ORIGINAL ARTICLE

Electroclinical Analysis of Postictal Noserubbing

Richard Wennberg

ABSTRACT: ***Background:*** Postictal noserubbing (PIN) has been identified as a good, albeit imperfect, lateralizing and localizing sign in human partial epilepsy, possibly related to ictal autonomic activation. ***Methods:*** PIN was studied prospectively in a group of consecutive patients admitted for video-EEG monitoring, with the laterality of noserubbing correlated with electrographic sites of seizure onset, intra- and interhemispheric spread, and sites of seizure termination. ***Results:*** PIN was significantly more frequent in temporal than extratemporal epilepsy ($p<0.001$; 23/41 (56%) patients and 41/197 (21%) seizures in temporal lobe epilepsy compared with 4/34 (12%) patients and 12/167 (7%) seizures in extratemporal epilepsy). The hand used to rub the nose was ipsilateral to the side of seizure onset in 83% of both temporal and extratemporal seizures. Seizures with contralateral PIN correlated with spread to the contralateral temporal lobe on scalp EEG ($p<0.04$). All extratemporal seizures with PIN showed spread to temporal lobe structures. One patient investigated with intracranial electrodes showed PIN only when ictal activity spread to involve the amygdala: seizures confined to the hippocampus were not associated with PIN. PIN was not observed in 63 nonepileptic events in 17 patients. Unexpectedly, one patient with primary generalized epilepsy showed typical PIN after 1/3 recorded absence seizures. ***Conclusions:*** This study confirms PIN as a good indicator of ipsilateral temporal lobe seizure onset. Instances of false lateralization and localization appear to reflect seizure spread to contralateral or ipsilateral temporal lobe structures, respectively. Involvement of the amygdala appears to be of prime importance for induction of PIN.

RÉSUMÉ: Analyse électroclinique du frottement du nez postictal. ***Introduction:*** Le frottement du nez postictal (FNP) a été identifié comme étant un bon signe, quoiqu'imparfait, de latéralisation et de localisation dans l'épilepsie partielle humaine, pouvant être relié à une activation autonome ictale. ***Méthodes:*** Le FNP a été étudié prospectivement chez un groupe de patients consécutifs admis pour monitorage ÉEG vidéographique. La latéralité du FNP a été corrélée aux sites électrographiques du début des crises, à la propagation intra et interhémisphérique et aux sites de fin des crises. ***Résultats:*** Le FNP était significativement plus fréquent dans l'épilepsie temporale que dans l'épilepsie extratemporale ($p<0.001$; 23/41 (56%) des patients et 41/197 (21%) des crises dans l'épilepsie temporale par rapport à 4/34 (12%) des patients et 12/167 (7%) des crises dans l'épilepsie extratemporale). La main ipsilatérale au côté où la crise débutait était utilisée pour frotter le nez dans 83% des crises temporales et des crises extratemporales à l'ÉEG de surface ($p<0.04$). Toutes les crises extratemporales accompagnées de FNP présentaient une propagation aux structures du lobe temporal. L'investigation d'un patient au moyen d'électrodes intracrâniennes a montré que le FNP était présent seulement quand l'activité ictale se propageait à l'amygdale: les crises limitées à l'hippocampe n'étaient pas associées au FNP. Le FNP n'a pas été observé dans 63 événements non épileptiques chez 17 patients. Un patient avec une épilepsie généralisée idiopathique a présenté un FNP typique après 1 absence sur 3 documentées par ÉEG. ***Conclusions:*** Cette étude confirme que le FNP est un bon indicateur du début des crises temporales ipsilatérales. Des cas de fausse latéralisation et localisation semblent refléter une propagation de la crise aux structures temporales contralatérales ou ipsilatérales respectivement. L'implication de l'amygdale semble être d'importance capitale dans l'induction du FNP.

