Loss of the sense of self-ownership for perceptions of objects in a case of right inferior temporal, parieto-occipital and precentral hypometabolism

Roland Zahn[a,c], Jochen Talazko[b,d], Dieter Ebert[a]

a Department of Psychiatry and Psychotherapy

b Department of Nuclear Medicine
Albert-Ludwigs-University Freiburg, Hauptstr. 5, D-79104 Freiburg, Germany

c The University of Manchester, School of Psychological Sciences, Neuroscience and Aphasia Research Unit, Manchester, M13 9PL, UK

d Institute for Radiology and Nuclear Medicine, Schwarzwald-Baar-Klinikum, Röntgenstr. 20, D-78054 Villingen-Schwenningen, Germany

Correspondence to: Dr. Roland Zahn, The University of Manchester, School of Psychological Sciences, Neuroscience and Aphasia Research Unit, Zochonis Building, Office T7a, Manchester, M13 9PL, UK

Tel: +44-(0)161-27-57338
Fax: +44-(0)161-27-52873
e-mail: roland.zahn@manchester.ac.uk

Short title: *"Loss of self-ownership"*

This paper has been published by Karger publishers in Psychopathology (2008), 41: 397-402.

Keywords: ***Social Cogntion; Self-consciousness; Temporal Lobe; Parahippocampal gyrus; FDG-PET; Decompression Sickness; Neurophilosophy***

Abstract

Philosophers define the ‘minimal self’ as the immediate awareness of being the agent and owner of one’s actions and perceptions. Here, we describe a patient with a selective loss of one part of this ‘minimal self’, namely the immediate sense of self-ownership for perceptions of objects. In contrast, his sense of self-ownership for body perceptions and for self–agency during actions remained intact. 18-Fluoro-Deoxy-Glucose-Positron-Emission-Tomography revealed predominantly right inferior temporal hypometabolism in comparison with healthy controls (parahippocampal and fusiform gyri), additionally dysfunction of the right parieto-occipital junction and precentral cortex were detected. Taken together, we demonstrate selective changes in the quality of the sense of self-ownership for perceptions of objects but not actions and an intact sense of self-agency which points to anatomically separable systems underpinning different aspects of the ‘minimal self’. The associated hypometabolism in inferior temporal, parieto-occipital and motor regions but not in medial prefrontal areas most consistently associated with self-referential processing are most parsimoniously explained when self-consciousness is not assumed to be an anatomically localised cognitive function, but instead is conceived as emerging from integration across anatomically distributed networks of regions with different functional specializations, not all of which need to be special for the ‘self’.

Introduction

Several recently developed philosophical approaches to the self and self-consciousness promise to enhance the exchange of ideas between philosophy of mind and cognitive neuroscience [1-3]. The anatomical evidence on regions participating in cognitive functions underlying our experience of self has been comprehensively reviewed [1,2,4]. Philosophical notions of self-consciousness as discussed by Gallagher [3] distinguish different aspects of self experience. We will focus here on the so called 'minimal self' comprising probably the most basic aspect of self-consciousness which can be investigated. Phenomenologically the 'minimal self' is a consciousness of oneself as an immediate subject of experience, unextended in time. This minimal self-awareness can theoretically be further divided into: 1) the sense of self-agency as the sense that I am the one who is causing or generating an action, and 2) the sense of self-ownership as the sense that I am the one who is undergoing an experience [3].

It may be debated whether these primarily philosophical constructs only have a theoretical basis or whether they are neurobiologically valid and can be experimentally demonstrated. To confirm the latter at least two empirical methods exist: First, an experimental design can be developed to modulate the phenomenal experience of the sense of self-agency or the sense of self-ownership in a subject. This approach has been successfully used to demonstrate that quantitative changes to the sense of self-agency can be experimentally induced without changing the sense of self-ownership for actions [5]. In a next step the neurobiological basis of these phenomenal experiences can be established by using brain imaging for example. Experimental manipulations, however, can only modulate the quantity but not the quality of experience of the minimal self, since the sense of self-ownership accompanies every action or perception by definition (as we always know that we have or have not a perception or that we do or do not commit an action). The second way to

investigate a postulated neurobiological basis of the 'minimal self' is to describe the psychopathology of neurobiological disorders in which selective losses of sense of self-ownership or self-agency occur. The most prominent candidate for this approach so far is schizophrenia which has been characterized as a loss of sense of self-agency already by Bleuler [6] who first developed a disease category of schizophrenia comparable to our modern classification. Experimental approaches have successfully demonstrated abnormalities in the sense of self-agency in schizophrenic patients [2,7-10]. A diminished sense of self-agency, however, usually does not occur in isolation in schizophrenia but is usually accompanied by cognitive impairments in multiple domains.

