Case Report

Various Types of Second Degree A-V Node-Ventricular Block (Block in Block) Observed in a Case of Complete A-V Block with Atrial Fibrillation and Bilateral Bundle Branch Block

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Block can occur in any part of the cardiac conduction system from the sinus node to the Purkinje fibers. Conventionally, block of antegrade conduction from the primary pacemaker has been classified into S-A and A-V block by its location. These blocks have been classified into three degrees. First degree block represents conduction delay, second degree block an intermittent discontinuation of impulse propagation and third degree block a complete dissociation between a higher and a lower pacemaker. Both entrance and exit block are forms of unidirectional block in parasystoles and other extrasystoles. In entrance block, a parasystolic center is protected (protection block) by a unidirectional block from the transmission of impulses originating from the basic rhythm. In exit block, outward transmission of the parasystolic impulses is prevented. The lower pacemaker may arise in the A-V node, bundle of His or bundle branches. The block between this lower pacemaker and the His-Purkinje system has been referred to as "block in block" (Volhard). This block in block can also be of first, second and third degree.

In complete A-V block with atrial fibrillation, an impulse originating in the A-V node (mid-nodal region) propagates through the common bundle and bundle branches (node-His region and His-Purkinje system) to the ventricles, just as an impulse originating in the sinus node propagates through the sino-atrial junction, intra-atrial pathways and atrio-nodal junction to the A-V node. Therefore, the P wave in regular sinus rhythm corresponds to the QRS complex in complete A-V block with atrial fibrillation. Since excitations in the A-V node are not recorded electrocardiographically, first degree block in A-V node-ventricular (N-V) pathway can not be demonstrated by the conventional electrocardiograms. Second degree N-V block can be recognized by an intermittent duplication or multiplication of the RR interval (type II) or by a progressive shortening of the RR interval followed by a prompt prolongation of the RR interval (type I). Third degree N-V block may manifest itself as ventricular asystole or a ventricular escape rhythm according to a failure or success of ventricular escape. Concealed retrograde discharge of the A-V nodal pacemaker by ventricular premature beats can also be recognized by the resumption of the RR interval.

In this paper, a case is reported in which not only type II but also type I, second degree N-V block were observed with other arrhythmias during transient complete A-V block with atrial fibrillation and bilateral bundle branch block.

Case Report

I. Clinical Course

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S. T., a 78-year-old female with rheumatoid arthritis. Varying doses of Prednisolone and Phenylbutazone had been given orally for joint pain, and 75 mg of Dipyridamole daily for coronary sclerosis. The hospital course was uneventful until she had mental troubles with her newly-appointed nurse on March 30, 1969. From then she complained of insomnia, nausea, general malaise and precordial discomfort. She began to improve after the newly-appointed, ill-natured nurse was fired on April 17, 1969. From then on she was attended by her own daughter. During the period between March 24 and April 21, the patient developed various arrhythmias described below.

II. Serial Electrocardiographic Changes

On March 24, the ECG showed regular sinus rhythm with complete RBBB which persisted throughout the observation period. On April 3, when she first experienced precordial discomfort, the ECG showed first degree A-V block, intermittent second degree A-V block type II and transient third degree A-V block as shown in Fig. 1.

Figure 1 can be interpreted as follows: The PR interval in the upper strip averages 0.36 sec. In the lower strip, the 3rd, 4th, 5th and 6th (buried in the QRS complex) P waves are blocked, and 2 nodal escape beats occur as indicated in the diagram. The combination of the PR prolongation and complete RBBB in the upper strip indicates the presence of third degree block in the right bundle branch and first degree block in the left bundle branch, provided that the conduction in the node-His region is intact. Namely, this combination corresponds to the second stage of bilateral bundle branch block classified by Lopez. The combination of RBBB and transient third degree A-V block in the lower strip likewise indicates the presence of third degree block in the left bundle branch, which corresponds to the third stage of bilateral bundle branch block of Lopez. On April 4, A-V dissociation occurred with occasional concealed conduction as shown in Fig. 2.

Figure 2 can be interpreted as follows: The P wave and the QRS complex are completely dissociated, and the P wave marches through the QRS complexes. Concealed antegrade conduction of the sinus impulse discharges the A-V nodal pacemaker, suppressing the next impulse formation in it. Time relation between the concealed conduction and the dropped beat in the diagram is depicted arbitrarily. On April 5, A-V dissociation changed into atrial fibrillation with complete A-V block. The A-V nodal escape rhythm was occasionally interrupted by ventricular premature beats followed by a pause which was not fully compensatory.

