Journal of Neurology, Neurosurgery, and Psychiatry 1986;49:451–454

Short report

Pantomime agnosia

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SUMMARY Visual agnosia is impaired visual recognition not explained by defective visual acuity, visual fields, visual attention, or general mental ability. Two nonapraxic patients with lesions in the left occipital lobe could imitate pantomimes they could not recognise. Although both patients had a hemianopia, sparing of gesture imitation shows that no visual defect accounts for their inability to recognise pantomimes. Both patients were amnestic and alexic but had no general impairment of cognitive ability to account for a pantomime-recognition disorder. These patients seem to have agnosia for pantomime. From the computed tomograms from these patients, we propose that inferior visual association cortex is critical for gesture comprehension, whereas superior portions of these structures are critical for imitating or performing pantomime to an object presented visually.

Liepmann and Maas[1] suggested that the left hemisphere contains movement formulae that bilaterally program selection, timing, and spatial relationships of skilled motor movements. Heilman[2] suggested that these engrams are stored in the inferior left parietal lobe. Conceivably, brain lesions may destroy these engrams, spare but disconnect them from primary sensory/sensory association areas, or disconnect them from primary motor/motor association areas. Based on this schema a variety of behavioural abnormalities may be predicted reflecting impairment of different components of the gestural function system.

Our research group[3] proposed that the movement formulae programs skilled motor movements in much the same way as Wernicke's area programs linguistic acts. These movement formulae not only mediate skilled motor movement but also have a role in discriminating features of gestural movements that are distinctive and therefore crucial to gestural reception and production. As in aphasia caused by destruction of Wernicke's area, input processing as well as production could be disrupted by destruction of the movement formulae. In contrast, patients whose movement formulae engrams are preserved but disconnected from motor areas, although apraxic, could perform a gestural discrimination task to differentiate between well-performed and poorly performed motor acts. Our findings from studying gesture discrimination in patients with discrete cerebral lesions confirm that the inferior parietal lobule is crucial for discriminating and comprehending motor acts.[3]

Dejerine postulated that the angular gyrus contained a centre critical for comprehending visual/verbal material[4] and described a man who could write but not read.[5] He postulated that damage to visual areas or visual pathways or both could disconnect the angular gyrus from visual input. The intact angular gyrus would allow the patient to write, but because the visual areas were disconnected from the angular gyrus, the patient could not read. Lesions that induce alexia without agraphia are usually in posterior portions of the left hemisphere, including inferior aspects of the left occipital and temporal lobe, splenium of the corpus callosum or forceps major.[6] More anterior and deep lesions may disconnect both right and left visual areas from language areas.[7] Similar lesions may disconnect visual areas from the movement formulae also stored in the inferior parietal lobe of the left hemisphere. A visuomotor dissociation may induce visuomotor apraxia in which patients cannot pantomime the use of objects they see or imitate pantomimes (but can perform to command), or may induce agnosia of pantomime, or both. We examined two patients who had alexia with-

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Received 29 January 1985 and in revised form 28 July 1985.
Accepted 3 August 1985

