Study of anosognosias

JOHN CUTTING

From the Institute of Psychiatry and King's College Hospital, London

SUMMARY Anosognosia (denial of weakness) and "anosognosic phenomena" (other abnormal attitudes to a weak limb) were studied in 100 acute hemiplegics. Both conditions were associated with lesions of either hemisphere. Apathy, visual field defect, and impaired picture identification were particularly prominent in anosognosia. A failure to integrate information from one side of the body was regarded as fundamental to the condition; explanations in terms of "unilateral neglect" and "agnosia" are discussed.

Babinski introduced the word "anosognosia" in 1914 as a description of patients who ignored their hemiplegia. In current usage the term embraces a variety of abnormal attitudes. Chief among these is an explicit verbal denial that the limb is weak. Some patients, although admitting to their disability on direct questioning, minimise its extent, often in a jocular fashion (anosodiaphoria, Babinski, 1914). Yet others experience the limb as strange or not belonging to them, or even attribute ownership to another person (somatoparaphrenia, Gerstmann, 1942). Some express hatred of the limb (misoplegia, Critchley, 1962), some give it a nickname (personification, Juba, 1949), and some overestimate the strength of an unaffected limb (anosognosic overestimation, Anastasopoulos, 1961). A false belief that the limb is moving (kinaesthetic hallucinations, Waldenström, 1939) or that a separate limb has appeared in another part of the body (phantom supernumerary limb, Ehrenwald, 1930) may occur. Although these phenomena are generally regarded as related in some way, there is no agreement on an overall classification. Frederiks' (1969) division into "explicit denial" and "anosognosic behavioural disturbances" (incorporating all the other phenomena) is attractive, and will be used in this report. For the sake of convenience, these will be referred to respectively as "anosognosia" and "anosognosic phenomena."

The association of anosognosia with a left hemiplegia has been found so often that some authors have attributed to right hemisphere damage a preeminent role in its development (Bogen, 1969; Galin, 1974). It is true that anosognosia for a right hemiplegia has only rarely been recorded. Excluding left handed patients and those with bilateral lesions, only four case reports remain (Von Hagen and Ives, 1937; Nathanson et al., 1952; Weinstein and Kahn, 1955; Welman, 1969). However, the presence of aphasia in patients with left hemisphere lesions complicates the issue. Gross and Kaltenbäck (1955) found that 91% of right hemiplegic patients with a field defect and sensory loss, features which had predicted anosognosia in their counterparts with a left hemiplegia, were totally aphasic in the first week after onset. They concluded, therefore, that right hemiplegics at risk for developing anosognosia were the very patients in whom aphasia precluded its determination. In the light of these difficulties, the association between anosognosia and right hemisphere damage may be more apparent than real.

The independence of anosognosia from a general cognitive impairment is suggested by individual reports of patients with intact orientation (Waldenström, 1939; Gilliatt and Pratt, 1952). However, three of the only four series of patients (Nathanson et al., 1952; Weinstein and Kahn, 1955; Ullman, 1962) recorded disorientation in every instance of anosognosia, and the authors of the fourth series (Gross and Kaltenbäck, 1955) maintained that although 18% were correctly oriented, they suffered from a milder degree of "confusion," which they termed "lack of critical awareness of surroundings."

Resolution of these central issues might lead in part to a clearer appreciation of the nature of
anosognosia. At present, theories range from psychodynamic interpretations of motivation (Weinstein and Kahn, 1955) to detailed neurophysiological explanations of the role of central sensory pathways (Frederiks, 1969; Waldenström, 1939). Other factors, in particular "neglect," have been less investigated, and the aim of the present study was to examine the basis for the major theoretical positions by correlating deficits in higher mental functions with the emergence of anosognosia and its phenomena in a consecutive series of patients with an acute hemiplegia.

