SINO-AURICULAR BLOCK, INTERFERENCE DISSOCIATION, AND DIFFERENT RECOVERY RATES OF EXCITATION IN THE BUNDLE BRANCHES

BY

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Disturbances in the propagation of the heart beat between the auricle and the two ventricles are of frequent occurrence and well known under the terms A-V dissociation or heart block. The term sino-auricular block is the standard name for an apparent block between the sinus node and the auricle, a hypothetical incident to explain transient auricular hypotension. The term auricular A-V dissociation, and conductivity following their refractory period, is the standard name for an apparent disturbance in A-V and intraventricular conduction invited some speculation.

Report of Case

A woman, 60 years of age, registered at the clinic with the main complaints of varicose veins, painful feet related to corns, mild shortness of breath, slight substernal discomfort with moderate exertion, and backache. No history was elicited that digitalis or other medicines had been taken. Auscultation of the heart revealed no murmurs. The blood pressure was 140 systolic and 90 diastolic. There were extensive varicosities of the veins of both legs and a stasis ulcer on the lower part of the left leg. The roentgenogram of the thorax showed moderate generalized cardiac enlargement. The roentgenogram of the spinal column showed a dorsal kyphosis and moderate osteo-arthritis changes. The cardiac diagnosis was coronary sclerosis and angina pectoris.

Electrocardiographic study. On the short electrocardiographic sequences obtained for routine evaluation there was noted an intermittent type of A-V dissociation during which occasional A-V conduction occurred. Only a few such complexes were present and the initial electrocardiographic diagnosis was probable A-V dissociation with interference dissociation, the occasional conduction being related to a super-normal phase of conduction. The patient returned the following day for more extensive study. For about the first fifteen minutes of electrocardiographic sampling, the normal sinus rhythm was present and this was not affected by pressure on either carotid sinus. Then spontaneously a cardiac irregularity appeared
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and a continuous record was obtained for approximately three minutes, during which about twenty-five premature beats were noted. The developed records showed phenomena similar to those of the preceding day but the abnormal mechanisms could be more definitely interpreted.

The various disturbances in rhythm and conduction are illustrated in Fig. 1 and 2. In Fig. 1, there are shown in the four tracings from above downward respectively, first, normal sinus rhythm with slight sinus arrhythmia, the P-P intervals varying from 1.24 to 1.56 sec.; second, sequences of S-A block, the heart being more rapid and the regular P-P intervals showing only slight variation and averaging 0.96 sec.; and third and fourth, nodal rhythm with interference dissociation in the simultaneously taken right and left precardial leads. The third tracing shows three premature ventricular beats, the first having the complex of right bundle branch block, the second that of left branch block, and the third a normal QRS complex. The fourth tracing shows two premature right bundle branch complexes and one left, the latter being interpolated between two normal QRS complexes.

In Fig. 2, the top sequence shows the usual or classic picture of interference dissociation in which the nodal rhythmicity rate is faster than the sinus rate, and a retrograde block is present. The R-R intervals of the nodal rhythm measure 1.68 sec. and the P-P intervals of sinus rhythm 1.76 sec.; thus, the record shows that the P waves gradually appear later and later after the R wave until finally the junctional tissues are found non-refractory and an interference beat occurs, and in the record portrayed there is an associated prolonged P-R interval.

Fig. 1.—Electrocardiograms discussed in the text. Four sequences are shown. In the lower two pairs of records, simultaneous tracings from two precardial positions are shown, position 1 being in the fourth interspace just to the right of the sternum, and position 6 being in the fifth interspace in the left midaxillary line. The indifferent electrode was on the right arm.

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and left bundle branch block. In the lower tracing of Fig. 2 are shown three premature beats, the first with a left bundle branch block complex, the second with a normal QRS complex, and the third with a right bundle branch block, and then return of normal sinus rhythm. It is to be noted that the premature ventricular beats have a definite time relationship to a preceding P wave and, depending upon the interval between the P wave that is to be followed by conduction and the preceding R wave of the nodal beat, the ventricular complex will show left bundle branch block, right bundle branch block, or a normal QRS complex.
Particular attention needs to be paid to the left bundle branch block complexes in the third tracing in Fig. 1 and in the bottom tracing of Fig. 2 where the T wave is deformed (marked X) by an apparent P wave of abnormal shape. These P waves occurred commonly after the left bundle branch block complexes but never with the right bundle branch block complexes. While these P waves possibly are indicative of auricular extrasystoles, the constant association with the left bundle branch complexes and the preceding prolonged P-R interval suggest that such P waves represent a re-entry into the auricle, in fact indicating auricular reciprocal beating.

