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Dual atrioventricular nodal pathways
A common electrophysiological response[1]

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Evidence of dual atrioventricular nodal pathways (a sudden jump in H_1–H_2 at critical A_1–A_2 coupling intervals) was shown in 41 out of 397 patients studied with atrial extrastimulus techniques. In 27 of these 41, dual pathways were demonstrable during sinus rhythm, or at a cycle length close to sinus rhythm (CL_1). In the remaining 14, dual pathways were only demonstrated at a shorter cycle length (CL_2). All patients with dual pathways at cycle length$_1$ who were also tested at cycle length$_2$ (11 patients) had dual pathways demonstrable at both cycle lengths. In these 11 patients both fast and slow pathway effective refractory periods increased with decrease in cycle length. Twenty-two of the patients (54%) had either an aetiological factor strongly associated with atrioventricular nodal dysfunction or one or more abnormalities suggesting depressed atrioventricular nodal function. Evaluation of fast pathway properties suggested that this pathway was intranodal.

Seventeen of the patients had previously documented paroxysmal supraventricular tachycardia (group 1). Eight patients had recurrent palpitation without documented paroxysmal supraventricular tachycardia (group 2), and 16 patients had neither palpitation nor paroxysmal supraventricular tachycardia (group 3). Echo zones were demonstrated in 15 patients (88%) in group 1, no patients in group 2, and 2 patients (13%) in group 3.

The normal atrioventricular nodal conduction curve relating A_1–A_2 to H_1–H_2, generated with atrial extrastimulus technique is smooth (Krayer, Mandoki, and Mendez, 1951; Mendez, Gruhzit and Moe, 1956; Hoffman *et al.*, 1963; Wit *et al.*, 1970; Goldreyer, 1972; Van Cappelle, Du Perron, and Durrer, 1971; Ferrier and Dresel, 1973, 1974). A broken curve, with abrupt reproducible increase in H_1–H_2 responses over a critical range of A_1–A_2 coupling intervals suggests the presence of dual atrioventricular nodal pathways (Moe, Preston, and Burlington, 1956; Mendez *et al.*, 1965; Schuilenburg and Durrer, 1969; Denes *et al.*, 1973; Rosen, Mehta, and Miller, 1974; Wu *et al.*, 1974). We have reported extrastimulus studies suggestive of dual atrioventricular nodal pathways in a patient with two PR intervals, and in a limited number of patients with atrioventricular nodal re-entrant paroxysmal supraventricular tachycardia (Denes *et al.*, 1973; Rosen *et al.*, 1974; Wu *et al.*, 1974). In these earlier studies, the presence of dual atrioventricular nodal pathways was related to the patients' clinical and/or electrocardiographic findings.

Our recent experience suggests that electrophysiological responses suggestive of dual atrioventricular nodal pathways are a common occurrence and often without apparent relation to the patients' clinical or electrocardiographic findings. In this report, we describe our total experience with patients having extrastimulus studies suggestive of dual atrioventricular nodal pathways. Clinical, electrocardiographic, and electrophysiological observations are reported in this group, in order to elucidate further the significance of this interesting electrophysiological phenomenon.

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Subjects and methods

Forty-one patients out of a total number of 397 patients

studied with atrial extrastimulus technique between July 1971 and July 1974 showed electrophysiological evidence of dual atrioventricular nodal pathways. These are the subject of the present report. Patients with electrocardiographic evidence of pre-excitation (short PR interval (<0.12 s), and/or presence of a delta wave) were excluded. Historical, physical, electrocardiographic, and electrophysiological findings were reviewed in each patient. Of these 41 patients, 8 have been the subjects of previous reports (Denes *et al.*, 1973; Rosen *et al.*, 1974; Wu *et al.*, 1974).

