



Asymmetric Catalepsy After Right Hemisphere Stroke

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Summary: We describe the appearance of left hemineglect and striking cataleptic posturing, more prominent in left-sided extremities, in a patient without psychiatric illness. Neuroimaging demonstrated a large posterior right hemisphere infarct involving the parietal, occipital, and temporal lobes, the insula, and caudate. Additional movement abnormalities that comprise the full catatonia syndrome were absent, including stereotypy, mannerisms, ambivalence, automatic obedience, mutism, negativism, and echopraxia. Catatonia has been reported to be produced by lesions of diverse etiology affecting the frontal lobe, limbic system, diencephalon, or basal ganglia. In these cases, catalepsy has been manifest only rarely, and motor signs that are present are generally bilateral. This case demonstrates that asymmetric catalepsy can be produced by right hemisphere stroke, and provides partial support for earlier clinical literature relating catalepsy and the parietal lobe. **Key Words:** Catalepsy—Waxy flexibility—Catatonia—Neglect—Parietal lobe.

Catalepsy denotes a tendency to maintain postures induced by the examiner (1,2). Of the varied manifestations of the catatonic syndrome, catalepsy is perhaps the most specific, and certainly the most dramatic. *Catatonia* labels a cluster of psychomotor symptoms and signs that may appear with idiopathic psychiatric disturbances, drug reactions, metabolic derangements, and localized structural lesions of the CNS (3,4). The classic features of catatonia include mutism, stupor, catalepsy, negativism (including gegenhalten), mannerisms, stereotypy, bizarre postures, echolalia, echopraxia, automatic obedience, and catatonic excitement (furor) (2,4). Among the idiopathic psychiatric disease states, catatonia is more commonly associated with affective disorder than with schizophrenia. Catalepsy is present in 20–30% of patients who exhibit catatonia in association with an idiopathic psychiatric disturbance (5).

We describe a patient without psychiatric illness who developed striking cataleptic posturing, more prominent in left-sided extremities, after right hemisphere stroke.

CASE REPORT

A 75-year-old, right-handed man presented with complaints of clumsiness of the left hand and becoming lost in his house. There was no history of psychiatric disease or exposure to neuroleptic agents. The patient did not have known cardiac disease, nor major risk factors for atherothrombotic stroke.

General medical examination results were unremarkable.

Mental status examination disclosed severe left hemispatial neglect, constructional impairment, spatial dysgraphia, difficulty with comprehension and repetition of prosody, dressing difficulty, occasional hand-for-object substitutions bilaterally during execution of learned motor tasks, and inappropriate cheerfulness.

Elemental neurological examination demonstrated right gaze preference, and initial left hom-

A videotape segment accompanies this article.

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onymous hemianopia, left hemihypoesthesia (dense to pin and temperature, moderate to light touch), minimally impaired left-sided vibration and joint sense, and symmetric reflexes with flexor plantar responses. There was minimal weakness of finger, wrist, and elbow extension on the left, mild hypomimesis and some paucity of spontaneous movement, and no tremor. On testing for pronator drift, the left arm moved upward minimally. Resting tone was not elevated; tone increased with activation more on the left than the right, without cogwheeling. The left hand was held in an abnormal posture at rest, with flexion at the wrist. Dysmetria of finger-nose movements of the left hand was noted, improving when the patient visualized the hand in the right hemispace. Gait was slightly wide based, with a tendency to circle toward the right; the left hand maintained a dystonic posture during walking. No grasp or avoiding reaction was noted.

Cataleptic posturing was noted, prominently in the left upper extremity, to a lesser degree in the right upper extremity and left lower extremity, sparing the right lower extremity (Fig. 1). Without prompting, the patient maintained postures produced by examiners' positioning, even when uncomfortable, for up to 5 min. In addition, after completing a requested motion (e.g., holding arms up for pronator drift testing) or a self-generated motion (e.g., pointing), the patient would often maintain the last posture of his movement for several minutes. The limbs would immediately release from these sustained postures on further command from



FIG. 1. Photograph of patient maintaining postures induced by the examiner during free conversation. Both upper extremities and the left lower extremity show perseveration of posture. This phenomenon could not be elicited from the right lower extremity.

the examiner or spontaneously if the patient employed the extremity for gesturation during free conversation. When the posturing behavior of his limbs was pointed out to him, he would state he had not noticed they were in unusual positions. He would then place the limbs in repose, but shortly thereafter posturing would resume.

