record a proven case of SSPE in a 15-year-old boy who deteriorated for 20 months and then made an impressive spontaneous improvement which had lasted for 3½ years at the time of their report. Vandvik *et al*¹⁴ suggested treatment with transfer factor, but the results have not been encouraging.¹⁵⁻¹⁸ The patient reported here received this treatment but continued to deteriorate for 21 months afterwards, and it seems unlikely to have contributed to the long-term survival. There have been reports of remission occurring following treatment with isoprinosine^{19,20} and with amantadine.^{21,22} Huttenlocker *et al*²⁰ reported sustained remission with clinical improvement for up to six years in six patients treated with isoprinosine, and Robertson *et al*²² remission and mild improvement for one to seven years in four out of eight patients treated with amantadine.

There is no doubt that this patient will remain permanently severely disabled. However, the mild but sustained clinical improvement over the past five years, the disappearance of generalised repetitive complexes from the EEG, and the dramatic fall in serum measles antibody titre all suggest that the disease is not just in remission, but that the pathological process has ceased. The possibility of this event occurring spontaneously must be borne in mind when assessing the therapeutic effects of any new treatment for this disorder.

CR BARRACLOUGH
The Midland Centre for
Neurosurgery and Neurology,
Smethwick,
West Midlands, UK

References

- Freeman JM. The clinical spectrum and early diagnosis of Dawson's encephalitis: with preliminary notes on treatment. *J Pediatr* 1969;**75**:590-603.
- Haddad FS, Risk WS, Jabbour JT. Subacute sclerosing panencephalitis in the Middle East: Report of 99 cases. *Ann Neurol* 1977;**1**:211-7.
- Risk WS, Haddad FS. The variable natural history of subacute sclerosing panencephalitis. *Arch Neurol* 1979;**36**:610-4.
- Eicke WJ, Ziegler W. Ein Beitrag zur Frage der atypischen Encephalitis. *Nervenarzt* 1950;**21**:312-8.
- Malamud N, Haymaker W, Pinkerton H. Inclusion encephalitis with a clinico-pathologic report of three cases. *Am J Pathol* 1950;**26**:133.
- Krucke W. Über eine besondere Form der spontanen Encephalitis. *Nervenarzt* 1957;**28**:289-301.
- Landau WM, Luse SA. Relapsing inclusion encephalitis (Dawson type) of eight years duration. *Neurology (Minneapolis)* 1958;**8**:669-76.
- Osetowska E, Torck P. Subacute sclerosing leucoencephalitis. *World Neurol* 1962;**3**:566-79.
- Kurtzke JF. Inclusion body encephalitis: a non fatal case. *Neurology (Minneapolis)* 1956;**6**:371-6.
- Simpson JA. Subacute inclusion-body encephalitis: A possible association with infective hepatitis. *Lancet* 1961;**2**:685-7.
- Pearce JMS, Barwick DD. Recovery from presumed subacute inclusion-body encephalitis. *Br Med J* 1964;**2**:611-3.
- Cobb WA, Morgan-Hughes JA. Non-fatal subacute sclerosing leucoencephalitis. *J Neurol Neurosurg Psychiatry* 1968;**31**:115-23.
- Resnick JS, Engel WK, Sever JL. Subacute sclerosing panencephalitis: spontaneous improvement in a patient with elevated measles antibody in blood and spinal fluid. *N Engl J Med* 1968;**279**:126-9.
- Vandvik B, Frøland SS, Høyeraal HM, Stien R, Degre M. Immunological features in a case of subacute sclerosing panencephalitis treated with transfer factor. *Scand J Immunol* 1973;**2**:367-74.
- Meulen V ter, Grob PJ, Kreth WH, Kackell YM, Kibler R. Transfer factor therapy in patients with SSPE (Abst). *Arch Neurol* 1975;**32**:501.
- Blaese RM, Hofstrand H, Krebs H, Sever J. Evaluation of transfer factor in the therapy of SSPE (Abst). *Arch Neurol* 1975;**32**:502.
- Valdimarsson H, Agnarsdottir G, Lachmann PJ. The treatment of SSPE with transfer factor (Abst). *Arch Neurol* 1975;**32**:503.
- Pabst HF, Cumming JDA. Simultaneous immunotherapy of two cases of SSPE (Abst). *Arch Neurol* 1975;**32**:503-4.
- Streletz LJ, Cracco J. The effect of isoprinosine in subacute sclerosing panencephalitis (SSPE). *Ann Neurol* 1977;**1**:183-4.
- Huttenlocker PR, Mattson RH. Isoprinosine in subacute sclerosing panencephalitis. *Neurology (Minneapolis)* 1979;**29**:763-71.
- Haslam RHA, McQuillen MP, Clark DB. Amantadine therapy in subacute sclerosing panencephalitis: a preliminary report. *Neurology (Minneapolis)* 1969;**19**:1080-6.
- Robertson WC, Clark DB, Markesbery WR. Review of 38 cases of subacute sclerosing panencephalitis: Effect of Amantadine on the natural course of the disease. *Ann Neurol* 1980;**8**:422-5.