The sense of self-ownership during actions has recently been investigated by using functional neuroimaging [11] and mainly confirmed the important role of parietal cortices in distinguishing self and other during action as has been demonstrated for self-agency [8,12,13]. There is also evidence for the importance of medial prefrontal cortices for the sense of self-agency during actions [14].

To our knowledge, however, there is no description of a disorder with selective loss of the sense of self-ownership so far. Furthermore the experimental approaches to self-ownership primarily addressed the experience of ownership for body parts during actions and not the ownership for perceptions of external objects. Here, we describe a patient with an isolated loss of the sense of self-ownership for perceptions of objects with an intact sense of self-ownership in the proprioceptive domain and an intact sense of self-agency.

Case report

A 23 year old male (D.P.) complained of (what he called) 'double visions'. He reported their acute onset after an attack of tachycardia and dyspnoea accompanied by the fear of asphyxiation during a long-distance flight on his overseas holiday. He had been diving 10 days before. After having returned from his holiday, 5 weeks after the incident he first

reported to the neurological outpatient clinic at the University of Freiburg, who referred the patient to our psychiatric outpatient clinic with no sign of a neurological disorder (normal MRI, EEG and neurological exam). Psychopathological and neurological examination revealed that D.P. did not see doubled objects in the literal sense – instead he described his sensations as follows: When looking at or concentrating on a new visual object, he is able to see the object as a single object, but that the way he perceived had markedly changed in a way which he had never experienced before. It appeared to him that he was able to see everything normally but that he did not immediately recognize that he was the one who perceives and that he needed a second step to become aware that he himself was the one who perceives the object. In contrast, his actions felt unchanged and he was always immediately aware of his actions as the one who acts and perceives his body while acting. He also denied changed passive perceptions of his body, changes in his body image or in the awareness of being the one who is initiating an action. There were no apparent changes in his perception of other people's movements. Visual perceptions were not accompanied by any delusions of control or thought insertions, obsessions like obsessive doubts, compulsions or fear. Social interaction and communication was normal which was also confirmed with the patient's parents with whom he was currently living. In the history there was no psychiatric or serious medical condition. D.P. had finished his baccalaureate with good marks and after completing his civilian service, he was working in a supermarket to earn money for a 1 year overseas trip which he had terminated after the incident. There were no psychosocial stressors or traumata in the history. The patient was socially normally integrated. Daily life activities were not impaired and D.P. was planning to apply for University. Day to day memory was normal.

A neuropsychological standard test examination 2 months after the first referral showed normal results despite unchanged psychopathology. We excluded attention and executive deficits by performing the reactive cognitive flexibility and divided attention subtests from the Test Battery for Attentional Performance ([15], above the age- and

education-corrected percentile rank 50 for response time and errors), three letter word fluency (LPS 6 [16], above average performance: > mean + 2 standarddeviations, age-corrected) and Trail Making Test A ([17], age- and education-corrected percentile rank 99). Short-term and working memory were above average: digit span forwards (age-corrected percentile rank 57) and backwards (age-corrected percentile rank 86) from the revised Wechsler Memory Scale [18]. Semantic memory, lexical retrieval and visual object recognition were intact as measured by 100 % correct responses on picture naming using line drawings of living and nonliving objects (further described in [19]). Episodic verbal memory was intact as measured by word list learning ([17], Auditory Verbal Learning Test: 14/15 words recalled already after 2nd round). Visuo-constructive abilities and nonverbal episodic memory were normal as assessed by the immediate (35.5 points = above age-corrected average) and delayed (24 points = above age-corrected average) Complex Rey Figure copy [17].

