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 but were not available to recognise relatives. He was treated with antipsychotic medication which quietened paraesthesiae in the legs. Later the patient started behaving abnormally; he shouted incoherent.

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 to sensory deficits. Deep tendon reflexes were normal. Both plantar reflexes were extensor. There was no neck stiffness.

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 feature 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.

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 abdominal pain and paraesthesiae in the legs. Later the patient started behaving abnormally; he shouted irreverently, 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.

The patient was treated with mannitol and dimercaprol could not be instituted due to renal failure.

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

JAYANTI MANI NALIN CHAUDHARY MAKARAND KANJALKAR PRAVINI U SHAH
Department of Neurology, KEM Hospital, Parel, Mumbai 400012, India
Correspondence to: Dr J Mani, Department of Neurology, Ward 10, KEM Hospital, Parel Mumbai 400012, India.

“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. In the lower limbs, the anomaly is uncommon except for the accessory deep peroneal nerve. Recently, an exclusive innervation of the extensor digitorum brevis by the tibial nerve, “all tibial foot” has been reported. 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. 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 coincinnervation 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.

MARIKO YAMASHITA
Department of Neurology, Osaka Saiseikai Nakatsu Hospital, Osaka, Japan

TAKAHIRO MEZAKI
Department of Neurology, Sakakibara Hakuho Hospital, Hisai, Japan

TORU YAMAMOTO
Department of Neurology, Osaka Saiseikai Nakatsu Hospital, Osaka, Japan

Correspondence to: Dr Mariko Yamashita, Department of Neurology, Osaka Saiseikai Nakatsu Hospital, 2–10–39 Shihata, Kitaku, Osaka, 530-0012, Japan.


(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.
functions were normal. On day 5 her CSF had C-reactive protein (9.0 mg/dl) were raised. 

blood cell count (15 600/µl) and serum no meningeal signs were present. Her white 

lar ataxia. She complained of headache, 

there were headaches, nausea, and vertigo. The regional distribution of her symptoms 

34 5 23 4 32 21 20 19 18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1

Thin layer chromatography with immunostaining. Thin layer chromatography plates stained with (A) orcinol/sulphuric acid for hexose; (B) IgG from our patient, subsequently stained by peroxidase 

cells were 7/µl. Motor conduction velocity in the right tibial nerve on Day 11 was slightly 

i.e. to test whether the anti-GT1a antibodies do not express GT1a, and anti-GQ1b antibodies 

3 Linda D, Berlit P. The intrinsic foot muscles are rarely innervated by the tibial nerve (“all 

4 Glocker FX, Deuschl G, Lucking CH. Trau-

5 Linden D. Traumatic lesion on the common peroneal nerve with complete foot drop and preserved disi-

612.0x792.0

functions were normal. On day 5 her CSF had 

C-reactive protein (9.0 mg/dl) were raised. 

blood cell count (15 600/µl) and serum no meningeal signs were present. Her white 

lar ataxia. She complained of headache, 

there were headaches, nausea, and vertigo. The regional distribution of her symptoms 

34 5 23 4 32 21 20 19 18 17 16 15 14 13 12 11 10 9 8 7 6 5 4 3 2 1

Thin layer chromatography with immunostaining. Thin layer chromatography plates stained with (A) orcinol/sulphuric acid for hexose; (B) IgG from our patient, subsequently stained by peroxidase 

first she noticed diplopia and dysphagia. Two weeks after admission, her ocular movements were almost normal, ptosis remained only in the right eye, the strength of the neck muscles had increased to 4, and deep reflexes became detectable. All these symptoms disappeared within 3 months of onset. Serum antiganglioside antibody titres were determined using an enzyme linked immuno-

sorbent assay (ELISA). On day 11, the titres of anti-GM1, GM2, GD1a, GD1b IgG, and 

IgM antibodies were <500; whereas the titre of the IgG anti-GT1a antibody had increased to 

16 000 and that of the anti-GQ1b antibody was 500 (normal ranges were set at <500). No 

IgM antibodies to GT1a or GQ1b were detected. Thin layer chromatography with 

immunostaining confirmed that her serum IgG reacted with GT1a but not with GQ1b or 

GD1a (figure 2). An absorption study showed that her IgG anti-GT1a antibodies were not absorbed by GQ1b, GD1a, or GM1 (data not shown). Three months after onset, the respective serum titres of the anti-GT1a and anti-GQ1b antibodies had decreased to 500 and <500.

The regional distribution of her symptoms was the same as that of the pharyngeal-

cerebrospinal fluid (CSF) antibodies. We report on a patient with this variant 

and Guillain-Barré syndrome. Some of these antibodies are associ-

ated with certain clinical variants or signs of 

Guillain-Barré syndrome. Chiba et al 

detected IgG anti-GT1a and anti-GQ1b antibodies in three patients with acute orophary-

geal palsy. The presence of IgG anti-GT1a antibodies in these patients and in ours was 

associated with the emergence of acute polyneuropathy with marked lower cranial 

nerve involvement. IgG anti-GT1a antibodies which do not cross react with GQ1b may be 

closely related to the proclivity of Guillain-

Barré syndrome to manifest oropharyngeal 

palsy or to the cause of its pharyngeal-cerebrospinal 

variant. The IgG anti-GT1a antibody present in Fisher’s syndrome may recognise a 

structure common to GT1a and GQ1b gangliosides, whereas in the pharyngeal-

cerebrospinal variant it would react with another epitope specific to GT1a.

This research was supported in part by grants in aid 

from the Ono Medical Research Foundation, Uehara Memorial Foundation, and Takeda 

Foundation (Japan) for the Promotion of Science, the Nakabayashi Trust for ALS Research, the Ryoichi 

Naito Foundation for Medical Research, and by a Research Grant for Neuroimmunological Diseases from the Ministry of Health and 

Welfare. 

