Nonparoxysmal A-V Nodal Tachycardia

By Alfred Pick, M.D., and Pedro Dominguez, M.D.

Acceleration of impulse formation in the ordinarily subsidiary A-V nodal pacemaker is manifested in clinical electrocardiograms in a paroxysmal and a nonparoxysmal variety. The former occurs as a rule in normal hearts, the latter in pathologic conditions, frequently, but not invariably also causing A-V block. The electrocardiographic and clinical aspects of nonparoxysmal nodal tachycardia were studied in 30 cases and the relative importance of the disturbance was evaluated in 3 conditions in which it is found most frequently, namely, digitalis effect, acute rheumatic fever, and recent posterior wall infarction.

MANIFESTATIONS of the pacemaker function of the A-V node are, according to current concepts, classified into 2 types¹:

1. A “passive” form in which the A-V node escapes the control of the primary pacemaker because the latter releases impulses at a slower rate than the A-V node, or because primary (sinus) impulses, discharged at a normal rate, are prevented by a block from reaching the secondary pacemaker and suppressing its activity.

2. An “active” form in which the A-V node gains control of ventricular or atrial activation by virtue of a sudden and marked enhancement of impulse formation resulting in single or a run of premature beats—rapid rhythms of the latter type being termed paroxysmal A-V nodal tachycardias. In interpreting clinical electrocardiograms the range of rates of “passive” nodal rhythms was found to vary between about 35 and 50 and that of paroxysmal nodal tachycardias between about 150 and 220.

The present report deals with a type of enhanced A-V nodal activity that does not conform with the above criteria in 2 respects: (1) the rate of the nodal discharge is only moderately accelerated, from about 70 to 130 and (2) the ectopic rhythm lacks the sudden onset and abrupt termination characteristic of the paroxysmal forms of nodal tachycardias. The existence of this type of ectopic impulse forma-

From the Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

Aided by grants from the Michael Reese Research Foundation and the Herbert G. Mayer Memorial Fund for Cardiovascular Research.

