LETTERS

International summer school for neuropathology and epilepsy surgery in Chengdu, China, August 29–September 1, 2016

To the Editors:

In China, approximately 9 million people have epilepsy, which in 30–40% of patients is considered drug-resistant. Epilepsy surgery has been recognized as effective treatment option for a carefully selected cohort of patients, that is, patients with mesial temporal lobe epilepsy (MTLE). Neuropathologic workup of epilepsy surgery specimens must not only be considered as gold standard to confirm the etiopathology in a given patient, but also to help provide predictive measures for long-term disease management or when counseling a patient. What's more, scientific research of resected and well-characterized human brain tissue will be made available for a systematic exploration of the underlying pathomechanism of chronic epilepsy.

The gap in evidence-based neuropathologic diagnosis of epilepsy surgery specimens is, however, huge in China. Most neuropathology specialists must also work for general pathology services. In addition, there are no facilities for continuous medical education in microscopy and state-of-the-art histology protocols, which became mandatory in the area of epilepsy surgery (see International League Against Epilepsy [ILAE] consensus classification systems for focal cortical dysplasia [FCD][1] and hippocampal scle-

Figure 1.
INES group.
Epilepsia © ILAE

Figure 2.
Each afternoon, our tutors (Drs. Ingmar Blumcke, Roland Coras, and Hajime Miyata) instructed participants on how to identify and classify histopathologic patterns specific for each major disease category, that is, tumors, malformations of cortical development, or hippocampal sclerosis. The use of special stainings and immunohistochemistry to detect characteristic histopathologic patterns was also discussed in great detail.
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rosis [HS][2]; ILAE consensus recommendation for neuropathology workup).[3] To improve competence and practical skills in diagnostic workup and decision making, the ILAE, together with the Chinese Association Against Epilepsy (CAAE), developed and provided a first educational course and training program for neuropathology and epilepsy surgery in Chengdu, Sichuan province, People's Republic of China.

Thirty participants registered at the 4-day workshop, arriving from various epilepsy centers across China (Fig. 1). Participants were consultants, residents, or fellows, and also had varied medical training background (neuropathology, neurosurgery, or neurology). Each day started with a series of lectures introducing the broad spectrum of neuropathologic lesions, for example, HS, FCD, and neurodevelopmental tumors, and also discussing etiology-related challenges in the presurgical evaluation and surgical treatment from a clinician's perspective. Simultaneous translation of all lectures from Chinese to English (and reverse when lecture held in English) were offered to avoid the communication gap. Advanced training at the multihead microscope (Fig. 2), projecting microscope (Fig. 3), or using a virtual microscopy platform was performed in the afternoon to empower histopathology skills and standard operational procedures. We thank our distinguished guest tutors Dr. Ingmar Blumcke (Erlangen, Germany), Dr. Roland Coras (Erlangen, Germany), and Dr. Hajime Miyata (Akita, Japan) for their continuous efforts. Distinguished experts were also invited from epilepsy centers across China to comprehend our course).

Details of the program of this first workshop can be obtained from the organizers (Prof. Dong Zhou zhoudong66@yahoo.de and Dr Jinmei Li jinmeili-neuro @qq.com). First day: diagnostic principles in neuropathology workup and ILAE classification of HS. Second day:

Figure 3.
Participants also had the opportunity to review and discuss cases from their own clinical practice at a multihead microscope.
Epilepsia © ILAE

vascular malformations and principles in magnetic resonance imaging (MRI) in focal epilepsy. Third day: ILAE classification of FCD and malformations of cortical development. Fourth day: long-term epilepsy–associated brain tumors. The application of special immunohistochemical stainings were presented and discussed in detail for different lesion entities, e.g., various neuronal and glial marker proteins.[3] A booklet summarizing the content of workshop lectures was made available to each participant, including a summary of cases presented during practical microscopy teaching sessions.

All participants acknowledged the course as being most helpful for their daily medical routine, applying ILAE's consensus classification systems, and recognizing the

spectrum of HS subtypes, FCD subtypes, and tumor entities. Another important outcome will be the foundation of a Chinese neuropathology task force under the umbrella of CAAE (endorsed by the ILAE Task Force for Neuropathology). The task force will develop a training and teaching curriculum for neuropathology in epilepsy surgery centers in China. This network will also develop a web-based virtual microscope platform to discuss difficult-to-classify cases among neuropathology colleagues from associated Chinese epilepsy centers, and also to continuously train neuropathology colleagues to become specialized in epilepsy surgery.

Disclosure

None of the authors have any conflicts to declare. 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.

