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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise, strictly relevant and interesting to the reader.

CARDIAC ARREST IN A YOUNG WOMAN TREATED WITH AMITRIPTYLINE COMBINED WITH LEVOPROMAZINE

DEAR SIR,

Some authors such as Herzmann (1978) compare the influence of the tricyclic antidepressants (TAD) on the heart to that of quinidine, talking about a quinidine-like effect as a result of which atrioventricular block may occur or which may lead to ventricular fibrillation and even cardiac arrest. Most cases of cardiac arrest among patients treated with TAD and, less frequently, with neuroleptics take place in older people or in people with a history of heart disease. There is a much smaller number of reports of sudden disturbance of cardiac rhythm, ventricular fibrillation and even death in young patients treated with the above mentioned drugs but who did not previously suffer from heart disease (Moccetti, 1977; Hollister, 1978). Some authors express doubt that such cases exist, e.g. Sack (1977). The risk of cardiac complications becomes greater when the doses of TAD are large or when TAD is given in combination with neuroleptics (Hollister, 1978). There has been no mention in the literature of clinical death following cardiac arrest as a result of an unfavourable interreaction between amitriptyline and levopromazine (an aliphatic phenothiazine) given in therapeutic doses. It is worth stressing the fact that the case discussed here concerned a young woman who had never had heart disease. For both these reasons the case seemed worth reporting.

A 34-year-old single female engineer was admitted in depressive stupor. She had been previously healthy and free of heart disease, and many routine ECGs in psychiatric departments had been normal. On admission she had a normal tracing, with tachycardia. Eight days after admission amitriptyline was started in doses rising to 250 mg daily on the 10th day of treatment. Because of insomnia she also had levopromazine 100 mg at night. She improved markedly, being in better contact and less depressed. On the 12th day she had unheralded cardiac and respiratory arrest at 10.30. Anaesthetists were by chance at hand and immediately began resuscitation by intubation, assisted respiration and cardiac massage, followed by defibrillation, which restarted the heart at 11.15. The patient's ECG then showed normal axis, sinus rhythm at 96 per minute, non-specific ST deflections, T_1 plane, and T waves in leads V_2 to V_6 flattened. That afternoon the patient recovered consciousness and recognized her parents. At 20.05 ventricular fibrillation returned very briefly but ceased without intervention. A xylocaine infusion was started, at 22.55 defibrillation again had to be applied for ventricular fibrillation, and at 24.00 tracheotomy for accumulation of bronchial secretions. The next day the patient's general condition improved greatly, and five days after that the ECG was normal.

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NEUROENDOCRINE RESPONSES AS AN INDICATOR OF RECURRENCE LIABILITY IN PRIMARY AFFECTIVE ILLNESS

DEAR SIR,

The assessment on clinical grounds of the liability of affective disorders to recur has not been satisfactory. Clearly, if we had a laboratory test which would detect biological propensity for relapse before it manifests in psychopathology and behaviour, the management of recurrent affective disorders could be