Organic Delusional Syndrome: Tentative Neuropsychological Mechanism of Delusions

Yoshitaka Ohigashi and Makiko Yamada

Abstract Three organic delusions, namely, "persecutory delusions in psychotic disorder following traumatic brain injury (PDFTBI)," "Capgras syndrome, a major form of delusional misidentification syndrome," and "anosognosia for left hemiplegia or somatoparaphrenia," are discussed in this chapter. Concerning persecutory delusions in PDFTBI, we underscore the role of the temporal pole lesion that may segregate the function of the amygdala from visual information processed in the temporal lobe. The isolated function of the amygdala is speculated to cause an undiscerning oversensitive response to any incoming emotional stimuli. With regard to Capgras delusion, the most conventional neuropsychological account of "the mirror-image model of prosopagnosia" is reevaluated, and several important critiques are mentioned. Last, we have attempted to provide a novel explanation of anosognosia for left hemiplegia and somatoparaphrenia by refining definitions of "body consciousness" and "body schema" and by taking account of Edelman's reentry hypothesis for the genesis of consciousness. In the end, it is concluded that ingenious neuropsychological approaches are indispensable for the understanding of organic delusional syndromes.

Keywords Anosognosia • Capgras syndrome • Neuropsychology • Persecutory delusion • Somatoparaphrenia

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Introduction

Various types of organic delusions can be observed in neurological patients, yet their mechanisms are still to be determined. In this chapter, three major organic delusions are discussed, and we attempt to provide the neuropsychological mechanisms of each delusion. The first topic is the "persecutory delusion" that is observed in psychotic disorders following traumatic brain injury (PDFTBI). The second is the "delusional misidentification syndrome" (DMS), represented mainly by Capgras syndrome. The third is "anosognosia for left hemiplegia" [1] and "somatoparaphrenia" [2], both of which are generally observed following large right hemispheric lesions.

Given that the persecutory delusion and delusional misidentification can occur independently in neurological settings, these delusions are based on the distinct neurolopsychological mechanisms. Although the lesion in the temporal pole seems to be responsible for persecutory delusion in PDFTBI, abnormal functions in the limbic structures together with the right frontal lobe could be related to Capgras syndrome. The third type of delusion, anosognosia for left hemiplegia and somatoparaphrenia, is of special interest. Body schema and body consciousness are distinctive in nature. As the former is supposed to possess symbolic or semiotic features, it must be represented mainly in the left hemisphere. In contrast, the latter can be represented in the bilateral hemisphere in such a way that the right hemisphere represents whole-body consciousness while the left hemisphere supports only the contralateral, that is, the right side of body consciousness. As a consequence of a large right hemisphere lesion, a patient's bilateral body consciousness might be reduced, producing a residual consciousness of only the right side of his/her body. We consider that this is the fundamental feature of anosognosia for left hemiplegia and the phenomenon of somatoparaphrenia.

Persecutory Delusion and Delusional Perception in PDFTBI

Psychotic disorder following traumatic brain injury (PDFTBI), originally reported by Fujii and Ahmed [3, 4], is the psychotic state that consists predominantly of persecutory delusions and auditory hallucinations with an absence of negative symptoms. Abnormalities in the temporal and frontal areas are often associated with PDFTBI. The latency between traumatic brain injury and the onset of psychotic symptoms varies between patients, but the recent consensus falls around 4 to 5 years.

Our major interest here is whether delusional perception in PDFTBI is related to the patient's biased-emotion evaluations of others, which may provide insight regarding the neuropsychological mechanisms of persecutory delusion. In the following, we present two PDFTBI patients with temporal lobe lesions. Based on our examination of their ability to estimate emotion intensity from facial expressions, the critical role of the temporal pole for delusional perception is suggested.

