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# Hearing of a Presence

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## Abstract

**Here we describe a patient with epilepsy (secondary to left parieto-temporal brain damage) suffering from the paroxysmal unilateral experience of hearing a person in her near extrapersonal space. The paroxysmal auditory experience was associated with a deficit in spatial auditory perception and other paroxysmal disorders of somatognosia. Based on these findings, it is suggested that the paroxysmal hearing of a person nearby corresponds to an auditory disorder of somatognosia.**

## Introduction

Paroxysmal disorders of the body image or somatognosia (paroxysmal somatognosic disorders, PSD) include a variety of usually short lasting, illusory experiences about the location and position of one's body or body parts in space (Menninger-Lerchenthal, 1935; Schilder, 1935; Lhermitte, 1939, 1951; Critchley, 1951; Hécaen, 1973; Landis, 1997). They generally occur in patients with posterior parietal or posterior temporal lobe dysfunction and are characterized by the illusion of (a) the absence of a body part (e.g. of an arm; Critchley, 1951; Hécaen and Ajuriaguerra, 1952), (b) the transformation of a body part (such as shrinking or extension; Hécaen and Ajuriaguerra, 1952; Ionasescu, 1959; Lunn, 1970; Nithingale, 1982; Landis, 1997), (c) the dislocation or disconnection of a body part (Critchley, 1951; Hécaen and Ajuriaguerra, 1952), (d) phantom limbs and supernumerary limbs (i.e. Menninger-Lerchenthal, 1935; Hécaen and Ajuriaguerra, 1952; Mayeux and Benson, 1979; Halligan *et al.*, 1993; Landis, 1997; Ramachandran and Hirstein, 1998; Brugger *et al.*, 2000), (e) autoscopia (seeing an image of one's own body; i.e. Menninger-Lerchenthal, 1935; Hécaen and Ajuriaguerra, 1952; Dewhurst and Pearson, 1955; Kamiya and Okamoto, 1982; Brugger *et al.*, 1997; Landis, 1997; Blanke *et al.*, 2002), and (f) the "feeling of a presence" (vivid feeling that there is another person nearby; Jaspers, 1913; Critchley, 1951; Kamiya and Okamoto, 1982; Ardila and Gomez, 1988; Grüsser and Landis, 1991; Brugger *et al.*, 1996). Despite these phenomenological differences (ranging from whole body manifestations to isolated arm or leg manifestations), PSDs have been considered as representing one pathophysiological category and have been classified under the name of paroxysmal somatognosic disorders (PSD; Hécaen and Ajuriaguerra, 1952). This classification is based on their common functional association with touch, proprioception, and vision, as well as their common anatomical association with parietal and temporal lobe damage (Hécaen and

Ajuriaguerra, 1952; Lunn, 1970; Brugger *et al.*, 1997; Ramachandran and Hirstein, 1998).

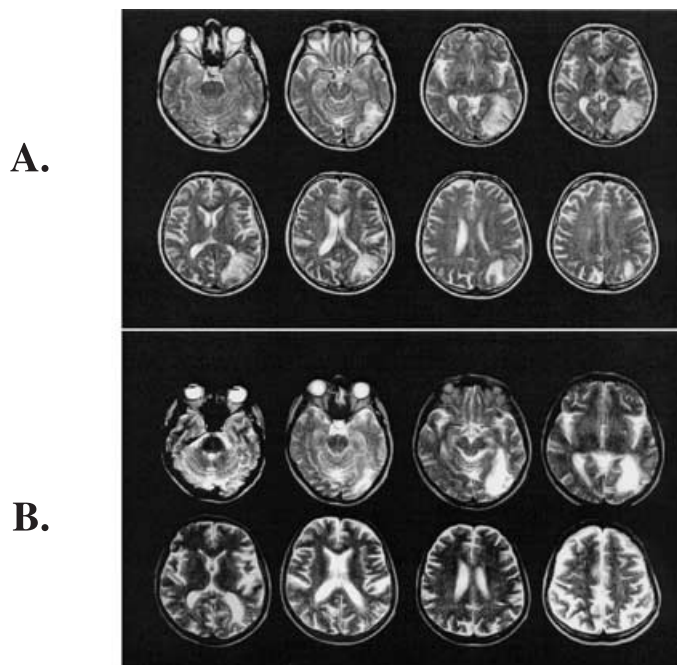
Interestingly, there are no descriptions of auditory PSDs. This is astonishing, since audition, like vision and somatosensation, is involved in the construction of the body image (Menninger-Lerchenthal, 1935; Làdavos, 2002). Moreover, electrophysiological studies in the macaque at the subcortical level (Sparks, 1986; Stein and Meredith, 1993) and in parietal and temporal cortex (Duhamel *et al.*, 1998; Bremmer *et al.*, 2001; Schroeder *et al.*, 2001) suggest that several cerebral areas combine auditory, tactile, proprioceptive and visual information in a coordinated reference frame for personal and peripersonal space. This is also suggested by neuroimaging work (Bremmer *et al.*, 2001; Foxe *et al.*, 2002) and neuropsychological studies (Làdavos *et al.*, 2001; Làdavos, 2002). It might thus be hypothesized that neurological damage to parieto-temporal areas might not only lead to visual and/or somatosensory forms of PSD, but also to auditory forms.

The present report describes a patient with epilepsy, following left parieto-temporal brain damage, suffering from the paroxysmal experience of hearing a person in the contralateral near extrapersonal space. We describe the phenomenology of the auditory experience, its association with other PSDs, its etiology and lesion location, and the associated neuropsychological deficits. Based on these results we argue that the persuasive hearing of a person nearby might represent an auditory PSD.

