Junctional Tachycardia
Mechanisms, Diagnosis, Differential Diagnosis, and Management

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IN THE ABSENCE of functioning anomalous connection between atria and ventricles, the A-V junction serves as the only link between these structures, allowing the atrial impulse to be transmitted to the ventricles. The A-V junction may be defined as consisting of the atrial approaches to the A-V node, the A-V node itself, and the penetrating portion of the His bundle. Total transection of the A-V junction at any point should produce A-V block, and stimulation in the presence of a normal His-Purkinje system should produce a narrow QRS.

The electrophysiologic function of the A-V junction is primarily that of conduction. However, in addition, the A-V junction possesses intrinsic automaticity, allowing it to serve as a subsidiary pacemaker in the event of failure of proximal impulse formation or propagation. Under pathologic conditions, the A-V junction can serve as an ectopic pacemaker, or as a site of reentry.

Electrophysiologic Considerations

Experimental studies demonstrating automaticity in the A-V junction were extensively reviewed by Scherf and Cohen.1 Earlier workers produced A-V junctional escape rhythms after depressing sinus node function by various surgical, chemical, mechanical, and thermal maneuvers. In addition, experimental A-V junctional rhythms were produced by increasing sympathetic tone or by warming specific areas in the A-V junction. The site of origin in the A-V junction was determined by noting the timing of atrial and ventricular contraction, and also by noting the initial sites of negativity with direct recording. Zahn suggested classification into upper, middle, and lower A-V nodal rhythms.2 In upper nodal rhythms, atrial contraction preceded ventricular contraction (fig. 1A); in middle nodal rhythms, atria and ventricles contracted simultaneously (fig. 1B); and, in lower nodal rhythm, ventricular contraction preceded atrial contraction (fig. 1C). This classification has been useful descriptively, but is not accurate since it does not take into account variations in antegrade and retrograde conduction time.3

The recording of cellular action potentials from the specialized conduction system allowed a more direct approach to the determination of the nature and sites of cardiac automaticity. Automatic cells are characterized by the presence of slow depolarization during diastole (phase 4 diastolic depolarization). Sinus node cells possess this characteristic as do other areas in the specialized conduction system where automaticity may be considered normally latent.

Electrophysiologists have divided the A-V node into three regions based upon both anatomic location and on the characteristics of the recorded action potentials. These are the A-N region (atrionodal), the N region (nodal), and the N-H region (nodal-His). Diastolic depolarization has been definitely demonstrated in the N-H regions, possibly in the A-N regions, but not in the N region.4 Diastolic depolarization is also demonstrable in cells comprising the His bundle. A-V junctional rhythms due to enhanced automaticity may arise in one or more of the following areas: the A-N region, the N-H region, the coronary sinus, and the His bundle. The term A-V junctional rhythms is a more accurate description than A-V nodal rhythms,5,6 since these rhythms do not appear to arise in the N region of the A-V node.

Diagnosis of A-V Junctional Rhythms

If antegrade and retrograde conduction are intact, a beat arising from the A-V junction produces both a retrograde P wave and QRS of supraventricular configuration. The timing of this retrograde P wave in relation to the QRS is dependent upon the site of origin of the impulse, as well as the antegrade and retrograde conduction times.1, 3, 5, 6 A beat arising in the proximal A-V
junction region is more likely to have a retrograde P wave preceding the QRS, while a beat arising distally is more likely to have a retrograde P wave following the QRS.

If only antegrade conduction is intact, then junctional rhythms will be just characterized by QRS complexes. If retrograde conduction is intact, then junctional rhythms will produce retrograde P waves. Junctional impulses may also occur without either antegrade or retrograde conduction. These are electrocardiographically silent unless they disturb impulse formation or conduction in subsequent beats.7,8

P-Wave Morphology

Typically, the P waves in junctional rhythms are inverted in leads II, III, and aVF, and upright in lead aVR. The P-wave amplitude in lead I is usually small and of variable contour. The frontal axis of the P wave is generally between −60° and −80°. P-wave configuration in the precordial leads is variable, with inverted P waves being noted in none, some, or all precordial leads.

