Atrial dissociation

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A case of transient atrial dissociation developing in a woman with acute myocardial infarction is presented. The mechanism of this condition and its differentiation from other conditions which may mimic it are discussed.

In atrial dissociation a unilateral atrial rhythm which is never conducted to the ventricles exists independently of the basic atrial rhythm. The two rhythms do not interfere with each other (Chung, 1971). The condition is thought to be caused by complete interruption of impulse conduction between the right atrium and the whole (interatrial block) or part (intra-atrial block) of the left atrium, and implies disease of the Bachmann and the other interatrial bundles (Cohen and Scherf, 1965). Atrial dissociation has usually been observed in critically ill patients with severe congestive heart failure or atrial infarction, and is generally regarded as a grave sign (Chung, Walsh, and Massie, 1965; Khan, 1972). Despite the experimental demonstration of atrial dissociation (Scherf and Siedeck, 1934), and the publication of a number of electrocardiograms allegedly illustrating this condition in patients (Scherf, 1955; Chung, 1971), several authors have doubted its existence (Higgins, Phillips, and Sumner, 1966; Aygen et al., 1972). This doubt has attached particularly to the commonest type of atrial dissociation in which the supernumerary P waves (P') are of low frequency. In several such supposed cases it has been shown that what appear to be P' waves are really small electrocardiographic artefacts associated with inspiration and thought to be caused by the action potentials of the diaphragm (Soler-Soler and Angel-Ferrer, 1974) or the accessory muscles of respiration (Higgins et al., 1966).

We present a case of atrial dissociation in a patient with acute myocardial infarction who was not in congestive heart failure and in whom the P' waves were not associated with respiration or any other detectable source of electrocardiographic artefact. In addition to atrial dissociation a brief episode of ventricular irregularity was recorded associated with the appearance on the electrocardiogram of a second atrial ectopic focus (P') displaying some characteristics of atrial parasyostole.

Case report

The patient was a 50-year-old woman who was admitted two hours after the onset of central chest pain which radiated into both arms. She was known to have had a murmur but there was no history of hypertension or cardiac failure and she was not receiving any medical treatment. On physical examination the patient was not dyspnoic (respiratory rate 16/min), and the heart was regular (rate 82/min). The blood pressure was 200/110 mmHg (26.6/14.6 kPa). There was a loud pansystolic murmur indicating mitral regurgitation. There were no pulmonary crepitations and the jugular venous pressure was not raised.

The 12-lead electrocardiogram recorded on admission showed sinus rhythm (rate 78/min). The PR interval measured 0.13 s in lead II. The P waves measured 0.10 s in lead II and were notched in leads II, aVR, aVF, and V2 to V6. The PQ segments were isoelectric in all leads. The T waves were inverted in leads I, aVL, and V6 and were flat in leads V4 and V5. A provisional diagnosis of non-transmural myocardial infarction was made and the patient was monitored to give an electrocardiographic tracing similar to standard lead II.

Two hours after admission when the ventricular rate was 74/min, what appeared to be supernumerary P waves (P') were noted at a rate of 79/min. (Fig. 1A). The electrodes were not loose and no one was touching the patient or the electrodes; the patient was not hiccupping and there respiratory rate was 14/min. Atrial dissociation was suspected and the electrocardiogram was recorded continuously. The original P waves were unchanged and still captured the ventricles consistently with an unchanged PR interval. The P' waves were taller and narrower with a duration of only 0.05 s and a rate of 79/min initially. During the next 20 minutes the rates of both P and P' varied independently of one another, P' sometimes being slightly faster than P (Fig. 1A) and sometimes slower (Fig. 1B and C). Examples of P' moving right through the cardiac cycle were recorded (Fig. 1B and C).

During the recording the ventricular response became irregular transiently. An additional pacemaker (P') had developed. This new pacemaker was negative in contour and differed from P' in being broader and in sometimes being conducted to the ventricles. Initially P' followed
P after a fixed coupling interval and interfered with it in the manner typical of atrial premature contractions (Fig. 2A). Later the 'coupling' interval varied in several instances and the P' wave did not interfere with either P or P', suggesting that it had become parasystolic. At this stage the discharge rate of the additional focus (shortest interectopic interval) was 133/min. On one occasion it captured the ventricles continuously for approximately 3 seconds producing a short run of tachycardia (Fig. 2B and C).