Electrophysiological studies were performed in the post-absorptive, non-sedated state. All cardiac drugs were discontinued 48 hours before study. Informed consent was obtained from each patient. His bundle electrograms were recorded by a catheter technique (Dhingra, Rosen, and Rahimtoola, 1973). Refractory periods were measured with the atrial extrastimulus method during sinus rhythm, or at a cycle length slightly shorter than sinus (CL_1) (Denes *et al.*, 1973). In 25 patients, the effect of shortening of cycle length on refractory periods was also examined. In these patients, a second paced cycle length (CL_2) was selected, which was shorter than CL_1, but longer than the cycle length inducing atrioventricular nodal Wenckebach periods.

Definitions

Established criteria for electrocardiographic diagnosis of myocardial infarction and arrhythmias were used (Lipman and Massie, 1965; Katz and Pick, 1956). 'Documented arrhythmias' were defined as spontaneous arrhythmias with electrocardiographic documentation. Two PR intervals were diagnosed when surface electrocardiograms revealed two non-overlapping ranges of PR intervals (Rosen *et al.*, 1974).

Dual atrioventricular nodal pathways were defined with atrial extrastimulus technique. Dual pathway curves were characterized by a sudden jump in H_1–H_2 at a critical range of A_1–A_2 coupling intervals (Denes *et al.*, 1973; Rosen *et al.*, 1974; Wu *et al.*, 1974). None of these curves conformed to previously described normal atrioventricular nodal curves. The portion of the curve to the right of the jump represented the fast pathway, and to the left, the slow pathway. The slope of the slow pathway could be positive (decrease in H_1–H_2 with decreasing A_1–A_2 intervals), flat (no change in H_1–H_2 with decreasing A_1–A_2), or negative (increase in H_1–H_2 with decreasing A_1–A_2). Though repeated scanning with the atrial test stimuli close to the A_1–A_2 interval producing the jump frequently demonstrated an overlap of H_1–H_2 corresponding to the fast and slow pathways, intermediate H_1–H_2 intervals were not elicited in this zone of overlap (Rosen *et al.*, 1974). Curve fitting analysis was used in any of the cases where the diagnosis of dual pathways was equivocal (Denes *et al.*, 1973).

The fast pathway effective refractory period was defined as the longest A_1–A_2 that failed to conduct via the fast pathway, even in those cases where overlap between fast and slow pathway curves occurred. The fast pathway functional refractory period was defined as the shortest attainable H_1–H_2 interval on the fast pathway curve. The effective refractory period of the slow pathway was the longest A_1–A_2 not conducted via the slow pathway. When atrial functional refractory period limited slow pathway conduction, the slow pathway effective refractory period could not be measured, and was considered to be less than the shortest conducted A_1–A_2 interval. The slow pathway functional refractory period was the shortest attainable H_1–H_2 interval on the slow pathway curve.

Atrioventricular nodal re-entry was diagnosed by noting atrial echoes with low to high activation sequence related to achievement of a critical AH interval (Goldreyer, 1972; Denes *et al.*, 1973; Wu *et al.*, 1974). This occurred with or without sustained paroxysmal supraventricular tachycardia (Wu *et al.*, 1974).

Results

Clinical data (Table 1)

There were 26 male and 15 female patients, mean age 50 years (range 13 to 81 years). Eighteen (44%) patients had no evidence of organic heart disease, 7 (17%) had arteriosclerotic cardiovascular disease, 7 (17%) had hypertensive cardiovascular disease, 3 (7.3%) had primary conduction disease (intraventricular conduction defect without other evidence of organic heart disease), 3 (7.3%) had congenital heart disease (1 postoperative ventricular septal defect, 1 postoperative coarctation of the aorta, and 1 secundum atrial septal defect), and 3 (7.3%) had hypothyroidism. Cardiomegaly was present in 32 per cent (13/41) of the patients.

