for the study would be great, although preliminary results from a recent study suggest that they are not insurmountable.1

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To the Editor

Dear Sir;

We read with interest the paper by Kidd and Thompson entitled “Screening for neurostatus in intensive care units.”1 We would like to comment on their methods and findings. In their study, Kidd and Thompson assessed 335 patients in 34 units and reported that 11 patients (3.3%) were felt to have a neurostatus. They concluded that the screening method used was reliable and that a randomised controlled trial was required to compare the method they used with the current routine practice in those units.

In our study, we assessed 213 patients in 14 units and found that 8 patients (3.8%) were felt to have a neurostatus. This is in agreement with the findings of Kidd and Thompson and suggests that their screening method is similar to the routine practice in the units we assessed.

However, the method used by Kidd and Thompson has certain limitations. First, the screening method relies on the subjective assessment of the medical staff. This may lead to bias and variability in the results. Second, the method does not provide any evidence of the severity or the underlying cause of the neurostatus. Finally, the method does not provide any information on the prognosis of the patients.

Therefore, we believe that a randomised controlled trial comparing Kidd and Thompson’s screening method with the current routine practice is necessary to establish the reliability and validity of the method.

Sincerely,

[Your Name]

[Your Institution]

Reference

ried with no pupillary change, it was most probably realised through the activity of the oculomotor nerve although we did not perform EMG recording of the muscles.

The afferent limb of this reflex is probably attributable to proprioceptive impulses rising in the deep tissues of the arm such as muscles, ligaments, tendons, and joints participating in flexion of the arm. It is unlikely that pain receptors played a part, because the patients did not open their eyes to painful stimuli.

The "arm EOR" was accompanied by extension of the head and contralateral limbs suggesting a decerebrate response. Decerebrate response usually occurs in brainstem lesions with at least partial and bilateral midbrain lesions, but occasionally in severe diffuse bilateral hemispheric damage due to posthypoxic encephalopathy. This response is interpreted as a release phenomenon of the brainstem activity from higher extrapyramidal control. It includes not only extension of the limbs and body, but also some pathological reflexes of the head such as clenching of the jaw ("a bulldog reflex"), which was seen in our patient 1, or on the other hand, jaw opening. These reflexes must require activity of the appropriate cranial nucleus and its efferent pathways. The "arm EOR" may be regarded as the requisite of a decerebrate responses involving the cranial nerves. It at least suggests preservation of the central caudal nucleus, which is thought to be responsible for the eyelids.

We report with confidence that there exists an EOR elicited by flexion of the arm. It may be a manifestation of decerebrate response, especially in diffuse bilateral hemispheric damage after acute hypoxic-ischaemic encephalopathy. This phenomenon may be mediated through the proprioceptive system, although it has been considered that the pain system is the most important role in occurrence of both decerebrate response and wakefulness.

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Association of antineural antibodies in a patient with paraneoplastic cerebellar syndrome and small cell lung carcinoma

Three autoantibodies have been well characterised in paraneoplastic neurological syndromes and proved to be helpful in the diagnosis. Anti-Hu (or ANNA-1) antibodies were reported in patients with encephalomyelitis-sensory neuropathy, complex and small cell lung cancer, anti-Yo (or PCA-1) antibodies were identified in women with paraneoplastic cerebellar degeneration and gynaecological tumours, and anti-Ri (or ANNA-2) antibodies were first reported in patients with opsoclonus/ataxia and breast cancers.

Our patient with paraneoplastic cerebellar syndrome and small cell lung carcinoma had circulating anti-Hu and anti-Ri autoantibodies. The serum samples and the CSF of this patient also contained anti-CV2, another autoantibody recently described in patients with paraneoplastic neurological syndromes.

A 56 year old woman with a 50 pack-year history of cigarette smoking had an unremarkable medical history until October 1994, when she developed acute vertigo. Brain CT showed no abnormalities. She then developed mild instability which remained stable until March 1995 when her gait became difficult with frequent falls. In June 1995, neurological examination showed bilateral horizontal nystagmus, dysarthria, and severe statokinetic cerebellar syndrome. The rest of the neurological and general physical examination was normal. Routine laboratory and immunological analyses were normal. Erythrocyte sedimentation rate was 40 mm. Cerebrospinal fluid contained 10 white blood cells/mm³ (40% lymphocytes and 60% neutrophils), 50 mg/dl protein (IgG 8.1 mg/dl, IgA 0.8 mg/dl, and IgM 0.4 mg/dl), and 43 mmol/dl glucose. Cytology was negative for malignant cells. Viral, bacterial, and fungal cultures from blood, CSF, and urine were negative. Serological studies of blood and CSF, including HIV, Lyme, syphilis and hepatitis B and C were negative. An EEG was normal. Brain MRI showed mild vermian and cerebellar atrophy. Computed tomography of the chest, abdomen, and pelvis disclosed two mediastinal lymphadenopathies and one node in the right adrenal gland. Bronchoscopy was normal. A mediastinoscopy showed several lymphadenopathies adhered to the trachea, the biopsy of which was consistent with small cell anaplastic carcinoma. Treatment included intravenous immunoglobulins (0.4 g/kg/day) and methylprednisolone (1 g/day) for five consecutive days, once a month, from July to September 1995. The patient received chemotheraphy from July to December 1995 (cyclophosphamide, etoposide, cisplatine, and epirubicine) and after chemotheraphy, she underwent bilateral lung hilus irradiation (44 grams) and mediastinal irradiation (16 grams). Clinical follow up was marked by progressive deterioration of cerebellar ataxia and, in February 1996, she was bedridden and totally dependent. There was tumour progression and the patient died suddenly in April 1996. Necropsy was not permitted.

Serum and CSF were examined for the presence of antineural antibodies, using immunohistochemical and western blot techniques with rat and human brain, as previously reported. Immunochemical studies showed the presence of antibodies which reacted with the nuclei, and to a lesser degree the cytoplasm of neurons, and with the cytoplasm of a subpopulation of oligodendrocytes in the white matter of the rat cerebellum, brainstem, and spinal cord as seen in patients with anti-CV2 antibodies. Western blot analysis of isolated Purkinje cells, recombinant HuD (an Hu antigen), CD62 (a Yo antigen), and Nova (an Ri antigen) showed a high titre of both anti-Hu and anti-Ri antibodies (figure A).
Eye opening reflex triggered by flexion of an arm: a manifestation of decerebrate response in diffuse bilateral hemispheric damage.

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