THE ELECTROPHYSIOLOGIC EFFECTS OF DISCRETE CRYOSURGERY FOR THE TREATMENT OF SUPRAVENTRICULAR TACHYCARDIA DUE TO AV NODAL REENTRY

MASATOSHI IKESHITA, M.D.*, WILLIAM L. HOLMAN, M.D., JOHN G. LEASE, B.A.
DAVID C. SABISTON, JR., M.D., AND JAMES L. COX, M.D.

The electrophysiologic effects of multiple cryosurgical lesions along the circumference of Koch's triangle were evaluated as a treatment of AV nodal reentrant tachycardia in 31 dogs. These animals were divided into a control group (n = 8) and a cryosurgical group (n = 23), and were studied using incremental atrial pacing and programmed atrial premature stimulation.

In the cryosurgical group, significant prolongation of AV nodal conduction time, initiation of Wenckebach periodicity at a slower atrial pacing rate, and an increase in the functional refractory period of the AV node were documented postoperatively. In contrast, there were no significant differences between preoperative and postoperative AV node conduction characteristics in the control group. Four dogs in which the effective refractory period of the AV node could be measured showed an increase of 28–104 msec following cryosurgery. In 2 dogs, discontinuous conduction curves (i.e., dual AV nodal pathways) were demonstrated preoperatively. Postoperatively, a uniform conduction curve was demonstrated. In conclusion, these results strongly suggest that this new cryosurgical method is an effective surgical procedure for the interruption of the reentrant circuit responsible for AV nodal reentry while preserving AV conduction.

REENTRY, as first described by Mines in 1912¹, is a basic mechanism of many tachyarrhythmias, and may occur in any part of the conduction system. The clearest verification of the presence of a reentrant circuit responsible for paroxysmal tachycardia is the surgical division of an accessory pathway in the Wolff-Parkinson-White syndrome.² Reentry occurring within the atrioventricular node (AV node) has been demonstrated electrophysiologically in

animals^{3–6} and in humans.^{7–9} Surgical interruption, cryoablation, or catheter ablation^{10–12} of the His bundle has been used for patients with medically refractory AV nodal reentry. These procedures do not abolish AV nodal reentry, but rather they confine it to the atria and protect the ventricles from the effects of the atrial tachycardia. In addition, these procedures require the implantation of a permanent ventricular pacemaker. The purpose of this study is to assess the effects of a new cryosurgical technique for the ablation of the AV nodal reentrant circuit itself while preserving atrioventricular conduction.

Key Words:

Dual AV nodal pathway
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The Department of Surgery, Duke University Medical Center, Durham, North Carolina, *Department of Thoracic and Cardiovascular Surgery, Nippon Medical School, Tokyo, Japan

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Mailing address: James L. Cox, M.D., Associate Professor of Surgery Duke University Medical Center Durham, North Carolina 27710

Fig.1. Multiple discrete cryosurgical technique. Multiple cryo-lesions along the circumference of Koch's triangle were produced from site 1 to site 9 sequentially during normo-thermic cardiopulmonary bypass.

METHODS AND MATERIALS

Experimental Protocol: Thirty-one adult, mongrel dogs were divided into control ($n = 8$) and cryosurgical groups ($n = 23$) to study the effects of cryosurgery on the AV node. Two dogs in the cryosurgical group were given reserpine (0.5 mg/kg/day for 2 days) and atropine (0.8 mg IV) preoperatively to block autonomic influences on the AV node. The animals, weighing 26–32 g, were anesthetized with morphine and thiamylal sodium (Surital) and were ventilated with 40% oxygen via an endotracheal tube attached to a volume-cycled respirator (Bennett MA-1). A right thoracotomy was performed through the 5th intercostal space, and bipolar atrial pacing electrodes were sewn to the right atrial epicardium 5 mm anterior to the sino-atrial node. Two bipolar electrodes were sewn to the epicardium of the right atrial appendage and right ventricular outflow tract for the recording of electrograms. A tripolar electrode catheter was introduced retrograde into the right carotid artery and positioned in the non-coronary sinus of the aortic valve for recording of the His bundle electrogram.¹³ Data from limb lead II, right atrial, His bundle, and right ventricular electrograms were simultaneously recorded during the electrophysiological studies. Limb lead II was recorded within a

range of 0.5 to 300 Hz and all other electrograms were recorded from 50 to 1000 Hz.

