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Effects of age and underlying brain dysfunction on the postictal state

William H. Theodore*

Clinical Epilepsy Section, National Institutes of Health, Bethesda, MD, USA

Abstract

There is relatively little information on the underlying parameters that affect clinical features of the postictal period. Age-related physiological changes, including alterations in cerebral blood flow and metabolism, neurotransmitter function, and responses of the brain to seizure activity may affect postictal clinical phenomena. Some conclusions can be drawn. Elderly adults and children, particularly in the presence of diffuse cerebral dysfunction, may have more prolonged postictal confusion. Postictal dysphasia strongly suggests a dominant hemisphere focus, more often temporal, and Todd's paralysis is always contralateral to the epileptogenic zone. Much additional information could be derived from the vast amount of video/EEG monitoring data available.

Keywords

Postictal; Children; Elderly; Metabolism; Blood flow; Lesion; Focal neurologic signs

1. Pathophysiology

There are relatively limited data on the effect of clinical parameters on the postictal state. However, the physiological changes that occur may be more complex than simple models based on functional “depression” assume [1]. After relatively brief complex partial seizures (CPSs), hypometabolism may persist, and even decline further, for 24–48 hours after a seizure, particularly in inferior temporal regions [2]. Reduced cortical excitability measured by transcranial magnetic stimulation may also last up to 24 hours [3]. In addition to changing cerebral blood flow (CBF) and glucose metabolism (CMRglc), neurotransmitter release and receptor availability may be altered. Opiate receptor availability may be increased for up to 8 hours after a seizure, possibly reflecting an overshoot below basal levels after the ictal endogenous opiate released occupied available binding sites [4]. It is interesting that SPECT scans during postictal psychosis reveal bifrontal and bitemporal hyperperfusion [5].

A focal lesion might be expected to predispose patients to particular postictal signs, just as it can influence ictal phenomena. A mass lesion such as an astrocytoma might have different effects than an arteriovenous malformation. Tumors and malformations, for example, have differing effects on the functional anatomy of language measured with fMRI activation studies, but potential effects on postictal aphasia have not been investigated [6]. MRI immediately after a seizure in a patient with a tumor may show transiently increased T2 or gadolinium enhancement, incorrectly suggesting lesion progression [7].

* Clinical Epilepsy Section, National Institutes of Health, Building 10, Room 7C-103, Bethesda, MD 20892, USA. Fax: +1 301 402 2871. theodorw@ninds.nih.gov..

Differences in CBF, CMR_{glc}, and neuroreceptor availability might have age-related clinical effects as well. Children have higher cerebral blood flow and metabolism than adults, with values gradually declining through adolescence until about 18 years of age [8,9]. Similar data have been reported for maturation of the GABA–benzodiazepine receptor complex [10]. The elderly show age-related declines in CBF, cerebral metabolism, and muscarinic, dopamine, and serotonin receptors [11–16].

Age-related differences in seizure etiology could interact with underlying physiological processes. Children may be more likely to have, for example, malformations of cortical development. The elderly have a high incidence of epilepsy that may be related to cerebrovascular disease, primary dementias, or acquired metabolic disturbances [17]. Global or focal neurological impairment may predispose patients to prolonged postictal confusion or more prominent focal signs. Age-related differences in drug metabolism, as well as varying effects of withdrawal of specific antiepileptic drugs during video/EEG monitoring, might affect the length, and clinical expression, of the postictal period.

This article reviews data on the effects of age, epileptic focus location, and underlying lesions on several clinical manifestations of the postictal period.

2. Postictal duration

Postictal periods in temporal and frontal lobe epilepsy usually are short [18]. In 132 postictal periods following CPSs in adults, mean duration was 89 seconds, although the longest was 767 seconds [19]. Postictal automatisms occurred in 19 seizures. There was no relation of clinical parameters such as age, age at epilepsy onset, or etiology to postictal duration; the oldest patient was 58. One study suggested that patient older than 60 were less likely to be “responsive” after CPSs [20]. Patients with epilepsy onset after age 18 had a tendency toward longer postictal duration [21]. Seizures starting in the dominant hemisphere may be associated with longer postictal recovery [22].

