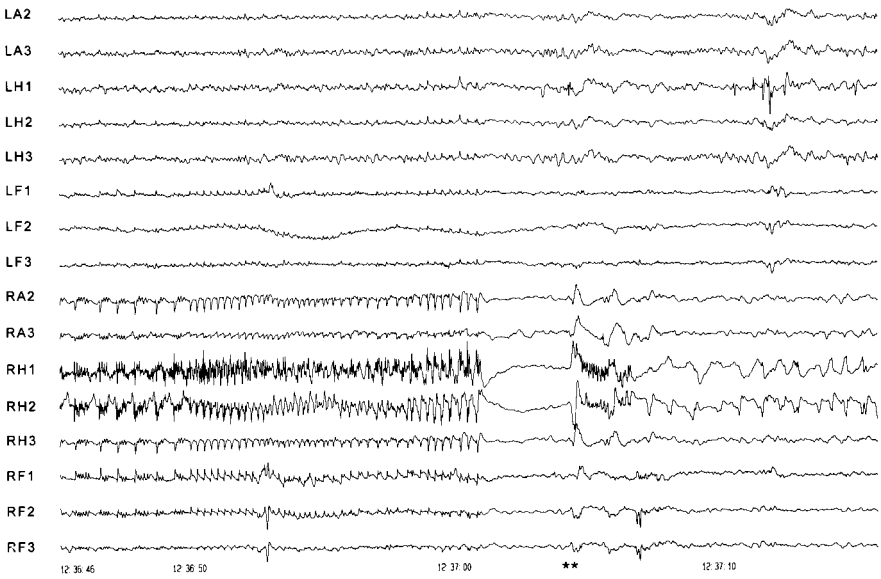

Figure. Intracranial depth electrode recording in patient with right mesial temporal epilepsy. Focal electrographic onset occurred at RH1,2 (right anterior hippocampus) 52 seconds earlier (not shown). The ictal activity remained confined to RH1,2 before clinical onset 25 seconds later, coinciding with spread to ipsilateral amygdala (RA2,3) and orbital frontal contacts (RF1,2). Coughing occurred at ictal offset (**); noserubbing occurred 54 seconds later. Average referential montage.

tients were excluded from the analysis because all recorded seizures 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 remaining two cases had structural lesions in the uncus (one neoplasm, 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 localization. Two of the patients could not be classified with certainty as either mesial or neocortical.

Forty-nine patients with extratemporal epilepsy and 22 patients 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 neocortical 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), either in the same (9 of 60) or different (13 of 60) seizures.

One patient with PIC was investigated with bilateral frontal and temporal depth electrodes. All 18 recorded clinical seizures, and all seizures with PIC (and PIN), showed onset localized to the right anterior hippocampus with subsequent spread to the amygdala and then right orbital frontal region (figure). Seizures restricted to the hippocampus were always subclinical and did not evoke PIC or PIN.

PIC was less common in patients with extratemporal epilepsy, seen in 2/49 patients ($p < 0.05$; χ^2, one-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 correlation 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%,[7] 10%,[1] and 40%.[2] 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[6]). Also, as described previously,[1] 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[3] 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 study,[7] however, no significant differences in lateralization or intratemporal localization of seizure onsets with PIC have been reported.[1,2,7]

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

Lawrence J. Hirsch, MD; Ronald G. Emerson, MD; and Timothy A. Pedley, MD

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 generalized 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 anomia 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-