Following cannulation of the femoral artery, superior vena cava, and inferior vena cava, cardiopulmonary bypass was instituted to maintain esophageal temperature between 35 and 37°C. A right atriotomy was performed to expose the lower atrial septum. The area encompassed by Koch's triangle was mapped using a bipolar handheld electrode during atrial pacing for delineating the location of the His bundle.¹⁴ In the control group of animals, the cryoprobe (4 mm diameter) was applied to 9 separate sites along the Tendon of Todaro, the anulus of the tricuspid valve, and the region between the coronary sinus and the anulus of the tricuspid valve for 2 minutes each without cooling. In the cryosurgical group, cryolesions were produced at these sites by cryothermic exposure of 2 minutes at -60°C (Fig. 1). If complete AV block was observed during cryothermic exposure, cooling was terminated prematurely. The cryosurgical instrument (Frigitronics, Inc., Shelton, Conn.) utilized internally expanding nitrous oxide as the coolant. Following closure of the right atriotomy, all animals were weaned from cardiopulmonary bypass without the use of inotropic drugs. Potassium was administered for post cardiopulmonary bypass hypokalemia. Post-operative electrophysiologic data were obtained

TABLE I ELECTROPHYSIOLOGICAL DATA (IN MSEC) BEFORE AND AFTER MULTIPLE CRYOSURGERY

No.	BCL		AH		HV		FRP _{Atrium}		Antegrade				Wenckebach Periodicity	
									FRP _{AVN}		ERP _{AVN}			
	B	A	B	A	B	A	B	A	B	A	B	A	B	A
Control Group														
1	350	350	74	76	28	24	140	168	216	228	<140	<168	≤190	≤190
2	350	350	84	54	30	24	156	146	236	186	<156	<146	210	≤190
3	350	350	60	44	28	32	156	150	210	198	<156	<150	≤190	≤190
4	350	350	78	74	32	34	148	172	264	266	<148	<172	220	240
5	350	350	82	78	23	22	150	158	240	248	<150	<158	200	200
6	350	350	62	52	26	24	160	154	262	238	190	154	240	210
7	300	300	86	83	34	35	138	160	220	238	<138	<160	200	210
8	350	350	56	66	29	30	150	164	222	240	<150	<164	≤190	200
Cryosurgery group														
1	400	450	65	159	32	33	<160	<330	244** 296	404	190** 160	330	250	390
2	300	300	47*	85*	26*	25*	138	156	190	230	<138	<156	≤190	210
3	350	300	68	98	30	33	128	<148	232	280	<128	200	200	270
4	350	350	55	73	28	30	152	158	196	250	<152	<158	≤190	230
5	350	350	74	84	26	20	162	<190	226	296	<162	190	210	270
6	350	350	64	72	26	24	148	174	222	238	<148	<174	200	210
7	350	350	70	68	36	38	180	162	248	230	<180	<162	≤190	200
8	325	325	65*	80*	28*	26*	162	160	204	248	<162	<160	≤190	210
9	350	350	90	106	29	26	170	162	264	312	<170	<162	240	290
10	300	300	68	166	30	32	126	182	200	290	<126	230	≤190	280
11	350	350	55	92	38	35	168	176	216	240	<168	<176	≤190	210
12	350	350	50	80	24	26	160	180	226	266	<160	<180	≤190	260
13	350	350	66	105	30	30	150	148	234	240	<150	<148	≤190	210
14	350	350	52	65	38	34	170	140	236	246	<170	<140	≤190	200
15	350	350	63	79	37	34	148	170	214	240	<148	<170	≤190	200
16	350	350	71	100	40	34	160	142	216	232	<160	146	≤190	200
17	350	350	77	81	31	29	180	156	238	252	<125	<156	≤190	200
18	350	350	62	78	31	30	168	178	252	276	<168	<178	200	230
19	350	350	75	96	35	34	178	170	240	252	<178	<170	200	220
20	350	350	69	97	24	35	164	148	228	262	<164	<148	200	220
21	350	350	80	104	40	42	196	172	248	272	<196	<172	200	250
Cryosurgery Group with Administration of Reserpine														
1	350	350	108	196	33	34	186	214	290	396	<186	280	260	360
2	350	350	89	108	36	29	174	174	300** 374	318	240** 200	260	290	300
	400	400	81	98	30	30	180	184	304** 378		240** 200			

