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CHAPTER 4.5

The impairment of the body image in the unilateral neglect syndrome

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Introduction

In this chapter we shall discuss some theoretical issues on the mental representation of the body, illustrating the main competences of the left and the right hemisphere in this cognitive domain. The attention will mainly focus on the "non semantic" components of the body representation which are particularly related to the right hemisphere. Finally, the symptoms associated to the desegregation of the body schema which typically occur when unilateral neglect is present, will be described in their neuropsychological and anatomical aspects.

The body representation: Theoretical issues

The concept of body representation is a topic of debate not only in the psychological domain. It is also extensively discussed for example at the philosophical level. In neuropsychology the impairment of the body representation may cause a wide range of symptoms very different in their nature. Various hypotheses have been proposed on the mechanisms underlying the several disorders of the body representation. Some of them are spatial in their nature (De Renzi & Faglioni 1963; De Renzi & Scotti 1970; Reed & Farah 1995), other relay on more conceptual/linguistic theories (Denes 1989; Semenza 1988; Semenza & Goodglass 1985).

Recent works suggest that our knowledge of the body is mediated by at list three different types of representations (Buxbaum & Coslett 2001; Coslett, Saffran, & Schwoebel 2002; Schwoebel & Coslett 2005; Schwoebel, Coslett, & Buxbaum 2001).

The ***body image*** includes semantic, propositional, and lexical information such as the name of body parts, the association between body parts and artefacts and the function of different parts. This information are verbally coded and are accessible to consciousness.

The ***body structural description*** is a representation that includes information about the shape and contours of the body and the local relationship between different body parts. This representation derives primary from visual input.

The ***body schema***, the third type of body representation, is a dynamic representation of the body in the space modulated by the motor systems which controls action and posture. The "body schema" is an on-line representation grounded on the integration of somatosensory, vestibular and visual stimuli that provide information about the body posture and its interaction with the space.

This three types of representation may be selectively impaired and cause different deficits. While an impairment at the "body image" level may produce the Gerstmann syndrome (Gerstmann 1924), autotopagnosia (Pick 1908, 1915, 1922) is supposed to reflect a disorder at the "body structural description" level of representation. Finally, a "body schema" deficit may cause neglect related to the personal-body space (Coslett et al. 2002).

Disorders of the body representation

Body related deficits are usually classified in bilateral or unilateral. In general, while lesions of the left hemisphere cause bilateral symptoms such as autotopagnosia or finger agnosia, lesions of the right hemisphere induce unilateral symptoms (Poeck & Orgass 1971).

Bilateral deficits

A typical bilateral body–related deficit is autotopagnosia (Pick 1908, 1915, 1922) which manifests as the inability to point both verbally and under imitation commands to body parts on its own body or on other persons or on a human schematic configuration. Especially because this deficit is not exclusively concerning their own body Gerstmann proposed the term somatotopagnosia instead of autotopagnosia (Gerstmann 1942). Conversely in this syndrome the ability to name and recognize the body segments is preserved. Several times this specific impairment is associated to mental deterioration or other cognitive deficits such as aphasia, visuo-spatial and motor deficits and the inability to perceive the global stimulus in its subcomponents (reviews in Denes 1989; Poeck & Orgass 1971). The frequent co-occurrence with other symptoms makes sometimes difficult the identification of "pure" cases of autotopagnosia. Indeed there is evidence in the literature of rare pure cases not associated to other confounding cognitive dysfunctions (Denes, Cappelletti, Zilli, Dalla Porta, & Gallana 2000; Guariglia, Piccardi, Puglisi Allegra, & Traballesi 2002; Ogden 1985; Semenza 1988; Sirigu, Grafman, Bressler, & Sunderland 1991). Patients show normal performance on point to both animals' and other objects' parts, this confirms that autotopagnosia is a selective deficit of a cognitive process strictly related to the body representation (Denes et al. 2000; Ogden 1985; Semenza 1988; Sirigu et al. 1991). Lesions typically associated to this symptom are in the left frontal lobe (Denes 1989).

