Denial of Illness in Stroke

Simon J. Ellis, MRCP, and Marian Small, PhD

**Background:** The effects of stroke on classically nondominant hemisphere functions have received less attention than those on the dominant hemisphere, but visuospatial neglect and denial of illness both produce significant morbidity.

**Summary of Comment:** The early literature on denial of illness is discussed and the etiological theories are examined. These are explanations based on deficits of higher mental function, impaired sensory input (especially proprioceptive), an abnormal representation of body image, psychodynamic defense mechanisms, and/or premorbid personality factors.

**Conclusions:** Denial of illness is an important consequence of stroke. No explanation thus far proposed is entirely satisfactory. The consequences on rehabilitation and strategies for therapy have not been adequately investigated. *(Stroke 1993;24:757–759)*

**KEY WORDS** • cerebrovascular disorders • denial

Many of the consequences of stroke are apparent to a casual observer, but visuospatial neglect and denial of illness need to be specifically sought. Failure to appreciate these phenomena, which are particularly associated with nondominant hemisphere infarcts, results in an underestimation of the morbidity in this population.

It is easy to see how hypochondriasis can cause disruption and morbidity, yet its converse, denial of illness, also results in inappropriate behavior and much distress. One of the most florid forms of denial of illness is that seen in hemiplegic patients who may believe that there is nothing wrong with them. From the frequency of reports of this phenomenon in the literature one might conclude that it is not common. However, by its nature it rarely appears spontaneously in the history, it has to be elicited by specific questioning, and many patients demonstrate elements of denial once they are sought.

Anton1 and Pick2 were the first to give brief descriptions of denial of illness in 1898. The term anosognosia was subsequently coined by Babinski3 in 1914 to describe this condition in two patients who failed to acknowledge their left-sided hemiplegia. Etymologically, the term anosognosia describes any condition or illness that is not acknowledged by the patient. The term denial of illness more accurately reflects the active and delusional nature of the disorder, whereas “anosognosia” implies a purely passive phenomenon.

Unawareness of disability can occur in neurological disorders such as Anton’s syndrome (unawareness of blindness), amnesia, aphasia, head injury, hemiplegia, and hemianopia,4 as well as in disorders of noncerebral etiology such as heart disease,5 lung cancer,6 spinal cord injuries,7 and severe burns.8 Denial can be very specific; stroke patients may acknowledge memory impairment and left upper limb paralysis but refuse to recognize paralysis of the leg.9 It may be limited to neurological disability, as a case in which heart disease is admitted but the more recent and obvious hemiplegia is denied.10 Very recently it has been concluded that global cognitive dysfunction is not necessary for denial of hemiplegia, as patients’ acknowledgment of their own condition and paresis was selectively impaired in a state of clear consciousness.13

Early studies stressed the importance of sensory loss on the affected side, especially proprioception, in association with denial.14 However, denial of hemiplegia can occur in the absence of sensory loss, as was found in 13% of a series of 31 patients.15 Sensory loss per se, therefore, cannot be sufficient to produce this condition. The anatomic correlates of denial of hemiplegia, as demonstrated by computed tomography, implicate inferoposterior parietal areas, indicating that the condition is related to damage to structures outside the primary sensory cortex.16

---

From the MRC Research Centre in Brain and Behavior, Department of Clinical Neurology, Oxford University, Radcliffe Infirmary, Oxford, UK.

Address for correspondence: Simon J. Ellis, Oxford University, Department of Clinical Neurology, Radcliffe Infirmary, Oxford, OX2 6HE, UK.

Received June 26, 1992; final revision received January 21, 1993; accepted January 22, 1993.
Recently a “discovery theory” has been proposed which suggests that the presence of both cognitive loss and sensory loss provides an explanation for denial of hemiplegia. According to this theory, the failure to discover the paralysis results from loss of proprioceptive mechanisms as well as an additional cognitive deficit that prevents the necessary observations and inferences.

