tients were excluded from the analysis because all recorded sei-
zes either secondarily generalized or were auras with no other
clinical manifestations. Thirty-five of 40 mesial temporal patients
had MRI evidence of mesial temporal atrophy or sclerosis, three
were identified through depth electrode recordings, and the re-
mainder of two cases had structural lesions in the uncus (one ne-
oplasm, one cavernoma). Seventeen of 19 neocortical temporal
patients had MRI or pathologic evidence of a structural lesion,
either neoplastic (four), hamartomatous (two), gliotic (nine), or
vascular (two), and two had no identifiable structural lesion but
scalp EEG findings incompatible with a mesial temporal localiza-
tion. Two of the patients could not be classified with certainty as
either mesial or neocortical.

Forty-nine patients with extratemporal epilepsy and 22 pa-
tients with nonepileptic events (pseudoseizures) were investigated
during the same study period.

Results. PIC was observed in eight of 62 patients (12.9%) with
temporal lobe epilepsy in 30 of 287 seizures (10.4%). Sixty
seizures were recorded in the eight patients with PIC: five with
right mesial, two with right neocortical, and one with left neocor-
temporal temporal epilepsy. PIC invariably occurred within 30 seconds of
seizure termination, often at or just before ictal offset (17 of 30
seizures). PIN was seen in 28 of 62 patients (45.2%), in 61 of 287
seizures (21.2%). All eight patients with PIC also demonstrated
PIN (p = 0.0034; Fisher’s exact test, two-tailed), one of whom also
had PIN. PIC was not observed in patients with nonepileptic
events.

Discussion. All patients with temporal lobe epilepsy and PIC
also had PIN in the same or other seizures, a significant correla-
tion indicating that the two phenomena may be markers for a
subset of patients whose seizures induce autonomic activation of
respiratory secretions to an extent greater than that seen in other
patients with temporal lobe epilepsy.

PIC is less common than PIN, occurring in 13% of patients
with temporal lobe epilepsy in this study. Previous reports have
described the incidence of PIC in patients with temporal lobe
epilepsy as 9%, 10%, and 40%. PIC tends to occur earlier after
ictal offset than PIN (100% within 30 seconds of offset with PIC,
compared to 61% within 30 seconds of offset with PIN). Also, as
described previously, PIC frequently occurs at or just before ictal
offset, which may indicate a need to regain partial awareness in
the postictal period to initiate PIN which is not required for PIC.

PIC is more common in temporal than extratemporal epilepsy
and is not seen with pseudoseizures. A trend toward right-sided
lateralization and mesial temporal localization was seen in this
and another temporal lobe epilepsy study, however, no significant differences in lateral-
ization or intratemporal localization of seizure onsets with PIC
have been reported.

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Prolonged “postictal” aphasia: Demonstration of persistent ictal activity with intracranial
electrodes

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We present a case of a 40-year-old woman with recurrent episodes of
prolonged postictal aphasia lasting for days to weeks. Although there was no scalp EEG correlate, intracranial recordings demonstrated ongoing ictal activity, predominantly in the posterior left
temporal lobe, during several days of her typical "postictal" aphasia.

Case history. A 40 year-old woman with no risk factors for
epilepsy had intractable complex partial and secondarily general-
ized seizures since age 15 years. These were often followed by
postictal aphasia that lasted for days to weeks. Despite MRI evidence of mesial temporal sclerosis, an earlier left anterior temporal lobectomy helped only transiently. During an evaluation for additional epilepsy surgery, left hemisphere subdural grid and strip electrodes were inserted (figure, A). She had a typical complex partial seizure with secondary generalization. Electrical onset was in the anterior superior temporal gyrus (electrodes LT2 to 4). IV lorazepam was administered within 2 minutes and quickly terminated clinical seizure activity. However, intracranial recordings demonstrated ongoing ictal activity throughout the left temporal lobe. Additional IV lorazepam and fosphenytoin were given, and no further clinical seizures occurred. She became alert but had severe deficits in naming, repetition, comprehension, and reading, similar to prior episodes, as well as right agraphesthesia and hemianopia. Subdural electrodes demonstrated ongoing ictal activity (rhythmic theta and alpha) in the basal and posterolateral temporal neocortex (highlighted areas in the figure, A), occasionally spreading more superiorly.

Inferior and anterior temporal lobe scalp electrodes (F9, T9, P9, AT1, and Fp1) were added. Over the next several days, intracranial ictal activity gradually fragmented, but bursts of spikes and polyspikes continued to occur every 1 to 3 seconds in the same area. Her aphasia remained severe. Simultaneous recordings from proximate scalp electrodes did not reflect ictal activity throughout most of postictal days 2 through 6. Two samples taken 48 hours after her convulsion are shown in the figure, B and C. Figure B shows typical activity during this period, with no scalp EEG correlate. Figure C is an example from the rare times when a subtle scalp EEG correlate could be seen, consisting of low-voltage pseudoperiodic discharges; this was most evident when subdural recordings showed spread to suprasylvian electrodes (G38, G46). No further clinical seizures occurred. The patient was treated with high doses of four anticonvulsant medications. Her hemianopia and agraphesthesia on the right resolved in less than 1 week. The aphasia improved slowly 7 to 10 days after her convulsion as the intracranial EEG discharges gradually subsided. Comprehension and naming deficits were the last to improve. She then underwent resection of the anterior superior temporal gyrus, where all recorded seizures had originated. Mild to moderate anoma persisted postoperatively.

