■ Electroclinical reasoning report

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Epileptic Disorders

Jerking during absences: video-EEG and polygraphy of epileptic myoclonus associated with two paediatric epilepsy syndromes

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ABSTRACT

Objective. Epileptic myoclonus (EM) is reported in many paediatric epilepsies from neonatal period to adolescence. Myoclonus can be the only seizure type or may occur among others, independently or in combination as a single ictal event. We report two children presenting with absences associated with myoclonus, predominating on one side, in a setting of two different types of absence seizures and two different electro-clinical syndromes.

Methods. Patients were explored with long-duration video-EEG coupled to surface EMG polygraphy. EEG was visually analysed and complemented by jerk-locked back-averaging.

Results. Two types of seizure, encompassing myoclonus and absence, were identified: myoclonic absences in the context of epilepsy with myoclonic absences and atypical absences with atonic component (negative myoclonus) in the context of encephalopathy related to status epilepticus during slow sleep (ESES). In the latter case, rhythmic upper limb jerking, mimicking positive myoclonus, corresponded to recovery of muscular tone after each negative myoclonus.

Significance. Due to the rhythmic recovery of muscle tone, subsequent rhythmic negative myoclonus may exhibit a similar clinical picture to that of rhythmic positive myoclonus. Video-EEG recording coupled to EMG polygraphy is essential in order to precisely characterize motor manifestations during seizures with myoclonus. [*Published with video sequences*].

Key words: myoclonic absences; atypical absences with atonic component; encephalopathy related to status epilepticus during slow sleep (ESES); negative myoclonus

VIDEOS ONLINE

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Epileptic myoclonus (EM) is a frequent seizure type in many paediatric epilepsies, occurring from the neonatal period to adolescence. It can manifest as an exclusive seizure type associated with a syndrome (myoclonic epilepsy of infancy) or occur independently among other types, such as, for example, epilepsy with myoclonic-atonic seizures, Dravet syndrome, and juvenile myoclonic epilepsy. Myoclonus can also be embedded in another seizure type

(myoclonic absence, myoclonic-tonic seizure, and atypical absence with an atonic component) [1-3].
Herein, we report absence seizures associated with myoclonus for which electrophysiological studies disclosed opposing pathophysiological mechanisms and different nosology.

