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Chapter 22

Illusory perceptions of the human body and self

OLAF BLANKE[1,2,*], SHAHAR ARZY[1,2], AND THEODOR LANDIS[2]

[1]*Laboratory of Cognitive Neuroscience, Brain Mind Institute, Ecole Polytechnique Fédérale de Lausanne (EPFL), 1015 Lausanne, Switzerland*

[2]*Department of Neurology, University Hospital, 1211 Geneva, Switzerland*

For my part, when I enter most intimately into what I call myself, I always tumble, on some particular perception or other, of heat or cold, light or shade, love or hatred, pain or pleasure. I can never catch myself at any time without a perception, and never can observe anything but the perception.

David Hume (1711–1776)

22.1. Doubles

Illusory reduplication of the patient's own body refer to complex manifestations during which human subjects experience a second own body or self in their environment. Here we refer to this illusory second own body or self as a double. Doubles may be seen, felt, or heard, may be multiple or even concern the inner organs of the patient. Doubles have fascinated mankind from time immemorial and – often under the term autoscopic phenomena – several distinct forms have been described that can be separated based on phenomenological, functional, and anatomical criteria. The main forms of doubles are the visual own-body reduplications: autoscopic hallucination (AH), heautoscopy (HAS), and out-of-body experience (OBE) as well as the rarer forms including polyopic heautoscopy and inner heautoscopy. These are referred to here as visual doubles. Other own body reduplications include feeling of a presence (sensorimotor doubles), hearing of a presence (auditory doubles), and negative heautoscopy (negative doubles).

Doubles are abundant in folklore, mythology, and spiritual experiences (Rank, 1925; Menninger-Lerchenthal, 1946; Todd and Dewhurst, 1962; Sheils, 1978; Arzy et al., 2005; Metzinger, 2005). In more recent times, doubles became a frequent and popular topic in the romantic literary movement of the nineteenth century (Rank, 1925; Dewhurst and Pearson, 1955; McCulloch, 1992). Reflecting these popular trends, detailed case descriptions (Muldoon and Carrington, 1929; Yram, 1972; Alvarado, 1992) and medical reports (Du Prel, 1886; Féré, 1891; Sollier, 1903a) began to appear. Since then, doubles have been repeatedly described in patients suffering from neurological or psychiatric disease (Menninger-Lerchenthal, 1935; 1946; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Todd and Dewhurst, 1955; Lukianowicz, 1958; Leischner, 1961; Fredericks, 1969; Devinsky et al., 1989b; Grüsser and Landis, 1991; Dening and Berrios, 1994; Brugger et al., 1997). Doubles have been related to various neurological diseases such as epilepsy, migraine, neoplasia, infarction, and infection (Menninger-Lerchenthal, 1935; 1946; Lippman, 1953; Devinsky et al., 1989a; Grüsser and Landis, 1991; Dening and Berrios, 1994; Brugger et al., 1997; Podoll and Robinson, 1999) and psychiatric diseases such as schizophrenia, depression, anxiety, and dissociative disorders (Menninger-Lerchenthal, 1935; Lhermitte, 1939, Bychowski, 1943; Hécaen and Ajuriaguerra, 1952; Todd and Dewhurst, 1955; Lukianowicz, 1958; Dening and Berrios, 1994; Simeon, 2004; Bünning and Blanke, 2005; Mohr and Blanke, 2005).

Yet, despite this large number of observations of doubles in neurological disease they occupy a neglected position in neurobiology and behavioral neurology.

*Correspondence to: Olaf Blanke, MD, PhD, Laboratory of Cognitive Neuroscience, Brain–Mind Institute, Ecole Polytechnique Fédérale de Lausanne (EPFL), 1015 Lausanne, Switzerland. E-mail: olaf.blanke@epfl.ch, Tel: +41-21-6939621; Fax: +41-21-6939625.

In addition, given the rarity of these manifestations, the widespread neurological literature, and the complex phenomenology, doubles have only recently been investigated systematically. This is surprising when looking at the large number of studies investigating visual and nonvisual illusory reduplication of body parts such as phantom limbs (Ramachandran and Hirstein, 1998; Halligan, 2002), which has led to the neuroscientific investigation and description of many of the underlying neurocognitive mechanisms for body part reduplications. Importantly these latter findings have not only enhanced our understanding of phantom limbs, but have also improved our models of corporeal awareness and bodily processing (Ramachandran and Hirstein, 1998; Brugger et al., 2000; Halligan, 2002). The scientific value of a thorough understanding of visual illusory reduplication of the entire body can thus not be overstated given its potential importance in understanding the central mechanisms of corporeal awareness, embodiment, and self consciousness.

Here we will first review phenomenological, functional, and anatomical similarities and differences of the three main forms of visual reduplication: out-of-body experience, autoscopic hallucination, and heautoscopy. The separation into three distinct autoscopic phenomena was initially developed by Devinsky et al. (1989a) and subsequently extended by Grüsser and Landis (1991), Brugger and colleagues (Brugger et al., 1997; Brugger, 2002), and Blanke et al. (2004). These authors agreed that the combined classification of the well-known phenomenon of out-of-body experience with the less known phenomena of autoscopic hallucination and heautoscopy is important since during all three autoscopic phenomena the subject has the impression of seeing a second own body (or double) in extrapersonal space. It has been speculated that these phenomenological characteristics point to similar as well as distinct neurocognitive mechanisms in the different forms of autoscopic phenomena (Brugger et al., 1997; Blanke et al., 2004). Second, we will review the rarer forms of autoscopic phenomena including multiple visual doubles (polyopic heautoscopy) and inner visual doubles (inner heautoscopy). The description of these visual doubles is followed by a discussion of sensorimotor doubles (feeling of a presence), auditory doubles (hearing of a presence), and negative doubles (negative heautoscopy) due to neurological disease.

The feeling of a presence is defined as the convincing feeling that there is another person close by without actually seeing that person (Brugger et al., 1996; Blanke et al., 2003) and has been called previously "leibhafte Bewusstheit" (Jaspers, 1913), "hallucination du compagnon" (Lhermitte, 1939) or "feeling of a presence" (Brugger et al., 1996; Blanke et al., 2003). Although several patients with the feeling of a presence due to focal brain damage have been described (for review see Brugger et al., 1996), we do not consider the feeling of a presence as an autoscopic phenomenon because it is characterized by a nonvisual body reduplification as opposed to the three main forms of autoscopic phenomena which are all characterized by a visual body reduplication (see below; for alternative classifications of autoscopic phenomena see: Sollier, 1903a; Menninger-Lerchenthal, 1935; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Grüsser and Landis, 1991; Brugger et al., 1997).

22.2. Visual doubles

22.2.1. Out-of-body experience

During an out-of-body experience people seem to be awake and feel that their "self," or center of awareness, is located outside of the physical body and somewhat elevated. It is from this elevated extrapersonal location that the subjects experience seeing their body and the world (Blackmore, 1982; Irwin, 1985; Devinsky et al., 1989a; Brugger, 2002; Blanke et al., 2004). The subjects' reported perceptions are organized in such a way as to be consistent with this elevated visuospatial perspective. The following example from Lunn (1970, case 1) illustrates what individuals commonly experience during an out-of-body experience: "Suddenly it was as if he saw himself in the bed in front of him. He felt as if he were at the other end of the room, as if he were floating in space below the ceiling in the corner facing the bed from where he could observe his own body in the bed. [. . .] he saw his own completely immobile body in the bed; the eyes were closed."

An out-of-body experience can thus be defined as the presence of the following three phenomenological elements: the feeling of being outside one's physical body (disembodiment); the presence of a distanced and elevated visuospatial perspective; and the seeing of one's own body (autoscopy) from this elevated perspective. These three aspects are shown graphically in Fig. 22.2.

22.2.2. Autoscopic hallucination

During an autoscopic hallucination people experience seeing a double of themselves in extrapersonal space without the experience of leaving their body (no disembodiment). As compared to out-of-body experiences, individuals with autoscopic hallucination see the world from their habitual visuospatial perspective and experience their "self," or center of awareness inside their physical body (Fig. 22.2). The following example of

Case Study 22.1

Out-of-body experience
Blanke et al. (2002)

The present description of a patient with an OBE is interesting as it shows that OBEs can be induced by focal electrical stimulation of the human brain at the right temporoparietal junction (Fig. 22.1). In addition it highlighted shared functional and anatomical mechanisms between OBEs and other illusory own body perceptions such as illusory limb shortening and movement, as well as vestibular processing.

In this 43-year-old right-handed woman OBEs were induced by focal electrical stimulation of the junction of the right angular gyrus and the posterior superior temporal gyrus (Fig. 22.1). The patient underwent intracranial presurgical epilepsy evaluation for intractable seizures. Focal electrical stimulation at currents of 3.5 mA (for 2 seconds) induced OBEs that lasted for 2 seconds. OBEs were characterized by disembodiment, elevated visuospatial perspective, and autoscopy. During these OBEs the patient experienced that she was localized under the ceiling almost as if touching the ceiling with "her" back and looking down on her (autoscopic) body that was lying motionless on the bed. All stimulations at this current intensity were associated with an instantaneous feeling of "lightness" and "floating" about two meters above the bed. The elevated self was experienced as a complete body, although the patient was only sure about the presence of trunk, head, and shoulders. Repeated stimulations induced identical OBEs in the intrigued and surprised patient who had never experienced an OBE previously. With respect to the autoscopic body the patient reported that "I see myself lying in bed, from above, but I only see my legs and lower trunk" (i.e. negative HAS). In addition to seeing her body and the bed, the patient experienced seeing the present physicians and the table next to the bed. The visual experience was described as highly realistic and not dreamlike. Initial stimulations at the same site, but with smaller currents (2.0–3.0 mA), induced vestibular responses, in which the patient reported that she was "sinking into the bed" or that she has the impression of "falling from a height." Interestingly, if the patient was asked to look at certain parts of her body during focal electrical stimulation she experienced other illusory own body perceptions:if looking at her limbs that were stretched out during electrical stimulation (4.0–4.5 mA), she had the impression that the inspected body part was transformed, leading to the illusory visual perception of limb shortening. If the limbs were bent at the elbow or knee she reported that her legs appeared to be moving quickly towards her face, and took evasive action (4.0–5.0 mA). Finally, with closed eyes, the patient had neither an OBE nor visual body-part illusions but perceived her upper body as moving toward her legs (4.0–5.0 mA).

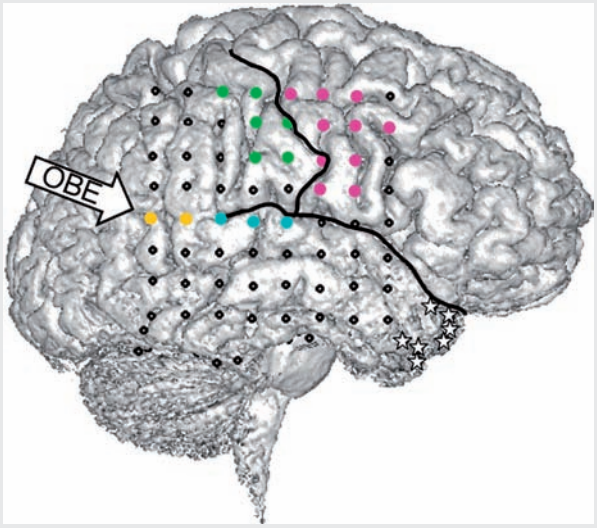

Fig. 22.1. Out-of-body experience induced by focal electrical stimulation. The out-of-body experience in this patient was induced by electrical stimulation of the right temporoparietal junction. The image shows a three-dimensional surface reconstruction of the right hemisphere of the brain from magnetic resonance imaging. Subdural electrodes (dots) were implanted in the brain of an epileptic patient undergoing presurgical evaluation (see Case Study 22.1). The locations at which focal electrical stimulation evoked behavioral responses are shown: magenta, motor responses; green, somatosensory responses; turquoise, auditory responses; yellow, site at which out-of body experiences, body part illusions and vestibular responses were induced (arrow). Stars indicate the location of the epileptic focus in the anterior and medial temporal lobe. Reprinted from Blanke et al. (2002) with permission by The Nature Publishing Group.

The patient suffered from complex partial seizures since the age of 32 years. Based on invasive presurgical epilepsy evaluation by subdural grid electrodes, the epileptic focus was located in the anterior and medial temporal lobe and thus ~5 cm anterior to the site that induced OBEs.

Case Study 22.2

Autoscopic hallucination
Zamboni et al. (2005)

The present case description of an autoscopic hallucination illustrates how much visual detail may be contained in the illusory image (of the autoscopic body) that patients experience to see. Somewhat atypically the autoscopic hallucination in this case was not paroxysmal, but persisted for several months allowing for the patient to describe the autoscopic body in great detail.

A 30-year-old, right-handed female reported seeing in a permanent fashion her own image as though she was looking into a mirror. Wherever she looked, this mirror image was always in front of her, at a distance of about one meter from her eyes. If a solid object was placed between the autoscopic image and the patient, she said that she can still see the image, but nearer to her, on the surface of the object. She described that the autoscopic image was transparent, yet somewhat blurred, setting "on a sheet of glass" resting against whatever object she was looking at. The image was life-sized and usually included head and shoulders, but could extend as far as the legs if the patient explored it by moving the gaze downward over the figure. It was always dressed exactly like the patient. Like a real mirror image, the autoscopic image or body replicated her bodily movements, in particular her face and arm movements. Interestingly, while one of the examiners put his hand on the patient's shoulder the patient reported that she could perceive something on the image's shoulder similar to a hand. The image disappeared when she closed her eyes. The autoscopic hallucination was not associated with an emotional state and the patient appeared somewhat indifferent to its presence and disappeared progressively after 3 months.

The patient suffered from hemorraghic infarction to the occipital poles extending to the right parieto-occipital junction (as demonstrated by magnetic resonance imaging) due to gestosis and eclampsia, three month before the above described hallucination. The neurological examination during the period of the autoscopic hallucination showed right lower limb weakness, left visuospatial hemineglect, optic ataxia, ocular apraxia, impaired depth perception, severe object agnosia, prosopagnosia, and alexia.

an autoscopic hallucination is taken from Kölmel (1985, case 6). ". . . the patient suddenly noticed a seated figure on the left. 'It wasn't hard to realize that it was I myself who was sitting there. I looked younger and fresher than I do now. My double smiled at me in a friendly way.'"

22.2.3. Heautoscopy

The third form of autoscopic phenomena is heautoscopy, which is an intermediate form between autoscopic hallucination and out-of-body experience. Individuals experiencing a heautoscopy also have the experience of seeing a double of themselves in extrapersonal space. However, it is difficult for the subject to decide whether he/she is disembodied or not and whether the self is localized within the physical body or in the autoscopic body (Blanke et al., 2004). In addition, the subjects often report seeing, in an alternating or simultaneous fashion, from different visuospatial perspectives (physical body, double's body) as reported by patient 2B in Blanke et al. (2004) (see Fig. 22.2). "[The patient] has the immediate impression as if she were seeing herself from behind herself. She felt as if she were 'standing at the foot of my bed and looking down at myself.' Yet, [. . .] the patient also has the impression of "seeing" from her physical [or bodily] visuospatial perspective, which looked at the wall immediately in front of her. Asked at which of these two positions she thinks herself to be, she answered that 'I am at both positions at the same time.'"

To summarize, the three forms of autoscopic phenomena differ with respect to the three phenomenological characteristics of disembodiment, visuospatial perspective, and autoscopy. Whereas there is no disembodiment in autoscopic hallucination and always disembodiment in out-of-body experiences, subjects with heautoscopy generally do not report clear disembodiment, but are often unable to localize their self. Thus, in some patients with heautoscopy the self is localized either in the physical body, or in the autoscopic body, and sometimes even at multiple positions. Accordingly, the visuospatial perspective is body-centered in autoscopic hallucination, extracorporeal in out-of-body experience, and at an extracorporeal and body-centered position in heautoscopy.