## Case report

### *Medical history*

This right-handed nun had been healthy up to the age of 65, when she was sent to our hospital for the sudden appearance of



**Fig. 1.** Lesion location. A. Depiction of left hemispheric brain damage caused by bleeding of probable hypertensive origin. T2 weighted MRI five years prior to paroxysmal body illusions (1996). The lesion includes the posterior part of the superior and middle temporal gyrus as well as the angular gyrus and the parieto-occipital junction. B. T2-weighted MRI showing brain damage during the actual symptomatology (2001). Note the absence of more recent brain damage and cortico-cortical atrophy in the left posterior hemisphere.

speech and vision difficulties. The neurological and neuropsychological exams found a right complete hemianopia and pure alexia. The MRI showed a hematoma at the left parieto-temporo-occipital junction (Fig. 1A) and EEG focal slowing over the left parieto-temporal region. Hemianopia and pure alexia recovered within four months.

One year later (1997) the patient returned to our clinic for complex partial seizures with secondary generalization. She suffered from severe alexia, moderate visual agnosia and right-sided complete hemianopia. Computer tomography and MRI were not modified and only showed left posterior atrophy concordant with the vascular brain damage in 1996. The EEG revealed focal slowing over the left parieto-temporal region. Antiepileptic treatment was initiated (phenytoine, 200 mg/d). She had no further seizures during hospitalization and her alexic and visual symptoms diminished. However, her seizures became more frequent and she was rehospitalized for repetitive complex partial seizures with secondary generalization and aggravation of her alexic and visual deficits (right complete hemianopia). The MRI was unmodified, but the EEG showed sharp waves and spikes over the left parieto-temporal region. New antiepileptic medication (valproate 2000 mg/d) was prescribed with success.

In 2001 she was re-hospitalized for partial status epilepticus with frequent secondary generalizations. Initially she suffered from psychomotor agitation, disorientation and reduced vigilance. A CT scan and MRI did not show any new lesion (Fig. 1B). The lumbar puncture was normal. Prolonged post-

ictal status was assumed, although the EEG only showed focal slowing over the left parieto-temporal region. Intravenous and oral anti-epileptic treatment was started and her clinical condition improved rapidly. However, moderately severe aphasia with semantic and phonological paraphasias, strong alexia and moderate agraphia were found and only slowly evolved into pure persisting alexia (for detailed neuropsychological examination see below). Visual field testing by automatic perimetry found a partial right inferior quadrantanopia. The neurological examination did not reveal any sensori-motor deficits. Because of complex multisensory experiences (described below) 24-hour Video-EEG was performed, showing frequent interictal activity characterized by spike-waves, sharp waves and slow waves over the left mid-to-posterior temporal region (Fig. 2A). In one instance, rhythmic discharges over the occipito-temporal region (Fig. 2B) were noted. During this latter period there were no observable clinical signs or sensory manifestations.

