Importance. The increasing use of continuous electroencephalography (EEG) monitoring in the intensive care unit has led to recognition of new EEG patterns that are of unclear or unknown significance.

Objective. To describe an EEG pattern, lateralized rhythmic delta activity (LRDA), encountered in critically ill subjects and determine its clinical significance in this setting.

Design, Setting, and Participants. Retrospective review at an academic medical center of EEG recordings, medical records, and imaging studies of critically ill patients with LRDA and comparison with subjects with lateralized periodic discharges (also known as periodic lateralized epileptiform discharges), subjects with focal nonrhythmic slowing, and controls.

Intervention. Electroencephalography or continuous electroencephalography.

Main Outcomes and Measures. Cross-sectional prevalence of lateralized rhythmic delta activity; EEG characteristics; etiology, clinical, and radiological correlates; and risk of early seizures.

Results. We identified LRDA in 4.7% of acutely ill subjects undergoing EEG or continuous EEG monitoring. It was often associated with other focal EEG abnormalities, including lateralized periodic discharges in 44% of cases. The most common conditions associated with LRDA were intracranial hemorrhage and subarachnoid hemorrhage. Lateralized rhythmic delta activity was an independent predictor of acute seizures, with 63% of subjects having seizures during their acute illness, a proportion similar to subjects with lateralized periodic discharges (57%) and significantly higher than associated with focal nonrhythmic slowing (20%) or in control subjects (16%). Most patients (80%-90%) in the LRDA and lateralized periodic discharges groups who had seizures while undergoing continuous EEG monitoring had only nonconvulsive seizures, whereas this was the case for only 17% of patients in the other groups. Lateralized rhythmic delta activity and lateralized periodic discharges were both associated with lesions involving the cortex or juxtacortical white matter.

Conclusions and Relevance. Lateralized rhythmic delta activity in critically ill patients has a similar clinical significance as lateralized periodic discharges. It reflects the presence of a focal lesion and is associated with a high risk of acute seizures, especially nonconvulsive.
Continuous electroencephalography (CEEG) recording is increasingly used to detect nonconvulsive seizures, which are more common than convulsive seizures in critically ill subjects. In addition to detecting nonconvulsive seizures, these recordings have revealed the existence of periodic and rhythmic electroencephalography (EEG) patterns whose significance remains uncertain.\(^1\)\(^2\)\(^3\)

The purpose of this article was to describe a focal or lateralized type of rhythmic delta activity (RDA) encountered in critically ill subjects and determine its clinical significance in this setting. We termed this pattern lateralized RDA (LRDA), in accordance with the American Clinical Neurophysiology Society Standardized Critical Care EEG Terminology.\(^7\) We describe its characteristics and compare its etiology and clinical and radiological correlates with lateralized periodic discharges (LPDs) by American Clinical Neurophysiology Society nomenclature, also known as periodic lateralized epileptiform discharges (PLEDs), and focal polymorphic [nonrhythmic] slowing [herein referred to as “focal nonrhythmic slowing”]. We also compare the risk of acute seizures associated with LRDA with the risk of seizures associated with LPDs, with focal nonrhythmic slowing, and in control subjects.

**Methods**

The Yale Human Investigation Committee approved this research. We maintain a prospective database of urgent EEGs and CEEGs and code the findings based on the American Clinical Neurophysiology Society Standardized Critical Care EEG Terminology,\(^1\)\(^2\)\(^3\) including LRDA. We retrospectively searched our database between May 2011 and May 2012 for the key words “lateralized rhythmic delta activity.” Lateralized RDA was defined according to this terminology as the “repetition of a unilateral or bilateral synchronous but asymmetric waveform with relatively uniform morphology and duration, and without an interval between consecutive waveforms, at a frequency less than or equal to 4 Hz.” Minimal duration was 6 cycles (hence, at least 1.5-6 seconds, depending on frequency) and there were no inclusion or exclusion criteria for morphology. Interrater agreement for both terms (lateralized and rhythmic delta activity) has been found to be excellent.\(^4\) A similar search was conducted for “lateralized periodic discharges” and “focal slowing” to constitute groups for comparison. We also selected all subjects whose EEG did not show any focal, periodic, or rhythmic abnormalities (referred to as “controls”). All terms are routinely used by attending neurophysiologists at our institution and to enter EEG data from the reports in our database. In addition to the EEG reports and recordings, medical records and imaging reports were reviewed systematically.

