Dissociation of the subjective and objective bodies: Out-of-body experiences following the development of a posterior cingulate lesion

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An out-of-body experience (OBE) is a phenomenon whereby an individual views his/her body and the world from a location outside the physical body. Previous studies have suggested that the temporoparietal junction (TPJ), the brain region responsible for integrating multisensory signals, is responsible for OBE development. Here, however, we first present a case of OBE after brain tumour development in the posterior cingulate cortex (PCC). The patient was a 46-year-old right-handed female; she underwent brain surgery. She reported that she had experienced OBEs several times monthly (during daily life) before surgery but never after surgery. She defined her OBEs explicitly; she drew pictures. Her OBEs exhibited phenomenological, overt dissociation of the subjective and objective bodies. We discuss the mechanisms underlying this phenomenon and the relationship between OBEs and the PCC in terms of anatomical and functional brain connectivity. Our case sheds some light on the mechanism involved in creating spatial (dis)unity between the self and the body.

The self is typically perceived as localized within the bodily boundary or even as the body per se (Merleau-Ponty, 1945/2012). However, neurological patients reporting out-of-body experiences (OBEs) have revealed that self-alterations associated with disembodiment reflect disruption of the spatial unity between the self and the body. An OBE occurs when an individual seems to view his/her body and the world from a location outside the physical body (Blanke, Landis, Spinelli, & Seeck, 2004; Devinsky, Feldmann, Burrowes, & Bromfield, 1989). The core feature of an OBE is the feeling of spatial separation of the self and body (Brugger, 2002). An OBE may be followed by a floating or flying sensation with elevation of the disembodied perspective. The current view suggests that transient

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abnormal integration of visual, somatosensory and vestibular signals explains the disembodiment (Lopez & Elzière, 2018). Although OBEs may exhibit various aetiologies (e.g., psychiatric diseases, migraine or sleep paralysis), few neurological patients with OBEs caused by brain damage have been reported (Blanke et al., 2004; Blanke, Ortigue, Landis, & Seeck, 2002; Bos, Spoor, Smits, Schouten, & Vincent, 2016; Brandt, Kramme, Storm, & Pohlmann-Eden, 2009; Brugger, Regard, & Landis, 1997; Daly, 1958; De Ridder, Van Laere, Dupont, Menovsky, & Van de Heyning, 2007; Devinsky et al., 1989; Greyson, Fountain, Derr, & Broshek, 2014; Lunn, 1970; Maillard, Vignal, Anxionnat, Taillandier, & Vespignani, 2004; Yu et al., 2018). In terms of the neural basis of OBEs, almost all cases exhibit involvement of the temporoparietal junction (TPJ), as revealed by electrical stimulation and lesional studies (Blanke et al., 2004; Bos et al., 2016; Maillard et al., 2004). One study featuring voxel-based lesional symptom mapping of focal brain damage triggering OBEs revealed that the maximal lesional overlap was in the TPJ, including the right angular and posterior superior temporal gyri (Ionta et al., 2011). The TPJ integrates multisensory signals (visual, somatosensory and vestibular), contributing to the maintenance of an embodied sense of self (i.e., a sense of spatial unity between the self and the body; Blanke, 2012). However, some studies have suggested that additional brain regions contribute to OBEs. Here, we present the first case of a patient with a posterior cingulate lesion and OBEs and discuss whether OBEs might involve the posterior cingulate cortex (PCC).

Case report

Medical history
Our patient was a 46-year-old right-handed female. After graduating from university, she worked at a trading company for 9 years, and then became a homemaker. In October 2014, she suffered from memory problems, recurrent migraine headaches, vertigo and transient (1 min) anarthria. She consulted a local hospital; magnetic resonance imaging (MRI) revealed brain tumours, primarily in the left medial parietal lobe and PCC (Figure 1). She was admitted to our hospital and underwent awake craniotomy to remove the tumours in December 2014. The final diagnosis was a ganglioglioma (WHO grade I; Louis et al., 2016). The results of the neuropsychological examinations performed before and after surgery are presented in Table 1. The memory test (WMS-R) revealed deterioration of the patient’s memory before surgery, but this was ameliorated after surgery (Table 1), which is consistent with previous findings showing the relevance of the PCC to memory deficits (Hayashi et al., 2018; Weaver et al., 2015). Neurological assessment revealed that the contralesional (right) side exhibited somatosensory weakness prior to surgery. Related to the contralesional somatosensory deficit, she reported an experience of touch-colour synaesthesia. Before surgery, when she was stimulated on the contralesional side of the body, where the tactile sensation was weaker than that on the ipsilesional side, she experienced a white colour; when stimulated on the ipsilesional side the body, where the tactile sensation was stronger than that on the contralesional side, she perceived an orange colour. During and after surgery, both the somatosensory deficits and the touch-colour synaesthesia disappeared.

