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Left Tactile Agnosia Amelioration by Prism Adaptation Sustains Unilateral Spatial Neglect-Based Hypothesis

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Abstract

Objective: Left tactile agnosia has never been described as part of the neglect syndrome so far. We observed two patients with left tactile agnosia, following a right hemisphere stroke and test if impairment in their unilateral tactile object recognition could be due to Unilateral Spatial Neglect.

Method: patients were submitted to three different experimental tests assessing the tactile recognition of micro and macro structural characteristics of shapes, the number of hands' explorative movements and the effects of prism adaptation on tactile object recognition impairment.

Results: patients showed a left tactile agnosia, which was neither related to impairment in microstructural nor macrostructural recognition of shapes. Similarly to controls, patients performed more exploratory movements with the left hand, which resulted to be impaired in object recognition, suggesting that tactile apraxia was not the cause of this tactile agnosia. After 10 days treatment with prism adaptation procedure, both neglect symptoms and left tactile agnosia improved.

Conclusions: The absence of other causes of left tactile agnosia and the efficacy of prism adaptation on it, support the functional relation between tactile agnosia and tactile neglect.

Keywords: Tactile Agnosia; Neglect

Introduction

Unilateral spatial neglect (USN) is a behavioural disorder occurring after stroke, mainly of the right hemisphere [1,2], with an estimated prevalence of 40% or greater [3]. Brain damage in USN results from the disruption of a large network of white matter pathways connecting parietal and frontal lobe, including the posterior parietal cortex (PPC), frontal lobe, cingulated gyrus, striatum, thalamus, or specific brain-stem nuclei [3,4]. USN is associated with a greater risk for falls, longer rehabilitation stays, and poor functional recovery [1,2,5,6]. USN syndrome is characterized by the inability to report, orient, or respond to stimuli (objects or people) appearing on the side contralateral to the brain lesion, which cannot be attributed to either sensory or motor defects. Clinical presentation of USN is characterized by a defective awareness of the contralesional side of the body (personal neglect), of environment within reaching distance (extrapersonal neglect), or beyond reaching distances (far extrapersonal neglect) [7,8]. Anosognosia for contralesional motor and sensory deficits is a frequent component deficit of USN syndrome [8]. Delusional beliefs concerning the contralesional side of the body (somatoparaphrenia) and unawareness of hemyplegic limbs (hemiasomatognosia) may be present as well [9]. Another important clinical feature of USN is extinction to simultaneous stimuli which refers to the failure of verbally reporting the most contralesional of a pair of simultaneous stimuli, usually presented in the visual or tactile modality, while maintaining an intact or largely preserved ability of reporting the same contralesional stimulus when presented alone. Extinction can occur both within and between different sensory modalities and it is often detected in the recovery phase of neglect [5]. In the tactile modality, extinction is considered a manifestation of tactile neglect [8]. Neglect symptoms spontaneously recover in many patients a few weeks post stroke, but may persist in a substantial number of chronic cases and several rehabilitation procedures have been reported to be effective [10].The most frequently used are vestibular stimulation, reported to improve tactile and auditory deficits, optokinetic stimulation that can ameliorate proprioceptive deficits of position sense [11] and visuo-motor prismatic adaptation (PA), reported to long lastingly improve tactile deficits as well as several other USN manifestations [6,12-15]. Briefly, the technique is based on the employment of prisms which deviate the visual field 10 degrees rightward and induce an after effect leftward compensatory reorientation of the spatial representation that results in neglect amelioration by mean of a supposed bottom up effect. Particularly, resetting of the oculo-motor system would lead to an improvement in high-order visuo-spatial representation able to ameliorate neglect manifestations [16]. Although other authors consider that, as PA affects different sensory modalities, a multisensory conception of neglect would be supported [17], according to which mechanisms aimed at integrated coding of spatial information derived from different senses may also be preserved in neglect and can provide potential multisensory mechanisms for compensating modality-specific symptoms [18].

