Two alien hand syndromes

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Article abstract—Review of the clinical characteristics and neuroanatomy of 20 reported cases of alien hand syndrome (AHS) and a patient of our own confirm that AHS is actually two distinct syndromes. Frontal AHS occurs in the dominant hand; is associated with reflexive grasping, groping, and compulsive manipulation of tools; and results from damage to the supplementary motor area, anterior cingulate gyrus, and medial prefrontal cortex of the dominant hemisphere and anterior corpus callosum. Callosal AHS is characterized primarily by intermanual conflict and requires only an anterior callosal lesion. The occurrence of frontal AHS in the dominant limb can be explained by an increased tendency for dominant limb exploratory reflexes coupled with release from an asymmetrically distributed, predominant nondominant-hemisphere inhibition. Callosal AHS is best explained by hemispheric disconnection manifested during behaviors requiring dominant-hemisphere control.

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In 1908, Goldstein[1,2] reported a 57-year-old woman who felt her left hand had a will of its own. She complained that the hand behaved in a fashion out of the patient's control, and at one point the left hand grabbed and choked the patient's throat, and it took great strength on her part to pull it off. Postmortem examination[2] revealed multiple vascular brain lesions involving the right hemisphere, including one involving the corpus callosum.

Akelaitis[3] described a patient with a corpus callosotomy whose left hand involuntarily performed in a manner opposite to what she voluntarily desired to do with the right hand. He called this "diagonistic dyspraxia," now known as the alien hand syndrome (AHS).[4,5]

The symptoms of AHS vary. All patients have actions of an extremity that are experienced as involuntary and frequently contrary to the patient's stated intention. The normal hand typically restrains the alien one.

The neuropathologic substrate of AHS is either medial frontal[6,7] or callosal,[5,8,9] but the occurrence of AHS in the left extremity of postcommissurotomy patients has been hypothesized to be the result of retraction of the medial frontal right hemisphere during surgery.[4,5] This would suggest that AHS is a clinically and pathologically homogeneous entity resulting from medial frontal dysfunction.[6]

Some investigators have argued that AHS consists of frontal and callosal forms, with differing clinical and anatomic features. They suggest the frontal form results from a dominant medial frontal lesion, occurs in the dominant limb, and is associated with the grasp reflex, impulsive groping, and compulsive manipulation of tools.[7,10-12] The callosal forms of AHS, according to this view, do not show these features. We report a case, review the literature, and present an hypothesis that AHS consists of two syndromes.

Case report. The patient is a 68-year-old right-handed man with a history of diabetes who suddenly developed a headache with confusion and mild right hemiparesis. CT and MRI revealed a left medial frontal and callosal infarction in the distribution of the anterior cerebral artery (figure).

The headache and hemiparesis rapidly resolved, and the neurologic examination became normal within 2 weeks of onset, except for the following signs and symptoms. In the right hand, the patient had a prominent grasp response. This hand was at times in constant motion. He demonstrated frequent groping movements of the right hand, such that any nearby object, including bedclothes, sheets, objects, or the patient's own leg or genitals, were impulsively grasped. Once grasped, these objects could not be released. At one point, the patient was putting on pajamas and the right hand grabbed and tore them. He reported that he had "wrestled the right hand to the ground."

He viewed these actions as unwanted, unintentional, and uncontrollable. He described the right hand as "the bad one, it has a mind of its own," and that it was "always trying to get into the act." A very specific complaint, which was chronic and persisted for a year after the infarction, was that the patient felt the right hand anticipated future actions and performed movements