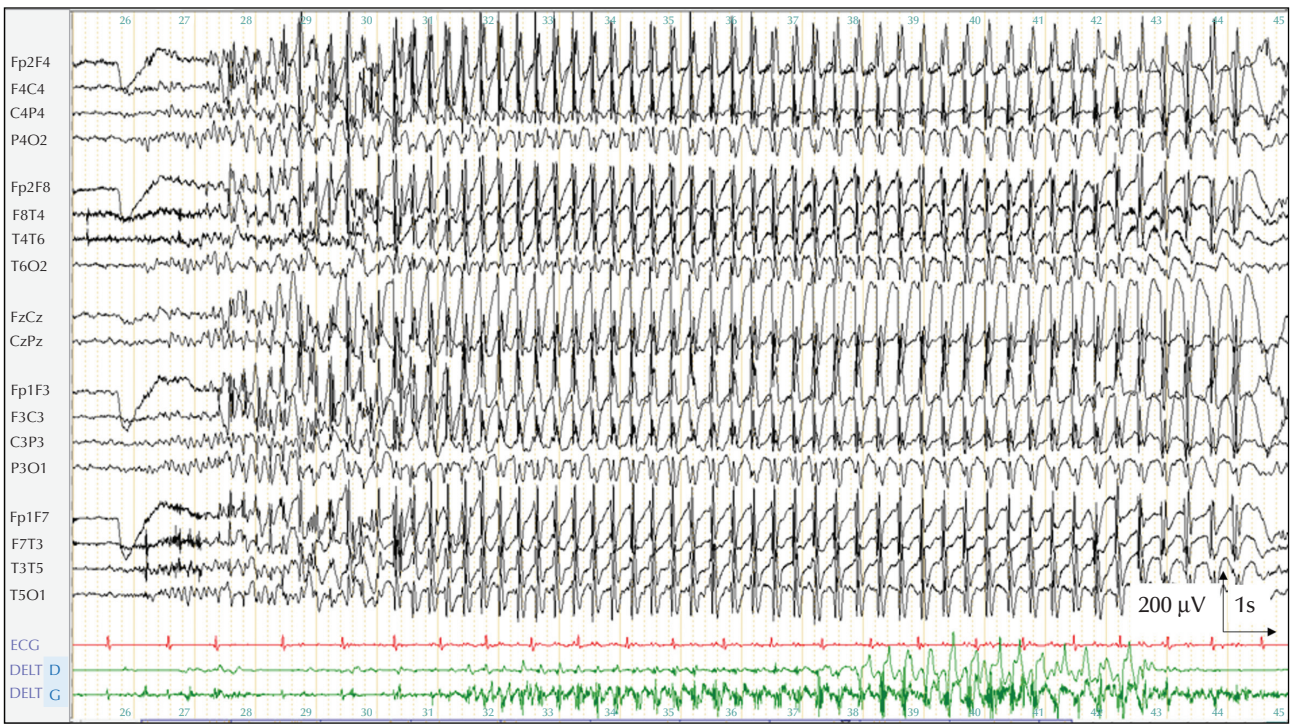
Patients and methods

Patient 1 was a boy, a third child of healthy, non-consanguineous parents, born at term after *Caesarean* section due to abnormal heart rate. Psychomotor development was marked by a global delay affecting mainly reading, writing and attention skills. Since the age of four years, he presented with stereotyped episodes of behavioural arrest and impairment of awareness associated with rhythmic jerks of the upper limbs lasting up to 20 seconds. Interictal EEG at the age of seven years showed high-amplitude bi-frontal fast activity and high-voltage right frontal spikes. Seizures occurring spontaneously or triggered by hyperventilation were characterized by a sequence of bilateral synchronous high-voltage 3-3.5-Hz spike-waves (SW), predominating over the right frontal area, lasting up to 20 seconds. Brain MRI, as well as cerebral PET scan, were normal. Genetic testing revealed Klinefelter syndrome. Epilepsy remained drug resistant despite four antiepileptic drugs (AEDs) (valproic acid, levetiracetam, lamotrigine and zonisamide).
Patient 2 was a girl, a third child of healthy non-consanguineous parents, born at term after normal pregnancy and uneventful delivery. Psychomotor development was normal. At the age of three years, she presented with seizures with repetitive blinking and back and forth head nodding with impairment of awareness. Repeated EEGs performed without polygraphy showed right central-temporal spikes and SW focus activated during drowsiness and slow sleep. Seizure frequency increased a few months after onset, up to 10 per day, with impairment of awareness, head nodding, and rhythmic left upper limb jerks. Several AEDs (clobazam, lamotrigine, valproic acid, levetiracetam, ethosuximide) were ineffective and the diagnosis of "drug-resistant myoclonic epilepsy" was erroneously proposed while she was receiving lamotrigine, valproic acid and clobazam. At the age of four years, she was hospitalized for recurrent seizures, as described above, lasting up to 30 seconds and causing drop attacks. Awake EEG was similar to the initial EEGs, however, the sleep EEG showed an activation of SW during >50% of N-REM sleep duration. 3T brain MRI was normal. The child presented with concomitant psychomotor regression, predominantly affecting fine motor skills.
Long-duration video-EEG was performed at the age of eight and five years in Patient 1 and 2, respectively, using 21 silver chloride cup electrodes placed according to the international 10/20 system (reference electrode on FPz, impedances < 10 KOhm). Polygraphy including electromyogram (EMG), recorded with two cup electrodes placed 2 cm apart, on both deltoids and on the neck in Patient 2, electrocardiogram and respiration were also included. Signals were amplified (1,000x), band-pass filtered at 0.16-97 Hz and digitized at 256 Hz, using the Deltamed Coherence EEG system. Back-averaging was locked to the onset of the myoclonic jerks in both patients using two different subsets of repetitive myoclonic events and one subset of isolated myoclonic events in Patient 2. Analysis was performed using mean reference montage. Latency was measured from the peak (positive or negative) of the spike preceding the myoclonus up to the beginning of the muscular contraction or silence using the measurement tool, Coherence software (Coherence, Deltamed/Natus, France). An analysis window was set to two seconds preceding, and to three seconds following, the marked events (Coherence, Deltamed/Natus, Paris, France).

Results

Patient 1

The inter-ictal EEG showed a well-organized awake and sleep background activity and rare, isolated, bilateral frontal-temporal spikes and SW predominating on the right or more seldom on the left side. During seizures, the child presented with incomplete impairment of awareness, a fixed gaze, deviation of the head to the right and rhythmic clonic movements of the head, eyelids and upper limbs, predominating on the left side for 15-20 seconds (video sequence 1). On EEG, seizures started with bilateral frontal-temporal spikes and polyspike and waves with a higher amplitude on the right side (asymmetry was not constant), followed by rhythmic bilateral synchronous SW at 3 Hz with maximal amplitude over the right frontal-temporal areas, lasting 20-30 seconds and ending abruptly and bilaterally. EMG showed regular, bilateral rhythmic deltoid contractions, predominating on the left, lasting for around 70 ms at the beginning of the seizure, then increasing in duration (up to 200 ms), superposed on a gradual increase in muscular tone of both deltoids predominating on the left (*figure 1*, *figure 2A*, *B*). After each myoclonus, there was a brief interruption of muscular activity

