

# Treatment of Slow Heart Rates Following Acute Myocardial Infarction

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Recent advances in the intensive care of patients suffering from acute myocardial infarction have shown that the immediate mortality may be lowered by the prompt recognition and treatment of cardiac arrhythmias (Hellerstein and Turell, 1958; *Lancet*, 1960; Brown *et al.*, 1963; Day, 1963; Lindsay and Speikerman, 1964; Kurland and Pressman, 1965). While ventricular fibrillation is the usual cause of sudden death, a frequent precursor appears to be a slow rate due to slow nodal rhythm or complete atrio-ventricular block (Brown *et al.*, 1963), with a tendency to episodes of asystole or ventricular tachyarrhythmia. Despite the effectiveness of some drugs such as atropine, steroids, and isoprenaline, the over-all mortality in patients with a slow rate following myocardial infarction is high (Solarz, Berkson, and Pick, 1958; Gale and Enfroy, 1959; Dall and Buchanan, 1962; Hall, 1962; Vogel, 1961; *Brit. med. J.*, 1965; Smith and Anthonisen, 1965). This may be explained in part by the failure to keep a constant increase in heart rate and the increase in myocardial oxygen consumption caused by many of these drugs, particularly isoprenaline (Winterscheid *et al.*, 1963). A better chance of survival seems likely if the slow rate is treated by artificial pacing, since the heart rate can then be accurately controlled without drugs which may irritate the myocardium. Unlike most patients with chronic atrio-ventricular block where the underlying cause is frequently unrelated to coronary disease, acute atrio-ventricular block following myocardial infarction is usually temporary, as is sinus bradycardia or slow nodal rhythm. It seems that the combination of a low cardiac output secondary to the slow rate together with a damaged irritable myocardium often results in ventricular fibrillation. If the cardiac output can be raised by increasing the heart rate, the risk of ventricular

fibrillation should be reduced. In addition cardiac depressant drugs can be safely used to suppress ectopic rhythms once satisfactory pacing has been established.

Although defects in A-V conduction are relatively uncommon in patients with acute infarction, the mortality ranges between 39 and 100 per cent (Dimond, Dunn, and Brosius, 1960; Courter, Moffat, and Fowler, 1963; De Saint Pierre, Toscani, and Massi, 1963; Friedberg, Donoso, and Stein, 1964; Robinson *et al.*, 1965) and has now been considerably lowered by the prompt use of electrical pacing of the heart (Delman, Schwedel, and Escher, 1963; Samet, Jacobs, and Bernstein, 1963; Levy and Albert, 1964; Bruce *et al.*, 1965). We report our experience of pacing 12 patients for slow rates secondary to acute myocardial infarction.

## SUBJECTS AND METHODS

All patients in the past 5 years who had acute myocardial infarction have been continuously monitored on a 3 in. (7.6 cm.) oscilloscope, and equipment has been readily available for acute resuscitation. With the sudden onset of Stokes-Adams attacks due to asystole, the situation can be temporarily controlled by external pacing but may produce skin burns and is extremely painful for the patient on return of consciousness, and may require general anaesthesia (Zoll, 1952; Leatham, Cook, and Davies, 1956). As soon as possible a unipolar C.50, No. 5 electrode catheter (U.S. Catheter Corporation), is passed via an antecubital fossa vein into the right atrium, and this procedure is greatly aided by the use of a high definition image intensifier with a field of 7 in. (17.8 cm.) or more. The electrode tip is passed through the tricuspid valve and then into the pulmonary artery, thereby demonstrating that the electrode has not inadvertently been placed into the coronary sinus. The tip is then withdrawn and manoeuvred into a position as low down and as far out in the apex of the right ventricle as possible. Many positions at the apex of the right ventricle may have to be tried before a stable pacing site is found, and the threshold for pacing is then measured