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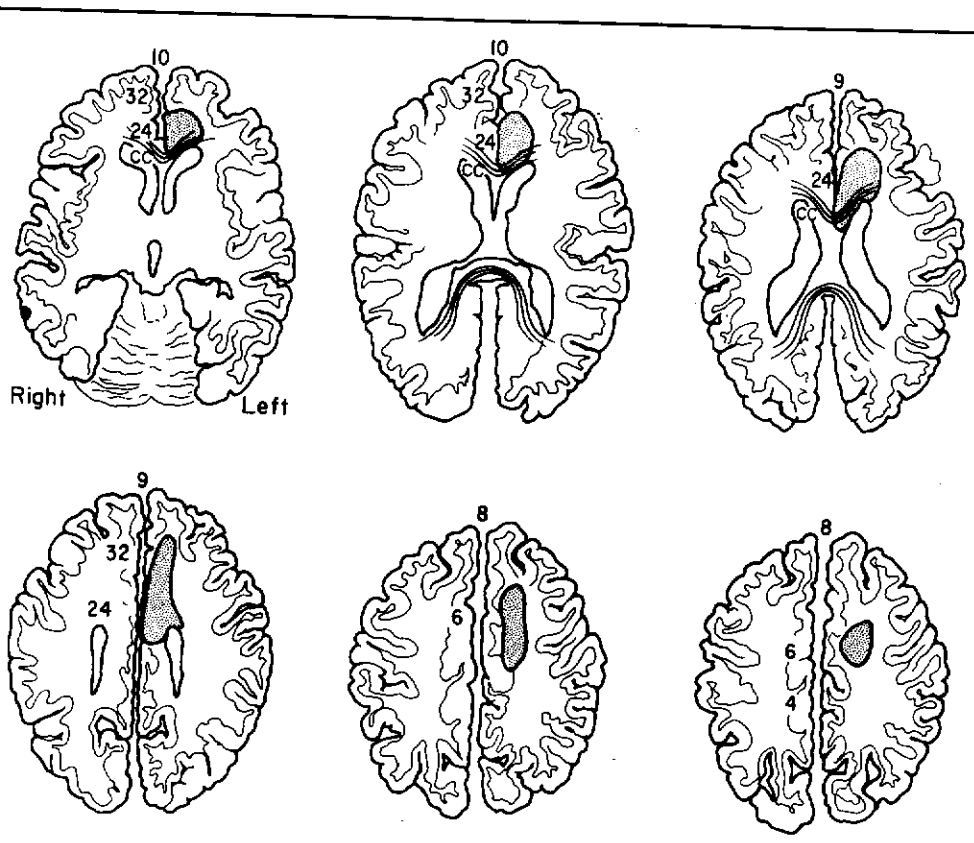

Figure. Template of horizontal sections of brain of case-report patient with dominant AHS. Templates were adapted from neuroanatomic atlases.[22,23] Speckled regions represent areas of infarction. Numbers refer to Brodmann's areas. CC = corpus callosum.

prior to the patient actually intending them. The left hand did not show this tendency.

Praxis of the right hand was normal for pantomime, imitation, and object use. There was a severe ideomotor apraxia of the left hand to command, which improved (but not to normal) with imitation and use of actual objects. Tasks requiring bimanual cooperation such as tying shoes, buttoning shirts, and cutting with a knife and fork were normal unless interrupted by sudden groping movements of the right hand. Abnormal movements were rare during bimanual tasks and were much more common when the left hand was performing a unimanual task or when the patient was not engaged in any specific task.

Tactile naming of objects placed in the left hand was normal, as was cross-copying of hand gestures passively imposed on either hand with eyes closed.

Methods. In addition to the above patient, we analyzed 20 reported cases of AHS.[1,2,6-21] AHS was defined as unwilled and uncontrollable movements of an extremity not due to a movement disorder. Reports included in this analysis were those that provided adequate clinical detail to determine clinical subtype and sufficient neuropathologic detail to localize the responsible lesion.

The MRI of the present patient and published CTs or MRIs of reported cases were mapped onto templates adapted from neuroanatomic atlases.[22,23] Where brain images were not published, descriptions of the lesion were used to determine lesion locus.

The patients were divided into four groups: (1) unilateral left-hemisphere lesions, (2) unilateral right-hemisphere lesions, (3) bilateral-hemisphere lesions, and (4) lesions confined to the corpus callosum. Impulsive groping was defined as groping movements toward a stimulus based on the mere proximity of the stimulus which did not require tactile stimulation.[24] Compulsive manipulation of tools (CMT) was defined as unilateral involuntary handling and utilization of familiar objects.[7] Intermanual conflict was defined as simultaneous or alternating conflicting or independent action of the alien limb during voluntary unilateral actions of the normal limb or during bimanual tasks.[5] Stimulus responsiveness was determined by whether the abnormal actions were in response to visual or tactile stimuli. Restraining actions were the tendency of the normal limb to hold down or restrain in some manner the alien hand. We also noted the presence or absence of limb apraxia in either hand.

Results. The results of the anatomic analysis are reported in table 1. When unilateral lesions are considered, left-hemisphere lesions predominate and occurred in a 9:2 ratio when compared with right-hemisphere lesions. All left-hemisphere lesions were frontal and generally involved the medial premotor region including the supplementary motor area (SMA) as well as the anterior cingulum, medial prefrontal cortex, and anterior corpus callosum. These cases had AHS of the dominant hand and typically had reflexive grasping, impulsive groping, and often had CMT.

