CRITICAL REVIEW

Atrioventricular Junctional Rhythm: Classification and Clinical Significance*

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A classification of atrioventricular (A-V) junctional impulse formation is presented, based on recent observations in clinical and experimental cardiology. Several examples of intermittent and persistent manifestations of A-V junctional impulse formation are correlated with experimental findings to illustrate the complexity of these arrhythmias. Their clinical significance and management are briefly discussed, and the role of cardiac glycosides in the genesis of these rhythm disorders is reemphasized.

The atrioventricular (A-V) junction is usually defined as "that portion of the specialized A-V transmission between the approaches to the A-V node in the lower right atrium and the bifurcation of the His bundle" (Fig 1). This region, although small, consists of several types of specialized fibers with dissimilar action potential characteristics and conductivity (Fig 2). By its strategic location, impulse formation originating in this region would interfere with either the forward transmission of atrial impulses or retrograde conduction of "ventricular" impulses in some instances, whereas it may gain control of the atria or ventricles, or both, in other instances.

These different manifestations of A-V junctional impulse formation result from a complex interaction of many factors, including the timing and localization of impulse formation, the conductivity of tissues above (toward the atria) and below (toward the ventricles), and even the mode of impulse formation (whether automaticity or reentry).

Our knowledge of the impulse formation and conduction in the A-V junction was extensively reviewed by Scherf and Cohen in 1964. However, recent studies, with precise mapping of the A-V junction with microelectrodes, and clinical studies utilizing His bundle electrograms suggest that the

FIGURE 1. Diagram of A-V junction and intraventricular conducting system. (de Azevedo IM, Watanabe Y, Dreifus LS: Heart Lung 1:626-638, 1972, published by permission.)
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S-A NODE

ATRIA

AN

A-V JUNCTION

N

NH

HIS BUNDLE

PURKINJE FIBER

VENTRICLES

200msec

Figure 2. Characteristic action potential configurations in various fiber types. Action potential duration is progressively prolonged from atria to peripheral Purkinje fiber. (de Azevedo IM, Watanabe Y, Dreifus LS: Heart Lung 1:626-638, 1972, published by permission.)

These classic concept of the disturbances of impulse formation and conduction must be modified. The purpose of this paper is to offer a more precise classification of A-V junctional impulse formation (Table 1). Several examples of these rhythms, as well as electrophysiologic correlation and clinical significance, will be discussed.

Classification of A-V Junctional Impulse Formation

Impulse formation near and within the A-V junction has historically been based on the polarity of the P wave and on the timing of the P wave relative to the QRS complex. Hence, through the years, many descriptive terms were introduced based on these criteria for identification of the site of impulse formation; they included coronary sinus rhythm, coronary nodal rhythm, and upper, middle and lower nodal rhythm. Recently, impulse formation arising in the region from the approaches to the A-V node and to the beginning of the branching portion of the A-V bundle has been termed junctional rhythm. This classification resulted from Hoffman's observation that automatic activity did not occur within the N region of the A-V node. However, later studies identified automatic activity within the other two layers (AN and NH) of the A-V node. Longitudinal and transverse dissociation (we called it inhomogeneous conduction) was possible within all three layers of the A-V node, which could lead to reentry mechanism within these regions. Hence, strictly speaking, the approaches, the A-V node proper, and areas both proximal and distal to the fibrous A-V ring could be responsible for junctional rhythms, whether due to automatic activity or re-entry or to combinations of both mechanisms.

Clearly, the temporal relation of the retrograde P wave to QRS depends not only on the site of impulse formation but also on the relative speed of conduction in one direction versus the other. Retrograde atrial excitation may not necessarily produce negative P waves in leads 2, 3 and aVF, at least under certain experimental conditions. The precise localization of a pacemaker within the A-V junction is difficult even with the use of multiple microelectrode recordings in isolated hearts, and is almost impossible in clinical cases, in which a far less accurate recording of the His bundle electrogram is the only method available. His bundle studies have identified the site of impulse formation as above or below the site of the close bipolar catheter electrode, but some notable exceptions to these observations demonstrated that the His spike was dependent on the relative time of the impulse toward the catheter.