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Postictal noserubbing or nosewiping (PIN) has recently been described as a lateralizing and localizing sign in human partial epilepsy.[1-4] Specifically, the occurrence of PIN is highly suggestive of ipsilateral partial seizure onset, usually localized to the temporal lobe. Geyer et al[4] proposed a link between PIN and olfactory auras, however, others have found no such association and the most prevalent speculation is that PIN represents a reaction to increased nasal secretions associated with ictal limbic activation of central autonomic pathways.[1-3]

PIN was first brought to the attention of this author some years ago as an interesting, albeit imperfect, indicator of side of partial seizure onset (Drs. P. Gloor, L.F. Quesney, personal communications). In an attempt to understand why the lateralizing and localizing value of PIN is imperfect and, in so doing, perhaps gain insight into the mechanisms underlying this

From the Division of Neurology, University Health Network, Toronto Western Hospital, University of Toronto, Ontario, Canada.

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Reprint requests to: R. Wennberg, Toronto Western Hospital, EC8 - 022, 399 Bathurst St., Toronto, Ontario, Canada M5T 2S8.

clinical phenomenon, the frequency of occurrence of PIN and its association with sites of electrographic seizure onset, spread and termination was studied prospectively in consecutive patients admitted over a two year period for video-EEG monitoring at the Toronto Hospital, University of Toronto.

Patients and methods

The study group consisted of all patients admitted to the Epilepsy Monitoring Unit (EMU) at the Toronto Hospital between April 1997 and April 1999. A majority of these patients suffered from medically intractable partial seizure disorders and were admitted for localization of seizure onset zone(s) to determine potential surgical candidacy. The remainder were investigated for diagnosis of seizure-like episodes, i.e. epileptic versus nonepileptic spells.

Patients were monitored with continuous video-EEG using scalp plus or minus sphenoidal electrodes. Scalp EEG recordings used the international 10-20 system of electrode placement plus zygomatic and subtemporal electrodes F9, F10, T9, T10, P9, P10. Three patients were investigated with intracranial electrodes: two with extratemporal epilepsy and one with bitemporal epilepsy as determined by previous scalp EEG investigations. The two extratemporal cases had no PIN events. The patient with bitemporal epilepsy, who demonstrated PIN, was investigated with bilateral depth electrodes implanted orthogonally through the second temporal gyrus with the deepest tips aimed at the amygdala and anterior hippocampal region, respectively, plus an additional depth electrode inserted orthogonally through the orbital frontal region bilaterally.

PIN was identified on videotape archived each day by EMU technologists. Video records typically included a number of minutes (usually two to five minutes) of postictal recording.

EEG recordings were scored for time and localization of seizure onset, the presence or absence of intra- and/or interhemispheric spread and time of electrographic offset. These EEG results were compared with the timing and laterality of PIN.

Seizures which underwent secondary generalization were not included in the analysis. Auras with no other evident clinical manifestations apparent on the video were also not included in the analysis.

Patients were classified into temporal or extratemporal groups based on the combination of electrographic, neuroimaging (MRI) and neuropsychological test results. Further localization in the extratemporal group was documented when sufficiently clear on the basis of the same findings. Lateralization was occasionally unclear in the extratemporal group, however, this did not affect the results of this study as lateralization was always clear in the extratemporal patients who demonstrated PIN. Patients with temporal lobe epilepsy were separated into two groups, mesial temporal and neocortical temporal, on the basis of neuroimaging and/or pathological findings or, in one case, based on the results of intracranial EEG recording. Thirty-one of 33 mesial temporal patients had neuroimaging evidence of mesial temporal atrophy or sclerosis, one mesial temporal case was identified through depth electrode recordings and the remaining mesial temporal case had a cavernous hemangioma restricted to the region of the right uncus. Five of the neocortial temporal cases had neuroimaging or pathological evidence of a temporal neocortical tumor, one had a neocortical cavernous hemangioma, five had encephalomalacic or gliotic lesions, either post-traumatic, postencephalitic or congenital and one had no identifiable structural lesion but scalp electrographic findings incompatible with a mesial temporal localization. Two of the temporal lobe patients could not, with certainty, be classified as either mesial or neocortical on the basis of the neuroimaging, scalp EEG or other findings. Twenty-three of the temporal lobe patients with study seizures had undergone surgery at the time of this writing (20 with mesial temporal and three with neocortical temporal epilepsy): intraoperative electrocorticography and surgical pathology results confirmed the preoperative localization in each case and all patients in the operated group became either seizure free or have had a greater than 90% reduction in seizure frequency.