Patients with lesions confined to the corpus callosum, in contrast, had AHS of the nondominant limb and clinically demonstrated primarily intermanual conflict. Patients with bilateral frontal lesions also had callosal lesions, and most did not demonstrate reflexive grasping or other features

Table 1. Cases of alien hand syndrome

	Left hemisphere	Corpus callosum	Right hemisphere	Alien hand	Handedness	GR	IGR	CMT	IC
Unilateral LH lesions									
Present case	A Cing, MPF, CR MPM (partial)	GCC, BCC		R	R	+	+	+	+
Goldberg et al[6]									
Case 1	MF	GCC		R	R	+	+	—	+
Case 2	A Cing, MPF, MPM	GCC		R	R	+	+	—	—
Mori and Yamadori[7]	A Cing, MPF, MPM	GCC		R	R	+	+	+	—
Notoya et al[10]	A Cing, MF	GCC		R	NR	—	+	+	—
NcNabb et al[13]	MF	GCC, BCC		R	R	+	+	—	+
Motomura et al[12]	A Cing, MPF, MPM	GCC		R	R	+	+	+	—
Stuss and Benson[14]	A Cing, MPF, MPM, P Cing	GCC, BCC, SCC		R	R	+	+	—	+
Magnani et al[21]	A Cing, MPF, MPM	GCC, BCC		R	NR	+	+	—	+
Unilateral RH lesions									
Goldstein[1,2]		GCC, BCC	A Cing, P Cing, IP	L	NR	—	—	—	+
Levine and Rinn[20]		SCC	Temporo-occipital thalamic	L	R	—	+	—	—
Bilateral lesions									
Tanaka et al[11]	A Cing, MPF	GCC, BCC	A Cing, MPF	L	R	—	—	—	+
Goldenberg et al[16]	A Cing, MPF	GCC, BCC	A Cing, MPF	L	R	+	—	—	+
Banks et al[15]									
Case 1	MF	GCC	MF	L	R	—	+	—	—
Case 2	MPF	GCC, BCC	MPF	L	R	—	—	—	+
Mukai et al[18]	MPF	GCC	MPF	L	R	—	—	—	+
Only CC lesions									
Beukelman[8]		GCC		L	R	—	—	—	+
Watson and Heilman[9]		GCC, BCC		L	R	—	—	—	+
Toshiya et al[17]		GCC		L	R	—	—	—	+
Leiguarda et al[19]									
Case 1		GCC, BCC		L	R	—	—	—	+
Case 3		GCC, BCC, SCC		L	R	—	—	—	+

GR Grasp reflex.
IGR Impulsive groping.
CMT Compulsive manipulation of tools.
IC Intermanual conflict.
CC Corpus callosum.
A Cing Anterior cingulate.
MPF Medial prefrontal.
CR Corona radiata.
MPM Medial premotor cortex including medial area 6 (supplementary motor area).
MF Medial frontal, otherwise unspecified.
P Cing Posterior cingulate.
GCC Genu corpus callosum.
BCC Body corpus callosum.
SCC Splenium corpus callosum.
IP Inferior parietal.
+ Present.
— Absent or not reported.
NR Not reported.

typical of frontal AHS and clinically resembled the group with lesions confined to the corpus callosum. Patients with unilateral right-hemisphere involvement had callosal lesions as well, which tended to be more posteriorly located when compared with left-hemisphere patients. The clinical characteristics of these patients also tended to resemble the callosal type of AHS.

Discussion. Review of previous cases indicates that the AHS should be divided into two clinically and anatomically distinct subtypes, the characteristics of which are summarized in table 2. Different mechanisms may account for these subtypes. Several explanations have been offered for the occurrence of grasping, groping, and related behaviors after frontal lesions. Denny-Brown described compulsive tactile exploration after frontal lobe ablation in the monkey[25] and humans[26] and used the term "magnetic apraxia"[26] to describe this behavior. He suggested that cingulate gyrus, area 8, and SMA were responsible for avoidance responses, and damage to these regions resulted in an exaggeration of positive exploratory behaviors subserved by the parietal lobe.[25,26] His localization of the frontal lesions which produced compulsive tactile exploration in the monkey is in accord with findings in human AHS. A similar explanation has been offered to account for bilateral utilization behavior (compulsive grasping and use of common objects) due to frontal lobe lesions in the human.[27]

The SMA is the portion of Brodmann's area 6

Table 2. Characteristics of the two alien hand syndromes

	Frontal type	Callosal type
Hand involved	Dominant[6,7,10,12-14]	Nondominant[8,9,17,19]
Associated grasp reflex	Common[6,7,12-14]	Rare[16]
Lesion site	Left mediofrontal and callosal[6,7,10,12-14,21]	Callosal with bilateral frontal[11,15,16,18] or without frontal[8,9,17,19] damage
Frequency of movements	Frequent, compulsive[6,7,10,12-14]	Occasional
Stimulus responsiveness	To visual or tactile stimulation[6,7,10,12-14]	Activated by action of dominant hand[11]
Intermanual conflict	Occasional[6,13,14]	Frequent, independent,[11,15,17] or oppositional[8,9,11,15,16,19]
Restraining actions	Common[6,7,10,13,14]	Rare
Limb apraxia	Occasional in either hand[13,15]	Commonly apraxia of the involved (left) limb[8,9,11,15-17,19]
Compulsive manipulation of tools	Common[7,10,12]	Never

