CME

Prevalence and clinical characteristics of postictal psychiatric symptoms in partial epilepsy

Andres M. Kanner, MD; Arnoldo Soto, MD; and Hilary Gross–Kanner, OTR

Abstract—*Objectives:* To identify the prevalence and clinical characteristics of postictal psychiatric (PPS) and cognitive (PCS) symptoms in patients with refractory partial epilepsy and to investigate whether interictal psychiatric and cognitive symptoms worsened in severity during the postictal period. *Methods:* Using a 42-item questionnaire, the authors determined the prevalence and clinical characteristics of PPS and PCS that occurred after >50% of seizures in 100 of 114 consecutive patients with refractory partial epilepsy during a 3-month period. The postictal period was defined as the 72 hours that followed a seizure. The prevalence of all interictal psychiatric and cognitive symptoms was identified and the frequency with which they worsened postictally determined. *Results:* A mean of 2.8 ± 1.8 PCS (median = 3) and 5.9 ± 5.3 PPS (median = 5) was identified, which included postictal symptoms of depression (PSD) in 43 patients, anxiety (PSA) in 45, postictal psychotic symptoms (PIP) in 7, hypomanic symptoms in 22, neurovegetative symptoms in 52, and fatigue in 37. Most patients experienced more than one type of PPS. Independently of the occurrence of PPS, 38 patients reported a worsening of interictal psychiatric and cognitive symptoms postictally. A history of depression and anxiety significantly increased the number of PSD, PSA, and PIP. *Conclusions:* Postictal psychiatric symptoms are common among patients with refractory partial epilepsy, and the severity of interictal psychiatric and cognitive symptoms commonly worsens during the postictal period.

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Postictal psychiatric symptoms (PPS) have been recognized since the 19th century, following their description by Gowers[1] and Hughlings Jackson.[2] In the early part of the 20th century, Kraepelin[3] reported on peri-ictal and postictal mood changes, while Williams noted the persistence of ictal symptoms of depression into the postictal period for as long as 3 days.[4] Nevertheless, there are very few data on the prevalence of PPS, and a systematic analysis of their clinical characteristics has never been done. PPS may present as a single symptom (i.e., irritability, suicidal ideation) or as clusters of symptoms, mimicking those of an anxiety, depressive, or psychotic disorder. PPS do not necessarily occur immediately after a seizure; in fact, they are more likely to appear following a symptom-free period ranging from 12 to 72 hours.[5-11]

Given the relatively frequent psychiatric comorbidity in patients with poorly controlled epilepsy,[12-14] it is necessary to establish whether PPS represent "de novo" psychiatric symptoms or reflect a "transient worsening" of interictal psychiatric symptoms or a "transient reactivation" of past psychiatric disorders. This study was carried out with the following principal aims: 1) to establish the prevalence and clinical characteristics of PPS and postictal cognitive symptoms (PCS) occurring as habitual phenomena, that is, following at least 50% of seizures, in a group of patients with poorly controlled partial epilepsy; and 2) to investigate whether interictal psychiatric and cognitive symptoms worsened in severity during the postictal period. Secondary goals of the study included 1) determining whether a past psychiatric history is a risk factor for or worsens PPS and PCS and 2) establishing whether the presence of PPS worsens postictal cognitive disturbances. We define the postictal period as the 72-hour period after a seizure or cluster of seizures.

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Methods. We prospectively investigated the occurrence of PPS and PCS during a 3-month period in 114 consecutive outpatients with a history of pharmacoresistant partial epilepsy followed at

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From the Division of Epilepsy and Clinical Neurophysiology and Rush Epilepsy Center, Department of Neurological Sciences, Rush Medical College, Rush University Medical Center, Chicago, IL.

