Two Cases of Complex A-V Interference-Dissociation

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As was theoretically classified by Miller & Sharrett, interference-dissociation can occur between 2 pacemakers in any part of the heart. However, there have been reported few clinical instances with interference-dissociation between 2 foci both located within the A-V conducting system. Recently we have experienced 2 patients with A-V interference-dissociation among more than 3 pacemakers, resulting from the depression of the sinus node. These cases might be worth demonstrating herein.

Case Reports

Case 1. An 18-year-old office worker, male, was admitted to our Clinic on Oct. 7, 1961, with the chief complaints of bradycardia and attacks of vertigo. He has a history of severe suppuration in the right arm at the age of 3 years, after which bradycardia (40 to 50 per min.) has been noticed. Since 16 years of age occasional attacks of vertigo and headache lasting several minutes occurred, accompanied by oliguria and edema. Recently he was found to have systolic hypertension (190mm.Hg) and albuminuria, and was recommended to be admitted to the hospital.

Upon the hospitalization, the patient was well-nourished and well-developed in apparent good health. Pulse was irregular with a rate of 42 per minute and his blood pressure in the supine position was 154/62mm.Hg. Laboratory examination revealed slight albuminuria without other abnormal findings, and normal renal function. Fecal and hematological examinations, blood chemistry and liver function tests were within normal limits. There were no signs of active rheumatism. The chest film disclosed no abnormality.

ECG: Fig. 1 is the electrocardiogram on admission. The P waves have retrograde contour (inverted in II, III and $V_f$) with variable time relationship to QRS complexes. Except for one ventricular beat in II, this arrhythmia may be understood presumably as a nodal rhythm with irregular RR intervals. But precise analysis cannot be made on this ECG alone.

Fig. 2 is a long tracing of Lead II at other time. It shows more complex arrhythmia than Fig. 1 and at least 3 different configurations of P waves can be recognized. The flat and notched P waves ($P_A$) with fixed PQ intervals

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of 0.16 second (P_3, P_9 and P_{11} of the upper strip and P_3, P_5, P_7 and P_9 of the lower strip) may be assumed as sinus P, tentatively. There are 2 types of inverted P waves; the one is wider and more plump with almost fixed QP interval of 0.10 second (P_B), and the other is rather diphasic with narrower downward deflection with variable PQ or QP interval (P_C). The difference between P_B and P_C is clearly demonstrated also in an esophageal lead (Fig. 3).

In Fig. 3, P_4, P_6, P_8 and P_{10} can be considered as retrograde P waves from an ectopic ventricular focus with fixed QP intervals of 0.19 second. P_7 and P_{12} which are of similar configuration to them, follow the QRS complexes with fixed QP intervals of 0.11 second, while P_{11} and P_{13} have the different contour and unfixed time relationship to QRS complexes. So, it may be concluded that P_7 and P_{12} correspond to P_B in Fig. 2 and P_{11} and P_{13} to P_C.
Fig. 4 is an analytical diagram of the relationship between PQ or QP intervals of each type of P waves and the preceding RR intervals. It shows that PA which is assumed to be sinus P with constant PQ interval of 0.16 sec., is preceded by the shortest cycle length, and PB is a retrograde atrial activation conducted with almost fixed QP interval of 0.10 sec. from a nodal center which escapes after the longest RR interval. Pc which has the different contour from BB with variable time relationship to QRS complex, falls on slightly shorter RR interval than PB. This fact supports the assumption of the other retrograde atrial activation from a lower atrial or upper nodal center, which is synchronised by QRS complex produced by the escaped lower nodal center (similar to the center of PB). The analytical figures below ECGs in Fig. 2 and 3 were made on these assumptions.

The most interesting point in this arrhythmia is the fact that 2 escaped centers within or near A-V node with almost isofrequent rhythm can exist, resulting in complex type of A-V interference-dissociation. Another explanation may be wandering pacemaker which is sometimes assumed in the interpretation of similar arrhythmias in the literature of the past. But, in this case, the above-mentioned analysis is preferable because RR or PP intervals sometimes show rather abrupt change and the configuration of the P waves can be classified into 3 definite groups.

Fig. 5, an ECG (Lead II) of this case taken on another occasion, might be interpreted indicative of wandering pacemaker within the A-V node because of the gradual change of time relationship of P waves to QRS complexes, but the underlying mechanism will be similar to those in Fig. 2 and 3.

Another comment on this case concerns with the sinus activation. Tentative interpretation that PA is of sinus origin may leave some room for discussion, when the mode of appearance of P waves is precisely examined. Because the rate of PA is very slow with some irregularity, which might be evidence for
brady-arrhythmia due to sinus depression or some degree of S-A block. As compared with the conspicuous irregularity of PB-PA or Pc-PA intervals, however, that of PA-PA intervals including escaped nodal beat is by far inconspicuous to the extent of implying the possibility that a parasystolic atrial ectopic center may be responsible for PA. After exercise or ephedrine injection (Fig. 6) the pace of PA was temporarily accelerated with disappearance of PB and Pc, but no other P waves, which might be assumed to be real sinus P, could be obtained. In this case, the existence of sinus depression is not deniable, though the origin of PA cannot be definitely decided.

