

References

1. Roeltgen D, Heilman KM. Apraxic agraphia in a patient with normal praxis. *Brain Lang* 1983;18:35–46.
2. Friedman RB, Alexander MP. Written spelling agraphia. *Brain Lang* 1989;36:503–517.
3. Alexander MP, Fisher RS, Friedman R. Lesion localization in apraxic agraphia. *Arch Neurol* 1992;49:246–251.
4. Coslett HB, Gonzalez Rothi LJ, Valenstein E, Heilman KM. Dissociation of writing and praxis: two cases in point. *Brain Lang* 1986;28:357–369.
5. Hodges JR. Pure apraxic agraphia with recovery after drainage of a left frontal cyst. *Cortex* 1991;27:469–473.
6. Sugishita M, Ishijima B, Hori T. “Pure” agraphia after CM-thalamotomy. *Rinsho-shinkeigaku* 1973;9:568–574.
7. Araki S, Kawamura M, Isono O. Reading and writing deficit in cases with localized infarction of the left anterior thalamus. *No-to-Shinkei* 1990;42:65–72.
8. Talairach BJ, Tournoux P. Co-planar stereotaxic atlas of the human brain. New York: Thieme Medical Publishers, 1988:12.
9. Crary MA, Heilman KM. Letter imagery deficits in a case of pure apraxic agraphia. *Brain Lang* 1988;34:147–156.
10. Roeltgen D. Agraphia. In: Heilman KM, Valenstein E, eds. *Clinical Neuropsychology*, 3rd ed. New York: Oxford University Press, 1993:63–89.

Unilateral pure thalamic asterixis: Clinical, electromyographic, and topographic patterns

Article abstract—Eleven patients (nine with infarctions and two with primary hematomas) with isolated thalamic lesions and contralateral asterixis were examined using a standard electromyographic and neuroimaging protocol. Asterixis was a short-duration phenomenon associated with a hemiataxia hypesthesia syndrome in all patients. Electromechanical synchronization was constant for the two silent period types. The anatomic data strongly suggest that ventral lateral or lateral posterior thalamus are concerned in the pathophysiology of thalamic asterixis. **Key words:** Asterixis—Negative myoclonus—Thalamus—Electromyography—Stroke.

NEUROLOGY 2000;54:2339–2342

L. Tatu, MD; T. Moulin, MD; V. Martin, MD; G. Monnier, MD; and L. Rumbach, MD

Asterixis is defined as an abrupt, brief, arrhythmic lapse of tonically contracting muscles.¹ It is best seen in the extended wrist but can also be observed in the dorsiflexed foot or on the face. Electromyographically, asterixis is characterized by brief periods of electrical silence in muscles activated to sustain posture, and it is now considered as a form of negative myoclonus.^{2,3} Unilateral asterixis is a reliable sign of structural neurologic disease and is associated with various focal brain lesions in which the thalamus is frequently involved.^{4,5}

We report 11 cases of unilateral asterixis related to an isolated contralateral thalamic vascular lesion. Electromyographic patterns were studied for all patients. Anatomic and radiologic correlations were performed to assess the precise lesion topography.

Methods. Between 1990 and 1998, every patient admitted for stroke to the Neurology Department of the Besançon University Hospital was systematically examined for asterixis. The patients, eyes closed, were asked to hold their arms outstretched with their hands dorsiflexed at the wrist and their fingers extended and abducted. The patients were also asked to dorsiflex the ankle and to show

their teeth in order to demonstrate a leg or face asterixis. The duration of asterixis was noted.

Among 35 patients with unilateral asterixis and stroke, 28 had at least one thalamic involvement and 11 (nine with infarction and two with primary intracerebral hematoma [PIH]) had an isolated thalamic lesion and underwent a standard evaluation allowing inclusion in this study.

Electromyography (EMG) was performed in all patients with asterixis. EMG of forearm or finger flexor and extensor muscles and ankle flexor and extensor muscles was made with surface electrodes. An accelerometer was placed on the dorsum of the hand or fingers and the dorsum of the foot. For the face, the surface electrodes were placed over the levator labii superioris, the orbicularis oris, and the depressor anguli oris muscles. According to the analysis of Ugawa et al.,⁶ EMG silences were classified as silent period type I and type II. In type I, the silent period followed usual EMG background activities. In type II, the silent period followed a somewhat large EMG discharge preceded by a short EMG silence. The characteristics of this synchronization of mechanical effect and silent periods were studied in all patients.