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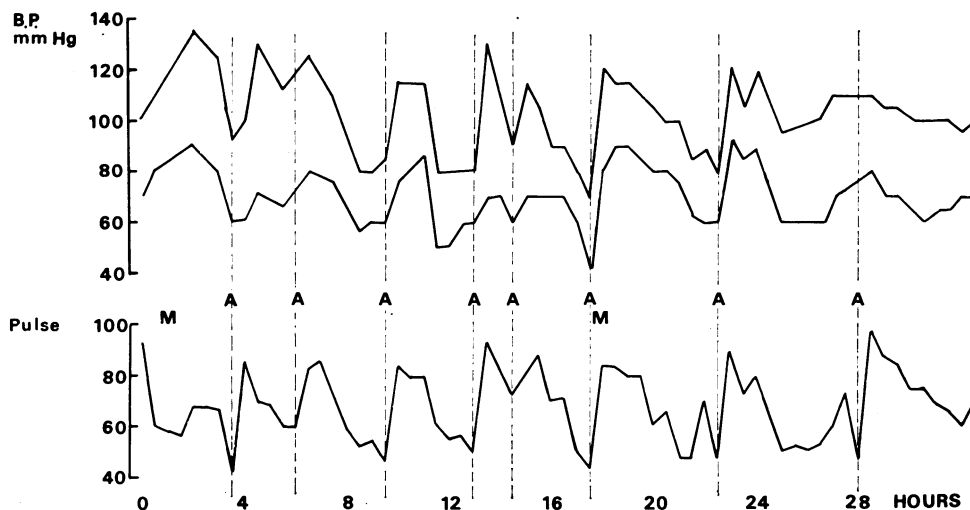


FIG. 1.—Sinus bradycardia, with a fall in systemic blood pressure following acute myocardial infarction treated by repeated intravenous atropine 0.6 mg. (A). The systemic pressure was recorded with a sphygmomanometer. 10 mg. morphine (M) was given for severe chest pain on two occasions. An increase in heart rate and systemic blood pressure followed every dose of atropine but the over-all response was irregular.

and should be less than 1 volt at 2 milliseconds duration. The patient is then encouraged to breathe deeply and cough, and if these manoeuvres interrupt pacing another position will have to be found. The positive electrode is sutured to the exposed subcutaneous tissue alongside the electrode catheter, which is firmly tied into the antecubital fossa with silk ligatures, and the skin incision is closed. Both electrodes are then connected to a pacemaker unit which has variable controls for changing rate and power. The patient's electrocardiogram and pulse are observed throughout, and means for immediate external pacing, defibrillation, and pulmonary ventilation kept available.

Although an antecubital fossa vein is the easier and faster route for insertion of the electrode catheter in an emergency, displacement is possible when the patient's arm is moved unless the arm is carefully splinted. Since artificial pacing may have to be continued for several weeks, the external jugular route is probably better since the electrode catheter is then more stable.

**Drug Therapy.** Most patients developing A-V block or sinus bradycardia received prior treatment with atropine, ephedrine, or isoprenaline and steroids (Fig. 1). Lack of adequate response or the production of an unstable rhythm, particularly with isoprenaline, necessitated artificial pacing. Once satisfactory pacing has been established, ectopic beats or ventricular arrhythmias may be controlled by procaine amide or xylocaine, given preferably as a continuous intravenous infusion, and digitalis may be safely used. On occasions when procaine amide or xylocaine has failed to control ventricular arrhythmias,  $\beta$ -blocking agents, particularly propranolol (inalderl), have been effective.

**Patients Treated.** The clinical details of 12 patients are shown in the Table. There were 11 men and 1 woman between the ages of 39 and 73 (average 57 years). It was thought that 11 patients had had recent cardiac infarction; the other presented with severe angina and syncopal attacks associated with short periods of slow nodal rhythm. The length of history of slow rates was short in 7 patients (less than 3 days).

**Electrocardiograms.** Acute diaphragmatic infarction was present in 5 patients, antero-septal in 2; there was no localized infarct on the electrocardiogram in 4 others, but the combination of a history of severe retrosternal pain, shock, and the subsequent course of their illness, was strongly suggestive of acute myocardial infarction.

The remaining patient (Case 8) had a 7-day history of recurrent attacks of severe retrosternal chest pain followed by brief periods of loss of consciousness. Before an attack his electrocardiogram was normal (Fig. 2), and after an attack T wave inversion appeared in leads I and aVL (Fig. 3). The electrocardiographic events which occurred during an attack of chest pain with a brief loss of consciousness were recorded on a continuous electrocardiogram tape recorder (lead II), and showed that soon after the onset of angina there was sinus bradycardia followed by nodal rhythm with acute S-T elevation in lead II (Fig. 4); at this stage the patient became unconscious and pulsation could not be detected in the carotid or femoral arteries though the heart rate was 50 a minute. These changes rapidly regressed on recovery of sinus rhythm. Six months later coronary arteriography showed a localized area of narrowing in the dominant left main coronary artery.