Tactile object recognition (TOR) is an everyday life exerted skill, which permits the recognition of common objects out of sight. However, neuropsychology has devoted only relatively little attention

to this issue up to now [19]. A selective impairment of TOR, without impaired tactile sensation, namely tactile agnosia (TA), usually results from lesions of parietal lobes [19]. The mechanisms involved in tactile object analysis and the anatomical correlates of those mechanisms have not been precisely defined. TOR is likely to involve a number of stages including the initial encoding of elementary sensory data, the integration of sensory information to form a coherent tactile representation of the object and the association of that tactile representation with semantic knowledge about the object. In this scheme, TA might result from a defect at a stage of sensory integration (apperceptive TA) or a subsequent stage at which tactile representations acquire meaning (associative TA) [19-23]. Finally, tactile anomia, is characterized by impaired naming of a palpated and recognized object, presumably due to tactile-verbal disconnection. Tactile apraxia is a different impairment in TOR, characterized by inadequate hand movements during objects exploration, inducing an insufficient examination of the object itself [20-23]; on the contrary, patients with TA per se seem to employ a substantially normal number of exploratory movement for shape identification. Furthermore, the ability to recognize basic features such as size, weight and texture of an object may be dissociated from the ability to name or recognize objects [19] and it has also been suggested that the computation of macro-geometrical and micro-geometrical tactile object properties can be dissociable, macro-geometrical tactile analysis depending on intact programming of exploratory hand movements, while microgeometrical (e.g roughness) properties seem to be independent [24].

Human lesion and functional imaging studies generally implicate superior parietal and occipito-temporal cortex in shape processing and the parietal operculum in texture discrimination [25-27]. A recent fMRI study suggests that TOR involves a complex network including parietal and insular somato-sensory association cortices, as well as occipito-temporal visual areas, prefrontal and medio-temporal supramodal areas, medial and lateral secondary motor cortices. Somatosensory association areas seem to be prominent in the recognition component of TOR, rather than visual cortex, as reported in previous studies. Neural activation for naturalistic TOR is distinct from that one produced by visual object recognition. Activation of the ventrolateral somatosensory pathway may be homologous to the ventrotemporal pathway strongly associated with visual object recognition [28]. Somatosensory cortical areas seem to have a functional hierarchy, with sensorimotor areas involved in more perceptual aspects of TOR and inferior parietal regions, including SII, involved in higher-level somatosensory processing [29]. Patients with lesions sparing somatosensory cortical areas without basic or complex sensibility deficits are usually observed in clinical practice. As the majority of patients described in the literature have bilateral or left hand impaired TOR, little is known about the possible relationship between TA and left neglect, in particular between TA and neglect in the tactile modality [30]. As the parietal cortex results to be relevant for high-level supramodal representations and crucially associated with the disruption of high-level supramodal spatial representations [31,32], we assumed that a lesion occurring in the right parietal cortex would possibly disrupt complex spatial representations both in the tactile and in the visual modalities, leading to both contralateral TOR deficits associated with some USN symptoms. We hypothesize that neglect, particularly in its more subtle and under-diagnosed clinical manifestations, may be responsible, in some patients, for impairing the spatial representation of objects manipulated by left hand out of sight, inducing unilateral TA.

Moreover, we tested the hypothesis of a remission of left TA by PA exposure, a rehabilitation procedure known to be effective on different sensory modalities and supposed to elicit multisensory mechanisms relevant in compensating both neglect and TOR impairment.

Methods

We describe two right hemisphere damaged patients, admitted to our Neuropsychology Unit due to difficulties in recognizing objects with manipulation and omissions in reporting spatial items located in the left hemispace. They were both right-handed and suffered a right hemisphere stroke, six months earlier, after which they developed a transient left hemiparesis associated with a left visual field deficit. Patients were examined in our Unit, submitted to baseline examination, followed by two different experimental procedures.

