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Short report

Persistent denial of handicap after infarction of the right basal ganglia: a case study

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SUMMARY A case is reported of persistent denial of handicap following stroke. Hemiplegia was due to infarction involving only sub-cortical structures, and there was no associated visual or sensory neglect or inattention, and no evidence of dementia.

It is not uncommon in clinical practice to encounter patients who are severely handicapped by some neurological deficit, and yet are apparently unaware that they have any disorder. The best known examples are the cortical blindness without insight described by Anton,[1] and the denial of hemiplegia to which Babinski[2] first gave the name *anosognosia*. This lack of awareness of deficit should be distinguished from a lack of appropriate concern for any existing deficit (*anosodiaphoria*) which is seen in those who are aware intellectually that they are disabled. This distinction was drawn by Babinski in his original description, but has not always been recognised since.

Although there is general agreement about the existence of these phenomena, there is uncertainty as to their exact nature. Two particular inconsistencies emerge from the literature. The first is that it is unclear whether denial of an *impairment or deficit* (such as hemiplegia) is the same thing as denial of the *consequencies of impairment* that is, denial of handicap or illness. Frederiks[3] stated explicitly that "Agnosognosia must not be identified with the denial-of-illness syndrome", and suggested that the former might be understandable more in neurological and the latter in psychological terms. Other authors have been less certain; Cutting[4] claimed to adopt Frederiks' nomenclature in his study, but did not mention denial of illness/handicap as a separate phenomenon from anosognosia. Willanger *et al*[5] identified three varieties of the phenomenon: obstinate denial of hemiplegia, inconsistent denial of hemiplegia, and inconsistent evaluation of own condition, the latter apparently involving denial of handicap with acknowledgement of paresis. However they concluded that the difference between the three was one of degree rather than quality.

The second major uncertainty is whether denial of deficit and/or handicap is simply a manifestation of neglect-inattention, or whether it can occur independently. Critchley[6] was unclear about this matter, separating unilateral neglect from denial of hemiparesis in his classification of disorders of body image, but noting how frequently the syndromes overlap. Sandifer[7] stated "... neglect of half the body and of minor disabilities involving it are examples of the milder forms of anosognosia", and the recent monograph by Kirschner[8] similarly discusses denial and neglect under the same heading. Although Frederiks[3] and Cutting[4] explicitly distinguished between denial of hemiplegia and neglect of the paralysed half, they classify both phenomena as aspects of the wider syndrome of anosognosia.

There are clinical reasons for these inconsistencies, which are the result of the fact that anosognosia usually occurs in association with large lesions, and in the acute phase of the illness. In fact, Cutting observed "... factors unique to the acute stage of a cerebrovascular insult must be regarded as essential to the development of anosognosia". Under such circumstances many neurological and psychological deficits

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co-exist and it may be difficult to judge which phenomena are essential to the presence of anosognosia, and which are merely common accompaniments of large acute lesions.

We present a case of persistent denial of handicap arising from hemiparesis, in which the lesion responsible for the paralysis was a small deep-seated infarct. The unusual features of the case throw some light on the nature of this clinical problem.

Case report

The patient was an 89 year old woman, who lived alone and functioned independently prior to the onset of her stroke. In January 1986 she suffered an episode of transient left hemiparesis lasting 1–2 hours. The next day she was found unconscious on the floor and was admitted to hospital, where she had fully regained consciousness within 36 hours. When seen 5 days later she was alert but unable to recall the circumstances of admission. She had a left hemiparesis with no arm movements and MRC grade 1 movement only in the leg. There was no sensory loss in the limbs. Cranial nerves were normal apart from left UMN VII palsy, and visual fields were full to confrontation. Design copying was accurate and there was no evidence of visual or tactile neglect/inattention. She appeared mentally alert and coherent at interview, and scored 7/10 on Hodkinson's Mental Test[9] (that is, in normal range). At this stage no mention was made by the examining doctor of her attitude to her weakness.

Full blood count, glucose, and lipids were normal; syphilis serology was negative. CT performed next day showed an infarct involving the right internal capsule and extending laterally into the globus pallidus and putamen and superiorly into part of the corona radiata. In addition there was a smaller and subclinical lesion adjacent to the anterior horn of the left lateral ventricle and a minor degree of periventricular lucency (fig 1).

She was seen on one occasion 2 months later, at which time she was living in a nursing home, markedly handicapped by hemiparesis and wheelchair-bound (Barthel ADL[10] score 4/20). The staff reported that she was consistently denying her problems, despite being alert and well orientated. At this stage a fuller examination of the denial was not undertaken.

The examination reported here was undertaken 6 months after the original stroke. At this time the patient was still chairbound, unable to weight bear and dependent for feeding, dressing, use of toilet and transfer from chair; Barthel ADL score 4/20. She had a persistent dense left hemiplegia with normal pain and touch sensation. We undertook a systematic examination of her mental state in the following order: spontaneously expressed awareness of deficit and its consequences; effect of demonstration of weakness on awareness of deficit; judgement of disability and its consequences in others; assessment of agnosia, neglect, inattention; assessment of cognitive function and mood.

