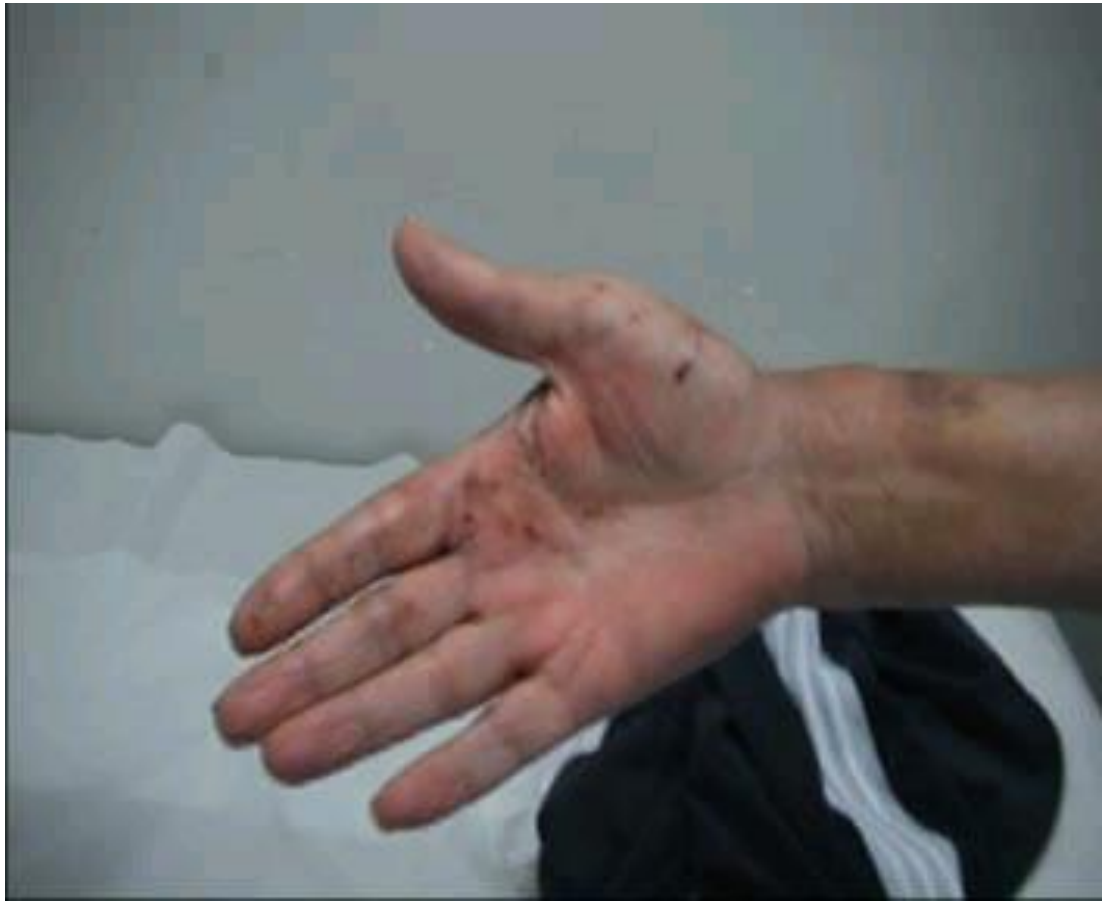
- 69 ans
- Zona dermatomes C6 et C7
- MAI du MSD
- IRM impossible (Pacemaker)
- Amélioré par levetiracetam



