Lazy Sinus Syndrome

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Mrs R S, aged 66. Housewife

History: She had diphtheria at the age of 3 years and since childhood complained of palpitations; attacks were associated with giddiness and episodes of transient unconsciousness. Her symptoms had become more frequent during the last two years so that she was virtually incapacitated.

In September 1969 she was admitted to hospital. She had no symptoms as an inpatient but the ECG showed a slow nodal rhythm. She was started on treatment with Saventrine, 30 mg q.d.s., without benefit. In November 1969 she was readmitted to hospital. Her arrival on the ward coincided with an attack of palpitations.

Examination and investigation: She was semiconscious with an irregular heart rhythm of variable rate. A soft mid-systolic murmur was audible at the apex of the heart. No evidence of cardiac failure.

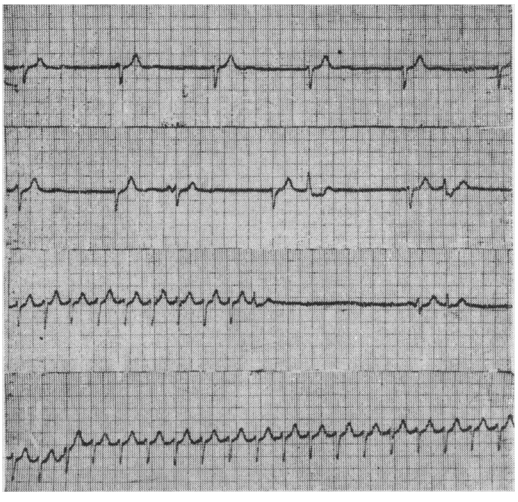

Fig 1 *ECG on admission*

ECG showed slow nodal rhythm with paroxysmal supraventricular tachycardia and short periods of asystole. Little evidence of sinus node activity, but all evident P waves were followed by a normal QRS complex. That atrioventricular conduction was normal was confirmed by the normal P–R interval on atrial pacing (Figs 1 & 2).

Treatment and progress: Transvenous ventricular pacing (later replaced by permanent demand pacing on 15.12.69) was instituted for two reasons: (1) It seemed possible that an irritable supraventricular focus might be consistently depolarized by a pacing stimulus so that tachycardia would not develop. (2) If it proved impossible to prevent paroxysms in this way, the pacemaker would allow large amounts of anti-arrhythmic agents to be used without producing bradycardia. Ectopic atrial activity in fact was not controlled by pacing and it was only after using propranolol 60 mg q.d.s. that the ectopic dysrhythmia was more effectively suppressed.

Discussion: Sinus node inertia is a well documented form of rhythm disturbance, usually attributed to increased vagal tone. The unusual feature in this case is the permanency of the inertia and the severity of the symptoms produced. An interesting possibility is that destruction of the sinus node developed following the attack of diphtheria in infancy.

The problems of management in this type of situation have been described in detail by Sowton (Sowton *et al.* 1964, Bloomfield & Sowton 1967, Sowton *et al.* 1969). The use of propranolol introduces the risk of depression of myocardial function. In addition, the drug may produce severe bradycardia, and iatrogenic heart block may occur. Pacing has long been established as a method of suppressing dysrhythmias, usually on a temporary basis. In the long term, it suffers from the disadvantage that an unacceptably rapid rate may be required to prevent recurrence of the tachycardia. A combination of both pacing and anti-arrhythmic agents to a large extent overcomes the disadvantages of either method of treatment alone. Bloomfield & Sowton (1967)

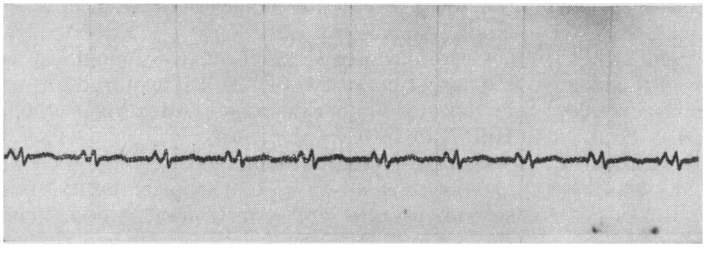

Fig 2 *ECG after atrial pacing*

have reported a reduction in cardiac output when patients with pacemakers are given propranolol but there has been no such problem in this case. If supraventricular tachycardia cannot be controlled by a combination of pacing and medical therapy, then surgical division of the conducting system and implantation of a ventricular pacemaker should be considered.