Thrombosis of cerebral venous sinuses due to a catheter for parenteral nutrition

Sir: Venous thrombosis in patients with an indwelling venous catheter is a well known iatrogenic condition. We report a case of cerebral venous sinuses thrombosis in a patient receiving parenteral nutrition.

In March 1979, after a subtotal intestinal resection for mesenteric infarction, a 41-

year-old patient was fed by chronic parenteral nutrition using a silicone catheter implanted into the right external jugular vein and entering into the superior vena cava; the other end of the catheter was inserted under the skin along a distance of 10 cm. In October, the catheter was removed after inflammatory signs had appeared suggesting a right external jugular vein thrombosis. The patient successfully underwent treatment for eleven days with heparin and ampicillin. Intra-venous heparin subsequently was replaced by sub-cutaneous calcium heparinate (5000 units every 12 hours). On November 3rd, the patient was re-admitted to the emergency ward with violent headache, behavioural changes and progressive somnolence. He soon developed a left hemiparesis and hemi-hypoesthesia including the face. He then developed partial motor status epilepticus involving the face and the left upper limb. Intra-venous diazepam and phenytoin therapy was unsuccessful. Lumbar puncture at the admission was normal. EEG showed a slowed background rhythm with a high voltage delta rolandic focus in the right hemisphere. A right carotid arteriography showed, on the late films, the absence of opacity of the superior longitudinal sinus and of the right lateral sinus. There was no sign of collateral venous pathways. He was treated with pentothal infusion and fibrinolysis with streptokinase for 36 hours. Subsequently he recovered and a repeat angiogram was normal.

Venous thrombosis may be an iatrogenic condition in patients with indwelling catheters. Subclavian vein thrombosis were reported in three patients in a series of 770 cardiac stimulator implantations¹ and in one patient of another series of 298 implantations.² Cerebral venous thrombosis is much less frequent, since it was reported in only two patients who underwent cardiac pacing.^{3,4} Chronic parenteral nutrition which requires indwelling catheters for several months may constitute a similar potential risk. To our knowledge, however, the case we report is the first instance of cerebral venous thrombosis complicating chronic parenteral nutrition.

I VAN DE MORTEL
C DEGAUQUE
Hôpital Civil de Verviers,
Rue du Parc 29,
B-4800 Verviers, Belgium.
A THIBAUT
Department of Neuro-Radiology,
Hôpital de Bavière,
Boulevard de la Constitution 66,
B-4020 Liege, Belgium.

References

- ¹ Grogler FM, Frank G, Greven G *et al.* Complications of permanent transvenous cardiac pacing. *J Thorac Cardiovasc Surg* 1975;69:895-904.
- ² Seremetis MG, de Guzman VC, Lyons WS, Peabody JW. Cardiac pacemakers: clinical experience with 289 patients. *Am Heart J* 1973;85:739-48.
- ³ Floyd WL, Mahaley MS. Cerebral dural venous sinus thrombosis following cardiac pacemaker implantation. *Arch Intern Med* 1969;124:368-72.
- ⁴ Girard DE, Reuler JB, Mayer BS, Nardone DA, Jendrzewski J. Cerebral venous sinus thrombosis due to indwelling transvenous pacemaker catheter. *Arch Neurol* 1980;37:113-4.

Accepted 18 December 1982.

A case of blindness following carbon monoxide poisoning, treated with dopamine

Sir: Severe neurological sequelae are common after carbon monoxide poisoning and frequently have a bad prognosis. We report a case of carbon monoxide poisoning that resulted in visual failure, treated with an intravenous infusion of dopamine to increase the systemic arterial pressure.