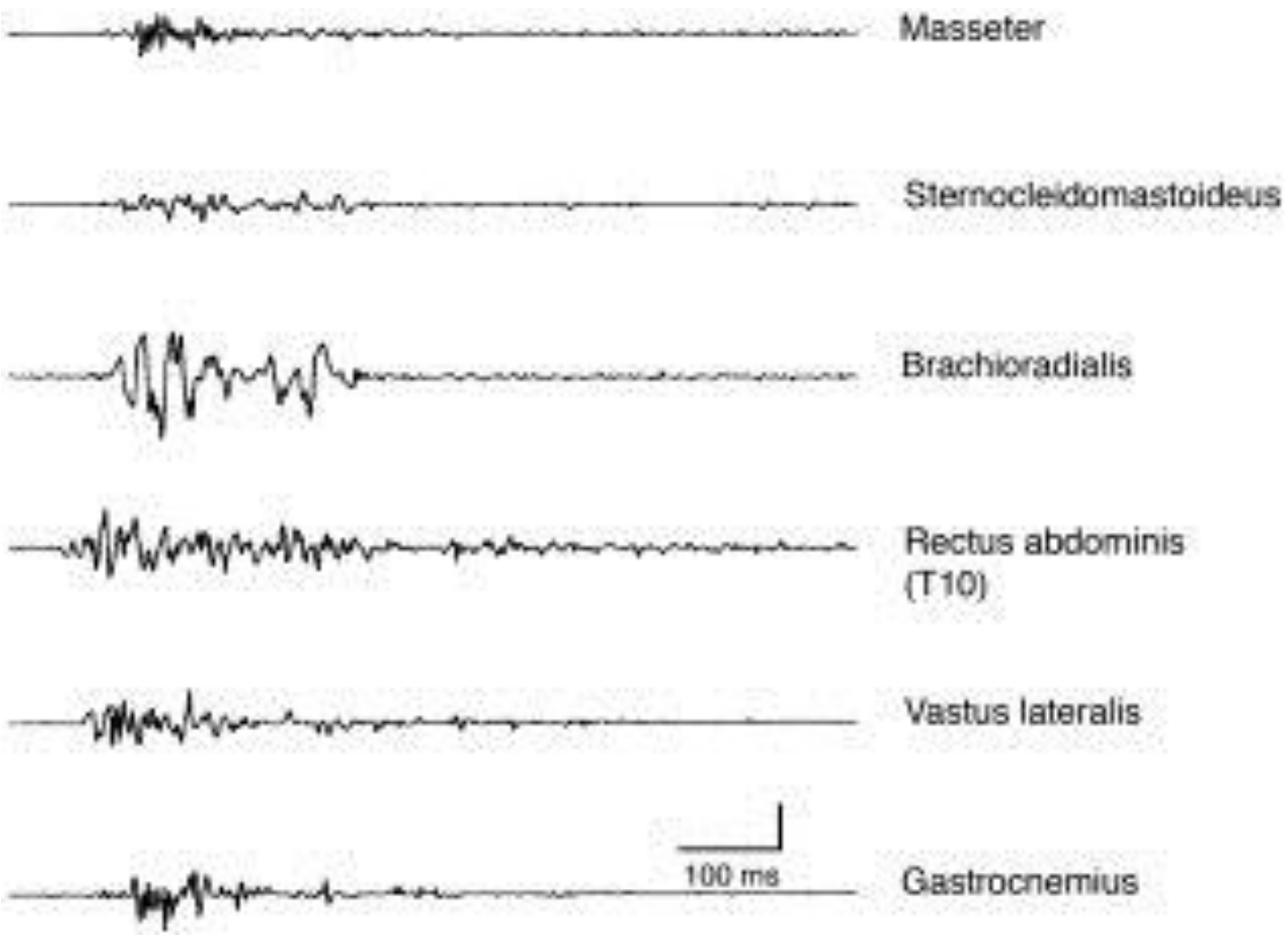
Estreano, Neurology 2007

Myoclonies spinales segmentaires

- Myoclonies rythmiques, de topographie limitée aux muscles innervés par 1 ou 2 segments médullaires contigus
- Uni ou bilatérales
- Le plus souvent rythmique mais arythmie possible
- 1/min à 240/min
- Durée de 100 ms à 1 s
- Parfois :
  - stimuli-sensitives
  - persistance pendant sommeil
- Rechercher une lésion médullaire
- TTT : Clonazepam…