Clinical data collection included demographics, major medical and neurological diagnoses, neurological examination during EEG monitoring, presence or absence of clinical seizures during the 24 hours preceding admission or during hospital stay, history of epilepsy, metabolic disturbances, and imaging results. Comparisons between groups were performed with a 2-sided t test, 2-sided Fisher exact test, \(\chi^2\) test, and multivariate logistic regression using Matlab (MathWorks) and SAS (SAS Institute Inc). Correction for multiple comparisons was done using the method of the false discovery rate.\(^5\) The corresponding threshold for a rate of type I error of 0.05 was 0.006. Thus, we used a cutoff P value <0.006 for significance, with a trend defined as .006 <\(P\) < .05.

**Results**

**Prevalence of LRDA**

Among 558 individuals older than 1 month who underwent urgent EEG or CEEG, we identified 27 subjects (4.7%) with LRDA (Figure 1A and Figure 2A). During the same period, we identified 49 subjects (8.6%) with LPDs, 136 (23.9%) with focal nonrhythmic slowing, and 241 control subjects (43.2%) (with no focal, periodic, or rhythmic discharges). There was significant overlap between groups (see EEG Characteristics of LRDA subsection). Reason for EEG study and location of subjects (neurological, general medical, or surgical unit) did not differ significantly between groups. Demographic data are summarized in Table 1 and were similar across all groups.

**EEG Characteristics of LRDA**

Twenty-three subjects with LRDA had 1 focus, 2 had 2 bilateral independent foci, and 1 had 2 ipsilateral independent foci. Thus, a total of 30 foci of LRDA were identified.

Most commonly, LRDA was seen as brief or very brief (<1 minute in 28 of 30 subjects; <10 seconds in 19 of 30 subjects) intermittent runs of monomorphic, 50- to 200-μV sinusoidal or sharply contoured delta activity (Figure 1A and Figure 2A). The typical frequency was most often 1 to 2 Hz or 2 to 3 Hz (in 18 of 30 and 8 of 30 subjects, respectively) (eFigure in Supplement). Faster frequencies (up to 3-4 Hz) and longer duration (up to 30 minutes) were observed occasionally. Comparatively, LPDs were slower (≤1 Hz in 45 of 49 subjects) and occurred in longer runs (between 5 minutes and 1 hour in 33 of 49 subjects and >1 min in 47 of 49 subjects) than LRDA (eFigure in Supplement).

The intermittent presence of embedded spikes, or more commonly sharp waves, was noted in 5 cases of LRDA but the pattern was different from rhythmic spike-and-wave complexes (Figure 2A; “LRDA+S” by American Clinical Neurophysiology Society nomenclature). Lateralized periodic discharges were significantly more frequent in subjects with LRDA than in subjects with focal nonrhythmic slowing (Table 2). Similarly, LRDA tended to be more frequent in subjects with LPDs than in patients with focal nonrhythmic slowing. Sporadic epileptiform discharges were equally common in all 3 groups.

Foci of LRDA were most commonly located anteriorly (25 of 30 were frontal or temporal; 18 frontal, 7 temporal, 1 central, 3 parietal, and 1 occipital) but this distribution did not differ from the distribution of LPDs (39 of 54 were frontal or temporal; 26 frontal and 13 temporal).

In patients whose CEEG was initiated on the day of onset of their acute illness, LRDA was identified within the first 24 hours of monitoring in 79% (15 of 19), a proportion slightly lower
than LPDs (91%; 31 of 34). While all LPDs were first observed within 48 hours of monitoring, LRDA developed after 48 hours in 10% (2 of 19) of cases. Overall, however, the timing of first recording of LRDA was not significantly different from LPDs ($P = .34$).

Because the duration of CEEG monitoring varied significantly between patients and few had a repeated EEG at distance from the acute findings, we could not reliably determine the persistence of LRDA and LPDs over time. However, in 2 patients with prolonged recording, LRDA was noted to persist up to at least 7 and 14 days, respectively.

**Etiology of LRDA**

Almost all subjects with LRDA, LPDs, or focal nonrhythmic slowing had either an acute or remote cerebral injury (Table 1 and eTable in Supplement). This is in contrast with control subjects, among whom a metabolic, toxic, or sepsis-related encephalopathy was found in almost one-third of the cases. Six subjects of 27 (22%) with LRDA had a history of epilepsy. This proportion did not differ significantly from subjects with LPDs (14%) and focal nonrhythmic slowing (16%).

