THE HEMODYNAMIC EFFECTS OF ARTIFICIAL PACING IN COMPLETE HEART BLOCK

COMPlicATING ACUTE MYOCARDIAL INFARCTION

by

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INTRODUCTION

Complete heart block (CHB) developing after acute myocardial infarction is a complication which carries a high mortality. Although heart block is almost always transient in those who survive, the haemodynamic consequences of bradycardia and the danger of ventricular asystole during the period of disordered atrio-ventricular conduction jeopardise the patient's chance of survival. With the development of intensive coronary care, temporary transvenous endocardial pacemaking has become a widely used method of treatment of this disturbance of conduction (Kimball and Killip 1965, Escher 1967, Sowton 1967, Lassers and Julian 1968). Rational management by pacemaking must be based on knowledge of the circulatory changes associated with heart block and with artificial pacing in the particular circumstances of acute myocardial infarction. Although the haemodynamic effects of pacing in chronic heart block are well documented (Escher et al. 1964, Judge et al. 1964, McGregor and Klassen 1964, Samet et al. 1964, Segel et al. 1964, Sowton 1964), there have been no studies in acute myocardial infarction. This report describes the haemodynamic changes associated with advanced heart block complicating acute myocardial infarction and their response to asynchronous ventricular pacing at varying rates.

METHODS

Clinical Material

Thirteen patients with atrio-ventricular block complicating acute
myocardial infarction were studied in the Coronary Care Unit of the Royal Infirmary, Edinburgh (table 1). The clinical diagnosis of myocardial infarction was confirmed in all cases by electrocardiographic and serum enzyme changes. All had developed CHB within 4.4 days of the onset of infarction and the mean duration between the onset of symptoms and the development of CHB was 28 hours. All patients were studied within 24 hours of the onset of CHB and most within 6 hours. All patients had CHB within 30 minutes prior to the beginning of the study: 11 remained in CHB throughout the studies on the first day, but 2 developed second degree block immediately prior to study. No patient had received digitalis, a diuretic, a sympathomimetic agent or an antiarrhythmic drug. Analgesics had not been given within 3 hours of any study and only one patient had received oxygen within one hour of the study.

Six patients were studied with and without pacing on subsequent days while still in heart block, and 5 of these were studied after return of sinus rhythm. Two patients died while in CHB: one of shock in spite of effective pacing and one of asystole unresponsive to pacing. Normal AV conduction returned in the remaining 11. These 11 patients survived to be discharged from hospital.

During the 5 month period in which these patients were investigated, 8 other patients with CHB complicating acute infarction were managed in the coronary care unit. Seven of these 8 patients died, however, before a pacing electrode could be inserted and studies performed.

**Techniques and Measurements**

In 9 patients cardiac outputs were calculated by the Fick principle. In the remaining 4 patients, cardiac outputs were determined by dye-dilution methods. Bipolar pacing electrodes (U.S. Catheter Co.) were inserted per-
cutaneously into the right subclavian vein and manipulated under fluoroscopic control to the apex of the right ventricle. This procedure and all studies were carried out at the bedside in the coronary care unit. The electrode was connected to a variable rate, variable current continuously discharging external pulse generator incorporated in the Sanborn "Visomonitor" unit. A single lumen, or in the case of the dye-dilution studies, a triple lumen catheter was then advanced from an antecubital vein until its tip lay in the main pulmonary artery. Right atrial pressures were recorded through the proximal lumen of the triple lumen catheter or by means of a third catheter passed via an arm vein or the subclavian vein. Systemic arterial pressures and blood samples were obtained through a nylon catheter inserted percutaneously into a brachial artery and advanced centrally. Pressures were transduced with Sanborn 267B manometers or Bell and Howell 4-327-L221 strain-gauge manometers and recorded either on a Mingograf 243 ink-jet recorder or on a Devices M4 direct-writing recorder. Zero reference level for the manometers was taken as 5 cm below the level of the manubrium sterni.

Expired air was breathed through a dry gas meter, the volume recorded in liters on a direct-writing recorder and the oxygen and carbon dioxide content analysed by the Lloyd-Haldane method. During a 5 minute expired air collection period, arterial and mixed venous blood samples were withdrawn continuously and analyzed for oxygen tension, carbon dioxide tension and pH. The oxygen saturation of the blood was derived from Dill's desaturation table and the oxygen content was calculated from the saturation and the haemoglobin capacity.