Table 1—Classification of A-V Junctional Impulse Formation

<table>
<thead>
<tr>
<th>Intermittent manifestation of A-V junctional impulse formation</th>
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<tbody>
<tr>
<td>1 A-V junctional escape beats</td>
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<tr>
<td>2 A-V junctional premature systoles</td>
</tr>
<tr>
<td>a Coupled extrasystoles</td>
</tr>
<tr>
<td>b Parasytole</td>
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<tr>
<td>3 Paroxysmal A-V junctional tachycardia</td>
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<tr>
<th>Persistent manifestation of A-V junctional impulse formation</th>
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<tbody>
<tr>
<td>1 A-V junctional rate &lt; 70/min (A-V junctional rhythm)</td>
</tr>
<tr>
<td>a with dependent activation of atria and ventricles</td>
</tr>
<tr>
<td>b with independent activation of atria and ventricles</td>
</tr>
<tr>
<td>2 A-V junctional rate &gt; 70/min (nonparoxysmal A-V junctional tachycardia)</td>
</tr>
<tr>
<td>a with dependent activation of atria and ventricles</td>
</tr>
<tr>
<td>b with independent activation of atria and ventricles</td>
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In addition, subjunctional impulse formation can now be localized within the various fascicles of the His-Purkinje system.

On the other hand, it is clear that any classification of A-V junctional rhythms, although purely arbitrary, can be suitable for clinical purposes. Many classic terms such as "coronary sinus" and "coronary nodal rhythms," and "upper, middle and lower rhythms," have been replaced by a simple category, "A-V junctional rhythm," since these differences are not warranted in view of recent electrophysiologic studies.

**Intermittent Manifestation of A-V Junctional Impulse Formation**

Figure 3 is an interesting example of junctional escape beats in the presence of S-A block and A-V block. Sinus rhythm is present at a rate of approximately 70 per minute in both upper strips aVL and V2. The P-R interval is 0.20 second. The second and the sixth QRS in aVL, and the fourth and seventh in the V1, are not preceded by P waves; they appear late (R-R intervals from 1.18 to 1.26 second) and represent junctional escape beats. The basic R-R intervals measure 0.86 second, and the prolonged R-R intervals after the escape beats represent the return cycle of the A-V junctional escape pacemaker. In lead 2 (lower strip, as shown by diagram) sinus rhythm is present at a rate of 65 per minute. After the fourth QRS complex, which appears slightly early, there is a long pause of 1.52 second, which is terminated by a QRS complex not preceded by a P wave, with the same contour of the basic rhythm, and which represents a junctional escape. This occurs because an S-A block is present, with the Wenckebach phenomenon. Immediately after the QRS of escape beat, a nonconducted sinus P wave is seen on the S-T segment, as shown by the diagram. The longer pause of 1.60 second is terminated again by junctional escape, followed by a blocked P wave. The P-P intervals are shorter than twice P-P of the basic rhythm, and show progressive shortening, characteristic of an S-A block with Wenckebach periodicity. On the other hand, the sinus impulse is...
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FIGURE 5. Example of A-V junctional parasystole.

blocked when it arrives immediately after the escape of the junctional pacemaker (beats 5 and 6). These escape mechanisms represent physiologic A-V junctional automaticity, and may appear in the presence of (1) increased vagal tone; (2) depression automaticity of the S-A node, especially following ectopic atrial excitation; (3) sick sinus syndrome; and (4) A-V as well as S-A block. The return cycle of the S-A node should not exceed 1.5 second.

In Figure 4, sinus rhythm is present at a rate of approximately 75 per minute, with sinus arrhythmia. The P-R interval is 0.13 second. In lead II, after the second QRS complex, a premature beat occurs with a coupling interval of 0.50 second, and is not preceded by a P wave. The similar contour of the basic rhythm represents a junctional premature systole. After the early QRS complex, a P wave from the sinus origin is seen on the S-T segment and is blocked, falling within the absolute refractory period of the A-V junction. In the lower strip (lead 2), the junctional premature systole occurs earlier (0.48 second), as shown by the diagram; the sinus impulse falls within the relative refractory period of the A-V junction and is conducted with a prolonged P-R interval. The electrophysiologic mechanisms that produce these extrasystoles are still unclear, although a reentry movement is a possibility in view of various experimental studies.

