

Psychic tonus, body schema and the parietal lobes: A multiple lesion case analysis

C.M.J. Braun*, S. Desjardins, S. Gaudet and A. Guimond

Centre de Neurosciences de la Cognition and Department of Psychology, Université du Québec à Montréal, Canada

Abstract. The psychic tonus model (Braun and colleagues, 1999, 2002, 2003, 2006) states that the left hemisphere is a “booster” of internal experience and behavior in general, and that the right hemisphere is a “dampener”. Twenty-five patients with a “positive” extreme disturbance of body schema (somatoparaphrenia) and 37 patients with a “negative” disturbance of body schema (autotopagnosia or Gerstmann’s syndrome), all following a unilateral parietal lesion, were found in the literature and were analyzed to test predictions from Braun’s “psychic tonus” model. As expected, patients with a positive syndrome had a right hemisphere lesion significantly more frequently, and those with a negative syndrome had a left hemisphere lesion significantly more frequently. Thus the psychic tonus model of hemispheric specialization, previously supported with regard to psychomotor baseline, libido, talkativeness, memory, auditory and visual perceptual tonus, now incorporates the tonus of representation of the body (body schema) in the parietal lobes.

Keywords: Body schema, psychic tonus, hemispheric specialization, parietal lesions, hypergnosia, hypognosia

1. Introduction

1.1. *Psychic tonus: a hemispheric specialization interpretation of positive and negative symptoms*

Braun and colleagues (see Braun [1] for an in depth account) have developed a model of hemispheric specialization initially based on effects of focal lesions which they have termed “psychic tonus”. This model proposes a new framework (but see Galin [2] for some premonitory ideas) for interpretation of positive and negative symptoms. The model draws equally from psychiatry as it does from neurology, as well as the biopsychology of normal and abnormal function in humans and animals. The model is based on demonstrations to the effect that mood [3], psychomotor baseline [3], libido [4], talkativeness [5], immune func-

tion [6], memory [7], visual symptoms [8] and auditory symptoms [9] are all modulated in similar opposed ways by the two hemispheres, in right handers. The normal left hemisphere increases psychic tonus (inferred from right hemisphere lesions), while the right decreases it (inferred from left hemisphere lesions). In short, an organism with high psychic tonus will be mentally and behaviorally activated. Even the immune system is up-regulated by left hemisphere activation. An organism with low psychic tonus will be all the contrary and will be characterized by right hemisphere activation.

The issue of whether perceptual function also falls under the orbit of hemispheric specialization for psychic tonus remains pending. Braun and colleagues [10] found that there were significantly more reports of cases of post lesion visual hallucination with right hemisphere lesions than left in the behavioral neurology literature. It therefore appears reasonable to predict that post lesion perceptual anomalies should generally manifest a dissociation according to lesion side. Left hemisphere lesions should produce negative perceptual anomalies, a dulling of perceptual representation which we shall term *hypognosia*, while right hemisphere lesions should produce positive perceptual

*Address for correspondence: Claude M.J. Braun, PhD, Full professor, Centre de Neurosciences Cognitives UQAM, C.P 8888, Succ. Centre-Ville, Montréal, Québec, Canada, H3C 3P8. Tel.: +1 514 987 3000 (4814); Fax: +1 514 987 8952; E-mail: Braun.Claude@UQAM.CA; URL: <http://www.er.uqam.ca/nobel/r31210/home.html>.

anomalies such as hallucinations, a lushness of perceptual representation, which we shall term *hypergnosia*. More prosaically, “positive” symptoms are understood here to consist of occurrence of undesired, unsolicited representations which ought not occur in a normal subject. We understand “negative” symptoms as inability to mobilize representations which should easily be mobilized at will by a normal person.

One problem with using neurological patients to test the veridicity of the “psychic tonus” concept is that extreme manifestations are required. We acknowledge that such cases are very rare, and that many cases, if not most, may carry bilateral lesions. Pure and extreme perceptual disorders resulting from well documented unilateral brain lesions are too rare to be assembled in a single analysis from any one clinical data base (neurology or psychiatry files of any one institution). That is why the exponentially increasing explosion of case reports today provides a data base fit for inference testing. The present report will limit itself to the somesthetic modality, and to cases with parietal lobe lesions exclusively.

Hemispheric specialization for somesthetic perception has been far less investigated than it has been for vision. Several authors have noted that hysterical somatic symptoms are more typically manifest on the left side of the body (see [11] for a review). This is compatible with the psychic tonus model but it is not the ideal test. As will be explained in the next sections, the ideal test of the psychic tonus model in the somesthetic modality would consist of determining the laterality of focal lesions causing a massively “positive” distortion of body schema versus a massively “negative” distortion. As far as we could determine, body schema has not been subjected to any such analysis crowned by inference tests of lesioned patients designed to test theoretical models of hemispheric specialization.

1.2. *Psychic tonus and “body schema”*

In their review of post lesion hallucinosis, Braun and colleagues (2003) observed that pure somesthetic hallucination tends to result from right parietal lesions, a finding which has been noted by several predecessors [12]. However, post lesion somesthetic hallucination has been too rare to support inference testing regarding its localisation. Likewise, pure post lesion autotopagnosia or loss of body schema has been noted to nearly always result from left parietal lesions [13,14]. The purpose of the present report was thus primarily to assemble an exhaustive review of single cases with ra-

diological documentation of a focal unilateral parietal lesion leading either to a positive form of distortion of “body schema” (bilateral somesthetic hallucinosis) or to a negative form (bilateral somesthetic agnosia).

