Neuropsychological and neural correlates of autobiographical deficits in a mother who killed her children

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Neuropsychological and neural correlates of autobiographical deficits in a mother who killed her children

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We report a case of a delusional patient who had killed two of her children in an attempted ‘extended suicide’. She was convinced of a genetic defect that caused autobiographical memory and emotional deficits and made life ‘senseless’. Neuropsychological tests revealed dysfunctions in remembering emotional details of personal episodes and theory of mind. Water positron emission tomography (15O) with a paradigm used in a former study by Fink et al. (1996) with healthy controls elicited abnormal activations during autobiographical memory retrieval characterised by a lack of prefrontal and limbic activity. We conclude that these imaging findings reflect neural correlates of the self-reported and objectified autobiographical dysfunctions. Furthermore, they indicate that beliefs or prejudices may have a major impact on the brain’s processing of the personal past.

Keywords: Neuroscience; Crime; Autobiographical memory; Theory of mind; Positron emission tomography.

INTRODUCTION

Autobiographical-episodic memory is the highest memory system that consists of personal events with a clear relation to time, space and context; in Tulving’s words, the conjunction of subjective time, autonoetic consciousness and the experiencing self represents episodic memories, which are typically emotionally toned (Tulving, 2002, 2005). Representing our personal past, autobiographical-episodic memory allows subjective time travel and is fundamental for building the feeling of one’s own identity (Conway & Pleydell-Pearce, 2000). Accordingly, disorders of autobiographical memory usually lead to disastrous consequences of the individuals’ lives as they lose their personal past and consequently their personality (e.g., Markowitsch, 2003a, 2003b). Emotional deficits also commonly occur in those patients, as one function of autobiographical memory is to guide emotional behaviour on the bases of previous personal experiences having an emotional connotation (Kopelman, 2000; Serra, Fadda, Buccione, Caltagirone, & Carlesimo, 2007).

Retrograde amnesia for the personal past, covering either the whole life-span or distinct parts of the biography, can be caused by brain damage, predominantly if limbic and/or prefrontal structures are affected. In addition, psychic stress and traumas can also result in severe autobiographical memory disorders, a condition referred to as dissociative, psychogenic, or functional amnesia.
In summary, autobiographical deficits, whether they are pronounced, as seen in patients with amnesia caused by brain damage or with dissociative amnesia, or whether they are more specific, as seen in depression or schizophrenia, can be linked to emotional dysfunctions and changes of the feeling of one’s own self and may have distinct functional brain correlates.

It is also noteworthy that criminal offenders can have neuropsychological dysfunctions including deficits in emotional processing and memory (Deeley et al., 2006; Raine et al., 2005). In neuroscientific research, there is growing interest in investigating neural mechanisms underlying violent behaviour (Markowitsch & Siefer, 2007). Using modern brain imaging techniques, both structural and functional abnormalities were reported in individuals with antisocial personality disorder (Kiehl et al., 2001; Müller et al., 2003) as well as in murderers (Davidson, Putnam, & Larson, 2000; Raine, Stoddard, Bihrl, & Buchsbaum, 1998; Raine, Lencz, Bihrl, LaCasse, & Colletti, 2000). Typically, prefrontal and limbic brain regions are affected (Abbott, 2001; Brower & Price, 2001; Bufkin & Luttrel, 2005). However, it has not been reported yet that the personal burden due to autobiographical deficits or other cognitive dysfunction can lead to violent behaviour to oneself or others.

Here we report a so far unique case of a forensic delusional patient who had killed two of her own children in an act of ‘extended suicide’. In her own view, she suffered from a genetic defect that led to an inability to build up an identity and feel emotionally connected to her past (which according to the theory described above reflects autobiographical memory dysfunction), and which consequently led to the same problems in her children. This train of thought was the self-reported reason for the offence. The aim of the current investigation was to reveal potential cognitive-mnestic and functional brain correlates of the patient’s self-reported deficits. For this purpose, we administered an extensive neuropsychological test battery, and we measured the regional cerebral blood flow (rCBF) with positron emission tomography (PET) during an autobiographical memory paradigm that had been used in a former PET study with healthy control subjects (Fink et al., 1996).