Presented in part at the Fifth Interamerican Congress of Cardiology, Havana, Cuba, November, 1956.
<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yrs.)</th>
<th>Clinical diagnosis</th>
<th>Cause of ectopic rhythm</th>
<th>Ectopic rate (beats/min.)</th>
<th>Basic rhythm</th>
<th>A-V block present</th>
<th>Resulting disturbance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>HHD, CHF</td>
<td>Digitalis</td>
<td>75</td>
<td>AF</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>2</td>
<td>51</td>
<td>H &amp; AHD old ant. infarct</td>
<td>Digitalis</td>
<td>78</td>
<td>SR</td>
<td>Yes</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>RHD, CHF</td>
<td>Digitalis</td>
<td>75</td>
<td>AF</td>
<td>Yes</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>Pneumonia, CHF</td>
<td>Digitalis</td>
<td>73</td>
<td>SR</td>
<td>Yes</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>5</td>
<td>67</td>
<td>AHD, CHF</td>
<td>Digitalis</td>
<td>80</td>
<td>SR (with S-A block)</td>
<td>Yes</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>AHD</td>
<td>Digitalis</td>
<td>94</td>
<td>SR</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>9</td>
<td>61</td>
<td>H &amp; AHD, CHF</td>
<td>Digitalis</td>
<td>110</td>
<td>AF</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>11</td>
<td>70</td>
<td>AHD</td>
<td>Digitalis</td>
<td>79</td>
<td>ST</td>
<td>Yes</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>12</td>
<td>83</td>
<td>AHD, CHF</td>
<td>Digitalis</td>
<td>94</td>
<td>AF</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>13</td>
<td>43</td>
<td>RHD, CHF</td>
<td>Digitalis</td>
<td>111</td>
<td>SA</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>14</td>
<td>65</td>
<td>AHD, CHF</td>
<td>Digitalis</td>
<td>68 → 75</td>
<td>AF</td>
<td>Yes</td>
<td>Complete A-V dissoc.</td>
</tr>
<tr>
<td>(fig. 2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>75</td>
<td>AHD, CHF</td>
<td>Digitalis</td>
<td>100</td>
<td>ST</td>
<td>?</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>16</td>
<td>84</td>
<td>HHD, CHF</td>
<td>Digitalis</td>
<td>75</td>
<td>SA</td>
<td>No</td>
<td>Retrograde conduction → incomplete A-V dissoc.</td>
</tr>
<tr>
<td>17</td>
<td>9</td>
<td>ARF</td>
<td>Carditis</td>
<td>130</td>
<td>ST</td>
<td>No</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>18</td>
<td>15</td>
<td>RHD—reactivated</td>
<td>Carditis</td>
<td>107–130</td>
<td>ST</td>
<td>No</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>19</td>
<td>7</td>
<td>ARF</td>
<td>Carditis</td>
<td>136</td>
<td>ST</td>
<td>No → Yes</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>(fig. 4)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>3</td>
<td>ARF</td>
<td>Carditis</td>
<td>85</td>
<td>SA</td>
<td>No</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>21</td>
<td>7</td>
<td>ARF</td>
<td>Carditis</td>
<td>88</td>
<td>SA</td>
<td>No</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>22</td>
<td>12</td>
<td>ARF</td>
<td>Carditis</td>
<td>120 → 60</td>
<td>ST → SA</td>
<td>No</td>
<td>Incomplete → intermittent A-V dissoc.</td>
</tr>
<tr>
<td>(fig. 5)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>23</td>
<td>ARF</td>
<td>Carditis</td>
<td>100</td>
<td>SA</td>
<td>No</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>24</td>
<td>62</td>
<td>MI</td>
<td>Recent post. infarct</td>
<td>75</td>
<td>SA</td>
<td>No</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>25</td>
<td>75</td>
<td>MI</td>
<td>Recent post. infarct</td>
<td>71</td>
<td>SA</td>
<td>No</td>
<td>Intermittent A-V dissoc.</td>
</tr>
<tr>
<td>26</td>
<td>40</td>
<td>MI</td>
<td>Recent post. infarct</td>
<td>75</td>
<td>SA</td>
<td>No</td>
<td>Intermittent A-V dissoc.</td>
</tr>
</tbody>
</table>

**Table 1.—Clinical and Electrocardiographic Findings in Thirty Cases with Nonparoxysmal A-V Nodal Tachycardia**
NONPAROXYSMAL A-V NODAL TACHYCARDIA

Table 1.—Continued

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yrs)</th>
<th>Clinical diagnosis</th>
<th>Cause of ectopic rhythm</th>
<th>Ectopic rate (beats/min)</th>
<th>Basic rhythm</th>
<th>A-V block present</th>
<th>Resulting disturbance</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>55</td>
<td>MI</td>
<td>Recent post. infarct</td>
<td>70</td>
<td>SR</td>
<td>Yes</td>
<td>Incomplete A-V dissoc.</td>
</tr>
<tr>
<td>28</td>
<td>67</td>
<td>Acute ehole-cystitis</td>
<td>?</td>
<td>107</td>
<td>?</td>
<td>No</td>
<td>Retrograde conduc-</td>
</tr>
<tr>
<td>29</td>
<td>25</td>
<td>Anxiety</td>
<td>?</td>
<td>93</td>
<td>SR</td>
<td>No</td>
<td>Retrograde conduc-</td>
</tr>
<tr>
<td>30</td>
<td>32</td>
<td>Trilogy of Fallot</td>
<td>?</td>
<td>96-107</td>
<td>SA</td>
<td>No</td>
<td>Retrograde conduc-</td>
</tr>
</tbody>
</table>

HHD, Hypertensive heart disease.  
AHD, Arteriosclerotic heart disease.  
RHD, Rheumatic heart disease.  
CHF, Congestive heart failure.  
ARF, Acute rheumatic fever.

established cause of the nodal tachycardia. Thus, the first and largest group consists of 16 cases in which a nonparoxysmal nodal tachycardia was encountered in the course of protracted or acute digitalization. The second group comprises 7 instances during acute rheumatic fever. In a third group of 4 cases the disturbance was found during early stages of posterior wall infarction. A final group of 3 consists of instances with 3 different and completely unrelated diagnoses and in which the cause of the protracted acceleration of the subsidiary pacemaker could not be established on clinical grounds.