Abbreviation: BCL = basic cycle length; AH = AH interval; HV = HV interval; FRP_{Atrium} = functional refractory period of atrium; FRP_{AVN} = functional refractory period of A-V node; ERP_{AVN} = effective refractory period of A-V node; Wenckebach Periodicity = longest atrial cycle length at which Wenckebach periodicity occurred; B = before surgery; A = after surgery.

*Value which was measured at BCL of 350 msec

**Value of fast pathway (the upper row) and slow pathway (the lower row) which measured in the case with dual A-V nodal pathway.

Fig. 2. Effect of multiple discrete cryosurgical lesions on antegrade AV nodal conduction time. PRE-OP = preoperative; POST-OP = postoperative; NS = not significant.

Fig. 3. Effect of multiple discrete cryosurgical lesions on antegrade His-Purkinje conduction time. PRE-OP = preoperative; POST-OP = postoperative; NS = not significant.

at intervals up to 3 hours after application of the cryolesions.

Electrophysiological Studies and Definitions: Antegrade conduction properties of the AV node that were evaluated included the AV nodal conduction time (AH interval), the longest atrial cycle length at which Wenckebach periodicity occurred, and AV node refractory periods as evaluated by incremental atrial pacing and

Fig. 4. Effect of multiple discrete cryosurgical lesions on antegrade conduction through the AV node. Longest CL at Wenckebach = longest atrial cycle length at which Wenckebach periodicity occurred; triangles indicate values less than or equal to; circles indicate exact values; PRE-OP = preoperative; POST-OP = postoperative; NS = not significant.

programmed atrial premature stimulation.

Incremental atrial pacing was initiated at a cycle length (i.e., S_1-S_1 interval) slightly less than the sinus cycle length, and the cycle length was then decreased by 10 msec decrements down to a cycle length of 200 msec. Programmed atrial premature stimulation was introduced after every eighth paced beat. The coupling interval (S_1-S_2 interval) was decreased by 5 or 10 msec decrements until the effective refractory period of the atrium was reached. The pacing stimuli were approximately twice diastolic threshold and 2 msec in duration.

The responses recorded on the His bundle electrogram to a basic pacing stimulus (S_1) were designated A_1 for the lower right atrial septum, H_1 for the His bundle, and V_1 for the ventricle. The responses to an atrial premature stimulus (S_2) were designated A_2 , H_2 , and V_2 for the same regions respectively. AV conduction curves were generated from programmed atrial premature stimulation data by plotting the H_1-H_2 intervals or A_2-H_2 intervals against the A_1-A_2 intervals. The effective refractory period (ERP) of the AV node was defined as the longest A_1-A_2 interval that did not propagate the atrial impulse to the His bundle. The functional

Fig.5. Effect of multiple discrete cryosurgical lesions on functional refractory period of the AV node (AV node FRP). PRE-OP = preoperative; POST-OP = postoperative; NS = not significant.

refractory period (FRP) of the AV node was the shortest H_1-H_2 interval at which propagation occurred through the His bundle. Dual AV nodal pathways were defined by a discontinuous conduction curve that showed the H_1-H_2 interval increasing by at least 40 msec over a decrement of 10 msec or less in the A_1-A_2 interval with a concomitant increase in the conduction time represented by the A_2-H_2 interval and no change in the H_2-V_2 interval. The portion of the curve to the right of the discontinuity in the H_1-H_2 interval represents the fast pathway, and to the left, the slow pathway.^{7,9} The effective and functional refractory periods of the fast and slow pathways were defined as previously described.