Patient 1

Following to carotid embolism, a 63-year-old man developed a right temporo-parietal-occipital ischemic stroke with left hemianopia and mild paresis in the left arms. MRI scan performed six months after the stroke, is shown in Figure 1, showing a right posterior parietal ischemia in the territory of the middle cerebral artery. Carotid Doppler US exam showed diffuse atherosclerotic lesions. The patient was sent to rehabilitation unit for the left hemiparesis and eventually recovered. The neurological examination, performed six months later, showed a left homonymous lower quadrantanopia, left personal neglect and extinction to the left double simultaneous stimulation (DSS). No sensory impairment in the tactile, including graphestesia, kinesthetic and vibratory modality was detected. The patient clinically demonstrated a personal left neglect in shaving and in eyeglass wearing. Neuropsychological assessment showed an impairment of long term visuo-spatial memory, constructive apraxia, left personal neglect (Table 1).

Test	Patient 1	Patient 2	Cut off
Token Test	28,5	29,5	26,5
Spatial span	3,5	3,25	3,5
Digit span	3,75	6	3,75
Short story	12	2,5	7,5
Red copy	10,75	30,5	28,87
Rey recall	6,5	10	15

COWA (f.p.l.)	33	29	17
COWA (categories)	33	33	25
Benton facial rec. tes	42	42	40
Oral apraxia	20	20	16
Ideomotor apraxia	64	67	62
Line bisection (right deviation)	2 cm	1,2 cm	0.5
Bell's test (left omission)	17	2	3
Draw copy (left omissions)	0	3	0
Left/right tactile extinctions	10-Jun	10-May	0

Table 1: Neuropsychological assessment of patients 1 and two (tests scores and cut offs). In bold are pathological scores.

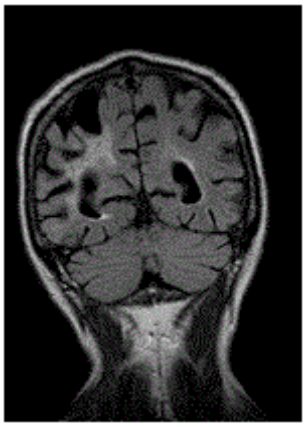

Figure 1: Patient 1-MR T2 examination: right posterior parietal hyperintense area.

Patient 2

Following to a right temporo-parietal hematoma due to a rupture of a right middle cerebral artery aneurysm, a 56-year-old woman developed a left hemiparesis and left homonymous lateral hemianopia. CT scan is shown in Figure 2 and shows two different temporal images of the hematoma. Figure 2A, the first week after the stroke with an hyperdense lesion and B, in the chronic phase (six months later) showing the hypodense quadrangular lesion involving the right insular and parietal region. After surgical clipping of the aneurysm the patient also developed focal motor seizures of the left arm, which were treated with phenobarbital 100 mg per day. She came to our observation six months later. After a period of motor rehabilitation, she completely recovered the paresis, no sensory defects in the tactile, chinestetic and vibration modality were detected. She also did not present agraphestesia. Upper left quadrantanopia, extinction to visual DSS, left personal neglect, long-term verbal memory impairment, temporal disorientation and constructive apraxia were diagnosed (Table 1). She also presented anosognosia of the tactile defects, impulsivity and irritability.

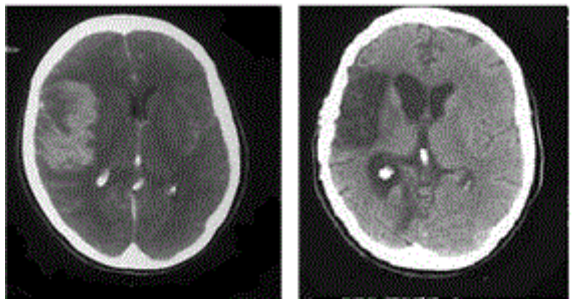

Figure 2: Patient 2 (A) Acute CT scan, showing right parietal and insular intraparenchymal haematoma (B) CT scan: chronic hypodensity

Both patients were reported by relatives not to be able to recognize common objects if they were manipulated with the left arm only. A written informaed consent was obtained by both patients, the research was conducted according to the Ethical Commettee of our Hospital.

Patients were submitted to a TOR task by mean of a naming task and two Experimental procedures.