Fig. 1. First and third degree A-V block with nodal escape rhythm (bilateral bundle branch block)

First degree A-V block in the upper strip and transient third degree A-V block in the lower strip with a QRS complex of complete RBBB, which indicates the presence of third degree block in the right bundle branch and first and third degree block in the left bundle branch. For details see text. Throughout this paper, A represents the atria, AV the A-V nodal pathway and V the ventricles.

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as shown in Fig. 3.

Figure 3 can be interpreted as follows: An interval between the 2 basic nodal escape beats is ca. 1.70 sec. as observed in the lower strip. However, an interval between the ventricular premature beat and the following basic nodal escape beat is also 1.68 to 1.70 sec. This indicates that the A-V nodal pacemaker is discharged by concealed retrograde conduction of the ventricular premature impulse, resetting the A-V nodal cycle, as illustrated in the diagram. Therefore, the ventricular premature beat is not fully compensatory, being followed by a coupling interval and a basic A-V nodal cycle. The 3rd ventricular premature beat in the lower strip is interpolated as a result of a failure of the concealed retrograde conduction, as indicated in the diagram. On April 7, the ECG still showed atrial fibrillation, complete

A-V block, A-V nodal escape rhythm and intermittent ventricular bigeminy. On that day, the ventricular premature beat was followed by a pause which was fully compensatory, as shown in Fig. 4.

Figure 4 can be interpreted as follows: In the upper strip, the ventricular premature beat is followed by a pause which is fully compensatory. This indicates that the A-V nodal pacemaker is protected from retrograde discharge by the ventricular premature beat, and that propagation of the next A-V nodal impulse is blocked by refractoriness in the node-His region produced either by the ventricular premature beat or by spontaneous prolongation of the absolute refractory period, as observed in the lower strip in the form of 2:1 N-V block. The coupling interval of the ventricular premature beat is constant between 0.50 to 0.52 sec. This

Temporal relation between concealed conduction and a dropped beat is depicted arbitrarily. The P wave marches through the QRS complex.

Basic A-V nodal QRS complexes of a RBBB pattern and ventricular premature beats of a LBBB pattern with a fixed coupling interval. The ventricular premature beats are followed by a pause which is not fully compensatory. For details see text. EF represents the ectopic focus in the ventricle. All time intervals in the diagram are expressed in a hundredth of a second.
indicates the presence of re-entry of the basic A-V nodal impulse into the ectopic focus in the ventricle. The fact that all the basic QRS complexes show a complete RBBB pattern and all

Fig. 4. Ventricular bigeminy in complete A-V block with atrial fibrillation.
II. Ventricular premature beats with a fully compensatory pause.

In the upper strip, the ventricular premature beats are followed by a pause which is fully compensatory. In the lower strip, 1:1 N-V conduction is followed by 2:1 type II second degree N-V block. For details see text. EF represents the ectopic focus in the ventricle.

1) Basic A-V Nodal Beat of a RBBB Pattern
2) Penetration of the Basic A-V Nodal Impulse into the Ectopic Focus
3) Ventricular Premature Beat of a LBBB Pattern

Fig. 5. Basic A-V nodal rhythm and ventricular premature beats by re-entry

The impulse of the basic A-V nodal rhythm is able to propagate to an excitable area through the left bundle branch, producing a complete RBBB pattern. Meanwhile, the localized area in the right bundle branch below the block which has been refractory becomes excitable again. At this point, an impulse of the basic A-V nodal rhythm reaches this previously refractory area and is able to penetrate it. Then, the ectopic focus in the previously depressed area undergoes depolarization, and the area initially excited by the basic A-V nodal impulse becomes once again excitable. Thus, the excitation impulse in the ectopic focus once again propagates to the ventricles. As a result, an extrasystole appears prematurely and has a constant coupling interval.
the ventricular premature beats have a complete atypical LBBB pattern indicates that an impulse of a ventricular premature beat originates in the right bundle branch below the blocked area, as indicates in Fig. 5. Therefore, the combination of the basic A-V nodal QRS complex of a RBBB pattern and the ventricular premature beat of an atypical LBBB pattern (Fig. 6) also indicates the presence of bilateral bundle branch block. The ECG on April 9 was similar to that on April 7. On April 11, the ECG showed atrial fibrillation, multiple second degree N-V block type II, second degree N-V block type I and ventricular asystole of varying durations as shown in Figs. 7 and 8.

