rapid cycling with a periodicity of cycles of 3 weeks. She did not improve on combined antidepressant/neuroleptic treatment, amitryptiline, 25 mg tid and 75 mg at night, and zuclopenthixol, 4 mg four times a day. This treatment resistance led to the addition of lithium.

During the third week of lithium therapy, at the low dose of 600 mg sdd and stable serum lithium level of 0·6 mmol/l, sleeplessness, concentration difficulties, dysarthric speech and gait ataxia developed. Cessation of lithium led to a rapid reversal of neurotoxic symptoms within 2 weeks. Neurological examination, EEG, ECHOgraphic investigation and CSF examination were unremarkable afterwards. Cerebral CT, indicated by this neurotoxic episode and persistent depressive symptomatology with apathy, however, revealed a frontobasal bilobar tumour, homogeneously enhancing, and situated in the midline, which proved to be a frontal meningioma.

Lithium is increasingly used in psychiatry, often in elderly patients, and based on recent findings that it is able to augment the effect of anti-depressants.

We have shown that the occurrence of reversible neurotoxic phenomena at normal serum lithium levels, which we have seen in several patients in the past 2 years, may point to a possibly treatable intracranial pathology. In one of our patients lithium neurotoxicity pointed to cerebral infarction, in the other to frontal meningioma.

These findings are in accordance with those of Bekker (1987) concerning lithium-augmentation in geriatric patients; he found that, on introducing lithium, 42% of his cerebrovascular-compromised patients, who had transient ischaemic attacks, cerebrovascular accidents or multiple infarcts in their history, developed signs of neurotoxicity, compared with 14% of patients without this pathology.[3]

Why this symptomatology occurs in these patients is unclear. One theory is that there is an abnormal affinity for lithium in pathologic brain tissue,[4] which is based on showing that rat neuroblastoma and glioma cells are able to contain high levels of lithium.[5] This theory is corroborated by one report of a lithium concentration nearly thrice the serum level in human glioblastoma tissue.[6]

We hypothesise, that these findings speak in favour of the existence of a diminished capacity for lithium removal in pathologically transformed brain tissue. Such a diminished capacity might lead to islands of high lithium concentration, influencing the surrounding (normal) brain and giving rise to the specific phenomenon of lithium neurotoxicity at normal serum lithium levels.

Though risk factors concerning neurotoxicity of lithium may be known,[2] neurologists and psychiatrists should be aware of a "lithium neurotoxicity sign", which may be observed at normal serum levels of lithium and which may point to potentially treatable intracranial pathology.

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Thirst and compulsive water drinking in medial basal limbic epilepsy: an electroclinical and neuropathological correlation

Sir: A sensation of thirst and compulsive water drinking are unusual ictal phenomena that have been reported to occur in association with complex partial epilepsy.[1] Previous studies have indicated an association of this ictal behaviour and epileptiform activity in the temporal lobe.[1] The following report describes a patient with stereoptypic, reproducible clinical events consisting of an intense desire for water. Ictal scalp recorded EEG studies demonstrated right anterior temporal electrographic seizure activity and histopathology of the right hippocampus revealed hippocampal sclerosis. This case provides confirmatory evidence that an alteration in water intake may occur during partial or localisation-related seizures associated with pathology in the mesiobasal region of the temporal lobe.

A 39-year-old right-handed woman was evaluated for consideration of surgical treatment of her epilepsy. The patient developed generalised tonic/clonic seizure activity at 11 years of age. Growth and development were normal and the aetiology of her seizure disorder could not be determined. When aged 21 years she began to experience complex partial seizures that remained refractory to antiepileptic drug therapy. All of the patient's seizures were stereotypic and began with the sensation of "lightheadedness" and palpitations, followed by an urge to drink water. She would run to a sink to consume fluids, often by placing her head directly under the faucet. She could consume 6–8 glasses of water during a seizure. Every seizure episode was associated with thirst, even if the patient had recently completed eating and drinking. Subsequent to a seizure the patient recalled her desire to drink. The patient always kept a large pitcher of water next to her bed. She did not drink in response to a dry mouth or disagreeable sensation in the mouth or throat. If water was not available she would consume cola or juice. On one occasion she could not locate a source of water and ran in "terror", injuring herself. The ingestion of fluids did not appear to terminate her seizure activity. The patient was forced to surrender her position as a store clerk because of her bizarre drinking behaviour during her seizures. Previous diagnostic considerations had included psychogenic polydipsia, panic attacks, diabetes mellitus and diabetes insipidus.

