

THE ANATOMY OF ANOSOGNOSIA FOR HEMIPLEGIA: A META-ANALYSIS

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ABSTRACT

Anosognosia for hemiplegia is the denial of the contralesional motor deficits that may follow brain damage. Although this disturbance has been reported in the neurological literature since the beginning of the last century, only few longitudinal studies have addressed the issue of the anatomical substrate of the disorder. Here we present a comprehensive review of the literature on anosognosia for hemiplegia from 1938 to 2001, taking into account some of its clinical, epidemiological and anatomical aspects. In particular, an attempt has been made to identify the intra-hemispheric lesion locations most frequently associated to the denial behaviour.

Our review shows that anosognosia for hemiplegia most frequently occurs in association to unilateral right-sided or bilateral lesions of different brain areas (cortical and/or subcortical). It seems to be equally frequent when the damage is confined to frontal, parietal or temporal cortical structures, and may also emerge as a consequence of subcortical lesions. Interestingly, the probability of occurrence of anosognosia is highest when the lesion involves parietal *and* frontal structures in combination, if compared to other combinations of lesioned areas. This pattern of lesions suggests the existence of a complex cortico-subcortical circuit underlying awareness of motor acts that, if damaged, can give rise to the anosognosic symptoms.

Key words: anatomy, anosognosia, brain damage, denial, hemiplegia, neglect

INTRODUCTION

Anosognosia for hemiplegia (namely, the denial of contralesional motor deficits in brain-damaged patients: Babinski, 1914; Papagno and Vallar, 2003, for historical review), is a common neurological symptom, usually reported after focal lesions of the right hemisphere. This disturbance has both 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 from motor disorders after right-brain damage (Gialanella and Mattioli, 1992). Anosognosia also has theoretical implications for the study of higher cognitive functions. Indeed, the detailed study of patients' denial can disclose implicit mental contents and shed light on the neural structures underlying conscious mental processes (Berti et al., 1998).

Despite the neuropsychological relevance of this symptom, the problem of its anatomical correlates has seldom been addressed in longitudinal studies. There are two main issues related to the cerebral localization of anosognosia for hemiplegia. The first concerns the side of the brain most frequently damaged. Most studies agree that the lesion usually involves the right hemisphere. Although this figure might be due to the difficulty of testing left brain-damaged patients with aphasia, recent studies investigating the memory for right and left hemiplegia after inactivation of either hemisphere with intracarotid amytal injection, found a higher

prevalence of denial after right hemisphere injection. This strongly suggests a right hemisphere involvement in the monitoring of motor performance (Gilmore et al., 1992; Carpenter et al., 1995; Adair et al., 1995; Breier et al., 1995). Much more problematic is to find an agreement amongst different studies relative to the intra-hemispheric localization of the damage. Indeed, many different regions, cortical and subcortical, have been indicated as having a crucial role in causing the disorder. Earlier descriptions of the anatomy of anosognosia reviewed by Gerstmann (1942) and more recently by Ellis and Small (1997) were not consistent. A lesion to either the right parietal lobe or to the right optic thalamus involving the right thalamo-parietal radiations has been considered as a necessary prerequisite for the presence of anosognosia (Potzl, 1925; Barkman, 1925). In two cases reported by von Hagen and Ives (1937), one patient had a putaminal haemorrhage, whereas the other had a large infarct in the vascular territory of the middle cerebral artery. Nielsen (1938) also associated the presence of anosognosia with damage to the thalamus, or to the thalamo-cortical projections. In other cases, however, these regions were reported to be spared (Muller, 1905; Pineas, 1931).