Patients and methods

One hundred patients with an acute hemiplegia were seen over a period of two years in a general hospital. Weekly visits were paid to all medical wards, and the notes of all patients with a provisional diagnosis of a cerebrovascular accident were inspected. Cases were rejected if the hemiplegia was more than eight days old, if there was no recorded limb weakness, if any weakness had totally recovered by the time the patient was seen by the author, or if the patient was unconscious on the day of the visit. About 50 patients were excluded in this way, and the remaining 100 were considered to be representative of acute hemiplegics without prolonged unconsciousness or rapid resolution admitted to a general hospital.

No detailed analysis of the localisation within a hemisphere or of the pathology of the lesion was planned, but some comment on this is appropriate. The exclusion of any patient whose weakness had been present for longer than a week ensured that a cerebrovascular accident was likely. Investigations were incomplete at the time of the first visit, but notes were inspected subsequently, and in four cases there was evidence that a tumour and not a vascular lesion had been responsible. No patient with bilateral weakness was included, but three had pyramidal tract signs on the side opposite to that currently affected. This suggested that bilateral brain damage was present in 3%.

GENERAL EXAMINATION

General features (age, sex, handedness, occurrence of a previous paresis, and duration and side of present hemiplegia) were recorded. A neurological examination with emphasis on degree of weakness, sensory loss and visual field defects was then carried out. Weakness was rated on a four point scale (1=slight, 2=moderate, 3=severe, 4=total). Sensory loss was recorded as present or absent; the extent and nature was noted and, although a crude scale was drawn up, this was not included in the analyses because it only mirrored the absolute rates of loss.

HIGHER MENTAL FUNCTION

Six aspects of higher mental function were systematically studied. Orientation, attention, and memory were assessed by asking patients to orientate themselves in time and place, to repeat digits, and to remember a name and address and details of their admission. Mood was observed and recorded as pathological (euphoric, apathetic, or depressed) if it appeared inappropriate to the circumstances. Personal neglect was regarded, at Critchley's (1953) suggestion, as a disinclination of the contralateral hand to cross the midline. In the presence of weakness it is difficult to separate personal neglect from an abnormal attitude to a paralysis, and in left hemisphere lesions, from disturbances of body naming. Some indication of this, however, for patients with a left hemiplegia, was achieved by asking them to touch parts of the body on the left side (little finger, thumb, elbow, ear) with the right hand. Visuospatial neglect was estimated by inspecting their drawings of a face, a clock, and a map of England with six cities. The simple neglect test of Albert (1973), which requires patients to score out lines on a piece of paper, was also used; those with neglect omit lines on one side. Visual perception was tested by presenting 20 pictures of moderately uncommon objects—for example, speedometer, accordion—and asking for a correct identification, name, or functional description. The task was entirely visual and performance was easily contrasted with the patients' abilities in the other language tests given. The test was given to 30 controls (10 Korsakoff subjects, 10 alcoholics, and 10 patients with peripheral neuropathy), and as the lowest identification score among these was 14 out of 20, any score below this was suggestive of impaired visual perception. The mean scores for any groups were also calculated.

LANGUAGE

Language was assessed for expressive deficits, receptive deficits (by asking questions of increasing complexity requiring nonverbal answers, for example, point to where the illumination of the room comes from), and for impairment of the abstract attitude (by asking for interpretations of five proverbs, each scored out of 2). The general, neurological, and higher mental function assessment will be referred to as the profile of a patient. An anosognosia questionnaire (Table 1) was designed to cover the range of anosognosic phenomena, and to probe anosognosia itself.
Table 1  Anosognosia questionnaire