Out-of-body experiences
The patient reported that she experienced OBEs on several occasions in October and November 2014. In early October, she had suffered from frequent headaches, and, when
sleeping at night, heard low notes such as those made by a male baritone singer. She interpreted the notes as music because they lay on a musical scale. Thereafter, she experienced OBEs several times a month during daily life. Her OBEs occurred indoors, for instance, she experienced OBEs when she was cooking in the kitchen. She had never experienced OBEs before, and they disappeared after surgery. She remembered the OBEs in detail, and the experience was so vivid that she drew pictures (Figure 2). We interviewed her carefully; we report our findings below:

1. She viewed her body from above and 1–1.5 m behind the physical body (the parasomatic body is shown on the left of Figure 2a).
2. Other than below the feet, she could view all parts of her physical body from behind.
3. She felt that the parasomatic body was larger than the physical body.
4. The parasomatic body could not move.
5. Although she recognized her own body from behind, as revealed by the statement: ‘I can see myself moving’ in Figure 2b, she also recognized the parasomatic body as herself (as depicted by the oblique line with the descriptors ‘real form’, ‘myself’ and ‘myself who is thinking’) (Figure 2b).

Discussion

We describe a patient who experienced OBEs after the development of a PCC lesion. Her OBE reports were phenomenologically extremely detailed; they allow us to discuss a possible origin of OBEs in terms of PCC function. Commencing with the phenomenology, the patient first heard low musical notes made by a male during sleep. This is an auditory hallucination that often accompanies OBEs (Brandt et al., 2009): hearing a ‘presence’, that is an auditory manifestation characterized by the impression of the physical presence of another (Blanke et al., 2003). Description (1) above shows that the patient experienced a parasomatic body that was elevated above the ‘real’ body; this is both a common and a
### Table 1. Neuropsychological profile

|                          | Before surgery | After surgery | Maximum score |
|--------------------------|----------------|---------------|---------------|
| **General intelligence** |                |               |               |
| MMSE                     | 30             | —             | /30           |
| RCPM                     | 31             | 36            | /36           |
| **Executive function**   |                |               |               |
| FAB                      | 17             | 17            | /18           |
| **Memory**               |                |               |               |
| Digit forward            | 9              | 8             | —             |
| Digit backward           | 5              | 5             | —             |
| Verbal memory<sup>a</sup> | 9-10-          | 10-           | /10-10-10     |
| Visual memory (immediate recall)<sup>b</sup> | 5             | 6             | /7            |
| Visual memory (recognition)<sup>b</sup> | 15            | 15            | /15           |
| **RBMT**                 |                |               |               |
| Profile score            | 23             | 20            | /24           |
| Screening score          | 11             | 10            | /12           |
| **WMS-R**                |                |               |               |
| Verbal memory index      | 80             | 112           |               |
| Visual memory index      | 98             | 106           |               |
| General memory index     | 83             | 112           |               |
| Attention/concentration index | 115          | 101           |               |
| Delayed recall index     | 81             | 94            |               |
| **Language**             |                |               |               |
| Verbal fluency           | 22             | 18            | —             |
| Naming                   | 19             | 19            | /20           |
| Repetition               | 5              | 5             | /5            |
| Auditory comprehension   | 10             | 10            | /10           |
| **Visuospatial function**|                |               |               |
| ROCFT (copy, immediate recall, delayed recall) | 34- 31- 30     | 36- 30- 28   | /36           |
| Necker cube (copy)       | 9              | 7             | /10           |
| Line bisection test      | —2%, 0.6%, —0.6% | 0.9%,1%, 1% | Positive values indicate leftward bias |
| **COGNISTAT**            |                |               |               |
| Orientation              | 10             | —             | /10           |
| Attention                | 10             | —             | /10           |
| Comprehension            | 10             | —             | /10           |
| Repetition               | 11             | —             | /11           |
| Naming                   | 10             | —             | /10           |
| Construction             | 11             | —             | /11           |
| Memory                   | 10             | —             | /10           |
| Calculation              | 10             | —             | /10           |
| Similarities             | 10             | —             | /11           |
| Judgment                 | 11             | —             | /12           |

*Note. COGNISTAT = Neurobehavioral Cognitive Status Examination; FAB = Frontal Assessment Battery; MMSE = Mini-Mental State Examination; RBMT = Rivermead Behavioural Memory Test; RCPM = Raven’s Coloured Progressive Matrices; ROCFT = Rey–Osterrieth’s Complex Figure Test; WMS-R = Wechsler Memory Scale Revised.*

