Distinct illusory own-body perceptions caused by damage to posterior insula and extrastriate cortex

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Recent research in cognitive neuroscience using virtual reality, robotic technology and brain imaging has linked self-consciousness to the processing and integration of multisensory bodily signals. This work on bodily self-consciousness has implicated the temporo-parietal, premotor and extrastriate cortex and partly originated in work on neurological patients with different disorders of bodily self-consciousness. One class of such disorders is autoscopic phenomena, which are defined as illusory own-body perceptions, during which patients experience the visual illusory reduplication of their own body in extra-personal space. Three main forms of autoscopic phenomena have been defined. During autoscopic hallucinations, a second own body is seen without any changes in bodily self-consciousness. During out-of-body experiences, the second own body is seen from an elevated perspective and location associated with disembodiment. During heautoscopy, subjects report strong self-identification with the second own body, often associated with the experience of existing at and perceiving the world from two places at the same time. Although it has been proposed that each autoscopic phenomenon is associated with different impairments of bodily self-consciousness, past research on neurological patients and the development of experimental paradigms for the study of bodily self-consciousness has focused on out-of-body experiences and the association with temporo-parietal cortex. Here, we performed quantitative lesion analysis in the—to date—largest group of patients with autoscopic hallucination and heautoscopy and compared the location of brain damage with those of control patients suffering from complex visual hallucinations. We found that heautoscopy was associated with lesions to the left posterior insula, and that autoscopic hallucinations were associated with damage to the right occipital cortex. Autoscopic hallucination and heautoscopy were further associated with distinct symptoms and deficits. The present data suggest that the autoscopic hallucination is a visuo-somatosensory deficit implicating extrastriate cortex and is, despite the visual hallucination of the own body, not associated with major deficits in bodily self-consciousness. Based on the symptoms and deficits in patients with heautoscopy and the implication of the left posterior insula, we suggest that abnormal bodily self-consciousness during heautoscopy is caused by a breakdown of self-other discrimination regarding affective somatosensory experience due to a disintegration of visuo-somatosensory signals with emotional (and/or interoceptive) bodily signals. These brain mechanisms are distinct from those described for out-of-body experiences. The present data extend previous models of autoscopic phenomena and provide...
Brain mechanisms of autoscopic phenomena

Introduction

Autoscopic phenomena [from the Greek autos (self) and skopeo (looking at)] are dramatic illusory own-body perceptions and encompass a wide range of experiences involving the visual illusory reduplication of one’s own body in extrapersonal space. Three main forms of autoscopic phenomena have been defined, and these include out-of-body experiences (Blanket et al., 2004; Brandt et al., 2005; De Ridder et al., 2007; Heydrich et al., 2011), autoscopic hallucinations (Maillard et al., 2004; Zamboni et al., 2005; Blanke et al., 2008) and heautoscopy (Brugger et al., 1994, 2006; Tadokoro et al., 2006). Although autoscopic phenomena have been reported in various focal and generalized disorders of the CNS for a long time (Menninger-Lerchenthal, 1946), they have only recently been investigated with the modern tools of cognitive neuroscience and neurology (Easton et al., 2009; Bolognini et al., 2010).

During an out-of-body experience, the patient has the subjective feeling of being awake and experiences the ‘self’ or centre of awareness, as being located outside the physical body, at a somewhat elevated level (abnormal self-location). It is from this elevated extrapersonal location that the patient’s body and the world are perceived (abnormal first-person perspective) (Devinsky et al., 1994, 2001; Devinsky et al., 2006; Tadokoro et al., 2006). A recent lesion analysis of autoscopic hallucination and heautoscopy are less well understood. During autoscopic hallucination, patients experience seeing an image of their body in extrapersonal space as if they were looking into a mirror, while self-location, self-identification and the first-person perspective remain unaffected (Table 1) (Féré, 1891; Brugger, 2002). Autoscopic hallucinations are mostly of brief duration (for exception, see Zamboni et al., 2005), often accompanied by visual hallucinations or visual illusions, and associated with visual field deficits (Kölmell, 1985; Blanke and Mohr, 2005) that may be lateralized to the affected visual field (Kölmell, 1985). Moreover, patients often experience seeing their own face or the upper part of the trunk and only rarely their entire body (Blanke and Castillo, 2007).

Autoscopic hallucinations due to various neurological disorders such as migraine (Lippman, 1953) and focal epilepsy (Blanke et al., 2004; Maillard et al., 2004), as well as ischaemic and neoplastic brain damage of the occipital and/or occipito-parietal lobe

Table 1 Classification criteria for heautoscopy, out-of-body experience and autoscopic hallucinations, as well as lesion location suggested by previous case reports and small case series

<table>
<thead>
<tr>
<th></th>
<th>Autoscopic hallucination</th>
<th>Out-of-body experience</th>
<th>Heautoscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-location</strong></td>
<td>Centred at physical body, stable</td>
<td>Centred at illusory body, stable</td>
<td>Centred at physical and/or illusory body, unstable</td>
</tr>
<tr>
<td><strong>Self-identification</strong></td>
<td>With physical body</td>
<td>With illusory body</td>
<td>With physical and/or illusory body</td>
</tr>
<tr>
<td><strong>First-person perspective</strong></td>
<td>Centred at physical body, stable</td>
<td>Centred at illusory body, stable</td>
<td>Centred at physical and/or illusory body</td>
</tr>
<tr>
<td><strong>Second own body</strong></td>
<td>2D image of own body, often of the face and upper trunk</td>
<td>3D image of whole own body</td>
<td>3D image of whole own body</td>
</tr>
<tr>
<td>(autoscopic body)</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td><strong>Vividness/realism</strong></td>
<td>Bilateral, occipital, temporal</td>
<td>Right, temporal, parietal</td>
<td>Left, temporal, parietal</td>
</tr>
<tr>
<td><strong>Lesion location</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Menninger-Lerchenthal, 1961; Gruessner and Landis, 1991), aber-represents a vestibular disorder (Bonnier, 1905; Ionasescu, 1960; depression (Lukianowicz, 1958; Arenz, 2001). Functionally, many counts such as externalization of the ‘somatic ego’ (Lunn, 1970), schizophrenia (Lukianowicz, 1963) and typhoid fever (Fere, 1891; Menninger-Lerchenthal, 1946), m-