**Comment**

The acceptance of the concept of a sino-auricular block is dependent only on the evidence of sudden excitation could not be readily measured. The frequent close association of a P wave with the R wave when a supposed nodal rhythm was present might theoretically be related to sino-ventricular conduction and partial sino-auricular block. The progressive increase in the R-P times in the sequence shown in Fig. 2 could be related to a progressive increase in S-A conduction time. The conducted beat of the interference dissociation sequence, if such premises were accepted, could then represent reciprocal rhythm. While a progressive increase in S-A time or a Wenckebach phenomenon has been claimed, on good evidence (Decherd et al., 1946), to occur, it is most unlikely that the phenomenon of sino-ventricular conduction without auricular response would have occurred without many examples of dropped auricular beats having been observed. Orthodox views, however, regarding the halving of the auricular rate, and all the objections of Lewis (1925) to the term and its implications still pertain. In the case reported herein, the disturbance in sinus rhythm resulting in marked auricular slowing allowed a lower rhythm to drive the ventricle and permitted the development of circumstances that give the picture of interference dissociation. If there were specialized conducting pathways between the sinus node and the A-V node, as Eyster and Meek (1914 and 1922) interpreted their experiments to indicate, and if one were to assume the presence of an entrance block into the auricle, a curious phenomenon of a sino-ventricular beat without an auricular contraction could occur. As Lewis et al. (1914) incidentally noted in their criticism of Eyster and Meek's investigations, excitation potentials related to sino-auricular-ventricular nodal conduction without contingent auricular nature of nodal rhythm with and without retrograde conduction to the auricle would seem to be well agreed upon, and they are explained and illustrated in standard textbooks on electrocardiography. The relatively short intervals in ventriculo-auricular and auriculo-ventricular sequential beats during heart block has probably attracted the attention of many investigators, and the problem was reviewed by Wolferta and McMillan (1929), who emphasized that the auricular elements of the sequences were represented by abnormally shaped and usually inverted P waves. The possible auricular re-entry beats in this case (Fig. 1 and 2) associated with conduction defects in the main bundle and left bundle block complexes are particularly interesting in that there was never retrograde conduction to the auricle during the long sequences of nodal rhythm.
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The only published electrocardiographic records seen that approach the nature of the tracings in the case reported herein are those shown in Fig. 196 of Scherf and Boyd's Clinical Electrocardiography (1946). The tracing is interpreted as showing extreme sinus bradycardia through 2:1 sinus block, with escaped beats and interference dissociation, with the conducted sinus beats showing an aberrant QRS complex owing to abnormal spread within the ventricle. The tracings demonstrating interference except for one conducted beat where there is impaired bundle branch conduction.

In the present case the number of observations seems sufficient to establish evidence of an absolute pattern of conduction in the main bundle and bundle branches dependent upon the time the P wave came after the R wave of the preceding nodal beat. The switch from left bundle branch block to right bundle branch block occurred at a critical time interval and was associated with a sharp decline in total A-V dissociation published by Jervell (1934) show complexes of the bundle branch form, but in these tracings nearly normal QRS complexes may occur with the same R-P and P-R intervals as those in which the intraventricular conduction defect is present.

The case in the paper by Cowan (1939) may have been similar to that of the case reported herein in that the records were said to show "variable sino-auricular block, the auricular rhythm being very irregular and infrequent." One tracing that was reproduced shows complete A-V dissociation conduction (Fig. 3). With the sequences showing left branch block there was a definite tendency for the total A-V conduction time to be shorter as the P wave fell farther away from the preceding nodal complex, but with the right bundle block complexes the total A-V conduction time remained relatively constant (Fig. 4).

One of the main difficulties in appraising the nature of the conduction of the interference beat is the lack of knowledge of the exact origin of the nodal beat within the junctional tissues. If such know-

![Diagram]

Fig. 3.—The diagrammatic illustration shows the relationship of P waves, represented by the black rectangles, to the preceding R wave of the nodal beat in respect to whether they are followed by left or right branch block complexes or a normal QRS complex. See text for explanation.
ledge were available the refractoriness of the upper and lower portions of the junctional tissues relative to one another might have been better estimated. The difficulties of interpreting the effects of blocked retrograde impulses on the refractoriness and possible supernormal phases of junctional tissues and myocardium have been emphasized by Langendorf (1948) and Mack et al. (1947).

One explanation of the phenomenon of the rapid alternation in the type of bundle branch block is illustrated in Fig. 5. It is assumed that the nodal beat originated in the upper portion of the junctional tissues so that the main bundle precedes that of the bundle branches in phases of excitation and refractoriness. The earliest P waves followed by conduction are shown to occur in the partially refractory state of the main bundle, and when the excitatory process reaches the bundle branches, the left bundle branch is completely refractory and the right partially refractory. Left branch block associated with partial right branch block is then present. The term "partial right branch block" is used as an equivalent to the term "partial heart block" when the latter means an abnormal prolongation of the P-R interval.