KENICHI KASHIHARA

Department of Neurology, Okayama University 

Medical School, Okayama, Japan

MICHIAKI KOGA

Department of Neurology, Dokkyo University School of 

Medicine, Tochigi, Japan

Correspondence to: Dr K Kashihara, Department of 

Neurology, Okayama University, 2–5–1 Shikata-cho, Okayama 700–0914, Japan.

1 Chiba A, Kusunoki S, Obata H, et al. Serum anti-GQ1b IgG antibody is associated with ophthalmoplegia in Miller Fisher syndrome and 

Guillain-Barré syndrome: clinical and 

immunohistochemical studies. 


2 Ropper AH. Unusual clinical variants and signs in 

Guillain-Barré syndrome. Arch Neurol 1986; 

45:1150–2.

3 Mizoguchi K, Hase A, Obi T, et al. Two species of 

antiganglioside antibodies in a patient with a 

pharyngeal-cerebrospinal variant of 

Guillain-Barré syndrome. J Neurol Neurosurg 


4 O’Leary CP, Veitch J, Durward WP, et al. Acute oropharyngeal palsy is associated with antibodies 

to GQ1b and GT1a gangliosides. J Neurol 


Dexamethasone is not necessarily unsafe in primary supratentorial 

intracerebral haemorrhage

Controversy surrounds the role of steroids in the treatment of intracerebral haemorrhage.1 

Theoretically, the short term use of dexam-

ethasone is justified because it lessens the 

risk of complications, especially infections and 
gastrointestinal haemorrhage.2

1 Chiba A, Kusunoki S, Obata H, et al. Serum anti-GQ1b IgG antibody is associated with ophthalmoplegia in Miller Fisher syndrome and 

Guillain-Barré syndrome: clinical and 

immunohistochemical studies. 


2 Ropper AH. Unusual clinical variants and signs in 

Guillain-Barré syndrome. Arch Neurol 1986; 

45:1150–2.

3 Mizoguchi K, Hase A, Obi T, et al. Two species of 

antiganglioside antibodies in a patient with a 

pharyngeal-cerebrospinal variant of 

Guillain-Barré syndrome. J Neurol Neurosurg 


4 O’Leary CP, Veitch J, Durward WP, et al. Acute oropharyngeal palsy is associated with antibodies 
to GQ1b and GT1a gangliosides. J Neurol 

So far, only two randomised, controlled trials concerning this controversy have been reported. One trial of 40 patients assumed to have intracerebral haemorrhage, found no beneficial effects of steroids.1 However, this study had 22 patients with haemorrhagic infarction and a loss of haemorrhage area, and the outcome measures used had little clinical relevance regarding functional ability of the patients. The other trial, from Thailand,2 was well designed, but had to be terminated prematurely. An interim analysis disclosed lack of benefit and presence of clinically important adverse effects. Patients in the dexamethasone group had more frequent infections, gastrointestinal haemorrhage, and diabetogenic effect, than the placebo group. There was a possible longer early survival in a subgroup with less severe stroke. Good recovery was noted in 17% of patients in the dexamethasone group, compared with 10% of patients in the placebo group, giving a difference of 7% in favour of the dexamethasone group.

In our experience, the complication rates are not as high with dexamethasone as reported by Pourugarin et al.3 To consider the issue of safety of dexamethasone in primary supratentorial intracerebral haemorrhage, we undertook a double blind, randomised, placebo controlled trial as a pilot project.

Twenty-six patients in the age group 40–80 years, with primary supratentorial intracerebral haemorrhage confirmed by brain CT, presenting within 5 days of onset were included. Patients with a history of previous disabling stroke or contraindications to steroid treatment were excluded. Informed consent was taken from relatives of all patients before admission into the trial. Dexamethasone was given intravenously, at a dose of 4 mg 6 hourly for 12 days, followed by 4 mg 12 hourly for two days and 2 mg 12 hourly for 2 days. Placebo injections of saline were given in the same dosage, from ampoules of similar size and shape, and with comparable labels, filled with a colourless solution indistinguishable from dexamethasone. No one involved with the study knew whether a particular patient was receiving dexamethasone or placebo. All patients received injectable rintidine, 50 mg 8 hourly, for the period of the trial. The remainder of patient care was specified by their attending neurologist. The allocated treatment was stopped if the attending neurologist thought that the patient had developed a complication likely to be caused by, or aggravated with dexamethasone. No other antiedema measure (mannitol/glycerol) or neurosurgical intervention was undertaken, except in one patient who was found to have a ruptured anterior communicating artery aneurysm, diagnosed after randomisation.

Details of history and physical examination conducted at the time of admission were recorded. Haematoma size was calculated using the formula given by Kothari et al.4

Outcomes measures of all patients were assessed by one of us (PD), using the Glasgow outcome scores5 at day 7 and at discharge. Fasting blood sugar was done one week after starting the treatment, to detect any diabetogenic effect. The development of fever after entry into the study was considered to be indicative of infection, irrespective of whether the focus of infection was detected or not. Statistical methods used were χ² test with continuity correction for categorical variables and a two tailed t test for continuous data.

The clinical characteristics, complications, and outcome of the patients are shown in the table. Both groups were well matched for age, hours from haemorrhage to treatment, blood pressure, volume and location of haematoma, and presence of intraventricular extension of blood. However, there were more comatose patients (Glasgow coma scale score<7) in the dexamethasone group (5/12) than in the placebo group (3/14).

The complication rates were higher in the placebo group than in the dexamethasone group (placebo group, 10 v dexamethasone group, 4), but the difference was non-significant. One patient in each group was found to be between 21 days post randomisation, and their allocated treatment was stopped within 3 days, but they continued to remain in the allocated group for analysis.

