SHARED LOCI FOR MIGRAINE AND EPILEPSY ON CHROMOSOMES 14q12-q23 AND 12q24.2-q24.3

John J. Millichap, Douglas R. Nordli, Jr., Chicago: We read with interest the recent article by Polvi et al.,1 who described shared loci on chromosomes 14q and 12q for migraine and epilepsy in a large Finnish family. Ten of the 60 family members lacked the most typical clinical symptoms of PS, nausea, retching, and vomiting, as well as the prerequisite occipital interictal spikes on EEG.2 The authors respond that the patients in their series lacked these signs and symptoms.3

Author Response: Auli L.M. Siren, Helsinki; Heikki Rantala, Oulu; Anne Polvi, Helsinki; Maija Wessman, Helsinki; Anna-Elina Lehesjoki, Helsinki, Finland: We thank Drs. Millichap and Nordli for their interest in our article.1 We investigated and discussed the clinical features and classification of the SUA attacks. Three reasons led us to use the term SUA. First, review of medical files and EEG reports and interviews of parents and family members revealed no other signs than unconsciousness resembling autonomic seizures or autonomic status epilepticus.3 Second, no EEG was recorded during any of the SUA events, so we cannot be definitive about the nature of the events. Third, it is possible that other autonomic signs or symptoms occurred but were not recorded. Since the most typical clinical and EEG findings of PS were lacking in our patients with SUA, we think it is very improbable that they had PS.

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Author response: Auli L.M. Siren, Helsinki; Heikki Rantala, Oulu; Anne Polvi, Helsinki; Maija Wessman, Helsinki; Anna-Elina Lehesjoki, Helsinki, Finland: We thank Dr. Panayiotopoulos et al. for their letter and expert opinion on the nature of SUA being similar to SL-PS in PS.1

We adopted a conservative view on the SUA events due to lack of EEG data and the narrow spectrum of autonomic features during the events reported in our patients. PS, as a recently acknowledged entity, is most likely underdiagnosed6 and this might have happened in our study. The upcoming article by Koutroumanidis et al.4 will hopefully further clarify this topic.

Although this family is a gem for genetic studies on epilepsy and migraine, to our knowledge no affected individuals with epilepsy or SUA exist in the next generation (descendants of individuals 17–29). When new data on the genetics of PS become available, we are ready to study the SUA/SL-PS patients.

Author disclosures are available upon request (journal@neurology.org).
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