TOR was examined in both patients in the same time frame as Neuropsychological examination; Experiment 1 and 2 were performed two days later. We used a naming task of 11 natural objects of common use, manipulated with closed eyes, first with left hand, then with right hand. Visual naming of the same object was requested, at the end of the task, in order to exclude visual naming impairment. Ten age matched controls performed the task and showed no difference between hands in object recognition (mean correct responses: 10.6 for right hand; 10.63 for left hand, p n.s., Mann Whitney).

Experimental Procedures

Two experiments were designed in order to assess if TOR impairment in patients was related either to impaired recognition of macrostructure characteristics (Experiment 1) or to impaired explorative movements (Experiment 2). After them a 10 days rehabilitation procedure using prisms was effected.

Experiment 1

In order to assess if TOR impairment was due to a deficit in recognizing macroscopic tactile characteristics of objects, 30 sandpaper shapes (15 x 10 cm each; 10 letters, 10 numbers, 10 geometrical shapes) were built and used for a tactile recognition task with left hand (LH) and right hand (RH). Sandpaper surface was chosen as a microstructure equally present in all the shapes, in order to permit the dissociation of a possible impairment in macrostructure recognition, often reported in cases of TA. The shapes were randomly presented to the patients in both hands in a passive movement recognition task (to test the recognition ability independently from the active manipulation). In this task, the examiner gently moved each shape three times according with the horizontal, vertical and diagonal axis over the palm of each hand; thereafter, patients were requested to name the shape. Both patients and 10 healthy age matched controls performed the task.

Experiment 2

In order to assess if the impairment in object recognition with LH was attributable to a deficit in the number of exploratory movements, we submitted our patients to a tactile recognition task of the previously described 30 shapes after active manipulation of them with both hands. Shapes were randomly presented. The number of active movements with both hands registered and the number of correctly recognized shapes recorded.

Prism Adaptation Treatment

In order to test the hypothesis that TOR impairment in LH could represent a subtle manifestation of neglect, particularly in the tactile modality, we looked for a possible remission of left TA by using prism adaptation rehabilitation (PA) with leftward negative after-effect, a procedure whose effectiveness is reported for several neglect symptoms [6,12-14].

Treatment started one week after the neuropsychological examination and the Experiments 1 and 2 and was conducted by a trained neuropsychologist according to the procedure described by Angeli et al. [33]. Briefly, 10 daily sessions of 20 minutes were conducted for each patient during which pointing tasks performed with the right hand were performed by the patients wearing prisms.

Stimuli to be pointed were presented at an arm distance by the examiner in both hemifields. Both patients, after the first day, showed the expected after effect with leftward compensatory deviation in pointing responses.

In order to assess the clinical effects of PA, we used the Behavioural Inattention Test battery (BIT) and Tactile Objects Recognition (TOR) before and after PA in patient 1 and 2.

A subsequent follow up was performed six months later, showing no changes in both TOR and BIT results.

Results

Tactile object recognition: Visual recognition of objects was correct for both patients, instead they showed a significantly lower performance in correctly naming the objects with left hand (mean 5.5/11 correct response) compared to right hand (mean 9.5/11 Mc Nemar Test; $p < 0.05$) (Table 2), suggesting a unilateral TOR impairment.

	RH	LH
Patient 1	10/11	5/11
Patient 2	9/11	6/11

Table 2: Number of recognized objects with Right and left hands by patients.

Experiment 1: Tactile recognition of geometrical shapes: in patients the performance was significantly poorer with left hand, compared with right hand (mean correct responses 9/30 e 13/30, mean 11/30 with LH, 17/30 e 24/30, mean 20.5/30 with RH $p<.05$ Mc Nemar test). Control subjects performed similarly in the correct responses with left and right arm (mean LH 27/30; mean RH 28/30; p n.s.). Basing on this result, patients showed an impairment in TOR of the LH, which was neither related to impaired microstructural nor macrostructural recognition of shapes.

Experiment 2: In patients, the mean number of exploratory movements was not different between hands: RH: 218, LH: 263.5 p n.s. Patients performed more exploratory movements with LH, although the number of correctly recognized shapes was shown to be significantly lower with LH: number of correct responses 18.5/30 with LH, 25.5/30 with RH; $p<.0001$, indicating that, despite their intention to explore actively the shape, the recognition with LH was poorer compared to RH.