**Clinical Correlates of LRDA**

Seventeen subjects with LRDA (63%) were stuporous or comatose. This proportion was similar to subjects with LPDs but tended to be higher than in subjects with focal nonrhythmic slowing (Table 1). Nineteen subjects (70%) with LRDA had a localizing neurological examination, which was always consistent with the side of LRDA. This proportion was similar in the LPDs and focal nonrhythmic slowing groups but was significantly lower in controls.

No obvious clinical manifestations were seen during runs of LRDA. Because most subjects had an abnormal baseline neurological examination (altered mental status or focal deficits), we cannot rule out the possibility that some runs of LRDA were clinical seizures. Against this hypothesis, electrographic seizure patterns observed in these subjects differed from LRDA (see earlier and Figure 1B and Figure 2B), and in some cases, the electrographic seizures were accompanied by clinical manifestations whereas LRDA never was.

**Radiological Abnormalities**

Brain imaging was performed in 26 subjects with LRDA (18 magnetic resonance imaging and 8 computed tomography...
scans) and found to be abnormal in 24 of them (Table 3). In cases with a single focal lesion, LRDA was localized in the same region as the lesion in all but 2 cases (17 of 19); 1 had an ipsilateral thalamic hemorrhage with parietal LRDA, and another had an extensive upper brainstem hemorrhage that involved the thalamus with frontal LRDA. Compared with lesions in the focal nonrhythmic slowing group, lesions in subjects with LRDA tended to be less often restricted to the deep white matter and more frequently involve the deep gray structures. No significant difference was found between the LRDA and LPDs groups with regard to the localization of the lesions.

Table 1. Demographics, Etiology, and Clinical Correlates in Subjects With LRDA and Comparison Groups With LPDs, Focal Nonrhythmic Slowing, or Controlsa

<table>
<thead>
<tr>
<th>No. (%)</th>
<th>LRDA (n = 27)</th>
<th>LPDs (n = 49)</th>
<th>Focal Nonrhythmic Slowing (n = 136)</th>
<th>Controlsb (n = 241)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean (SD)</td>
<td>52.3 (25.2)</td>
<td>63.4 (20.6)</td>
<td>58.0 (23.2)</td>
<td>57.0 (17.1)</td>
</tr>
<tr>
<td>Female</td>
<td>11 (41)</td>
<td>24 (49)</td>
<td>59 (43)</td>
<td>99 (41)</td>
</tr>
<tr>
<td>History of epilepsy</td>
<td>6 (22)</td>
<td>7 (14)</td>
<td>22 (16)</td>
<td>23 (10)</td>
</tr>
<tr>
<td>Remote CNS lesion</td>
<td>13 (48)</td>
<td>19 (39)</td>
<td>58 (43)</td>
<td>61 (25)</td>
</tr>
<tr>
<td>Acute CNS lesion</td>
<td>19 (70)</td>
<td>32 (65)</td>
<td>67 (63)</td>
<td>94 (39)</td>
</tr>
<tr>
<td>Acute encephalopathy</td>
<td>0</td>
<td>0</td>
<td>15 (11)</td>
<td>74 (31)</td>
</tr>
<tr>
<td>Stupor or coma</td>
<td>17 (63)</td>
<td>25 (51)</td>
<td>51 (37)</td>
<td>110 (46)</td>
</tr>
<tr>
<td>Localizing examination</td>
<td>19 (70)</td>
<td>30 (61)</td>
<td>84 (62)</td>
<td>30 (12)</td>
</tr>
</tbody>
</table>

Abbreviations: CNS, central nervous system; LPDs, lateralized periodic discharges; LRDA, lateralized rhythmic delta activity.

a All comparisons are made with the LRDA group.

b Controls consisted of all subjects whose electroencephalogram did not show any focal, periodic, or rhythmic abnormalities.

c A cutoff P value <.006 was used for significance.

A trend, defined as .006 < P < .05.

Figure 2. Encephalomyelitis, Bilateral Independent Rhythmic Delta Activity, and Bilateral Independent Seizures in a 22-Year-Old Man

A, Ten-second electroencephalography (EEG) page showing a run of lateralized rhythmic delta activity characterized by rhythmic 2/s, 70-μV, unilateral activity maximal over the right frontotemporal region. There was no clinical correlate. Note the presence of embedded sharp waves (*) and the sharply contoured morphology of some of the delta waves (underlined). B, Twenty-second EEG epoch showing a focal seizure arising from the same region as the lateralized rhythmic delta activity and spreading to the left hemisphere. The seizure onset is characterized by quasiperiodic sharp waves (denoted by *) followed by rhythmic sharp theta activity (underlined) that then evolves. A 1-Hz high-pass filter and 70-Hz low-pass filter were applied to the EEGs. The notch filter was off.
Seizures in Subjects With LRDA

