

act of dressing induced the fits; no seizures were ever provoked by tactile stimulation or by any other single maneuver used during dressing. The emotional gratification of being completely dressed could be the cause of the seizures.

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### Jacksonian Somatosensory Seizures as the Sole Manifestation of Chronic Subdural Hematoma

*To the Editor.*—We present the first reported case, to our knowledge, in which jacksonian somatosensory seizures were the sole manifestation of a contralateral chronic subdural hematoma. The somatosensory seizures resolved following evacuation of the hematoma.

*Report of a Case.*—A previously well, 43-year-old, right-handed male welder began to note transient paresthesias of the left hand, arm, and face. With each episode, which lasted from 30 to 60 s, the symptoms would spread from the distal left phalanges to include serially the entire hand, arm, shoulder, face, and torso on that side. This temporal sequence never varied. Initially occurring once or twice daily, the episodes gradually increased in frequency to ten or more per day. The

Coronal section from computed tomographic scan, showing right parietal subdural hematoma and its associated contrast-enhancing membrane (arrow).



paresthesias were accompanied by a sensation of heaviness in the left arm, but frank weakness was denied, as was headache, visual symptoms, ataxia, or focal motor activity.

The patient was unaware of antecedent trauma. Family history was unremarkable. Examination two weeks after the onset of symptoms disclosed no evidence of cranial injury. The patient's sensorium was clear. Functions of cranial nerves were intact. Strength, coordination, and gait were normal. All sensory modalities were preserved, including graphesthesia, stereognosis, point localization, and two-point discrimination. Muscle stretch reflexes were normal, and there were no pathologic reflexes. An interictal EEG demonstrated prominent delta activity over the right parietal region. A coronal computed tomographic scan (Figure) disclosed a right parietal chronic subdural hematoma, which was drained. Postoperatively, the patient experienced a marked reduction in the frequency of seizures. He has remained free of seizures during 12 months of treatment with phenytoin sodium.

*Comment.*—Although the cardinal symptoms of chronic subdural hematoma are increased intracranial pressure, alteration in mental status, and hemiparesis,<sup>1,2</sup> seizures of any type are a relatively uncommon initial symptom. To our knowledge, there has been no prior report of chronic subdural hematoma manifested solely by somatosensory seizures. The only prior implication of subdural hematoma in somatosensory seizures involved a patient in whom such seizures were only one of several manifestations of the underlying hematoma.<sup>3</sup> Notwithstanding the lack of associated neurologic signs, the presence of somatosensory seizures proved to be of localizing value in our case.

Recent reports have shown that chronic subdural hematoma may give rise to symptoms that closely simulate transient ischemic attacks (TIAs).<sup>4,7</sup> The symptoms of our patient might have been confused with recurrent TIAs. The TIA-like symptoms reportedly associated with chronic subdural hematoma, however, last variably from three minutes to an hour or more, in contrast to the relative brevity of the episodes experienced by our patient.

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### Endocrine Dysfunction in Temporal Lobe Epilepsy

*To the Editor.*—The report by Herzog and colleagues, "Neuroendocrine Dysfunction in Temporal Lobe Epilepsy" (ARCHIVES 1982;39:133-135), offers a potential neuroendocrinologic basis for impaired fertility in epileptic women<sup>1</sup> and the frequent occurrence of hyposexuality—primarily erectile dysfunction—in men with temporal lobe epilepsy (TLE).<sup>2</sup> We would be interested to know whether the patient who had an elevated serum prolactin level had recently experienced a seizure. Although we agree that interictal prolactin studies are normal in this clinical population, we found postictal hyperprolactinemia after complex partial seizures in all cases studied<sup>3</sup>; thus, the temporal relationship between serum prolactin determinations and the occurrence of seizures is critical. Although the clinical consequences of episodic hyperprolactinemia are uncertain, impotence and decreased libido are the most common accompaniments of sustained hyperprolactinemia in men.<sup>4</sup> Further studies of prolactin secretion in TLE seem warranted.

Using an intravenous (IV) bolus of luteinizing hormone-releasing hormone (LH-RH) in male temporal lobe epileptics with sexual dysfunction, we found a spectrum of responses, both normal and abnormal. With a different technique of LH-RH infusion, Herzog et al found similar results in patients with TLE who were sexually asymptomatic. Interestingly, the mean age of Herzog's patients was 28.3 years, compared with 41.4 years for our normosexual patients with TLE and 50.0 years for those with hyposexuality. Thus, neuroendocrinologic dysfunction seems to antedate overt sexual dysfunction in TLE. This conclusion may have far-reaching implications concerning aggressive medical therapy of TLE in early life and alternatives to medical therapy if those at greatest risk can be identified. In our study, there was a positive