A parasystolic pacemaker located in the A-V junctional region could also cause premature systoles (Fig 5). In lead Y, sinus rhythm is interrupted by frequent premature systoles showing inverted P waves preceding the QRS complexes. When the coupling intervals are shortened, the QRS complexes

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show morphologic evidence of aberrant intraventricular conduction. As indicated in the diagram, the coupling intervals are variable and the interectopic intervals are multiples of the shortest intervals. Thus, an A-V junctional parasystole is identified at a rate of approximately 38 per minute. Although the precise mechanism for parasystole is still unknown, automaticity is the most likely explanation.\(^\text{13}\)

In Figure 6, an example of junctional paroxysmal tachycardia is shown. In the top strip (lead 2), there is a regular rhythm; although the P waves are not clearly seen, they are easily identified in the right atrial lead (A). His deflections precede and follow QRS. The retrograde H spike is of different contour (H'). The A-H and H'-A intervals have the same duration, while the V-H' interval is almost double the H-V interval. The diagram shows the characteristics of orthograde and retrograde conduction. The atrial electrogram indicated that P waves were superimposed on T waves of lead 2 of the electrocardiogram. Unfortunately, the onset of the reciprocal tachycardia, generally preceded by premature systoles, was not seen. In this case, the impulse is conducted with reduced velocity in retrograde fashion from V to His (H-V = 55, V-H' = 106). The atrial-His and His-atria conduction remain constant. Functional longitudinal dissociation of the A-V junction may explain reciprocal junctional tachycardia. A crucial amount of conduction delay in either a forward or a retrograde direction is essential in the genesis of this type of arrhythmia. The ventricular rate during such tachycardia usually exceeds 150 per minute, and both the onset and termination are abrupt.

![Figure 7. A-V junctional rhythms with dependent activation of atria and ventricles. (de Azevedo IM, Watanabe Y, Dreifus LS: Heart Lung 1:626-638, 1972, published by permission.)](image)

![Figure 8. Example of A-V junctional rhythm with independent activation of atria and ventricles. Atrial fibrillation is present, although ventricular rate is quite regular at 60 per minute (upper two strips). (Lower two strips). Rate is 30 per minute. Hence, exit block from A-V junctional pacemaker is present together with atrial fibrillation.](image)
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Figure 9. Exit block from A-V junctional pacemaker in rabbit heart. N1 = action potential from perinodal atrial fiber; N2 = action potential (polarity inverted) from NH region. X on schematic map shows site of impulse formation. Action potential record and diagram illustrate both forward (node-ventricular) and retrograde (node-atrial) propagation failure of fourth and fifth impulses, respectively. (Dreifus LS, Watanabe Y, Byer B: Ann NY Acad Sci 167:950, 1969, published by permission.)

PERSISTENT MANIFESTATION OF A-V JUNCTIONAL IMPULSE FORMATION

A-V Junctional Rhythm

Formerly, the rhythms depicted in Figure 7A, B and C were called upper, middle and lower A-V nodal rhythms, respectively. In all these instances, the atria and the ventricles were considered to be activated by an impulse originating in the A-V junctional region (dependent activation). The relative timing of the P and QRS is determined by the retrograde versus the forward conduction time from the junctional pacemaker. Normal QRS duration in these records suggests impulse formation above the branching portion of the His bundle, and the rate (less than 70 per minute) probably indicates physiologic automaticity of the A-V junction.

