Verbal asomatognosia

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Article abstract—Verbal asomatognosia is a form of neglect in which a patient denies ownership of a limb contralateral to a brain lesion. To establish the neuroanatomic substrate of this syndrome, we analyzed the CTs of 12 right-hemisphere stroke patients with neglect and verbal asomatognosia and 4 patients with neglect but without asomatognosia. Of 16 cortical and subcortical brain regions analyzed, supramarginal gyrus and its subcortical connections within posterior corona radiata were most consistently involved in the asomatognosia cases. One or both of these regions were spared in all cases of neglect without asomatognosia. Our data confirm Nielsen's localization of asomatognosia to the right supramarginal gyrus and thalamoparietal peduncle. Converging lines of evidence from experiments in humans and monkeys suggest that damage to area PF may be necessary for the production of personal neglect of a limb.

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Asomatognosia is a syndrome of distorted awareness of a part of the body. Its manifestations vary from simple failure to dress an arm or shave half of the face (nonverbal asomatognosia) to verbal denial of ownership of a limb, usually the arm (verbal asomatognosia). Both forms of asomatognosia are accompanied by hemispatial neglect.

The neuroanatomic substrate of the verbal form of the syndrome has yet to be resolved. In this study, we studied the neuroanatomic basis of verbal asomatognosia.

Methods. A series of neurology inpatients with acute right-hemisphere stroke were examined. The patients were required to be sufficiently cooperative to be able to answer questions and perform a line bisection and line cancellation task. These 2 tests were performed, and patients were deemed to manifest neglect if they demonstrated gross asymmetry on these tasks.

The patients were then examined in the following manner. The normal right arm was lifted, and the patient was asked, "What is this?" A patient was required to accurately recognize the normal limb as his own to be included in the study. The examiner then lifted, by the elbow, the limb contralateral to the lesion and moved the patient's hand and forearm into the hemispace ipsilateral to the lesion. The patient was again asked, "What is this?" A patient was judged to have verbal asomatognosia if the limb was misidentified. Care was taken to keep the examiner's hand and arm out of the patient's ipsilateral hemispace.

A number of patients with right-hemisphere lesions with neglect but without verbal asomatognosia were included for comparison. Attempts to increase the size of this comparison group were made difficult by the surprisingly high frequency of asomatognosia in acute neglect patients.

All patients underwent CT, and these studies were analyzed by a neuroradiologist who was blind to the nature of the investigation. Individual lesions were mapped onto templates adapted from a neuroanatomic atlas.[1] The templates represented 9 horizontal sections at varying levels at a 15-degree angle relative to the canthomeatal line. A series of composite templates that included all individual templates at each level were constructed as well (figures 1 and 2). The templates were divided into 16 anatomic regions, and each region was rated to be 0 (spared), 1 (mildly involved), 2 (moderately involved), or 3 (severely involved) (table).

Results. Lesion data for all patients are contained in the table. There were 16 patients with neglect included in this study. Twelve of these had asomatognosia. Patients with asomatognosia were older (70 versus 52 years) and tended to have larger lesions (table) than patients without asomatognosia. Duration of lesions until time of examination did not differ between the groups (1.9 versus 1.3 days). All patients in both groups had both hemiparesis and hemisensory syndromes, and no difference between groups was apparent on these variables. All patients in both groups demonstrated hemispatial neglect on line bisection or cancellation tasks.

Within the asomatognosia group, the most common misidentification was calling the limb "your hand" or "your arm." Several of these patients also referred to the limb as "the doctor's" hand. One patient referred to the limb as "a breast" and a "deodorant." One patient called it "my mother-in-law's" hand.

Supramarginal gyrus and posterior corona radiata deep to supramarginal gyrus were the 2 anatomic structures most highly associated with asomatognosia (table). However, no cases had lesions confined to either or both of these regions. In controls with neglect but