A 28-year-old man was exposed to carbon monoxide accidentally. He was rescued rapidly, but was unconscious for 10 minutes. On regaining consciousness he could not see. When admitted to the local hospital he was confused and disorientated, but obeyed commands. Visual acuity was reduced to perception of light. There were no other focal signs. Treatment consisted of 60% oxygen by mask, 500 ml. of 20% mannitol and 40 mg frusemide intravenously. COHb level 4 hours after exposure was 26%, indicating an initial level of about 50%. Eight hours after exposure he was orientated and able to count fingers. At 20 hours he could read a newspaper and was discharged "well" at 24 hours, with a retrograde amnesia of 10 minutes and a post-accident amnesia of 4 hours. He remained well until 5 days after the accident when, over several hours, he developed bitemporal headache, photophobia and blurring of vision. The next day he was unable to see beyond perception of light. He was re-admitted to hospital and examination was normal, apart from reduction in visual acuity to perception of light.

A history was obtained at that time of episodes of migraine in childhood with headache, photophobia and blindness lasting up to 2 hours. The last episode of this had occurred 13 years previously. There had been no improvement 48 hours later and he was transferred to the Royal Hallamshire Hospital, Sheffield. Examination was normal apart from reduction of visual acuity to perception of light. A CT scan and CSF were normal. An EEG showed almost continuous delta activity with absence of fast activity in the posterior quadrants and of photic following responses. A regional cerebral blood flow study demonstrated reduced flow to both temporal areas (on right 17 ml/min/100 g and on left 10 ml/min/100 g).

Our hypothesis was that the patient had a pre-existing critical circulation to the visual cortex, or association areas, as indicated by the history of migraine with associated episodes of blindness, and that the tissue hypoxia caused by the carbon monoxide was sufficient to cause loss of function of this region of the brain. It was thought that cerebral vascular autoregulation was likely to be impaired in the damaged area of the brain and that by increasing the systemic arterial pressure the perfusion of this part of the brain could be improved.

The systemic blood pressure was increased from 130/80 to 180/85 mm Hg by an infusion of dopamine. A dose of 17 μ g/kg/min was necessary to maintain this pressure. Arterial pressure, central venous pressure and urine output were continually monitored. After receiving the dopamine infusion for 6 hours the patient could read the top line of a Snellen Chart held at 70 cm; at 12 hours he had 6/60 vision, and at 36 hours had 6/6 vision with normal visual fields. The dopamine was then reduced over 12 hours with no loss of visual acuity. The only visual deficit was of some difficulty with scanning and with performing a Friedman's field analysis. Subsequent EEGs showed a gradual return to normal with reappearance of alpha rhythm and photic following responses over the succeeding days, and at follow-up a month later he was well.

Patients who have been exposed to carbon monoxide may apparently recover from the poisoning and then develop neurological problems such as fits, cortical blindness, ataxia, apraxia, dysphasia and intellectual impairment¹ in the next few weeks. The severity may fluctuate over the succeeding weeks and then gradual improvement may occur over 3 years. A

follow-up study showed that 3 years after exposure to carbon monoxide, 43% of patients still had significant memory impairment and 33% had a change in personality.²

The mechanism of late neurological sequelae to carbon monoxide poisoning is uncertain. It is possible that there is an increase in vascular resistance, perhaps caused by a build-up of toxic metabolites in the hypoxic brain, or by cellular swelling, resulting in obstruction of small vessels. This phenomenon was called the "No Re-flow" by Ames *et al.*³ in 1968. In essence it was found that in areas of rabbit brain that had been made temporarily ischaemic, subsequent capillary flow was reduced, despite restoration of normal perfusion pressure. Electron microscopy on these brains⁴ showed cellular swelling of perivascular glial cells and the presence of cellular blebs arising from the capillary endothelium. The conclusion was that these two factors caused partial capillary obstruction which was compounded by locally increased blood viscosity caused by influx of intravascular fluid into the cells as a result of failure of the "sodium pump".

If the raising of the blood pressure to 180/85 mm Hg in our patient did aid a return to normal function of the visual cortex, it is possible that it did so by increasing capillary perfusion and thus re-establishing normal blood flow which, once re-established, remained normal when the perfusion pressure was lowered to normal.

In a similar case reported by Garland and Pearce in 1967,¹ a 21-year-old man was exposed to carbon monoxide and apparently recovered, but on the fifth day after exposure, was re-admitted with visual acuity reduced to perception of hand movements. In this case the visual acuity gradually returned over a four week period with no specific therapy.

We suggest the consideration of dopamine treatment in further cases of carbon monoxide poisoning with neurological sequelae. Obviously this treatment must not distract attention from the acute treatment with immediate 100% oxygen, if possible hyperbaric, until the carbon monoxide is cleared from the body. Measures to control raised intra-cranial pressure should also be instituted.

JOHN S DUNCAN
JOHN GUMPERT

*The Department of Neurology,
Royal Hallamshire Hospital,
Sheffield, S10 2JF, UK*