A Case Showing Atrial Dissociation and Other Various Kinds of Arrhythmias

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SUMMARY

A case is reported in which atrial dissociation occurred after oral isoproterenol administration to increase the heart rate in the presence of complete atrioventricular block. Two types of P wave, one with small upward deflection and the other with deep downward spike-like deflection, were present. The latter wave appeared at a high rate of 300/min, suggesting the existence of atrial flutter in the left atrium alone.

Additional Indexing Words:
Atrial dissociation  Isoproterenol  Atrial flutter

A case is reported which showed atrial dissociation in addition to other various kinds of arrhythmias.

CASE REPORT

A 52-year-old housewife was admitted to the Nippon Medical School Hospital on November 14, 1968. She was suffering from marked bradycardia with intermittent episodes of unconsciousness lasting for a few minutes, which had started to recur in July of that year. She had had several attacks of tachycardia since the age of 18 and presence of right bundle branch block was pointed out in July 1968.

On admission her consciousness was slightly cloudy. Arterial blood pressure measured 110/69 mmHg and the heart rate was 27 per minute and irregular. Chest X-ray film revealed significant cardiac enlargement with a cardiothoracic ratio of 63%.

Electrocardiogram showed marked bradycardia with very small P waves appearing at almost regular interval of about 0.58 sec, as seen in Fig. 1. No relationship existed between the P and QRS waves, indicating the presence of complete atrioventricular block. In addition, ventricular premature contractions occurred sporadically. Phonocardiogram showed a diastolic murmur in the third intercostal space. From these observations, a diagnosis of Adams-Stokes syndrome due to complete atrioventricular block accompanied by aortic insufficiency was made.

In spite of the continuous intravenous drip infusion of isoproterenol with

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dexamethasone from 9.25 PM of the day of admission to 7.30 PM of the following day, the heart rate increased to only 39 per minute. Thirty mg of isoproterenol was then given additionally via the oral route. After 30 min, a complicated arrhythmia appeared as seen in Fig. 2. This will be discussed later in detail. This arrhythmia continued for about 4 min, then returned to the atrioventricular block as previously observed.

On November 22, sinus rhythm was restored spontaneously (Fig. 3 left), although even after that day, several episodes of paroxysmal supraventricular tachycardia appeared (Fig. 3 right), occasionally accompanied by an atrioventricular block (Fig. 4). She was discharged on January 20, 1969. On March 8, 1969, however, a complete atrioventricular block recurred and a demand type myocardial pacemaker was implanted on April 25. The patient died suddenly on June 12, 1969.

Autopsy revealed marked cardiac hypertrophy with heart weight of 750 Gm.
Fig. 3. Left. Regular sinus rhythm observed on November 22, 1968, with right bundle branch block and marked left axis deviation.
Right. Supraventricular tachycardia observed on December 18, 1968.

Fig. 4. Electrocardiogram taken on November 18, 1968, showing atrial tachycardia with atrioventricular block.

A fresh hemorrhagic necrosis, 1 cm × 3 cm in size, was found in the subendocardial muscle layers of the left ventricle. The atrial myocardium, however, showed no marked changes either macroscopically or microscopically. The valves were intact and no further information was available to help in the interpretation of the diastolic murmur. Histological studies of the conduction system showed fibrosis interrupting all of the 3 fascicles.

Analysis of the electrocardiogram (Fig. 2): the strips which were almost continuously recorded. The upward tall waves seen in B, C, and D can be regarded as QRS deflection which appeared at almost fixed interval of 1.82 sec. The upward but very small waves (P) appearing at almost regular interval of 0.60 sec as seen in B, C, and D can only be interpreted as atrial activity. There is no regular rela-
tionship between the P and QRS waves, demonstrating the presence of a complete atrioventricular block. The shortening of the PP interval observed transiently in B can be ascribed to atrial premature contractions.