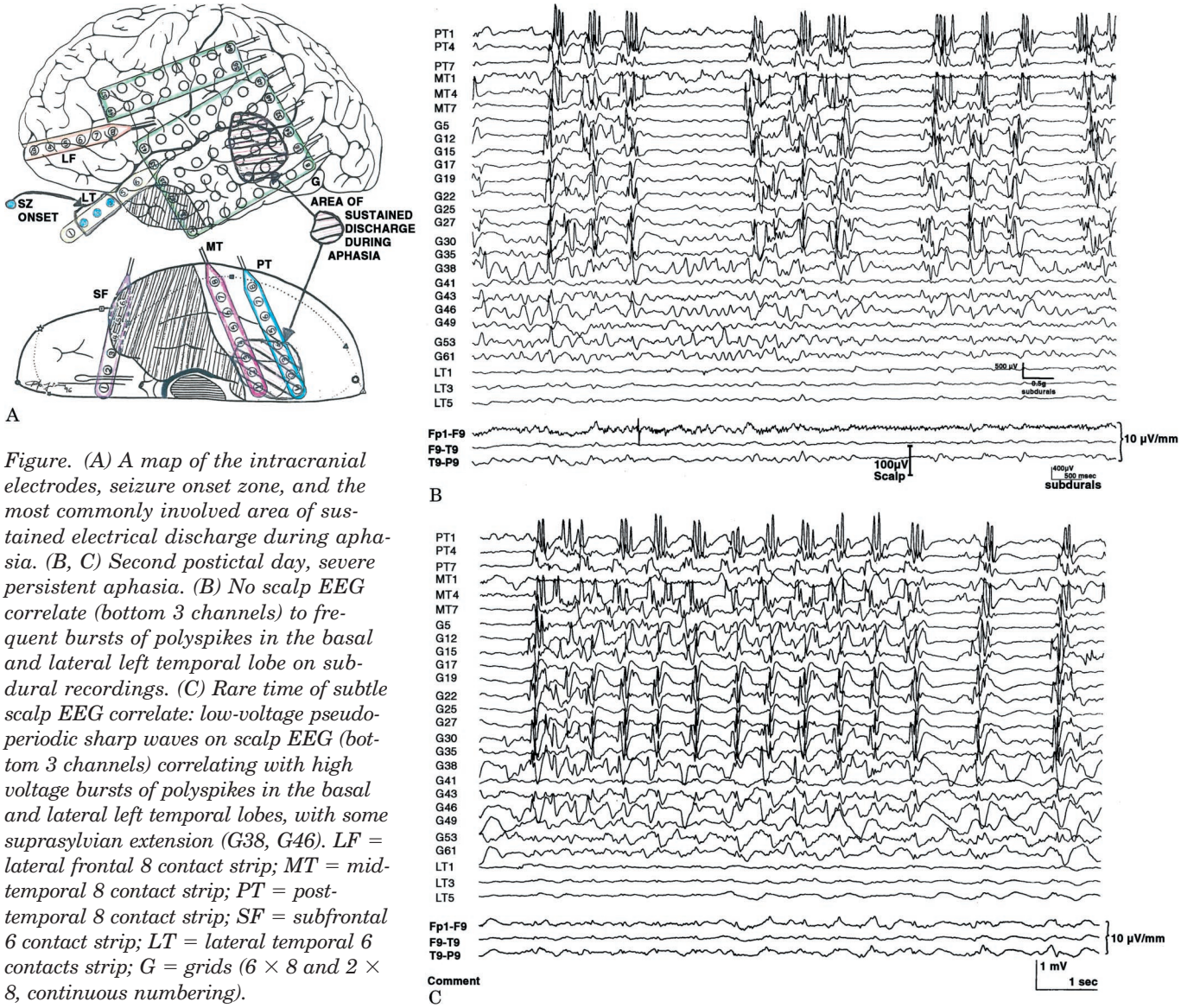

Figure. (A) A map of the intracranial electrodes, seizure onset zone, and the most commonly involved area of sustained electrical discharge during aphasia. (B, C) Second postictal day, severe persistent aphasia. (B) No scalp EEG correlate (bottom 3 channels) to frequent bursts of polyspikes in the basal and lateral left temporal lobe on subdural recordings. (C) Rare time of subtle scalp EEG correlate: low-voltage pseudoperiodic sharp waves on scalp EEG (bottom 3 channels) correlating with high voltage bursts of polyspikes in the basal and lateral left temporal lobes, with some suprasylvian extension (G38, G46). LF = lateral frontal 8 contact strip; MT = midtemporal 8 contact strip; PT = posttemporal 8 contact strip; SF = subfrontal 6 contact strip; LT = lateral temporal 6 contacts strip; G = grids (6 × 8 and 2 × 8, continuous numbering).

ity, including discharges that could not be detected at the scalp.[3,4] We believe it is important to consider the possibility that prolonged "postictal" deficits may be due to ongoing ictal discharges, 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 (PLED) on the scalp EEG are actually ictal.[5] 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.[6] This 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.

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Cerebral artery air 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 air 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 in the esophagus. She underwent an elective fiberoptic endoscopy under conscious sedation, which confirmed a malignant stricture at the esophagogastric 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.

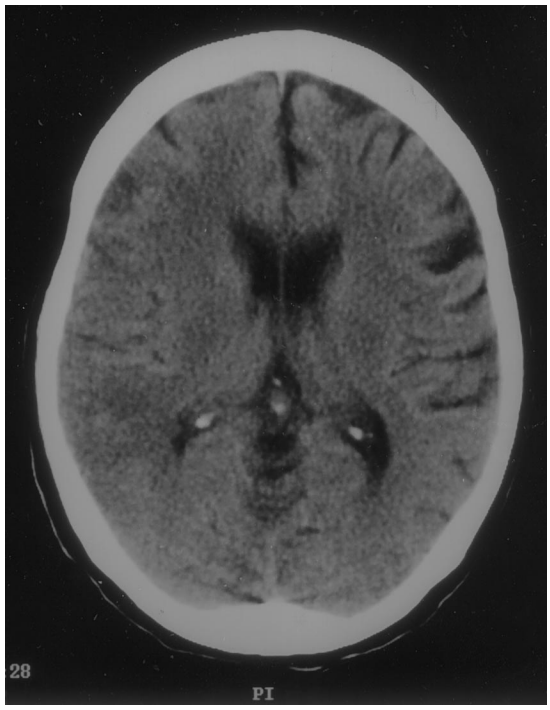

Figure. Unenhanced CT scan of head shows right hemispheric swelling and effacement of gyri on that side. Free parenchymal air is seen in the subfrontal region.

Discussion. Cerebral air embolism may occur with barotrauma[1] and during neurosurgical procedures,[2] especially when performed in a sitting position. This also has been reported as a complication of cardiac catheterization, as well as in other diagnostic andtherapeutic procedures.[3] 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.[4]

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.[5] 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.[3] An alternative mechanism for paradoxic air embolism—intrapulmonary shunts as well as transcapillary route with large air emboli—has been proposed.[6] 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 antithrombotic and anti-inflammatory properties may be of potential benefit when given prophylactically in subjects at risk for cerebral air embolism.[5,6]

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