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]

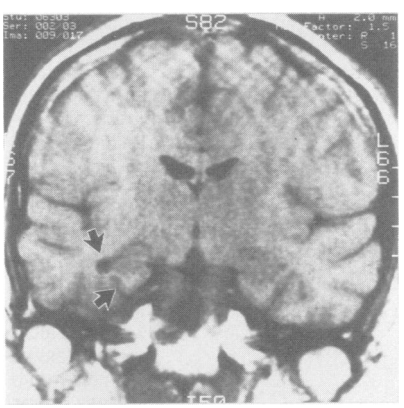

Fig *A coronal T1 weighted image magnetic resonance scan shows atrophy of the right parahippocampal gyrus (arrow) and enlargement of the right temporal horn of the lateral ventricle (arrow). (Note the right temporal lobe is on the left side of the photograph.)*

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

Table *Findings in 10 patients with mild trichinosis*

No	Sex	Age (yr)	Incubation period (days)	Fibrillation	Mean duration MUP (% decreased)	Percentage polyphasic potentials (N < 15%)	T (N = 215–457)	Mean amplitude (mV) (N = 0·24–0·44)	Ratio T/A (μV) (N = 0·71–1·23)	nFD (N = < 2)	% end-plates increased jitter (N < 10%)	Mean jitter (μs) (N < 39·5)	CK level (N < 260 IU)	Number eosinophils (N < 500)
1	M	47	6	+	10	33	391	0·28	1·39	NP	NP	NP	1033	1008
2	M	22	8	+	20	25	394	0·27	1·45	NP	NP	NP	1351	1808
3	M	30	4	+	24	25	400	0·27	1·48	5·0	30	58·5	167	3200
4	M	27	10	+	15	20	517	0·27	1·91	NP	NP	NP	446	1230
5	F	61	9	+	45	45	NP	NP	NP	5·3	20	52·0	121	1410
6	M	31	7	+	NP	NP	479	0·29	1·65	4·2	15	48·3	173	2880
7	F	28	10	+	28	20	NP	NP	NP	3·8	20	39·9	483	1370
8	M	21	7	+	10	15	NP	NP	NP	2·6	10	43·8	1420	1130
9	F	48	14	+	35	30	536	0·34	1·57	3·1	10	37·8	1176	660
10	F	37	16	−	NP	NP	630	0·32	1·96	3·8	0	33·0	121	700

M = male; F = female. Fibrillation potentials (+) = present; (−) = absent. Mean duration of the MUP (% decrease from normal mean) and percentage of polyphasic potentials in biceps brachii muscle. Automatic analysis of the EMG in biceps brachii muscle (T = number of turns; mean amplitude in mV. and T/A (μV) ratio). Single fibre EMG study in extensor digitorum communis muscle with data of nFD, percentage of end-plates with increased jitter and mean jitter values in μs. N = normal values; NP = not performed.

cases), although examination only revealed it in one case and in another was doubtful. Clinical examination suggested that weakness was mainly secondary to muscle pain. Sensation and tendon reflexes were normal. Other signs and symptoms were: periorbital and facial oedema in all patients, fever in nine subjects, conjunctivitis in four cases, headache in three and paraesthesiae in only one patient. The number of eosinophils was raised in all cases, and CK values were elevated in six (table) at the time of the first electrophysiological examination. All cases were clinically asymptomatic 6–8 weeks after the onset of the illness. The patients were treated with thiabendazole (25–50 mg/kg/day, for 5 days) and prednisone (0·3–0·5 mg/kg/day, for 1 week).

Muscle biopsy was performed in case 1 (biceps brachii), and showed *Trichinella spiralis* without encysted larvae within the muscle fibres. The diagnosis in the other patients was confirmed by positive trichinella serology.

Conventional EMG was performed in distal and proximal muscles of the upper and lower limbs. Full interference pattern was found in all tested muscles. Mean duration of the motor unit potentials (MUPs) and number of polyphasic potentials are shown in the table. Automatic analysis of the EMG[4] showed increase of the Turns-Amplitude (T:A) ratio (table). The SFEMG was performed in extensor digitorum communis muscle (EDC)[5] and fibre density (FD) was expressed as normalised FD (nFD), which excludes the changes due to age.[6] The values of nFD were increased in all examined patients (table). The SFEMG was newly performed in cases 5 and 8. The nFD increased from 5·3 to 9·7 and from 2·6 to 3·8, whereas the mean jitter decreased from 52·0 to 38·4 μs and from 43·8 to 31·7 μs respectively. The remaining patient refused a new EMG study.

The motor and sensory conduction velocities performed in upper and lower limbs were within the normal range for age.

The laboratory and electrophysiological data were correlated (Spearman's correlation coefficient). Decrement of the MUPs duration was significantly ($p < 0.01$) correlated with the raised CK values. Significant correlation ($p < 0.05$) was also found between nFD and mean jitter values.

The main electrophysiological findings reported here are consistent with myopathy in all patients. EMG changes were mild to moderate in eight cases, and moderate to severe in two. Peripheral nerve abnormalities were not found. Our findings in the conventional EMG were those typically reported in inflammatory myopathies. Quantitative EMG[4] also showed myopathic features.