CASE STUDY

Two years before our examination, the 49-year-old female, right-handed patient AA, a nurse, was admitted to a forensic clinic after having murdered
two of her three children (9 and 10 years old) by first sedating them with doxepin at home and then drowning them in a river. She had then tried to commit suicide by cutting her arteries but was found early enough to be rescued. The offence was interpreted as an act of ‘extended suicide’. According to the court expertise, which referred to an extensive psychiatric and neurological examination, she suffered from an affective disorder in the form of a long-lasting severe depressive episode with psychotic symptoms (F 32.3 according to ICD-10) and was thus found not guilty by reason of insanity. CT scans had shown no structural brain alterations.

AA’s prominent psychotic symptom consisted of the delusion that she (and, consequently, also her three children) suffered from a genetic defect that made her life ‘senseless’. She reported that this defect resulted in an inability to build up an identity and to feel emotionally connected to her past. She said that she was unable to feel emotions. Furthermore, she reported that she could not identify other people’s emotions or intentions by interpreting their facial expressions or by grasping information from the intonation in speech. Although her behaviour in the clinic was regarded as ‘social’ and she took part in social events and even had the role of a ‘spokesperson’ in that setting, she also stated that she was unable to communicate with other people.

AA was convinced that death was the only way out and that it was right to kill her children. She also regarded the life of her oldest daughter, who was not at home when she committed the offence, as ‘senseless’ and believed that death would be better for her. Her descriptions of the offence were reported to have been unemotional.

At the time of our examination, 2 years after the offence, her mood was described as retained and not depressive any more. She had discontinued antidepressant medication about 1 year prior to our examination. Besides the delusion about her genetic defect, other psychotic symptoms were not detectable. There was no evidence of psychopathy according to the psychologist’s evaluation and the Psychopathy Checklist-Revised (PCL-R, Hare, 2003) (score of 8 out of 40 points).

AA had great effort in finding objective evidence for her assumed defect, and she was convinced that so far no one had diagnosed her disease correctly. She attentively studied relevant popular scientific literature and had contacted a number of researchers, longing for an examination. It was also her initiative to contact one of the authors (HJM).

METHODS

Neuropsychology

An extensive neuropsychological test battery was administered to AA (Table 1). Besides an assessment of basic cognitive functions, instruments were used that test functions which, according to the patient’s self reports, could be dysfunctional, that is autobiographical memory (concluded from the statement that she was unable to build an identity and to feel emotionally connected to her past), emotional processing, and theory of mind (inferred from reports of an inability to decode other people’s emotions or intentions). It is noteworthy that the patient herself, as a layman, did not use the terms ‘autobiographical memory’ or ‘theory of mind’.

In detail, the neuropsychological test battery included standardised instruments to assess the patient’s general cognitive state (DemTect, Kalbe et al., 2004) and intelligence (Leistungsprüfsystem, Horn, 1983), executive functions (interference condition of the Word Colour Interference Test, Bäumler, 1985), verbal fluency using the letters F, A, and S (Strauss, Sherman, & Spreen, 2006), speed of information processing (conditions ‘reading colour words’ and ‘naming colours’ of the Word Colour Interference Test), and selective attention (d2 test, Brickenkamp & Zillmer, 1998).

Anterograde memory was assessed with the Wechsler Memory Scale-Revised (WMS-R, German version by Härtling, Markowitsch, Neufeld, Calabrese, & Deisinger, 2000). Two further tests were used to determine the possible influence of the patient’s emotional state on memory performance: The Affective Word Test (Fujiwara et al., in press) in which emotional and neutral words have to be recalled, and the emotional and neutral photographs (e.g., those used by von Cramon, Markowitsch, and Schuri, 1993). In this latter task, 40 out of 80 photographs of various scenes have to be recognised after a 15-min delay. Half of the photographs had been rated as particularly touching emotionally by 20 normal subjects.

Autobiographical memory was assessed with the Bielefeld Autobiographical Memory Inventory (Fast & Markowitsch, in press), a semistructured interview which is based on the technique proposed by Kopelman, Wilson, and Baddeley (1989) and in which autobiographical facts and autobiographical episodic memory from specified time intervals of the whole life span have to be recalled and evaluated regarding vividness, detailedness, and emotionality.
Further tests for remote memory were the Kieler Remote Memory Test (Kieler Altgedächtnistest, Leplow, Blunck, Schulze, & Ferstl, 1993; Leplow, Dierks, Merten, & Hänsgen, 1997) which assesses knowledge of famous events, and the Famous Faces Test (Fast, Fujiwara, & Markowitsch, in preparation)

