LETTERS TO THE EDITOR

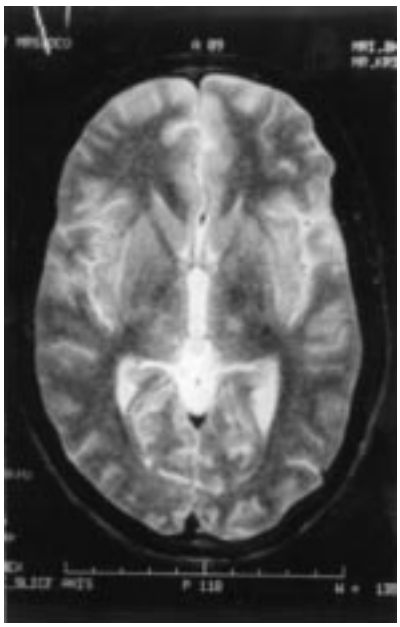
Cerebellar ataxia due to lead encephalopathy in an adult

Lead has been used by humans as long as recorded history for various purposes ranging from jewellery to weapons and construction materials, paints, and pigment manufacture. Lead colic was known to ancient physicians since the time of Hippocrates, but encephalopathy was first described as late as 1925; it is especially common in children. Here we report a rare case of lead encephalopathy associated with ataxia in a 30 year old battery plate manufacturer.

He had been working for the past 12 years in a factory making battery plates. He presented with history of abnormal behaviour and unsteadiness of gait for 8 days accompanied by colicky abdominal pain and paraesthesiae in the legs. Later the patient started behaving abnormally; he shouted irrelevantly, became violent, and refused to recognise relatives. He was treated with antipsychotic medication which quietened him. Two days later he had difficulty in walking. His gait was unsteady and speech was incoherent.

Enquiry disclosed two earlier episodes of abdominal pain with abnormal behaviour in the past year which responded to treatment, the details of which were not available. Two other factory workers had also had episodic abnormal behaviour but were not available for interrogation. There was no history of fever or symptoms suggestive of raised intracranial pressure, seizures, or focal motor or sensory deficits. The patient denied consumption of alcohol on a regular basis.

On admission the patient was afebrile, pulse 82/min, BP 116/76 mm Hg. There was mild pallor and a suspicious bluish grey discoloration of the gums. He was conscious and oriented, but extremely restless. He was totally anarthric. A detailed evaluation of higher functions was not possible due to the restlessness and anarthria, but from the limited evaluation, comprehension appeared intact. There was no evidence of hallucinations or delusions. The optic fundi were normal. There was no nystagmus and the lower cranial nerves were normal except for slow movements of the tongue.



T2 weighted MRI showing hyperintensities in both thalami.

Motor system examination disclosed no significant weakness. There were prominent cerebellar signs in the form of truncal ataxia, impaired finger to nose and knee-heel tests, and dysdiadochokinesia. There was no tremor and no sensory deficits. Deep tendon reflexes were normal. Both plantar reflexes were extensor. There was no neck stiffness.

The history of abdominal colic and behavioural abnormalities in a person working with battery plates and also of similar complaints in coworkers, raised a clinical suspicion of lead toxicity.

Blood lead concentration estimated on the next day was 89 μg/dl (normal range 10–15 μg/dl), which confirmed lead toxicity. The patient had hypochromic microcytic anaemia (Hb 8.3 g). There was no basophilic stippling. Urine examination was normal and negative for porphyrins. There was no azotaemia (BUN 10.0 mg/dl, serum creatinine 1.0 mg/dl), Serum electrolytes were normal Na 133 (normal 132-–144) meq/l, K 4.6 (3.6–5.0) meq/l, Cl 98 (96–108) meq/l, Ca 10.0 (9-ll) mg/dl. Liver function tests were normal.

T2 weighted MRI disclosed bilateral hyperintense lesions in both thalami (figure). The EEG did not show any focal or background rhythm abnormalities. Peripheral nerve conduction studies were within normal limits.

The patient was treated with mannitol and intravenous fluids. Oral chelation therapy was started with penicillamine on day 2. EDTA or dimercaprol could not be instituted due to unavailability. The patient showed significant improvement in behaviour, and speech returned by day 3 of admission, although extremely slurred. The unsteadiness and dysarthria improved steadily until discharge 2 weeks later. Repeat estimation of lead concentration at discharge was 64 μg/dl. The patient showed complete neurological recovery on follow up 6 months after discharge. He was able to perform his routine duties, but decided to look for a different job. A repeat blood lead concentration was 52 g/dl.

