Long-term Control of Intractable Supraventricular Tachycardia by Ventricular Pacing

EDGAR SOWTON, RAPHAEL BALCON, TOM PRESTON, DAVID LEAVER, and MAGDI YACOUB

From Institute of Cardiology, London W.1

The prevention of rapid dysrhythmias in patients without heart block by pacing the heart above a critical rate has been utilized on a short-time basis for some years, particularly in patients with recent cardiac infarction (Sowton, Leatham, and Carson, 1964; Heiman and Helwig, 1966), or in those who have undergone cardiac surgery (Eraklis, Green, and Watson, 1965). The technique of overdriving in this way is usually applied only as a temporary measure but is applicable also to the long-term treatment of patients with tachycardia (Sowton, 1968; Burchell and Meredith, 1969; Dack, 1969). In this report we describe our clinical experience over the past two years with this method of treatment of supraventricular tachycardia.

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**TABLE I**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Type of tachycardia</th>
<th>Intermittent bradycardia before pacing</th>
<th>Angina during tachycardia</th>
<th>Ventricular rate during tachycardia</th>
<th>Period of symptoms before pacing (yrs)</th>
<th>Other diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>48</td>
<td>Wolff-Parkinson-White; atrial fibrillation</td>
<td>Only when on propranolol</td>
<td>Yes</td>
<td>200/min.</td>
<td>10</td>
<td>(i) Mild mitral valve disease</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>57</td>
<td>Paroxysmal supraventricular tachycardia; atrial fibrillation</td>
<td>When digitalized</td>
<td>No</td>
<td>180/min.</td>
<td>1</td>
<td>(ii) L.t. hemiparesis during tachycardia</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>55</td>
<td>Paroxysmal supraventricular tachycardia; atrial fibrillation</td>
<td>No</td>
<td>Yes</td>
<td>200/min.</td>
<td>3</td>
<td>Obesity; mild hypertension (B.P. 170/100 mm. Hg)</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>47</td>
<td>Paroxysmal supraventricular tachycardia</td>
<td>Venticular asystole on deep inspiration or carotid sinus massage</td>
<td>No</td>
<td>160/min.</td>
<td>18</td>
<td>Transient l.t. hemiparesis during tachycardia</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>58</td>
<td>&quot; &quot;</td>
<td>No</td>
<td>Yes</td>
<td>150/min.</td>
<td>2/12</td>
<td>Cardiac infarction 5 yr. and 2 mth. previously</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>60</td>
<td>&quot; &quot;</td>
<td>No</td>
<td>Yes</td>
<td>240/min.</td>
<td>Several</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>55</td>
<td>&quot; &quot;</td>
<td>No</td>
<td>Yes</td>
<td>200/min.</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

**Patients and Procedure**

Seven patients with intractable supraventricular tachycardia have been treated in this way for periods of up to 2 years. None had heart block at any time before pacemaker treatment. Ages ranged from 48 to 60 (mean 54 years) and there were 5 men and 2 women (Table I). In all cases recurrent attacks of supraventricular tachycardia had failed to respond to drug treatment despite doses increased to the limits of tolerance, as indicated in Table II. In all cases where these drugs were not contraindicated, full doses of digoxin, quinidine, procainamide, and propranolol had been given alone and in combination without success, often over several years. Three of the 7 patients had intermittent atrial fibrillation with ventricular rates of about 200 a minute despite digitalization. Digoxin had been given to all 3 in increasing doses to the point of toxicity without preventing the episodes of tachycardia. One patient had frequent periods of supraventricular tachycardia which could be
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TABLE II

