

Clinical/Scientific Notes

Vomiting out of proportion to dizziness in ischemic brainstem strokes

C.M. Fisher, MD

At the onset of ischemic strokes involving the lower brainstem, vomiting may be prominent when dizziness and other neurologic abnormalities are minimal or absent. Errors in interpretation may result in the misdiagnosis of a gastrointestinal disorder. The stroke literature does not clearly refer to this clinical point. The present note draws attention to the phenomenon both for practical diagnostic reasons and to illustrate a distinct form of vestibular vomiting.

Case 1. A 52-year-old hypertensive, diabetic man with a history of myocardial infarction awakened at 1 AM with nausea and vomiting. He vomited hourly until arrival at the Emergency Department at 8 AM. He had no history of dizziness, headache, abdominal pain, or diarrhea. On examination the man was alert and afebrile; his abdomen felt normal; and his neurologic status was intact except for peripheral neuropathy. His blood pressure was 220/118 mm Hg. Blood chemistry test results were normal. Nifedipine (sublingual), promethazine, and hydralazine (intravenous) were administered. Acute gastroenteritis was suspected and the man was admitted to the Medical Service.

On day 1 further vomiting occurred and the man requested medication for headache once. By 10 PM, 12 hours after admission, his blood pressure fell to 110/60 mm Hg; he became diaphoretic and reported that, momentarily, the door appeared to slant sideways and his wife, standing near the bed, looked like she was upside down. Examination within 15 minutes showed no new abnormality. Vision was chronically impaired by diabetic retinopathy treated with laser and the fundi could not be seen. The vomitus was coffee-ground and the differential diagnoses included gastrointestinal bleeding. There had been no dizziness. A CT scan of the head was normal. On day 2 nausea continued with occasional vomiting. On returning to bed after voiding, the patient was dizzy briefly, for the first time, and "the world tilted like it did last night." His blood pressure, treated vigorously, was 135/70 mm Hg. On days 3 and 4 nausea continued with occasional vomiting and, for the second time, medication was requested for headache. Hypertensive encephalopathy was considered. On day 5 hoarseness and dysphagia appeared abruptly, and in the following 12 hours a complete lateral medullary syndrome developed. The man's restlessness precluded satisfactory MRI scanning. Respiratory complications followed and cardiac arrest occurred 12 hours later. An autopsy was not performed. Thrombosis of the left vertebral artery was suspected.

Comment. In the incipient stage, intractable nausea and vomiting were quite out of proportion to dizziness. Headache, neither persistent nor severe, was ascribed to hypertension. Tilted vision and upside-down vision, although infrequently reported in the literature, would probably have led a neurologist to the right diagnosis.

Case 2. A 70-year-old hypertensive man, while traveling on a commercial airliner, vomited forcefully six times in 20 minutes. He sat with his eyes closed; he was pale, diaphoretic, and cold. On arrival after 2 1/2 hours he walked, with support, to a wheelchair. He disclaimed dizziness. His blood pressure was 165/85 mm Hg. Examination at the Emergency Department was normal and the man was admitted to the Medical Service with the diagnosis of gastroenteritis. Forty-eight hours later transient diplopia occurred and a neurologic consultant found a fine, rapid, horizontal nystagmus on gaze to the right, a slower, coarser nystagmus on gaze to the left; the nystagmus subsided after 24 hours. An MRI scan and vertebrobasilar angiogram were normal. Occlusion of a basilar artery penetrating branch was the presumed diagnosis.

Discussion. In labyrinthine (end organ) disorders, nausea and vomiting are generally proportionate to dizziness (vertigo). This relationship does not always hold for ischemic lesions in the brainstem vestibular system, as these two cases illustrate. With ischemic lesions the vestibular deficit may be less restricted to one side, thereby preventing the relative contralateral overactivity that underlies end organ dizziness, the sum total of the disturbed afferent impulses resulting in a pattern that stimulates the vom-

iting center with little or no vertigo.^{1,2} There is some parallel with the dynamic neural arrangement in motion sickness.