### TABLE 1
Results of the neuropsychological test battery

<table>
<thead>
<tr>
<th>Domain</th>
<th>Test/Instrument</th>
<th>AA's performance</th>
<th>Reference score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DemTect</td>
<td>16</td>
<td>15.3 (2.8)</td>
</tr>
<tr>
<td>Cognitive state/ Intelligence</td>
<td>Leistungsprüfssystem (LPS-K)</td>
<td>PR: 54</td>
<td>PR: 50</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive functions, attention</td>
<td>Verbal Fluency: FAS-test, sum of words</td>
<td>PR: 80</td>
<td>PR: 50</td>
</tr>
<tr>
<td>and speed of information processing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Word Colour Interference Test: reading colour words</td>
<td>T: 56</td>
<td>T: 50 (20)</td>
</tr>
<tr>
<td></td>
<td>colour naming</td>
<td>T: 55</td>
<td>T: 50 (20)</td>
</tr>
<tr>
<td></td>
<td>interference trial</td>
<td>T: 48</td>
<td>T: 50 (20)</td>
</tr>
<tr>
<td></td>
<td>Selective Attention Test (d2); total minus errors</td>
<td>PR: 46</td>
<td>PR: 50</td>
</tr>
<tr>
<td>Anterograde memory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>WMS-R (indices); verbal memory</td>
<td>95</td>
<td>100 (15)</td>
</tr>
<tr>
<td></td>
<td>visual memory</td>
<td>94</td>
<td>100 (15)</td>
</tr>
<tr>
<td></td>
<td>general memory</td>
<td>94</td>
<td>100 (15)</td>
</tr>
<tr>
<td></td>
<td>attention/concentration</td>
<td>106</td>
<td>100 (15)</td>
</tr>
<tr>
<td></td>
<td>delayed recall</td>
<td>116</td>
<td>100 (15)</td>
</tr>
<tr>
<td></td>
<td>Affective Word Test: affective judgements (errors)</td>
<td>1</td>
<td>1.3 (1.3)</td>
</tr>
<tr>
<td></td>
<td>free recall</td>
<td>6</td>
<td>4.1 (1.8)</td>
</tr>
<tr>
<td></td>
<td>recognition</td>
<td>13</td>
<td>13.0 (1.6)</td>
</tr>
<tr>
<td></td>
<td>false positives</td>
<td>0</td>
<td>1.4 (1.4)</td>
</tr>
<tr>
<td></td>
<td>Recognition of Emotional and Neutral Photographs:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>emotional</td>
<td>64%</td>
<td>95%</td>
</tr>
<tr>
<td></td>
<td>neutral</td>
<td>50%</td>
<td>88%</td>
</tr>
<tr>
<td>Retrograde memory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bielefeld Autobiographic Memory Inventory:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>semantic</td>
<td>80%</td>
<td>90 (11)%</td>
</tr>
<tr>
<td></td>
<td>episodic: free</td>
<td>53%</td>
<td>89 (11) %</td>
</tr>
<tr>
<td></td>
<td>episodic: details</td>
<td>20%</td>
<td>85 (16) %</td>
</tr>
<tr>
<td></td>
<td>period of life: pre-school age</td>
<td>50%</td>
<td>90 (12) %</td>
</tr>
<tr>
<td></td>
<td>primary school age</td>
<td>55%</td>
<td>94 (15) %</td>
</tr>
<tr>
<td></td>
<td>secondary school age</td>
<td>55%</td>
<td>934 (6) %</td>
</tr>
<tr>
<td></td>
<td>early adulthood (up to 35 years)</td>
<td>36%</td>
<td>64 (5) %</td>
</tr>
<tr>
<td></td>
<td>latest past</td>
<td>23%</td>
<td>91 (15) %</td>
</tr>
<tr>
<td></td>
<td>total</td>
<td>43%</td>
<td>96 (9) %</td>
</tr>
<tr>
<td></td>
<td>Kieler Remote Memory Test</td>
<td>43%</td>
<td>cut-off: &gt;40%</td>
</tr>
<tr>
<td></td>
<td>Famous Faces Test:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>verbal, recognition</td>
<td>90%</td>
<td>94 (8)</td>
</tr>
<tr>
<td></td>
<td>verbal, semantic information</td>
<td>90%</td>
<td>89 (14)</td>
</tr>
<tr>
<td>Affective processing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ekman &amp; Friesen: naming</td>
<td>11</td>
<td>11 (1)</td>
</tr>
<tr>
<td></td>
<td>multiple choice</td>
<td>16</td>
<td>22 (2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tübinger Affect Battery:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>facial identity discrimination</td>
<td>100%</td>
<td>99 (3)</td>
</tr>
<tr>
<td></td>
<td>facial affect discrimination</td>
<td>93%</td>
<td>92 (6)</td>
</tr>
<tr>
<td></td>
<td>facial affect naming</td>
<td>93%</td>
<td>95 (6)</td>
</tr>
<tr>
<td></td>
<td>facial affect selection</td>
<td>100%</td>
<td>97 (4)</td>
</tr>
<tr>
<td></td>
<td>facial affect matching</td>
<td>73%</td>
<td>94 (5)</td>
</tr>
<tr>
<td></td>
<td>nonemotional prosody discrimination</td>
<td>73%</td>
<td>100 (0)</td>
</tr>
<tr>
<td></td>
<td>emotional prosody discrimination</td>
<td>100%</td>
<td>100 (0)</td>
</tr>
<tr>
<td></td>
<td>name the emotional prosody</td>
<td>93%</td>
<td>95 (8)</td>
</tr>
<tr>
<td></td>
<td>match emotional prosody to an emotional face</td>
<td>93%</td>
<td>96 (7)</td>
</tr>
<tr>
<td>Theory of mind</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reading-the-Mind-in-the-Eyes-Test (max. score = 18)</td>
<td>12</td>
<td>15 (2)</td>
</tr>
<tr>
<td></td>
<td>Happé stories:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Theory of Mind Stories (max. score = 16)</td>
<td>10</td>
<td>15 (1)</td>
</tr>
<tr>
<td></td>
<td>Control ‘Physical’ Stories (max. score = 16)</td>
<td>10</td>
<td>12 (2)</td>
</tr>
<tr>
<td></td>
<td>Multiple-Choice-Theory-of-Mind-Test</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(max. score = 16)</td>
<td>9</td>
<td>14 (2)</td>
</tr>
</tbody>
</table>