In children seen in an emergency room, the median time to recovery of full consciousness was considerably longer: 38 minutes [23]. However, patients who received antiepileptic drugs, particularly benzodiazepines, as well as those with symptomatic as opposed to idiopathic or febrile seizures, had significantly longer recovery times [23,24]. Children who underwent MRI within 48 hours of a prolonged generalized febrile seizure had evidence of large hippocampi and prolongation of T2 relaxation time; scans performed between 48 hours and 5 days after the seizure showed large hippocampi but normal T2, suggesting resolving edema [25]. Children may be more susceptible than adults to postictal brain structural perturbations, although a comparative study has not been performed.

3. Postictal cognitive disturbances

Rapid return to full consciousness, without clear postictal confusion, may be more common in frontal than temporal lobe epilepsy, potentially increasing the chance of diagnostic confusion with primary generalized epilepsy [18]. Patients with prolonged (≤ 10 days) postictal confusion were likely to have diffuse cortical atrophy and mild baseline cognitive dysfunction in a small series [26]. The episodes always occurred after a seizure cluster or status epilepticus.

Patients with postictal dysphasia tended to have been older at seizure onset, and were significantly older at the time of study [19]. In another study, age did not affect postictal language disturbances [27]. An unusual transient postictal transcortical aphasia has been reported in a 67-year-old patient [28]. Fluent, nonfluent, or global postictal dysphasia is more common in patients with dominant hemisphere temporal foci [22]. In patients with nondominant temporal foci, postictal dysphasia was significantly longer when seizures

propagated to the dominant temporal lobe [29]. The effect of structural lesions is uncertain. Postictal dysphasia occurred in 7% of patients with nontumoral, as compared with 18% of patients with tumoral, parietal lobe epilepsy [30,31].

Postictal “transient global amnesia” is said to occur rarely in elderly patients because of seizures, implying that patients thought to have a vascular etiology should undergo electroencephalography [32]. Thirty patients had a late onset “epileptic amnesic syndrome” that appeared to be a postictal manifestation of frequent brief CPSs [33]. Older adults presenting with memory disturbances, particularly if they appear to be intermittent, should have thorough EEG studies.

4. Motor phenomena

“Todd's paralysis” is one of the most well-known sequelae of seizures [34]. Some small series suggest that patients with structural lesions, and the elderly, may be more likely to have transient focal weakness after a seizure [35]. In addition to pure motor disturbances, ipsilateral ataxia may occasionally accompany weakness [36].

Postictal weakness was found in 44 of 513 patients (13.4%) during video/EEG monitoring [37]. It was always unilateral and always contralateral to the seizure focus; median duration was 173.5 seconds (range: 11 seconds–22 minutes). Eighty-eight percent of seizures associated with postictal weakness were accompanied by ipsilateral ictal motor phenomena. There was no difference in the frequency of lesions or focus location between patients with and without postictal paralysis. However, the weakness lasted longer in patients without lesions (median: 356 seconds vs 140 seconds) than in patients with a structural abnormality on MRI (including mesial temporal sclerosis) [37]. Todd's paralysis was seen in 22% of patients with nontumoral parietal lobe epilepsy compared with 32% of patients with parietal lobe foci caused by tumors [30,31].

Other studies, based on chart review rather than videotape analysis, found a lower incidence of postictal weakness and suggested a preponderance of frontal foci [38,39]. Postictal weakness did not occur in nonepileptic events [39].

“Inhibitory seizures” (transient neurological impairment not associated with focal seizures or loss of consciousness) occur in a small proportion of patients admitted for evaluation of potential stroke [40]. They are much more likely to occur in the elderly, and can be difficult to distinguish from transient ischemic attacks. Speech disturbances, confusion, and partial amnesia were the most common symptoms [40]. In contrast to transient ischemic attacks, EEG findings, usually intermittent rhythmic delta activity, are evident during or after the episodes. These events are particularly interesting because they seem to occur in the “borderlands” of the ictal and postictal states.

Postictal nose rubbing is said to be significantly more frequent in temporal than extratemporal epilepsy; in 83% of cases the hand used to rub was ipsilateral to the focus [41].

5. Visual disturbances

Visual features, particularly postictal homonymous hemianopsia or blindness, lasting from minutes to days, are more common in childhood, according to some investigators [42–45]. Seizure foci usually are occipital or occipitotemporal. Some patients have paroxysmal headaches as well, and misdiagnosis as migraine may occur. There does not seem to be a clinical difference between patients with and those without occipital lesions; some had encephalitis or cerebral malaria. A review of occipital epilepsy found a mean age at onset of 17; two patients presenting in their fifties had trauma and a glioma [46].