The resting electrocardiogram was normal in 11 (27%) of the patients, and 15 (37%) had an intraventricular conduction defect. (The high incidence of patients with bundle-branch block presumably

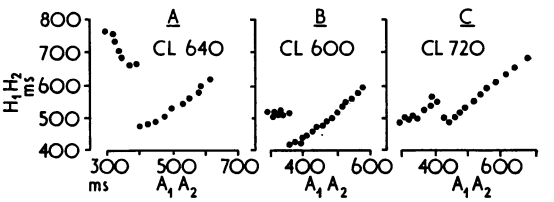

FIG. 1 *Three types of dual atrioventricular nodal curves. In each panel, A_1–A_2 coupling intervals are plotted on the abscissa and H_1–H_2 responses on the ordinate. CL = cycle length. Intervals are expressed in ms. Note a sudden jump in H_1–H_2 at a critical A_1–A_2 coupling interval. The portion of the curve to the right of the jump represents the fast pathway, and to the left, the slow pathway. Panel A shows a slow pathway curve with negative slope, Panel B shows a slow pathway curve with flat slope, and Panel C shows a slow pathway curve with positive slope.*

TABLE 1 *Clinical and electrocardiographic findings*

Case No.	*Age*	*Sex*	*Symptom*	*Chest x-ray heart size*	*Cardiovascular diagnosis*	*ECG*	*Documented arrhythmia*
1	50	F	Palpitation	Normal	Hypothyroidism	Low voltage, ST-T	PSVT
2	55	M	Palpitation	Normal	NHD	WNL	PSVT
3	62	F	Palpitation	Normal	NHD	ST-T	PSVT
4	44	M	Palpitation	Normal	NHD	ST-T	PSVT
5	62	M	Palpitation	Normal	HCVD	AMI, 1° AVB, LAD	PSVT
6	68	F	Palpitation	Normal	NHD	ST-T	PSVT
7	52	M	Palpitation	Normal	NHD	WNL	PSVT
8	50	M	Palpitation	Normal	NHD	WNL	PSVT
9	54	M	Palpitation	Normal	NHD	WNL	PSVT
10	20	F	Palpitation	Normal	NHD	WNL	PSVT
11	60	F	Palpitation	Normal	NHD	ST-T	PSVT
12	63	F	Palpitation	Normal	NHD	ST-T	PSVT
13	51	F	Palpitation	Normal	Hypothyroidism	WNL	PSVT
14	32	M	Palpitation	Normal	NHD	WNL	PSVT
15	22	F	Palpitation	Normal	ASD	IRBBB	—
16	28	M	Palpitation	Normal	NHD	WNL	PSVT
17	26	M	Palpitation	Normal	NHD	WNL	PSVT
18	73	M	Palpitation	Cardiomegaly	HCVD	LBBB, 1° AVB	PSVT
19	32	F	Palpitation	Normal	NHD	Low atrial rhythm	—
20	22	F	Palpitation	Normal	NHD	WNL	—
21	68	M	Palpitation	Normal	NHD	WNL	—
22	54	F	Palpitation	Cardiomegaly	HCVD, SSNS	ST-T	Sinus bradycardia, atrial flutter
23	55	M	Palpitation	Cardiomegaly	ASHD	IMI	Ventricular tachycardia
24	59	M	Angina, palpitation	Cardiomegaly	ASHD	IMI	Atrial flutter
25	40	M	Dyspnoea	Cardiomegaly	ASHD	IMI	—
26	64	M	Dyspnoea	Cardiomegaly	ASHD	CRBBB and RAD	—
27	54	M	Dyspnoea	Cardiomegaly	Post-surgical coarctation	LVH	—
28	68	M	Angina, palpitation	Cardiomegaly	ASHD	CRBBB and LAD, IMI	Ventricular tachycardia
29	58	M	Angina	Normal	ASHD	IMI	—
30	70	F	Dyspnoea	Cardiomegaly	HCVD	CRBBB and LAD	—
31	58	M	Dyspnoea	Cardiomegaly	HCVD	CRBBB and LAD, 1° AVB	
32	59	M	Angina	Normal	ASHD	RDBBB	—
33	81	F	Syncope	Normal	PCD, SSNS	CRBBB and LAD	Sinus bradycardia
34	22	M	Asymptomatic	Normal	PCD	CRBBB and LAD	—
35	22	M	Asymptomatic	Normal	PCD	CRBBB and LAD	—
36	21	F	Asymptomatic	Normal	NHD	1° AVB	—
37	13	M	Asymptomatic	Normal	Post-surgical VSD	CRBBB and LAD	—
38	98	M	Dyspnoea	Cardiomegaly	HCVD	CRBBB and LAD	—
39	24	F	Dyspnoea	Normal	NHD	1° AVB	—
40	67	M	Dyspnoea	Cardiomegaly	Hypothyroidism	CRBBB and LAD	—
41	69	M	Dizziness	Cardiomegaly	HCVD	LBBB	Sinus bradycardia

