated, considering the extent of the data collected.

Seven individuals from the AAD groups met the criterion of substantial overall impairment ($z > 2.33$, $P < 0.01$) on at least one of the four tasks. Their scores are summarized in Table 1. Three patterns emerged:

1. Overall impairment of emotion recognition, with poor performance on at least three of the four tasks
2. Impairment on the face test, with performance on the other three tests less severely affected
3. Impairment on the sentence task only

We now consider what these patterns of impairment imply for the questions addressed in our study. With respect to the first issue of the effects of asymmetrical amygdala damage on recognition of emotional expressions, the fact that seven individuals were identified as having significant impairments in emotion recognition ($P < 0.01$) for at least one of the four tasks indicates that a minority of individuals with asymmetrical amygdala damage do have difficulty recognizing emotion.

Second, the profiles of these seven individuals reveal no evidence for a lateralization of function between the left amygdala and right amygdala. Three individuals had asymmetrical damage to the right amygdala, and four individuals had asymmetrical damage to the left amygdala. There were no obvious differences between the profiles of individuals with left or right amygdala lesions.

With respect to the third issue, the amygdala's role in processing visual and auditory expressions of emotion, results are more interesting. At first sight, the two principal patterns of impairment correspond to what has been reported in the literature on bilateral amygdala damage, namely, domain-specific impairment of facial expression recognition (pattern 2 [cf. 12,17]) or multimodal impairment affecting both auditory and visual stimuli (pattern 1 [cf. 15,16]). However, closer inspection of the data for cases R3, L2, and L3 suggests caution. Although it is clear from their profiles that deficits in facial expression recognition were the most prominent, they all scored more than 1 SD below the mean in at least one other domain. In other words, their performance on the non-face tasks was not convincingly normal, leaving open the possibility of a more general impairment more akin to pattern 1.

This suspicion was borne out by a further analysis used to explore the fourth issue, that of whether the amygdala is specifically involved in the recognition of some emotions and not others. To explore this at the individual level, the performance of the seven participants was broken down according to recognition of each of the five basic emotions

Table 1
Patterns of performance of individuals^a showing substantial impairments ($P < 0.01$) in overall performance on at least one of the four tasks

	Pattern 1: overall impairment			Pattern 2: impaired mainly on face test			Pattern 3: impaired on sentence test
	R1	R2	L1	R3	L2	L3	L4
Face	-7.66 ^d	-5.23 ^d	-3.15 ^d	-2.45 ^d	-4.88 ^d	-5.58 ^d	-0.02
Sentence	-7.39 ^d	-2.66 ^c	-7.39 ^d	-1.40	-1.40	-1.09	-3.29 ^c
Sound	-3.80 ^d	-0.18	-3.44 ^d	0.00	-1.27	-1.09	0.54
Prosody	-2.34 ^c	-1.84 ^b	-1.72 ^b	0.42	-2.09 ^b	-1.21	0.29

^a Three right AAD (R1, R2, R3) and four left AAD (L1, L2, L3, L4). Performance is expressed as z -score differences from the comparison group mean for total accuracy in each task.

^b Degree of impairment: $z > 1.65$, $P < 0.05$.

^c Degree of impairment: $z > 2.33$, $P < 0.01$.

^d Degree of impairment: $z > 3.10$, $P < 0.001$.

used in each test. The resulting profiles are outlined in Table 2.

For individuals grouped as pattern 1 (R1, R2, and L1), impairments affecting each of the five basic emotions were found, consistent with the conception of an overall impairment of emotion recognition. For pattern 2 (participants R3, L2, and L3), there was also no clear emotion-specific pattern. Across the different tests, participant R3 was impaired in recognizing anger and happiness; L2 was impaired in recognizing fear, anger, disgust, and sadness; and L3 was impaired in recognizing fear, anger, and sadness. Moreover, all three of the participants grouped into pattern 2 showed some impairment on tasks other than facial expressions when their performance across each emotion was investigated in detail. This further undermines the view that their problems are associated exclusively with facial emotion. For pattern 3 (participant L4), the problem was restricted to emotional sentences, but encompassed all emotions except anger.