Seventeen subjects with LRDA (63%) had acute seizures during their stay, including 12 subjects with witnessed clinical seizures during or immediately prior to their admission and 12 with seizures recorded on EEG (7 had both). The incidence of seizures in subjects with LRDA (63%) was similar to subjects with LPDs (57%) and significantly higher than in subjects with focal nonrhythmic slowing (20%) and control subjects (17%) (Figure 3A). Similar differences were noted when considering only seizures that were identified during CEEG (Figure 3A). In addition, subjects with LRDA alone (i.e., without LPDs; n = 15) had the same risk of acute seizures (47%) as subjects with LPDs alone (i.e., without LRDA, n = 37) (49%). The incidence of seizures in subjects with both LRDA and LPDs was 84% (10 of 12 subjects).

In subjects who underwent CEEG, the risk of seizure occurrence and/or recurrence during the monitoring when LRDA was identified in the first hour was similar to the risk with LPDs and significantly higher than the risk with focal nonrhythmic slowing and in controls (Figure 3B).

All seizures seen on CEEG had a focal onset that coincided with the location of LRDA except in 1 subject, where the seizure onset was still in the same hemisphere. One subject (with encephalitis) had bilateral independent seizure onsets that colocalized with bilateral independent RDA. In 2 subjects, the electrographic pattern seen at the onset of seizures consisted of RDA and thus the brief runs of LRDA seen between seizures could be considered as aborted ictal discharges. Representative EEG pages from 1 of these subjects are depicted in Figure 1B. In the 10 other subjects with LRDA and recorded seizures, the ictal onset pattern was distinct from RDA and consisted of faster activity. A representative case is presented in Figure 2B.

There was a trend toward an association between the presence of embedded spikes/sharp waves and the risk of seizures (4 of 5 with LRDA and spikes/sharp waves and 13 of 22 with LRDA but no spikes/sharp waves) and runs of LRDA lasting more than 10 seconds (9 of 10 subjects with LRDA >10 seconds had seizures and 8 of 17 subjects with LRDA had seizures ≤10 seconds). The localization of LRDA did not change the risk of seizures: frontal LRDA was associated with a similar risk of seizures as nonfrontal LRDA (9 of 17 subjects and 8 of 10 subjects, respectively).

When considering all subjects, focal nonrhythmic slowing, LRDA, LPDs, sporadic epileptiform discharges, and clinical seizures prior to CEEG were all found to be predictors of recurring or newly occurring seizures during monitoring by univariate analysis. We subsequently performed a multiple logistic regression analysis that showed that LRDA (adjusted odds ratio [OR], 3.3; 95% CI, 1.2-9.6), LPDs (adjusted OR, 14.1; 95% CI, 5.6-35.8), sporadic epileptiform discharges (adjusted OR, 4.3; 95% CI, 1.5-12.0), and clinical seizures (adjusted OR, 4.6; 95% CI, 1.9-11.4) were all independent predictors of subsequent seizures during CEEG.

Of the 11 and 20 subjects with LRDA and LPDs who had seizures while undergoing CEEG, 9 (81%) and 18 (90%) had only nonconvulsive seizures, whereas this proportion was much lower in subjects with focal nonrhythmic slowing only or control subjects (both 1 of 6; 17%) (Figure 3C). In those with LRDA or LPDs, 10 of 11 patients (91%) and 19 of 20 patients (95%) had at least some nonconvulsive seizures, 3 of 6 (50%) and 4 of 6 (67%) of those with focal nonrhythmic slowing only or no periodic, rhythmic, and focal abnormalities, respectively.

The rate of seizure control during CEEG monitoring was good in all groups (18 of 20 subjects with LPDs, 10 of 11 subjects with LRDA, and 10 of 11 subjects with focal nonrhythmic slowing).