1.3. *Disorders of body schema*

The term “body image” is often a misnomer in clinical neuroscience. Most clinical neuroscience authors, when talking about “body image” are not talking about body *image* at all, but rather about body *schema*, i.e., of somesthetic representation of one’s own body rather than of visual representation. Though the term body image persists, we contend that it should be replaced by “body schema”. Indeed, the congenitally blind have body schema, i.e., high level representation of body parts. It is significantly less precise than in normals, but it is far from autotopagnosia [15] and this is achieved despite complete absence of visual imagery, even from birth. So obviously visualisation need not be critical in higher order representation of the body. Also, estranged body schema is probably more easily expressed in “visual” terms by patients with expressions such as “I see my body split in two”, “I see a third limb on myself”, etc., when in probable fact, they “feel” those things somesthetically, and translate that feeling into expressions designed to be better understood by the interlocutor. Indeed, when a patient complains of a supernumerary limb, the first comments of the clinician are likely to be: “do you see it? show me where it is . . . etc.”. Suffice it to state that, for the purposes of the present report, we take the term “body schema” to designate a primarily somesthetic high level representation of the body.

The quintessential positive disorder of body schema is somatoparaphrenia [16]. When it is pure and is caused by a unilateral lesion, the localisation of the lesion is typically right parietal (as we shall explicate in the results section). In the extreme form of somatoparaphrenia complex somesthetic hallucinations involve feelings of supernumerary or abnormally sized or deformed body parts, of feelings of being touched, or of involuntary movement involving both sides of the body. Other terms referring to less obviously “positive” anomalies of body schema include dysmorphobia (or body dysmorphic syndrome), autoscopia, micro and macrosomatognosia, phantom limb, auto-reduplication, self misidentification syndrome (reverse Capgras or Fregoli syndrome, see [17], somatisation disorder, and perhaps heautoscopy and autoscopia though the latter two are visual by definition.

The quintessential and most extreme “negative” disorder of body schema is autotopagnosia [18]. It consists of a complete inability to localize or represent one’s own body parts on both sides, a bilateral high level somesthetic agnosia, which often comprises bilateral anosagnosia as well. When it is pure and is caused by a unilateral lesion, the lesion is typically left parietal. Other terms referring to similar but less encompassing negative anomalies of body schema include Gerstmann’s syndrome (especially the finger agnosia and right-left confusion elements), asymbolia for pain, anosagnosia, etc. Hécaen and Albert [19] considered Gerstmann’s syndrome a mild form of autotopagnosia. Gerstmann’s syndrome is relevant to the “psychic tonus” model primarily because one of its symptoms is an intrinsically bilateral distortion of body schema”, namely right-left confusion. We have found that when investigated for such, finger agnosia in Gerstmann’s syndrome is also typically bilateral (see the results section), making that syndrome all the more relevant for the present report. Pure Gerstmann’s syndrome is nearly always caused by a left parietal lesion. Since many cases of Gerstmann’s syndrome have been published, this syndrome was also reviewed systematically for the present report.

1.4. *Unilateral neglect syndromes and the “psychic tonus” model*

There is overwhelming evidence in behavioral neurology to the effect that unilateral neglect, in any modality – including somesthetic (even with regard to representation of the body), results far more often from right than left lesions, and that the typical lesion producing hemineglect is inferior parietal. We believe that there is probably some truth to this, though exaggerated. A pure test of tactile hemineglect must exclude any motor exploration component. Tactile extinction fits that requirement. Gainotti and colleagues [20] found that large cohorts of patients with right and left telencephalic lesions presented no lateralized difference in tactile extinction after correction for errors on the body side ipsilateral to the lesion. Schwartz and colleagues [21] obtained similar results with 274 patients with unilateral brain damage. This explains why many authors may have observed more tactile extinction after right hemisphere lesions: patients with massive left hemisphere lesions tend to get excluded from neuropsychological studies because they are too aphasic to understand elementary instructions, and/or left hemisphere lesioned patients present more bilateral neglect. In short, we

believe the so-called “right hemisphere hemineglect” phenomenon is none other than a standard contralesional functional loss which is far less observed in left hemisphere cases simply because the neglect is bilateral in those cases. Nevertheless, the generally recognized propensity of right parietal lesions to produce hemineglect could be construed as the main objection (or perhaps the more exact word would be “challenge”) which could be leveled, in the “perceptual domain”, against the “psychic tonus” model of hemispheric specialization. Indeed, the psychic tonus model predicts negative cognitive-perceptuomotor symptoms after a left hemisphere lesion, rather than a right hemisphere lesion. Our answer to this is as follows. The dominant models of hemineglect are variants of what could be called the class of “subcortical mediation of orienting”. Heilman and colleagues for example, propose a lateralized “corticolimbic reticular loop” and Kinsbourne proposes a “brainstem asymmetry”, of lateral orientation [22]. These models insist on primitive pre-representational factors to the detriment of representational factors. One argument for this class of models is that hemineglect is often furtive: it tends to disappear spontaneously after a few weeks after the lesion – suggesting that it is due to a simple primitive mechanism that can be compensated for by representational feedback (the patient learns to invest extra attentional energy to the previously neglected side). Another argument for this class of models is that patients with left hemineglect are hyper-oriented to the right side more than they are neglectful of the left side (they look more and they grope more on that side [23]). A third argument in support of this class of models is that subcortical lesions often produce hemineglect, including in the tactile modality [24].

