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Gait Apraxia: Further Clues to Localization

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Key Words
Gait apraxia · Supplementary motor area · Gait ignition failure · Primary progressive freezing gait

Abstract
Background/Aims: Gait apraxia characterized primarily by gait ignition failure has been linked to lesions involving the dorsomedial frontal lobes, but the precise locus within this general region has not been determined. It has previously been hypothesized by Thompson and Marsden that disease, disconnection, or dysfunction of supplementary motor area (SMA) may account for the similarities in the gait disorders observed in Binswanger's disease, hydrocephalus, frontal lobe lesions, and Parkinson's disease. We reevaluate this hypothesis. ***Methods:*** Clinical description and MRI of 2 subjects with gait apraxia characterized primarily by gait ignition failure. ***Results:*** Both subjects had incapacitating gait disorders characterized by particular difficulty with initiating gait and making turns. Both had MRI-demonstrated lesions of the SMA region, parasagittal convexity premotor cortex, or subjacent white matter bilaterally, one due to primary CNS lymphoma, one due to a lobar atrophy. ***Conclusions:*** In both these cases, the lesions were substantially more limited and focal than any reported heretofore in the literature on gait apraxia or freezing of gait. The clinicopathologic correlation in these cases provides partial support for the Thompson and Marsden hypothesis, but also may implicate parasagittal convexity premotor cortex in the genesis of gait apraxia.

Gait apraxia can be defined as loss of ability to properly use the lower limbs in the act of walking that cannot be attributed to deficits in elementary sensory, motor, or cerebellar function, or psychiatric disease [1, 2]. We suggest, after Denny-Brown [3], that the sine qua non of gait apraxia is gait ignition failure: particular difficulty in initiating gait and making turns, often with a tendency to freeze, with relative preservation of straight-line gait once initiated. Gait apraxia has been linked to bilateral lesions of the medial frontal lobes [1, 3, 4], but more precise localization within this general region has not been possible. We report 2 cases that may provide further clues to the location of the necessary and sufficient lesion for gait apraxia to occur.

Case Reports

Case 1

A 76-year-old man presented with a 10-month history of insidiously progressive imbalance. He had been wheelchair-bound for 1 week. He had experienced several falls. He complained of difficulty knowing where his legs were. There was no history of dizziness, urinary urgency, or incontinence. His wife conceded some loss of spontaneity over the last few weeks. He had enjoyed good health all his life. There was no history of hypertension, diabetes, or more than occasional minimal alcohol consumption.

On examination, he was somewhat taciturn. He recalled none of 3 objects after several minutes of distraction. On tests of prefrontal function, he produced 6 words beginning with the letter F in 1 min, but he had no difficulty performing contrasting pro-

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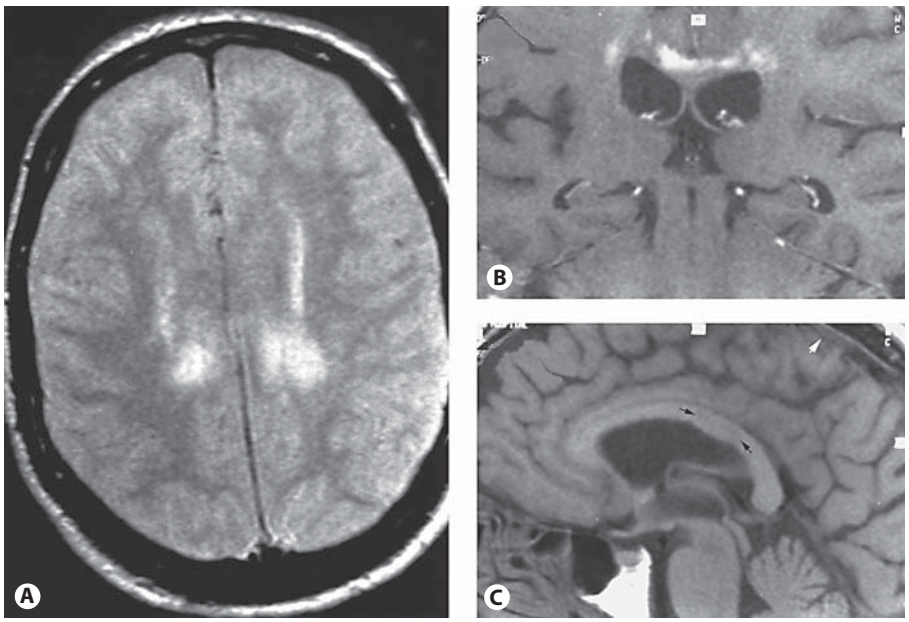

Fig. 1. Case 1. **A** Axial T_2-weighted MRI. **B** Coronal T_1-weighted MRI after gadolinium administration. **C** Sagittal T_1-weighted MRI. Black arrowheads delineate margins of lesion in corpus callosum. The white arrowhead indicates approximate location of precentral sulcus.

