No impairment of recognition and experience of disgust in a patient with a right-hemispheric lesion of the insula and basal ganglia

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An influential single case study (Calder, Keane, Manes, Antoun, & Young, 2000, Nature Neuroscience, 3, 1077–1078) recently showed a marked multimodal impairment in the recognition and experience of disgust in a patient with a left-hemispheric lesion of the basal ganglia and the insular cortex. Here, we investigated whether a similar deficit will be observed in a patient with a comparable lesion. The patient showed no impairments in the recognition or experience of disgust and also no notable impairments in the recognition and experience of other emotions, across a range of stimuli, as compared to healthy comparison subjects. Thus, either deficits in disgust processing are not reliably observed in patients with lesion of the insula and basal ganglia regardless of the laterality of the lesion; or right-hemispheric lesions, in contrast to left-hemispheric lesions, do not seem to induce impairments in the processing of disgust.

1. Introduction

There is an ongoing debate on the neural substrates underlying the processing of basic emotions (e.g., Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000; Adolphs, Tranel, & Damasio, 2003; Calder, Lawrenc, & Young, 2001; Damasio et al., 2000; Harciarek, Heilman, & Jodzio, 2006; Phillips et al., 1997). For example, a disproportionate, but not exclusive role of circumscribed brain areas in emotion processing has been emphasized by studies investigating the recognition of disgust (Calder, 2003; Calder, Keane, Manes, Antoun, & Young, 2000; Sprengelmeyer et al., 1996). Neurological diseases that affect basal ganglia and insula – such as Huntington’s disease (HD) or Wilson’s disease – are often associated with impairments in the recognition of disgust stimuli (Hennenlotter et al., 2004; Kipps, Duggins, McCusker, & Calder, 2007; Sprengelmeyer et al., 1996; Thieben et al., 2002; Wang, Hoosain, Yang, Meng, & Wang, 2003). Furthermore, several functional imaging studies have shown activation of the insular cortex and the basal ganglia during the recognition and experience of disgust (Calder et al., 2007; Jabbi, Bastiaansen, & Keysers, 2008; Jabbi, Swart, & Keysers, 2007; Phillips et al., 1997, 1998; Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998; van der Gaag et al., 2007). A significant role of these brain areas is indicated by several studies investigating the processing of disgust stimuli in patients with lesions of specific brain regions (e.g., Adolphs et al. 2003), even though insula activation has also been shown during the processing of emotions other than disgust (e.g., Jabbi et al., 2007; Schienle, Stark, et al., 2002; Straube, Kolassa, Glauser, Mentzel, & Miltner, 2004; Straube, Mentzel, & Miltner, 2005, 2006; van der Gaag et al., 2007). One study demonstrated – using depth implant electrodes in the ventral section of the insula of patients with epilepsy – selective responses to disgusted facial expressions relative to other facial emotions (Krolak-Salmon et al., 2003).

However, lesion studies with subjects with circumscribed lesions to specific brain regions are necessary to critically test the assumed functions of these brain areas. To our knowledge, there is only one single case study by Calder et al. (2000) investigating the processing of disgusting stimuli in a patient with a left hemisphere infarction involving the insula and basal ganglia. This patient showed a clear impairment in the recognition and processing of disgust in several stimulus categories as well as impaired emotional expression. This well-controlled study strongly suggests a significant role of the insula and/or basal ganglia, at least in the left hemisphere, for the recognition and experience of disgust. Furthermore, Adolphs et al. (2003) reported a patient with large bilateral lesions of several brain areas including the insula. This patient showed several deficits in emotion processing, but processing of disgust stimuli was disproportionately impaired for the recognition of dynamic emotional expressions and of short stories. The authors suggested that bilateral insula lesions might be responsible for this finding.
Calder et al. (2001; see also Calder, 2003) proposed an insula-basal ganglia system as the neural substrate for disgust processing. Based on this idea and the previous single cases, however, it is still unclear whether the impairment in disgust processing is specifically associated with lesions of the left hemisphere, or whether similar findings are also observed in patients with lesions of the insula and basal ganglia in the right hemisphere. Kipps et al. (2007) found that recognition of disgust correlated with left insula volume in pre-symptomatic individuals with HD (i.e., gene carrier). Based on functional imaging studies, however, there are no consistent findings on whether the impairment of disgust processing should be bound to left-hemispheric lesions only. Whereas some studies have demonstrated a role for the left insula (Hennenlotter et al., 2004; Sprengelmeyer et al., 1998; Wicker et al., 2003), others have reported bilateral (Jabbi et al., 2008; 2007; Phillips et al., 1997, 1998; Schienle, Stark, et al., 2002; Stark et al., 2007; van der Gaag et al., 2007) or even predominantly right-hemispheric activation of the insula in response to disgust-related stimuli (Phillips et al., 1997). Thus, functional neuroimaging data provides little evidence for a robust lateralization of disgust processing.