MRI was performed in all patients using the center-to-center bicommissural plane as a reference. The site of the thalamic lesion and, for thalamic infarction, the arterial territory were systematically assessed.⁷ Standard EEG was carried out in all patients but without simultaneous EEG-EMG studies. Sensitive-evoked potentials were not measured. All patients were questioned on possible drug-induced asterixis. Laboratory studies were performed in order to rule out metabolic disorders.

From the Fédération des Sciences Neurologiques, Centre Hospitalier Universitaire Jean Minjot, Besançon, France.

Received October 19, 1999. Accepted in final form February 29, 2000.

Address correspondence and reprint requests to Dr. Laurent Tatu, Service de Neurologie, CHU Jean Minjot, 25030 Besançon, France; e-mail: laurent.tatu@univ-fcomte.fr

Table Clinical, electromyographic, and radiologic characteristics

Patient no. (diagnosis)	Sex	Age, y	Asterixis location	Asterixis duration, d	Silent periods	VPL	VL	LP	CM	ANT	PU
1 (Infarction)	F	20	Left U	21	I	+	+	+			
2 (Infarction)	M	73	Right U	90	I + II	+	+		+		
3 (Infarction)	F	76	Left U	8	I + II	+	+	+			+
4 (Infarction)	M	70	Left U	3	I	+	+	+	+		
5 (Infarction)	M	58	Right U	1	I	+	+	+	+		
6 (Infarction)	F	75	Left U	2	I	+	+			+	
7 (Infarction)	M	77	Right U	3	I	+	+	+	+		+
8 (Infarction)	F	89	Right U	90	I + II	+	+	+			
9 (Infarction)	F	68	Right U	8	I + II	+	+	+			
10 (Hematoma)	F	70	Right U + L + F	6	I	+	+	+	+		+
11 (Hematoma)	M	68	Left U	8	I	+	+	+	+		

VPL = ventroposterolateral nucleus; VL = ventral lateral nucleus; LP = lateral posterior nucleus; CM = centromedian nucleus; ANT = anterior nucleus; PU = pulvinar; U = upper limb; L = lower limb; F = face.

Results. *Clinical features.* The baseline characteristics of our patients are presented in the table. Asterixis was present in the upper limb for all patients; in one patient we observed a “hemias-terixis.” Unilateral asterixis was always associated with hemiataxia and hypesthesia. For each patient, unilateral asterixis was present at the initial examination performed at a mean of 7.2 hours (range, 2 to 20 hours). Asterixis duration was less than 8 days in seven patients. In two patients, asterixis was still found 3 months after stroke onset.

Electromyography patterns. All patients had EMG patterns indicative of asterixis. We found isolated type I silent periods in seven patients and both type I and type II silent periods in four patients. Type II silent periods were never found in isolation. The duration of type I silent periods ranged from 50 to 120 msec except for Patient 4, in whom some silences lasted more than 150 msec. The total duration of type II silent periods ranged from 135 to 240 msec. The durations of the different parts of the silent periods were as follows: first silence, 35 to 65 msec; second silence, 75 to 115 msec; and intermediate burst, 35 to 55 msec. The mechanical effect observed on the accelerometer was synchronized with EMG silent periods. For type I silent periods, the mechanical effect began at a point located 70% into the electric silence period (figure 1A). For type II silent periods, the mechanical effect was synchronized with the second electric silence and began 70% into this period (figure 1B).

Standard electroencephalographic patterns. Standard EEG was normal in all patients; no epileptic discharge was observed.

Topography of thalamic vascular lesions. Lesions were always located contralateral to the asterixis (see table). PIH involved the whole ventral lateral thalamus. For the nine patients with ischemic lesions, the ventroposterolateral and ventral lateral nuclei were involved in all and the lateral posterior nucleus in seven (figure 2). Ischemic lesions were limited to the territory of the thalamogeniculate arteries in all patients except in Patient 6, in whom the territory of the posterior communicating artery was also involved.

Discussion. The first descriptions of unilateral asterixis in focal thalamic lesions concerned thalamotomies.⁸ Since this initial article, other sporadic cases of acute unilateral thalamic asterixis documented with MRI related to infarction⁵ or PIH⁴ have been reported.

In our patients, unilateral asterixis was always present at the first neurologic examination and usually concerned the upper limb. Thalamic “hemias-terixis,” as in Patient 10, is an uncommon condition.⁴ The duration of thalamic asterixis in our patients was as short as in those cases previously reported, from 1 to 12 days.

