**Case Reports**

**Double A-V Nodal Rhythm**

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The A-V node may function as pacemaker by producing either relatively slow nodal escape rhythms or more rapid non-paroxysmal or paroxysmal tachycardias.1,2) Although A-V nodal escape rhythm and nodal tachycardia are frequently encountered in clinical medicine, only a few cases2,10) of double nodal rhythm (or tachycardia) have been reported in the literature.

Because of the rarity and clinical importance of this arrhythmia, 5 cases of double A-V nodal rhythm have been selected from the files of the Barnes Hospital Heart Station for presentation in this paper.

**Case Reports**

*Case 1:* F.W., a 69 year old white male was admitted to Barnes Hospital because of psychosis and mild congestive heart failure due to arteriosclerotic heart disease. He developed clinical signs of digitalis intoxication and double A-V nodal rhythm in the electrocardiogram during hospitalization. The patient improved after discontinuation of digitalis therapy for 10 days and sinus rhythm was restored. In the electrocardiogram reproduced in Fig. 1, there can be seen predominantly double A-V nodal rhythm and areas of sinus rhythm with intermittent A-V dissociation. One A-V nodal pacemaker (classified as group A) activates the atria in retrograde fashion with the rate of 52 per min. (P-P interval is 1.16 sec.) These retrograde P waves are inverted in leads II, III and aVF and upright in lead aVR. The other A-V nodal pacemaker (classified as group B) activates the ventricles producing the QRS complexes with the rate of 65 per min. (R-R interval 0.92 sec.). The ventricular deflections originating from the A-V node are slightly aberrantly conducted11,12) in comparison with the normal sinus beats, but the QRS interval of the aberrant beats is less than 0.10 sec. This phenomenon is clearly seen in lead V3. There are areas of sinus rhythm with a rate of 63 per min. (R-R interval 0.98 sec.) in leads II (B), aVR, aVF, and V3 and during the sinus rhythm there is intermittent A-V dissociation. It is interesting to note that there are occasional atrial fusion beats (labeled FB) in leads aVR and aVF which are intermediate in form between sinus P waves and retrograde P wave originating from the Group A nodal pacemaker. The tenth QRS complex in lead V3 is probably a conducted A-V nodal beat produced by the group A nodal pacemaker which exhibits some

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delay in antegrade conduction but no evidence of aberrant conduction. There is one ventricular premature beat in lead III (A).

Case 2: W.C., a 59 year old white male was admitted to Barnes Hospital because of pneumonia and mild congestive heart failure. The patient developed double A-V nodal tachycardia and clinical signs of digitalis excess. He improved after discontinuation of digitalis therapy for 2 weeks, and his rhythm returned to normal sinus rhythm. His tracing (Fig. 2) shows double A-V nodal rhythm without any sinus activity. The P waves are inverted in lead II and upright in lead aVR indicating that they are retrograde P waves. There is no relationship between the inverted P waves and the QRS complexes. The atria are activated by an A-V nodal pacemaker (classified as group A) in retrograde fashion, and the ventricles are activated by a second pacemaker (group B) in the A-V node. The rate of the group A pacemaker is faster (112 per min.) than the group B (92 per min.). Both A-V nodal pacemakers are beating at a much faster rate than the inherent automaticity of impulse formation in the A-V node, which results in double non-paroxysmal nodal tachycardias.13, 14

Case 3: B.F., a 73 year old white male was admitted to Barnes Hospital because of bronchitis, pulmonary emphysema and congestive heart failure. During hospitaliza-
tion he developed a double A-V nodal rhythm (Fig. 3) which was thought to be due to digitalis excess. The A-V nodal rhythm was converted to sinus rhythm after discontinuation of digitalis therapy for 2 weeks. In Fig. 3, the P waves are inverted in lead III and upright in lead aVR and appear at a rate of 40 per min. These P waves are conducted in a retrograde fashion from the A-V node (group A). The ventricular rate is 78 per min. and the QRS deflections originate from a second A-V nodal pacemaker (group B). Thus, the atria and ventricles are activated independently by 2 different pacemakers within the A-V junctional tissues. The rate of the group B pacemaker is faster than the inherent automaticity of impulse formation in the A-V node, so that a non-paroxysmal A-V nodal tachycardia exists.

**Case 4:** O.J., a 3 year old girl was admitted to Barnes Hospital for repair of atrial and ventricular septal defects and pulmonic stenosis, but surgical repair was not successful. An electrocardiogram (Fig. 4) recorded during surgery showed a double nodal rhythm. The atria are activated in retrograde fashion by an A-V nodal pacemaker (group A) beating at a rate of 123 per min. The ventricles are activated by another pacemaker in the A-V node (group B) with a rate of 58 per min. The group A rhythm can be considered a non-paroxysmal nodal tachycardia,\(^{13},^{14}\)
although the inherent rate of impulse formation within the A-V node in the children is faster than adult.