Impaired scores in AA’s performance (deviation of more than 1 SD from reference data) are indicated in bold.
in which famous persons from politics, sports, and cultural life have to be recognised.

Affective processing was tested with a set of 28 black and white photographs taken from Ekman and Friesen (1975) which show facial emotional and neutral expressions and which had to be described first and then matched to corresponding terms in a multiple choice design (anger, disgust, fear, joy, sadness, surprise, or neutral). Furthermore, the Tübinger Affect Battery (Breitenstein, Daum, Ackermann, Lütgetheinman, & Müller, 1996), a German version of the Florida Affect Battery (Bowers, Blonder, & Heilman, 1998), was used. It includes two sets of tasks testing the ability to process facial affect and emotional prosody, respectively. Finally, theory of mind, i.e., the ability to infer other people’s mental states such as emotions, thoughts and intentions, was administered with the Reading-the-Mind-in-the-Eyes-Test (Baron-Cohen, Wheelwright, Hill, Raste, & Plumbe, 2001, German version by Kalbe et al., unpublished material), with a story comprehension task including theory of mind stories and control (non-theory of mind) stories (German translations of the material introduced by Happé, 1994) as well as with the Multiple-Choice-Theory-of-Mind-Test (Kalbe et al., unpublished material) in which character’s thoughts in short scenes have to be inferred in a multiple choice paradigm.

**Positron-Emission-Tomography (PET)**

To find possible neural correlates of the episodic autobiographical memory dysfunctions that were derived from AA’s reports and were confirmed by neuropsychological tests (see below), an autobiographical memory paradigm was chosen that had been used by a former study by Fink et al. (1996) with healthy controls. AA’s individual results could thus be compared to those of historical controls scanned according to exactly the same protocol. The PET design included 12 sequential measurements of relative regional cerebral blood flow (rCBF). It consisted of three study conditions: one control condition (REST [A]) with eyes closed and no auditory or visual stimulation, and two experimental conditions: in the FICTITIOUS (B) condition, auditory sentences were presented that contained episodic autobiographical information of a person our patient AA did not know, but which had been introduced to her about 1 h before PET scanning. In the AUTOBIOGRAPHICAL (C) condition, sentences were read to AA that contained episodic information taken from her own life but not referring to the offence. All episodes were brief and significant events of the other person’s life (FICTITIOUS, e.g., ‘With my French exchange student I mixed cocktails’ or ‘My first car was a VW beetle’) and her own life, respectively (AUTOBIOGRAPHICAL, e.g., ‘From my Russian pen pal I received a small parcel’ or ‘A cat that frequently crept around our garden became our pet’). AA received the instruction to imagine what happened to the person in the described situations (FICTITIOUS) or to imagine what happened to herself in the described situations (AUTOBIOGRAPHICAL). AA’s episodes were partly taken from the patient’s history and partly referred to information she had given in the Autobiographical Memory Inventory. The fictive episodes of the other person were matched to those taken from AA’s life concerning topics and emotionality. The presentation order of the study condition was ABCCBAACBBCA.