The reason why a subgroup of individuals with AAD demonstrate deficits in emotion recognition remains unclear. Close inspection of medical and neuropsychological reports revealed no obvious relation of clinical features or neuropsychological deficits to the specific pattern of impairment of emotion recognition with the exception of participant L4, whose poor performance on sentences

may have been associated with language impairments (Tables 3 and 4).

4. Discussion

The results suggest that AAD does not consistently result in impairments in emotion recognition, lending support to previous evidence that AAD does not invariably impair recognition of facial expressions [11]. Other findings of impairments in emotion recognition may be related to the damage being more extensive, such as with bilateral destruction of the amygdala. Adolphs et al. [12] found a moderate negative correlation between extent of amygdala damage and overall performance in recognition of facial expressions. A substantial variability in performance across participants may have contributed to the present null finding at the group level. The fact that surprise was not included as a target emotion may also have made fear easier to recognize, as it is most readily confused with surprise [14,28]. In addition, our decision to measure recognition accuracy rather than have participants rate intensity of emotion may account for the difference in the results between this study and some others [3,12].

Although no significant differences between groups were found in accuracy over the four tasks, seven participants with amygdala damage were impaired in recognizing

Table 2
Individual profiles for recognizing emotions across the four tasks^a

Emotion	Task	Pattern 1: overall impairment			Pattern 2: impaired mainly on face test			Pattern 3: impaired on sentence test
		R1	R2	L1	R3	L2	L3	L4
Fear	Face	-6.72b	-2.52c	-1.68d	0.00	-1.68d	-2.52c	0.84
	Sentence	-9.52b	-3.03c	-8.22b	0.87	-1.73d	-1.73d	-1.73d
	Sound	-3.44b	-0.88	-3.02c	0.40	0.40	-1.31	0.83
	Prosody	-1.50	0.05	-1.50	0.05	-1.89d	0.44	0.44
Anger	Face	-4.03 ^b	-3.30 ^b	-2.58 ^c	-2.58 ^c	-3.30 ^b	-4.75 ^b	1.58
	Sentence	-2.14 ^d	-2.14 ^d	-3.54 ^b	-2.14 ^c	-1.44	-0.74	0.24
	Sound	-4.01 ^b	1.02	-1.21	-0.10	-2.89 ^c	-0.65	0.32
	Prosody	-0.62	-1.05	-2.33 ^c	0.24	-1.91 ^d	-2.34 ^c	-0.74
Disgust	Face	-3.80 ^b	-3.80 ^b	-2.26 ^d	-1.49	-2.26 ^d	-1.49	-1.49
	Sentence	-4.45 ^b	-2.65 ^c	-5.35 ^b	0.05	0.96	0.96	-1.74 ^d
	Sound	-5.55 ^b	-4.06 ^b	-7.04 ^b	0.42	-1.08	0.42	-1.08
	Prosody	-1.60	-2.02 ^d	-0.75	0.52	-2.44 ^c	-1.17	0.52
Sadness	Face	-2.44 ^c	-1.75 ^d	-0.38	-1.07	-2.44 ^c	-3.12 ^b	0.30
	Sentence	-1.54	0.31	-2.16 ^d	-0.31	-0.93	-0.93	-2.78 ^c
	Sound	-0.77	0.90	-1.34	0.34	-0.78	-0.78	-1.34
	Prosody	-4.18 ^b	-3.36 ^b	-1.70 ^d	-0.88	-0.05	-1.70 ^d	-0.05
Happiness	Face	NA	NA	NA	NA	NA	NA	NA
	Sentence	-11.22 ^b	-1.22	-5.22 ^b	-3.22 ^b	-1.22	-1.22	-3.22 ^b
	Sound	-0.45	0.22	-1.78 ^d	-1.11	-0.45	-0.45	0.89
	Prosody	-3.04 ^c	-2.37 ^c	-0.37	1.63	-1.04	-0.37	-0.37

NA, indicates that a ceiling effect on recognition of happy faces meant that meaningful *z* scores could not be calculated in each task.

^a Performance is expressed as *z* scores for recognition of emotion.

^b Degree of impairment: $z > 3.10$, $P < 0.001$.

^c Degree of impairment: $z > 2.33$, $P < 0.01$.