Not all workers agree with this classical description of A-V junctional P waves. Moore et al., studying P-wave morphology in dogs, suggested that rhythms arising from the area of the coronary sinus or from the A-V node were characterized by upright P waves in leads II, III, and aVF.9 Waldo and co-workers, studying patients with A-V dissociation developing during open-heart surgery, described junctional P waves which were diphasic (−/+), or predominantly (+) in leads II, III, and aVF.10 In these cases, the initial negative portion of the P wave was often masked in the T wave, giving the impression on the surface electrocardiogram that junctional P waves were of antegrade contour.

Several specific categories of A-V junctional rhythms have been designated based upon P-wave contour and/or P-R interval. These include: (1) Coronary sinus rhythm, characterized by retrograde P waves with normal P-R intervals.1 Lancaster et al. and Lau et al., pacing the interior of the coronary sinus, produced P waves conforming to this description.11, 12 However, Waldo demonstrated that stimulation of other atrial sites also produced P waves of retrograde contour with normal P-R intervals, which suggests that this appearance was not specific for rhythms arising from the coronary sinus.13 Thus, the use of the term “coronary sinus rhythm” is probably inappropriate.
These rhythms could better be labeled as either inferior or low atrial rhythms. Some of these may arise in tissues of the A-V junction. (2) Left atrial rhythms, suspected to arise from specialized conduction tissue in the left atrium and characterized by P-wave inversion in V1.18 However, coronary sinus pacing produces P-wave inversion in V6, and left atrial pacing may or may not produce a similar change.11, 12, 15 Because of this, the use of the term “left atrial rhythm” for rhythms characterized by retrograde P waves with additional P-wave inversion in V6 is probably inappropriate. These rhythms should be classified as either low atrial or A-V junctional. (3) Coronary nodal rhythm, characterized by normal P-wave contour with short P-R intervals.1-16 This rhythm has been suspected of arising from the tail of the sinus node close to the coronary sinus. Waldo was able to reproduce the electrocardiographic pattern of coronary nodal rhythm by pacing a number of right atrial endocardial sites.13 In addition, the presence of accessory conduction tracts bypassing the A-V node can allow the development of sinus rhythms with normal P-wave configuration and short P-R intervals. The term “coronary nodal rhythm” is inaccurate, since these rhythms can either represent ectopic atrial rhythms arising from a number of sites, or sinus rhythm with bypass of the A-V node.

Part of the confusion regarding the various types of A-V junctional P waves reflects changing concepts regarding atrial depolarization. Current evidence suggests that atrial depolarization is not radial, as originally proposed by Sir Thomas Lewis, but is asymmetric with internodal tracts playing a significant role in the distribution of the atrial impulse.17 This implies that one cannot accurately predict the site of origin of an impulse by vectorial analysis of the P wave. The difficulty is further compounded by the possible occurrence of conduction lesions in the internodal tracts, which could alter P-wave contour.18 There is no information in man as to the specific alterations in P contour associated with single or combined lesions in the internodal tracts.

QRS Morphology

In the absence of intraventricular conduction defects, the QRS of a junctional beat is usually narrow, since the impulse arises above the branching portion of the His bundle and is simultaneously distributed to both ventricles. In the presence of intraventricular conduction defects, the QRS will be appropriately altered.

Junctional beats may occasionally manifest QRS abnormalities in axis and/or duration, not related to previously existing conduction defects. This occurs in several circumstances, namely: (1) When junctional premature beats follow long cycle lengths, the aberrant QRS in this circumstance reflects functional bundle-branch block related to prolongation of the refractory periods in the His-Purkinje system due to the preceding long cycle. (2) The QRS may be aberrant when there are several consecutive junctional beats or for the total duration of a junctional tachycardia. This can represent the perpetuation of a functional bundle-branch block due to repetitive concealed conduction,19 or it can represent rate-dependent bundle-branch block. (3) Aberrant QRS complexes may occur during a junctional escape rhythm.20 This can represent the effect of diastolic depolarization in one of the bundle branches with decreased membrane responsiveness in late diastole, or could reflect either longitudinal dissociation in the His bundle or use of accessory pathways allowing asymmetric distribution of the cardiac impulse to the ventricles. Studies utilizing the His bundle recording technic suggest that many of the so-called junctional escape rhythms with aberrant conduction are really rhythms arising in the bundle branches or more distal ventricular Purkinje system.21 In such cases, the abnormal escape beats are preceded by H potentials with short H-V intervals, or have H potentials simultaneous or following the onset of the QRS. These H potentials are retrograde, the timing in relation to the QRS depending upon the site or origin of the escape beat and conduction time antegrade to the ventricles and retrograde to the His bundle.

