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Catatonia secondary to acute Chagas' encephalitis

Sir: We have read the contribution by MP Barnes, *et al*¹ with great interest. It emphasises the need to view catatonia as merely a symptom and not a disease, and that full neurological investigation is warranted to disclose any organic brain lesion. Our recent experience with a patient suffering from Chagas' (American trypanosomiasis) encephalitis presenting with catatonia, encouraged us to report our findings.

This 25 year old man acutely developed abnormal behaviour and intermittent fever six months after renal transplantation. He had been treated with cyclosporin and methylprednisolone. When the psychiatric symptoms developed, he was on maintenance doses of both drugs and his renal function was normal. At age 12 years, he had been seen by a psychiatrist because of social withdrawal and a diagnosis of schizoid personality was made. On admission, he lay motionless and mute with an expressionless face; he would reply occasionally and appropriately in monosyllables when questioned. He also had catalepsy and maintained a stereotyped posture. There were no abnormalities on neurological examination, except for reduced muscle tone. Initially, symptoms were attributed to a psychiatric cause. An EEG showed diffuse slowing. CSF was clear, with a protein content of 1.2 g/l and no cells. The complement fixation (Machado Guerreiro) test for Chagas' disease was negative. A CT scan was normal.

There was progressive clouding of consciousness, leading to stupor and coma within two weeks. A frontal brain biopsy showed necrotic parenchymal tissue with macrophages full of amastigotes positive for specific *Trypanosoma cruzi* antiserum (Sternberger's PAP Technique), and a few lymphocytes as well as reactive peripheral astrocytes. Despite treatment with Nifurtimox (15 mg/kg/day), there was no improvement and he died one month after onset of catatonia.

To the best of our knowledge, this is the first report of catatonia induced by Chagas' disease, and we suggest that it should be included in the list of neurological infections liable to cause catatonia. Chagas' encephalitis causes an acute non-suppurative encephalomyelitis,² with small inflammatory foci spread uniformly in the white and grey matter. Acute Chagas' disease is mainly restricted to children, but can occur in immunodeficient adults due to contaminated blood transfusion.^{3,4} In our patient, the negative complement fixation

test seemed attributable to immunosuppressive treatment.

Since catatonia is often caused by an underlying brain disease,⁵ every effort should be made to exclude an organic lesion. A brain biopsy may be the only means of diagnosing a life-threatening condition.

GUSTAVO SEVLEVER

ANA HÍA TARATUTO

M CAROLINA DE LAS CARRERAS

ROMÓN LEIGUARDA

MARTÍN NOGUÉS

Instituto de Investigaciones Neurológicas

Dr Raul Carrea

Ayaoucho 2166

1112 Buenos Aires, Argentina

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Behavioural manifestations of third ventricular colloid cysts

Sir: Dr Arnold Goran, a Diplomate of the American Board of Neurological Surgery, recently referred me to the section entitled "Matters Arising" in the April 1986 issue of *J Neurol, Neurosurg Psychiatry*. In Dr Winer's reply to Dr Backlund, he makes the statement "unfortunately, in the United States there is difficulty finding a neurosurgical centre which performs CT guided biopsy."

I would like to strongly disagree with Dr

Matters arising

Winer's statement. He, obviously, has not been staying abreast of neurosurgical developments in this country. Since the development of the Brown-Roberts-Wells CT stereotaxic guidance system, which has been available since 1981, and the modification of the classical Leksell stereotaxic unit, CT-guided stereotaxy is literally available in over 200 neurosurgical centres in the United States. I believe, if Dr Robert Winer took a look around him, it is likely that within the short distance of 100 miles he would find a neurosurgical centre, skilled in the use of this technique.

I might refer Dr Winer to the case records of the Massachusetts General Hospital published in the *N Engl J Med* on 5 June 1986. In the presentation of a 55-year-old right-handed woman with blurred vision, the pathologic diagnosis of a primary central nervous lymphoma was made with CT stereotaxic localisation and biopsy using the Brown-Roberts-Wells frame.