Psychopathological examination revealed no other symptoms apart from the distress caused by the primary symptom. According to the performed structured clinical interview for DSM-IV-R [20] the clinical picture did not fit to any specific psychiatric diagnosis, but we assumed a cognitive disorder due to a medical condition. The following examinations were performed without any pathological findings: neurological examination, analysis of blood and liquor including drug screening, electroencephalography, cranial magnetic resonance tomography (Siemens Magnetom, 1.5 Tesla, Fluid Attenuated Inversion Recovery-, Diffusion-, T1- and T2-weighted images). Cardiological examination revealed an open foramen ovale but no evidence of a deep venous thrombosis as an origin of a crossed embolus was found. The 18F-Deoxy-Glucose-Positron-Emission-Tomography (FDG-PET) findings 2 months after first referral are reported in figure 1 and table 1. Remarkably, the psychopathological finding remained stable and no further psychopathology emerged over the course of a 1 year follow up. The following therapies were applied without any effect:

Antidepressants (reboxetine, venlafaxine, amitryptiline), antipsychotics (risperidone, olanzapine), memantine and recompression therapy in a hyperbaric chamber (normally only applied soon after suspected decompression sickness after diving). We finally diagnosed a cognitive disorder, not otherwise specified. The aetiology of the demonstrated areas of hypometabolism with normal MRI exams remained obscure. However, a small cerebral infarction due to a crossed cardio-embolic event needs to be discussed as a possible cause. This is because the time delay of 5 weeks between MRI, thrombosis diagnostics and the onset of symptoms might have contributed to the negative diagnostic findings.

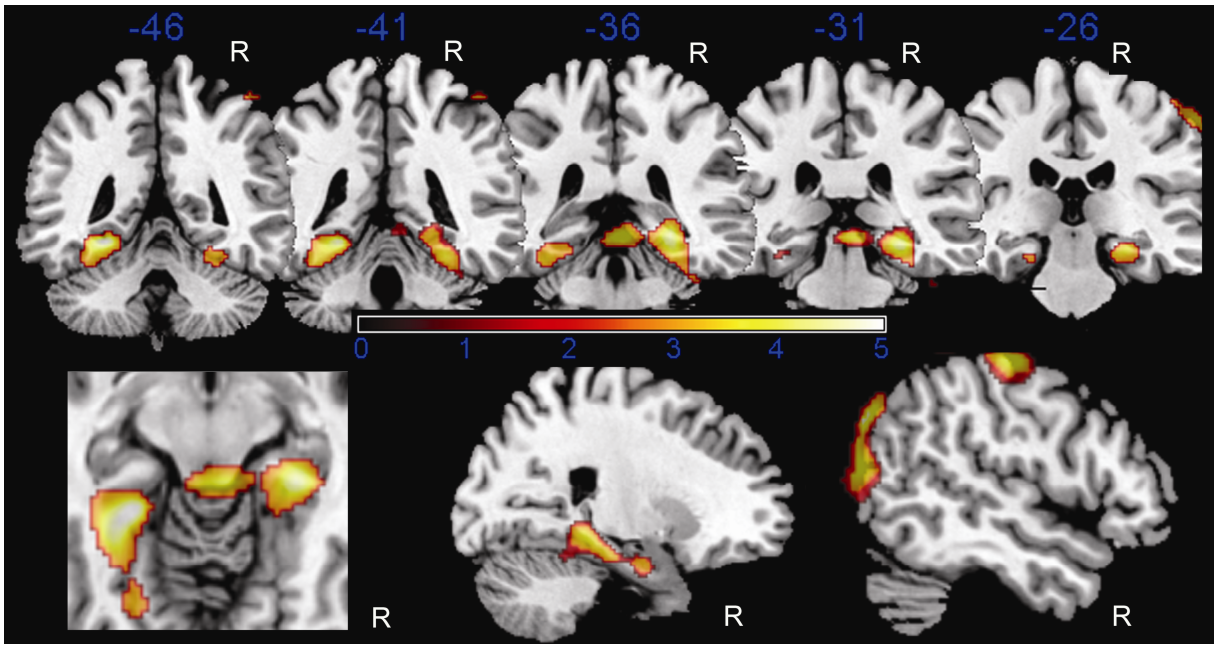

Figure 1 Significant areas of hypometabolism (voxel level: P=0.01, uncorrected, 15 voxels for visual display, only areas surviving uncorrected P=0.001 and Family-Wise-Error corrected P=0.05 over a 12 mm sphere around the peak voxel are reported in table 1 and text) for patient D.P. vs. 12 normal controls are shown. Data were projected on a standard brain using MRIcron (http://www.sph.sc.edu/comd/rorden/mricron/)[35] after statistical analysis using Statistical Parametric Mapping (SPM5, http://www.fil.ion.ucl.ac.uk/spm/, [36]), smoothing with a 12x12x12 mm Gaussian kernel and a global cerebral metabolic rate for glucose normalization to a mean of 50 µmol/100ml/min. A voxel by voxel t-statistic for detection of hypometabolic areas was computed (methods further described in [19]).