In analyzing electrocardiographic and clinical data of these cases primary attention was paid to the following points: (a) the mode of onset, perpetuation, and termination of the nodal tachycardia, (b) the various secondary effects of the disturbance with regard to the pre-existing fundamental rhythm, (c) the presence or absence of a pre-existent, simul- taneous, or subsequent disturbance of A-V conductivity, and (d) the correlation of serial electrocardiograms with all the clinical data. Representative examples of nonparoxysmal nodal tachycardia and of resulting simple or complex disorders of rhythm are illustrated in figures 1-5.

In figure 1 (case 30) a gradual acceleration, from 96 to 107, is seen in both the atrial and ventricular rates. Concomitantly, the P waves become at first smaller and then inverted, while the P-R interval shortens from 0.16 to 0.12 second. Toward the end of the tracing, the P waves gradually return to their original upright contour and P-R lengths to 0.16 second, whereas the rate remains at 107. Thus, here, unlike paroxysmal nodal tachycardia, the A-V nodal pacemaker accelerates pari passu with speeding up of the sinus pacemaker. At first the 2 compete for the control of the atria with atrial fusion beats resulting; soon the nodal tachycardia exceeds the sinus tachycardia and nodal impulses dominate both the atria and ventricles; eventually the nodal pacemaker loses, and the sinus pacemaker gains speed. As a consequence, after another short period of competition, the sinus pacemaker once again controls both the atria and ventricles.

In figure 2 (case 14), on 12-1, adequate digitalization is evident from the ST-T configuration, the slow ventricular rate (averaging 55) during atrial fibrillation, and the occurrence of 2 nodal escapes (E); the latter are diagnosed on the basis of the equal R-R intervals (1.28 seconds, corresponding to a rate of 47)—the longest of the entire record.

On 12-4 and 12-5, the ST-T deflection is more marked and the ventricular rate is regular throughout. In the presence of atrial fibrillation this regularity indicates complete A-V dissociation. The nodal cycle is shorter than before, corresponding on 12-4 to a rate of 62, and 72 on the following day.

The record on 12-15 (10 days after digitalis was stopped) resembles that on 12-1, in that the ST-T abnormality is less marked and the ventricular action is irregular except for 2 nodal escapes (E). However, the average ventricular rate (75) as well as the nodal escape rate (65) are faster than originally.

The sequence of events in this series of records demonstrates the 2-fold action of digitalis upon A-V junctional tissues, depression of impulse transmission and enhancement of impulse formation. Here the 2 effects can be clearly separated. Impairment of conductivity preceded the acceleration of rhythmicity but, after withdrawal of the drug, the latter outlasted the former. The development of A-V dissociation coincided with the progressive speeding up of nodal impulse formation. Hence, it appears that the rapid sequence of nodal impulses preventing recovery of A-V junctional tissues was the actual factor responsible for the onset of the dissociation rather than a complete block of A-V conduction.
FIG. 1. The mode of onset and termination of a nonparoxysmal A-V nodal tachycardia, leading to retrograde activation of the atria (case 30).

12-1-52 ON DAILY MAINTENANCE DOSE OF 0.25 DIGOXIN ORALLY

12-4

12-5

12-15 NO DIGITALIS SINCE 12-5

FIG. 2. Digitalis-induced nonparoxysmal A-V nodal tachycardia, as a cause of A-V dissociation in atrial fibrillation (case 14).

