CASE REPORT

Congenital Abnormalities of the Conduction System in Two Patients with Tachyarrhythmias

SAROJA BHARATI, M.D., ROBERT BAUERNFIEND, M.D., MELVIN SCHEINMAN, M.D., BARRY MASSIE, M.D., MELVIN CHEITLIN, M.D., PABLO DENES, M.D., DELON WU, M.D., MAURICE LEV, M.D., AND KENNETH M. ROSEN, M.D.

SUMMARY Serial sections of the conduction system (CS) were performed in two patients with recurrent tachyarrhythmias. Case 1, a 34-year-old female who had dual atrioventricular (AV) nodal pathways with recurrent paroxysmal supraventricular tachycardia, committed suicide. Autopsy revealed an abnormally formed atrial septum with insertion of eustachian valve on the AV part of the pars membranacea. The intercuspispid portion of the pars membranacea was muscular. The AV node was located adjacent to the membranous part of the ventricular septum rather than the central fibrous body. In addition, there was an accessory anterior AV node on the parietal wall of the right atrium. Case 2, a 13-year-old boy with history of recurrent ventricular tachycardia, died suddenly. CS revealed a right-sided, markedly septated bundle. The first part of right bundle branch was divided into three parts, which later joined together. Both cases showed fatty infiltration of the atrial septum, more than normal for the age of the patients. The relationship of the recurrent tachyarrhythmias to the congenital abnormalities in the CS in the two cases and the fatty infiltration is reviewed.

EXCEPT FOR THE Wolff-Parkinson-White (WPW) syndrome, an anatomic substrate has rarely been found to explain cardiac tachyarrhythmias.1, 2 We report the electrophysiologic and anatomic findings in two patients with documented recurrent tachyarrhythmias. These findings are important because they suggest additional anatomic basis for tachyarrhythmias and encourage more careful analysis of cardiac specialized conduction system in arrhythmic patients.

Case One

Clinical Summary

The patient was a 34-year-old female with a history of psychiatric disorder, heroin abuse, and multiple suicide attempts. She also had a 5-year history of paroxysmal supraventricular tachycardia (PSVT) which recurred despite treatment with digoxin, propranolol and quinidine. Most episodes were difficult to terminate, usually lasted several hours and were associated with fatigue and dyspnea. Physical examination during sinus rhythm revealed no abnormal cardiovascular findings. She was referred to San Francisco General Hospital for further studies in October 1976 after a bout of tachycardia resulted in profound hypotension, requiring emergency direct current cardioversion. She died in January 1977, under circumstances suggesting multiple drug overdose.

Review of Electrocardiograms

ECGs during sinus rhythm demonstrated short P-R intervals in the range of 0.10-0.12 sec, narrow QRS complexes with normal morphology except for non-specific ST- and T-wave changes (fig. 1). PSVT was characterized by QRS complexes identical to those observed during sinus rhythm, more pronounced ST- and T-wave changes, and rates in the range of 200 beats/min (fig. 1). No P waves were visible during the tachycardia (fig. 1).

Electrophysiologic Studies

The patient underwent electrophysiologic studies at San Francisco General Hospital in October 1976. At the time of study the patient was in sinus rhythm with a cycle length of 1009 msec. P-A and H-V intervals were normal (31 and 52 msec, respectively), while the A-H interval was short (43 msec, normal 54-130 msecs).3 Catheter-induced right bundle branch block was observed throughout the study. Rapid atrial pacing from the high right atrium revealed that as paced cycle lengths were decreased from 700 to 550 msec, A-H intervals increased only from 65 to 75 msec. At a
paced cycle length of 500 msec the A-H interval suddenly increased to 195 msec, suggesting failure of a fast atrioventricular (AV) nodal pathway. As atrial paced cycle lengths were further decreased to 350 msec, only a small additional prolongation of A-H intervals to 210 msec was observed. At an atrial paced cycle length of 300 msec, type I second-degree block occurred proximal to the His bundle recording site.