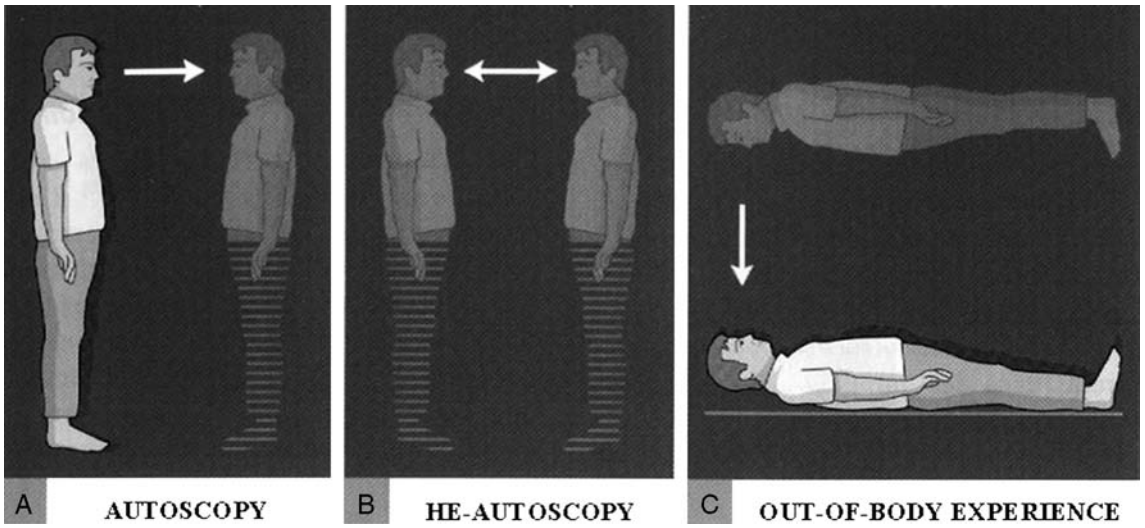

Fig. 22.2. Phenomenology of autoscopic phenomena a. Autoscopic hallucination: experience of seeing one's body in extracorporeal space (as a double) without disembodiment (experiencing the self as localized outside one's physical body boundaries). The double (right figure) is seen from the habitual egocentric visuospatial perspective (left figure). b. Heautoscopy: an intermediate form between autoscopic hallucination and out-of-body experience; the subject experiences seeing their body and the world in an alternating or simultaneous fashion both from an extracorporeal perspective and from their bodily visuospatial perspective; often it is difficult for the subject to decide whether the self is localized in the double or in their own body. c. Out-of-body experience: during an out-of-body experience the subject appears to "see" themselves (bottom figure) and the world from a location above their physical body (extracorporeal location and visuospatial perspective; top figure). The self is localized outside one's physical body (disembodiment). The directions of the subject's visuospatial perspective during the AP are indicated by the arrows (modified from Blanke, 2004).

Case Study 22.3

Heautoscopy
Brugger et al. (1994)

The following HAS case underlines that HAS is not only associated with a reduplication of the patients' body, but also by a reduplication of the self as these patients often cannot indicate in which of the two experienced bodies their self is localized and often claim to be localized at two positions simultaneously or in rapid alternation. Reduplication of the self is not present in AH and OBE.

A 21-year-old right-handed man woke up one morning and described the following experience. When he got up with a feeling of dizziness, he turned around and saw himself still lying in bed. He was angry about "this guy who I knew was myself and who would not get up and thus risked being late at work." He tried to wake up the body in the bed first by shouting at it then by trying to shake it and then by repeatedly jumping on the autoscopic body in the bed. His double did not show any reaction. Only then did the patient realized that he should be puzzled about his double and became more and more scared by the fact that he did not know any more who of the two bodies he really was (or where his self was located). This was especially due to the fact that he experienced his self-location to be alternating between the two bodies. Thus, several times he experienced being the one lying in bed and having the double look down on him from above the bed and even beating him. His only intention was described as trying to become one person again: standing next to the window (from where he could still see his other body lying in bed) he decided to jump out the window "in order to stop the intolerable feeling of being divided into two" hoping that "this desperate action would frighten the one in bed and thus urge him to merge with me again." The next thing he remembers is waking up in the hospital.

This patient was known for complex partial seizures since the age of 15 years due to a dysembryoblastic neuroepithelial tumor in the mediobasal part of the left temporal lobe. The neurological examination revealed diminished right-sided hand agility, a severe deficit in verbal memory, but not in visuospatial memory.

The impression of seeing one's own body is present in all autoscopic phenomena (for further details see Brugger et al., 1997; Blanke et al., 2004). Only during autoscopic hallucination does the subject immediately realize the hallucinatory nature of the experience, whereas heautoscopy and out-of-body experiences are generally described as highly realistic experiences (Brugger et al., 1994; Brugger, 2002; Blanke et al., 2004).

In a recent study Blanke and Mohr (2005) analyzed a larger number of neurological cases with out-of-body experiences, heautoscopy, and autoscopic hallucinations related to confirmed brain damage. These authors systematically analyzed 113 reported medical autoscopic phenomena cases from the English, German, French, and Italian literature and finally considered 41 cases with autoscopic phenomena (20 cases with autoscopic hallucination, 10 with heautoscopy, 11 with out-of-body experience) allowing a more detailed analysis of phenomenology (especially of the autoscopic body), associated neurological findings, etiology, and lesion site for out-of-body experience, autoscopic hallucination, and heautoscopy separately. With respect to phenomenology, these authors observed that a partially seen autoscopic body and its position in the visual field differed between the different forms of autoscopic phenomena. First, a partial autoscopic body was mostly experienced by patients with autoscopic hallucinations (63%) who always saw the upper part of the autoscopic body including head, neck, and upper trunk (while arms, legs, and lower trunk were missing). Second, in autoscopic hallucination the position of the autoscopic body in the visual field was frequently lateralized to the side of other visual hallucinations and hemianopia (Brugger et al., 1996), whereas the autoscopic body in heautoscopy and out-of-body experience was generally in the central visual field. Thus, these data do not agree with Green (1968) and Brugger et al. (1996) who observed a frequent lateralization of the autoscopic body also for out-of-body experiences. This might be due to several reasons. Green (1968) carried out her study in healthy subjects and we only investigated neurological patients with confirmed brain damage that was mostly unilateral. As Brugger et al. (1996) studied psychiatric and neurological patients, and also included neurological patients with nonfocal brain damage as well as patients without confirmed brain damage, differences in patient selection might explain the phenomenological differences between the different studies. Finally, the autoscopic body is seen as standing or sitting in autoscopic hallucination and heautoscopy, whereas it is in supine position in out-of-body experiences (Blanke et al., 2004). These positions were also found for the actual body position of the patient prior to the autoscopic phenomena suggesting that the position in which the patient experiences seeing the autoscopic body directly reflects the patient's own body position prior to and during out-of-body experience, heautoscopy, and autoscopic hallucination. A supine body position was also found by Green (1968) in 75% of her out-of-body experience subjects and, interestingly, most techniques that are used to voluntarily induce out-of-body experiences propose that subjects use a supine and relaxed position (Blackmore, 1982; Irwin, 1985). On the contrary, the data of Blanke and Mohr (2005) confirmed the mainly upright body position in patients with autoscopic hallucinations and heautoscopy as found by Dening and Berrios (1994).

Whereas the above described variables allow the differentiation of autoscopic hallucination from heautoscopy and out-of-body experience, the following five phenomenological characteristics of the autoscopic body allow distinguishing out-of-body experience and heautoscopy. First, whereas patients with out-of-body experiences and autoscopic hallucinations experience seeing the autoscopic body in front-view, patients with heautoscopy often see the autoscopic body in side- or back-views. Ionasescu's (1960, case 7) patient, who was a hairdresser experienced rotating around his customer (while cutting his hair) and then saw his autoscopic body from the side. Blanke et al.'s patient (2004, case 2b) saw herself from behind as did Devinsky et al.'s patient (1989a, case 9). Brugger et al. (1994) describe a patient who saw the autoscopic body in many different views. Second, this variability of views of the autoscopic body in heautoscopy is also reflected in the various motor actions that the latter is experienced performing. Thus, patients with heautoscopy report that the autoscopic body walks, runs, sits down, even shouts at the patient, and beats him with his fists (for a very vivid description of a patient's experience see Brugger et al., 1994). On the contrary, the autsocopic body during out-of-body experience and autoscopic hallucination does not move or act. Third, heautoscopy is often associated with the experience of sharing thoughts, words, or actions, which are less frequent in out-of-body experiences and autoscopic hallucinations. Indeed, patients with heautoscopy experience hearing the autoscopic body talk to them (Brugger et al., 1994) or that both bodies communicate by thought (Blanke et al., 2004, case 5). Others patients stated that the autoscopic body is performing the actions they were supposed to do (Devinsky et al., 1989a, case 9) or fights with other people that could be of potential danger to the patient (Blanke et al., 2004, case 5). Fourth, whereas the visuospatial perspective was unambiguously localized and experienced as unitary by all patients with autoscopic hallucinations and out-of-body experiences (as was

used to classify both phenomena), patients with heautoscopy frequently experience seeing from several different visuospatial perspectives (Brugger, 2002; Blanke et al., 2004). Indeed, patients 2b, 4, and 5 of Blanke et al. (2004) experience seeing from two different physical positions as did Brugger et al.'s patient (1994). Finally, patients with heautoscopy frequently report to "be split into two parts or selves" or feel as if "I were two persons" (Pearson and Dewhurst, 1954). Others reported that they were localized at two places at the same time (bilocation; Blanke et al., 2004, cases 2b, 5). In Brugger et al.'s patient (1994) bilocation occurred in rapid succession between the autoscopic and physical body and Lunn's patient (1970) describes himself (during heautoscopy) as a "split personality." The latter five variables of the autoscopic double (different views; actions; sharing of thoughts, words, or actions; multiple visuospatial perspectives; bilocation or splitting of the self) were all associated with heautoscopy. Thus, although out-of-body experience and heautoscopy share many associated hallucinations and some aspects of the autoscopic body, they differ in these latter five, more complex, variables suggesting that they are caused by different central mechanisms. These phenomenological difference are corroborated by functional and anatomical differences.

22.2.4. Clinical presentation

Although most of the aforementioned authors agree that autoscopic phenomena relate to a pathology of own body perception and/or corporeal awareness, it is not known which of the many involved corporeal senses are primarily involved in the generation of autoscopic phenomena and whether there are differences between the different forms of autoscopic phenomena. Whereas many authors have argued that autoscopic phenomena are due to a multisensory disturbance or disintegration, most authors have argued that autoscopic phenomena are caused by different sensory disturbances classifying autoscopic phenomena as visual disorders, proprioceptive disorders, vestibular disorders, or body schema disorders.

Some authors postulated a dysfunction of visual processing (Féré, 1891; Naudascher, 1910). Visual theories considered autoscopic phenomena to be visual or "specular" hallucinations based on the fact that they were experienced and described by most patients spontaneously as visual manifestations (Féré, 1891; Naudascher, 1910). In addition, especially autoscopic hallucinations may sometimes be lateralized in the visual field and are frequently experienced as visual pseudohallucinations (Brugger et al., 1997; Brugger, 2002; Blanke et al., 2004). However, a number of arguments show that a purely visual explanation cannot account for autoscopic phenomena in general. First, although all three forms of autoscopic phenomena are described spontaneously as visual, they are frequently experienced as veridical (especially heautoscopy and out-of-body experience) and not as pseudohallucinations (Menninger-Lerchenthal, 1935; 1946; Hécaen and Ajuriaguerra, 1952; Brugger et al., 1997; Blanke et al., 2004). Secondly, patients and healthy people reported that the impression of reality and self-recognition is preserved even if visual details of the autoscopic body during the autoscopic phenomena differ from the patient's actual appearance (such as clothes, age, haircut, size, coloring of the body (Sollier, 1903a; 1903b; Lhermitte, 1939; Lukianowicz, 1958; Crookall, 1964; Green, 1968; Irwin, 1985; Kölmel, 1985; for discussion see Blanke et al., 2004). In some patients, self-recognition may even be immediate if the patient only sees their back during the autoscopic phenomena (Devinsky et al., 1989a; Blanke et al., 2004). These data point to the importance of nonvisual, body-related, mechanisms in autoscopic phenomena, such as proprioceptive and/or kinaesthetic processing as already argued by Sollier (1903a; for later discussions see also Menninger-Lerchenthal, 1935; Lhermitte, 1939; Brugger et al., 1997; Blanke et al., 2004). In line with phenomenological differences, these authors proposed that the involvement of disturbed processing may differ between the different forms of autoscopic phenomena. Paul Sollier (1903a) for instance differentiated heautoscopy (or "autoscopie dissemblable") from autoscopic hallucination (or "autoscopie spéculaire") of previous authors such as Féré (1891) suggesting that both autoscopic phenomena forms might relate to different cerebral mechanisms. He postulated the latter to be a mere visual hallucination, whereas he assumed the former to be a proprioceptive–kinaesthetic disturbance associated with a strong psychological affinity between physical and autoscopic body. For proprioceptive–kinaesthetic processing he coined the term "cénesthesia" (as the body's visceral and deep sensations) stating that autoscopic hallucination and heautoscopy are due to different degrees of the "projection of the body's visceral and deep sensations in the space on the outside of the body" (Sollier, 1903a, pp. 34–44). Several authors have also highlighted the role of proprioception and kinesthesia in autoscopic phenomena by noting that some patients report shared movements between their physical and autoscopic body (autoscopic echopraxia; Menninger-Lerchenthal, 1935; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Lukianowicz, 1958; Brugger et al., 1997). Another sensory system, which has been linked to autoscopic phenomena, is the vestibular system that

conveys sensations of the body's orientation in three-dimensional space to the brain. Whereas Bonnier (1904) and Skworzoff (1931) noted the frequent association of vestibular sensations of either peripheral or central origin with autoscopic phenomena, others proposed that a central vestibular dysfunction might be an important mechanism for the actual generation of autoscopic phenomena (Menninger-Lerchenthal, 1935; 1946; Grüsser and Landis, 1991; Brugger et al., 1997). Menninger-Lerchenthal (1935) extended this view and pointed to the importance of vestibular disorders in the generation of visual illusions and visual dysfunctions, as well as autoscopic phenomena. Blanke et al. (2004) suggested, on clinical grounds, a differential implication of vestibular processing in the different forms of autoscopic phenomena. These authors suggested systematic differences in the strength of vestibular dysfunction in autoscopic hallucination, heautoscopy, and out-of-body experiences. The potential role of the vestibular system for autoscopic phenomena is also supported by descriptions of vestibular sensations during autoscopic phenomena in healthy populations (i.e. Crookall, 1964; Green, 1968; Yram, 1972; Blackmore, 1982; Irwin, 1985; Metzinger, 2003). Blanke et al. (2004) suggested that out-of-body experiences were associated with a gravitational (otholithic) vestibular disturbance, whereas the vestibular dysfunction in patients with heautoscopy was more variable and often characterized by rotational components, and vestibular dysfunction was absent in patients with AS. Finally, many patients with autoscopic phenomena also experience paroxysmal visual body-part illusions (Ehrenwald, 1931; Menninger-Lerchenthal, 1935; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Ionasescu, 1960; Lunn, 1970; Dening and Berrios, 1994) and this has led several authors to argue for a similar or closely related functional and anatomical origin of visual body part illusions and visual illusions of the entire body (Menninger-Lerchenthal, 1935; 1946; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Ionasescu, 1960; Brugger et al., 1997).

Recent findings from Blanke and Mohr (2005) suggest that different patterns of hallucinations and neurological deficits are associated with out-of-body experience, heautoscopy, and autoscopic hallucination arguing for different functional mechanisms in each form. Thus, vestibular hallucinations and body schema disturbances, as well as the absence of hemianopia were associated with out-of-body experiences and heautoscopy, whereas lateralized visual hallucinations and hemianopia without vestibular hallucinations and no body schema disturbances were associated with autoscopic hallucination. In addition, the visual hallucinations of patients with autoscopic hallucinations were lateralized to the side of hemianopia. Auditory hallucinations were mainly observed in patients with out-of-body experiences. Other manifestations such as tactile hallucinations, aphasia, and sensorimotor deficits were infrequent in all autoscopic phenomena. Based on this pattern of associated hallucinations and neurological deficits, Blanke and Mohr (2005) argued that it is possible to differentiate the mainly visual autoscopic hallucinations from out-of-body experience and heautoscopy confirming earlier case descriptions of autoscopic hallucination as a visual or "specular" hallucination or pseudohallucination by Féré (1891) and Paul Sollier (1903a). Next to a confirmation of Féré's earlier theory of visual mechanisms in autoscopic hallucination, Blanke and Mohr's (2005) analysis also provided evidence for a vestibular and body schema pathology. However, this was not found for all autoscopic phenomena, but specifically for heautoscopy and out-of-body experiences (Menninger-Lerchenthal, 1935; 1946; Brugger et al., 1997; Blanke et al., 2004).

22.2.5. Etiology

In comparison with the rich phenomenology of the abovementioned studies, much less information is available about the etiology and especially anatomy of autoscopic phenomena, which is partly due to the fact that many cases were reported in the first half of the twentieth century. With respect to etiology, autoscopic phenomena have been reported in various focal and generalized diseases of the central nervous system. Generalized neurological etiologies include cerebral infections such as meningitis and encephalitis, intoxications, as well as generalized epilepsies (Menninger-Lerchenthal, 1935; Lhermitte, 1939; Bychowski, 1943; Hécaen and Ajuriaguerra, 1952; Lukianowicz, 1958; Devinsky, 1989a; Dening and Berrios, 1994; Brugger et al., 1997; Blanke et al., 2004). Autoscopic phenomena following focal brain damage also emerge from a large variety of etiologies including focal epilepsy (Devinsky, 1989a), traumatic brain damage (Todd and Dewhurst, 1955), and migraine (Lippman, 1953), vascular brain damage (Kölmel, 1985), neoplasia (Todd and Dewhurst, 1955), dysembryoblastic neuroepithelial tumor (Blanke et al., 2004) and arteriovenous malformation (Devinsky et al., 1989a).