8-23-52

8-25


In figure 3 (case 26) the record of 8-23 shows complete A-V dissociation except for the first and last beats, which represent sinus beats conducted with a normal P-R of 0.14 second. During the A-V dissociation the ventricles are activated by an accelerated A-V nodal pacemaker (rate 75). Both sinus and ectopic beats reveal typical earmarks of posterior wall infarction but differ slightly in contour; this is attributed to the use of preferential A-V pathway by the nodal impulses. The A-V dissociation commences when the rate of the irregular sinus pacemaker slows to 67; it continues while the sinus rate varies between 68 and 79 and disappears as soon as the sinus speeds up to 83.

The record of 8-25 shows a sinus bradycardia of 55, yet all beats are conducted with a normal P-R of 0.14 second. Evidently the nodal acceleration that caused the A-V dissociation on 8-23 must have subsided.

Involvement of A-V nodal function by recent
posterior wall infarction is a common event, usually manifested by a transient A-V block. In this instance only the property of impulse formation was affected while conductivity was entirely spared—as evident from the normal P-R interval of the conducted beats on both days. The failure of retrograde activation of the atria during the nodal tachycardia and the absence of ventricular captures during the ensuing A-V dissociation are the result of persistent physiologic A-V interference of sinus and nodal impulses operating at close rates.

In figure 4 (case 19), on 8-26, A-V dissociation is present because an accelerated A-V nodal pacemaker operates at a faster rate (130) than the simultaneously accelerated sinus pacemaker (125). The A-V dissociation is incomplete, since conductivity of the A-V node (in a forward direction) is unimpaired, as evident from the periodic recurrence of 3 or 4 successive ventricular captures (indicated by cutoff symbols). The last in this series of conducted sinus impulses has a P-R of 0.16 second; in the others, the P-R is prolonged to 0.34 or 0.36 second—a consequence of the normal state of relative refractoriness of A-V junctional tissues early in the nodal cycle. The R-R interval of these first captures is, contrary to the rule, prolonged instead of being shortened. This unusual finding compels the conclusion that: (a) part of the conduction delay of these captures took place in pathways below the site of the A-V nodal pacemaker which was prematurely discharged by the passing sinus impulse, and (b) the conduction time in this stretch of the A-V junction did not exceed the duration of the postponed nodal cycle.

On the following day (8-27), A-V dissociation is still present but the ventricular action is precisely regular—the A-V dissociation has become complete. Since on both days sinus and nodal rates are the same, and hence R-P relationships vary in a corresponding manner, the absence of ventricular captures on 8-27 must be ascribed to a new additional factor, namely prolongation of the refractory period of A-V junctional tissues impairing their conductivity.

That this was actually the case becomes evident in 2 subsequent tracings on 8-29 and 9-4, which reveal, following the disappearance of the nodal tachycardia, a slowly subsiding first degree A-V block (P-R 0.26 and 0.20 respectively).

This series of records, therefore, clearly shows the 2 manifestations of involvement of the A-V node by acute rheumatic fever and, in addition, that the 2 nodal functions, rhythmicity and conductivity, may be affected separately or together.

The diagram in figure 5B summarizes the clinical, laboratory, and electrocardiographic data of case 22 over a period of 16 days; the numbers in circles indicate the days on which the 3 electrocardiograms in figure 5A were obtained. The acute rheumatic process is evident from the temperature curve, the involvement of the joints, the development of an apical systolic murmur, the initial elevation of the leukocyte count and of the C-reactive protein (CRP), and the rising antistreptolysin titer (ASL). The broad shaded area represents the range of sinus arrhythmia, the black area the rate of a corresponding A-V nodal pacemaker, as noted in daily electrocardiograms. This correlation shows that at the height of the acute process the accelerated nodal rate permanently exceeded the range of the simultaneously accelerated sinus pacemaker with resulting persistent (incomplete) A-V dissociation (upper record in A). As rheumatic activity subsided, the rate of the 2 pacemakers dropped to similar average
values; consequently A-V dissociation was observed only intermittently when the sinus rate, temporarily, became slower than that of the still accelerated and less irregular nodal pacemaker (middle record in A). Eventually, after a week, with further clinical improvement of the patient, signs of nodal activity disappeared from the electrocardiograms. Presumably the A-V node had returned to its normal function as a subsidiary pacemaker with an inherent rate of less than 60 (lowest record of A).