Blanke has also been linked to various neurological (Lippman, 1953; Lukianowicz, 1958; Devinsky et al., 1989; Brugger, 2002; Blanke and Mohr, 2005), which may even persist if the autoscopic body only partly reflects the patient’s visual bodily appearance (Brugger, 2002; Blanke and Mohr, 2005). It has been argued that such illusory self-identification may be related to the frequent report of echopraxia [e.g. the experienced imitation of the patient’s movements by the autoscopic body (Lukianowicz, 1958; Brugger et al., 2006)] or feelings of detachment from emotional and bodily processing concerning the patient’s physical body (Menninger-Lerchenthal, 1935; Lukianowicz, 1958; Devinsky et al., 1989; Brugger, 2002).

A further difference exists between heautoscopy with respect to out-of-body experiences and autoscopic hallucinations; patients with heautoscopy may report to experience existing at two places at the same time (bi-location), often associated with alternating or simultaneous self-locations and first-person perspectives at the physical and the autoscopic body (Sollier, 1903; Brugger et al., 1994; Brugger, 2002; Blanke et al., 2004). Heautoscopy has also been linked to various neurological (Lippman, 1953; Blanke et al., 2004) and psychiatric conditions (Lukianowicz, 1958, 1963). These include temporal lobe epilepsy (Devinsky et al., 1989; Brugger et al., 1994; Tadokoro et al., 2006), neoplasia originating in the insular cortex (Brugger et al., 2006), typhoid fever (Fere, 1891; Menninger-Lerchenthal, 1946), migraine (Lippman, 1953), schizophrenia (Lukianowicz, 1963) and depression (Lukianowicz, 1958; Arenz, 2001). Functionally, many hypotheses have been proposed, suggesting that heautoscopy represents a vestibular disorder (Bonnier, 1905; IONESCEA, 1960; Menninger-Lerchenthal, 1961; Grüssner and LANDIS, 1991), aberrant visual memory (Dewhurst and Pearson, 1955), dissociative disease (Devinsky et al., 1989) and descriptive psychological accounts such as externalization of the ‘somatic ego’ (Lunn, 1970), projection of suppressed desires (Fere, 1891) or pathological grief reaction (Menninger-Lerchenthal, 1935).

Recent models account for heautoscopy, autoscopic hallucinations and out-of-body experiences within a common model, proposing that heautoscopy is based on abnormal integration of multisensory signals in personal space (as mentioned earlier in the text) as well as extrapersonal space (of visuo-vestibular signals) (Blanke et al., 2004, 2008). However, we note that these accounts of heautoscopy and autoscopic hallucinations are almost entirely based on single case reports (Brugger et al., 1994, 2006; Arenz, 2001; Zamboni et al., 2005) or small case series (Fere, 1891; Devinsky et al., 1989; Blanke et al., 2004). Moreover, data regarding the exact lesion location of autoscopic hallucinations and heautoscopy are missing because, to date, no quantitative lesion analysis [e.g. lesion overlap, voxel-based lesion symptom mapping (Bates et al., 2003; Rorden et al., 2007)] has been carried out.

Major advances in lesion analysis have permitted us to analyse, with high spatial resolution, whether symptoms are associated with circumscribed brain regions. These approaches are based on statistical analysis at the group level and voxel-wise quantitative statistical analysis (Bates et al., 2003; Rorden et al., 2007; Ionta et al., 2011). Here, we performed quantitative lesion analysis using MRicron (http://www.sph.sc.edu/comd/rorden/mricron) (Rorden et al., 2007) and compared the distribution of brain lesions in the—to date—largest sample of patients with heautoscopy and with autoscopic hallucinations with those of control patients. This was combined with an in-depth analysis of several phenomenological aspects and neurological deficits in patients with heautoscopy and autoscopic hallucinations.

Based on earlier work (Blanke and Mohr, 2005) and differences in associated symptoms, we had three major predictions concerning brain damage. We hypothesized that autoscopic hallucinations and heautoscopy would be caused by damage to distinct brain regions (lesion overlap analysis). Moreover, given the strong alteration of bodily self-consciousness in heautoscopy (abnormal self-location, self-identification and first-person perspective), we predicted that brain damage in patients with heautoscopy will be significantly different from that in our control group of patients with complex visual hallucinations but preserved bodily self-consciousness, and affects regions in proximity to those recently described in abnormal states of bodily self-consciousness (Ionta et al., 2011). Finally, we hypothesized that the lesion overlap in patients with autoscopic hallucinations will not differ from that in a control group, as patients with autoscopic hallucinations and the control group both suffer from frequent visual symptoms and have preserved bodily self-consciousness.

Patients and methods

Patients

We included nine patients suffering from heautoscopy (mean age: 37.8 years, four female, all right-handed) and seven patients suffering from autoscopic hallucinations (mean age: 33.8 years, four female, all right-handed) due to circumscribed structural brain lesions and/or

(Maillard et al., 2004; Zamboni et al., 2005), have been reported. Based on the frequent association with visual field deficits and other visual hallucinations, it has been argued that autoscopic hallucinations are a visual disorder (Fere, 1891; Sollier, 1903; Menninger-Lerchenthal, 1935; Hubert and Ajuriaguerra, 1952), and several dysfunctional visual or vision-related mechanisms have been proposed: abnormal visual imagery (Coleman, 1934), hypnagogic visual hallucination (Lukianowicz, 1958), aberrant plasticity after cortical damage in the early visual cortex (Zamboni et al., 2005) or a release phenomenon (Devinsky et al., 1989). More recently, it has been proposed that autoscopic hallucinations are a disorder of multisensory integration in personal space (due to conflicting cortical signal integration from vision, proprioeception and touch) (Blanke et al., 2004; Maillard et al., 2004; Bolognini et al., 2010).