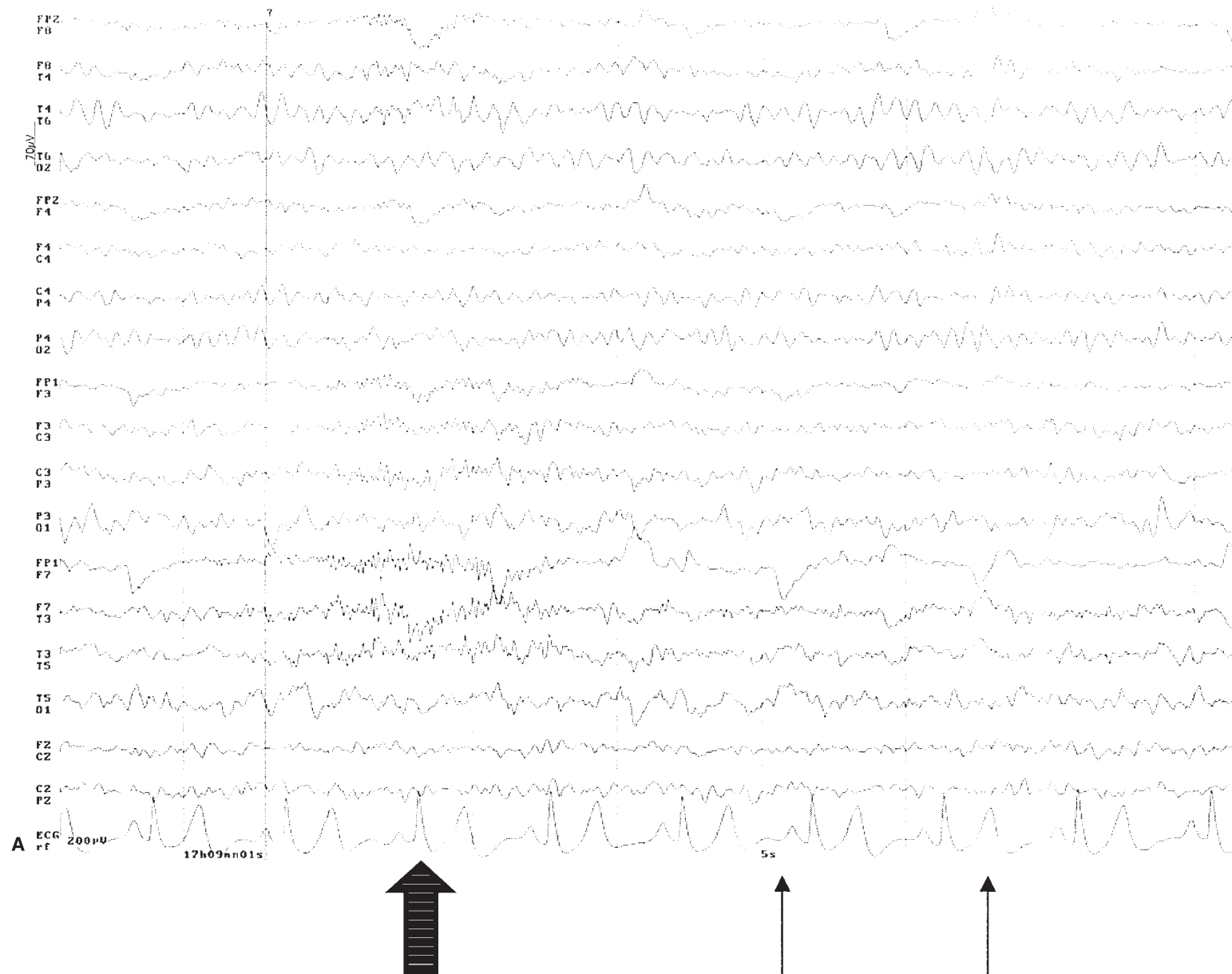
## Paroxysmal experiences

### *Hearing of a presence (HP)*

The patient suffered from complex auditory manifestations characterized by the impression of the physical presence of one or two people behind her that were talking. During the most impressive and longest episode, she was sitting in the hospital church during the religious service 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 (Fig. 3A). Their speech was incomprehensible, and she did not know what they were saying. She could not indicate the gender of the “people”, nor their age, nor whether their voices were characterized by any emotional state. However, she became progressively annoyed by their continuous whispering. Since their conversation did not end, she finally turned around to tell them to be quiet. However, to her surprise there was no one sitting behind her. After having turned back the patient was extremely irritated, since the experience still continued and did so until she finally got up and left the hospital church.

She reported similar experiences in her hospital room. While sitting in her chair, she often had the sudden feeling as if someone were standing behind the chair talking to her. The “person” was heard or experienced to her right. The words were incomprehensible and the “person” was whispering. Although she had not heard anyone enter the room, the feeling of a physically present “person” talking to her was so strong that she turned around in order to see whether in fact anyone was there. As before, no one could be seen. The patient could not indicate the gender of the heard “person”, nor his/her age, nor whether the voices were characterized by a particular emotion.

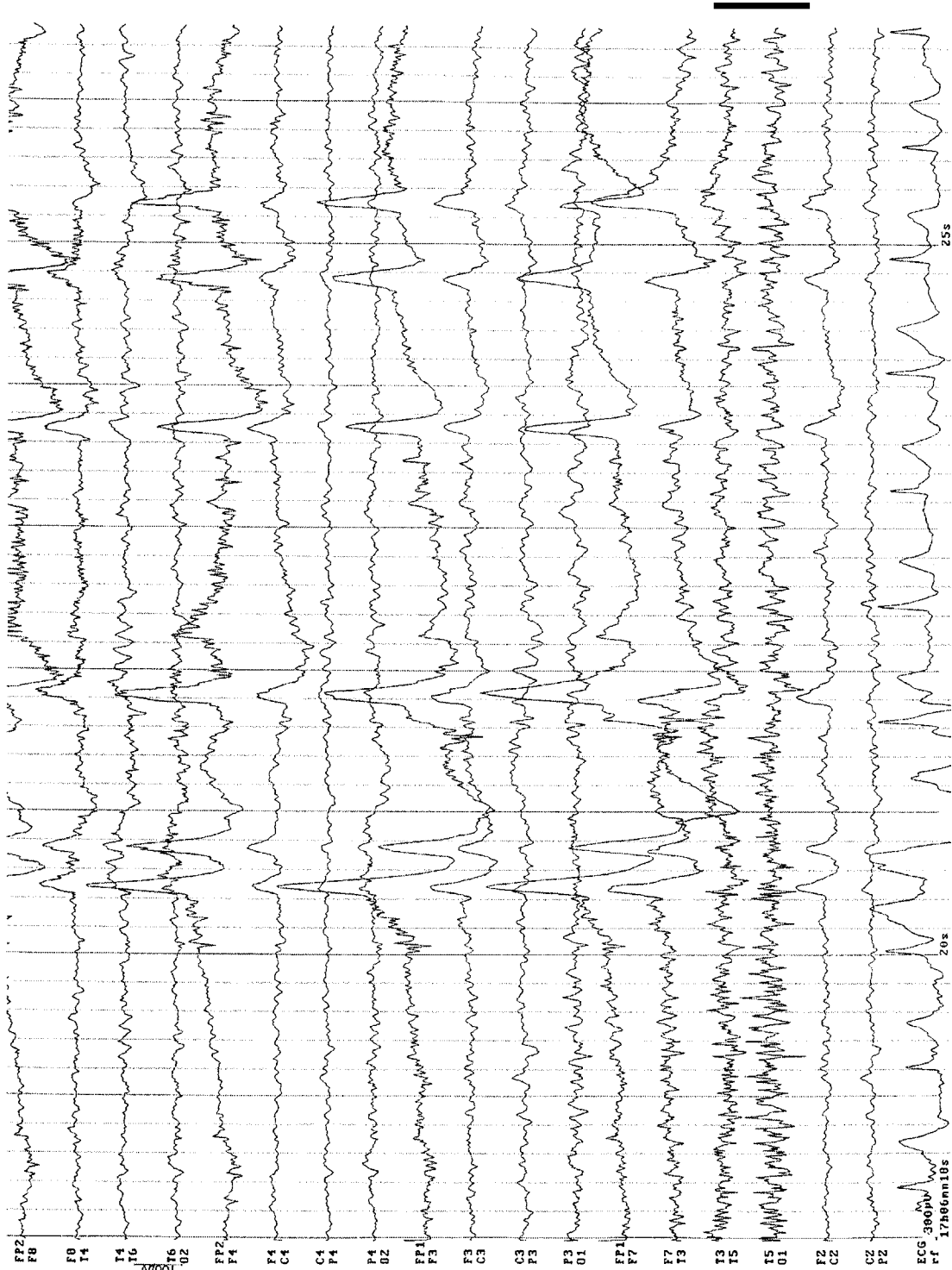
Finally, for approximately a week during hospitalization and several times daily, she suffered from simple auditory hallucinations characterized by humming or buzzing. These