<table>
<thead>
<tr>
<th>General questions</th>
<th>Procedure if denial elicited on general questions</th>
<th>Anosognosia phenomena</th>
<th>Questions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Why are you here?</td>
<td>(Arm picked up)</td>
<td>Anosodiaphoria</td>
<td>Is it a nuisance? How much trouble does it cause you? What caused it?</td>
</tr>
<tr>
<td>What is the matter with you?</td>
<td>Can you lift it? The patient clearly has some problem with this?</td>
<td>Denial elicits</td>
<td>Do you ever feel that it doesn’t belong? Do you feel that it belongs to someone else?</td>
</tr>
<tr>
<td>Is there anything wrong with your arm or leg?</td>
<td>Can’t you see that the two arms are not at the same level?</td>
<td>Strange feelings</td>
<td>Do you feel the arm is strange or odd? Do you dislike the arm? Do you hate it?</td>
</tr>
<tr>
<td>Is it weak, paralysed or numb?</td>
<td>(Asked to lift arms)</td>
<td>Misopelia</td>
<td>Do you have strong feelings about it? Do you ever call it names?</td>
</tr>
<tr>
<td>How does it feel?</td>
<td></td>
<td>Personification</td>
<td>Do you ever feel it moves without your moving it yourself?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hallucinations</td>
<td>How’s the other arm?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Overestimation</td>
<td>Do you ever feel a strange arm lying beside you separate from the real arm?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Supernumerary limb</td>
<td></td>
</tr>
</tbody>
</table>

Results

CHARACTERISTICS OF ENTIRE GROUP

Fifty-two patients had a right hemiplegia. Of these, 30 were so aphasic that it was impossible to assess anosognosia. The remaining 22 had sufficient preservation of language for an assessment to be carried out. They were free of receptive aphasia but five had expressive difficulties. They will be referred to as the “testable” group. Forty-eight patients had a left hemiplegia; none of these had aphasia. The three groups—aphasic right hemiplegics, testable right hemiplegics, and left hemiplegics—are compared in Table 2. The main findings were that the testable group had less sensory disturbance, a lower incidence of field defects, and fewer abnormalities of higher mental function. Language deficits were more common in right hemiplegics. Mood change was depressive in right, but predominantly apathetic or euphoric in left hemiplegics.

