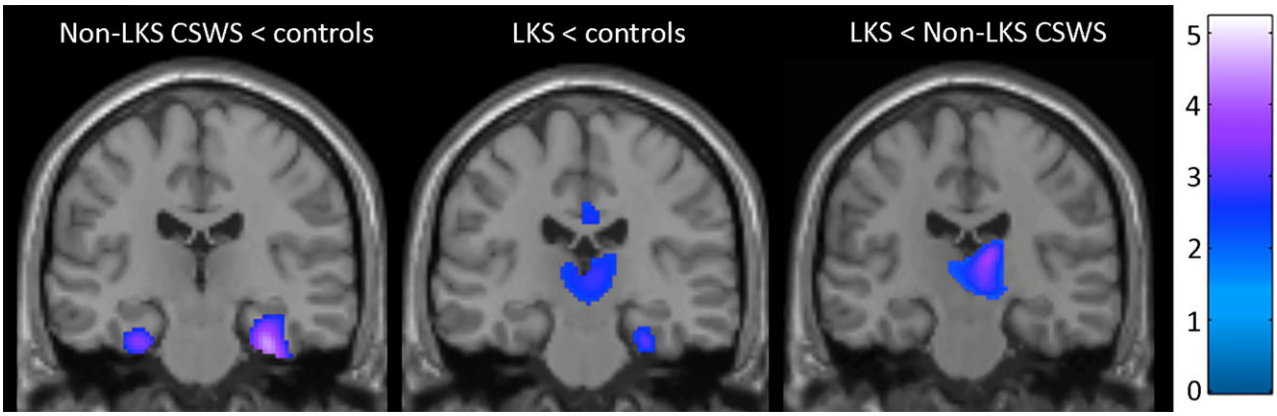

Figure 1.
Results of the statistical comparisons between non-LKS CSWS patients and pseudo-controls (left), LKS patients and pseudo-controls (middle), and LKS and non-LKS CSWS patients (right). For illustration purpose, images are thresholded at $p < 0.01$ uncorrected.
Epilepsia © ILAE

Acknowledgments

Xavier De Tiège is a postdoctoral clinical master specialist at the Fonds de la Recherche Scientifique (FRS-FNRS, Brussels, Belgium).

Disclosure

None of the authors has any conflict of interest to disclose. 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.

Nicola Trotta[1]
Noémie Ligot[1]
Frédérique Archambaud[2,3]*
Serge Goldman[1]
Patrick Van Bogaert[1]
Catherine Chiron[2,3]
Xavier De Tiège[1]
xdetiege@ulb.ac.be
[1]*Laboratoire de Cartographie fonctionnelle du Cerveau (LCFC), UNI – ULB Neuroscience Institute, Université libre de Bruxelles (ULB), Brussels, Belgium;*
[2]*Inserm U1129, Université Paris Descartes, Paris, France; and*
[3]*CEA, I2BM, Service Hospitalier Frédéric Joliot, Orsay, France*

**In memoriam*

References

1. Agarwal R, Kumar A, Tiwari VN, et al. Thalamic abnormalities in children with continuous spike-wave during slow-wave sleep: an F-18-fluorodeoxyglucose positron emission tomography perspective. *Epilepsia* 2016;57:263–271.
2. Ligot N, Archambaud F, Trotta N, et al. Default mode network hypometabolism in epileptic encephalopathies with CSWS. *Epilepsy Res* 2014;108:861–871.
3. Archambaud F, Bouilleret V, Hertz-Pannier L, et al. Optimizing statistical parametric mapping analysis of 18F-FDG PET in children. *EJNMMI Res* 2013;3:2.
4. Benedek K, Juhasz C, Muzik O, et al. Metabolic changes of subcortical structures in intractable focal epilepsy. *Epilepsia* 2004;45:1100–1105.

In response: No evidence of thalamic metabolic abnormality associated with continuous spike-and-wave during sleep

To the Editors:

We thank Trotta et al. for their critique and appreciate their efforts to evaluate thalamic metabolism quantitatively in their cohort of patients with continuous spike-and-wave in sleep (CSWS) using F-18-fluorodeoxyglucose positron emission tomography (FDG-PET).[1] Although their findings contrast with our results, it is noteworthy that their methodology remains fairly distinct.[2] Even in our cohort, not everybody had thalamic abnormality. In those who did, the abnormalities ranged from hypometabolism to hypermetabolism, as well as "increased asymmetry only" with thalamic FDG uptake in the apparently normal range. This emphasizes the complexity of the issue, with thalamic metabolism perhaps getting modulated by various clinical factors, which may confound the results of small sample-size studies with the nonhomogeneous patient population. Furthermore, a voxel-based analysis may not be the most appropriate approach for evaluating the aforementioned variability in thalamic FDG uptake due to possible averaging of the uptake values at the group level. Results of individual region-of-interest

analysis with subsequent analytical approach, as employed in our study, might have been more comparable with our findings.

