British Journal of Psychiatry (1988), **153** (suppl. 2), 7–11

The Neurobiology of the Body Schema

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The history of a concept of a body schema is linked with the fact that the discovery that there is such a thing as a permanent, albeit vague perception that one has of one's own body, has been mainly in observations of its pathological manifestation in the clinic.

There is much misunderstanding in this context about the body schema concept. The terms 'body schema', 'body experience' and many other terms such as 'body image', 'body concept', 'somatopsyche', 'image of the body ego' and 'body awareness' are often used indiscriminately with ever-changing meanings. The practical point is whether one wishes to use the concept either in the neurological or psychological sense. When used in a neurological sense it is hoped that the findings will help to contribute to a topographical neurological diagnosis.

In general terms, the body schema is used for the awareness of spatial characteristics of one's own body, this awareness being formed by current and previous sensory information. The term body experience, however, is more comprehensive including psychological and situational factors as well as emotions and intentional factors – the body schema being a fact of perception, whereas the body experience is a fact of psychological experience.

It is generally accepted that the parietal lobes of the brain play a major part in the construction of the normal body schema as indicated by the frequency of disorders of body schema that is seen in patients with parietal lobe disorders. However, since the concept of the body schema is dependent upon both current and previous sensory information, the roles of the somatoaesthetic afferent system and the thalamus cannot be neglected.

Body schema is not a single concept and it would appear that it is impossible to lose all awareness of the body schema in neurological disease, but individual subcomponents of the body schema may, of course, be involved at different anatomical sites.

The unilateral misperception of one's own body is described as hemiasomatognosia following the detailed descriptions of L'Hermitte in 1939. Clinically this can be divided into two forms, the conscious form and the non-conscious form. In the conscious form of the disorder the phenomenon is usually transient or paroxysmal and can occur in association with other cerebral paroxysmal events, for example in patients with migraine or epilepsy. Both right- and left-sided cerebral lesions are known to cause this particular form of hemiasomatognosia. However, the conscious form is somewhat uncommon compared with the non-conscious form.

The non-conscious form of hemiasomatognosia is characterised clinically not by a conscious experience but by a specific and often spectacular disorder of behaviour – the patient behaving as though one half of his body was non-existent. The original early descriptions of Gerstmann in the 1930s remain classical clinical descriptions of the condition.

The patient classically shows no concern for one half of his body and if he is, for example, asked to elevate the affected limb, his facial expression may suggest that he has accomplished the task successfully, although no limb movement is seen to occur. A very prominent feature is the fact that even under normal visual control, the movement still remains absent. Therefore, if the patient directs his vision towards the affected limb and can apparently perceive that the limb is not moving, he still provides a facial expression suggesting that he has satisfactorily complied with the command to elevate the limb. The majority of patients with non-conscious hemiasomatognosia have associated features suggesting involvement of the parietal lobe, with anosognosia for hemiplegia and often unilateral spatial agnosia or unilateral visual neglect.

We now consider therefore the subdivisions of the non-conscious form of hemiasomatognosia, i.e. anosognosia for hemiplegia, 'neglect syndromes' and Gerstmann's syndrome. Anosognosia for hemiplegia is by definition always associated with hemiplegia in which the patient is not aware of the paralysed limbs and in which, on asking the patient about his paraparesis or paraplegia, he specifically denies its existence.

The earliest descriptions of this syndrome were by Anton (1893, 1898, 1899) and Babinski (1918, 1923) in the early part of this century and they stressed the fact that if the examiner is not familiar with the phenomenon of anosognosia, he may simply attribute the patient's ignorance about the fact of his own hemiplegia to some non-specific mental impairment. The condition occurs with right cerebral hemisphere lesions leading on to a left-sided stroke and it usually

occurs in the presence of a hemisensory defect and an appropriate hemianopic defect. There may be associated features of a neglect syndrome but these are not necessarily present. In the majority of patients there has been an infarct in the territory of the right middle cerebral artery, although in some patients who have had this condition, a small capsular infarct without evidence of cortical involvement has been described. The condition is usually seen in the acute phase following a stroke, and it is usually a transient phenomenon which occurs during the first 1–2 weeks after the onset of the stroke. Fredericks (1985) in his extensive studies on intellectual functioning following cerebral events has drawn attention to the fact that in his experience anosognosia for hemiplegia is only seen in acute vascular events and is not seen in the developing symptoms of a tumour.