DRUG AND PACEMAKER TREATMENT

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Failed treatment before pacing (excluding short-term drugs)</th>
<th>Type of pacemaker</th>
<th>Period of pacing (mth.)</th>
<th>Propranolol dose during pacing (mg./day)</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Digoxin; quinidine; phenytoin; procaainamide; propranolol</td>
<td>(i) Cordis demand</td>
<td>25</td>
<td>200</td>
<td>&quot;Pulitation&quot; during atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(ii) Cordis ventricular-triggered</td>
<td>25</td>
<td>—</td>
<td>No attacks since pacing started</td>
</tr>
<tr>
<td>2</td>
<td>Digoxin; propranolol; saventrine</td>
<td>Cordis demand</td>
<td>16</td>
<td>360</td>
<td>Very occasional short attacks of tachycardia</td>
</tr>
<tr>
<td>3</td>
<td>Digoxin; quinidine; procaainamide; propranolol; dextro-propranol; atropine</td>
<td>Cordis ventricular-triggered</td>
<td>9</td>
<td>80</td>
<td>Tachycardia of 140–150/min. when propranolol omitted</td>
</tr>
<tr>
<td>4</td>
<td>Digoxin; atropine; dextro-propranolol; ICI 50172</td>
<td>ICI 50172</td>
<td>12</td>
<td>300</td>
<td>No attacks since pacing started</td>
</tr>
<tr>
<td>5</td>
<td>Digoxin; propranolol procainamide; ICI 50172</td>
<td>Medtronic demand</td>
<td>1</td>
<td>360</td>
<td>No attacks since pacing started</td>
</tr>
<tr>
<td>6</td>
<td>Digoxin; propranolol; quinidine; ICI 50172; dextro-propranol</td>
<td>Cordis ventricular-triggered</td>
<td>Die 24 hr. post-op.</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>Digoxin; quinidine; procaainamide; propranolol</td>
<td>Cordis ventricular-triggered</td>
<td></td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

interrupted temporarily by vagal manoeuvres, though these frequently resulted in a syncopal episode. Carotid sinus massage or even deep inspiration caused total asystole (Fig. 1). Attacks of tachycardia were accompanied by acute dyspnoea suggesting left ventricular failure in every case and by typical ischaemic cardiac pain in all except 2 patients (Cases 2 and 4). The episodes were severe and frequent enough to cause gross disturbance in the patients' lives, and several had been confined to bed for considerable periods (up to a month) because of recurrent episodes of tachycardia. Control of acute episodes had usually been achieved by admission to hospital for DC countershock in combination with large doses of intravenous lignocaine, but this provided only very short remissions from tachycardia. Control of these episodes had usually been achieved by admission to hospital for DC countershock in combination with large doses of intravenous lignocaine, but this provided only very short remissions from tachycardia. Two patients (Cases 1 and 3) had suffered left hemipareses during paroxysms of tachycardia, which had left a slight residual disability in Case 1.

Pacemaker treatment was instituted for three reasons. First, it seemed possible that an irritable supraventricular focus might be consistently depolarized by the pacemaker impulse so that tachycardia did not develop. Secondly, it proved impossible to prevent paroxysms in this way the pacemaker would allow large doses of anti-dysrhythmic drugs to be used without producing bradycardia, maintaining cardiac function until an effective dose had been reached. Thirdly, if both these mechanisms proved unsuccessful pacing would already be well established by the time surgical division of the conducting tissue was considered, providing the pacing electrodes were situated on the ventricle.

Though atrial stimulation is capable of functioning in the first manner indicated above, and was in fact used for short periods in some of these patients, the likelihood that large doses of drugs would also be given and the possibility that iatrogenic AV dissociation might be produced influenced us to site the final electrode in the ventricle in all cases. Subsequent progress of some patients indicates that atrial pacing would probably have been effective, and this technique has subsequently been used in other patients not included in the present report.

An initial trial of pacing was carried out in hospital with right atrial and right ventricular transvenous electrodes. The patients were continuously monitored and pacing was continued for a minimum of 48 hours; an exercise test was included in the assessment. If it appeared likely that the method would be useful, a pacemaker was implanted with transvenous electrodes in the ordinary way. A demand or ventricularly-triggered pacemaker was used in every case to reduce the risk of competition in these patients with irritable hearts if the dysrhythmia recurred. After the implant no anti-

Fig. 1.—Electrocardiogram of Case 4. A rapid supraventricular tachycardia is interrupted by atrial and ventricular asystole during deep inspiration.
dysrhythmic drugs were given unless tachycardia returned or ectopic beats were noted on the monitor. If the patient experienced no return of his paroxysms drugs were not used.