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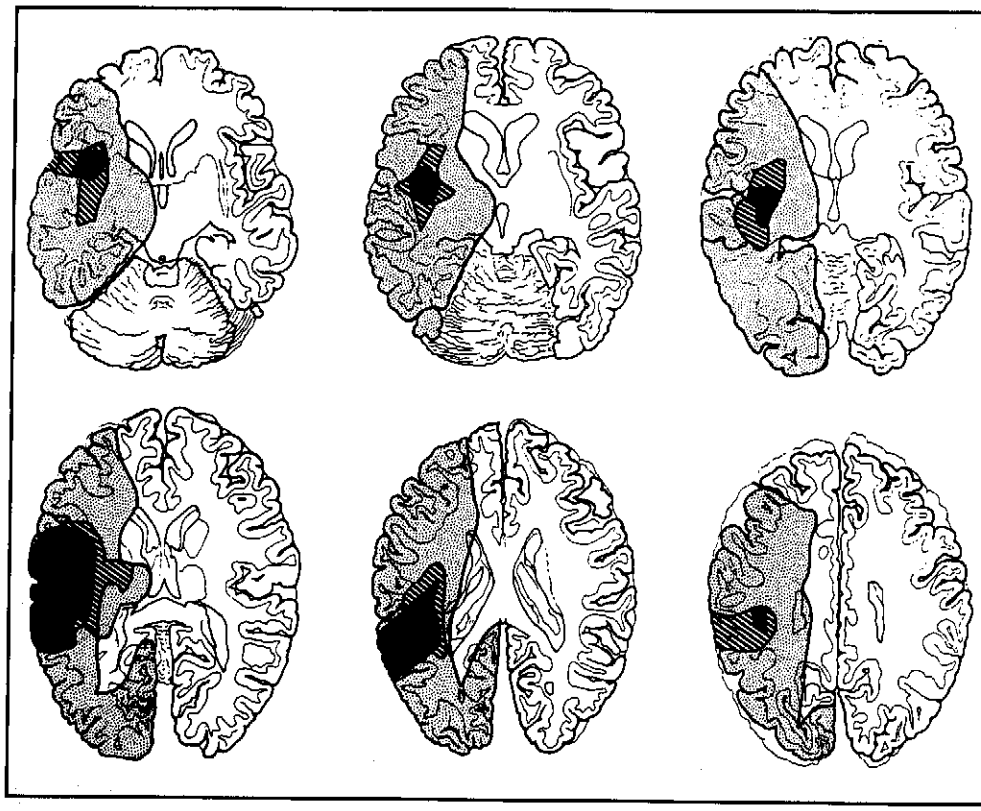

Figure 1. Composite templates of 12 asomatognosia patients. The horizontal sections shown are those in which a minimum of 6 patients overlapped. Stippled regions represent involvement in at least 1 case, striped areas represent overlap of 6 or more cases, and black areas represent overlap of 10 or more cases.

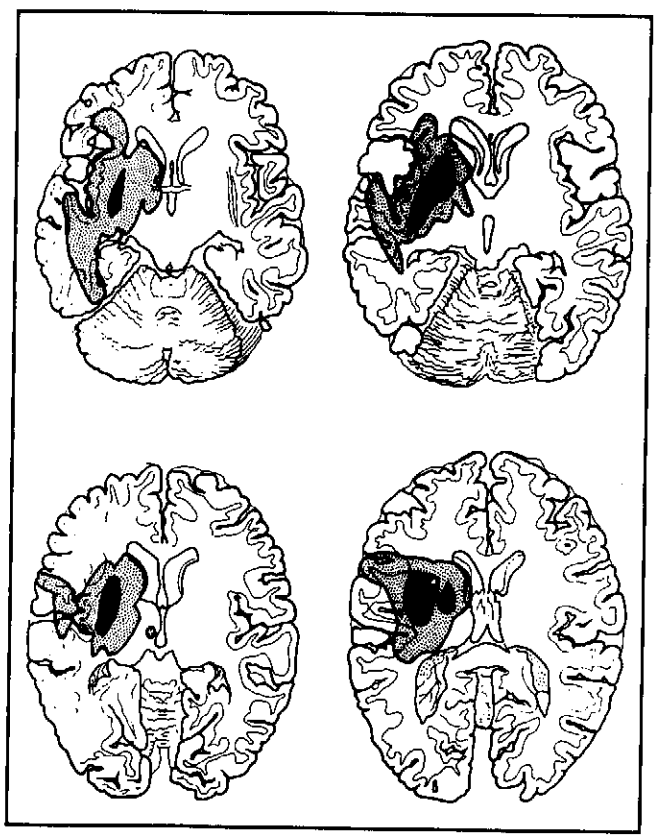

Figure 2. Composite templates of 4 patients without asomatognosia. The horizontal sections shown are those in which a minimum of 3 patients overlapped. Stippled areas represent overlap of at least 1 case, and black areas represent overlap of 3 or more cases.

without asomatognosia, supramarginal gyrus was spared in all cases, and posterior corona radiata was spared in 3 of 4 cases.

Other frequently involved brain regions in asomatognosic patients were insular cortex and lentiform nucleus. However, the involvement of these structures was less than the damage to supramarginal gyrus and posterior corona radiata. Furthermore, 3 controls had severe involvement of insula and lentiform nucleus and 1 control had severe involvement of lentiform nucleus, and yet all failed to display asomatognosia. In addition, 1 patient with asomatognosia had insula completely spared.

Angular gyrus had a relatively low frequency of involvement in asomatognosia cases and was spared entirely in 3.

Discussion. Babinski coined the term "anosognosia" to describe lack of awareness of disease, specifically hemiparesis.[2] Potzl[3] and Ives and Nielsen[4] described cases of denial of ownership of hemiplegic left limbs, and Gerstmann[5] introduced the term "autosomatagnosia" to distinguish this condition from anosognosia, which he felt was a more general condition applied to denial of other defects besides hemiplegia, such as cortical blindness.

All cases of verbal asomatognosia reported in the literature are the result of damage to the right hemisphere.[4,6-9] We have examined numerous patients with left hemisphere strokes where examination was adequate in spite of aphasia, and have never found verbal asomatognosia for the right arm.