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Address correspondence and reprint requests to Dr. A.M. Kanner, Department of Neurological Sciences, Rush University Medical Center, 1653 W. Congress Pkwy., Chicago, IL 60612; e-mail: Andres_M_Kanner@rush.edu

Table 1 *Prevalence of postictal psychiatric and cognitive symptoms in 100 patients with intractable partial epilepsy*

Postictal symptoms	Prevalence
Symptoms of depression, total	43
Irritability	30
Poor frustration tolerance	36
Anhedonia	32
Hopelessness	25
Helplessness	31
Crying bouts	26
Feelings of self-deprecation	27
Feelings of guilt	23
Neurovegetative symptoms, total	62
Excessive somnolence	43
Loss of appetite	36
Loss of sexual interest (not related to fatigue)	26
Symptoms of anxiety, total	45
Constant worrying	33
Agoraphobic symptoms	29
Due to fear of seizure recurrence	20
Self-consciousness	26
Psychotic symptoms, total	7
Hypomanic symptoms, total	22
Thought racing	15
Fatigue	37
Cognitive symptoms, total	82
Difficulty in concentration	71
Problems with memory	66
Confusion	65
Disorientation	46
Thought blockage	42
Only cognitive symptoms, total	14
No postictal symptoms, total	12

This table includes only individual symptoms reported by at least 20% of patients.

the Rush Epilepsy Center that had undergone a video-EEG monitoring study at least 4 months prior to study enrollment. Sixty-seven were women and 47 were men with a mean age of 34.1 ± 10 years and a mean duration of their seizure disorder of 21.1 ± 11.5 years. Among these 114 patients, 14 were on antidepressant drugs and 100 were on no psychotropic drugs. We used only the data of these 100 patients to address the four objectives outlined above as the impact of antidepressants on PPS is not known.

We investigated the presence of PPS and PCS with a standard 42-item questionnaire designed to identify 30 psychiatric and 5 cognitive symptoms. The selected PPS were derived from the diagnostic criteria of mood, anxiety, and psychotic disorders, according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed; DSM-IV).[15] Only 1 of the 30 PPS (1 of 6 psychotic symptoms ["Do you feel as if you are getting special messages from the radio or television?"]) was not reported by any patient. Table 1 shows the PPS and PCS reported by at least 20% of patients. The complete list of PPS and PCS, their prevalence, and their median duration can be reviewed in the supplementary material on the *Neurology* Web site (go to www.neurology.org).

A reliability analysis of the psychiatric items of the questionnaire yielded a Chronbach α coefficient of 0.878, indicating a very good internal consistency. Psychiatric items were submitted to a principal component factor analysis with varimax rotation, items loaded on four factors. A detailed description of the psychometric properties of this instrument will be published in a separate manuscript. The questionnaire can be reviewed in the supplementary material on the *Neurology* Web site.

Psychiatric and cognitive symptoms were coded as follows: 1) postictal symptom if its occurrence was restricted to the postictal period; 2) interictal symptom with postictal exacerbation if patients perceived that its severity worsened significantly during the postictal period; 3) interictal symptom if patients reported that its severity did not differ between interictal and postictal periods. The prevalence of each symptom was rated as follows: never (=0), following <50% of seizures (=1), following >50% of seizures (=2), following all seizures (=3). Only symptoms with ratings of 2 and 3 were included in this study. Patients were also asked to estimate an average duration for each PPS and PCS. Investigators administered the questionnaire to minimize the patients' confusion about interictal and postictal symptoms and discard those symptoms for which their recollection was questionable.

Postictal symptoms were grouped into seven symptom categories: postictal symptoms of depression (PSD; n = 9), anxiety (PSA; n = 5), psychosis (PIP; n = 5), hypomania (PHM; n = 2), neurovegetative symptoms (PNV; n = 7), fatigue (n = 1), and PCS (n = 5). We coded neurovegetative symptoms (changes in sleep, appetite, and sexual drive) and fatigue as separate symptom categories to avert false-positive increases in the number of PSD, PSA, or PHM, as they are primary symptoms of mood and anxiety disorders but are also common postictal symptoms.