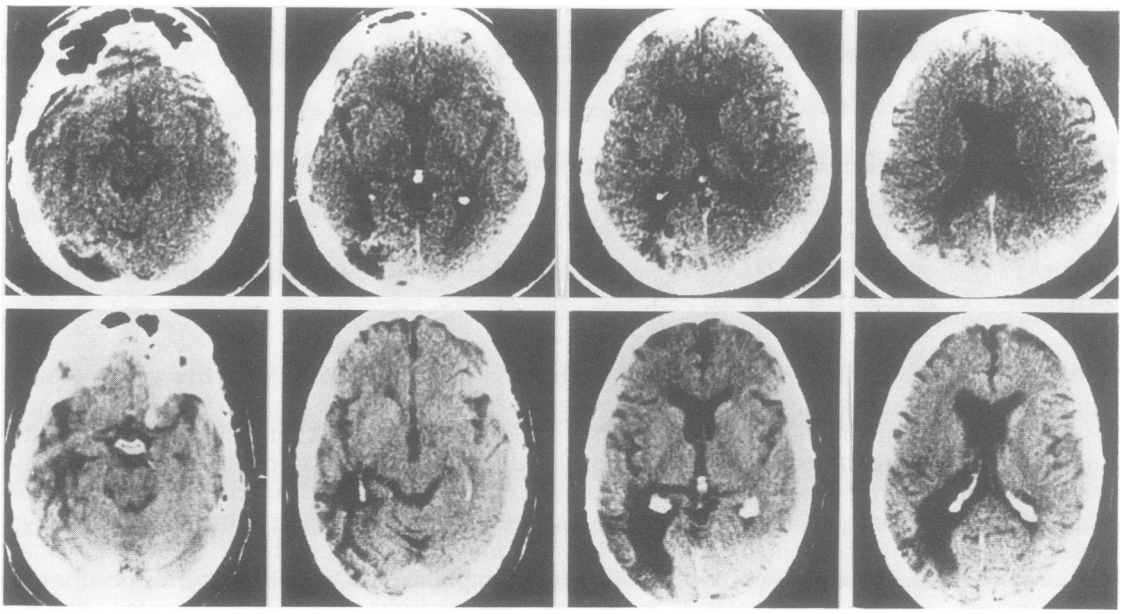

Fig *CT scan. Top four views are from patient 1 and bottom four are from patient 2.*

out agraphia from posterior left hemisphere lesions, to learn whether they would show visuomotor apraxia or agnosia for pantomime.

Methods

Subjects

Patient 1, a 65-year-old right-handed man, 38 years before this admission, had an intracranial haemorrhage probably secondary to left occipital and posterior temporal arteriovenous malformation (AVM). We admitted him for evaluation of increasingly severe headaches accompanied by blurred vision. CT without contrast (fig) disclosed an abnormal region consistent with AVM in the left occipital region immediately posterior to the occipital horn of the left lateral ventricle. The patient was cooperative and oriented. Digit span was seven forward and three backward. He could not recall any of three objects after being distracted for 5 minutes. He had no colour anomia, right-left confusion, finger agnosia, acalculia, apraxia, neglect, astereognosis, or agraphaesthesia.

The clinical impression of normal language function was confirmed by his performance on the Western Aphasia Battery (WAB).[8] The patient had a profound defect preventing his appreciation of complex visual arrays, but he could match simple stimuli. Appreciation of line drawings was much worse than that of 3-dimensional objects; object-naming to palpation was normal. He also had alexia without agraphia, with reading responses characteristic of spelling dyslexia. Neurologic evaluation showed a right homonymous hemianopia with corrected vision of 20/40 and 20/200. Mild weakness and clumsiness on the right was noted.

Patient 2, a 68-year-old right-handed man, was admitted to the hospital because of increasing irritability and emotional lability. Six years earlier he had an intracerebral haemorrhage and underwent emergency posterior temporal craniotomy with evacuation of a large intracerebral and intraventricular haematoma. Postoperatively, he had mild fluent aphasia with "decreased auditory comprehension, and occasional jargon," as well as alexia with agraphia. All deficits resolved quickly and completely except for significant alexia and a confrontation naming disorder. CT at 3 and 6 years after onset disclosed "ventricular asymmetry with dilatation of the left lateral ventricle mainly in the trigone and occipital polar areas." Encephalomacia was noted in the watershed area between the left middle and posterior cerebral arteries (fig).

He was cooperative and appropriate throughout testing and was awake, alert, and oriented to person, place, and time. He could name only one of four recent Presidents and after 5 minutes could remember only one of three objects. Digit span was seven forward and four backward. No difficulties were noted in calculations, right/left orientation, colour naming, stereognosis, graphaesthesia, mapping, constructions, or finger gnosis; nor was any neglect noted. Language during spontaneous conversations, as well as formal testing (WAB[8]), was normal except for confrontation naming. Performance on the Boston Naming Test[9] was severely impaired (+15 of 60 possible). Of his error responses 60% indicated that he recognised the picture ("takes you one flight" for escalator). At no time did his performance improve with semantic cueing, but phonemic cueing increased accuracy 47% of the time. It was evident that the disturbance was anomia rather that agnosia. The patient had right homonymous hemianopia and moderate-to-severe bilateral sensory/neural hearing loss. The remainder of his findings were normal.