Also in more recent studies anosognosia has been found to be associated with both superficial and deep lesions, or with damage confined to deep structures. For instance, Bisiach, Vallar, Perani, Papagno and Berti (1986) studied anosognosia for upper limb motor disorders and visual field defects. In both cases anosognosia was found to occur more

frequently in association with lesions involving the infero-posterior parietal region and/or deep structures (thalamus and basal ganglia), although the composite contour maps of the lesions of the six patients presenting anosognosia for the motor impairment also showed some degree of frontal involvement. Similarly, Starkestein et al. (1993) found that patients with anosognosia had higher frequency of temporo-parietal, basal ganglia and thalamus lesions than patients without anosognosia. On the other hand, Small and Ellis (1996) and Ellis and Small (1997), by grouping patients according to the presence/absence of anosognosia and neglect, found that the majority (70%-79%) of anosognosic patients had a lesion to the basal ganglia, whereas only 30% of the patients without anosognosia had a lesion involving this region. Therefore, although anosognosia for hemiplegia has been often considered a parietal lobe disturbance (Critchley, 1953) the results emerging from either group studies or single case report, when one considers them in isolation, do not seem to indicate the prevalence of a specific brain area involved in causing the denial behaviour.

In the present paper we reviewed an extensive literature on anosognosia for hemiplegia with the aim of defining some epidemiological and clinical aspects of the disorder. In particular, by analyzing a great number of cases where either a radiological report or a brain image study of the lesion was available, we tried to ascertain whether it was possible to identify a specific brain area responsible for the occurrence of anosognosia for hemiplegia.

METHOD

In the present paper we considered neurological/neuropsychological studies on brain-damaged patients where different aspects of anosognosia for hemiplegia were discussed. The database was selected using both PubMed Services and the references reported in the different papers we collected. Fifty-two studies from 1938 to 2001 that focused on the topic of our search were found. From this database we selected group studies and single case report according to the following criteria: Group studies were selected either when data relative to the prevalence of anosognosia (see below) were reported or when an anatomical report was available for each patient. Single case studies were selected only if the anatomical data were present. When available, we also considered the patient's age and intellectual status, the presence/absence of neglect, the aetiology and the duration of illness. It must be acknowledged that a meta-analysis approach has the limitation that the sampling and the methods of definitions of the different disturbances can vary across studies. Therefore, we have to take into account the possibility that some negative cases were excluded

as a result of tests that were not sensitive enough to detect one of the disorders at issue. This is crucial when one wants to select pure anosognosic cases, dissociated from spatial neglect. In the present study we considered as pure anosognosic cases only those patients where the absence of neglect was diagnosed after the execution of quantitative tests that evaluated different aspects of the spatial disorders. In those studies where neglect was considered to be absent, but no quantitative results were reported, data on neglect were indicated in the tables as 'not available' and the patients were not included in the 'pure anosognosic cases'.

Prevalence of Anosognosia for Hemiplegia

Anosognosia was usually evaluated using an interview that assesses the patient's awareness of contralesional limb weakness. Although there can be differences in the procedure used in the different studies, we considered a patient to be anosognosic when an explicit verbal denial of the paresis was observed. In the more recent papers of our database, structured interviews and quantitative scores related to the *severity* of the denial behaviour are also available. For the prevalence of anosognosia we considered only those studies where the number of patients affected by *complete* contralesional hemiplegia was reported. We then divided those studies in two groups. The first group comprised those studies in which patients were selected independently of lesion side (i.e. the sample comprised both right and left brain-damaged patients and patients with bilateral lesions, see Table I). The second group was composed of those studies in which patients were selected for having a right hemisphere lesion (see Table II).

It is worth noting that papers in which the prevalence of anosognosia was given in relation to the whole sample of patients who entered the study, and not in relation to the number of patients *showing the motor impairment*, were not considered. Indeed, they could not provide any information about the real prevalence of the denial behaviour in those patients who were really affected by the symptom (contralesional hemiplegia) they denied.

Anatomo-Clinical Correlations

We considered only those studies (either group studies or single case study) where the lesion location was reported for each anosognosic patient (23 studies). Data came from either autoptic examination or CT and MRI scans. Occasionally, either the scan films or the lesional maps were reproduced in the paper. The brain regions that were said to be affected are reported in the tables. Because for cortical lesions it was not possible to trace the localization at the level of single

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