

# Sinus node dysfunction in acute myocardial infarction

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*The frequency, clinical course, and prognosis of sinus node dysfunction in 431 patients with acute myocardial infarction admitted to the coronary care unit were studied. Sinus node dysfunction occurred in 20 patients. In 13, the principal manifestation consisted of severe sinus bradycardia. In the remaining 7, periods of bradycardia alternating with episodes of supraventricular tachycardia were noted. Though several of the patients with sinus bradycardia required intravenous atropine or temporary pacing, normal sinus rhythm returned in virtually all during follow-up. The clinical course of patients with both bradycardia and tachycardia was less benign, during the acute phase and during follow-up; 5 of the 6 survivors required continued antiarrhythmic therapy or permanent pacing. The differences in the clinical course between these two groups of patients may reflect distinct underlying pathological changes. The findings in this study suggest that in contrast to sinus bradycardia, the occurrence of bradycardia-tachycardia syndrome during the acute phase of myocardial infarction may have important prognostic implications.*

Sinus node dysfunction is being increasingly recognized as a cause of serious cardiac arrhythmias and frequently presents difficult problems in diagnosis and therapy. Characteristic manifestations of sinus node dysfunction include persistent bradycardia, episodes of brief or sustained sinus arrest, and the bradycardia-tachycardia syndrome (Ferrer, 1973; Rubenstein *et al.*, 1972). Previous clinical and clinicopathological studies have shown that all these manifestations of sinus node dysfunction may be seen during the acute phase of myocardial infarction (Rokseth and Hatle, 1971; Lippestad and Marton, 1967). However, little is known concerning the subsequent clinical course and prognosis of such patients. In the study reported here, the frequency, clinical course, and prognosis of sinus node dysfunction were analysed in 431 consecutive patients with acute myocardial infarction admitted to the coronary care unit.

## Patients and methods

The charts and rhythm files of 431 consecutive admissions to the coronary care unit between January 1973 and July 1974 formed the material for this study. Since the admission policy to the coronary care unit in this

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institution dictates the exclusion of patients with severe congestive failure and cardiogenic shock, the patients included in this study were free from these major complications of myocardial infarction at the time of admission.

The cardiac rhythm of each patient was continuously monitored at a central console by trained technicians. A 40-s tape delay permitted the documentation of the onset and termination of any major change in the cardiac rhythm. At the end of each of the three 8-hour shifts, a summary of the arrhythmias, including representative rhythm strips, were incorporated into the patient's rhythm file. When arrhythmias requiring therapy with drugs occurred, the time, the dosage, and the route of administration of the drugs were also recorded.

Each chart and rhythm file was reviewed for evidence of sinus node dysfunction, which was considered to be present in patients with one or more of the following abnormalities: (a) persistent sinus bradycardia with rates less than 50 beats a minute during at least two consecutive 8-hour shifts; (b) sinoatrial block, 'sinus pause', or 'sinus arrest'; and (c) bradycardia-tachycardia syndrome. Patients in whom any one of the preceding arrhythmias was known to have been present before the myocardial infarction were excluded. Similarly, patients taking digitalis, propranolol, or reserpine were also excluded. Transient bradycardia associated with pain, or the administration of analgesic drugs was not considered evidence of sinus node dysfunction.

In patients with sinus node dysfunction, the presence of acute myocardial infarction was confirmed using the usual criteria of a typical clinical history and sequential electrocardiographic and enzyme abnormalities. The presence of previous myocardial infarction was also noted. The time of onset of sinus node dysfunction, its duration, and the presence of associated arrhythmias were also recorded. The hospital charts were reviewed to obtain data during the hospital stay after discharge from the coronary care unit. Follow-up information was provided by the private physician from his records.

### Results

Twenty patients fulfilled the criteria for sinus node dysfunction as set forth in the section on methods. There were 11 men and 9 women and their ages ranged from 39 to 79 years. Based on the major electrocardiographic manifestation during the acute phase, the 20 patients were subdivided into the following two categories: (a) those with severe sinus bradycardia, and (b) those with the bradycardia-tachycardia syndrome.