ANOSOGNOSIA

Twenty-eight patients denied a left sided weakness

Table 2  Profiles of right aphasic, right testable, and left hemiplegic patients

<table>
<thead>
<tr>
<th></th>
<th>Right aphasic</th>
<th>Right testable</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>30</td>
<td>22</td>
<td>48</td>
</tr>
<tr>
<td>Age</td>
<td>68</td>
<td>64</td>
<td>68</td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>50%</td>
<td>55%</td>
<td>65%</td>
</tr>
<tr>
<td>Left handers</td>
<td>—</td>
<td>14%</td>
<td>10%</td>
</tr>
<tr>
<td>Previous hemiplegia</td>
<td>8%</td>
<td>14%</td>
<td>8%</td>
</tr>
<tr>
<td>Duration (days)</td>
<td>3.6</td>
<td>4.2</td>
<td>3.8</td>
</tr>
<tr>
<td>Degree of weakness</td>
<td>3.8</td>
<td>2.3</td>
<td>2.5</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>—</td>
<td>36%</td>
<td>88%</td>
</tr>
<tr>
<td>Field defect</td>
<td>—</td>
<td>9%</td>
<td>77%</td>
</tr>
<tr>
<td>Disorientation</td>
<td>—</td>
<td>32%</td>
<td>56%</td>
</tr>
<tr>
<td>Mood—total abnormal</td>
<td>—</td>
<td>23%</td>
<td>56%</td>
</tr>
<tr>
<td>—apathetic</td>
<td>—</td>
<td>0%</td>
<td>36%</td>
</tr>
<tr>
<td>—depressed</td>
<td>—</td>
<td>23%</td>
<td>8%</td>
</tr>
<tr>
<td>—euphoric</td>
<td>—</td>
<td>0%</td>
<td>12%</td>
</tr>
<tr>
<td>Personal neglect</td>
<td>—</td>
<td>0%</td>
<td>21%</td>
</tr>
<tr>
<td>Visuospatial neglect</td>
<td>—</td>
<td>0%</td>
<td>50%</td>
</tr>
<tr>
<td>Visuoperceptual defect</td>
<td>—</td>
<td>36%</td>
<td>53%</td>
</tr>
<tr>
<td>mean score</td>
<td>—</td>
<td>14.8</td>
<td>13.0</td>
</tr>
<tr>
<td>Language—expressive dysfunction</td>
<td>—</td>
<td>23%</td>
<td>0%</td>
</tr>
<tr>
<td>—abstract score</td>
<td>—</td>
<td>2.0</td>
<td>3.9</td>
</tr>
<tr>
<td>Anosognosia</td>
<td>—</td>
<td>14%</td>
<td>58%</td>
</tr>
<tr>
<td>Anosognosia phenomena</td>
<td>—</td>
<td>41%</td>
<td>29%</td>
</tr>
<tr>
<td>Total (no associated anosognosia)</td>
<td>—</td>
<td>9%</td>
<td>4%</td>
</tr>
<tr>
<td>Anosodiaphoria</td>
<td>—</td>
<td>14%</td>
<td>23%</td>
</tr>
<tr>
<td>Nonbelonging</td>
<td>—</td>
<td>0%</td>
<td>6%</td>
</tr>
<tr>
<td>Strange sensations</td>
<td>—</td>
<td>23%</td>
<td>4%</td>
</tr>
<tr>
<td>Misopelia</td>
<td>—</td>
<td>0%</td>
<td>2%</td>
</tr>
<tr>
<td>Personification</td>
<td>—</td>
<td>0%</td>
<td>4%</td>
</tr>
<tr>
<td>Kinaesthetic hallucinations</td>
<td>—</td>
<td>0%</td>
<td>4%</td>
</tr>
<tr>
<td>Overestimation</td>
<td>—</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Supernumerary phantom</td>
<td>—</td>
<td>0%</td>
<td>0%</td>
</tr>
</tbody>
</table>
(58% of left hemiplegics); there were three instances of denial of a right hemiplegia (14% of testable, 6% of all right hemiplegics). There was a spectrum of attitudes, ranging from vehement denial that anything was wrong at all, to admission that a stroke had occurred but denial that this had resulted in limb weakness. Most would reluctantly agree that something was wrong, usually described as "stiffness" or "heaviness." Some patients, in the course of the interview, would admit to weakness, only to deny it minutes later. Others volunteered the fact that they had suffered a "stroke," often attributing the source of this information to the doctor. When questioned as to the effects of this "stroke," they denied weakness, and instead gave some other consequence, for example a "faint." A sample response is given. "Nothing wrong. Sometimes it's a bit stiff. It needs exercise. Something hurts. Something aches." The behaviour and statements of patients when cross-examined provided some insight into their thought processes. The impression gained was that by manipulating the situation one could achieve an admission that there was some abnormality, but still fail to convince them that the explanation for this weakness was in a limb. When presented with overwhelming evidence, they resorted to evasion ("Doctors know more about it than I do"), or inconsequential remarks ("I thought it was a stroke once, but now I've seen chaps in the ward with them").