The third form of autoscopic phenomenon is heautoscopy and has been conceptualized as an intermediate form between autoscopic hallucinations and out-of-body experience. As in out-of-body experiences and autoscopic hallucinations, the patient with heautoscopy has the impression of seeing an image of his body in extrapersonal space. However, it is often difficult for the patient with heautoscopy to decide whether they are disembodied and whether the centre of conscious experience is localized within the physical body or the autoscopic body (Table 1) (Blanke et al., 2004). This is associated with strong self-identification and close affinity with the autoscopic body (Devinsky et al., 1989; Brugger, 2002; Blanke and Mohr, 2005), which may even persist if the autoscopic body only partly reflects the patient’s visual bodily appearance (Brugger, 2002; Blanke and Mohr, 2005). It has been argued that such illusory self-identification may be related to the frequent report of echopraxia [e.g. the experienced imitation of the patient’s movements by the autoscopic body (Lukianowicz, 1958; Brugger et al., 2006)] or feelings of detachment from emotional and bodily processing concerning the patient’s physical body (Menninger-Lerchenthal, 1935; Lukianowicz, 1958; Devinsky et al., 1989; Brugger, 2002).

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localized neural dysfunction due to focal epilepsy (Tables 2 and 3). Inclusion criteria were that heautoscopy or autoscopic hallucinations of neurological origin were caused by either focal brain damage (measured with MRI or CT) or a circumscribed zone of seizure onset (confirmed by intracranial EEG recording). Furthermore, we required the availability of a sufficient amount of detail about the autoscopic hallucinations or heautoscopy so that they could be classified with certainty. The patients were recruited at the Department of Neurology at Geneva University Hospital or from other clinical research groups, where the original neuroradiological data were available for analysis. Several of the patients have been reported previously by different authors in the form of case reports or small case series (Brugger et al., 1994, 2006; Maillard et al., 2006; Bolognini, 2004; Zamboni et al., 2010).

The control group consisted of 14 patients with complex visual hallucinations who were recruited during the same time period at the Geneva University Hospital (Supplementary Table 1). Complex visual hallucinations consisted of people and/or faces without disturbance of bodily self-consciousness (normal self-location, self-identification and first-person perspective) and were also due to circumscribed brain lesions.

**Classification of autoscopic phenomena**

Based on the criteria used previously (Brugger, 2002; Blanke and Mohr, 2005), we classified cases as having heautoscopy or autoscopic hallucinations based on the available data concerning the first-person perspective (e.g. from where the patients reported to perceive the world), self-location (e.g. the location in space where the patients experience to be) and self-identification (e.g. the degree to which the patients identify with a body).

**Phenomenology and associated symptoms**

We assessed the phenomenology of heautoscopy and autoscopic hallucinations and, if reported, the presence of mirror-reversal of the autoscopic body and scene, as well as echopraxia. We further analysed the associated symptoms, such as visceral sensations (nausea, vomiting, palpitations), vestibular sensations (rotation, sensation of falling or flying, lightness and heaviness), visual field deficits and simple visual hallucinations (e.g. colours, light flashes), somatosensory deficits and associated emotions. Results of an extensive neuropsychological examination were also analysed (Blanke et al., 2004).

**Lesion mapping and spatial normalization**

Brain pathology was confirmed using a multimodality imaging approach relying on a combination of MRI ($n = 28$, 93%), CT ($n = 4$, 13%), ictal and interictal scalp EEG ($n = 14$, 46%), intracranial EEG using subdural electrodes ($n = 2$, 6%), PET ($n = 8$, 26%), ictal and/or interictal single-photon emission computed tomography ($n = 4$, 13%) and/or intracranial electric stimulation ($n = 3$, 10%, Tables 1 and 2 and Supplementary Table 1) (Knowlton, 2004; Kurián et al., 2007). MRI brain scans were normalized to the smoothed T1 template using SPM5 (http://www.fil.ion.ucl.ac.uk/spm/software/spm5) (Ashburner and Friston, 2005). As unified segmentation models give

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**Table 2 Patient characteristics in patients with heautoscopy**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Lesion site</th>
<th>Lesion side</th>
<th>Lesion analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAS 1</td>
<td>Epilepsy (dysplasial tumour)</td>
<td>Temporal lobe, insula</td>
<td>Left</td>
<td>MRI, EEG, PET</td>
</tr>
<tr>
<td>HAS 2</td>
<td>Epilepsy</td>
<td>Temporal lobe, insula</td>
<td>Left</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>HAS 3</td>
<td>Epilepsy (dysplasial tumour)</td>
<td>Temporal lobe, mesio-basal</td>
<td>Left</td>
<td>MRI, EEG, PET</td>
</tr>
<tr>
<td>HAS 4</td>
<td>Epilepsy (focal dysplasia, after resection)</td>
<td>Temporo-parietal lobe, insula</td>
<td>Left</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>HAS 5</td>
<td>Migraine (atrophy)</td>
<td>Parieto-occipital lobe</td>
<td>Bilateral</td>
<td>MRI</td>
</tr>
<tr>
<td>HAS 6</td>
<td>Epilepsy (lesional)</td>
<td>Insula and tempo-parieto-occipital lobe</td>
<td>Left</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>HAS 7</td>
<td>Epilepsy (astrocytoma)</td>
<td>Temporal lobe, insula</td>
<td>Right</td>
<td>CT, MRI, EEG, PET</td>
</tr>
<tr>
<td>HAS 8</td>
<td>Epilepsy (astrocytoma)</td>
<td>Temporal lobe, insula</td>
<td>Left</td>
<td>CT, EEG</td>
</tr>
<tr>
<td>HAS 9</td>
<td>Epilepsy (hippocampal sclerosis)</td>
<td>Temporal lobe, mesial</td>
<td>Left</td>
<td>MRI, SPECT</td>
</tr>
</tbody>
</table>

* Enough imaging data were available for accurate tracing onto a normalized standard template brain. No normalization of the original data was possible in these cases.