Abbreviations: ASHD = arteriosclerotic heart disease; HCVD = hypertensive cardiovascular disease; PCD = primary conduction disease; NHD = no heart disease; ASD = atrial septal defect; VSD = ventricular septal defect; CRBBB = complete right bundle-branch block; LAD = left axis deviation; IMI = inferior wall myocardial infarction; AMI = anterior wall myocardial infarction; LVH = left ventricular hypertrophy; WNL = within normal limits; RAD = right axis deviation; ST-T = ST-T wave changes; IRBBB = incomplete right bundle-branch block; PSVT = paroxysmal supraventricular tachycardia; 1° AVB = first degree atrioventricular block; SSNS = sick sinus node syndrome; RDBBB = rate dependent bundle-branch block; LBBB = left bundle-branch block.

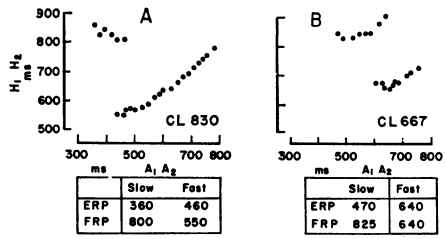

FIG. 2 *An example of the effect of shortening cycle length on fast and slow pathway refractory periods. CL=cycle length; ERP=effective refractory period; FRP=functional refractory period. Intervals are expressed in ms. Panels A and B represent dual A-V nodal curves at two different CL's in the same patient. Note the increase in ERP and FRP of both slow and fast pathways with decrease in CL.*

reflects the large number of patients with intraventricular conduction defect studied at our laboratory under an NIH contract.) In 3 patients, two PR intervals were observed on the surface electrocardiogram. One of these cases has been reported in detail in a separate publication (Rosen *et al.*, 1974).

Electrophysiological data

In 27 of the 41 patients, dual pathways were demonstrable with atrial extrastimulus technique at CL_1 (Fig. 1 and 2A). Of these 27, 11 were also tested at a shorter paced cycle length (CL_2), and all had dual atrioventricular nodal pathways demonstrable (Fig. 2B). In 14 of the 41 patients, atrioventricular nodal conduction curves were smooth at CL_1, and dual pathways were only demonstrated at CL_2 (Fig. 3A and B).

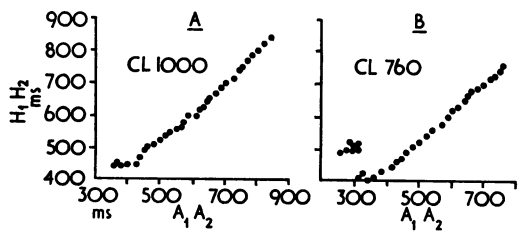

FIG. 3 *Unmasking of dual atrioventricular nodal curves with decreasing cycle length. Panel A shows a smooth atrioventricular nodal curve at a CL of 1000 ms. Panel B shows dual pathway curves in the same patient at a shorter CL (760 ms).*

In the 27 patients with dual pathway curves at CL_1, AH interval during sinus rhythm (fast pathway conduction time) ranged from 62 to 240 ms (mean $\pm$ SD 109 $\pm$ 44 ms). The fast pathway effective refractory period in these patients ranged from 290 to 610 ms (417 $\pm$ 94 ms). The fast pathway functional refractory period ranged from 400 to 760 ms (510 $\pm$ 97 ms). The slow pathway effective refractory period could be measured in 16 of the 27 patients with dual pathways at CL_1 and ranged from 285 to 530 ms (353 $\pm$ 87 ms). In the remaining 11 patients with dual pathways at CL_1, atrioventricular conduction was atrial limited. The slow pathway functional refractory period in these 27 patients ranged from 450 to 870 ms (618 $\pm$ 131 ms). The slow pathway had a negative slope in 8 (30%) patients (Fig. 1A), a flat slope in 14 (52%) patients (Fig. 1B), and a positive slope in 5 (18%) patients (Fig. 1C).