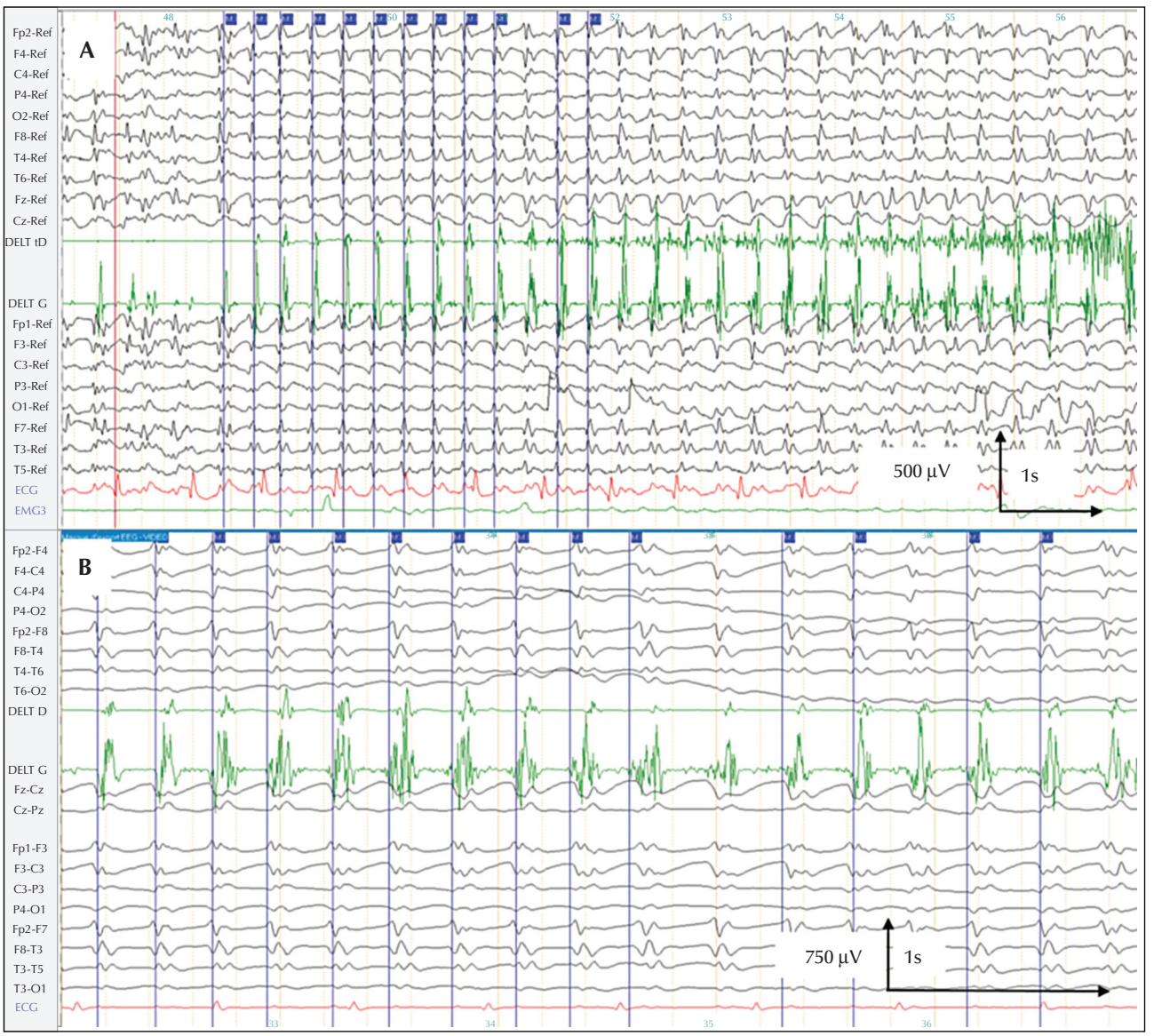


■ **Figure 1.** Ictal EEG (video sequence 1) with EMG on both deltoids (DELT D: right; DELT G: left) in Patient 1. Seizure starts with bilateral frontal-temporal spikes and polyspike and waves with a more marked amplitude on the right side, followed by rhythmic bilateral synchronous spike waves (SW) at 3 Hz with maximal amplitude over the right frontal-temporal areas, lasting around 20 seconds. Note bilateral rhythmic contractions predominating on the left deltoid (DELT G).

(*figure 2A, B*). The negative and positive components of the SW were almost synchronous on the two hemispheres, and the positive transient was maximal at Fz and presented with similar morphology and polarity at F3 and F4 (*figure 3A, B, C*). The onset of myoclonic jerks was easily identifiable initially during the absence, but was then blurred by increasing underling muscular tone (*figures 1, 2*). Back-averaging was therefore performed using the first myoclonic jerks and showed bilateral synchronous spikes with a positive component at frontal and negative at temporal-occipital electrodes, predominating on the right and preceding the myoclonic jerk by 20-30 ms (bilateral tangential dipoles with inversion of polarity near central electrodes) (*figure 3 A-C*).

Patient 2

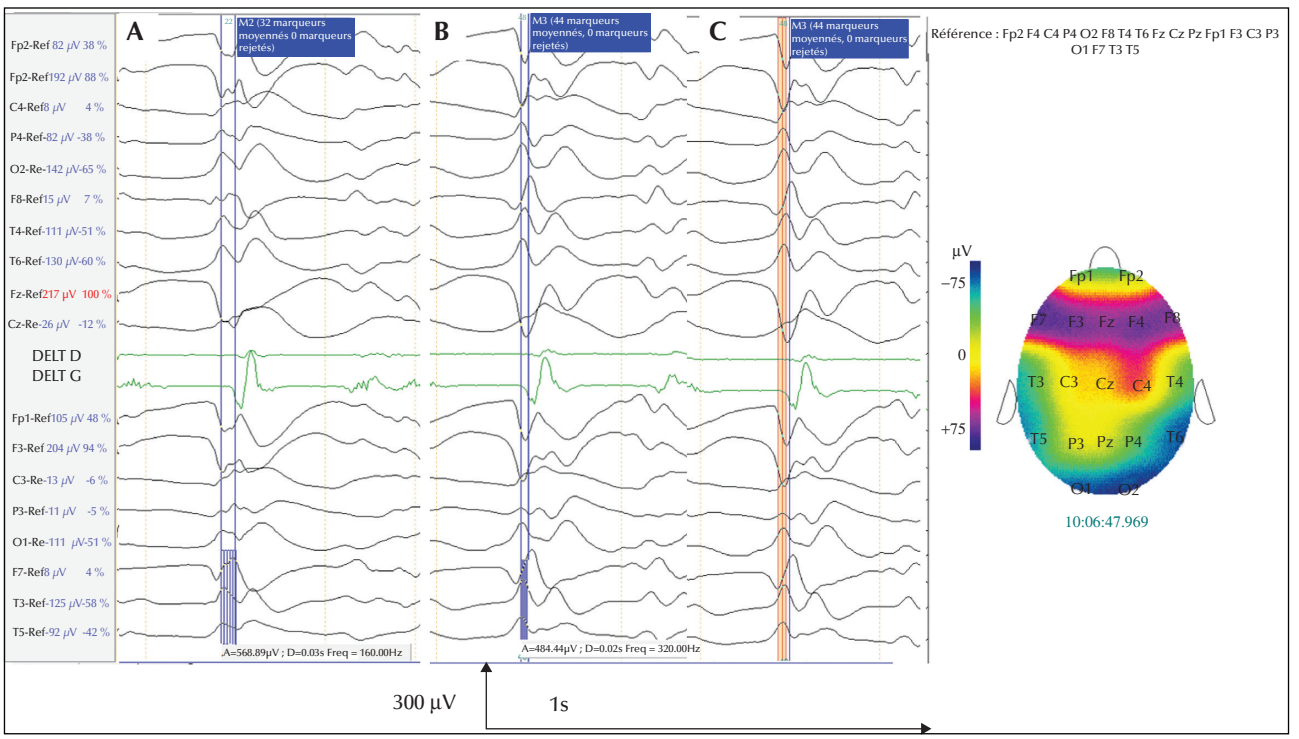
The interictal EEG recording during wakefulness showed normal background activity with right central-parietal SW. Seizures consisted of impaired awareness, repetitive blinking and saccadic brief atonic manifestations of the trunk and upper limbs, recognizable when the child was sitting, causing nodding involving the head and arms, with sudden backwards or forwards falls (depending on the position of the child). When the patient was lying down and moving, only blinking and rhythmic left upper limb jerks were observed (video sequence 2). The atonic component was more pronounced in the left upper limb and was followed by rhythmic jerks corresponding to the recovery of the muscular tone, identifiable when the child moved her arms or when the arms were outstretched (video sequence 3, *figure 4*). Ictal video-EEG showed a sequence of diffuse, high-amplitude, 2-3-Hz SW during up to 20 seconds with maximal amplitude over the right central-parietal area (*figure 4*). The positive transient of the spike-wave shape on the right preceded the left one (*figure 5A*) figure 5. During seizures, the EMG showed a rhythmic muscle contraction of variable duration and amplitude, depending on the underlying muscular activity and predominating on the left deltoid (*figures 4, 5*). With outstretched arms during SW sequences, the duration of jerks was approximately 300 ms, whereas muscular silence on left deltoid EMG lasted for 190-300 ms (*figure 5A, B*). Back-averaging locked to the beginning of each contraction, revealed a central right negative SW with the spike preceding