(1) In spontaneous conversation she could not explain her presence in the home. She said she did not walk because she had hurt her right foot (sic) and had been told to rest it by the staff. When pressed she admitted that her arm might be

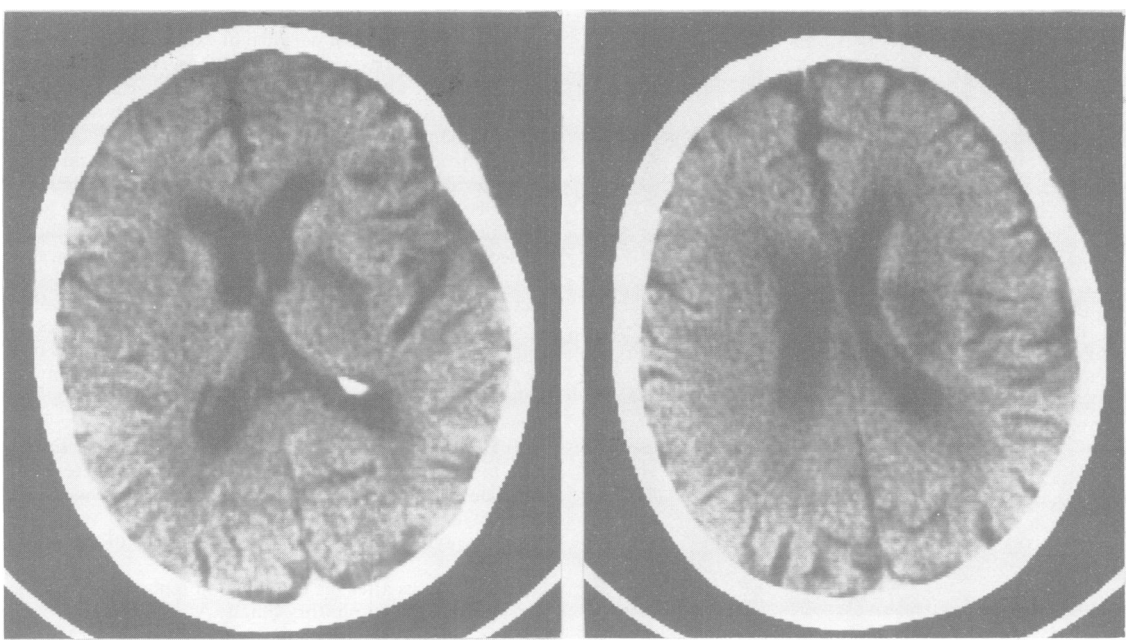

Fig 1 *CT scan (6 days after stroke) demonstrates an infarct involving the anterior limb of the right internal capsule, globus pallidus and putamen and superiorly, the corona radiata.*

Table *Self-ratings of strength in the left side at different stages during the assessment (right side taken as 10/10 for comparison)*

	Spontaneous (pre-examination)	*Immediately post-examination*	*5 minutes post-examination*
Shoulder	6/10	4/10	6/10
Elbow	4/10	0/10	6/10
Hand/wrist	2/10	0/10	6/10
Hip	6/10	2/10	6/10
Knee	6/10	2/10	6/10
Ankle/foot	6/10	2/10	6/10

weak, but suggested that this was because she had broken it some years previously. We asked her to grade the strength in her limbs on a scale of 1–10, taking the strength in her normal right limbs as grade 10 as a standard for comparison. Her ratings did suggest that she felt her arm was weak, and more so than her leg which she regarded as "not too bad" (table). Despite this awareness of some deficit, she denied all handicap, claiming she would be able to walk, feed and dress herself unaided, and even drive a car although "the left side might be a bit awkward".

(2) During physical examination, when it was demonstrated to her that her left arm was completely paralysed and her leg nearly completely so, she modified her ratings slightly. She now rated her arm 0/10 at elbow and hand, and her leg 2/10. However, when she was asked to rate her limb strength again after 5 minutes distraction, she scored 6/10 in all groups. Again she denied all handicap, both immediately after examination and 5 minutes later. For example, although she rated strength in her leg as 2/10 in all groups and had demonstrated that she could not move her knee or ankle even with gravity removed, she insisted that she could walk upstairs unaided if she were allowed to.

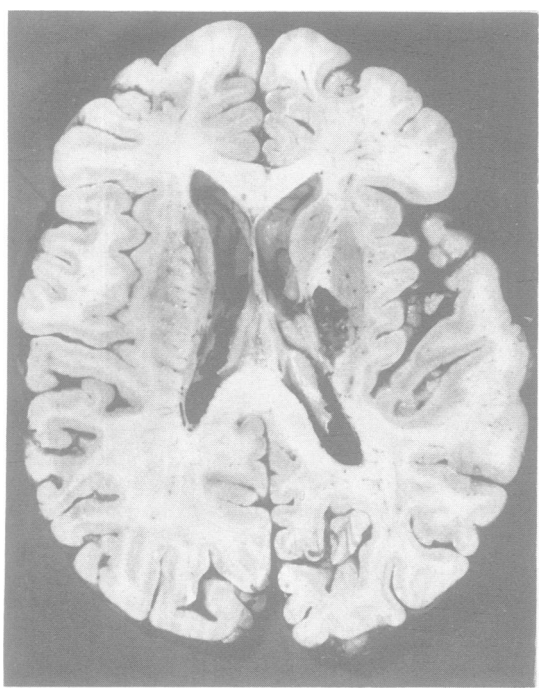

Fig 2 *Axial brain slice at the level of the insula showing infarct confined to the right internal capsule, and basal ganglia.*