In all, seven patients died (five patients in the dexamethasone group died due to herniation within the first week and two patients in the placebo group died; one died of herniation in the first week, and the other died due to septicaemia on day 36). There was no difference between the groups in the number of patients who had a good outcome (2/12 in the dexamethasone group v 2/14 in the placebo group).

There could be many reasons why Pourugarin et al.5 found a higher frequency of adverse effects due to dexamethasone. Firstly, it may have been a chance finding. If they had continued the study further, the placebo group may have had similar complications such as infections or hyperglycaemia, thereby making the difference between the two groups non-significant. Secondly, the dexamethasone group may have had a higher proportion of serious patients who are more likely to develop complications such as infections, gastrointestinal haemorrhage, or hyperglycaemia. This may occur despite stratified randomisation, particularly when the numbers are few in the individual stratum. Arguably, similar factors in our placebo group may explain our findings. This may be true, but it emphasises the few and hence not very reliable data on whether dexamethasone is too unsafe to be used.

We conclude that dexamethasone in the regimen used in our trial is not likely to produce an unacceptably high rate of adverse effects in patients with primary supratentorial intracerebral haemorrhage. Whether or not dexamethasone or other corticosteroids are beneficial in this setting needs further study. The study by Pourugarin et al.4 points to a higher proportion of patients making complete recovery by the 21st day in the treatment group (odds ratio 0.7, 95% CI, 0.2–2.4), this needs to be either confirmed or refuted.

P DESAI

Department of Neurology, All India Institute of Medical Sciences, New Delhi, India

Correspondence to: Dr K Prasad, Department of Neurology, All India Institute of Medical Sciences, New Delhi 110029, India. Telephone 0991 11 651 6872; fax 0991 11 686 2663; email kprasad@medinsternet.in


Clinical characteristics, complications, and outcome of patients

<table>
<thead>
<tr>
<th>Age (mean (SD))</th>
<th>58.3 (11.7)</th>
<th>59.8 (7.4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemorrhage to treatment (h, mean (SD))</td>
<td>30.3 (31.8)</td>
<td>27.2 (29.7)</td>
</tr>
<tr>
<td>Glasgow coma scale scores:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3–7</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>8–11</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>12–14</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Volume of haematoma (ml, mean (SD))</td>
<td>34 (36.4)</td>
<td>35.7 (35.3)</td>
</tr>
<tr>
<td>Location of haematoma:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal ganglia</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Thalamus</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Lobar</td>
<td>2*</td>
<td>35</td>
</tr>
<tr>
<td>Intraventricular leakage of blood</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Mean (SD) arterial pressure (mm Hg)</td>
<td>123.9 (13.9)</td>
<td>133.8 (20.2)</td>
</tr>
<tr>
<td>Complications:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Gastrointestinal bleeding</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Diabetogenic effect</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Reasons for stopping allocated treatment:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal bleed</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Abdominal distension</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Jaundice</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Wrong diagnosis</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Outcome at discharge:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Due to herniation</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Due to infection</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Vegetative</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Independent</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Death</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

* One patient had haemorrhagic infarct. † One patient had aneurysmal bleed. § Infection which was not present or suspected at the time of admission to the study. ‡ Includes overt gastrointestional bleed (haematemesis, bloodstained gastric aspirate or melena), and clinically important gastrointestional bleed (overt gastrointestinal bleed with either, a fall in systolic blood pressure>20 mm Hg within 2 hours of bleeding; fall in haemoglobin >2 g/dl; blood pressure reduction >10 mm Hg; and increase in heart rate >20 beats/minute on orthostatic change; or need for blood transfusion). ¶ Fasting sugar >160 mg/dl, requiring regular insulin in a patient who was not previously considered to have diabetes, and who was not receiving glucose.

Postpartum cerebral venous thrombosis, congenital protein C deficiency, and activated protein C resistance due to heterozygous factor V Leiden mutation

Activated protein C resistance (APC-R) due to factor V Leiden mutation is the most common thrombophilia associated with cerebral venous thrombosis. It is present in 10% to 20% of patients but usually in association with other constitutional or acquired prothrombotic conditions. We present a case of postpartum cerebral venous thrombosis in a patient with protein C deficiency and APC-R due to heterozygous factor V Leiden mutation. In addition to puerperium, the role of intravenous steroids is questioned in this case.

A 33-year-old woman was admitted because of severe subacute headaches, nausea, and drowsiness. She was not taking oral contraceptives. Her medical history disclosed recent delivery of a second child 3 weeks before and an asthma attack 5 days before entry, treated with intravenous methylprednisolone (120 mg daily). Family history disclosed that the patient's mother had had postpartum lower limb deep vein thrombosis. On admission, clinical examination disclosed papilloedema. There was no fever and no ear, nose, or throat infection. Brain CT showed a right temporal hypodensity, a delta sign, and small ventricles. Brain MRI (with MRA) demonstrated recent superior sagittal sinus, and right and left lateral sinus thrombosis. High dose intravenous heparin was immediately initiated. Heparin treatment was switched to warfarin after 10 days. At 3 months, neurological examination was normal. Follow-up MRA showed complete recanalisation of the superior sagittal sinus and the right lateral sinus, and partial recanalisation of the left hypoplastic lateral sinus. Oral anticoagulation was maintained at International Normalised Ratio (INR) between 2 and 3. One year later, the patient is still symptom free under this treatment.

The proband was investigated and examined (figure and table). The proband (II.2) exhibited qualitative protein C deficiency and APC-R due to heterozygous factor V Leiden mutation. Protein S activity and antithrombin III concentrations were normal. Search for antineuronal antibodies, anticardiolipin antibodies, and lupus anticoagulant, dysfibrinogenemia, or plasminogen deficiency was negative. Plasma homocysteine concentration was normal.

The father (I.1) had APC-R due to heterozygous factor V Leiden mutation. The mother (I.2) had a qualitative protein C deficiency. The first sister (I.1) had no APC-R nor protein C deficiency. The second sister (II.3), who had one pregnancy without any thrombotic event, had both protein C deficiency and APC-R due to heterozygous factor V Leiden mutation.