Table 1 PET, SPM5 Pat. < Normal Controls

Hemi-sphere	Area	x	y	z	BrodmannArea	Cluster size at P=0.01	T - score
R	Parahippocampal gyrus/fusiform gyrus	28	-33	-7	35/36/37	778	4.72
R	Parieto-occipital junction	51	-72	35	39/19	815	5.17
R	Precentral/premotor cortex	61	-8	43	4/6	658	6.03
R	Cerebellum – culmen	4	-33	-5	-	195	4.29
L	Parahippocampal /fusiform gyrus	-24	-43	-6	35/36/37/20	694	5.52

PET=18-fluorodeoxyglucose positron emission tomography. Only areas exceeding a voxel level threshold of Z > 3.09 and ***P=***0.001 uncorrected with a minimal cluster size of 15 voxels at P=.01 and surviving FWE-corrected P=.05 over a small volume of a 12 mm diameter sphere around the peak voxel are reported. MNI coordinates were transformed to Talairach space (using Matthew Brett's formula, http://www.mrc-cbu.cam.ac.uk/Imaging/Common/mnispace.shtml).

Discussion

To the authors` knowledge, this is the first report of an isolated loss of the sense of self-ownership. As mentioned in the introduction the disappearance of such a phenomenon may serve as an indirect proof of its previous existence and, indirectly, points to an identifiable neurobiological basis. That means that this aspect of the 'minimal self' as a philosophical construct probably has a neurobiological basis which exists independently from philosophical theories. The finding thereby challenges the widely accepted philosophical conviction of the immunity to error through misidentification relative to the first-person pronoun [21,22]: The access to my self in first-person experience is not necessarily immediate and non-observational, and it is not just a logical consequence of the structure of language and grammar. Instead, as shown in the case report, the access may involve a perceptual or reflective act of consciousness different from the notion of the immunity principle. Critics may argue that we did not describe a loss of self-ownership, since self-consciousness was only delayed but not abolished. However, the minimal self is defined by being unextended in time and even if the sense of self-ownership was not lost entirely, at least its defining characteristics and quality were affected. Furthermore our case shows an unexpected dissociation of an impaired sense of self-ownership only for perceptions of objects but not for

perceptions of the own body and actions. Although this dissociation is not predicted by philosophical notions of self-ownership it can be plausibly explained within a neuro-cognitive framework further discussed below.

We demonstrated abnormal functioning in inferior temporal, parieto-occipital and precentral regions which was associated with the loss of the sense of self-ownership for perceptions of objects. Although a single case can never reveal whether abnormalities in a brain region are sufficient or even necessary to evoke abnormal experiences, we may speculate that the identified temporal and parieto-occipital regions of hypometabolism are at least part of the network of brain regions underpinning the sense of self-ownership. The observed selectivity of impairment for self-ownership of object but not body- or action-perceptions points to a distributed system of brain regions which are involved in different aspects of the sense of self-ownership. This is further corroborated by the fact that hypometabolism occurred predominantly within right inferior temporal and parieto-occipital regions known to be crucial for visual object and visuo-spatial representation but not within inferior parietal regions previously demonstrated to be relevant for self-ownership of bodily actions [11] and which have been implicated as key to distinguishing between self and other [12]. The parahippocampal gyrus has been demonstrated to represent visual scenes [23] and the inferior temporal cortex is known to be necessary for representations of objects [24] as part of the ventral visual "what" stream, whereas the parieto-occipital cortex is part of the dorsal visual or "where" stream for object representation [24]. The role of the parahippocampal gyrus in associative (episodic) memory is still debated [25]. Normal performance on episodic memory and picture naming (which requires intact visual object recognition) shows that temporal hypometabolism demonstrated here cannot be compared to structural lesions following for example herpes simplex encephalitis where patients with bilateral inferior temporal lobe damage typically show severe impairments on picture naming

for animals [26] (included in our naming test, details see [19]) which may be partly confounded with general impairments of visual object recognition [27].