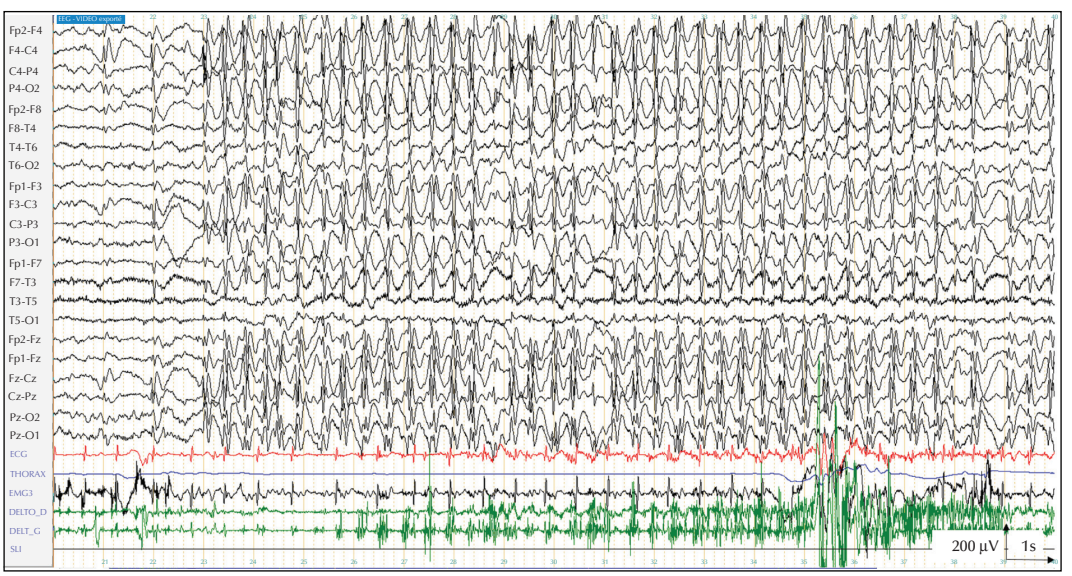
■ **Figure 2.** Ictal EEG with EMG on both deltoids (DELT D: right; DELT G: left) in Patient 1 with narrow window (A) illustrating the complex relationship between spikes and EMG contractions, and bilateral rhythmic deltoid contractions predominating on the left, increasing in duration and superposed on a gradual increase in muscular tone of both deltoids predominating on the left. Note an inhibitory component of the tonic activity between myoclonia interrupted by post-myoclonic silent periods. (B) Focus on spikes and their relationship awith EMG contractions.

the contraction with a variable latency of 240-330 ms (*figure 6A, B,-C*). Back-averaging locked to the onset of the left deltoid silence showed a negative SW at the right central electrode with a latency of 20-40 ms (*figure 7A, B, C*). The patient also presented with isolated atonic episodes of trapezius and both deltoids, predominating on the left, lasting for around 250 ms, following a right central SW with a latency of 50-60 ms (video sequence 3, *figure 8A, B, C*).

