Epileptic Disord 2007; 9 (4): 449-52

Ipsilateral blinking seizures during left fronto-temporal ictal pattern on scalp EEG

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Received June 13, 2007; Accepted August 29, 2007

ABSTRACT – We report an infant with left eye blinking seizures accompanying a left (ipsilateral) fronto-temporal scalp EEG ictal pattern. The epileptogenic lesion was a left frontal encephalomalacia along the ventriculo-peritoneal shunt tract. The shunt was inserted for treatment of communicating hydrocephalus. This case illustrates the lateralizing value of the ictal blinking. Review of the literature suggests that seizures with unilateral blinking are likely to be produced by activation of ipsilateral trigeminal fibers innervating subdural intracranial structures and pial vessels in temporal and frontal lobes. Ipsilateral blinking could also be produced by activation of the ipsilateral cerebellar hemisphere.

Key words: ictal ipsilateral blinking, epilepsy, children, lateralizing signs

Unilateral blinking not associated with facial clonic twitching is a rare ictal phenomenon with an estimated frequency of approximately 1.5% (Benbadis *et al.* 1996) in children and adults. The mechanism for this ictal phenomenon is unknown, but ipsilateral cerebral hemisphere or cerebellar connections have been implicated (Mesiwala *et al.* 2002). We report an infant with partial seizures, with exclusive left eye blinking semiology accompanying a left fronto-temporal electrographic ictal pattern on scalp EEG. This case suggests the lateralizing value of unilateral ictal blinking.

Case study

A 3-month-old male underwent video-EEG evaluation to characterize episodes that had begun two days prior to admission. The episodes were stereotypical, and were described as sudden left eye blinking, high-pitched cry, flushing, sweating, and stiffening of the whole body for 30-45 seconds. He had a total of eight episodes in the two days prior to evaluation. The infant was one of an identical twin pregnancy, born at 29 weeks gestation to a 32-year-old mother whose pregnancy was complicated by polyhydramnios. Twins were delivered by Cesarean section because of premature labor and fetal heart rate decelerations. The infant's Apgar scores were 3 at one minute, 5 at five minutes, and he required resuscitation. Head circumference at birth was 27.5 cm. A right hemispheric germinal matrix hemorrhage with intraventricular extension was diagnosed by cranial ultrasound in the first week of life. Serial cranial sonograms revealed progressive hydrocephalus with dilatation of the lateral, third, and fourth ventricles. A ventriculo-peritoneal shunt (VPS) was introduced at seven weeks of age via a left frontal craniotomy.

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doi: 10.1684/epd.2007.0130

Neurological examination at the time of admission showed a well developed infant who was in no pain or distress. General physical and other systemic examination revealed no abnormal findings. Neurological exam revealed macrocephaly (head circumference 40 cm), a decompressed anterior fontanel, and brisk deep tendon reflexes with bilateral ankle clonus.

Video EEG monitoring performed three days after the onset of the spells revealed interictal epileptiform discharges in the left frontal, temporal and parietal regions, at a frequency of 1 every 2-3 minutes. A total of 10 stereotyped seizures were recorded. At the onset of the EEG seizures, the patient showed no change in behavior: 7-22 (mean = 15.8) seconds after the ictal EEG onset, left eye blinking was noted as an isolated symptom in all the seizures. The blinking did not involve the contralateral side of the face or ipsilateral lower part of the face. No other motor phenomenon was noted during the seizure. The infant cried during a seizure with an emotional tenor suggesting intact awareness.

The ictal scalp EEG onset during eye blinking seizures showed repetitive sharp waves in the left frontal and temporal region (maximum F7 electrode; *figure 1A* and *B*) that evolved in amplitude, frequency and distribution over the temporal region (T7 and F7), intermixed with the rhythmic artifact due to ipsilateral blinking (see artifact distribution in *figure 1A* and *B*). None of the seizures spread to the right hemisphere or became generalized. The EEG seizure duration was 45-178 (mean 110.6) seconds. The patient was treated with phenobarbital with an excellent response within 24 hours.

MRI *(figure 2)* performed three days after the onset of seizures showed communicating hydrocephalus with a VPS tract and surrounding encephalomalacia in the left frontal region. Bilaterally decreased white matter volume was also noted suggesting periventricular leukomalacia. Residual blood degradation products were seen in the bilateral anterior frontal horns, left occipital horn, and fourth ventricle.

Discussion

The patient presented is a 3-month-old boy with unilateral left eye blinking as a major feature of his seizures. Lateralization of the sign to the ipsilateral brain hemisphere is supported by the ictal onset in the left fronto-temporal region without secondary generalization. Active, emotional crying during the seizure suggested preservation of consciousness. In addition, the MRI demonstrated a VPS catheter with surrounding encephalomalacia in the left frontal lobe near the ictal onset zone. The ictal EEG pattern and location of VPS in the left frontal region suggest a causative relationship of seizures to the region of cortical injury induced by the shunt (Bourgeois *et al.* 1999).