grams. Language, limb praxis, visuospatial function, and affect were normal and he exhibited a warm personality. Cranial nerves II–XII were intact but for the presence of a mild visual grasp. He lost two lines of visual acuity on a pocket Snellen chart in association with sinusoidal head movement (suggesting mild impairment in the vestibulo-ocular reflex). Strength was normal but for a trace of hip flexor weakness. There was mild bilateral paratonia. There was no upper extremity drift during sustention and fine motor movements of the hands were normal. He exhibited a pronounced tendency to retropulsion in the sitting position. Finger-to-nose maneuver was performed normally with eyes open or closed. Heel-to-shin maneuver was executed normally. Gait was moderately wide based and he had a great deal of difficulty initiating walking and making turns. On turns, his stride, already mildly shortened, became very short, and he tended to retropulse. Even while walking straight ahead he exhibited considerable instability and preferred to keep both hands on the examiner to maintain his balance. There was a severe apraxia of sitting. Vibratory sensation was slightly reduced in the great toes bilaterally. Deep tendon reflexes were 2–3 and symmetric and plantar responses were equivocal bilaterally.

An MRI scan (fig. 1) demonstrated a small, contrast-enhancing lesion of the corpus callosum extending up along the midline into the parasagittal white matter bilaterally, immediately below posterior portions of the superior frontal gyri corresponding to supplementary motor area (SMA). Tissue obtained on brain biopsy was consistent with primary CNS lymphoma (B-cell type).

Case 2

A 71-year-old man with a 2-year history of chronic lymphocytic leukemia presented with an 8-month history of insidiously progressive difficulty with gait and balance. He also reported occasional slurring of his speech, particularly when reading aloud, and progressive decline in the quality of his handwriting. His leukemia was under satisfactory control on a regimen of chlorambucil, fludarabine, and prednisone. He had a history of coronary disease and mild hypertension. He had never consumed more than minimal amounts of alcoholic beverages.

On neurologic examination, higher neural functions (including upper extremity praxis) were intact but for the production of only 8 words in 1 min on a letter fluency task (letter F). Cranial nerves II–XII were intact. He lost one line of visual acuity on a pocket Snellen chart in association with sinusoidal head movement. He exhibited a slight tendency to slurring of consonants. There was moderate reduction of vibratory sensation in the great toes bilaterally with preservation of position sense. Graphesthesia was normal in the feet. Motor examination was completely normal but for mild atrophy of the extensor digitorum brevis bilaterally. The patient was able to tap his feet quite well. Finger-to-nose and heel-to-shin maneuvers were performed normally. Gait was characterized by slight widening of the base, mild reduction in stride, and severe difficulty in initiating walking and making turns. There was a slight tendency to retropulsion and a mild sitting apraxia. Deep tendon reflexes were 1–2 and symmetric. Plantar responses were equivocal bilaterally. An MRI scan demonstrated focal atrophy in the parasagittal region bilaterally involving the precentral gyri and posterior portions of the superior frontal gyri (fig. 2).

Discussion

In both cases, the pathology, by its very nature, was likely to extend beyond that exhibited on the MRI scans. In both cases there was also some evidence of clinical pathology affecting other regions of the nervous system: case 1 exhibited impairment in declarative memory acquisition, and both exhibited mild impairment in ves-

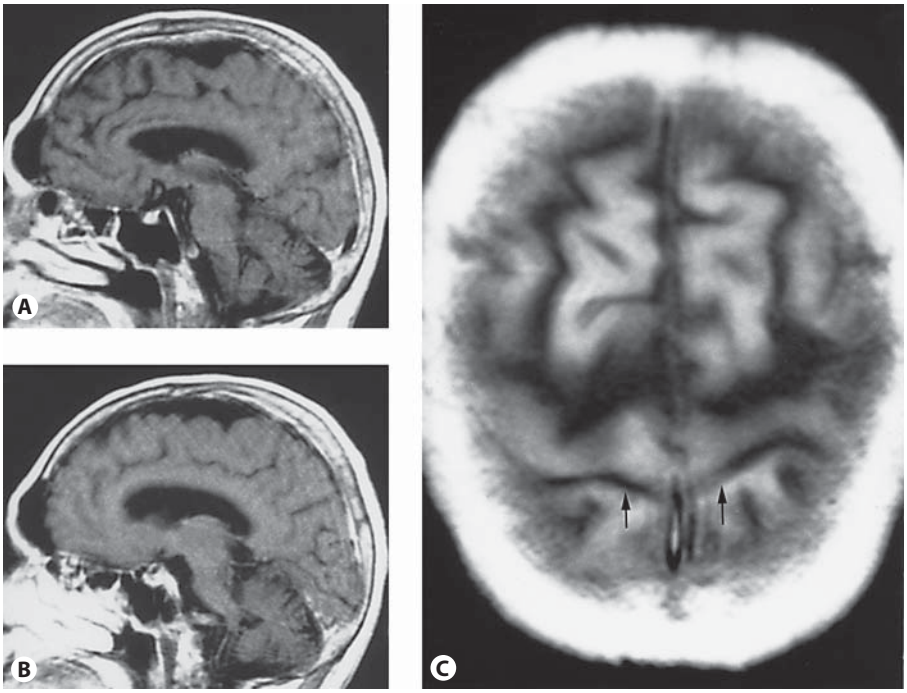

Fig. 2. Case 2. **A**, **B** Right and left parasagittal T_1-weighted images. **C** Axial T_1-weighted image; arrows indicate central sulcus.