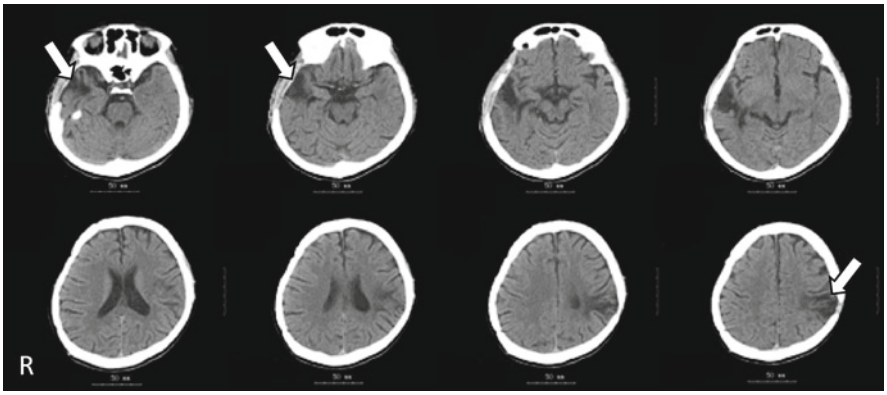

Fig. 1 Computed tomography (CT) findings of case SA

Case SA was a 23-year-old female ambidextrous college student. She had a traffic accident at age 16 years and had been unconscious for about 10 days. She received a craniotomy to remove a left parietal hematoma. Her lesions were detected in the right temporal pole and the left parietal lobe (Fig. 1). When she was discharged from hospital after 3 months, she showed mildly impaired memory retention, reduced calculation ability, articulation disorder, and mild right hemiparesis. After the accident, she was noticed by her family and friends to behave and talk like a child. She complained of her difficulty in memory retention and concentration; however, she had spent a quasi-normal daily life without many serious problems.

About 4 years later she began to experience hallucinations and delusions. She complained: "my father looks like a different person," "I feel as if TV is speaking of me," and "I'm very scared and always feel as if I'm chased by someone." She also experienced functional auditory hallucinations, such as "I hear the sound of typing computer keyboard as human voice."

Case DR was a 36-year-old right-handed woman. She received a college education. A traffic accident at age 14 resulted in skull fracture and intracranial hemorrhage with a loss of consciousness for 3 days. Her major complaint was headache, insomnia, and irritability. After 5 years after the accident, she began to develop persecutory delusion, such as "I hear some noise that speaks evil of me" and "my vulgar idea is detected by other people." She was first diagnosed as "schizophrenia" in a psychiatric department but was now rediagnosed as PDFTBI. Computed tomography (CT) and single-photon emission computerized tomography (SPECT) indicated apparent lesions in the left anterior temporal lobe and the left posterior frontal cortex (Fig. 2).

The ability of both patients to evaluate emotional intensity from others' faces was investigated using the Emotional Intensity Scale (EIS [5]) and compared with that of healthy subjects. In this task, subjects were asked to rate the perceived intensity of a given emotion word (six basic emotions in total) for each facial expression of six basic emotions (see [5] for detailed procedure). Results indicated that case SA perceived a sad facial expressions not only as "sadness" itself but also as high intensities of