An even more selective bilateral deficit is finger agnosia which consists of the inability to recognize, identify, name and localize hand's fingers (Gerstmann 1924).

Autotopagnosia does not appear as a semantic deficit as subjects know for example what a nose is and are perfectly able to associate objects related to it (e.g. handkerchief). Thus autotopagnosia seems to be a selective deterioration of the structure of the body intended as the correct localization of its segments in its schema.

Unilateral deficits: From somatosensory perception to body ownership

As mentioned before, this deficits are typically associated to right brain damage. Thus, it is very common that patients with left neglect may present a wide variety of body schema impairments. This spectrum of disorders includes a range of symptoms from ***personal neglect***, which is the inability to orient toward, explore and perceive the contralesional side of the body, to the lack of awareness for motor and sensory impairment of the contralesional half of the body (***anosognosia***). Vallar has distinguished in a recent review on neglect, ***defective*** and ***productive*** symptoms related to the personal (bodily) space. The first group of deficits includes for example hemisomatoagnosia which is due to a defective awareness of the contralesional side of the body, and anosognosia which manifests with the denial of the motor, visual and somesthetic deficits. Among the productive manifestations there is somatoparaphrenia (Vallar 1988). All those symptoms will be treated separately.

Personal neglect

Patients with extrapersonal neglect typically fail to respond to stimuli presented in the contralesional space and to explore that side of the space in absence of elementary sensory or motor deficits (Heilman 1979). However spatial neglect may also concern the contralesional half of the body. In this case subjects are unable to orient toward, explore, perceive and represent this part of the body (personal neglect, Zingerle 1913). When personal neglect is clinically evident patients become unable to use common objects on the left side of the body so they may wash, shave, comb hair only on the right side (Critchley 1953). Sometimes they do not use the contralesional limb even in absence of primary motor deficits, mimicking a left hemi-paresis (motor neglect, Critchley 1953). Personal and extrapersonal neglect may be clinically double dissociated although relatively few cases with personal neglect without extrapersonal neglect have been described in literature both in the acute and in the chronic phase (Beschin & Robertson 1997; Bisiach, Perani, Vallar, & Berti 1986; Guariglia & Antonucci 1992; McIntosh, Brodie, Beschin, & Robertson 2000; Zoccolotti & Judica 1991). Interestingly enough only a single case (L.D.) of right personal without extrapersonal neglect, anosognosia and somatoparaphrenia after a left hemispheric lesion has been reported (Peru & Pinna 1997). The evidence of these behavioural dissociations suggests that the representation of different parts of the space (extrapersonal, peripersonal versus personal, bodily space) may be subserved by functionally distinct and independent systems (Bisiach et al. 1986; Guariglia & Antonucci 1992).

The body related deficit is not easily quantifiable. In the classical test proposed by Bisiach and co-worker patients are required to touch the contralesional parts of the body (hand) with their ipsilateral hand without visual control (Bisiach et al. 1986). Patients' performance is scored in a four-point scale ranging from 0 (the patient promptly reaches for the target; absence of personal neglect) to 3 (absence of movements towards the target; severe personal neglect). Intermediate score are assigned when the target is reached with hesitation and searching behaviour (score:1) or when the search is interrupted before the target is reached (score:2). Score 2 and 3 respectively correspond to medium and severe personal neglect. Personal neglect may also be assessed using a more ecological test such as the "comb and razor test" (Zoccolotti & Judica 1991) in which patients perform three simple and common daily activities such as using a comb, a razor (men) or a facial compact (women), and wearing glasses. Patients' performance is scored in a four-point scale ranging from 0 (normal performance) to 3 (severe deficit). A modified and more quantified version of this test has been proposed by Beschin and Robertson (Beschin & Robertson 1997): patients are asked to perform personal grooming behaviour for a fixed period of time (30 second) and the proportion of activity made on the left side of the body is estimate using a simple formula (% left = left activity / left + ambiguous + right activity). The formulation of this test has been further improved by McIntosh and co-workers (McIntosh et al. 2000) using a different formula in which personal neglect is considered as a lateral bias of behaviour rather then as a lateralised deficit (% bias = left – right activity / left + ambiguous + right activity). The more sensitive the used test the higher the number of reported cases of personal neglect (Beschin & Robertson 1997; McIntosh et al. 2000) compared with previous evidence (Bisiach et al. 1986; Pizzamiglio et al. 1992; Zoccolotti & Judica 1991).