^d Degree of impairment: $z > 1.65$, $P < 0.05$.

Table 3
Medical profiles of the seven individuals with impairments in emotion recognition

	Pattern 1: overall impairment			Pattern 2: impaired mainly on face test			Pattern 3: impaired on sentence test		
	R1	R2	L1	R3	L2	L3	L4	L4	L4
Gender	Female	Female	Male	Male	Female	Male	Male	Male	Male
Age	25	46	44	30	39	46	17	17	17
Diagnosis	C-P ^a	C-P, 2 °G	C-P	C-P	C-P, 2 °G	C-P, 2 °G	C-P, 2 °G	C-P, 2 °G	C-P, 2 °G
Onset of seizures	Infancy	17	Infancy	Infancy	Infancy	Infancy	Infancy	Infancy	Infancy
MR scan	Right MTS	Bi MTS	Left MTS	Bi HS	Left HS	Left MTS	Right MTS	Right MTS	Left TA
EEG activity	Right frontal, temporal, and parasagittal	Right temporal	Left temporal	Right temporal	Bitemporal	Left temporal	Left temporal	Bitemporal	Bitemporal
WADA	Language: left Memory: impaired bi	Not done	Language: left Memory: impaired bi	Language: left Memory: impaired right	Language: left Memory: impaired left	Language: right Memory: impaired left	Language: right Memory: impaired left	Language: Not done	Language: Not done
Postsurgical	No	No	No	No	Yes	Yes	Yes	Yes	Yes

^a C-P, complex partial seizures; 2 °G, secondary generalization; Bi, bilateral; MTS, medial temporal sclerosis; HS, hippocampal sclerosis; TA, temporal atrophy.

emotion in at least one domain. This suggests that a minority of individuals with AAD do have difficulty recognizing emotional expressions. This minority included three individuals with right AAD and four with left AAD, a ratio that provides no evidence for lateralization of function with respect to emotion recognition. Furthermore, no distinction between deficits in auditory and visual domains was detected between those with right and those with left damage. Impairments in recognition of facial expression were the most common, but individuals who at first sight might have been taken to show face-specific impairments turned out, on closer investigation, to also be impaired to different extents in other domains. This supports previous findings that the amygdala is involved in processing facial expressions [4,5,11], but also suggests that the amygdala has a role recognizing emotions in other domains.

As has been noted in other studies of the consequences of amygdala damage, impairments of facial expression recognition were unrelated to problems in perceiving faces picked up by the Benton Test of Facial Recognition [21]. Recognition of facial expressions does not seem to reflect a more general visual impairment or a problem in perceiving faces per se.

Individuals with emotion recognition impairments had the most consistent difficulty recognizing negative emotions, expressions previously found to be associated with the amygdala, but there was no convincing evidence of selective impairments restricted to specific emotions (such as fear and anger). Although individuals with bilateral amygdala damage and highly selective impairments have been reported in the literature [3,15,16,29], a study of a group of individuals with bilateral amygdala damage showed a more heterogeneous pattern [6]. It is clear that although highly selective impairments affecting the recognition of fear, in particular, can be detected after amygdala damage, they are displayed in such a striking manner only by a minority of individuals. This is not to deny previous demonstrations of selective impairments, but it does draw attention to the need to understand better the reasons for the variability in outcome.

In the present study, the subgroup of individuals with impairments in emotion recognition was heterogeneous in their medical and neuropsychological profiles. Although some of the variability in emotion recognition deficits in the present group of participants might be thought to be linked to the extent of extra-amygdala pathology, we have noted that a degree of variability also characterizes cases with lesions restricted to the amygdala region. Further research is therefore required to define the risk factors associated with such impairments.