Twenty-six of 39 extratemporal patients were lateralizable to one or the other hemisphere (20 right hemisphere, 12 of which were localized to the frontal lobe; six left hemisphere, three localized to the frontal lobe). The remaining 13 extratemporal patients showed bilateral/multifocal abnormalities, seven of whom could be classified as symptomatic (secondary) generalized epilepsy. Only one of the extratemporal patients underwent resective surgery during the study period. Four of the bilateral/multifocal extratemporal patients had nonresective surgical treatment in the form of bilateral deep brain stimulation of either the centromedian or anterior nucleus of the thalamus.

One patient with primary generalized 3 Hz spike and wave epilepsy was investigated because of a referring question of possible nonepileptic events.

Nonepileptic events were recorded in 17 patients, five of whom also had definite epilepsy (two right temporal and three right extratemporal). Eleven patients had only nonepileptic spells: four of these had a past history of head trauma and one had a previous sagittal sinus thrombosis and intracerebral hemorrhage. The remaining patient in this group was found to have a REM sleep disorder.

Statistical analyses used either the Chi-squared test with correction for continuity or, where indicated, the Fisher exact test. All p values are for two-tailed tests.

Results

General results

One hundred and fourteen patients were admitted to the EMU during the study period. Twenty-six had no study events, either because no events were recorded during admission (seven patients) or, most commonly, because all recorded seizures were simple auras and/or seizures with secondary generalization (19 patients: eight left temporal, three right temporal, eight extratemporal).

A summary of results and comparison with other recent reports of PIN is presented in the Table. PIN was seen in 23/41 patients (56%) with temporal lobe epilepsy and in 41/197 recorded temporal lobe seizures (21%). PIN was seen more commonly in patients with left temporal epilepsy and in seizures with left temporal onsets although the differences were not statistically significant ($p>0.2$ and $p>0.1$, respectively). PIN occurred in 15/27 mesial and 8/12 neocortical temporal lobe cases, a distribution showing no statistical difference ($p>0.7$).

Table: Reported incidence and lateralization of PIN in temporal and extratemporal epilepsy

Study	Temporal PIN			Extratemporal PIN		
	patients (%)	seizures (%)	ipsilateral (%)	patients (%)	seizures (%)	ipsilateral (%)
Hirsch et al[1]	28/47 (60)	74/171 (43)	23/25 (92)[a,b]	13/40 (33)	22/148 (15)	?
Rajan et al[2]	11/35 (31)	?	28/31 (90)[c,d]	0/18 (0)[e]	?	-
Leutmezer et al[3]	39/76 (51)	104/263 (40)	90/104 (87)[c]	3/25 (12)	11/181 (6)	6/11 (55)[c]
Geyer et al[4]	46/100 (46)	105/229 (46)	41/46 (89)[a]	5/50 (10)	?	?
Wennberg	23/41 (56)	41/197 (21)	34/41 (83)[c]	4/34 (12)	12/167 (7)	10/12 (83)[c]

[a] percentage of *patients* with PIN who used the hand ipsilateral to seizure onset to rub the nose
[b] excluded patients who had episodes of both ipsilateral and contralateral PIN
[c] percentage of *seizures* with PIN where the patient used the hand ipsilateral to seizure onset to rub the nose
[d] approximate percentage; included episodes of ictal noserubbing
[e] one extratemporal patient reported to have ictal noserubbing

The hand used to rub the nose was highly correlated with the side of temporal lobe seizure onset ($p<0.001$; $\chi^2=14.69$).