In addition, we found hypometabolism in cerebellar and motor regions which have been implicated in predicting the sensory consequences of own movements necessary for the sense of self-agency in the motor domain [28] [29,30]. It is interesting to speculate why, despite these abnormalities, the sense of self-agency for movements was not compromised in our patient, one possibility is that regional dysfunction within the cerebellum and right precentral cortex were quite focal and may have been compensated for by left hemispheric or adjacent regions, such compensation mechanisms can be effective even in patients with large irreversible lesions due to stroke and severe language impairment (e.g. [31]).

Most studies of self-consciousness have used measures of self-reference as the experimental factor of interest. These studies point to cortical midline structures (medial prefrontal and medial parietal cortex) as the substrates of self-referential processes (reviewed in [1]). On the other hand, temporal binding of distributed neural assemblies has been postulated as the neural correlate of the experience of the self [32]. There is solid evidence for the importance of temporal binding of distributed neural networks underlying the experience of perceptual gestalt [33,34]. Therefore our finding of abnormalities in areas known to be important for object perception as associated with a qualitative change in the experience of self-ownership of visual objects is very plausible when this sense of self-ownership is assumed to arise by the temporal binding and integration of object representations within the inferior temporal lobe and other higher-order representations within cortical midline structures [1] and the inferior parietal lobes [12].

Taken together, our finding of selective changes in the quality of the sense of self-ownership for perceptions of objects but not actions and an intact sense of self-agency points to anatomically separable systems underpinning different aspects of the ‘minimal self’. The

associated hypometabolism in areas of the ventral and dorsal visual stream but not within areas previously linked to self-consciousness (inferior parietal and midline structures) are most parsimoniously explained when self-consciousness is not assumed to be an anatomically localised cognitive function, but instead is conceived as emerging from integration across anatomically distributed networks of regions with different functional specializations, not all of which need to be specific to the 'self'.

With regard to the general psychopathology of disorders of self-awareness, our case points to the necessity to distinguish between phenomenologically different symptoms of abnormal self-experience. A refined psychopathological phenomenology of self-awareness may help to aid in the distinction of schizophrenia in which a loss of the sense of self-agency is known to occur from abnormalities of the "minimal self" due to other causes in which there is a selective loss of the sense of ownership for perceptions as described here.

Acknowledgements

RZ was supported by a German Academy of Natural Scientists Leopoldina Fellowship (BMBF-LPD 9901/8-122) and a Stepping Stones Research Fellowship of the Faculty of Medical & Human Sciences of the University of Manchester.

References

1. Northoff G, Heinzel A, de Greck M, Bermpohl F, Dobrowolny H, Panksepp J: Self-referential processing in our brain--a meta-analysis of imaging studies on the self. Neuroimage 2006;31:440-457.
2. Kircher TT, David AS: Self-consciousness: an integrative approach from philosophy, psychopathology and the neurosciences. in Kircher TT, David AS (eds): The Self in Neuroscience and Psychiatry. Cambridge: Cambridge University Press, 2003, 445 - 475.
3. Gallagher II: Philosophical conceptions of the self: implications for cognitive science. Trends Cogn Sci 2000;4:14-21.
4. Gillihan SJ, Farah MJ: Is self special? A critical review of evidence from experimental psychology and cognitive neuroscience. Psychol Bull 2005;131:76-97.
5. Sato A, Yasuda A: Illusion of sense of self-agency: discrepancy between the predicted and actual sensory consequences of actions modulates the sense of self-agency, but not the sense of self-ownership. Cognition 2005;94:241-255.
6. Bleuler E: Dementia praecox oder Gruppe der Schizophrenien. in Aschaffenburg G (ed): Handbuch der Psychiatrie. Leipzig: Deuticke, 1911.
7. Frith CD, Blakemore SJ, Wolpert DM: Abnormalities in the awareness and control of action. Philos Trans R Soc Lond B Biol Sci 2000;355:1771-1788.
8. Sirigu A, Daprati E, Pradat-Diehl P, Franck N, Jeannerod M: Perception of self-generated movement following left parietal lesion. Brain 1999;122 (Pt 10):1867-1874.
9. Spence SA, Brooks DJ, Hirsch SR, Liddle PF, Meehan J, Grasby PM: A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). Brain 1997;120:1997-2011.
10. Kircher TT, Leube DT: Self-consciousness, self-agency, and schizophrenia. Conscious Cogn 2003;12:656-669.
11. Shimada S, Hiraki K, Oda I: The parietal role in the sense of self-ownership with temporal discrepancy between visual and proprioceptive feedbacks. Neuroimage 2005;24:1225-1232.
12. Decety J, Sommerville JA: Shared representations between self and other: a social cognitive neuroscience view. Trends Cogn Sci 2003;7:527-533.
13. Leube DT, Knoblich G, Erb M, Kircher TT: Observing one's hand become anarchic: an fMRI study of action identification. Conscious Cogn 2003;12:597-608.
14. David N, Bewernick BH, Cohen MX, Newen A, Lux S, Fink GR, Shah NJ, Vogeley K: Neural representations of self versus other: visual-spatial perspective taking and agency in a virtual ball-tossing game. J Cogn Neurosci 2006;18:898-910.
15. Zimmermann P, Fimm B: Testbatterie zur Aufmerksamkeitspruefung. In, Psytest, 2005.
16. Horn W: Leistungspruefsystem L-P-S. ed 2, Goettingen, Toronto, Zuerich, Verlag fuer Psychologie Dr. C.J. Hogrefe, 1983.
17. Lezak M: Neuropsychological Assessment. Portland, Oregon Health Sciences University, 1995.
18. Härting C, Markowitsch HJ, Neufeld H, Calabrese P, Deisinger K: WMS-R Wechsler Gedächtnis Test - Revidierte Fassung. Goettingen, Hogrefe Verlagsgruppe, 2000.
19. Zahn R, Juengling FD, Bubrowski P, Jost E, Dykierek P, Talazko J, Hull M: Hemispheric asymmetries of hypometabolism associated with semantic memory impairment in Alzheimer's disease: a study using positron emission tomography with fluorodeoxyglucose-F18. Psychiatry Research Neuroimaging 2004;132:159 -172.
20. Association AP: Diagnostic and statistical manual of mental disorders. ed 4th, Washington D.C., APA, 2000.