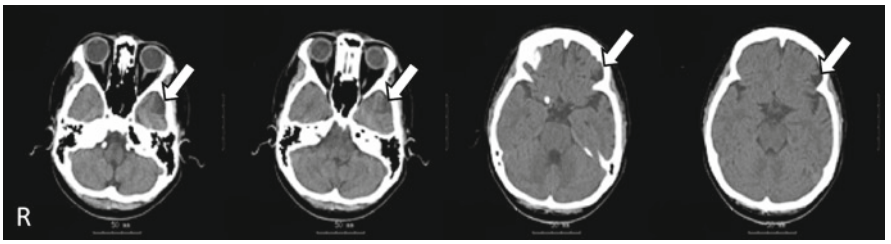

Fig. 2 CT findings of case DR

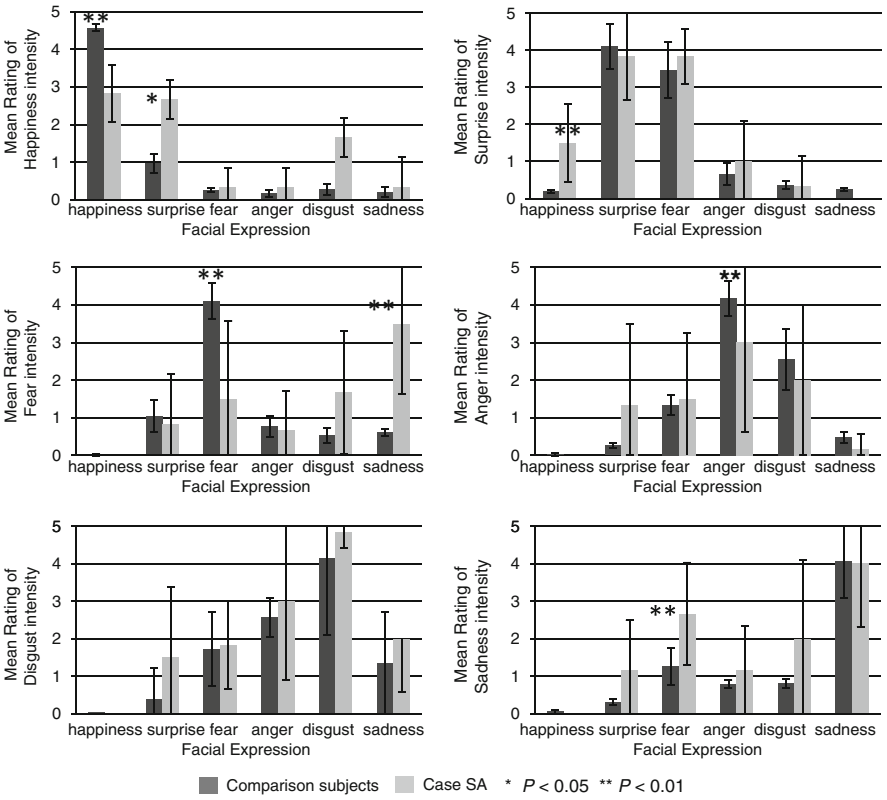

Fig. 3 Results of Emotional Intensity Scale in case SA. *Horizonta axis*, evaluated facial expressions; *vertical axis*, intensity of an emotion word

"surprise" and "fear" (Fig. 3). This patient showed a tendency to perceive facial expressions in heightened emotions of "surprise," "disgust," and "happiness" compared to normal subjects. Case DR perceived emotions of fear, anger, and sadness in almost all facial expressions except for happiness (Fig. 4).

