Nonparoxysmal Junctional Tachycardia
Complicating Acute Myocardial
Infarction

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SUMMARY
Twenty-one instances of nonparoxysmal junctional tachycardia (NPJT) were observed in 203 consecutive patients with acute myocardial infarction. The arrhythmia complicated both anterior and inferior wall infarction and was associated with an ominous prognosis, particularly in patients with anterior wall infarction.

The NPJT is probably a manifestation of the severity of the lesion. The exact mechanisms of the enhanced A-V junctional activity remain obscure. The possible role of increased levels of epinephrine and norepinephrine and localized increases of potassium in the A-V junction from adjacent necrotic myocardium is suggested.

Additional Indexing Words:
Arrhythmia Junctional tachycardia Nodal tachycardia

A trioventricular (A-V) junctional tissue possesses properties of impulse conduction and of latent automaticity.1 Spontaneous impulse formation may be passive, resulting from either slowing of the primary pacemaker or failure of the dominant impulse to traverse the junction. Or, under certain, usually pathologic, situations, the automaticity of the A-V junction may be suddenly enhanced, and an A-V junctional tachycardia with rate exceeding that of the primary pacemaker appears. The limits of ventricular
total and rates in passive A-V junctional rhythms vary from 35 to 50/min and in paroxysmal A-V junctional tachycardia from 150 to 220/min. In addition, Pick and Dominguez2 described a third junctional tachyarrhythmia, namely, nonparoxysmal junctional tachycardia (NPJT), which differs from either of the above in that the ventricular rates are in the range of 70 to 130/min, the onset and termination of the arrhythmia are gradual, and as a rule a causal factor responsible for the arrhythmia can be recognized. NPJT has been attributed primarily to four conditions: digitalis toxicity, acute rheumatic fever, open-heart surgery, and acute inferior wall infarction.1-4 In each of these instances, the arrhythmia is a manifestation of a pathophysiologic disorder involving the junctional tissue.2

The purpose of this report is to present the incidence of this arrhythmia in patients with acute myocardial infarction, to point out that the arrhythmia complicates both inferior and anterior wall infarction, and to focus attention on the high mortality which appears to accompany this arrhythmia when due to myocardial infarction. In addition, possible
theoretic mechanisms responsible for the arrhythmia are suggested.

**Methods**

During the period of this study, 203 patients with unequivocal acute myocardial infarction were admitted to the coronary care unit. Continuous oscillographic monitoring was carried out in all, with hourly rhythm strips recorded in some while the ECG was taped without interruption in others. In each case the age and sex, anatomic area of infarction, atrial and ventricular rates, onset of NPJT, other complications of infarction, and the final outcome were noted. Special attention was paid to the relationship, if any, of the NPJT to drugs which are known to accelerate the junction, for example, digitalis or isoproterenol.

In this group of 203 patients, 21 developed NPJT with a ventricular rate above 60/min.5

**Results**

The pertinent clinical information for each of the 21 patients with NPJT is given in table 1. Of the 21 patients 13 had an acute inferior wall infarction, seven had an acute anterior wall lesion, and one had both anterior and inferior wall involvement. Thirteen of the 21 patients died, a mortality rate of 62%. Of the 13 patients with inferior infarction, six died, a mortality of 46%. All of the seven patients with anterior myocardial infarction died, and all manifested cardiogenic shock. The one patient with anterior and inferior wall infarction survived.

In addition to the location of the infarction, the mortality appeared to be related to the ventricular rate of the NPJT, in that the higher the rate, the greater the mortality. All patients with a ventricular rate of 80/min or above died (table 1). In all but two cases, the onset of the arrhythmia appeared within the first 36 hours or less, the majority being present on admission to the coronary care unit. Digitalis or isoproterenol therapy had not preceded the onset in any of the patients.

The following manifestations of NPJT were observed:
1. Independent atrial and ventricular rhythms with the more rapid atria under the control of the SA node and the slower ventricles under the control of the junction, with depression of A-V conduction making the A-V dissociation possible (fig. 1). This condition occurred in 12 of the 21 patients. Ten of the 12 had inferior infarction, and four died with cardiogenic shock.

2. A-V dissociation with the ventricular rate exceeding the atrial rate (fig. 2). In such situations the A-V dissociation may be maintained without any depression of A-V conduction.

Figure 1

A-V dissociation with an atrial rate of 132 and ventricular rate of 80. No captures are seen, indicating that some degree of A-V conduction depression contributes to the A-V dissociation.