The psychic tonus model has a wider angle than these diverse explanations of hemineglect. It proposes that representational neglect can be bilateral (e.g., autotopagnosia) following a unilateral telencephalic lesion of the left hemisphere, and that the same lesion located in the right hemisphere is more likely to produce the contrary syndrome, a syndrome of pathologically exuberant bilateral representation (e.g., somatoparaphrenia). Since we demonstrate that this is what typically occurs (see the results section), we are led to concur with authors who interpret hemineglect as a pre-representational defect of orientation involving subcortical brain structures. Thus, lesions of the right inferior parietal lobe ought to produce somatoparaphrenia or related symptoms – as well as unilateral left hemineglect, and these two symptom complexes ought to be considered relatively independent – despite the fact

that the lesion engendering them can be the same. We agree with Heilman and Kinsbourne that the parietal cortical lesion producing hemineglect probably causes that symptom via loss of connections to subcortical structures, but we add that somatoparaphrenia is a high level representational disorder probably caused directly by damage to parietal cortex. The psychic tonus model proposes that left parietal lesions ought to produce bilateral neglect (of which autotopagnosia is the severest form), without hallucinosis, and as stated above, that is exactly what most often occurs.

Many authors believe that somatoparaphrenia is nothing other than compensation for hemineglect [12, 25–28]. However, somatoparaphrenia is not always a hallucination of an extra body part on the left, it can be on the right body side or both sides or the body meridian, and hemiplegia and hemineglect are not necessarily present (see our results section). Somatoparaphrenia is clearly a “positive” syndrome because it typically consists of spontaneous and exuberant representation of something (e.g., a third limb) that is not in the “normal” repertoire.

2. Method

The selection criteria of the cases to be assembled were optimized to assure meaningful interpretation of localisation of brain damage in view of testing the “psychic tonus” model of hemispheric specialization with regard to “body schema”. First, to assure that cases with right versus left pathology would be comparable and relevant, only etiologies involving the parietal lobes were considered. Careful attention was paid to lesion localization within the hemisphere to assure against a lesion size artefact. Several other possible intervening variables (age, gender, presence/absence of EEG, presence/absence of neurological exam, date of publication, presence/absence of aphasic symptoms, lesion etiology, presence/absence of hemineglect, psychiatric comorbidity, hemibody where the hallucination occurs) were also systematically tabulated for all cases.

3. Results

3.1. Positive distortions of body schema

The first table reviews published cases of the most extreme form of positive distortion of body schema resulting from a unilateral parietal lesion, with or without

hemineglect. Cases with hemineglect are slightly less telling (for a test of the psychic tonus model of hemispheric specialization) than cases without hemineglect because there probably is indeed a relation between unilateral hallucinosis and unilateral hemineglect – consisting of compensation for sensory deprivation. Such cases present a mixture of positive and negative symptoms – to the extent that hemineglect can be construed as a “negative” symptom (though we think it should not be considered negative psychic tonus). We found only one case presenting both somatoparaphrenia and autotopagnosia and he was excluded because of the presence of both positive and negative body schema symptoms. Other less obviously “positive” bilateral higher order syndromes of distorted body schema (e.g., somatisation disorder, reverse Capgras syndrome) have been reported much less frequently to result from unilateral brain lesions. We found only two cases of reverse Capgras syndrome following a unilateral lesion, and though both had right hemisphere lesions, these were not parietal [29,30]. We found only three cases of somatisation disorder after a unilateral parietal lesion [31–33]. All three had a right hemisphere lesion. However, in the next table, we review only cases with somatoparaphrenia following a unilateral parietal lesion. See Table 1.

Of the 25 cases of somatoparaphrenia following a unilateral parietal lobe lesion of table 1, 24 had a lesion on the right and one on the left (binomial: $p < 0.0005$, two tailed).

We believe left and right hemisphere lesions have equiprobable chances of occurring and being selected for case reports (in the absence of selection for any type of mental disturbance). For example, Montour-Proulx and her colleagues [59] assembled 635 previously published post-lesion cases without selection for any psychiatric symptom. In their data base, 328 cases were left hemisphere damaged and 307 were right hemisphere damaged (binomial probability against equiprobability: $p > 0.12$, two tailed).