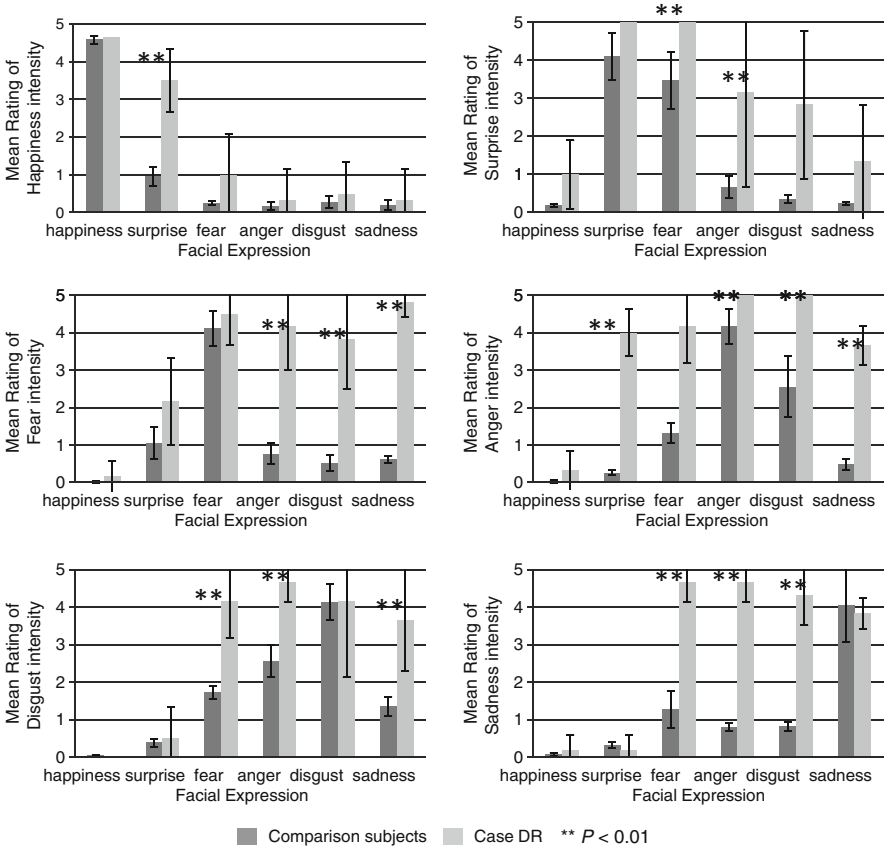

Fig. 4 Results of Emotional Intensity Scale (EIS) in case DR

Several common features between the two cases can be pointed out. (1) Their common lesion site was the temporal pole. (2) Psychotic states appeared 4 to 5 years after head injuries. (3) Persecutory delusion with hallucination was the predominant clinical picture in both cases. (4) Results of EIS suggested the confused judgments of facial expressions, characterized by a negatively biased-emotion estimation.

The temporal pole is known to play a crucial role in high-level recognition because it is anatomically located at the endpoint of the auditory and visual "what" stream. A general function of the temporal pole is supposed to be to couple emotional responses to highly processed sensory stimuli through a tight connection with the amygdala [6]. Thus, the temporal pole is regarded as a relay point that links the final "what" perception to emotional responses in the amygdala. The lesion sites in our cases suggest that the route from the temporal pole to the amygdala could be disrupted, and that the functions between these two sites might be isolated. As indicated by EIS results, the patients overestimated negative emotions regardless of the actual emotional signals of the facial expression. This overestimation of negative emotion has been observed in the case of ictal fear [7], which implied that the

amygdala was not dysfunctional but rather hypersensitive. Consistent with this amygdala hypersensitivity account, a negatively biased perception in our cases could have persisted through limbic kindling, which might have created a trait-like misinterpretation of others' minds, or persecutory delusions.

In sum, the temporal pole lesions in patients with PDFTBI may segregate the function of the amygdala from visual information processing. This isolation may cause the amygdala to respond to emotional stimuli overly intensively and unselectively. We regard this as one possible cause of delusional perception and persecutory delusion in PDFTBI.

Delusional Misidentification Syndrome: Capgras Syndrome

Capgras syndrome is the most common form of delusional misidentification, originally described by Capgras and Reboul-Lachaux [8]. This disorder is characterized as the delusional belief that familiar persons have been replaced by identical impostors. This condition was once (or is still now) explained by the psychoanalytical view that posits this disorder as a defense mechanism against unconscious prohibited desires. However, accumulating neurological and neuropsychological evidence suggests that organic factors are important in the pathogenesis of Capgras syndrome [9].

