A n out-of-body experience (OBE) is defined as an experience in which a person who is seemingly awake sees his or her body and the world from a location outside the physical body. Spontaneous OBEs occur not only in patients with clinical conditions, such as near death,2 cardiogenic syncope,5 and epilepsy,13 but also in the healthy population.14 The neurocognitive processes of an OBE are very complicated and may mainly be related to an integration failure of proprioceptive, tactile, and visual information with respect to one’s own body and the disintegration between personal and extra-personal space. An experiment by Ehrsson indicated that visual information from the first-person perspective and multisensory correlation are fundamental for OBE formation.10 We herein report the case of an OBE in an epileptic child in response to focal cortical dysplasia (FCD) in the right temporoparietal junction (TPJ), which was misdiagnosed as psychosis for 1 year. Through this case, we hope to strengthen the hypothesis that temporoparietal cortex dysfunction is the fundamental etiology of an OBE.

Case Report

History and Examination. This 15-year-old right-handed boy’s first symptoms, a staring spell with loss of responsiveness lasting 1–2 minutes, occurred during sleep when he was 9 years of age. When he regained responsiveness, he told his mother that he had been projected out of his physical body to an elevated position under the ceiling, staring at his own body. He was conscious and could describe what had happened to him. These attacks occurred once every 2 months, and each episode was an OBE. One year later, after multiple similar experiences, he had an episode of clonic movement of his limbs in addition to the staring and OBE and was admitted to the local hospital. At that time psychosis was diagnosed. Over the next year, the patient continued to have frequent symptoms and experienced seizures characterized by clonic movement of the left upper extremity and left facial twitching in addition to an OBE. These episodes usually lasted for 1–10 minutes, followed by a short period of numbness in the left upper extremity. Very rarely, the seizures presented as clonic movement of all 4 extremities and loss of consciousness without an OBE. After trying 4 antiepileptic drugs (AEDs), the patient continued to have seizures. He was then referred to our epilepsy center for further evaluation. Besides epilepsy, the patient had no other significant medical problems such as head trauma, infection, surgery, or mental abnormality. His verbal, performance, and full-scale IQs were within normal range. Brain MRI did not reveal any abnormal findings.

KeY WoRDS • out-of-body experience • refractory epilepsy • focal cortical dysplasia • epilepsy surgery

Abbreviations used in this paper: AED = antiepileptic drug; EEG = electroencephalography; FCD = focal cortical dysplasia; OBE = out-of-body experience; TPJ = temporoparietal junction.

This article contains some figures that are displayed in color online but in black-and-white in the print edition.
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Scalp interictal electroencephalography (EEG) recorded sharp waves and spikes in the right hemisphere, especially in the right temporal and parietal lobes (Fig. 1). However, it was difficult to localize the epileptogenic zone in the right frontal, temporal, and parietal lobes. Subdural electrodes were therefore placed in the right frontal lobe, temporal lobe, parietal lobe, and central cortex. Intracranial EEG activity was continuously monitored, and 5 habitual seizures were recorded, 3 of which were OBEs with partial seizures. Ictal EEG showed continuous spikes on the central parietal cortex that originated from the right TPJ when the patient experienced an OBE (Figs. 2 and 3).

Operation. Through brain mapping via electrical stimulation of the cortex using subdural electrodes, sensory and motor cortices were precisely localized (Fig. 4). During stimulation, the patient experienced agnosia of the extremities but not an OBE. The epileptogenic zone on the right TPJ (3 × 2.5 cm) and part of the right postcentral gyrus (1.5 × 1.0 cm) were carefully removed, while the eloquent cortex was kept intact.

Postoperative Course. The pathological examination showed Type IIA FCD. The patient did not have agnosia of the extremities postoperatively. Neither did he have any further OBEs or seizures postoperatively. For 2 years, he was treated with AEDs, which were then gradually tapered and ultimately discontinued. He has been seizure free for 4.5 years since the surgery.

Discussion

The incidence of OBE in epilepsy patients has never been studied and reported. In the period from January 2001 to January 2013, 1500 patients with intractable epilepsy underwent surgical treatment at our epilepsy center. The epileptogenic zone was located in the TPJ in 31 patients. Of these patients, only 1 had an OBE.

Tong discussed the link between vestibular illusions and OBEs and analyzed the possible mechanism behind OBEs in his excellent paper. Clearly, Penfield is the ear-

![Fig. 1. Preoperative scalp interictal EEG tracings. Left: Sharp waves and spikes in the right hemisphere, especially the right temporal and parietal lobes. Right: The attack originated from the right hemisphere.](image1)

![Fig. 2. Intracranial ictal EEG: continuous spikes on the right central parietal cortex.](image2)

![Fig. 3. Intracranial ictal EEG: originated from the right temporoparietal junction.](image3)
An experience closely related to OBE is autoscopy, which is characterized by the experience of seeing one’s body in extrapersonal space. Both experiences are classified as autoscopic phenomena. During an OBE, the subject “sees” him- or herself and the world from a location other than his or her physical body, whereas the subject during autoscopy remains within the boundaries of his or her physical body. Previous literature has documented some autoscopy cases, but an OBE with epilepsy, especially in pediatric patients, has been rarely reported.