PIN was significantly less frequent in patients with extratemporal epilepsy and in seizures of extratemporal onset (both $p<0.001$; $\chi^2=13.16$ and 12.79, respectively), occurring in 4/34 extratemporal patients (12%) and in 12/167 extratemporal seizures (7%). The hand used to rub the nose was more likely to be ipsilateral to the site of seizure onset (83% ipsilateral) although the numbers were insufficient for this trend to reach statistical significance ($p>0.3$).

Three of 27 patients with partial epilepsy and PIN were lefthanded (two with left mesial temporal sclerosis and one with right frontal epilepsy). Handedness was not correlated with the hand used to rub the nose (dominant hand used in 57% of seizures; $p>0.3$). Twelve of 27 patients (44%) with partial epilepsy and PIN showed contralateral ictal dystonia[5] (9/12) or tonic posturing (3/12, with or without some low amplitude clonic movements) in at least one recorded seizure (not necessarily a seizure with PIN). One patient with ictal dystonia showed contralateral ictal neglect[6] in another seizure. No discernable postictal Todd's paralysis was evident in any of the patients with PIN as could best be determined from the videotape. None of the study patients had olfactory auras.

The patient with primary generalized epilepsy had numerous subclinical bursts of generalized spike and wave activity as well as three brief clinical seizures associated with absence, upward deviation of the eyes and rapid eyelid flutter. Surprisingly, following one of the three clinical seizures the patient exhibited PIN with the right (dominant) hand identical to that seen in the patients with partial epilepsy.

PIN was not seen in any of 63 recorded nonepileptic events in 17 patients.

PIN was not seen beyond two minutes after electrographic offset. PIN occurred within 30 seconds of seizure offset in 33/54 seizures and within 60 seconds of offset in 51/54 seizures. The remaining three episodes of PIN occurred within two minutes of seizure offset. In one case of contralateral PIN the patient was observed to be leaning on his ipsilateral arm at the time of the noserubbing behavior. The ipsilateral arm appeared to be free to move in all other cases of contralateral PIN. As described in previous reports,[1,3,4] the noserubbing was often performed rather vigorously, and in a minority of cases repeated once or twice after 10-30 seconds.

Ictal noserubbing was observed in five seizures in four patients (two temporal and two extratemporal). The hand ipsilateral to seizure onset was used in 4/5 events: the other seizure showed contralateral ictal noserubbing one second prior to offset (the same right frontal onset seizure had contralateral PIN).

Electroclinical correlations

Episodes of ipsilateral PIN were slightly more likely to show unilateral seizure offsets with no evidence of spread to the contralateral hemisphere (24/44 seizures, 55%) than they were to show bilateral seizure offsets recorded either independently or synchronously over the frontal temporal regions or with a more generalized distribution. In contrast, contralateral PIN was significantly correlated with seizure spread to the contralateral temporal lobe ($p<0.04$, Fisher exact test). Ictal activity spread to involve both hemispheres in 8/9 seizures with contralateral PIN, with offset recorded either with bilateral synchrony (6/8) or in a bilaterally independent fashion (2/8): one with final offset contralateral to seizure onset and one with final offset ipsilateral to seizure onset. The remaining seizure with contralateral PIN was a case of right mesial temporal epilepsy with no evidence of contralateral electrographic spread. This patient had, in addition to the one episode of contralateral noserubbing, six episodes of ipsilateral PIN.

Analysis of the extratemporal cases included one patient with a previously resected right parietal-occipital arteriovenous malformation who demonstrated PIN in two of three recorded seizures. Both of these seizures showed extratemporal onset over the right posterior quadrant, maximal over the midparietal region with subsequent spread to involve the temporal lobe structures, where offset was recorded over the right temporal region prior to ipsilateral PIN (Figure 1). Another extratemporal patient with right frontal epilepsy had both ipsilateral (6/8 recorded seizures) and contralateral (2/8 recorded seizures) PIN. In this patient, all