One most conventional neuropsychological account is "the mirror-image model of prosopagnosia" [10], based on the dual-route theory of facial recognition (Fig. 5). According to this model, two anatomically independent routes to face recognition, namely, overt and covert recognition pathways (Fig. 5a, b, respectively), are damaged in a mirror-reversed manner between prosopagnosia and Capgras syndrome. As Fig. 5 illustrates, although the overt route interruption causes prosopagnosia, the covert route disconnection yields Capgras syndrome. This claim is derived exclusively from undifferentiated skin conductance responses (SCRs) toward known and unknown faces in Capgras patients, in contrast to normal SCRs toward unrecognized-yet-known faces in prosopagnostic patients. Disconnection between the face-processing areas and the amygdala has been considered to represent a lack of affection/familiar feeling toward known faces in Capgras syndrome. This disconnectionist account has been favored by several researchers ([12], etc.); however, it seems that quite a few serious objections impede upholding this idea.

Major criticisms include the following. (1) Although this model claims that the SCR is a measure of covert recognition, the relationship between SCRs and the true experience of patients is far less clear [13, 14]. (2) SCRs usually index a generalized arousal state following an unexpected external stimulus that is often threat- or fear related; this contradicts the assumption that SCRs signify a "familiar feeling" in the mirror-image model. (3) Correspondingly, although this model regards the amygdala functions as a "familiar feeling," the widely accepted view of this region is mainly to detect threat-related information. (4) The disconnection in the covert route is assumed in Capgras syndrome, but the actual anatomical disconnection has not

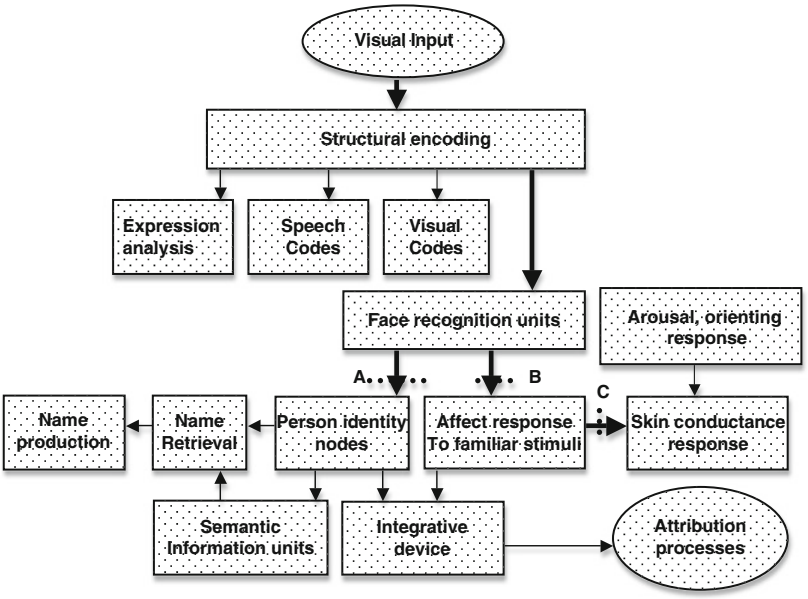

Fig. 5 Model of face recognition and Capgras delusion, formulated by Ellis and Young [10] (figure is a reprint from [11], modified for this publication). Disconnection in the overt recognition route (**a**) yields prosopagnosia, whereas that in the covert recognition route (**b**) produces Capgras syndrome

been identified. (5) The model explains the face recognition processing but ignores the occasional coexistence of misidentification of objects or places. (6) The model does not account for patients' resistance to modify their delusional belief in the presence of very strong evidence against it.

Among different explanations for DMS provided by other researchers, a two-factor model, proposed by Coltheart and his colleagues [15], takes into consideration the foregoing issue (see "6," above). In their model, although the first factor is composed of the failure of autonomic responsiveness to familiar faces, the second abnormal factor is the patient's resistance to revise the delusional belief, which is speculated to arise from a disrupted belief evaluation system associated with the right frontal cortex. In accord with their idea, neuroimaging studies revealed that the right prefrontal cortex was involved in mediating the ability to detect or resolve conflicts in thinking [16, 17]. In this sense, we also hypothesize that the delusion in Capgras syndrome as well as other DMSs might represent a dysfunction of the cognitive-conflict resolution mechanism, which is supported by the right frontal lobe. Further support for this view is observed from evidence that right frontotemporal lesions are predominant in patients with Capgras syndrome in the setting of focal

brain damage [18–20]. The reduction in event-related potential (ERP) in the right frontal lobe was also reported in deluded patients [21].