Following the collection of postictal and interictal data, all patients underwent a semistructured psychiatric interview designed to identify any past psychiatric history of mood, anxiety, attention deficit, and psychotic disorders. Finally, we investigated whether patients had ever received any psychopharmacologic therapy or other type of treatment specifically targeted to their PPS.

Data analyses. We used χ^2 statistics to investigate significant association between dichotomous outcome measures, and we used analysis of variance and Student *t*-test for continuous measures. We used the Spearman correlation coefficient in the investigation of correlations among the PPS categories. Given the number of analyses, significance was set at a p value of ≤ 0.005.

Results. *Neurologic data.* Table 2 summarizes the epilepsy data of our 100 patients, specifically, the type of seizures, location of ictal foci, seizure frequency, and etiology of the seizure disorder. There were no significant associations between any PPS category and any of the epilepsy variables listed in table 2 or between the patients' age and gender.

Psychiatric history. We identified a lifetime prevalence of psychiatric disorders in 54 patients. These consisted of mood disorders in 33 patients, anxiety disorders in 4, mixed mood and anxiety disorders in 12, and attention deficit hyperactivity disorder in 5. No patient had experienced a history of a psychotic disorder. Eleven patients reported one or more psychiatric hospitalizations.

Prevalence of postictal symptoms. Among the 100 patients, we identified a mean of 8.8 ± 6.5 habitual postictal symptoms (range 0 to 25; median 8.5) corresponding to 2.8 ± 1.8 PCS (range 0 to 5; median 3) and 5.9 ± 5.3 PPS (range 0 to 22; median 5). Seventy-four patients experienced at least one type of PPS; 68 reported PPS and PCS and 6 only PPS. An additional 14 patients experienced only PCS, whereas 12 did not report any postictal symptom. Sixty (81%) of these 74 patients experienced PPS belonging to more than one symptom category (i.e., PSD + PIP). As shown in table 3, the most frequent combination included PSA, PSD, and PNV.

Table 2 *Summary of neurologic data, n = 100*

Seizure focus	Temporal lobe	75
	Frontal lobe	13
	Parietal or occipital lobe	7
	Multilobar	5
Side of focus	Left/right	42/33
	Bilateral independent foci	23
	Not lateralized	2
Seizure type	Only complex partial seizures	49
	Generalized tonic-clonic seizures with/without complex partial seizures	50
	Simple partial seizures	1
Seizure frequency	>1/mo	78
	Seizures in clusters, >1/d	63
Etiology	Cryptogenic	42
	Mesial temporal sclerosis	36
	Posttraumatic epilepsy	7
	Structural lesion (tumor, arteriovenous malformation, cyst, focal dysplasia)	15

Clinical characteristics of PPS and relation to past psychiatric history. PSD. We identified a mean of 4.8 ± 2.4 PSD (range 2 to 9; median 5) in 43 patients. All of the PSD investigated, with the exception of postictal suicidal ideation, were reported by at least 20% of patients (see table 1). With the exception of postictal crying, the median duration of each PSD was 24 hours (see also the supplementary material on the *Neurology* Web site). Thirty-two patients experienced PSD of at least 24 hours' duration (18 experienced a minimum of six PSD lasting ≥24 hours), five reported PSD of 1 to 23 hours' duration, and only three patients had PSD lasting <1 hour. The duration of PSD was not significantly associated with a psychiatric history.

As shown in table 3, PSD always occurred in combination with other PPS categories, though correlations were identified only with PSA ($r = 0.556$, $p < 0.0001$), PIP ($r = 0.3$, $p = 0.002$), and PNV ($r = 0.37$, $p < 0.0001$).

Among these 43 patients, 25 had a history of a mood disorder and 11 of an anxiety disorder. There was an association between a history of depression and the following PSD: self-deprecation ($\chi^2 = 10.8$, $df = 1$, $p = 0.001$) and guilt ($\chi^2 = 16.9$, $df = 1$, $p < 0.0001$). In addition, a history of anxiety was associated with the presence of postictal guilt ($\chi^2 = 10.7$, $df = 1$, $p = 0.001$). Furthermore, there was a greater number of PSD in the presence of a history of depression ($F = 8.5$, $p = 0.004$) and anxiety disorders ($F = 8.4$, $p = 0.005$).