In the 14 patients with smooth curves at CL_1, AH intervals during sinus rhythm ranged from 72 to 180 ms (95 $\pm$ 28 ms). Atrioventricular nodal effective refractory period could only be measured in 4, and ranged from 245 to 380 ms (309 $\pm$ 55 ms). In the remaining 10 patients, atrioventricular conduction was atrial limited. Atrioventricular nodal functional refractory period in the 14 patients ranged from 390 to 530 ms (453 $\pm$ 62 ms).

In the 14 patients with dual pathways only at CL_2, fast pathway effective refractory period and functional refractory period ranged from 265 to 500 ms (361 $\pm$ 68), and 310 to 590 ms (446 $\pm$ 81 ms), respectively. Slow pathway effective refractory period could be measured in 7, and ranged from 300 to 420 ms (346 $\pm$ 59 ms). In the remaining 7 patients, atrioventricular conduction was atrial limited. Slow pathway functional refractory period ranged from 400 to 740 ms (554 $\pm$ 111 ms).

In the 11 patients in whom dual pathway curves were obtained at both CL_1 and CL_2, the effect of decrease in CL on the fast and slow pathway refractory periods could be determined. Mean $\pm$ SD CL_1 and CL_2 in these 11 patients were, respectively, 768 $\pm$ 76 and 641 $\pm$ 44 ms. The mean $\pm$ SEM fast pathway effective refractory period at CL_1 and CL_2 were, respectively, 391 $\pm$ 29 and 428 $\pm$ 37 ms ($P < 0.05$) (Fig. 2A and B, and Fig. 4). The mean fast pathway functional refractory periods at CL_1 and CL_2 were, respectively, 501 $\pm$ 28 and 489 $\pm$ 20 ms (NS). The mean slow pathway effective refractory periods at CL_1 and CL_2 were, respectively, 341 $\pm$ 36 and 374 $\pm$ 46 ms ($P < 0.1$) (Fig. 2A and B, and Fig. 4). The mean slow pathway functional refractory periods at CL_1 and CL_2 were, respectively, 612 $\pm$ 33 and 621 $\pm$ 36 ms (NS).

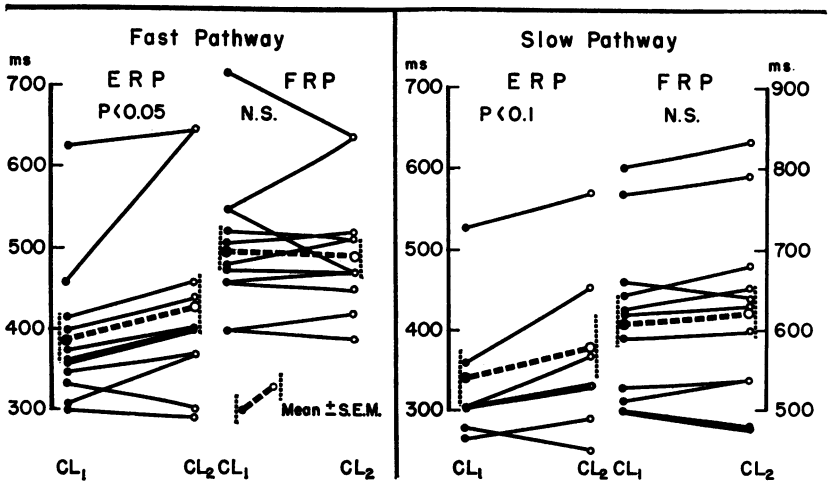

FIG. 4 *The effect of decreasing cycle length on fast and slow pathway refractory periods. Mean ± SEM CL_1 and CL_2 are, respectively, 768 ± 24 and 641 ± 13 ms. Shown in each panel are individual values for each patient, as well as the mean ± SEM (broken line) at CL_1 and CL_2 for the group (11 patients). Slow pathway ERP could only be determined in 7 patients, because atrial FRP limited atrioventricular conduction in 4. Note the significant increase in fast pathway ERP with shortening of CL.*