In all our patients, hemiataxia hypesthesia syndrome was associated with unilateral thalamic asterixis. The term *hemiataxia hypesthesia* is used to describe cerebellar ataxia and ipsilateral sensory abnormalities caused by a lateral thalamic stroke. Sensory disturbances of the syndrome are due to the ventroposterolateral thalamic nucleus lesion whereas cerebellar ataxia results from the interruption of dentatorubrothalamic fibers in ventral lateral thalamic nucleus infarction.⁵

Vascular lesions implicated in unilateral thalamic asterixis are only described as posterolateral thalamic lesions.^{4,5} In our series, the ventroposterolateral and ventral lateral nuclei were always involved underlying the hemiataxia hypesthesia syndrome. Nevertheless, the lateral posterior nucleus was involved in seven of nine patients with ischemic lesions. The role of the ventroposterolateral nucleus in the pathophysiology of thalamic asterixis seems to be less important because limited infarctions of this nucleus usually lead to a pure sensory stroke with no unilateral asterixis.⁹ These data suggest that ventral lateral and lateral posterior nuclei provide a key to understanding the pathophysiology of thalamic asterixis.

Since the work of Young and Shahani,³ asterixis has been regarded as a form of negative myoclonus

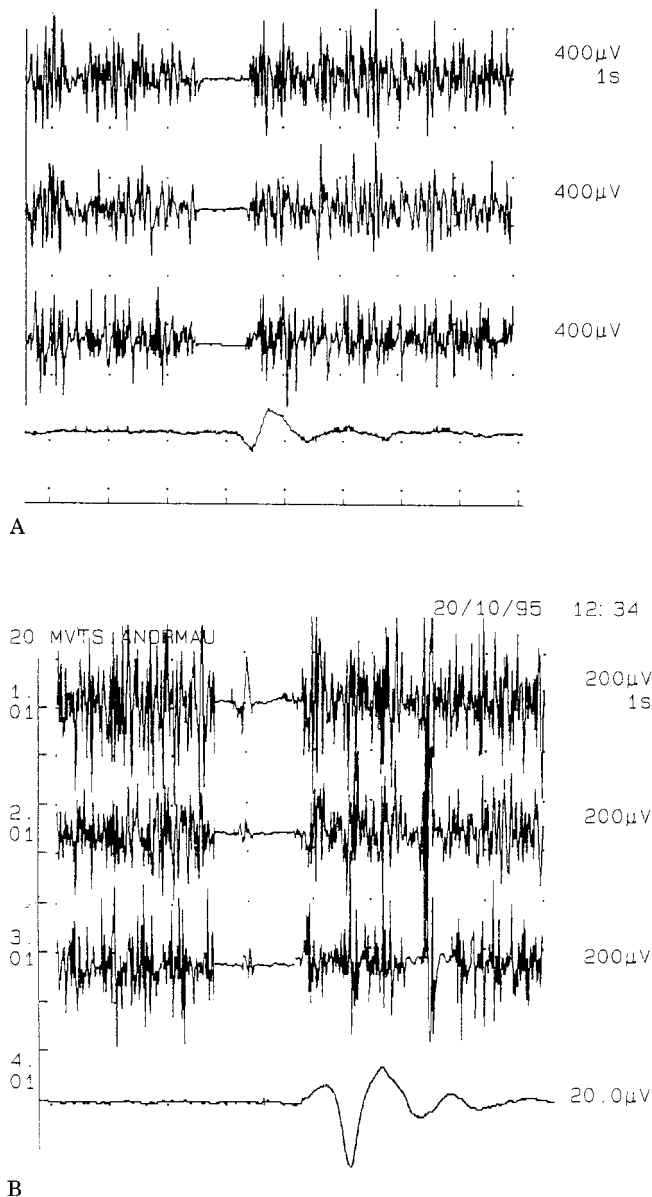


Figure 1. Electromyographic silence was recorded in the extensor digitorum (line 1), the flexor digitorum superficialis (line 2), and the first interosseus muscles (line 3). The mechanical effect on accelerometer is shown in line 4. (A) In a type I silent period, mechanical effect began at a point located 70% into the electric silence period. (B) In a type II silent period, mechanical effect was synchronized with the second electric silence and began 70% into this period.

corresponding to a failure in tonic neuronal activity necessary to maintain a posture. Silent periods underlying unilateral asterixis defined by EMG usually last from 50 to 220 msec.² The duration of electric silences observed in epileptic negative myoclonus are longer, reaching 400 msec,¹⁰ thus allowing a clear distinction from asterixis. In our patients, type II silent periods were always associated with type I periods, as in the initial report.⁶ Type I silent periods were isolated in seven patients. The significance of

