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 disoriented, 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 oriented 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 µg/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 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 impairment 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.

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 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 of these brains showed cellular swelling and 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 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, 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.

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Morbidity in patients with intracranial gliomas

Sir: In 1980 the factors affecting mortality in patients with cerebral gliomas were considered. Local prognostic factors which were found to be associated with longer survival from first symptoms, a younger age, epilepsy as a presenting feature, a grade of glioma less than IV (glioblastoma multiforme), partial removal of the tumour, radiotherapy and chemotherapy. Although survival is important, the quality of life is equally, if not more so. A problem is to decide whether the benefits of active investigation and treatment (operation, radiotherapy and chemotherapy) outweigh the discomfort, morbidity and mortality of the procedures. Many patients near death would prefer, if possible, to be at home rather than spend their last few weeks in hospital. We studied the morbidity of the disease in 110 patients with histologically proven gliomas who attended between 1961 and 1977. One hundred and four patients were followed up until 1981 or until death, but six were still alive when last seen and are now lost to follow up. Of these 110, 42 had Grade III, 39 Grade IV and 18 Grade I and II astrocytomas. The remaining 11 had other gliomas. This population of patients is heterogeneous in respect of possible prognostic factors, but we think represents a typical population of patients with gliomas.

Morbidity was assessed retrospectively until 1976 and then prospectively. A simplified rating scale based on that of Karnofsky and Burchenal was used. The categories used were full function (Scale 90-100), independent (70-80), dependent but at home (40-60) or in hospital (10-30).

The prognosis in terms of the morbidity rating at first symptoms, on first admission to hospital or at the time of operation was studied and, as expected, showed that patients with a high morbidity had a poor early prognosis. However, these data did not give a realistic evaluation of long-term morbidity. In the table, therefore, the morbidity of 70 patients (64%) who survived five months from first symptoms (and would on the whole have completed operative and treatment procedures) is shown at each year after first symptom in relationship to survival. The data show that very few patients who survive five months are able to live at home and that the majority of those who survive can lead independent lives, often with full function. Very few patients get trapped in the tragic situation of being in hospital or requiring prolonged care from relatives. In this, they are luckier than patients following severe head injuries or strokes.

The data also indicate that the prognosis for patients with primary brain tumours is bad. The mode of onset of disease was reviewed by McKern and Thomas, but morbidity between time of diagnosis, operation or death was not considered. In this study, patients who did survive often led a relatively normal life for a long time. When their condition deteriorated, their decline tended to be rapid. There is no evidence that operation or treatment in the first five months could, in themselves, leave a patient in a prolonged state of disability, although the immediate morbidity due to these procedures is impossible to assess.

In summary, the mortality in a group of 110 patients with gliomas was high. However those who survived the first few months led a relatively normal life for a reasonable time. When their condition deteriorated decline was rapid and they rarely remained long in the situation of needing considerable care. These results should encourage doctors to treat patients with gliomas.

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References


Trigeminal and facial nerve involvement resulting from ischaemia of the petrosal branch of the middle meningeal artery

Sir: We describe a woman with diabetes mellitus who suddenly experienced total facial paralysis on the right side accompanied by ipsilateral anaesthesia in the region of the trigeminal nerve. It is speculated, on the basis of anatomical, experimental and clinical data, that this association resulted from a vascular mechanism involving the petrosal branch of the middle meningeal artery. Thus the present case report may also provide some clues as to the aetiology of idiopathic facial paralysis.

A 56-year-old woman with mild hypertension and diabetes mellitus was admitted in November 1979 following the sudden onset of facial weakness and paraesthesiae confined to the right side of the face. On admission, blood pressure was 160/95 mm Hg, and neurological examination revealed complete right-sided peripheral facial palsy and loss of sensation over the whole distribution of the right trigeminal nerve, with striking impairment of appreciation of pain and temperature. The corneal reflex was absent while the Vth nerve motor function was preserved. Funduscopic examination showed microaneurysms, soft exudates, and narrowing of the retinal vessels. All laboratory tests were unremarkable except for an increased blood glucose level. The EEG, radiographs and CT scans of the skull, radiographs of the chest and cervical spine, full audiometric