Unilateral ictal blinking has been previously reported in the literature. Benbadis *et al.* (1996) reported 14 patients, age 18 months to 50 years, with a total of 38 seizures in which ictal unilateral eye blinking occurred as one component of ictal semiology. Ictal onset was lateralized to the ipsilateral hemisphere in 10 patients, nine being from the left hemisphere. The authors estimated the positive predictive value for ipsilateral ictal EEG localization to be 83% (Benbadis *et al.* 1996). The authors were unable to localize accurately the symptomatogenic zone for ictal eye blinking, however, they postulated that it probably originated in the frontal region.

Another study showed that ipsilateral blinking can be produced by stimulation of the basal region of the temporal lobe. Lesser *et al.* (1985) reported the occurrence of ipsilateral eye blinking after subdural cortical stimulation of the basal hemisphere structures. They hypothesized that stimulation at the edge of the plate generated ipsilateral pain or blinking due to activation of sensory sub-dural trigeminal fibers or the intracranial portions of the trigeminal and facial nerves. When ipsilateral blinking was noted during electric stimulation of electrodes in the center of the subdural plate, the response was attributed to activation of the trigeminal fibers traveling with the pial vessels. Therefore, it is possible that local vascular changes over the fronto-temporal region during spontaneous seizures could have the same effect (Lambert *et al.* 1997), leading to the ipsilateral blinking. This is further supported by the close anatomical association between the sensory innervation of the forehead muscles and middle cerebral artery and its branches (O'Connor, 1986).

Other mechanisms for the production of ipsilateral blinking during seizures have been suggested. The trigeminal facial reflex has been implicated in the production of the ipsilateral blinking response in humans when the trigeminal fibers are directly stimulated intracranially (Sindou *et al.* 1994). However, extracranial stimulation of the trigeminal fibers at the cornea or supraorbital region produce a bilateral blinking response (the blink reflex) (Aramideh and Ongerboerd 2002).

A few case reports have also suggested the involvement of the ipsilateral cerebellum in the mechanism of ictal blinking. One study reported an infant with left eye blinking events in the presence of a left cerebellar gangliglioma (Mesiwala *et al.* 2002). Another report described decreased ipsilateral blinking in patients with lesions in the cerebellar hemisphere (Gerwig *et al.* 2003). An fMRI study showed activation of the ipsilateral cerebellar hemisphere in response to unilateral blinking in healthy human volunteers (Dimitrova *et al.* 2002). These studies postulated that ipsilateral blinking abnormalities were due to disturbed sensory trigeminal input to the cerebellum.

In conclusion, our case and the review of the literature suggest that seizures with prominent and exclusive unilateral blinking probably originate from the ipsilateral frontal and temporal regions. The potential mechanism, as re-