TABLE

CLINICAL AND CARDIOGRAPHIC DATA OF 12 PATIENTS TREATED FOR SLOW HEART RATE AFTER ACUTE MYOCARDIAL INFARCTION

Patient No.	Age (yr.)	Sex	Clinical diagnosis	Electrocardiogram	Drug treatment	Duration of endocardial pacing (days)	Result		Post-mortem findings
							Died (days)	Survived	
1	60	M	Acute cardiac infarction	Asystole	Isoprenaline	30 min.	After 30 min.		Severe coronary artery disease; old and recent posterior cardiac infarctions
2	58	M	Acute cardiac infarction	Acute diaphragmatic infarct; complete heart block; ventricular tachycardia; asystole	Isoprenaline; atropine	11	11		No necropsy
3	59	M	Acute cardiac infarction	Acute diaphragmatic infarct; sinus rhythm; intermittent 2:1 heart block	Atropine; steroids; anticoagulants	15		In sinus rhythm, active, and well after 8 mth.	
4	65	M	Acute cardiac infarction	Sinus rhythm with intermittent slow nodal rhythm; ischemic changes	Atropine; long-acting isoprenaline; anticoagulants	9	9		Severe coronary artery disease; antero-septal cardiac infarction
5	67	M	Acute cardiac infarction	Sinus rhythm with intermittent complete heart block; ventricular ectopics/tachycardia and fibrillation	Ephedrine; isoprenaline; long-acting isoprenaline; steroids	19		Long-term pacing needed; active and well after 8 mth.	
6	49	M	Acute cardiac infarction	Diaphragmatic cardiac infarction; complete heart block; asystole; ventricular tachycardia and fibrillation	Procaine amide; propranolol	3	3		Severe coronary artery disease; anterior and diaphragmatic cardiac infarction
7	39	M	Acute cardiac infarction	Diaphragmatic cardiac infarction; complete heart block	Anticoagulants	5		In sinus rhythm, active and well after 2 yr.	
8	45	M	Acute coronary insufficiency	Slow nodal rhythm; ischemic changes	Long-acting isoprenaline; atropine; anticoagulants	11		In sinus rhythm; active and well after 16 mth.	
9	57	M	Acute cardiac infarction	Complete heart block	Ephedrine; isoprenaline; adrenaline	3			No necropsy
10	64	F	Acute cardiac infarction	Diaphragmatic cardiac infarction; complete heart block; ventricular fibrillation		1	1		Severe coronary artery disease; diaphragmatic cardiac infarction
11	73	M	Acute cardiac infarction	Antero-septal cardiac infarction; complete heart block; asystole	Isoprenaline	7	10		Severe disease anterior descending coronary artery; antero-septal cardiac infarction
12	54	M	Acute cardiac infarction	Diaphragmatic cardiac infarction; slow nodal rhythm; complete heart block	Isoprenaline	19		In sinus rhythm; active and well after 3 mth.	

*Duration of Artificial Pacing.* Temporary endocardial pacing was carried out for periods ranging from half an hour to 19 days, with an average of 8 days. The preliminary plan had been to discontinue pacing when normal sinus rhythm returned, but in 2 patients com-

plete heart block recurred, and subsequently pacing was continued for several days after sinus rhythm had been re-established. The electrode was left in position for a further 7 days in order that pacing might be restarted if the conduction defect recurred.

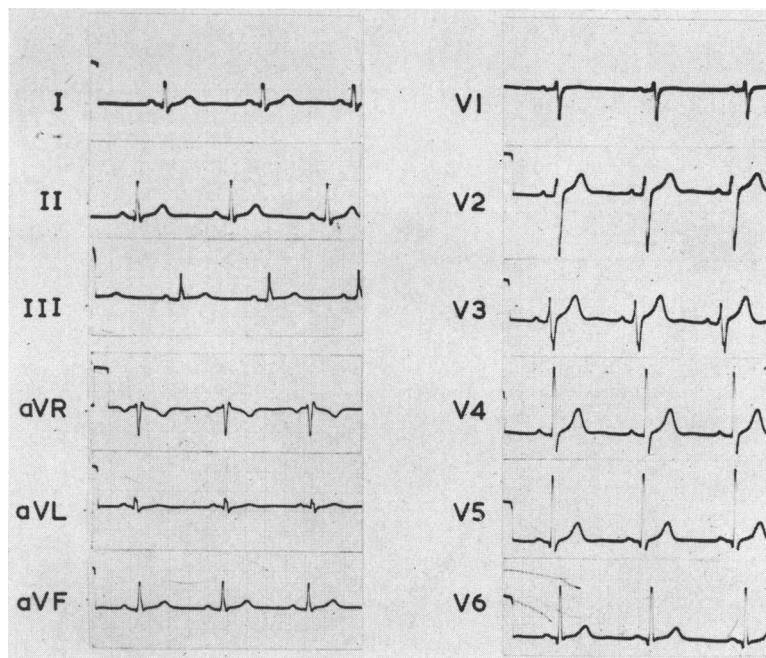


FIG. 2.—Electrocardiogram in Case 8 before attack of angina associated with syncope.