<sup>a</sup>Miyake paired verbal association learning test.; <sup>b</sup>Randt memory test.
characteristic feature of OBEs (Blanke et al., 2004; Bos et al., 2016). The elevated location reflects vestibular dysfunction caused by brain damage (Lopez & Elzière, 2018). The distance from the physical to the parasomatic body (1–1.5 m) implies that the OBE lay within the peripersonal space (di Pellegrino & Lädavas, 2015); this reflects the plasticity of the bodily boundary (Noel, Pfeiffer, Blanke, & Serino, 2015). Description (2) suggests the ambiguity of the visual appearance of the physical body, especially below her feet, as depicted in Figure 2a. This implies the possibility that the seen body could be a visual hallucination. Generally, others’ feet cannot be seen from above and behind; she reported the invisibility of her physical body during the OBE similarly. Indeed, a previous case report revealed the presence of autoscopic hallucinations together with visual agnosia (Zamboni, Budriesi, Nichelli, Budriese, & Nichelli, 2005), suggesting that anomalous visual processing is involved in this phenomenon. Description (3) indicates that the parasomatic body was larger than the physical body. This suggests macrosomatognosia-like symptoms, a condition in which individuals perceive themselves as abnormally large (Frederiks, 1963). Macrosomatognosia exhibits several aetiologies, including migraine, epilepsy and stroke (Podoll & Robinson, 2000; Weijers, Rietveld, Meijer, & De Leeuw, 2013). In terms of the neural mechanism involved, an earlier neuroimaging study suggested that body size perception involves the postcentral and intraparietal sulci (Ehrsson, Kito, Sadato, Passingham, & Naito, 2005). Thus, it is possible that damage to the parietal lobe in our patient may have elicited macrosomatognosia-like symptoms. The spatial separation of the parasomatic and physical bodies suggests that she felt that she (i.e. the parasomatic body) was not confined within the ‘conventional’ spatial coordinates of the physical body because she observed her own physical body outside of it. An ambiguous boundary between the parasomatic body and the environment induced by the feeling of detachment from the physical body may be related to the sense of elevated location and macrosomatognosia-like symptoms.

Description (4) indicates motor dysfunction of the physical body. The novel features of OBEs are evident. Although OBEs are commonly accompanied by floating or flying sensations (considered to reflect anomalous vestibular sensations; Blanke et al., 2004; Lopez & Elzière, 2018), our patient reported that she (the parasomatic body) could not move, whereas the physical body could. She did not report any subjective, anomalous vestibular sensation such as floating or flying. This indicates that OBEs do not necessarily include such sensations. In fact, the patient did not exhibit continuous vestibular
dysfunction, although she suffered from vertigo at symptom onset. Furthermore, a focus on the functional difference between the parasomatic and physical bodies reveals a difference in bodily representation. Description (4) implies that the body responsible for motor function is independent of the body charged with self-localization in space. The former corresponds to the concept of a body schema (i.e. a system of sensorimotor capacities; Gallagher, 2005), and the latter to the concept of body image, which is considered to be divided into preceptual (body structural description) and conceptual (body semantics) aspects of the body (de Vignemont, 2010; Schwoebel & Coslett, 2005). An earlier study showed that body schema and body image could be mutually dissociated (Paillard, 1999); it is possible that OBEs result from a separation of the concepts. Description (5) features self-location in terms of the perceived bodily position in space (Maselli, 2015). The patient recognized the physical body in front of and below her as herself, and she simultaneously accepted the parasomatic body as herself. This is a prototypical feature of OBEs (Blanke et al., 2004; Devinsky et al., 1989). More interestingly, she described the parasomatic body as her ‘real form’ (a type of bodily representation), but also described the parasomatic body both abstractly and conceptually as ‘myself who is thinking’. The patient clearly differentiated the observing body (a subject) from the observed body (an object), indicating that the body potentially has both subjective and objective aspects. Based on these descriptions, the semantic and conceptual aspects of the conscious self seemed to be located in disembodied (i.e. empty) space. However, this does not reflect a Cartesian dualism, but rather indicates that the visual perspective is independent of the physical body that is accompanied by sensorimotor function. When we perceive the external world, we can adopt different perspectives depending on the bodily boundary. For example, we can imagine and understand how others perceive the world spatially and psychologically from their perspectives; this is termed the ‘theory of mind’ (Aichhorn, Perner, Kronbichler, Staffen, & Ladurner, 2006), ‘mentalizing’ (Amodio & Frith, 2006) or ‘perspective taking’ (Vogeley & Fink, 2003). Many studies have found that a disembodied perspective is a useful biological function, allowing us to know how to perceive the world (Arnold, Spence, & Auveray, 2017) and interact with others (Frith, 1999). However, those experiencing OBEs perform worse than others on tasks requiring adoption of a disembodied perspective towards an avatar (Blanke et al., 2005; Braithwaite, Samson, Apperly, Broglio, & Hulleman, 2011), suggesting that the anomalous nature of the disembodied perspective is linked to the OBE mechanism. Indeed, an earlier neuroimaging study suggested that adoption of a disembodied perspective reflects TPJ activation (Schurz, Aichhorn, Martin, & Perner, 2013); the TPJ is the brain region considered responsible for OBEs (Blanke & Arzy, 2005). Moreover, a previous report found that self-location involving a visual first-person perspective reflected TPJ activity (Ionta et al., 2011), suggesting that one’s visual perspective was associated with a sense of being localized in space. This interpretation can be applied to our case. Experiencing the parasomatic body with a sensation of disembodiment makes it impossible to apply a normal perspective (Blanke et al., 2005; Braithwaite et al., 2011).