The finding that a minority of individuals with TLE and amygdala damage demonstrated impairments in emotion recognition might be linked to the numerous potential factors that can influence emotion recognition, and this is consistent with the biopsychosocial framework of chronic conditions [30]. For example, Reynders and others [31] investigated social cognition and quality of life

Table 4
Neuropsychological profiles of the seven individuals with impairments in emotion recognition

Test	Measure	Pattern 1: overall impairment			Pattern 2: impaired mainly on face test			Pattern 3: impaired on sentence test
		R1	R2	L1	R3	L2	L3	L4
NART	Predicted IQ score	92	90	85	102	85	120	84
WAIS-R/ WISC-III	Verbal IQ score	69	65	82	83	88	106	75
	Performance IQ score	68	69	81	104	86	93	65
WMS-R/ WMS-III	Verbal memory index	55	71	69	73	89	54	56
	Visual memory index	60	65	72	88	92	47	100
	Attention index	56	76	96	87	107	68	108
COWAT	Language	9	6	7	18	13	NT ^a	NT
FAS	Verbal fluency (%ile)	<1	11–22	1122	11–22	11–22	NT	NT
Stroop	Executive skills (%ile)	<2	15	9	51	100	2	NT
Benton	Face recognition	34	41	49	46	41	49	43
HADS	Anxiety	8	11	8	8	6	16	9
	Depression	4	6	5	5	2	12	9

^a NT, not tested, or data not available.

with epilepsy based on clinical evidence of individuals with TLE and ictal fear compared with individuals with TLE but no ictal fear and individuals with idiopathic generalized epilepsy rather than amygdala pathology. They found that all three epilepsy groups had difficulty recognizing fear, and this was associated with impaired social judgment of trustworthiness, duration of epilepsy, and quality of life. This suggests that other factors as well as amygdala damage are important in the complex process of recognizing emotions. Other evidence in support of this includes the impact of TLE and hippocampal damage on emotional memory [32]. Unexpected results were found, in that both patients with left TLE and patients with right TLE demonstrated impairments in face memory, whereas only patients with left TLE were significantly impaired in facial expression memory, and this was related to impaired recognition of facial expressions in left TLE and age. The investigators hypothesized that functional reorganization of verbal memory in left TLE may compromise visual memory. They also suggested that patients with right TLE may be able to verbalize visual material more successfully than those with left TLE. However, this does not explain previous studies, for example Adolphs et al. [12], that demonstrated that patients with right TLE are more likely to show impairments in emotion recognition.

Furthermore, our study investigated the impact of reduced amygdala volume on emotion recognition. Other studies have shown that enlarged amygdala volumes may be associated with psychopathology impacting on behavior. For example, Trimble and van Elst [33] found that psychosis in epilepsy might be linked to enlarged amygdala volumes. The investigation of enlarged amygdala volumes and emotion recognition in epilepsy may be a useful addition to the literature in the future.

Our study has several limitations. It would have been advantageous to undertake vMRI with the pain comparison group, but this was not possible, in part, because members of the group were attending an independent

support group. However, we believe that the robustness of sampling was comparable to that of previously published studies, and potential participants with a known history of neurological or psychiatric disorders were excluded. We were careful to select a comparison group with a sociocultural and demographic profile similar to that of the participants with epilepsy. In this way, we were able to control for these potentially confounding factors. Pre- and postsurgical patients were included, which may have impacted our results. However, there were approximately equal numbers of both in the two epilepsy groups, and participants who had experienced a seizure within 24 hours of the experiment were excluded. In addition to patients having lateralized TLE and mesial temporal sclerosis clinically decided as appropriate for surgery, we also chose to exclude participants with less than a 20% difference in amygdala volumes. Our rationale for this was that we were specifically interested in AAD and were concerned that some previously published studies had not measured relative amygdala volumes quantitatively, thus potentially confounding the results.

The aim of this study was to investigate the effects of AAD in TLE on recognition of emotional expression across auditory and visual domains. The study was designed to address four key issues identified from previous research on emotion recognition and the role of the amygdala. The outcome provided further information regarding the role of the amygdala by investigating multiple domains of emotion recognition and testing a relatively large number of participants with known amygdala damage.

In conclusion, this study indicates that a minority of individuals with AAD do have impairments in emotion recognition. No uniform pattern was detected when clinical and neuropsychological profiles were made of these individuals. There was no evidence of lateralization of function between the left and right amygdala, and impairments in emotion recognition were found across visual and auditory domains. Furthermore, impairments were not specific to

particular emotions, but found globally across all five emotions. Although the reasons for variability in the impact of amygdala damage on emotion recognition remain to be determined, elucidating the factors involved is of potential importance to informing decisions about the treatment of epilepsy.

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