22.2.6. Anatomy

Regarding their underlying anatomy, autoscopic phenomena of focal origin primarily implicate posterior brain regions and with respect to lobar anatomy most studies found the temporal, parietal, or occipital lobe to be involved (Hécaen and Ajuriaguerra, 1952; Todd

and Dewhurst, 1955; Lunn, 1970; Devinsky et al., 1989a; Blanke et al., 2004; Blanke and Arzy, 2005). Some of these authors have either suggested a predominance of temporal lobe involvement (Devinsky et al., 1989a; Grüsser and Landis, 1991), a predominance of parietal lobe involvement (Menninger-Lerchenthal, 1935; 1946; Hécaen and Ajuriaguerra, 1952), or no brain localization at all (Lhermitte, 1939). Menninger-Lerchenthal (1935) even speculated on different anatomical substrates for the different autoscopic phenomena, suggesting that autoscopic hallucination originates at the junction of the parietal and occipital lobe (junction of Brodmann's areas 21 and 40), heautoscopy from the angular and supramarginal gyrus (Brodmann's areas 40 and 41), and out-of-body experiences from the superior parietal lobule (Brodmann's area 7). These anatomical dissociations have been partly confirmed by Blanke et al. (2004) showing that autoscopic phenomena might be related to damage to the temporoparietal junction (TPJ; Fig. 22.3).

Unfortunately, the small number of analyzed patients in this latter study did not allow lesion analysis for each of the three forms of autoscopic phenomena. With regard to predominant hemispheric involvement the reported data are quite divergent. Some authors found no hemispheric predominance for autoscopic phenomena (Hécaen and Ajuriaguerra, 1952; Fredericks, 1969; Devinsky et al., 1989a; Dening and Berrios, 1994), while others have suggested a right hemispheric predominance for autoscopic phenomena (Menninger-Lerchenthal, 1935; 1946; Grüsser and Landis, 1991; Brugger et al., 1997). For autoscopic phenomena in psychiatric disease see Bünning and Blanke (2005) and Mohr and Blanke (2005).

An analysis of 41 cases with autoscopic phenomena suggested that all three autoscopic phenomena may be due to either right or left hemispheric brain lesions (Blanke and Mohr, 2005) although there were differences with respect to primarily involved hemisphere and brain region. Out-of-body experiences were mostly due to right hemispheric brain damage (67%), whereas more frequent left hemispheric brain damage was found for patients with heautoscopy (67%). The fact that previous studies have analyzed the lesion location for all autoscopic phenomena together, might thus explain why some authors reported no hemispheric predominance (Hécaen and Ajuriaguerra, 1952; Fredericks, 1969; Devinsky et al., 1989a; Dening and Berrios, 1994). The data by Blanke and Mohr (2005) would point to a right hemispheric predominance for autoscopic hallucination and out-of-body experience, something suggested only for autoscopic hallucination by previous authors (Menninger-Lerchenthal, 1935; 1946; Grüsser and Landis, 1991; Brugger et al., 1997). Regarding the intrahemispheric lesion site of autoscopic phenomena a high predominance of temporal lobe involvement in all autoscopic phenomena (55–82%) was found by Blanke and Mohr (2005) corroborating older literature (Devinsky et al., 1989a; Grüsser and Landis, 1991; Dening and Berrios, 1994). The parietal lobe was also found frequently and equally often involved in all forms of autoscopic phenomena (45–55%; Blanke and Mohr, 2005). Only patients with autoscopic hallucinations had significantly more involvement of the occipital lobe concordant with the above described association with visual hallucinations and hemianopia. Occipital lobe involvement in autoscopic hallucinations was already suggested based on the fact of frequent bright coloring of the autoscopic body in autoscopic hallucinations that contrasted with the colorless, pale, and misty appearance of the autoscopic body in heautoscopy

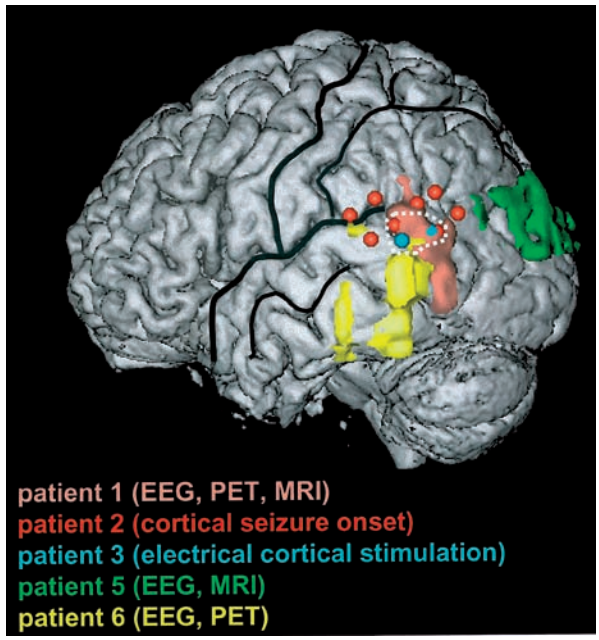

Fig. 22.3. Lesion location in patients with autoscopic phenomena. Autoscopic phenomena are linked to interference with the temporoparietal junction. The figure shows the results of lesion overlap analysis in the five patients with autoscopic phenomena from Blanke et al. (2004). Each patient is indicated in a separate color. The area of lesion overlap (patient 1,5,6), of intracranial seizure onset (patient 2), or of the site of electrical cortical stimulation (patient 3) of each patient is mapped onto the right hemisphere of Patient 6. Lesion overlap for all patients centred on the temporoparietal junction (area indicated by dashed white line). Thick black lines indicate the sylvian fissure and the central sulcus; thin lines indicate superior temporal sulcus, postcentral sulcus and intraparietal sulcus. Modified with permission from Blanke et al. (2004).

(Brugger et al., 1997). Based on this it might be suggested that patients with autoscopic hallucination might have more posterior brain damage in occipitoparietal and occipitotemporal cortex, whereas patients with heautoscopy and out-of-body experience have rather temporoparietal lesions including the TPJ.

22.2.7. Theoretical considerations

These data suggest that autoscopic phenomena may result from a disintegration in personal space (due to conflicting tactile, proprioceptive, kinesthetic, and visual information) and a second disintegration between personal and extrapersonal space (due to conflicting visual and vestibular information) (Blanke et al., 2004; Bünning and Blanke, 2005; Mohr and Blanke, 2005). These authors proposed that, while disintegration in personal space was present in all three forms of autoscopic phenomena, differences between the different forms of autoscopic phenomena were mainly due to differences in strength and type of the vestibular dysfunction. Indeed, Blanke et al. (2004) suggested that out-of-body experiences were associated with a strong vestibular disturbance, whereas heautoscopy were associated with a moderate and more variable vestibular disturbance and autoscopic hallucination only with a mild or even absent vestibular disturbance. The here reviewed phenomenological, neurological, and anatomical data suggest the importance of a vestibular dysfunction and body schema disturbance in heautoscopy and out-of-body experience and suggests that a vestibular dysfunction is absent or only weakly present in autoscopic hallucination. Moreover, the high frequency of visual hallucinations and of hemianopia in autoscopic hallucination suggests that deficient visual processing rather than vestibular processing might be the main causative factor for disintegration in personal space and/or extrapersonal space. This is also in agreement with the anatomical findings showing that autoscopic hallucination patients have significantly more occipital lobe involvement as compared to patients with heautoscopy or out-of-body experiences. The phenomenological differences between heautoscopy and out-of-body experience suggest that each form of autoscopic phenomena relies on different neurocognitive mechanisms. These more complex phenomenological differences were found despite the highly similar sensory hallucinations and neurological deficits that were associated with heautoscopy and out-of-body experience. Yet in contrast to out-of-body experiences, heautoscopy was associated with the presence of many different views of the autoscopic body, many actions, the sharing of thoughts, words, and agency, multiple visuospatial perspectives, and bilocation of the self. We therefore suggest that the association of greater phenomenological variability of the autoscopic body (with respect to views and actions) with the increased frequency of shared thoughts, voices, and agency between autoscopic and (the patient's) physical body (i.e. echopraxia) might be due to a greater (or more variable) implication of abnormal kinesthetic/proprioceptive information processing in heautoscopy. This is contrasted in out-of-body experiences by the silent and static autoscopic body, the disembodiment, the 180° inversion and the elevated and distanced visuospatial perspective of the self (with respect to the extracorporeal environment) that are probably related to vestibular disturbances (Blanke et al., 2004; Bünning and Blanke, 2005; Mohr and Blanke, 2005). Thus, it seems to the subject with an out-of-body experience that (1) their body position and visuospatial perspective is distanced (about 2–3 meters) and rotated (by 180°) with respect to the actual physical position (Fig. 22.2). In addition, during heautoscopy, the sharing of thoughts, voices, and agency might make it difficult for the patient to decide where the physical agent (Gallagher, 2000; Decety and Sommerville, 2003) is localized (i.e. in the physical body or in the autoscopic body). This is increased by two visuospatial perspectives that either alternate or are simultaneously present between autoscopic and physical body in heautoscopy. This situation makes it almost impossible for the heautoscopy patient to decide where the observing self is localized and might lead to the experience of two "observing" selves (Blanke et al., 2004, case 2b). It might thus be argued that heautoscopy is not only an experience characterized by the reduplication of one's body, but also by a reduplication of one's self. As strikingly reported by Brugger et al. (1994) the high risk of suicide during this terrifying experience cannot be overstated as some of these patients with heautoscopy try by all means to re-establish their unitary self.

22.3. Multiple visual doubles

22.3.1. Polyopic autoscopic phenomena

Polyopic autoscopic hallucination or polyopic heautoscopy is present when patients report seeing more than one autoscopic double in extracorporeal space, that is, a multiple rather than a single reduplication of one's own body. Probably the first account of polyopic heautoscopy is to be found in Müller's (1826) seminal work on visual hallucinations. Returning home late from work, this exhausted university professor suddenly found himself in front of 15 persons, all clearly recognized as doubles of himself although being of different ages and wearing different clothes he himself had used to wear in former times. A case of an autoscopic hallucination with

multiple optical images is reported by Roubinovitch (1893; quoted by Menninger-Lerchenthal, 1935). This author's patient saw three identical mirror images of himself which he compared to the reflections he would have seen standing in front of a mirror with three wings.

Other earlier autoscopic phenomena of polyopic nature can be found in Oesterreich (1910) and Leischner (1961). As analyzed recently by Brugger et al. (2006) polyopic autoscopic phenomena are characterized as follows. In a third of the cases autoscopic phenomena were characterized by either two or three doubles, but most often by a large number of doubles that, in some cases, filled up the entire room (Mayer-Gross, 1928) or the interior of the patient's body (Heintel, 1965). If polyopic autoscopic phenomena are characterized by a large number of doubles they are generally seen as quite

Case Study 22.4

Polyopic heautoscopy
Brugger et al. (2006)

This case highlights that HAS is not only associated with a single reduplication of the patient's body (as in autoscopic hallucination, heautoscopy, or out-of-body experience). Same patients may also experience seeing multiple autoscopic bodies with varying degrees of physical resemblance and psychological affinities.

A 41-year-old right-handed pottery maker woke up one night and noticed that he had split into three distinct parts: there was the left half of his body which felt quite normal, a right half which, physically and psychologically, felt detached from the left, and a man adjacent to his right side, which he felt to be a part of himself. It was as if he and the man were "sharing the same soul." This feeling was very convincing despite the fact that there was no similarity in physical appearance (for instance, the man was blond while the patient's hair is black). Puzzled by this, the patient began to walk up and down in his bedroom. As he did so, he at once discovered what he later referred to as "the family." He gives the following account: "When I walked around, I repeatedly looked towards the gentleman on my side and wondered if I could recognize his face. This was impossible since on looking towards the right side he also turned his head to the right. I could note however, that the man was blond and about 50 years old. All of a sudden, I noticed that, even more to the right, there was a whole group of people. At a distance of 2 meters I saw an approximately 50-year-old lady with blond braids. Still another 4 meters away, there were two girls [both approximately age 20] and some 20 meters from me, still in a straight line with all the other persons, there was a boy [unspecified age]. I knew right from the beginning that these persons were intimately linked with one another, they were father, mother, daughters and son." [In reality, the patient's wife was younger and had dark short hair. His only two children were two sons, aged 10 and 16]. The patient reported that, with the appearance of the "family," the gap between left and right halves of his body ceased to exist. He continued to feel a strong sense of belonging towards the man at his right side, which gradually expanded also to the woman and, to a lesser extent, to the girls. The boy was only vaguely seen and sometimes vanished in the darkness of the far right end of the bedroom. Notably, all "family" members imitated the patient's movements, yet the "daughters" and the "son" were also able to move on their own. The patient described: "When I walked, the family walked with me; when I bent my knees, the others bent theirs; when I looked to the right, so did all the others. The girls were talking to one another, and sometimes they would look towards me waving their hands as if inviting me to join their world... Naturally, I could not see the persons any longer on closing my eyes, but the feeling remained that, pieces of myself were located in those places I knew them to stay. It was a feeling of being awfully frittered away!" When his "real" wife was sitting at his right side, the "family" would temporarily vanish and he perceived himself to be one person in one place again. However, he noted a clumsiness and weakness of the entire right half of his body. As soon as his wife moved from his side, the imaginary persons would immediately reappear in their respective places. According to the patient's wife account, the patient's speech was barely understandable throughout the experience and contained many neologisms.

The patient suffered from partial seizures due to a left insular astrocytoma that extended into the adjacent frontotemporal cortex (as demonstrated by computed tomography). Neurological examination revealed a mild right-sided sensory hemisyndrome, logorrhea, an elevated mood with fluctuating denial of illness, and an isolated deficit in the recall of verbal material.

small in size (Dewhurst and Pearson, 1955), whereas the cases with a smaller number of doubles are mostly experienced as having the same size as the patient. Echopraxia (or sharing of action between the autoscopic bodies and the patient's body) was noted by two previously reported patients with autoscopic phenomena (Staudenmaier, 1912/1968; Lance 1976). The doubles are generally localized in the central visual field (lateralization in the visual field was only described by two patients: Dewhurst and Pearson, 1955; Ley and Stauder, 1950). If mentioned, the perceived distance of the double from the patient was generally very small and thus in the peripersonal or personal space as is the case in most patients with autoscopic phenomena. With respect to etiology about two thirds of the cases were of neurological origin, a third due to psychiatric disease (one case was reported during puerpurium). Of the neurological cases, the large majority were of focal origin either due to vascular infarction or focal epilepsy. In these focal neurological cases the lesion was localized as often in the right as in the left hemisphere.

The recent case reported by Brugger et al. (2006) corroborated the importance of nonvisual, body-related, mechanisms, also for polyopic heautoscopy (Sollier, 1903a; 1903b; Menninger-Lerchenthal, 1935; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Brugger et al., 1997; Blanke et al. 2004) by showing an alteration of own-body awareness (or depersonalization), detachment of parts or of the entire left half of the patient's body (or dyssomatognosia), and vestibular illusions. Interestingly, Brugger et al.'s patient report confirmed that self-recognition and self-identification, as well as psychological affinity between patient and double occurs even if visual details of the body's double differ from the patient's actual appearance. There were only few physical similarities between this patient and the closest double (Case Study 22.4). Yet, the patient felt a strong psychological affinity towards him, as if the double were a part of him, and as if they were to share thoughts and feelings. Interestingly, self-recognition, self-identification, and psychological affinity depended on the distance between the patient and the experienced location of the double. Thus, the doubles at greater distances (from the patients's body) were perceived initially as different people and not as part of his own body. Only later, once this patient experienced the doubles as a group as well as closer with respect to his body did he state that he experienced them as part of his self (Brugger et al., 2006). It might thus be suggested that the visuospatial characteristics of the experienced scene such as the distance between patient and double relates to such psychological processes as self-recognition, self-identification, and psychological affinity with the double. This relation might become especially evident in polyopic heautoscopy where multiple doubles with different characteristics are experienced simultaneously at different locations and distances from the patient's body.

What are the functional mechanisms leading to the perception of multiple visual doubles? Very little clinical information is currently available on this rare autoscopic phenomenon as is also the case for other multiple supernumerary body disturbances (Ehrenwald, 1930). Interestingly, the patient described by Brugger et al. (2006) initially observed only one single right-sided double, but discovered the other more distant right-sided doubles upon moving his eyes onto the closest double. This potential relationship between eye movements and polyopic heautoscopy might be important especially since eye movement related mechanisms are considered one of the major pathomechanisms in classical polyopia (Bender, 1945; but see Cornblath et al., 1998).

22.4. Inner visual doubles

22.4.1. Inner heautoscopy

A number of patients have been described that claim to see the inner organs of their own body and this experience has been called inner heautoscopy. Schilder (1935), Hécaen and Ajuriaguerra (1952), and Dening and Berrios (1994) only briefly mentioned inner heautoscopy, whereas Menninger-Lerchenthal (1935) and Lhermitte (1951) and especially Sollier (1903b) discussed several cases of inner heautoscopy in greater detail. There is a histrionic element to inner heautoscopy and most cases have been described about hundred years ago (Comar, 1901; Bain, 1903; Sollier, 1903a; 1903b).