21. Wittgenstein L: The blue and brown books. Blackwell, 1958.
22. Shoemaker S: Identity, cause, and mind. Cambridge, Cambridge University Press, 1984.
23. Downing PE, Chan AW, Peelen MV, Dodds CM, Kanwisher N: Domain specificity in visual cortex. Cereb Cortex 2006;16:1453-1461.
24. Ungerleider LG, Haxby JV: 'What' and 'where' in the human brain. Curr Opin Neurobiol 1994;4:157-165.
25. Mayes A, Montaldi D, Migo E: Associative memory and the medial temporal lobes. Trends Cogn Sci 2007;11:126-135.
26. Gainotti G: What the locus of brain lesion tells us about the nature of the cognitive defect underlying category-specific disorders: A review. Cortex 2000;36:539-559.
27. Borgo F, Sgaramella TM, Penello B, L'Erario R, Toso V: A componential analysis of visual object recognition deficits in patients with Herpes Simplex Virus Encephalitis. Brain and Cognition 2000;43:53-56.
28. Blakemore SJ, Frith CD, Wolpert DM: The cerebellum is involved in predicting the sensory consequences of action. Neuroreport 2001;12:1879-1884.
29. Blakemore SJ, Wolpert DM, Frith CD: Central cancellation of self-produced tickle sensation. Nat Neurosci 1998;1:635-640.
30. Leube DT, Knoblich G, Erb M, Grodd W, Bartels M, Kircher TT: The neural correlates of perceiving one's own movements. Neuroimage 2003;20:2084-2090.
31. Zahn R, Schwarz M, Huber W: Functional activation studies of word processing in the recovery from aphasia. J Physiol Paris 2006;99:370-385.
32. Lutz A, Thompson E: Neurophenomenology. Journal of Consciousness Studies 2003;10:31-52.
33. Engel AK, Singer W: Temporal binding and the neural correlates of sensory awareness. Trends Cogn Sci 2001;5:16-25.
34. Tononi G, Edelman GM: Consciousness and complexity. Science 1998;282:1846-1851.
35. Rorden C, Brett M: Stereotaxic display of brain lesions. Behavioural Neurology 2000;12:191-200.
36. Signorini M, Paulesu E, Friston K, Perani D, Colleluori A, Lucignani G, Grassi F, Bettinardi V, Frackowiak RS, Fazio F: Rapid assessment of regional cerebral metabolic abnormalities in single subjects with quantitative and nonquantitative [18F]FDG PET: A clinical validation of statistical parametric mapping. Neuroimage 1999;9:63-80.