REFERENCES
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Dr Celia Oakley said that logically the patient should have been treated by chronic atrial rather than ventricular pacing on a demand basis, but chronic atrial pacing was technically unreliable and this was why conventional ventricular demand pacing was chosen. This patient's dysrhythmias responded well to relatively modest doses of propranolol so that it could be hoped she would have no further problems.

Patients with the lazy sinus syndrome might not also be troubled by rapid ectopic rhythms, and some then responded to long-acting isoprenaline which could successfully 'kick' the lazy sinus into more frequent activity. In the present case isoprenaline had failed because it also inspired the ectopic foci to greater activity. Propranolol alone was no help because of the excessively slow heart rate, and this was the reason for the combination of pacing with antidysrhythmic treatment. As the patient had at no time shown any sign of myocardial pump dysfunction, she had not suffered any adverse side-effects from the use of β-blocking drugs.

Reversible Organic Dementia due to Normal-pressure Communicating Hydrocephalus

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Miss A T

In 1958, at age 48, she gave a six-month history of stiffness and heaviness of the legs. She had a mild dementia, a spastic dysarthria and there was spastic weakness of both legs with extensor plantar responses. An air-encephalogram (AEG) showed mild symmetrical dilatation of the lateral ventricles and a diagnosis of cerebral atrophy (cause undetermined) was made.

By 1966 there had been a slow progression of her previous symptoms, but the physical signs were unchanged. AEG now showed pronounced symmetrical dilatation of both lateral ventricles but, despite repeated manœuvres, no air passed over the hemispheres. Following the AEG there was a marked deterioration in her mental and general state.

Although the CSF pressure was normal the diagnosis of atrophy was revised to one of probable chronic communicating hydrocephalus because of (1) failure of air to pass over the hemispheres, (2) air remaining in the ventricles four weeks after AEG, (3) gross clinical deterioration after AEG.

To corroborate the diagnosis isotope encephalography was performed by the lumbar route. This technique was described by Bannister *et al.* (1967) in an earlier report of this case. Isotope had entered the ventricles seven hours after injection, and only after 48 hours was activity there decreasing and beginning to appear over the surface of the hemispheres.

Diagnosis of normal-pressure communicating hydrocephalus was thus confirmed and a ventriculocaval shunt was inserted with the valve opening at all pressures above 45 mm. Within a few days there was a marked improvement in her speech and mental state and a decrease in the spasticity. The improvement was maintained throughout 1967 and 1968, but since early 1969 there has been a gradual deterioration in gait, though her mental improvement has been maintained.

Comment

Normal-pressure communicating hydrocephalus was first described by Adams *et al.* (1965). Mental deterioration and disorder of gait are constant features whilst urinary incontinence occurs in a high proportion and bilateral pyramidal signs are seen in advanced stages. The CSF pressure is not raised; it is always less than 200 mm of CSF.

AEG appearances are characteristic, showing dilatation of ventricular system and failure of air to pass into the cerebral subarachnoid space. However, the scan appearances after intrathecal injection of radioactive iodinated human serum albumin (RISA) are more specific, and this technique has several advantages over air encephalography (Bannister *et al.* 1967).

Once diagnosed, relief of this condition is regularly obtained either by shunt procedures or by repeated lumbar punctures (Adams *et al.* 1965, Hill *et al.* 1967).

Normal-pressure communicating hydrocephalus is thus an important clinical entity, but several aspects are poorly understood. The etiology and pathogenesis are obscure where neither

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