increased efforts to recognize SPF and special therapeutic strategies for their avoidance or compensation, in addition to traditional pharmacotherapy or epilepsy surgery. Such treatment would increase the therapeutic potential and can help produce a better quality of life for the patients (at least in ''responders''). Furthermore the approach provides new possibilities for the patients to contribute to their own therapeutic process.

Conflict of interest: We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. None of the authors has any conflict of interest to disclose.

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Epilepsy and deletion syndromes of chromosome 18: Do not forget the short arm!

To the Editors:

Frequent clinical features of 18p- syndrome include mental retardation, postnatal growth retardation and dysmorphisms. Although this chromosomal syndrome does not appear to have a distinctive phenotype, epileptic seizures are uncommon in affected individuals. A recent review on chromosome 18 aberrations and epilepsy concluded that epilepsy is rare in individuals with 18p deletion syndromes (DS), while it occurs from 10% to 31% in subjects with 18q DS (Grosso et al., 2005).

We report a novel finding in a young male patient with normal karyotype (46,XY) and del(18)(p11.1pter). ish(18qter→18p11;tel 18p-) de novo who developed intractable epilepsy.

The child's family history was negative for epilepsy, febrile convulsions, mental retardation, and/or other neurological problems. At birth, child's weight was 2,820 g (10th centile), his head circumference was 35 cm (<45th centile), his length 48 cm (10–25th centile) and APGAR scores were 7 at 1 min and 8 at 5 min. Signs of psychomotor delay were noticed at an early age: he sat at 10 months, walked at 18 months and have never developed language skills.

At the first examination, the child was hypotonic and showed a pattern of facial dysmorphisms: brachycephaly, frontal bossing, hypertelorism, epicanthal folds, narrow nasal bridge, everted upper lip, microretrognathia; furthermore, pectus excavatum was noted. He had difficulties in social interaction, made little eye contact, and presented self-injury behavior. He presented a severe cognitive impairment and was tested with Autism Diagnostic Observation Schedule matching criteria for autistic disorder. Brain MRI and metabolic diseases screening were normal.

At age 6, the boy developed nocturnal seizures with prominent tonic and postural manifestations, automatisms, and occasional secondary generalization. Seizures became diurnal with frequent falling, not modified by antiepileptic drug (AED) therapy. Episodes persisted with a frequency of more than ten times per months. An electroencephalographic recording during sleep showed spikes and sharp waves localized of frontal-central leads and occurring synchronously or less often asynchronously (Fig. 1).

At age 9, the patient was referred for a genetic consultation. High-resolution chromosome analysis of lymphocytes showed a de novo deletion of the short arm of chromosome 18.

At present, despite AED treatment with sodium valproate and clobazam, the boy still has two to three seizures per week, often on awaking. The clinical features of the episodes include tonic posturing, loss of contact and versive movements. Frequent falling during seizure and autonomic signs were also observed. Previous AEDs therapies with carbamazepine, oxcarbazepine, lamotrigine and topiramate were discontinued for poor seizure control and idiosyncratic side effects.

With the exception of a Spanish 18p DS child presenting West syndrome (Vaquerizo et al., 1995), and one case with electroencephalographic anomalies but without seizures (Wester et al., 2006), epilepsy associated with deletion of the short arm of chromosome 18 has never been reported.

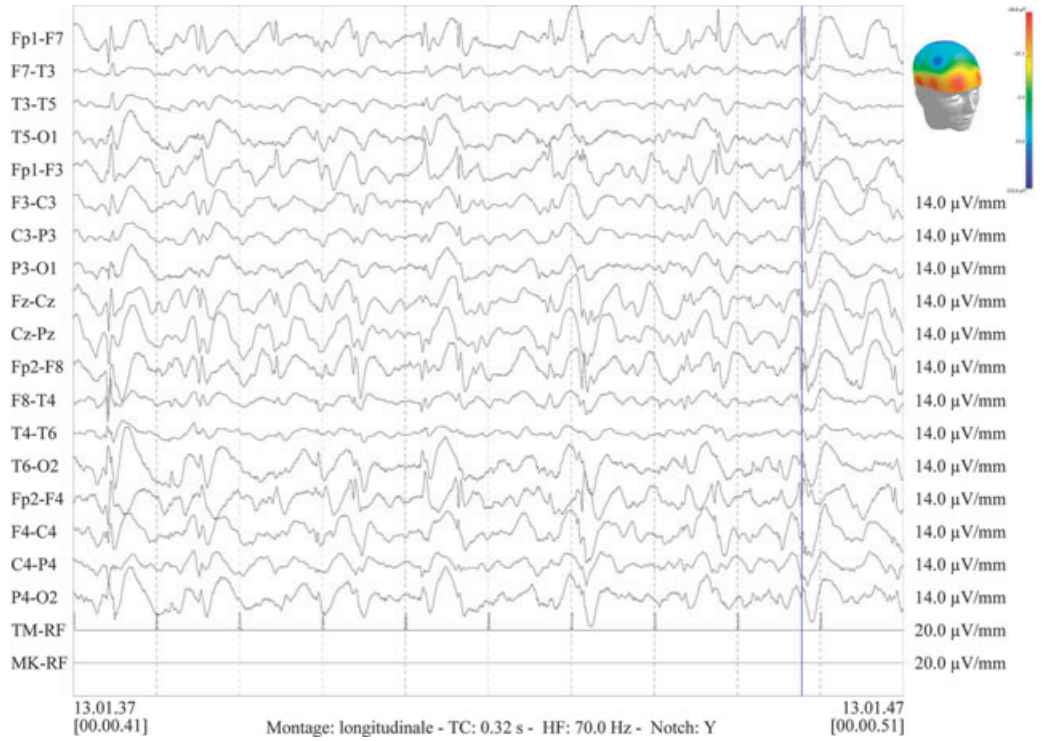

Figure 1.
Sleep EEG recording (stage II) performed at 7 years. EEG showing interictal spike and slow waves localized over frontal-central leads that occur synchronously. In the right side: Amplitude map of paroxysmal activity (maximal activity under F electrodes).
Epilepsia © ILAE

There are a few descriptions of the characteristics of epilepsy in patients with this chromosomal aberration, nor are the characteristics of the seizures or EEG features available.

In conclusion, it is very complicated to define whether the epileptic seizures are part of 18p DS, and to our knowledge, this is the first report of intractable epilepsy in a case with 18p DS. Additional reports are necessary to better define the characteristics and outcome of epilepsy in this chromosomal aberration.

Conflict of interest: We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. None of the authors has any conflict of interest to disclose.

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Delayed responses in TMS-EEG are different from SPES

To the Editors:

We read with great interest the paper by Valentin and colleagues recently published in *Epilepsia* (Valentin et al., 2008). This report was the first to evaluate transcranial magnetic stimulation electroencephalography (TMS-EEG) responses in epileptic patients, in comparison to healthy