Pacing after acute myocardial infarction

ALAN HARRIS
B.Sc., M.D., M.R.C.P.

Cardiac Department, St George's Hospital, London, S.W.1.

Summary

Experience of endocardial pacing in eighty-two patients with atrioventricular block after myocardial infarction is described. A unipolar pacing wire was passed into an antecubital fossa vein and passed into the right atrium and wedged into the apex of the right ventricle under radiological control using an image intensifier. All patients were attached to an external demand unit set at 60/min which will pace if there is asystole longer than 1 sec. The disadvantages of pacing are discussed.

A fall in mortality occurred from 71% of unpaced patients to 38% of thirty-seven patients where demand pacing was given. When suppressant drugs (lignocaine, procainamide and quinidine) were given in addition to pacing the mortality dropped to 11% of twenty-seven patients. The disadvantages of pacing are few and it is considered that suppressant drugs are only used with safety when a pacing system has been installed.

ACUTE myocardial infarction is complicated by complete heart block in 5-8% of all patients (Brown, Hunt & Sloman, 1969) and when untreated carries a mortality rate ranging between 48-71%. Transvenous endocardial pacing for the management of Adams-Stokes disease is now established as a satisfactory method of treatment. More recently temporary transvenous pacing has been successfully used for the treatment of the acute disturbances of atrio-ventricular conduction which may follow acute myocardial infarction (Epstein *et al.*, 1966; Harris & Bluestone, 1966; Lassers & Julian, 1968). During the past 9 years we have treated ninety-five patients with second degree or complete atrioventricular block complicating acute myocardial infarction. The majority of these patients have been referred to our unit from other hospitals and hence there must be some element of selection which might have influenced our results. Details of the first fifty-five patients and the technique used have been reported (Sutton, Chatterjee & Leatham, 1968).

Method

We prefer to use a US Catheter Corporation uni-

polar C 5650 or bipolar C 5651 transvenous wire passed via a medially placed antecubital fossa vein exposed under local anaesthesia. There are some who advocate the percutaneous subclavian vein approach but this technique requires a high degree of skill to avoid local complications such as pneumothorax or damage to the brachial plexus. We prefer not to use the external jugular veins for temporary pacing but to preserve these in case long-term pacing is eventually required. The transvenous wire is passed into the right atrium using a high-definition image intensifier. The bipolar electrode wire avoids the need for a separate positive electrode but is slightly more rigid than the unipolar wire. The electrode wire is passed through the tricuspid valve, usually by making a loop off the right atrial wall, and then into the pulmonary artery, thereby demonstrating that the electrode wire has not been inadvertently placed in the coronary sinus. The electrode is then withdrawn from the pulmonary artery and its tip manoeuvred into position as low down and as far out in the apex of the right ventricle as possible (Fig. 1) and 'wedged' into position by gentle pressure. Sometimes many positions at the apex of the right ventricle have to be tried before a stable site is found. During the manipulation of the electrode tip within the right ventricle, ectopic rhythms may be induced but they usually disappear once pacing is started. The wire is attached to an external demand pacing box (Device Implants Ltd.) and the threshold for pacing (voltage required) is measured and this should be less than 1 volt at 2 sec duration. Pacing is then commenced at just above the measured threshold and the stability of the electrode tip in the right ventricle is checked by encouraging the patient to breathe deeply, and cough; if these actions interrupt pacing even for one cycle, another position is found. If the electrode wire is 'wedged' with unnecessary force then perforation of the myocardium will occur (Fig. 2) leading to early or late loss of pacing, but rarely causing tamponade.

A further test of satisfactory electrode tip impaction is obtained by recording the endocardial electrocardium, by attaching all four limb leads of a battery-powered electrocardiograph to the patient.

FIG. 1. Chest X-ray showing a satisfactory endocardial electrode position. The electrode tip is 'wedged' at the apex of the right ventricle. The course of the wire through the right atrium is free from redundant loops.

FIG. 2. Chest X-ray showing myocardial perforation by the electrode tip which is lying beneath the pericardium.

and the chest lead switched to V_1 connected to the free end of the endocardial electrode wire. When the endocardial electrode is well impacted into the right ventricular wall an injury pattern with elevation of the S-T segment of at least 2-3 mV is usually obtained (Fig. 3). The patient should then be screened again to observe that no redundant wire has been left in the right atrium (Fig. 4) since later a loop may

form in the electrode wire within the heart resulting in the withdrawal of the electrode tip from a stable position and intermittent pacing may result.

Once an ideal pacing position is obtained the electrode wire is firmly tied to the antecubital fossa vein and neighbouring tissues with several silk sutures. If a unipolar wire has been used then an indifferent electrode will have to be placed in the

FIG. 3. Endocardial electrocardiogram showing in (A) a good injury potential indicating the electrode tip is satisfactorily 'wedged' and in (B) a poor injury potential indicating the electrode tip is not in satisfactory contact with the endocardium.