While the anatomy of peri and extrapersonal neglect has been extensively studied (review in Vallar, Bottini, & Paulesu 2003) only few studies have concerned the anatomical substrate of personal neglect. The difficulty to identify isolated cases of personal neglect (i.e. dissociated by extrapersonal neglect) has made very complex any exhaustive and definitive conclusion on the anatomical correlates of this specific symptom. It seems, in fact, that personal neglect is associated with posterior brain lesions involving the infero-posterior parietal areas or subcortical regions such as the basal ganglia, the thalamus and white matter fibre tracts (Bisiach et al. 1986; Guariglia & Antonucci 1992; Peru & Pinna 1997); it is important to note however that the reported anatomical correlates concern only very few cases of pure personal neglect. The areas involved are those classically associated with peri and extrapersonal neglect as well. Studies on primates demonstrate that the monkey's brain contains well distinguished regions subserving the exploration of different sectors of space (Rizzolatti, Berti, & Gallese 2000; Rizzolatti & Camarda 1987; Rizzolatti, Matelli, & Pavesi 1983). The hypothesis of a similar organization in the human brain, although already suggested (Berti & Frassinetti 2000; Cowey, Small, & Ellis 1994; Halligan & Marshall 1991), deserves more research especially to better define possible dissociations within the peripersonal space which includes the bodily space. A more systematic investigation of the anatomical correlates of this spatial sector is needed with appropriate tests to distinguish personal neglect by other confounding clinical manifestations such as hypokinesia and with a proper lesional mapping methodology including statistical comparisons of the extension of the lesions between the different groups of patients (Rorden & Karnath 2004). A more theoretical question might be arose on which personal neglect is as its definition and identification mainly relies on clinical descriptions. However the boundary between personal neglect, sensory imperception, motor neglect (pseudo-hemiplegia) still needs to be clarify.

Anosognosia for hemiplegia

Patients affected by neurological disorders may not acknowledge their deficits despite unambiguous evidence. This behaviour has been described in sensory-motor domains (e.g. cortical blindness, cortical deafness, hemianopia and hemiplegia), cognitive domains (e.g. language and memory deficits), in schizophrenia and Alzheimer's disease (see Prigatano & Schacter 1991). One instance of this phenomenon can be found in right-brain-damaged patients, affected by left-sided hemiplegia, who may deny their paralysis and claim that their contralesional limbs can still move. This distorted or absent perception of what affects *one side of the body* has been termed anosognosia or denial of motor deficit (Babinski 1914). Anosognosia for hemiplegia has important clinical and theoretical implications. From a clinical point of view, anosognosia can have a negative impact on rehabilitation. Indeed, the denial of left side hemiplegia has been shown to be the worst prognostic factor for functional recovery of the motor disorders in right brain-damaged patients (Gialanella & Mattioli 1992). Thus, a better understanding of the mechanisms underlying the denial symptoms might help in the clinical treatment of these patients. Anosognosia also has *theoretical* implications for the study of higher cognitive functions. It has been shown that the detailed study of patients' denial can disclose implicit mental contents and can shed light on the structure underlying conscious mental processes (Berti, Làdavas, Stracciari, Giannarelli, & Ossola 1998).

The symptomatology of anosognosia for hemiplegia can vary in different patients. Sometimes when explicitly questioned about the condition of their limbs, anosognosic patients may display different degrees of denial ranging from emotional indifference (anosodiaforia), in which the motor problems may be admitted but without any concerns, to resolute and intractable unawareness of the disease. Additionally, productive symptoms, as verbal confabulations about their limbs, and delusional beliefs may coexist. In this latter case, patients may claim that the limbs are far from the body or belong to someone else, e.g. to another patient or to the doctor (*somatoparaphrenia*). The content of the confabulation can be very bizarre and patients may even claim that somebody else is lying on their beds or may show violent attitude against those 'alien' limbs (*misoplegia*).