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**Table 3 Patient characteristics in patients with autoscopic hallucination**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Lesion site</th>
<th>Lesion side</th>
<th>Lesion analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>AH 1</td>
<td>Epilepsy (glioblastoma)</td>
<td>Parieto-occipital lobe</td>
<td>Left</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>AH 2</td>
<td>Epilepsy (focal dysplasia)</td>
<td>Parietal lobe</td>
<td>Right</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>AH 3</td>
<td>Ischaemic lesion (eclampsia)</td>
<td>Occipital lobe</td>
<td>Right</td>
<td>MRI</td>
</tr>
<tr>
<td>AH 4</td>
<td>Epilepsy (parasitical lesion)</td>
<td>Occipital lobe</td>
<td>Right</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>AH 5</td>
<td>Epilepsy (intracerebral haematomata)</td>
<td>Parieto-occipital lobe</td>
<td>Right</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>AH 6</td>
<td>Epilepsy (oligodendrogloma)</td>
<td>Occipital lobe</td>
<td>Right</td>
<td>MRI, EEG</td>
</tr>
<tr>
<td>AH 7</td>
<td>Tumour (postoperative lesion)</td>
<td>Occipital lobe</td>
<td>Right</td>
<td>MRI</td>
</tr>
</tbody>
</table>

* Enough imaging data were available for accurate tracing onto a normalized standard template brain. No normalization of the original data was possible in these cases.
the most precise registration of lesioned structural images (Crinion et al., 2007), no cost-function masking was necessary. Functional imaging (PET, single-photon emission computed tomography) was normalized using SPM5 and co-registered to the normalized MRI scans. Intracranial electrodes were co-registered to the normalized MRI scans for each patient using the Cartool software developed by Denis Brunet (http://brainmapping.unige.ch/Cartool.htm). Lesions were subsequently traced manually, slice by slice, either on the individual normalized brain scans or on the T1-weighted images using MRicron (Rorden et al., 2007). The manual tracing on the template brain was only done when confidence could be achieved for matching corresponding slices between the lesioned brain and the template brain. Thus, structural lesions were analysed by MRI, and if MRI was not available, by CT.

In a few patients, intracranial electrical stimulation and intracranial recordings were available and used to localize the seizure onset zone (Patient HAS 2 and Controls 1, 2 and 11). In this group of patients, the lesion site was defined as the location of the implanted electrodes (on the standard T1 template) where the seizure onset was found (plus an additional radius of 10 mm around the ictal onset zone). No patients with unclear lesion boundaries, generalized seizures or metallic artefacts were included in the analysis. Lesion volumes (volume of interest) were determined as the sum of all voxels compromising the traced lesion in all slices and were spatially smoothed using a 5-mm full-width at half-maximum Gaussian kernel and a threshold of 0.5.

**Lesion overlap and statistical analysis**

For lesion overlap and statistical analysis, we used MRicron and non-parametric mapping, which is part of the MRicron software package (Rorden et al., 2007). In a first step, simple voxel-based lesion overlap analysis establishing the anatomical subregions of maximal lesion overlap for heautoscopy and autoscopic hallucinations was performed. In a second step, non-parametric voxel-based lesion symptom mapping analysis (Bates et al., 2003), contrasting autoscopic hallucinations and heautoscopy against the control group, was performed on the hemisphere that was significantly more often affected (as confirmed by the binomial test, see later in the text). The control group was matched for the hemispheric predominance and the cerebral vascular territories, as defined by the lesion overlap (e.g. left anterior for heautoscopy and right posterior for autoscopic hallucinations). We used the Liebermeister test and corrected the results for multiple comparisons using a 5% false discovery rate (FDR). The Liebermeister test is a non-parametric implementation of a two-group comparison on a binary variable. It is more appropriate than the $\chi^2$ test (Rorden et al., 2007). We only included voxels affected in at least 30% for all subsequent analyses. Right versus left hemispheric involvement was tested with a binomial distribution, with an expected frequency of 0.5.

The distribution of phenomenological data and associated symptoms and neurological findings was analysed using the $\chi^2$ test and the Fisher exact test, respectively (Blanke and Mohr, 2005).

**Results**

**Phenomenology**

For illustration, several characteristic clinical, phenomenological and neuroradiological findings are described for two patients with heautoscopy and two patients with autoscopic hallucinations. Details for the remaining patients are in the online Supplementary material.

**Heautoscopy**

**Patient 1**

Patient 1 was a 44-year-old right-handed man known for pharmaco-resistant epilepsy and complex partial seizures. Neurological examination was normal. Interictal EEG showed slow waves with spikes over the left anterior and medial temporal region. Ictal EEG showed a seizure onset in the left anterior and medial temporal lobe. CT and MRI revealed a cystic lesion in the left temporal lobe, including parts of the left insula, enhancing contrast medium in its posterior parts. A dysembryoblastic tumour was diagnosed (see Fig. 1A for individual lesion analysis).

The episodes always started with an epigastric aura and a sensation of intense fear. He then saw a man in his right visual field. Although vision was blurred, the patient could tell that this man was very familiar to him. The man spoke in an incomprehensible way, while the patient (according to his relatives) suffered from language problems at these moments. The patient reported that he increasingly felt that the man he was seeing was himself. He felt as if he was ‘duplicated’ (abnormal self-identification). During the full episode, the patient did not experience abnormal self-location and always experienced to perceive the world and the autoscopic body from the normal first-person perspective. Postictally, the patient was depressed and often cried.

**Patient 2**

Patient 2 was a right-handed 15-year-old girl suffering from pharmaco-resistant epilepsy. During invasive presurgical evaluation, the seizure onset was localized to the left medial temporal lobe, followed by rapid spread of the ictal activity to the left insula. MRI revealed a left hippocampal sclerosis. Postictal neuropsychological testing revealed a discrete deficit in verbal memory (see Fig. 1B for individual lesion analysis).