FIG. 4. Chest X-ray showing a redundant loop of electrode wire in the right atrium which later resulted in loss of pacing.

subcutaneous tissue and similarly fixed. The main disadvantage with the use of an arm vein is the ease of displacement of the electrode tip from the 'wedged' position if unrestricted arm movement is permitted. We therefore always restrict arm movement with a special harness but satisfactory restriction can be obtained with a fracture board.

Occasionally the situation may demand that transvenous pacing be carried out without X-ray screening facilities. This may be quite rapidly achieved by passing the electrode wire from an antecubital fossa vein, especially the left, and checking the position of the electrode tip by recording the intra-cardiac cavity electrocardiogram which clearly differentiates the right atrium, right ventricle and pulmonary artery position of the tip of the electrode wire (Chatterjee *et al.*, 1969). When the typical right ventricular cavity electrocardiogram is recorded the electrode wire is advanced gently and impacted and often a good injury current is recorded and a low pacing threshold obtained. Pacing is then commenced and the position of the electrode wire radiologically checked with a portable chest X-ray.

All patients are attached to an external demand unit set at 60/min, which will pace if there is asystole longer than 1 sec, at no more than twice the minimum voltage required to stimulate the right ventricle. Demand pacing with low power will theoretically

lower the risks of inducing ventricular fibrillation by inappropriate stimulation during ectopic rhythms or during sinus competition (R on T). A unipolar electrode is recommended because a greater intracardiac potential (which inhibits the demand pacemaker) is recorded with a unipolar than with a bipolar electrode thereby reducing the risk of inappropriate stimulation due to low intracardiac potential. A number of patients have been observed to suddenly develop competing fixed-rate pacing although at the time their own heart rate was satisfactory; this was found to be due to a fall in the intracardiac potential (not usually reflected in the conventional external electrocardiogram) which often occurs following acute myocardial infarction (Chatterjee *et al.*, 1970).

All our patients now have suppressant therapy once the pacing system is satisfactory, usually intravenous infusion of lignocaine (1–3 mg/min) for 3 days and then long acting quinidine as quinidine bisulphate (Kinidin Durules, Astra Chemicals) 0.25 g four times daily, or procainamide 250–500 mg q.d.s. Oral quinidine seems to be more effective than oral procainamide in suppressing ventricular ectopic beats. Because of the risk of precipitating ventricular asystole, oral ventricular depressant drugs must not be started until the pacing system is installed. Continuing ectopic beats despite increased dose of suppressant therapy are treated by increasing the pacing rate, but care must be taken when ventricular function is borderline since too fast a rate may induce heart failure.

Heart failure is treated by diuretics and digitalis and lack of improvement with atrio-ventricular pacing (requiring a second transvenous wire to pace the atrium) in order to obtain the benefit of atrial transport.

The threshold of stimulation is checked daily, and pacing voltage should be set at not more than twice the threshold value. A rise of threshold from any cause to above 2 volts at 2 msec is not treated by increasing the power of the stimulus but by repositioning the electrode wire to achieve a threshold as low as possible.

Endocardial potentials should be checked daily to avoid inappropriate stimulation, particularly in patients with shock and heart failure. Inappropriate stimulation from low cavity potential requires increased sensitivity of the demand pacemaker or continuous pacing at a higher rate to avoid competition. Increasing the sensitivity of the inhibiting circuit of the pacemaker will increase the risk of inhibition of the pacemaker by external electrical interference from, for example, electric razors, cleaning machines, physiotherapy appliances or even by potentials from skeletal muscle activity (Fig. 5). The demand system is left in position for 1 week after return to sinus rhythm (average 2 days) and

FIG. 5. The effect of the pectoral muscle electromyogram (EMG), during arm movement, on the endocardial electrocardiogram (ICP) in an unpaced subject (unipolar system). Demand pacing with a sensitive box is interrupted by the pectoral EMG.

P. J. Reynolds pace beat counter is connected with an external demand pacemaker (set at 50) for the final 24 hr to exclude intermittent block, which requires prolongation of pacing care. If the pace-counter shows no pacemaker action for 24 hr, the pacing system is removed but suppressant drugs are continued.

Results

The first eighty-two patients were divided into three groups according to the date of treatment and the technique (Table 1). The thirty-two patients who were unpaced (Table 1) were admitted to hospital before our coronary care units were opened and were not referred to the cardiac unit. Table 1 shows that the mortality in the paced groups has fallen from 66 to 11%, although the population of patients with poor prognosis, judged by anterior infarction, heart-failure and shock, was little different in the three groups. Factors responsible for the improvement in group C were thought to be early pacing with avoidance of syncope, impairment in cardiac output,

demand-pacing with awareness of the possibility of inappropriate stimulation, and the free use of suppressant drugs.

Early pacing

In this series of eighty-two patients syncope was associated with a mortality of 52%, compared with 20% in those without syncope (Table 2). Conclusions of the effect of syncope must be provisional because these patients are also the worst risk group, but early pacing to avoid syncope, and hence further deterioration in the clinical condition, seems to be a prime objective. Furthermore, the installation of a pacing system in a convulsing patient can be difficult.

Cardiac output

Improvement in the clinical state with pacing is often remarkable, but, if shock and heart failure continue, indicating a bad prognosis (43% mortality), atrioventricular pacing may improve cardiac output because of appropriate atrial systole (Martin & Cobb, 1966).