Waxy flexibility persisted with only slight diminution during the 7-day hospital course. Trials of carbidopa/levodopa (Sinemet) and bromocriptine produced no change in symptoms. At a follow-up visit 2 weeks after onset, the patient's family reported that they had observed unusual posturing at home, especially of the left arm. For example, when the patient rested from raking leaves, he was noted to maintain the left arm in an unusual elevated posture for an extended period, thrusting the rake into the air. The family stated that the patient had never exhibited such behaviors before his stroke. At follow-up 1 year after onset, catalepsy could no longer be elicited, although a tendency to maintain the left hand in a flexed, dystonic posture was noted.

Acute cranial computed tomography scan and follow-up cranial magnetic resonance scan obtained 6 days after symptom onset demonstrated infarction in the posterior territories of the right middle cerebral artery (Fig. 2). Template analysis (6) disclosed that the lesion primarily involved the right angular and supramarginal gyri; the superior, middle, and inferior temporal gyri; the posterior insula; and the anterolateral occipital lobe (Fig. 3). A small infarct in the body of the right caudate was also noted. An electroencephalogram on hospital day 2 demonstrated continuous delta range slowing over the right hemisphere, without epileptiform activity.

Etiologic workup failed to determine the source of stroke.

DISCUSSION

There are more than 25 case reports in the literature of catatonia associated with focal brain injuries (2,4,7). Lesion sites most commonly are frontal, temporolimbic, or subcortical (diencephalon, basal ganglia, or brainstem), and are bilateral. Catalepsy is distinctly uncommon among these patients, and the catatonic motor signs that are present are generally symmetric. Three aspects of our patient's presentation are distinctive: (a) the appearance of catalepsy isolated from other signs of the catatonia syndrome, (b) the asymmetry of cat-

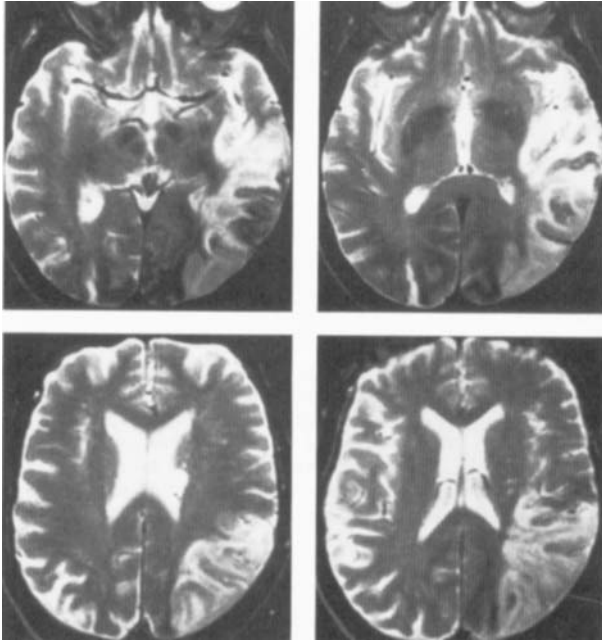


FIG. 2. Representative axial images from T2 signal-weighted magnetic resonance imaging scan (relaxation time 2,500 ms, echo time 80 ms), demonstrating infarction in the posterior territories of the right middle cerebral artery. (Right side of images is right side of brain.)

aleptic posturing, and (c) the association with a well-documented unilateral parietotemporal injury.

Our patient did not exhibit the mental state changes usually associated with catatonia, of either the "excited" or "retarded" type. He also failed to display classic motoric manifestations of catatonia other than maintenance of posture. We suggest that his condition represents an instance of catalepsy *sine* catatonia. Catalepsy appearing in isolation in this manner needs to be distinguished from a number of other pathologic motor phenomena.