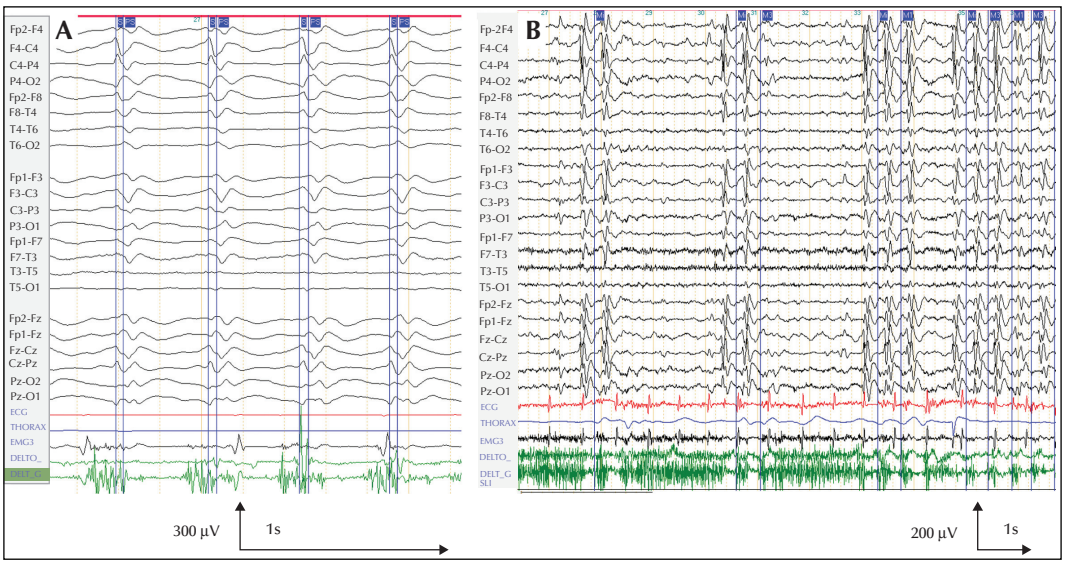
EEG during NREM sleep showed diffuse synchronous sub-continuous SW of 1-2 Hz, predominant on the right central-parietal area with a SW index during NREM sleep (defined as the percentage of time occupied by spike and wave discharges) of >50% of N-REM sleep duration (*figure 9*).



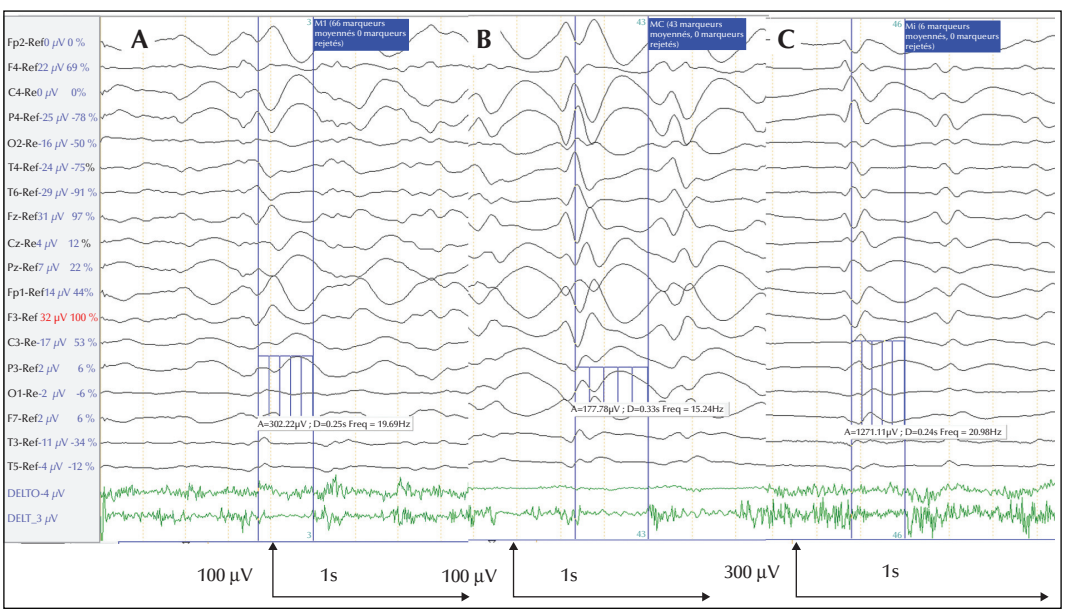
■ **Figure 3.** Back-averaging locked to the onset of myoclonic jerks during the myoclonic absence in Patient 1 using two different subsets of the first myoclonic jerks, (A) (n=32) and (B) (n=44), revealing bilateral synchronous spikes with a positive component at frontal (Fz, F3, F4) electrodes and a negative component at temporal-occipital electrodes, predominating on the right and preceding the myoclonic jerk by 20-30 ms. (C) The pre-myoclonic spike is represented on a 2D map illustrating bilateral tangential dipoles with inversion of polarity near central electrodes, predominant on the right.