Upside-down vision might be interpreted somewhat analogously.^{1,3} Normally, objects are still seen as correctly oriented when the observer is in an upside-down position, indicating a complex compensatory brain process involving vestibular function. Brainstem ischemia must create an abnormal neural pattern that is comparable, and that is interpreted as the environment being visually upside down, since all other sensory information, somatosensory and proprioceptive, indicates that the body is not upside down.

From the Neurology Service, Massachusetts General Hospital, Boston, MA.

Received March 13, 1995. Accepted in final form April 27, 1995.

Address correspondence and reprint requests to Dr. C.M. Fisher, Neurology Service, Massachusetts General Hospital, Boston, MA 02114.

References

1. Young LR. Perception of the body in space: mechanisms. In: Brookhart JM, Mountcastle VB, Darian-Smith I, eds. Handbook of physiology, vol III, part 2. Bethesda, MD: American Physiological Society, 1984:1023-1066.
2. Goldberg JM, Fernandez C. The vestibular system. In: Brookhart JM, Mountcastle VB, Darian-Smith I, eds. Handbook of physiology, vol LLL, part 2. Bethesda, MD: American Physiological Society, 1984:977-1022.
3. Ropper AH. Illusion of tilting of the visual environment. *J Clin Neuroophthalmol* 1983;3:147-151.

Transient hemiballism/hemichorea due to an ipsilateral subthalamic nucleus infarction

S. Crozier, MD; S. Lehericy, MD, PhD; P. Verstichel, MD; C. Masson, MD; and M. Masson, MD

Hemiballism usually results from vascular lesions of the subthalamic nucleus (STN), its afferent or efferent pathways, or its projection areas (thalamus, striatum, and lenticular nucleus). Most often, the lesions are contralateral to the abnormal limb, but there may be ipsilateral lesions (review reference 1). Precise correlation between the ipsilateral lesion and the abnormal movements is uncertain, because the lesions are usually multiple and often bilateral. We report a case of transient hemiballism/hemichorea that occurred with an isolated ischemic lesion of the ipsilateral STN.

Case report. A 63-year-old man, without previous history of neurologic or systemic disease, was awakened on May 5, 1994, by a sudden onset of abnormal movements of the left side of the body. Involuntary movements involved the proximal aspect of the upper and lower limbs and were violent, irregular, and associated with flexion and rotation. There were also irregular choreic movements of the left hand. All these movements were exaggerated when the patient was speaking. Muscle tone was decreased in the affected limbs. General physical examination was normal except for elevated blood pressure (180/100 mm Hg).

Investigation revealed normal serum analysis, including lipids and coagulation factors. Antiphospholipid antibodies were negative. Cardiac echography, B-mode carotid and vertebral ultrasonography, and Holter study were also normal.

Chlorpromazine (50 mg/day), introduced the first day, resulted in great reduction of abnormal movements over the next 24 hours. Then, only rare movements of wide range persisted.

CT scan performed the first day was normal. Postcontrast MRI scan, 5 days after the onset of abnormal movements, showed a small enhancement in the left subthalamic area. Follow-up MRI examination, performed at 3 months, confirmed the presence of an isolated small infarction (45 mm³) within the left subthalamic area (figure). The stereotaxic coordinates of the center of the lesion (calculated using three-dimensional T1-weighted sections, 2 T, 1.7 mm thick) were as follows: 3 mm below the anterior commissure-posterior commissure (AC-PC) line, 2 mm behind the midline of the AC-PC line (25 mm length), and laterally 10 mm from the midline (width of the third ventricle, 3 mm). These coordinates corresponded to those of the subthalamic nucleus.²

Discussion. Because the previously reported cases of hemiballism, presumably caused by ipsilateral lesions, had multiple or bilateral lesions,¹ the temporal sequence of onset of abnormal



Figure. Postcontrast coronal T_1 -weighted MRI showing a hypointense area (infarction) in the left subthalamic nucleus area (arrow).

movements and appearance of the lesion was not established unequivocally.