Many interpretation of the denial behaviour, although theoretically distant, have in common the fact of not considering the disturbance as a *specific* cognitive disorder. For instance, anosognosia has been explained in terms of a generalized defensive

mechanism or psychological denial that should protect the patients from the disease. However, many different data falsify this hypothesis. First, anosognosia for hemiplegia is more frequent after right-brain damages and during the acute and post-acute phases of the illness (Bisiach & Geminiani 1991; Pia, Neppi-Mòdona, Ricci, & Berti 2004). These finding does not support a defense mechanisms view because a defensive mechanism should be active for both right and left hemiplegic disturbances and should increase with time: a goal-directed mechanisms would take time to consolidate. Furthermore, anosognosia can be temporally eliminated by caloric stimulation of one ear (Geminiani & Bottini 1992). Again, a defensive reaction should not be influenced by vestibular activation. Finally, data showing that anosognosia can be selective (see below) are themselves against a motivational account of the disorder. Other interpretation, although admitting that anosognosia is a disorder related to the cognitive system, explain it away as due to the presence of multiple concomitant neurocognitive disorders. Many authors pointed to the role of somatosensory deficit associated with intellectual impairment or memory problems (e.g. Levine, Calvanio, & Rinn 1991) or considered anosognosia as due to neglect of contralateral side of space involving also the detection of the left side of the body (Bisiach & Berti 1987). These accounts of anosognosia would receive support only by finding the coexistence of the denial behaviour with severe somatosensory deficits, intellectual impairment, memory problems or neglect. On the other hand, this account would be falsified by finding double dissociations between all these deficits. Berti et al. (Berti, Làdavas, & Della Corte 1996), Small and Ellis (Small & Ellis 1996) and Berti et al., (Berti et al. 2005) found that, although most patients with anosognosia are affected by sensory problems of the plegic arm, there are a few anosognosic patients who showed neither sensory impairment nor intellectual derangement. Moreover, most patients do not show any sign of memory loss or neglect symptoms (Berti et al. 2005; Berti et al. 1996). Conversely, many patients with sensory, memory and intellectual deficit are not anosognosic. These double dissociations show that, although the concomitant presence of various neurological/neuropsychological impairments may aggravate and shape the manifestations of anosognosia, their presence is neither necessary nor sufficient to cause the disorder (Berti 2001). Therefore, anosognosia cannot be explained away ascribing it to other deficits, but instead it seems to be a *specific* cognitive disorder affecting self-awareness of one side of the body.

There are data suggesting that anosognosia can be also *selective* (i.e. domain-specific). Indeed, it has been described limited to one limb (either the upper or the lower, Berti et al. 1996; Berti et al. 1998), affecting only some kind of movements (Marcel, Tegner, & Nimmo-Smith 2004) and evident in particular kind of task response and not in other: there are patients who are anosognosic in personal reports, when they are asked why they are in the hospital and what is the matter with their left arm or leg, but in a self-evaluation task they judge with very low score their capacity of making movements with the contralesional limbs. On the other hand, there are patients who verbally admit their motor impairment, but give very high score to their potential ability of clapping hands or using the left hand for moving an object (Berti et al. 1996; Berti et al. 1998; Marcel et al. 2004; see also House & Hodges 1988). Finally, it has also been shown (Berti et al. 1996) that when patients have different concomitant neurological disorders (e.g. neglect dyslexia, drawing neglect and motor impairment), awareness can be damaged in one domain and not in the other. For instance, we found that some patients were anosognosic for the motor problem, but admitted their spatial deficit (drawing neglect or neglect dyslexia), whereas other patients did not admit their cognitive deficit but were aware of the hemiplegia.