TABLE 2 *Atrioventricular nodal dysfunction*

Case No.	*Aetiology*	*AH (ms)*	*ERP (ms)*	*FRP (ms)*	*Wenckebach (beats/min)*
5	HCVD	240	480 (SP)	650 (FP)	110
6	NHD	106	285 (SP)	550 (FP)	160
7	NHD	100	310 (SP)	520 (FP)	120
9	NHD	64	340 (SP)	540 (FP)	108
12	NHD	62	290 (SP)	525 (FP)	180
16	NHD	92	380 (AVN)	510 (AVN)	140
18	HCVD	180	480 (AVN)	530 (AVN)	130
20	NHD	106	360 (SP)	550 (FP)	120
21	NHD	80	<390 (AVN)	520 (AVN)	160
23	IMI	85	245 (AVN)	310 (AVN)	200
24	IMI	74	300 (SP)	455 (FP)	160
25	IMI	104	310 (SP)	510 (FP)	140
26	IMI	70	<340 (AVN)	420 (AVN)	160
27	Post-surg. coarct.	90	<280 (AVN)	390 (AVN)	160
28	IMI	80	370 (SP)	490 (FP)	140
29	IMI	170	530 (SP)	720 (FP)	110
30	HCVD	120	<370 (SP)	700 (FP)	100
31	HCVD	153	<285 (SP)	475 (FP)	160
34	PCD	84	<470 (AVN)	520 (AVN)	120
37	Post-surgical VSD	110	460 (SP)	520 (FP)	120
39	NHD	232	370 (SP)	760 (FP)	110
40	Hypothyroidism	110	<350 (SP)	530 (FP)	130

Abbreviations: ERP = effective refractory period; FRP = functional refractory period; SP = slow pathway; FP = fast pathway.

Relation of dual pathways to presence of atrioventricular nodal dysfunction (Table 2)

Twenty-two patients (54%) had either an aetiological factor strongly associated with atrioventricular nodal dysfunction or one or more electrophysiological abnormalities suggesting depressed atrioventricular nodal function. Six patients (15%) had old inferior wall myocardial infarction (De Soyza *et al.*, 1974), and 2 patients (5%) had previous surgery for correction of congenital anomalies.

Electrophysiological evidence for atrioventricular nodal dysfunction was considered to be present on the basis of one or more of the following: 1) prolonged AH interval (> 130 ms) during sinus rhythm (Dhingra *et al.*, 1973) which was found in 5 patients (12%). 2) Prolonged atrioventricular nodal effective refractory period or slow pathway effective refractory period (> 380 ms) at CL_1 (Denes *et al.*, 1974a), which was found in 4 patients (10%). 3) Prolonged atrioventricular nodal functional refractory period or fast pathway functional refractory period (> 507 ms) at CL_1 (Denes *et al.*, 1974a), which was found in 16 patients (39%). 4) Atrial pacing rates of 110 or less inducing atrioventricular nodal Wenckebach periods (Dhingra *et al.*, 1973), which was found in 5 patients (12%). Seventeen patients (41%) had one or more electrophysiological abnormalities of atrioventricular nodal function.

Relation to paroxysmal supraventricular tachycardia

Seventeen patients had electrocardiographic documentation of spontaneous paroxysmal supraventricular tachycardia (group 1), 8 patients had recurrent paroxysmal palpitation without documentation of spontaneous paroxysmal supraventricular tachycardia (group 2), and 16 patients had neither palpitation nor documented paroxysmal supraventricular tachycardia (group 3).