All electrophysiologic data obtained were recorded on magnetic tape (Ampex) through bioelectric amplifiers (Hewlett-Packard Model 8811A) and were directly recorded on an 8-channel Elema mingograph recorder at a paper speed of 250 mm/sec.

RESULTS

Electrophysiologic data including antegrade AV conduction times, the longest atrial cycle length at which Wenckebach periodicity occurred,

Fig.6. Demonstration of preoperative dual AV nodal pathway utilizing programmed atrial premature stimulation. Shortening of the A_1-A_2 intervals results in lengthening of the A_2-H_2 interval (Panel A). When a sudden increase in the A_2-H_2 and H_1-H_2 intervals are seen at an A_1-A_2 interval of 240 msec (Panel B), conduction occurs through the slow pathway. At an A_1-A_2 interval of 200 msec, the premature atrial impulse is blocked on the slow pathway above the His bundle (Panel C).

the effective and functional refractory periods of the AV node, and conduction curves were obtained in all animals (Table I).

Atrioventricular Conduction Characteristics

AV conduction times at an atrial paced cycle length of 350 msec were measured in the 7 animals in the control group and 18 animals in the cryosurgical group. In the control group, the AH interval was 71 ± 4.3 msec preoperatively and 63 ± 5.1 msec postoperatively ($p = NS$) (Fig. 2). In the cryosurgery group, the AH interval increased from 66 ± 2.5 msec preoperatively to 85 ± 3.1 msec postoperatively ($p < 0.001$). Comparison of the postoperative AH intervals in the control group (63 ± 5.1 msec), to the AH interval in the cryosurgical group (85 ± 3.1 msec) showed a significant increase of the AH interval in the cryosurgery group

Fig. 7. Preoperative and postoperative conduction curves demonstrating the ablation of dual AV nodal conduction. Premature atrial coupling intervals (A_1-A_2) are plotted against H_1-H_2 intervals (left panel) and A_2-H_2 intervals (right panel). The slow pathway that was demonstrated preoperatively was not present postoperatively. Open circles indicate the preoperative response to premature stimulation; closed circles indicate postoperative responses to premature stimulation. Fast = fast pathway; Slow = slow pathways; FRP = functional refractory period of AV node; ERP = effective refractory period of AV node.

($p < 0.001$).

In the control group, the HV interval was 28 ± 1.1 msec preoperatively and 27.1 ± 1.8 msec postoperatively ($p = \text{NS}$) (Fig. 3). In the cryosurgery group, the HV interval was 31.5 ± 1.3 msec preoperatively and remained unchanged at 30.7 ± 1.3 msec postoperatively ($p = \text{NS}$). Likewise, comparison of the postoperative HV intervals between the control group (27.1 ± 1.8 msec) and cryosurgery group (30.7 ± 1.3 msec) showed no significant difference ($p = \text{NS}$). In the control group, the longest atrial cycle length at which Wenckebach periodicity occurred was 205 ± 6.3 msec preoperatively and 203.8 ± 6.0 msec postoperatively ($p = \text{NS}$) (Fig. 4). In the cryosurgery group, the longest atrial cycle length at which Wenckebach periodicity occurred was 196 ± 2.6 msec preoperatively and 228 ± 30.1 msec postoperatively ($p < 0.001$). The atrial cycle length causing Wenckebach periodicity postoperatively in the control group (203.8 ± 6.0 msec) was significantly less than that causing Wenckebach periodicity postoperatively in the cryosurgery group (228 ± 30.1 msec) ($p < 0.05$).

Refractory Period Determinations: The FRP of the AV node was measured in all animals and was calculated at the same paced cycle length of 350 msec (Fig. 5). In the control group, the

FRP of the AV node was 236 ± 8.1 msec preoperatively and 229 ± 10.6 msec postoperatively ($p = \text{NS}$). In the cryosurgery group, the FRP of the AV node was 231 ± 4.3 msec preoperatively and 256 ± 5.8 msec postoperatively ($p < 0.001$). The postoperative FRP of the AV node in the control group (229 ± 10.6 msec) was significantly less than that in the cryosurgery group (256 ± 5.8 msec) ($p < 0.05$).