Alghough Ellis &Young's original one-stage account was recently incorporated into two-factor model, called "an interactionist model" [11], the puzzle still remains as to what special mechanisms create the delusional misidentification. The examination of neurological patients suggests the involvement of the limbic structure, yet its functional relationship with the misidentification phenomenon must be determined in future research. Considering that the amygdala is an alarm system that deals with not only threat-related stimuli but also anything ambiguous for an organism, we dare advocate a new proposal of misidentification in Capgras syndrome. If the same argument that we made for delusional perception in PDFTBI can be applied to Capgras syndrome, we speculate that regardless of known or unknown persons (or objects), the maladaptive function of the hypersensitive amygdala creates a feeling too strongly suspicious to be rejected; that is, "a deluded misidentification in Capgras syndrome."

Anosognosia and Somatoparaphrenia

Anosognosia for left hemiplegia was first described by Babinski [1] as the denial of left hemiplegia. Somatoparaphrenia was reported by Gerstmann [2] as delusional beliefs concerning a contralesional side of body (the left side in most cases), which is characterized by a pathological alteration of the ownership of the limbs. In a famous monograph, *The Parietal Lobes* [23], Critchley classified the content of distortion of the body image as follows: (1) unilateral neglect, (2) lack of concern (anosodiaphoria), (3) unawareness of hemiparesis (anosognosia), (4) defective appreciation of the existence of hemiparesis, (5) denial of hemiparesis, (6) denial of hemiparesis with confabulation, (7) loss of awareness of one body-half (asomatognosia), (8) undue heaviness, deadness, or lifelessness of one half, (9) phantom third limb, (10) personification of paralyzed limb, and (11) misoplegia (the last two categories were included in 1955 and 1974, respectively). In his categorization, anosognosia and various somatoparaphrenia were not sharply demarcated but closely related. In 1972, Hécaen [24] distinguished hemisomatognosic disorder into three categories: (1) anosognosia for the left hemiplegia, (2) hemi-asomatognosia raging from simple neglect to amnesia, unawareness of one side of the body, and (3) feeling of absence of one's body part or one side of the body, including disownership and phantom limbs. Apparently, these disorders still lie on a continuum.

Here we attempt to provide a neuropsychological mechanism of anosognosia (unawareness of left hemiparesis) and a feeling of disownership of the left side of the body, based on a novel framework of consciousness proposed by Edelman [25] and on our refined definitions of "body schema" and "body consciousness." Body schema represents a symbolic semiotic or linguistic body concept, possibly