located on the mesial surface of the frontal lobe.[28] The SMA is involved in the control of motor planning and initiation[29-33] as well as motor inhibition.[29,34,35] In addition to direct projections to primary motor cortex (MI),[36,37] SMA has extensive cortical[36,38] and subcortical[39-41] connections, particularly to areas subserving motor control[40] and is reciprocally connected with contralateral SMA and MI.[33,36,37,42] Lesions of SMA are known to cause contralateral grasping in monkeys[43] and humans.[44,45]

Goldberg et al[6] and Goldberg[33] suggested that damage to the SMA is the critical lesion in frontal AHS. Based on phylogenic and anatomic considerations,[33,41,46-48] some have suggested a dual system of control of primary motor cortex (MI), consisting of a medial premotor area (SMA) and a lateral premotor area, termed the "arcuate premotor area" (APA). The medial system is internally dependent, predictive, and projectional, whereas the lateral system is externally contingent and object oriented.[33,49] According to Goldberg,[33] a unilateral lesion in SMA and anterior corpus callosum could release ipsilateral MI from any medial or contralateral premotor inhibition, disrupting the balance between the SMA and APA systems, resulting in excessive environmentally driven manual exploration and AHS.

None of the above accounts explain the predominance of left-hemisphere lesions in frontal AHS, nor do they explain the suggested greater frequency of the contralateral grasp reflex after left frontal lesions.[50] One possible explanation is that forced grasping, AHS, and utilization behavior represent positive exploratory behaviors[25-27] in which the left hemisphere has a leading role.[51,52] These behaviors could be preferentially released by a left r
frontal lesion through what Denny-Brown[25]
"transcortical release."

All of the frontal AHS cases had anterior ca
involvement, and it has been shown that left
al frontal damage without callosal damage do
result in AHS.[7] This suggests that transco
inhibition from the right frontal lobe upon left
must be disturbed for frontal AHS to emerge
finding that right,[52] frequently retrorolan
lesions more commonly cause an ipsilateral in
tive grasp reaction than left-hemisphere le
suggests that the emergence of these ref
requires a release from right hemispheric inh
ry mechanisms mediated in part through tra
losal mechanisms. Verfaellie and Heilman[35] r
ed one case of contralateral manual disinhi
(though not AHS as described here) in a pa
with a right medial frontal lesion. This disi
tion was not present in a patient with a left m
frontal lesion. They suggested that bilateral
inhibition was initiated from the right but ne
left SMA, which exerted only unilateral contro

If the sole mechanism of dominant-limb
were a disruption of right-hemisphere inhib
we would expect dominant-limb AHS after ca
lesions alone and bilateral AHS after right m
frontal lesions, which does not occur. It is ther
likely that frontal AHS of the dominant lir
caused by a greater tendency for dominant
spheric reflexive exploration mediated via th
hemisphere, which is released from an asymm
cally distributed, predominant right-hemisp
inhibition of the left hemisphere.[52] There ma
an additional explanation for our failure to
contralateral frontal AHS after right fro
lesions. Since contralateral limb akinesia is g
est after lesions involving the right hemisph
there is the possibility that left limb akinesia
vented the appearance of left frontal AHS
right frontal lesions.

The occurrence of nondominant AHS in cal
cases suggests a different mechanism from
offered to explain frontal AHS. The finding
callosal cases were nonsurgical and had no
frontal retraction, and many had no right fro
lesions, suggests this subtype does not req
right frontal dysfunction for its occurrence.
callosotomized monkeys, conflicting bima
responses have been reported,[55,56] and there is
to distinguish these behaviors from those obse
in callosal AHS in humans. In human call
AHS, however, the abnormal movements o
exclusively in the nondominant limb. This sugg
a failure of medial frontal transcallosal inhibi
of the nondominant hemisphere during ta
requiring dominant-hemisphere motor contro
verbal mediation. The almost universal presen
callosal apraxia[57,58] of the nondominant limi
these cases (table 2) supports the interpreta
that hemispheric disconnection underlies call
AHS.

Finally, some authors have reported involuntary movements described as "alien hand" phenomena in association with cortical basal ganglionic degeneration.[59,60] These movements are most often described as repetitive, rhythmic, and spasmodic, with a tendency to levitate and assume unusual postures, and associated with dystonia and myoclonus. Although these involuntary movements may be similar to those observed in association with frontal or callosal lesions, we have excluded these cases from this study on the basis of this association with other movement disorders. However, the possible relationship between these entities deserves further investigation.

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