The change between preoperative and postoperative ERP of the AV node could be measured in only one animal in the control group and in 4 animals in the cryosurgery group. In the remaining animals, the ERP of the AV node could not be measured because of the shorter FRP of the atrium. In one animal in the control group, the postoperative ERP of the AV node decreased by 36 msec as compared with the preoperative value. In the 4 animals in the cryosurgical group, the postoperative ERP of the AV node showed increases of 28, 72, 94, and 104 msec for each animal as compared with the preoperative value.

Dual Atrioventricular Nodal Pathways (Table I): Preoperative AV nodal conduction curves were continuous in 29 of the 31 animals. In the remaining 2 animals, both in the cryosurgical group, discontinuous conduction curves, suggesting dual AV nodal pathways, occurred

without atrial echoes as demonstrated by programmed atrial premature stimulation. Following cryosurgery, only a single continuous conduction curve was demonstrated in each animal. These postoperative conduction curves were shifted upward and to the right on the graphs of the H_1-H_2 or the A_2-H_2 intervals plotted against the A_1-A_2 intervals. In Dog 23 of the cryosurgery group, discontinuous conduction curves were obtained at both basic cycle lengths of 350 and 400 msec preoperatively. With a basic cycle length of 400 msec, progressive shortening of the H_1-H_2 intervals and lengthening of the A_2-H_2 intervals were observed as the A_1-A_2 intervals were shortened down to an interval of 250 msec. At that point, the H_1-H_2 and A_2-H_2 intervals were 308 and 140 msec respectively. At an A_1-A_2 interval of 240 msec, sudden prolongation of the H_1-H_2 interval to 378 msec and the A_2-H_2 to 220 msec was observed. At A_1-A_2 intervals of 240–210 msec, long H_1-H_2 and A_2-H_2 intervals were present with conduction block from the atrium to the His bundle occurring at an A_1-A_2 interval of 200 msec. Atrial echoes were not observed at any A_1-A_2 interval (Fig. 6). The conduction curves thus demonstrated the presence of both a fast and a slow conduction pathway in the AV node. The fast pathway had an FRP of 304 msec and an ERP of 240 msec. The slow pathway had an FRP of 378 msec and an ERP of 200 msec.

Postoperatively, progressive shortening of the H_1-H_2 interval and lengthening of the A_2-H_2 interval greater than the preoperative values were obtained until an A_1-A_2 interval of 270 msec was reached. At an A_1-A_2 interval of 260 msec, conduction block from the atrium to the His bundle developed while atrial capture persisted. Postoperative conduction showed a single continuous curve with an FRP of 318 msec and ERP of 260 msec. This curve was shifted upward and to the right as compared with the preoperative fast pathway conduction curve (Fig. 7).

DISCUSSION

Until recently, ablation of the His bundle was the most frequently employed surgical treatment for refractory paroxysmal supraventricular tachycardia due to AV nodal reentry.^{10–12} This surgical procedure has been largely replaced in the past 18 months by catheter ablation of the His bundle.¹² While this technique does not

require a formal operation, it is not without risks and it still requires that a permanent ventricular pacemaker system be implanted. Pritchett, et al¹⁵ reported one clinical case in which surgical dissection of the lower atrial septum in the region of the AV node inadvertently abolished paroxysmal supraventricular tachycardia due to AV nodal reentry while preserving AV conduction. The authors postulated that this procedure might disrupt the delicate time relationships required to maintain the tachycardia. However, the surgical results in that patient were fortuitous, and no definite method for reproducing that result has been developed. In this paper, we report a new cryosurgical technique confined to the lower atrial septum that is directed towards ablating the reentrant circuit while preserving AV conduction. The effects of this procedure on the AV node were investigated in detail electrophysiologically.