Patients with inner heautoscopy claim to see their inner organs in extracorporeal space (Bain, 1903; Sollier, 1903a; 1903b) or rarely within their own body from an extracorporeal visuospatial perspective (Heintel, 1965). Modern accounts of inner heautoscopy are rare (Carlson, 1977, case #4; Magri and Mocchetti, 1967; Peto, 1969). Internal heautoscopy may also be encountered during shamanic rituals (Eliade, 1951/1964, p.62; cited in Brugger et al., 1997) and has been reported in certain populations. (Irwin, 1985 reported that Eskimos see their body as a skeleton under certain conditions). With respect to medical reports, Comar (1901; case #1) described a 18-year-old female patient who reported seeing her heart, and another patient (case #2) who claimed seeing her hip joint. Brugger et al. (1997) described a patient who saw the interior of his torso including blood circulating in vessels and another patient who saw his skeleton. The case described by Heintel (1965) is interesting as she did not describe

seeing her inner organs in extracorporeal space, but many different mirror images of her own body (of different sizes) inside her body. It thus seems as if this patient experienced seeing doubles inside her body from a disembodied visuospatial perspective that is generally reported by subjects with an OBE.

Paul Sollier (1903b) described several patients who experienced autoscopic hallucination or heautoscopy in association with inner heautoscopy. He described inner heautoscopy as "becoming conscious of one's inner organs, in their form, their placement, their structure, [and] their functioning (p.45)" and described a patient claiming to see at different times her heart, lungs, intestines, uterus, muscles, and even her brain (Sollier, 1903, pp. 68–79). Sollier thought inner heautoscopy to be functionally related to other autoscopic phenomena such as autoscopic hallucination and heautoscopy. Recently, Bradford (2005) suggested a relationship between inner heautoscopy and Cotard syndrome. Critchley (1950, p. 338) characterized "hysterical inner heautoscopy" as "a pathological accentuation of the body-image." Bradford (2005) summarized several patients with inner heautoscopy generally as a "late middle-aged, usually female [patient], pacing the wards of public psychiatric hospitals, describing and bemoaning the extraction or diseased state of their viscera, and, in keeping with their complaints of damnation to Hell, occasionally complaining of excessive bodily heat ('I am burning... I am on fire')" He adds that "morbid transformations of the viscera are reported more commonly than changes in the skeletal structure." Lhermitte (1939) mentions that inner heautoscopy might be related to sensations of referred pain (Sinclair et al., 1948). In referred pain, pain of inner organs is experienced at distinct spatial positions on the patient's body (Lhermitte, 1939, pp. 228–232). Thus, cardiac pain is often experienced in the left hand and arm, pain from the gall bladder in the right shoulder, and kidney pain in the testes. Cardiac pain may even be experienced in phantom limbs (Cohen and Jones, 1943; cited in Lukianowicz, 1958). One could thus assume that inner heautoscopy is related to pathological interference with shared representations of visceral and somatosensory body parts in the brain. Clinical evidence suggests that the insular cortex and the superior temporal gyrus might harbour such shared representations. Thus, the conscious (nonvisual) experience of one's inner organs is frequently reported by patients with temporal lobe epilepsy (Isnard et al., 2000). This includes a variety of "visceral sensations" (Penfield and Jaspers, 1954) such as epigastric sensations, abdominal aura, palpitations, and more rarely nausea, vomiting, suffocation, thirst, or constipation. Given that the insula contains cortical representations of the inner organs (Ostrowsky et al., 2000; Shelley and Trimble, 2004; Isnard and Maugière, 2005), that visceral sensations have been induced by electrical stimulation of the insula and the superior temporal gyrus (Penfield and Jaspers, 1954; Ostrowsky et al., 2000; Isnard and Maugière, 2005), and the fact that the lesion site of autoscopic hallucination, heautoscopy, and out-of-body experience most often affects the temporal lobe, one might suggest that inner heautoscopy may be related functionally and anatomically to a dysfunction of these shared cortical representations of inner organs with certain parts of the body surface and the entire body. The description of patient A.Ki. might be relevant in this respect as electrical stimulation at various points of his right insula induced sensations that included large parts of the body surface as well as visceral (abdominal) sensations (Penfield and Jaspers, 1954, pp. 426–431). Also, as reviewed by Dorpat (1971), not only the amputation of a limb, but also the resection of inner organs such as uterus, stomach, and rectum may lead to phantom sensations for the removed inner organs. Inner heautoscopy may thus be considered a visualized phantom sensation for inner organs due to disturbed central mechanisms with respect to visceral own body representations much as autoscopic hallucinations and phantom limb sensations are due to disturbed central mechanisms of body and limb representations. Finally, it might also be relevant concerning the involved mechanisms in inner heautoscopy to mention that the insula is a key region of the vestibular cortex (Guldin and Grüsser, 1998; Brandt and Dieterich, 1999) as it has been argued that disturbed vestibular processing is a key mechanism in autoscopic phenomena.

With respect to etiology, inner heautoscopy has most often been described in patients suffering from hysteria (Comar, 1901; Bain, 1903; Sollier, 1903a; 1903b). Dening and Berrios (1994) mentioned that inner heautoscopy is often associated with agitated depression (Dening and Berrios, 1994), but may also be observed in patients with neurosyphilis and psychiatric disease (Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952). To our best knowledge, internal heautoscopy has not been reported in neurological patients with circumscribed brain damaged.

We conclude that (1) the histrionic element in inner heautoscopy, (2) the rarity of cases and especially recent descriptions, and (3) the absence of cases with confirmed brain damage and detailed neuropsychological examination does not justify classifying inner heautoscopy with the other autoscopic phenomena. These observations also make clear that – at this point – any functional theory is pure speculation, although many associations–such as with other autoscopic phenomena, referred pain, phantom sensations of inner organs, and

Cotard syndrome, as well as visceral representations in temporal and insular cortex – might be meaningful.

22.4.2. Negative heautoscopy and negative doubles

Negative heautoscopy is defined as the failure to see one's own body when looked at either directly or in a mirror (Menninger-Lerchenthal, 1935; Lhermitte, 1951; Hécaen and Ajuriaguerra, 1952; Devinsky et al., 1989a; Dening and Berrios, 1994; Brugger et al., 1997). As for inner heautoscopy, case descriptions in neurological patients are rare, although a few reports of negative heautoscopy due to focal brain damage exist. Negative heautoscopy is discussed separately below (see negative doubles).

22.5. Somatosensorimotor doubles

22.5.1. Feeling of a presence

The "feeling of a presence" refers to the illusion that somebody is close by although nobody is around (Jaspers, 1913; Lhermitte, 1939; Critchley, 1950; 1955; Brugger et al., 1996). It is defined as the convincing feeling that there is another person close by without the patient actually being able to see that person (Brugger et al., 1996; Blanke et al., 2003) and was initially described by Karl Jaspers as "leibhafte Bewusstheit" (Jaspers, 1913). Later authors have named this experience of a somatosensory double "hallucination du compagnon" (Lhermitte, 1939), idea of a presence (Critchley, 1950), or more recently "feeling of a presence" (Brugger et al., 1996; Blanke et al., 2003). This experience of feeling another human person close by is often described as highly realistic and vivid, but may also be experienced as dreamlike and ephemeral. It is mostly a transient experience, yet might sustain for a longer time. It often disappears when patients try to ascertain themselves that there is "nobody there" by looking towards the felt location of the "presence." Although the patients do not experience seeing the "presence," they are convinced of the presence of the somatosensory double and can classically describe its spatial localization very accurately (James, 1961; Brugger et al., 1996).

Indeed, the "presence" is almost always experienced on one side of the patient's body (Féré, 1891; Jaspers, 1913; Menninger-Lerchenthal, 1935; Lippman, 1953; Critchley, 1955; Williams, 1956; Lukianowicz, 1960; Brugger et al., 1996; Blanke et al., 2003), in peripersonal space, and most often less than 1 m from the patient's body (Brugger et al., 1996; Blanke et al., 2003). Importantly, some patients may also mention a psychological affinity with the "presence" (Critchley, 1955; Brugger et al., 1996), or a sharing of actions (or echopraxia) or that the presence has the same body position as the patient (Jaspers, 1913; Engerth and Hoff, 1929; Brugger et al., 1996; Blanke et al., 2003). These latter points have led most previous authors to consider the feeling of a presence as a disorder of own body perception and led to its inclusion with other illusory own body reduplications

Case Study 22.5

Feeling of a presence
Brugger et al. (1996, case 2)

A 55-year-old right-handed woman reported several times a day the brief sensation of having "a shadow" in her right peripersonal space. She described that "the shadow is always in front of me, about 50 cm to the right. I feel that it is very familiar to me, and I kind of know that it is a male shadow." She did not see the shadow yet she could "feel" it, although she knew that there is nothing there. The shadow was described as stable or stationary, was not experienced as performing any action, did not talk to the patient and never imitated the patient's movements. The experience was not occurring during or after the patient's epileptic seizures. Often the feeling of a presence was associated by feelings of dizziness, vertigo, and headache. Notably, while her husband died some month afterwards, the patient began to refer to the presence as her deceased husband.

Six months before admission the patient developed headaches, rotational vertigo, and left-sided motor seizures. The neurological examination revealed left-sided hypoesthesia, a visuospatial memory deficit, mild apraxia, perseveration, and visual agnosia. Computed tomography demonstrated a space occupying lesion in the right temporal lobe. Surgical treatment rendered the patient seizure-free under anticonvulsant treatment. She continued to daily experience the feeling of a presence at least for a period of 6 months.

by most authors (Menninger-Lerchenthal, 1935; Lhermitte, 1939; Lippman, 1953; Hécaen and Ajuriaguerra, 1952; Critchley, 1955; Williams, 1956; Lukianowicz, 1960; Brugger et al., 1996; 1997; Blanke et al., 2003). Despite the fact that the patients deny seeing the double it is often described as "a shadow" or as a "black man" at the brink of vision (Critchley, 1950; Brugger et al., 1996; Blanke et al., 2003) that can be associated with autoscopic phenomena (Lukianowicz, 1960, case 2; Maack and Mullen, 1983; Brugger et al., 1996; Blanke et al., 2004, case 3 and 5).

Concerning associated hallucinations, vestibular hallucinations (Brugger et al., 1996, cases 2, 3, and 4; Blanke et al., 2004, case 5) and body schema disturbances have been observed quite often, whereas visual, tactile, and auditory hallucinations were only rarely noted. With respect to associated neurological deficits, the feeling of a presence is often associated with hemiparesis or hemiplegia (Féré, 1891; Gloning et al., 1957; Nightingale, 1982; Brugger et al., 1996, case 1) as well as somatosensory deficits (Hall, 1918; Gloning et al., 1957; Brugger et al., 1996, cases 1 and 2). In addition, patients with feeling of a presence may suffer from hemineglect (Critchley, 1979, case b; Brugger et al., 1996, case 1) or aphasia (Hall, 1918; Brugger et al., 1996, case 1; Blanke et al., 2003). Often other body schema disturbances such as limb disconnection, displacement, asomatognosia (Critchley, 1979, case b; Brugger et al., 1996, cases 1 and 4; Blanke et al., 2003), or somatoparaphrenia are present (see below). Some patients have also been reported to suffer from hemianopia (or quadrananopia) (Critchley, 1979, case b; Brugger et al., 1996, cases 1 and 4; Blanke et al., 2003).

22.5.2. Etiology

Feeling of a presence was described in various neurological disturbances, mostly epilepsy (Féré, 1891; Critchley, 1950; 1955; Williams, 1956; Gloning et al., 1957; Critchley, 1979; Hermann and Chhabria, 1980; Benson et al., 1986; Ardila and Gomez, 1988; Brugger et al., 1996; Blanke et al., 2003) but also migraine (Lippman, 1953; Todd and Dewhurst, 1955), neoplasm (Hécaen and Ajuriaguerra, 1952; Nightingale, 1982; Brugger et al., 1996), head injury (Lukianowicz, 1960), or acute hypoxia (Sherrard, 1978; Messner, 1980). In psychiatry it was described in patients with schizophrenia (Jaspers, 1913; Havens, 1962; Mahaluf et al., 1987), depression (Lukianowicz, 1960) and organic psychosis (Nightingale, 1982). However, it may also be present in normal subjects, especially during long periods of loneliness and exhaustion such as in mountaineers, explorers, sailors, and castaways (Smythe, 1934; Critchley, 1950; Suedfeld and Mocellin, 1987; Brugger et al., 1999; Kellehear, 1990).

22.5.3. Anatomy

Several patients have been described in whom feeling of a presence occurred in association with focal brain damage. Although it has been observed in patients with damage to any lobe, it is most often associated with posterior parietal damage (Kurth, 1941, case 2; Critchley, 1950; 1953; Hécaen and Ajuriaguerra, 1952; Gloning et al., 1957; Nightingale, 1982; Brugger et al., 1996, cases 1, 3 and 4; Blanke et al., 2003). However, other lobes such as the occipital or temporal lobe were also implicated, mostly in association with parietal lobe damage (Hécaen and Ajuriaguerra, 1952; Critchley, 1979, case b; Brugger et al., cases 1, 3, and 4). Finally, several patients with temporal lobe epilepsy and feeling of a presence have been described (Williams, 1956, case 7; Brugger et al., 1996, case 2).

With regard to predominant hemispheric involvement the reported data are quite divergent. Some authors found no hemispheric predominance for feeling of a presence (Brugger et al., 1996), others have suggested a right hemispheric predominance (Féré, 1891; Kurth, 1941) or left hemispheric predominance (Hall, 1918; Hécaen and Ajuriaguerra, 1952).

22.5.4. Theoretical considerations

A number of observations support the assumption that the "presence" is actually related to the patient's abnormal own body perception. Thus, a feeling of familiarity or close psychological affinity, as is often found in heautoscopy, is frequently mentioned (Critchley, 1955; Brugger et al., 1996). The patient also experiences the "presence" in close proximity to their body (Strindberg, 1897; Critchley, 1950; Brugger et al., 1996; Blanke et al., 2003) and even as imitating the patient's own body movements (Jaspers, 1913; Engerth and Hoff, 1929; Brugger et al., 1996). The "presence" is by some patients described as their "alter ego" (Critchley, 1950; 1955) and patients might even refer explicitly to the "presence" as their own double (Engerth and Hoff, 1929; Critchley, 1950). Feeling of a presence thus shares many phenomenological and clinical characteristics with autoscopic phenomena like autoscopic hallucination and heautoscopy and in some patients both phenomena are observed (Lukianowicz, 1960; Maack and Mullen, 1983; Brugger et al., 1996; Blanke et al., 2004). Other patients might find it difficult to clearly state whether they see their double or whether they feel a presence.

22.5.4.1. Autoscopic phenomena

Concerning the relation with autoscopic phenomena, Menninger-Lerchenthal (1935) referred to the feeling of a presence as "heautoscopy without optical image" and Bychowski (1943) also compared the experience of an autoscopic body or visual double to a "visual feeling of a presence." Of the autoscopic phenomena, the feeling of a presence shares many characteristics with heautoscopy. This concerns the psychological affinity (Critchley, 1955; Brugger et al., 1996) and the sharing of action that is reported by some patients between their bodies and the "presence" (Jaspers, 1913; Engerth and Hoff, 1929; Brugger et al., 1996; Blanke et al., 2003). Also, both latter illusory body reduplications are frequently associated with other body schema disturbances. Temporoparietal damage is often found in both conditions. Despite these similarities with heautoscopy, the feeling of a presence also shares some aspects with autoscopic hallucination, as both conditions are often lateralized (Féré, 1891; Jaspers, 1913; Menninger-Lerchenthal, 1935; Lippman, 1953; Critchley, 1955; Williams, 1956; Lukianowicz, 1960; Brugger et al., 1996) and may be associated with hemianopia (Critchley, 1979; Brugger et al., 1996; Blanke et al., 2003). Despite these similarities with autoscopic phenomena several differences should also be mentioned. During the feeling of a presence the double is not experienced visually and it is for this reason that we have classified it among nonvisual doubles and not with autoscopic phenomena. Also, the feeling of a presence, in the cases that we have analyzed here, is always lateralized, whereas the autoscopic body in autoscopic hallucination is only lateralized in ~50% of patients and almost never lateralized in heautoscopy (Blanke and Mohr, 2005). In addition, the feeling of a presence is frequently associated with contralesional deficits in somatosensory and motor function (Brugger et al., 1996) that have only infrequently been found in autoscopic phenomena (Blanke and Mohr, 2005). Based on the shared phenomenological and neurological characteristics with autoscopic phenomena we suggest that the feeling of the presence also relates to a double disintegration of multisensory information (Blanke et al., 2004). As most characteristics are shared with heautoscopy for which a primary dysfunction in proprioceptive processing has been proposed (Blanke and Mohr, 2005) and based on the observation that the feeling of a presence is often associated with sensorimotor deficits we suggest that it is associated with a dysfunction of motor–proprioceptive mechanisms. This is also compatible with the reported damage to parietal cortex. In the following section we discuss two clinical conditions that might be helpful in further unraveling the neural underpinnings of the feeling of a presence: phantom limbs and somatoparaphrenia.