Atrial extrastimulus testing (coupled to a basic driven cycle length of 500 msec) was performed, but the study was incomplete because of intermittent failure of atrial capture. However, dual AV nodal

pathways were suggested by the demonstration of a sudden increment in A2-H2 at A1-A2 coupling intervals between 445 and 425 msec$^+$ (fig. 2). Testing at shorter coupling intervals demonstrated a continuous slow pathway curve. The fast pathway effective refractory period was thus 425 msec, while slow pathway conduction was limited by an atrial functional refractory period of 260 msec. Single atrial echoes were observed with coupling intervals of 320–260 msec. With rapid ventricular pacing, ventriculooatrial (VA) conduction was intact at paced cycle lengths as short as 350 msec. Sustained PSVT was induced at ventricular paced cycle lengths of 400 and 350 msec. The cycle length of PSVT was 330 msec, and atrial and ventricular electrograms occurred simultaneously.

Electrophysiologic studies were repeated after 1 g of procainamide was administered intravenously (experimental protocol). Atrial extrastimulus testing (again coupled to a basic driven cycle length of 500 msec) generated a discontinuous A1-A2, H1-H2 curve with a sudden jump in A2-H2 at an A1-A2 coupling interval of 325 msec (fast pathway effective refractory period) (fig. 3). Slow pathway conduction was limited by an atrial functional refractory period of 260 msec. A1-A2 coupling intervals of 260–320 msec resulted in induction of sustained PSVT with a cycle length of 370 msec (fig. 4).

Postmortem Findings

Aside from the findings in the heart, we noted pulmonary congestion and edema, severe cerebral edema and hemorrhagic gastritis. Birefringent foreign material was present in the liver, spleen and lungs. The serum digoxin level was 62.4 ng/ml and serum procainamide level was 19.9 μg/ml.

Heart

The heart weighed 290 g. The chambers were not hypertrophied or enlarged. The limbus was very thick, and its arch continued as a thickened ridge to the AV groove in the region of the medial part of the anterior leaflet and proximally it continued as a thickened ridge in the region of the tubercle of Lower (fig. 5). There was distinct endocardial fibroelastosis in this region. In contrast, the crista terminalis (posterior crest) was a relatively small structure. The eustachian valve was a thickened structure and the space of His was aneurysmally dilated (fig. 5). An aneurysmal dilatation in the inferior wall of the right ventricle measuring about 2 cm was present at the junction of

FIGURE 4. Case 1. Induction of paroxysmal supraventricular tachycardia with atrial extrastimulus testing after procainamide. CL = cycle length; HBE = His bundle electrogram. With an A1-A2 coupling interval 320 msec, there is long A2-H2. The failure of the fast pathway coincides with induction of paroxysmal supraventricular tachycardia. Atrial electrograms cannot be identified with certainty during QRS complexes. Since atrial activation is well recorded in this patient, it is unlikely that there is atrioventricular dissociation after tachycardia induction, so that atrial activation is almost certainly simultaneous with ventricular activation.
Methods

AV node was branched. The right was cut into each block. The heart was grossly showed no changes.

Microscopic Examination

Methods

The sinoatrial (SA) node and its approaches, the AV node and its approaches, the AV bundle and bundle branches up to the region of the moderator band were serially sectioned, and every 10th section was retained. The atrial preferential pathways were serially sectioned and every 40th section was retained. The right AV rim was completely serially sectioned and every 10th section was retained. The left AV rim was not serially sectioned. The remainder of the heart was cut into blocks and two sections were taken from each block. Consecutive sections of the conduction system and the right AV rim were stained with hematoxylin and eosin, Weigert-van Giesen or Gomori trichrome stains. Alternate sections of the remainder of the heart were stained with hematoxylin and eosin and Weigert-van Giesen stains. Thirteen hundred sections were examined in this manner. The findings in the conduction system were compared with those of six hearts aged 31-40 years, with no pathologic changes.9, 9

Findings

Conduction System.

SA Node. There was focal edema.

Approaches to the SA Node. Marked fatty infiltration was present for this age (fig. 7) involving Bachmann’s bundle, the crista terminalis (posterior crest) and the right atrial appendage.

Atrial preferential pathways. There was marked fatty infiltration (fig. 8).

Approaches to the AV Node. Marked fatty infiltration of the approaches was present for this age (fig. 8). There was slight thickening and narrowing of the AV nodal artery. The eustachian valve was anchored on the AV portion of the pars membranacea, and not on the central fibrous body (fig. 9).

AV Node. The node lay on the right side of the AV portion of the pars membranacea and not on the central fibrous body (fig. 8). The node was more lobulated and elongated than usual, and showed a slight increase in connective tissue. Fibers of James entered the end of the node from the upper approaches, as seen normally.

AV Bundle, Penetrating Portion. There was a slight infiltration of fat. No connections between the atrial septal musculature and the bundle were found.