Thirteen patients reported habitual postictal suicidal ideation; eight experienced passive and active suicidal thoughts, whereas five reported only passive suicidal ideation. No patient ever acted on these symptoms. Ten of these 13 patients (77%) had a history of either major depression or bipolar disorder, and this association was significant ($\chi^2 = 21.8$, $df = 1$, $p < 0.0001$).

Table 3 *Combination of the different PPS categories*

Categories	Type of PPS	No. of patients
Single category	Depression	0
	Anxiety	1
	Psychosis	0
	Hypomanic	3
	Neurovegetative	10
	Subtotal	14
Two categories	Depression + anxiety	5
	Anxiety + neurovegetative	8
	Depression + neurovegetative	9
	Hypomanic + neurovegetative	5
	Depression + hypomanic	1
	Anxiety + hypomanic	1
	Subtotal	29
Three categories	Depression + anxiety + neurovegetative	17
	Depression + anxiety + hypomanic	1
	Depression + hypomanic + neurovegetative	1
	Anxiety + hypomanic + neurovegetative	1
	Subtotal	20
Four categories	Depression + anxiety + psychosis + neurovegetative	2
	Depression + anxiety + psychosis + neurovegetative + neurovegetative	5
	Depression + anxiety + neurovegetative + hypomanic	4
	Subtotal	11
	Total	74

Postictal cognitive symptoms and postictal fatigue were not included in this table.

PPS = postictal psychiatric symptoms.

PSA. We identified a mean of 2 ± 1 PSA (range 1 to 5; median 2) in 45 patients (see table 1). Three of the five PSA were reported by at least 20% of patients, whereas two symptoms, panicky feelings and compulsions, were reported by 10%. The median duration of individual PSA ranged from 6 to 24 hours (see also the supplementary material on the *Neurology* Web site). In 30 patients, at least one PSA lasted ≥24 hours (15 patients [33%] reported a cluster of four PSA of at least 24 hours); 10 patients reported at least one PSA of 1 to 23 hours' duration

and 5 patients had PSA lasting <1 hour. As with PSD, the duration of PSA was not associated with a psychiatric history.

As shown in table 3, PSA were identified in combination with other PPS categories in 44 (98%) patients, but correlations existed with PSD ($r = 0.556$, $p < 0.0001$), PIP ($r = 0.3$, $p = 0.002$), and PNV ($r = 0.38$, $p < 0.0001$).

Among the 29 patients with postictal agoraphobia, 18 (62%) attributed these symptoms to the fear of seizure recurrence. Nevertheless, the presence of this fear was not related to the actual occurrence of seizures in clusters ($p = 0.12$, Fisher exact test).

A prior history of anxiety disorder was identified in 15 patients (33%). There was an association between a history of anxiety disorder and the occurrence of two PSA: constant worrying ($\chi^2 = 22.7$, $p < 0.0001$) and panicky feelings ($\chi^2 = 9.3$, $p = 0.002$). In addition, there was a statistical trend in the association between a history of anxiety ($F = 8.3$, $p = 0.006$) and depressive disorders ($F = 6.8$, $p = 0.01$) and a greater number of PSA.

PIP. Seven patients experienced 2.6 ± 1.1 PIP (range 1 to 5; median 2; see table 1). Five patients reported referential thinking (people are staring and talking about me), two auditory hallucinations, four paranoid delusions, three religious delusions, and one visual hallucinations. The median duration of individual PIP ranged from 0.2 to 36 hours (see also the supplementary material on the *Neurology* Web site). In four of these patients, at least one PIP lasted a minimum of 24 hours, two patients experienced symptoms lasting between 1 and 23 hours, and one patient reported symptoms of <1-hour duration. The duration of PIP was not associated with a psychiatric history. These seven patients also experienced PSD and PSA, and five patients reported PHM. Not surprisingly, correlations existed with PHM ($r = 0.33$, $p = 0.001$), in addition to PSD and PSA, as shown above.