We would like to highlight that several authors have reported structural thalamic abnormalities in patients with CSWS and that this relationship has been recognized for over a decade.[3–5] This association of a structural thalamic abnormality (visible on magnetic resonance imaging [MRI], not requiring statistical processing) and CSWS cannot be completely discounted. Similarly, in our patient group with CSWS and normal MRI findings, a priori qualitative analysis of glucose uptake on FDG-PET (by visual analysis) revealed thalamic abnormalities in 6 of 23 patients (patients 2, 6, 8, 10, 11, and 15). Despite the differences in the quantitative methods used between Trotta et al. and our group, it is hard to ignore this obvious pattern of abnormalities visible to the naked eye. Therefore, we do not concur with their conclusion that metabolic thalamic abnormalities are *not* associated with CSWS, although we do believe that this needs further exploration to be fully explained and understood.

Although studies on functional thalamic abnormalities in patients with CSWS are being investigated, various clinical and genetic factors may impact the development and pathophysiology of CSWS and thalamic abnormalities. Indeed, use of a standardized methodology with a larger patient population would be the way to go forward. Of course, given the rarity of this condition, this may be accomplished only by collaborative work of clinicians and scientists with expertise and interest in CSWS and anatomic/functional neuroimaging.

We acknowledge that thalamic abnormalities are seen in patients with epilepsy without CSWS; however, the proportion of patients who had thalamic abnormalities in our study far exceeds the expected proportion otherwise.[6] Thus, there appears to be a more specific role of thalamic metabolic abnormalities in patients with CSWS. We concur with their comment that the cortex is likely to be the primary generator of the epileptic discharges in CSWS patients and do not believe otherwise, but we disagree that the thalamus is merely a propagation node. Our hypothesis is that an abnormal thalamus leads to aberrant thalamocortical interactions, which in turn leads to CSWS, although this remains to be further validated.

Acknowledgments

None.

Disclosure

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

Rajkumar Agarwal[1,2]
ragarwal@med.wayne.edu

Ajay Kumar[1,2,3,4]
Vijay N. Tiwari[1,2,4]
Harry Chugani[1,2,4]
[1]Carman and Ann Adams Department of Pediatrics; Divisions of [2]Neurology; [3]Radiology; and [4]PET Center, Children's Hospital of Michigan and Wayne State University, Detroit, Michigan, U.S.A.

References

1. Trotta, Ligot N, Archambaud F, et al. No evidence of thalamic metabolic abnormality associated with continuous spike-and-wave during sleep. *Epilepsia* 2016;57:1007–1008.
2. Agarwal R, Kumar A, Tiwari VN, et al. Thalamic abnormalities in children with continuous spike-wave during slow-wave sleep: an F-18-fluorodeoxyglucose positron emission tomography perspective. *Epilepsia* 2016;57:263–271.
3. Guzzetta F, Battaglia D, Veredice C, et al. Early thalamic injury associated with epilepsy and continuous spike-wave during slow sleep. *Epilepsia* 2005;46:889–900.
4. Sánchez Fernández I, Takeoka M, Tas E, et al. Early thalamic lesions in patients with sleep-potentiated epileptiform activity. *Neurology* 2012;78:1721–1727.
5. Incorpora G, Pavone P, Smilari PG, et al. Late primary unilateral thalamic hemorrhage in infancy: report of two cases. *Neuropediatrics* 1999;30:264–267.
6. Chang CP, Yen DJ, Yu SM, et al. Unilateral thalamic hypometabolism in patients with temporal lobe epilepsy. *J Formos Med Assoc* 2008;107:567–571.

Elite athletes and epilepsy

To the Editors:

We read with interest the article entitled "Epilepsy, seizures, physical exercise, and sports: A report from the ILAE Task Force on Sports and Epilepsy."[1] The authors suggest an approach to issuing medical fitness certificates for people with epilepsy (PWE) based on assessing the potential risks of seizure occurrence in different sports, taking into account factors such as an individual's usual seizure characteristics (e.g., seizure type, triggers, pattern, and severity) and willingness to accept some degree of risk.

Despite a general move toward encouraging—rather than limiting—involvement in sports for PWE,[2] unfounded beliefs and fears regarding risks of injury and seizure exacerbation persist and can lead to the imposition of inappropriate restrictions by physicians or sporting bodies.[3–6] For athletes who develop epilepsy, such prohibitions can have career-ending consequences.

We suggest an approach using objective clinical data to counter potential barriers to participation in high-level sports for PWE and to empower PWE to make informed choices.