22.5.4.2. Phantom limbs

Feeling of a presence has been associated with phantom limb phenomena by most authors reporting on the feeling of a presence (Menninger-Lerchenthal, 1935; Lhermitte, 1939; Hécaen and Ajuriaguerra, 1952; Brugger et al., 1996), which are the vivid impression that a missing body part is not only still present but in some cases painful (Ramachandran and Hirstein, 1998). Phantom phenomena were long considered to derive from irritation in severed axon terminals in the stump by the presence of scar tissue and neuromas (for review see Melzack, 1990). However, there is now a wealth of empirical evidence demonstrating cortical reorganization following limb amputation leading to disintegration of multisensory information (Ramachandran and Hirstein, 1998) as also proposed for autoscopic phenomena (Blanke et al., 2004). Moreover, although phantom limb phenomena most often occur in pathology, different phantom sensations and related phenomena might be easily induced in normal subjects. Examples are the Pinocchio effect (Lackner, 1988; Ramachandran and Hirstein, 1998) or the rubber hand illusion (Botvinick and Cohen, 1998) arising through multisensory conflict and relying on information processing in parietal and frontal cortices (Ehrsson et al., 2004; 2005). In these own body illusions, visual sensations and psychological affinity (or feelings of ownership) are projected onto parts of the external world through ambiguous proprioceptive–visual input (Ramachandran and Hirstein, 1998). It has been speculated that the brain is required to homogenize these different multisensory sensations to one coherent body representation and treat the discrepant or ambiguous information as noise (Ramachandran et al., 1995). If the discrepancy is not corrected, a phantom limb (or supernumerary phantom limb) may occur. If interference is rather with central mechanisms that represent the entire body of the subject, phantoms of the entire body may occur and this may be experienced as a somatosensory double (feeling of a presence) or as a visual double (autoscopic phenomena) (Blanke et al., 2004; Brugger, 2005).

22.5.4.3. Somatoparaphrenia

Previous authors suggested that autoscopic phenomena might share functional and neural mechanisms with somatoparaphrenia (Menninger-Lerchenthal, 1935; Hoff and Pötzl, 1935/1988; Hécaen and Ajuriaguerra, 1952; Gloning et al., 1963; Brugger, 2005). Although, somatoparaphrenia most often affects only certain (mostly contralesional) body parts such as the hand and arm of

the patient, we here present several neurological cases who reveal that somatoparaphrenia may also concern the contralesional half of a patient's body and even their entire body. We will draw especially on these latter cases to highlight some potential similarities between the feeling of a presence and somatoparaphrenia.

The term somatoparaphrenia was coined by Josef Gerstmann (1942) in an attempt to isolate it from two other phenomena, anosognosia and asomatognosia, that were often associated in patients with visuospatial neglect. (In Gerstmann's terminology asomatognosia was called autosomatoagnosia.) Gerstmann defined somatoparaphrenia as "specific psychic elaborations (marked by formation of illusions, confabulations, and delusions) with respect to the affected members or side of the body." Following Gerstmann's definition, somatoparaphrenia should be distinguished from asomatognosia that he defined as the patient's "imperception of the affected limbs or body half, in various degrees from simple forgetting to obstinate denial of their existence". Here we will not discuss the third symptom that Gerstmann discussed, anosognosia. Anosognosia has been defined in many variants, but the most common and most related in the present context is neurological patients' unawareness or nonexperience of contralesional hemiplegia (Babinski, 1923; Gerstmann, 1942; Cutting, 1978). Somatoparaphrenia is characterized by a number of apparently strange perceptions and beliefs with respect to the patient's extremities or body half of which the most common are that patients believe that their own arm or body half belongs to another person or that they have a third arm (supernumerary phantom limb; Halligan et al., 1993). Gerstmann's classification was only partly adapted by subsequent authors especially with respect to the distinction between asomatognosia and somatoparaphrenia (Hécaen and Ajuriaguerra, 1952; Feinberg et al., 1990). This is probably due to the many intermediate forms as well as the presence of asomatognosia and somatoparaphrenia in the same patient (Hécaen and Ajuriaguerra, 1952). Here, we will concentrate on patients with somatoparaphrenia that affect the entire body in order to stress its potential link with illusory reduplications of the entire body.

Pötzl (1925), Hoff and Pötzl (1935/1988), Menninger-Lerchenthal (1935), and later Gerstmann (1942) and Gloning et al. (1963) proposed that somatoparaphrenia may not only affect a limb or body part, but also a body half of the patient and even lead to limb or whole body reduplication.

For instance, Lhermitte (1939, p.130) described somatoparaphrenia in a patient with visuospatial neglect due to right hemisphere brain damage who perceived her left own hand as the hand of somebody else. Yet, as many of these patients, she also claimed that this hand belonged to (the body) of a person that is close by and that she assumed to be in her hospital room. Most often patients with somatoparaphrenia will thus claim that their own extremity is not just an unknown extremity, but the extremity of another person. And this extremity belongs generally to a neighbor in the hospital room (Lhermitte, 1939), a doctor (Gerstmann, 1942), the husband (Assal, 1983), or other family members or friends

Case Study 22.6

Feeling of a presence associated with somatoparaphrenia
Pötzl (1925)

In addition to somatoparaphrenia this patient also felt the presence of another person in his bed (feeling of a presence) suggesting that both phenomena might share functional and neural mechanisms.

This 56-year-old male patient repeatedly reported that his left hand and arm belonged to somebody else. This was especially the case when his hand was held in front of him and he mentioned that it was the hand of a stranger that he sees, probably belonging to another patient in the room. He also stated that "I don't know how this hand got here" or "the hand seems so long, so lifeless, as dead as a snake." He also claimed that there was an unknown person that was lying in his bed (to his left side) and that this person wants to push him out of the bed.

This 56-year-old male patient suffered from hemorrhagic brain damage to the right inferior parietal lobule including supramarginal and angular gyri, parts of the superior temporal gyrus and insula, as well as underlying white matter. Autopsy also revealed an older right thalamic lesion. The neurological examination revealed left-sided plegia and hemianesthesia without hemianopia and severe hemineglect associated with anosognosia.

(Weinstein et al. 1954, case 1). Thus, these patients *indirectly* attribute this somatoparaphrenic hand to another person in spatial (and emotional) proximity. Other patients have mentioned the presence of another person that is close by more *directly*. Thus, Pötzl (1925; patient #1; Case Study 22.6) described a patient with left-sided hemiplegia and somatoparaphrenia who not only claimed that his left arm belonged to an unknown person, but also that there was another person lying in his bed to his left and that this person tried to push him out of the bed (p.119). Pötzl (1925) described a second patient with left-sided hemiplegia and somatoparaphrenia also claiming that his left arm belonged to a stranger. As this patient also claimed that there was a supernumerary left arm (see also Ehrenwald, 1930; Halligan et al., 1993), Pötzl (1925) and later Hoff and Pötzl (1935/1988) argued that reduplication of an extremity and of an entire body in patients with somatoparaphrenia may share functional mechanisms and that the delusional other in somatoparaphrenia is closely related to the feeling of a presence. Further such cases with somatoparaphrenia and the feeling of a presence can be found in the literature. Engerth and Hoff (1929) describe a 71-year-old man with left-sided hypoesthesia, hemianopia (with hemianopic hallucinations), and anosognosia who experienced a constant left-sided person who was most often localized next or behind the patient. In addition, the patient noted that this person had the patient's posture and size and only appeared when the patient was standing or walking. This dependence on posture and action of the patient has also been described in recently reported patients with feeling of a presence (Blanke et al., 2003) and, notably, heautoscopy (Blanke and Mohr, 2005). Lhermitte (1939) described a 72-year-old female patient with left-sided hemiplegia and hemianesthesia that claimed that her left body half belonged to another person that was lying in the same bed as her. More such patients with the association of somatoparaphrenia and the feeling of a presence were reported by Anton (1898), Zingerle (1913), Halligan (1995), and Cereda et al. (2002). The fact that this "stranger's body" is experienced in a highly realistic fashion is underlined by the fact that many of these patients are afraid or annoyed by the presence of this stranger. By trying to throw them out of the bed these patients often find themselves on the floor. This difficulty to distinguish between self and other is reminiscent of severe cases of heautoscopy (see Case Study 22.3) where the patient desperately tries to get rid of the unwanted stranger by often very dangerous (self-mutilating) actions. Some patients not only report sensorimotor doubles, but report seeing their double (autoscopic phenomena) on the contralesional side (Hoff, 1931). Still other patients may even describe that another person's body (such as their father) has partly invaded one half of their body (Nightingale, 1982, Case Study 22.7).

Based on these observations and the association of somatoparaphrenia with parietal lobe damage we argue that doubles that are reported by patients with

Case Study 22.7

Somatoparaphrenia
Nightingale (1982)

Somatoparaphrenia is mostly confined to the patient's upper extremity and patients claim that their generally plegic arm belongs to another (most often familiar) person. The present case illustrates that somatoparaphrenia may also affect an entire half of the patient's body and may be associated with illusory reduplication of the entire body.

A 46-year-old right-handed man felt that the left side of his body was different from the right half. He explained that the left side of his body had slipped behind the right side so that the latter became more prominent than the former. Moreover, the left side seemed to him somewhat evil and controlled by external agents (such as the devil or his father). The right side of his body was perceived as "self" and "good." These two sides were in constant conflict about his behavior. The left body side tried to instruct him to perform evil acts that his "self" or right body side felt to be incorrect. These experiences were accompanied by the patient hearing compelling voices, coming from his left extracorporeal space. Rarely, he experienced left-sided complex visual hallucinations and the presence of another person to his left (feeling of a presence).

This patient suffered from complex partial seizures with secondary generalization since the age of 30 years. Despite removal of a parasagittal meningioma that was adjacent to right parietal cortex and anticonvulsant treatment the patient continued to have frequent seizures. The patient is known for a period of moderate depression following the death of his father at the age of 40 years. There were no signs of schizophrenia.

somatoparaphrenia may relate phenomenologically, functionally, and anatomically to the feeling of a presence and accordingly also with other own body reduplications. As somatoparaphrenia is strongly associated with right hemispheric brain damage, whereas the feeling of a presence is encountered with damage to either hemisphere, it is likely that the mostly left-sided sensorimotor doubles in somatoparaphrenia and right-sided sensorimotor doubles relate to different functional mechanisms (Brugger et al., 1996; 1997). This is also suggested by clinical differences between right- and left-sided sensorimotor doubles with respect to etiology and clinical evolution.

22.5.4.4. Delusional misidentifications syndromes

Finally, some authors (Signer, 1987) have proposed that the feeling of a presence (as well as autoscopic phenomena) may also relate functionally to delusional misidentifications syndromes concerning the patient's body and self, either as imposter or double. These include the subjective doubles syndrome (Christodoulou, 1978a; Case Study 22.8) and as a subtype of the Capgras syndrome (Berson, 1983; Kamanitz et al., 1989; Silva and Leong, 1991; Silva et al., 1993; Feinberg and Roane, 2005). Patients with the subjective doubles syndrome are convinced that another person is posing as the patient, whereas patients with Capgras syndrome may claim that not only other persons, but also they themselves are replaced by identical substitutes (Capgras and Reboul-Lachaux, 1923; Kamanitz et al., 1989; Silva and Leong, 1991; Silva et al., 1993). Other forms may also include patients who fail to recognize themselves in a mirror (Hécaen and Ajuriaguerra, 1952; Ajuriaguerra et al., 1963; Foley and Breslau, 1982) and, in addition, mistake their mirror reflection for an imposter (Gluckman, 1968; Feinberg and Shapiro, 2000).

Other authors thought it important to distinguish between delusional misidentification syndromes and autoscopic phenomena based on several clinical differences (Sims, 1986). Thus, Sims mentions that autoscopy (or autoscopic phenomena in general) (1) has a perceptual element, which is generally absent in delusional misidentification syndromes, (2) that the autoscopic patient experiences the double as "their real self" whereas the Capgras patient is convinced that the double is an imposter, and that (3) autoscopy is a pseudohallucination, which delusional misidentification syndromes are not. Yet, as discussed in this review, we would like to underline here that only some illusory reduplications are experienced as pseudohallucinations (autoscopic hallucinations) and the double is only rarely experienced as the location of the "real self." Weinstein

Case Study 22.8

Subjective doubles syndrome
Christodoulou (1978b)

Patients suffering from subjective doubles syndrome claim that another person has taken on the same appearance as the patient, but has kept the other person's character traits and leads a life of their own. Some patients stated that several others have taken on their appearance. Other patients claim that another person with their habitual appearance has taken the same personality as the patient. Both subtypes are probably related to the more common syndromes of Capgras and Frégoli.

An 18-year-old woman developed insomnia, agitation, depression, loosening of associations, lack of sexual inhibition, and experience of déjà vécu. Yet, she also stated that a female neighbor acquired physical characteristics identical to the patient's characteristics. The subjective double was described as having the "same face, same build, same clothes, same everything." The patient also stated that the neighbor accomplished this by wearing special make-up, a wig, and a mask. In another episode, while the patient was hospitalized, she insisted that at least two other female patients had transformed themselves into her by taking on the patient's appearance. The patient even attacked one of them trying to "pull the mask" off the other patient's face. In a letter to her father she explained: "In here there is a girl as fat and as tall as I am. At night when everyone is asleep, she puts on a wig and a mask and walks from room to room stealing things in order to incriminate me. One night I woke up and saw her with my own eyes. It is unfortunate that due to my confusion I failed to run to the window to shout to the people, 'look here, this is me, and this is my double with a wig and a mask.'"

The patient had an unspecified seizure disorder since the age of 8 years. Before the above described hospitalization her psychiatric history was unremarkable. A paternal uncle suffered from a paranoid schizophrenia. Neurological examination, routine laboratory check, cerebrospinal fluid and computed tomography were normal.

et al. (1954) and, more recently, Signer (1987) even speculated about common mechanisms between reduplicated body parts, bodies, and paramnesias for place and event (Röhrenbach and Landis, 1995). Yet, although it is likely that some common mechanisms may be involved between delusional misidentication syndromes, delusional mirror recognition and misidentification, and paramnesias, we have not elaborated this any further here due to the many clinical differences of the latter conditions with illusory own body reduplications.

In conclusion, these observations on the feeling of a presence suggest that it shares phenomenological, functional, and neural mechanisms with visual doubles (especially heautoscopy) and delusional doubles (somatoparaphrenia) and is probably due to multisensory mechanisms and sensorimotor disintegration. It might thus be speculated that the investigation of these three conditions through detailed neuropsychological examination is likely to further our understanding of the central mechanisms of own body representations, self processing, and self–other distinction as previous research helped elucidating the nature of (supernumerary) phantoms limbs.

22.6. Auditory doubles

22.6.1. Hearing of a presence

Are there auditory doubles? Have there been reports of neurological patients who claim to have the highly realistic experience of hearing a double of themselves or another person in extracorporeal space? Menninger-Lerchenthal (1935) and Gloning et al. (1963) have suggested that illusory own body reduplications should also exist in the auditory domain, yet have not presented clinical evidence for this nor further developed this hypothesis.

Audition like vision, balance, and somatosensation is involved in the construction of the body image (Làdavas, 2002; Blanke et al., 2003; Pavani et al., 2003; Holmes and Spence, 2004). Moreover, electrophysiological studies in the macaque at the subcortical level (Stein et al., 1993) and in parietal and temporal cortex (Duhamel et al., 1998; Bremmer et al., 2001; Schroter-Kunhardt, 2002) suggest that several cerebral areas combine auditory signals with tactile, proprioceptive, and visual information in a coordinated reference frame for personal and extrapersonal space. This has also been found by neuroimaging work (Bremmer et al., 2001; Foxe et al., 2002; Holmes and Spence, 2004) and behavioral studies in brain damaged and healthy subjects (Làdavas et al., 2001; Làdavas, 2002; Pavani et al., 2003; Holmes and Spence, 2004) in humans. In light of these findings and the earlier speculations by Menninger-Lerchenthal (1935) and Gloning et al. (1963), it might thus be hypothesized that neurological damage to temporoparietal areas might not only lead to visual and sensorimotor doubles, but also to auditory doubles.