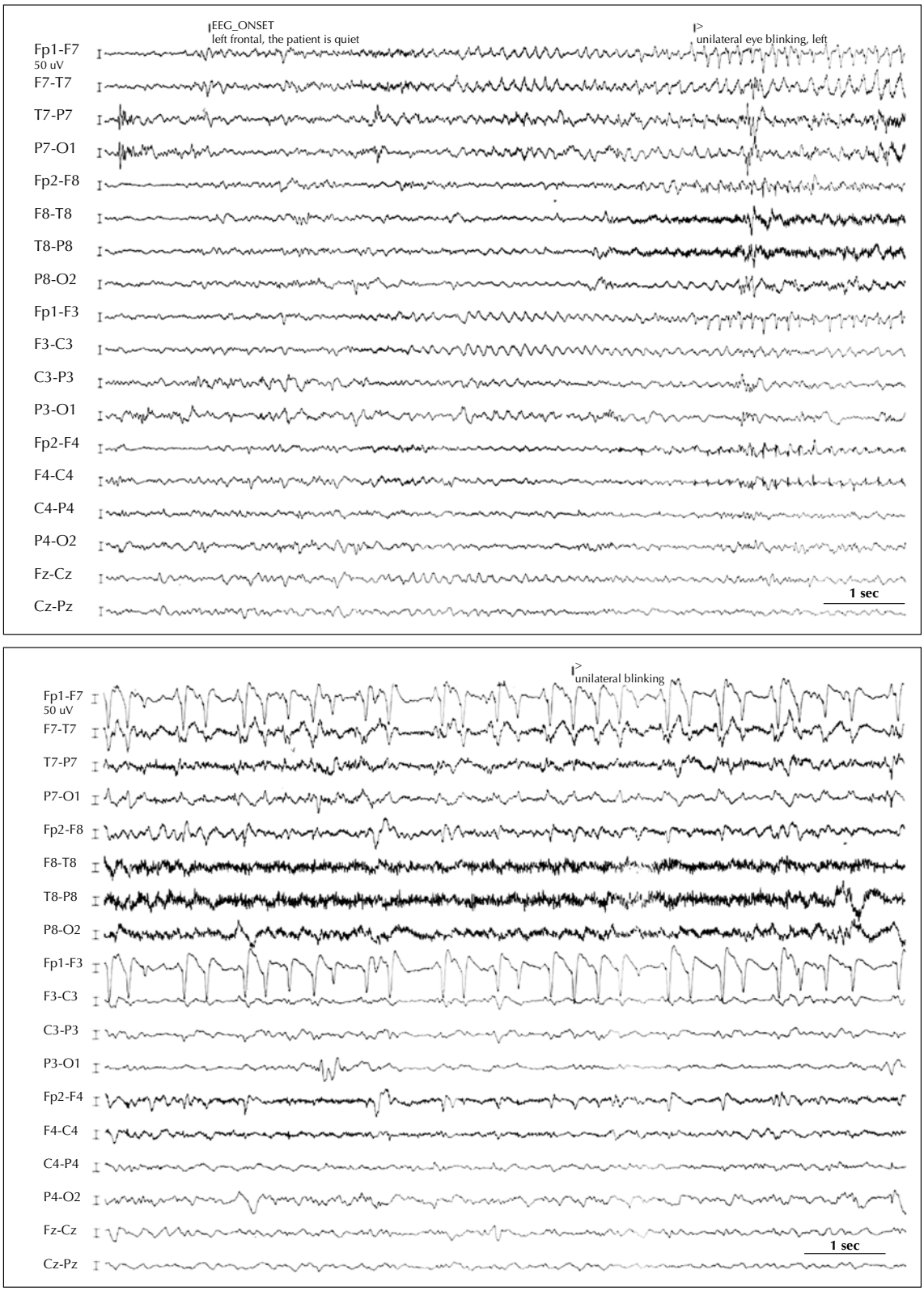

Figure 1. A) and **B**) showing the EEG of the seizure with unilateral blinking. The ictal EEG onset is maximally distributed at the left fronto-temporal region (F7>FT9) (**A**). The unilateral blinking artifact is later seen (Fp1-F7 and Fp1-F3 channels intermixed with ictal EEG [**B**]) pattern in the left temporal and frontal regions.

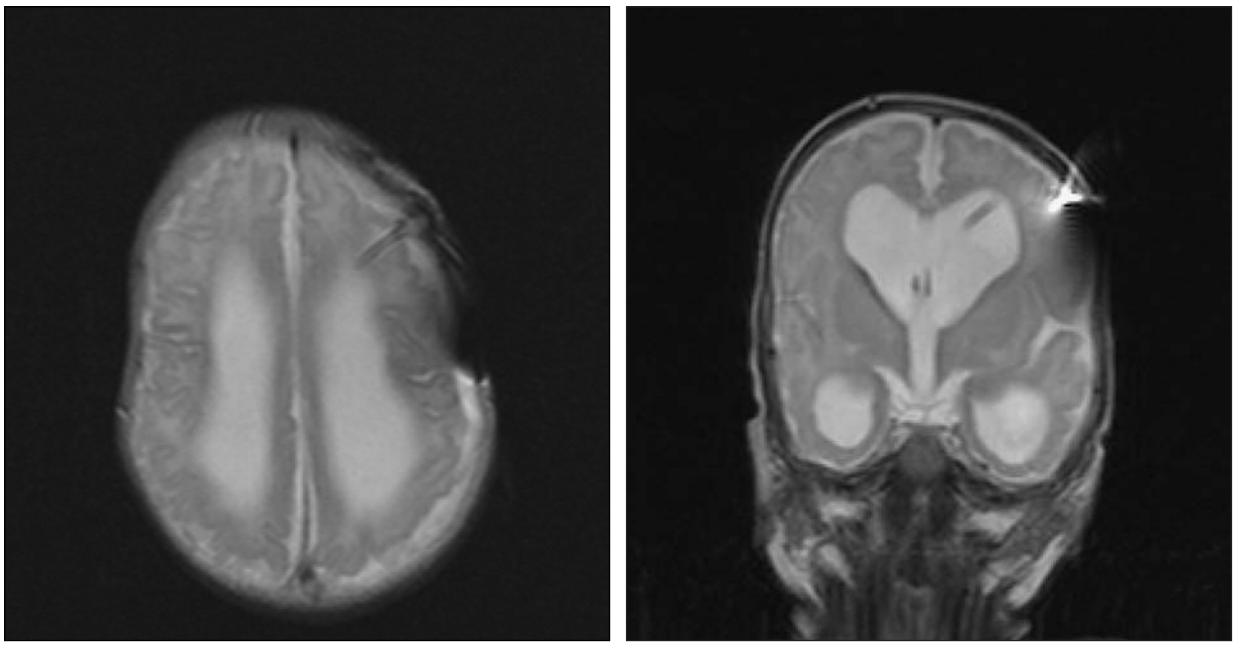

Figure 2. Brain MRI with axial (**A**) and coronal (**B**) T2-weighted images showing communicating hydrocephalus and left frontal craniotomy. A ventriculo-peritoneal shunt and encephalomalacia along the shunt tract are seen. Brain MRI also shows bilateral white matter loss and abnormal signal suggesting periventricular leukomalacia.

vealed by the electric stimulation of subdural electrodes during brain mapping, is activation of the trigeminal fibers innervating the subdural intracranial structures and pial vessels in the ipsilateral fronto-temporal regions. Lesions in the cerebellar hemisphere may also produce ipsilateral abnormalities in eye blinking. □

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