3.2. Negative distortions of body schema

The next table reviews published cases of the most prototypically negative and complete variant of distortion of body schema, namely autotopagnosia -resulting from a unilateral parietal lesion. By definition, all these cases presented bilateral symptoms. Because several authors consider that autotopagnosia is a semantic aphasia [51], cases without aphasia represent a more interesting test of the psychic tonus model of hemi-

Table 1
Cases of somatoparaphrenia following unilateral parietal lesions

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
72 Female H?	Right temporo-parietal	Infarct	Somatoparaphrenia (supernumerary left limb), left hemineglect	No mention of EEG or seizures	?	Worthington et al., 1996 [28]
64 Female LH	Right > Left parietal	Meningioma	Somatoparaphrenia (2 legs on each side, uninterrupted for two weeks), slight left visuospatial hemineglect	A few partial seizures of the left leg are mentioned, but EEG is not mentioned	Not confused or disoriented	Vuilleumier et al., 1997 [34]
41 Male RH	Right parieto-temporal	Hematoma	Somatoparaphrenia (supernumerary feet, hands and fingers on the left), left hemineglect that disappeared after 4 months	No mention of EEG or seizures	Impulsivity in keeping with his previous life-style, "no evidence of psychotic paranoid ideas or depressive symptoms"	Halligan et al., 1995 [26]
50 Female RH	Right parietal	Hematoma after clipped aneurism due to a artero-venous malformation	Bilateral somatoparaphrenia (macropsia of self and others, misidentification of limb), alien hand syndrome (LH), no hemineglect	No signs of paroxysm in EEG, only slowing over lesion	?	Leiguardia et al., 1993 [35]
85 Female H?	Right parietal and thalamic	Infarct	Somatoparaphrenia (misidentification of left body), no hemineglect	No mention of seizures or EEG	?	Paulig et al., 2000 [36]
64 Male H?	Right parieto-temporo-occipital	Infarct	Somatoparaphrenia (left arm is a live baby), no hemineglect	No mention of seizures or EEG	No history of psychiatric illness, agitation and delusions following the stroke, visual and auditory hallucinations	Richardson, 1992 [37]
36 Male RH	Right parieto-occipital	Oligodendroglioma	Somatoparaphrenia (autoscopy in left field), no hemineglect	EEG revealed only slowing over the lesion, no evidence of epilepsy	?	Maillard et al., 2004 [38]
46 Male RH	Right parietal	Meningioma	Bilateral somatoparaphrenia (his right body slides behind his left, features of the right face are "more prominent" than the left), the devil controls left side, auditory and visual hallucinations, mild left hemineglect	The patient had epilepsy but the somatoparaphrenia was continuous and the interictal EEG was normal	Heavy alcohol use, depression, psychosis and delusions	Nightingale et al., 1982 [39]

Table 1, continued

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
69 Female H?	Right parieto-temporo-occipital	Infarct	Somatoparaphrenia (misidentification of sentient left arm and thumb), anosagnosia, left hemineglect	No mention of EEG or seizures	Logorhea	Rode et al., 1992 [27]
63 Male RH	Right parieto-fronto-temporal	Infarct	Bilateral somatoparaphrenia (midline limb), apraxia, spatial agraphia, acalculia, left hemineglect	Right fronto-temporal slowing of EEG	Psychotic episode, delusions, mania, psychosis, confabulation, depression and paranoia	Berthier et al., 1987 [40]
86 Female RH	Right parieto-fronto-temporal	Emboly	Somatoparaphrenia (misidentification of sentient left hand), anosagnosia, left hemineglect	No mention of EEG or seizures	No history of psychiatric illness	Assal, 1983 [41]
84 Female RH	Right fronto-parieto-occipital	Infarct	Somatoparaphrenia (attributes sentient left limb to someone else), visual left hemineglect	No EEG or seizures reported	?	Bisiach et al., 1991 [42]
77 Female RH	Right fronto-parieto-temporal +putamen and pallidum	Hemorrhage	Somatoparaphrenia (her left sentient hand belongs to someone else), left hemineglect, anosognosia	No EEG or seizures reported	“Oriented in time and space and did not show any other sign of mental deterioration”	Bottini et al., 2002 [12]
85 Female H?	Right parietal and thalamic	Infarct	Somatoparaphrenia (misidentification of left body), left hemineglect	No mention of seizures or EEG	?	Paulig et al., 2000 [36]
76 Female H?	Right parieto-occipital	Infarct	Somatoparaphrenia (content unspecified), left hemiplegia, left neglect	No mention of EEG or seizures	?	Ramachandran, 1996 [43]
57 Female H?	Right hemisphere	Several hemorrhages	Somatoparaphrenia (supernumerary hands), no hemineglect	EEG slowing over the right hemisphere, no mention of seizures	Depression	Weinstein et al., 1954 [44]
77 Male RH	Right occipito-parieto-temporal	Infarct	Somatoparaphrenia (supernumerary arms) on the right hemibody, left hemineglect	No mention of EEG or seizures	?	Sellal, 1996 [45]
70 Female H?	Right parieto-temporal	Atrophy (ganglionic degeneration)	Somatoparaphrenia (misidentification of the left hand), no hemineglect	EEG slowing over the lesion, right hypoperfusion on SPECT, no mention of seizures	?	Carrilho et al., 2001 [46]