Propranolol 20 mg and methyldopa 250 mg were given orally. The P' waves ceased and the ventricular response became regular again. A bipolar electrode catheter was passed into the right atrium in order to study the atrial rhythm in more detail, but the P' waves ceased before the electrode was in position. The patient was monitored electrocardiographically for two days by means of both the intra-atrial and chest electrodes but atrial dissociation did not recur. The diagnosis of myocardial infarction was confirmed by a rise and fall in the serum aspartate aminotransferase and lactic dehydrogenase. The patient went home in stable sinus rhythm and three months later was back at full-time work.

Discussion

The earlier published reports on atrial dissociation have been well reviewed by Chung et al. (1965), and the diagnostic criteria and differential diagnoses are discussed by Chung (1971). The present case shows unusually clearly the unilateral ectopic atrial rhythm independent of the basic (sinus) rhythm and shows no conduction of the ectopic rhythm to the ventricles (Fig. 1). The P' waves were much faster than the prevailing respiratory rate and were unrelated to respiratory or other movements. They were not followed by the transient microfibrillary waves which commonly follow artefacts arising from the diaphragm (Soler-Soler and Angel-Ferrer,

FIG. 1 Atrial dissociation. The normal P waves (P) and supernumerary P waves (P') do not interfere with each other and P' is never conducted to the ventricles. (A) P' faster than P. (B and C, continuous recording) P' is slower than P.
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FIG. 2 Electrocardiogram showing atrial dissociation and in addition a third atrial pacemaker ($P''$). (A) Fixed coupling interval (480–500 msec) between $P$ and $P''$, characteristic of atrial premature contractions. (B and C, continuous recording) Varying ‘coupling’ interval (100–520 ms) between $P$ and $P''$ suggesting atrial parasystole. Note the short episode of tachycardia caused by $P''$ at a rate of 133/min.

1974) and so were not caused by diaphragmatic action potentials. Nor were the $P''$ waves produced by dial telephones or other electrical instruments since none were present near the patient. They occurred while no one was touching the patient or the electrodes, and the latter had not become loose (Deitz et al., 1957).

The $P''$ waves were not examples of non-conducted atrial premature contractions since there was never any interference between them and the $P$ waves and since there was not a constant coupling interval between the $P$ and $P''$ waves. They were not parasystolic since they never conducted to the ventricles even when falling in the non-refractory period (Chung, 1971). First-degree interatrial block of variable severity, and interatrial Wenckebach block are ruled out by the uninterrupted march of $P''$ through $P$ and through the rest of the cardiac cycle regardless of which wave was the faster (Fig. 1): no example of $P$ or $P''$ being dropped was recorded. In the absence of other demonstrable causes for the $P''$ waves, the diagnosis of atrial dissociation seems established.

The exact cause of the atrial dissociation in this patient is unclear. The patient had an acute myocardial infarct and probable pre-existing mitral regurgitation. Serial electrocardiograms did not show changes diagnostic of atrial infarction. The duration of the $P$ waves was not abnormal but their notched configuration suggests slight underlying delay
interatrial conduction. The transience of the atrial dissociation (approximately 45 min) indicates that permanent damage of interatrial conducting pathways did not occur. The lack of change in the original P waves after the appearance of P', and the short duration of P' (0.05 s) suggest that only part of one atrium became dissociated from the rest of the atrial musculature. This could have resulted from relatively localized and transient atrial ischaemia. One must assume that the dissociated parts of the atria were mutually protected against each other's impulses by 'entrance' and 'exit' blocks as suggested by Deitz et al. (1957).

It is impossible to decide the exact origin of the P' waves. The negative deflection of P' and the initial fixity of the P/P' interval (Fig. 2A) suggest a low atrial source, producing apparently orthodox atrial bigeminy. However, the appearance of P' in Fig. 2 (B and C), with its inconstant conduction to the ventricles, the apparent lack of interference between P' and P and P', and the consequent lack of fixity of the P/P' 'coupling' interval, raise the possibility that it was a parasystolic focus. Such a focus must differ from one arising in a dissociated part of the atria in having complete 'entrance' block but incomplete 'exit' block (Chung, 1968). Whatever the cause of the irregular ventricular response noted in Fig. 2, it is noteworthy that atrial dissociation persisted throughout it without any interruption of the P and P' waves. To our knowledge this is the first such tracing to be described.

We wish to thank Professor Evan Fletcher who kindly reviewed the manuscript.

References


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