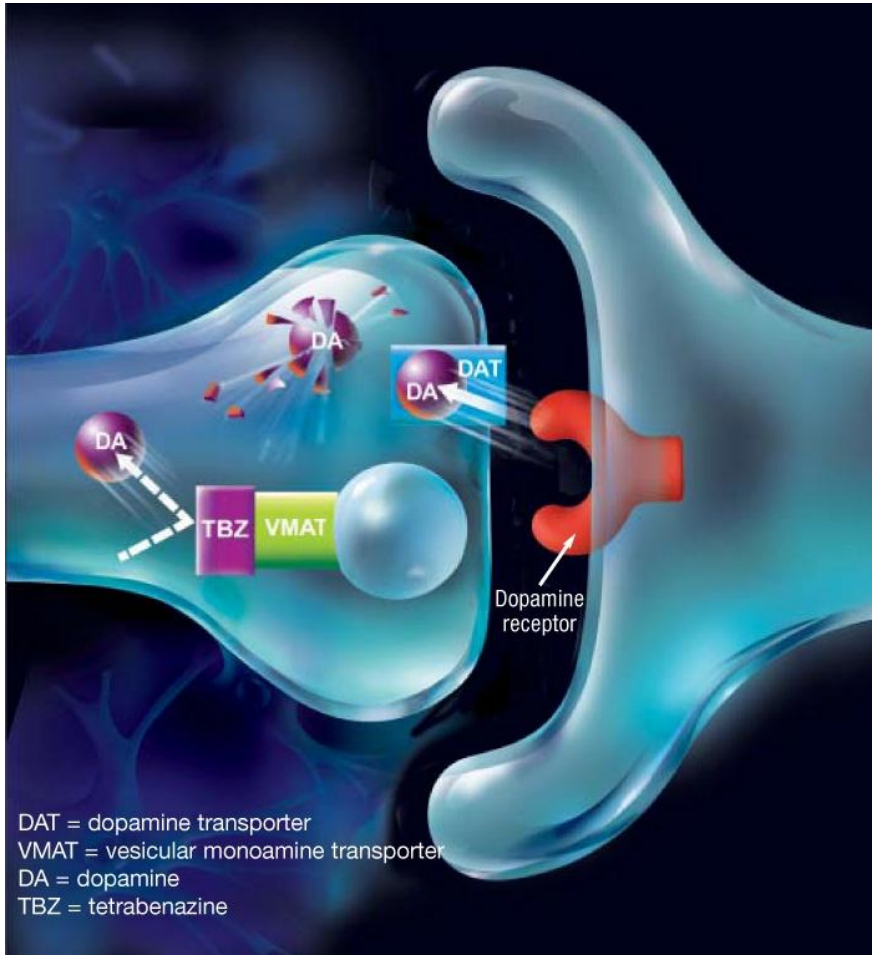
Myoclonies proprio-spinales

- Générateur médullaire le plus souvent dorsal
- Influx anormal se propage sur l'ensemble de l'axe médullaire, recrutant les muscles du tronc et de la racine des membres
- Flexion spontanée du tronc et parfois de la racine des membres et du cou
- Maximum repos et couché, gênent l'endormissement et persistent pendant le sommeil
- Stimuli-sensitives 50% des cas
- Durée des myoclonies longue, jusqu'à 4 secondes
- Sensation prémonitoire 50% des cas
- Causes : pathologies médullaires, ciprofloxacine, cannabis, « idiopathiques »…


Masseter
Sternocleidomastoideus
Brachioradialis
Rectus abdominis
(T10)
Vastus lateralis
100 ms
Gastrocnemius

Tetrabenazine

- Bloque le transporteur des monoamines VMAT2
- 25 à 150 mg/j
- Mouvements anormaux involontaires hyperkinétiques