M PETER HEILBRUN
School of Medicine,
50 North Medical Drive,
Salt Lake City,
Utah 84132,
USA

Syncope and sudden death attributed to carbamazepine

Sir: I read with interest the recent report of Stone and Lange on a young epileptic with sudden death attributed to carbamazepine.¹ They cited the various articles illustrating the electrophysiological actions of this anti-epileptic on the heart, including two isolated cases of carbamazepine-associated bradycardia.^{2,3} Separately, I am reporting a rare patient with complex partial seizures of confusion accompanied by profound sinus bradycardia (down to 10 beats a minute during the attacks).⁴ Results of cardiac testing on our patient were normal. He was prescribed carbamazepine after he could not tolerate phenytoin; plasma therapeutic levels and complete seizure control were achieved. After one year, medication was discontinued and in subsequent follow up, seizures have not recurred. He tolerated carbamazepine well and at no time disorders of cardiac rhythms were documented on his medical visits. He took no concomitant medications. Should this patient's bradycardia had resulted from his individual propensity to develop bradyarrhythmias (seizure-induced or otherwise), I expect the

complications in question should have operated from the use of the drug; furthermore, antecedent cardiac testing failed to show baseline abnormalities.⁴ My patient was prescribed carbamazepine because of the well-known optimal therapeutic effects of this agent on complex partial seizures. I confess, however, Drs Stone and Lange's detailed review on the subject left me with guarded concern about prescribing carbamazepine in patients with associated cardiac conduction defects. Nevertheless, I venture to say, based on this single experience that the noxious cardiac effects of carbamazepine may be only significant in the presence of underlying heart disease. If the heart is normal, encountering ictal arrhythmias should not be a contraindication for prescribing carbamazepine in epileptics.

DANIEL E JACOME
Department of Medicine,
Palmetto General Hospital, and
Hialeah Hospital,
Hialeah, Florida; and
Department of Neurology,
University of Miami,
School of Medicine,
Miami, Florida, USA

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Necrotising haemorrhagic encephalomyelopathy in an adult: ? Leigh's disease

Sir: May I be allowed to ask a question and to make a comment on the case reported by Dr Delgado and colleagues¹ entitled "Necrotising haemorrhagic encephalomyelopathy in an adult: ? Leigh's disease"? I would like to ask if this patient's work or interests brought him into contact with any chemicals which might have been responsible for the described changes. My question arises from the growing realisation that such cases, whether in children or in adults, give every indication for being expressions of an acute tissue energy deprivation, and there seem to be many

environmental as well as genetic causes for this type of metabolic disturbance. The pattern of the damaged brain regions found in the reported case is very similar, as the authors rightly point out, to that found in Wernicke's encephalopathy, and although the latter is rarely as severe as this, it may occasionally be.² An alcoholic cause appears to be excluded here, but the possibility of another environmental agent being responsible remains, and it is for this reason that I pose the question.

From experimental studies it is now coming to be realised that there are a number of chemicals that may produce acute tissue energy deprivation states that end up with tissue damage of the same type and the same general distribution within the neuraxis as seen in Wernicke's disease. The list of agents known from experimental studies to act in this way is growing and there appear to be several points along the energy generation pathways that may come under attack. Thus, the glycolytic pathway can be blocked at separate points by 6-chloro-6-deoxy-glucose³ and (indirectly) by 6-aminonicotinamide,⁴ causing acute, vasculonecrotic lesions in cortex, basal ganglia, brain stem centres and in spinal cord grey matter.^{5,6} However, perhaps more importantly in the present context, there are also compounds that undergo nitro-reduction in the tissues and in the presence of divalent oxygen, such as would be encountered in highly oxygenated nervous tissue, enter a redox cycling state that brings with it adverse metabolic consequences, which include disturbance to electron transport, over-utilisation of cellular NAD(P) and GSH, and the generation of free radical species. This situation occurs with the drugs metronidazole (5-nitroimidazole) and miso-nidazole (2-nitroimidazole), and with certain nitrofurans, and has also been described following administration to animals of both nitrobenzene and 1,3 dinitrobenzene, chemicals widely used in industry that have found their way onto the domestic market in different forms. All these last chemicals produce in experimental animals, with slight species differences, acute vasculonecrotic lesions⁸⁻¹¹ closely similar to the changes seen in acute thiamine deficiency in man and in animals. In view of the growing interest, both clinical and experimental, in this group of disorders, it would be helpful if an answer to my question could be given by the authors of the article noted above. It may be that the question was asked of the patient and the answer was in the negative, but in the interest of good case recording we should have this information.