In other cases propranolol was given in increasing doses until paroxysms of tachycardia had been eliminated both at rest and on effort. Even when large doses of propranolol were given to these patients with poor cardiac function, side-effects were minimal and no patient developed cardiac failure. Presumably this complication was avoided because the ventricular rate was maintained by the pacemaker.

**RESULTS**

Control of tachycardia has been achieved in all cases, though propranolol was eventually needed in 5 of the 7 patients. The underlying abnormality persisted in the patients, and temporary inhibition of the implanted pacemaker revealed persistence of the original conditions (Fig. 2). In one patient with Wolff-Parkinson-White syndrome transient periods of atrial fibrillation occurred and the subsequent irregularity was appreciated by the patient as palpitation. During these episodes the ventricular rate did not increase appreciably owing to the propranolol and did not slow because of the pacemaker, which functioned faultlessly without producing competition (Gibson and Sowton, 1969). In one patient (Case 3) attacks of tachycardia were initially controlled by propranolol 240 mg./day but recurred after 4 months. The propranolol was increased to 360 mg./day, and the dysrhythmia recurred only once in the next 6 months, the attack lasting about 5 minutes and terminating spontaneously (Fig. 3).

The over-all results in the group show that paroxysms of tachycardia have been controlled in 6 patients over an average time of 15 months. In 1 patient (Case 3) occasional episodes have occurred but have been controlled by an increase in propranolol dosage, and in 1 patient (Case 5) episodes still occur if propranolol is omitted but they are short lasting and considerably slower than they were before this method of treatment was instituted.

**Fig. 2.—Electrocardiogram during pacing of Case 1.** Low voltage (2V) electrical stimuli from a second pacemaker have been applied to the skin over the heart, and these inhibit the implanted demand pacemaker. During this period the underlying Wolff-Parkinson-White complexes can be seen.

**Fig. 3.—Electrocardiograms of Case 3.** In the lowest strip the first, second, and fourth beats are paced, but the third is spontaneous and has triggered the implanted pacemaker.
PACING RESULTS

There has been no premature pacemaker failure in this group. The longest time a pacemaker remains implanted with continuing normal function is 25 months (Case 2). One patient developed tenderness and probable local infection at the pacemaker site after 12 months, but this was controlled with antibiotics, and normal pacemaker function continued until the unit was replaced after 24 months (Case 1). In one other patient (Case 3) the ventricular-triggered pacemaker failed to sense spontaneous QRS complexes for a short period so that it functioned as a fixed rate unit. Normal triggering was present at recent follow-up appointments, and the pacemaker continues to function normally after 16 months in use.

In one patient (Case 7) the technique was not successful and surgical division of the conducting tissue was attempted as an emergency procedure.

Case 7. This man aged 55 had a 5-year history of paroxysmal supraventricular tachycardia, with attacks becoming more frequent and lasting longer during the past 18 months. Attacks usually followed respiratory infection and he suffered from asthma. He had several recent admissions to hospital for periods of up to 6 weeks during which the tachycardia was temporarily terminated by DC countershock.

Four weeks previously tachycardia developed and he was admitted to the Brook General Hospital under the care of Dr. Bruce Pearson 2 weeks later. Drug treatment with digoxin, intravenous procainamide, lignocaine, and propranolol was ineffective and DC shock produced a remission lasting only a few seconds. He was transferred to the National Heart Hospital where his ventricular rate was found to be over 200 a minute. He was severely orthopnoeic and complained of chest pain though this was not severe. The cardiac impulse could not be felt and the jugular venous pressure was raised 5 cm. above the sternal angle. The blood pressure varied but was low, with the systolic level at about 80 mm. Hg, and there was generalized purpura over the trunk. There had been haemoptysis for the past week.