In 1942 Gerstmann18 recognized that sensory impairment alone does not explain denial of illness. He suggested that beyond the primary sensory area there is a more complex mechanism in which a body scheme or body image is represented. This proposed mechanism is localized chiefly in the parietal region, where tactile, kinesthetic, and visual experiences contribute to an integration of a body scheme: a kind of inner diagram of one’s body as a whole. During a dynamic process, bodily alterations are recognized from afferent sensory impulses that initially occur outside central consciousness but then become conscious cognition. Denial of hemiplegia is explained as a specific impairment in this higher level of neural integration that regulates body consciousness. Such a defect results in unawariness of a functional change without necessarily involving any general intellectual loss.

Because denial of disability can occur in the absence of brain insult, a psychological “defensive” explanation has been proposed.19,20 Seventy-one percent of patients with denial of hemiplegia were judged to have moods inappropriate for the circumstances (51% euphoric, 10% apathetic, 10% depressed), but only 6% of patients who recognized their hemiplegia exhibited abnormal moods.15 It was also noted that there was no generalization of denial of illness, in that patients would readily admit to a heart attack or a stroke but would not acknowledge weakness of a limb. Such a defensive mechanism may be reflected in personality factors. When postmorbid personality was examined, patients who denied illness were found to be compulsive and in need of prestige from others when compared with control subjects. They had a history of denying inadequacies and regarded illness as an imperfection.21 However, an explanation based on personality factors was thought unlikely by Willanger et al13 because of the frequency of denial of hemiparesis (25% of 55 consecutively selected patients).

Denial of hemiplegia has been thought of as a phenomenon of the nondominant hemisphere,18 but differences in the frequency of observation of denial may reflect the concomitant aphasia with lesions of the dominant side. In a study of 100 consecutive patients with hemiplegia, there were 31 cases of denial.15 Thirty of the 52 patients with right hemiplegia were so aphasic that assessment was impossible. Three (14%) of the remaining 22 patients with right hemiplegia who could be assessed had denial, whereas the incidence was 58% in the left hemiplegic group. The issue of exactly how far severe receptive aphasia masks denial in patients with dominant hemisphere damage remains unclear. Cattell’s15 estimation of the incidence of denial with left hemiplegia (58%) is higher than those of other authors,13,22 but this may be due to his earlier examination of patients (less than 8 days from onset).

That an association between denial and neglect exists has also been subject to study. Inattention to space contralateral to a brain lesion, known as unilateral visuospatial neglect, is typified by the patient’s ignoring visual stimuli on one side or producing drawings that are crowded to one side of the page. In a series of right hemisphere stroke patients, 46% were found not to attend to left-sided stimuli, and 36% had denial. There was a significant association between neglect and denial. An association was also found with lesion size in denial and neglect, indicating that both phenomena occur with more extensive lesions.22 However, a double dissociation between denial and neglect has been reported. In 97 patients with right hemisphere damage, 32 had denial with little or no neglect, whereas four had neglect without denial.16 Denial for hemiplegia is therefore not necessarily a manifestation of unilateral neglect. That no one factor has been identified as being invariably associated with denial of illness suggests that several factors either individually or acting in concert may be required to produce this phenomenon.

There have been few reports concerning the spontaneous rate of recovery from this disorder. In a follow-up series of patients with right hemisphere stroke who were initially tested at 7 days and then at 2- to 4-week intervals, the mean time required to recover from denial of illness was 11 weeks.23 However, some studies have reported that the phenomenon remains present for several years after the event.10 Recently a surprising discovery has been reported: irrigation with iced water of the external auditory canal contralateral to the lesion can produce transient, complete remission of denial of illness.24

Seneca25 noted two millennia ago that in some circumstances “it is difficult to recover from illness just because we are unaware of it.” Although the phenomenon may superficially protect the patient from some of the psychological trauma of coming to terms with the diagnosis, it can be disturbing and confusing for relatives. It often results in hemiplegic patients attempting inappropriate and harmful activities, such as trying to get out of bed or walk unaided. It may also be difficult to initiate active physical therapy while the patient continues to deny that there is a problem. In association with visuospatial neglect, denial of illness contributes to some of the morbidity of stroke not directly attributable to the hemiparesis.

References
Ellis and Small  Denial of Illness in Stroke 759

Denial of illness in stroke.
S J Ellis and M Small

Stroke. 1993;24:757-759
doi: 10.1161/01.STR.24.5.757

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/24/5/757