The diagram (Fig. 5) shows rapid recovery in the left branch so that an excitatory process traversing the main bundle at a later period travels more quickly through the left branch than through the right and a right branch block is manifest. A supernormal phase of conduction in the left branch might be added to the basis for the explanation but seems unnecessary. If the diagram as drawn were truly representative of the conditions, there would be a partial left branch block in addition to a manifest right branch block.

![Fig. 4. A graph to illustrate the relationship between the R–P period (the time interval between the beginning of the P wave that is to be followed by a ventricular complex and the preceding R wave of the nodal beat) and the P–R interval (the A–V conduction time). The sharp reduction in total A–V conduction when left branch block is replaced by right branch block is clearly shown.](http://heart.bmj.com/Br Heart J: first published as 10.1136/hrt.11.3.230 on 1 July 1949. Downloaded from http://heart.bmj.com)
The term "partial branch block" is somewhat confusing because the complete cardiographic record obtained will be dependent upon conduction in both bundle branches. For example, if the left bundle branch is normal, then depressed conduction in the affected right bundle branch gives rise to the picture of various degrees of incomplete right bundle branch block as originally discussed from the experimental and clinical cardiographic viewpoints by Wilson and Herrmann (1920). If both the bundle branches have similarly depressed zones of conductivity, the P–R interval is prolonged and the QRS complex is of normal duration. If there is complete loss of conductivity in one bundle branch, for instance, the left, and depressed conduction in the other, the right, then as happened in the case reported herein, there is an increased P–R interval and complete left branch block.

Excellent illustrations of bilateral branch block have been published by Bain (1941), Case 3 in his article being of particular interest. Incomplete heart block is shown, the usual mechanism being a 2:1 heart block with right branch block, but occasionally two auricular beats are conducted in sequence, the second of which shows a prolonged P–R interval and a left, rather than a right, branch block. It seems reasonable to suppose in Bain's case that retarded conduction was present constantly in the right bundle branch, and when the left bundle branch conducted normally, right bundle branch block was manifest. However, when the left bundle branch was completely blocked, the right bundle branch conducted after a delay, resulting in an increased P–R interval and left branch block.

In the case reported herein it would seem a
justifiable assumption that the depressed zones of conduction in the bundle branches were associated with a pathological state in the upper part of the ventricular septum. Utilizing the conception of unidirectional block related to the orientation of slightly depressed to severely depressed zones in the bundles, as outlined by Herrmann and Ashman (1931), one might explain the observed conduction defects in the following way. The first excitatory processes to be conducted meet a severely depressed area in the right bundle branch which, however, is eventually traversed, while in the left bundle branch there is a slightly depressed zone above a severely depressed area and complete block in the left branch occurs. A tenth of a second or so later, the left bundle branch has recovered, the excitatory process passes quickly through it, and a right bundle block complex appears. In general, it would appear that the right bundle branch conduction defect simulates type 1 A-V block while the left bundle branch conduction defect simulates type 2 A-V block as classified by Mobitz (1928), type 1 being a progressive increase in conduction time culminating in complete block, and type 2 being the sudden appearance of complete block without preceding increase in conduction time.

The possible relationship of the conduction disturbances to the clinical diagnosis of coronary insufficiency may be allowed if it be accepted that the blood supply to the conducting tissues were jeopardized. When normal sinus rhythm was present, there was never any auriculo-ventricular or intraventricular conduction defect. One might presume on a theoretical basis a further phasic decrease in blood supply during ventricular systole during which period the P wave that was to be followed by the bundle branch block QRS occurred. Such a supposition concerning the effect of systole on the blood supply to an ischemic zone is the direct opposite to that proposed by Wolferth (1928) in explaining the A-V conduction in his case. If the phenomena observed were the effect of anoxemic anoxia, the question of a possible elucidation of the problem from the work of Harris and Matlock (1947) arises. These investigators reported that the threshold of excitability was lowered and conduction rates increased in moderate anoxia while in severe anoxia the reverse effect was obtained. If the results were applicable to functional tissues, one might explain the phenomenon of the sudden increase in conductivity and perhaps even an increase of excitability in the left ventricle in this case by assuming that a hypoxic environment changed from a severe state to a moderate one.

**Summary**

A patient having intermittent sino-auricular block and interference dissociation was found to have also bundle branch block associated with the majority of the interference beats. The bundle branch block was either right or left, dependent upon the time relationships of the auricular beat to be conducted and the preceding R wave of the nodal (idioventricular) rhythm. The possibility of an intraventricular supernormal phase of recovery in conductivity in the left bundle branch might be utilized in explaining the phenomena, but it is not necessary if different recovery rates in excitation of the two bundle branches are hypothesized.

**References**