Treatment with intravenous lignocaine (1 g./8hr.) dextropropranolol (50 mg. i.v.), and ICI 50172 (50 mg.) did not affect the tachycardia. A C 51 bipolar electrode catheter was passed to the right atrium and the heart slowed to 115 a minute by paired pacing with 120 msec. separation between impulses (Fig. 4). Despite an initial improvement peripheral vascular failure developed, and he was transferred to the operating theatre for emergency division of the conducting tissue.

At operation (Mr. M. Yacoub), attempts were made to produce block by diathermy with a finger inside the right atrium. Supraventricular rhythm persisted and normothermic bypass was instituted. Sutures were placed in the region of the AV node and the bundle, and diathermy was used both above and below at the tricuspid ring. AV dissociation was produced, but when the patient came off bypass supraventricular rhythm reappeared and bypass was reinstituted.

Stitches were then placed in the bottom of the membranous septum between the right and the non-coronary

![Image](https://i.imgur.com/3Q5Q5Q.png)

**Fig. 4.**—Electrocardiogram of Case 7. The supraventricular tachycardia (upper strip) was slowed by paired pacing from an electrode in the right atrium (lower strip). The delay between impulses was 120 msec.
cusps and the whole area was extensively treated by diathermy. Bypass was stopped and the rhythm was found to be varying between complete heart block and supraventricular tachycardia. A Cordis ventricular-triggered pacemaker was implanted, the electrodes being attached to the right ventricle. A particular feature during the operation was that the PR interval apparently shortened after attempts to destroy the bundle or AV node.

After operation the rhythm continued to vary, and though pacing was technically satisfactory the blood pressure was low (mean 40 mm Hg). Temporary improvement followed with an isoprenaline/adrenaline intravenous drip, but after resuscitation from one cardiac arrest he died in ventricular asystole on the second day.

At necropsy (Professor R. E. B. Hudson) 80 sections were cut from the region of the coronary sinus. The AV node was penetrated by a suture and was very haemorrhagic. The bundle of His was virtually necrotic in its distal half, with several sutures penetrating it. Both right and left branches were effectively destroyed by haemorrhagic necrosis and penetration by sutures. The ventricular muscle was unremarkable, but the endocardium over the left atrium was greatly thickened, and the aortic valve cusps showed some fibrous thickening.

**Discussion**

The use of propranolol in the long-term treatment of supraventricular tachycardia is frequently successful in reducing or preventing attacks (Gettes and Surawicz, 1967; Frieden *et al.*, 1967) but introduces a grave risk of depression of cardiac function, particularly since large doses may be needed. In addition, the drug may produce severe bradycardia when the patient returns to sinus rhythm, and iatrogenic heart block may occur. Propranolol alone is contraindicated on these grounds for the group of patients who experience varying attacks of tachycardia and bradycardia, though the combination of propranolol and pacing has been successfully used in several centres (Sandoe and Flensted-Jensen, 1969). The group of patients in the present series differs in that AV block was not present before drug treatment, though 1 patient had sinus bradycardia alternating with supraventricular tachycardia (Case 2), and another patient had episodes of ventricular asystole precipitated by deep inspiration or by carotid sinus massage (Case 4); in this patient atropine had failed to prevent attacks of asystole or tachycardia. It has been shown (Bloomfield and Sowton, 1967; Donoso *et al.*, 1967; Lund-Larsen *et al.*, 1968) that cardiac output is reduced during effort when patients with fixed-rate pacemakers are given propranolol, but this has not presented a clinical problem in the present series.

Artificial pacing has long been known as a method of suppression of dysrhythmias, usually on a temporary basis. As a long-term method it suffers from the disadvantage that an unacceptably fast rate may be needed in some patients to prevent recurrence of the tachycardia. Disadvantages of each method of treatment are to a large extent overcome by the combination of both pacing and large doses of anti-dysrhythmic drugs even in a group of severely ill patients. Life-threatening tachycardia has been successfully controlled for up to 2 years with return of housebound patients to regular work.