Our patient presented an extensive postpartum cerebral venous thrombosis with a double inherited coagulation defect: protein C deficiency and APC-R due to heterozygous factor V Leiden mutation. To our knowledge, the coexistence of these two thrombophilias has not been previously described in a patient with cerebral venous thrombosis. Genetic studies showed that APC-R due to factor V Leiden mutation was transmitted by the patient's father and that protein C deficiency was transmitted by the patient's mother.

The protein C anticoagulant pathway is triggered when thrombin binds to the endothelial cell receptor thrombomodulin. This interaction converts thrombin into a potent protein C activator while blocking the fibrinogen clotting and platelet activating activity of thrombin. Activated protein C then serves as an anticoagulant by inactivating factors Va and VIIIa. In our patient, acquired causes of protein C deficiency could be excluded. We diagnosed a congenital protein C deficiency with an autosomal dominant mode of inheritance. A type I deficiency could be postulated according to the reduction of both antigen concentration and activity of protein C, reduction of activity but normal antigen concentration being present in type II. Only a few cases of cerebral venous thrombosis with congenital protein C deficiency have been reported so far. Inherited protein C deficiency seemed to be the sole risk factor for thrombosis in some cases whereas other prothrombotic conditions such as the postpartum period were present in other cases. Additional risk factors for thrombosis might be necessary for thrombotic manifestations to appear in patients with protein C deficiency as the postpartum state is usually asymptomatic with an estimated prevalence of 1 in 200 to 300.

In 1993, Dahlbäck et al described a new pathological condition termed APC-R, characterised by a poor anticoagulant response to activated protein C. This coagulation disorder is often associated with a single point mutation in one or both alleles of the factor V gene (adenine substituted for guanine at nucleotide 1691, the so-called Leiden mutation) at a site of cleavage by activated protein C which delays inactivation of coagulant factor Va. The prevalence of the factor V Leiden mutation varies by geography and ethnicity, ranging from 2% to 15% in healthy white people. Factor V Leiden mutation is inherited as an autosomal dominant trait and most heterozygous people do not have clinical thrombotic complications. The odds ratio for venous thrombosis has been calculated to be 3.8-fold for heterozygous patients. An important similarity of patients with APC-R and cerebral venous thrombosis is the frequency of other associated risk factors for thrombosis. Sixteen of 18 reported cases showed another genetic or acquired thrombosis risk factor: oral contraceptives in eight, pregnancy/puerperium in four, nephrotic syndrome in one, intravenous steroids in one, immobilisation in one, primary antiphospholipid antibody syndrome in one, and antithrombin III deficiency in one. These data suggest that the association of APC-R with an other prothrombotic state is crucial in the occurrence of cerebral venous thrombosis. In our patient, APC-R was associated with congenital protein C deficiency and two other acquired prothrombotic risk factors, postpartum and intravenous steroid therapy. The thrombotic risk of intravenous steroids is questioned in our patient as she had not experienced any thrombotic event during her first pregnancy and postpartum period. It is noticeable that the patient's sister (II.3) with APC-R and protein C deficiency had had one child without venous thrombosis. The prothrombotic risk of intravenous steroids should be considered in patients with this condition.
APC-R. This case report also points out that the presence of APC-R should not delay the search for other causes in patients with cerebral venous thrombosis. The detection of single or multiple inherited coagulation defects has major practical consequences for the secondary prevention of these patients and for the primary prevention of the other family members, as these abnormalities usually have an autosomal dominant inheritance pattern. Lifelong management to prevent further venous thrombotic events in patients with isolated APC-R is not well delineated up to now, particularly regarding the duration of anticoagulation. Patients with combinations of APC-R and protein C or protein S deficiency have a higher risk of thrombosis than those with APC-R alone.1,1 Lifelong oral anticoagulation may be advocated in patients with cerebral venous thrombosis and multiple inherited coagulation defects, such as protein C deficiency and APC-R.

LAURENT DEREX
FREDERIC PHILIPPE
NORBERT NIGHOGHSSIAN
PAUL TROUILLAS
Service d’Urgences Neurovasculaires, Hôpital Neurologique, Lyon, France

MICHELINE BERRUYER
Laboratoire Central d’Hématologie et de Cytogénétique, Hôpital Cardio-Vasculaire et Pneumologique, Lyon, France

Correspondence to: Dr Laurent Derex, Service d’Urgences Neurovasculaires, Fr Paul Trouillas, Hôpital Neurologique, 59 Boulevard Pinel, 69003 Lyon, France


Intravenous immunoglobulin dependent inflammatory radiculopathy presenting as lumbar canal stenosis

A patient with symptoms and signs of lumbar canal stenosis showed non-malignant, nerve root hypertrophy on MRI. The patient responded dramatically but temporarily to intravenous immunoglobulin (IVIg).

Hypertrophy of nerve roots is recognised as a cause of “spinal stenosis” syndrome.1 The association has been previously described with hereditary causes such as neurofibromatosis, Refsum’s disease, and hereditary motor and sensory neuropathy (HSMN) type 1 and 3.2 There have been recent reports of chronic inflammatory demyelinating polyneuropathy (CIDP) presenting as a spinal stenosis syndrome.1,2 We report on a patient with an acquired inflammatory radiculopathy who presented with a lumbar canal stenosis syndrome only responsive to IVIg.

A 60 year old white man presented with a 2 year history of progressive numbness and stiffness of his legs with difficulty in walking. He had noticed the discomfort in his legs to be exacerbated after walking 100 metres and relieved by rest. He could cycle long distances without discomfort. He specifically denied any upper limb symptoms or sphincter disturbance. He had mild hypertension treated with nifedipine. There was no relevant family history.