However, the right insula has been suggested as playing a role in providing an integrative center for interoceptive processes (Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Craig, 2002; Straube, Schmidt, Weiß, Mentzel, & Milten, 2009) and to be involved in the processing of emotion in general (Adolphs et al., 2000; Craig, 2002). Thus, one would expect that patients with right-hemispheric insula and basal ganglia lesions show at least similar deficits in disgust processing to patients with left-hemispheric lesions. However, due to the assumed role of the right insula in emotion processing, there should be even more general deficits in emotional function.

In the present study, we investigated this in a patient with lesions of the insula and basal ganglia in the right hemisphere. The lesion pattern of this patient was comparable to the lesion pattern of the patient with insula and basal ganglia lesions of the left hemisphere investigated by Calder et al. (2000). If the insula and basal ganglia are reliably involved in disgust processing regardless of the lateralization of the lesion, the present patient should show at least some deficits in the processing of disgust stimuli, as compared to a sample of healthy control subjects.

2. Methods

2.1. Subjects

2.1.1. Single Case M.K

The patient was recruited following participation in a rehabilitation program for motor deficits due to stroke, one of several neuropsychological projects our group conducts with chronic stroke patients (e.g. Miltner, Bauder, Sommer, Dettmers, & Taub, 1999; Richter, Miltner, & Straube, 2008; Straube, Schulz, Geipel, Mentzel, & Milten, 2008). M.K. is a right-handed German-speaking 26-year-old male who, prior to his stroke, was working as a mechanic. At the time of this study, he was unemployed. His stroke occurred in 1999, 9 years prior to the start of this study, and affected the middle cerebral artery of his right hemisphere. The lesion was confirmed by a high-resolution T1-weighted anatomical volume (192 slices, TE = 5 ms, matrix = 256 x 256 mm, resolution = 1 mm x 1 mm x 1 mm; duration = 12 min) obtained by a 1.5 T magnetic resonance scanner (“Magnetom Vision plus”, Siemens, Medical Systems, Erlangen, Germany). The lesion is shown in Fig. 1. For this study, an experienced neuropsychologist (C.P.) also diagnosed it as a perisylvian infarction of the right middle cerebral artery. The complete right insula and all parts of the right basal ganglia, including the head of the caudate nucleus, the caudate body, putamen, and pallidum, were lost. Claustrum, external and extreme capsula were also lesioned. The lesion further comprised about 1/3 of the superior temporal gyrus (the anterior pole was preserved, but the lesion included the planum temporale). All other areas in the right temporal lobe, including the amygdala, were structurally intact. All opercula were affected: the parietal oeculopel, the rolandic operculum and the triangular part of the frontal operculum. Besides damage to the frontal and rolandic operculum, the right frontal cortex showed lesions in the ventral precentral gyrus. Most of the ventral postcentral gyrus was also damaged. As there was no infarction of the anterior cerebral artery, the most upper parts of the motor and somatosensory cortices were intact. The internal capsule was lesioned except for a very small bridge of tissue in the posterior limb. The lateral part of the thalamus was destroyed. Those parts of the thalamus that depend on blood supply from the posterior cerebral artery were intact as well as basal parts of the thalamus. The lesion extended with gliotic signal alterations upwards into the lateral and dorsal white matter compartment including the corona radiata. M.K. suffered from moderate paresis of the left upper and lower extremities and reported impairments in audition, which prevented additional assessments of his emotional auditory processing capacity. He was well oriented and had no history of psychiatric or additional neurological disorders. His verbal intelligence, measured by the MWT-A (Lehrl, Merz, Burkard, & Fischer, 1991), was slightly below average (verbal IQ: 83). Standard neuropsychological tests [Sub-tests (Alertness with and without warning, Divided attention, Go/Nogo, and Neglect test) of the Test Battery for Attention Performance (TAP, Zimmermann & Fimm, 1993); a German version of WMS-R (Hüting, Markowitsch, Neufeld, Calabrese, & Deisinger, 2000)] revealed impairments of attention and memory. The patient had no signs of neglect (neglect test of the TAP) or of basal visual perceptual impairments [recognizing famous faces; subtest “Bilder ergänzen” of the German version of the WIFA-R (Tewes, 1991)]. Background neuropsychological data are summarized in Table 1. We also tested his conceptual knowledge about basic emotions by requesting descriptions of two typical situations where people feel sad, angry, surprised, happy, disgusted, or fearful. The patient showed no impairment of knowledge about these emotions, since he could easily describe two situations for each emotion. The transcript of the patient’s description of typical situations where people feel disgust (as well as other emotions) is provided in supplementary data. We also asked the patient whether he experiences disgust in his everyday life and we asked whether he could remember specific experiences from the recent past. He answered that he experiences disgust rather frequently and that he remembered two situations from the recent past. In the first situation, he experienced disgust when confronted with a dirty public toilet and in the second, he was disgusted by a dirty toilet in the apartment of a friend. Thus, based on these retrospective answers, the patient did not describe any impairment in the experience of disgust.

2.1.2. Control subjects

We also recruited nine healthy, right-handed male control subjects who were of similar age and who had passed similar levels of education to M.K. (mean: 23.8 years; range: 21–31 years). All subjects provided informed consent to participate in the study. The experimental procedures were approved by the Ethics Committee of the Friedrich-Schiller-University of Jena, and the investigation was performed in...
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