Attachment of the electrode to the atrium is theoretically preferable and has in practice been successfully achieved by Kastor *et al.* (1967). Using a preformed curved Cordis pervenous electrode in the right atrium, these authors were able to obtain stable atrial pacing without the necessity for thoracotomy. In the present report only ventricular electrodes were utilized to avoid risks associated with possible subsequent development of AV block in a patient with a rapid rate, either due to additional drug therapy or related to atrial pacing. Even in normal subjects, complete heart block can be produced at rapid rates (Warner and Lewis, 1961; Lister *et al.*, 1965), and Lau *et al.* (1967) have reported a similar temporary AV block in both normal and accessory conduction pathways in 1 subject with Wolf-Parkinson-White syndrome. More experience with long-term atrial pacing will be needed before the magnitude of these theoretical risks can be properly assessed.

The combined use of long-term pacing and drugs to control multiple rapid dysrhythmias has been reported previously, both by ourselves (Sowton *et al.*, 1964) and by others (Avenhaus, Grohmann, and Nordmann, 1967). McCallister, McGoon, and Connolly (1966) reported the first use of an implanted pacemaker for long-term control of paroxysmal ventricular tachycardia and fibrillation. No additional drugs were used and the pacing was set at 100 a minute. Moss *et al.* (1968) described the first long-term use of left atrial transvenous pacing for control of recurrent ventricular fibrillation in a patient whose pacemaker stimulated at 110 a minute; additional procainamide was given. A left atrial electrode site was also used by Cohen *et al.* (1967) who reported successful prevention of recurrent ventricular tachycardia and fibrillation in a 15-year-old girl who was also given propranolol. Furman, Escher, and Solomon (1967) reported their use of standby pacing in patients with supraventricular dysrhythmias and spontaneous or drug-induced bradycardia, while the combination of propranolol and pacing was also used by Wennevold and Sandoe (1968) in the treatment of a patient with ventricular tachycardia.

Surgical division of the conducting tissue represents a logical extension of treatment if life-threatening supraventricular tachycardia cannot be pre-
vented. The recurrence of supraventricular rhythm in our own patient despite destruction of the anatomical conducting pathways emphasizes the difficulties and raises the possibility that transmission of impulses from the atrium to the ventricle might have occurred by simple contact or mechanical movement. This experience differs from those reported by Giannelli et al. (1967), by Slama et al. (1967), by Dekker, Büll, and Schuilenburg (1965), and by C. Ranzi (1968, personal communication), who were able to produce complete block successfully, with satisfactory implantation of pacemakers.

**Suggested Programme for Pacing of Intractable Tachycardia**

When a decision has been made that drug treatment alone is unsatisfactory a suitable course of action might be as follows.

1. If AV dissociation has never been a feature of the presentation, either before or during drug treatment, then atrial pacing should be established on a temporary basis.
2. If AV dissociation has been present at any time or large doses of suppressant drugs are contemplated, then temporary ventricular pacing should be started.
3. The pacing rate should be increased until the dysrhythmia does not recur at rest or during moderate exercise over several days.
4. Anti-dysrhythmic drugs may be added to the regimen in increasing doses to suppress the dysrhythmia without requiring an unduly rapid pacing rate (say 100/min.).
5. A permanent system can then be implanted to provide the conditions already established.
6. If supraventricular tachycardia cannot be controlled during the temporary pacing, surgical division of the conducting system and implantation of a ventricular pacemaker should be considered.

**Summary**

Six patients severely ill with intractable, drug-resistant, paroxysmal supraventricular tachycardia have been treated for up to two years with a combination of propranolol and demand pacing. In all cases, the dysrhythmias were satisfactorily controlled. One further patient died after surgical division of the conducting system. The method can be used to treat patients with rapid supraventricular tachycardia, irrespective of associated dysrhythmias. A suggested scheme for the management of such patients is presented.

We would like to express our gratitude to colleagues who have referred patients with their permission to reproduce details in this publication.

**References**


Sowton, Balcon, Preston, Leaver, and Yacoub