Trauma-Induced Spinal Vascular Event Producing Hemipseudoathetosis

S Frank, R Barbano

Movement Disorders
ement Disorders Society

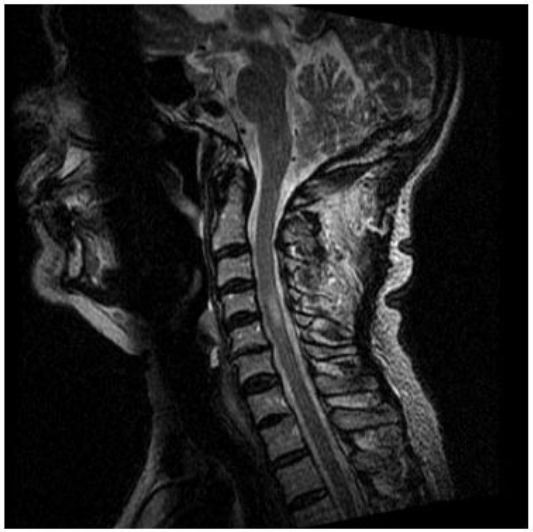


FIG. 1. The sagittal T2 sequence of the cervical spine demonstrates a high signal intramedullary lesion.

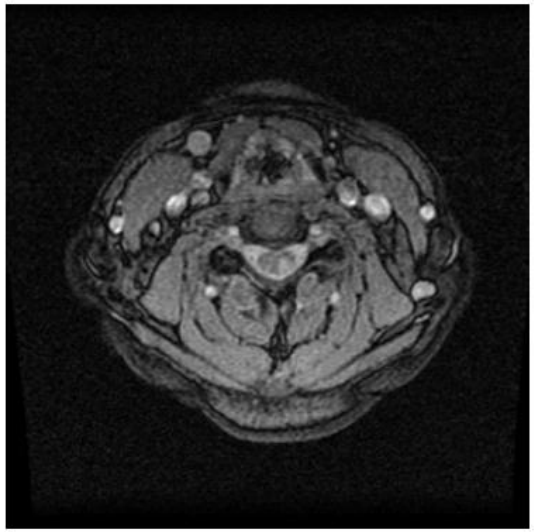


FIG. 2. The axial T2 sequence at the C4 level demonstates the lesion eccentric to the left.

Spinal pseudoathetosis:

A rare, forgotten syndrome, with a review of old and recent descriptions

J. Ghika, MD, and J. Bogousslavsky, MD

Article abstract—We report a patient with a cervical spinal astrocytoma who presented with athetotic hand movements. We postulate that acute pain, related to a syrinx around the operative site, contributed to the athetosis. These movements occur only after protracted, severe loss of proprioception, often in conjunction with an acute, generally painful crisis. Athetotic movements can disappear without change in proprioceptive or motor deficits when pain lessens.

NEUROLOGY 1997;49:432–437

In 1871, Hammond[1] coined the term *athetosis* (from Greek, meaning "without fixed position") to describe a group of involuntary movements characterized by "an inability to retain the fingers and toes in any position in which they might be placed and by their continual motion". Athetosis is a clinical syndrome in which slow, writhing, vermicular, sinuous, cramplike movements seem performed with great deliberation and force.[1] Alternating posture and/or motor patterns, which are monotonous but vary in intensity and frequency with time, are characteristic of the clinical picture.[2-4] Caricatures of movements such as piano playing, withdrawal from an object, and alternation of grasping and avoiding reactions that

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Athétose/Pseudoathétose

- Athétose : incapacité à garder immobiles les doigts et orteils
- Varie en fréquence et intensité
- Souvent associé à une posture anormale en hyperextension
- Si troubles proprioceptifs : pseudoathétose
- Décrit après lésions sur l'ensemble des voies proprioceptives

Pseudoathetosis: Report of Three Patients

M Spitz, AA Costa Machado,
R do Carmo Carvalho, F Martins Maia,
M Santoro Haddad, D Calegaro,
M Scaff, E Reis Barbosa

Movement Disorders

CME

Clinical characteristics and topography of lesions in movement disorders due to thalamic lesions