No patient had experienced a history of interictal psychosis. A psychiatric history was not a risk factor for PIP, but a history of anxiety disorder was associated with a greater number of PIP ($F = 20.8$, $p < 0.0001$).

PHM. PHM included excessive energy and racing thoughts, which were identified in 22 patients: Fifteen patients reported racing thoughts and nine increased energy, but only two reported both symptoms. The median duration of both symptoms was 2 hours (0.1 to 48 hours). In contrast to PSD, PSA, and PIP, only six patients experienced PHM lasting ≥24 hours. The occurrence of PHM correlated significantly only with that of PIP, as shown above. A psychiatric history was not a risk factor for PHM.

PNV. As stated before, PNV are among the most commonly reported postictal symptoms, above all postictal somnolence and loss of appetite. Sixty-two patients experienced 2.3 ± 1.1 PNV (range 1 to 5; median 2), and in 12, they were the only PPS category reported. Table 1 shows the PNV reported by at least 20% of patients. In addition, early night insomnia was reported by 11% of patients, middle night awakening by 13%, early morning awakening by 11%, and excessive appetite by 10%. These are four symptoms not typically associated with the postictal state. The median duration of individual PNV ranged from 15 to 39 hours (see also the supplementary material on the *Neurology* Web site).

PNV were identified in 50 patients (81%) with other types of PPS, and as already shown above, PNV correlated significantly with PSD and PSA. A psychiatric history did not worsen or act as a risk factor for PNV.

Postictal fatigue. Thirty-seven patients reported postictal fatigue with a median duration of 24 hours (0.1 to 108 hours). Its presence was associated with PSD lasting at least 24 hours ($\chi^2 = 10.9$, $df = 1$, $p = 0.001$). Postictal fatigue had no impact on the number of other PPS categories or PCS, however.

A cautionary note is in order: A prevalence rate of 37% of postictal fatigue is lower than expected. Nevertheless, this prevalence rate has to be coupled with an additional 18% of patients who reported interictal fatigue with postictal exacerbation (see below).

Relationship between PPS and prior psychiatric hospitalizations. There was a statistical trend between the presence of PSD and a history of psychiatric hospitalization ($\chi^2 = 6.8$, $df = 1$, $p = 0.009$). Of note, symptoms usually recognized with suicide-related symptomatology accounted for this association; these included postictal suicidal ideation ($\chi^2 = 13.1$, $df = 1$, $p < 0.0001$, odds ratio [OR] 14.7, 95% CI 3.4 to 57.9), helplessness ($\chi^2 = 10.9$, $df = 1$, $p = 0.001$, OR 9.2, 95% CI 2.2 to 37.9), and guilt ($\chi^2 = 10.9$, $df = 1$, $p = 0.001$, OR 9.4 , 95% CI 2.4 to 36.3).

Prior investigation and treatment of PPS. The existence of PPS had been investigated in only seven patients prior to this study, and only one was offered treatment specifically directed to the remission of his PSD.

Interictal psychiatric symptoms with postictal exacerbation. Thirty-eight patients experienced a mean of 3.1 ± 2.7 (range 1 to 15, median 3) psychiatric symptoms during the interictal period, which included a mean of 2.4 ± 1.7 symptoms of depression (range 1 to 9; median 2), 1.5 ± 1.1 anxiety symptoms (range 1 to 4; median 1), and 1.5 ± 0.8 neurovegetative symptoms (range 1 to 3; median 1). Among these 38 patients, 4 experienced only neurovegetative symptoms, whereas the other 34 had symptoms of depression (n = 24), anxiety (n = 3), or mixed anxiety/depression (n = 6); 15 of these 34 patients also experienced neurovegetative symptoms and 20 patients experienced interictal fatigue.