Antegrade and Retrograde Conduction in Junctional Rhythms

Prolongation of either antegrade or retrograde conduction from the site of impulse formation in the A-V junction (first-degree block) will alter the relationship of retrograde P wave and QRS. Second-degree block between the A-V junctional focus and the atrium can occur.22 This may be of type I (Wenckebach) with progressive R-P prolongation terminating in dropped retrograde P waves (fig. 2B). Type II retrograde block (Mobitz) without progressive R-P prolongation is less common (fig. 2C).

Antegrade conduction disturbance may also complicate junctional rhythms. These can be difficult to diagnose if they occur in the presence of
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A-V dissociation since in these cases the conduction disturbance does not affect the relationship of P waves and QRS, but only the periodicity of QRS complexes. Antegrade Wenckebach periods are associated with characteristic Wenckebach periodicity, i.e., groups of QRS complexes with progressively shortening R-R intervals followed by pauses which are less than the sum of two consecutive short cycles. Combinations of antegrade and retrograde conduction disturbances may coexist in junctional rhythm giving rise to complex arrhythmias.

Junctional tachycardia is one of the causes of A-V dissociation, the mechanism frequently being a combination of both physiologic interference in the A-V node between antegrade sinus impulses and retrograde junctional impulses, and also depression of conduction. In A-V dissociation complicating junctional tachycardia, the atrial and ventricular rates are independent, with the ventricular rate being somewhat faster than the atrial (fig. 3B). A-V dissociation is frequently incomplete with occasional ventricular captures from appropriately timed sinus beats allowing antegrade conduction through the A-V node (figs. 3B, C). The occurrence of captures depends upon the atrial and ventricular rate and the A-V refractory period. Retrograde conduction is more frequently impaired than antegrade conduction in junctional tachycardia with A-V dissociation. This is necessary, for if retrograde conduction were completely intact then junctional tachycardia should be accompanied by 1:1 retrograde atrial capture (figs. 2A, 3A).

Junctional tachycardia with A-V dissociation occurs with other supraventricular rhythms besides sinus rhythm, such as sinus bradycardia, atrial fibrillation, atrial flutter, atrial tachycardia, or a second junctional tachycardia characterized by retrograde P waves (fig. 3B, C, D). The occurrence of junctional tachycardia with additional supraventricular tachycardia may be considered a double tachycardia (fig. 3D).

In patients with sinus rhythm and junctional tachycardia, A-V dissociation is frequently isorhythmic. In these instances, the ventricular rate apparently influences the atrial rate, with the dissociated sinus P wave fluctuating cyclically back and forth around the QRS complex. Waldo et al., studying 11 patients with “isorhythmic dissociation”

Figure 2

Variation of retrograde conduction with NPJT. (A) NPJT with intact retrograde conduction. Note retrograde P wave following every QRS. (B) NPJT with retrograde Wenckebach periods. The R-P interval gradually increases from 0.16 to 0.20 sec during the first five QRS complexes. Retrograde block occurs after the sixth QRS, which is then followed by a sinus escape and ventricular capture (C). The eighth QRS is followed by a fusion P wave (f), with fusion occurring between sinus escape and retrograde P. (C) NPJT with type II retrograde block. The first two QRS complexes are followed by P waves with a fixed R-P interval. There is retrograde block after the third QRS, which then allows the sinus to escape with an antegrade P wave and ventricular capture (C). Following this sequence, 2:1 retrograde block is noted with sinus escape and ventricular captures.

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complicating open-heart surgery, suggested that synchronization of QRS and P resulted from retrograde atrial capture from junctional beats during the periods when junctional rate surpassed sinus rate.\textsuperscript{13} They further postulated that the retrograde P waves had either (–/+ +) polarity or predominantly positive polarity simulating A-V dissociation (as opposed to junctional rhythm with retrograde atrial capture). In their cases, the only period of true dissociation occurred in the transition between sinus rhythm and junctional rhythm. In our experience, the transition from antegrade to retrograde P waves in junctional rhythms has generally been readily apparent (fig. 3A).

