immediately instituted but over the following 25 minutes he developed recurrent episodes of ventricular tachycardia, ventricular fibrillation, bradycardia and asystole. Before ventilation his serum potassium was 4.3 mmol/L; at the time of the initial ventricular tachycardia his serum potassium had risen to 8.6 mmol/L. Following resuscitation he was comatose with fixed dilated pupils. The next day there was evidence of anoxic brain damage with myoclonic jerks. Dolls head eye movements were initially present but were then lost and all brain stem reflexes were lost gradually over the course of six days at which time brain stem death was confirmed.

The most probable cause of the ventricular arrhythmias in this patient was suxamethonium induced hyperkalaemia. Hypoxaemia was very unlikely to have been a contributory factor because the partial pressure of oxygen was normal and the patient was given 100% oxygen before the procedure. Autonomic dysfunction in the Guillain-Barré syndrome may be severe enough to cause sudden death.³ Our patient had a sinus tachycardia before intubation but had no other signs of autonomic dysfunction. The ventricular tachycardia occurred before intubation so autonomic reflexes related to endotracheal intubation could not have caused this arrhythmia. Hyperkalaemia during the ventricular tachycardia after the suxamethonium administration indicates that suxamethonium induced hyperkalaemia was the probable cause of the arrhythmia.

Suxamethonium induced hyperkalaemia has been described in intracranial lesions,¹⁴ spinal cord lesions,⁵ peripheral nerve disorders,¹ muscular disease,⁶ prolonged immobilisation¹ and many non-neurological conditions including infections,⁷ burns⁸ and cold injury.⁹ Administration of suxamethonium causes a transient but slight increase in serum potassium in normal patients.⁴ This may be a result of the release of potassium from the skeletal muscles during repolarisation.

In denervated or traumatised muscle, the muscle cell membrane becomes supersensitive to suxamethonium thus accounting for the sudden release of substantial amounts of intracellular potassium after suxamethonium is given. There appears to be a critical period between day five and day 15 when supersensitivity is maximum in traumatised muscle. This is less clear in denervated muscle when the sensitivity increases dramatically by day five but can continue for weeks and longer in the chronic and relapsing denervation syndromes.¹⁰

Our case clearly demonstrates that in clinical practice suxamethonium should not be used in chronic or relapsing polyneuropathies. In addition, because of the varying reports of the "at risk" time span and because of the incompletely understood mechanism of the aetiology of the hyperkalaemia, we suggest that suxamethonium should be avoided in all cases of Guillain-Barré syndrome.

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Peripheral neuropathy associated with lithium toxicity

It is well known that lithium intoxication produces central nervous system damage,¹ but its effect on the peripheral nervous system is less well documented. We report a patient with generalised peripheral neuropathy following lithium intoxication, who recovered over several weeks. EMG and nerve conduction studies showed this to be an axonal neuropathy.

A 69 year old woman with long standing depression had been on 1000 mg/day of lithium carbonate for several years. She was admitted with a three day history of lethargy, withdrawal and depressed mood. She had stopped eating or drinking and on the day of admission become drowsy. On examination she was dehydrated, febrile at 37.5°C but vital signs were normal. She was obtunded but could move all four limbs to command against gravity and tendon reflexes were normal. Initial investigations showed impaired renal function (sodium 146 mmol/l, potassium 4.5 mmol/l, urea 48.3 mmol/l and creatinine 378 micromol/l), and a lithium level of 1.89 mmol/l (normal range 0.6-1.2 mmol/l). A full screen for infection was normal, and her pyrexia quickly settled.

Despite cautious correction of renal function with intravenous fluids, her conscious level continued to deteriorate, and she became unresponsive three days after admission. Her limbs were hypotonic, but still withdrew to pain, and she became areflexic with her plantar responses remaining flexor. With conservative management she slowly regained consciousness and ten days after admission was opening her eyes spontaneously. Gag reflex was present and she could swallow normally. Her limbs, however, remained flaccid and areflexic. There was distal loss of all sensory modalities in all four limbs. Haemoglobin was 11.3 g/dl, white blood cell count was 6,100/mm³, serum albumin 32 g/l and glucose 5.6 mmol/l. Her renal function had now returned to normal (sodium 143 mmol/l, potassium 4.2 mmol/l, urea 3.2 mmol/l and creatinine 50 micromol/l). Other routine biochemical and haematological tests were normal, as were creatine kinase, serum vitamin B12 and porphyrin studies. The cerebrospinal fluid was

