

Novel Representation of Astasia Associated With Posterior Cingulate Infarction

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Background and Purpose—The representation elicited in the cingulate motor area has been demonstrated in animals, but remains unclear in humans. In particular, the representation and pathogenic mechanisms of the posterior cingulate cortex are poorly understood, especially in humans. We describe a case of posterior cingulate infarction associated with contralateral astasia.

Case Description—A 67-year-old right-handed man with a 10-year history of hypertension suddenly presented with right-sided pulsion on attempting to stand or sit. On the following day, he could not maintain a sitting position. The patient immediately fell to the floor because of instability, characterized by marked right-sided pulsion despite no muscle weakness, sensorial deficits, or cerebellar ataxia. Magnetic resonance imaging of the brain showed abnormal intensity in the posterior parts of the cingulate, with no other clinically significant lesions.

Conclusions—Because the cingulate motor area is connected to the vestibulocerebellar system through the thalamic nuclei, disruption of this connection by posterior cingulate infarction may result in astasia. (*Stroke*. 2006;37:e3-e5.)

Keywords: astasia ■ behavioral neurology ■ cingulate gyrus ■ infarction ■ stroke

Several cortical motor areas have been identified on the medial wall of the hemispheres of the brain. These include a group of premotor regions located in the cingulate sulcus, collectively termed the cingulate motor area (CMA). Cytoarchitectural studies have established that the CMA has 2 separate motor representation areas: the rostral CMA (area 24c) and the caudal CMA, encompassing both the dorsal CMA (CMA_d, area 6c) and the ventral CMA (CMA_v, area 23c). Each area has direct projections to the spinal cord and connections with the premotor cortex and primary motor cortex.¹ Physiological studies indicate that anatomical connections of the CMA with the motor system are functionally linked to limb-movement generation. The representation elicited in the CMA has been demonstrated in animals but remains unclear in humans. Damaged to the anterior cingulate cortex in humans is associated with reduction in spontaneous, compulsive, and emotional motor responses. Transient akinesia and mutism have been observed in large bilateral anterior cingulate cortex lesions with slight encroachment on adjacent cortical regions, such as the supplementary motor area.² However, the representation and pathogenic mechanisms of the posterior cingulate cortex are poorly understood, especially in humans. We describe a case of posterior cingulate infarction associated with contralateral astasia and discuss underlying neurological mechanisms.

Case Reports

A 67-year-old right-handed man with a 10-year history of hypertension suddenly noticed repetitive scrubbed move-

ments of the fingers of the right hand, lasting for >15 minutes. He subsequently presented with right-sided pulsion on attempting to stand. On the following day, right-sided pulsion occurred while sitting, as well as while standing. Finally, he could not maintain a sitting position and was admitted to the hospital. He never lost consciousness or had any sensory complaints. On admission, the blood pressure was 162/104 mm Hg, and the pulse rate was 106 beats/minute and regular. He was alert and fully oriented, with no impairment of higher functions, including auditory comprehension, orientation, cognition, and mentation. He could sit up only by pulling himself up with both hands while holding onto the side rails of his bed; however, on attempting to sit by releasing his hands, the patient immediately fell to the floor because of instability, characterized by marked right-sided pulsion. The muscle strength of the trunk and extremities was not decreased. There was no ataxia on either side. The deep tendon reflexes were normal, with no pathological reflexes. The muscle tone of the extremities was normal. Cranial nerves, superficial sensation, joint position senses, and vibratory senses were not impaired. Paper-and-pencil tests of extrapersonal neglect (line bisection and figure copy) were normal. The results of routine laboratory blood examinations were normal. Anti-DNA, antinuclear, antiphospholipid, and anticardiolipin antibodies and the results of lupus anticoagulant testing were negative. On day 2, fluid-attenuated inversion recovery image and diffusion-weighted and T₂-weighted

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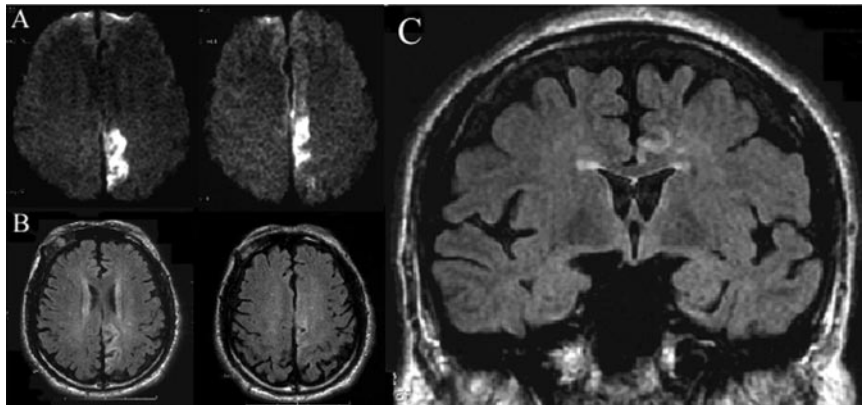
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Diffusion-weighted (A) and FLAIR magnetic resonance images (B) of the brain, showing high intensity in posterior parts of the cingulate. (C) FLAIR image showing high intensity in only the cingulate gyrus with no other clinically significant lesions.