Table 2. Associated EEG Findings in Subjects With LRDA, LPDs, and Focal Nonrhythmic Slowing a

<table>
<thead>
<tr>
<th>No. (%)</th>
<th>LRDA (n = 27)</th>
<th>LPDs (n = 49)</th>
<th>Focal Nonrhythmic Slowing (n = 136)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sporadic epileptiform discharges b</td>
<td>7 (26)</td>
<td>14 (29)</td>
<td>34 (25)</td>
</tr>
<tr>
<td>Lateralized periodic discharges</td>
<td>12 (44)</td>
<td>NA</td>
<td>22 (16)</td>
</tr>
<tr>
<td>Lateralized rhythmic delta activity a</td>
<td>NA</td>
<td>12 (25)</td>
<td>19 (14)</td>
</tr>
</tbody>
</table>

Abbreviations: EEG, electroencephalography; LPDs, lateralized periodic discharges; LRDA, lateralized rhythmic delta activity; NA, not applicable.

* The Table indicates the number (percentage) of subjects in whom a secondary EEG pattern was found in addition to the main pattern.

b A trend, defined as .005 < P < .05.

Table 3. Localization of Brain Lesions on Imaging Studies in Subjects With LRDA, LPDs, and Focal Nonrhythmic Slowing a

<table>
<thead>
<tr>
<th>No. (%)</th>
<th>LRDA (n = 26)</th>
<th>LPDs (n = 43)</th>
<th>Focal Nonrhythmic Slowing (n = 97)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortex ± white matter</td>
<td>16 (62)</td>
<td>34 (79)</td>
<td>54 (56)</td>
</tr>
<tr>
<td>White matter only</td>
<td>3 (11)</td>
<td>2 (5)</td>
<td>31 (32) b</td>
</tr>
<tr>
<td>Deep gray nuclei</td>
<td>7 (27)</td>
<td>10 (22)</td>
<td>8 (8) b</td>
</tr>
</tbody>
</table>

Abbreviations: LPDs, lateralized periodic discharges; LRDA, lateralized rhythmic delta activity.

* The Table indicates the number (percentage) of subjects who had a lesion involving the cortex and adjacent white matter, the white matter only, and/or the deep gray nuclei. All comparisons are made with the LRDA group.

b A trend, defined as .05 < P < .01.

Abbreviations: EEG, electroencephalography; LPDs, lateralized periodic discharges; LRDA, lateralized rhythmic delta activity; NA, not applicable.
Discussion

Our study shows that LRDA is a common EEG pattern in critically ill patients. In this group, it indicates the presence of an epileptogenic brain lesion, most often involving the cortex or the juxtacortical white matter. Similar to LPDs (or PLEDs), it is associated with a high risk of acute seizures, in particular electrographic seizures that can only be detected with CEEG.

Comparison of LRDA With Other Types of Pathological Delta Activity

Delta activity in the EEG of an alert or comatose individual is a sign of disturbed cerebral function. Focal polymorphic slowing, especially if delta range and persistent, usually indicates the presence of a localized lesion involving the white matter but can sometimes be seen transiently as a postictal phenomenon. Our findings in the group with focal nonrhythmic slowing are in agreement with this view. We also found that LRDA significantly differed from focal (nonrhythmic) slowing in 2 aspects. First, LRDA was found to be an independent predictor of recurring seizures, with a risk of seizures significantly higher than with focal nonrhythmic...
slowing. Second, the underlying brain lesion tended to be more likely to involve the cortex, juxtacortical white matter, and/or deep gray structures rather than the deep white matter only. These differences indicate that LRDA and focal non-rhythmic slowing represent distinct EEG phenomena and that the rhythmicity changes the clinical implications of lateralized delta activity.

Intermittent rhythmic delta activity was described by Cobb in 1945 and several subtypes are recognized. Frontal intermittent rhythmic delta activity (FIRDA) is usually bilateral synchronous and symmetrical and is most often encountered in subjects with diffuse encephalopathy and less often with lesions of the frontal lobes, deep midline lesions, or hydrocephalus or in subjects with primary generalized epilepsy. Asymmetrical and even unilateral FIRDA has been described and usually denotes the presence of a focal brain lesion, rather than of encephalopathy. We suggest that this type of unilateral FIRDA is frontal LRDA and highly associated with seizures. In contrast to the low incidence of seizures associated with FIRDA reported in the literature and confirmed in our database (9% of patients and 10 of 17 patients in our series), LRDA is associated with a high rate of acute seizures (53% of 17 patients in our series), similar to (though a bit lower than) nonfrontal LRDA (80% of 10 patients).

One form of LRDA that has been well described in the literature is highly associated with seizures: temporal intermittent RDA, as opposed to temporal intermittent RDA, which is considered to be fairly specific for mesial temporal lobe epilepsy, LRDA is associated with various types of brain lesions, most commonly hemorrhagic and ischemic stroke, and is not restricted to the anterior temporal region. Furthermore, temporal intermittent RDA has been described in ambulatory subjects with chronic epilepsy and does not necessarily indicate a risk of imminent seizures, whereas we found that LRDA in acutely ill individuals predicts acute seizures. In our series, 7 subjects had temporally predominant LRDA and 5 of them (85%) had acute seizures.