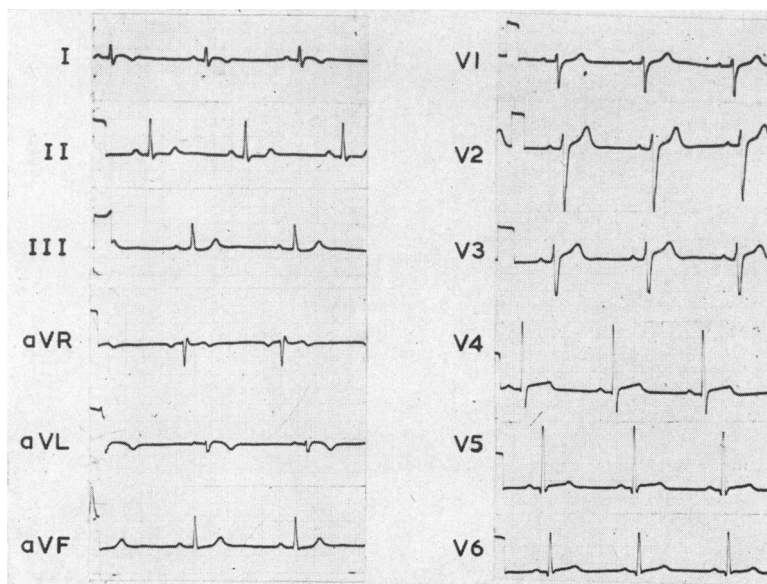


FIG. 3.—Electrocardiogram in Case 8 obtained 30 minutes after an attack of angina associated with syncope, showing T wave inversion in leads I and aVL, with S-T segment flattening in V4-V6.

### RESULTS

Five patients survived; 4 were paced until sinus rhythm returned after an average of 10 days (range 5-15 days), and the other remained in complete heart block which required long-term endocardial pacing.

Seven patients died, 5 of myocardial failure, despite pacing until the time of death, and 2 patients died suddenly 10 days and 21 days, respectively, after sinus rhythm had returned and endocardial pacing had stopped.

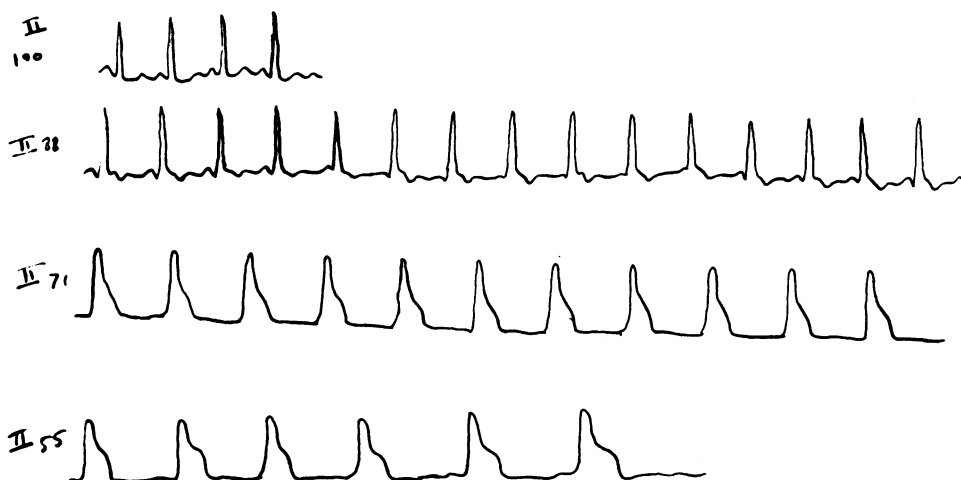


FIG. 4.—Record of lead II in Case 8 during an attack of chest pain associated with syncope. The heart rate slowed from 100 to 55 a minute with the development of acute S-T segment elevation and nodal rhythm. These episodes were later completely prevented by endocardial pacing.