The "perseveration of posture" of catalepsy differs from customary forms of motor perseveration. The "inappropriate intrusion of a previous action or response in ongoing activity" is the hallmark of classic perseveration (8), often associated with frontal systems injuries (9). Although several subtypes of perseverative behavior have been recognized (10,11), all are characterized by the recurrent performance of an action, as opposed to the persistence of posture after completion of an action or after passive positioning seen in catalepsy. Cataleptic behavior also contrasts with akinetic-rigid syndromes of extrapyramidal origin and hypokinetic-hypometric syndromes seen after frontal and right

hemisphere injury (12). In these, the preponderant difficulties are with the initiation, speed, and gain of movements. Difficulty with returning to rest after completion of movement, if present, does not predominate.

The sustained persistence of posture seen in catalepsy may be viewed as a phenomenologic opposite of "motor imperistence," the inability to sustain postures most commonly associated with frontal and/or right hemisphere lesions (13). Cataleptic posturing with a parietal insult should also be distinguished from the "parietal hand" phenomenon. The incoordination and dysmetria that can be associated with isolated parietal lesions has been reported to sometimes be accompanied by dystonic posturing of the hand (14), but not persistence of induced postures of multiple limbs.

One notable feature of our patient's catalepsy was its asymmetry, with perseveration of posture more prominent in left-sided extremities, contralateral to the cerebral lesion. In reviewing the literature, we have not discovered previous reports of strongly asymmetric catalepsy, with the exception of a 19th-century account of the experimental induction of one-sided perseveration of posture in a hypnotic subject (15).

Catatonic persistence of attitude has been associated with parietal lesions in rare case reports, but generally without confirmation of localization by modern neuroimaging techniques. Critchley, in his monograph on the parietal lobe (16), listed among features suggestive of parietal involvement in cases of hemiparesis "a tendency towards the adoption of catatonic-like postures by the affected upper limb." However, no report of particular cases accompanied this general observation. Denny-Brown and Chambers, in their survey of parietal lobe function (17), discussed cataleptic persistence of attitude as an extreme manifestation of the "avoiding reaction"—a withdrawal response seen after parietal lesions analogous to the grasping reaction seen with injuries to frontal cortices. They described briefly one patient, a 59-year-old woman with a history of presumed right parietal injury since the age of 15 years and a sudden new stroke producing additional clinical signs of left parietal damage, whose limbs "remained for many minutes in any posture passively applied to them." No supportive neuroimaging or postmortem findings were available to confirm the clinical localization. Of note, Denny-Brown and Chambers also reported experiments in monkeys in which cataleptic posturing was pro-