22.6.2. Clinical presentation, etiology, anatomy

Auditory hallucinations cover a variety of elementary experiences such as hearing noises or sounds (humming, buzzing, tapping, ringing, etc) and complex experiences such as voices, conversations, or music (Cole et al., 2002). Complex auditory hallucinations are most often characterized by the hearing of a voice or voices that are generally called auditory verbal hallucinations. About 70% of schizophrenic and a variety of other psychiatric and neurological patients suffer from auditory verbal hallucinations (Stephane et al., 2001). Voices during auditory verbal hallucinations are most often experienced as addressing the subject directly and called 2nd person auditory verbal hallucinations (Frith, 1996). Less frequently voices during auditory verbal hallucinations may be experienced as the subject's own voice (1st person auditory verbal hallucinations) or as hearing two or more other people taking to each other (3rd person auditory verbal hallucinations). The content of auditory verbal hallucinations may vary as does the localization of the voice which maybe at varying positions in personal or extrapersonal space. In addition, most patients experience these variably localized auditory verbal hallucinations as voices and not as a present person that speaks to them. Auditory verbal hallucinations in neurological patients have been reported most often in spontaneous seizures and been localized to the temporal cortex (Bancaud, 1987). Auditory verbal hallucinations may also be evoked directly by electrical cortical stimulation in patients with pharmacoresistant epilepsy (Penfield and Perot, 1963; Halgren et al., 1978; 1983), which has the advantage of greater spatial precision and experimental control. The electrically induced experiences were reported to be highly similar to those described by psychiatric patients (2nd person auditory verbal hallucinations) and mostly characterized by hearing voices inside the head or at varying locations. Yet very few patients reported a precise localization of the auditory source as well as hearing a talking person. Penfield and Perot (1963) reported this in two of 21 patients with stimulation-induced and seizure-induced auditory verbal hallucinations (case 12 and 29). Both epileptic patients reported that they not just heard a localized "voice," but heard a physically present person in the contralateral space or in the backspace that spoke to them. Moreover, the "heard persons" had a precise location and distance from the patient's body and in both patients either the feeling of a presence (case 12) or

the visual experience of a second body (case 29) were noted as well. One may wonder whether this was an autoscopic body, although this is not further detailed. Thus, a 24-year-old woman (case 12) had seizures since the age of 20 years characterized by a sensation (of something or somebody) in her back, complex auditory and visual hallucinations, and fear followed by secondary generalization. She described hearing a man that spoke behind her and that she could not understand what he was saying. Her seizure focus was localized to the left parietal lobe (arteriovenous malformation). Electrical cortical stimulations at the left posterior aspects of the superior temporal gyrus (at the temporoparietal junction) induced hearing of a presence described as "I could hear someone talking" and "there was [somebody] talking or murmuring, but I could not understand it." During further stimulations she detailed that she heard a man who was standing behind her and who was once identified as her father. Note that somatoparaphrenic doubles or extremities are also often identified as relatives of the patient. The second patient of Penfield and Perot (1963) with hearing of a presence was a 25-year-old man (case 29) who suffered from seizures since the age of 19 years characterized by vertigo and auditory verbal hallucinations (a voice calling him by his first name). (Interestingly, several authors have suggested that 2nd person auditory verbal hallucination of being called by one's first name relate to autoscopic phenomena (Menninger-Lerchenthal, 1935, pp. 131–132; Schilder, 1914). See Perrin et al. (2005) for a recent neuroimaging study on the neurobiology of hearing one's first name.) His seizure focus was localized to the perisylvian region including temporal and parietal cortex (arteriovenous malformation). Electrical cortical stimulations at the right posterior (and middle) aspects of the superior temporal gyrus induced hearing of a presence. This was described as "it is just like someone [is] whispering in my left ear" and "again someone [is] trying to speak to me, a single person," "I could not understand what he said." Interestingly stimulations at the superior temporal gyrus also lead to the visual impression seeing of a person in front of him ("someone was there in front of me"). Other stimulations at sites on the superior temporal gyrus and middle temporal gyrus led to different auditory hallucinations and experiential phenomena. Hearing of a presence was also reported by Gloor et al. (1982, case 3). More recently, Blanke et al. (2003) also described a patient with hearing of a presence probably due to epileptic seizures following hemorrhagic brain damage at the left TPJ (Case Study 22.9).

Case Study 22.9

Hearing of a presence
Blanke et al. (2003)

A right-handed 65-year-old nun reported complex auditory hallucinations characterized by the impression of hearing for various periods one or two people talking behind her. During one especially impressive and long period she was sitting in the hospital church when she suddenly had the feeling that she heard two "people" whispering behind her. Both "people" were sitting on a bench approximately one meter behind her and on her right. She could not understand what they were saying. She could not indicate the gender of these "people" or any other character of their voices. While turning around she noticed that there was no one sitting behind her. Yet, after she turned her head back forward, she continued to hear two people whispering behind her back on the left side. This persisted until she left the hospital church. She reported similar experiences in her hospital room (and after hospital discharge for a period of several years). These instances were always characterized by the auditory perception as if someone was suddenly standing behind her and to her right and talking in an incomprehensible manner to her. In addition, she suffered from simple auditory hallucinations characterized by humming or buzzing also localized on the right side (either lateral or behind her) or bilaterally. She also experienced several times a day a "shadow" on her right side (feeling of a presence) and other right side dyssomatognosic illusions.

The patient developed complex partial seizures with secondary generalization due to a hematoma at the left parietotemporo-occipital junction at the age of 60 years. When hospitalized for the hearing of a presence, the neurological examination revealed right-sided auditory spatial agnosia (deficit in the localization of auditory targets), moderate aphasia with semantic and phonological paraphasias, severe alexia, and moderate agraphia. There were no signs of apraxia or of visual agnosia. MRI did not show any new lesion, but EEG revealed frequent interictal epileptic activity characterized by spike-waves, sharp waves and slow waves over the left mid-to-posterior temporal region. In one instance, rhythmic discharges over the occipitotemporal region were noted.

With respect to the underlying anatomy, Penfield and Perot (1963) localized auditory verbal hallucinations to the superior and middle temporal gyri of either hemisphere with a left-sided predominance. Others have confirmed these findings, but also induced auditory verbal hallucinations by electrical cortical stimulation of inferior temporal and mesial temporal structures (Penfield and Perot, 1963; Halgren et al., 1978; 1983). The three cases of hearing of a presence are concordant with the findings by Penfield and Perot (1963).

22.6.3. Theoretical considerations

The four cases with hearing of a presence (Penfield and Perot, 1963; Gloor et al., 1982; Blanke et al., 2003) thus closely resemble each other and are in contrast to classically reported auditory verbal hallucinations in epileptic patients. In addition, they suggest that HP can phenomenologically be dissociated from other auditory verbal hallucinations. Indeed, psychotic patients often find it difficult to say whether the "voice" is inside or outside their head (Nayani and David, 1996; David, 1999) and mostly experience auditory verbal hallucinations inside their head or body (Junginger and Frame, 1985; Chadwick and Lowe, 1994; Nayani and David, 1996). This was also found for most stimulation-induced auditory verbal hallucinations in epileptic patients (Penfield and Perot, 1963) and differs from the phenomenology described by these four patients described here who localized a talking person (or persons) at a precise location in their backspace. This auditory lateralization and auditory distance from the patient's body was corroborated by neuropsychological findings showing that the heard person(s) were localized on the side where spatial auditory agnosia and other dyssomatognosic sensations were found (Blanke et al., 2003). Although, some psychotic patients are able to describe characteristics of the voice such as content, affective tone, and identity, they usually lack spatial attributes such as location in extrapersonal space (Junginger and Frame, 1985; Chadwick and Lowe, 1994). This has even led to the proposition that auditory verbal hallucinations of psychotic origin classically lack any localization (Strauss, 1962). Even if in rare instances external auditory verbal hallucinations may be lateralized and localized in psychiatric patients, their spatial attributes are extremely variable. They are experienced at variable distances and variable locations from the patients' bodies and often described at delusional locations (Chadwick and Lowe, 1994; Nayani and David, 1996; David, 1999).

Based on these differences and neuropsychological findings, Blanke et al. (2003) suggested that the hearing of a presence might relate to auditory–spatial disorders rather than auditory disorders (related to the identification of the nonspatial characteristics of a sound). The coappearance of hearing of a presence and feeling of a presence in three of the here presented patients as well as previously reported patients with feeling of a presence (Jaspers, 1913; Critchley, 1954; Grüsser and Landis, 1991; Brugger et al., 1996) also suggests their close functional relationship. It could be argued that the hearing of a presence is not a disorder of own body perception (referring to disorders in the perception and cognition of the patient's own body), since these four patients never experienced their "own voice" or their "own body" as talking behind themselves. Similar arguments have been proposed for the feeling of a presence. Yet, as noted by Brugger et al. (1996) and others (Jaspers, 1913; Menninger-Lerchenthal, 1935), although patients suffering from feeling of a presence also do not feel their own body at two locations at the same time, the felt (or heard) body is always experienced in a very persuasive way (at the fringe of vision) and is often associated with a strong feeling of a strangeness towards one's own body (depersonalization; Dening and Berrios, 1994; Brugger et al., 1997) and a psychological affinity with the felt body. In addition, in rare instances the feeling of a presence is associated with autoscopy (Brugger et al., 1996; 1997) suggesting a close link between visual and nonvisual body reduplications. Several functional and neural mechanisms have been proposed to account for auditory verbal hallucinations. Research proposed that auditory verbal hallucinations might be due to either an auditory dysfunction (McKay et al., 2000), a language dysfunction (Hoffmann, 1986; Frith and Done, 1988), a failure to monitor inner speech (McGuire et al., 1995), or dysfunctional reality monitoring (Bentall, 1990). Based on the rare, but concordant phenomenological and neuropsychological data in patients with hearing of a presence we speculate that it might result from a paroxysmal failure to integrate auditory body-related information with somatosensory and visual body-related information. This information is needed in order to create neural representations of personal and peripersonal auditory space (di Pellegrino et al., 1997; Làdavas et al., 2001; Farnè and Làdavas, 2002) and the mechanisms of hearing of a presence Au13 are probably related to, but distinct from, mechanisms causing more common forms of auditory verbal hallucinations. These data suggest that within the group of illusory own body reduplications that concern the whole body, one should discern between visual doubles (autoscopic phenomena), sensorimotor doubles (feeling of a presence), and auditory doubles (hearing of a presence: the persuasive hearing of a person nearby).

22.7. Negative doubles

Negative heautoscopy is defined as the failure to see one's own body either when looked at directly or in a mirror (Menninger-Lerchenthal, 1935; Lhermitte, 1951; Hécaen and Ajuriaguerra, 1952; Devinsky et al., 1989a; Dening and Berrios, 1994; Brugger et al., 1997). Although negative heautoscopy is not an own body reduplication in the strict sense it is classically grouped among autoscopic phenomena. This is due to the fact that negative heautoscopy shares many phenomenological characteristics with other forms of autoscopic phenomena. Most authors mentioned negative heautoscopy only briefly with respect to other forms of autoscopic phenomena (Lhermitte, 1951; Hécaen and Ajuriaguerra, 1952; Devinsky et al., 1989a; Dening and Berrios, 1994), some mentioned that negative heautoscopy is a distinct autoscopic phenomenon (Bradford, 2005; Blanke and Mohr, 2005), whereas others have included it more prominently (Grüsser and Landis, 1991; Brugger et al., 1997; Brugger, 2005). This may largely be due (as for inner heautoscopy) to the fact that case descriptions, especially recent ones in neurological patients, are rare. Yet a few reports of negative heautoscopy due to focal brain damage do exist and as we will argue in the remainder of this section that negative heautoscopy may have functional links with other neurological conditions such as asomatognosia and depersonalization.

22.7.1. Clinical presentation

The most well-known description has probably been given by Guy de Maupassant in his short story "Le Horla" (Maupassant, 1886/1961) and was quoted by Lhermitte, Critchley, and many other neurological authors. After describing many instances of persecution, fear, and hallucinations Maupassant writes "I could not see myself in the mirror! It was empty, transparent, deep [. . .] I was not reflected in it [. . .] and I was standing in front of it." A medical history with negative heautoscopy has been described by von Stockert (1934). This patient was "alarmed by the sudden impression of the left half of his body being absent. When he would look at himself with horror, he would indeed notice that the left half was not there. At these moments he felt somewhat comforted by the visual confirmation [of not seeing his left body] of his somatosensory impressions" (cited in Brugger et al., 1997). Interestingly, this patient claimed not only that he could no longer see his own left

Case Study 22.10

Negative heautoscopy
Arzy et al. (in press)

Negative heautoscopy refers to failure to perceive one's own body either in a mirror or when looked at directly. Given the rarity of negative HAS for the entire body we detail here the experience of a recently reported patient in whom negative heautoscopy only affected one extremity. We suggest that the involved pathomechanisms are similar and might further relate to those involved in asomatognosia.

A 51-year-old, right-handed woman, without neurological or psychiatric antecedents reported that for several minutes she did not see her left arm and left hand any more while she did normally see all other parts of her body. While at work she suddenly felt dizzy and noticed that parts of her left arm had "disappeared." She thus did not see her left upper extremity from her elbow on downwards. She was quite frightened, but realized to her astonishment that she could see the table on which she had rested her "disappeared" arm as if she could see the table "through the left arm." She saw her left arm only above her elbow where she saw a clear cut border. In addition, she could not move her left arm or hand while being normally able to move her right arm. She noted no changes with respect to any other body parts. Only after several minutes did she experience that her left arm and hand changed again being progressively "restored" until the arm was "complete" again and occluding the table beneath it. Only some minutes later was she able to move her arm normally again.

The neurological examination showed moderate left-sided hypoesthesia for arm and lower face, a mild executive deficit in Luria's alternating sequences test, verbal semantic fluency, and in the mental rotation of human body parts. There were no signs of visuospatial neglect. Magnetic resonance imaging showed two small ischemic lesions in the premotor and the primary motor cortices.

body half, but also noted that, when looking at other people that they lacked the right side of their bodies. Sollier (1903a) reports a case of negative heautoscopy in a 14-year-old hysteric patient and Magri and Mocchetti (1967) describe a 61-year-old patient who suffered from complex partial seizures and reported that he could not see his mirror reflection anymore. Brugger et al. (1997) note that some patients have been described who suffer from negative heautoscopy in association with other autoscopic phenomena. A more recent patient was briefly mentioned by Brugger (2005; unpublished observation). This female patient suffered from panic attacks consisting of episodes during which she could not see the left half of her body (negative heautoscopy for her left hemibody). EEG revealed abortive spike-wave complexes over the right parietocentral area and carbamazepine treatment seemed to have abolished all symptoms. Other patients have been described that noted that parts of their body were detached, missing, or invisible with respect to the rest of their body. Indeed, Gloning et al. (1954) described a patient with simple partial seizures and left-sided sensorimotor deficits who noted that during his simple partial seizures his right body half was one meter in front of his normally localized left-sided body. Brugger et al. (2006) describe a patient who noticed that his body was split along the midline with an empty area between both body parts. Finally, Blanke et al. (2002; 2004) described a patient who during an out-of-body experience only saw the lower parts of her body (autoscopic body). Whereas this partial vision of the autoscopic body is rather rare during out-of-body experiences and heautoscopy, it is quite frequent during autoscopic hallucinations and concerns generally the lower body (for review see Blanke and Mohr, 2005). It might thus be proposed that these latter negative illusory own body experiences reflect related functional and neural mechanisms.

The authors argued that this patient's negative experience shares many characteristics with asomatognosia and may be defined as a visual form of asomatognosia. Au4 Extending Arzy et al.'s (in press) argumentation to the entire body (as in negative heautoscopy), one might argue that the entire visually perceived body may also be missing, disappear, or "fall out of corporeal awareness." The reviewed cases suggest that negative heautoscopy may affect the entire body, but mostly seems to affect only one half of the patient's body or only a certain body part (mostly the upper extremity; Grüsser and Landis, 1991; Brugger et al., 1997; Bradford, 2005). It should also be noted that the autoscopic body in autoscopic phenomena is not infrequently seen as missing certain body parts (Nouët, 1923; Genner, 1947; Maximov, 1973; Blanke et al., 2002; 2004; for review see Blanke and Mohr, 2005).

22.7.2. Etiology and anatomy

Bradford (2005) writes that negative heautoscopy is an "instance of conversion reaction, a hysteria driven and attenuated form of asomatognosia." Critchley (1953, p. 240) stated that negative heautoscopy is "very rare" and may be an "expression of a psychotic illness." Yet, a few cases due to focal brain damage have also been reported. Although most lesions affected the right hemisphere, lesion sites included parietal and frontal cortex, thalamus, and splenium. For instance, von Stockert's (1934) patient suffered from a right-sided thalamic tumor that invaded the splenium and the patient described by Magri and Mocchetti (1967) suffered from complex partial seizures with secondary generalization due to a calcification in her right parietal lobe. The patient reported by Arzy et al. (in press) suffered from Au5 two small ischemic lesions in right motor and premotor cortex and Brugger's patient (2005) showed abnormalities over the right centroparietal region.