Case 5: H.W., a 67 year old white male was admitted to Veterans Administration Hospital, Washington University Service, because of congestive heart failure due to arteriosclerotic heart disease. He developed clinical signs of digitalis intoxication and expired subsequently. Two electrocardiograms taken when the patient was intoxicated by digitalis are shown on Fig. 5 and Fig. 6. Fig. 5 shows sinus rhythm
with complete A-V block and areas of double A-V nodal rhythm. There are 2 different P waves; namely, upright P waves of sinus node origin and inverted retrograde P waves of A-V node (group A) origin. None of the sinus P waves are conducted to the ventricles indicating that there is complete A-V block. The ventricles are activated by a second pacemaker in the A-V node (group B). It is interesting to note that despite the antegrade A-V block of the sinus impulses retrograde conduction of the B nodal beats occurs twice to produce atrial capture beats (marked AC). There is one atrial fusion between an antegrade sinus P wave and a retrograde P wave of the group A nodal rhythm. The rate of the sinus rhythm is 67 per min.; that of the group A nodal rhythm, 80 per min.; and that of the group B nodal rhythm, 52 per min. The group A nodal rhythm is a relatively slow non-paroxysmal nodal tachycardia. The electrocardiogram appearing in Fig. 6 was taken a few days later and shows a double A-V nodal rhythm with occasional nodal premature beats. The P waves are inverted in leads II and III and upright in lead aV_R and occur at a rate of 62 per min. (group A). The ventricles are activated by another A-V nodal pacemaker (group B) and appear at a rate of 77 per min. It is interesting to note that there are frequent nodal premature beats in the group B nodal rhythm. An alternative explanation for these premature beats would be that they are reciprocal beats as shown in the diagram (No. 2), but it is impossible to establish which is the actual mechanism of production of these premature beats in this record.
DISCUSSION AND COMMENT

In all of the electrocardiograms of the 5 patients reported in this paper, there were inverted P waves in leads II, III and aVF and upright P waves in lead aVR. While inverted P waves have been observed both clinically,¹,²,¹⁵ and experimentally¹⁶ in ectopic rhythms originating low in the atria or in the presence of aberrant intra-atrial conduction,¹⁵,¹⁷ they are frequently produced by retrograde conduction of the A-V nodal impulses into the atria. The length of the P-R interval of such beats has been used to differentiate between a low atrial and an A-V nodal origin of such inverted P waves but in the presence of A-V dissociation or antegrade block such a differentiation becomes impossible. However, one may assume that the ectopic pacemaker is most likely located in the upper portion of the A-V node since the A-V node is the natural pacemaker secondary to the sinus node in its degree of rhythmicity. In double nodal rhythm the second ectopic pacemaker (group B) activating the ventricles probably is located in the lower part of the A-V node since none of these impulses conducted to the atria except for the 2 atrial capture beats in Fig. 5. Moreover, the QRS interval of the ectopic ventricular beats is less than 0.10 sec., although one cannot exclude entirely the possibility that the ectopic focus is located in the ventricular septum near the A-V node. Thus, it would seem justifiable to assume that the group A pacemaker driving the atria is located in the upper part of A-V node and the group B pacemaker producing the ventricular rhythm is located in the lower part of the A-V node. In A-V nodal rhythms, A-V dissociation commonly occurs. Almost always the atria are controlled either by the sinus node or by an ectopic rhythm in the atria (as in atrial fibrillation, flutter and tachycardia) and the ventricles are controlled by the A-V node. Whenever the primary pacemaker fails to produce impulses (sinus arrest) or fails to conduct through the S-A or A-V junctional tissues due to S-A block or A-V block, A-V nodal rhythm occurs as an escape mechanism, although rarely idioventricular rhythm may occur. The upper A-V nodal rhythm (group A) in Fig. 1, 3 and probably Fig. 6 and the lower A-V nodal rhythm (group B) in Fig. 4 and 5 (see the Table I) most likely represent A-V nodal escape rhythms in view of their slow rates. On the other hand, the faster rate of the group A nodal rhythms in Figs. 2, 4, and 5 and the group B rhythms in Figs. 1, 2, 3, and 6 represent non-paroxysmal nodal tachycardias with rates varying between 65 and 125 per min. If the A-V node is acting as a pacemaker resulting from acceleration of impulse formation beyond its inherent automaticity, A-V nodal tachycardia occurs as a paroxysmal or non-paroxysmal form.¹³,¹⁴ The rate varies between 150 and 200 per min. in paroxysmal tachycardia and 70 and 130
Table I. An Analysis of Clinical Features of 5 Cases with Double A-V Nodal Rhythm in Relation to Their Electrocardiographic Findings