Older patients can have occipital seizures associated with postictal visual loss [47]. Case reports suggest that symptoms may last longer or even be permanent in older patients or as a sequela of status epilepticus [48].

6. Postictal psychoses

Postictal psychoses are one of the most troubling manifestations of epilepsy. In a comparison of 59 patients with a history of postictal psychosis and 94 without this history, the former had a significantly higher incidence of extratemporal foci or bilateral foci, as well as a family history of psychiatric disorders, and possibly encephalitis [49]. There was no difference in age, sex, or MRI findings. Other investigators have noted the prevalence of bilateral EEG foci as well [50]. Some, in contrast, found that frontal or temporal foci were more common [51,52]. Overall, seizure focus and laterality, age at epilepsy onset, and duration have not been shown to be significant factors [53]. Postictal mania, in contrast, was associated with dominant frontal foci [52]. Withdrawal of “mood-stabilizing” antiepileptic drugs such as carbamazepine might be more likely to provoke psychopathology during video/EEG monitoring [54].

7. Conclusion

There is relatively little information on the underlying parameters that affect clinical features of the postictal period. Elderly adults and children, particularly in the presence of diffuse cerebral dysfunction, may have more prolonged postictal confusion. These observations may be useful when pharmacological intervention is being considered, particularly because the elderly may be on multiple medications and have reduced drug clearance. Two features may be of lateralizing value. Postictal dysphasia strongly suggests a dominant hemisphere focus, more often temporal, and Todd's paralysis is always contralateral to the epileptogenic zone. Perhaps surprisingly, postictal motor phenomena do not seem to be more common in patients with structural lesions. Given the enormous amount of video/EEG monitoring data available, the limits of our knowledge are surprising.

References

1. Fisher RS, Schachter SC. The postictal state: a neglected entity in the management of epilepsy. *Epilepsy Behav* 2000;1:52–9. [PubMed: 12609127]
2. Leiderman DB, Albert P, Balish M, Bromfield E, Theodore WH. The dynamics of metabolic change following seizures as measured by positron emission tomography with fludeoxyglucose F 18. *Arch Neurol* 1994;51:932–6. [PubMed: 8080394]
3. Badawy R, Macdonell R, Jackson G, Berkovic S. The peri-ictal state: cortical excitability changes within 24 h of a seizure. *Brain* 2009;132:1013–21. [PubMed: 19251759]
4. Hammers A, Asselin MC, Hinz R, et al. Upregulation of opioid receptor binding following spontaneous epileptic seizures. *Brain* 2007;130:1009–16. [PubMed: 17301080]
5. Leutmezer F, Podreka I, Asenbaum S, et al. Postictal psychosis in temporal lobe epilepsy. *Epilepsia* 2003;44:582–90. [PubMed: 12681009]
6. Bookheimer S. Pre-surgical language mapping with functional magnetic resonance imaging. *Neuropsychol Rev* 2007;17:145–55. [PubMed: 17484055]
7. Finn MA, Blumenthal DT, Salzman KL, Jensen RL. Transient postictal MRI changes in patients with brain tumors may mimic disease progression. *Surg Neurol* 2007;67:246–50. [PubMed: 17320628]
8. Chiron C, Raynaud C, Mazière B, et al. Changes in regional cerebral blood flow during brain maturation in children and adolescents. *J Nucl Med* 1992;33:696–703. [PubMed: 1569478]
9. Takahashi T, Shirane R, Sato S, Yoshimoto T. Developmental changes of cerebral blood flow and oxygen metabolism in children. *AJNR Am J Neuroradiol* 1999;20:917–22. [PubMed: 10369366]
10. Chugani DC, Muzik O, Juhász C, Janisse JJ, Ager J, Chugani HT. Postnatal maturation of human GABAA receptors measured with positron emission tomography. *Ann Neurol* 2001;49:618–26. [PubMed: 11357952]