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Added value and limitations of electrical source localization

To the Editors:

In two interesting articles, Russo et al.[1,2] showed the utility of three-dimensional electroencephalography source imaging (3D-ESI) with low-resolution electroencephalographic data (32 channels) in the pediatric noninvasive presurgical evaluation. In their first study, 3D-ESI localized the sources within the surgical resection cavity (SRC) in 65% of all cases, and in 78.6% of magnetic resonance imaging (MRI)–negative cases, which are particularly challenging cases for epilepsy surgery. The main methodological issue raised in their first paper is that not all the operated patients considered here were significantly improved (Engel class I or II) after surgery, suggesting that some of the sources localized in the SRC may actually have been mislocalizing. Unfortunately, the details of the population outcome after surgery were not given in the first publication. In the second, this is particularly crucial, since 2 years after surgery, 7 of the 14 reported MRI-negative cases were Engel class III or IV.

Surprisingly, Russo et al. "found no comparative studies investigating 3D-ESI in MRI-negative and MRI-positive cases." In a previous study,[3] we prospectively showed that interictal ESI had a better concordance with the stereo electroencephalography (SEEG)–defined ictal-onset zone in MRI-negative than in MRI-positive cases[3] (respectively, 100% in MRI-negative subjects versus 83% in MRI-positive cases). Russo et al. also showed a better localizing value of low-resolution ESI when the SRC was localized within the temporal lobe (84.6% vs. 48% in extratemporal lobe). This result is somehow intriguing, knowing that low-resolution ESI generally has lower spatial sampling precisely in the basal temporal region,[3] with an increasing source-localization accuracy from 31 to 64 and 123 electrodes.[4] However, this discrepancy is regrettably not discussed, and we wonder whether it could be related to the limitations of the reference methods.

Finally, the methods used to calculate the specificity calls for some comments. Specificity was indeed defined as the ratio of patients with source localization outside the SRC and poor outcome to all patients with an unfavorable outcome after surgery. Although the use of SRC as reference is especially meaningful in assessing sensitivity, since this constitutes an unambiguous proof of correct seizure-onset localization, it is more debatable when it comes to assess the specificity. A drawback of this latter definition is indeed that surgical failure does not necessarily rule out the epileptogenicity of resected tissue but is often due to its partial resection.[5] Another drawback of this definition is the consideration of any ESI localization beyond the resection volume in cases of surgical failure as a "true" localization, whereas nonresected areas encompass both epileptogenic and nonepileptogenic regions, which results in overestimating the specificity. We strongly believe that these points should be discussed in order to provide a balanced evaluation of this powerful diagnostic tool that is more and more widely used.

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.

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Response: Added value and limitations of electrical source localization

To the Editors:

In their commentary on our two articles[1,2] Rikir et al. offer several insightful observations and pose a series of questions regarding the reliability of electrical source imaging (ESI). The respondents suggest that a negative outcome in patients whose three-dimensional electroencephalographic source imaging localizes within the surgical resection cavity (SRC) implies mis-localization, We believe this conclusion could also logically apply to every localizing tool utilized in the epilepsy presurgical evaluation including ictal single-photon emission computed tomography (SPECT), interictal fluorodeoxyglucose–positron emission tomography (FDG-PET), subtraction ictal SPECT co-registered to magnetic resonance imaging (MRI) (SISCOM), and magnetoencephalography (MEG). It is now well accepted that no single methodology is perfectly localizing and that a concordance of different data is required before offering epilepsy surgery to patients.

Rikir et al. underline their previous prospective study,[3] in the adult population demonstrating concordance of ESI with stereo electroencephalography (SEEG) in the epilepsy surgical evaluation. In particular they showed a better concordance with the SEEG-defined ictal-onset zone in MRI-negative than in MRI-positive cases (respectively, 64% in MRI-negative subjects versus 18% in MRI-positive cases, considering the fully concordant data). However, the authors do not provide sufficient postsurgical data to allow a comparison of their results with ours. In fact, of their 28 subjects reported, 10 did not undergo resection, many for functional reasons, and 4 remaining subjects had a poor outcome (Engel class III or IV). We previously pointed out in our first publication that not all the operated patients were significantly improved (Engel class I or II).

Furthermore, although delineation of the EZ and mapping are listed in their study, all of their patients with malformations of cortical development (MCDs) underwent SEEG, although the indication for SEEG was not well defined. We typically do not implant electrodes in MRI-positive patients unless there is a need to map nearby eloquent cortex or there are discordant data. We believe that this protocol is fully consistent with the recent International League Against Epilepsy (ILAE) guidelines for invasive EEG.[4]

Rikir et al. also commented on our finding of improved localization in the temporal versus extratemporal dipoles with low-resolution three-dimensional (3D)-ESI. We typically place electrodes beyond standard channels only after reviewing all patient data, including semiology, EEG, and MRI data. Nonuniform electrode placement has been evaluated in simulated dipoles[5] and provides local high-resolution recording. This technique likely eliminates many of the known difficulties associated with localizing temporal sources. Prior to monitoring, we add sub temporal electrodes bilaterally in cases that do not have a well-documented semiology or EEG abnormalities. This strategy overcomes many of the limitations of the standard 10-20 placement for sampling the basal temporal regions.