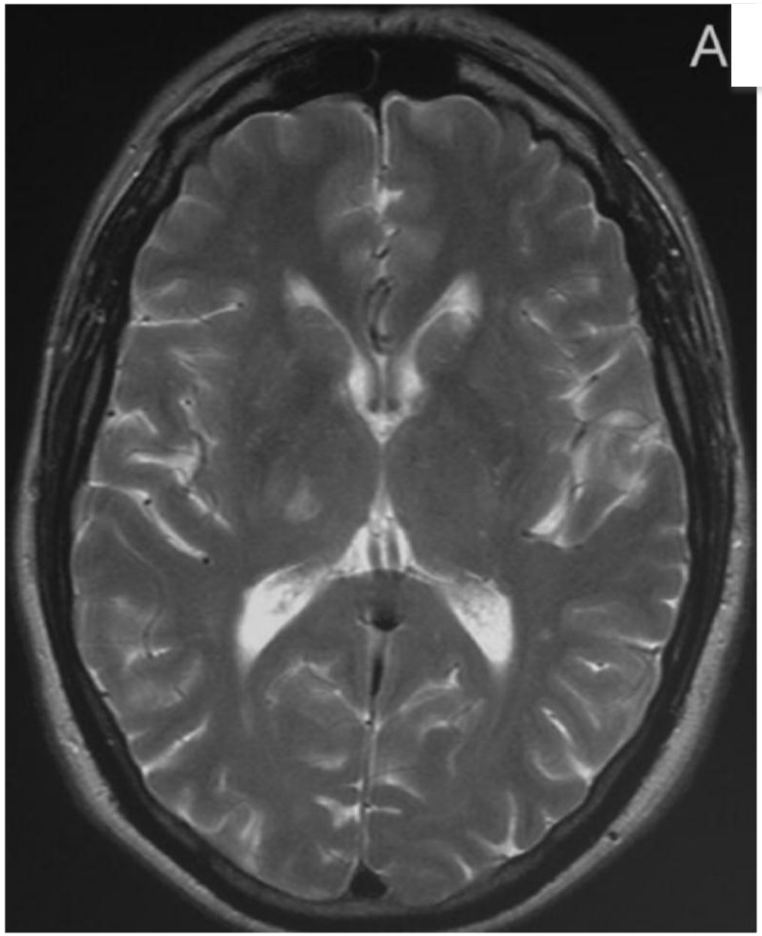
S. Lehéricy, MD, PhD; S. Grand, MD; P. Pollak, MD; F. Poupon, PhD; J.-F. Le Bas, MD; P. Limousin, MD, PhD; P. Jedynak, MD; C. Marsault, MD; Y. Agid, MD, PhD; and M. Vidailhet, MD

Article abstract—*Objective:* To determine which thalamic subnuclei are involved in symptomatic unilateral movement disorders due to localized thalamic infarction, and the clinical characteristics of these abnormal movements. *Methods:* The authors studied 22 patients with thalamic infarcts for their clinical presentation and the topography of the lesions, using three-dimensional T1-weighted MRI sequencing and stereotaxic analysis of the lesions. *Results:* Patients were divided into four groups: 1) absence of abnormal involuntary movements (AIM) (nine patients); 2) isolated dystonic posture (two patients); 3) myoclonic dystonia (five patients); and 4) tremor or myoclonus (six patients). In patients with AIM, thalamic lesions were contralateral to the abnormal movements, involving the thalamogeniculate territory, centered on the ventral intermediate (Vim) and ventral caudal (Vc) nuclei. No significant difference in the volumes or center of mass of the lesions was found between patients with tremor and myoclonus and patients with dystonia, although the central nucleus and the internal part of the Vim nucleus were more consistently damaged in dystonic patients. *Conclusion:* Movement disorders related to thalamic lesions included: 1) myoclonic dystonia with predominating myoclonus and "thalamic" hand associating dystonic posture and slow, pseudo-athetoid movements, both related to lesions in the Vim and Vc nuclei of the thalamus; and 2) postural and action tremor, also related to lesions in the Vim, similar to tremor associated with midbrain lesions, as a result of abnormal functioning of the cerebello-thalamic pathways.