Lead intoxication most often occurs in 1 to 3 year old children due to chewing of lead paint. Acute encephalopathy is a serious complication in children, which can be fatal or leave permanent neurological sequelae.[1]

Lead toxicity is much less common in adults. It is mainly an occupational hazard due to inhalation of lead fumes or physical contact with lead in processes that require remelting of lead, such as painting, lead smelting, pottery glazing, and storage battery manufacture. The emissions of vehicles using leaded petroleum is a recognised source of environmental pollution in urban areas.[2] The usual manifestations of lead poisoning in adults are colic, anaemia, and peripheral motor neuropathy. Encephalopathy is rare. Whitfield *et al* (1972) reported the largest series of 23 adults with lead encephalopathy; all these followed consumption of illicit liquor contaminated by lead (moonshine).[3] Ten of these patients had altered sensorium; 18 of them had seizures. Other symptoms included dizziness, syncope, disorientation, and blindness. One had papilloedema. None of the patients had ataxia. Ataxia was a prominent feature in our patient and has been described as a feature of lead encephalopathy in children.[1 4] This could be secondary to raised intracranial pressure or to direct involvement of the cerebellum. Lead acts as a cellular toxin by inhibiting mitochondrial respiration. The increased resistance of the adult to encephalopathy and ataxia is believed to be due to the capacity of the mature brain to sequestrate lead away from its mitochondrial site of action within the cerebral and cerebellar neurons.[5]

T2 weighted MRI in our patient disclosed hyperintensities in both thalami (figure). Schroter *et al* reported high signal intensities in the periventricular white matter, basal ganglia, insula, posterior thalamus, and pons.[6]

Our patient did not have basophilic stippling of peripheral red blood cells. Basophilic stippling of erythrocytes is reported in 91% of patients by Whitfield *et al*,[3] but was seen in only 40% of cases reported by Greengard *et al*.[4] It is more pronounced in the bone marrow than in the peripheral blood[7] and may be missed unless carefully looked for.

The extent of clinical recovery was out of proportion to the decline in blood concentrations. Free erythrocyte protoporphyrin and the urinary concentrations of γ-aminolevulinic acid (ALA) or coproporphyrin are better clinical correlates of lead toxicity, rather than estimations of blood lead concentration. In our patient only the urinary porphobilinogen could be estimated.

Severe medical illness, alcohol, dehydration, and emotional stress are known to precipitate symptoms of lead poisoning, but we were unable to identify any such factors in our patient.

To summarise, frank encephalopathy due to lead intoxication has become increasingly rare in adults. We report a patient with lead encephalopathy who presented with behaviour problems and cerebellar ataxia.

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"All tibial foot" with sensory crossover innervation between the tibial and deep peroneal nerves

One of the most common and well studied innervation anomalies in the upper limbs is the Martin-Gruber anastomosis.[1 2] In the lower limbs, the anomaly is uncommon except for the accessory deep peroneal nerve.[1 2] Recently, an exclusive innervation of the extensor digitorum brevis by the tibial nerve, "all tibial foot" has been reported.[3–5] We experienced a similar patient with "all tibial foot", who, in addition, showed sensory anomaly.

A 23 year old man with encephalitis had nerve conduction studies (NCSs) to exclude coexistent peripheral neuropathy. The studies were normal except for the anomalous innervation in the bilateral lower limbs. Peroneal nerve stimulation at the ankle, fibular head, and popliteal fossa elicited only a negligible compound muscle action potential (CMAP) over the extensor digitorum brevis. The accessory deep peroneal nerve was not demonstrated by stimulation behind the lateral malleolus.[1 2] A normal CMAP from the extensor digitorum brevis was elicited by stimulating the tibial nerve at the ankle and popliteal fossa (figure A). Although CMAP of the anterior tibial muscle was normally elicited by stimulating the common peroneal nerve at the fibular head, a small CMAP was recorded also by the stimulation of the tibial nerve at the popliteal fossa (figure B).

Sensory studies of the sural, superficial peroneal and medial plantar nerves were normal. Stimulation of the deep peroneal nerve at the ankle gave rise to a normal antidromic sensory nerve action potential (SNAP) in the skin between the first and second toes, where an obvious SNAP was recorded even after the stimulation of the tibial nerve behind the medial malleolus (figure C).

Our patient had "all tibial foot" for the motor innervation, the anomalous dual innervation of the anterior tibial muscle, and the sensory coinnervation of the skin between the first and second toes by the tibial and deep peroneal nerves. Findings were similar on both sides. We speculate that, in our patient, the deep peroneal nerve becomes almost pure sensory after branching motor fibres for the anterior tibial muscle, and that the extensor digitorum brevis is innervated by the tibial nerve. Further, the tibial nerve had a motor branch for the anterior tibial muscle and a sensory branch to supply the area typically innervated by the deep peroneal nerve. Although rare, we should keep in mind this anomaly in the practice of nerve conduction studies.