AV Bundle, Branching Portion. The bundle passed

the medial and inferior leaflets. The muscle of Lancisi was large. The intercuspid portion of the pars membranacea was muscular (fig. 6). The mitral valve presented an unusual focal distribution of chordae with an increase in hemodynamic changes for the age of the patient. Otherwise the heart grossly showed no changes.

FIGURE 5. Case 1. Right atrial and right ventricular view of the heart. RA = right atrium; RV = right ventricle; AH = aneurysm of the space of His; C = mouth of coronary sinus; ARV = aneurysm of sinus of right ventricle; E = eustachian valve. Arrows point to accentuation of the middle internodal pathway.

FIGURE 6. Case 1. Left ventricular view of the heart. A = aorta; LV = Left ventricle. Arrows point to intercuspidal part of the pars membranacea infiltrated with muscle.
to the left side of the septum. It showed slight fatty infiltration.

Right Bundle Branch. The bundle passed more horizontally than usual, but was otherwise normal.

Left Bundle Branch. There were no abnormalities.

Myocardium. All parts of both atria showed marked fatty infiltration, as did the right ventricle. Slight fatty infiltration of the left ventricle was also noted. There was acute focal degeneration of muscle.

Summit of the Ventricular Septum. For the age of this patient there was considerable increase in connective tissue. There was also distinct sclerosis of the large arterioles in this region (fig. 9). The infundibular muscle infiltrated the right side of the pars membranacea.

Right AV Rim. The atrial muscle on the anterior parietal wall, close to the septum and the right atrial appendage, had a distinct AV node-like structure (fig. 10) consisting of small cells which were serpiginous and intertwining. The cytoplasm of these cells was only slightly more pale than that of the ordinary atrium. The nuclei were longitudinally oval. There was a distinct increase in elastic and collagenous tissue in this structure compared with the ordinary atrial musculature. Adjacent to this structure, the cell structure of the atrium resembled that of the approaches to the regular posterior AV node. The subendocardial cells adjacent to the approaches resembled Purkinje-like cells. There were no communications between atrium and ventricle in the right AV rim.

Case Two

Clinical Summary

The patient was a 13-year-old male referred to the University of Illinois Hospital with a 6-year history of documented recurrent ventricular tachycardia. Most
episodes were short and well-tolerated, but several lasted for many hours, resulting in weakness and epigastric pain, and eventually required cardioversion. The episodes of ventricular tachycardia recurred despite treatment with combinations of procainamide, quinidine, digoxin, propranolol and diphenylhydantoin. Physical examination during sinus rhythm was normal, as were chest x-ray, echocardiogram and electrocardiographic stress test. Right heart catheterization results were normal except for mildly elevated pulmonary artery and right ventricular systolic pressures (35 mm Hg each; 15 to 28 mm Hg each is normal). The patient died suddenly in November 1976.

Review of Electrocardiograms

ECGs during sinus rhythm revealed a P-R interval of 0.16 sec and narrow QRS complexes with normal morphology except for Q waves in lead III and left ventricular hypertrophy by voltage criteria (fig. 11). Ventricular tachycardia was characterized by rates of 150–220 beats/min, left bundle branch block QRS morphology, and AV dissociation with capture and fusion beats (fig. 11). Some rhythm strips demonstrated intermittent non-sustained burst of ventricular tachycardia (fig. 11).

Electrophysiologic Study

When electrophysiologic studies were performed (October 1976) the patient was in sinus rhythm with a cycle length of 650 msec. P-A and A-H intervals were normal (40 and 75 msec, respectively), but the H-V interval was prolonged (65 msec; 31–55 msec is normal). AV nodal function, determined by rapid atrial pacing and extrastimulus testing, was normal.

Only short bursts of ventricular tachycardia were
recorded. These were characterized by absent VA conduction and absence of H potential with QRS complexes of the tachycardia type. Ventricular tachycardia could not be initiated with right ventricular incremental pacing or extrastimulus testing.\(^\text{11}\)

**Postmortem Findings**

Findings were restricted to the heart and lungs. Gastric contents were found diffusely throughout the tracheobronchial tree. The lungs otherwise showed no changes.

**Heart**

The heart was enlarged, weighing 357 g. All chambers were hypertrophied and enlarged. There was a rete chiari in the right atrium. The heart was otherwise normal.