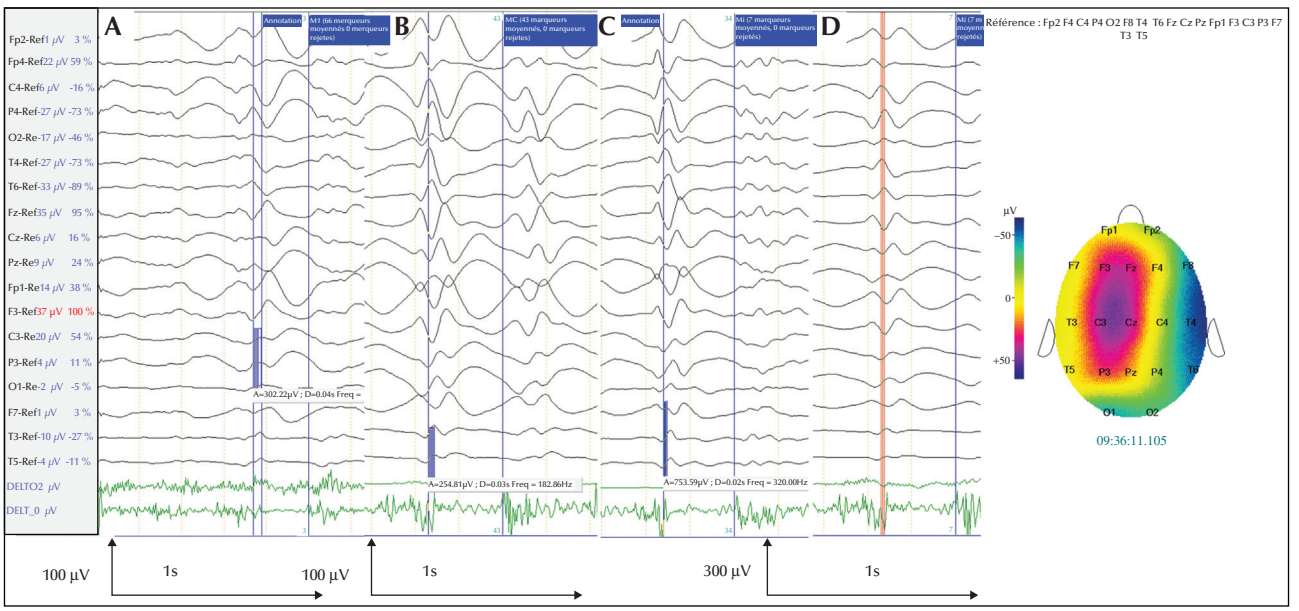
■ **Figure 4.** Ictal EEG (video sequence 2) with EMG of both deltoids and the neck (DELT D: right; DELT G: left; EMG3: neck) in Patient 2 showing a sequence of high-amplitude 2-3-Hz SW during up to 20 seconds with maximal amplitude over the right central-parietal area. Note bilateral rhythmic contractions predominating on the left deltoid corresponding to a sudden recovery of muscle tone after negative myoclonus on both deltoids.



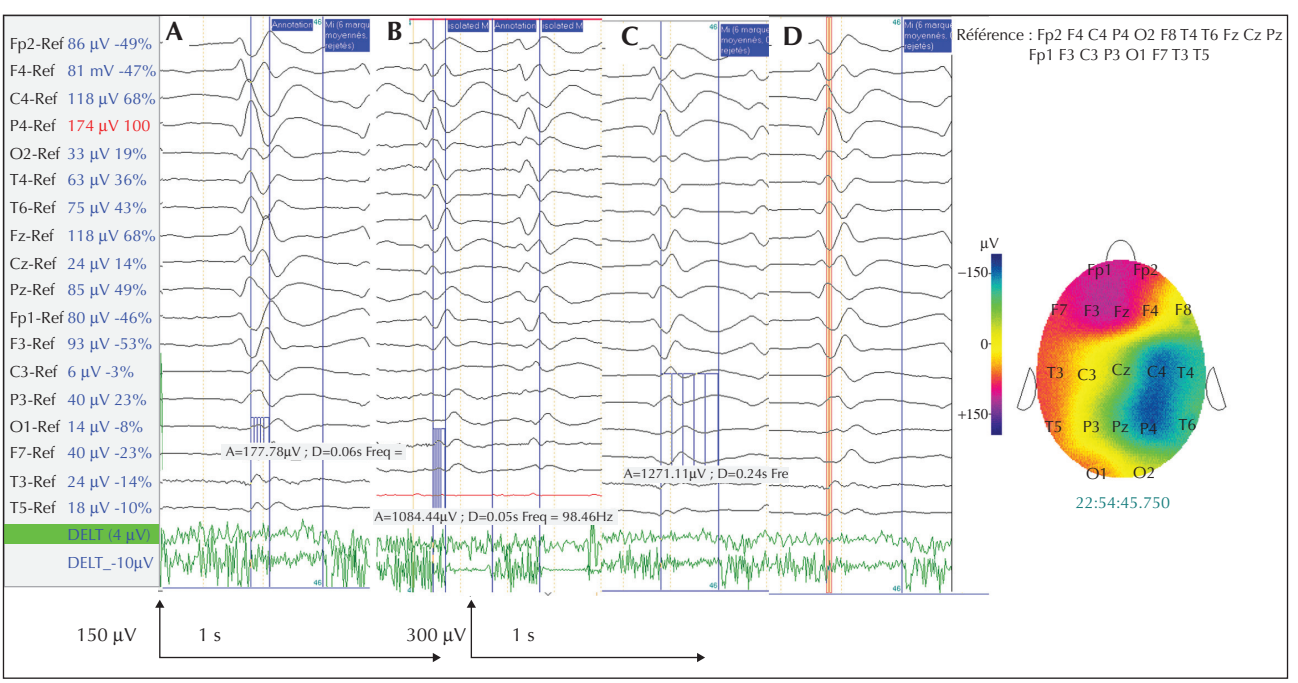
■ **Figure 5.** Ictal EEG (video sequence 2) with EMG of both deltoids and the neck (DELT D: right; DELT G: left; EMG3: neck) in Patient 2 with (A) the narrow window showing the sequence of diffuse, high-amplitude, 2-3-Hz SW with maximal amplitude over the right central-parietal area. The positive transient of the spike-wave shape on the right preceded that of the left. Note the relationship between the SW and the muscular contractions corresponding to the sudden recovery of muscle tone of both deltoids after the negative myoclonus. (B) Ictal EEG (video sequence 3) with EMG of both deltoids and the neck (DELT D: right; DELT G: left; EMG3: neck) in Patient 2 showing short bursts of bilateral, high-amplitude 2-3-Hz SW with maximal amplitude over the right central-parietal area. On EMG, note the contractions of both deltoids during the sequences of the SW and isolated negative myoclonias of the left deltoid and trapezius following a right central SW. EMG of the left deltoid and he neck showed a negative myoclonus lasting between 150 ms and 270 ms.



