At this time the T4 concentration had fallen to slightly below normal and the T3 concentration was within the normal range for the first time, although the TRH test remained suppressed (basal TSH 1.3, at 20 min 1.2, and at 60 min 1.0 mU/l). A thyroid scan showed uptake confined largely to the left lobe of the gland. Thyroid microsomal and thyroglobulin antibodies were detected (+ + and + ± respectively). In May 1978 the patient had gained more weight and complained of tiredness. The concentration of T4 had fallen further while that of T3 remained normal, and the basal serum TSH was now raised. There was no change in the thyroid scan, which was thought to be consistent with extensive thyroiditis on the right side. A month later the hormone concentrations were unchanged.

Comment

Patients with Hashimoto's thyroiditis may be hyperthyroid, euthyroid, or hypothyroid depending on the duration of the disease and the stage at which they are first seen and assessed. For example, in a review of 217 patients² 77.9% were euthyroid, 18.4% were hypothyroid, and 3.7% were hyperthyroid. Hyperthyroidism due to T3 toxicosis seems to be unusual. We were unable to find another report of a convincing case of T3 toxicosis proceeding to hypothyroidism in the absence of treatment. Our patient, on no antithyroid medication, progressed in 11 months from a state of undoubted T3 toxicosis to hypothyroidism associated with the expected rise in serum TSH concentrations.

¹ Doniach, D, Vaughan Hudson, R, and Roitt, I M, *British Medical Journal*, 1960, **1**, 365.

² Fisher, D A, et al, *Journal of Clinical Endocrinology and Metabolism*, 1975, **40**, 795.

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Profound bradycardia in response to atrial fibrillation in the Wolff-Parkinson-White syndrome

In the Wolff-Parkinson-White (WPW) syndrome an anomalous pathway bypasses the physiological filtering characteristics of the atrioventricular node. Atrial fibrillation may therefore result in a rapid ventricular response that may cause syncope or sudden death. In the patient with WPW syndrome described here atrial fibrillation paradoxically resulted in syncope due to profound ventricular bradycardia.

Case report

A 41-year-old chef who was an extremely fit rugby player complained of several syncopal spells, frequent dizziness, and a six-year history of occasional bouts of palpitations. On examination no physical abnormalities were detected except for a resting bradycardia. His chest radiograph and blood values were normal. The resting 12-lead electrocardiogram (ECG) showed atrial bradycardia (35 beats/min) with abnormal P-wave morphology and a P-R interval of 0.16 second. The QRS complexes were broad (0.17 second) with initial slurred delta waves, positive in leads I and V6 and negative in lead V1, typical of WPW syndrome type B. A stress test showed excellent exercise tolerance and an appropriate increase in atrial rate. A 24-hour taped ECG showed a basic heart rate that varied from 35 to 120 beats/min and, in addition, pauses in the basic rhythm of up to 2.5 seconds long.

An atrial pacing study was performed which showed abnormal sinus node function with unduly long sinus node suppression (2.6 seconds) after rapid atrial pacing and impaired atrioventricular conduction resulting in 2:1 atrioventricular block at an atrial rate of 100 beats/min. A formal study of the conducting capacity of the anomalous bypass tract, responsible for the WPW syndrome, showed that this tissue was not capable of conducting at intervals shorter than 0.53 second. The induction of atrial fibrillation by rapid atrial

pacing resulted in the dramatic response (see figure) of extreme ventricular bradycardia with R-R intervals of up to 11 seconds in duration and uniformly pre-excited QRS complexes, indicating conduction to the ventricles by the bypass tract. This bradycardia was associated with symptoms identical with those for which the patient had been admitted. In view of this response to atrial fibrillation, impaired sinoatrial function, and atrioventricular conduction, a ventricular demand pacemaker was implanted. During follow-up of almost four months there have been no further episodes of near-syncope or blackouts.

Comment

A substantial proportion of patients with the WPW syndrome suffer spontaneous paroxysms of atrial fibrillation.¹ If the anomalous pathway which links atrium to ventricle can conduct atrial impulses at a high frequency then the ventricles may be stimulated too rapidly to maintain an adequate cardiac output. This is a mechanism commonly invoked to explain episodes of ventricular fibrillation, syncopal spells

Profound ventricular bradycardia with pre-excited QRS complexes during atrial flutter-fibrillation. HRAE, DCSE, PCSE, and HBE are electrograms recorded from the high right atrium, the distal and proximal coronary sinus, and His-bundle regions. I, AVF, V1, and V6 are simultaneous surface ECG leads.

and sudden death in these patients.² If the bypass tract cannot sustain high-frequency conduction atrial fibrillation is thought unlikely to provoke serious ventricular arrhythmias.³

In this patient reduced sinoatrial automaticity and atrioventricular nodal conduction might be ascribed to increased vagal tone in a physically fit individual⁴ and would not in itself indicate the need for cardiac pacing. In the presence of atrial fibrillation profound ventricular bradycardia sufficient to induce syncope cannot be wholly related to the autonomic state of a physically fit individual but must imply widespread conduction disease and the failure of subsidiary pacemaker mechanisms. The low ventricular rate is all the more remarkable in a patient with two atrioventricular conduction pathways but might be explained by repetitive and incomplete conduction into these pathways.⁵

The treatment of WPW syndrome and atrial fibrillation may require antiarrhythmic drug suppression of atrioventricular conduction in both the normal and abnormal pathways to prevent symptoms related to a rapid ventricular response. In this case such treatment might have proved disastrous. Simple atrial pacing techniques allowed an improved assessment of this patient's problem, showed an unsuspected syncopal mechanism, and suggested that the correct treatment should be ventricular pacing rather than antiarrhythmic drugs.

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¹ Dreifus, L S, et al, *American Journal of Cardiology*, 1976, **38**, 149.

² Dreifus, L S, et al, *Circulation*, 1971, **43**, 520.

³ Wellens, H J, and Durrer, D, *American Journal of Cardiology*, 1974, **34**, 777.

⁴ *British Medical Journal*, 1974, **1**, 575.

⁵ Castellanos, A, et al, *British Heart Journal*, 1973, **35**, 811.

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