#### Sinus bradycardia

This group included 13 patients whose predominant clinical feature consisted of persistent sinus bradycardia with rates ranging between 30 and 50 beats a minute. Five patients also experienced brief episodes of supraventricular tachycardia lasting no longer than several seconds at a time. One patient was noted to have sinoatrial block. The average age of patients with sinus bradycardia was 69 years (range 42 to 79 years). The acute infarction was inferior in 11 and anterior in 2. A history of previous myocardial infarction was elicited in 5 patients. Sinus bradycardia was noted within 24 hours of admission in all patients and lasted from 24 hours to several days. In one patient, it lasted throughout the hospital course. Intravenous injections of atropine were used in all and resulted in a prompt but transient increase in the heart rate in 8. In 5 patients in whom atropine did not produce an increase in the heart rate, a proper evaluation of the effectiveness of the drug could not be made since the doses employed were small (0.4 mg). Two patients required temporary pacing. Significant associated arrhythmias consisted of ventricular fibrillation in 1, ventricular tachycardia in 3, and atrioventricular block in 2. None of the patients developed congestive heart failure, and none died in hospital.

One patient was lost to follow-up, and another died from recurrent myocardial infarction five

months after discharge. In the remaining 11 patients, clinical data and electrocardiograms were available for periods ranging between 10 and 30 months after discharge. Ten patients have had no bradycardia or any of the other manifestations of sinus node dysfunction. One patient, who was noted to have episodes of sinoatrial block during the acute phase of myocardial infarction, has continued to have sinus bradycardia though he remains asymptomatic (Fig. 1).

#### Bradycardia-tachycardia syndrome

Of the 7 patients in this group, 4 had inferior infarcts, 2 had anterior infarcts, and the remaining patient had a non-transmural infarct. Their ages ranged between 39 and 77 years, with an average of 70 years. None gave a history of previous myocardial infarction. The clinical course in these 7 patients was characterized by prolonged episodes of atrial tachyarrhythmias alternating with periods of sinus bradycardia and slow escape rhythms. One patient, who was admitted with sinus bradycardia, developed atrial fibrillation shortly after admission and, within a few hours, developed ventricular standstill which did not respond to attempts at resuscitation. In 5 of the 6 remaining patients, tachyarrhythmias became manifest within 24 hours of admission and lasted throughout the period of hospital stay. In 1 patient, episodes of tachyarrhythmias subsided within 48 hours of admission. The tachycardias were variable in duration, lasting from a few seconds to several hours. Spontaneous cessation of tachycardias was followed by prolonged periods of sinus standstill (Fig. 2) requiring the insertion of temporary pacemakers in 5 patients. Though radiological evidence of mild left ventricular failure was observed in many during tachycardia, none developed significant congestive failure or hypotension.

Five of the 6 survivors in this group have continued to experience recurrent episodes of atrial tachycardia, flutter, or fibrillation during the 30-month follow-up period. Four have required permanent pacing in addition to a combination of antiarrhythmic drugs, and one has remained well on antiarrhythmic drugs alone. The only patient who has required neither drugs nor a permanent pacemaker was the one noted to have tachycardias only for a 48-hour period during the acute phase of infarction. Thus, in contrast to the patients with sinus bradycardia, the occurrence of bradycardia-tachycardia syndrome during the acute phase was associated with persistence of this arrhythmia during follow-up.

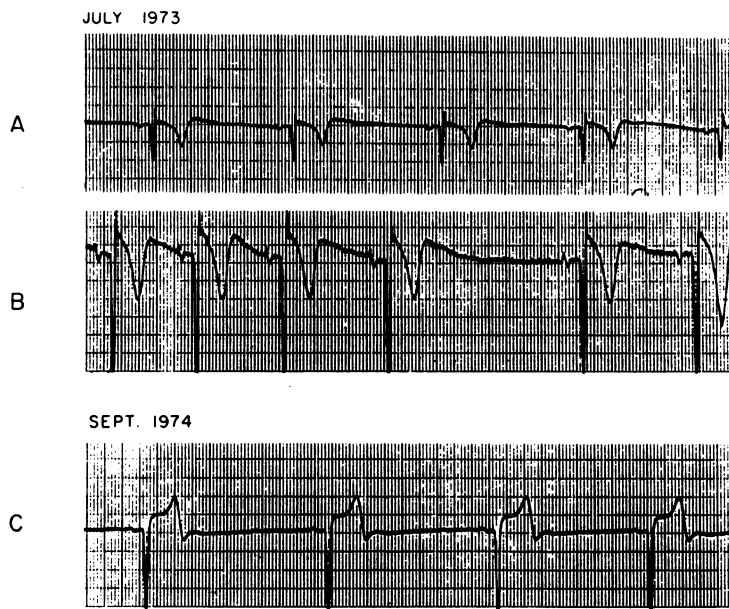


FIG. 1 Representative rhythm strips from a patient with acute inferior myocardial infarction and persistent sinus bradycardia. Panels A and B were recorded during the acute phase and show severe sinus bradycardia (A), and episodes of sinoatrial block (B). Severe sinus bradycardia has persisted during repeated follow-up examinations (panel C) though the patient remains asymptomatic (see text for discussion).