**Fig. 2.** Location of epileptogenic discharges. A. Focal spikes (thick arrow) and slow waves (thin arrow) over the left temporal region (electrodes T3-T5-F7). EEG was recorded with 19 electrodes positioned according to the 10–20 system (Deltamed equipment) during 24-hour continuous Video-EEG recording. EEG is shown in a bipolar montage. B (1.2.) shows prolonged focal discharges (vertical bar) over the left temporo-occipital region (electrodes T5-O1, T3-T5; onset indicated by a thick arrow) preceded by a generalized flattening (onset is indicated by a thin arrow). There were no observable clinical changes during this period, nor sensory manifestations indicated by the patient.



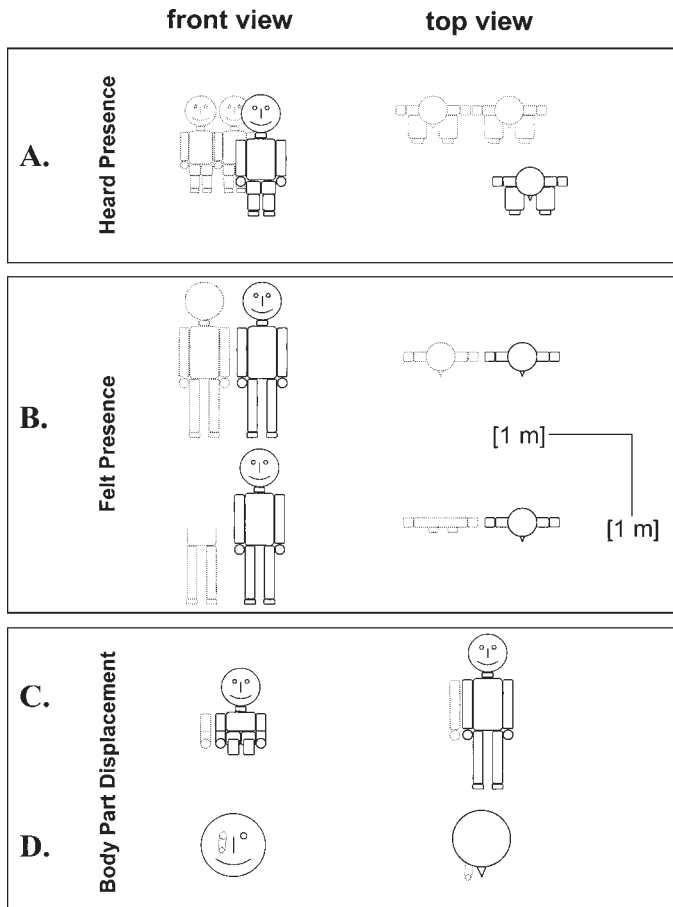
Fig. 2. (continued)



**B.2**

Fig. 2. (continued)

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**Fig. 3.** Graphical representation of the different paroxysmal body illusions. A. Heard presence. B. Felt presence. C. Arm dislocation. D. Eye deformity and displacement. All body illusions are shown in front view (left column) and in top view (right column). The illusory body/body parts are indicated by dashed lines, whereas the position and location of the real body is depicted in black lines. Approximate distance and location with respect to the real body is indicated. Body illusions are depicted in the (real) body positions in which they were experienced (A: felt presence, standing; B: heard presence, sitting; C: arm dislocation, lying in bed; D: eye dislocation, sitting (only the head is shown)).

sounds were experienced either on the right side (lateral or behind her) or bilaterally. She was sometimes able to localize the sounds as coming from behind the walls of her room or from a cupboard. Several times, the patient asked her roommate if she also heard the illusory sounds. The roommate heard no such sounds.

### *Feeling of presence (FP)*

Several times per day the patient experienced the presence of a person, which she described as “a shadow”. She stated that most of the time “it is only the lower half of a person”, from the waist down (hips, legs and feet). Sometimes she experienced a complete human shadow. She described the figure as three-dimensional, not as an image, and grayish-black. At first, she was extremely frightened by this experience, and she preferred not to look directly at the “shadow”. It was always on her right side, never behind her, nor in front of her. Initially,

she could not say whether it was a human figure, but later thought it to be “a person” and a woman rather than a man. She could not say if the “person” had socks and shoes, or a dress. The size of the illusory legs was identical to hers. The distance between her and the shadow was short, but it did not touch her. The distance was always the same and approximately 20–30 cm (Fig. 3B). She only had this experience when she was standing and especially when she was walking. It was never felt when she was sitting in a chair or lying in bed. She not only felt that the “person” followed her, but had the strong impression that it moved only when she moved (with the same speed and direction) and stopped when she stopped walking. The patient explained that when she moved her right leg she experienced that the “person” also moved her right leg. Movements of her hands, mouth or tongue did not lead to any observable modifications of the figure. She had the experience both during the day and in the evening. There were no auditory or visual manifestations. The sensation was never accompanied by autoscopy, heautoscopy or the impression of leaving her body (out-of-body experience). There were no feelings of a doubled self or depersonalization. The patient was frightened by the experience and initially reluctant to talk about it. She refused to see a psychiatrist and realized the illusory character of the experience. However, she questioned a psychiatric origin.