The fact that anosognosia is not only *specific*, but also *selective* implies that it cannot be explained as a generalized disturbance of awareness (related, for instance, to massive damage to prefrontal areas) because this would imply a lack of monitoring for all concomitant deficits, both in the somatosensory domain (unawareness for upper and lower limb paresis) and in the cognitive domain (unawareness for hemiplegia and all concomitant neglect and related disorders). On the contrary, the selectivity of anosognosia strongly suggest that awareness can have a composite structure, revealing even at the level of thought processes the modular organization of the cognitive system (Bisiach & Berti 1995). This hypothesis would predict different neural basis for different form of awareness and not a unique cerebral localization for monitoring processes (such as pre-frontal areas). A recent review of the literature on the neural basis of anosognosia for hemiplegia has shown that its occurrence is related to frontal and parietal lobes damages. The authors suggested that anosognosia can be conceived as a disorder of motor awareness implemented in a fronto-parietal circuit related to space and motor representation where the parietal component may be responsible for the spatial computation necessary to act in space (Pia et al. 2004). However, an important limit of this study was the lack of anatomical details because most of the studies cited in the review did not report lesional maps of the damaged brains. This prevented to draw conclusions about the specific Brodmann areas involved in this putative fronto-parietal circuit. A prospective study, however, investigated the lesion distribution in a group of patients showing left spatial neglect, left hemiplegia and anosognosia for the motor deficit, with those of patients showing neglect, left hemiplegia but not anosognosia. The study clearly showed that the brain areas whose lesion causes anosognosia are localized mainly in the pre-motor frontal cortex (Berti et al. 2005). These areas are known to be closely linked to motor programming both in humans and monkeys (Rizzolatti, Luppino, & Matelli 1998; Tanji 1996), motor imagery (Jeannerod 1994; Roth et al. 1996) and even interpretation of others' actions (Jackson & Decety 2004; Rizzolatti & Craighero 2004). Our data expands this knowledge by providing evidence of an involvement of pre-motor areas in the conscious monitoring of body acts and is relevant for models of motor control in particular and consciousness in general. Indeed, the involvement of pre-motor areas in self-monitoring of body movements implies that, at least for motor functions, monitoring is neither the prerogative of some kind of central executive system, hierarchically superimposed to sensory-motor and cognitive functions, nor a function that is physiologically and anatomically separated from the primary process that has to be monitored. Instead, the anatomical correlates of anosognosia show that monitoring can be implemented in the same neural network

responsible for the process that has to be controlled and not in a central-superimposed and anatomically-separated system. The study also showed that some pre-motor areas were selectively spared by the lesions (e.g. the supplementary motor areas). The authors speculated that the possibility of normally activating these areas might generate a distorted representation of one side of the body generating the false belief of being able to move. Finally, because body movements occur in egocentric space, the association observed between anosognosia, neglect and parietal lesion may reflect the damage to common components of a fronto-parietal network, specifically related to spatiomotor integration. The lesion to a single component of this network gives raise to selective, and spatially constrained, disorders of awareness, either related to the conceiving of contralesional extrapersonal space (causing neglect) or of contralesional awareness of body space (giving raise to anosognosia and related disorder).