The initial ictal sensation was characterized by an ascending epigastric sensation, nausea and the urge to vomit. The patient further mentioned a generalized feeling of extreme warmth (as if her body was burning) and that she was not able to breathe (as if someone was trying to strangle her). This was followed by the visual impression that a transparent body was leaving her body. The patient indicated that she felt that this body was her ‘soul’ leaving her body and that she could actually see a white, but transparent, body above her. The autoscopic body (her ‘soul’) was described as looking like the patient; in particular, she mentioned that she could clearly recognize the face and the upper parts of the trunk. Despite the highly realistic nature of these experiences, the patient remained critical of them and was aware that she was lying in the hospital bed. Towards the end of the seizure, the patient reported to feel that ‘the soul’ re-entered the body. The patient was agitated throughout the entire episode. To summarize, the patient reported strong self-identification with the physical as well as the autoscopic body, but did not experience any changes of self-location and the first-person perspective.
Autoscopic hallucinations

Patient 1

The patient was a right-handed 30-year-old man who suffered from complex partial seizures due to a glioblastoma in the left parietal lobe. The clinical examination was normal. The interictal EEG revealed a slowing over the left parietal lobe without any epileptiform activity. After resection of the tumour, no further seizures were noted.

During one episode, the patient saw his autoscopic body standing on top of a taxi for 10 s. The autoscopic body appeared as if observing the scenery. There was no change of the first-person perspective (e.g. the patient did not see the scene from the taxi), no sensation of disembodiment and no affinity with the autoscopic body (normal self-location and self-identification). The episodes were initially characterized by the sensation of losing balance, together with palpitations and a weakness of the right arm.

Patient 2

The patient (Maillard et al., 2004) was a right-handed 36-year-old woman known for intractable partial epilepsy. Seizures occurred weekly and included motor automatisms and tonic posturing of the trunk and upper and lower limbs bilaterally. She further described three kinds of initial ictal symptoms: palinopsia (persistence of an image of an object that she had actually seen a few seconds before), macroasomatognosia (sensation of inflation of the nose, head and sometimes whole body) and autoscop y. The interictal EEG showed subcontinuous right parieto-central paroxysms, and the ictal EEG (associated with the autoscopic hallucinations) showed epileptic discharge over the right parieto-central area. MRI showed focal cortical thickening and subcortical increase in FLAIR signal in the right inferior parietal gyrus, consistent with the diagnosis of focal dysplasia.

The patient described seeing the image of her face and her chest (sometimes her whole body) as in a mirror. The autoscopic body had a vague oval contour, was of normal size and colour and showed no particular expression. Self-location, the first-person perspective and self-identification remained normal.

Summary of heautoscopy autoscopic hallucinations and heautoscopy

All patients with heautoscopy reported a strong affinity and self-identification with the autoscopic body (significantly different from autoscopic hallucinations, $P < 0.01$; see later in the text). The autoscopic body was seen in all cases, not only in front view but
also in side and back views ($P < 0.01$). Five patients with heautoscopy reported alterations of the direction and the position of the first-person perspective (55%, $P = 0.029$). Only two patients with heautoscopy reported bi-location (22%, not significant). None of the patients reported to see the autoscopic body in a mirror-reversed way ($P = 0.01$), and three patients reported echopraxia (33%, not significant). Fig. 2 summarizes the phenomenological characteristics of the patients with heautoscopy and autoscopic hallucinations.

None of the patients with autoscopic hallucinations reported abnormal self-location, first-person perspective or self-identification; self-location and the origin of the first-person perspective as well as self-identification were always centred in the physical body. Patients described the autoscopic body as a mere visual body or a mirror reflection without experiencing any particular affinity (normal self-identification). The autoscopic body was usually seen in front of them (85%) and in a mirror-reversed way (57%). Shared movement (echopraxia) was reported by one patient (14%). For more detailed statistical results, see the online Supplementary material.

**Control group**

The control group for heautoscopy consisted of eight patients (mean age: 31.5 years, four female, six right handed, two ambidextrous) suffering from complex visual hallucinations due to damage of the left temporal, temporo-parietal or frontal cortex (Supplementary Table 1). Hallucinations included seeing a shadowy person, children, persons moving back and forth, two female persons (daughter and wife) and faces. Another six patients (mean age: 53.3 years, two female, all right handed) suffering from damage to the right posterior parietal and/or right occipital cortex were used as a control group for the patients suffering from autoscopic hallucinations. The latter patients also suffered from complex visual hallucinations and all reported seeing people (e.g. daughter, little people and faces). None of the patients of the control groups reported any particular affinity or self-identification with the seen persons, or a change of the first-person perspective or self-location.

**Associated symptoms and neurological deficits**

Fig. 3 shows the associated symptoms and neurological deficits in the patients with heautoscopy and autoscopic hallucinations. Five patients with heautoscopy experienced strong emotional sensations (fear, pleasure, anger) with the autoscopic phenomenon (55%), whereas none of the patients with autoscopic hallucinations reported a particular emotional state ($P = 0.029$). Visceroceptive sensations (33%, not significant), vestibular sensations (55%, $P = 0.09$) and feelings of echopraxia (not significant) were more frequent (but not statistically significant) in the heautoscopy group as compared with the group with autoscopic hallucinations.

The neurological examination was abnormal in six patients with heautoscopy (67%) and in five patients with autoscopic hallucinations (72%, not significant), but differed in the type of deficit. Five patients with autoscopic hallucinations (72%) had a (mostly) contralesional visual field deficit or associated visual symptoms (and usually perceived the autoscopic image in the part of the visual field that was affected). Visual deficits were only found in two patients with heautoscopy (22%, $P = 0.05$). A sensorimotor deficit was present in five patients with heautoscopy (55%), but only one patient with autoscopic hallucinations (14%, $P = 0.09$). Neuropsychological testing yielded a deficit in five patients with heautoscopy (55%; including verbal memory and visuo-spatial...
deficits, frontal signs), whereas all patients with autoscopic hallucinations had a normal neuropsychological examination ($P = 0.02$). For more detailed statistical results, see Fig. 3 and the online Supplementary material.