Effect of Multiple Discrete Cryolesions on the AV Node with a Single Continuous Conduction Curve

In 29 animals with preoperative single, continuous conduction curves as demonstrated by programmed atrial premature stimulation, multiple discrete cryolesions placed around Koch's triangle produced marked prolongation of antegrade AV nodal conduction, produced Wenckebach periodicity at slower atrial pacing rates, and increased the refractory period of the AV node while preserving atrioventricular conduction. These electrophysiologic findings after cryosurgery of the lower atrial septum suggest that the cryolesions may alter the functional properties of the AV node and/or of the AV node input pathways sufficiently to prevent AV nodal reentry. In addition, these alterations in AV conduction may decrease the ventricular response rate to supraventricular tachycardia and may ultimately be beneficial in the treatment of a wide variety of non-AV node reentry supraventricular tachycardia without requiring permanent pacemaker system implantation.

Effect of Multiple Discrete Cryolesions on the AV Node with a Discontinuous Conduction Curve

Preoperative discontinuous conduction curves were demonstrated in two animals suggesting that they had dual AV nodal pathways. However, neither a single atrial echo nor sustained AV

nodal reentrant tachycardia could be induced in either animal.

As described by Moe and Mendez^{3,4} the atrial echo phenomenon and AV nodal reentrant tachycardia occur on the basis of the formation of a reentrant circuit within the AV nodal region that is thought to be due to longitudinal dissociation of the AV node into two pathways with different functional properties. Experimental studies utilizing action potential recordings from microelectrodes in the AV nodal region have also shown evidence for AV nodal reentrant circuits.^{5,6} In addition, clinical studies with His bundle recordings during programmed atrial premature stimulation have demonstrated evidence supporting dual AV nodal pathways due to functional longitudinal dissociation in association with AV nodal reentrant tachycardia.^{7,8}

One reported series of patients with paroxysmal supraventricular tachycardia revealed that 69% had AV nodal reentrant tachycardia and 68% of these patients showed discontinuous conduction curves typical of dual AV nodal pathways.⁸ Therefore, the presence of a dual AV nodal pathway is associated with the occurrence of AV nodal reentrant tachycardia.

Generally the slow (α) pathway demonstrates slower conduction and a shorter refractory period than the fast (β) pathway. Both pathways seem to be linked by proximal and distal common pathways to form the reentrant circuit. If antegrade conduction is blocked in the fast pathways, conduction occurs down the slow pathway. If the conduction time down the slow pathway exceeds the effective refractory period of the fast pathway, then retrograde conduction will occur over the fast pathway. This returning impulse results in an atrial echo, and if it is again conducted down the slow pathway, sustained tachycardia will occur.⁹ Therefore, the prevention or termination of AV nodal reentrant tachycardia could be achieved by: 1) depressing or blocking the antegrade slow pathway conduction or the retrograde fast pathway conduction, 2) prolongation of the effective refractory period in a part of the reentrant circuit in excess of the total conduction time of the reentrant circuit, or 3) disruption of the delicate time balance required to maintain the reentry by altering the conduction time and refractory period.

In two animals with preoperative dual AV nodal pathways, multiple discrete cryolesions of the AV nodal region resulted in a single continu-

ous conduction curve postoperatively. This curve was shifted upward and to the right as compared to the preoperative curve. The slow pathway was, therefore, apparently ablated by the cryosurgery although AV conduction was preserved.

The observations suggest that the multiple discrete cryolesions either: 1) functionally blocked the antegrade conduction in the slow pathway, 2) equalized the effective refractory periods of the fast and slow pathway, or 3) made the effective refractory period of the slow pathway greater than that of the fast pathway.

We support the idea of actual ablation of the slow pathway because the cryolesions are known to produce homogeneous well-demarcated myocardial injury.¹¹ Whether or not any part of the atrium is required to initiate or maintain AV nodal reentrant tachycardia is a point of controversy that has not yet been resolved in experimental animals or in humans.^{4-6,16} Regardless of its precise anatomic location, the present study suggests that the multiple cryolesions most likely ablate a portion of the reentrant circuit that is responsible for AV nodal reentrant tachycardia.¹⁷

The clinical application of this new technique may not be practicable at this present yet for reasons that an evidence to abolish and prevent the attacks of AV nodal reentrant tachycardia is not provided actually and several of nine cryolesions may not be needed to achieve the same results in the present study.

