Intractable paroxysmal tachycardia caused by a concealed retrogradely conducting Kent bundle

Demonstration by epicardial mapping and cure of tachycardias by surgical interruption of the His bundle

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Electrophysiological and epicardial mapping studies are described in a patient without pre-excitation who had intractable recurrent paroxysmal supraventricular tachycardia. Electrophysiological studies revealed fixed VA conduction times during both rapid ventricular pacing and coupled ventricular stimulation. Catheter mapping of atrial activation during retrograde conduction and during induced paroxysmal supraventricular tachycardia revealed early distal coronary sinus activation (posterior left atrium) relative to the low septal, low lateral, and high lateral right atrium. These studies suggested the presence of a concealed left-sided bypass tract.

The patient underwent surgical interruption of the His bundle for control of paroxysmal supraventricular tachycardia. Epicardial mapping of the atria (during ventricular pacing) confirmed the presence of a concealed left-sided bypass tract. Surgery produced antegrade AV block (while retrograde conduction was maintained) and total cure of paroxysmal supraventricular tachycardia. This is the first reported case of a concealed retrograde extranodal pathway documented by epicardial mapping.

Recent studies in patients with re-entrant paroxysmal supraventricular tachycardia without apparent pre-excitation suggest that re-entry can either be within the AV node or involve the normal pathway and a concealed extranodal pathway (Denes et al., 1973; Coumel and Attuel, 1974; Neuss, Schlepper, and Thormann, 1975; Spurrell, Krikler, and Sowton, 1974a, b; Zipes, DeJoseph, and Rothbaum, 1974). In the latter cases, the extranodal pathway is capable of only retrograde conduction and, therefore, concealed on the surface electrocardiogram. Differentiation between intra- and extranodal re-entry has both diagnostic and therapeutic implications.

In the present report, we describe the electrophysiological and epicardial mapping studies in a patient without ventricular pre-excitation who had recurrent, intractable supraventricular tachycardia, and who underwent surgical ablation of the His bundle. Preoperative electrophysiological studies suggested the presence of a left-sided concealed extranodal pathway capable of only retrograde conduction. Epicardial mapping of the atria at the time of cardiac surgery confirmed the presence of an anomalous left-sided connexion. After surgical section of the His bundle, complete antegrade AV block was produced with maintenance of intact retrograde conduction.

Case report

The patient was a 44-year-old male Jehovah’s witness, with documented recurrent paroxysmal supraventricular tachycardia for 15 years. Electrocardiograms between attacks were within normal limits, with a PR interval...
Because of failure of drug therapy, an attempt was made to control the tachycardia with atrial pacing, but was unsuccessful. A permanent externally activated demand epicardial ventricular pacemaker was implanted, when studies suggested that this mode of therapy would result in interruption of tachycardias. However, within several weeks of pacemaker implantation, intractable recurrent paroxysmal supraventricular tachycardia again occurred. The patient was then referred to the University of Illinois Hospital for further evaluation.

Electrophysiological studies

Electrophysiological studies were performed in January 1975, after informed consent was obtained. Medicines were discontinued 72 hours before study. His bundle electrograms were recorded with catheter techniques (Scherlag et al., 1969). In addition, a quadripolar catheter was positioned in the high right atrium for recording and stimulation. A hexapolar catheter was positioned so that the distal two poles were located at the right ventricular apex, while the proximal electrodes were contiguous to the low lateral right atrium. A bipolar catheter was placed in the distal coronary sinus.

Basic conduction intervals were recorded during sinus rhythm at a cycle length of 800 ms and were as follows: PA of 42 ms, AH of 84 ms, and HV of 45 ms. With high right atrial pacing, 1:1 AV conduction was noted up to a paced rate of 200/min, with an increase in AH interval from 110 to 200 ms. AV nodal Wenckebach periods were noted at a paced rate of 210/min. There was no evidence of an antegradely conducting anomalous pathway with pacing of the low lateral right atrium, the coronary sinus, and low septal right atrium (Denes et al., 1974). With right ventricular pacing at rates of 90 to 170/min VA conduction (ventricle to low septal right atrium) remained constant at 230 ms. The sequence of atrial depolarization during retrograde conduction was distal coronary sinus, preceding both low septal right atrium and high right atrium by 40 ms.

Antegrade refractory periods were measured with atrial extra-stimulus technique from the high right atrium at cycle lengths of 600 and 430 ms and are listed in the Table. The AV conduction curves (A1-A2, H1-H2) were smooth.