With regard to our definition of specificity, we agree that it is difficult to determine the best definition in complex cases. In both studies in which the Rotating Dipole (RD) solution was considered outside the SRC, it was found to be fully outside. Given that the RD should represent the starting point of the ictal discharge, it is insufficient to focus on incomplete resection of the epileptic zone (EZ) rather than the specificity of the 3D-ESI. Furthermore, in our second study we analyzed the moving dipole

solution to minimize poor outcome due to partial resection of the EZ with excellent results, and surprisingly the moving dipole was useful only when the RD was inside the SRC.

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.

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Electroconvulsive therapy and epilepsy

To the Editors:

Recently, Bryson et al.[1] published a paper entitled "Temporal lobe epilepsy following maintenance electroconvulsive therapy—Electrical kindling in the human brain?" In their manuscript, they described five patients who received electroconvulsive therapy (ECT) and developed temporal epileptiform discharges on electroencephalography (EEG) despite no previous history of epilepsy. They reported that three patients had epileptic seizures. After cessation of ECT their EEG findings normalized and no further clinical seizures occurred. These authors concluded that maintenance ECT is potentially hazardous. They recommended that EEG should be performed regularly for patients receiving long-term ECT.[1]

Herein, I would like to argue that their study has a major limitation: they did not reintroduce ECT after they stopped it and when the patients' EEG findings normalized to verify the existence of any cause and effect relationship between ECT and epilepsy that they have hypothesized in their manuscript. Of course, one may argue that it was not ethically feasible to do so, but this does not eliminate that major limitation in order to establish a cause and effect relationship between ECT and epilepsy. Ironically, ECT has been employed as a treatment for refractory epilepsy and status epilepticus in a few anecdotal reports, sometimes successfully.[2–4] In addition, ECT has not been found to cause epilepsy in two large studies.[5,6] In one study of 166 patients who had received ECT, the prevalence of epilepsy did not differ significantly from that in the general population.[5] In another study of 619 patients, there was no report of spontaneous seizures.[6] I should also mention that there are some other studies that suggested otherwise and are consistent with the Bryson's observation.[7] It is probably fair to say that epidemiologic data do not suggest that ECT causes epilepsy. However, when a patient who is receiving ECT develops spontaneous epileptic seizures, one is challenged with the question as to whether ECT caused it. To answer to this question, we should bear in mind the possibility of coexistent epilepsy and psychiatric disorders, the chance of seizures happening as adverse effects of psychiatric drugs, and other potentially confounding factors (e.g., family history of epilepsy).

In brief, this is too premature to suggest that ECT is potentially hazardous and that routine EEG should be performed for patients receiving this therapy based on this observation. Well-designed studies are required to establish any potential relationship between ECT and epilepsy.

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Disclosure of Conflict of Interest

Ali A. Asadi-Pooya, M.D., consultant: Cerebral Therapeutics, LLC and UCB Pharma; Honorarium: Hospital Physician Board Review Manual; Royalty: Oxford University Press (Book publication). I confirm that I have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Response: Can ECT cause "kindling" in the human brain ?

To the Editor:

Thank you for your interest in our report in which we describe five patients who developed florid temporal epileptiform abnormalities on electroencephalography (EEG), including three who developed clinical epileptic seizures while receiving electroconvulsive therapy (ECT) and despite no history of epilepsy.[1] Dr Asadi-Pooya has queried whether a causal relationship can be inferred from these findings, as previous studies have not found an association between ECT and epilepsy, and ECT has been used to control seizures in some patients.

Studies that have reported no association between ECT and epilepsy, including the two referenced by Dr Asadi-Pooya, differ significantly from our series in patient cohort and methodology.[2,3] In the study by Ray, the median number of ECT sessions was 7, and 57% of patients received between 6 and 8 sessions. EEG findings were not documented, and assessment was performed retrospectively using patient files. In the study of Blackwood et al.,[3] the mean number of ECT sessions was 16.8 (range 1–75) and the mean duration between last ECT course and clinical assessment was 18 months. Conversely, the mean number of ECT sessions in our series was 174.6 (range 36–348) and all patients were assessed during their ECT course. Of two patients receiving fewer than 100 sessions, both had total treatment durations <12 months. Our patients were assessed under particularly intensive and prolonged treatment regimens. Of interest, these regimens fit more closely with animals models of kindling, as raised in the introduction of Blackwood et al. Specifically, frequent and regular electrical stimulations appear most effective at inducing this phenomenon.[4]

Dr Asadi-Pooya has pointed out that there is evidence that ECT has anticonvulsant properties in certain situations. However, this does not exclude the possibility of a proconvulsant effect through other mechanisms. As raised in our discussion, there is neuroimaging evidence to support a differential impact of ECT on different brain networks.[5] Thus ECT may suppress seizures in networks sustaining generalized convulsions while promoting seizure threshold in mesiotemporal (limbic) networks.