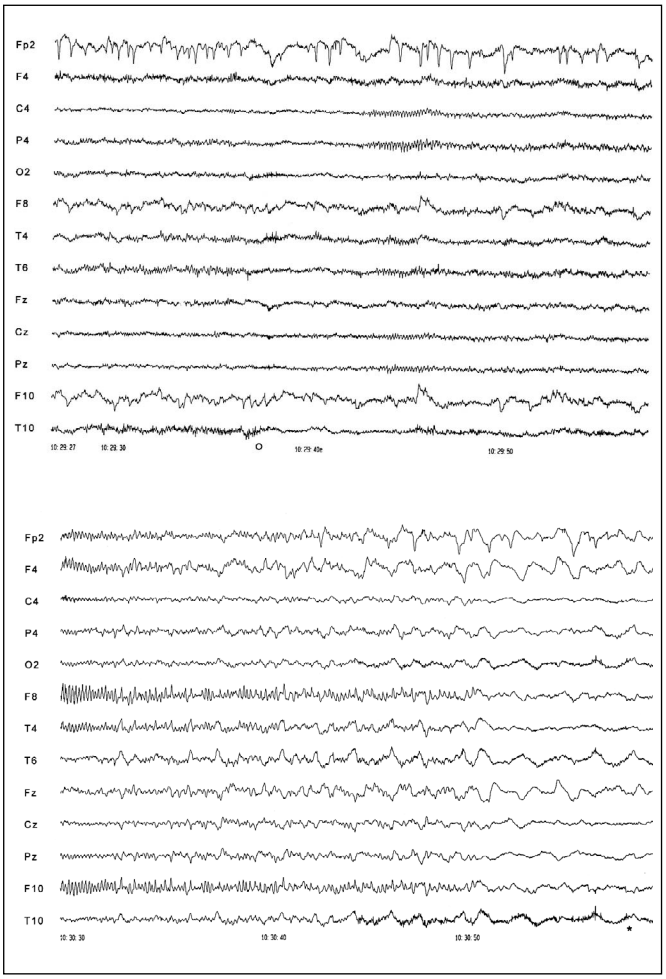

Figure 1: *A. (Top) Clinical and electrographic seizure onset at* "O" *as attenuation and subsequent rhythmic 6 Hz ictal activity recorded over the right posterior quadrant (maximal P4 > C4 > Pz, Cz, T6) in patient with a previously resected right parietal-occipital arteriovenous malformation. B. (Bottom) Termination of same seizure shows spread of ictal activity to anterior temporal structures prior to offset (F8 > F10). PIN occurred at* "★". *Average referential montage.*

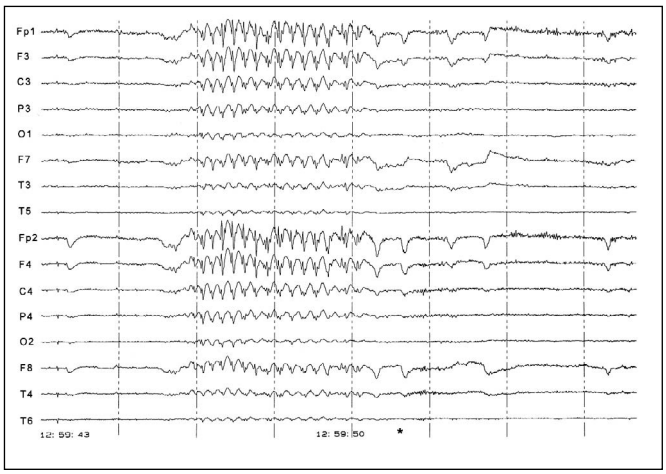

Figure 2: *Brief 3 Hz generalized spike and wave seizure associated with absence, upward eye deviation and eyelid flutter, followed by PIN at* ★ *and again 19 seconds later. Ipsilateral mastoid referential montage. Time bar interval = 2 s.*

recorded seizures showed a distinct onset over the right frontal polar region with subsequent spread to involve first the midfrontal area and ultimately the right temporal lobe structures prior to unilateral offset (in the six seizures with ipsilateral PIN) or further progression to bilateral frontal temporal offset (in the two seizures with contralateral PIN). The remaining two patients with extratemporal epilepsy and PIN included one patient with band heterotopia where PIN was observed in 1/11 bihemispheric seizures showing an electrographic maximum over the left posterior temporal region, and a second patient with right parietal seizures with 1/3 recorded events showing ipsilateral PIN with ictal involvement of the entire right hemisphere prior to offset.

