Intractable recurrent ventricular tachycardia in dilated cardiomyopathy controlled by a vasodilating β blocker

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A 30 year old white female with dilated cardiomyopathy presented with episodes of exercise induced ventricular tachycardia (VT) associated with symptoms of palpitation, chest tightness, dizziness, and dyspnoea. The resting ECG showed multifocal ventricular extrasystolic beats and lateral T wave inversion. The echocardiogram revealed a dilated left ventricle (end diastolic internal dimension of 6-8 cm) with moderate global hypokinesia and functional mitral regurgitation. Coronary angiography revealed no discrete arterial stenosis. Histology of the endomyocardial biopsy in the left ventricle showed features consistent with idiopathic dilated cardiomyopathy. Ambulatory ECG showed multifocal ventricular extrasystoles and a salvo of VT.

Amiodarone was introduced and the patient improved symptomatically although occasional exertional palpitation, dizziness, and dyspnoea persisted. Electrophysiological study revealed VT consistent with right ventricular outflow origin. During a subsequent cardio-pulmonary exercise test the patient developed symptomatic VT at the peak workload of stage V Bruce protocol when her sinus rhythm heart rate was 176 beats/min and oxygen consumption 31.3 ml/min/kg (fig). The VT was associated with palpitation, dyspnoea, and mild dizziness, and resolved spontaneously on resting.

Attempts were made to abolish the VT with therapy using amiodarone in combination with a class IB antiarrhythmic agent (mexiletine) or β blockers (atenolol, sotalol), but the VT paradoxically occurred at earlier stages during exercise tests. After three years of amiodarone treatment, the patient became hypothyroid necessitating withdrawal of amiodarone. Symptomatic deterioration ensued despite treatment with propafenone and atenolol. There was no obvious deterioration of left ventricular function on echocardiographic examination.

Following cessation of the amiodarone (six months), propafenone, and atenolol therapy, carvedilol was introduced at increasing doses from 3-125 to 6-25 to 12-5 mg twice a day. Symptoms of palpitation, dizziness, and exertional dyspnoea completely resolved. On repeating the incremental exercise test the patient was able to complete two minutes of stage V of the Bruce protocol, reaching a peak heart rate of 176 beats/min, without developing any ventricular arrhythmia. It was not possible to induce VT despite a further repeat of maximal exercise test.

Carvedilol is a nonselective β adrenoceptor inhibitor with vasodilatory properties through its α adrenoceptor inhibition. Trials have shown that acute and chronic carvedilol administration improve resting and exercise haemodynamics in patients with dilated cardiomyopathy.1 Symptomatic improvement has similarly been recorded.12 In patients with New York Heart Association class II–IV heart failure already taking diuretics and angiotensin converting enzyme inhibitors, carvedilol reduced mortality by 65% compared with placebo.3

There was a reduction in the number of premature ventricular contractions with carvedilol administration in patients with hypertension, stable angina, and chronic ischaemic heart failure.4 No study has so far assessed the effect of carvedilol on sustained VT. Indeed, this is the first report of carvedilol being effective in the treatment of VT in a patient with dilated cardiomyopathy. It was safe without causing significant deterioration.
in cardiac function or exercise capacity. Further studies into the antiarrhythmic therapeutic potential of carvedilol are warranted.