Table

Tasks	*Correct responses (%)*		
	Patient No		*Control subjects*
	1	*2*	
1 Pantomime to verbal command	93*	80*	100
2 Pantomime function of shown picture	0	100	100
3 Gesture imitation	87	92	100
4 Name gesture performed by examiner	63	63	6
5 Gesture discrimination	68	58	100
6 Gesture discrimination	63	69	90
7 Gesture comprehension	50	70	98

*These scores are within the nonapraxic range for normative data reported previously.[10]

Special testing

Both patients received special tests of ability to process gestural information presented visually. These tasks were also presented to 36 men (ages, 40 to 80 years; mean, 60 years) with no history of neurological disease. The tasks included pantomime to verbal command (15 items), pantomime to picture command (20 items), gesture imitation (74 items), and gesture naming (30 items). In another task of gestural discrimination (32 items), the subject was to choose from among three alternatives which gesture best depicted the use of a target object. The foils were either movement foils (for example screwdriver for key) or limb orientation foils (for example lasso for stirring). The control subjects performed flawlessly on all tasks except the gesture-naming task in which the group mean was 95·7% accuracy (table).

During the pantomime to verbal command task, neither patient displayed ideomotor apraxia or verbal-motor disconnection apraxia with either hand.[11] To test for visuomotor disconnection apraxia,[12] we gave the pantomime-to-picture-command task. Patient 2 performed flawlessly; patient 1, because of his previously described difficulty in processing pictures, could not perform a single pantomime when shown pictures. He did, however, accurately pantomime the use of 14 of 18 objects he was shown but did not hold.

Both patients imitated gestures reasonably well (task 3). Additionally, these patients performed normally on pantomiming-to-verbal-command (task 1). In contrast, they performed poorly when they were visually presented these same gestures and were asked to label them (task 4) or discriminate them from visually similar gestures (task 5).

Further testing included paradigms used in previous studies to assess gesture discrimination and comprehension (table). In the discrimination task,[3] they watched a videotape and were to choose among three acts which one best depicted a particular target gesture. In 16 trials the foils were clumsily produced aberrations of the target act. In the second video study[10] they were presented 20 trials of an actress performing pantomimes and were to indicate the act being performed. In both these video tasks the patients performed significantly lower than the lowest normal.

Discussion

These two nonapraxic patients with lesions in the dominant occipital lobe had difficulty in understanding pantomimes. Visual agnosia has been defined as impaired visual recognition not explained by a defect in visual acuity, visual fields, visual attention, or general mental ability. Both patients could imitate pantomimes they could not recognise. Therefore, although they both had hemianopia, this or other visual defects could not account for their inability to recognise pantomimes. Although the patients were amnestic and alexic, they did not have a general impairment of mental ability that could account for this defect. Additionally, because this defect was present not only in gesture naming but also when the response involved only discrimination among gestural alternatives, anomia could not be the mechanism. Agnosia for gesture (pantomimes) therefore seems to be the disorder.

One patient had difficulty recognising pictures of objects (suggesting a partial agnosia), but could recognise actual objects; the other patient, although anomic, appeared free of agnosia. Therefore, although visual agnosia may coexist with a gestural agnosia, they seem to be dissociable phenomena.

Finkelnburg[13] and Head[14] attributed impaired gesture and pantomime recognition to a generalised symbolic defect, asymbolia. Duffy and Pearson[15] noted that inability to recognise pantomime was often associated with aphasia and correlated with naming ability, aural comprehension, and general linguistic competence. Like Finkelnburg[13] and Head,[14] Duffy and Pearson[15] also attributed inability to recognise pantomime to an impaired central symbolic activity. Although one of our patients had anomic aphasia, the other was not aphasic, which indicates that their inability to recognise pantomime could not be attributed to asymbolia in general or again to anomia particularly. In additon, Heilman *et al*[3] and Varney[16] described patients who had severe language disorders but could recognise pantomimes. Thus, although aphasia and impaired pantomime recognition may correlate, these phenomena are dissociable.

Varney[17] studied aphasic patients and noted that disorders of pantomime recognition always occurred with reading defects (alexia) but that reading defects were not always associated with disordered pantomime recognition. Lesions of the dominant inferior parietal lobe, like those of the occipital lobe reported here, induce not only alexia,[4 5 18] but also defective comprehension of pantomime.[3] However, unlike occipital lobe lesions, those in the parietal lobe are associated with production defects such as agraphia and apraxia. Unfortunately, Varney[17] did not test for these disorders nor specify the loci of the lesions in his patients.