Table: Refractory periods (ms)

<table>
<thead>
<tr>
<th></th>
<th>CL</th>
<th>600</th>
<th>430</th>
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<tbody>
<tr>
<td>Atrial ERP</td>
<td>220</td>
<td>230</td>
<td></td>
</tr>
<tr>
<td>Atrial FRP</td>
<td>270</td>
<td>250</td>
<td></td>
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<tr>
<td>AV node ERP</td>
<td>270</td>
<td>260</td>
<td></td>
</tr>
<tr>
<td>AV node FRP</td>
<td>380</td>
<td>330</td>
<td></td>
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<tr>
<td>Ventricular ERP</td>
<td>—</td>
<td>280</td>
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<tr>
<td>Bypass ERP</td>
<td>&lt;280</td>
<td></td>
<td></td>
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<tr>
<td>AV echo zone</td>
<td>305-270</td>
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CL = Cycle Length; FRP = Functional Refractory Period; ERP = Effective Refractory Period.
Retrograde refractory period determinations were performed using the ventricular extrastimulus technique (V) from the right ventricular apex at a cycle length of 600 ms. V-A remained constant at 230 ms, despite shortening of V-V from 580 to 285 ms. The sequence of atrial activation was identical to that described above during incremental ventricular pacing. VA conduction was limited by ventricular refractoriness at an S1-S2 interval of 280 ms.

Both single atrial echoes and sustained paroxysmal supraventricular tachycardia were noted during pacing-induced AV nodal Wenckebach periods, with achievement of a critical AH of 260 ms (Goldreyer and Damato, 1971). An echo zone (single echoes ± episodes of sustained paroxysmal supraventricular tachycardia) was defined with atrial extrastimulus technique between A1-A2 coupling interval of 305 to 270 ms at a cycle length of 430 ms (Fig. 1). The critical AH for induction of echoes during coupled stimulation was 195 ms (Fig. 1B) (Goldreyer and Damato, 1971).

Induced sustained paroxysmal supraventricular tachycardia was characterized by a narrow QRS, an HV of 45 ms, and a cycle length of 367 ms. Retrograde activation times during induced paroxysmal supraventricular tachycardia were as follows (measured from the onset of the QRS to the onset of the respective atrial electrogram): V-distal coronary sinus (110 ms), V-high right atrium (145 ms), and V-low septal right atrium (160 ms) (Fig. 1B).

Short periods of functional left and right bundle-branch block were also observed during episodes of induced paroxysmal supraventricular tachycardia. The cycle length during tachycardia with left bundle-branch block was always 25 ms longer than the cycle length during narrow QRS paroxysmal supraventricular tachycardia and with functional right bundle-branch block. Ventricular extrastimuli (V) were coupled to the QRS (V) of the tachycardia. At V-V intervals greater than 345 ms, no reset of the atrial electrogram was noted. At V-V intervals between 345 and 290 ms, the corresponding A to A1 interval progressively shortened as the V-V was decreased (Fig. 2). At V-V intervals between 345-325 ms, the ventricular extrastimulus (V) depolarized the atria (A) without depolarizing the His bundle (Fig. 2A). At V-V between 285 to 280 ms, V depolarized the atrium and interrupted the tachycardia (Fig. 2C).

The presence of a left-sided concealed extranodal pathway with ability for only retrograde conduction was suggested by the following observations: 1) absence of antegrade pre-excitation on all electrocardiograms and during atrial pacing at multiple sites (Denes et al., 1974). 2) Fixed V-A intervals during incremental and coupled ventricular pacing (Wellens and Durrer, 1974a; Narula, 1974). 3) Abnormal retrograde activation sequence (with distal coronary sinus preceding all other atrial electrograms) during ventricular pacing and during supraventricular tachycardia (Svensen et al., 1974). 4) Lengthening of cycle length of paroxysmal supraventricular tachycardia during functional left bundle-branch block (Coulm and Attuel, 1974; Spurrell et al., 1974a; Neuss et al., 1975). 5) Ability to capture the atria from the ventricles during paroxysmal supraventricular tachycardia without depolarizing the His bundle (Zipes et al., 1974; Neuss et al., 1975). 6) Smooth antegrade AV nodal curves (Wu and Denes, 1975).

During paroxysmal supraventricular tachycardia, the left-sided anomalous pathway was used for retrograde conduction and the AV node for antegrade conduction.