<table>
<thead>
<tr>
<th>Case No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
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<tbody>
<tr>
<td>Age</td>
<td>69</td>
<td>59</td>
<td>73</td>
<td>3</td>
<td>67</td>
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<tr>
<td>Sex</td>
<td>M</td>
<td>M</td>
<td>M</td>
<td>F</td>
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<tr>
<td>Cardiomegaly</td>
<td>LVH</td>
<td>-</td>
<td>LVH</td>
<td>RVH, LAE, RAE</td>
<td>LVH</td>
</tr>
<tr>
<td>Probable Cause of Nodal Rhythm</td>
<td>Digitalis Intoxication</td>
<td>Digitalis Intoxication</td>
<td>Digitalis Intoxication</td>
<td>IASD, IVSD</td>
<td>Digitalis Intoxication</td>
</tr>
<tr>
<td>Digitalis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Sinus Mechanism and Rate</td>
<td>+ (63)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Fig. 5</td>
</tr>
<tr>
<td>Rate of Group A Nodal Rhythm</td>
<td>52</td>
<td>112</td>
<td>40</td>
<td>123</td>
<td>80</td>
</tr>
<tr>
<td>Rate of Group B Nodal Rhythm</td>
<td>65</td>
<td>92</td>
<td>78</td>
<td>58</td>
<td>52</td>
</tr>
<tr>
<td>Atrial Fusion Beat</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Other E.K.G. Abnormality</td>
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<td>DE</td>
<td>Old Ant. MI, DE</td>
<td>-</td>
<td>ACB, DE</td>
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<td>ASHD</td>
<td>MI, ASHD</td>
<td>IASD, IVSD</td>
<td>ASHD</td>
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<tr>
<td>Associated Disease</td>
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<td>Pneumonia</td>
<td>Emphysema Diabetes M.</td>
<td>-</td>
<td>-</td>
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<tr>
<td>Result</td>
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<td>Improved</td>
<td>Improved</td>
<td>Expired</td>
<td>Expired</td>
</tr>
</tbody>
</table>

+: Present  
-: Absent  
M: Male  
F: Female  
VPC: Ventricular premature contraction  
DE: Digitalis effect  
ASHD: Arteriosclerotic heart disease  
LVH: Left ventricular hypertrophy  
Ant. MI: Anterior myocardial infarction  
Diabetes M.: Diabetes mellitus  
RVH: Right ventricular hypertrophy  
RAE: Right atrial enlargement  
LAE: Left atrial enlargement  
IASD: Intra-atrial septal defect  
IVSD: Intra-ventricular septal defect  
ACB: Atrial capture beat  
RB: Reciprocal beat  
The rate indicates the number of beats per sec.

per min. in non-paroxysmal tachycardia. The presence of non-paroxysmal nodal tachycardia almost always indicates organic heart disease or
digitalis intoxication, whereas paroxysmal nodal tachycardia occurs in healthy hearts.\(^13\),\(^14\) In A-V nodal tachycardia, in general, the ventricular rate is equal to or faster than the atrial rate producing A-V dissociation, except when A-V dissociation occurs in the presence of atrial flutter and fibrillation. If there are capture beats (almost always ventricular capture beats but rarely atrial capture beats), then the A-V dissociation is termed incomplete. If there are no capture beats, then complete A-V dissociation exists. It is very interesting to note that 2 atrial capture beats occur in Fig. 5 (lead II-b, c) so that temporarily the atria and ventricles are activated from the same focus located in the lower part of the A-V node (group B) despite the fact that the lower A-V nodal rhythm is much slower than the upper A-V nodal rhythm. On the whole, this phenomenon is a rare occurrence since the upper pacemaker always produces impulses more rapidly than the lower pacemaker so that the atria are refractory whenever impulses from the lower pacemaker attempt to reach the atria. If, however, the atria are receptive to the lower nodal impulses, either because they are non-refractory as the result of beating at a slow rate or because they are in the supernormal phase, then atrial capture beats may occur. In a rare form of A-V dissociation, the atria and ventricles are activated by different ectopic foci located in the A-V node with the result that double A-V nodal rhythm occurs. Occasionally the atria may be activated by the sinus node intermittently in the presence of double nodal rhythms (Fig. 5), and atrial fusion beats (between antegrade sinus impulses and retrograde nodal impulses) may occur (Fig. 1). Although the majority of reported cases of double A-V nodal rhythms showed a more rapid lower A-V nodal pacemaker than upper pacemaker, only one half of our cases showed this phenomenon. It is interesting to note that the rate relationship between the upper and lower A-V nodal rhythm in case 5 is completely reversed on different occasions (Figs. 5 and 6). The presence of nodal premature beats in double nodal rhythm (Fig. 6) is an unusual phenomenon but has been reported also by others.\(^2\),\(^10\),\(^18\) In Fig. 6, atrial activity was never disturbed regardless of the presence of nodal premature beats. In 4 out of 5 cases of our series, the double A-V nodal rhythm was thought to be due to digitalis intoxication during digitalis therapy for congestive heart failure and this has also been the experience of others.\(^4\),\(^5\),\(^7\),\(^8\) In one case, this arrhythmia was most likely associated with congenital heart disease; a similar case has also been reported.\(^4\)

**Summary**

Five cases of double A-V nodal rhythm have been discussed in detail and the relevant literature has been reviewed. Double A-V nodal rhythm was
thought to be due to digitalis intoxication during digitalis therapy for congestive failure in 4 cases.

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REFERENCES