REFERENCES

1. MINES GR: On dynamic equilibrium in the heart. *J Physiol* 46: 349, 1913
2. SEALY WC, WALLACE AG, RAMMING KP, GALLAGHER JJ, SVENSON RH: An improved operation for the definitive treatment of the Wolff-Parkinson-White syndrome. *Ann Thorac Surg* 17: 107, 1974
3. MOE GK, PRESTON JB, BURLINGTON H: Physiologic evidence for a dual A-V transmission system. *Circ Res* 4: 357, 1956
4. MENDEZ C, MOE GK: Demonstration of a dual A-V nodal conduction system in the isolated rabbit heart. *Circ Res* 19: 378, 1966
5. WIT AL, GOLDREYER BN, DAMATO AN: An in vitro model of paroxysmal supraventricular tachycardia. *Circulation* 43: 862, 1971
6. JANSE MH, VAN CAPELLE FJL, FREUD GE, DURRER D: Circus movement within the A-V node as a basis for supraventricular tachycardia as shown by multiple microelectrode recordings in the isolated rabbit heart. *Circ Res* 28: 403, 1971

7. DENES P, WU D, DHINGRA RC, CHUQUIMIA R, ROSEN KM: Demonstration of dual A-V nodal pathways in patients with paroxysmal supraventricular tachycardia. *Circulation* **48**: 549, 1973
8. WU D, DENES P, AMAT-Y-LEON F, DHINGRA R, WYNDHAM CRC, BAUERNFEIND R, LATIF P, ROSEN KM: Clinical, electrocardiographic and electrophysiologic observations in patients with paroxysmal supraventricular tachycardia. *Am J Cardiol* **41**: 1045, 1978
9. DENES P, WU D, AMAT-Y-LEON F, DHINGRA R, WYNDHAM CR, ROSEN KM: The determinants of atrioventricular re-entrance with premature atrial stimulation in patients with dual A-V nodal pathways. *Circulation* **56**: 253, 1977
10. SEALY WC, ANDERSON RW, GALLAGHER JJ: Surgical treatment of supraventricular tachyarrhythmias. *J Thorac Cardiovasc Surg* **73**: 511, 1977
11. KLEIN GJ, SEALY WC, PRITCHETT ELC, HARRISON L, HACKEL DB, DAVIS D, KASELL J, WALLACE AG, GALLAGHER JJ: Cryosurgical ablation of the atrioventricular node-His bundle: Long-term follow-up and properties of the junctional pacemaker. *Circulation* **60**: 440, 1979
12. GALLAGHER JJ, SVENSON RH, KASELL JH, GERMAN LD, BARDY GH, BROUGHTON A, CRITELLI G: Catheter technique for closed-chest ablation of the atrioventricular conduction system. *NEJM* **306**: 194, 1982
13. URTHALER F, JAMES TN: A comparison of His bundle electrograms recorded from the aortic root and from a plaque sutured near the His bundle. *J Lab Clin Med* **85**: 711, 1975
14. KRONGRAD E, MALM JR, BOWMAN FO, HOFFMAN, BF, KAISER GA, WALDO AL: Electrophysiological delineation of the specialized atrioventricular conduction system in patients with congenital heart disease. *J Thorac Cardiovasc Surg* **67**: 875, 1974
15. PRITCHETT ELC, ANDERSON RW, BENDITT DG, KASELL J, HARRISON L, WALLACE AG, SEALY WC: Reentry within the atrioventricular node: Surgical cure with preservation of atrioventricular conduction. *Circulation* **60**: 440, 1979
16. JOSEPHSON ME, KASTOR JA: Paroxysmal supraventricular tachycardia. Is the atrium a necessary link? *Circulation* **54**: 430, 1976
17. HOLMAN WL, IKESHITA M, LEASE JG, SMITH PK, FERGUSON TB, Jr, COX JL: Elective prolongation of atrioventricular conduction by multiple discrete cryolesions: A new technique for the treatment of paroxysmal supraventricular tachycardia. *J Thorac Cardiovasc Surg* **84**: 554, 1982