NEUROLOGY 2001;57:1055–1066

Homme de 58 ans

- Droitier
- AVC il y a 5 ans
- Ne se souvient pas de la symptomatologie à l'époque
- Mouvement anormal du MSD survenu quelques mois après l'AVC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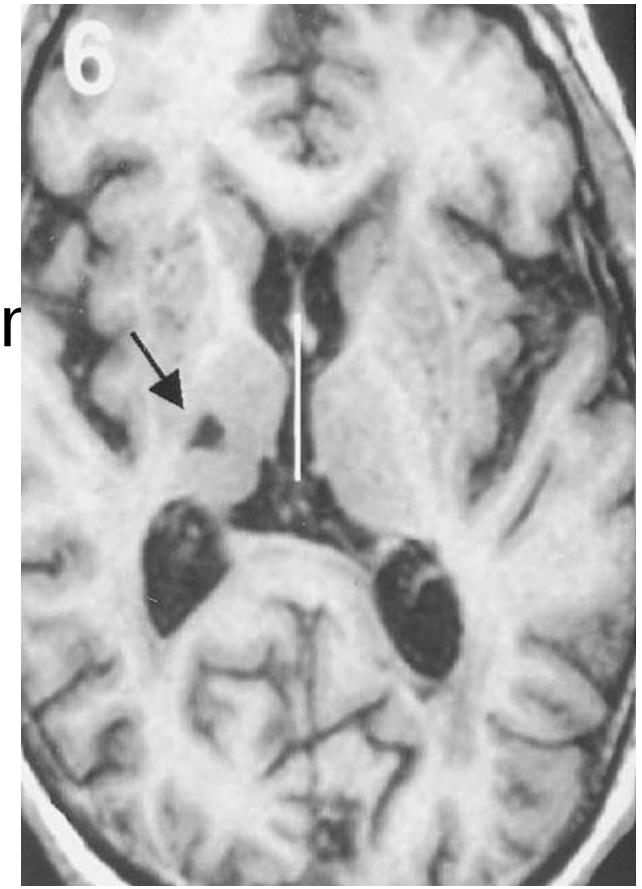
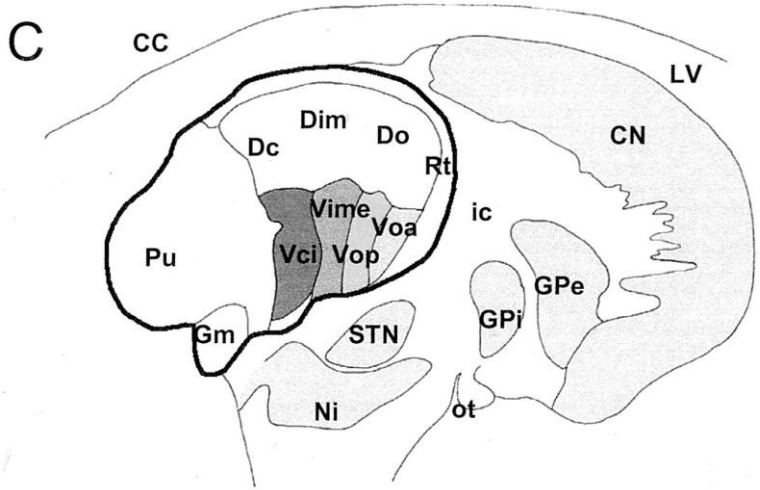
Mouvements anormaux et AVC thalamique

- Dystonie myoclonique
- Myoclonies isolées
- Plus rarement dystonie isolée
- Pseudo-athétoïde
- Territoire thalamo-géniculée, Vir





Lehericy et Vidailhet Neurology 2001

Striatopallidal and Thalamic Dystonia

A Magnetic Resonance Imaging Anatomoclinical Study

Stéphane Lehéricy, MD, PhD; Marie Vidailhet, MD; Didier Dormont, MD; Laurent Piérot, MD; Jacques Chiras, MD; Pilar Mazetti, MD; Claude Marsault, MD; Yves Agid, MD, PhD

Objective: To determine which brain structures are involved in symptomatic unilateral dystonia caused by localized cerebral infarction.

