MEDICAL MEMORANDA

Alprenolol in Resuscitation

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Sympathetic beta-blocking agents such as alprenolol are not for routine use in resuscitation, but they may be life saving in certain cases. We report three cases of myocardial infarction in which alprenolol was successfully used with direct-current shock when other measures had failed to terminate recurring ventricular fibrillation.

Case 1

A 44-year-old man who had two myocardial infarctions and who was taking thyroid 100 mg daily for hypothyroidism after thyroidectomy had a cardiac arrest. Ventricular fibrillation was successfully defibrillated. Thirteen days later, as he was about to leave hospital, he had another attack of ventricular fibrillation. Sinus rhythm was restored but not maintained for longer than 15 to 30 seconds after each defibrillation, despite the fact that acidosis was corrected, the patient well oxygenated, and given adequate doses of lignocaine. After one hour alprenolol 10 mg was given intravenously. The first 200 W/s direct-current shock three minutes later terminated the fibrillation. The patient recovered without any signs of brain damage. Lignocaine and procainamide (500 mg six hourly) were not enough to control tachyarrhythmias, and alprenolol 100 mg daily was added to the treatment. Sinus rhythm was then maintained. Thyroid had been discontinued in view of the tachyarrhythmias. The patient was discharged after six weeks, but anginal attacks increased and after four days he was readmitted because of intractable chest pain. Five days later he had another cardiac arrest. Once again resuscitation was prolonged and defibrillation had to be repeated eight times. Recovery was uneventful. For the past 18 months the patient has been maintained on thyroid 50 mg and alprenolol 300-400 mg daily. Anginal attacks have been rare and less severe.

Case 2

A 52-year-old man with a long history of anginal attacks was admitted to hospital with a transmural anteroseptal infarction. A fortnight later he had an attack of ventricular fibrillation. Resuscitation lasted over four hours, and the patient had to be defibrillated 20 times despite the administration of lignocaine 800 mg and the correction of acidosis. Glucagon 6 mg and hydrocortisone 1,000 mg were also given. After intravenous alprenolol 9 mg and atropine 1.5 mg had been given defibrillation terminated the attacks and the patient recovered. Eight days later, on two occasions he again had to be resuscitated. On the first occasion alprenolol 4 mg was given intravenously before successful defibrillation. Alprenolol had already been given by mouth for two days previously but in a dose of only 50 mg three times a day. This was increased to 100 mg four

times a day, and lignocaine was also continued. Fourteen days later the patient had bursts of ventricular tachycardia as well as ventricular fibrillation which required defibrillation 17 times. Procainamide 500 mg by mouth four times a day and hydrocortisone 500-1,000 mg were added to the treatment. Irritability of the myocardium continued until the procainamide was replaced by phenytoin 100 mg four times a day. For the past six months the patient has remained in sinus rhythm.

Case 3

A 56-year-old man with a history of coronary disease went into ventricular fibrillation 16 days after admission to hospital with a transmural anteroseptal infarction. Fibrillation continued despite correction of acidosis, adequate doses of lignocaine and glucagon, and several attempts at defibrillation. After an hour alprenolol 5 mg and atropine 1 mg were given intravenously before a 300 W/s direct-current shock, which terminated the fibrillation. The patient was maintained on lignocaine and alprenolol was given twice during the first night. Two days later a sudden attack of multiple extrasystoles was followed by ventricular tachycardia and fibrillation. Defibrillation gave only temporary relief and after two hours the patient remained in permanent asystole. At necropsy a large, fresh septal and anterolateral infarction was found.

Discussion

Beta-adrenergic receptor blocking agents have been used in exceptional cases to terminate ventricular fibrillation (Sloman *et al.*, 1965; Rothfeld *et al.*, 1968; Ikram, 1968). By depressing the sympathetic drive on the cardiac beta-receptors they may reduce myocardial contractility to such an extent that the subsequent decrease in cardiac output (further enhanced by acidosis) may lead to persistent hypotension and asystole. Theoretically this risk should be less if some beta-adrenergic stimulating activity could be maintained, and that is why we chose alprenolol in our cases (Abland *et al.*, 1967). Acidosis was always corrected before alprenolol was given, and atropine was given with alprenolol to antagonize the rate-slowing effect of adrenergic beta-blockade (Standard *et al.*, 1968; Johnstone, 1970).

With this treatment it was possible to terminate very resistant ventricular fibrillations in three cases, lasting in one case over four hours, in which lignocaine, correction of acidosis, and oxygenation failed to maintain sinus rhythm after defibrillation. Our conclusion is that alprenolol may be lifesaving in these cases, provided acidosis is corrected first and atropine is also given intravenously.

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