Discussion. We describe a woman with recurrent episodes of secondarily generalized seizures followed by days to weeks of aphasia that eventually improved. Although scalp EEG did not reveal ictal activity, intracranial electrodes demonstrated that this "postictal" aphasia was correlated with an ongoing ictal discharge.

This is certainly not the first report of prolonged aphasia from epileptiform activity,1,2 nor of a prolonged postictal deficit. However, we are unaware of another report of an isolated clinical seizure followed by a prolonged postictal deficit (aphasia in this patient) without a scalp EEG correlate that proved to be due to ongoing ictal activity utilizing intracranial recordings. It is not surprising that this can occur, as there are documented cases of cognitive impairment during highly focal intracranial electrical activ-
It is important to consider the possibility that prolonged "postictal" deficits may be due to ongoing ictal activity, as additional anticonvulsant medication may be indicated.

There were rare periods in this case when there were subtle pseudoperiodic discharges on the scalp EEG during ongoing ictal activity intracranially, and persistent aphasia. This supports the view that in some instances, periodic lateralized epileptiform discharges (PLEDs) on the scalp EEG are actually ictal. It is possible that functional imaging with PET or SPECT can help make this differentiation, as focal increased blood flow or metabolism during a prolonged postictal deficit or during PLED may suggest an ictal state. Thia was not performed in our patient.

We conclude that prolonged "postictal" deficits may sometimes be due to ongoing ictal activity that is not evident on scalp EEG.

References

Cerebral artery embolism following an esophagogastroscopy: A case report

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Cerebrovascular events during endoscopy are uncommon. Air embolism should be considered in the differential diagnosis of any stroke, particularly if it develops during or after an endoscopic procedure. We report this unusual complication in a woman who had a cerebral artery embolism during an esophagogastroscopy procedure.

Case report. An 80-year-old woman presented with a history of progressive dysphagia, initially for solid foods and later for liquids, and severe weight loss during the previous 6 months. She was taking enalapril for hypertension. Initial investigations revealed the presence of a malignant stricture at the esophagogastroduodenal junction, along with a small esophagotracheal fistula. Multiple small biopsies were performed, confirming the presence of esophageal carcinoma. Her level of consciousness deteriorated immediately following the procedure. She became unresponsive, but remained hemodynamically stable. Examination revealed a left hemiparesis involving the face, arm, and leg, with flexion of the right upper limb in response to pain. An emergent unenhanced CT scan of the head revealed parenchymal air in the right hemisphere (figure). Right-sided sulci were effaced, which was suggestive of an acute right middle cerebral artery infarct. Cerebral air embolism was diagnosed. She was treated conservatively with 100% oxygen therapy. A transthoracic echocardiogram with bubble contrast showed no evidence of a right-to-left intracardiac shunt. She was discharged from the hospital in a vegetative state 2 weeks after admission.

Discussion. Cerebral air embolism may occur with barotrauma during surgical procedures, especially when performed in a sitting position. This also has been reported as a complication of cardiac catheterization, as well as in other diagnostic and therapeutic procedures. A few cases have been reported in the absence of intracardiac defects. Penetrating duodenal ulcers may form fistulas with colon, pancreatic or bile duct, or aorta. Other cases have been reported with duodenocaval fistulas after trauma and local radiation. Because of unique hepatic venous drainage, systemic air embolism is uncommon with gastrointestinal endoscopy procedures. Venous air embolism occurs only when the liver is bypassed, e.g., with portosystemic shunts in portal hypertension. Arterial air embolism is even more rare because of capillary filtration in the lungs. In certain circumstances, this filter is bypassed through a right-to-left intracardiac shunt. However, in our patient, such a shunt was not found. A small tracheoesophageal fistula around the stricture was seen. There have been reports of a fatal cerebral arterial gas embolism caused by a large venous gas embolism, though no intracardiac defects or shunt mechanisms could be demonstrated. An alternative mechanism for paradoxic air embolism—intrapulmonary shunts as well as transcapillary route with large air emboli—has been proposed. This probably best explains the mechanism in our patient.

The neurologic manifestations are myriad and include altered consciousness, seizures, and focal deficit. CT scanning is valuable in detecting this if performed early, as IV air is rapidly absorbed and delays would result in a failure of diagnosis. CNS dysfunction results from mechanical obstruction of arterioles, leading to cerebral ischemia and the thrombotic-inflammatory response of air-injured epithelium. This results in a transient decline of cerebral blood flow and neural function. Treatment should be started as soon as there is a strong clinical suspicion for the diagnosis, including the removal of the air source, 100% oxygen by face mask, and hyperbaric oxygen. The aim of this treatment is to rapidly reduce the volume of air embolus. Recent experimental data suggest that agents with anti-thrombotic and anti-inflammatory properties may be of potential benefit when given prophylactically in subjects at risk for cerebral air embolism. From the Departments of Medicine—Neurology (Drs. Akhtar and Mozaffar), Gastroenterology (Dr. Jafri), and Pathology (Dr. Mozaffar), the Aga Khan University Medical College, Karachi, Pakistan.
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