Design: Three-dimensional T_1-weighted magnetic resonance imaging sequence and stereotactic analysis were used to analyze the topography of the lesions. Stereotactic localization of thalamic lesions was conducted according to the atlas of Hassler with a Voxtool software (Advantage Windows Workstation, General Electric, Milwaukee, Wis) workstation system.

Patients: Eight patients with hemidystonia, segmental dystonia, or focal dystonia were selected from among 51 consecutive patients (between January 1988 and May 1993) with symptomatic unilateral dystonia.

Results: Patients had dystonic spasms (n=4) or myoclonic dystonia (n=4). Lesions associated with dystonic spasms were located in the striatopallidal complex, and those with myoclonic dystonia were in the thalamus contralateral to the dystonia. Lesions of the striatopallidal complex involved the putamen posterior to the anterior commissure in all patients and extended variably into the dorsolateral part of the caudate nucleus, the posterior limb of the internal capsule, or the lateral segment of the globus pallidus. These lesions were centered in the "sensorimotor" part of the striatopallidal complex, with a trend toward a somatotopical distribution. Lesions of the thalamus were located in the ventral intermediate and ventral caudal nuclei, while the ventral oral anterior and posterior nuclei (which receive pallidal efferents) were largely spared.

Conclusion: These results suggest that striatopallidal and thalamic dystonia may have different pathophysiologic bases.

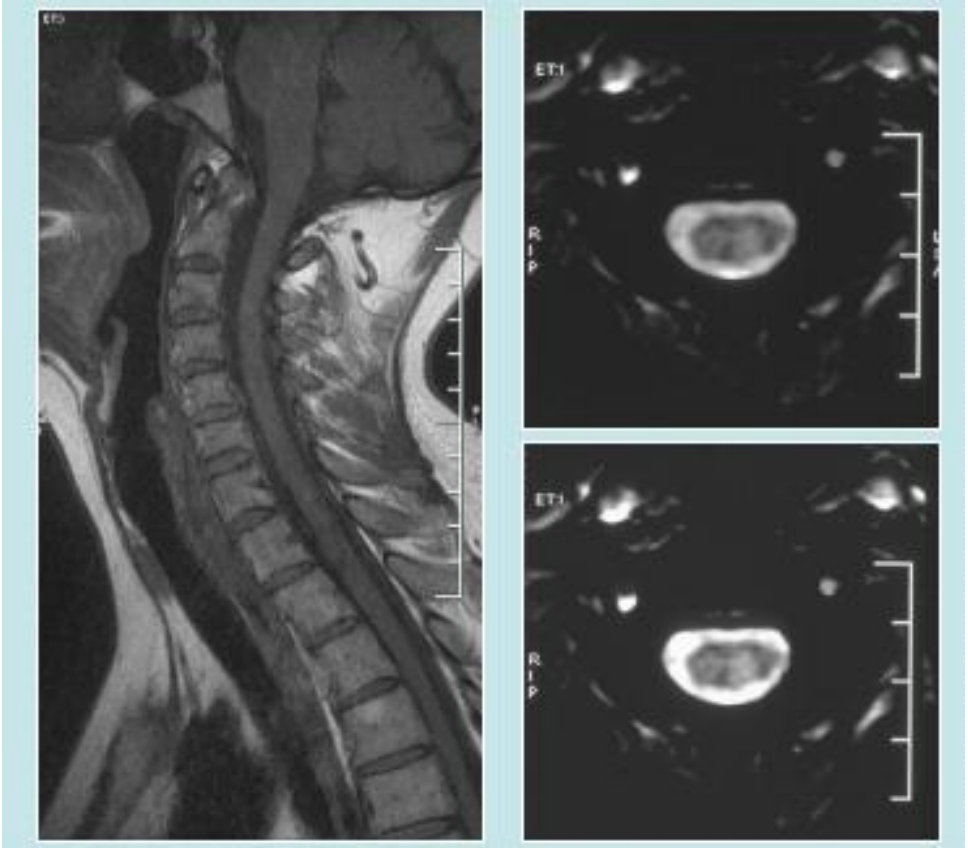
(Arch Neurol. 1996;53:241-250)