Nielsen[6] suggested that asomatognosia resulted from

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Region*	1
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SMT	1
PLIC	2
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able. Lesion location of neglect patients with and without asomatognosia

Region*	Patients with asomatognosia													Patients without asomatognosia				
	1	2	3	4	5	6	7	8	9	10	11	12	Total	1	2	3	4	Total
PCR	3	3	3	3	3	2	3	3	3	3	3	3	35	0	0	3	0	3
SM	3	3	3	3	3	3	3	1	3	3	3	3	34	0	0	0	0	0
I	3	3	3	3	0	3	3	3	3	3	3	3	33	3	0	3	3	9
L	3	3	3	3	1	3	2	3	3	3	1	3	31	3	3	3	3	12
SMT	1	3	3	3	3	0	3	0	3	3	2	3	27	2	0	1	0	3
PLIC	2	3	3	3	1	0	3	1	3	3	2	3	27	0	1	3	0	4
C	0	3	3	1	1	3	3	0	3	2	2	3	24	0	0	0	0	0
ACR	0	3	3	3	0	3	0	0	3	3	2	3	23	1	1	3	1	6
ANG	1	3	3	3	3	0	3	0	3	0	3	0	22	0	0	0	0	0
ALIC	0	3	3	0	0	0	0	0	3	3	1	3	16	0	3	3	2	8
GIC	0	3	3	2	0	0	0	0	2	3	0	3	16	0	1	3	0	4
MIF	0	3	1	0	0	1	0	0	3	0	0	0	8	0	1	0	0	1
O	0	1	1	0	0	0	1	0	0	0	3	0	6	0	0	0	0	0
T	0	0	1	1	0	0	0	0	1	1	0	1	5	0	0	1	0	1
BTO	1	1	1	0	0	0	1	0	0	0	0	0	4	0	0	0	0	0
Caud	0	2	2	0	0	0	0	1	0	0	0	0	5	0	3	0	1	4
Total	17	40	39	28	15	18	25	12	36	30	25	31	—	9	13	23	10	—

* Regions listed in descending degree of involvement for patients with asomatognosia.

0 Spared.
1 Minimally involved.
2 Moderately involved.
3 Severely involved.
PCR Posterior corona radiata.
SM Supramarginal gyrus.
I Insula.
L Lenticular nucleus.
SMT Superior and middle temporal gyrus.
C Pre-and postcentral gyrus.
ANG Angular gyrus.
PLIC Posterior limb internal capsule.
ACR Anterior corona.
ALIC Anterior limb internal capsule.
GIC Genu internal capsule.
MIF Middle and inferior frontal gyrus.
O Occipital region.
T Thalamus.
BTO Basal temporooccipital region.
Caud Caudate nucleus.

destruction of the nondominant supramarginal gyrus or its subcortical connections via the thalamoparietal peduncle. In our investigation, there was a high frequency of involvement of supramarginal gyrus and posterior corona radiata in verbally asomatognosic patients, and these regions were spared in the non-asomatognosic patients. Other regions frequently involved in the asomatognosia group, such as insula and lentiform nucleus, were also involved in the non-asomatognosia group, suggesting that damage to these structures could be dissociated from the occurrence of asomatognosia. Our data support Nielsen's localization of the syndrome.

In this investigation, we found that patients with asomatognosia differed from patients without it by larger lesions and greater age. However, in the asomatognosia group, lesions were not random, but localized to supramarginal gyrus and its subcortical connections. Greater age of the asomatognosia group supports the study of Levine et al,[10] which found atrophy correlated with worse neglect.

Rizzolatti et al[11,12] produced personal neglect in monkeys with ablation of area 7b but preservation of sensory postcentral gyrus. The animals were indifferent to restraint and the application of noxious clips to the contralateral limb, resembling asomatognosia in humans.

Region 7b[13] corresponds to region PF in the monkey.[14,15] Area PF in humans relates to the supramarginal gyrus,[16-18] linking supramarginal gyrus to personal neglect in monkeys and humans.

Bisiach et al[19] tried to anatomically dissociate personal from extrapersonal neglect. Patients with extrapersonal neglect without personal neglect had lesions of primarily inferoposterior parietal region. Patients with extrapersonal and personal neglect had more anterior lesions involving more of supramarginal gyrus, suggesting lesions that include supramarginal gyrus may be necessary for producing personal neglect.

We found verbal asomatognosia only in association with right-hemisphere lesions. Since verbal asomatognosia is a form of neglect, the greater frequency and severity of neglect with right-hemisphere lesions[20-24] might explain this asymmetry. However, neglect may occur, though to a lesser extent, after left-hemisphere lesions.[25] Patients with left-hemisphere lesions might be too aphasic to express verbal asomatognosia.[26,27] However, we have examined many non-aphasic right hemiplegic patients without verbal asomatognosia. Another explanation is that verbal asomatognosia results from an interaction between a speaking left hemisphere and a neglected left hemispace. The nondominant hemisphere, after a left cerebral infarction, may have a similar attitude toward the neglected ipsilateral right hemispace, but the limited verbal capacities of the right hemisphere do not allow its verbal expression. In this circumstance, the speaking left hemisphere may not share the views of its nondominant partner, and therefore verbal alienation toward the paralyzed limb is not expressed.

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