ANOSOGNOSIC PHENOMENA

Fourteen patients with a left hemiplegia (29%) showed anosognosic phenomena in the absence of anosognosia itself; a further four demonstrated both. Nine patients with a right hemiplegia (41% of testable, 18% of all) exhibited anosognosic phenomena. None had associated anosognosia. Individual phenomena are described. Two patients with a right, and two with a left hemiplegia demonstrated anosodiaphoria. The experience of nonbelonging of a limb was present in 11 left hemiplegics and three right hemiplegics. They offered motor ("It disobeyed me"), sensory ("I've got no feeling"), or visual explanations ("My fingers shrank to short fat fingers"). Three patients, all left hemiplegics, attributed ownership to another person ("I felt it could have been a nurse's hand, a neighbour's, my wife's, a doctor's"). Strange sensations, not to the extent of nonbelonging, were reported by three left hemiplegics ("lifeless," "cold and clammy"). Misoplegia was expressed by two left and five right hemiplegics ("I'll hit it with a two-pound hammer when I get back to work; its a bloody nuisance"). Personification was seen in one left hemiplegic. She claimed that the nurse had originally called the leg "Fred," and she then called her arm "Little Fred," she further stated that she could be called "one-handed Pandy." Kinaesthetic hallucinations occurred in two left hemiplegics. One said that he "tried gripping, and if I am not looking, I feel as if I'm moving it, but when I look, I'm not." Neither denied their weakness. Overestimation of the strength of the unaffected arm occurred in two left hemiplegics. The circumstances were different in each case. One, although admitting to weakness in the left arm, insisted that the examiner test the strength of his right arm, which he said was "very strong." Another, with anosognosia, offered this increased strength as one of a series of inconsistent explanations when faced with the falling away of his left arm. A supernumerary phantom limb was not clearly identified in this study. Its presence might have been inferred from the behaviour of one left hemiplegic who fumbled in his axilla on cross-examination.

GENERAL, NEUROLOGICAL, AND HIGHER MENTAL FUNCTION PROFILE

The profiles of three groups of patients are presented in Table 3. Tests of significance ($\chi^2$ or $t$ test, whichever appropriate) were used to compare anosognosics with those with phenomena only, and the latter with "normal" subjects.

General features

Sex emerged as a moderately significant factor. Men were more likely to show anosognosia or a "normal" attitude, and women the phenomena. It should be remarked that men were over-represented in the left hemiplegic group as a whole, a curious finding as the sex distribution for right hemiplegia was equal. Age distinguished those with a "normal" attitude from other groups; the former were younger. Left handedness occurred in some patients from all three groups, and no conclusions can be drawn concerning the effect of laterality on the development of anosognosia. A history of a previous hemiplegia was more common in patients with phenomena and the duration of hemiplegia from onset was longer in the same group.

Neurological features

Degree of weakness was compared across the groups, and cannot be said to affect the development of an abnormal attitude. Sensory loss did not emerge as a significant factor in the two comparisons, but there was a clear trend for its inci-
Abstract

The presence of disorientation significantly separated the three groups. Further analysis revealed that in right hemiplegics, all three anosognosics and four of the nine with phenomena were disoriented; four of the remaining five with phenomena were impaired on tests of attention or memory. In the left hemiplegics, a lower rate of cognitive dysfunction prevailed. Of 28 anosognosics, 19 were disoriented and a further four had memory impairment; of 14 with phenomena, six were disoriented and only a further one patient had deficient memory. Mood change of any kind distinguished the groups to a significant extent; apathy was virtually restricted to those with anosognosia, while euphoria and depression were the forms predominantly seen in those with phenomena. Personal neglect was only seen in one patient who did not have anosognosia, but only appeared in one-third of those with anosognosia. Visuospatial neglect failed to emerge as a significant discriminator, but there was a trend for anosognosics to show this more than other groups. Visuoperceptual deficit appeared a potent factor in separating anosognosia from those with phenomena. However, it was not entirely certain that failure to identify simple pictures reflected a pure disturbance of visual perception, independent of such variables as neglect or inattention. Abstract language was considerably worse in those with phenomena, an unexpected finding which suggests that impairment of metaphorical abilities is not crucial to the development of anosognosia itself, but appears to play a part in at least some of the phenomena.

Discussion

Incidence

An abnormal attitude towards a recent hemiplegia was common with damage to either hemisphere. Anosognosia and anosognosic phenomena were seen, respectively, in 58% and 29% of left hemiplegics, and in 14% and 40% of right hemiplegics. The estimation of the incidence of anosognosia for a left hemiplegia is slightly higher than that of other authors, who give figures ranging from 30% to 50%. Anosognosic patients in the present study were seen after only three to four days had elapsed from the initial ictus, and this early examination may have been responsible for the high incidence. The other authors mentioned (Nathanson et al., 1952; Gross and Kaltenbäck, 1955; Ullman, 1962) recorded a longer mean interval between the onset of the stroke and their assessment. As chronic hemiplegics rarely exhibit anosognosia (Gilliatt and Pratt, 1952), factors unique to the acute stage of a cerebrovascular insult must be regarded as essential to the development of anosognosia.