Normally there would seem to be no particular reason to ask a hemiplegic patient whether he was actually paralysed, but when the question is posed, even indirectly, unless the examiner is aware of the existence of anosognosia for hemiplegia, he may well come to the conclusion that the patient's denial, not only of hemiplegia but of any illness at all, may have a psychological basis.

There have been various theories about the pathogenesis of anosognosia. For example, Goldstein (1928), Weinstein & Khan (1955) and Ullmann (1962) have held that anosognosia is an expression of a general mental disorder resembling diffuse cortical syndrome and they have suggested that the patient's premorbid personality has a lot to do with his subsequent reaction to illness. Babinski himself has stressed that anosognosia has such a specific localising value that it has to be a focal cerebral syndrome. Gerstmann (1930) believed that like most agnosias, anosognosia for hemiplegia was a result of a specific disconnection from the speech area and Weinstein & Khan (1955) went on to suggest that the fact that anosognosia only occurred in left-side hemiplegia, was on the basis of the patients with right-sided hemiplegia being confused, silent or using jargon, this latter being regarded as the link between aphasia and anosognosia. However, Fredericks (1985) has drawn attention to the fact that denial of hemiplegia is brought on only when a pertinent question is asked by the examiner, but when left to his own devices the patient does not deny spontaneous existence of the hemiplegia. He has also shown that the patient has a form of kinaesthetic hallucination in that when asked to lift up the hemiplegic limb, he gives the impression of having done so from facial expression, even though the limb has not moved, and in particular when the vision is directed to the unmoved limb, he still seems to give the visual impression of having moved the limb. This would suggest that there has been a kinaesthetic hallucination of limb movement and from this Fredericks has argued that this syndrome is caused by unstructured and unfiltered somatoaesthetic information coming from the hemiplegic limb, which because of the infarct in the right hemisphere, has not been adequately processed. The fact that the syndrome tends to disappear with time is due, he suggests, to the slow resolution of the swelling phase of most strokes.

The next most common form of non-conscious hemisomatognosia are the various 'neglect syndromes'. These differ from anosognosia for hemiplegia in the fact that they are present on a long-term stable basis and in that, although seen in the acute phase of the stroke, they do persist as a permanent effect. In addition, although commonly seen in right parietal abnormalities, they are also frequently seen in left hemisphere lesions. The commonest manifestations of the neglect syndromes are hemi-inattention and hemispatial neglect. In all these patients it is assumed that the peripheral motor and sensory mechanisms are intact.

It is possible to distinguish hemi-inattention from hemiloss for example, of vision or touch since patients with inattention, when their attention is directed to the affected limb, can often perceive the abnormality. Thus, when doing visual field tests, for example in the presence of a left hemivisual inattention, the left eye, when tested on its own, will give normal results, but when tested with simultaneous stimuli, there would be an apparent extinction. The same is true for tactile stimulation.

The mechanisms underlying hemi-inattention seem to become increasingly complex with each passing year. Figure 1 is derived from the work of Heilman *et al* (1985) on the interaction between sensory attention and the tonic arousal pathways. Sensory input coming from somatosensory, auditory and visual pathways divides into two major distributions. These are:

(a) the pathway to the thalamic relay nucleii and

(b) the pathway to the mesencephalic reticular formation.

Following pathway (a) through the thalamic relay nucleii, sensory information is passed to the sensory cortex and hence to the association cortex. The association cortex, which has itself a feed-back loop to the thalamic nucleii, sends its main output to the pre-frontal association cortex. This area has major interconnections at the posterior cingulate area (limbic) and with the inferior parietal lobule.