Although reintroducing ECT and observing a recurrence of epileptiform changes on EEG would strengthen a causal link, we felt this would be unethical. The dramatic resolution of EEG abnormalities on cessation of ECT argues that it is most likely that the EEG changes are causally related. To the best of our knowledge, the alternative possibilities raised by Dr Asadi-Pooya were excluded: psychiatric drugs were not reduced (in one patient the clozapine dose was increased), neuroimaging findings were normal, and there was no known past or family history of epilepsy.

Following these initial observations, our psychiatry colleagues have a lower threshold for ordering EEG on patients receiving prolonged courses of ECT, and we have observed temporal lobe epileptiform discharges in several other patients. However, we agree that a larger study is required to determine the prevalence of these changes. All subjects would require an epilepsy clinical history, neuroimaging, and EEG prior to commencement of the ECT course, and at regular intervals during the treatment course. If epileptiform changes or seizures developed, cessation of ECT would be recommended and follow-up EEG could be performed.

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.

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ANNOUNCEMENTS

32nd International Epilepsy Congress

2–6 September 2017
Barcelona, Spain
Congress website: www.epilepsybarcelona2017.org

Regional Congresses

4th East Mediterranean Epilepsy Congress

16–18 February 2017
Luxor, Egypt
Information: luxor@epilepsycongress.org

3rd African Epilepsy Congress

5–7 May 2017
Dakar, Senegal

12th Asian and Oceanian Epilepsy Congress

21–24 June 2018
Bali, Indonesia
Website: www.epilepsybali2018.org

13th European Congress on Epileptology

26–28 August 2018
Vienna, Austria
Website: www.epilepsyvienna2018.org
Call for session proposals

Upcoming Chapter Congresses

10th Qatar Epilepsy Symposium

3–4 February 2017
Doha, Qatar

ECON 2017: 18th Joint Annual Conference: Indian Epilepsy Association & Indian Epilepsy Society

17–19 February 2017
Patna (Bihar), India
Website: www.econ2017.com

Annual Meeting of the Austrian and German Society for Epileptology and the Swiss Epilepsy-League

3–6 May 2017
Vienna, Austria
Website: www.epilepsie-tagung.de

Other Congresses

5th International EMINS

13–14 January 2017
Dubai

To participate, or to suggest preferred topics for the congress, contact the EMINS Board: Dr Jihad Inshasi, MD, FAAN, FEAN: drjihadinshasi@gmail.com; or emins.uae@gmail.com

7th EPODES Advanced II: Comprehensive Epileptic Surgery Course

16–20 January 2017
Brno, Czech Republic
Announcement | Program
Website: www.ta-service.cz/epodes2017

Padua 2017: Recent Advances in Epileptology

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Padova Orto Botanica, Italy
Flyer | website: www.epilessiapadova2017.org

The 11th World Congress on Controversies in Neurology (CONy)

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6th London-Innsbruck Colloquium on Status Epilepticus and Acute Seizures

6–8 April, 2017
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www.statusepilepticus.eu

1st North American Workshop on Neuropathology and Epilepsy Surgery

27–30 April 2017
Presented by Cleveland Clinic Epilepsy Center
Cleveland, OH, USA
Flyer

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www.epilepsy2017.com

First International Training Course on Neuroimaging of Epilepsy

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Poster | Information
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15–18 June 2017
Miami Beach, FL, USA

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Lifelong course of diseases of the child's nervous system.
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Website: www.epns2017.com

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Advanced International Course: Bridging Basic with Clinical Epileptology – 6

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Information

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Clinically oriented, and focused on comprehensive aspects of diagnosis and treatment of epilepsy.
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28 August–1 September, 2017
Mon St Benet, Barcelona, Spain
Announcement | WONOEP Report
Abstract Submission form: Abstract submission closes 15 January

XIII World Congress of Neurology

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Kyoto, Japan
Website: www.2017.wcn-neurology.com/

7th Eilat International Educational Course: Pharmacological Treatment of Epilepsy

15–20 October 2017
Jerusalem, Israel
Website: www.eilatedu2017.com – Course and bursary information, applications

2018 Congresses

31st International Congress of Clinical Neurophysiology (ICCN): of the International Federation of Clinical Neurophysiology (IFCN)

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ICCN 2018 Website: http://iccn2018.acns.org/

6th Global Symposium on Ketogenic Therapies for Neurological Disorders

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