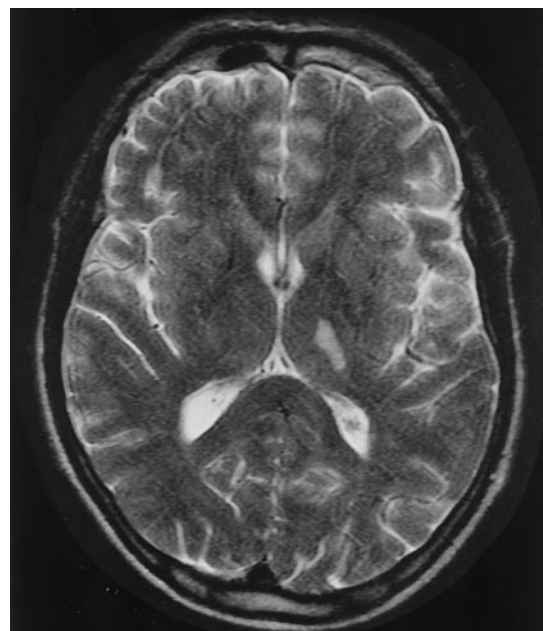


Figure 2. MRI reveals left thalamic infarct involving ventroposterolateral, ventral lateral, and lateral posterior nuclei, and pulvinar.

these two types of silences is unknown. Moreover, information on the relationship between mechanical effect and EMG patterns is scarce. In our study, we found that the onset of mechanical effect regularly began at a point located 70% into the period of electric silence (or the second electric silence for type II silent periods).

Our series demonstrates that the association of unilateral asterixis and hemiataxia hypesthesia syndrome is highly suggestive of an association of a lateral and ventral thalamic stroke. Unilateral thalamic asterixis is usually a transient phenomenon with well-defined EMG patterns. Our data strongly suggest that ventral lateral and lateral posterior thalamic nuclei should be taken into consideration in the pathophysiology of this movement disorder.

References

1. Adams RD, Foley JM. The neurological changes in the more common types of severe liver disease. *Trans Am Neurol Assoc* 1949;74:217-219.
2. Shibasaki H. Pathophysiology of negative myoclonus and asterixis. In: Fahn S, ed. *Advances in neurology*, volume 67: negative motor phenomena. New York: Raven Press; 1995: 199-209.
3. Young RR, Shahani BT. Asterixis: one type of negative myoclonus. In: Fahn S, ed. *Advances in neurology*, volume 43: myoclonus. New York: Raven Press; 1986:137-156.
4. Donat JR. Unilateral asterixis due to thalamic hemorrhage. *Neurology* 1980;30:83-84.
5. Melo TP, Bogousslavsky J. Hemiataxia hypesthesia: a thalamic stroke syndrome. *J Neurol Neurosurg Psychiatry* 1992; 55:581-584.
6. Ugawa Y, Shimpo T, Mannen T. Physiological analysis of

asterixis: silent period locked averaging. *J Neurol Neurosurg Psychiatry* 1989;52:89–93.

7. Tatu L, Moulin T, Bogousslavsky J, Duvernoy HM. Arterial territories of human brain: cerebral hemispheres. *Neurology* 1998;50:1699–1708.
8. Young RR, Shahani BT, Kjellberg RJ. Unilateral asterixis pro-

duced by a discrete CNS lesion. *Trans Am Neurol Assoc* 1973; 101:306–307.

9. Pacciaroni M, Bogousslavsky J. Pure sensory syndromes in thalamic strokes. *Eur Neurol* 1998;39:211–217.
10. Guerrini R, Dravet C, Genton P, et al. Epileptic negative myoclonus. *Neurology* 1993;43:1078–1083.

Thalamic stimulation for the treatment of midline tremors in essential tremor patients

Article abstract—The authors prospectively collected unblinded data from 27 consecutive patients following thalamic stimulation. A significant reduction of midline tremor was achieved after unilateral surgery, but a staged contralateral surgery had an additional effect. A subgroup analysis showed significant beneficial effects for head, voice, tongue, and face tremor. The most frequent reversible side effects were disequilibrium, dysarthria, and paresthesias. We observed more pulse generator adjustments for speech problems in the bilaterally implanted group. **Key words:** Thalamic nuclei—Stimulation—Essential tremor.