Figure 7 can be interpreted as follows: In the first strip (lead II), there are periods of ventricular asystole of varying durations. It may be due to A-V nodal arrest. However, as indicated in the diagram, it may be due to 2 : 1, 3 : 1 and 4 : 1 second degree N-V block type II. The block may be present in the node-His region or in the left bundle branch. This finding corresponds to second degree S-A block type II. Also in the 2nd strip (lead III), there are periods of ventricular standstill of varying duration. In complete A-V block with atrial fibrillation, ventricular standstill may result from a marked prolongation of stimulus propagation from the A-V node to the ventricular muscle (first degree N-V block), blockage of all A-V nodal impulses to the ventricles (third degree N-V block) or from a failure of impulse generation in the A-V nodal pacemaker (A-V nodal arrest). Advanced, multiple second degree N-V block such as 4 : 1 to 6 : 1 N-V block type II which is observed in the 2nd strip (lead III) may also be responsible for ventricular asystole. In the 3rd and 4th strips (leads aVR and aVL), there are periods of 2 : 1 second degree N-V block. A type II, second degree N-V block may be due to an intermittent prolongation of the absolute refractory period of the node-His or the His-Purkinje system, by which an antegrade conduction of the A-V nodal impulse is blocked for one beat. Or the cause of advanced second degree N-V block type II may be due to a rapidly succeeding stimulation of the vagus, especially of the left, as indicated by Balsano et al. A failure of ventricular pacemaker to escape may be due to a repetitive concealed discharge of the node-His region by the A-V nodal impulses or an increase in the local depression of conductivity in the vicinity of an ectopic ventricular focus (failure of reentry).

Figure 8 can be interpreted as follows: In the upper strip, there is a 2 : 1 second degree N-V block followed by a progressive shortening of the RR interval (the Wenckebach period). At the end of the upper strip, there is even a progressive prolongation of the RR interval (the counter-Wenckebach period). This Wenckebach period corresponds to the Wenckebach period in S-A block. The presence of the counter-Wenckebach period in A-V conduction has been indicated by the present author in a case of bilateral bundle branch block. In that case, the PR interval was progressively prolonged at first and then progressively shortened without second degree A-V block. In contrast to the progressive RR shortening in the Wenckebach period in N-V conduction, the RR interval was progressively prolonged in the counter-Wenckebach period in N-V conduction. This may be due to a phasic alteration.

![Figure 6](image_url)

**Fig. 6.** Coupling of the basic A-V nodal beat of a RBBB pattern with a ventricular premature beat of an atypical LBBB pattern.

The basic A-V nodal QRS complex of a RBBB pattern is coupled with a ventricular premature beat of an atypical LBBB pattern.

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Fig. 7. Multiple second degree N.V. block type II and ventricular standstill. Advanced multiple N.V. block type II in the upper 2 strips and 2:1 N.V. block in the lower 2 strips. For details see text.
A progressive decrease in the RR interval is succeeded by a progressive increase in the upper strip and terminated by type II, second degree N-V block in the middle and lower strips. For details see text. N represents the A-V nodal pacemaker, NH the node-His region and V the ventricles.
in the tension of the vagus of unknown etiology. In the middle and lower strips, there is a progressive decrease in the RR interval, followed by a period of nearly constant RR interval (first degree N-V block) and then by a prompt increase in the RR interval, nearly twice as long as the preceding RR interval (2:1 second degree N-V block). This phenomenon repeated itself in a longer tracing. As indicated in the diagram, the earlier portion of this tracing resembles second degree S-A block type I, the middle portion first degree S-A block and the last portion 2:1 second degree S-A block type II. The Wenckebach period which is followed by a period of constant 1:1 conduction and terminated by second degree block type II can be referred to as second degree block type III. These conduction disturbances may be present in the node-His region or the left bundle branch because the right bundle branch is completely blocked. According to Langendorf and Pick, a type I conduction disturbance in one bundle branch cannot become manifest as long as conduction through the other bundle branch is possible and conduction through the latter is faster than the slowest conduction through the former. With complete interruption of one bundle branch and unimpaired conduction above the bifurcation of the common bundle, Wenckebach periods of conduction in the other bundle branch will become manifest. The ECG on April 12 was almost the same as that on the preceding day. On April 15 and 16, the ECG showed atrial fibrillation, complete A-V block, A-V nodal escape rhythm and ventricular bigeminy as observed on April 5 and 7. On April 17, atrial fibrillation disappeared and sinus rhythm was resumed. As shown in Fig. 9, the sinus rhythm was associated with first degree A-V block and occasionally interrupted by a blocked sinus or a blocked atrial premature beat. On April 18, sinus rhythm with advanced first degree A-V block as observed on April 3 was intermittently interrupted by second degree S-A block type II and by a blocked atrial premature beat. On April 21, the ECG returned to a regular sinus rhythm with complete RBBB as observed on March 24.