Cases of ipsilateral hemiballism have also occurred after neurosurgical lesions of the thalamus in Parkinson's disease.¹ In these patients, the onset of abnormal movements was chronologically related to the production of the lesion in one or the other hemisphere, but all had previous contralateral stereotaxic thalamic lesions.

Hoogstraten et al³ reported a case of biballism due to a unilateral lesion; and to our knowledge, Moersch and Kernohan⁴ reported the only case of hemiballism related to an isolated ipsilateral lesion of the basal ganglia. This patient had severe left-sided hemiballism associated with ipsilateral STN infarction, without contralateral basal ganglia lesion, on histologic examination.

In the present case, neuroimaging studies showed that the abnormal movements were contemporary with the infarction, as the lesion initially enhanced after contrast administration and enhancement disappeared at 3 months. Stereotaxic analysis located the infarction within the ipsilateral STN and no other lesion was present.

The pathophysiology of hemiballism is still poorly understood. Even less understood is ipsilateral hemiballism. Based on their model of stereotaxic thalamic lesions in parkinsonian patients, Dierssen et al¹ have postulated that a previous contralateral lesion was necessary for the occurrence of hemiballism secondary to the ipsilateral lesion. In these patients, the ipsilateral lesion played a "triggering" role combined with the previous contralateral basal ganglia lesion, resulting in the expression of the abnormal movements. In our patient, the presence of a preexisting contralateral basal ganglia lesion, which was too small to detect on MRI scans, cannot be excluded. The possibility that the STN exerts a bilateral control of motor function may represent another explanation to the occurrence of ipsilateral hemiballism. This is supported by unilateral stereotaxic neurosurgery in Parkinson's disease frequently resulting in bilateral improvement of abnormal movements.⁵ Anatomic pathways related to this bilateral control are unknown. To our knowledge, there is no evidence of crossed subthalamopallidal projections or intersubthalamic connections.⁶ The pedunculopontine nucleus, however, which is involved in motor functions, is bilaterally connected to the STN.⁷ Whether these pathways play a role in the production of ipsilateral hemiballism remains to be determined.

Acknowledgment

We thank Professor J. Féger for his useful comments and suggestions.

From the Departments of Neurology (Drs. Crozier, Verstichel, C. Masson, and M. Masson) and Radiology (Dr. Lehericy), Hôpital Beaujon, Clichy, France.

Received February 16, 1995. Accepted in final form May 4, 1995.

Address correspondence and reprint requests to Dr. Verstichel, Service de Neurologie, Hôpital Beaujon, 92110 Clichy, France.

References

1. Dierssen G, Gioino GG, Cooper IS. Participation of ipsilateral hemisphere lesions in the pathology of hemichorea and hemiballismus. *Neurology* 1961;11:894-898.
2. Hassler R. In: Schaltenbrand G, Bailey P, eds. Introduction to stereotaxis with an atlas of the human brain, vol 2. Stuttgart, Germany: Thieme, 1977:plates 1-57.
3. Hoogstraten MC, Lakke JPWF, Zwartz MJ. Bilateral ballism: a rare syndrome: review of the literature and presentation of a case. *J Neurol* 1986;233:25-29.
4. Moersch FP, Kernohan JW. Hemiballismus—a clinicopathologic study. *Arch Neurol Psych* 1939;41:365-372.
5. Laitinen LV, Bergenheim AT, Hariz MI. Leskell's posteroventral pallidotomy in the treatment of Parkinson's disease. *J Neurosurg* 1992;76:53-61.
6. Carpenter MB, Carleton SC, Keller JT, Conte P. Connections of the subthalamic nucleus in the monkey. *Brain Res* 1981;224:1-29.
7. Lavoie B, Parent A. Pedunculopontine nucleus in the squirrel monkey: projections to the basal ganglia as revealed by anterograde tract-tracing methods. *J Comp Neurol* 1994;344:210-231.