Table 1, continued

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
75 Female H?	Right parieto-temporal	Atrophy	Somatoparaphrenia (misidentification of the left hand), no hemineglect	No mention of EEG or seizures	?	Carrilho et al., 2001 [46]
63 Male H?	Left > Right parietal	Atrophy	Somatoparaphrenia (misidentification of the right hand), right hemineglect	EEG slowing over the lesion, left > right temporo-parietal hypoperfusion on SPECT, no mention of seizures	?	Carrilho et al., 2001 [46]
60 Female H?	Right parietal	Infarct	Somatoparaphrenia (misidentification of the left hand), no hemineglect	No mention of EEG or seizures	?	Carrilho et al., 2001 [46]
35 Male H?	Right capsulo-lenticular, parieto-frontal	Hematoma	Somatoparaphrenia (supernumerary left arm or leg), left hemineglect	No mention of EEG or seizures	?	Donnet et al., 1997 [25]
61 Female H?	Right sub-parietal white matter	Hemorrhage	Somatoparaphrenia (supernumerary left arm), no hemineglect	No mention of EEG or seizures	No delusions or confabulation, reactive depression	Canavero et al., 1999 [47]
36 Male H?	Right parietal	Aneurysm	Somatoparaphrenia (misidentification of the left hemibody, supernumerary legs and arms), no hemineglect	No mention of EEG or seizures	?	Fredericks, 1963 [48]
26 Male H?	Right parietal	Penetrating wound	Somatoparaphrenia (autoscopy, sensation that his body was dislocated, macroscopy), no hemineglect	No mention of EEG or seizures	No previous experience of depersonalization or any other psychiatric disturbance	Lunn, 1970 [49]

Note. None of these cases were reported to have aphasic symptoms.

spheric specialization. Presence of aphasic symptoms was systematically noted. See Table 2.

All four symptoms of Gerstmann's syndrome (agraphia, acalculia, right-left confusion, finger agnosia) were thought by Gerstmann [16] to be high-order disorders of "body image". The rationale for acalculia being a disorder of body schema is that we learn to count with our fingers. The rationale for agraphia being a disorder of body schema is that the agraphia is postulated to be peripheral rather than central, i.e., putatively proprioceptive and/or apraxic. It is frequently argued that Gerstmann's syndrome always comprises and is essentially explainable as a "defective process of mental manipulation of images" [52–55]. Several authors

have believed that Gerstmann's syndrome is a form of aphasia [56–58] and one group has even specified that the aphasia is semantic and that the main problem is a breakdown of the part/whole dialectic [59]. Presence of aphasia weakens our test of the psychic tonus model because aphasia falls under the orbit of a form of hemispheric specialization (linguistic) which we fear may be orthogonal to the issue at hand (though we are not sure of that yet). We therefore documented any mention of aphasia. Others have argued that the syndrome does not exist in pure form. However, all the controversy does not detract from the fact that bilateral finger agnosia and right-left confusion (especially relative to self) are obvious defects of body schema of

Table 2
Cases of autotopagnosia or Gerstmann's syndrome following a unilateral parietal lesion

Age at onset, gender and hand preference	Lesion locus	Etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity/ aphasia	Reference
49 Male H?	Left parietal	Glioma	Autotopagnosia (bilateral), no aphasia	Diffuse slowing of EEG	No mention of psychiatric comorbidity, no aphasia	De Renzi & Faglioni, 1963 [60]
64 Male RH	Left parietal	Tumor	Autotopagnosia (bilateral), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	De Renzi & Scotti, 1970 [61]
59 Male RH	Left parietal	Cystic tumor (metastatic carcinoma)	Autotopagnosia (bilateral), Gerstmann syndrome, ideomotor apraxia, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Ogden, 1985 [62]
67 Male RH	Left parietal	Encephalomalacia	Autotopagnosia (bilateral), dyscalculia, dysgraphia, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, aphasia	Denes et al., 2000 [13]
74 Female LH	Right parieto-temporo-occipital	Hemorrhagic infarct	Autotopagnosia (bilateral), limb apraxia, neglect syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Verstichel et al., 1994 [63]
64 Female H?	Left parietal	Anaplastic astrocytoma	Autotopagnosia (bilateral), anomia	No mention of EEG or seizures	No mention of psychiatric comorbidity, anomia	Baldini et al., 1978 [5]
56 Male RH	Left parieto-occipital	Hemorrhage	Autotopagnosia (bilateral), mild dysphasia	Left EEG slowing without epileptiform signs	no mention of psychiatric comorbidity, mild dysphasia	Poncet et al., 1971 [59]
71 Female LH	Right parietal (right hemisphere dominant for language)	Encephalomalacia	Autotopagnosia (bilateral), aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, aphasia	Denes et al., 2000 [13]
77 Female RH	Left parieto-temporo-occipital	Infarct	Autotopagnosia (bilateral), ideomotor apraxia, aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, aphasia	Schwoebel et al., 2001 [14]
74 Female RH	Left parieto-occipital	Metastatic melanoma	Autotopagnosia (bilateral), mild aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, mild aphasia	Semenza, 1988 [64]
71 Male H?	Left parieto-occipital	Tissue softening	Complete autotopagnosia with consequent Gerstmann's syndrome	Normal neurological exam, no mention of imagery	No mention of psychiatric comorbidity, no aphasia	Nielsen, 1946 [65]
59 Male RH	Left parietal	Stroke	Gerstmann's syndrome, (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, mild word finding difficulties	Varney, 1984 [66]