DeRenzi *et al*[11] requested 150 patients with left hemisphere disease to imitate movements and demonstrate the use of visually displayed objects. These patients were also verbally asked to pantomime object use. DeRenzi *et al* found seven patients whose performance was worse in the visual than in the verbal modality. Although we first suspected that our patients would have difficulty performing pantomimes to visually presented objects and have difficulty imitating gestures, they performed these tasks well. The lesions in our patients involved the inferior portions of the visual association areas (that is Brodmann areas 18, 19, 37). Unfortunately, DeRenzi *et al* did not test for gesture comprehension and did not describe the anatomical lesions in their patients with visuomotor disconnection apraxia. The inferior visual association cortex is critical for gesture comprehension, but the superior portions of visual association cortex (Brodmann areas 18, 19) may be critical for imitating a pantomime or performing a pantomime to a visually presented object.

Although our patients had alexia without agraphia and an associated disorder of pantomime recognition, not all patients with alexia and agraphia have a disturbance of pantomime recognition. For example, we recently examined a patient who had alexia without agraphia from a left occipital lobe lesion; his performance on the discrimination and comprehension tasks was flawless. His lesion also involved the inferior visual association areas (that is, inferior portions of Brodmann areas 18, 19, 37). We cannot be certain that minor anatomic differences between lesions in these patients account for behavioural differences. However, differences in premorbid brain organisation may be responsible.

We are grateful to Dr Russell Bauer for information he provided on the visual object agnosia displayed by patient 1. This work was supported in part by Grant NS-20204 from the National Institutes of Health. The study was presented in part at the annual meeting of the International Neuropsychological Society, San Diego, California, USA, February, 1985.

References

[1] Liepmann H, Maas O. Fall von linksseitiger Agraphie und Apraxie bei rechsseitiger Lähmung. *Z Psychol Neurol* 1907;**10**:214–27.
[2] Heilman KM. Apraxia. In: Heilman KM, Valenstein E, eds. *Clinical Neuropsychology*. New York: Oxford University Press, 1979:159–85.
[3] Heilman KM, Rothi LJ, Valenstein E. Two forms of ideomotor apraxia. *Neurology (NY)* 1982;**32**:342–7.
[4] Dejerine J. Sur un cas de cécité verbal avec agraphie suivi d'autopsie. *Mem Soc Biol* 1891;**3**:197–201.
[5] Dejerine J. Contribution à l'étude anatomo-pathologique et clinique des differentes variétés de cécité verbale. *Mem Soc Biol* 1892;**4**:61–90.
[6] Damasio AR, Damasio H. The anatomic basis of pure alexia. *Neurology (Cleveland)* 1983;**33**:1573–83.
[7] Greenblatt S. Subangular alexia without agraphia or hemianopia. *Brain Lang* 1976;**3**:229–45.
[8] Kertesz A. *Western Aphasia Battery*. New York: Grune & Stratton, 1982.
[9] Kaplan E, Goodglass H, Weintraub S. *Boston Naming Test*. Philadelphia: Lea & Febiger, 1983.
[10] Heilman KM. Ideational apraxia—a redefinition. *Brain* 1973;**96**:861–4.
[11] DeRenzi E, Faglioni P, Sorgato P. Modality-specific and supramodal mechanisms of apraxia. *Brain* 1982;**105**:301–12.
[12] Rothi LJG, Heilman KM, Watson RT. Pantomime comprehension and ideomotor apraxia. *J Neurol Neurosurg Psychiatry* 1985;**48**:207–10.
[13] Duffy RJ, Liles BZ. A translation of Finkelnburg's (1870) lecture on aphasia as "asymbolia" with commentary. *J Speech Hear Disord* 1979;**44**:156–68.
[14] Head H. *Aphasia and Kindred Disorders*. London: Cambridge University Press, 1926.
[15] Duffy J, Pearson K. Pantomime in aphasic patients. *J Speech Hear Res* 1975;**18**:115–32.
[16] Varney NR. Pantomime recognition defect in aphasia: implications for the concept of asymbolia. *Brain Lang* 1982;**15**:32–9.
[17] Varney NR. Linguistic correlates of pantomime recognition in aphasic patients. *J Neurol Neurosurg Psychiatry* 1978;**41**:564–8.
[18] Albert ML. Alexia. In: Heilman KM, Valenstein E, eds. *Clinical Neuropsychology*. New York: Oxford University Press, 1979:59–91.