### *Illusion of contralateral arm dislocation*

On several occasions she had the vivid feeling as if her right arm was disconnected from the rest of her body (Fig. 3C). This occurred while she was lying in bed either at night or in dimly lit rooms. The disconnection was experienced at the level of her shoulder and there was a distance of approximately 10–20 cm between her torso and her right illusory upper extremity. The illusion was very convincing, so she immediately needed to touch her real right arm with her left hand in order to ascertain its correct position. Often she was only sure about its real position once she had turned on the light and inspected it visually. Only after looking at her true right arm did the illusion disappear. The shape, length, weight, and position of her right illusory arm were not modified with respect to her real arm, nor was the relative position of the parts of the arm. There were no illusory movements of the right upper extremity. She did not feel any weakness or other sensory symptoms in her right arm or leg.

### *Illusion of contralateral eye deformity and displacement*

On rare occasions, the patient reported the experience as if her right eye was slowly and progressively falling out of its socket. Eventually she experienced her right eye as if hanging approximately 10 cm below and anterior to her right orbit (Fig. 3D). It was still connected with her head at the orbit. She checked the position of her right eye with her hand, and also inspected it in the mirror only to find it in its normal place.



There were no changes to her left eye or to the rest of her face. There was no vertigo, diplopia, or other visual changes.

### Detailed neuropsychological examination

The patient was spatially and temporally well oriented. Language abilities were characterized by severe alexia (letter-by-letter reading), moderate agraphia (omission and reduplication of letters) as well as a moderate aphasia with semantic and phonological paraphasias. Naming (Boston Naming: 5/20 correct) and semantical verbal fluency (3 words, 1 minute duration) were deficient. Further aphasia testing was normal (auditive reading: 24/25 correct; anagram: 11/12 correct; antithesis: 19/19 correct). There were no significant deficits in attention (d2 test), memory (digit span: percentile 20; Corsi block-tapping span: percentile 75; Rey's words: percentile 70; Door's test: percentile 75 (Baddeley *et al.*, 1994)) and executive functions (Trail making test, figural fluency). There were no signs of apraxia or of visual agnosia (Poppelreuter overlapping figures test; illusory contours and colors).

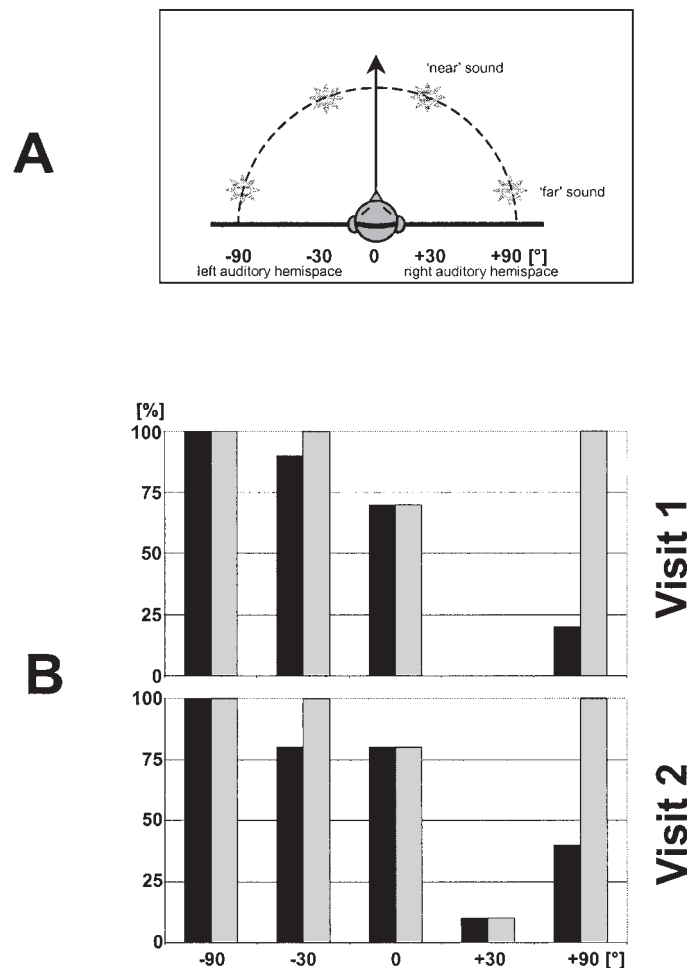
### Further clinical evolution and neuropsychological examination

Since being discharged from the hospital, one complex partial seizure, associated with postictal language deficits, occurred. She continued to suffer from HP and FP with a variable frequency ranging from 1–2 per month to 2 per week. HP and FP were always experienced on her right side. As the patient was mainly annoyed and only moderately frightened by both experiences she preferred to pay as little attention to them as possible. Nevertheless she indicated that she had HPs while she was at church, at a music concert, and at home. HPs were described as stereotyped (as above) and only encountered with the patient sitting down. Thus, the content of the HP consisted of one or two people, approximately one meter behind her and on her right, who uttered incomprehensible, whispered speech. The instances of FP were also described as above. Yet, the patient could not say whether the human-shaped “shadow” was complete or a partial “shadow” (see above). The patient only remembered instances of FP while she was standing or walking. No further details about the FP could be given by the patient.

A neuropsychological control examination was carried out 13 months after the first occurrence of HP and FP in 2001. This examination focused on auditory functions, but also controlled language and proprioception. The detection, discrimination, and recognition of sound sources was tested with the Montréal–Toulouse test (Protocole Montréal–Toulouse, évaluation des gnosies auditives, 1992). In an additional paradigm the patient's capacity to localize auditory stimuli in space was tested (see below).

With respect to auditory functions, a selective deficit for the localization of sound sources was found. The auditory-localization paradigm that was carried out in the present patient

has been applied in healthy subjects previously (Ducommun *et al.*, 2002). The paradigm and the results will be described first. The auditory stimuli were binaural white noise bursts (500 Hz low-pass filtered, 500 ms duration), digitized on a Power Macintosh 8100 fitted with an Audiomedia card II and running Sound Designer II and Protocol Powermix software. The sensation of stationary sound sources was created through interaural time differences (ITD). Stationary sounds were presented at five different locations (two in each auditory hemifield and one central stimulus; see Fig. 4A).