Levy and co-workers proposed another mechanism explaining isorhythmic A-V dissociation.\textsuperscript{23} Based upon both clinical and experimental observations, they suggested that isorhythmic dissociation reflected a biological feedback control system, with P-R interval determining stroke volume and blood pressure. They demonstrated that increase in blood pressure had an inverse effect on sinus rate, causing sinus slowing when the P-R interval became hemodynamically optimal. This sinus slowing allowed the P wave to migrate back into the QRS at which point pressure fell and the sinus again accelerated.

**His Bundle Recording in the Diagnosis of Junctional Rhythms**

The recording of His bundle potentials (H) with electrode catheters passed to the right heart has been helpful in the diagnosis of junctional rhythms. The presence of H potentials with normal or prolonged H-V intervals in front of either narrow or wide QRS complexes suggests that these complexes arise in or above the His bundle. The occurrence of retrograde P waves, either in front or following the QRS, suggests A-V junctional rhythm. Further confirmation of the retrograde nature of P waves may be obtained by simultaneous recording of bipolar atrial electrograms from the high and low right atrium. A progression of atrial conduction

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Figure 3

Nonparoxysmal junctional tachycardia (NPJT) with and without A-V dissociation. (A) NPJT with intact retrograde conduction. Note that as the A-V junctional pacemaker overtakes the sinus pacemaker, retrograde P waves become apparent (last three complexes). A-V dissociation is present only for the two beats between the three sinus captures and the three retrograde captures. (B) NPJT with incomplete A-V dissociation. The sinus rate is 100 beats/min and the ventricular rate is 116 beats/min. Atria and ventricles are dissociated except for the eighth P wave which occurs at a time when A-V junction is not refractory capturing the ventricles (C). (C) NPJT with incomplete A-V dissociation and sinus bradycardia. The atrial rate is 50/min while the ventricular rate is 72/min. The third and fifth P wave captures the ventricles. (D) Double tachycardia with NPJT at a rate of 84 beats/min, and PAT with an atrial rate of 150 beats/min. Complete A-V dissociation is noted. QRS widening is due to preexistent conduction defect.
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from low to high suggests retrograde atrial depolarization.

In atrial fibrillation, H potentials precede all conducted beats whether they propagate from the atrium or arise in the A-V junction. The diagnosis of junctional origin depends upon demonstration of regular R-R intervals.

In A-V dissociation, A-V junctional beats are preceded by H potentials with normal or prolonged H-V intervals without relationship to atrial activity.

Paroxysmal Junctional Tachycardia

Paroxysmal supraventricular tachycardia (PSVT) is characterized by the following: (1) abrupt onset (usually following a premature beat) and abrupt termination; (2) a ventricular rate usually between 180 and 220 beats/min; the range of rates may be as slow as 100/min or as fast as 250/min; (3) the presence of abnormal P waves (at identical rate) or absent P waves; (4) QRS complexes of supraventricular origin which are usually narrow, although with preexistent conduction defects or aberrant conduction may be wide; (5) the presence of precisely regular ventricular and/or atrial complexes; and (6) either a complete lack of response or abrupt termination with carotid massage.

PSVT may be subdivided into paroxysmal atrial tachycardia (PAT), in which P waves are abnormal but not of retrograde contour and occur in rapid succession with a consistent relationship to QRS, and paroxysmal junctional tachycardia (PJT), in which there are retrograde P waves or no P waves (fig. 1).

The differential diagnosis of PSVT includes: (1) Sinus tachycardia. This arrhythmia can be diagnosed by noting the lack of paroxysmal onset and termination, as well as slight slowing in response to carotid massage. (2) Atrial flutter with 2:1 block, where one or both flutter waves are concealed in the QRS and T. The demonstration of flutter depends upon transient increase in A-V block with vagal maneuvers allowing visualization of flutter waves, or by direct recording with atrial electrograms or esophageal recordings of flutter activity. (3) Atrial fibrillation with rapid ventricular response. Slight variation in R-R intervals suggest this arrhythmia. Demonstration of fibrillatory waves when the ventricular response is slowed by vagal maneuvers is also helpful. (4) Paroxysmal ventricular tachycardia. This is always characterized by QRS widening and usually by A-V dissociation. The ventricular rate is not as precisely regular as in PSVT. His bundle recordings may be useful in the differentiation of PSVT and paroxysmal ventricular tachycardia, with H potentials being apparent before each QRS in the former.