### Lesion overlap

**Heautoscopy**

The left temporal lobe (superior, middle and inferior temporal gyrus), including mesial temporal lobe (amygdala, hippocampus), and/or the left insula were affected in seven patients with heautoscopy. Two patients had left temporo-parietal lesions (including the angular gyrus and postcentral gyrus). One patient with heautoscopy suffered from exclusive left parietal lobe damage, and in one patient, the right insula was affected. The left hemispheric predominance was confirmed by statistical analysis ($P = 0.03$, binomial test, two-tailed). Lesion overlap analysis highlighted the left posterior insula (centred on MNI coordinates $x = -40, y = 1, z = -10$), which was found to be involved in five out of eight patients with heautoscopy with left brain damage (Fig. 4).

**Figure 3** Associated symptoms in heautoscopy (HAS) and autoscopic hallucinations (AH). Asterisks indicate a significant difference between the two groups ($\chi^2$ test and Fisher exact test, respectively).

**Figure 4** Lesion overlap in heautoscopy. Lesion overlap analysis highlighted the left posterior insula (centred on MNI coordinates $x = -40, y = 1, z = -10$), which was found to be involved in five out of nine patients with heautoscopy. The number of overlapping lesions is illustrated by colour, from violet ($n = 2$) to yellow (maximal lesion overlap, $n = 5$).
Autoscopic hallucinations

In patients with autoscopic hallucination (n = 7), the right hemisphere was affected in six patients, and the left hemisphere only in one patient (P = 0.12, binomial test, two tailed). The occipital lobe was affected in five patients with autoscopic hallucinations, the parietal lobe in one patient and the parieto-occipital lobe in two patients. The lesion overlap map highlighted a subregion in the right occipital lobe, more specifically the right superior occipital gyrus and the right cuneus (centred on MNI coordinates x = 20, y = −84, z = 20), as the area involved in five patients with autoscopic hallucinations. The number of overlapping lesions is illustrated by colour, from violet (n = 2) to red (maximal lesion overlap, n = 5). (B) 3D rendering of the lesion overlap in patients with autoscopic hallucinations.

**Discussion**

Here we demonstrate phenomenological differences as well as distinct neuroanatomical substrates for heautoscopy and autoscopic hallucinations. Heautoscopy was characterized by a strong disturbance of bodily self-consciousness, including altered self-identification and emotional changes and affinity with the autoscopic body that were frequently associated with changes of the first-person perspective and self-location. Moreover, our analysis associated abnormal vestibular sensations, neuropsychological deficits and contralesional sensorimotor, but not visual, deficits with heautoscopy. This was different during autoscopic hallucinations. Self-identification, self-location and the first-person perspective remained centred at the physical body and the pseudo-hallucinatory autoscopic body was often experienced in a mirror-reversed way, and frequently seen on the side of the contralesional visual field deficit. Autoscopic hallucinations were not associated with neuropsychological or sensorimotor deficits. Using state-of-the-art lesion symptom mapping techniques in the—to date—largest sample of patients suffering from heautoscopy and autoscopic hallucinations, we were able to demonstrate distinct neuroanatomical substrates for both autoscopic phenomena: heautoscopy was linked to the left posterior insula and adjacent cortical regions, whereas autoscopic hallucinations were not significantly different from the control group with complex visual hallucinations, which were due to lesion to the right parietal or occipital cortex (Z-score = 2.18, P > 0.05, corrected for FDR).

**Statistical lesion analysis**

These results were corroborated and extended by statistical lesion overlap comparison (non-parametric mapping) (Rorden et al., 2007). Lesion overlap contrast yielded maximal involvement of the left posterior insula (centred on MNI coordinates x = −40, y = 2, z = −11; Z-score = 3.31, P < 0.01, corrected for FDR) for heautoscopy as compared with the control group (Fig. 6). Autoscopic hallucinations did not significantly differ from the control group with complex visual hallucinations, which were due to lesion to the right parietal or occipital cortex (Z-score = 2.18, P > 0.05, corrected for FDR).

**Figure 5** Lesion overlap in autoscopic hallucinations. (A) The lesion overlap map highlighted a subregion of voxels in the right occipital lobe, more specifically the right superior occipital gyrus and the right cuneus (centred on MNI coordinates x = 20, y = −84, z = 20), as the area involved in five patients with autoscopic hallucinations. The number of overlapping lesions is illustrated by colour, from violet (n = 2) to red (maximal lesion overlap, n = 5). (B) 3D rendering of the lesion overlap in patients with autoscopic hallucinations.

**Figure 6** Voxel-based lesion symptom mapping in heautoscopy. Lesion overlap contrast yielded maximal involvement of the left posterior insula (centred on MNI coordinates x = −40, y = 2, z = −11; Z-score = 3.31, P < 0.01, corrected for FDR) for heautoscopy as compared with the control group. Only significant voxels are displayed.
associated with damage to the occipital cortex. Later in the text, we discuss the relevance of our findings in the context of the existing models for autoscopic phenomena and recent findings from cognitive neuroscience and neurology on body representation and bodily self-consciousness. We next discuss autoscopic hallucinations and then focus on heautoscopy and the role of the insular cortex as a multisensory integration area, comparing the present findings with the recent implication of the right temporo-parietal junction in bodily self-consciousness and out-of-body experiences (Ionta et al., 2011).