In 15 of the 17 patients (88%) in group 1, atrioventricular nodal re-entrance with echo zones could be demonstrated during electrophysiological studies. Single echoes were demonstrated in 5 patients and sustained paroxysmal supraventricular tachycardia was induced in 10 patients.

In 4 of the 8 patients in group 2, spontaneous arrhythmias other than paroxysmal supraventricular tachycardia were documented (paroxysmal ventricular tachycardia in 2 patients and paroxysmal atrial flutter in 2 patients). No arrhythmias were documented in the remaining 4 patients. Atrioventricular nodal re-entrance with atrial echoes or paroxysmal supraventricular tachycardia could not be induced in any of these 8 patients during electrophysiological studies.

In 2 of the 16 patients (13%) in group 3, single atrioventricular nodal re-entrant echoes without sustained paroxysmal supraventricular tachycardia were induced during electrophysiological studies.

Discussion

Rosenblueth (1958) and Moe *et al.* (1956), using extrastimulus technique, suggested that the atrioventricular node might undergo longitudinal dissociation into two pathways. Rosen *et al.* (1974) recently reported a patient with two PR intervals and dual atrioventricular nodal conduction times and refractory periods, findings strongly suggestive of dual atrioventricular nodal pathways. Denes *et al.* (1973), and Wu *et al.* (1974) reported atrioventricular nodal conduction curves (A_1–A_2, H_1–H_2) in patients with atrioventricular nodal re-entrant paroxysmal supraventricular tachycardia and normal PR intervals, suggesting dual atrioventricular nodal pathways. In these previous studies, the demonstration of dual atrioventricular nodal pathways was directly relevant to the patients' clinical or electrocardiographic findings.

The present study suggests that dual atrioventricular nodal pathways are a relatively common electrophysiological finding, being found in approximately 10 per cent of the patients undergoing extrastimulus testing in our laboratory. Though the patients studied in our laboratory frequently have conduction disease, the demonstration of dual pathways was frequently not anticipated and not always relevant to the patients' clinical or electrocardiographic findings.

In a patient with dual pathways, the ability to demonstrate these pathways depends upon the properties of the two pathways. The following conditions are necessary for the demonstration of dual pathways with atrial extrastimulus technique. 1) A fast pathway effective refractory period which is longer than the slow pathway effective refractory period; if the fast pathway had a shorter effective refractory period than the slow pathway, the slow pathway would be concealed. 2) An atrial functional refractory period which is shorter than the slow pathway effective refractory period; if the atria had a longer functional refractory period than the slow pathway effective refractory period, the slow pathway would be concealed.

The inability to demonstrate dual pathways during sinus rhythm in 14 patients of the present series may be explained by the above conditions. Shortening of cycle length decreased the atrial functional refractory period (Denes *et al.*, 1974a), and

as demonstrated in the present study, prolonged fast pathway effective refractory period. Both of these effects could allow unmasking of a slow pathway with atrial pacing, which was concealed at CL_1. This could account for demonstration of dual pathways only at CL_2 in some or all of these 14 patients. However, another factor must be considered. It also is possible that dual pathways were not present at longer cycle lengths (sinus rhythm) in these patients, and that non-homogeneity of atrioventricular conduction related to shortening of cycle lengths was a necessary prerequisite for functional dissociation of the atrioventricular node.

We have assumed that the slow pathway is intranodal. Whether the fast pathway is intranodal or extranodal (James tract or other bypass) is not yet clear (Denes, Wu, and Rosen, 1974b). The following fast pathway properties are consistent with an intranodal location. 1) An AH during sinus rhythm (fast pathway conduction time) which is within or greater than the normal range of AH intervals. 2) A fast pathway functional refractory period which is within or greater than the normal range for atrioventricular nodal functional refractory period. 3) A fast pathway effective refractory period which is within or greater than the normal range for atrioventricular nodal effective refractory period. 4) An increase in fast pathway effective refractory period with decrease in cycle length, the normal cycle length-refractory period relation of the atrioventricular node (Denes *et al.*, 1974a). Fast pathways in the present series conformed to the properties described above.