MRI of the brain showed high intensity in the posterior parts of the cingulate (Figure). There were no other abnormalities in the cerebrum, brain stem, or cerebellum. An electroencephalogram, transcranial Doppler ultrasonogram, and echocardiogram were all normal. The 24-Holter ECG showed no abnormal arrhythmia. The transesophageal echocardiography showed spontaneous echo contrast within the left atrium. The patient was given anticoagulant therapy and regained stability. One week later, he became able to sit, but he was unable to maintain a standing position because of right-sided pulsion. Three weeks later, he was able to walk, although his gait was slightly unstable. His steps were characterized by an irregular amplitude and direction, sometimes directed laterally and then suddenly stopping while attempting to take a right step, leading to pulsion. The gait disturbance differed from cerebellar ataxia, because the gait was not broad based or lurching. He could arise from a squat and maintain a closed stance. The results of the other neurological examinations were unremarkable, including a negative Romberg's sign, finger-to-nose test, heel-knee test, and shin-tapping test. The patient regained more stability and became able to sit and to walk alone. After 1 month, he was discharged, with no residual signs or symptoms.

Discussion

Our patient had posterior cingulate infarctions associated with marked truncal instability, characterized by contralateral pulsion in the absence of other clinically significant lesions in the premotor cortex, primary motor cortex, thalamus, brain stem, cerebellum, and corpus callosum. Stimulation of the cingulate is known to elicit bilateral and contralateral complex movements of the extremities accompanied by tonic postures, deviation of the head and eyes, vocalization, and automatic changes. Stimulation studies in humans have demonstrated stiffening of the wrist, supination of the forearm and hand, and mild flexion in the fingers.³ However, the representation as contralateral pulsion has not been confirmed in either humans or animals. We consider this manifestation an astasia, because our patients could not maintain a stationary sitting or standing position and had no other conditions that could have accounted for pulsion, such as pyramidal or sensorial deficits, including abnormal proprioceptive sensation, cerebellar ataxia, kinaesthesia, psychosis, or parkinsonism. To our knowledge, this is the first time to report an

association in humans between astasia and cingulate infarctions.

Astasia can be caused by lesions of the thalamic ventrolateral region, resulting in "thalamic astasia," or of the pons or callosal region.⁴⁻⁶ Festigial fibers of the vestibulocerebellar pathway project to the medial ventrolateral nucleus of the thalamus, and disruption of this pathway may cause thalamic astasia.⁷ Moreover, the nucleus ventroposterior lateralis and the nucleus ventrolateralis of the thalamus as determined by vestibular stimulation in animals are termed "vestibular thalamic nuclei."⁸ The CMA_d and CMA_v also receive a major thalamic input from the ventrolateral nucleus of the thalamus.⁹ The CMA_d and CMA_v may be connected to the vestibulocerebellar system through the thalamic nuclei. Disruption of this connection by posterior cingulate infarction, including the caudal CMA, may have been responsible for the astasia in our patient.

However, the CMA_d receives the major component of its thalamic input from the nucleus ventralis lateralis pars oralis, which receives inputs primarily from the caudate putamen, as well as the supplementary motor area.¹⁰ Supplementary motor area seizures are associated with severe gait disturbances characterized by markedly impaired postural reflexes.¹¹ Moreover, the CMA_v receives a substantial component of its input from the mediodorsal and intralaminar nuclei and ventralis lateralis pars caudalis, which receive input primarily from deep cerebellar nuclei.¹² Ictal single-photon emission-computed tomography in patients with nocturnal frontal epilepsy has demonstrated increased blood flow in the cingulate gyrus and cerebellar cortex, suggesting a direct connection between these regions.¹³ Disruption of these connections may also have partially contributed to the patient's astasia.

In conclusion, astasia is a novel representation elicited in the posterior CMA in humans by the disruption of the connection between CMA and the vestibulocerebellar system via thalamic nuclei.

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