The following details should be noted in the 3 electrocardiograms:

1. On 4-4, the repetitive recurrence of 2 successive ventricular captures after every third nodal beat causes ventricular group beating. Only the second of each pair of captures reveals the characteristic R-R shortening; in the first, R-R is, against the rule, prolonged—and this for reasons outlined in the description of figure 4, where a corresponding finding is illustrated.
2. On 4-11, A-V dissociation is transiently replaced by 5 successively conducted sinus impulses, since at this time the sinus became faster than the nodal pacemaker. However, the first of these conducted sinus impulses (third P wave) occurs after a pause that exceeds by 0.14 second the longest nodal interval in the record. The failure of a nodal beat to terminate this pause is attributed to concealed conduction\(^3\) of the second P wave. Obviously this sinus impulse penetrated to the site of the nodal pacemaker postponing its spontaneous discharge, but then failed to reach the ventricles.

3. In none of these 3 records (nor in any other of the patient's records) was there evidence of A-V block. Prolongation of the P-R of the captures on 4-4, as well as the failure of an attempted capture to be completed on 4-11, are attributable to a normal state of relative refractoriness of A-V junctional tissues early in the nodal cycle. Thus, in this instance of acute rheumatic fever, selective involvement of A-V nodal rhythmicity, without any impairment of conductivity is clearly demonstrated.

**RESULTS AND DISCUSSION**

*Electrocardiographic Features of Nonparoxysmal A-V Nodal Tachycardia*

Like any type of subatrial rhythm, nonparoxysmal nodal tachycardia may lead to retrograde activation of the atria provided the rate of discharge of the ectopic pacemaker exceeds the rate of the primary one and the retrograde conduction time through the A-V junction is short. This may be the case when the ectopic focus is located close to the atria, in the upper portion of the A-V node, but our material suggests that this is uncommon. Retrograde P waves were encountered in only 4 of the 30 cases (nos. 16, and 28-30 of table 1) and one of them is illustrated in figure 1 as an example of the mode of onset and termination of a nonparoxysmal nodal tachycardia. In this instance the transition of one rhythm into the other with interposition of atrial fusion beats superficially resembles a condition known as "wandering pacemaker." However, the latter is induced characteristically by slowing of the primary pacemaker whereas here the "shift in command" takes place as both sinus and nodal pacemaker speed up simultaneously, though to a different extent.

The usual consequence of a nonparoxysmal nodal tachycardia is 1 (or more) of the varieties (3) of A-V dissociation (figs. 2-5). The type of dissociation, whether persistent or intermittent on the one hand, and complete or incomplete on the other, will depend on the degree of enhancement of the nodal rate relative to the rate of the atria and on the state of refractoriness of the A-V junctional tissues.\(^9\)

Since in a nonparoxysmal nodal tachycardia the limits of acceleration (70-130) of nodal impulse formation coincide with the range of ordinary sinus rhythms, an approximation or equalization of the timing of impulses of the 2 pacemakers is a common occurrence. It may take place during normal sinus rates (fig. 4) as well as during a sinus tachycardia (fig. 4). If the sinus rate is regular the dissociation will persist throughout the duration of the ectopic rhythm; in the presence of a sinus arrhythmia it may become manifest in an intermittent form, that is only during the slower phases of the sinus action (figs. 3 and 5). If under any of these circumstances the ectopic rate closely approaches or actually equals that of the sinus pacemaker the dissociation will become complete (fig. 3); then the independence of atrial and ventricular beating can be accounted for entirely on the basis of normal states of refractoriness in the A-V junction or, in other words, by persistent simple A-V interference\(^9\) of the 2 impulses. If, on the other hand, the nodal acceleration exceeds the sinus rate, then incomplete or complete A-V dissociation may be the result. In the former, the occurrence of ventricular captures early in the nodal cycle and in a predictable manner may be considered evidence of normal A-V conductivity—and the physiologic handicap in the A-V junctional tissues to retrograde transmission of impulses\(^3\) may be invoked as the factor maintaining the dissociation (fig. 5). Should, however, under comparable conditions, ventricular captures fail to occur when expected, and the A-V dissociation be complete, the implication of a concomitant A-V block prolonging A-V refractoriness becomes inescapable. An excellent example of transition from one to the other of these 2 conditions is presented in figure 4.