11. Dewey SL, Volkow ND, Logan J, et al. Age-related decreases in muscarinic cholinergic receptor binding in the human brain measured with positron emission tomography (PET). *J Neurosci Res* 1990;27:69–75.
12. Rinne JO, Hietala J, Ruotsalainen U, et al. Decrease in human striatal dopamine D2 receptor density with age: a PET study with [¹¹C]raclopride. *J Cereb Blood Flow Metab* 1993;13:310–4. [PubMed: 8436624]
13. Iyo M, Yamasaki T. The detection of age-related decreases of dopamine D1, D2 and serotonin 5-HT2 receptors in living human brain. *Prog Neuropsychopharmacol Biol Psychiatry* 1993;17:415–21. [PubMed: 8475323]
14. Takada H, Nagata K, Hirata Y, et al. Age-related decline of cerebral oxygen metabolism in normal population detected with positron emission tomography. *Neurol Res* 1992;14(2 Suppl):128–31. [PubMed: 1355868]
15. Martin AJ, Friston KJ, Colebatch JG, Frackowiak RS. Decreases in regional cerebral blood flow with normal aging. *J Cereb Blood Flow Metab* 1991;11:684–9. [PubMed: 2050757]
16. Møller M, Jakobsen S, Gjedde A. Parametric and regional maps of free serotonin 5HT1A receptor sites in human brain as function of age in healthy humans. *Neuropsychopharmacology* 2007;32:1707–14. [PubMed: 17251909]
17. Jetter GM, Cavazos JE. Epilepsy in the elderly. *Semin Neurol* 2008;28:336–41. [PubMed: 18777480]
18. Laskowitz DT, Sperling MR, French JA, O'Connor MJ. The syndrome of frontal lobe epilepsy: characteristics and surgical management. *Neurology* 1995;45:780–7. [PubMed: 7723970]
19. Theodore WH, Porter RJ, Penry JK. Complex partial seizures: clinical characteristics and differential diagnosis. *Neurology* 1983;33:1115–21. [PubMed: 6684245]
20. Kellinghaus C, Loddenkemper T, Dinner DS, Lachhwani D, Lüders HO. Seizure semiology in the elderly: a video analysis. *Epilepsia* 2004;45:263–7. [PubMed: 15009228]
21. Villanueva V, Serratos JM. Temporal lobe epilepsy: clinical semiology and age at onset. *Epileptic Disord* 2005;7:83–90. [PubMed: 15929909]
22. Williamson PD, Thadani VM, French JA, et al. Medial temporal lobe epilepsy: videotape analysis of objective clinical seizure characteristics. *Epilepsia* 1998;39:1182–8. [PubMed: 9821982]
23. Allen JE, Ferrie CD, Livingston JH, Feltbower RG. Recovery of consciousness after epileptic seizures in children. *Arch Dis Child* 2007;92:39–42. [PubMed: 16820389]
24. McKenny-Fick NM, Ferrie CD, Livingston JH, Taylor JC, Allen JE. Prolonged recovery of consciousness in children following symptomatic epileptic seizures. *Seizure* 2009;18:180–3. [PubMed: 18835729]
25. Scott RC, Gadian DG, King MD, et al. Magnetic resonance imaging findings within 5 days of status epilepticus in childhood. *Brain* 2002;125:1951–9. [PubMed: 12183341]
26. Biton V, Gates JR, dePadua Sussman L. Prolonged postictal encephalopathy. *Neurology* 1990;40:963–6. [PubMed: 2345618]
27. Ficker DM, Shukla R, Privitera MD. Postictal language dysfunction in complex partial seizures: effect of contralateral ictal spread. *Neurology* 2001;56:1590–2. [PubMed: 11402125]
28. Goldberg-Stern H, Gadoth N, Ficker D, Privitera M. The effect of age and structural lesions on postictal language impairment. *Seizure* 2005;14:62–5. [PubMed: 15642503]
29. Yankovsky AE, Treves TA. Postictal mixed transcortical aphasia. *Seizure* 2002;11:278–9. [PubMed: 12027578]
30. Salanova V, Andermann F, Rasmussen T, Olivier A, Quesney LF. Tumoural parietal lobe epilepsy: clinical manifestations and outcome in 34 patients treated between 1934 and 1988. *Brain* 1995;118:1289–304. [PubMed: 7496787]
31. Salanova V, Andermann F, Rasmussen T, Olivier A, Quesney LF. Parietal lobe epilepsy: clinical manifestations and outcome in 82 patients treated surgically between 1929 and 1988. *Brain* 1995;118:607–27. [PubMed: 7600082]
32. Tassinari CA, Ciarmatori C, Alesi C, et al. Transient global amnesia as a postictal state from recurrent partial seizures. *Epilepsia* 1991;32:882–5. [PubMed: 1743160]
33. Gallassi R. Epileptic amnesic syndrome: an update and further considerations. *Epilepsia* 2006;47 (Suppl 2):103–5. [PubMed: 17105476]