Laterality

The apparent difference between the incidence of an abnormal attitude towards a right (54%) and left hemiplegia (87%) is complicated by the large group of 30 right hemiplegics who were aphasic. If all the aphasics are assumed to have held an abnormal attitude, not an unreasonable assumption, then the figure for all right hemiplegics...
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would rise to 81%. It certainly appears that right hemisphere damage is not essential to the development of an anosognosic attitude. There remains the question of whether it has a quantitatively or qualitatively different role than damage to the left hemisphere. Only six of 48 left hemiplegics were regarded as holding a normal attitude, while 10 of 22 testable right hemiplegics did so. This is slender support for a quantitative difference between hemispheres. Some of the findings lend at least meagre support to the idea that different factors may be responsible in each hemisphere. For instance, left hemiplegics could develop anosognosia or its phenomena in the absence of disorientation or even impaired memory; in contrast, all but one right hemiplegic with an abnormal attitude had obvious cognitive impairment.

ANOSOGNOSIC PHENOMENA

Although deficits in higher mental functions were more common in patients with phenomena than in normals, they were less remarkable than in anosognosics. In the main, it appeared that general factors distinguished the two groups. Those with anosognosic phenomena were more likely to be women and to have had a previous hemiplegia; a longer period had elapsed between the ictus and the author's examination than in the anosognosic group. The last finding is in agreement with other authors (Gilliatt and Pratt, 1952) that the phenomena arise at a later stage in the resolution of a stroke than anosognosia itself. There appears to be a sex difference in hemispheric distribution of language and visuospatial skills (McGlone and Kertesz, 1973), although it is difficult to relate the sex difference between anosognosia and its phenomena to this. The previous hemiplegia might have provided an experience which rendered their current state more understandable than to those for whom a hemiplegia was a novel occurrence. Of the phenomena, anosodiaphoria and the experience of “nonbelonging” were the most likely to show deficits of higher mental function, and had therefore the best claim to be regarded as “pathological attitudes.” Misoplegia and some of the rarer phenomena might be better regarded as “normal” adaptation phenomena (Critchley, 1953; Frederiks, 1969).

PATHOGENESIS OF ANOSOGNOSIA

Deficits in higher mental function and field defect were the most significant correlates of anosognosia. The higher level of significance obtained with field defect, apathetic mood, and visuoperceptual deficit suggests that these should be regarded as particularly important. These considerations allow a critical appraisal of the main theories concerning the nature of anosognosia.

An emphasis on “confusion,” accentuating a general tendency of ill people to repudiate their disability (Weinstein and Kahn, 1955; Ullman, 1962), does not provide a comprehensive account of anosognosia. A confusional state was not invariably associated with anosognosia and was not infrequent in patients without this state. Anosognosia is not adequately defined merely as “denial of illness;” these patients would admit to a “heart attack” and even a “stroke,” but fail to appreciate weakness of a limb. Kinaesthetic hallucinations (Waldenström, 1939; Frederiks, 1969) cannot be regarded as the fundamental element in the condition. In the first place, only two patients reported them, and neither had anosognosia itself. Secondly, there was evidence that visual factors (field defect, poor picture identification) were more relevant than kinaesthetic factors. Further, when hemiparetic patients were asked to raise both arms, those with anosognosia would accept that they came to rest at different levels but fail to appreciate that this might indicate weakness of one limb. It appeared that in this situation they could use some of the information pertaining to one side of the body but failed to integrate it into a judgment about weakness. Unqualified adherence to the idea of anosognosia as a “body image disorder” with the implication of a right hemisphere “centre” (Bogen, 1969; Galin, 1974) is not consistent with the present results. The term “body image disorder” is, however, thoroughly ambiguous, and while anosognosia can be accommodated by such a broad definition, the original proposal of Head and Holmes (1911) of a “body schema,” essentially a physiological model which could account for certain sensory deficits of cortical origin, lacks the robustness necessary to explain the diverse manifestations observed in the present series.