Of course a nonparoxysmal nodal tachycardia can develop in the presence of atrial fibrillation\(^20\) or flutter, and this can also lead to A-V dissociation. In our material this occurred in
about one third of instances in which the ectopic rhythm was attributable to digitalis medication (table 1). One instance is exemplified by figure 2, others have been illustrated elsewhere. Similar cases have apparently been considered as instances of "complete A-V block." While it is true that a nonparoxysmal nodal tachycardia in combination with rapid atrial rhythms usually leads to completely independent atrial and ventricular action, this independence is per se no proof that A-V conductivity is totally blocked. Complete A-V dissociation under such circumstances can, in our opinion, result from: the inability of retrograde nodal impulses to activate atria that are in a state of fibrillation, and the contribution of these accelerated retrograde nodal impulses to the state of reactivity of the A-V junction, over and above the refractoriness induced by the rapid impulses of atrial fibrillation (or flutter). Sometimes preservation of A-V conductivity can actually be demonstrated in the form of ventricular captures effected by occasional atrial fibrillation impulses traversing the A-V junction.

The identification of ventricular captures in an A-V dissociation induced by a nonparoxysmal nodal tachycardia may require meticulous measurements. As a rule their occurrence can be presumed whenever single, or several successive, early beats are found in an otherwise regular ectopic ventricular action. Such "premature" conducted complexes may precisely resemble the dominant ectopic beats, or the 2 may differ in shape and QRS duration due to an aberrant intraventricular spread of the conducted or of the ectopic impulses. If the atria respond to sinus impulses and P waves can be recognized, the diagnosis of ventricular captures can be fortified by the demonstration of a typical inverse relationship between R-P and P-R intervals. Such data can then be used to estimate the duration of the absolute and relative refractory phases of the A-V junction in the individual case and to rule out the presence of A-V block or to determine its type and degree.

Exceptionally, however, conduction of sinus impulses in predominant A-V dissociation may be recognized on the basis of sudden lengthening, rather than shortening, of an R-R interval. This will be the case when a sinus impulse after entering the A-V junction penetrates to the site of the nodal pacemaker and, preceding the spontaneous nodal discharge, interrupts the nodal cycle, but is then retarded (figs. 4 and 5) or completely stopped (fig. 5) in its further spread to the ventricles. The latter event, representing an attempted but not completed capture, has been described previously; the former, which may be termed "delayed capture," has to our knowledge not been reported as yet.

**Clinical Significance of Nonparoxysmal A-V Nodal Tachycardia**

Whereas paroxysmal acceleration of nodal impulse formation occurs, as a rule, in individuals free from demonstrable cardiovascular abnormalities, this is the exception for the nonparoxysmal variety. Thus, only in 2 of our cases (nos. 28 and 29) was clinical cardiac pathology not detected. In more than half of the cases nonparoxysmal nodal tachycardia was encountered as a consequence of digitalis therapy for congestive heart failure; in the others it occurred either in the course of acute rheumatic fever or in the early stages of posterior wall infarction. This apparent predilection for these conditions is not surprising considering the very frequent occurrence of another manifestation of A-V nodal pathology, namely A-V block in all 3 of these states.

However, disorders of the 2 functions of the A-V node, transmission and formation of impulses, need not be associated. Our material shows both their combined as well as their independent development. The predominant feature in digitalis-induced nonparoxysmal nodal tachycardia was its association with A-V block. Contrariwise, the majority of cases with acute rheumatic fever or posterior wall infarction developed A-V dissociation due to nonparoxysmal nodal tachycardia without any evidence of impaired A-V conductivity, even in serial records. Such observations contradict a recent statement that "A-V dissociation and nodal rhythm in acute rheumatic fever have never been described without preceding or following periods of fixed P-R prolongation."
Hence, the interesting hypothetical concept concerning the mechanism of A-V arrhythmias developed on this basis seems to have no validity from the clinical standpoint.