Somatoparaphrenia

On the basis of the symptomatological classification proposed by Vallar in 1998 (Vallar 1988), somatoparaphrenia (Gerstmann 1942) should be considered as a productive disorder characterized by a selective delusion about the paralyzed limbs. It occurs mostly in case of right hemispheric lesions, on our knowledge, in fact there is only one case described in the literature with this symptom induced by a left hemispheric lesion (Neilsen 1938); although it may be noted that the exploration of this disorder is mainly based on patients' interviews, and the frequent association of aphasia to left hemispheric damage may prevent a deep investigation of this phenomenon. Patients with somatoparaphrenia (Gerstmann 1942) typically claim that their contralesional limbs belong to another person, for example parents (Bisiach, Rusconi, & Vallar 1991; Bottini, Bisiach, Sterzi, & Vallar 2002) or may report experience of detachment or reduplication of body parts (Halligan, Marshall, & Wade 1995). Somatoparaphrenia is generally associated to unawareness or denial of hemiplegia (anosognosia) and neglect (Bisiach et al. 1991). The selectivity of somatoparaphrenia is corroborated by the fact that patients show normal believes about other parts of the body (Bisiach et al. 1991). The literature on this topic is quite scanty and generally consists of the description of this disorder. As a systematic investigation of the occurrence of somatoparaphrenia still lacks, there is no evidence of a specific anatomical correlates. There is not a convergent opinion on the origin of this peculiar disorder. Halligan and coll. (Halligan et al. 1995) for example hypothesize that the brain damage "contaminate" central reasoning processes, leading thereby to believes that stretch reason to the breaking point". If this is the truth it might be expected that somatoparaphrenia is particularly associated with lesion in the frontal cortex. Bisiach (Bisiach 1995) has attempted to explain the behaviour of somatoparaphrenic patients in term of a disruption of the Topological Representation of Egocentric Space (TRES) cognitive model of spatial configuration and in particular of body schema. This model assumes that the representation of space is implemented in three layers with different competences: layer 1 which is sensory driven; layer 2 which produces autochthonous not sensory driven spatial representations, and is partially inhibited by 1 when normally functioning; and layer 3 which only has a diagrammatic function as it symbolizes layers 1 and 2 products (Bisiach 1995). In this model body-related representation may remain at an unconscious level or at least "not conscious with the mainstream of the subject's ideation" (Bisiach 1995). The damage of this cognitive system provokes a mismatch between the sensory driven and the endogenous processes of body representation which explains the alternation of rational and delusional attitude of the most somatoparaphrenic patients towards their paralyzed limbs.

The body representation is the result of different cognitive processes which are hierarchically organized from the more "elementary" levels such as perception of visual, vestibular and somatosensory stimuli to the more cognitive in their nature processes such as the ownership of their own body. These components seem to be grounded in a modular structure as there is evidence that, at least in the right hemisphere, different neural systems subserve diverse cognitive functions concerning the perception, exploration and representation of the personal, peripersonal and extrapersonal space (see Vallar 1988). The interactions among these modules have still to be clarified and the coexistence or dissociation of elementary or more cognitive defective or productive deficits of the body representation reflect, at the behavioural level, the still unclear neurophysiological organization of the bodily and extrapersonal spatial configuration.

When a lesion in the right somatosensory parietal cortex occurs tactile imperception (hemianesthesia) for the contralateral limbs which is the typical clinical consequence, is sometimes accompanied by delusional phenomenon concerning the contralesional paralyzed body. This pathological condition offers the opportunity of exploring the role of the sense of body ownership in somatosensory awareness.

One of these cases, F.B., has been recently described (Bottini et al. 2002). This is a woman who suffered from a right subcortical haemorrhagic stroke, inducing a dense left hemiplegia and hemianesthesia associated to a wide range of body awareness deficits such as anosognosia, personal neglect and somatoparaphrenia: patient in fact claimed that her left paralyzed hand, on which she was not able to perceive any tactile stimuli, belonged to her niece. The global intellective functioning of FB was within the normal range. The relationship between her hemianesthesia and her delusional symptom was explored through a simple, apparently paradoxical verbal modulation: the patient was required to report tactile stimuli delivered to "her niece's hand" instead then to her left hand. In this case F.B.'s hemianesthesia dramatically recovered. Also in this case, as observed by other authors who report in somatoparaphrenic patients the tendency to give weak explanations to the examiners' questions about the delusional behaviour, FB was very elusive and only if very pressed she said that she was taking care of her niece's hand as she forgot it on the bed after having kindly massaged the painful shoulder of the aunt.

FB case may contribute in clarifying at least some aspects of body ownership. Body parts representations relies onto somatosensory and motor cortices and these representations are resistant to the absence of the corresponding body segments as demonstrated by the well known phenomenon of the phantom limb. The author suggest that somatoparaphrenia may be considered the opposite phenomenon as in the