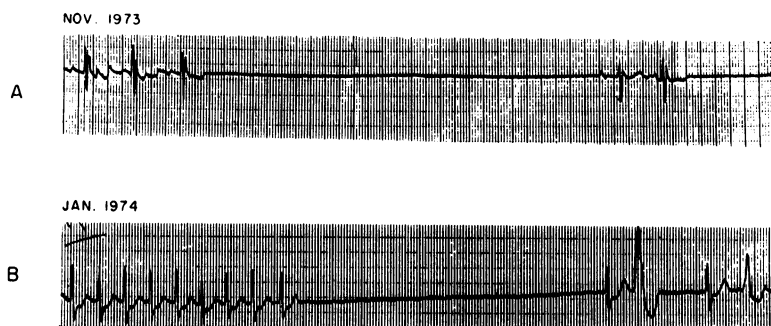


FIG. 2 Rhythm strips from a patient with inferior myocardial infarction and bradycardia-tachycardia syndrome. The top strip (A) was recorded 3 days after myocardial infarction, and the bottom strip (B), upon readmission 2 months later. Note periods of prolonged sinus standstill after cessation of the tachycardia.

### Discussion

Previous studies have reported an incidence of between 4 and 5 per cent for sinus node dysfunction in patients with acute myocardial infarction (Rokseth and Hatle, 1971; Rotman, Wagner, and Wallace, 1972). In the present study, sinus node dysfunction was noted in 20 out of 431 patients

with acute myocardial infarction, yielding an incidence of 4.6 per cent. Though the exclusion of patients with congestive failure and cardiogenic shock may have eliminated some additional patients with sinus node dysfunction, it must be kept in mind that the drugs used in the therapy of both these complications may give rise to arrhythmias of the type described, making the

diagnosis of sinus node dysfunction difficult.

Analysis of the clinical data revealed no relation between sinus node dysfunction and the age, sex, or a history of previous myocardial infarction. There was a preponderance of inferior infarction in the group as a whole. This relation was particularly striking in the case of sinus bradycardia, whereas in patients with bradycardia-tachycardia syndrome, the proportion of inferior to anterior infarcts was similar to that observed by James (1961) in a group with atrial arrhythmias. There appeared to be no relation between sinus node dysfunction and the severity of myocardial infarction as judged by the mortality or the subsequent development of congestive heart failure or hypotension. This is in contrast to the high incidence of such complications reported by Rokseth and Hatle (1971). Though the reasons for this difference are unclear, it is possible that the early use of temporary pacing in our patients played a role in preventing such complications.

The most interesting observation in this study concerned the dissimilarity in the subsequent clinical course between patients presenting with sinus bradycardia and those with the bradycardia-tachycardia syndrome. It is notable that 5 out of 7 patients with the latter arrhythmia required permanent pacing in addition to antiarrhythmic drugs, whereas none of the patients with sinus bradycardia alone required such measures. This difference may be explained by the differences in the underlying pathology and pathogenesis between these two types of arrhythmias after acute myocardial infarction. During the acute phase of myocardial infarction sinus bradycardia may be caused by a variety of factors including pain, fear, the administration of analgesic drugs, or depression of sinus node automatically induced by ischaemia, cholinergic influences, and products of tissue breakdown (Rotman *et al.*, 1972; Billette *et al.*, 1973). Though the criteria used in the selection of patients in this study excluded those with transient bradycardia as a result of pain, fear, and drugs, the

onset and duration of the arrhythmia in most instances were compatible with vagally mediated depression of the sinus node. In patients with the bradycardia-tachycardia syndrome on the other hand, pathological studies have shown characteristic lesions consisting of infarction of the sinus node and the adjacent atrium (Lippestad and Marton, 1967; James, 1961). As suggested by James (1961), such a combination of sinus node and atrial damage appears to provide an explanation for the occurrence of the bradycardia-tachycardia syndrome during the acute phase and its persistence after recovery. The atrial damage, by virtue of its proximity to the sinus node, may impair conduction from the sinus node and also predispose to re-entry and re-entrant tachycardias. Since the impairment of sinoatrial conduction may be expected to persist after healing of the infarction, the arrhythmia may recur after recovery from the acute infarct.

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