(3) As a test of her ability to assess the importance of neurological deficits, she was shown six black and white photographs portraying patients with differing disabilities, such as walking with a Zimmer frame, or sitting in a wheelchair. Her ranking of these according to their disability was accurate. For example, she ranked as "most disabled" the wheelchair-bound patient and as "least disabled" a patient walking unaided. When asked to select the person "most like her" she picked the patient in the wheelchair. Neither in her performance of this test nor in her recognition of everyday objects or of those around her was there any evidence of visual agnosia.

(4) None of the more bizarre disorders sometimes associated with this syndrome, such as denial of ownership or misoplegia,[4] were present, and attempted limb movement with eyes closed did not produce kinaesthetic hallucinations.

(5) Her spontaneous behaviour did not suggest any neglect of the limbs or of personal or external space. During the interview she frequently touched and rubbed her paralysed arm and altered its position using her good hand. Visual fields were full to confrontation, with no neglect on confrontation testing, reading, drawing spontaneously or design copying. Peripheral sensation was normal and there was no evidence of tactile neglect on bilateral stimulation.

(6) She was alert and cooperative and showed a good grasp of current affairs. On Folstein's Mini Mental State[11] she scored 26 (cut off for organic impairment 24) when first seen. Cognitive testing on two further visits revealed a fluctuating impairment of registration (for example recall of fictitious addresses, digit span forwards) but no evidence to suggest significant dementia. The home staff confirmed that she was orientated and alert with normal memory in her exchanges with them. When she did have memory lapses she showed no tendency to confabulate.

(7) Her mood state was assessed by Present State Examination,[12] a semi-structured psychiatric interview. Her only complaints were of a mild degree of depression related to a desire to be in her own home, but she had no vegetative depressive symptoms. There was no evidence of elation or euphoria.

(8) Six months after the examination reported above, she died after a short respiratory illness. A necropsy confirmed that she had died of bronchopneumonia. Serial slices of the brain confirmed the presence of the two lesions seen on the CT scan (fig 2). The larger infarct which was responsible for

the hemiparesis was entirely confined to deep subcortical structures, and on none of the slices was there evidence of a cortical lesion. Brain histology did not reveal Alzheimer changes, nor evidence of deep white matter ischaemia.

Discussion

In this case denial of handicap was unusually persistent, since it is classically described as an acute disorder with resolution occurring over a matter of weeks.[6 13] In other ways however the presentation was typical, with bland denial of handicap occurring in clear consciousness and despite demonstrations of severe paralysis, these demonstrations being dismissed with trivial rationalisations. By contrast with this obstinate denial of handicap, the denial of hemiplegia, which was also present, was more inconsistent and could be modified at least temporarily by demonstration of weakness at physical examination.

This denial was not associated with unilateral sensory or visual neglect-inattention, or with primary sensory loss, none of which were present either immediately after the stroke or 6 months later. As noted above, the denial of handicap persisted even when the paralysis in the limbs was brought to conscious awareness by examination. In other words, anosognosia and denial of handicap are not simply manifestations of unilateral neglect but are clearly dissociable disorders of higher cognitive function.

Although there was evidence of mild cognitive impairment, this was insufficient to account for the denial on the basis of a confusional state. This conclusion would be supported by her response to the photographs, which showed her ability to make correct judgements about the meaning of neurological lesions in others. The absence of the indifference or euphoria sometimes described in such cases, would not support an explanation couched solely in psychological terms (denial as a defence), although no such observation could entirely discount a psychodynamic theory.

The absence of any cortical damage in this case is of considerable theoretical interest. Recent findings have suggested that syndromes previously considered as due to cortical damage (such as aphasia[14 15] or neglect[16]) may be found when the only demonstrable structural lesions are confined to the subcortex. Denial has usually been assumed to be associated with right parietal lesions, but this case suggests that it too may arise in association with damage restricted to deep structures. Functional studies have demonstrated that such lesions may have a widespread effect on cortical metabolism, at least in the acute phase,[17] so the part played by the anatomical lesion in producing cognitive deficits remains to be clarified.

In our opinion, these findings support the suggestion that denial of hemiplegia (anosognosia) and denial of consequences of hemiplegia are closely related but not identical phenomena, which are best explained as due to a failure of central integration of information from the affected limb. Thus although primary sensations may be intact they are not assimilated to lead to a modification of central schemata, and their meaning for functioning of the limbs is lost to the individual. This account would be supported by the observation that an improvement could be achieved by drawing attention to the deficits, but that it was not sustained, and that misjudgements about the leg were more striking than were those about the arm, since the latter's function is more readily observed by the patient.

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