Table 2, continued

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
48 Female H?	Left parieto-occipital	Tumor (multiform spongioblastoma)	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No mention of EEG, seizures or psychiatric comorbidity	No mention of psychiatric comorbidity, no aphasia	Arbuse, 1947 [67]
60 Female H?	Left parieto-occipital	Infarct	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No paroxysms in EEG, slowing over lesion	No mention of psychiatric comorbidity, no aphasia	Perez-Blanco et al., 1989 [68]
64 Male RH	Right parietal	Infarct	Gerstmann's syndrome, (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Calvo-Romero, 2000 [69]
71 Female RH	Left fronto-parietal	Hematoma	Gerstmann's syndrome, (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, mild word finding impairment, paraphasic errors	Maeshima et al., 1998 [70]
85 Male ambidextrous	Left parietal	Ischemic stroke	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Carota et al., 2004 [52]
57 Female LH	Right parietal	Stroke	Gerstmann's syndrome, (bilateral finger agnosia documented), dysphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, dysphasia	Moore et al., 1991 [71]
69 Male ambidextrous	Left parieto-occipital	Hemorrhage	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Dozono et al., 1997 [72]
52 Male RH	Left parietal	Stroke	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No EEG or seizures reported	Euphoria, no aphasia	Sobota et al., 1985 [73]
44 Male RH	Left parietal	Penetrating head injury	Gerstmann's syndrome (bilateral finger agnosia documented), no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Mazzoni et al., 1990 [74]
59 Male RH	Left parietal	Infarct	Gerstmann's syndrome (bilateral finger agnosia documented), impairment of visual rotation, no apraxia or aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Mayer et al., 1999 [75]
72 Female RH	Left parietal	Glioblastoma multiforme	Gerstmann's syndrome with bilateral toe agnosia (bilateral finger agnosia documented), no aphasia	No mention of seizures or EEG	No history of psychiatric illness, no aphasia	Tucha et al., 1997 [76]

Table 2, continued

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
58 Male RH	Left parietal	Stroke	Gerstmann's syndrome, (bilateral finger agnosia documented), semantic dysphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, semantic dysphasia	Ardila et al., 2000 [56]
7 Male H?	Left parietal	Infarct	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Ohtagaki et al., 1998 [77]
12 Male H?	Left parietal	Atrophy	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Garty et al., 1989 [78]
64 Male RH	Left parietal (angular and supra-marginal gyri)	Infarct	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Roeltgen et al., 1983 [79]
65 Male RH	Left parieto-occipital	Infarct	Gerstmann's syndrome without alexia, apraxia or aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, word-finding difficulty	Levine et al., 1988 [57]
56 Male RH	Left parieto-temporal	Infarct	Gerstmann's syndrome, no aphasia	EEG slowing over lesion, no mention of seizures	No mention of psychiatric comorbidity, no aphasia	Kinsbourne et al., 1974 [80]
47 Male RH	Left parietal	Astrocytic glioma	Gerstmann's syndrome, dysphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, dysphasia	Kinsbourne et al., 1962 [81]
65 Male LH	Left parieto-occipital	Meningioma	Gerstmann's syndrome, no aphasia	EEG slowing over lesion, no mention of seizures	No mention of psychiatric comorbidity, no aphasia	Kinsbourne, 1962 [81]
7 Male RH	Left parieto-occipital softening	Infarct	Gerstmann's syndrome, ADHD, learning disability, no aphasia	EEG was normal bilaterally, no mention of seizures	No mention of psychiatric comorbidity, no aphasia	Fournier-Del Castillo et al., 2000 [82]
67 Male RH	Left parietal	Stroke	Gerstmann's syndrome, aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, aphasia	Sheimo et al., 1997 [58]
65 Male RH	Left parietal	Haematoma	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Trillet et al., 1989 [83]
52 Male RH	Left parietal	Haematoma	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Trillet et al., 1989 [83]

Table 2, continued

Age at onset, gender and hand preference	Locus of the lesion	Lesion etiology	Somatic and other symptoms	Clinical considerations concerning possible epilepsy	Psychiatric comorbidity	Reference
68 Male LH	Right parietal	Infarct	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Dehaene et al., 1997 [84]
79 Male RH	Left parietal	Infarct	Gerstmann's syndrome, no aphasia	No mention of EEG or seizures	No mention of psychiatric comorbidity, no aphasia	Gold et al., 1995 [53]

the agnosic type (though manipulation of images may be a problem as well, and the problem can be verbally mediated), and these two symptoms affect both sides of the body, making them highly relevant tests of the “psychic tonus” model. At any rate, the controversy over Gerstmann’s syndrome has resulted in a large set of case reports, useful for testing the psychic tonus model.

Cases of Gerstmann’s syndrome following a unilateral parietal lesion which are better suited to testing the psychic tonus model are those where the finger agnosia is documented as bilateral. In Table 3, we noted whether the cases were documented for bilateral agnosia or not. See Table 2.

Of the 37 cases of Table 2, all with a negative disorder of body schema, 32 had a left hemisphere lesion and 5 had a right hemisphere lesion (binomial: $p < 0.0005$, two tailed).

Any issue of a general bias in published case reports for lesions in one or the other hemisphere is eschewed by a test of the prediction of a crossed double dissociation. All the post lesion cases with positive syndromes (somatoparaphrenia) ($N = 25$) were therefore compared to all the post lesion cases with a negative syndrome (autotopagnosia, Gerstmann’s syndrome) ($N = 37$). The crossed double dissociation between lesion side and type of somesthetic disorder is highly significant ($\text{Chi}^2 = 40.77$, $p < 0.0005$, two tailed).

3.3. Secondary analyses for purposes of artefact control (all cases, $N = 62$)

The main results reported above appear to be highly significant and thus, to be concordant with the psychic tonus model. However, it is important to determine whether that inference test withstands challenges from eventual contamination from extraneous sampling bias.