Relative rCBF was measured by recording the regional distribution of cerebral radioactivity after the intravenous injection of $^{15}$O-labeled water. Measurements were performed on an ECAT-EXACT HR scanner (Siemens Medical Systems, Erlangen, Germany) in 3D-Mode. After correction for measured attenuation and scatter, 12 images consisting of 47 transaxial slices with a thickness of 3.125 mm were reconstructed using a filtered back-projection algorithm with a Hanning-filter (cut-off frequency of 0.4 pixels/cycle). For data analysis, images were ratio-normalised for differences in global CBF, filtered with a spherical Gaussian kernel of 8 mm FWHM, and difference images of the contrast AUTOBIOGRAPHICAL–REST, FICTITIOUS–REST and AUTOBIOGRAPHIC–FICTITIOUS were computed. Significance was assessed on $z$-transformed images using a global variance estimate from balanced noise images as previously described (Thiel et al., 2001). Voxels with $z$-scores greater than 3 or less than –3 were regarded as significantly activated or deactivated. $Z$-score images were transformed to the MNI152-standard brain using FSL (FMRIB, Oxford) and activation/deactivations are reported in Talairach coordinates.

**RESULTS**

**Neuropsychology**

An overview of the neuropsychological test results is given in Table 1. AA was well adapted and
cooperative throughout the examination. She was well oriented to time and place. Her cognitive performance was within or above average in tests for the general cognitive state, intelligence, executive functions, attention, and speed of information processing.

Anterograde memory performance as assessed with the WMS-R and also the Affective Word Test was in the normal range. Remote memory as tested with the Kieler Remote Memory Test and the Famous Faces Test was unaffected in AA. In contrast, marked deficits were evident in autobiographical memory. The Autobiographical Memory Inventory revealed that, while semantic autobiographical memory was only slightly below the average, the recall of personal episodes was thoroughly disturbed with a temporal gradient towards the present. The classification of episodes remembered revealed that AA mostly remembered general, recurring events, i.e., events that occurred over a longer period of time such as ‘With our athletic sports group we regularly participated in contests’ or ‘My friend lived with her grandparents and we used to play at their big kitchen table’. These general events and also the few single events AA remembered could not be described in detail. She also reported that she could not visualise these memories at all and that remembering them did not arouse any emotional involvement.

Contradicting results were observed regarding affective processing in AA. While the majority of subtests of the Tübingen Affect Battery and also naming of facial expressions using the pictures from Ekman and Friesen (1975) were within the normal range, AA scored below average both in the facial affect matching task of the Tübingen Affect Battery and in the comparable condition using the Ekman pictures (matching facial expressions to corresponding terms of emotion). Significant disturbance of the ability to infer other people’s mental states were observed in all used theory of mind tasks. It is noteworthy that AA’s performance in the control ‘physical’ stories of the Happé story task was unaffected.

**PET**

**Activation and deactivation patterns in patient AA**

Contrasting the two activation conditions, AUTOBIOGRAPHICAL–FICTITIOUS, revealed significant activations within the right posterior and left anterior cingulate gyrus as well as anterior part of the right insular cortex (Table 2 and Figure 1). Furthermore, several regions were deactivated significantly, i.e., the right anterior cingulate gyrus, the right medial and the lateral orbitofrontal cortex, the left inferior frontal gyrus and the left angular gyrus.

When the REST condition was subtracted from the AUTOBIOGRAPHICAL condition, activations were found in the left and right superior temporal gyrus as well as the temporal and frontal poles. Deactivations were demonstrated in the right anterior and middle cingulate gyrus and primary and visual association cortices of both hemispheres.