normal (no cells, protein 0.5 g/l). Nerve conduction studies confirmed the presence of a peripheral neuropathy: sensory action potentials were absent in the legs and reduced or absent in the arms (left median, F2 and F3 to wrist, and left radial—all absent; left ulnar, F5 to wrist, 3 µV). Motor action potentials were reduced in all four limbs. Motor conduction velocities were at the lower end of the normal range (left ulnar 53 m/s-1; left common peroneal 41 m/s-1). EMG studies showed fibrillations distally in the legs and widespread polyphasic units. Nerve biopsy was not performed. The neurophysiological tests were consistent with an axonal neuropathy.

Over the next four weeks there was considerable return of limb function, so that she could feed herself and stand with support. Repeat neurophysiological testing at this stage indicated a mild improvement in motor action potentials but no significant sensory change. She continued to improve with physiotherapy and was discharged home after three months in hospital.

Whilst muscle fasciculations and paraesthesias are recognised clinical sequelae of lithium intoxication, frank peripheral neuropathy is rare and we have only been able to find six documented cases in the literature.¹⁶ Temporal events and the exclusion of other causes of neuropathy incriminated lithium in our case, which appears to show many similarities with the other cases that have been reported. A polyneuropathy of the critically ill is well recognised,⁷ but our patient was never ventilated and multiple organ failure was not present. In addition, no underlying infection could be found to explain the initial mild pyrexia.

She was encephalopathic at the time of presentation and her neurological condition deteriorated after admission. Brain lithium content is not always reflected by serum lithium concentration and worsening of neurological symptoms after discontinuation of lithium is well documented, even when serum levels are within the so-called "therapeutic" range.⁸ An acute axonal dysfunction seems to be the hallmark of lithium-induced peripheral nerve damage but there is often considerable clinical recovery over a period of weeks to months.¹⁻³ It has been suggested that intracellular accumulation of lithium may interfere with the propagation of the action potential.⁹

Peripheral nervous system damage secondary to lithium is probably under-recognised. Abnormal nerve conduction velocities have been reported in manic-depressives and healthy volunteers taking lithium.¹⁰ In addition, a mild neuropathy could easily be overlooked in acute toxicity where the central nervous system manifestations are usually much more dramatic. Lithium toxicity should thus be considered in any patient receiving this drug who develops evidence of a peripheral neuropathy, including muscle fasciculations or paraesthesia, even in the presence of central nervous dysfunction.

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Bulimia in a patient with temporal lobe epilepsy

There have been no reports of focal neuro-anatomical lesions associated with bulimia nervosa. The following is a case report of a patient with bulimia, who was found to have a lesion of the left temporal lobe on MRI during evaluation for a seizure disorder.

The patient was a 33 year old right handed female college administrator who had complained of fainting spells since the age of six. These had occurred at least once or twice a year and, after puberty, often five to seven days premenstrually. She came to neurological attention after having had three such spells in four months. During one of these recent episodes rhythmical twitching of the right hand had been observed.

She typically experienced an aura of a strong odour of ether lasting up to one minute before each event, followed by unconsciousness for about 30 seconds. Infrequently she also had experienced nocturnal bladder incontinence as a child and as an adult.

An eating disorder with onset at the age of 28 had been diagnosed as bulimia nervosa. Since that time she had been having recurrent periods of daily binge-eating behaviour followed by self-induced vomiting which would last for up to a year, followed by periods of remission lasting four to six months.

Despite feeling loss of control over her eating behaviour, she was always aware of her actions and had never experienced amnesia, somnolence, or syncope during the binges or purges. She generally maintained her weight in the normal range of 55-60 kg with a maximum weight fluctuation of 14 kg. The severity of the behaviour had increased during the couple of months preceding the time of presentation.

There had also been alcohol abuse up until five years ago. There was no history of depression. The family history included the father who had alcoholism and a paternal cousin with multiple sclerosis.