Figure 2

Leads 2 and right intraatrial (RIA). A-V dissociation with an atrial rate of 100 and ventricular of 150. Because of the faster ventricular rate, the A-V dissociation can be maintained through physiologic interference without the necessity of invoking A-V depression. The QRS of sinus origin (not shown) had the same morphology as during the run of NPJT.
Figure 3

Continuous recording of lead V2 in a patient with an acute anterior myocardial infarction. AV dissociation is clearly present at the end of top row and beginning of the bottom row. The R-R interval during dissociation is 0.72 second. The somewhat aberrant QRS might at first suggest an accelerated idioventricular rhythm. However, foreshortening of the R-R interval following the fifth QRS, lower strip, to 0.68 with a fixed P-R interval indicates supraventricular conduction with an identical morphology of the QRS.

3. Gradual shifting of the ventricular control from junctional to the SA node (fig. 3).

4. Gradual emergence of the NPJT with both the atria and ventricles under control of the junctional focus (fig. 4, top row).

5. Sudden captures of the ventricles by the sinus impulse (fig. 4, bottom row).

6. Double junctional rhythms (fig. 5).

7. NPJT with exit block (fig. 6).

Discussion

As indicated earlier, under certain well-defined clinical conditions the A-V junction with latent pacemaker properties may, by virtue of inappropriately rapid phase-4 depolarization, become the dominant pacemaker site. The anatomic site of this junctional activity is probably the nodal-His or the bundle of His region. When this enhancement results in a rate of 70 to 150/min, the arrhythmia emerges and disappears gradually, and the QRS duration and morphology are similar to the normally conducted beats, then NPJT as defined by Pick and Dominguez can be said to be present. We feel that rates between 60 and 70/min represent acceleration of the junction, and thus all patients with an A-V junctional rate of 60/min or above are included in this study.

The electrocardiographic manifestations of NPJT may vary from case to case. Occasionally, the atria are under the control of the junctional focus, and retrograde P waves are recorded in leads 2, 3, and AVF (fig. 4). More often, however, the atria exhibit an independent rhythm, be it sinus, atrial tachycardia, flutter, or fibrillation. Double junctional tachycardia is recognized by inverted P waves in leads 2, 3, and AVF and a normal QRS, with a regular atrial and ventricular rhythm but at different rates and without temporal relationship between the two (fig. 5).

Most often in NPJT the ventricular rate is faster than the atrial. If the atria are controlled by the SA node and the ventricles by the A-V junction and both foci have similar but slightly variable rates, such as may be present with sinus arrhythmia, a gradual shift
Top strip, lead 2, recorded from a patient with an acute inferior infarction, shows a gradual emergence of a junctional rhythm and control of both chambers by the junctional focus. The gradual emergence of what most likely is an automatic rather than a reentrant focus is supported by atrial fusions (fifth and sixth P waves). The second strip (V2) was recorded in a patient with an acute anterior myocardial infarction. The basic rhythm is a sinus arrhythmia with A-V dissociation with a junctional R-R of about 0.60 second. The sixth and seventh QRS represent captures by sinus impulses.

A-V dissociation with both pacemakers located within the A-V junction (double junctional tachycardia) in the presence of acute inferior infarction. With exception of the third P in L2, which is sinus in origin, and perhaps the next to last P in L3, which may be a fusion, the remaining P waves in L2 and L3 are inverted, indicating junctional origin. The P-P varies slightly between L2 and L3 from 0.96 to 0.88 second. The R-R is constant at 0.66 second. There are four captures, one by the sinus impulse (L2) and three by the upper junctional focus. The constancy of P-P over a number of cycles and of the R-R without any fixed P-R or R-P relationship establishes the diagnosis of intranodal dissociation.
Figure 6

Acute subendocardial injury which evolved into an acute inferior infarction with A-V dissociation with a P-P of 0.72 and R-R of 0.68. A 2:1 exit block from the junctional focus is present in V5.

of the control of the ventricles from the SA node to the A-V junction and back to the SA node may be seen. This is demonstrated in figure 3. In fact, this gradual emergence and disappearance is one of the characteristic features of NPJT. If the appearance of NPJT is associated with retrograde activation of the atria from the junctional focus, the gradual shift of control from the A-V junction to the SA node or SA node to the A-V junction may result in various degrees of atrial fusion (fig. 4). Such a gradual emergence of NPJT without accurate coupling to the preceding QRS suggests that enhanced automaticity, rather than reentry, is the mechanism of NPJT.