The analyses to be presented next comprise tests of alternative explanations of the interaction between

somesthetic disorder type and lesion side. We operationalized this interaction as a dichotomous variable which we termed “concordance with the psychic tonus model”. A left lesion associated with autotopagnosia (Gerstmann syndrome or a right lesion associated with somatoparaphrenia was rated as 1, the other eventualities were rated as 2. In the case of a significant relation between “concordance with the psychic tonus model” and an extraneous variable, a second phase of analysis was to be implemented, subjecting the theoretical effect to partial correlation – retrenching the variance of the contaminant. This analytical framework supposes that only dichotomous variables may be processed. Consequently all control variables were to be eventually dichotomized if not naturally limited to two levels.

3.4. Hand writing preference

Hand writing preference is an important variable because non-right handers present reversed hemispheric specialization in 10 to 30% of cases. Biased sampling of hand preference could thus bring a caveat to the main result (concordance with the psychic tonus model by lesion side). In this study, there were 32 right handers of which 31 were concordant with the psychic tonus model and 8 left handers or ambidextrals or which 4 were concordant with the psychic tonus model (the other cases’ hand preference was not reported). The relation between these two variables is significant, the non-right handers being less concordant with the psychic tonus model (50%) than the right handers (96.9%). A partial correlation was therefore computed between lesion side and type of somesthetic disorder, controlling for hand writing preference. The psychic tonus effect remained significant despite statistical removal of the effect of handedness ($rp = 0.82$, $p < 0.0005$).

3.5. Other control variables

Locus of the lesion was analyzed because though parietal lobe lesions were a selection criterion, damage elsewhere was not an exclusion criterion, and thus an extra parietal lesion sampling artefact had to be ruled out. Lesion size was analyzed because larger lesions can be thought to produce loss of mental function more often than smaller lesions, and thus a sampling bias could contribute an artefact. Etiology of the lesion was of interest because some etiologies, like tumor, can sometimes irritate and excite brain tissue and create cognitive symptoms [85]. Head trauma can also introduce noise into the data base because it presents with diffuse bilateral damage that is too subtle to be radiologically localized. Cases with old (ex: congenital) lesions could present far more cognitive compensation, adding noise to the inference tests. Presence of a psychiatric comorbidity was analyzed because somatic hallucination or delusion, though less frequent than auditory or visual hallucination, are not rare in psychotics [86,87]. The age of the patient is of interest because a bilateral diffuse stress on the brain is associated with aging. Juvenile cases also present less hemispheric specialization than adults. Gender was of interest because women tend to have less focal cognitive representation in the hemispheres and also less functional lateralization. The date of publication is pertinent because MRI technology used nowadays as a diagnostic method is more precise than CT scan. The presence of an EEG was of concern because it brings more precision to lesion localization and reduces chances of contamination by epileptiform activity. The presence/absence of aphasic symptoms is an important control variable because of the well known hemispheric specialization for linguistic abilities (see [88] for a review). However, none of these variables was significantly related to “concordance with the psychic tonus model”.

3.6. More specific analyses (somatoparaphrenic group only, $N = 25$)

In the neurological literature, it is well known that positive symptoms often occur in the contralateral or the neglected hemibody [89]. In the current study, 20 cases out of 25 had their target symptoms occurring in the hemibody contralateral to the lesion ($\text{Chi}^2 = 7.64$, $p = 0.054$). There are however four cases of right hemisphere lesions, thus concordant with the psychic tonus model, for whom the symptoms occurred bilaterally. Power was lacking to measure statistically the

Table 3
Distribution of symptom lateralization

Hemibody where the target symptom occurred			
Right	Left	Bilateral	Unspecified
3	17	4	1

psychic tonus model inference with these cases only. See Table 3.

In the current data base, there were 12 non neglecting cases and 13 cases of hemineglect. This variable was not related to lesion side and was thus given no further consideration.

4. Discussion

The results of this investigation strongly support the psychic tonus model –extending it to somesthetic perception in the parietal lobes. Unilateral parietal lesions typically cause a positive disturbance of body schema when they are located in the right hemisphere and a negative disturbance of body schema when they are located in the left hemisphere.

Why would a focal left parietal lesion, and only that lesion, impair a patient’s finger gnosis (mental representation of the finger) at both hands as it does in Gerstmann’s syndrome? Primary sensory somesthetic representation has been mapped in many species and has always been found to be fully contralateral and to present the same topography in each hemisphere in each posterolateral gyrus. None of the published reports on Gerstmann’s syndrome has addressed the question of why both hands should manifest finger agnosia –except with perplexity [73] and when the bimanual nature of the agnosia is revealed, it is mentioned only incidentally or obliquely. The fact that autotopagnosia [62,90,91] or Gerstmann’s syndrome is so often due to a left hemisphere lesion has inspired several authors (e.g., [67, 74]) to state that body schema is left-hemisphere specialized – a thesis we categorically reject. Likewise, the fact that somatoparaphrenia can be due to a right sided lesion has led several other authors (e.g., [47, 92]) to conclude that body schema is right-hemisphere specialized – a thesis we also categorically reject. The laterality of lesions causing disorders of body schema is often acknowledged and stated to be perplexing by many authors not willing to commit themselves to a single hemispheric repository for body schema. Our psychic tonus model provides a clear explanation of the laterality of the lesion findings and the bilaterality of symptoms in the various syndromes of higher order

body schema -and will hopefully put an end to some of these expressions of perplexity.