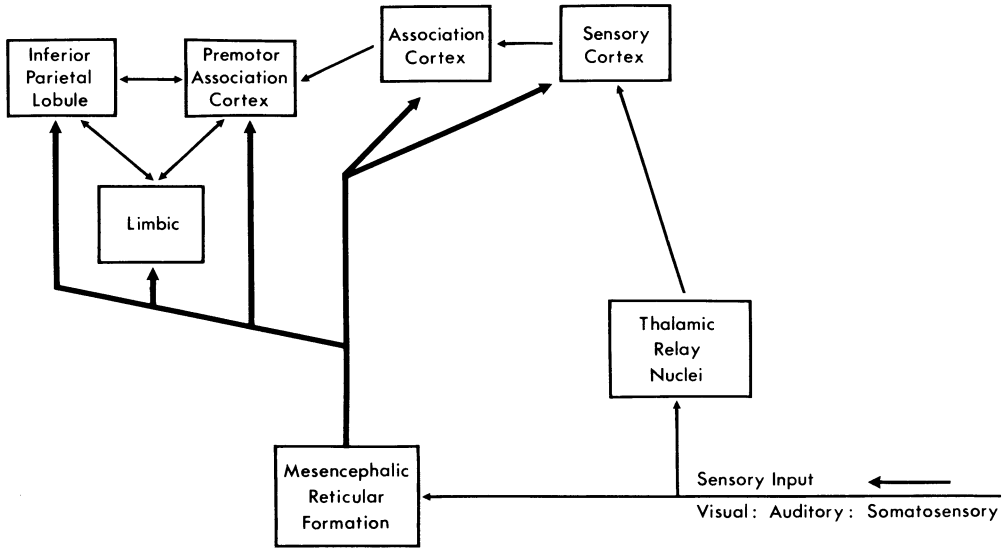

FIG. 1 Sensory attention and tonic arousal pathways. (Based on Fig. 1, p. 156 in The neglect syndrome, Ch. 12, by K. M. Heilman, E. Valenstein & R. T. Watson in *Handbook of Clinical Neurology*, Vol. 1(45): *Clinical Neuropsychology* (ed. J. A. M. Fredericks), Elsevier Science Publishers (1985) with the kind permission of K. M. Heilman).

The second main pathway, however, of the sensory input (b), goes straight into the mesencephalic reticular formation. Tonic arousal, originating from the mesencephalic reticular formation, stimulates virtually all points at the sensory system to allow the sensory information which is being provided via the thalamic relay nucleii to be acted upon.

Although these pathways underlying hemi-inattention seem complex, they do help to unravel some of the clinical situations encountered. Although it is usually claimed that hemi-inattention occurs in lesions of the parietal sensory cortex, either due to tumour or infarct, it has been described in lesions of the thalamus and lesions of the limbic system, in lesions affecting the prefrontal area and more particularly, it can be seen in the phase of reawakening from coma without any specific cortical abnormality.

Figure 1 shows how the interrelationship of these various pathways can explain hemi-inattention rising from subcortical and diffuse cortical abnormalities whether tumour or infarct.

Hemispatial neglect describes those patients who, when asked to perform a variety of behavioural tasks in space, neglect the hemispace contralateral to their lesion. For example, when asked to draw a picture of a flower, they draw only half of the flower and, if asked to draw a clock face, they classically do one-half of the clock face. This defect has been variously described as hemispatial neglect, visuospatial agnosia, visual spatial neglect and unilateral spatial neglect. Frequently patients in this situation also have a dressing apraxia and may have what Benson & Geschwind (1969) describe as paralexia, that is, if they are asked to read the word 'cowboy', they will read only 'boy'. A similar abnormality can be seen when they are using a typewriter in that they fail to type letters on the side of the keyboard contralateral to their lesions. This is described as paragraphia by Valenstein & Heilman (1979).

Assessment of hemispatial neglect has now become routine, e.g. asking the patient to draw a clock face or a stick man. However, the mechanisms underlying hemispatial neglect remain poorly understood. The fact that visual neglect can be seen in patients who are not hemianopic, implies that simple visual sensory defect is not sufficient to account for their symptoms.

The abnormal performance of patients in contralateral space suggests that brain mechanisms relating to the opposite hemispace has been disturbed. Heilman *et al* (1985) have suggested that each hemisphere is responsible, not only for receiving stimuli from contralateral space and for controlling the contralateral limbs, but also for attending and intending in contralateral hemispace independent of which hand is used. On this basis, neglect is seen as a failure of attention of the affected hemisphere leading to neglect in the contralateral hemispace.