- Homme de 52 ans
- 1990 : douleurs du MID, sciatique S1?
- 1998 : algoneurodystrophie du MID
- 2001 : douleurs atypiques du MID, botte plâtrée
- Juin 2002 : MAI du MID, début assez brutal

« Painful legs moving toes »

- Première description 1991
- Douleur premier signe, jours à années avant les MAI
  - « Brulures, écrasement », non systématisable
  - Hyperpathie, allodynie, algodystrophie
- MAI
  - Complexes, flexion, extension, adduuction, abduction
  - 1 à 2 Hz
- Le plus souvent bilatéral mais parfois unilatéral
- Causes
  - Atteinte radiculaire (mécanique, post zostérienne)
  - Traumatisme osseux ou parties molles
  - Atteinte médullaire

- Femme de 44 ans
- PR depuis 1976
- LEAD depuis 1998
- 2 épisodes de myélite cervicale en 2001 et 2005
- Episodes brefs, paroxystiques, 30 à 50 à fois/j de contracture MSG MIG durée quelques secondes
- Favorisés lorsque se lève brutalement
- IRM cérébrale normale



Dyskinésies paroxystiques

Mouvements dystoniques, choréiques, athétosiques, balliques

- Kinésigéniques
  - Uni ou bilatéral
  - Favorisées par un mouvement brusque
  - Brefs : qques s à 5 min
  - Jusque 100 épisodes par jour
  - Efficacité des anti-comitiaux
  - Idiopathique (génétique) ou symptomatiques (SEP)
- Non kinésigéniques
  - Uni ou bilatéral
  - Favorisé par café, alccol, thé
  - Longues : de 5 min à 4 heures
  - 2 à 3 épisodes par jour
  - Inefficacité des anti-comitiaux
  - Idiopathiques ou symptomatiques

Dyskinésies paroxystiques et atteinte médullaire

- Rare, la plupart des dyskinésies paroxystiques symptomatiques sont en effet liées à des atteintes encéphaliques

Paroxysmal Kinesigenic Dystonia and Spinal Cord Lesion

Multiple sclerosis and ischemia are frequent causes of symptomatic PKD (1,2). Dystonic attacks can be extremely frequent, even >100/day, and can be precipitated by a sudden movement, hyperventilation, or sensory stimulus.

The thalamus, putamen, and internal capsule are the most frequently affected sites of lesion as demonstrated by magnetic resonance images (2,3). PKD associated with pure spinal cord lesions is rare.

We report here a case of PKD associated with an intramedullary lesion and attacks that were precipitated not only by movement but by deep nociceptive stimulus.

Movement Disorders, Vol. 11, No. 4, 1996

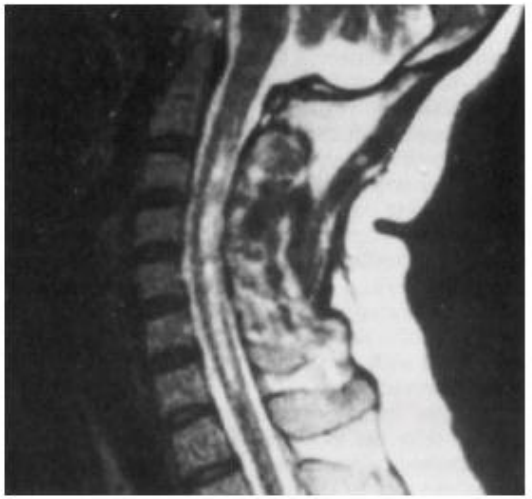


FIG. 1. T2-weighted magnetic resonance scan shows high signal intensity in the cervical spinal cord from C2 to C7.

Améliorées par Diamox