The patient was admitted for prolonged extracranial EEG studies with audiovisual monitoring. Neurological examination was normal. Multiple seizures were recorded. The episodes began with a motionless stare, followed by lip smacking and head turning to the left. The patient then placed her hands on her chest. At this time she would intermittently follow simple commands but not speak. She then pointed to her mouth and motioned

to the sink. She consumed 1–2 pitchers of water. Interictal EEG studies revealed intermittent epileptiform activity in the right anterior region. Ictal EEG studies confirmed the right anterior temporal localisation. Postictally she appeared confused and did not recall test items presented to her during the ictus. She did, however, admit to her thirst and consumption of water.

Electrolytes, serum and urine osmolality and fasting blood glucose were normal. A computed tomography (CT) scan was normal. A magnetic resonance (MR) scan showed an enlargement of the right temporal horn of the lateral ventricle and suggested atrophy of the right parahippocampal gyrus (fig). The patient underwent an en bloc right anterior temporal lobectomy. Hippocampal sclerosis was demonstrated. The neuronal loss and gliosis were widespread and prominent in the hippocampus, subiculum, and dentate gyrus. The amygdala and the lateral temporal neocortex were unremarkable. She has been asymptomatic and seizure free since surgery.

The urge to pour and drink water has been documented rarely to occur as an ictal behaviour in epilepsy. Remillard *et al* described 20 patients with complex partial epilepsy and ictal water drinking.[1] In these patients scalp recorded EEG demonstrated electrographic seizure activity in the temporal lobe, and depth electrode studies revealed the onset of seizure activity in the amygdala, hippocampus and parahippocampal gyrus in two patients.[1] This report did not include CT and MR studies or describe the pathological anatomy of this ictal syndrome.[1] The neuroimaging studies may have demonstrated an extratemporal structural abnormality associated with an aberration of water intake, such as a hypothalamic lesion.[1]

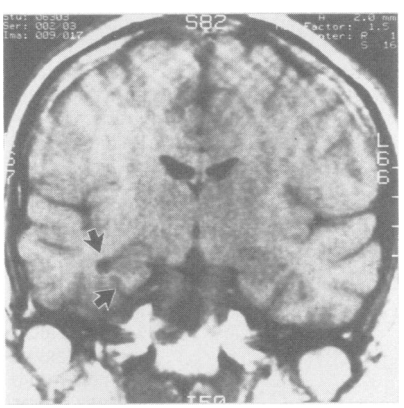

Fig *A coronal T1 weighted image magnetic resonance scan shows atrophy of the right parahippocampal gyrus (arrow) and enlargement of the right temporal horn of the lateral ventricle (arrow). (Note the right temporal lobe is on the left side of the photograph.)*

The anterior and ventromedial hypothalamus has an important role in thirst and water regulation.[2-6] In addition, efferent pathways from the hippocampus and amygdala that project to the hypothalamus have also been implicated in water balance and drinking behaviour.[3 7] Stimulation and lesion studies in the dog have implicated a role for the anteroventral amygdala in thirst and water drinking behaviour.[3] Amygdala efferent fibres in the stria terminalis and hippocampal efferents in the fornix converge on the hypothalamus.[7]

Thus, the hypothalamus is linked with limbic structures by a variety of multisynaptic pathways.[7] Epileptiform activity generated in the temporal lobe may propogate into the hypothalamus and other structures implicated in thirst and water regulation, producing ictal manifestations of abnormal water seeking behaviour.

This study demonstrates that complex partial seizures, accompanied by mesiobasal temporal lobe pathology, may indeed be associated with an ictal disturbance of water drinking. The clinical and neuropathological observations suggest that epileptiform activity may propogate into regions that are synaptically remote from hippocampal pathology and produce ictal manifestations of abnormal water-seeking behaviour. This case confirms the previous report regarding the temporal lobe localisation of ictal thirst, and in addition identified a pathological lesion associated with this unusual ictal behaviour.[1]

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Single fibre and quantitative EMG study in acute stage of human trichinosis

Sir: Human trichinosis is a parasitic infection now rarely reported, although the disease has not been eradicated. Electrophysiological information is scanty and usually only reports a myopathic pattern.[1-3]

We report the clinical findings, laboratory manifestations and, particularly, the results of a careful electrophysiological examination in 10 patients with only mild neuromuscular involvement by trichinosis. Correlation between electrophysiological and laboratory findings was made.

The symptoms began 4 to 16 days after consuming home prepared wild boar sausages (table). A large number of members of several families in the same small town were affected, although most cases were almost asymptomatic. Ten patients (table) had diffuse myalgias of variable duration, and some of them had subjective weakness. These 10 cases were electrophysiologically evaluated early, in the second or third week after onset of the symptoms (mean = 17, SD 3 days). A second electrophysiological examination was performed in two patients, 2 months (case 5) and 6 weeks (case 8) after the first exploration, when the patients were already asymptomatic.

The first manifestation of the disease was often characterised by nausea, vomiting, abdominal cramp and diarrhoea, always followed by myalgias. Myalgias had a variable duration, from 7 to 48 days. Subjective weakness was a common symptom (seven