Examination of the cranial nerves and upper limbs was normal. In the lower limbs there was symmetric wasting of the extensor digitorum brevis with grade 4/5 weakness of ankle dorsiflexion, eversion, and big toe extensors. Deep tendon reflexes were absent in the lower limbs. The plantar responses were flexor. Sensory examination disclosed pin prick, soft touch, and vibration sense to be reduced to knee level. Joint position sense was preserved.

Routine haematological and biochemical investigations were normal and there was no serum paraprotein. Prostate specific antigen, vitamin B12, venereal laboratory research test, and HIV serology were negative. Enzyme linked immunosorbent assay (ELISA) IgG for Borrelia was negative. Plain radiology of the chest and lumbar sacral spine was normal. Initial CSF examination disclosed a raised protein of 2.08 g/l, glucose 3.7 mmol/l, and a white cell count of 40 cells/mm³ (75% reactive lymphocytes). Cytology was normal. CSF angiotensin converting enzyme concentration was normal and acid fast bacilli culture negative. Nerve conduction studies and EMG showed dispersed F wave responses (right common peroneal 49–88 ms and right posterior tibial nerve 45–80 ms) together with denervation in the right tibialis anterior. Motor conduction velocities in the limbs were normal with preserved sensory action potentials. Lumbar spine MRI showed poorly defined hypertrophic nerve roots (figure).

On exploration of the intradural contents, the roots were “matted together” and not free floating in the CSF. Biopsies were taken of the ligamentum flavum, dura, and arachnoid, and this showed a mixed population of inflammatory cells in the ligamentum flavum sections. Examination of CSF on this occasion showed the same protein concentration of 2.08 g/l and a white cell count of 2 cells/mm³.

Oral prednisolone (40 mg/day) caused a deterioration in his symptoms. The patient was given a 5 day course of IVIg (0.4 g/kg/day) and made a dramatic recovery and within 3 days the motor and sensory examination was normal but the knee and ankle jerks were still absent. Treatment with IVIg provides clinical benefit lasting 2–3 weeks, and neither this pattern nor the dose of IVIg prescribed has been altered by giving azathioprine, cyclophosphamide, or cyclosporin.

The patient presented with an acquired, non-malignant, root hypertrophy. There are electrical features to suggest this may be a CIDP, although the reactive CSF showed a higher white cell count than usual,1,2 subsequent CSF white cell counts have been normal. Unlike previous cases of CIDP the root hypertrophy described in our patient has

![MRI of the lumbar spine showing ill defined hypertrophic nerve roots (black arrow).](http://jnnp.bmj.com/content/802/7/608/F1)
shown no benefit after oral immunosuppres- 
sion. Indeed, introduction of a moderately 
high dose of prednisolone caused a dramatic 
deterioration, a response that is recognised in 
CIDP.1 2 Unlike the modest clinical benefit 
seen by others after the administration of IVIg, 
our patient remains exquisitely sensitive to 
IVIg.

C E M MILLER 
J G LLEWELYN 
M D HOURIHAN

Deartment of Neurology and Neuroradiology, 
University Hospital of Wales and University of 
Wales College of Medicine, Heath Park, Cardiff, UK

Correspondence to: Dr CEM Miller, Department of 
Neurology and Neuroradiology, University Hospital of 
Wales, Heath Park, Cardiff CF4 4XW, UK. Telephone 0044 1222 
746441; fax 0044 1222 741466.

1 Ginsberg L, Platts AD, Thomas PK. Chronic 
5 Ad Hoc Subcommittee. Research criteria for diagnosis of chronic inflammatory demyelinating neuropathy (CIDP). Neurology 1991;41: 
617–18.

Sodium valproate for tinnitus

In 1935 Barany serendipitously discovered the temporary relief of tinnitus after ligno-
caine injection of nasal turbinates.1 Since that time, other agents known to suppress the activity of excitable membranes have been tried, including antihistaminic and anti-
convulsant drugs. Among such drugs, car-
bamazepine has the best documented efficacy in the treatment of tinnitus,2 but is generally unhelpful in unselected tinnitus populations and often discontinued due to adverse effects.3 A 53 year old man with viral cardionyopa-
thy developed severe (60dB) tinnitus after bilateral temporal lobe strokes. Various treat-
ments including masking and diazepam were unhelpful. Carbamazepine (200 mg nightly) was effective, but was withdrawn due to pro-
gressive hyponatraemia (120mM after two weeks of therapy), followed by the rapid recurrence of tinnitus. Sodium valproate (200 mg twice daily) was also promptly effect-
ive in suppressing tinnitus, and was well toler-
ated until his death due to cardiac arrhyth-
mia one month later.

In part due to its diverse aetiology, pharmacotherapy of tinnitus has met with very limited success.4 Uncontrolled trials in the French5 and Japanese6 literature have indicated benefit from sodium valproate in selected patients, but its use seems not to have been described in English apart from a specialist monograph.7 Tinnitus loudness and sensory neural pathy but not ligno-
caine response seem to predict response. Valproate may also differ from car-
bamazepine in that it seems better tolerated in an unselected tinnitus population.8 Con-
trolled studies of valproate for this common, often debilitating condition seem warranted.

DAVID B MENKES
PAUL M LARSON
Dundee University, New Zealand

Correspondence to: David B Menkes, PO Box 913, Dundee, New Zealand. Email david.menkes@ 
stonebow.dtu.ac.uk

3 Mansbach A-L, Freyens P. Acouphenes: don-
nees actuelles et traitement par valproate de sodium. Acta Otorhinolaryngol Belg 1983:67: 
697–705.
4 Vesterager V. Tinnitus—investigation and man-
5 Manabe Y, Sato T, Sakashita T, et al. Treatment of tinnitus based on lidocaine test and tinnitus loudness. Practica Otologica Kyoto Suppl 1993: 

Audible carotid dissection

Carotid dissection is a common cause of stroke in the young and can present with various clinical syndromes or symptoms. These may include stroke or transient ischae-
mic attack,2 ipsilateral ptosis, isolated or mul-
tiple cranial nerve palsies,3 carotidynia,4 hemicrania,5 scintillating scotomata, pulsatile 
tinnitus, or subjective bruit.6 I recently cared for a man who experienced an audible “creaking” sound heard even by his wife in the hours before a right middle carotid artery (MCA) infarct secondary to carotid dissec-
tion. I think that this sound represented the actual dissection.

