Title
EEG in Nonagenarians: Author Reply

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Interictal Epileptiform Discharges and Epilepsy Prediction

To the Editor:

I read with interest the article by Dr. So (2010) who, after an extensive review of the literature, concluded that the presence of interictal epileptiform discharges (IEDs) on the EEG carries a higher risk of "seizures" in patients with neurologic symptoms compared with healthy individuals with the same EEG pattern. Dr. So also asserts that the risk of "epilepsy" in patients with IEDs remains unknown.

Controversy exists surrounding the correct definition of sharp waves and spikes. A definition that has consistently appeared in the literature is as follows: "distinct waves or complexes distinguishable from the background activity, and with a sharp configuration." This definition, however, is ambiguous. A more objective definition was introduced years ago, at a symposium of the American Clinical Neurophysiological Society, as follows: "a waveform that is characterized by an amplitude of 2.5 times that of the underlying background with a duration of either less than 70 milliseconds (spikes) or longer than 70 milliseconds (sharp waves)." It should be noted that the terms sharp waves and spikes may not always be interchangeable in different clinical settings. For example, there have been anecdotal reports of a higher incidence of sharp waves rather than spikes in slowly growing lesions such as supratentorial tumors and in slowly progressive pathologic processes as observed in Alzheimer's disease. Conceivably, the differences in the definitions applied to IEDs may have partly accounted for the discrepancies noted in the EEG literature. Also, the "negative afterdischarge" as a component of the IEDs and its amplitude may be an important parameter, deserving further investigation.

Unlike sharp waves and spikes, "sharp slow waves" (to be distinguished from sharp wave complexes) have been vastly overlooked in the literature. Indications are that this pattern may have significant implications in connection with epilepsy diagnosis, cause, and severity.

The presence of IEDs poses a serious concern in epilepsy legal defense when aggression or crime may be interpreted as a manifestation of epileptic mental automatisms in an otherwise healthy offender with or without a history of aggressive or criminal behavior. In such instances, where neurologists may be asked to testify as expert witnesses, circumstances surrounding the crime, a thorough medicolegal history, detailed clinical data, and a period of hospital-based observation with video-EEG monitoring are required to resolve the issue by establishing or refuting criminal liability.

Finally, as Dr. So suggests, future studies of IEDs should eschew the "methodological shortcomings" of the past. Moreover, particular emphasis should be placed on parameters germane to IEDs, including morphology, topography, frequency, and activation, by provocative procedures (hyperventilation, photic stimulation, and sleep).

Abdorasool Janati, MD
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REFERENCE

So EL. Interictal epileptiform discharges in persons without a history of seizures: what do they mean? *J Clin Neurophysiol.* 2010;27:229–238.

Commentary on Letter to the Editor Entitled "Interictal Epileptiform Discharges and Epilepsy Prediction"

In Reply:

I thank Dr. Janati for his interest in my review article. Dr. Janati provided an alternate definition for interictal epileptiform discharges. I am pleased that Dr. Janati agrees that the task remains with future studies that are well designed to determine the association between these interictal discharges and subsequent risk of epilepsy in persons without prior seizure history.

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EEG in Nonagenarians

To the Editor:

I read with interest the article by Peltz et al. (2010) who reported on 12 previously healthy nonagenarians, the majority of whom (10) had diffuse background or focal EEG abnormalities.

It seems that the authors implicitly draw a distinction between "intermittent polymorphic delta slowing" in the temporal regions in their patients and a physiological variant in the elderly, characterized by occasional, random, low-voltage, and reactive polymorphic delta transients in the same topographical distribution, often with left-sided dominance. However, if the characteristics of these two patterns are similar (with the exception of frequency), then the polymorphic focal delta in the authors' series may in fact signify an advanced phase in the evolution of physiological focal delta transients of senescence, hence a normal variant for age.

Temporal, intermittent, rhythmic delta activity (TIRDA) as reported in this series may emanate from a deep hemispheric white matter pathology ipsilaterally and, as such, should not be considered an epileptic pattern. Thus, the pathophysiological mechanisms of TIRDA in this age group may be different from those of the TIRDA in younger patients with partial complex seizures.

The authors have reported physiological variants including mu rhythms and wicket spikes in their patients but do not specify whether they encountered other variants commonly seen in old age such as occasional brief bursts of rhythmic theta activity in the temporal regions and occasional random single sharp transients in the same areas. Also, it would be important to know whether there was a paucity of sleep spindles in this series, as expected in this age group. Furthermore, asymmetries of sleep parameters, if present, would have helped further define the pathologic significance of the "focal abnormalities."

It is surprising that neuroimaging data are conspicuously missing in this study, with no factual information available concerning neuropathological correlates of the EEG findings.