Psychodynamic, kinaesthetic, and “body image” theories do not, therefore, provide satisfactory accounts of anosognosia. Other suggestions can be divided into two groups, which emphasise either “neglect” or “agnosia.”

Gross and Kaltenbäck (1955) introduced the term “anosognosic complex” to describe the background of neglect, apathy, and inattention out of which they believed that anosognosia developed. Denny-Brown et al. (1952) saw in anosognosia an illustration of “amorphosynthesis,” which they claimed was a fundamental characteristic of parietal lobe disease of either hemisphere, and which consists of a tendency to extinguish one member of a stimulus pair and to lose insight into
perceptual processes. These are adequate descriptions of much of anosognosic behaviour. The development of unilateral neglect in monkeys (Kennard, 1939; Welch and Stuteville, 1958) and in man (Heilman and Valenstein, 1972) from contralateral lesions in the frontal lobe provides a relevant experimental model with which to compare anosognosia. The deficit in these reports was transient, affected stimuli from all sensory modalities and produced an apparent, but false, hemianopia. There was "an apparent lack of recognition of objects" attributed to "a disturbance of the more complex integrative processes of the frontal lobe" (Welch and Stuteville, 1958). The resemblance to anosognosia is striking. Each condition was transient, both visual and kinaesthetic sources of information were affected, and there was a marked failure to identify pictures of simple objects. However, a number of anosognosic patients acknowledged the presence of morbid change in the affected limb and cannot be regarded as demonstrating "neglect" unless one broadens the concept to mean a failure to balance information entering from the two sides (Denny-Brown et al., 1952; Heilman and Valenstein, 1972). Interpreting "neglect" in this way, one can allocate patients to points along a spectrum, with, at one end, severe forms giving rise to complete denial of any change in a limb, and, at the other, minor forms where the experience of morbid change is preserved but the capacity to make a judgment about the cause of this is disrupted in the central analysis.

Another approach is to regard the deficit as a form of agnosia. This idea was discussed by Sandifer (1946) and Roth (1949), and much of the discussion on the nature of anosognosia, particularly the role of "confusion" and lower-order sensory deficits, can be found in the literature on visual agnosia. Further, Geschwind (1965) argued that visual agnosia could be regarded as a language disorder and this point has been made for anosognosia by Weinstein et al. (1964). A further comparison can be drawn between the nature of visual agnosia and anosognosia. In the former, a distinction has been made between "apperceptive" and "associational" forms (Taylor and Warrington, 1971), and applying this idea to anosognosia it can be argued from the present results that most patients have normal apperception in their appreciation of morbid change, but disturbance in the associational sphere by their faulty choice of linguistic term to express their experience. Others, comparatively few in this study, had little or no appreciation of change in a limb and might be regarded as showing "apperceptive anosognosia."

In conclusion, I believe that the present study has advanced the understanding of anosognosia by pointing out the distinction between this and anosognosic phenomena, and by examining the relationship with laterality of hemispheric damage. In demonstrating the significant association with certain other neuropsychological deficits, I have indicated the poverty of some theoretical positions and the relevance of others. In the present state of knowledge, I believe that the two most comprehensive approaches attribute anosognosia to either "neglect" or agnosia. In favour of "neglect" was the prominence of apathy in the present study and the relevant animal experiments in the literature. I am, however, impressed by the associated visuo-perceptual deficits and by the number of patients who appreciated morbid change but failed, even in the absence of disorientation, to arrive at a judgment of weakness, and favour, therefore, an agnostic basis for the condition.

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