In addition, very large and downward spike-like waves (p) are observed in B, C, and D. Although the intervals between them are not regular, most of them are measured 0.5, 1.0, 1.5, or 2.0 sec.

In A of Fig. 2, there are 2 kinds of deflections: downward waves which appear frequently and upward waves which appear infrequently at interval of about 1.8 sec. Since the latter are regarded as expressions of ventricular excitation because of their frequency and shape which are similar to the QRS deflections seen in other tracings, the former should be regarded as the result of atrial excitation. However, such a deep downward configuration is very unusual for atrial excitation. Nevertheless, it is extremely rare that ventricular excitation should occur at such a high rate of approximately 300 per minute. These downward deflections are somewhat similar in shape to the waves denoted as p in the lower tracings. Consequently, the downward waves in A are taken as being the expression of atrial excitation, not of ventricular excitation.

From these findings coexistence of 2 kinds of atrial activities, i.e. atrial dissociation, can be concluded. The small upward waves are regarded as representing right atrial excitations because they resemble the P wave observed in the sinus rhythm indicated in Fig. 3; the other waves are ascribed to the left atrial ectopic activity because they direct downwards in lead I. The very high rate of about 300 per minute for the latter waves, as seen in A, suggests the presence of atrial flutter. However, it decreases to about 200 per minute in the latter half of A, indicating that this tachycardia is not simple atrial flutter. “Atrial tachysystole” must be a more appropriate expression for this irregularity.

DISCUSSION

Although various kinds of arrhythmias were observed in this case, the most striking one was the complicated arrhythmia suggesting atrial dissociation indicated in Fig. 2.

Two kinds of atrial dissociation have been described in the literature: one is due to “interatrial” or “intraatrial” conduction disturbance and the other to an ectopic pacemaker located in one of the atria.

In our case, interatrial disturbance is hardly conceivable because the pp interval (probably left atrial excitation) is sometimes shorter than the PP interval (probably right atrial excitation). This suggests that abnormal excitation of the left atrial ectopic center is more likely.

Deitz et al1) described 4 types of atrial dissociation: (1) 2 parallel sets of atrial waves; (2) flutter in one atrium and fibrillation in the other; (3) sinus or atrioventricular rhythm in the right atrium, flutter in the left, and (4) sinus or atrioventricular rhythm in the right atrium, fibrillation in the left.

According to this description, the atrial dissociation seen in B, C, and
D in Fig. 2 is classified as (1), and that in A as (3) or (4). The simultaneous recording of intracardiac electrograms of both atria would have made this clear in this case, but this procedure was not carried out because of the short duration of the arrhythmia.

Although Condrelli²) and Scherf and Siedeck³) produced the flutter experimentally in one atrium only, very few clinical observations have been reported. Especially, if we use the strict criteria for the diagnosis of atrial dissociation whereby 2 kinds of atrial activities are found in the same tracing, only the cases reported by Sanghvi,⁴) Bellet,⁵) Montella,⁶) and ourselves can be regarded as certain.

Furthermore, we have to pay attention to the artifacts in making diagnosis of atrial dissociation, because some periodical phenomena fall on the electrocardiographic curves, producing deflections which are almost indistinguishable from those caused by atrial activities.

Possible causes of artifacts usually include troubles in the electrocardiograph, rhythmic contractions of the skeletal muscles, or electrical noises induced from outside of the body. However, these causes are inconceivable in our case, because the complicated arrhythmia under discussion lasted for only a very brief period after the oral administration of isoproterenol.

Although the amplitude of a P wave of ectopic origin is low in the most reported cases of atrial dissociation, the amplitude of the downward deflection in our case measures about 0.5 mV. Similar high P waves of ectopic origin have been described in the cases of Snaghvi⁴) and Sivertssen et al.⁷)

As to the etiology of atrial dissociation, rheumatic fever, uremia, pneumonia, myocardial infarction, digitalis intoxication, and other several diseases have been reported. The production of atrial dissociation by isoproterenol, however, has not been described as far as we know.

References