A forty three year old, right handed lawyer with a presynucleal pharyngitis and severe cough for two weeks duration returned from work at 600 pm and began hearing periodic, high frequency, “creaking” sounds in his right ear. These sounds occurred every 1–2 hours lasting a few seconds each time. These sounds were not pulsatile or rhythmic. He had not experienced these sounds previously with his illness. When sitting at the dinner table, his wife too heard these peculiar sounds. On examination, he provided a detailed description of these sounds as the patient himself was lethargic. At midnight, he experienced a scintillating scotoma with right retroocular visual field loss by 1:30 am was lethargic with a left sided weakness. According to the wife and the patient these sounds had now ceased and did not recur.

On examination he had diminished atten-
tion, and mental status was otherwise normal. Neck auscultation was normal and there was no audible creaking sound. Fundus and visual fields were normal. A left lower facial droop was present with otherwise normal cranial nerve function. Left arm weakness and left leg paresis was present with associated hyper-
reflexia and extensor plantar response. He reported diminished sensation to pin, posi-
tion, and light touch and extinguished left sided touch on the palmar and plantar surfaces. Brain MRI at 12 hours showed a T1 hypointense and T2 hyperintense lesion in the anterior MCA distribution, thrombus in the MCA (M-1 segment), with a suggestion of focal narrowing in the upper cervical region of the right internal carotid on MRA. An angio-
gram confirmed a right internal carotid dissection and MCA thrombus. The patient was anticoagulated and with rehabilitation is ambulatory with partial use of his left arm.

Gynaecomasia in association with phenytoin and zonisamide in a patient having a CYP2C subfamily mutation

Anticonvulsant drugs can have various side effects on endocrine functions, such as impotence, hirsutism, infertility, and thyroid dysfunction. Gynaecomasia is caused by many types of drugs such as methyldopa, tricyclic antidepressant drugs, isoniazid, and spironolactone,1 but there have been only a few reports of gynaecomasia caused by anti-
convulsant drugs, including phenytoin2 and zonisamide.3 We recently encountered a young man with partial seizures, who
generically had a heterozygous mutation of both CYP2C9 and CYP2C19, normally responsible for biotransformation of phenytoin in the human liver microsomal P-450 system. He developed gynaecomastia after increasing the dose of phenytoin.

The patient was an 18-year-old boy, an 18-year-old man with a diagnosis of left parietofrontal lobe epilepsy since the age of 2 years, until which time his developmental milestones were normal. He had complex partial seizures occurring at least once every day despite various anticonvulsant drugs of usually sufficient dose including clorazepate, phenytoin, and carbamazepine, and zonisamide since the age of 9 years, when he had an epilepsy surgery for a focal, high intensity abnormality on T2-weighted MRI in the left parieto-occipital area. His seizures were not controlled. At the age of 18 years, the patient had chronic implantation of subdural electrodes for evaluating the intractable seizures, and based on the results of the invasive evaluation, he had focal resection in the left parietal and mesial parietocoronal areas. After surgery his seizures decreased in frequency to once every week, and he became 90% seizure-free, accompanied by a loss of awareness in only a third. Before and after the surgery he was taking phenytoin (175 mg/day), carbamazepine (300 mg/day), and zonisamide (400 mg/day) giving blood concentrations of 13 mg/l, 7.9 mg/l, and 13.2 mg/l, respectively. Two months after surgery, partly because of sleep deprivation, the patient had a cluster of complex partial seizures, some resulting in secondary generalised seizures, occurring seven times in 90 minutes. After a total of 375 mg phenytoin was intravenously loaded, his seizures were well controlled. Subsequently the medication was maintained at phenytoin (190 mg/day), carbamazepine (1100 mg/day), and zonisamide (400 mg/day), and the steady state, basal blood concentrations of these anticonvulsant drugs were 16.6 mg/l, 5.2 mg/l, and 9.7 mg/l, respectively.

About a month later, the patient noticed bilateral enlargement of his breasts associated with some tenderness restricted to the centre of the breast. The size was about 3 cm in diameter. No tumour or overrecession was seen. Blood concentrations of luteinising hormone, follicle stimulating hormone, prolactin, and oestradiol were all normal. His liver function was also within normal limits except for slight increases in γ-GTP. The patient had developed gingival hypertrophy and mild hypertrichosis. He was taking no medication other than the anticonvulsants.

On rare occasions gynaecomastia has been reported in patients taking phenytoin, and in some of them it disappeared after stopping phenytoin. The mechanism as to how gynaecomastia is caused by this drug is uncertain. It was pointed out that some drugs interfere with testosterone synthesis as well as by way of blocking the cytosol receptor of androgen in target tissues. It has been shown that liver enzyme inducing antiepileptic drugs such as carbamazepine and phenytoin are associated with increased productions of serum sex hormone binding globulin and decreased amounts of free androgen available in the tissue. However, for most drugs causing gynaecomastia, its mechanism is not well defined.

