the presenting features. All three patients were assessed and investigated by a neurologist, and no other diagnoses were obtained. Nystagmus, unsteady gait, and cranial palsies other than cranial nerve VI have, however, rarely been described in association.¹ The close temporal relation of the symptoms (including atypical symptoms and signs and lumbar puncture opening pressures) and successful treatment of the patients' myeloma strongly suggests an association but of course is not conclusive. Other mechanisms may be at play—the aetiology of mononeuritis multiplex in myeloma remains obscure.

Total protein content, sugar, and cytology of cerebrospinal fluid were all entirely within normal limits in all three cases. The computed tomography appearances were also normal (ventricles; no mass effect) apart from the empty sella as shown in our table.

We agree that dural sinus thrombosis has to be excluded in patients presenting with benign intracranial hypertension. Our radiologists were specifically asked to exclude the possibility on the basis of contrast enhanced computed tomography scans. Magnetic resonance imaging and arteriography would be more sensitive methods but we did not feel they were justified as the presenting features were chronic in onset, the protein concentration in cerebrospinal fluid was not raised, and there were no red cells to suggest venous infarction. Most importantly, however, it would not have altered our management, anticoagulation being a contentious issue. Hypercoagulation symptoms are rare in myeloma, which is not a myeloproliferative disorder; indeed, a bleeding tendency is common through interference at many levels of platelet and clotting function.²

From published studies, we are not clear of the incidence of dural sinus thrombosis in benign intracranial hypertension. We suspect that most neurologists do not regularly exclude this condition in patients with suspected benign intracranial hypertension unless the clinical presentation is acute; mortality from dural sinus thrombosis is even higher in patients with malignant disease.

We maintain that myeloma should be included in the differential diagnosis of causes of benign intracranial hypertension. Most other associations are also rare,³ and the clinical course of two of our three patients obviated the need for further treatment of their benign intracranial hypertension.

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- 1 Swash M, Oxbury J, eds. *Clinical neurology*. Vol 1. Edinburgh: Churchill Livingstone, 1991:358.
- 2 Penny R, Castaldi PA, Whitted HM. Inflammation and haemostasis in paraproteinaemias. *Br J Haematol* 1971;20:35-44.
- 3 Digre KB, Corbett JJ. Pseudotumour cerebri in men. *Arch Neurol* 1988;45:866-77.

Missed neuroleptic malignant syndrome

SIR,—We agree with the statement by Deborah S Renwick and colleagues that greater awareness of neuroleptic malignant syndrome among physicians dealing with patients taking neuroleptic drugs may improve outcome and reduce mortality.¹

After we came to realise our own missed cases we devised a protocol to raise awareness of the syndrome and its treatment among medical and nursing staff. With the backing of local drug and therapeutic committees this protocol was circulated widely within our own and neighbouring hospitals, and it has been incorporated into ward nursing procedures and medical staff induction programmes. Vigilance for all features of the disorder is increased, and one of the cardinal changes in practice is simply to monitor the daily

temperature of patients who are receiving energetic treatment with neuroleptics. Any pyrexia in such a patient is attributed to neuroleptic malignant syndrome until proved otherwise. Accordingly, we have been able to make early diagnosis and intervention in recent cases of the syndrome. We strongly recommend the use of such a protocol and would be pleased to supply copies to those interested.

We are grateful to Dr P Clarke for assistance with preparation of the protocol.

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- 1 Renwick DS, Chandraker A, Bannister P. Missed neuroleptic malignant syndrome. *BMJ* 1992;304:831-2. (28 March.)

SIR,—In their lesson of the week on missed neuroleptic malignant syndrome Deborah S Renwick and colleagues recommend that "serum creatine kinase should be measured early in patients with any feverish illness who are taking neuroleptic drugs."¹ The implication seems to be that creatine kinase is of diagnostic importance in this condition.

It is true that Levenson uses creatine kinase as one of his three main diagnostic criteria,² but the creatine kinase level is simply a non-specific indicator of muscular catabolism and, as such, can be raised in many conditions that may be included in the differential diagnosis of the neuroleptic malignant syndrome. These include damage to skeletal muscle from any cause (for example, intramuscular injections), malignant hyperpyrexia, infectious diseases affecting muscle, coma, convulsions, and any cause of muscular rigidity.³ As an illustration of this, we recently reported catatonia with greatly increased creatine kinase levels in a patient who had not taken neuroleptic drugs at any time during the illness.⁴

The creatine kinase level should be used only as an indicator of (non-specific) muscle damage. It is by no means pathognomonic of the neuroleptic malignant syndrome.