Next, we turn to the possible neural mechanism underlying OBEs in terms of PCC function. To the best of our knowledge, this is the first report of OBEs in a patient with a PCC lesion. Previous neuroimaging studies on OBEs of both healthy and neurologically compromised individuals found that the TPJ contributed to the convergence of multisensory signals (Blanke et al., 2004; Bos et al., 2016; De Ridder et al., 2007). However, as the role played by the PCC in OBEs remains unknown, the topic should be discussed. An earlier study suggested that patients with PCC lesions exhibited impaired
spatial orientation (termed ‘topological disorientation’; Katayama, Takahashi, Ogawara, & Hattori, 1999; Takahashi, Kawamura, Shiota, Kasahata, & Hirayama, 1997). This seems to share a phenomenological basis with the difficulty in localizing the self in space associated with a PCC lesion. Furthermore, in the resting brain, the PCC is functionally connected to the TPJ (Mars et al., 2012); the PCC and TPJ both lie in the posterior region of the default-mode network (one of the central resting brain networks; Zhang et al., 2017). Anatomically, the PCC projects to the macaque homologue of the inferior parietal lobule and the superior temporal sulcus, both of which are TPJ regions (Parvizi, Van Hoesen, Buckwalter, & Damasio, 2006). Given this PCC/TPJ connectivity, it is possible that within-body multisensory integration, which is mediated in the TPJ (Blanke & Arzy, 2005), was impaired by the PCC lesion in our patient. Indeed, the patient showed transient touch-colour synaesthesia (Steven & Blakemore, 2004), suggesting that the aberrant multisensory processing was mediated in the TPJ, as this symptom has been characterized as a failure of inhibition in mutual connection between the somatosensory cortex and TPJ, which mediates the perception of colour (Horiguchi, Wandell, & Winawer, 2016). Importantly, neuroimaging research on out-of-body illusions suggested that the PCC engages in self-location in space (Guterstam, Björnsdotter, Gentile, & Ehrsson, 2015). In the cited work, the self-location illusion was induced from the perspective of the body of a stranger, suggesting that self-location by reference to personal visual perspective reflects PCC activity. The evidence we present above explains the OBEs of our patient in terms of anomalous self-location in space by the PCC.

Finally, we raise the question of why there are no previous reports of OBEs developing after PCC lesions. It is possible that the anatomical connections between the PCC and TPJ are so robust that OBEs are rarely induced by such lesions. As maintenance by the TPJ of spatial unity between the self and the body is crucial in terms of human self-consciousness, the neural connection from the TPJ to various other regions may indeed be very strong. Furthermore, it may be that some subpopulations are OBE-prone (Nakul & Lopez, 2017). Not only brain damage, but also psychological dysfunctions such as depersonalization/derealization may precipitate OBEs. In future, simultaneous evaluation of the neurological and psychological aspects of OBEs will allow us to better understand OBEs.

In summary, we first present a case in whom OBEs developed after a PCC lesion. The case suggests that the PCC contributes to self-location depending on the visual perspective; this is in line with prior patient and neuroimaging findings and sheds some light on the mechanism involved in creating spatial (dis)unity between the self and the body.

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Conflicts of interest
All authors declare no conflict of interest.

Author contributions
Kentaro Hiromitsu: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Validation, Visualization, Writing – original draft, Writing – review &
Data availability statement
The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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