**Fig. 4.** Location discrimination of sound sources. A. Illustration that shows the positions of the auditory stimuli at five different eccentricities ( $-90^\circ$ ,  $-30^\circ$ ,  $0^\circ$ ,  $+30^\circ$ ,  $+90^\circ$ ). The sound sources (white noise bursts) were generated via interaural time differences. Midsagittal plane is shown by the vertical line. Stimuli were presented for 500 ms (for further details see text). B. Behavioral data for the localization of sound sources. Upper graph shows the results of the first examination of the patient, which were controlled two weeks later (lower graph). Correct performance (in %) is plotted on the y-axis for each of the five eccentricities ( $-90^\circ$  to  $+90^\circ$ ; x-axis). Black columns depict performance on the five-alternative forced choice task. Gray columns show results if performance is analyzed based on correct lateralization of responses (see text for further explanation). Note the similarity between both examinations (compare upper and lower graph) and that the auditory deficit was selective for the right contralesional hemisphere. Moreover, performance was better for far ( $+90^\circ$ ) than for near ( $+30^\circ$ ) contralesional sounds (compare especially gray columns at  $+30^\circ$  and  $+90^\circ$  eccentricity). No such difference was observed in the left ipsilesional space.

By introducing a constant phase lag between stimuli presented to each ear, we obtained sounds located near (ITD of 200  $\mu$ s) or far (ITD of 700  $\mu$ s) with respect to the midsagittal plane (no phase lag for central location). Healthy subjects perceive the lateral sound sources as being located at two different positions within each hemifield (approximately  $\pm 90^\circ$  and  $\pm 30^\circ$ ; where  $0^\circ$  is directly ahead), with the azimuth on the frontal interaural plane. Sounds were delivered through earphones at the intensity level judged to be most comfortable by the patient at the beginning of the experiment. Subjects (and the present patient) performed a five-alternative forced choice task ( $-90^\circ$ ,  $-30^\circ$ ,  $0^\circ$ ,  $+30^\circ$ ,  $+90^\circ$ ). In healthy subjects, performance is flawless for all five positions (Ducommun *et al.*, 2002) although subjects (and the present patient) judge the  $\pm 30^\circ$  positions to be more difficult than the  $\pm 90^\circ$  and  $0^\circ$  positions. Ten repetitions were carried out for each eccentricity and the order of appearance was randomized.

As shown in Fig. 4B (upper graph), localization of sound sources was severely deficient in the right hemispace (10% correct for both eccentricities), but normal in the left hemispace (95% correct, black columns). In addition, the deficit was stronger for sound sources that were presented at  $30^\circ$  eccentricity (0% correct) as compared to sound sources at a greater eccentricity in the right hemispace ( $90^\circ$ ; 20% correct; see black columns in Fig. 4B). The latter finding becomes more apparent if the percentage of correct responses is defined depending on whether the stimulus is correctly lateralized or centrally perceived (i.e. the response “ $+30^\circ$ ” for a “ $+90^\circ$ ” stimulus was counted as a correctly lateralized response, whereas the responses “ $0^\circ$ ”, “ $-30^\circ$ ”, “ $-90^\circ$ ” were counted as incorrect for the “ $+90^\circ$ ” stimulus; see gray columns in Fig. 4B). Thus, for both eccentricities in the left hemispace and  $+90^\circ$  in the right hemispace the performance was 100% correct. Yet, performance at  $+30^\circ$  was severely impaired and no stimulus was lateralized correctly (see Fig. 4B). These results were confirmed in a control examination carried out two weeks later (lower graph in Fig. 4B). The detection, discrimination, and recognition of auditory stimuli was tested with the Montréal-Toulouse Test. In this test, the discrimination of simple sounds is tested first by asking the patient to perform “same or different” judgements of two successively presented simple sounds (sound pair). The simple sounds within a pair may vary according to their pitch (1000 Hz or 1030 Hz), intensity (six decibel difference or equal to the first stimulus) or timbre (high frequency filtering at 2, 2.5, 3.15, 4, 5, 6.3, 8, and 10 Hz). The discrimination of pitch, intensity, and timbre is tested by presenting ten sound pairs for each auditory characteristic (three exemplary sound pairs are given prior to each test). Secondly, the patient is asked to perform “same or different” discriminations of two successively presented syllables (total of 40 pairs preceded by four exemplary sound pairs). This task assesses the patient’s ability to discriminate monosyllabic non-sense words (e.g. [it] versus [ut]). Thirdly, auditory recognition of familiar sounds is tested by a forced-choice paradigm. A total of twelve sounds is given (two exemplary sounds). The auditory signals are animal

sounds, music instruments, or everyday sounds (e.g. alarm clock). The patient indicates the response manually from a choice of four different cards. This task assesses the patient’s ability to identify and attribute a meaning to a familiar sound. The detection, discrimination, and recognition of auditory stimuli were normal (pitch, 100% correct; intensity, 80% correct; timbre, 90% correct; minimal phonological differences, 80% correct; auditory recognition, 100% correct).

Language deficits were characterized by persisting moderate alexia and mild agraphia. Naming was now in the normal range (Boston Naming: score: 19/20 correct). Dichotic listening (words) could only be carried out in French (second language of the patient) and was found to be deficient (although there was a strong right ear predominance; index of laterality:  $+30\%$ ). A detailed examination of limb proprioception did not reveal any deficits (performance was flawless for flexion and extension of the second digit, the arm at the elbow, the arm at the shoulder, the big toe, as well as the leg at the knee; tested on the right and left body side). Performance obtained in autotopognosia and somatognosia assessment was flawless (naming of and pointing to body parts on her own as well as on the experimenter’s body: 32/32 correct; Right-Left Orientation (Benton Form)).