In the FICTITIOUS minus REST contrast, inferior frontal and lateral temporal regions bilaterally as well as the left parietal association cortex were activated. The right sided primary visual cortex and the middle part of the cingulate gyrus of both hemispheres were deactivated. Furthermore, the left anterior and right posterior cingulate gyrus were deactivated. These latter deactivations most likely lead to the activations of these areas found in the AUTOBIOGRAPHICAL minus FICTITIOUS contrast.

**Comparison to normal healthy controls**

Compared to the historical healthy controls (Fink et al., 1969) there were no significant activations found in right medial temporal lobe structures (e.g., hippocampal formation, amygdala) for the contrast AUTOBIOGRAPHICAL–FICTITIOUS, whereas the left-sided activations observed in our patient were not reported in healthy control subjects.

In the contrast AUTOBIOGRAPHICAL–REST, again wide-spread activations of the left hemisphere in patient AA were found which were not observed in normal controls. Concerning the deactivations, the same occipital pattern of task related CBF-decrease was observed as in controls but the deactivation of the right cingulate was only seen in patient AA.

Differences were also seen for the contrast FICTITIOUS–REST. In healthy controls the parietal regions, which were activated in patient AA, were found to be inactivated. In contrast, the posterior cingulate region was activated in controls and deactivated in AA. The deactivation of the occipital cortex was observed in our patient as in controls.
We here describe the case of a forensic delusional patient AA who killed two of her own children in an act of extended suicide. The patient herself reported that her main dysfunction was not building an identity or feeling emotionally connected to her past (in the author’s view describing episodic autobiographical memory dysfunction, see Conway & Pleydell-Pearce, 2000) as well as an inability to feel and decode emotions (interpreted as theory of mind dysfunction) and to communicate. Our investigation revealed two main findings: (1) the neuropsychological profile is in accordance with the self-reported deficits. She suffered from selective autobiographical episodic memory changes. In detail, there was a lack of specificity and emotionality in remembering episodes from her past. Moreover, theory of mind deficits were observed. (2) Specific brain activation patterns linked to autobiographical memory retrieval as examined with PET indicate potential neural correlates of her self-reported dysfunctions.

Referring to the neuropsychological pattern, AA had intact retrograde memory both for general semantic information as well as facts of her biography. She also retrieved episodes from her life. However, the events retrieved usually had more general character in the way that they were not single episodes but happened repeatedly (e.g., ‘Every morning on the way to school…’). Thus, they do not reflect what Tulving (2002, 2005) is referring to.
as ‘episodic memories’ which have a clear relation to a specific time and a singular locus. Most importantly, she was unable to report affective connotations of autobiographical episodes and did not have a feeling of her own self when retrieving events. This was even evident when she reported the act of killing her children. The report included the temporal order of acting on the day of the offence, but no emotional approach was perceived. These circumstances also indicate that she did not vividly remember her biography as defined by Tulving, who proposed that episodic memories are emotionally toned and self-related. Accordingly, we suggest that AA suffered from a specific form of autobiographical memory dysfunction.

As outlined in the introduction, deficits in autobiographical remembering including the emotional connotation of episodes are the main symptom of dissociative amnesia (e.g., Brandt & van Gorp, 2006; Kopelman, 2000; Kritchevsky, Chang, & Squire, 2004; Markowitsch, 2003b). Therefore, one might hypothesise that AA’s deficits reflect symptoms of this kind of dissociative condition. On the other hand, the mnestic profile revealed is not directly comparable to the pattern typically found in patients with this form of amnesia. These patients commonly ‘forget’ a whole life span or episodes from the whole life including the sense of their own identity (Fujiwara et al., in press). In fact, our patient AA reported that she had no sense for her own self. Nevertheless, she could report episodes from all time periods of her life. Therefore, we do not think that she matches with the condition of dissociative amnesia, at least not in its typical form.