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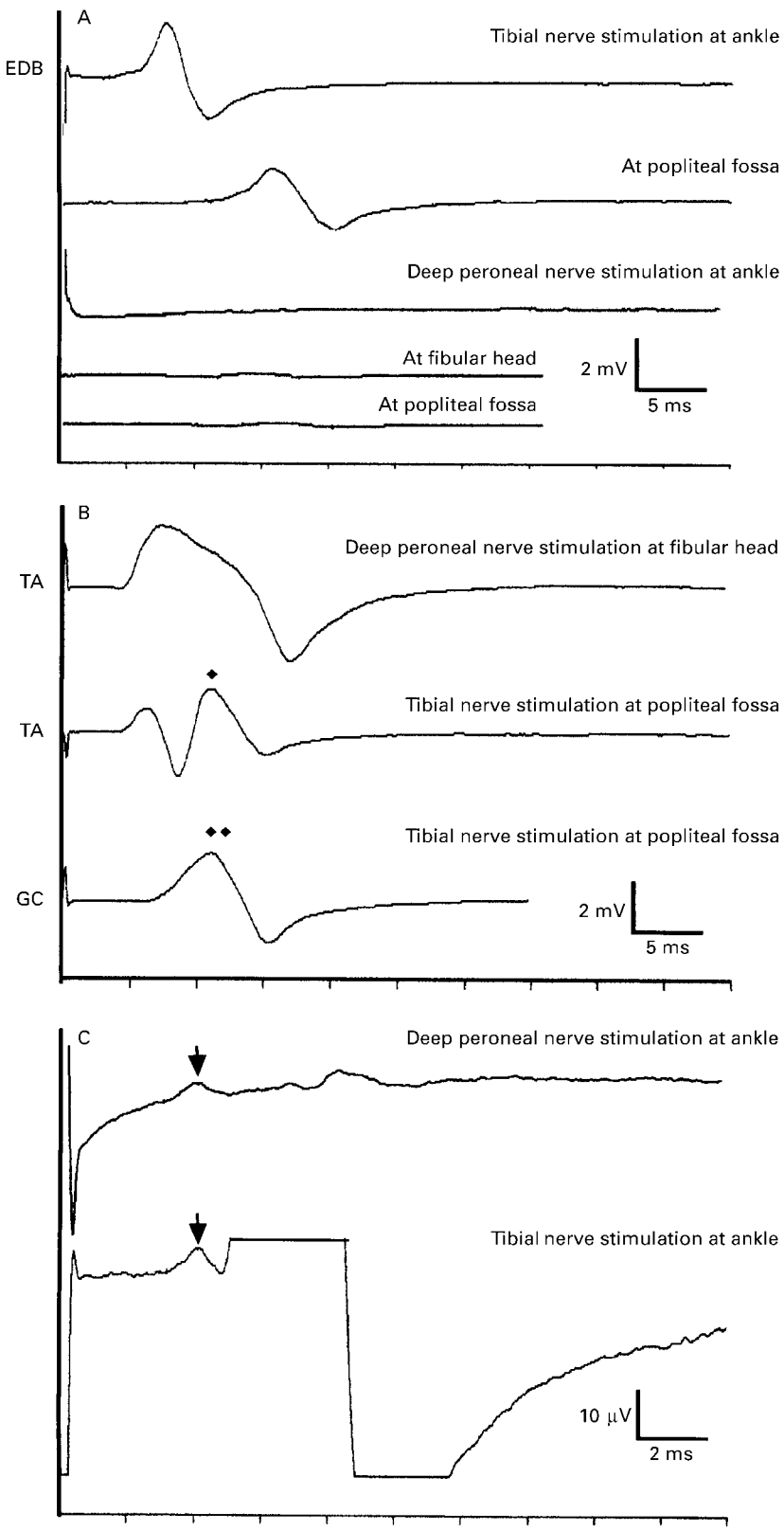

(A) Stimulation of the tibial and deep peroneal nerves at the ankle and popliteal fossa. Recordings from the extensor digitorum brevis. (B) Stimulation of the tibial and deep peroneal nerves at the popliteal fossa. Recordings from the anterior tibial muscle. The second negative peak (◆) is probably made by the volume conduction from the simultaneously contracting gastrocnemius muscle (◆◆). (C) Stimulation of the tibial and deep peroneal nerves at the ankle. In either case, sensory nerve action potential (arrow) was recorded from the skin between the first and second toes.