**Microscopic Examination**

**Methods**

The SA node and its approaches were serially sectioned, and every 10th section was retained. The atrial preferential pathways were serially sectioned and every 40th section was retained. The AV node and its approaches, the AV bundle, the bundle branches up to the region of the moderator band were serially sectioned and every 10th section was retained. All sections from the SA node to the bundle branches were consecutively stained with hematoxylin and eosin, Weigert-van Gieson, and Gomori trichrome stains. The remainder of the heart was cut into blocks and sections were taken from each block. Alternate sections were stained with hematoxylin and eosin and Weigert-van Gieson stains. Seven hundred four sections were examined in this manner. The findings in
Figure 10. Node-like structure in region of anterior parietal wall of right atrium. Weigert-van Gieson stain. (top) Low power × 17; (bottom) high power × 150. TV = tricuspid valve; RA = right atrium; RV = right ventricle. Arrows point to the nodal-like structure.
the conduction system were compared with those of three hearts aged 11–20 years for the SA node and from four hearts aged 11–20 years for the AV node, bundle and bundle branches.

Findings

SA Node. There were several small accumulations of mononuclear cells.

Approaches to SA Node. These were normal.

Approaches to AV Node. A slight infiltration of fat with mononuclear cells was present.

Summit of the Ventricular Septum. There was an increase in loose connective tissue with occasional old scars posteriorly. A slight infiltration of mononuclear cells at the base of the mitral valve was also noted.

Atrial Preferential Pathways. An increase in fat more than normal for this age was present. There was a focal increase of mononuclear cells in fat, muscle and around nerves.

AV Node. This was compact, small and partly engulfsed in the central fibrous body. The node formed the bundle abruptly and it was difficult to differentiate the node from the bundle (fig. 12).

Penetrating Bundle. This part of the bundle was situated entirely on the right side of the summit of the ventricular septum. It was very short, becoming the branching portion almost immediately and giving off the posterior fibers of the left bundle branch (fig. 12).

Branching Bundle. The branching bundle consisted of a compact right side and centrally located left side which was broken up by thick masses of connective tissue. At one point these connective tissue fibers completely divided the right and central parts of the branching bundle. They joined together again to form one mass. Distal to the disrupted region more fibers of the left bundle branch were given off. Then the bifurcation occurred (fig. 13). Thus, the AV node and bundle progressed into the right bundle branch while the central portion gave off further fibers of the left bundle branch (figs. 12 and 13). At one point the branching bundle fibers which were going to the left side dipped in and joined the ventricular septum (Mahaim fibers).

Right Bundle Branch. The first part of the right bundle branch (fig. 14A) divided into three parts (fig. 14B), which joined together again. The first part was moderately fibrotic. The second part showed slight fibrosis.

Left Bundle Branch. The fibers in the beginning of their course (posterior radiation) showed moderate fibrosis. The anterior radiation showed moderate fibrosis with smallness of cells. More distally, the left bundle branch showed considerable fibrosis. Some of the peripheral Purkinje cells were smaller than normal.

Right Ventricle. There was more fatty infiltration than usual for this age.

Left Ventricle. Recent focal necrosis of the left ventricle with some old scars was noted.

Discussion

The first patient demonstrated findings characteristic of the Lown-Ganong-Levine syndrome, i.e., a short P-R interval in a young female with repeated bouts of PSVT. As in previously studied cases, A-H interval was short, suggesting fast AV nodal conduction or partial AV nodal bypass. Previous studies in
patients with short PR interval and recurrent PSVT suggest that AV nodal reentry is the most common basis of arrhythmias. In these patients, discontinuous conduction curves are demonstrated, with induction of paroxysmal tachycardia coinciding with antegrade failure of a fast AV nodal pathway. The circus movement in these patients consists of antegrade conduction via a slow AV nodal pathway, and retrograde conduction via a fast AV nodal pathway. AV nodal reentrance appears to occur within the AV node in these patients, in that neither the His bundle nor atrium appear part of the reentrant circuit.

Electrophysiologic studies in the first patient were consistent with previous studies. Dual AV nodal pathways were suggested by sudden increase in A-H interval with atrial incremental pacing, and by demonstration of discontinuous conduction curves with atrial extrastimulus testing. AV nodal reentrance (rather than reentrance via a concealed Kent bypass tract), was suggested by initiation of the tachycardia coinciding with failure of conduction over a fast AV nodal pathway. In addition, simultaneous activation of the atria and ventricles during tachycardia, made unlikely participation of an accessory extranodal pathway. In addition, ventricular echo beats without intervening atrial depolarization again suggested that most of the atrium is not a necessary link in the tachycardia.