34. Binder DK. A history of Todd and his paralysis. *Neurosurgery* 2004;54:480–6. [PubMed: 14744294]
35. Meyer JS, Portnoy HD. Postictal paralysis: a clinical and experimental study. *Brain* 1959;82:162–85.
36. Bansal SK, Chopra JS. Reversible postictal ataxic hemiparesis. *Ital J Neurol Sci* 1991;12:75–9. [PubMed: 2013527]
37. Gallmetzer P, Leutmezer F, Serles W, Assem-Hilger E, Spatt J, Baumgartner C. Postictal paresis in focal epilepsies—incidence, duration, and causes: a video-EEG monitoring study. *Neurology* 2004;62:2160–4. [PubMed: 15210875]
38. Kellinghaus C, Kotagal P. Lateralizing value of Todd's palsy in patients with epilepsy. *Neurology* 2004;62:289–91. [PubMed: 14745071]
39. Urrestarazu E, Iriarte J, Alegre M, et al. Postictal paralysis during video-EEG monitoring studies. *Rev Neurol* 2002;35:404–7. [PubMed: 12373669]
40. De Reuck J, Van Maele G. Transient ischemic attacks and inhibitory seizures in elderly patients. *Eur Neurol* 2009;62:344–8. [PubMed: 19776589]
41. Wennberg R. Electroclinical analysis of postictal noserubbing. *Can J Neurol Sci* 2000;27:131–6. [PubMed: 10830346]
42. Kosnok E, Paulson GW, Laguna JF. Postictal blindness. *Neurology* 1976;26:248–50. [PubMed: 814481]
43. Shahar E, Barak S. Favorable outcome of epileptic blindness in children. *J Child Neurol* 2003;18:12–6. [PubMed: 12661932]
44. Genizi J, Zelnik N, Ravid S, Shahar E. Childhood epilepsy with occipital paroxysms: difficulties in distinct segregation into either the early-onset or late-onset epilepsy subtypes. *J Child Neurol* 2007;22:588–92. [PubMed: 17690066]
45. Caraballo R, Koutroumanidis M, Panayiotopoulos CP, Fejerman N. Idiopathic childhood occipital epilepsy of Gastaut: a review and differentiation from migraine and other epilepsies. *J Child Neurol* 2009;24:1536–42. [PubMed: 19955346]
46. Panayiotopoulos CP. Visual phenomena and headache in occipital epilepsy: a review, a systematic study and differentiation from migraine. *Epileptic Disord* 1999;1:205–16. [PubMed: 10937155]
47. Hadjikoutis S, Sawhney IM. Occipital seizures presenting with bilateral visual loss. *Neurol India* 2003;51:115–6. [PubMed: 12865542]
48. Joseph JM, Louis S. Transient ictal cortical blindness during middle age: a case report and review of the literature. *J Neuro-ophthalmol* 1995;15:39–42.
49. Alper K, Kuzniecky R, Carlson C, et al. Postictal psychosis in partial epilepsy: a case-control study. *Ann Neurol* 2008;63:602–10. [PubMed: 18481288]
50. Kanner AM, Ostrovskaya A. Long-term significance of postictal psychotic episodes: I. Are they predictive of bilateral ictal foci? *Epilepsy Behav* 2008;12:150–3. [PubMed: 18086458]
51. Adachi N, Ito M, Kanemoto K, et al. Duration of postictal psychotic episodes. *Epilepsia* 2007;48:1531–7. [PubMed: 17386048]
52. Nishida T, Kudo T, Inoue Y, et al. Postictal mania versus postictal psychosis: differences in clinical features, epileptogenic zone, and brain functional changes during postictal period. *Epilepsia* 2006;47:2104–14. [PubMed: 17201710]
53. Devinsky O. Postictal psychosis: common, dangerous, and treatable. *Epilepsy Curr* 2008;8:31–4. [PubMed: 18330462]
54. Ketter TA, Malow BA, Flamini R, White SR, Post RM, Theodore WH. Anticonvulsant withdrawal-emergent psychopathology. *Neurology* 1994;44:55–61. [PubMed: 8290092]