**Fig. 2** Atrial reset with premature ventricular stimulation during supraventricular tachycardia. V represents the last QRS before the introduction of the premature ventricular stimulus (V), CS represents the last coronary sinus electrogram before the ventricular extrastimulus. CS represents the premature coronary sinus electrogram corresponding to the ventricular extrastimulus (V). E indicates echo and H represents the His electrogram. The cycle length of the tachycardia is 365 ms. Paper speed is 100 mm/s, time lines at 1/s.

Panel A: The V-V is 330 ms. The corresponding CS-CS interval is at 330 ms. Note that the HH intervals remain undisturbed at 365 ms. This shows that an extranodal pathway must be present. Panel B: The ventricular premature beat is introduced at a V-V interval of 290 ms. The corresponding CS-CS interval shortens to 290 ms.

Panel C: The V-V interval is decreased to 285 ms; the corresponding CS-CS interval is shortened to 285 ms and blocked in the AV node. The tachycardia is then interrupted. The ventricular pacemaker escapes with retrograde conduction to the atria.
Epicardial mapping and surgery

Because of medical intractability, and because of the demonstration of a circus movement amenable to correction, the patient was referred to the Texas Heart Institute for elective transection of the His bundle. Operation was performed in March 1975. With the heart exposed, epicardial mapping of the atria was performed during right ventricular pacing using a previously described mapping technique (Svenson et al., 1974; Wyndham et al., 1974). The area of earliest atrial activation was noted at the lateral left atrium just adjacent to the left AV ring (Fig. 3). The patient was placed on cardiopulmonary bypass, and the His bundle was interrupted via a right atriotomy incision. Atrial pacing after closure of the right atriotomy revealed complete AV block. Ventricular pacing after closure of the atriotomy revealed intact retrograde activation. Repeat epicardial atrial mapping with ventricular pacing was identical to that before operation.

Subsequent clinical course

After operation, the patient recovered uneventfully. He has remained in heart block with pacemaker rhythm and he has been totally free of paroxysmal supraventricular tachycardia. He feels well and has resumed lecturing.

Discussion

AV re-entry can occur within the AV node or using the normal pathway and an extranodal pathway (Coulomb and Attuel, 1974; Denes et al., 1973; Neuss et al., 1975; Spurrell et al., 1974a, b; Zipes et al., 1974; Wellens and Durrer, 1974a; Svenson et al., 1974; Wu and Denes, 1975). The extranodal pathway can be manifest with ability for antegrade conduction (Wolff-Parkinson-White syndrome) or concealed on the surface electrocardiogram. Concealment of an extranodal pathway reflects unidirectional block with ability for only retrograde conduction. The diagnosis of a concealed extranodal pathway can be suspected by demonstration of the following electrophysiological findings: 1) absence of antegrade pre-excitation on all electrocardiograms and during atrial pacing at multiple sites (Denes et al., 1974). 2) Fixed ventriculoatrial conduction times during incremental and coupled ventricular pacing (Wellens et al., 1974; Narula, 1974). 3) Abnormal sequence of retrograde atrial activation (Svenson et al., 1974). 4) Increase in cycle length of induced paroxysmal supraventricular tachycardia during development of functional bundle-branch block ipsilateral to the suspected anomalous pathway (Coulomb and Attuel, 1974; Spurrell et al., 1974a; Neuss et al., 1975). 5) Retrograde capture of the atria during paroxysmal supraventricular tachycardia without discharging the His bundle (Zipes et al., 1974; Neuss et al., 1975). 6) Smooth antegrade conduction curves (A1-A2, H1-H2) with echo zones (Wu and Denes, 1975).

All of the above electrophysiological features were shown in the present case, suggesting the presence

![FIG. 3 Posterior schematic view of epicardial atrial mapping during right ventricular stimulation.](http://heart.bmj.com/)

 Isochronous lines are drawn at 10-ms intervals with reference to the earliest epicardial atrial activation (0 ms). Note that the posterolateral left atrium contiguous to the left AV ring is activated earliest.
of a concealed extranodal pathway. In addition, the
findings suggested that the extranodal pathway was
left-sided. The importance of our case resides in the
fact that epicardial mapping of the atria confirmed
the presence of a left-sided pathway, as predicted by
preoperative electrophysiological studies. Epicardial
mapping showed early retrograde activation of the
lateral left atrium contiguous to the left AV ring.