The patient investigated with bilateral intracranial depth electrodes was found to have right mesial temporal epilepsy and ipsilateral PIN. Multiple focal subclinical electrographic seizures restricted to the right anterior hippocampal contacts were recorded during her investigation. All 18 recorded clinical seizures, and all seizures with PIN, showed a focal right anterior hippocampal onset with subsequent spread to the amygdala and then right orbital frontal region.

Figure 2 shows the brief 3 Hz spike and wave absence seizure recorded in the patient with primary generalized epilepsy which was associated with typical PIN.

Discussion

PIN has been described to be a useful lateralizing and localizing sign in human partial epilepsy. The hypothetical mechanism may be a response to olfactory auras[4] or, more likely, a response to increased nasal secretions caused by ictal autonomic activity which becomes clinically manifest as the patient regains awareness in the postictal period.[1-3] The latter hypothesis has led to speculation about the anatomical region involved in this phenomenon and most discussion has centered on the amygdala,[1] well-known to have multiple connections with subcortical/brain stem autonomic structures.[7-9] The results of electrical stimulation studies performed by Ferrier[10] over a century ago are relevant in this regard: stimulation restricted to the region of the amygdala/uncus in rabbits, cats, dogs, and monkeys induced nasal torsion, as if "the nostril is irritated by some pungent odor" or "as if from irritation directly applied to the nostril". Stimulation at no other cortical or limbic site induced this response.

The results of this study confirm the findings of previous reports showing PIN to be a reliable marker of ipsilateral partial seizure onset typically localized to the temporal lobe. The percentage of patients with temporal or extratemporal epilepsy demonstrating PIN (56% and 12%, respectively) was similar to that seen in previous studies. The percentage of temporal lobe seizures demonstrating PIN was, however, lower than previously described. The reason for this is unclear but could represent inclusion of more seizures with subtle clinical manifestations in

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this study which may have been excluded as auras in other reports.

Consistent with previous reports,[2,4] PIN was never seen in the setting of nonepileptic spells. In contrast with previous reports where PIN has been described to not occur in the setting of generalized epilepsy,[2,4] the only patient included in this series with primary generalized epilepsy demonstrated PIN after one of three recorded seizures. It is not, however, entirely clear what is meant by generalized epilepsy in the other studies and it may be that most such patients had instead bilateral or multifocal epilepsy initiating their investigation in a monitoring unit.

Also, in keeping with previous reports, PIN was seen significantly more frequently in the setting of temporal as compared with extratemporal partial epilepsy. When occurring in the setting of extratemporal epilepsy the hand used to rub the nose was just as likely to be ipsilateral to seizure onset as was the case for temporal onset seizures, although the small number of extratemporal seizures with PIN did not allow this trend to reach statistical significance. Previous studies did not systematically analyze the electrographic findings associated with PIN. The extratemporal cases with PIN documented in this report provide evidence that temporal lobe structures need to be involved during seizure progression for PIN to occur. All cases of extratemporal seizure onsets with PIN showed subsequent seizure spread after onset to involve the temporal lobe.

The single case of temporal lobe epilepsy with PIN that was investigated with depth electrodes provides evidence supporting the speculation that the amygdala, or at least structures in the region of the amygdala, are of prime importance in the induction of PIN. Consistent with previous reports,[11,12] focal hippocampal seizures which did not spread to adjacent mesial structures never manifested clinical features. All clinical seizures with PIN showed spread to the ipsilateral amygdala, and subsequently to the ipsilateral orbital frontal region. Although these data cannot exclude the possibility that involvement of the orbitofrontal region is necessary to induce PIN, this would not be supported by the observation that frontal lobe seizures, in general, are much less likely to show PIN than temporal lobe seizures.