The present data show that autoscopic hallucinations are associated with visual deficits and caused by damage to the right superior occipital gyrus and the right cuneus in extrastriate visual cortex. This location of brain damage was similar to that in the control group, compatible with the known implication of extrastriate visual cortex in other complex visual hallucinations (Cogan, 1973; Manford and Andermann, 1998). Because complex visual hallucinations may be restricted to the affected visual hemifield and because this was observed in the present patients with autoscopic hallucinations, we suggest that autoscopic hallucinations are due to damage in the extrastriate visual cortex. This damage likely includes visual body perception regions such as the extrastriate body area (Downing et al., 2001; Astafiev et al., 2004), the fusiform form body area (Peelen and Downing, 2005) and the fusiform face area (Kanwisher et al., 1997), although this has not been directly tested in the present study. All regions have been linked to the perception and recognition of the human body, body parts and faces. Importantly, the right fusiform face area and fusiform body area also respond to one’s own face (Uddin et al., 2005) and one’s own body (Hodzic et al., 2009). The extrastriate body area has also been shown to respond to sensorimotor signals, compatible with a role of these regions beyond mere visual processing (Astafiev et al., 2004). However, we note that most of the lesions in the patients with autoscopic hallucinations were within the occipital cortex and that the maximal lesion overlap was more dorsal and posterior compared with the right fusiform body area and fusiform face area (Kanwisher et al., 1997; Peelen and Downing, 2005), as well as the right extrastriate body area (Downing et al., 2001). Accordingly, we cannot exclude that autoscopic hallucinations have resulted from interference with lower-level visual regions. As all three aspects of bodily self-consciousness were normal in patients with autoscopic hallucinations, the present data suggest that damage to the occipital cortex did not interfere with self-location, self-identification or the first-person perspective. As argued previously and extending related accounts of supernumerary phantom limbs (Ramachandran and Hirstein, 1998) and autoscopic hallucinations (Bolognini et al., 2010), we argue that autoscopic hallucinations and autoscopic (i.e. the seeing of one’s own body in extrapersonal space as is present during all autoscopic phenomena; Brugger et al., 1997; Brugger, 2002) are caused by disintegration between visual and somatosensory signals (Blanke et al., 2004; Blanke and Metzinger, 2009). Despite the inherent fascination and interest of the phenomenon for clinician and patient, the present data show that autoscopic hallucinations do not represent a disorder of bodily self-consciousness, as is the case in heautoscopy.

During heautoscopy, we found abnormal self-identification in all patients characterized by the experience of a strong emotional affinity towards and self-identification with the autoscopic body. Lesion overlap and statistical lesion analysis revealed that heautoscopy was caused by damage to or interference with the left posterior insula. The posterior insular cortex is a multisensory integration area, including somatosensory, motor, visual, auditory, vestibular and limbic signals (Augustine, 1996; Flynn, 1999). Schneider et al. (1993) observed large and often bilateral somatosensory receptive fields in the granular insular cortex. The posterior insula has also been implicated in disownership of body parts in neurological patients (e.g. somatoparaphrenia) (Baier and Karnath, 2008). Patients with somatoparaphrenia report the sensation that a certain body part, usually the left arm, is no longer their own, but belongs to another person (misattribution of a body part, abnormal self-identification) (Vallar and Ronchi, 2009). It has been suggested that the loss of ownership and the misattribution are a result of abnormal integration of sensorimotor and visual cues due to damage to the posterior insular cortex (Baier and Karnath, 2008). An implication of the insula in bodily self-consciousness is further supported by evidence from neuroimaging studies on manipulations of hand ownership and the related concept of agency through visuo-tactile and visuo-motor stimulations (Farrer et al., 2003; Tsakiris et al., 2007). These data and the present data on heautoscopy are compatible with the proposal that abnormal integration of somatosensory, visual and motor signals in the posterior insular cortex could result not only in misattribution of a body part (e.g. somatoparaphrenia, rubber hand illusion) but also in abnormal body ownership for a full body (e.g. self-identification with the autoscopic body).

However, a disintegration model based on somatosensory, motor and visual own-body signals as put forward for somatoparaphrenia (Baier and Karnath, 2008) and autoscopic hallucinations does not account for the observation that patients with heautoscopy experience a close emotional affinity towards the autoscopic body (Brugger et al., 1997) and the frequent association of heautoscopy with the sensation of detachment from own bodily processing (e.g. depersonalization) (Devinsky et al., 1989; Brugger et al., 1997) and viscerocceptive sensations (e.g. epigastric aura, vomiting, palpitation). We note that this aspect is critically absent in patients with autoscopic hallucinations and out-of-body experiences (Brugger et al., 1997; Blanke et al., 2004). Of relevance for heautoscopy, however, it has been suggested that the posterior insular cortex links somatosensory signals from the secondary somatosensory cortex with signals from limbic structures, such as the amygdala, the perirhinal cortex and the cingulate cortex (Friedman et al., 1986). This is supported by recent functional MRI work in humans by Ebisch et al. (2011), showing that activity in the left posterior insular cortex distinguished between the physical experience and observation of touch, but only if the touch was of affective significance (e.g. pleasant versus neutral touch). In line with these results, Morrison et al. (2011) found that activity in the posterior insular cortex is associated with both seeing and feeling pleasant touch. In addition, it has been suggested that activity in the insular cortex reflects abnormal perception of touch in the case of vision–touch synaesthesia (Blakemore et al., 2005), e.g. the case where the observation of another person
being touched is experienced as tactile stimulation on the equivalent part of one’s own body. Thus, the posterior insular cortex has been proposed not only to encode emotionally relevant somatosensory experience for both self and other, but also to distinguish whether an emotionally relevant somatosensory stimulus has been delivered to our body or to someone else’s body (Ebisch et al., 2011; Morrison et al., 2011).

Moreover, the posterior insular cortex has recently been implicated in the first-order cortical representation of pain and internal bodily states (viscerosensation), including homeostatic, gastrointestinal and cardiac signals (Augustine, 1996; Damasio et al., 2000; Craig, 2002, 2009). The further processing of this afferent viscerovisceral–autonomic information and the integration with limbic processing in the (anterior) insular cortex (together with the anterior cingulate cortex) are thought to be of crucial importance for emotions, interoceptive awareness and self-awareness (Damasio et al., 2000; Craig, 2002; Critchley et al., 2004; Picard, 2010). Extending earlier theories of emotion (James, 1884; Lange, 1922), studies have recently suggested that the mapping of internal bodily states and emotional experience in the insular cortex is crucial for conscious feelings generally and human self-consciousness (Damasio et al., 2000; Craig, 2002, 2009; Damasio, 2003). With respect to the present data on patients with heautoscopy, recent studies using functional MRI have shown increased insular activity not only during the subjective experience of one’s own feelings and emotions but also when a familiar other is experiencing the same emotion (Singer et al., 2004). It has thus been argued that these shared networks for self and other may form the basis for emotional perspective taking and empathy (Singer et al., 2009). Our observation that heautoscopy after insula damage is frequently associated with heightened or altered emotional states and viscerosensory sensations, such as palpitations, epigastric aura or vomiting [although only found in 33% of the present patient sample, but see Sollier (1903) for a viscerosensory account of heautoscopy], may be related to interference with such brain representations. Based on these findings, we speculate that damage to the posterior insular cortex results in a breakdown of self–other discrimination regarding affective somatosensory experience due to a disintegration of somatosensory and visual signals with emotional (and/or interoceptive) own-body signals. We speculate that the appearance of the autoscopic body and the referral of self-generated emotional states and feelings to the autoscopic body are a consequence of this disintegration, leading to abnormal emotional affinity and abnormally strong self-identification with the autoscopic body.