Although neglect can occur from both hemispheres, it is more often seen in lesions in the right

https://doi.org/10.1192/S0007125000298905 Published online by Cambridge University Press

hemisphere. The previous concept that lesions of the left hemisphere, which in addition to causing neglect also cause abnormalities of comprehension and therefore mask the symptoms of neglect, has for a long time been considered to be too simplistic. However, given that there is substantial asymmetry in the frequency of neglect seen in right-sided lesions *vs* left-sided lesions, is it possible therefore that the right side is in some way more specialised at dealing with attention and intention than is the left hemisphere? There is increasing experimental evidence to suggest that this may be the case.

Exactly the same pathways can be constructed with respect to the neglect syndromes as with attention in Fig. 1. In addition, these syndromes are seen not only with lesions in the parietal cortex but also in prefrontal association cortex and thalamic lesions either infarct or tumour. However, by far the most common area is the temporoparietal cortex, particularly in its posterior part, which is the watershed between the middle and posterior cerebral circulations. Lesions in this area, which are well behind the motor strip, can frequently occur without the presence of hemiplegia and this can lead to difficulty in their recognition.

Gerstmann's syndrome is the third of the common disorders of body schema. The syndrome consists of finger agnosia, acalculia, agraphia and right/left disorientation. Central to the syndrome is always finger agnosia. This was first described by Pick (1982) as autopagnosia but expanded greatly by Gerstmann's contributions in the 1920s. Examination for autopagnosia is simply part of the bedside examination. The patient is asked the name of, or to move, parts of his body touched by the investigator, or the investigator names parts of the patient's body and the patient has to then indicate or move them. The patient is asked to draw a human face or a human figure. This may reveal autopagnosic disturbances in that he omits parts of the body or draws them too small or distorted. Associated with this is the ability to distinguish left or right on one's own body, which can be tested by requests to the patient to name or move parts of the body which presuppose knowledge of right or left.

In addition to the inability to recognise and identify parts of the body, Geschwind (1965) pointed out that the syndrome can also include disturbed use of these parts, particularly in writing.

Attempts have been made to unify the components of Gerstmann's syndrome on the basis of disorders of body schema. However, the component parts are often seen independently and can be seen in association with other disorders of left hemisphere function and this has led Geschwind among others, to suggest that the basis of Gerstmann's syndrome is predominantly a language disconnection within the dominant hemisphere. Irrespective of this, the presence of Gerstmann's syndrome does have distinct localising value in that it is uniquely seen in lesions of the dominant hemisphere in the temporoparietal area.

Finally, we can consider microsomatognosia and macrosomatognosia. These disorders can be defined as disorders of the body schema in which the patient perceives part of his body as abnormally large or abnormally small. The patient will usually spontaneously complain of this disorder unless he is upset by the illusional character of the phenomenon. They are usually seen in association with other paroxysmal neurological disorders, particularly epilepsy or migraine.

Disorders of the body schema have a long association with neurology. Particularly since the advent of the CT scan, it has become possible to attempt to localise the areas of the brain responsible for these disorders. In terms of the body schema autopagonosia for hemiplegia seems to be well localised to the right hemisphere. Gerstmann's syndrome and its variants are well localised to the left hemisphere. The 'neglect syndromes' arise from either hemisphere but more commonly on the right than from the left. However, these rather simplistic concepts of loss of tissue leading to clinical symptoms have been shown to be much too basic and attention has now been directed to arousal, attention and intention within the hemispheres and mechanisms by which these interrelate. Undoubtedly, further investigation along these lines is going to increase greatly our understanding of higher cerebral function. However, these patients have to be identified and categorised correctly before they can be studied. Fortunately that can be done by the clinician without resource to split-field techniques and sophisticated equipment.

As things become more complicated, one wonders whether the delightful head maps of years ago might not some day have more than museum attraction.

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https://doi.org/10.1192/S0007125000298905 Published online by Cambridge University Press