The mechanism underlying PIN in the setting of generalized 3 Hz spike and wave activity is unclear but could presumably involve ictal activation of the amygdala with the spike and wave activity. Alternatively, the observation of PIN in one of three recorded seizures may have been a coincidence. Nevertheless, the clinical manifestation of the noserubbing was identical to that seen with partial seizures with the patient rubbing his nose once immediately postictally and again, more vigorously, 19 seconds later, such that one is inclined to believe a similar outflow system may have been activated.

The reason for which PIN was seen more frequently in left temporal lobe seizures than right during this study is not entirely clear but likely represents a chance distribution. One previous report described PIN to occur more frequently with right temporal lobe seizures[3] while other reports have shown no difference.[1,4] The difference in this study was not significant and may simply have represented the fact that more patients investigated with left temporal lobe epilepsy during this study were excluded from analysis because all recorded seizures were either simple auras with no outward clinical manifestations and/or seizures with secondary generalization.

If it seems relatively clear that ictal involvement of the temporal lobe and, more specifically, the amygdala, underlies the phenomenon of PIN, an unresolved issue is why the noserubbing is done preferentially with the ipsilateral hand. The two most obvious possibilities are that: (a) the laterality reflects lateralized autonomic activity induced by the ictal discharge in the form of increased nasal secretions ipsilateral to the side of seizure onset, with the noserubbing performed ipsilaterally because that is where the predominant piloerection and/or rhinorrhea exists, or, (b) the laterality represents a motor phenomenon with the ipsilateral hand used because of contralateral postictal paresis or neglect. The former hypothesis is supported by the observation that in all but one case of contralateral PIN electrographic seizure progression showed spread to the temporal lobe contralateral to the hemisphere of onset. Given that contralateral PIN was analyzed only with scalp EEG recordings it is possible that the one case without evident contralateral progression may have had spread to the contralateral limbic structures detectable with intracranial recording. Contralateral spread could induce bilateral nasal secretions which could then underlie occasional use of the contralateral hand in rubbing the nose after regaining awareness. This would be compatible with the observation that some patients with contralateral PIN (2/5) had other episodes with ipsilateral PIN, and the observation in one patient that strictly lateralized electrographic seizures were associated only with ipsilateral PIN, whereas contralateral PIN was observed in seizures which showed contralateral spread. The possibility that the laterality is representative of lateralized autonomic discharge is also supported by the observations of Ferrier[10] where stimulation of the amygdala/uncus in cats, dogs and monkeys caused ipsilateral movements of the nostril (although bilateral nasal torsion was seen with uncal stimulation in rabbits).

In support of the second hypothesis is the finding that nearly half of the patients with PIN were observed in at least one seizure to have ictal contralateral dystonia, neglect, or other motor involvement of the upper extremity which could theoretically be associated with postictal paresis or neglect and thus a preference to rub the nose with the ipsilateral arm. As these phenomena are typically observed only on videotape and not during realtime motor and sensory examination, ictal and postictal sensorimotor affliction of the contralateral upper extremity may be more prevalent than thought. Definitive differentiation between the two hypothetical mechanisms underlying the laterality of PIN would require both realtime ictal and postictal sensorimotor examination as well as bilateral measurement of piloerection and nasal secretion.

In summation, the occurrence of PIN in a patient with undiagnosed spells confirms that the ictal events in question are epileptic in origin and makes it highly likely that the site of seizure onset is ipsilateral to the hand used to rub the nose and usually localized to the temporal lobe. False localization and lateralization does occur rarely, however, and in these cases appears to be dependent on spread of ictal activity from extratemporal to temporal structures and to contralateral temporal structures, respectively. In addition, typical PIN can be seen after primary generalized absence spells and is thus not absolutely specific for partial epilepsy. Results from intracranial recording support previous speculation[1] that the amygdala may be the anatomical site most implicated in this curious clinical phenomenon.

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