As stated in Methods, interictal symptoms that worsened in severity during the postictal period were coded as interictal symptoms with postictal exacerbation. Symptoms in which the severity did not differ during interictal and postictal periods were coded as interictal symptoms. We identified interictal symptoms with postictal exacerbation in 36 of these 38 (94%) patients; in 19, all recorded symptoms were coded only as such, whereas the other 17 reported symptoms coded as both interictal symptoms with postictal exacerbation and as interictal symptoms.

Among these 36 patients, 30 (83%) also experienced de novo PPS. There was an association between the occurrence of interictal symptoms of depression with postictal exacerbation and PSD ($\chi^2 = 32.1$, $df = 1$, $p < 0.0001$). There was also an association between the presence of interictal symptoms of anxiety with postictal exacerbation and PSA ($\chi^2 = 16.6$, $df = 1$, $p < 0.0001$). Among these 30 patients, the number of PSD and PSA were greater than the number of interictal symptoms of depression (4.7 ± 2.1 vs 2.4 ± 1.7; $p < 0.0001$) and anxiety (2.0 ± 0.9 vs 1.5 ± 1.1; $p = 0.002$) with postictal exacerbation. Finally, among the 20 patients with interictal fatigue, 18 reported significant worsening during the postictal period.

Relation between PCS and PPS. Eighty-two patients experienced a median of 3.4 ± 1.4 PCS (range 1 to 5; median 4; see table 1). Fourteen patients reported only PCS, whereas the other 68 patients experienced PCS and PPS. The median duration of individual PCS ranged from 1 to 9 hours (0.05 to 108 hours). In general, the "estimated" duration of PPS appeared to be longer than that of PCS ($t = 8.5$, $p < 0.0001$). The presence of PSD was associated with worst postictal cognitive disturbances, as evidenced by a greater number of PCS ($F = 21.7$, $p < 0.0001$). On the other hand, a psychiatric history had no impact on either the occurrence or the number of PCS.

Thirty-seven patients reported interictal cognitive symptoms that worsened postictally; all of these patients also experienced de novo PCS. The number of PCS was greater than that of interictal cognitive symptoms with postictal exacerbation (3.4 ± 1.4 vs 2.3 ± 1.3; $p < 0.0001$).

Impact of antiepileptic drugs on PPS. At the time of this study, the 100 patients were taking a mean of 1.8 ± 0.7 antiepileptic drugs (AED). Nineteen patients were taking one AED with negative psychotropic properties (phenobarbital, primidone, vigabatrin). Being on this type of AED yielded a trend toward a greater likelihood of developing PIP ($\chi^2 = 5.3$, $df = 1$, $p = 0.02$).

Discussion. This study yielded four important findings: 1) PPS are relatively common among patients with refractory partial epilepsy, and in a majority they present as a combination of two or more symptom categories. 2) Interictal psychiatric and cognitive symptoms can commonly worsen in severity during the postictal period. 3) A psychiatric history can be a risk factor and can worsen certain, but not all, PPS. 4) The presence of PPS worsens the severity of postictal cognitive disturbances. A cautionary note is in order, however: Our data were obtained from a selected patient population with refractory partial epilepsy. Accordingly, they cannot be generalized to all patients with epilepsy.

In most patients, PPS present as a clustering of various symptom categories with combinations of depression, anxiety, and neurovegetative symptoms being the most frequent. This is not surprising, given the fact that anxiety and depression are the most common psychiatric disorders among patients with epilepsy.[12-14,16,17] Furthermore, 34% of our patients experienced interictal symptoms of depression, anxiety, or both. This prevalence rate is comparable with those reported in other patient series with refractory partial epilepsy.[12-14,16-19]

A psychiatric history worsened the severity of PPS, as a history of depressive and anxiety disorders was significantly associated with a greater number of PSD, PSA, or PIP. Furthermore, a history of depression was a risk factor for a number of PSD and PSA. Also, symptoms usually associated with increased suicidal risk (suicidal ideation, self-deprecation, hopelessness, and guilt) were also significantly associated with a history of past psychiatric hospitalizations. These findings deserve special attention, as patients with epilepsy have a five- to ninefold higher risk of suicide attempts than the general population.[20] Thus, recognition of these four PSD can be indicative of a (past) severe history of depression. Yet, the frequency with which postictal suicidal ideation leads to suicidal behavior is yet to be established.