22.7.3. Theoretical considerations

22.7.3.1. Asomatognosia

These abovementioned cases suggest that negative heautoscopy might share some functional mechanisms with asomatognosia (Magri and Mocchetti, 1967; Devinsky et al., 1989a). This might have also been the reason why Devinsky et al. (1989) and Magri and Mocchetti (1967) proposed the name asomatoscopy for negative HAS. Au14 Patients with asomatognosia generally describe that an arm or leg or an entire body half seems to be "missing" or that "the affected body parts may seem to disappear or to fall out of corporeal awareness" (Critchley, 1953, pp. 237–238). Evidence from patients with focal brain damage suggests that asomatognosia is linked to posterior parietal (or temporoparietal) lesions, especially in the right hemisphere (Critchley, 1953; Hécaen and David, 1945; David et al., 1946; Feinberg et al., 1990; Leiguarda et al., 1993; Feinberg et al., 2000; Sierra et al. 2002; So and Schauble, 2004). Experimental findings in patients with asomatognosia are rare, but several case studies have shown that asomatognosia may be modified by touching the "missing" body part or by looking at it suggesting multisensory mechanisms in asomatognosia and autoscopic phenomena (Critchley, 1953; Newport et al., 2001). Thus an asomatognosic patient described by Carp (1952) lost her sensation for the right half of her body and had to verify continuously its existence by looking at it. Whereas the missing body part in asomatognosia is generally experienced as a somatosensory loss (Critchley, 1953; Hécaen and David, 1945; David et al., 1946; Feinberg et al., 1990; Leiguarda et al., 1993; Feinberg et al.,

2000; Sierra et al. 2002; So and Schauble, 2004) the abovementioned cases of von Stockert (1934) and Arzy et al. (in press) suggest that asomatognosia may also exist as a visual loss. One might thus classify these cases either as partial negative heautoscopy or as visual asomatognosia. Khazaal et al. (2005) considered asomatognosia a special form of hemi-depersonalization (quoted after Brugger, 2005). The association between depersonalization and negative heautoscopy as well as other forms of autoscopic phenomena will be briefly considered next.

22.7.3.2. Depersonalization

Many authors have pointed out that autoscopic phenomena (except autoscopic hallucinations) are often associated with depersonalization (Menninger-Lerchenthal, 1935; Hécaen and Ajuriaguerra, 1952; Leischner, 1961; Devinsky et al., 1989a; Grüsser and Landis, 1991; Dening and Berrios, 1994; Brugger et al., 1997). Depersonalization is one of the four major dissociative disorders and defined as "an alteration in the experience of the self so that one feels detached from, and as if one is an outside observer of, one's mental processes or body" (DSM-IV, American Psychiatric Association). Dissociation including depersonalization is most common after severe stress as for example in military combat and automobile accidents (DSM-IV, American Psychiatric Association) that are also common precipitating factors of autoscopic phenomena (Devinsky et al., 1989a). As stated recently by Simeon (2004) not much is known about the neuroanatomical mechanisms involved in depersonalization. Penfield and Jaspers (1954), Gloor et al., (1982), and Devinsky and colleagues linked autoscopic phenomena (Devinsky et al., 1989a) and dissociative states (including depersonalization) (Devinsky et al., 1989b) to temporal lobe structures and epilepsy. Sierra and Berrios (1998) postulated that depersonalization is related to a widespread disturbance including prefrontal hyperactivation, limbic hypoactivation, and parietal dysfunction (Sierra et al., 2002). Simeon et al. (2000) linked activation at the right TPJ including right middle and superior temporal gyri, the right inferior parietal lobule as well as left occipital cortex to depersonalization.

Patients with autoscopic phenomena often suffer from depersonalization probably by being confronted with the experience of seeing one or more second own body or bodies in extracorporeal space (autoscopic hallucinations, heautoscopy, polyopic heautoscopy), of having the sensation of disembodiment (OBE), or of not feeling or seeing their body anymore (negative heautocopy, asomatognosia). Depersonalization may be especially strong when the patient does not see his own body (or body parts) through direct inspection or as reflected in a mirror as was the case in the patient reported by Arzy et al. (in press). This was also mentioned by Critchley (1953, p. 240) who stated that especially negative heautoscopy is "a severe example of the depersonalization syndrome." In conclusion, these observations on negative heautoscopy suggest that it shares several phenomenological, functional, and neural mechanisms with autoscopic phenomena and asomatognosia. We speculate that negative heautoscopy is also due to multisensory disintegration in parietal or temporoparietal cortex, especially in the right hemisphere, and that its neurological investigation might shed some light on depersonalization and dissociative states.

22.8. Conclusion

In science the most challenging phenomena are often the ones we take for granted in our everyday lives. Excellent examples are the self and the experienced spatial unity between self and body and thus the everyday experience of being spatially embodied. Both folk and psychological notions are challenged by the experience of one or more second own bodies or doubles that neurological patients describe in all their multisensory forms. The reviewed evidence from neurological patients experiencing these striking dissociations between self and body suggests that AP are culturally invariant phenomena, which can be investigated scientifically to further our understanding of the functional and neural mechanisms of corporeal awareness and self consciousness. Importantly, these findings will also help physicians in diagnosing and treating affected patients. The neuroscientific study of the self is in its infancy and there are currently no established models, very little data, and often not even the vocabulary to describe neuroscientific notions of the self and its relation to the subject's body. This complexity is especially evident when patients describe doubles to their physicians. We believe that the investigation of the phenomenological, functional, and neural mechanisms leading to the experience of a double in neurological patients (and healthy subjects) is likely to improve our neuroscientific models of embodiment, self, and subjectivity.

References

Ajuriaguerra J, Strejilevitch M, and Tissot R (1963). A propos de quelques conduites devant le miroir de sujets atteints de syndromes dementiels de grand age. Neuropsychologia 1: 59–72.

Alvarado CS (1992). The psychological approach to out-of-body experiences: A review of early and modern developments. J Psychol 126: 237–250.

Anton G (1898). Über Herderkrankungen des Gehirns, die vom Patienten selbst nicht wahrgenommen werden. Wien Klein Wochenschr 11: 227.

Ardila A and Gomez J (1988). Paroxysmal "feeling of somebody being nearby". Epilepsia 29(2): 188–189.

Arzy S, Idel M, Landis T, et al. (2005). Speaking with one's self. Autoscopic phenomena in writings from the ecstatic Kabbalah. J Consciousness Studies 12: 4–30.

Au8 Arzy S, Overney L, Landis T., et al. (in press). Neural mechanisms of Embodiment: Asomatognosia due to premotor cortex damage. Arch Neurol 63: 1022–1025.

Assal G (1983). Non, je ne suis pas paralysée, c'est la main de mon mari. Arch Suisses Neurol Neuroschir Psychiatr 133: 151–157.

Babinski A (1923). Sur l'anososgnosie. Rev Neurol 39(6):
Au15 731–731.

Bain A (1903). De l'autorepresentation chez les hysteriques Vigot, Paris.

Bancaud J (1987). [Clinical symptomatology of epileptic seizures of temporal origin]. Rev Neurol (Paris) 143(5): 392–400.

Bender MB (1945). Polyopia and monocular diplopia of cerebral origin. Arch Neurol Psychiatr 54: 323–338.

Benson DF, Miller BL, and Signer SF (1986). Dual personality associated with epilepsy. Arch Neurol 43(5): 471–474.

Bentall RP (1990). The illusion of reality: A psychological model of hallucinations. Psychol Bull 107: 82–95.

Berson RJ (1983). Capgras' syndrome. Am J Psychiatry 140(8): 969–978.

Blackmore SJ (1982). Beyond the Body. An Investigation of Out-Of-Body Experiences. Heinemann, London.

Blanke O (2004). Illusions visuelles. In: AB Safran, A Vighetto, T Landis, E Cabanis (Eds.), Neurophtalmologie. Masson, Paris, pp. 147–150.

Blanke O and Arzy S (2005). The out-of-body experience: Disturbed self-processing at the temporo-parietal junction. Neuroscientist 11(1): 16–24.

Blanke O and Mohr C (2005). Out-of-body experience, heautoscopy, and autoscopic hallucination of neurological origin. Implications for neurocognitive mechanisms of corporeal awareness and self-consciousness. Brain Res Brain Res Rev 50(1): 184–199.

Blanke O, Landis T, Spinelli L, et al. (2004). Out-of-body experience and autoscopy of neurological origin. Brain 127(Pt 2): 243–258.

Blanke O, Ortigue S, Coeytaux A, et al. (2003). Hearing of a presence. Neurocase 9(4): 329–339.

Blanke O, Ortigue S, Landis T, et al. (2002). Stimulating illusory own-body perceptions. Nature 419(6904): 269–270.

Bonnier P (1904). Le Vertige Masson, Paris.

Botvinick M and Cohen J (1998). Rubber hands 'feel' touch that eyes see. Nature 391(6669): 756.

Bradford D (2005). Autoscopic hallucinations and disordered self-embodiment. Acta Neuropsychologica 3: 120–189.

Brandt T and Dieterich M (1999). The vestibular cortex. Its location, functions, and disorders. Ann NY Acad Sci 871: 293–312.

Bremmer F, Schlack A, Duhamel JR, et al. (2001). Space coding in primate posterior parietal cortex. Neuroimage 14(1 Pt 2): S46–S51.

Brugger P (2002). Reflective mirrors: Perspective taking in autoscopic phenomena. Cogn Neuropsychol 7: 179–194.

Brugger P (2005). From mirror neurons to mirror. Acta Neuropsychologica 3: 190–201.

Brugger P, Agosti R, Regard M, et al. (1994). Heautoscopy, epilepsy, and suicide. J Neurol Neurosurg Psychiatry 57: 838–839.

Brugger P, Blanke O, Regard M, et al. (2006). Polyopic heautoscopy: Case report and review of the literature. Cortex 42(5): 666–674.

Brugger P, Kollias SS, Müri RM, et al. (2000). Beyond remembering: Phantom sensations of congenitally absent limbs. Proc Nat Acad Sci 97: 6167–6772.

Brugger P, Regard M, and Landis T (1996). Unilaterally felt presences: The neuropsychiatry of one's invisible doppelgänger. Neuropsychiatry Neuropsychol Behav Neurol 9: 114–122.

Brugger P, Regard M, and Landis T (1997). Illusory reduplication of one's own body: Phenomenology and classification of autoscopic phenomena. Cogn Neuropsychol 2: 19–38.

Brugger P, Regard M, Landis T, et al. (1999). Hallucinatory experiences in extreme-altitude climbers. Neuropsychiatry Neuropsychol Behav Neurol 12(1): 67–71.

Bünning S and Blanke O (2005). The out-of-body experience: Precipitating factors and neural correlates. Prog Brain Res 150: 331–350.

Bychowski G (1943). Disorders of the body-image in the clinical pictures of psychoses. J Nerv Ment Dis 97: 310–335.

Capgras J and Reboul-Lachaux J (1923). L'illusion des "sosies" dans un delire systématisé chronique. Bull Soc Clin Med Ment 11: 6–16.

Carlson DA (1977). Dream mirrors. Psychoanal Q 46(1): 38–70.

Carp E (1952). Troubles de l'image du corps. Acta Neurologica et Psychiatrica Belgica 52: 461–475.

Cereda C, Ghika J, Maeder P, et al. (2002). Strokes restricted to the insular cortex. Neurology 59(12): 1950–1955.

Chadwick PD and Lowe CF (1994). A cognitive approach to measuring and modifying delusions. Behav Res Ther 32(3): 355–367.

Christodoulou GN (1978a). Course and prognosis of the syndrome of doubles. J Nerv Ment Dis 166(1): 68–72.

Christodoulou GN (1978b). Syndrome of subjective doubles. Am J Psychiatry 135(2): 249–251.

Christodoulou GN (1991). The delusional misidentification syndromes. Br J Psychiatry Suppl(14): 65–69. Au10

Cohen H and Jones HW (1943). Reference of cardiac pain to a phantom left arm. Brit Heart J 2: 67.

Cole MG, Dowson L, Dendukuri N, et al. (2002). The prevalence and phenomenology of auditory hallucinations among elderly subjects attending an audiology clinic. Int J Geriatr Psychiatry 17(5): 444–452.

Comar G (1901). L'autorepresentation de l'organisme chez quelques hysteriques. Rev Neurol 9: 490–495.

Cornblath WT, Butter CM, Barnes LL, et al. (1998). Spatial characteristics of cerebral polyopia: A case study. Vision Res 38(24): 3965–3978.

Critchley M (1950). The body-image in neurology. Lancet 1: 335–340.

Critchley M (1953). The parietal lobes. Edward Arnold, London.

Critchley M (1954). Parietal syndromes in ambidextrous and left-handed subjects. Zentralbl Neurochir 14(1–2): 4–16.

Critchley M (1955). The idea of a presence. Acta Psychiatr Neurol Scand 30(1–2): 155–168.

Critchley M (1979). The Divine Banquet of the Brain and other Essays. Raven Press, New York.

Crookall R (1964). More Astral Projections. Analyses of Case Histories. Aquarian Press, London.

Cutting J (1978). Study of anosognosia. J Neurol Neurosurg Psychiatry 41: 548–555.

David AS (1999). Auditory hallucinations: Phenomenology, neuropsychology and neuroimaging update. Acta Psychiatr Scand Suppl 395: 95–104.

David M, Hécaen H, Passouant P, et al. (1946). Asomatognosie partielle et algie paroxystique, seuls signes cliniques d'un angiome pariétal partiellement calcifié. Guérison après extirpation chirurgicale. Rev Neurol 78: 236–238.

Decety J and Sommerville JA (2003). Shared representations between self and other: A social cognitive neuroscience view. Trends Cogn Sci 7(12): 527–533.

Dening TR and Berrios GE (1994). Autoscopic phenomena. Br J Psychiatry 165(6): 808–817.

Devinsky O, Feldmann E, Burrowes K, et al. (1989a). Autoscopic phenomena with seizures. Arch Neurol 46(10): 1080–1088.

Devinsky O, Putnam F, Grafman J, et al. (1989b). Dissociative states and epilepsy. Neurology 39(6): 835–840.

Dewhurst K and Pearson J (1955). Visual hallucinations of the self in organic disease. J Neurol Neurosurg Psychiatry 18(1): 53–57.

di Pellegrino G, Ladavas E, and Farne A (1997). Seeing where your hands are. Nature 388(6644): 730.

Dorpat TL (1971). Phantom sensations of internal organs. Comprehen Psychiatr 12(1): 27–35.

Duhamel JR, Colby CL, and Goldberg ME (1998). Ventral intraparietal area of the macaque: Congruent visual and somatic response properties. J Neurophysiol 79(1): 126–136.

Du Prel C (1886). Der Doppelgänger. Monatsschrift der übersinnlichen Weltanschauung. 1. Jahrgang.

Ehrenwald C (1931). Anosognosie und Depersonalisation. Nervenarzt 4: 681–688.

Ehrenwald H (1930). Verändertes Erleben des Körperbildes mit konsekutiver Wahnbildung bei linksseitiger Hemiplegie. Zeitschr Neurol 118: 89–97.

Ehrsson HH, Spence C, and Passingham RE (2004). That's my hand! Activity in premotor cortex reflects feeling of ownership of a limb. Science 305(5685): 875–877.

Ehrsson HH, Holmes NP, and Passingham RE (2005). Touching a rubber hand: Feeling of body ownership is associated with activity in multisensory brain areas. J Neurosci 25(45): 10564–10573.

Eliade M (1951/1964). Shamanism. Archaic techniques of ecstasy. Routledge & Kegan Paul, London.

Engerth G and Hoff H (1929). Ein Fall von Halluzinationen im hemianoptischen Gesichtsfeld. Beitrag zur Genese der optischen Halluzinationen. Monatsschr Psychiatr Neurol 74: 246–256.

Feinberg TE, Haber LD, and Leeds NE (1990). Verbal asomatognosia. Neurology 40(9): 1391–1394.

Feinberg TE and Roane DM (2005). Delusional misidentification. Psychiatr Clin North Am 28(3): 665–683, 678–669.

Feinberg TE, Roane DM, and Ali J (2000). Illusory limb movements in anosognosia for hemiplegia. J Neurol Neurosurg Psychiatry 68(4): 511–513.

Feinberg TE and Shapiro TE (2000). Misidentification-Reduplification and the right hemisphere. Neuropsychiatr Neuropsychol Behav Neurol 2(1): 39–48.

Féré C (1891). Note sur les hallucinations autoscopiques ou spéculaires et sur les hallucinations altruistes. Comptes Rendues Hebdomedaires des Séances et Mémoirs de la Société de Biologie 3: 451–453.

Foley JM and Breslau L (1982). A new syndrome of delusional misidentification. Ann Neurol 12: 76.

Foxe JJ, Wylie GR, Martinez A, et al. (2002). Auditory-somatosensory multisensory processing in auditory association cortex: An fMRI study. J Neurophysiol 88(1): 540–543.

Fredericks JAM (1969). Disorders of the body schema. In: PJ Vinken, GW Bruyn (Eds.), Disorders of Speech, Perception, and Symbolic Behavior. Amsterdam, North Holland, pp. 207–240.

Frith C (1996). The role of the prefrontal cortex in self-consciousness: The case of auditory hallucinations. Philos Trans R Soc Lond B Biol Sci 351(1346): 1505–1512.

Frith CD and Done DJ (1988). Towards a neuropsychology of schizophrenia. Br J Psychiatry 153: 437–443.

Gallagher II (2000). Philosophical conceptions of the self: implications for cognitive science. Trends Cogn Sci 4(1): 14–21.

Genner T (1947). Das Sehen des eigenen Spiegelbildes als epileptisches Äquivalent. Wien Klin Wochenschr 59: 656–658.

Gerstmann J (1942). Problem of imperception of disease and of impaired body territories with organic lesions. Arch Neurol Psychiatry 48: 890–913.

Gloning I, Gloning K, and Weingarten K (1954). Der Einfluss von kinästhetischen Impulsen auf Körperschemastörungen. Wien Z Nervenheilk 9: 481–495.