presence of the although paralyzed limb the subjects claim it does not belong them. Although FB apparently lost her ability to perceive touches on her left limb, she, under adequate semantic modulation, still demonstrated a residual spared somatosensory perception which probably depends on some elementary sensory functions comparable to the type mapped onto the somatosensory cortices. The problem arises when the patient has to recognize the hand on which she is still able to perceive touch as her own hand. At this point this residual sensory function does not seem to be able to elicit this sense of hand ownership. The verbal instructions of the examiners are coherent with her delusion ("Do you feel touches on your niece's hand?"), and cause a dramatic recover of the lack of perception. This behaviour is in many ways similar to allochiria which induces a symmetrical allocation of stimuli delivered on the contralesional bodily or peripersonal space to the ipsilesional side (Obersteiner 1882). In this case the phenomenon is more complex as FB needs to refer tactile stimuli on someone else's body image (a kind of "allantropia"...). Cases as FB suggest that the awareness of somatosensory perception intermingle with higher-level processes such as the sense of ownership.

Somatosensory neglect

At the more elementary level, lesions of the somatosensory parietal cortex may induce an impairment to report tactile stimuli delivered to the contralateral side of the body: remianesthesia (Paulesu, Frackowiak, & Bottini 1997; Pause, Kunesch, Binkofski, & Freund 1989; Roland 1987). Left hemianesthesia following right brain lesions, is frequently associated with some of the body awareness deficits previously described such as anosognosia, somatoparaphrenia and personal neglect. Left somatosensory deficit, at least in some cases, may reflect not only a primary sensory impairment but also a more complex and higher-order deficit of spatial representation of the body (review in Vallar 1997). This fact derives from many neurophysiological and clinical evidences. It has been for example demonstrated in a retrospective study on cohort of patients with stroke, that somatosensory deficits occurred significantly more frequently in right brain damaged subjects (Sterzi et al. 1993). Furthermore different physiological manipulations (caloric vestibular, electrical transcutaneous, and optokinetic stimulation) may produce a transient recovery of contralateral sensory deficits (Vallar, Bottini, Rusconi, & Sterzi 1993; Vallar, Rusconi, & Bernardini 1996; Vallar, Sterzi, Bottini, Cappa, & Rusconi 1990). Amongst these procedures the most extensively investigated, for its remarkably effect, is caloric vestibular stimulation (CVS) with iced water in the external ear canal contralateral to the brain lesion. Classical studies showed that in right brain damaged patients left CVS temporary ameliorates contralesional hemianesthesia (Bottini et al. 1995; Vallar et al. 1993; Vallar et al. 1990). Interestingly enough, the same stimulation in the right ear does not produce the same effect on left brain damaged patients with the notable exception of few cases of patients who manifest right visuo-spatial neglect with a probable non canonical hemispheric lateralization of spatial functions (Vallar et al. 1993). The observation of this behavioural asymmetry had suggested in case of right hemispheric lesion that hemianesthesia also contains an attentional-spatial component which is strictly related to the neglect syndrome, and that this particular component which is not typical of right hemianesthesia associated to left hemispheric lesion, may be modulated and transiently reduced by CVS. Another hypothesis might be taken into account, that the right hemisphere plays a special role in monitoring visuo-spatial functions in general and more specifically in the representation of the body in all its components including attention for tactile stimuli. To support this hypothesis, Meador et al. (Meador et al. 1988) have demonstrated that during pharmacological inhibition with sodium amobarbital, in case of right hemisphere inactivity the degree of tactile perception impairment (in particular extinction) was significantly more severe than in case of left inhibition. This result has been confirmed by a different experimental paradigm performed by Pardo and coworkers (Pardo, Fox, & Raichle 1991) who, exploring with PET the human anatomical areas involved in the vigilance aspects of normal attention to sensory stimuli, found a significant higher activation primarily in the prefrontal and superior parietal of the right hemisphere. Furthermore patients with left neglect may present physiological preserved response (skin conductance response or evoked potentials) to undetected contralesional tactile stimuli (Vallar, Bottini, Sterzi, Passerini, & Rusconi 1991; Vallar, Sandroni, Rusconi, & Barbieri 1991). This observation suggest that patient's hemianesthesia can't be entirely due to a primary sensory deficit. Another interesting evidence in this direction is that in right brain damaged patients, the degree of contralesional somatosensory deficits may be modulated by changing the position of body parts respect to the body midline (Smania & Aglioti 1995). In particular, when the left hand is placed in the right unaffected side of the space the detection of left tactile stimuli significantly improves, suggesting that tactile stimuli are also spatially coded.