Mechanisms of Paroxysmal A-V Junctional Tachycardia

PJT has generally been thought to be either due to reentrant mechanism or to a rapidly firing ectopic focus. “Reciprocating tachycardia” has been designated as a special type of PJT where electrocardiographic observations strongly support a reentrant mechanism.24 These cases of PJT are induced by atrial premature beats conducted slowly with long P-R intervals. This premature P and QRS is then followed by a retrograde P wave (atrial echo) and then by a run of spontaneous QRS complexes each followed by a retrograde P wave (fig. 4A). Patients with reciprocating tachycardia demonstrate ability for retrograde conduction and frequently have atrial echoes without paroxysms of tachycardia.

It was postulated that functional dissociation of the A-V node into two pathways allowed the development of these reciprocating rhythms.25 One of these pathways was available for antegrade and the other for retrograde conduction, thus setting up a circus movement. The premature beat inducing the tachycardia was blocked in one pathway, which, during slow conduction in the other pathway, recovered and was available for retrograde conduction (fig. 5).

Moe et al. reported a canine in which premature atrial stimulation could reproducibly induce paroxysms of PSVT.26 Janse et al.27 and Wit et al.28 both demonstrated experimental models of PSVT using isolated preparations of rabbit atria. Using microelectrode recordings of A-V nodal action potentials, both groups were able to demonstrate evidence for A-V nodal reentry in the A-N region during induced episodes of PSVT.

Bigger and Goldreyer, studying six consecutive patients with PSVT, demonstrated that spontaneous episodes of tachycardia were each induced by premature atrial contractions with slow conduction, and that these were followed by atrial echoes.29 Timed premature atrial stimulation could reproduce the arrhythmias with demonstration of an echo zone, an interval following the last normally conducted P wave in which stimulation produced atrial echoes and often paroxysms of tachycardia. The echo zone occurred later than the atrial vulnerable period. They suggested that timing of
retrograde P and QRS was a function of antegrade and retrograde conduction times. Frequently, the retrograde P and QRS were simultaneous. The rate of PSVT was related to A-V nodal refractoriness, the longer the refractory period the slower the rate during the tachycardia. Capture of the atria with appropriately timed stimuli could convert the tachycardia by interrupting the circus movement.

Goldreyer and Damato, using the His bundle recording technic, demonstrated that A-V nodal delay was necessary for inducing the tachyarrhythmias. They noted that a critical degree of P-H prolongation was necessary for induction of the echo phenomenon. The necessary P-H prolongation could be evoked by either premature atrial stimulation or during the course of pacing-induced Wenckebach periods. It seems likely that most episodes of PSVT are reentrant, with the site of reentry being the A-V node (fig. 4A, B). We have, however, seen several examples of PSVT, where A-V nodal reentry was apparently not involved. In these cases, the arrhythmia was provoked by premature beats without A-V conduction delay, with all subsequent beats of the tachycardia identical to the initiating beat. The junctional pacemaker manifested a warm-up phenomenon.

Figure 4

PJT spontaneous and induced. Shown are leads I, II, III, V1, and HBE. Atrial electrograms are labeled A and His bundle electrograms H. (A) There are two sinus beats followed by two atrial premature beats (APC). The second APC is conducted with a long A-H and is followed by an atrial echo and a run of PJT. (B) PJT induced by atrial pacing. Atrial pacing spikes are labeled with arrows. Shown are four paced beats followed by a timed extra stimulus (EX) which is conducted with a long A-H and is followed by a run of PJT. Atrial echoes are present but masked in the ventricular electrogram. These were subsequently demonstrated with intraatrial recording.

Figure 5

Ladder diagram demonstrating A-V nodal reentrant mechanism of PJT. An atrial premature beat with slow mechanism of PJT. An atrial premature beat with slow relationship of retrograde P and QRS depends upon the sites of reentry and the relative antegrade and retrograde conduction times.
with gradual acceleration of rate following initiation of the tachyarrhythmia (fig. 6). Carotid massage produced conversion of the arrhythmia. Rapid atrial pacing was also followed by suppression of the tachyarrhythmia, the duration of suppression being related to the duration of rapid pacing. Both the warm-up phenomenon and suppression following cessation of pacing were suggestive of a rapidly firing ectopic focus as cause of the tachycardia.

