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Face processing impairments after encephalitis: amygdala damage and recognition of fear

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Abstract—Face processing and facial emotion recognition were investigated in five post-encephalitic people of average or above-average intelligence. Four of these people (JC, YW, RB and SE) had extensive damage in the region of the amygdala. A fifth post-encephalitic person with predominantly hippocampal damage and relative sparing of the amygdala (RS) participated, allowing us to contrast the effects of temporal lobe damage including and excluding the amygdala region. The findings showed impaired recognition of fear following bilateral temporal lobe damage when this included the amygdala. For JC, this was part of a constellation of deficits on face processing tasks, with impaired recognition of several emotions. SE, YW and RB, however, showed relatively circumscribed deficits. Although they all had some problems in recognizing or naming famous faces, and had poor memory for faces on the Warrington Recognition Memory Test, none showed a significant impairment on the Benton Test of Facial Recognition, indicating relatively good perception of the face's physical structure. In a test of recognition of basic emotions (happiness, surprise, fear, sadness, disgust and anger), SE, YW and RB achieved normal levels of performance in comparison to our control group for all emotions except fear. Their results contrast with those of RS, with relative sparing of the amygdala region and unimpaired recognition of emotion, pointing clearly toward the importance of the amygdala in the recognition of fear.

Key Words: basic emotions; temporal lobes; amygdala; hippocampus; fear.

Introduction

The importance of the amygdala in social perception has become clear from animal and human studies [5, 9]. The consequences of human amygdala damage have been found to include problems in recognizing facial expressions of emotion [33, 34]. Among the basic emotions, it has been noted that recognition of fear can be particularly severely affected by amygdala damage [2, 3], and positron emission tomography (PET) and magnetic resonance imaging (fMRI) techniques have been used to demonstrate a response to facial expressions of fear in the normal amygdala [7, 22]. However, recent reports have suggested that bilateral damage to the amygdala may be required to produce such impairments [3], and that in addition such damage should either be congenital or acquired early in life [18].

The claim that bilateral amygdala damage is necessary is based on Adolphs *et al.*'s [3] findings that whereas studies of a person with bilateral amygdala damage due to an inherited metabolic disorder (Urbach–Wiethe disease) demonstrated impaired recognition of facial expressions of fear, investigations of a further six individuals with unilateral amygdala damage did not show any such impairment. Adolphs *et al.* [3] concluded, therefore, that bilateral and not unilateral amygdala damage impairs the recognition of fear from facial expressions. Alternatively, though, one might instead infer from their data that the

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results for Adolphs *et al.*'s [3] single case of bilateral damage were in some way atypical. Using the same procedures as Adolphs *et al.* [2], Hamann *et al.* [18] did not find impairments of expression perception in two cases of bilateral amygdala damage caused by herpes simplex encephalitis. This led Hamann *et al.* [18] to suggest that problems in recognizing fear after amygdala damage may depend upon congenital disease or early-acquired damage. The relatively low intelligence of the patients studied by Adolphs *et al.* [2, 3] and by Young *et al.* [33, 34] was also considered to be a possible contributory factor [18].

Hamann *et al.*'s [18] failure to find problems in emotion recognition in people with late-onset amygdala damage contrasts with our own observations of cases SE and DR, who each show such impairment. SE [10] sustained damage to the amygdala at the age of 55 years as a result of herpes simplex encephalitis, and DR [10, 33, 34] was 28 years old before she developed the epilepsy which eventually led to her having bilateral amygdala surgery. Although in the case of DR it might be thought that there could have been some pre-existing pathology predisposing her to develop epilepsy, there are no grounds to argue for any pre-existing brain pathology in SE's case.

We have therefore further examined this apparent discrepancy between our own observations and those of Hamann *et al.* [18]. Using the same logic as Hamann *et al.* [18], but adopting procedures used in our earlier studies of DR and SE, we investigated face processing and facial emotion recognition in five post-encephalitic people of average or above-average intelligence. Four of these people had extensive damage in the region of the amygdala. In addition, a further post-encephalitic person with a less common pattern of predominantly hippocampal damage and relative sparing of the amygdala participated. We were thus able to contrast the effects of temporal lobe damage including and excluding the amygdala region.

Case descriptions

We studied four people with amygdala damage due to encephalitis: JC, SE, YW and RB. These were contrasted with a fifth person, RS, who had suffered damage which was predominantly in the hippocampal region, with sparing of the amygdala. MRI scans are shown in Fig. 1, which includes coronal sections through the amygdala for all participants, an additional horizontal section to show the lack of further focal cortical damage in RB, and an additional coronal section passing through the hippocampus for RS. Summaries of damage to temporal lobes and other brain regions are given in Table 1.

JC

A 68-year-old woman who suffered from presumed herpes simplex encephalitis at the age of 67 years (some 18 months prior to testing). MRI showed extensive right-sided damage to the amygdala, hippocampus, parahippocampal gyrus, entorhinal and perirhinal cortex. Less extensive damage was also found in comparable regions on the left. Right inferior and middle temporal gyri were severely affected and the superior temporal gyrus was partially destroyed. Additionally, there was damage to the medial and anterior thalamus on the right. A lesion was evident in the right mesial infracalcarine region, but other neocortical regions appeared to be unaffected.

JC's speech is grammatical, fluent and generally appropriate. She complains of becoming forgetful as a consequence of her illness, in particular having difficulty 'matching names with faces', and of problems with topographical orientation. Although she is known to have experienced episodes of depression, her mood at the time of testing was unremarkable. She is cared for in a residential home.

SE

A 64-year-old man who suffered from presumed herpes simplex viral encephalitis at the age of 55. MRI showed extensive destruction of the right temporal pole, uncus, amygdala (including all nuclei), hippocampus, parahippocampal gyrus, and inferior and middle temporal

Fig. 1. Coronal T1-weighted magnetic resonance images through similar regions of the amygdaloid complex in JC (A), SE (B), YW (C), RB (D1) and RS (E1), together with a horizontal section for RB (D2) and a coronal image through RS's hippocampus reformatted along the long axis of that structure (E2). The arrows indicate areas of detectable damage to these structures. In each case the images are left/right reversed from the viewer's perspective. JC has bilateral damage to the amygdala, hippocampus and related structures, which in each case is more extensive on the right. In addition, there is destruction of temporal lobe neocortex and subjacent white matter, especially in the right hemisphere, where damage is widespread throughout the lobe. SE has bilateral amygdala damage, more severe on the right, along with extensive destruction of the right temporal pole, uncus, hippocampus, parahippocampal gyrus, and inferior and middle temporal gyri to the level of the insula. YW has extensive bilateral amygdala damage, as well as extensive destruction of the entorhinal, perirhinal and parahippocampal cortices, and the hippocampus in each hemisphere. RB's detectable damage is confined to the left side of his brain and includes the region of the left amygdala and hippocampus; the horizontal section shows the absence of focal neocortical damage outside these areas. RS shows extensive bilateral damage to the hippocampus but no detectable damage to either the amygdaloid complex or the cortices underlying either the amygdala or the hippocampus.