

tially devastating disorder as a cause for thunderclap headache, particularly when initial investigations seem unrevealing. In this particular case, hyponatremia was an early but subtle clue to this diagnosis. This case also underscores the value of MRI in detecting underlying causes for thunderclap headache, which may not be obvious on the initial CT or lumbar puncture.

From the Department of Neurology, Mayo Clinic, Rochester, MN.

Received August 8, 1997. Accepted in final form September 29, 1997.

Address correspondence and reprint requests to Dr. David W. Dodick, Department of Neurology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905.

Copyright © 1998 by the American Academy of Neurology

References

1. DeBuijn SFTM, Stam J, Kapelle LJ, for the CVST Study Group. Thunderclap headache as the first symptom of cerebral venous sinus thrombosis. *Lancet* 1997;348:1623-1625.
2. Slivka A, Philbrook B. Clinical and angiographic features of thunderclap headache. *Headache* 1995;35:1-6.
3. Wijdicks EFM, Kerkhoff H, Van Gijn J. Long-term follow-up of 71 patients with thunderclap headache mimicking subarachnoid hemorrhage. *Lancet* 1988;2:68-70.
4. Reid RL, Quigley ME, Yen SSC. Pituitary apoplexy: a review. *Arch Neurol* 1985;42:712-719.
5. Wakai S, Fukushima T, Teramoto A, et al. Pituitary apoplexy: its incidence and clinical significance. *J Neurosurg* 1981;55:187.
6. Mohr G, Hardy J. Hemorrhage necrosis and apoplexy in pituitary adenomas. *Surg Neurol* 1982;18:181.

Moriatic aphasia: A syndrome of acute carotid occlusion on the dominant side

F. Ghika-Schmid, MD; G. Assal, MD; J. Ghika, MD; and J. Bogousslavsky, MD

Case report. A 50-year-old, right-handed, French-speaking, Spanish worker was admitted 3 weeks after an acute right hemiparesis with "hyperproductive" global aphasia. He produced meaningless nonphonemic sounds ("brr," "prck," "pfft"), accompanied by an emotional display of unmotivated laughing, crying, childish behavior, and transient anger, lasting for hours, in a pseudo-hebephrenic manner. He also hummed "Gregorian chants." He could not follow simple commands, imitate gestures, read, or write. He showed utilization behavior, manipulation of objects, and an inappropriate tendency to attempt to undress the examiner, but no grasping or cephalic frontal reflexes.

On neurologic examination, severe right sensorimotor flaccid hemiparesis was found, with corticobulbar signs and left Horner's sign. On MRI, a subacute infarct was found in the deep territory of the left middle cerebral artery (MCA) and bilateral anterior cerebral arteries (ACAs) (figure), with hypoplasia of the A1 segment on the right. Doppler ultrasound of precerebral vessels was normal. On transthoracic echocardiography, atrial septal aneurysm was found, evocative of a possible cardioembolic source. ECG, chest radiograph, electrolytes, blood counts, and CSF were normal. He was treated with acetylsalicylic acid and clozapine and carbamazepine for seizures. His behavior improved over months, but the sensorimotor deficit remained unchanged.

On neuropsychological evaluation at 6 months he exhibited no anger but giggled playfully. He had right head tilt and poor visual scanning to the left, suggesting left spatial neglect. He did not communicate in an informative nonverbal way. Verbal expression was limited to "a-ge" and "brr," used profusely, without any attempt to communicate. He was unconcerned by his lack of communication. He could not name objects or repeat; comprehension was limited to simple orders. Writing was severely impaired. He oriented a text but demonstrated no reading or reading comprehension abilities. Automatic language was not preserved. He had severe buccolingual, ideomotor (on imitation), and constructional apraxia. He was unable to point to objects on verbal command, perform a word-object match, point to a nonsemantically related object among others, or designate Poppelreuter's embedded figures. He could orient a map but could not, when asked, point to cities. He failed matching emotional facial expressions of joy and sadness.¹

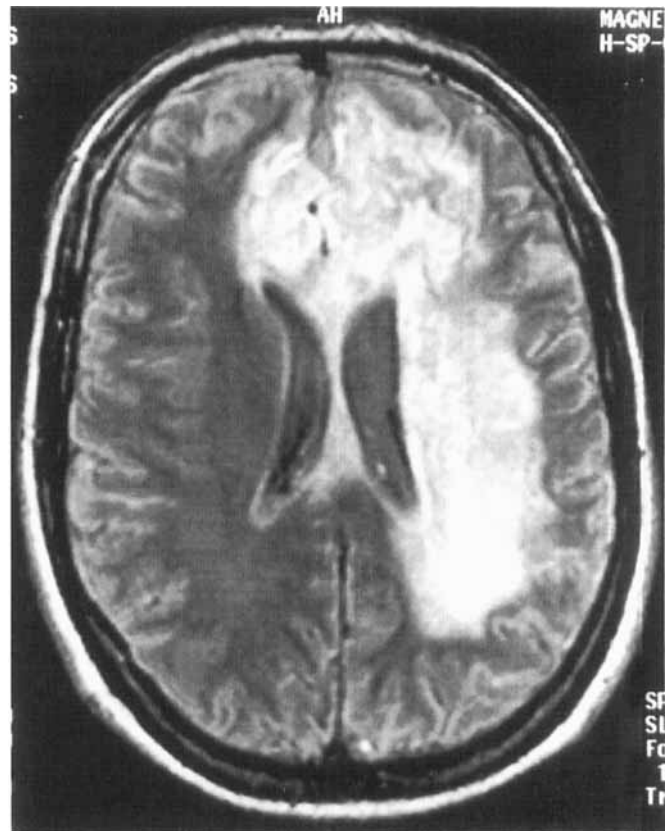


Figure. MRI transversal T2*-weighted sections showing subacute infarct in the territory of both ACAs and the left MCA.