Similarity to LPDs
We found that LRDA was similar in many aspects to LPDs (also known as PLEDs). Clinical and radiological correlates, as well as the incidence of acute seizures, were similar in both groups and were in line with the extensive literature on LPDs. The pathophysiological mechanisms of LPDs and RDA are only partially understood. Electroanatomical correlations support the hypothesis that LPDs and RDA are associated with injury to the cortex and juxtacortical white matter and/or the deep gray structures. In our series, the anatomical distribution of lesions was similar in the LPDs and LRDA groups and differed significantly from the focal nonrhythmic slowing group, in which there were a higher proportion of lesions limited to the white matter. This association provides further evidence that LRDA and LPDs share a common pathophysiological mechanism. Recordings in critically ill subjects with simultaneous scalp and intracortical electrodes have revealed that some instances of focal RDA seen on scalp EEG correspond to intracranial periodic epileptiform discharges or periodic bursts. However, there were some differences between LRDA and LPDs. Lateralized rhythmic delta activity occurred in shorter runs, typically less than 10 seconds and almost always less than 1 minute, whereas LPDs tend to occur in more prolonged runs, often lasting minutes to hours. The frequency of LRDA was most often between 1 and 3 Hz, faster than LPDs whose typical frequency is usually 1 Hz or less. LRDA and LPDs may occur independently but may coexist in the same individuals, who then have an even higher risk of acute seizures than subjects with only LRDA or only LPDs (82%, 63%, and 57%, respectively, in our series). For these reasons, we believe that the 2 patterns are at least partially distinct and convey different information regarding epileptogenesis.

In contrast to subjects with focal nonrhythmic slowing only, or to control subjects, seizures in subjects with LRDA and/or LPDs were almost always electrographic only, without overt clinical manifestations. This was much less common in the other groups, though about half of those patients had at least some nonconvulsive seizures. The lower proportion of clinical manifestations in the LRDA/LPDs groups is possibly due to the more severe injury associated with LRDA and LPDs, as attested by the higher proportion of subjects with significantly altered mental status or localizing examination that might prevent the clinical expression of seizures. It is also conceivable that more subjects in the LPDs group were receiving prophylactic anticonvulsant drugs that could also attenuate clinical manifestations (“electromechanical dissociation”). Another explanation is that subjects with LPDs or LRDA had more frequent seizures than the other subjects, as we observed (data not shown). During status epilepticus and with frequent seizures, clinical manifestations are known to progressively disappear. Finally, periodic and rhythmic discharges are thought to result from the dysfunction of a cortical-subcortical network, which may prevent synchronous caudal propagation of ictal activity through motor pathways and thus produce less motor activity.

This study is limited by its retrospective design and its relatively small size. Outcome measures could not be gathered for all subjects and imaging studies were not standardized. Thus, our findings should be replicated in an independent and preferably prospective cohort.

Conclusions
Lateralized rhythmic delta activity is a type of focal slowing encountered in critically ill subjects and associated with a high risk of seizures during acute illness (63%); almost all of these seizures (about 90%) are electrographic only and require EEG for detection. Lateralized rhythmic delta activity is similar in clinical significance to LPDs. Both patterns indicate the presence of focal cortical hyperexcitability and suggest the presence of a focal lesion involving the gray matter or juxtacortical white matter. Lateralized rhythmic delta activity differs significantly from focal nonrhythmic slowing. Future studies should aim to further clarify the differences between FIRDA
and its asymmetric version, frontal LRDA; investigate the pathophysiological mechanisms underlying LRDA and LPDs; determine the association of these patterns with ongoing neuronal injury, functional outcome, and subsequent epilepsy; and determine the role of prophylaxis and treatment of these EEG patterns.

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Analysis and interpretation of data: Gaspard, Rampal, Petroff, Hirsch.
Drafting of the manuscript: Gaspard, Petroff.
Critical revision of the manuscript for important intellectual content: All authors.
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Obtained funding: Gaspard.
Administrative, technical, and material support: Manganas, Rampal.
Study supervision: Rampal, Petroff, Hirsch.

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Correction: This article was corrected on September 11, 2013, to fix errors in Figure 3 and its legend.

REFERENCES