The clinical importance of a nonparoxysmal nodal tachycardia varies depending on circumstances of its development. In cases of recent posterior wall infarction it lasted for only a few days and, although 1 patient (no. 27) died after the onset of the nodal acceleration, it could not be considered an unfavorable sign of the condition of the patient at the time or of the final outcome of the attack. Indeed, the moderate acceleration of the ventricular rate with the onset of the ectopic rhythm may sometimes relieve symptoms caused by marked bradycardia, so common in the early stages of posterior wall infarction, which results from a pronounced slowing of the sinus rate or from a complicating advanced A-V block.

In acute rheumatic fever, on the other hand, a nonparoxysmal nodal tachycardia is a highly significant event, since it may represent the only electrocardiographic evidence of myocardial involvement, particularly in cases that do not develop an A-V block at any time. While it is true that nonparoxysmal nodal tachycardia is far less frequent than a simple P-R prolongation, it occurs more often than higher degrees of A-V block. Like the P-R prolongation, nonparoxysmal nodal tachycardia is closely linked to other signs of rheumatic activity and may be easily missed without daily electrocardiograms (fig. 5). It may occur only at the peak of activity (fig. 4) or it may persist into and subside gradually during the stage of recovery (fig. 5). Contrary to P-R prolongation nonparoxysmal nodal tachycardia is a transient phenomenon that does not outlast the clinical and laboratory evidence of rheumatic activity.

Perhaps most important from the clinical standpoint is the recognition of a nonparoxysmal nodal tachycardia induced by digitalis medication because of its frequency, because of its bearing on the therapeutic results, and because it is one of the manifestations of digitalis excess. Some of the problems involved are illustrated in fig. 3, which reveals the gradual acceleration of nodal activity—and ventricular rate—in a patient kept on a small maintenance dose of digitalis, as well as its deceleration after discontinuation of the medication. If, as in this instance, nonparoxysmal nodal tachycardia develops during atrial fibrillation and causes complete A-V dissociation, the ensuing regularization of the ventricular action can be readily mistaken at the bedside for conversion to sinus rhythm unless it is clarified by an electrocardiogram. Should the ectopic rhythm remain unrecognized and medication be continued, the result may be further "paradoxical" acceleration of the ventricular rate, a development that not only defeats the purposes of digitalis therapy but may lead from the stage of "digitalis excess" into one of actual digitalis intoxication.

Conclusions and Summary

Enhanced rhythmicity of the A-V node is manifested in clinical electrocardiography in a paroxysmal and nonparoxysmal form. These 2 types of rapid ectopic rhythms differ not only in the mode of onset and of termination but also in the degree of acceleration and especially in their clinical significance.

Although a nonparoxysmal A-V nodal tachycardia may cause retrograde activation of the atria it usually leads to A-V dissociation. Depending on the type of atrial dissociation, the relationship between atrial and ventricular rates and the state of refractoriness of the A-V junctional tissues, the A-V dissociation may be persistent or intermittent, and complete or incomplete. Incomplete dissociation becomes evident by sudden shortening, or lengthening, of some of the otherwise regular ventricular cycles.

Nonparoxysmal A-V nodal tachycardia is found predominantly in acute pathologic conditions known to cause moderate or advanced degrees of A-V block, in particular digitalis excess, acute rheumatic fever, and recent posterior wall infarction. It would therefore appear that both are manifestations of pathologic processes preferentially involving the A-V node. Sometimes only the property of impulse conduction is affected, sometimes that of impulse formation, and sometimes both these
functions are involved simultaneously, or successively.

Nonparoxysmal A-V nodal tachycardia represents an electrocardiographic entity with multiple facets and is an important factor to be considered in the assessment of digitalis excess and of rheumatic activity.