**Therapy of PJT: Acute Attack**

Since most episodes of PJT appear to reflect A-V nodal reentry, therapy of the acute attack should be predicated upon modification of properties of the circus pathways permitting this arrhythmia. Prolongation of A-V refractory periods by vagal maneuvers or antiarrhythmic medications appears to be effective. It must be remembered that PJT is usually self-limited and well tolerated in the healthy heart. The presence of angina or congestive failure is an indication for emergency therapy.

The specific therapeutic interventions effective in the conversion of PJT include: (1) *Vagal maneuvers*, such as carotid massage, performance of the Valsalva maneuver, and eyeball pressure. The intravenous administration of pressor agents, such as Neo-Synephrine or Aramine, with production of a vagal reflex may also produce conversion. Digitalization with increase of vagal tone may induce spontaneous conversion or enhance the effectiveness of carotid massage. Administration of acetylcholinesterase inhibitors such as Edrophonium or neostigmine may also be effective. (2) *Antiarrhythmic drugs* such as quinidine, procaine amide, diphenylhydantoin, and propranolol. It is difficult to recommend one of these drugs as clearly superior to the others since good comparative studies of effectiveness in PJT are not available. Propranolol is currently in vogue as the drug of choice. (3) *Electrical means of conversion*. DC cardioversion is generally successful in converting PJT to sinus rhythm. Atrial pacing with delivery of single or pairs of stimuli, or atrial pacing with fixed rates either slower or faster than the tachycardia, has been effective in conversion of PJT, presumably by interruption of circus pathways. Pacing technics can be coupled with recording of atrial and His bundle electrograms, yielding diagnostically useful information. We would recommend the use of pacing for conversion of PJT in laboratories skilled in electrophysiologic procedures, when simpler measures are ineffective.

**Therapy: Prevention of Recurrences**

Most attacks of PJT are sporadic, well tolerated, and self-limited. Patients can frequently be managed with reassurance alone and without recourse to antiarrhythmic regimens. In occasional cases, underlying preexcitation will be related to recurrent tachycardia. In these, therapy should be administered as recommended for Wolff-Parkinson-White syndrome. The remainder of this discussion is devoted to those patients with recurrent PJT, where attacks are either frequent and/or distressing.

The simplest management consists of chronic administration of drug therapy. Success has been reported with quinidine, procaine amide, propranolol, and oral diphenylhydantoin. Several workers have recommended combinations of antiarrhythmic drugs. The sporadic occurrence of PJT makes evaluation of the effectiveness of antiarrhythmic regimens difficult.

In cases refractory to drug management, more invasive therapy may be attempted. Both chronic atrial and ventricular pacing have been utilized successfully in the prevention of recurrent PJT. This therapy is most often indicated in patients

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**Figure 6**

PJT apparently secondary to ectopic firing. The first APC is not conducted slowly, but induces a run of PJT, with each P wave and QRS identical to the initiating APC. Note the gradual warm-up phenomenon (cycle lengths are listed in msec).
where episodes of PJT complicate bradycardia. Chronic pacing should not be attempted unless temporary pacing has been successful.

The carotid sinus stimulator has been used in the management of intractable PSVT. The stimulator is activated during paroxysms of tachycardia with conversion due to the vagal reflex produced. Giannelli has reported the surgical production of heart block in a case of recurrent PSVT, with prevention of further episodes but with the need for permanent pacing for control of bradycardia.

In general, even the more difficult cases can usually be managed with chronic antiarrhythmic therapy. In resistant cases, pacing technics can be utilized.

**Nonparoxysmal Junctional Tachycardia**

Pick and Dominquez described nonparoxysmal junctional tachycardia (NPJT), an arrhythmia characterized by a modest increase in ventricular rate, generally between 70 and 130 beats/min, and a lack of abrupt onset. NPJT usually occurs as a manifestation of digitalis intoxication, or in acute myocardial infarction, or with acute rheumatic fever. We have seen several patients with heart disease and NPJT where no precipitating cause could be demonstrated.