The anticonvulsant drug zonisamide has been clinically available in Japan for patients with seizures since 1989, and it is reported that three patients (age ranging from 3 years to 32 years) taking zonisamide (60 to 600 mg/day) developed gynaecomastia from 10 days to 1 year after starting zonisamide, and in all of them it disappeared after stopping zonisamide. Therefore, in the present patient, it is possible that both phenytoin and zonisamide played important parts in the development of gynaecomastia. However, the patient had been on zonisamide for the past 8 years, and its dosage was not increased before gynaecomastia developed. On the other hand, after phenytoin was loaded when he had frequent seizures, its maintenance dosage increased. Therefore, phenytoin is most likely a causative agent. Adolescent gynaecomastia usually occurs bilaterally, although asymmetric, in many boys as a physiological type, often during puberty with a mean onset age of 14 years, and it is correlated with transiently raised oestradiol of unknown origin before completion of puberty. However, there was no increase in oestradiol in the present patient.

The patient had a heterozygous point mutation in the defective allele of CYP2C9, and in such patients Vmax values in the pharmacokinetics of phenytoin were 40% lower than those in patients with a normal type CYP2C subfamily (see subject No 45 in fig 1 of Odani et al) and thus our patient’s maintenance dose of phenytoin (175 mg/day) was relatively small to achieve an appropriate blood concentration (14.3 mg/l). After having increased the maintenance dose of phenytoin by 15 mg/day, the blood concentration was kept <20 mg/l, and clinically he did not show toxic symptoms such as cerebellar ataxia and gaze evoked nystagmus. The presence of a heterozygous CYP2C subfamily mutation might have played some additional part in developing this rare side effect or in facilitating physiological gynaecomastia of the adolescent type.

This study was supported by Grants-in-Aid for Scientific Research (A) 09380831, (A) 08580803, and (C) 10670583 from the Japan Ministry of Education, Science, Sports and Culture, and a research grant for intractable epilepsy from the Japan Ministry of Health and Welfare.

AKIO IKEDA
Department of Brain Pathophysiology
HARUO HATTORI
Department of Pediatrics
ATSUKO ODANI
Department of Pharmacy
JUN KIMURA
Department of Neurology
HIROSHI SHIBASAKI
Department of Brain Pathophysiology, Kyoto University School of Medicine, Shogoin, Sakyo-ku, Kyoto, 606, Japan

Correspondence to: Dr Akio Ikeda, Department of Brain Pathophysiology, Kyoto University School of Medicine, Shogoin, Sakyo-ku, Kyoto, 606, Japan.

The authors reply:

We appreciate the interest of Hughes et al in our study on the neuropsychological prediction of dementia in Parkinson’s disease. The validity of the diagnosis of dementia was discussed in the paper. Hughes et al conjecture that the knowledge of initial neuropsychological tests scores would raise an observer bias. But the final diagnosis (dementia or not) was made by independent raters in 232 cases out of 86, and in every case we used a special case report form which did not even refer to the initial neuropsychological examination.

We agree that the incidence of dementia in our population cannot be interpreted as that of an inpopulation epidemiological study. We mentioned it as a mere additional descriptive characteristic.

We agree that the significance of the completion subtest was borderline in the multivariate analysis, as the confidence interval of the relative risk includes one, according to the calculation done by BMDP software. The level of significance is $<0.05$, as mentioned in the table, again according to the test performed by the BMDP package. This minor discrepancy is not unusual and should not affect the interpretation of our results in such an exploratory clinical epidemiological study.

With regard to age and age at onset, these two variables correlate strongly. The two have been reported as predictive factors for dementia in Parkinson’s disease. $^4$ Some authors consider that age at onset of Parkinson’s disease is a potentially more important variable. $^5$ Moreover, the inclusion of age instead of age at onset in the multivariate analysis modifies the results only slightly. The WAIS-R completion subtest and age remain significant and independent predictors of dementia with respective risk ratios of 3.344 for age $>68$ years (95% CI 1.7–17.1; $p<0.01$) and 10.929 for a completion score $<10$ (95% CI 2.5–48.3).

MARTIN NOGUES
Department of Clinical Neuropsychology, PLENI, Montevideo 2325 (1428) Buenos Aires, Argentina

Repeated syncopes and extended paediatric hydroxyringomyelia/Chiari I malformation

I have read with interest the letter recently published on Repeated syncopes and extended paediatric hydroxyringomyelia/Chiari I malformation. $^1$ The case reported is quite unusual, as syncope seems to be a very rare manifestation of this condition, even when associated with a Chiari malformation, at least in adults. From a series of 100 adult patients with syringomyelia diagnosed over the past 10 years at this institute, two thirds of whom had an associated Chiari type I anomaly, only one patient had syncope, which was triggered by sneezing. Another three patients had drop attacks without loss of consciousness, one with a Chiari type I anomaly, and two with an associated ventricular dilation (figure).

In the paper mentioned by the authors on cardiovascular reflexes in syringomyelia, $^2$ all patients with autonomic involvement, even those without syringobulbia, had an abnormal neurological examination, and sweating abnormalities and Horner’s syndrome were often encountered. The fact that neurological examination was normal in the patient described by Woelfle et al, $^3$ as well as the absence of Horner syndrome and sweating abnormalities, make autonomic dysfunction an unlikely explanation for the episodes described, although it may be a contributing factor. As syncope was associated with occipital headaches, transient brainstem compression may play a significant part. As the authors suggest, the precise mechanism of these episodes and of drop attacks is difficult to determine, and they may be multifactorial.