Her neurological and general examination were normal. Her mood was not depressed. A sleep deprived EEG showed a burst of theta slow waves in the left temporal leads during the awake state. An MRI of the brain showed a lesion in the left temporal lobe adjacent to the medial tip of the lateral ventricle in the region of the hippocampus. This was felt to represent a cystic dilatation of the ventricle or a small area of infarction (figure).

The patient was treated with carbamazepine. During the 18 months of treatment, she has had no further olfactory hallucinations, seizures, or syncopal episodes. She has had one episode of nocturnal incontinence. Her eating habits have been under good control and her weight has been stable. There have been no eating binges, purging, or feelings of compulsion to fast or eat large quantities of food.

This patient demonstrates a possible pathogenic relationship of temporal lobe disease to bulimia. Although the cause and pathogenesis of bulimia remain unknown, in addition to psychological factors, biological factors appear to play a role in the disorder.

Hyperphagia has been described in some neurological disorders including Huntington's disease, amygdectomy, frontal and hypothalamic tumours, frontal lobotomy, Pick's disease, epilepsy, Kleine-Levin syndrome, and von Economo's encephalitis. Hyperphagia in such patients, however,

clearly differs from the binge-purge diathesis seen in bulimia. Furthermore, bulimic patients tend to be selective in their eating behaviour, unlike the Kluver-Bucy syndrome, and maintain a normal weight. Aside from the relatively late onset of bulimia, this patient displays the behaviour, feelings, and clinical course typical of the more specific disorder of bulimia nervosa.

Studies of patients with bulimia nervosa using CT have reported ventricular dilation and sulcal enlargement; however, focal brain disease has not been demonstrated. MRI is a more sensitive technique for the detection of temporal lobe lesions in epilepsy, which could be of potentially greater yield in showing lesions in patients with eating disorders, as was seen in this case.

The temporal lobes have an integrative role in forming the brain's body image via afferent information from secondary sensory areas and the limbic system. Structural or biochemical disease of this part of the brain could thus play a role in the distorted self image which occurs in bulimia.

Reports of EEG abnormalities in bulimia range from 16-64%, depending mainly upon the diagnostic criteria used for EEG abnormality. In the series of 23 patients with compulsive eating treated by Rau and Green, a positive response to phenytoin was correlated with those patients having a combination of abnormal EEG plus other neurological symptoms such as dizziness or rage attacks.¹ A modest response to phenytoin was seen in a later double blind, placebo-controlled study of 20 patients; however, response was not correlated with pretreatment EEG abnormality.² Subsequent case studies of phenytoin treatment were less positive, and enthusiasm for anticonvulsant treatment of bulimia waned.

Epilepsy has yet to be conclusively demonstrated as a direct cause of bulimia. Disturbances of body perception are known to occur as experiences in patients with temporal lobe epilepsy, as well as compulsive water drinking and ictal vomiting. Ictal eating has not been reported; however, eating has been reported as a reflex cause of epilepsy, with seizures being most often complex partial in type and related to temporal lobe foci.³ Also, Remick reported three patients with postictal hyperphagia.⁴

The relationship of bulimia and temporal lobe epilepsy in this case is that of an interictal behaviour. The response of abnormal eating behaviour to carbamazepine in this patient could have been related to suppression of limbic kindling in the hypothalamus by the seizure focus.

Treatment of bulimia with carbamazepine has been reported in a double-blind crossover trial of six patients, of whom one showed a dramatic response to carbamazepine.⁵ This patient also had an associated mood disorder. A patient with bulimia, depression, and amnesic episodes with an EEG showing diffuse paroxysmal activity has also been reported to improve with carbamazepine treatment.⁶

The possibility that the temporal lobe lesion and secondary epilepsy in this patient were coincidentally related to bulimia cannot be excluded. The observations that her epilepsy worsened and came to attention during a flareup of bulimic behaviour and that both problems went into prolonged remission simultaneously with anticonvulsant therapy suggest a significant relationship. Furthermore, carbamazepine seems to be of potential

Figure MRI axial views showing A) low signal area in the medial left temporal lobe on T_1 -weighted image (TR = 857 ms, TE = 20 ms) and B) high signal area in the same region on T_2 -weighted image (TR = 3243 ms, TE = 80 ms).