Headaches after inadvertent lumbar puncture during bone marrow harvest

Frank Lieberman, MD, and Subhash Gulati, MD

Headaches in a cancer hospital are common and often require extensive diagnostic evaluation to exclude metastatic intracranial disease or infection. However, one does not expect to encounter headache in an individual with no other neurologic illness who donates bone marrow. We have seen two patients develop positional headaches, which were typical of low pressure headaches occurring after lumbar puncture, after bone marrow harvest. In both patients, the headaches resolved without specific therapy.

Case reports. Patient 1. A 44-year-old woman underwent elective bone marrow harvest to provide marrow for an HLA compatible sibling. She was morbidly obese. For several years, she suffered diffuse headache on awakening that would abate as she stood up and began her daily activities. The physician performing the bone marrow harvest noted the patient's obesity obscured the usual landmarks. When the surgical drapes were removed, several puncture marks were noted over the lateral sacrum. When the patient awoke from general anesthesia, she complained of severe headache accompanied by nausea and several episodes of vomiting. Sitting upright exacerbated the headache, which was relieved by lying supine. These new headaches were clearly different from the early morning headaches she had reported at the time of admission. Neurologic examination several hours after the headaches began demonstrated anisocoria with the right pupil being 3mm and the left 2.5mm. There was no papilledema. The cranial nerve examination was otherwise normal and the remainder of the neurologic exam was unremarkable. The headaches gradually abated over 5 days, and the anisocoria resolved.

Patient 2. A 50-year-old woman with stage IV diffuse T-cell lymphoma underwent bone marrow harvest to obtain stem cells for autologous bone marrow rescue after high-dose chemotherapy. Before the harvest, performed under general anesthesia, she had no neurologic complaints. The procedure was uneventful and the patient resumed normal activity. Severe band-like headache, which radiated into the nuchal region, began 48 hours later. The headache pain was worsened by sitting or standing and relieved by lying flat. The patient visited the emergency room after these headaches persisted unchanged for several days. Neurologic examination was unremarkable. The needle marks from the marrow aspiration were visible, including a puncture mark 3 cm left of the midline over the sacrum. A brain magnetic resonance imaging (MRI) scan, performed with and without gadolinium 10 days after the headaches began, was unremarkable. The headaches remitted after 2 weeks.

Discussion. Positional headache is a frequent complication of diagnostic lumbar puncture¹ and a well-recognized, although infrequent, complication of epidural anesthesia.² The headache improves when the patient lies flat and is exacerbated by sitting or standing. The headache is caused by low spinal fluid pressure due to leakage of CSF from the thecal sac. Symptoms and signs of cranial nerve dysfunction, including diplopia, tinnitus, facial numbness, and anisocoria, occur.³ MRIs of patients with low pressure headache sometimes demonstrate diffuse meningeal enhancement and downward brain displacement.⁴ Rarely, subdural

Neurology[®]

Transient hemiballism/hemichorea due to an ipsilateral subthalamic nucleus infarction

S. Crozier, S. Lehericy, P. Verstichel, et al.

Neurology 1996;46;267-268

DOI 10.1212/WNL.46.1.267-a

This information is current as of January 1, 1996

Updated Information & Services	including high resolution figures, can be found at: http://www.neurology.org/content/46/1/267.2.full.html
References	This article cites 6 articles, 1 of which you can access for free at: http://www.neurology.org/content/46/1/267.2.full.html##ref-list-1
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.neurology.org/misc/about.xhtml#permissions
Reprints	Information about ordering reprints can be found online: http://www.neurology.org/misc/addir.xhtml#reprintsus

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright Copyright 1996 by Advanstar Communications Inc.. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