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- 1 Renwick DS, Chandraker A, Bannister P. Missed neuroleptic malignant syndrome. *BMJ* 1992;304:831-2. (28 March.)
- 2 Levenson JL. Neuroleptic malignant syndrome. *Am J Psychiatry* 1985;142:1137-45.
- 3 Ford RD. *Diagnostic tests handbook*. Springhouse, Pennsylvania: Springhouse, 1987.
- 4 Craddock B, Craddock N, Milner G. CPK in NMS. *Br J Psychiatry* 1991;158:130.

SIR,—The lesson of the week on missed neuroleptic malignant syndrome presents an important, but incomplete, message.¹ The authors describe a patient with a feverish illness in whom the syndrome was diagnosed late, with a fatal outcome. When the diagnosis was made treatment with dantrolene was unsuccessful. But other treatments for the syndrome were apparently not tried. Some authors suggest that intravenous benzodiazepines, particularly lorazepam, may be helpful; others note that electroconvulsive therapy is useful. The idea that electroconvulsive therapy may help is not well known, the information being restricted to specialist journals as most cases of the neuroleptic malignant syndrome are reported in psychotic patients treated with neuroleptic drugs.²

A review of experience with various treatments in the neuroleptic malignant syndrome shows that the historic mortality associated with treatment with a specific drug (dantrolene or bromocriptine)

is 10% and that that associated with electroconvulsive therapy is similar. But the authors note that the deaths with electroconvulsive therapy were associated with the continued use of neuroleptic drugs during the course of treatment. In cases in which neuroleptic drugs were stopped the results were salutary. Electroconvulsive therapy, as presently administered under anaesthesia and with systemic monitoring, is remarkably safe. Colleagues and I have reported our successful experience with a severely ill, cachectic patient with a similar syndrome of fever, motor, and consciousness disorder.³

The pathophysiology of the neuroleptic malignant syndrome is considered to be a failure of dopaminergic transmission, hence the recommended use of bromocriptine. Electroconvulsive therapy increases brain dopaminergic activity, as reflected in numerous reports that it reduces symptoms of Parkinson's disease.^{4,5} Thus electroconvulsive therapy should be considered in cases of the neuroleptic malignant syndrome.

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- 1 Renwick DS, Chandraker A, Bannister P. Missed neuroleptic malignant syndrome. *BMJ* 1992;304:831-2. (28 March.)
- 2 Davis JM, Janicak PG, Sakkas P, Gilmore C, Wang Z. Electroconvulsive therapy in the treatment of the neuroleptic malignant syndrome. *Convulsive Therapy* 1991;7:111-20.
- 3 Fricchione GL, Kaufman LD, Gruber BL, Fink M. Electroconvulsive therapy and cyclophosphamide in combination for severe neuropsychiatric lupus with catatonia. *Am J Med* 1990;88:442-3.
- 4 Anderson K, Baldini J, Gottfries CG, Granerus AK, Modigh K, Svennerholm L, et al. A double-blind evaluation of electroconvulsive therapy in Parkinson's disease with "on-off" phenomena. *Acta Neurol Scand* 1987;76:191-9.
- 5 Fochtmann L. A mechanism for the efficacy of ECT in Parkinson's disease. *Convulsive Therapy* 1988;4:321-7.

AUTHORS' REPLY.—Max Fink refers to a patient with a syndrome similar to the neuroleptic malignant syndrome who was successfully treated with electroconvulsive therapy. The scientific literature contains other sporadic case reports of the use of electroconvulsive therapy in the neuroleptic malignant syndrome, but there have been no clinical trials to compare electroconvulsive therapy with the more conventional drug treatments for the syndrome (dantrolene and bromocriptine). Consequently the current role of electroconvulsive therapy in the syndrome is that of a second line agent, for use when conventional treatment has failed.

In the case we described the correct diagnosis of the neuroleptic malignant syndrome was obscured by the presence of a proved urinary tract infection, so that treatment with dantrolene was started only when the patient was already extremely unwell. The considerable risks of electroconvulsive therapy in patients critically ill with the syndrome include the induction of malignant hyperthermia by anaesthetic agents,¹ hyperkalaemia,² and potentially fatal cardiac arrhythmias.³ In this case we considered that these risks outweighed the possible potential benefits of the treatment.

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- 1 Vitkun SA, Boccia RV, Poppers PJ. Anesthetic management of a patient with neuroleptic malignant syndrome. *J Clin Anesth* 1990;2:188-91.
- 2 George AL, Wood CA. Succinylcholine induced hyperkalaemia complicating the neuroleptic malignant syndrome. *Ann Intern Med* 1987;106:172.
- 3 Addonizio G, Suzman VL. ECT as alternative treatment for patients with symptoms of the neuroleptic malignant syndrome. *J Clin Psychiatry* 1986;47:42-3.