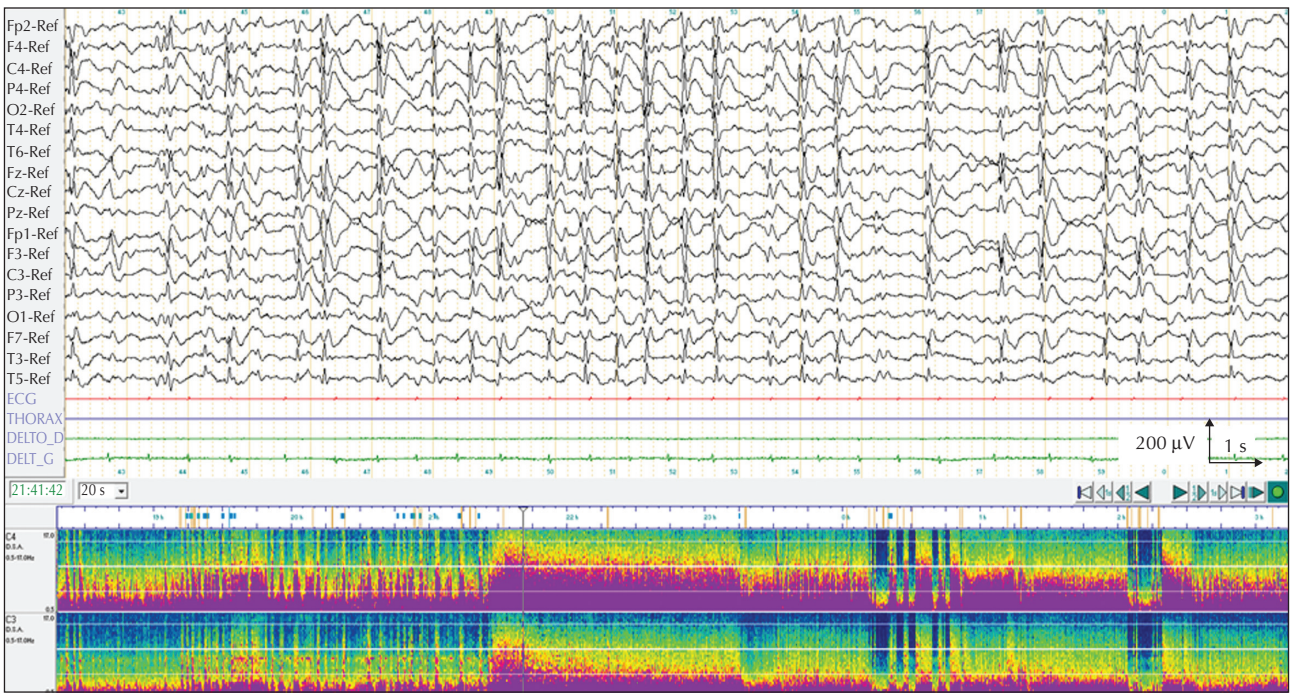
■ **Figure 6.** (A, B) Back-averaging in two subsets of jerks, (A) (n=66) and (B) (n=44), locked to the onset of the sudden recovery of muscle tone of both deltoids in Patient 2, revealed a central right negative SW in which the spike preceded the contraction with a variable latency of 240-330 ms. (C) Back-averaging using six isolated jerks.



■ **Figure 7.** Back-averaging locked to the onset of negative myoclonus of the left deltoid in Patient 2 using three subsets of events, (A) (n=66), (B) (n=43), (C) (n=7), showing a right central SW preceding the onset of the negative myoclonus by 20-40 ms. (D) 2D mapping of the same SW showing a right central tangential dipole.



■ **Figure 8.** (A, B) Back-averaging, locked to the onset of the isolated negative myoclonus on the left deltoid in Patient 2 (video sequence 3) showing a right central SW with latency of 50-60 ms. (C) Back-averaging locked to the onset of sudden recovery of muscle tone of both deltoids following a right central SW with a latency of 240 ms. (D) 2D map locked on the right central spike of the SW from (C) showing right frontal-central tangential dipole.



■ **Figure 9.** EEG during slow-wave sleep in Patient 2. Note the bilateral synchronous continuous spike waves of 1-2 Hz, predominant on the right during the whole duration of NREM sleep, visible on the spectral density analysis at C4 and C3 over a period of nine hours (lower panel).

Discussion

We report two types of rare seizures: myoclonic absences in epilepsy with myoclonic absences and atypical absences with atonic component in encephalopathy related to status epilepticus during slow sleep (ESES) [1, 3].

Both children presented with seizures with roughly similar clinical features, including incomplete impairment of awareness, repetitive blinking and asymmetrical jerks of the upper limbs. In addition, Patient 2 presented with a marked atonic component which was discernible on the neck on EMG (in the sitting position) and deltoid EMG (when the arms were outstretched or moving during seizures). Both patients presented with rhythmic contractions as well as briefs periods of silence, prominent on the left deltoid, on EMG. At first sight, ictal EEG aspects seemed to be comparable, showing sequences of diffuse rhythmic SW at 2-3 Hz in Patient 1, maximum over the right frontal-temporal area, and over the right central-parietal region in Patient 2.