The nFD and the jitter were abnormal in the patients with trichinosis reported here. A slight to moderate increase in FD in all tested cases and abnormal jitter in some of the patients have been previously reported in polymyositis,[7] where abnormalities in SFEMG were not correlated with clinical involvement and CK activity.[7] The results of SFEMG in our patients with trichinosis were the same as those reported in polymyositis,[7] and might be due to reinnervation following denervation. Preceding denervation could be caused by segmental necrosis of the muscle fibres.[5,7] Reinnervation in polymyositis is consistent with the fibre type grouping found in biopsied muscles.[7] Significant correlation between nFD and mean jitter in our patients might be due to the early stage of the regenerating activity, when the impulse transmission of the reinnervating complex was still uncertain. The follow-up in two cases is consistent with this hypothesis, because it indicated a large rearrangement of the motor unit (progressive increment in nFD) and more stable transmission of the impulse, perhaps owing to the maturation of the sproutings.

The significant correlation between decreased mean duration and raised CK could be due to drop-out of muscle fibres in the acute phase of the disease. Early examination might explain the poor correlation between enzymatic abnormalities, electrophysiological parameters and eosinophils count. A larger follow-up study could provide more information of the evolution of the electrophysiological parameters and long-time effects of the trichinosis on the motor unit architecture.

Our study proves that even in the acute stage of mild human trichinosis electrophysiological study shows early changes, and has a diagnostic yield for muscle involvement higher than raised CK values.

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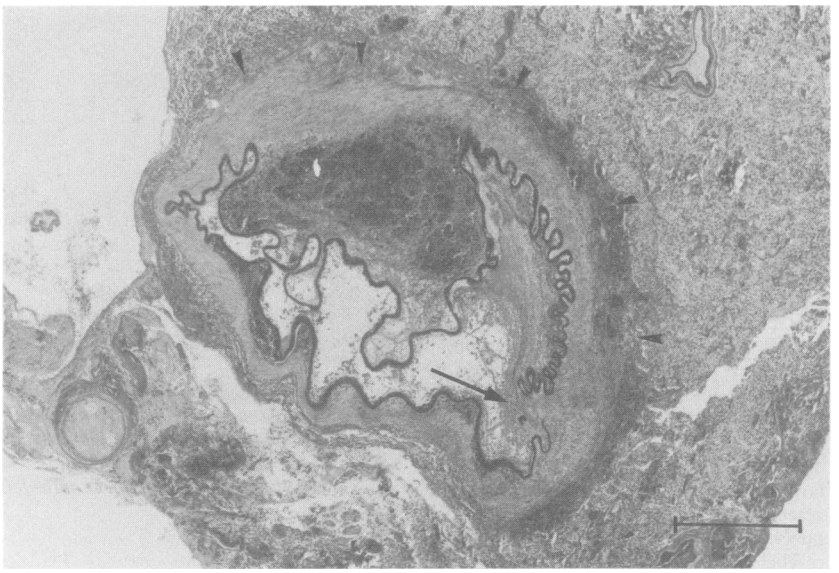

Fig *Cross-section of the middle cerebral artery with perivascular tumour fragments. The adventitial tissue is intimately connected with tumour stroma (arrow heads). A haemorrhage is situated underneath the internal elastic membrane. The large arrow indicates the defect in the internal elastic membrane. (Elastic van Gieson stain, bar = 50 μm.)*

Dissecting aneurysm of middle cerebral artery following resection of meningioma

Sir: Dissecting aneurysms of the intracranial arteries are rare. Spontaneous dissecting aneurysms have been reported in patients with arterial hypertension, cystic media degeneration, fibromuscular dysplasia, and in cases of Marfan's syndrome.[1 2] Blunt skull trauma may also lead to defects of the internal elastic membrane of intracranial vessels, but there is always the possibility of preexisting vascular lesions.[2] We present a case of post-traumatic dissecting aneurysm following partial resection of a large sphenoid ridge meningioma.

A 49 year old patient was admitted for the removal of a large meningioma. CT showed a tumour of the right sphenoid wing measuring 3·5 × 4 cm, with a 0·5 cm shift of the midline structures to the left. In preoperative angiography there was distension of the carotid siphon and the circle of Willis. The M1 sector of the ipsilateral middle cerebral artery was stenosed. At operation, the tumour was found surrounding the M1, M2, and M3 segment of the middle cerebral artery and the proximal segments of the anterior and communicans posterior arteries. Partial resection of the tumour was performed, leaving some surrounding the medial cerebral artery, which was seen pulsating during the operation. Twenty four hours after surgery, CT showed a slight hypodensity in the right temporal lobe. Three days after operation a large infarct in the area of the right middle cerebral artery was diagnosed by CT. The patient died on day 6 after surgery.

A large, approximately 5 day old infarct of the middle cerebral artery was found at postmortem examination. Small tumour fragments were still attached to the vessel wall. Histologically, an endotheliomatous meningioma had invaded the wall of the middle cerebral artery. There were multiple ruptures of the internal elastic membrane along a 5 mm segment, with underlying haematoma (fig). Some granulation tissue and iron-positive macrophages at the border of the haematoma were found. The other arteries were free of degenerative changes.

Some cases of dissecting aneurysms of intracranial arteries had recent head trauma, in other cases a generalised vasculopathy was observed.[1-4] In our case, this was clearly absent; all other vessels were free of pathological changes. The resection of a tumour intimately connected to the vessel wall appears to be the only plausible explanation for this dissecting aneurysm. In one previous case[5] a dissecting aneurysm was considered to be directly related to surgery and occurred after resection of an aneurysm of the middle cerebral artery. This is the first instance reported after surgery for a brain tumour.

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