**SUMMARIO IN INTERLINGUA**

Rhythmicitate accelerate del nodo atrio-ventricular es manifeste in le electrocardiographia in un forma paroxysmic e in un forma nonparoxysmic. Iste 2 typos de rapide rhytimo ectopic differen non solmente in lor modo de declaration e de termination sed etiam in le grado del acceleration e specialmente in lor signification clinic.

Ben que le nonparoxysmic tachycardia a nodo atrio-ventricular pote effectuar un activation retrograde del atrios, illo resulta usualmente in dissociation atrio-ventricular. Secundo le typo de activitate atrial, secundo le relation inter le frequentias atrial e ventricular, e secundo le stato de refractorietate del tessuto de junction atrio-ventricular, le dissociation atrio-ventricular pote esser persistente o intermittente, complete o incomplete. Incomplete dissociation deveni evidente per un substante accurtation o prolongation de certes del alteramente regular cyclos ventricular.

Nonparoxysmic tachycardia a nodo atrio-ventricular se trova predominante in acute conditiones pathologic que es cognoscitamente capace a causar moderate o avantitate grados de bloco atrio-ventricular, specialmente excessos de digitalis, acute febre rheumatic, e recente infarcimentos del pariete posterior. Per consequente il pare que ambes es manifestationes de processos pathologic que affice per preferentia le nodo atrio-ventricular. A vices, solmente le proprietate del conduction de impulsos es affiche, a vices le proprietate del formation de impulsos, e a vices ambe iste proprietates es affiche—simultaneae—o successivemente.

Nonparoxysmic tachycardia a nodo atrio-ventricular representa un entitate electrocardiographic con multiple facetas e es un factor importante que non debe esser negligeite in le evaluation de excessos de digitalis e de activitate rheumatic.

**REFERENCES**

2. **LEWIS, T.:** The Mechanism and Graphic Registration of the Heart Beat. Ed. 3. London, Shaw and Sons, 1925.
11. **WHITE, P. D.:** Ventricular escape with observations on cases showing a ventricular rate greater than that of the auricles. Arch. Int. Med. 18: 244, 1916.
15. **LEA, E.:** Complete heart block with higher ventricular than auricular rate. Lancet 1: 1289, 1915.
20. **LOWN, B., AND LEVINE, S.:** Current Concepts


The current interpretations of endocardial fibroelastosis as a genetically determined congenital lesion or a consequence of hypoxemia are examined in the light of the pathologic physiology of cardiac structure and by application of Laplace's law of hydrostatics. Since it has been repeatedly demonstrated that mechanical forces may exert an influence on the genesis and development of the elastica, especially if those forces are rhythmic and fluctuating, it would seem probable that the great tensions that are cyclically accentuated in the endocardium of the dilated heart would be good reason for the genesis of endocardial fibroelastosis. Since the hypothesis here advanced is based upon mechanical considerations, cardiomegaly with endocardial fibroelastosis may develop in utero, given the appropriate constellation of force relationships. It is significant, however, that unequivocal examples of congenital idiopathic cardiomegaly with endocardial fibroelastosis are extremely rare. Anoxemia cannot be a significant factor except terminally, since the almost complete and uniform absence of recognizable ischemic myocardial injury in these huge hearts excluded it as an etiologic factor.

The material studied consisted of examples of idiopathic cardiomegaly with endocardial fibroelastosis, anomalous left coronary artery arising from the pulmonary artery, miniature left ventricle resulting presumably from premature closure or stenosis of the foramen ovale, and miniature right ventricle. The presence of endocardial fibroelastosis can be explained on a mechanical basis in each instance; and furthermore, the theory appears to fit the peculiarities of cardiac structure and function found in these anomalies.

Maxwell
Nonparoxysmal A-V Nodal Tachycardia
ALFRED PICK and PEDRO DOMÍNGUEZ

Circulation. 1957;16:1022-1032
doi: 10.1161/01.CIR.16.6.1022
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1957 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/16/6/1022

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/