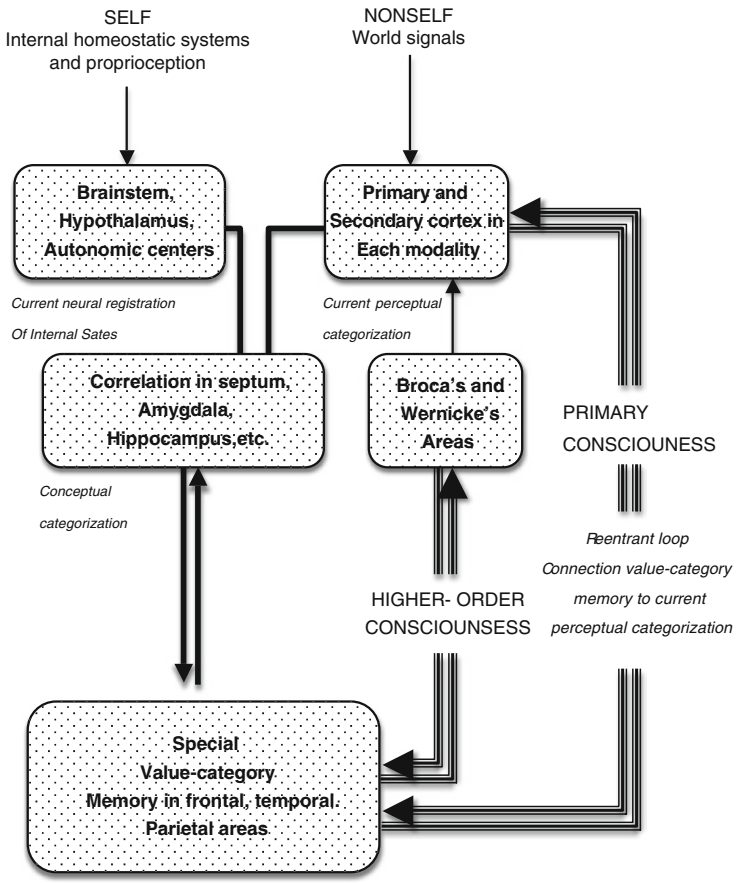

Fig. 6 Reentry genetic theory of consciousness by Edelman [25] (modified for this publication)

supported by the left parietal lobe. Phylogenetically, new "body schema," which would have coincided with the genesis of language, could be attributed to the higher-order consciousness within Edelman's reentry hypothesis for the genesis of consciousness [25] (Fig. 6). The close relationship between body schema and language is supported by the evidence that impaired body schema can provoke, for instance, bilateral finger agnosia or autotopagnosia. In contrast, body consciousness represents a basic and immediate body feeling that would be phylogenetically older than body schema. We would attribute body consciousness to the primary consciousness in Edelman's model, which may be supported predominantly by the right hemisphere.

We further speculate that body consciousness, which was originally distributed bilaterally and symmetrically, that is, the right body was in the left hemisphere and vice versa, gradually shifted to the right hemisphere as a consequent of a "body schema" lateralization in the left hemisphere. If so, it can be hypothesized that the right hemisphere represents the bilateral body consciousness, whereas the left hemisphere represents only a residual consciousness of the right side of the body. Recent lesion studies indicated that body consciousness would be represented by a neural circuitry of right hemisphere regions, including the temporo-parietal junction, posterior insula, and subcortical structures, such as basal ganglia [26–29]. Thus, damage to this neural circuitry in the right hemisphere may yield a loss of bilateral body consciousness whereas the right-body consciousness may survive without damage to the left hemisphere. Therefore, patients are aware of only the right side of the body, and the "anosognosia" and the "somatoparaphrenia" appear exclusively on the left side of the body.

In the patients' consciousness, the left body is no longer their own body. The conviction that they can move their own left arm would not work for them because the left arm no longer exists in their consciousness. In turn, they would say they can move it by showing their moving right arm, or would talk about their own left arm as if it did not belong to their body. These delusions are not false but quite real in their consciousness. We propose that this is the fundamental feature of "anosognosia for left hemiplegia" and the phenomenon of the "somatoparaphrenia."

Conclusion

In this chapter, we took a neuropsychological approach to understand organic delusional syndromes. One possible account for persecutory delusions in PDFTBI was the segregation of amygdala function caused by temporal pole lesions. The isolated amygdala may respond to any emotional stimuli because of inadequate visual information or rundown visual processing areas in the amygdala; this may create an overly reactive amygdala and shape the delusional perception in these patients. The pathogenesis of Capgras syndrome could be related to the abnormal functioning in the right hemisphere and the limbic areas; however, the most accepted neuropsychological account of "the mirror-image model of prosopagnosia" faces several critical problems, and the advanced theory warrants future research. Our novel explanation of anosognosia for left hemiplegia and somatoparaphrenia was provided within Edelman's reentry hypothesis for the genesis of consciousness, together with the refined definitions of "body consciousness" and "body schema."

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