#### DISCUSSION

The best method at the moment of lowering the mortality of acute cardiac infarction probably lies in the prompt recognition and treatment of cardiac arrhythmias and slow rates. This requires continuous monitoring of all patients for several weeks following acute myocardial infarction. Endocardial pacing should be used as soon as possible when evidence of atrio-ventricular conduction disturbance is obtained, and continued for at least 2 to 3 weeks following a return to sinus rhythm unless a ventricular inhibited pacemaker is available. This rather prolonged period of pacing is recommended, since 2 of our patients died suddenly several days after pacing had been discontinued. So far there have been no adverse effects from continuing endocardial pacing in the presence of sinus rhythm, though there is a theoretical possibility of producing ventricular fibrillation from R on T phenomenon (Wiggers, Wégria, and Piñera, 1940; Sowton, 1965). It seems that if the heart rate and cardiac output are satisfactorily maintained and ectopic foci are suppressed with procaine amide or  $\beta$ -blocking agents, there is very little risk in continuing pacing in the presence of sinus rhythm. The treatment of slow heart rates, following acute myocardial infarction, with steroids, atropine, or isoprenaline is often unsuccessful. The principal reason is often that failure to obtain an immediate and controlled increase in heart rate with drugs may end in disaster, since, in contrast to patients with chronic heart block who usually do not have significant coronary artery disease, patients with ischaemic heart disease do not survive recurrent periods of cardiac arrest. The

advantage of endocardial pacing is that the heart rate and cardiac output can be better controlled, and myocardial metabolism is not increased to the same extent as it might be with isoprenaline (Winterscheid *et al.*, 1963). Furthermore, once pacing has been established, ectopic rhythms, if still apparent, may be safely suppressed by procaine amide or  $\beta$ -blocking agents such as propranolol (inderal), even in the presence of complete heart block. If slow rates were treated with atropine or isoprenaline alone, it would be extremely dangerous to treat ectopic rhythms with cardiac depressants without some means of artificial pacing being available.

Seven patients had acute diaphragmatic infarction shown either by the electrocardiogram or at necropsy, confirming the more likely chance of conduction disturbances with this site of infarction, which is in agreement with the observation of Cohen, Doctor, and Pick (1958) and James (1962), and emphasizing the need for careful monitoring of these patients. However, the mortality in patients who develop conduction defects due to antero-septal infarcts is higher than in those patients who have conduction defects following postero-diaphragmatic or inferior infarction (Cohen *et al.*, 1958; Zion and Bradlow, 1964). Presumably this is due to the more extensive anterior necrosis necessary to interrupt atrio-ventricular conduction.

One patient who was having severe attacks of angina associated with syncope secondary to episodes of slow nodal rhythm had complete relief of symptoms with pacing which was continued for 7 days, though throughout this time he remained in sinus rhythm. Arbitrarily at the end of this

period, pacing was stopped and the patient has remained symptom free over the past 18 months. Selective coronary angiography showed a narrowed segment of the dominant left coronary artery. Maybe with borderline coronary flow, the slowing precipitated cardiac ischæmia causing further slowing, and pacing prevented this cycle of events.

#### SUMMARY

Eleven patients have been treated by artificial pacing for slow heart rates following acute myocardial infarction. One other patient with cardiac ischæmia was paced for syncopal attacks associated with severe angina and slow nodal rhythm. Of the 12 patients, 7 died; the cause of death in 5 was extensive myocardial infarction resulting in an irreversible fall in cardiac output despite satisfactory pacing, and in the other 2 a late recurrence of heart block. Drugs such as atropine and isoprenaline for slow rates following acute myocardial infarction have not proved entirely satisfactory, since their response is often unpredictable and dangerous arrhythmias may be produced. A good case can be made for always monitoring the cardiogram following cardiac infarction and treating any evidence of atrio-ventricular block by endocardial pacing. Since atrio-ventricular block may recur some days after recovery of normal conduction, artificial pacing should be continued for at least 3 weeks unless a reliable ventricular inhibited pacemaker is available. Ectopic foci may be safely suppressed during pacing by procaine amide or  $\beta$ -blocking agents.

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#### ADDENDUM

Since September 1965 we have had experience in the use of a ventricular (QRS) inhibited pacemaker (designed by Mr. J. G. Davies, Senior Technical Officer, Cardiac Department, St. George's Hospital, London) in a further 8 patients. All 8 patients have survived, which may in part be due to starting endocardial pacing as soon as electrocardiographic evidence of a disturbance of atrio-ventricular conduction was obtained. Once sinus rhythm is re-established at a satisfactory rate, the patient remains connected to the ventricular-inhibited pacemaker for a further 3 weeks. Providing a satisfactory heart rate is maintained, no electrical pacing of the heart will occur until the rate falls below a predetermined rate set on the ventricular inhibited pacemaker.

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