A majority of the 38 patients (96%) that reported interictal psychiatric symptoms and all patients with interictal cognitive symptoms noted that all or some of their symptoms worsened in severity during the postictal period. Furthermore, the presence of interictal symptoms with postictal exacerbation was significantly associated with the occurrence of de novo PSD and PSA. These findings may suggest possible common pathogenic mechanisms mediating the development of de novo PPS and the worsening in severity of interictal symptoms postictally, though the actual mechanisms are yet to be identified. Whether PPS, PCS, and the postictal exacerbation of interictal symptoms reflect a process equivalent to a "Todd paralysis" commonly identified with motor and sensory symptoms is an attractive hypothesis that needs to be studied in future research.

The "estimated" duration of PPS ranged from a few minutes to several days, with a median of 24 hours in all but one PSD, 6 to 24 hours in PSA, 6 to 16 hours in PIP, and 2 hours in PHM. A longer duration was not associated with a psychiatric history. Interestingly enough, the duration of PPS was significantly longer than that of PCS. Clearly, the lengthy duration of these symptoms contributes to the disability associated with seizures and must account to a significant degree for the poor quality of life of these patients. This is exemplified in our 18 patients who habitually experience a cluster of six or more PSD of at least 24-hour duration or the 7 patients with a combination of PIP, PSA, and PSD after >50% of their seizures.

The negative effect of postictal disturbances on quality-of-life ratings has been demonstrated in a study that investigated the relationship between seizure severity and health-related quality-of-life ratings in 340 patients with refractory epilepsy.[21] The findings of this study showed a significant correlation between time to recovery after a seizure and worse quality of life. The instrument used did not include PPS, however. Three of the frequently used seizure severity scales, the Liverpool Seizure Severity Scale,[22] the Chalfont–National Hospital Seizure Severity Scale,[23] and the Hague Seizure Severity Scale,[24] assess some aspects of the postictal period with items that focus on cognitive deficits and physical injury and symptoms but do not include PPS. A recently developed seizure severity questionnaire[25] included only one item on the impact of postictal "emotional effects (depression, anxiety, anger, etc.)." To date, the impact of PPS on the quality of life of patients has yet to be established and quantified.

Our data clearly establish a worsening effect of PPS on postictal cognitive disturbances, as the number of PCS increased significantly in the presence of PSD. These data exemplify the close interaction between psychiatric and cognitive disturbances and

further support the need to inquire about PPS even in the assessment of postictal cognitive disturbances.

The relatively high prevalence of PPS identified in our patients raises the question of their role in "shaping" the psychiatric symptomatology in intractable partial epilepsy. To the best of our knowledge, in none of the published studies aimed at identifying psychiatric disorders according to the DSM criteria did investigators ever discriminate among psychiatric symptoms with an interictal, preictal, or postictal occurrence. Had we attempted to formulate a psychiatric diagnosis according to DSM-IV criteria,[15] most of our patients would have ended with a diagnosis of "atypical depression or anxiety not otherwise specified." In fact, depression in epilepsy has been classified in this category by several authors.[12,16,17,26] Whether PPS account for the "atypical" presentation of mood disorders in epilepsy is yet to be established. This was not an aim of our study as our 42-item questionnaire was designed to identify distinct psychiatric symptoms but not to formulate psychiatric diagnoses.

The obvious treatment of PPS is the eradication of seizures. This, however, is not possible in patients with refractory epilepsy that are not candidates for surgical treatment. There are no controlled pharmacologic trial data on the treatment of PPS with psychotropic drugs, and the response of PSD and PSA to antidepressant medication is yet to be established.

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