The anatomic findings in case 1 are unique in that they represent the results of the first anatomic study of the cardiac conduction system in a patient with dual AV nodal pathways (with or without Lown-Ganong-Levine syndrome). These findings are: 1) the connection of the tendon of Todaro (anchor of the eustachian valve) to the pars membranacea rather than to the central fibrous body; 2) the invasion of the intercuspidal part of the pars membranacea by infundibular muscle; 3) the presence of the AV node on the right side of the AV part of the pars membranacea instead of the central fibrous body; 4) the presence of an

Figure 12. Case 2. Atrioventricular node and bundle of His. Weigert-van Gieson stain × 30. There is no sharp differentiation between the node partly engulfed in the central fibrous body and the markedly septated bundle of His. N = atrioventricular node; B = bundle of His; V = ventricular septum.
AV node-like structure in the right AV rim; 5) the prominence of the middle internodal pathway (limbus); 6) marked fatty infiltration of the entire atrial septum (all atrial preferential pathways but especially the middle), the parietal atrial walls, the approaches to the SA and AV nodes and the right ventricle; 7) aneurysms of the space of His and of the proximal part of the sinus of the right ventricle; and 8) greater-than-normal sclerosis of the left side of the cardiac skeleton.

The prominent middle internodal tract showed both muscle and fat tissue. It appeared that the amount of muscle was normal despite the marked invasion by fat tissue. The size of the muscle cells was not increased. Thus, the accentuation of this pathway grossly was due mostly, if not entirely, to an infiltration of fat.

It is difficult to differentiate many of the above findings from those found in what have been considered normal hearts. We have not seen the connection of the tendon of Todaro to the pars membranacea in normal hearts. However, two of us (SB and ML) have seen the AV node on the right side of the AV part of the pars membranacea, and the invasion of the intercusp portion of the pars membranacea by infundibular muscle, in hearts that were considered normal. (Of course, the fibers of James — the most downstream part of the
Figure 14. Case 2. Right bundle branch. Weigert-van Gieson stain × 45. (top) First part shows fibrosis with beginning lobulation; (bottom) end of first part shows division into three parts. Arrows in top figure point to right bundle branch; arrows in bottom figure point to three parts of right bundle branch.
AV node — are found in normal hearts.)

Again, node-like structures in the atrial aspect of the right AV rim have been identified in hearts considered to be normally formed. However, these structures (with the exception of the fibers of James) could actually be anatomic abnormalities unrealized until now, or if they are normal formations, they might produce abnormal conduction disturbances in some patients, as postulated for the fibers of James in some cases of the WPW syndrome.

If the rhythm disorder is related to the above findings, one might hypothesize that, in this case, there is an abnormal formation of the central portion of the fibrous skeleton of the heart, as manifested by the abnormal connection of the tendon of Todaro and the "abnormal" pars membranacea. This might produce abnormal stresses and strains in the central region of the heart, as manifested by the prominent middle preferential pathway and the accentuated sclerosis of the left side of the cardiac skeleton for the age of this patient. Associated with the abnormally formed central fibrous portion of the heart is the position of the AV node lying adjacent to the AV part of the pars membranacea, rather than the more immobile part of the central fibrous body. This structure in turn might be subjected to stresses and strains as manifested by the elongated, flattened configuration of the node.

The reason for the marked fatty infiltration of the myocardium involving in this case the atria and atrial septum, and especially the middle internodal pathway, is not understood. It is either a primary infiltration, with pressure and irritative effects on the myocardium, or an infiltration secondary to in some way altered myocardium. In either case, the literature contains reports of fatty infiltration and lipomatosis of the atrial septum being associated with arrhythmias.

We can then speculate on the possible anatomic base of the fast AV nodal conduction (short A-H interval), dual AV nodal pathways and AV nodal reentrant PSVT in this patient. Fast AV nodal conduction could reflect utilization of the prominent middle internodal pathway which was in continuity with atrial fibers (James tracts) entering the distal portion of the node. Or one could postulate a partial bypass of the node by James fibers alone for the fast AV nodal conduction. No other anatomic basis for fast conduction was detected. Specifically, we did not note anomalous connection between atria and His bundle as described by Lev et al. in a case of WPW and by Brechenmacher in Lown-Ganong-Levine syndrome.