Gloning I, Gloning K, and Weingarten K (1957). [A case of corporeal metamorphognosy]. Wien Z Nervenheilkd Grenzgeb 14(2–3): 228–235.

Gloning IG, Jellinger K, and Tschabiter H (1963). Über einen obduzierten Fall von optischer Körperschemastörung und Heautoskopie. Neuropsychologia 1: 217–231.

Gloor P, Olivier A, Quesney LF, et al. (1982). The role of the limbic system in experiential phenomena of temporal lobe epilepsy. Ann Neurol 12(2): 129–144.

Gluckman LK (1968). A case of Capgras syndrome. Aust NZ J Psychiatr 2: 39–43.

Green CE (1968). Out-of-Body Experiences. Hamish Hamilton, London.

Grüsser OJ and Landis T (1991). The splitting of "I" and "me": Heautoscopy and related phenomena. In: OJ Grüsser, T Landis (Eds.), Visual Agnosias and other Disturbances of Visual Perception and Cognition. MacMillan, Amsterdam, pp. 297–303.

Guldin WO and Grüsser OJ (1988). Is there a vestibular cortex? Trends Neurosci 21(6): 254–259.

Halgren E, Walter RD, and Crandall PH (1983). Experiential phenomena of temporal epilepsy. Ann Neurol 14(1): 93–94.

Halgren E, Walter RD, Cherlow DG, et al. (1978). Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. Brain 101(1): 83–117.

Hall PF (1918). Experiments in astral projection. J Am Soc Psychical Res 12: 39–60.

Halligan PW (1995). Phantom limbs: The body in mind. Cogn Neuropsychol 7: 251–268.

Halligan PW (2002). Phantom limbs: The body in mind. Au9 Cogn Neuropsychiatr 7: 251–268.

Halligan PW, Marshall JC, and Wade DT (1993). Three arms: A case study of supernumerary phantom limb after right hemisphere stroke. J Neurol Neurosurg Psychiatry 56(2): 159–166.

Havens LL (1962). The placement and movement of hallucinations in space: Phenomenology and theory. Int J Psychoanal 43: 426–435.

Hécaen H and Ajuriaguerra J (1952). L'Heautoscopie, Meconnassiances et hallucinations corporelles, Masson, Paris, pp. 310–343.

Hécaen HD and David M (1945). Syndrome pariétal traumatique: Asymbolie tactile et hémiasomatognosie paroxystique et douloureuse. Rev Neurol 77: 113–124.

Heintel H (1965). Heautoskopie bei traumatischer Psychose. Zugleich ein Beitrag zur Phänomenologie der Heautoskopie. Arch Psychiatr Nervenkrank 206: 727–735.

Hermann BP and Chhabria S (1980). Interictal psychopathology in patients with ictal fear. Examples of sensory-limbic hyperconnection? Arch Neurol 37(10): 667–668.

Hoff H (1931). Zur Frage der formalen Gestaltung optischer Halluzinationen im hemianopischen Gesichtsfeld Zeit ges. Neurol Psychiatr 137: 453–457.

Hoff H and Pötzl O (1935/1988). Transformations between body image and external world. In: JW Brown (Ed.), Agnosia and Apraxia: Selected Papers of Liepmann, Lange, and Pötzl. Lawrence Erlbaum, Hillsdale, NJ, pp. 251–262.

Hoffmann RE (1986). Verbal hallucinations and language production processes in schizophrenia. Behav Brain Sci 9: 503–548.

Holmes NP and Spence C (2004). The body schema and the multisensory representation(s) of peripersonal space. Cogn Process 5(2): 94–105.

Ionasescu V (1960). Paroxysmal disorders of the body image in temporal lobe epilepsy. Acta Psychiatr Scand 35: 171–181.

Irwin HJ (1985). Flight of Mind: A Psychological Study of the Out-Of-Body Experience. Scarecrow Press, Metuchen, NJ.

Isnard J, Guenot M, Ostrowsky K, et al. (2000). The role of the insular cortex in temporal lobe epilepsy. Ann Neurol 48: 614–623.

Isnard J and Maugière F. (2005). [The insula in partial epilepsy]. Rev Neurol 161: 17–26.

James W (1961). The Variety of Religious Experience. Coller McMillan, New York.

Jaspers K (1913). Über leibhaftige Bewusstheiten (Bewusstheitstäuschungen), ein psychopathologisches Elementarsymptom. Zeitschrift für Psychopathologie 2: 150–161.

Junginger J and Frame CL (1985). Self-report of the frequency and phenomenology of verbal hallucinations. J Nerv Ment Dis 173(3): 149–155.

Kamanitz JR, El-Mallakh RS, and Tasman A (1989). Delusional misidentification involving the self. J Nerv Ment Dis 177: 695–698.

Kellehear A (1990). The near-death experience as status passage. Soc Sci Med 31(8): 933–939.

Khazaal Y, Zimmermann G, and Zullino DF (2005). [Depersonalization–current data]. Can J Psychiatry 50(2): 101–107.

Kölmel HW (1985). Complex visual hallucinations in the hemianopic field. J Neurol Neurosurg Psychiatry 48(1): 29–38.

Kurth W (1941). Pseudohalluzination bei organischen Krankheiten. Acta Psychiatrica Nervenkr 112: 90–100.

Lackner JR (1988). Some proprioceptive influences on the perceptual representation of body shape and orientation. Brain 111: 281–297.

Làdavas E (2002). Functional and dynamic properties of visual peripersonal space. Trends Cogn Sci 6(1): 17–22.

Làdavas E, Pavani F, and Farne A (2001). Auditory peripersonal space in humans: A case of auditory-tactile extinction. Neurocase 7(2): 97–103.

Lance JW (1976). Simple formed hallucinations confined to the area of a specific visual field defect. Brain 99(4): 719–734.

Leiguarda R, Starkstein S, Nogues M, et al. (1993). Paroxysmal alien hand syndrome. J Neurol Neurosurg Psychiatry 56(7): 788–792.

Leischner A (1961). [Autoscopic hallucinations (heautoscopy).]. Fortschr Neurol Psychiatr 29: 550–585.

Ley H and Stauder KH (1950). Zur Neurologie und Psychopathologie des Morbus Bang. Zugleich ein Beitrag zum Phänomen der sogenannten "Ichverdoppelung." Arch Psychiatr Zeit Neurol 183: 564–580.

Lhermitte J (1939). Les phenomènes héautoscopiques, les hallucinations spéculaires et autoscopiques. In: L'image de notre corps. L'Harmattan, Paris, pp. 170–227.

Lhermitte J (1951). Les phenomènes héautoscopiques, les hallucinations spéculaires. In: G Doin (Ed.), Les Hallucinations. Clinique et Physiopathologie, Cie, Paris, pp. 124–168.

Lippman CW (1953). Hallucinations of physical duality in migraine. J Nerv Ment Dis 117(4): 345–350.

Lukianowicz N (1958). Autoscopic phenomena. AMA Arch Neurol Psychiatry 80(2): 199–220.

Lukianowicz N (1960). Visual thinking and similar phenomena. J Ment Sci 106: 979–1001.

Lunn V (1970). Autoscopic phenomena. Acta Psych Scand 46(Suppl. 219): 118–125.

Maack LH and Mullen PE (1983). The doppelganger, disintegration and death: A case report. Psychol Med 13(3): 651–654.

Magri R and Mocchetti E (1967). [Partial asomatoscopy (negative autoscopy) in epileptics. Nosographic classification and clinical contribution]. Arch Psicol Neurol Psichiatr 28(6): 572–585.

Mahaluf J, Canales G, and Cattenaci M (1987). Heautoscopia: Description de un caso clinico. Revue Psychiatrico Clinico 24: 35–39.

Maupassant G de (1986/1961). Le Horla. In: E Boyd, S Jameson (Eds.), 88 more stories by Guy de Maupassant. Cassell, London.

Maximov K (1973). Epilepsie occipitale avec hallucinations héautoscopiques. Acta Neurol Belg 73: 320–323.

Mayer-Gross W (1928). Psychopathologie und Klinik der Trugwahrnehmungen. In: O Bumke (Ed.), Handbuch der Geisteskrankheiten, Vol. I, Pt 1. Berlin, Springer, pp. 427–507.

McCulloch WH (1992). A certain archway: Autoscopy and its companions seen in Western writing. Hist Psychiatr 3(9): 59–78.

McGuire PK, Silbersweig DA, Wright I, et al. (1995). Abnormal monitoring of inner speech: A physiological basis for auditory hallucinations. Lancet 346(8975): 596–600.

McKay CM, Headlam DM, and Copolov DL (2000). Central auditory processing in patients with auditory hallucinations. Am J Psychiatry 157(5): 759–766.

Melzack R (1990). Phantom limbs and the concept of a neuromatrix. Trends Neurosci 13: 88–92.

Menninger-Lerchenthal E (1935). Das Truggebilde der eigenen Gestalt. Karger, Berlin.

Menninger-Lerchenthal E (1946). Der eigene Doppelgänger Bern.

Messner R (1980). Alleingang Nanga Parbat. Munchen, Knaur.

Metzinger T (2003). Being No One MIT Press, Cambridge.

Metzinger T (2005). Out of body experiences as the origin of the concept of a "soul." Mind and Matter 3: 57–84.

Mohr C and Blanke O (2005). The demystification of autoscopic phenomena. Experimental propositions. Curr Psychiatr Rep 7: 189–195.

Muldoon S and Carrington H (1929). The projection of the astral body. Rider, London.

Müller J (1826). Über phantastische Gesichtserscheinungen. Verlag, Koblenz, 79.

Naudascher MG (1910). Trois cas d'hallucinations spéculaires. Ann Médico-Psychol 68: 284–296.

Nayani TH and David AS (1996). The auditory hallucination: A phenomenological survey. Psychol Med 26(1): 177–189.

Newport R, Hindle JV, and Jackson SR (2001). Links between vision and somatosensation. Vision can improve the felt position of the unseen hand. Curr Biol 11(12): 975–980.

Nightingale S (1982). Somatoparaphrenia: A case report. Cortex 18(3): 463–467.

Nouët H (1923). Hallucination speculaire et traumatisme crânien. Encephale 18: 327–329.

Oesterreich TK (1910). Die Phänomenologie des Ich in ihren Grundproblemen. Barth, Leipzig.

Ostrowsky K, Isnard J, Ryvlin P, et al. (2000). Functional mapping of the insular cortex: Clinical implication in temporal lobe epilepsy. Epilepsia 41: 681–686.

Pavani F, Làdavas E, and Driver J (2003). Auditory and multisensory aspects of visuospatial neglect. Trends Cogn Sci 7(9): 407–414.

Pearson J and Dewhurst K (1954). [Two cases of heautoscopic phenomena following organic lesions.] Encephale 43(2): 166–172.

Penfield W and Jaspers H (1954). Epilepsy and the Functional Anatomy of the Human Brain. Churchill, London.

Penfield W and Perot P (1963). The brain's record of auditory and visual experience. A final summary and discussion. Brain 86: 595–696.

Perrin F, Maquet P, Peigneux P, et al. (2005). Neural mechanisms involved in the detection of our first name: A combined ERPs and PET study. Neuropsychologia 43(1): 12–19.

Peto A (1969). Terrifying eyes. A visual superego forerunner. Psychoanal Study Child 24: 197–212.

Podoll K and Robinson D (1999). Out-of-body experiences and related phenomena in migraine art. Cephalalgia 19(10): 886–896.

Pötzl O (1925). Über Störungen der Selbstwahrnehmung bei linksseitiger Hemiplegie. Zeit ges Neurol Psychiatr 93: 117–168.

Ramachandran VS and Hirstein W (1998). The perception of phantom limbs. The DO Hebb lecture. Brain 121(Pt 9): 1603–1630.

Ramachandran VS, Rogers-Ramachandran D, and Cobb S (1995). Touching the phantom limb. Nature 377(6549): 489–490.

Rank O (1925). Der Doppelgänger. Eine psychoanalytische Studie. Internationaler Psychoanalytischer Verlag, Leipzig.

Röhrenbach C and Landis T (1995). Dreamjourneys: Living in woven realities, the syndrome of reduplicative paramnesia. In: R Campbell, MA Conway (Eds.), Broken memories: Case studies in memory impairment. Blackwell, Oxford, pp. 93–99.

Schilder P (1914). Über Autoskopie, über die Lokalisation des Denkens und über die "Ichverdoppelungen" der Hysterie. In: Selbstbewusstsein und Persönlichkeitsbewusstsein. Eine psychopathologische Studie. Springer, Berlin, pp. 229–246.

Schilder P (1935). The Image and Appearance of the Human Body. London, Georg Routledge and Sons, Regan Paul Trench, Trubner & Co.

Schroter-Kunhardt M (2002). [Heautoscopy. Capgras phenomenon and rare hallucinations of own being. Comments on the contribution by D. Arenz]. Nervenarzt 73(3): 298–299; author reply 299.

Sheils D (1978). A cross-cultural study of beliefs in out-of-the-body experiences, waking and sleeping. J Soc Psychol Res 49: 697–741.

Shelley BP and Trimble MR (2004). The insular lobe of Reil – its anatamico-functional, behavioural and neuropsychiatric attributes in humans – a review. World J Biol Psychiatry 5: 176–200.

Sherrard C (1978). The Everest message. J Soc Psychical Res 49: 797–804.

Sierra M and Berrios GE (1998). Depersonalization: Neurobiological perspectives. Biol Psychiatr 44(9): 898–908.

Sierra M, Lopera F, Lambert MV, et al. (2002). Separating depersonalisation and derealisation: The relevance of the "lesion method." J Neurol Neurosurg Psychiatry 72(4): 530–532.

Signer SF (1987). Capgras' syndrome: The delusion of substitution. J Clin Psychiatry 48(4): 147–150.

Silva JA and Leong GB (1991). A case of "subjective" Fregoli syndrome. J Psychiatry Neurosci 16(2): 103–105.

Silva JA, Leong GB, and Weinstock R (1993). Delusions of transformation of the self. Psychopathology 26(3–4): 181–188.

Simeon D, Guralnik O, Hazlett EA, et al. (2000). Feeling unreal: A PET study of depersonalization disorder. Am J Psychiatry 157(11): 1782–1788.

Simeon D. (2004). Depersonalisation disorder: A contemporary overview. CNS Drugs 18: 343–354.

Sims A (1986). Psychopathology of schizophrenia with special reference to delusional misidentification. Bibl Psychiatr 164: 30–39.

Sinclair DC, Weddell G, and Feindel WH (1948). Referred pain and associated phenomena. Brain 71: 184–209.

Skworzoff K (1931). Doppelgänger-Halluzinationen bei Kranken mit Funktionsstörungen des Labyrinths. Zeitschr Nervenheilk 133: 762–766.

Smythe FS (1934). Everest 1933. Hodder & Stoughton, London.

So EL and Schauble BS (2004). Ictal asomatognosia as a cause of epileptic falls: Simultaneous video, EMG, and invasive EEG. Neurology 63(11): 2153–2154.

Sollier P (1903a). Les phénomènes d'autoscopie. Félix Alcan, Paris.

Sollier P (1903b). L'autoscopie interne. Revue Philosophique 55: 1–41.

Staudenmaier L (1912/1968). Die Magie als experimentelle Naturwissenschaft. In: Darmstadt Wissenschaftliche Buchgesellschaft, 106.

Stein BE, Meredith MA, and Wallace MT (1993). The visually responsive neuron and beyond: Multisensory integration in cat and monkey. Prog Brain Res 95: 79–90.

Stephane M, Barton S, and Boutros NN (2001). Auditory verbal hallucinations and dysfunction of the neural substrates of speech. Schizophr Res 50(1–2): 61–78.

Strauss EW (1962). Phenomenology of hallucinations. In: LJ West (Ed.). Hallucinations. Grune and Stratton, New York.

Strindberg A (1897). Inferno. Penguin Books, New York.

Suedfeld P and Mocellin JS (1987). The sensed presence in unusual environment. Environ Behav 19: 33–52.

Todd J and Dewhurst K (1955). The double: Its psychopathology and psycho-physiology. J Nerv Ment Dis 122(1): 47–55.

Todd J and Dewhurst K (1962). The significance of the Doppelganger (hallucinatory double) in folk-lore and neuropsychiatry. Practitioner 188: 377–382.

von Stockert FG (1934). Lokalisation und klinische Differenzierung des Symptoms der Nichtwahrnehmung einer Körperhälfte. Deut Zeit Nervenheilk, 1–13.

Weinstein EA, Kahn RL, Malitz S, et al. (1954). Delusional reduplication of parts of the body. Brain 77(1): 45–60.

Williams D (1956). The structure of emotions reflected in epileptic experiences. Brain 79(1): 29–67.

Yram (1972). Practical Astral Projection. Samuel Weiser, New York.

Zamboni G, Budriesi C, and Nichelli P (2005). "Seeing oneself": A case of autoscopy. Neurocase 11(3): 212–215.

Zingerle H (1913). Über Störungen der Wahrnehmung des eigenen Körpers bei organischen Gehirnerkrankungen. Monatsschrift Psychiatr Neurol 34: 3–36.

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