tibular function and of vibratory sensation in the great toes. Nevertheless, the gait disorder in both cases, characterized by particularly severe difficulty with initiation and turns, was far more severe than any of the other neurological abnormalities, and in both cases the lesions were substantially more limited and focal than any reported heretofore in the literature on gait apraxia or freezing of gait. Specifically, these 2 cases suggest that gait apraxia characterized predominantly by freezing of gait is produced not just by lesions of the dorsomedial frontal cortex, as has been previously suggested [1, 3, 4], but by lesions involving the SMA region or immediately subjacent white matter. This conclusion finds support in recent functional imaging studies during walking employing near-infrared spectroscopic topography [5]. In addition, our emerging understanding of the function of cortical regions anterior to SMA (pre-SMA and supragenual anterior cingulate cortex) suggests that they are involved in the more general process of gating plans for action, rather than such anatomically specific plans as might be involved in walking [see review in ref. 6] – further evidence that it is not dysfunction of midline frontal cortex in general that produces gait apraxia, but only dysfunction of the most posterior portions of this cortex. The concept that disconnection of SMA (as in case 1) may be as potent as destruction of SMA itself finds further support in the common clinical finding of gait apraxia with purely subcortical disease, e.g., communicating hydrocephalus.

Our conclusion that these cases implicate SMA in gait apraxia should not be construed as a statement that SMA is solely responsible for the functions that are impaired in gait apraxia. We suggest that SMA is only one network in a complex of neural networks involved in gait. Most obviously these include spinal cord pattern generators involved in the reciprocal oscillating movements of the legs, brainstem networks required to maintain adequate antigravity tone and make automatic corrections to assure balance, midline cerebellar networks, and portions of the basal ganglia involved in lower extremity function. The particular role of the cerebral cortex in gait can be inferred from the deficits observed with cerebral lesions: the initiation of walking, changing walking speed and direction, and adaptation of walking pattern to irregularities in the environment. A large body of research subsuming a number of fields [e.g., ref. 7] suggests that there is a division of labor within the frontal cortex, such that midline regions are involved in endogenously driven plans, while convexity regions are involved in the adaptation of these plans to environmental contingencies (supported by the extensive connectivity of these regions with posterior sensory association cortices). In the cases discussed here, the relevant convexity regions are parasagittal portions of premotor cortex, which could also be im-

plicated in the lesions. Thus, the gait apraxia in these 2 cases might reflect involvement or disconnection of frontal regions involved in endogenously driven walking plans (SMA), frontal regions involved in adaptation of walking to environmental features that require a change in walking pattern (parasagittal convexity premotor cortex), or both.

The MRI in case 2 suggests a focal atrophy. Focal atrophies are known to be caused by several degenerative disorders [8, 9]. A comparable focal atrophy (probably due to corticobasal degeneration), centered on area 7, has been reported; in that case, gait was preserved, but the patient exhibited an apparent deficit in aligning her own body coordinate system with the environmental coordinate system, producing incapacitating apraxia of sitting and lying, as well as difficulty with finger-to-nose maneuver with eyes closed [10]. The presentation of case 2 is consistent with the syndrome of primary progressive freezing gait (PPFG), which has been shown to be caused by several disorders, including pallidonigroluysian degeneration, diffuse Lewy body disease, progressive supranuclear palsy, and corticobasal degeneration, the latter two diseases also implicated in focal atrophies [11]. The locus of pathology in PPFG most responsible for the gait disorder is uncertain: anatomic imaging studies have demonstrated only minimal nonspecific abnormalities [12] and functional imaging studies have been similarly unrevealing [13]. However, Rossor et al. [4] have reported gait apraxia with freezing of gait in association with the histology of corticobasal degeneration most severe in the dorsomedial frontal cortex.

In both our cases, the MRIs implicate posterior portions of the superior frontal gyri, corresponding to SMA but also parasagittal convexity premotor cortex, in the pathogenesis of gait apraxia and suggest that gait apraxia can occur without more extensive involvement of the superior frontal gyri. To what extent this gait apraxia might reflect dysfunction of SMA, of adjacent parasagittal convexity premotor cortex, or of deafferented brainstem and spinal regions involved in gait cannot be determined from these cases. The potential contribution of other pathology that might have been present in these 2 cases (e.g., the disorders associated with PPFG), albeit not well defined, is also uncertain.

Thompson and Marsden [14] remarked that the similarity of many of the features of the gait disorder seen in Binswanger's disease, hydrocephalus, and frontal lobe lesions to that seen in Parkinson's disease suggests that similar mechanisms may be involved in all of these conditions. They further suggested that the common mechanism may be involvement of the SMA, directly or via disconnection in Binswanger's disease, hydrocephalus and frontal lobe lesions, or functionally in Parkinson's disease. The lesion location in our 2 cases supports this hypothesis but adds the possibility that involvement of parasagittal convexity premotor cortex might be important.

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