## Discussion

Based on the present observation we suggest that the unilateral hearing of a person nearby (hearing of a presence, HP) reflects an auditory PSD. As discussed below this is based on phenomenological, neuropsychological, etiological and anatomical evidence.

Before discussing the relation of HP with PSDs, we begin by proposing that HP can phenomenologically be dissociated from other complex auditory hallucinations. Complex auditory hallucinations cover a variety of experiences, but are most often characterized by the hearing of a “voice or voices” called auditory verbal hallucinations (AVH). AVHs are frequent in psychotic patients (Lowe, 1973; Junginger and Frame, 1985; Chadwick and Birchwood, 1994; David, 1999; Nayani and David, 1996) and have also been induced artificially by electrical stimulation of the temporal lobe in epileptic patients (Penfield and Perot, 1963; Gloor *et al.*, 1982). However, 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 AVHs inside their head or body (Junginger and Frame, 1985; Chadwick and Birchwood, 1994; Nayani and David, 1996). This was also found for most stimulation-induced AVHs in epileptic patients (Penfield and Perot, 1963). This observation in both patient groups clearly differs from the phenomenology described by the present patient who localized a talking person (or persons) at a precise location in her backspace. 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 Birchwood, 1994). This has even led to the proposition that AVHs of psychotic origin classically lack any localization (Strauss, 1962). Even if in rare instances external AVHs may be lateralized and localized, 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 Birchwood, 1994; Nayani and David, 1996; David, 1999). Our patient, on the contrary, experienced all HPs at the same location and distance from her body (on the same side where a deficit for the localization of sound sources was found and where the FP as well as arm dislocation occurred, see below). Precise localization of the auditory source was also rare in the study by Penfield and Perot (1963) and was found in two of 21 stimulation-induced complex auditory hallucinations (case 12 and 29). Interestingly, both latter 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. The latter phenomenon was also reported by Gloor *et al.* (1982, case 3). The three cases thus closely resemble the auditory experiences of our patient and are in contrast to classically reported AVHs in epileptic patients. Based on the phenomenological differences between the hearing of another person at a specific location nearby (here called HP) and the hearing of a non-localized and generally internal voice (AVH), we speculate that they are mediated by functionally different neural systems. However, this needs to be regarded with caution since phenomenological data are particularly prone to validity and reliability problems, simply because they are, by definition, subjective. However, the above proposed distinction and the pathophysiological relation of HP to somatognosia is strengthened by further observations in our patient, most importantly, (1) her auditory-spatial neuropsychological deficits and (2) the presence of other non-auditory PSDs.

Firstly, the hearing of another person on the right side at a specific location was associated with a severe deficit in the localization of auditory sources in that same hemispace. Thus, the present patient severely mislocalized right-sided auditory sources, but was almost flawless for left-sided sound sources. This auditory-spatial deficit was especially prominent for stimuli close to her sagittal body midline as she performed better for more distant right-sided sound sources. Although the localization of sound sources was not tested at the exact position at which HPs were experienced, the predominantly affected location and affected hemispace of the auditory dysfunction are largely concordant with the experienced auditory-spatial characteristics of the HP. The fact that performance in the left hemispace was normal for both locations also shows that the deficit for right near auditory sources ( $+30^\circ$ ) cannot be explained by a higher task difficulty for near sound sources. Interestingly, the patient's auditory deficit was selective for spatial processing, since identification, discrimination, and recognition of simple and complex auditory stimuli were

normal. This suggests that HP might relate to auditory-spatial disorders rather than auditory disorders related to the identification of the non-spatial characteristics of a sound.

Secondly, the co-appearance of HP and FP (Jaspers, 1913; Critchley, 1951; Grüsser and Landis, 1991; Brugger *et al.*, 1996) as well as their absence previous to epilepsy and brain damage in the present patient suggest their close functional relationship. FP has been described in neurological patients with epilepsy (Hécaen and Ajuriaguerra, 1952; Nithingale, 1982; Kamiya and Okamoto, 1982; Ardila and Gomez, 1988; Grüsser and Landis, 1991; Brugger *et al.*, 1996) and migraine (Lippmann, 1952; Podoll and Robinson, 1999) as well as psychiatric patients (Jaspers, 1913; Hécaen and Ajuriaguerra, 1952; Brugger *et al.*, 1996) and are defined as the non-visual, distinct paroxysmal feeling of another person or being that is experienced in the near extrapersonal space. Interestingly, HP and FP seem to depend differently on the patient's body position, with HP occurring while the patient is sitting and FP while she is standing (or walking). This dependency on body position further suggests that both phenomena relate to proprioception and somatognosia. A close functional association of HP and FP is also suggested by the fact that they were always experienced unilaterally and on the same side. Although HP and FP were experienced at different distances from the patient's body (within 1 m), they were both characterized by the very persuasive experience that there is a real person nearby. Preciseness of sensory source localization and persuasiveness of the experience have been observed previously for stimulation-induced HPs (Penfield and Perot, 1963; Gloor *et al.*, 1982) as well as in FP following neurological disease (Hécaen and Ajuriaguerra, 1952; Nithingale, 1982; Ardila and Gomez, 1988; Kamiya and Okamoto, 1982; Brugger *et al.*, 1996). Nithingale (1982) reported the symptomatic association of FP, body transformation, and auditory complex hallucinations, but did not explicitly relate auditory manifestations to somatognosia and PSD. It could be argued that the HP in the present patient is not a disorder of somatognosia (referring to disorders in the perception and cognition of one's own body), since our patient never experienced her "own voice" or her "own body" (parasomatic body) as talking behind herself. Similar arguments have been proposed for FP. Yet, as noted by Brugger *et al.* (1996) and others (Jaspers, 1913; Menninger-Lerchenthal, 1935), although patients suffering from FP also do not feel their own body at two locations at the same time, the parasomatic 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 called depersonalization (Denning and Berrios, 1994; Brugger *et al.*, 1996, 1997). In addition, in rare instances the FP is associated with autoscopia (Brugger *et al.*, 1996, 1997) suggesting a close link between visual and non-visual forms of whole body PSDs. Finally, the fact that our patient also suffered from illusions of contralateral arm dislocation and displacement, as well as contralateral eye transformation also supports our assumption that HP, FP and body transformations are all