Many patients with heautoscopy also suffer from abnormal self-location and first-person perspective such as alternating self-location and first-person perspective between the physical and the illusory body and sensation of bi-location. We argue that these changes are caused by additional abnormal integration of vestibular signals (as proposed previously by Grüsser and Landis, 1991; Blanke et al., 2004; and Blanke and Mohr, 2005) with other bodily signals. Our data suggest that the former disintegration (somatosensory–visual signals with emotional–interoceptive signals) is present in all patients with heautoscopy, whereas the vestibular disturbance was only found in about half of them. Previous work revealed that heautoscopy is frequently associated with vestibular disturbances (Blanke and Mohr, 2005) and was confirmed in the present study (95%). The posterior insular cortex in the right and left hemisphere is part of the ‘vestibular cortical network’, together with the temporoparietal junction, anterior parietal cortex and premotor cortex (Guldin and Grüsser, 1998; Lopez and Blanke, 2011). Other illusory own-body perceptions, such as out-of-body experiences (Blanke et al., 2004), the misattribution of body parts (Heydrich et al., 2010) and depersonalization (Sang et al., 2006), are also frequently associated with vestibular sensations and have been linked to the temporoparietal junction (Simeon et al., 2000; Blanke et al., 2004; Heydrich et al., 2010 and the posterior insular cortex (Landtblom et al., 2011). Blanke et al. (2004) proposed that abnormal integration of mainly otolithic vestibular signals with other bodily signals (from vision, proprioception, touch) results in the abnormal elevated self-location and first-person perspective, characteristic of out-of-body experiences. Moreover, links between the vestibular system and bodily self-consciousness have also been revealed experimentally. Thus, vestibular stimulation has been shown to alter body ownership and somatosensory processing, both in patients with somatoparaphrenia (Bisiach et al., 1991; Rode et al., 1992) and healthy participants (Lopez et al., 2008, 2010, 2012; Ferre et al., 2011). Thus, it has been suggested that vestibular processing might be a central aspect of body ownership and embodiment (Lenggenhager et al., 2008; Lopez et al., 2008). During heautoscopy, vestibular sensations are variable, often related to the semicircular canals, and less prominent as compared with out-of-body experiences. We argue that—although changes in self-location and the first-person perspective in heautoscopy are less prominent than those during out-of-body experiences—their more variable and dynamic character (and association with abnormal emotional–interoceptive signals) may be related to the sensation of bi-location that is present in heautoscopy, but absent in out-of-body experiences, the latter being characterized by a clear psychological separation between the autoscopic and the physical body.

Why was left, but not right, damage to the posterior insula associated with heautoscopy? A previous literature review without quantitative lesion analysis also linked the left temporoparietal cortex to heautoscopy (Blanke and Mohr, 2005). This lateralization is compatible with the presence of auditory verbal hallucinations in patients with heautoscopy that have been linked to the left hemisphere and the left temporoparietal cortex in particular (Hubl et al., 2004). Auditory verbal manifestations are generally absent in patients with autoscopic hallucinations and out-of-body experiences. We can currently only speculate why right posterior insula damage was not associated with heautoscopy. As suggested by Craig et al. (2009), there may be functional differences concerning self-processing in right versus left insular cortex. Such right versus left insula differences may also concern language (left) versus spatial (right) processing differences or vestibular processing differences (left, semicannals; right, otoliths) (Blanke, 2012). We also note that previous neuroimaging work in healthy subjects reported bilateral temporoparietal activations in experimentally induced changes in self-location and first-person perspective (Ionta et al., 2011). Future work is necessary to investigate right versus left
temporo-parietal activations (including the insula) with respect to emotional, vestibular, language and spatial processing.

In conclusion, we argue that heautoscopy is caused by damage to the left posterior insular cortex, leading to a disintegration of exteroceptive bodily signals (somatosensory, visual) with emotional and/or visceral corporeal signals. Such disintegration results in abnormal self-identification and heightened emotional affinity that patients with heautoscopy experience for the autoscopic body. Projecting self-generated emotional states and feelings onto the autoscopic body, while also experiencing detachment of emotional sensations and somatosensory processing for the own body (e.g. inner hollowness and depersonalization [Brugger, 2002]), is thus the fundamental pathomechanism in heautoscopy and is accompanied with the appearance of not just a seen second own body, but a ‘true’ double, often experienced as another self. Such emotional–somatosensory disintegration may lead to levels of self-identification that are elevated for both the physical and the autoscopic body, making self–other distinction and self-location ambiguous. If accompanied by additional abnormal vestibular signals, further changes in first-person perspective and self-location may result, leading to bi-location and the sensation of self-duplication, likely the strongest form of heautoscopic dissociation. Neurological and neuropsychological symptoms and lesion location differed in autoscopic hallucinations, highlighting visual and visuo-somatosensory mechanisms in extrastriate and occipital cortex. Given the normality of bodily self-consciousness during autoscopic hallucinations, we speculate that autoscopic hallucinations are a disorder of own-body representation due to visual–tactile disintegration caused by damage to the ventral visual pathways in proximity to the extrastriate body area, fusiform body area or fusiform face area.

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Supplementary material

Supplementary material is available at Brain online.

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