NEUROLOGY 2000;54:2342–2344

A.A. Obwegeser, MD; R.J. Uitti, MD; M.F. Turk, MSN; A.J. Strongosky, BA; and R.E. Wharen, MD

Essential tremor (ET) is one of the most common movement disorders. It most frequently affects the upper extremities, followed by head and voice.¹ Thalamic deep brain stimulation (DBS) of the ventral intermediate thalamic nucleus (Vim) effectively reduces limb tremor.^{2,3} Reports on the effect of thalamic stimulation on midline tremors are limited.⁴

Methods. Twenty-seven consecutive ET patients underwent thalamic DBS for intractable ET. Diagnosis of ET was based on the criteria by Louis.⁵ The indication for surgery was disabling tremor despite optimal medical therapy, which included propranolol, primidone, and gabapentin at maximal tolerable doses. Bilateral surgery was performed in cases of severe bilateral tremor.

The primary outcome measure was tremor evaluation based on the Fahn-Tolosa-Marin tremor rating scale carried out within two days before surgery and three months following each surgery. The stimulator was deactivated for at least 1 hour before tremor assessment (“off”). Tremor with activated stimulator was measured (“on”), when DBS provided maximal tremor control without side effects. Dr. Uitti evaluated all patients. No attempt was made to blind the examiner or patient.

The first surgery was performed contralateral to the body-side expected to bring the most benefit to the patient. If the patients had marked bilateral limb tremor, a second electrode was implanted approximately 4 months following the first surgery. Dr. Wharen performed each procedure.

The surgical procedure followed the guidelines proposed by other authors.^{2,4} In brief, a stereotactic target in the Vim was planned with MR guidance. Microelectrode recording was used for the first 13 patients. In all patients test stimulations were performed to evaluate tremor relief and side effects. After determining optimal position, the stimulation electrode (Model 3387, Medtronic, Minneapolis, MN) was secured to the skull and connected to the subclavicular pulse generator under general anesthesia.

Within 2 weeks after surgery the patient returned for further stimulator programming. If necessary, the stimulation parameters were adjusted for optimal tremor control with minimal side effects. If patients later observed side effects or a loss of tremor control, they returned for further pulse generator adjustment. All side effects were recorded. Initial paresthesias, occurring when the stimulator was turned on and subsiding within a few seconds, were not considered a side effect.

A Wilcoxon signed rank test was used to determine statistical probability for differences in patient scores. A Fisher's Exact test compared side effects. All *p* values were calculated for 2-sided tests. *p* Values less than 0.05 were considered to be significant. Values are mean (\pm SD).

Results. Patient data. Fourteen patients had a unilateral procedure and 13 patients had bilateral surgery (12 in staged procedures). Mean follow-up was 11 months in patients with unilateral surgery only and 12 months after bilateral surgery. With the exception of one patient who had simultaneous bilateral implantation, all patients were operated on the dominant hemisphere first. Age at first surgery was 73(\pm 5.2) years old and duration of tremor 27(\pm 15) years.

Two weeks before surgery 12 patients were given no antitremor drugs. The drugs used in the remaining 15 patients were propranolol, primidone, and gabapentin. Six of these patients completely stopped their medication at least 24 hours before the baseline evaluation. The remain-

From the Departments of Neurosurgery (Drs. Obwegeser and Wharen), and Neurology (Drs. Uitti, Turk, and Mr. Strongosky), Mayo Clinic Jacksonville, Florida; and the Department of Neurosurgery (Dr. Obwegeser), University Innsbruck, Austria.

Received December 9, 1999. Accepted in final form March 17, 2000.

Address correspondence and reprint requests to Dr. Robert E. Wharen, Mayo Clinic Jacksonville, 4500 San Pablo Road, Jacksonville, FL 32224; e-mail: wharen@mayo.edu

Neurology[®]

Unilateral pure thalamic asterixis: Clinical, electromyographic, and topographic patterns

L. Tatu, T. Moulin, V. Martin, et al.
Neurology 2000;54;2339-2342
DOI 10.1212/WNL.54.12.2339

This information is current as of June 27, 2000

Updated Information & Services	including high resolution figures, can be found at: http://www.neurology.org/content/54/12/2339.full.html
References	This article cites 8 articles, 5 of which you can access for free at: http://www.neurology.org/content/54/12/2339.full.html##ref-list-1
Citations	This article has been cited by 4 HighWire-hosted articles: http://www.neurology.org/content/54/12/2339.full.html##otherarticles
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.