disorders of body cognition or somatognosia. The association of PSDs that are experienced for the whole body and those of body parts is rather rare (Menninger-Lerchenthal, 1935; Lunn, 1970; Brugger *et al.*, 1997), but has been observed previously. Hécaen and Ajuriaguerra (1952) have argued that this rare functional association provides strong evidence in favor of a similar neural substrate. Based on these largely concordant phenomenological and neuropsychological data in the present patient (as well as etiological and anatomical evidence, see below) we hypothesize that HP might result from a paroxysmal failure to integrate information from audition and somatosensation. This integration is needed in order to create neural representations of personal and peripersonal auditory-somatosensory space. During HP, such paroxysmal disintegration between personal and peripersonal auditory-somatosensory space might then lead to the experience of hearing a person (i.e. one's own "projected" body) in a specific position in contralesional peripersonal space. Pathological integration also occurs in neurological patients with crossmodal extinction (visual-tactile: di Pellegrino *et al.*, 1997; auditory-tactile: Làdavvas *et al.*, 2001; Farnè and Làdavvas, 2002). In these conditions, visual or auditory stimuli in ipsilesional peripersonal space have been shown to interfere with the detection of contralesional tactile stimuli (in personal space). Interestingly, this auditory-tactile interference in extinction was shown to be strongest for auditory stimuli in peripersonal space and in backspace (Farnè and Làdavvas, 2002) and thus at the location where the HP and the auditory-spatial deficit were found in the present patient. It is thus suggested that HP might result from a paroxysmal failure to integrate information from audition and somatosensation within personal and peripersonal auditory-somatosensory space. Alternatively, HP could result from the concomitant appearance of complex auditory hallucinations and a FP, which were both present separately in the present patient. HP would thus be dependant on a PSD, FP, but not a PSD itself as HP would be the consequence of the combination of both former experiences.

Thirdly, the EEG recordings and the clinical evolution suggest that the unilateral HP and other forms of PSDs might have resulted from epileptic discharge, which is by far the most common neurological etiology of PSDs (Menninger-Lerchenthal, 1935; Hécaen and Ajuriaguerra, 1952; Devinsky *et al.*, 1989; Grüsser and Landis, 1991; Brugger *et al.*, 1996). In addition, the localization of brain damage and epileptic discharge in the left posterior parieto-temporal region is concordant with the region proposed by previous investigators to cause PSDs (Menninger-Lerchenthal, 1935; Hécaen and Ajuriaguerra, 1952; Lunn, 1970; Nithingale, 1982; Devinsky *et al.*, 1989; Grüsser and Landis, 1991; Denning and Berrios, 1994; Brugger *et al.*, 1996).

In conclusion, the association and concordance of the spatial characteristics of HP with other PSDs, and with the neuropsychological auditory-spatial deficits in the present patient, suggest that the hearing of a presence might reflect an auditory PSD (or the consequence of a PSD) that results

from damage to the parieto-temporal junction. This suggests that within the group of PSDs that concern the whole body, as well as a visual form (autoscopy) and a primarily somatosensory form (feeling of a presence, FP), an auditory form can be defined that is characterized by the persuasive hearing of a real "person" nearby (hearing of a presence, HP). In accordance with reports about multisensory processing and the integration of somatosensory and auditory information at the cortical level as found by single unit recordings in the monkey (Duhamel *et al.*, 1998; Bremmer *et al.*, 2001; Schroeder *et al.*, 2001), neuropsychology (Làdavvas *et al.*, 2001; Farnè and Làdavvas, 2002; Làdavvas, 2002), and functional neuroimaging in humans (Fuxe *et al.*, 2002), it might be postulated that HP represents an illusion related to a disturbance of auditory and somatosensory integration in personal and peripersonal space.

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## Hearing of a presence

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### Abstract

Here we describe a patient with epilepsy (secondary to left parieto-temporal brain damage) suffering from the paroxysmal unilateral experience of hearing a person in her near extrapersonal space. The paroxysmal auditory experience was associated with a deficit in spatial auditory perception and other paroxysmal disorders of somatognosia. Based on these findings, it is suggested that the paroxysmal hearing of a person nearby corresponds to an auditory disorder of somatognosia.

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### Neurocase Reference Number:

#533/02

### Primary diagnosis of interest

Epilepsy

### Author's designation of case

None

### Key theoretical issue

- Epileptic discharge (secondary to left parieto-temporal brain damage) led to the repetitive illusory hearing of a person in contralesional peripersonal space

*Key words:* auditory; illusion; somatognosia; multisensory integration; epilepsy

### Scan, EEG and related measures

EEG, MRI

### Standardized assessment

Boston Naming Test, Semantical Controlled Word Association Test, d2 Test, Digit Span, Corsi Test, Rey Auditory Verbal Learning Test, Door's Test, Trail Making Test, 5-Point Test, Protocole Montréal-Toulouse

### Other assessment

Auditory localization Test

### Lesion location

- Left parieto-temporal cortex

### Lesion type

Bleeding

### Language

English