time) after the fourth dose. This cleared within 24 hours after stopping EACA. The patient remained unconscious for about three months before she died.

Comment

The rash in this patient seemed to be causally related to the drug. The purpuric element of the rash was probably part of the hypersensitivity reaction and not related in any way to the antifibrinolytic effect of EACA. We do not think there has been a British report of a rash associated with EACA treatment, but we have heard that two such cases have been reported to Swedish manufacturers of the drug.

We thank Kabi-Vitrum (UK), the manufacturers of Epsikapron, for their co-operation, and Dr J M S Pearce for permission to report this case.

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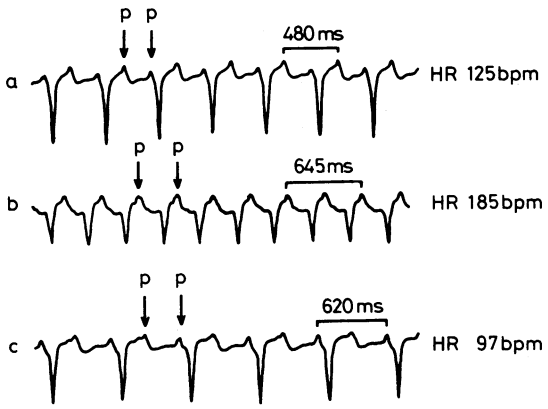
Potentially dangerous effect of disopyramide on atrioventricular conduction in a patient on digitalis

The antiarrhythmic drug disopyramide prolongs the refractory period of atrial myocardium.[1] It is therefore sometimes useful in treating ectopic atrial tachycardia. Disopyramide also has atropine-like properties[2] which may lead to enhanced atrioventricular (AV) nodal conduction. The combination of slowing the atrial rate with "improved" AV nodal function may lead to 1:1 conduction of the tachycardia when previously it had been 2:1. This causes a potentially dangerous increase in ventricular rate. A similar increase may occur when disopyramide is given to patients in atrial fibrillation.[3] For this reason patients should be digitalised before attempted conversion of atrial tachycardia with disopyramide. This report concerns the failure of digitalis to protect a patient from this unwanted effect.

Case report

The patient, a 68-year-old woman, had a long history of rheumatic mitral stenosis. She had been well having no treatment other than warfarin until two months before admission, when she developed a fast heart rate and dyspnoea on minimal exertion. An electrocardiogram (ECG) showed atrial tachycardia with 2:1 conduction. Digoxin and propranolol failed to control the rhythm so she was admitted for a trial of disopyramide. On admission she was taking digoxin 0·25 mg and frusemide 40 mg once a day, potassium supplements, and warfarin. On examination she was euthyroid: pulse rate 125/min, regular; jugular venous pressure raised 3 cm; blood pressure 140/80 mm Hg. Both ventricular impulses were slightly increased and the murmurs of moderate mitral stenosis and incompetence were present. There were no signs of pulmonary oedema. Blood urea concentrations, electrolytes, and thyroid function tests were normal; haemoglobin concentration was 14·7 g/dl, and serum digoxin 1·4 mg/l. Chest radiographs showed mitral cardiac contour with clear lung fields, and an ECG showed atrial tachycardia with 2:1 conduction (figure (*a*)).

Disopyramide was given intravenously in a total dose of 1·5 mg/kg over five minutes with continuous ECG monitoring. Thirty seconds after the injection she developed 1:1 conduction (figure(*b*)). The atrial rate had slowed from 250 to 185. She became breathless and hypotensive. Three minutes later she reverted to 2:1 conduction with an atrial rate of 194 (figure (*c*)).



Electrocardiogram before and after intravenous disopyramide: (a) before; (b) immediately after; (c) three minutes after. Lead VI shown at paper speed of 25 mm/s. p Represents p waves. Time for two atrial cycles shown.

Comment

The feature of interest in this case is that digitalis failed to protect the patient from the atropinic effect of disopyramide. Without an intracardiac study the relative contributions of enhanced AV conduction and slowing of the atrial rate cannot be disentangled nor can the effect of the digitalis on the AV node be assessed. Although the serum digoxin concentration may not be clearly related to its effect on AV nodal conduction, it is the only means available to most physicians to ensure adequate digitalisation. Caution should therefore be exercised when giving disopyramide to patients with atrial tachycardia even when they are taking digitalis. The same caution probably applies equally to treatment with quinidine, whose affect on electrophysiological pathways is very similar to that of disopyramide. This potential hazard should not detract from the usefulness of disopyramide in a condition which may be difficult to treat.

We thank Dr D Verel for permission to report this case.

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Corrections

Anabolic effect of human parathyroid hormone fragment on trabecular bone in involutional osteoporosis: a multicentre trial

We regret that in the first paragraph of the Patients and methods section of the above paper (7 June p 1340), 50 mg (2000 IU) daily should have read 50 μg (2000 IU) daily, and 12·5 mg (500 IU) daily should have read 12·5 μg (500 IU) daily. In the table the column referring to urinary hydroxyproline mmol should have read μmol.

Peroperative venography to ensure accurate sapheno-popliteal vein ligation

An error occurred in the paper by Dr J T Hobbs (28 June, p 1578). The measurements in the sixth line of the methods section should be 30 cm × 40 cm.