MRI of a 50 year old woman with syringomyelia, Noonan’s syndrome, and frequent drop attacks. There is a cerebellar cortex and moderate dilatation of the fourth ventricle. There is no Chiari anomaly.
In the first edition of this text in 1992 Professor Aicardi argues for the continuing role of clinical history and examination in the face of rapidly advancing techniques of neuroimaging and neurophysiology. In his preface to the second edition he describes the need for someone to marshall and make sense of the deluge of information available in a world in which only computerised networks can keep pace with new developments and new data in medical sciences. The experience in the field of childhood neurology of Professor Aicardi and his co-authors enables the organisation of this volume of information, and the direction of the reader to the most important and significant data. This second edition succeeds in its aim to incorporate the major developments of the past 6 years and provide an access route to further information in the literature, while retaining the overall outline of the first edition. The book is primarily clinically oriented, comprehensively describing the neurological diseases of childhood in sufficient detail to enable diagnosis, prognosis, and management. It will be of value to all physicians with an interest in childhood neurological disorders, including general paediatricians, neuropsychiatrists, and other physicians interested in developmental medicine. The book is divided into 11 main sections, covering childhood neurology from fetal development through to developmental and neuropsychiatric disorders of older children and adolescents. This last section is written by Professor Gillberg from Göteborg and isprefixed by a succinct and very useful chapter on normal mental and behavioural development. For the section on cerebral palsy Professor Aicardi is joined by Martin Bax and they note the lack of longitudinal data on the natural history of cerebral palsy, unfortunately a common problem in paediatric neurological disorders, together with a lack of randomised controlled trials of therapies. The chapter on metabolic diseases is co-written with Helene Ogier from Paris and this area of increasing importance is very clearly presented and illustrated. Otherwise the book is single author and extensively referenced, with an emphasis on recent articles. It is well illustrated, particularly with high quality neuroimaging reproductions. The numerous tables are comprehensive and of great practical use to the physician attempting to construct a differential for obscure diagnoses. This book is one of the best of its kind and, as with the first edition, will continue to take first place on the bookshelf of all paediatric neurologists. It is also highly readable and will remain the “friendly companion even at the bedside” that Professor Aicardi aims for, a role not yet overtaken by computer technology.

LOUISE HARTLEY


This book is well known to many neurologists and represents the foremost book for this area of neurological practice. The latest edition is dedicated to the life and accomplishments of the late professor Anita Harding and serves as a very fitting tribute to this remarkable neurologist.

The book is divided into 23 sections and contains nearly 1500 pages of text in the form of 77 chapters. It is therefore impossible to do justice to a book of this nature in a short book review, but for detail and clarity, there are few books to compete with this tome. The book opens with an account of some of the more general issues in genetics which is especially helpful for the non-specialist as it helps explain the approach in tackling neurological disorders from a genetic point of view. Indeed, this ultimately is the problem with a book of this type, in that the field moves forward with such speed that chapters soon become out of date. For example, the chapter on Huntington’s disease does not discuss recent animal models of this disease using expanded CAG repeats and the significance of intranuclear inclusions.

Furthermore, whereas the expanded triplet repeat in Friedreich’s ataxia is well discussed, no details are presented on the role of frataxin. These, however, are minor points in what is clearly an excellent reference book. Each condition is concisely documented with good illustrations and up to date reference lists, and thus readers can easily remind themselves about various conditions. There are occasional omissions—for example Pelizaeus-Merzbacher disease does not appear in this book even though the genetics of this condition are now becoming clearer.

This book is a must for all libraries but from an individual point of view it may seem extravagant to own a copy especially in a field that moves so fast. Specialists in neurological genetics would probably want to own a copy and update it manually as new information emerges. For the more general neurologist, a large neurological textbook probably offers a better buy as it covers all the conditions in this book and more. However, I loved this book and enjoyed dipping into it to catch up on the latest developments in the ever increasing array of neurogenetic disorders. Although many of these disorders are still rare, it seems that more and more neurological conditions will be found to have a genetic basis, and so an understanding of genetics, especially at the molecular level, will be an essential part of any neurologist’s training. So why not start now with this book?

ROGER BARKER


The continuing rapid expansion of neuroradiology, with new techniques and improvements in more well established techniques, have sharpened the tools with which to examine neurological and psychiatric diseases of old age. Investigation of, for example, the MRI findings in vascular dementia and depression, measurements of medial temporal lobe structures in Alzheimer’s disease, and functional imaging studies of schizophrenia, have led to new insights into diagnosis, prognosis, and symptomatology of these ill understood diseases. However, there are two obstacles in the understanding of this expanding area of research for the interested neurologist and psychiatrist—mainly, understanding of the basis of the technology and relating the research findings to best clinical practice. The remit of this text covers both these deficiencies.

For the non-physician getting to grips with the basic principles and methodologies of neuroimaging can be daunting. The first section of this book explains the basic principles behind the hardware of the imaging department and this is aided by many excellent diagrams. The general clinical indications and safety issues of structural (CT and MRI) and functional (PET, SPECT and EEG) imaging techniques are well reviewed and illustrated.

The second section of the text explores the research questions and summarises the answers so far in the field of old age psychiatry. Interpretation of imaging research in abnormal elderly patients, with regard to subject selection, imaging technique and the relation to normal age, is one of the main dilemmas in this field. This is fully discussed in the admirable chapter on normal aging, which commences this review of the research. Other chapters on Alzheimer’s disease, vascular dementia, other dementias, delirium, affective disorders, and schizophrenia of late onset continue this well referenced text. Besides presenting the data for the clinician, this comprehensive review will also be appreciated by researchers in this field.

The third part of this book returns to the application of these results to clinical practice. Both an American and European perspective on the clinical interpretations of the above data are presented and the conclusion can be quoted “Our ability to image the brain, however, has in some cases outpaced our ability to understand the clinical implications of the structural and functional findings seen using modern imaging techniques”. In other words this interesting research which has been so excellently summarised here has not yet to make an real impact on routine clinical practice.

CLARE GALTON
Cerebellar ataxia due to lead encephalopathy in an adult

JAYANTI MANI, NALIN CHAUDHARY, MAKARAND KANJALKAR and PRAVINA U SHAH

J Neurol Neurosurg Psychiatry 1998 65: 797-798
doi: 10.1136/jnnp.65.5.797

Updated information and services can be found at:
http://jnnp.bmj.com/content/65/5/797

These include:

References
This article cites 5 articles, 1 of which you can access for free at:
http://jnnp.bmj.com/content/65/5/797#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/