Discussion. Our patient's atypical global aphasia, with massive hyperproduction of meaningless sounds, affective facial expressions, emotional lability, and childish and manipulative behavior shares features with moria (stupidity).² This new type of aphasia, which we termed "moriatic aphasia," has not been previously reported.

Inappropriate cheerfulness and lack of concern were predominant in our patient.² He most closely resembles the third patient of Jastrowitz² with a left frontal tumor, who was not aphasic but was agitated, jocular, and gave bizarre answers as if he did not understand. Our patient had comprehension impairment but also behaved in an unexpected way for an aphasic. He paid no attention to nonverbal cues from the examiner, consistently maintaining a cheerful manner. This may be the nonverbal counterpart of "Witzelsucht" (jocularity) seen in frontal lobe damage.³ The difficulty in examining moriatic patients, because of bizarre behavior, was documented in the initial description.² A tendency to sing, as reported with rude songs ("gemeine Lieder"),² was present in our patient's word-free production. His attempts to undress his examiners combines features of the sexual disinhibition of moria² with those of environmental dependency.⁴

Our patient's global aphasia can be called "emotional logorrhea," with abundant stereotyped onomatopoeias and expressive mimics. This recalls jargon aphasia of Wernicke's aphasia, in which patients, as was the case in our patient, are often unconcerned by their lack of ability to understand.⁵ This contrasts with Broca's aphasia, in which stereotypia such as "Tan-Tan" are used for communication.⁶ The simplification of verbal stereotypia to "a-ge" and "brr" over time is consistent with the gradual improvement observed with frontal lesions.³

Emotional lability may be part of his corticobulbar involvement, but lability was also described after anterior hemisphere strokes.⁷ Jastrowitz² also noted alternation of prostration and agitation.

The combination of moria, suggestive of orbito-mesial fron-

tal involvement,^{2,3} and unusual hyperproductive global aphasia with uninformative emotional logorrhea and impaired right-hemispheric language abilities, atypical for left fronto-temporo-parietal involvement, was evocative of an additional right frontal involvement, confirmed by MRI. We suggest calling this unique clinical picture "moriotic aphasia." This syndrome may be useful to recognize because it raises the possibility of coupled orbito-mesial bifrontal (ACA) and left fronto-temporo-parietal (MCA) lesions, such as in left carotid occlusion in the presence of a right hypoplastic A1 segment.

From the Service de Neurologie (Drs. Ghika-Schmid, Ghika, and Bogousslavsky) and the Division Autonome de Neuropsychologie (Drs. Ghika-Schmid and Assal), Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland.

Received August 18, 1997. Accepted in final form October 7, 1997.

Address correspondence and reprint requests to Dr. Ghika-Schmid, Department of Neurology, CHUV BH13, 1011 Lausanne, Switzerland.

Copyright © 1998 by the American Academy of Neurology

References

1. Ekman P, Friesen WV. Constants across cultures in the face and emotion. *J Pers Soc Psychol* 1971;17:124-129.
2. Jastrowitz M. Beiträge zur Localisation im Grosshirn und über deren praktische Verwerthung. *Dtsche Med Wochensh* 1988;14:108-112.
3. Levin HS, Goldstein FC, Williams DH, Eisenberg HM. Frontal lobe function and dysfunction. New York: Oxford University Press, 1991.
4. Lhermitte F. "Utilisation behaviour" and its relation to lesion of the frontal lobes. *Brain* 1983;106:237-255.
5. Rubens AB, Garrett MF. Anosognosia of linguistic deficits in patients with neurological deficit. In: Prigatano GP, Schacter DL, eds. Awareness of deficit after brain injury: clinical and theoretical issues. New York: Oxford University Press, 1991:40-52.
6. Broca P. Remarques sur le siège du langage articulé, suivies d'une observation d'aphémie. *Bull Soc Anatomique (Paris)* 1861;2:330-357.
7. Morris PL, Robinson RG, Raphael B. Emotional lability after stroke. *Aust NZ J Psychiatry* 1993;27:601-605.

Neurology®

Moriatic aphasia: A syndrome of acute carotid occlusion on the dominant side

F. Ghika-Schmid, G. Assal, J. Ghika, et al.

Neurology 1998;50;1511-1512

DOI 10.1212/WNL.50.5.1511

This information is current as of May 1, 1998

Updated Information & Services	including high resolution figures, can be found at: http://www.neurology.org/content/50/5/1511.full.html
References	This article cites 5 articles, 0 of which you can access for free at: http://www.neurology.org/content/50/5/1511.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 . All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

