and chlorothiazide reintroduced when plasma lithium falls to non-toxic concentrations.

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Maprotiline hydrochloride and grand-mal seizures

We report three cases in which grand-mal seizures occurred for the first time during treatment with maprotiline hydrochloride (Ludiomil).

Case reports

Case 1—A 28-year-old mother of two children had been subject to bouts of depression since the birth of her first child six years before and was prescribed maprotiline 150 mg at night. Four months later she had a grand-mal seizure, which was not preceded by any aura or partial seizure activity. There was no history of head injury or alcoholic excess and no family history of epilepsy. Her only other medication was the contraceptive pill (Ovranette). Physical examination and routine investigations, including skull radiography and biochemical tests, showed nothing abnormal. Maprotiline was stopped. Three weeks later electroencephalography (EEG) showed a post-central dysrhythmia, which became generalised with hyperventilation. EEG at six months showed no noticeable change, but the recording at 20 months showed only a mild, brief theta dysrhythmia. She experienced no further seizures during the follow-up period.

Case 2—A 37-year-old postmistress was taking maprotiline 75 mg at night during her third bout of depression. After four weeks of treatment she had a well-documented grand-mal seizure without aura or focal onset. She was also taking an oral contraceptive (Ovran) and trifluoperazine, which her general practitioner had prescribed for nervous tension. All medication was stopped after the seizure. There was no personal or family history of fits, and no other cause for her seizure was found. Results of routine biochemical investigations and skull radiography were normal. EEG 21 days after the seizure showed a raised-voltage paroxysmal theta dysrhythmia with rapid generalised epileptiform breakdown occurring on photic stimulation. The recording two months later showed only intermittent interruption of normal rhythms, and nine months after the attack it was entirely normal. No further seizures occurred during 18 months of follow-up but she became deeply depressed one month after stopping maprotiline and was admitted to a psychiatric ward. On this occasion she was successfully treated with a tricyclic preparation (amitriptyline).

Case 3—A 36-year-old housewife, recently separated from her husband, had been taking maprotiline 75 mg at night for one week when she experienced a well-documented grand-mal seizure. There was no aura or focal onset and no relevant personal or family history. She was taking no other medication. Results of physical examination, skull radiography, and biochemical investigations were normal. EEG two weeks after the seizure showed a theta dysrhythmia with raised voltage and a frequency of 4-6 c/s. These paroxysms were prolonged during hyperventilation. EEG 11 weeks later showed a return to normal with only a mild theta dysrhythmia. She had no further seizures during four months of follow-up.

Comment

Grand-mal seizures may occur after overdosage of maprotiline,[1] and convulsions in patients taking therapeutic doses have been noted[2–4] but not fully documented. In one series[5] 3-4% of patients taking tricyclic antidepressants in therapeutic doses had grand-mal seizures, but no figures are available for maprotiline, which is a tetracyclic compound.

When seizures occur during treatment with maprotiline the drug should be stopped. Investigation and close follow-up is required to eliminate another cause for the seizure. If an antidepressant is still required a tricyclic drug should be introduced with caution.

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SHORT REPORTS

Gold lung

In the past two years there have been three reports of unusual pulmonary reactions to gold treatment affecting six patients, which have all conformed to a similar pattern. We present a further case in which the histological appearance suggested adenocarcinomatous deposits, but the patient recovered when the gold was stopped and steroids were given.

Case report

A 41-year-old West Indian district nurse developed rheumatoid arthritis in 1964. Her symptoms were controlled by ibuprofen alone until April 1977, when she was started on sodium aurothiomalate injections 50 mg weekly. Her erythrocyte sedimentation rate was 60 mm in first hour (Westergren), Rose-Waaler 1/64, antinuclear factor 1/10, DNA antibodies < 10 units. The chest radiograph was normal, and radiographs of the hands and feet showed erosions of the metacarpophalangeal and metatarsophalangeal joints.

Eight weeks after starting treatment, having had 400 mg of gold, she developed pleuritic pain on the left and complained of shortness of breath. She did not have a cough. There was no rash or clubbing. There were coarse late inspiratory crackles at the left base anteriorly and laterally. A chest radiograph showed patchy consolidation in the region of the lingula, but a perfusion scan was normal. The patient refused admission. She was treated with ampicillin. One week later coarse inspiratory crackles were heard in all areas of both lungs and there were patchy, diffuse opacities in all areas on the chest radiograph. She was admitted for further investigation.

Investigations showed: white cell count $6{\cdot}4 \times 10^9$/l (neutrophils 65%, eosinophils 5%); P_{CO_2} 4·6 (4·6-6·0) kPa (34·5 (34·5-45) mm Hg); P_{O_2} 9·7 (12-15) kPa (72·7 (90-113) mm Hg); forced expiratory volume in one second (FEV_1) 1·0 l (predicted 2·8 l); vital capacity (VC) 1·1 l (predicted 3·6 l). Carbon monoxide transfer factor (T_LCO) could not be measured satisfactorily while the results of skin tests and precipitins were negative. An open lung biopsy was performed.

Histology—The sections showed numerous intra-alveolar inflammatory cells and macrophages. The alveolar walls were fibrotic and the alveolar