Recalling more general than specific events of the personal past has been referred to as ‘over-generalised memory’. This phenomenon is often accompanied by less frequent retrieval of episodic-specific emotional connotations, as seen for example in patients with depression (Barnhofer et al., 2002; Decker, Hermans, Raes, & Eelen, 2003; Van Vreeswijk & De Wilde, 2004; Williams, 1996), persecutory delusions (Kaney et al., 1999), or schizophrenia (Riutort, Cuervo, Danion, Peretti, & Salame, 2003; Sonntag et al., 2003). These groups of patients also frequently show theory of mind dysfunctions (Harrington, Siegert, & McClure, 2005b; Lee, Harkness, Sabbagh, & Jacobson, 2005). It is noteworthy that it has been proposed that there is a relationship between a tendency to over-generalised memory and theory of mind reductions (Corcoran & Frith, 2003). This might explain the convergent neuropsychological pattern that was observed in our patient AA, who suffered from theory of mind abnormalities beyond the specific

![Figure 1. [15O]H2 PET images of patient AA with activations (a) and deactivations (b) in the main contrast AUTOBIOGRAPHICAL–FICTITIOUS.](image-url)
autobiographical memory alterations mentioned above. Potentially, deficits in inferring others’ mental states, especially emotions, can compromise retrieval of specific events, as it is difficult to develop a feeling of one’s own emotional state without being able to access main features of social interactions. In other words, attributions to others can serve as cues for remembering contextual details of an event (for further discussion see Corcoran & Frith, 2003). Additionally, the theory of mind reductions may also contribute to AA’s delusional symptoms, given that this specific link has been shown in patients with paranoid delusions (Craig, Hatton, Craig, & Bentall, 2004; Harrington, Langdon, Siegert, & McClure, 2005a).

As potential neural correlates of AA’s autobiographical memory complaints (circumscribed as the inability to build an identity and feel connected to her past), the PET investigation revealed a lack of activation in brain structures that have previously been linked to autobiographical memory retrieval. It is noteworthy that the lack of a direct control group is a limitation of our study. However, AA’s results could be compared to historical control subjects of a former study (Fink et al., 1996) which used exactly the same PET paradigm.

The main deviation of AA’s activation patterns compared to our historical control subjects and further studies were consistent in less recruitment of right hemisphere regions and more activation in left-sided areas, which are not observed in normal controls. For instance, there are several studies indicating that the prefrontal cortex is crucially engaged in retrieval processes. However, in patient AA, no prefrontal activations were found while she attempted to retrieve episodes from her past. By contrast, the medial and lateral orbitofrontal cortices were even deactivated in the AUTOBIOGRAPHICAL–FICTITIOUS contrast. This pattern potentially reflects AA’s specific autobiographical memory dysfunction, as there is convergent evidence for the involvement of both the lateral and the medial orbitofrontal cortex section in the retrieval of the emotional tone of personal episodes (Brand & Markowitsch, 2006; Cabeza et al., 2004; Conway et al., 1999; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005; Levine, 2004; Maguire, Henson, Mummery, & Frith, 2001; Markowitsch, Vandekerckhove, Lanfermann, & Russ, 2003; Ryan et al., 2001).

Concerning the lateral orbitofrontal cortex, there is also evidence for its engagement in discriminating lies from truth (Langleben et al., 2005; Markowitsch et al., 2000; Spence et al., 2001) and in telling lies and deceptions. Our patient’s deactivation in this region further emphasizes possible difficulties in processing details of episodic memories and in using these details for discriminating real and fictitious episodic information.

Beyond the prefrontal regions, structures of the medial temporal lobe, e.g., the hippocampal formation and the amygdala, were also not activated during AA’s autobiographical memory retrieval attempts. Previous studies with healthy individuals consistently found these structures activated when remembering personal episodes (Addis, Wong, & Schacter, 2007; Cabeza & St. Jacques, 2007; Fink, 2003; Fink et al., 1996; Maguire & Frith, 2003; Piefke, Weiss, Zilles, Markowitsch, & Fink, 2003; Piefke, Weiss, Markowitsch, & Fink, 2005; Piefke & Fink, 2005; Vandekerckhove, Markowitsch, Mertens, & Wiermann, 2005; Viard et al., 2007).