Little is known regarding treatment of recurrent
supraventricular tachycardia in patients with con-
cealed extranodal pathways. In our experience, these
cases are characterized by smooth conduction
curves, as opposed to the discontinuous conduction
curves often noted in patients with intra AV nodal
dual AV nodal pathway re-entrant paroxysmal
supraventricular tachycardia (Wu and Denes, 1975).
Previous studies concerning induction of re-
entrant tachycardia in the catheterization laboratory
before and after drug administration suggest that
patients with smooth conduction curves are more
likely to have potentiation of induction after drugs
which depress AV nodal conduction (ouabain and
propranolol) (Wu et al., 1974; Wu et al., 1975).
This probably reflects the fact that some of these
smooth curve patients have concealed extranodal
pathways underlying their paroxysmal suprance-
tricular tachycardia (Wu and Denes, 1975). The
administration of a drug depressing the AV node
allows a given atrial extrastimulus to provoke greater
AV nodal delays after drug administration. This
additional delay potentiates the echo phenomenon,
since the extranodal pathway is given more time for
recovery from antegrade concealed penetration.
Drugs such as procainamide and quinidine, which
depress antegrade anomalous pathway conduction,
should theoretically prolong retrograde refractori-
ness for the concealed pathway, so that these become
incapable of sustaining re-entrant paroxysmal supra-
ventricular tachycardia (Wellens and Durrer, 1974b).
However, the present patient was unres-
sponsive to procainamide and quinidine administra-
tion.

Both atrial and ventricular pacing have been used
with varying degrees of success in the management
of patients with intractable paroxysmal supranen-
tricular tachycardia (Kitchen and Golldrearyer,
1972). These were unsuccessful in the present case.
The failure of ventricular pacing appeared to
reflect the very narrow zone of coupling intervals
(5 ms), in which a ventricular extrastimulus could
interrupt sustained paroxysmal supraventricular
tachycardia. Long periods of random ventricular
pacing would be necessary before a random stimulus
fell within this critical zone. Programming of a
pacemaker to provide a stimulus in this zone could
also fail, since it is likely that the zone might well
change in timing from day to day.

Before the use of current diagnostic electro-
physiological techniques, surgical cure of in-
tractable paroxysmal supraventricular tachycardia
had been achieved by destruction of the His bundle
(Gianelly et al., 1967). Surgery has also been used
successfully in patients with manifest pre-excitation
to interrupt re-entrant circus movements (Sealy and
Wallace, 1974). Surgical ablation of anomalous
pathway after demonstration with epicardial map-
ning can prevent recurrent circus movement
paroxysmal supraventricular tachycardia (and also
prevent rapid ventricular rates during paroxysmal
atrial fibrillation). Similar cure of circus movement
tachycardia has also been achieved in patients with
pre-excitation occurring after surgical division of
the normal pathway (Dreifus et al., 1968; Wellens
et al., 1974).

This is the first reported case of a patient with
intractable re-entrant paroxysmal supraventricular
tachycardia caused by a proven concealed extranodal
pathway cured by surgery. In this case, we chose to
interrupt the His bundle, since this was the easiest
of the possible procedures for achieving cure of the
tachycardia. The fact that the patient was a
Jehovah's witness and could not receive blood
transfusions was another factor in reaching this
decision. This operation had the disadvantage of
producing chronic surgical heart block.

The other surgical approach to a case such as the
present one would be to ablate surgically the con-
cealed retrograde pathway. Theoretically, this would
be ideal, since it would leave the normal conduction
system intact without producing permanent heart
block. However, technical difficulties might be
anticipated in regard to localization of an anomalous
pathway when this pathway was only capable of
retrograde conduction. We believe that the feasi-
bility of surgery for retrograde concealed pathways
needs further exploration.

One additional point of interest relates to the
current electrophysiological status of the present
patient. He has chronic complete heart block with
intact retrograde conduction. He thus resembles
previously reported patients with chronic acquired
complete heart block and intact retrograde conduction
(Castillo and Samet, 1967; Goldreyer and
Bigger, 1970). Previous authors have commented on
the possibility that these patients have single
lesions producing unidirectional heart block. The
present case suggests that some patients with heart
block and intact retrograde conduction have ac-
quired heart block involving the normal pathway,
with a concealed retrograde pathway allowing
retrograde conduction.
We thank Dr. Bernard Kosowsky of St. Elizabeth's Hospital, Boston, for referring the patient, and Drs. Robert Hall and Denton Cooley for allowing us to perform the epicardial mapping at the Texas Heart Institute.

References


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