TABLE 1. Mortality of acute myocardial infarction complicated by heart block

	No. of cases	Death (%)	Anterior infarct. (%)	Shock and heart failure (%)
<i>Unpaced:</i>				
1963-66	32	71	38	—
<i>Paced:</i>				
A. 1961-66				
Fixed rate pacing	18	56	27	66
B. 1966-68				
Demand pacing	37	38	16	57
C. 1968-69				
Demand pacing and suppressant therapy	27	11	22	66

TABLE 2. Prognostic factors in eighty-two patients with post-infarct atrioventricular block (second or third degree)

	No.	Mortality (%)
Anterior infarct	17 (20%)	76
Inferior infarct	65 (80%)	23
Wide QRS	26 (31%)	73
Narrow QRS	56 (69%)	16
Onset known < 24 hr after pain	25 (36%)	32
Onset known > 24 hr after pain	45 (64%)	38
Syncope	34 (42%)	52
No syncope	48 (58%)	20
Good clinical state	22 (26%)	13
Shock and failure	60 (73%)	43
Return to sinus rhythm	60 (73%)	16
No return to sinus rhythm	22 (27%)	82

Demand pacing

The fall in mortality with demand-pacing in 1966 coincided with a reduction of pacemaker power to just above threshold level, tested daily. The theoretical safety of demand pacing received a setback when inappropriate stimulation due to transient falls in the endocardial potential, used to inhibit the pacemaker, was found in some patients with shock (Fig. 6). It is possible that the death of some of the patients with shock who failed to improve with pacing could have been related to low cavity potentials and inappropriate stimulation producing ventricular dysrhythmias.

Suppressant drugs

The free use of suppressant drugs has been the

major factor in reducing the high mortality associated with ectopic beats and ventricular dysrhythmias which may continue despite satisfactory pacing (Table 3). Increasing the pacing rate alone was only transiently effective in suppressing ectopic rhythms and intravenous lignocaine was far superior, though sometimes both were required.

The disadvantages of pacing are summarized in Table 4. Electrode displacement would have been less frequent if subclavian or external jugular veins had been used for electrode insertion, but the antecubital vein approach is much easier for non-specialized staff, particularly in a sick patient, and is less worrying to the patient. There were four patients on demand pacemakers who were improving yet unexpectedly developed ventricular fibrillation and

FIG. 6. The upper trace shows the RV endocardial potential which at times is too small to inhibit the demand pacemaker unit. The lower trace shows sinus rhythm and two inappropriate pacemaker stimuli. RV = right ventricle, III = lead III of the electrocardiogram, Thr Dem = minimum endocardial potential required to inhibit the demand pacemaker.

TABLE 3. Deaths among thirty-nine patients (47% of the eighty-two patients) with ventricular dysrhythmia not abolished by pacing

	No. of patients	Deaths (No.)	Deaths (%)
Suppressant drugs	23	3	13
No suppressant drugs	16	11	68

TABLE 4 Disadvantages of pacing in eighty-two patients

	No.	Deaths
VT or VF during electrode insertion	6	0
Electrode displacement	19	0
Suspected perforation (rise of threshold)	3	0
Phlebitis	6	0
Ectopic beats induced by pacing	2	0
Unexpected VF (demand)	4	3
Unexpected VF (fixed rate)	1	1

VT=ventricular tachycardia, VF=ventricular fibrillation.

three died. Inappropriate stimulation without adequate ventricular suppressant therapy was probably a major factor.

Following a period of ventricular pacing the unpaced electrocardiogram shows massive T wave inversion and ST depression which persists for a varying length of time depending on the duration of

FIG. 7. The pre-paced electrocardiogram shows sinus rhythm with normal S-T segments and T waves. After 7 days of continuous ventricular pacing, an unpaced electrocardiogram shows conspicuous T inversion in II, III, aVF, and V₃-V₇.

pacing (Figs 7 and 8). If the period of pacing is only a few minutes' duration (Fig. 9), then the ST and T wave changes rapidly regress, but if pacing has continued for several days, then the changes may persist for weeks. There is, however, nothing to suggest that the changes which occur in the ST segments and T wave after depolarization from an

FIG. 8. Series of electrocardiograms obtained from the same patient during 15 days of endocardial pacing. The T wave inversion progressively increased in II, III and aVF, reaching a maximum on day 15.

FIG. 9. A series of electrocardiograms obtained from the same patient pre-pacing and after short intervals of endocardial pacing. Immediately after 10 min of endocardial pacing T inversion occurred in V_3 - V_4 which fully regressed within 10 min. A further 10 min of pacing produced T inversion again.

abnormal site are of any clinical importance, except that they may be misinterpreted as evidence of progressive coronary disease.

In conclusion, immediate low-power demand

pacing for second and third degree block following infarction, together with the liberal use of myocardial depressant drugs, appears to be lowering the mortality of this serious complication of myocardial infarction. With the safeguards described, the disadvantages of pacing are few and it is only when a pacing system has been installed that it is safe to use suppressant drugs.

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