**DISCUSSION**

According to Romhilt et al., the sinus node artery was found to arise from the right coronary artery in 60.8 per cent and from the left

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*Fig. 9. Block of sinus premature beats at the S-A and A-V junctions*

A sinus impulse is blocked at the sino-atrial junction in the lower strip and at the atrio-nodal or atrio-ventricular junction in the upper strip.
circumflex artery in 37.6 per cent. The blood supply to the A-V node is from the right coronary artery in 84.7 per cent and from the left circumflex artery in 15.3 per cent. Although the presence of coronary arteriosclerosis cannot be ruled out in this patient, it seems less probable that structural damage due to coronary artery occlusion has been of major importance in or direct cause of the pathogenesis of N-V block observed because there was neither clinical nor laboratory signs of myocardial infarction. Electrocardiographically, it is impossible to distinguish between A-V node inactivation due to structural damage and functional blocking. However, as S-A block, N-V block may be transitory and may be caused by various stimuli such as strong vagal effect.

The inhibition of the sinus node pacemaker activity by the vagus has been accepted, but the simultaneous depression of the A-V nodal pacemaker cannot be easily explained since vagal effects on the lower pacemakers are still subjected to question. However, according to Balsano et al., the sinus node is chiefly innervated by the right vagus and the A-V node chiefly by the left vagus. Stimulation of the right vagus chiefly influences the sinus node, inducing either S-A block, sinus arrest or atrial fibrillation according to its intensity. Meanwhile, stimulation of the left vagus affects chiefly the A-V node, depressing conduction and inducing either partial block or ventricular arrest. The simultaneous occurrence of atrial fibrillation and N-V block in this patient strongly supports the presence of marked vagotonia. The mental tension caused by a quarrel with a newly-appointed, ill-natured nurse must have been the cause of this vagotonia. Or it may be that periods of ventricular asystole are due to repetitive concealed discharge of the node-His region by A-V nodal impulses, by which N-V block or a failure of re-entry is induced to reduce ventricular responses.

It was confirmed by Merideth and Titus that there are 3 pathways between the sinus and the A-V node in the human heart: anterior, middle and posterior atrial pathways. While, the A-V node has been divided into 3 regions by Paes de Carvalho: atrio-nodal, mid-nodal and node-His regions. The mid-nodal region is connected upward to the sinus node through the atrio-nodal region and the intra-atrial conduction system and downward to the ventricles through the node-His region and the His-Purkinje system. Therefore, N-V block can be induced by block between the mid-nodal and the node-His region or by block in the His-Purkinje system. The N-V block can also be of first degree, second degree (including type I, type II and type III) and third degree. Watanabe and Dreifus demonstrated that second degree A-V block due to excessive quinidine, high potassium or combination of both was characterized by failure of transmission below the node-His region or in the His-Purkinje-ventricular conduction system. Although it was often stated that the weakest link in the A-V conduction chain lies in the atrio-nodal junction, the lowest excitability and the greatest lag in recovery were observed in the mid-nodal region by Merideth and Titus. Also it was suggested by Hoffman et al. that delay in A-V transmission of premature atrial activity might be localized in part or in to the His bundle, the bundle branches or the more peripheral Purkinje fibers. Vasalle et al. induced asystole in animals with block of the A-V node by driving the ventricles at a rate higher than the idioventricular rate. Meanwhile, Merideth and Titus indicated that the cumulative depression of conductivity at higher driving frequencies is related to parallel changes in excitability. Although it is difficult to apply these experimental results to the clinical cases, a successive parasympathetic stimulation of the A-V nodal pacemaker or repetitive concealed conduction of the A-V nodal impulses into the node-His region may be one of the causes of ventricular asystole.

**Summary**

A 78-year-old female transiently developed atrial fibrillation with complete A-V block and bilateral bundle branch block after she had a quarrel with a newly-appointed, ill-natured nurse. During this period the ECG showed various types of second degree A-V node-ventricular (N-V) block such as multiple second degree N-V block type II, inducing ventricular asystoles of varying durations and second de-
gree N-V block type I (the Wenckebach period) showing a progressive shortening of and then a prompt increase in the RR interval. A special form of second degree N-V block in which a period of progressive shortening of the RR interval was followed by a period of constant RR interval and then terminated by second degree N-V block type II was referred to as second degree N-V block type III. Occasionally, the Wenckebach period in second degree N-V block was followed by a progressive increase in the RR interval which was designated as the counter-Wenckebach period.

REFERENCES


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