The ventricular rate in NPJT is frequently only slightly increased over the sinus rate. The presence of physiologic interference, with or without associated depression of retrograde conduction, predisposes to A-V dissociation which is frequent. A-V dissociation in NPJT is often incomplete, with capture of the ventricles by sinus impulses that fall outside the A-V refractory period (fig. 3B). NPJT may also occur with atrial fibrillation, flutter, or atrial tachycardia (fig. 2D). NPJT less commonly occurs with intact retrograde conduction, or with retrograde conduction with 2° retrograde block (fig. 2A–C).

The diagnosis of NPJT depends upon the demonstration of a gradual acceleration of junctional pacemaker. The junctional beats may have associated retrograde P waves. More frequently, the diagnosis of NPJT is made in the presence of A-V dissociation. In this latter case, the diagnosis depends upon the demonstration of a series of supraventricular beats with regular R-R intervals, unrelated to the atrial rhythm. The periodicity of these regular R-R intervals is often disturbed by: (1) ventricular captures if A-V dissociation is incomplete (fig. 3B, C), (2) antegrade exit block from the A-V junctional pacemaker producing Wenckebach periodicity or sudden dropped beats, or (3) resetting of the A-V junctional pacemaker by premature beats (concealed conduction).

NPJT must be differentiated from A-V block, since both arrhythmias may lead to A-V dissociation. In addition, NPJT and A-V block may sometimes coexist. If A-V dissociation is demonstrated and the QRS is narrow, the diagnosis can be clarified by noting atrial and ventricular rates. If atrial rate is faster than ventricular rate, and the latter is below 60 beats/min, then the diagnosis is A-V block with junctional escape. If atrial rate is faster than ventricular rate, but the latter is above 60 beats/min, then the rhythm is A-V block with superimposed NPJT. If the ventricular rate is faster than the atrial rate, then the rhythm is probably NPJT. In this latter situation, the differentiation between physiologic interference and depressed A-V conduction is difficult.

NPJT may be confused with accelerated idioventricular tachycardia. Both rhythms behave similarly; however, the latter is always characterized by aberrant QRS complexes. The difficulty lies in the differentiation of accelerated idioventricular tachycardia from NPJT with aberrant conduction. The presence of fusion beats supports the diagnosis of ventricular arrhythmia. Recording of His bundle potentials may be useful in differentiating these rhythms.

**Mechanisms of NPJT**

NPJT appears to reflect an increase in A-V junctional automaticity, which allows the A-V junction to become the dominant pacemaker controlling the ventricles. Digitalis can enhance phase 4 diastolic depolarization in His-Purkinje cells. Several factors common to myocardial infarction may also increase His-Purkinje automaticity including catecholamine release, ischemia, and stretch. It is not clear why an individual patient with either digitalis intoxication or myocardial infarction should develop NPJT due to enhanced His bundle automaticity as opposed to a bundle-branch tachycardia or ventricular tachycardia arising in the more distal His-Purkinje system. NPJT could also reflect the development of automaticity in the N or A-N regions of the A-V node. There is little experimental justification for proposing the former site, but there is some evidence for the latter site.

The mechanisms allowing the development of NPJT have not been as clearly delineated as those
producing PJT. Further work is necessary in understanding the mechanism of NPJT.

**Management: NPJT**

NPJT usually occurs in the presence of organic heart disease. The hemodynamic consequences of this rhythm are usually minimal, since the QRS is supranodal and the ventricular rate is only modestly increased. The major disturbance reflects only a loss of atrial transport function.

Since NPJT is usually secondary to either digitalis intoxication or myocardial infarction, therapy should be directed toward the primary problem. In the former case, digitalis should be discontinued, since NPJT may be a forerunner of a more serious arrhythmia. Potassium can be administered if the patient is hypokalemic. In acute myocardial infarction, the arrhythmia is generally self-limited and does not necessitate therapy. If loss of atrial transport function is hemodynamically troublesome, administration of atropine may restore sinus rhythm by increasing sinus rate and facilitating A-V conduction. However, atropine can provoke a faster NPJT, which could be deleterious.

**References**

17. James TN: The connecting pathways between the sinus node and A-V node and between the right and the left atrium in the human heart. Amer Heart J 66: 498, 1963
27. Janse MJ, Van Capelle FJJ, Freud GE, Durrer D: Circus movement within the A-V node as a basis of supraventricular tachycardia as shown by multiple microelectrode recording in the isolated rabbit heart. Circ Res 28: 403, 1971

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