In the presence of sinus or atrial tachycardia or flutter, NPJT is diagnosed by a normal QRS with a regular rhythm and a lack of temporal relationship between the atrial and ventricular complexes. This is demonstrated in figure 2 where an intraatrial electrogram was recorded simultaneously with a surface lead 2. The tracing demonstrates an atrial rate of 110/min and a ventricular rate of 150/min with an A-V dissociation. Similarly, in the presence of atrial fibrillation, the appearance of a regular ventricular rhythm with a rate within the range of NPJT suggests this diagnosis.

Occasionally, the independent atrial impulse will fall outside the refractory period of the A-V junction and will activate (capture) the ventricle (see fig. 4). This is less likely to happen with rapid atrial rhythms, such as atrial tachycardia, flutter, or fibrillation, because concealment of the “ever” available impulse into the A-V junction results in refactoriness, which, when coupled with the refractoriness generated by the A-V junctional focus, prevents ventricular capture.

NPJT with a 1:1 exit from the ectopic focus and, consequently, a regular ventricular rate when accompanied by a normal QRS complex poses few diagnostic problems. However, in the presence of an exit block (fig. 6) and consequent variation in heart rate, the A-V junctional origin of the tachycardia may not be easily recognized.

Nonparoxysmal junctional tachycardia has to be differentiated from accelerated idioventricular rhythms (“slow” ventricular tachycardia), a very common and relatively benign complication of myocardial infarction. The differential point is that in NPJT the QRS morphology is identical to the supraventricular impulse or only slightly aberrant. The presence of fusion and capture complexes makes a diagnosis of idioventricular rhythm fairly secure. There remains the possibility that idioventricular rhythm originating in the
septum may result in QRS complexes identical to those originating in the atrium, and consequently, a differentiation may not be possible. However, such a combination of circumstances is very rare indeed.

The exact etiology of NPJT is uncertain. It is possible that the arrhythmia is the result of the systemic electrolyte and metabolic disturbances which accompany severe myocardial damage. Consequent to myocardial infarction there may be an increase in circulating epinephrine and norepinephrine which in the hypoxic state have a more pronounced effect on the A-V junction than the SA node.7-9 Whether these factors contribute to the genesis of NPJT remains to be seen. However, the high incidence of cardiogenic shock and relatively high mortality in this series suggest very strongly that the extent of infarction and attending severe pathophysiologic changes play an important role in the genesis of the NPJT. In addition, because of the association with the shock state, a reflex increase in adrenergic drive could play a substantial role. This would be particularly indicated in the patients who showed depressed A-V conduction. In this group of patients, both the sinus and junctional rates were increased, suggesting that both pacemakers were influenced.

Another possibility is that the A-V junctional enhanced automaticity is produced by a localized increase in potassium in the area of the A-V junction due to its release from the adjacent necrotic tissue.10 NPJT has been induced in dogs by elevating plasma levels of potassium.11 The cation reduces the resting potential of the A-V junctional cells12 and tends to lower the threshold potential. If the latter condition predominates, the rate of the A-V junctional pacemaker will be enhanced. NPJT has been observed clinically in a young boy with unsuspected renal failure whose serum potassium was abruptly and inadvertently elevated.13

The arrhythmia was recorded in 10% of the patients in this series. While earlier reports described this arrhythmia as a complication of acute inferior wall infarction,1 2 it is noteworthy that in this series eight of 21 patients or 38% had an acute anterior wall lesion. Earlier studies suggest that the appearance of NPJT in the presence of an acute infarction may not carry a significantly higher mortality.1 2 In our series six of the 14 patients with inferior wall infarction died, a mortality rate of 43%, and seven of eight patients with anterior wall involvement died, a mortality rate of 87%. Furthermore, if one assumes that the patient who had both anterior and inferior wall infarction and survived had a predominantly inferior wall infarction, then the mortality rate for the anterior wall infarction group would be 100%.

The discrepancy between our observations and those of earlier workers may well be due to a paucity of studies of this specific arrhythmia in acute myocardial infarction.

The more rapid ventricular rates are probably a reflection of more extensive myocardial damage with its attendant pathophysiologic aberration and thus the higher mortality.

It appears from this study that NPJT in the presence of an acute myocardial infarction, especially if the ventricular rate is rapid and the location of the infarction anterior, is related to the severity of the myocardial damage and carried with it a serious prognosis.

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