In the present case, right temporoparietal epilepsy due to Type IIA FCD was clearly diagnosed. Although OBE phenomena in patients with certain medical problems have been reported, the underlying mechanisms remain unknown. It has been shown that the TPJ and its neighboring structures are implicated in body-related information processing such as vestibular perception\textsuperscript{9} and multisensory integration,\textsuperscript{8} as well as the perception of human bodies and body parts.\textsuperscript{1} Grüsser and Landis proposed that paroxysmal vestibular dysfunction may be an important mechanism for the generation of an OBE.\textsuperscript{12} Blanke et al. suggested that OBE and vestibular sensations are caused by functionally and anatomically related neuronal populations.\textsuperscript{5} Importantly, the core region of the vestibular cortex is situated at the TPJ and/or the posterior insula.\textsuperscript{3} The TPJ is also implicated in visuospatial neglect, a clinical condition that has been shown to disturb the patient’s egocentric spatial relationship with extrapersonal space or visuospatial perspective. In addition, the TPJ is activated during egocentric perspective changes in healthy subjects.\textsuperscript{16} Functional MRI studies have demonstrated the importance of the TPJ in vestibular processing, multisensory integration, and the perception of human bodies or body parts.\textsuperscript{13} Blanke and Mohr observed temporal lesions in about 80% of patients with autoscopic phenomena and parietal lesions in about 50%, suggesting a prominent role of the TPJ in OBE.\textsuperscript{4} Using evoked potential mapping, Blanke et al. showed the selective activation of the TPJ when healthy volunteers imagined themselves in the position and visual perspectives that are generally reported by people experiencing spontaneous OBEs.\textsuperscript{3} When the TPJ is exposed to transcranial magnetic stimulation, the mental transformation of the body in healthy volunteers is impaired. This study by Blanke and colleagues provided evidence of the crucial role of the TPJ in typical OBEs. These previous findings underline the importance of the TPJ in normal and pathological visuospatial perspective taking and concur with the present anatomical results in OBE. In the same paper, Blanke et al. described an OBE in an epilepsy patient whose epileptogenic zone was on the left TPJ. In our case, the OBE occurred repeatedly during the early period of the partial seizures of temporo-parietal epilepsy, further confirming the clinical significance of the TPJ in OBE induction.

In addition to vestibular dysfunction, other research suggests that OBE may also be related to a failure to integrate proprioceptive, tactile, and visual body-related information (disintegration in multisensory personal space) in a coherent central representation of one’s body (body schema). Blanke et al. speculated that during OBE the integration of proprioceptive, tactile, and visual information of one’s body fails because of discrepant central representations by the different sensory systems.\textsuperscript{3} This may then lead to the experience of seeing one’s body or body parts in a position that does not coincide with the felt position of one’s body, as was proposed for the affected body part in supernumerary phantom limbs. The TPJ is the critical cortex in vestibular processing, multisensory integration, and the perception of human bodies or body parts. The integration of proprioceptive, tactile, and visual information with respect to one’s body with vestibular information is important for constant updating of the movement and position of single body parts and the entire body, as well as the body’s position in extrapersonal space. Partial impairment of vestibular processing, multisensory integration, and perception of the body or body parts under conscious conditions are necessary for OBE induction. The partial seizure network caused by the epileptic lesion in the child in the featured case very likely triggered the process of OBE.

In the present case, the patient had only an OBE and no other symptoms in the early stage, which caused the misdiagnosis of psychosis for at least 1 year. The patient had no other symptoms or abnormal findings on MRI. The OBE in this patient did not present in conjunction with generalized seizures but was related to partial seizures caused by the FCD lesion in the TPJ area, suggesting that partial impairment of vestibular processing, multisensory integration, and perception of the body or body parts under conscious conditions is necessary for OBE induction. The partial seizure network triggered the process of OBE. Notably, the pathological lesion in this right-
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handed patient was in the right temporoparietal cortex, which is different from the left temporoparietal lesion in a previous case report.5

Conclusions

Impaired neurocognitive processing at the TPJ may be the key in OBE. The TPJ is a crucial structure for the conscious experience of the normal self, mediating spatial unity of self and body.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: F Fang. Acquisition of data: Yan. Drafting the article: T Fang. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: F Fang.

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