If both fast and slow pathways were intranodal, they could reflect either functional longitudinal dissociation, or anatomical septation of the node. Of the 41 patients reported in this study, 22 had atrioventricular nodal dysfunction as manifested by abnormal electrophysiological findings (prolonged conduction times or refractory periods) and/or aetiological factors directly associated with atrioventricular nodal injury (previous diaphragmatic infarction or intracardiac surgery) (De Soyza *et al.*, 1974). It is possible that in some of the patients, pathological lesions might be responsible for anatomical division of the node into two pathways. In the 3 patients with hypothyroidism, one could postulate myxoedematous infiltration of the atrioventricular node as a possible cause of dual pathways.

The presence of dual atrioventricular nodal pathways predisposes to the occurrence of atrioventricular nodal re-entrant paroxysmal supraventricular tachycardia (Denes *et al.*, 1973; Wu *et al.*, 1974). An atrial premature impulse blocked in the fast pathway may conduct antegradely via the slow pathway, and return to the atria via the fast pathway. Thus, it is not surprising that 17 of the 41 patients with dual pathways had documented episodes of paroxysmal supraventricular tachycardia. In 15 of these 17 patients, atrioventricular nodal re-entrance with the echo phenomenon was induced during atrial premature stimulation. In 10 of these 15, sustained atrioventricular nodal re-entrant paroxysmal supraventricular tachycardia could be induced during electrophysiological study.

The following conditions seem to be necessary for the induction of single atrial echoes (due to atrioventricular re-entrance) in a patient with dual pathways. 1) An antegrade fast pathway effective refractory period longer than the slow pathway antegrade refractory period. 2) A slow pathway anterograde conduction time long enough for the pathway to recover for retrograde conduction. 3) A final common pathway distal to the fast and slow pathways, so that the impulse can re-enter the fast pathway. 4) Ability for the fast pathway to conduct in retrograde direction. For sustained atrioventricular nodal re-entrant paroxysmal supraventricular tachycardia to develop in these patients, conditions 1) to 4) must be met. In addition, there must be a proximal common pathway so that the retrograde fast pathway impulse can re-enter the slow pathway in an antegrade direction. There must also be critical relations between fast and slow pathway conduction times and refractory periods, so that the circus movement is not extinguished.

Atrioventricular nodal conduction is much influenced by autonomic nervous influences. Since the necessary relation between fast and slow pathway conduction time and refractory periods appear to be relatively critical for the development of sustained re-entrance, the lack of induction of sustained reentry in the patients with known documented paroxysmal supraventricular tachycardia is not surprising. One has only to postulate that autonomic influences have critically changed refractoriness or conduction time in one or both pathways, and the ability for sustained re-entrance could be lost. The demonstration of dual pathways in 8 patients with paroxysmal palpitation without documented paroxysmal supraventricular tachycardia is intriguing. Though in 4 of the patients, other arrhythmias (ventricular tachycardia and atrial flutter) appeared to relate to palpitation, in the remaining 4, dual pathways were the most striking abnormality of conduction demonstrated. It is our opinion that the demonstration of dual pathways in these 4 suggests that sporadic episodes of atrioventricular nodal reentrant paroxysmal supraventricular tachycardia may account for the history of palpitation. Sixteen of the patients had neither palpitation nor docu-

mented paroxysmal supraventricular tachycardia. It is not known whether these 16 patients are at risk for development of clinically significant spontaneous paroxysmal supraventricular tachycardia in the future.

The present study sheds little light on one of the most intriguing questions concerning dual atrioventricular nodal pathways in man: 'Are dual atrioventricular nodal pathways a property of the normal atrioventricular node in a healthy heart?' Our data in patients without organic heart disease and without paroxysmal supraventricular tachycardia are too limited to answer this question. Our current data suggest that patients with recurrent paroxysmal supraventricular tachycardia without other cardiac abnormality frequently have dual atrioventricular nodal pathways. However, in these patients, it is possible that either congenital or acquired abnormality of atrioventricular nodal function predisposes to longitudinal dissociation of the atrioventricular node into dual pathways.

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