Longitudinal dissociation with reentrant PSVT, demonstrated in this patient, could have occurred totally within the AV node, within the AV node using adjacent atrial tissue, or using the node (fast pathway) and the accessory node (slow pathway). The latter reentrant circuit would necessarily use a small amount of atrial tissue. The abnormal placement of the AV node adjacent to the mobile pars membranacea rather than to the central fibrous body could predispose to internodal AV nodal reentrance. In one case of WPW, Lev et al. found an abnormal central fibrous body.

We can only speculate on the cause of the arrhythmia in the first case. We believe that the presence of what appear to be minor abnormalities in the formation of the heart and the conduction system, and the resultant fatty infiltration, may be responsible for the arrhythmias in this case, and may also explain some supraventricular arrhythmias in general. Thorough examination of the conduction system in more cases of tachyarrhythmias and dual AV nodal pathways is necessary for more definitive statements.

Otherwise normal hearts may have dual AV nodal pathways. These pathways may also be found in patients with hypertension, coronary disease and hypothyroidism. There are probably various mechanisms in the production of dual AV nodal pathways, both congenital and acquired.

In case 2, the ventricular tachycardia was first diagnosed at the age of 6 years, and may have occurred in infancy. The QRS pattern of the tachycardia (left bundle branch block pattern) is most consistent with a tachycardia originating in the right ventricle, or a tachycardia originating in the more proximal conduction system with preferential conduction via the right bundle branch. A left ventricular origin cannot be ruled out. The tachycardia was self-initiating during sinus rhythm (see rhythm strips) and not reproducible in the catheterization laboratory. Thus, we cannot prove a reentrant mechanism.

In case 2 there was a right-sided, abnormally formed, markedly septated bundle, with the first part of the right bundle branch divided into three segments, moderate fibrosis of both bundle branches, and the AV node partially embedded in the central fibrous body. The atrial preferential pathways and the right ventricle showed fatty infiltration greater than normal for this age, although this was not as startling as in case 1. The heart had hypertrophy of all chambers, with recent and old scars in the summit of the ventricular septum.

One may hypothesize in case 2 that the congenital abnormality of the conduction system was related to the tachycardia. Several mechanisms may be postulated: 1) reentrance in the septated His bundle with preferential delivery to the right bundle branch. In this case, one might have expected demonstration of H potentials before tachycardic beats, and these were not seen; 2) reentrance in the septated right bundle, with preferential delivery to the right bundle branch. In either case, the circus movement, localized in the proximal His-Purkinje system, might be inaccessible to extrastimuli (because of limiting refractoriness proximal and distal to the site of circus movement) so that a reentrant mechanism was not demonstrated with extrastimulus technique; 3) enhanced automaticity in the abnormally formed conduction system. The hypertrophy of the heart, the recent and old scars in the ventricular myocardium and the fatty infiltration of the right ventricle and internodal pathways might be considered to be secondary to the tachycardia. However, as in case 1 (but to a lesser extent),
we do not know the role of fatty infiltration of the atrial septum in the genesis of the arrhythmia.

Clinically, this case was similar to a group of patients recently reported by Pietras and co-workers.\(^2\) They described nine patients with chronic recurrent right ventricular tachycardia (tachycardia complexes with left bundle branch block pattern), no clinically apparent organic heart disease, and essentially negative diagnostic right and left heart catheterization (including coronary arteriography). It is possible that the idiopathic chronic recurrent right ventricular tachycardia, described in Pietras' cases, reflects an underlying congenital abnormality of the conduction system, perhaps similar to that described in our case 2.

Various abnormalities in formation of the conduction system which in the past have been considered normal variations may, in certain persons, be responsible for obscure arrhythmias and even sudden death. This has previously been suggested by James.\(^2\) In addition, various minor abnormalities of the heart previously overlooked or considered to be normal may be related to abnormalities in conduction.

References

23. Cameron GR: Pathology of the Cell. Springfield, Ill, Charles Thomas, 1951, pp 316-328
Congenital abnormalities of the conduction system in two patients with tachyarrhythmias.
S Bharati, R Bauernfiend, M Scheinman, B Massie, M Cheitlin, P Denes, D Wu, M Lev and K M Rosen

Circulation. 1979;59:593-606
doi: 10.1161/01.CIR.59.3.593
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/59/3/593

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/