Future studies on this topic should define the parameters applicable to focal slowing including amplitude, reactivity to eye opening, accentuation or attenuation by drowsiness and sleep, response to hyperventilation, prevalence and duration of TIRDA, and prevalence of polymorphic focal delta. Also, frequency of focal sharp transients and sleep characteristics should be the subject of further investigation. These data should be complemented by neuroimaging studies, and by a long period of outpatient observation combined with follow-up EEGs, to confirm the hypothesis of Peltz et al. that the EEG patterns described in their study are indeed abnormal in nonagenarians.

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REFERENCE

Peltz CB, Kim HL, Kawas CH. Abnormal EEG's in cognitively and physically healthy oldest old: findings from the 90+ study. *J Clin Neurophysiol.* 2010;27:292–295.

EEG in Nonagenarians: Author Reply

In Reply:

We greatly appreciate the comments from Dr. Janati on our recent article.

Distinguishing normal, age-related temporal theta slowing from abnormal intermittent polymorphic delta slowing in the extreme elderly is challenging. In our study, only EEG activity that was disproportionately delta, not isolated to drowsiness, repeated multiple times in the recording, and lasting at least 1 second was called intermittent polymorphic delta slowing. However, as Dr. Janati pointed out, it is possible that the temporal slowing we report is a more advanced form of the temporal theta slowing commonly found in younger elderly. In this case, the temporal delta slowing considered "abnormal" at younger ages may, in fact, be "normal" in mentally and physically healthy people aged 90 years and older.

The source of the temporal intermittent rhythmic delta activity (TIRDA) found in our asymptomatic, oldest-old participants is unknown. However, we note that previous studies have found that focal intermittent rhythmic delta generally has been ascribed to abnormalities of thalamocortical pathways and interactions. We agree with Dr. Janati that additional studies in the oldest-old using neuroimaging may provide some information regarding the source of TIRDA in this extreme age group.

In our article, we reported that a few participants exhibited mu rhythms and wicket spikes. Dr. Janati inquired about other normal variants commonly seen in older age groups; however, we did not see any of these activity patterns in our small group of oldest-old participants. Future studies with larger numbers of participants will be able to more fully determine the frequency of the other variants in the oldest-old.

We agree with Dr. Janati that additional research is necessary to untangle what is normal in EEGs in the oldest-old.

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EEG Source Analysis—Time for a Common Language

To the Editor:

The use of dipole and distributed modeling to help characterize the epileptogenic zone in patients with focal epilepsy is an evolving but equally promising science. These models are based on signal information contained in the noninvasively acquired (scalp) EEG recording. The method lends an extra degree of dimensionality to the two-dimensional EEG trace because the electrical source is modeled in three-dimensional electrical space. With the aid of coregistration techniques, the spatiotemporal behavior of the modeled electrical source can be analyzed in the same frame as the patient's MRI, positron emission tomography, single photon emission computed tomography, or EEG-functional magnetic resonance imaging result. In this sense, EEG source analysis brings with it an unmatched temporal resolution to the "multimodal" search for the epileptogenic zone (or, more pragmatically, the spike- or seizure-onset zone). The patients who stand to gain most from these combined technologies are those who represent to our hospitals and emergency rooms, patients with medically refractory focal epilepsy. One of the upshots of EEG source analysis is that it has highlighted the degree to which localizing information contained in the standard EEG recording is underused, information that could be used to guide, limit, or even eliminate the use of invasive EEG monitoring (subdural grids and depth electrodes) in surgical candidates.

The last decade has seen a relatively sharp jump in the level of mathematical sophistication attached to the use of these models. The current problem though is that counterpart clinical studies have struggled to keep pace in establishing the clinical utility and the clinical validity of the various models across the various focal epilepsy syndromes encountered in routine clinical practice. Perhaps as a consequence, there is little standardization of the technique. Most clinical investigators choose an approach or algorithm with which they are most familiar and apply it to a particular patient cohort. Comparative clinical studies are genuinely lacking. It is likely that this lack of standardization of approach has hampered the translation of EEG source analysis to the clinical mainstream.

One simple (but not inconsiderable) step would be for current researchers to come down on an agreed-upon term to label the science. In a list that is far from exhaustive, we now have Dipole Modeling, Distributed Modeling, Dipole Source Analysis, EEG Source Imaging, EEG Source Localization, Electrical Source Imaging, Inverse Modeling, Equivalent Dipole Modeling, and Current Density Reconstruction.

Because these methods are fundamentally trying to "model" the spike or seizure-onset zone, this term should perhaps be retained. In this sense, EEG source analysis should not be construed as a method that directly "images" the putative epileptic focus (in the manner that MRI directly images the putative epileptic lesion), so the term "imaging" might best be avoided. The more generic label "EEG Source Modeling" could be used to encompass dipole and distributed methods and their variants (present and future). The same kind of argument might be applied to Magnetoencephalographic (MEG) Source Modeling methods.

Is it time then for a common language, such as EEG Source Modeling, to label this discipline? While it might be regarded as a trivial point, the use of an agreed-upon term to describe the science is surely a necessary step toward its potential acceptance in the wider clinical community.

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