Case. 2 A 46-year-old male entered our Clinic on June 13, 1960, with the chief complaints of dizziness, edema, exertional palpitation and shortness of breath. In his middle-school days, he suffered from pleurisy on the right side. At age 32, he experienced acute pancreatic necrosis and was operated on. In May, 1960, he had occasional episodes of vertigo, especially while riding a bicycle. Exertional palpitation and shortness of breath also appeared, associated with edema of the lower extremities.

Laboratory examination on admission showed slight impairment of liver function. The venous pressure was 210 mm. H₂O at the cubital vein. Cardiac shadow in chest X-ray film was enlarged to both sides and heart sounds were normal without any murmurs. By administration of diuretics, edema and cardiac enlargement disappeared, and elevated venous pressure was reduced to normal level. However, without improvement of brady-arrhythmia, he was discharged. No digitalis preparations were administered during hospitalization.

ECG: Fig. 7 shows the ECG taken by his home doctor before hospitalization. It reveals an idioventricular rhythm, and upright P waves in aVF and inverted P waves in aVR are seen immediately after every ventricular complex. This ECG may be interpreted as isofrequent A-V interference-dissociation due to sinus bradycardia and escaped ventricular rhythm. The ECG after admission shows sino-atrial block with ventricular escape (Fig. 8), which may, therefore, account for the fundamental mechanism of his brady-arrhythmia seen in Fig. 7.

Fig. 9 shows more complex arrhythmia. The upper lead designated as E₄₀ is an esophageal one, and the lower is aVF. As clearly demonstrated in the analytical diagrams below, there are noticed mutual transitions of the following rhythms: sinus rhythm, upper nodal escaped rhythm, A-V interference-dissociation between escaped P originating from the lower atrial or the upper A-V nodal center and the QRS complexes of lower nodal origin, and also A-V interference-
Fig. 7 ECG of case 2, taken before hospitalization.

Fig. 8 ECG of case 2, taken after admission.

Fig. 9 ECG of case 2. $E_{40}$ and $aV_F$ are simultaneously recorded (distorted with respiration).

dissociation between the aforesaid P and the QRS complexes of ventricular origin. By exercise or ephedrine injection, this arrhythmia was brought back to sinus rhythm.

**DISCUSSION**

It is theoretically indisputable that the phenomenon of interference dissociation may occur among several foci near A-V node including atrial and ventricular ectopic centers. But a few literature have been published concerning this phenomenon. This is partly because synchronization of paces of these ectopic centers is needed as a special causative condition,
and partly because this phenomenon may be transient and be seldom, if ever, diagnosed.

In 1932, Luten & Jensen reported 2 cases of A-V interference-dissociation between 2 rhythms with both centers located in the A-V conducting system, as examples of ventricular bigeminy in A-V nodal rhythm, suggesting that digitalis plays a role as one of the contributing factors. Later, a case of the same kind was reported by Schott, who attributed the mechanism of these ectopic inverted P waves to intra-auricular conduction disturbance in the A-V nodal rhythm. Barker described one case of such A-V dissociation, in which gradual change of configuration of inverted P waves and of time relationship of P to QRS complexes, was interpreted as wandering pacemaker within the A-V node, referring to an alternate explanation of interference-dissociation with 2 centers within the A-V node. Castellanos et al. reported and precisely analyzed 2 similar cases; the one has 2, the other has 3 pacemakers in the A-V node with concealed conduction. They emphasized that this kind of arrhythmia may result from digitalis intoxication in old patients with arteriosclerotic heart disease and congestive heart failure. In addition to the cases of double A-V nodal tachycardia described by other authors, Pick et al. have recently recognized 5 cases of such nodal tachycardia among 27 cases of various types of A-V nodal tachycardia with block, which were mostly produced by digitalis excess.

In our cases presented, it is noteworthy that there exist 2 ectopic centers in the A-V node, with the respective rate of 41 to 43 (case 1) or 34 to 36 (case 2) per minute in the upper pacemaker, and of 44 to 47 (case 1) or 34 to 36 (case 2) in the lower pacemaker, and one of these ectopic rhythms dominates the whole heart at times, or interferes with the other, or with sinus rhythm, or, moreover, in case 2, with ventricular ectopic rhythm.

All the cases in the literature being reviewed, one of the interesting points is the rate of A-V nodal pacemakers. The instances, in which the lower pacemaker has slow rate under 70 per minute, are only 3 cases: the case of Schott, case 2 of Castellanos and the case of Sanghvi.

A-V nodal pacemaker with the rate of over 70 per minute should belong to the category of A-V nodal tachycardia. However, A-V interference-dissociation with slow rate of A-V nodal pacemakers should be a different electrocardiographical entity from double A-V nodal tachycardia indicating enhanced A-V nodal activity that digitalis intoxication often produces.

Our 2 cases, not in old age, had no particular organic heart disease and received no digitalis preparations. In consideration of the causative factors of sinus depression, digitalis intoxication should not be so strongly
emphasized for the occurrence of this kind of arrhythmia.

SUMMARY

Two cases of complex arrhythmia were reported showing A-V interference-dissociation among more than 3 pacemakers, 2 of which are located within the A-V conducting system. Such complex type of A-V interference-dissociation is very rare in the literature.

REFERENCES