Figure 8 shows independent activation of atria and ventricles. In this example, there is atrial fibrillation, and the ventricles are under the control of a junctional pacemaker at a rate of 64 per minute. In V2, the R-R intervals become more prolonged and measure 1.83 second, almost twice that seen in 1 and 2, probably because of exit block from the subsidiary junctional pacemaker. Examples of exit block from a pacemaker located in a specific region of the A-V junction have been demonstrated recently with microelectrode techniques (Fig 9). In junctional rhythm with exit block, the cause of block is attributed mainly to a small action potential amplitude with a slow rate of depolarization, a mechanism favoring decremental conduction.

Nonparoxysmal A-V Junctional Tachycardia

When the rate of junctional impulse formation exceeds 70 per minute, an accelerated mechanism is suggested. In contrast to the paroxysmal variety (Fig 6), the nonparoxysmal type develops gradually and disappears also slowly. This variety is most often the result of excessive cardiac glycosides. Figure 10 shows an example of nonparoxysmal junctional tachycardia with dependent activation of the atria and ventricles. A junctional pacemaker is present at the rate of approximately 85 per minute, and the atria are activated in a retrograde direction. A progressive prolongation of the retrograde conduction is seen, and when a critical retrograde delay occurs, reciprocation occurs, as seen in the first,

Figure 10. Example of nonparoxysmal A-V junctional tachycardia with dependent activation of atria and ventricles and reciprocal beats.

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An A-V junctional nonparoxysmal tachycardia with predominant A-V dissociation is shown in Figure 11. In the upper strip, lead I, the R-R intervals measure 0.80 second and alternate with pauses of 0.92 second, caused by concealed conduction of sinus impulses, which are superimposed on T waves and penetrate deeper into the A-V junction, as illustrated by the diagram. The lower diagram shows that the R-R intervals sometimes become shorter and represent ventricular capture beats (third, sixth, and ninth QRS complexes). The rate of the junctional pacemaker increases from 100 to 115 per minute.

Figure 12 shows concealment within the A-V junction. Short (234 msec) and long (262 to 264 msec) A-V intervals alternate. The mechanism for this alternation is apparent in the action potential recording from the NH region, in which every fourth atrial beat fails to evoke a propagated response. Thus, there is 4:3 conduction across the A-V node, with block of the fourth impulse above the NH fiber. Progressive prolongation of the intranodal conduction time of the Wenckebach type is evident prior to the failure of nodal transmission. However, the second of these three impulses successfully traversed the A-V node is blocked below the distal NH region, permitting only the first and third atrial impulses to be conducted to the ventricles. The His-Purkinje conduction time in the transmission of the third atrial impulse (140 msec) is most probably a result of partial penetration of the second impulse into the His-Purkinje system.17

Figure 13 shows so-called bidirectional tachycardia. There is regular alternation of predominantly upright and downward QRS complexes, at the rate of approximately 167 per minute. Alternation of narrower and wider QRS complexes is also evident. The atrial activity is hardly discernible, and atrial fibrillation cannot be ruled out. Although the true electrophysiologic mechanism of this variety of arrhythmia is still unknown, an A-V junctional (or
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Figure 13. Example of so-called bidirectional tachycardia. We are indebted to Dr. L. Shocket for permission to publish this figure.

supraventricular) tachycardia with alternating left anterior and left posterior division block (in addition to a conduction delay in the right bundle branch system) is one likely explanation. A basic supraventricular tachycardia with coupled ventricular premature systoles causing bigeminy is an alternative mechanism, at least in some instances. However, in this particular example, the former explanation is the most probable.

SIGNIFICANCE AND MANAGEMENT OF DISORDERS OF A-V JUNCTIONAL IMPULSE FORMATION

From the various forms of A-V junctional impulse formation described, it is apparent that some are grossly abnormal and require urgent therapy, while others are benign and need no specific treatment. Excessive digitalis therapy appears to be the most important factor engendering disorders of impulse formation and conduction in the A-V junction and withdrawal of cardiac glycosides is the first step to take in those cases.

