surface. Coverage of the south hemisphere was left to ad hoc electrode placements. In view of the existing body of data indicating that EEG space domain includes both north and south hemispheres, serial bipolar montages for spherical coverage have been developed and implemented. They were designed to supplement transverse bipolar montages in common use. The full electrode array requires addition of 3 pairs of infralateral orbital (IO1 and IO2), mastoid (MS1 and MS2) and cerebellar (CB1 and CB2) electrodes. Serial bipolar chains consist of Fz-F4-F8-IO2-IO1-F7-F3-Fz, Cz-C4-T4-MS2-MS1-T3-C3-Cz and Pz-P4-T6-CB2-CB1-T5-P3-Pz (anterior, middle and posterior basal transverse, respectively). Elimination of CB1 and CB2 will result in a convenient 18-channel polygraphic montage. Anterior and middle basal electrodes are easy to apply and stable in standard EEG recording settings. For reference derivations, MS1 and MS2 replace A1 and A2. While they were aimed at facilitating evaluation of temporal lobe disturbances, basal transverse montages may help display subtle features of potential significance associated with frontal lobe pathology or generalized paroxysms. Cumulating experience will be illustrated in clinical examples.

94. Hypothermia and triphasic waves. – J.T. Petrella [b] and R.P. Brenner [a,b] (Departments of [a] Psychiatry and [b] Neurology, University of Pittsburgh, Pittsburgh, PA)

Triphasic waves (TWs) due to hypothermia are rare as only 3 cases have been reported. We describe 1 case with profound hypothermia in whom TWs persisted approximately 9 h following normal body temperature restoration.

Case report. A 70-year-old woman with mild hypertension, diabetes, and old right middle cerebral artery infarct, was admitted to UPMC after being found alone in her car. Her rectal temperature was 28°C, and her pulse rate 56/min. There was no evidence of fecal or urinary incontinence, or tongue biting. She was lethargic and did not respond to verbal stimuli. Laboratory tests, including liver and renal function tests, were unremarkable. A CT scan revealed an old right hemispheric infarct. The patient was rewarmed to normothermia within 3 h.

EEGs were performed 12 h and 24 h after admission. The first demonstrated generalized background slowing (4–6 Hz) and periodic TWs, occurring every 0.5–1 sec. The patient's neurological examination was similar to that on admission. The second EEG revealed mild slowing (7 Hz) without TWs. The patient's mental status had improved considerably, although she was slow to respond to questioning. She recovered without sequelae.

95. Regional cerebral flow measured by SPECT in a child with a seizure, migraine and occipital spike-wave complexes. – P.W. Kaplan and J. Petronis [a] (Departments of Neurology and [a] Medicine, Francis Scott Key Medical Center, Johns Hopkins School of Medicine, Baltimore, MD)

Hughlings Jackson and Gowers referred to the interplay of migraine and seizures as "the borderland of epilepsy." Occipital seizures in children are characterized by visual ictal symptoms and interictal occipital spike-waves; whereas in basilar migraine, EEG shows occipital slowing without occipital spikes.

We present a 12-year-old boy with formed visual hallucinations, déjà vu, nausea, photophobia and pulsating headache, followed by a tonic-clonic seizure. The mother had a history of migraines; there was no family history of seizures. CSF, head CT and MRI scans were normal. EEG showed continuous polymorphic occipital delta activity with right occipital spike-waves at 1–2 Hz. Technetium-99m HMPAO scans at 4 days and 9 weeks showed marked bilateral occipital perfusion defects; a transcranial Doppler study was normal.

Occipital seizures may cause migraine; conversely, migraine-induced basilar vasospasm may lead to epileptogenicity. Although regional cerebral blood flow may decrease in migraine but increase with focal epileptiform activity, this patient showed decreased occipital perfusion suggesting basilar ischemia as a cause of epileptogenicity. EEG, SPECT and Doppler sonography may help clarify the nature of migraine-epilepsy interrelationships.

96. Reversible cortical blindness, delirium, seizures and occipital spikes with hypercalcemia. – P.W. Kaplan [a,b] and G. DalPan [b] ([a] Department of Neurology, Francis Scott Key Medical Center, Baltimore, MD, and [b] Department of Neurology, Johns Hopkins University School of Medicine, Baltimore, MD)

Hypercalcemia causes delirium, hallucinations, lethargy, and occipital spikes, but rarely cortical blindness. We report the first case documenting reversible, hypercalcemia-induced cortical blindness with cerebral perfusion defects and occipital spike-waves.

A 66-year-old woman developed hypercalcemia from multiple myeloma and ingestion of calcium-containing antacids. Following the onset of a delirium with visual hallucinations (calcium 13.6 mg/dl) there was a brief tonic-clonic seizure and calcium was lowered to 8.3 mg/dl with pamodronate. Examination revealed conjugate gaze, full extraocular movements, reactive pupils, normal fundi, but no sight for objects or movement. Computerized axial tomography was normal. Single photon emission computerized tomography showed multiple perfusion defects in the right occipital, left parietal and frontal regions. No responses were obtained on pattern reversal visual evoked potentials. EEG showed diffuse, bilateral slowing with high-voltage spike-wave discharges every 1–3 sec over the occipital cortex, predominating on the right. Serum calcium was further lowered to 6.4 mg/dl, delirium resolved, and vision returned.

Hypercalcemia may cause seizures and reversible cortical blindness with occipital spike-waves by means of calcium mediated, serotonin-induced vasospasm (Allen et al., J. Neurosurg., 1976, 44: 585–593).

97. Persistent bitemporal beta activity in a 2 year old. – C.E. Yen, F.F. Gilliam and R.J. Baumann (Department of Neurology, College of Medicine, University of Kentucky, Lexington, KY)

A 2-year-old girl sustained bilateral watershed parieto-occipital infarctions at birth. She has severe developmental delay, and is markedly microcephalic despite orbital frontal craniotomy for presumed craniosynostosis. She has spells of asymmetrical upper extremity posturing accompanied by left head deviation. On examination, she is non-verbal, follows no commands, has spastic quadriplegia. MRI shows bilateral parieto-occipital encephalomalacia.

EEG (on no medication) demonstrates a symmetrical 25–50 μV mixture of alpha-theta-delta parasagittal activity. Persistent rhythmic 50–100 μV beta activity of 20–25 Hz and occasional high amplitude independent spike-waves are seen in the temporal regions bilaterally. Occasionally, 8–10 Hz alpha activity is seen in the identical distribution. Her typical spells are unaccompanied by any electrographic changes.

Focal beta activity is seen with structural lesions and post craniotomy. In children, it is generally an ictal or interictal expression of a seizure disorder. We are not aware of reports of persistent bitemporal beta activity. We doubt this is just a breach rhythm. We cannot exclude the possibility her spells are frontal seizures, but bitemporal beta is an unlikely interictal correlate. This child appears to demonstrate persistent bilateral but focal beta activity in the absence of either a correlative focal structural lesion or definite seizure disorder.

98. Clinical correlations of theta coma. – C.E. Yen (Department of Neurology, University of Kentucky, Lexington, KY)

Since the initial description of theta coma by Suter (Neurology, 1973, 23: 445) several authors have described theta patterns in comatose patients following anoxia and severe head trauma, associated with a poor prognosis.