Some activations seen in AA conformed with findings in healthy control subjects. In the main contrast (AUTOBIOGRAPHICAL–FICTITIOUS), activations were found within the cingulate gyrus (posterior on the right and anterior on the left side) as well as in the anterior part of the right insula. In previous studies, the anterior cingulate gyrus was found to be involved in a number of emotional and motivational processes, but also in attention and executive functions such as set-shifting and cognitive flexibility (Allman, Hakeem, Erwin, Nimchinsky, & Hof, 2001; Kondo, Osaka, & Osaka, 2004; Lie, Specht, Marshall, & Fink, 2006; Otsuka, Osaka, Morishita, Kondo, & Osaka, 2006; Shima & Tanji, 1998). It also plays a crucial role in social cognition (Frith, 2002, 2007). Furthermore, it is fundamentally involved in a network underlying episodic memory encoding and retrieval (e.g., Brand & Markowitsch, 2003; Markowitsch, 1995, 2000; Piefke et al., 2003; Svoboda, McKinnon, & Levine, 2006; Vandekerckhove et al., 2005). Beyond this, the anterior section of the cingulate gyrus is also associated with the detection of conflicts and resulting erroneous responses (Bush, Luu, & Posner, 2000; Carter et al., 1998; Gehring & Knight, 2000; Hester, Fassbender, & Garavan, 2004; Luu, Flaisch, & Tucker, 2000; van Veen & Carter, 2006; Yeung, Holroyd, & Cohen, 2005). Given that the activation design used in our patient study comprised items which were either autobiographical or fictitious, the task required the patient to detect which category each item belonged to. Therefore, the activation of the anterior cingulate gyrus...
potentially reflects processing this kind of cognitive conflict, as demonstrated by previous studies mentioned above.

Regarding the activation in the right posterior cingulate gyrus and precuneus, this finding might indicate that our patient did recognise the sentences describing personal episodes as belonging to her own past. The precuneus has been found to be part of a network for self-referential processing or self-reflective consciousness (e.g., Cavanna & Trimble, 2006; Johnson et al., 2002; Kjaer, Nowak, & Lou, 2002; Uddin, Kaplan, Molnar-Szakacs, Zaidel, & Iacoboni, 2005; Vogele et al., 2004). Activation of this region was also seen in studies of episodic and autobiographical memory retrieval (e.g., Lou et al., 2004; Maddock, Garrett, & Buonocore, 2001; Pieck et al., 2003; Sugura, Shah, Zilles, & Fink, 2005; Svoboda et al., 2006). Furthermore, it may be involved particularly in the retrieval of visualisable information (Fletcher et al., 1995; Fletcher, Shallice, Frith, Frackowiak, & Dolan, 1996), though this is controversial.

Nevertheless, one has to notice that both the anterior and the posterior part of the cingulate gyrus were activated in the contrast AUTOBIOGRAPHICAL minus FICTITIOUS but deactivated in the contrast FICTITIOUS minus REST. This pattern suggests that the cognitive conflict of detecting true and fictitious information, as argued above, did not occur – or at least not as strongly – while processing fictitious episodes. This means that the patient most likely had no problem to decide whether an event was fictitious or not and that performing the FICTITIOUS condition did not require self-referential processing. On the other hand, recognising a true event as belonging to her own biography was more directly tapping into processing self-relevant information and may thus have been more effortful compared to processing the fictitious events. However, this point remains speculative, as no behavioural measure was included in the paradigm. The result that the cingulate gyrus was not activated in the AUTOBIOGRAPHICAL minus REST contrast may result from self-referential processing also in the REST condition; a phenomenon which is also frequently referred to as ‘default mode’ (Greicius, Krasnow, Reiss, & Menon, 2003; Hampson, Driesen, Skudlarski, Gore, & Constable, 2006).

One interesting question is whether the psychopathological symptoms in our patient, namely delusions, were related to the neural alterations described above. Functional imaging studies examining autobiographical memory in delusional patients are sparse. However, existing data have not shown changes in activation patterns which are similar to what we observed in our patient. For example, Blackwood et al. (2004) found abnormalities of cingulate gyrus activation (which was not abnormal in AA) while determining self-relevance in schizophrenic patients with active persecutory delusions. It may thus be assumed that the delusions themselves were not related to the changes we observed in the present study. However, since our paradigm or similar ones so far have not been used in patients with delusions, the exact relationship between deficits in autobiographical memory and delusions remains to be further characterised. This endeavour, however, is beyond the scope of this case report.

In summary, our patient suffered from specific neuropsychological deteriorations covering the ability to remember her own past emotionally and vividly, as well as deficits in inferring other people’s mental states. In addition, functional imaging elicited an abnormal activation pattern characterised by a lack of prefrontal and limbic, especially medial temporal engagement in episodic memory retrieval. To conclude, this study demonstrates that self-reported autobiographical dysfunctions accompanied by theory of mind deficits – which according to our patient were the reason for the offence in order to prevent her children from a ‘senseless life’ – can have neuropsychological and functional brain correlates.

REFERENCES

AUTobiographical deficits in a mother who killed her children