In the category of intermittent manifestation of A-V junctional impulse formation, A-V escape beats are an expression of physiologic automaticity of the junctional tissues and are beneficial in preventing prolonged periods of ventricular asystole. A-V junctional premature systoles rarely require specific therapy. Usually, mild sedation or administration of small doses of quinidine or a β-receptor blocking agent such as propranolol, will effectively eliminate these problems. The significance of A-V junctional parasystole is, on the other hand, still not entirely clear, as this arrhythmia has been observed in both diseased and seemingly normal hearts. Therefore, whether this type of A-V junctional impulse formation should be treated probably depends on the underlying pathophysiologic condition of the heart. Paroxysmal A-V junctional tachycardia also occurs sometimes in apparently healthy subjects. Such reciprocal tachycardia is initiated by either atrial or ventricular premature systoles in most instances. Hence, abolition of these premature beats would, at least theoretically, prevent recurrence of this arrhythmia, and the treatment of paroxysmal junctional tachycardia may be reduced to the management of atrial or ventricular extrasystoles. However, in certain patients in whom an associated conduction disturbance within the A-V junction is suspected, a different method of treatment may be necessary. Often carotid sinus pressure or other maneuvers to increase the vagal tone could terminate this arrhythmia, possibly by depressing conduction in the A-V junction and interrupting the reciprocal movement during either forward or retrograde transmission of such impulses. If a paroxysmal junctional tachycardia recurs frequently, causes hemodynamic deterioration, and is considered to be the result of an excessive intake of cardiac glycoside, this drug should be withdrawn and intravenous administration of potassium salts should be seriously considered. Treatment with propranolol has been extremely beneficial in patients with reciprocal tachycardia.

In the category of persistent manifestation of A-V junctional impulse formation, A-V junctional rhythms, with dependent activation of the atria and the ventricles, often occur in normal subjects. Usually no therapy is indicated. In contrast, A-V junctional rhythms with independent activation of the atria and the ventricles are most commonly seen in the presence of high-grade A-V blocks. When the rhythm is considered a result of intranodal conduction block showing normal intraventricular conduction (normal QRS), it may not require specific treatment or may require only temporary pacing. These conditions often follow an acute inferior wall infarction or excessive administration of cardiac glycosides, and are usually transient. In other cases, in which the high-grade A-V block is caused by a subnodal conduction disturbance, the QRS is pro-
longed and it is often difficult to distinguish between A-V junctional and idioventricular rhythms. Recent observations of so-called fascicular rhythms (escape rhythms originating in the fascicular portions of either the anterior or posterior fascicles of the left bundle branch)\textsuperscript{24} illustrate these difficulties, even with the use of His bundle recordings. Nevertheless, this variety frequently necessitates the insertion of a permanent electronic pacemaker.

Of the persistent manifestations of A-V junctional impulse formation, nonparoxysmal A-V junctional tachycardia is almost never encountered in the absence of cardiac disorders. This type of tachycardia most often results from excessive digitalis therapy, and may even be a more common variety of digitalis toxicity than the well-known "paroxysmal atrial tachycardia with block."\textsuperscript{15} This nonparoxysmal junctional tachycardia offers a poorer prognosis than the paroxysmal variety and the A-V junctional rhythms. Generally, nonparoxysmal A-V junctional tachycardia associated with intracardiac surgery, acute myocardial infarction or myocarditis regresses spontaneously, and either does not require specific therapy or may successfully be treated with propranolol.\textsuperscript{10} In contrast, those resulting from excess of cardiac glycosides are associated with the poorest prognosis, since the mortality may increase to almost 80 percent when the glycoside therapy is continued.\textsuperscript{16} Thus, withdrawal of digitalis, with or without supplemental treatment with potassium, is the first choice of therapies. Although some cases of bidirectional tachycardia may be caused by the presence of two independent pacemaking foci, one supraventricular and the other ventricular,\textsuperscript{22} rather than an A-V junctional tachycardia with altered intraventricular conduction, this arrhythmia almost always occurs in patients with a severe organic lesion of the heart, and is associated with high mortality.

REFERENCES
6 Watanabe Y, Dreifus LS: Newer concepts in the genesis of cardiac arrhythmias. Am Heart J 76:114, 1968
8 Bix HH: A-V nodal rhythm with various types of A-V block. Mt Sinai J Med NV 2:42, 1953