It seems to be a fact that when parietal neocortex is damaged unilaterally, control circuits of psychic tonus can become skewed, and a corresponding disorder of body schema can ensue, positive or negative, as a function of which hemisphere is damaged. Similar phenomena are observed for the other lobes and their respective functions: visual for the occipital lobes [8], auditory, sexual and paralinguistic for the temporal lobes [4,5,7,9], psychomotor for the frontal lobes [3]. These phenomena form an ensemble, a behavioral, motivational and attitudinal system which boils down to a certain type of energy expenditure/economy or approach/avoidance dynamic. Other metaphors of psychic tonus might include contrasts such as “up-beat/downtrodden” or “galvanized/inhibited”. For the moment, our favorite designation of psychic tonus is to view it as a mechanism of energy management [1, 93]. In normal behavior, the interplay of these opposed hemispheric mechanisms is finely tuned to situational demands – though we suspect that it oscillates slowly (recall that the immune response is involved, and we expect that various hormones and neurotransmitters are involved as well). However, after a unilateral lesion, the patient becomes set in a mode which is no longer delicately adaptive.

The findings presented here demonstrate that localized lesions can produce fractional effects on psychic tonus, such that only one representational modality may be affected – in the present case, somesthetic. Overall, the elements of psychic tonus tend to be correlated in normal and pathological conditions. Thus after a right hemisphere lesion, if a patient becomes manic, he/she (like any congenital manic) is also likely to be (but not necessarily) agitated, talkative, hypersexual, immunofacilitated, delusional and/or hallucinated. After a left hemisphere lesion, if a patient becomes depressed, he/she (like any congenital depressive) is likely to be (but not necessarily) lethargic, hypolalic, hyposexual, immunosuppressed and/or agnostic (see [1,3–5, 7–10] for reviews of the evidence).

It could be thought that the main mediator of the post lesion sticky switch is one or several neurotransmitters. Three good candidates are central norepinephrine serotonin and dopamine (all known to modulate functions subsumed under psychic tonus). Robinson and colleagues [94] ligated the middle cerebral artery of rats. Assays of brain catecholamines revealed 30 percent reductions of norepinephrine in the injured and uninjured cortex and locus coeruleus and a 20 percent reduction

of dopamine in the substantia nigra in the right lesioned rats who had also become hyperactive. In contrast rats with left middle cerebral artery ligations did not become hyperactive and did not show any significant change in catecholamines in any of the brain areas studied. Similar asymmetry occurs in serotonin concentrations as a function of stroke side in humans [95]. It remains to be determined whether the latter effect is hemispherically symmetrical or not (a PET ligand study). In fact however we believe that such a neurotransmitter-mediated mechanism is not the principal determinant of hemispheric specialization for psychic tonus effects reported here and elsewhere. Indeed we have found that in non lesional epileptics with unilateral foci ictal hallucination is highly significantly more often observed in cases with left than right foci [96]. This corroboration of hemispheric specialization for psychic tonus cannot be explained as a chronic and major change of neurotransmitter concentration in the brain.

Dominant contemporary methodology in cognitive neuroscience has perhaps not been fully prepared to unveil such mechanisms. Indeed, fMRI cannot be expected (at least at present and without specific precautions) to reveal clearly and unequivocally lateralized (hemispherically specialized) processing, in the normal brain, of higher order body schema, and indeed it generally does not, for example when movement of one limb is required to be imagined [97,98]. Normal high level representation of the body (or of any other cognitive or perceptual function) comprises a balance of positive and negative representation, soliciting both hemispheres for the typical task, thus activating both hemispheres (but each in different aspects of the processing: for example one hemisphere might prepare the movement and the other might inhibit the contralateral mirror movement). A normally optimized high level representation, requiring imagination, polysensory integration, sensorimotor integration, etc., must balance dispositions toward too much imagination (overshooting, tangentiality, profuseness, fabulation, hallucination, etc.) against dispositions toward too much conservatism (inhibition, paucity, inattention, apathy, sluggishness, agnosia, etc.). A typical fMRI experiment subtracts one task from another in view of isolating a specific cognitive operation, thereby canceling out psychic tonus. Most importantly, metabolic imaging studies of cases of extreme distortion of psychic tonus, specifically of body schema, do not form a corpus that can put a hemispheric specialization model to test. For intimate, subjective, complex experience such as the “body schema” the lesion method can provide insights

for behavioral neuroscience where more expensive and prized methods such as metabolic brain imaging need to refine the tasks used in their protocols. With regard to body schema, one intriguing fMRI study required normal subjects to distinguish self movement from movement of another person. This task activated the right parietal lobe, but no theoretical explanation was provided for the laterality of the effect [99]. The “psychic tonus” model would interpret the task as a requirement for inhibition of body schema. Another intriguing fMRI study required normal subjects to localize their own body parts. This activated the left parietal lobe [89]. The “psychic tonus” model would construe this task as a requirement for “activation” of one’s body schema. However, distinguishing other people’s movement from our own might call upon “inhibition” of one’s body schema.

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