Video-EEG and EMG-locked back-averaging revealed, however, a major difference in electroclinical phenomenology. Patient 1 presented with muscle jerks following frontal spikes with a latency of 20-30 ms, compatible with a myoclonus of epileptic origin, whereas in Patient 2, rhythmic jerks of the upper limbs were preceded by a spike with a time delay of 240-330 ms which does not correspond to corticospinal conduction (20 ms for upper limbs) [4-6]. In Patient 2, back-averaging locked to the onset of the muscular silence revealed, however, a right central SW complex, of which the spike preceded the event by around 20-60 ms, compatible with a negative myoclonus of epileptic origin (ENM). The motor manifestations in Patient 2 were therefore interpreted as a series of ENM which were each followed by a sudden recovery of muscle tone, perceived as a positive myoclonus. In ENM, the atonic component as well as the recovery of muscular tone can be overlooked when the patient is lying down or sitting with arms at rest. It was therefore crucial to ask the patient to outstretch arms in order to make the muscular tone and subsequent negative phenomena (silent period) visible on EMG polygraphy. To our knowledge, the clinical and electrophysiological correlate of the recovery of muscle tone after ENM have not been studied.

The myoclonic absence comprises a series of rhythmic myoclonic jerks of the upper limbs with impaired awareness and a pattern of typical absence on ictal EEG. The muscular background tone increases progressively during the seizure resulting in progressive elevation of the upper limbs [7]. After

each myoclonus, there is a very brief interruption of muscular activity due to a post-myoclonic silent period, soon replaced by the reappearance of tonic contraction and subsequent myoclonic jerks [8]. Asymmetry and even regional onset, as observed in our patient, are not rare [1].

In fact, distinction between clonus and myoclonus, when the latter occurs rhythmically, remains ambiguous. Previous classifications define clonus as a regular and prolonged myoclonus involving the same muscle group at a frequency of 2 to 3 Hz [9]. More recent classifications emphasize that clonus is a "sustained" rhythmic jerk, whereas myoclonus is a regular non-sustained jerk [2]. Indeed, "myoclonic absence" is an illustrative example of the difficulty of classification, as myoclonus in this condition is rather a rhythmic muscle contraction, thus corresponding to the definition of clonus (2.5-4.5 Hz). Some authors have even suggested that these seizures should be named differently [10]. Moreover, in the recent seizure classification, myoclonic absences are included as a group of "non-motor" generalized seizures [2].

In Patient 2, presenting with atypical absences with an atonic component, the NM occurred with a latency of 20-50 ms after the negative right central spike of the SW complex, compatible with ENM [11]. ENM is defined as an interruption of tonic muscle activity for <500 ms, which is time-locked to an epileptic EEG abnormality, without evidence of an antecedent positive myoclonia in the agonist-antagonist muscles [11]. Diagnosis of ENM therefore requires exclusion of positive myoclonus preceding the onset of the EMG silent period. We did not observe any positive myoclonus on EMG nor on the video preceding the ENM, particularly when the child presented with bursts or sequences of SW while she was at rest. Our analysis was, however, limited by the number of EMG recordings (only deltoids and neck) which precluded exclusion of a positive myoclonus involving facial muscles or eyelids preceding the ENM. Low-sampling frequency (256 Hz) also limited the accuracy of back-averaging and latency measurement.

Studies on the electrophysiological features of ENM, induced or not by antiepileptic drugs such as carbamazepine and lamotrigine, in atypical benign focal epilepsy in childhood and ESES, have previously been reported [12-16]. Authors found a latency between the spike peak and onset of ENM of 8-80 ms, depending of the intensity of the ENM (drop attack versus ENM limited to one upper limb), the amplitude of the spike, as well as the slow-wave component of the SW complex which was also significantly larger for drop attacks than localized ENM [12, 15]. Parmeggiani *et al*. [13] described an increase in the amplitude of the slow wave within the SW during the ENM and a strong correlation between the total duration of the atonic phenomenon and the total duration of the slow-wave component. They also reported that sustained, continuous muscle contraction of the orbicularis oris was frequently interrupted by brief muscular silent periods (100-400 ms), time-locked to a contralateral central-temporal SW, resulting in repetitive blinking [13].

ENM was recognized by a Task Force of the International League Against Epilepsy on Classification and Terminology as a seizure type in 2001, however, it is no longer included as a seizure type in the last seizure classification. ENM falls, therefore, within the category of generalized or focal motor "atonic seizures" [10, 17]. Myoclonic, atonic and myoclonic-atonic, as well as absence seizures, are frequent in childhood epilepsy syndromes and may present with misleading features and classification problems. Recording of EEG coupled to EMG, using jerks and silence-locked back-averaging, may be essential to properly assess the type of seizures. The precise characterization of ictal motor phenomena is not only crucial for the diagnosis of the seizure type and the electroclinical syndrome, but also for aetiological considerations and appropriate treatment choice. ■

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None of the authors have any conflict of interest to declare.

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Legend for video sequences

Video sequence 1

Myoclonic absence: impairment of awareness, a fixed gaze, deviation of the head to the right and rhythmic clonic movements of the head, eyelids and upper limbs predominating on the left side for 15-20 s.

Video sequence 2

Atypical absence with atonic component. When patient was lying, only blinking and rhythmic left upper limb jerks were observed.

Video sequence 3

Negative myoclonias, each one followed by rhythmic jerks corresponding to the recovery of the muscular tone identifiable only when the child moved her arms or when the arms were outstretched.

Key words for video research on www.epilepticdisorders.com

Phenomenology: myoclonic absence (video 1), atypical absence with atonic component (videos 2 and 3), patient sitting and outstretching her arms (videos 2 and 3), negative myoclonus (video 3)
Localization: generalized (videos 1, 2, 3)
Syndrome: epilepsy with myoclonic absences (video 1), encephalopathy related to status epilepticus during slow sleep (eses) (videos 2 and 3)
Aetiology: unknown (videos 1, 2, 3)

TEST YOURSELF

(1) What are the main epilepsy syndromes in childhood that may include, among seizures types, the negative cortical myoclonus?

(2) In clinical practice, what is the technical set up used to enhance negative myoclonus during EEG recording?

(3) What is the EEG correlate of cortical negative myoclonus?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".