Control subjects performed similarly in the number of correctly recognized shapes after active manipulation (LH 27.62, RH 28.75; p.n.s.). Also the number of exploratory movement was not statistically different between hands, though higher in LH also for healthy controls (mean LH 231.6; RH 215.75; p n.s.). These results support an exclusion of tactile apraxia as a possible cause of a defective left TOR impairment.

On the basis of our experiments' results, left TOR impairment in our patients seems not to be related to an impairment in either macrostructural or microstructural characteristics of tactile recognition. Furthermore an impairment in active manipulation of objects was excluded. Rather, patients seem to use more exploratory movements with the LH, although the recognition of the shape results to be poorer, compared to RH.

Prism adaptation results: Both patients showed a significant improvement after PA, in both neglect severity and TOR (Student's t test $p<.05$), as shown in Table 3.

	BIT		TOR	
	Before	**After**	**Before**	**After**
Patient 1	139/146	142/146	5	8
Patient 2	35/146	87/146	6	11

Table 3: Behavioural Inattention Test battery (BIT) and Tactile Objects Recognition (TOR) in LH before and after PA.

Discussion

The main result of our study is that, independently from elementary sensation impairment, impaired perception of micro or macro-characteristics of objects and from an impairment in actively exploring

the touched objects, the patients here described have a clearly dissociated LH TA, whose origin can be ascribed to a subtle manifestation of left neglect. This is supported by the observed remission of TA after PA treatment. Both patients present other clinical manifestation of neglect (tactile extinction, visual extinction in one patient, extra-personal neglect in several tasks), moreover the improvement of both classical neglect clinical signs as well as left TA, point to a common substrate of both disorders.

Literature data suggest that TA can result from an impairment of shape representation specific for the tactile modality, distinct from impairments of elementary sensory tactile perception. Tactile shape perception may be disrupted independently from general spatial ability, tactile spatial ability, manual shape exploration or even the exact perception of metric length in the tactile modality [19]. Consistent with literature data, our findings suggest that LH TA, in patients with parietal lesions, is neither due to somatic hypoesthesia nor to tactile apraxia. On the basis of this evidence we suppose that neglect, particularly in the tactile modality, may be responsible for impairment of spatial representation of objects manipulated by left hand out of sight.

Previous works support the efficacy of PA in all symptoms of unilateral neglect Rode et al. however, TA has never been considered and treated as a direct expression of left neglect till now. Lesions in the posterior parietal areas are present both in left unilateral neglect and in TA [19] and the here described patients have an involvement of parietal lobe in their brain lesions. The network of significant brain regions associated with improvement of left neglect performance, produced by PA, involves a complex neural network, including right posterior parietal cortex, besides right cerebellum, left thalamus, left temporo-occipital cortex and left medial temporal cortex [34]. The process may not only act on sensory-motor levels, but also on a higher cognitive level of mental space representation and/or exploration, to some extent explaining the improvement observed in TA, which recognizes the involvement of somatosensory association areas in higher-level somatosensory cognition [29]. PA rehabilitation was successful in ameliorating both LH TOR, as well as neglect in both patients. PA may improve TOR by long term reorganization of space representation in the tactile modality, as well as in other visuo-spatial lateralized tasks. PA efficacy further support the hypothesis that left neglect may directly underlie LH TA. The complexity of this condition notwithstanding, neglect can affect various senses concurrently [31,32]. Sensory stimulation and sensorimotor adaptation techniques, aimed at alleviating neglect, have also been shown to affect several sensory modalities [17]. Interestingly, all these effects have been interpreted as evidence in favour of a supramodal modulation of spatial processing, but they also support a multisensory conception of neglect [17]. In this framework, the effects of PA on TOR, may be viewed as a modulation of preserved multisensory networks relevant in both spatial and tactile unilateral representation of objects in the brain.

Although limited to a few cases, we think that these findings may be of interest and clinical research in this field should be implemented in the future. Particularly, left hand recognition of common objects in the absence of sensory or motor impairment should be assessed,. PA should be undertaken, as a good clinical responsiveness may be obtained.

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