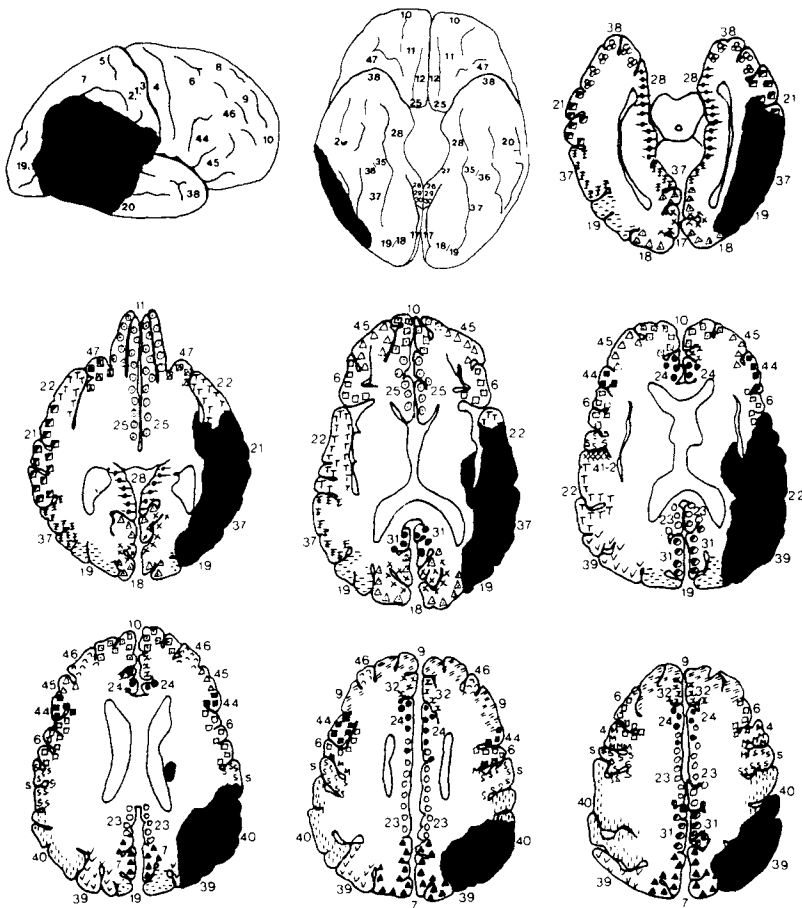


FIG. 3. Template analysis of lesion based on T1, proton density, and T2-weighted magnetic resonance imaging sequences. The lesion primarily involved the angular and supramarginal gyri, the superior, middle, and inferior temporal gyri, the posterior insula, the anterolateral occipital lobe, and the body of the right caudate.

duced by surgical excision of parietal cortices. One recent case with modern neuroimaging is also supportive of a link between parietal damage and persistence of posture: a full-blown catatonic syndrome, including catalepsy, was observed in association with bilateral parietal infarction (18).

Caution must be exercised in considering anatomic and physiologic bases for the cataleptic defect in our patient. The wide extent of his hemispheric infarction precludes precise anatomical localization. Moreover, postinfarction catalepsy is rare, and middle cerebral artery infarcts are common, suggesting that an unusual combination of lesions and mechanisms, perhaps both extrapyramidal and cortical, are required to generate cataleptic behavior. The collection of additional cases will help to clarify these issues.

Nonetheless, several possibly complementary mechanisms for the cataleptic defect in our patient may be conjectured. One possibility is that our patient's persistence of posture was the result of an interaction of parietal neglect with hypokinesia of

extrapyramidal origin. The literature on the modifying effects of supervening vascular lesions on the manifestations of idiopathic Parkinson's disease and other akinetic-rigid syndromes is incomplete. Pyramidal lesions are well known to cause disappearance or diminution of tremor during the period of most severe weakness. Parkinson himself described the first such case (19). Infarctions of the ventrolateral thalamus have acted as "vascular thalamotomies" in parkinsonian patients, relieving tremor and rigidity contralaterally (20). Similarly, cerebellar infarction (21) and anterior choroidal artery occlusion (22) have been noted to abolish extrapyramidal rigidity and tremor. However, parietotemporal infarcts that spare primary motor systems have not been systematically investigated in this regard. Our patient's asymmetric catalepsy may have been the collective product of a mild extrapyramidal hypokinetic syndrome modified by severe asymmetric neglect from a posterior parietal infarction.

The entity of cortical-basal ganglionic degenera-

tion (CBGD) is often associated with asymmetric limb rigidity and apraxia, in some instances resembling cataleptic posturing (23,24). Our patient exhibited a small lesion in the body of the right caudate. His ischemic cortical and subcortical injuries may have compromised the same functional systems impaired by idiopathic degeneration in CBGD. Against this potential mechanism, our patient did not demonstrate the alien limb phenomenon, focal myoclonus, postural-action tremor, and severe dystonia that patients with CBGD frequently develop, and his isolated caudate injury spared many of the subcortical sites typically affected in CBGD.

Another candidate mechanism recognizes that frontal system lesions are frequently associated with elements of the catatonic syndrome. Our patient's lesions in the posterior insula, inferior parietal lobule, and a portion of primary somatosensory cortex may have interrupted input from posterior to anterior regions to a degree severe enough to produce functional deafferentation of frontal cortices. Similarly, it may be noted that our patient's lesions involved posterolateral temporal cortices to a significant degree, and that temporal lesions have been associated with the catatonic syndrome in several cases. However, these patients generally differed from ours in having bilateral rather than unilateral lesions and in having prominent involvement of mesial-limbic temporal structures.

Studies of motor system physiology have been primarily directed at analyzing functional elements responsible for the planning, initiation, implementation, and ongoing feedback control of motor programs (25,26). Systems for recognizing the accurate completion of a motor program and initiating a return to resting posture have not been well described. This case suggests that an important element of such a neural system may be localized in parietotemporal cortices.

LEGENDS TO VIDEOTAPE

Segment 1. Moderate waxy flexibility while conversing with examiners.

Segment 2. Persistence of postures after pronator drift testing, more sustained on the left.

Segment 3. Praxis testing with persistence of end-task postures on the left.

Segment 4. Interview with patient regarding posturing.

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