It has also been shown that even in the presence of extensive right hemispheric lesion in the primary somatosensory cortex, (SI) cold left CVS may induces a clear cut transient remission of left hemianesthesia activating a neural network (insula, putamen, inferior frontal gyrus in the premotor cortex) in the right hemisphere, suggesting that compensatory mechanisms to recover tactile imperception and somatosensory awareness still rely in the right hemisphere although it has been largely damaged (Bottini et al. 1995). This functional asymmetry is also corroborated by evidence in normal subjects who show bilateral cortical activation more extensive in the right hemisphere during left cold CVS compared with the opposite stimulation (Bottini et al. 2001; Bottini et al. 1994). Recently, in order to better clarify these evidences, it has been systematically explored the effect of CVS differently lateralized in hemianesthetic, right and left brain damaged patients, whose anatomical lesions were mapped to exclude any volumetric difference in the somatosensory areas which might provoke a bias in the behavioural effect of CVS (Bottini et al. 2005). The expected remission of left hemianesthesia during left cold CVS was confirmed compared with no reaction in case of right CVS on right brain damaged subjects. Of more interest, and completely new, was the result on the left damaged population, as, a part the expected inefficacy of right cold CVS, the same kind of manipulation ***to the left*** induced a temporary remission of right hemianesthesia. These behavioural data on patients were combined with

neuroimaging observation in normal subjects and in one left brain damaged patient. Normal subjects underwent tactile stimulation to the right and the left hand during fMRI to identify the somatosensory areas involved by this stimulation of the same kind of which performed on patients before and after CVS. This part of experiment demonstrated a pattern of activation of the contralateral nucleus of the thalamus, of somatosensory SI area and the supplementary motor area, and Secondary Somatosensory Cortex, SII bilaterally. A significantly greater activation for ispilateral hand stimuli has been found in the right hemisphere compared with the left in the parietal operculum (SII area). The right hemianaesthetic patient with an extensive left hemisphere lesion involving left SII, the postcentral gyrus, the posterior limb of the internal capsule and a large portion of the thalamus with a complete destruction of the ventroposterior somatosensory nuclei was also submitted to fMRI during tactile stimulation before and after having left cold CVS. In this patient the areas modulated by CVS involved the right temporoparietal junction including area SII and the supramarginal gyrus which were significantly activated after left CVS and touches delivered to the right hand with a significant *touch-by CVS-by hand* interaction effect.

This right hemisphere effect contributes to better clarify the right hemispheric specialization in body representation (in particular in hand representation). The fact that in the patient with an extensive left hemispheric lesion starting from the thalamus to the cortical somatosensory cortices, touches on the ipsilesional hand, induce activation in SII in the right hemisphere, demonstrates that neurons in this region, have ipsilateral receptive fields. This is a convincing evidence that the right hemisphere contains a more complete representation of the whole body space compared whit the left hemisphere.

Concluding remarks

Behavioural and neuroimaging data support the hypothesis that body schema results from the integration of different signals. In the body representation processes it is possible to identify progressive hierarchical levels of analysis, from elementary operation, such as somatosensory processing to more complex computation, such as the construction of the feeling of ownership of body parts. However, the boundary between these processes is sometimes fuzzy. For example, the possibility of modulating apparently elementary neurological deficits, such as tactile imperception, through physiological stimulations that allow transient conscious perception of the tactile stimuli, suggests that even low level deficits contain a not obvious higher component. Some aspects of the mutual interaction between the different levels that construct the feeling of having a body acting in the space is starting to become clearer due to the progress in identifying the neurophysiological correlates of the different levels of body representation in normal subjects and in patients with different body representation disorders. The neural correlates of some aspects of pathological behaviours (such as somatoparaphrenia) are still unclear although clinical and neurophysiological findings seem to suggest that at least in part hemianestesia, deficits of the sense of ownership and related disorders, may share some common mechanisms of spatial representation.

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