In the case of the dye-dilution studies, indocyanine green was used as indicator and injected into the right ventricle via the middle lumen of the
triple-lumen catheter. Blood was sampled from the arterial catheter through a Waters Xc-302 cuvette-densitometer and curves recorded on a Servoscribe direct-writing ink recorder. Calibration was carried-out at the end of each study by drawing known concentrations of dye in samples of the patient's blood through the cuvette.

The following formulae were employed:

Systemic Vascular Resistance = (Mean arterial BP - Mean right atrial BP x 80)/Cardiac index x 100 (dyne sec cm²/sq.m)

Left Ventricular External Work = MSEF x Cardiac index x 13.6/1000 (kg-m/min/sq.m)

Tension Time Index = MSEF x Duration of systole x Heart rate (mm Hg sec/min)

Where MSEF = Mean Systolic Ejection Pressure obtained by planimetry.

When direct right atrial pressure was not recorded it was considered for the purpose of calculation of systemic vascular resistance to be 6 mm Hg if the jugular venous pressure was not elevated clinically. If the venous pressure was elevated, a clinical estimate was made and this figure substituted.

Statistical analysis was carried-out employing Student's t-test with the method of paired comparisons.

Plan of Investigation

Fick Principle Cardiac Output Studies: Whenever possible, control measurements were made in heart block and pacing was then performed for a period of 20 to 30 minutes before the measurements were repeated. Serial studies were performed at 3 or 4 different rates: i.e. in the range 1) 60 to 80/min, 2) 81 to 100/min, 3) 101 to 120/min, 4) 121 to 140/min. In each instance observations were repeated after a period of 20 to 30 minutes stabilisation at the new rate of pacing.
Dye-Dilution Studies: In the 4 patients in whom the cardiac output was measured by dye-dilution, continuous intravascular pressures were recorded and dye-dilution curves were performed at 5 minute intervals throughout the study. Initial control observations were made for 15 minutes. The pacemaker was then turned on and measurements continued for a further 15 minutes when the rate was increased.

**RESULTS**

Cardiac Index

In 2 cases (J.N. and A.S.) baseline measurements in CHB were not made because the patients required pacing urgently. The remaining 11 patients were studied in heart block on the first day. Three of these were studied again while still in heart block on subsequent days. The mean cardiac index in these 11 patients studied on 16 occasions in heart block was 2.22 ± 0.84 L/min/sq.m. This increased by 28% with pacing at the rate associated with the highest cardiac index (maximal output rate) to 2.85 ± 0.83 L/min/sq.m \( (p<0.001) \) (table 2, figure 1). In 9 patients the cardiac index in heart block was less than 2.50 L/min/sq.m. In these 9 patients pacing at maximal output rates resulted in an increase of 45% from a mean value of 1.74 ± 0.40 L/sq.m to 2.54 ± 0.72 L/min/sq.m \( (p<0.001) \). The cardiac index was greater than 2.50 L/min/sq.m on the first day of CHB in 2 patients and had risen above 2.50 L/min/sq.m in 2 others by the second and third day respectively. In these patients the increase in cardiac index from a mean level in heart block of 3.27 ± 0.49 to 3.54 ± 0.65 L/min/sq.m with pacing at maximal output rates was not statistically significant.
In the 11 patients studied on 16 occasions on the first and subsequent
days of heart block, cardiac index increased with pacing in 14 instances, fell
in one instance and did not change in another. The single patient (G.R.) whose
cardiac index fell with pacing on the second day of CHB, had an index of
2.98 L/min/sq.m with a ventricular rate of 52/min. This fell with pacing at
a single rate of 104/min to 2.67 L/min/sq.m. However, this patient had pre-
viously had an index of 1.29 L/min/sq.m in CHB with a ventricular rate of
44/min on the first day of CHB which had increased to 2.47 L/min/sq.m with
pacing at 80/min. The patient (J.R.) whose index failed to increase signifi-
cantly was in second degree heart block at the time of study with a cardiac
index of 1.81 L/min/sq.m and a ventricular rate of 50/min.

Patient R.C. was studied for a control period of 15 minutes in heart
block and then paced at 83/min for 65 minutes with repeated cardiac output
measurements throughout the 80 minute period of study (figure 2). The maximal
response to pacing at this rate was reached within 10 minutes and outputs
remained steady over the 65 minute period.

Figure 3a shows the effect of increasing ventricular rate on the cardiac
index. It includes only the 8 patients paced at 3 or more rates. In 2 of
these (J.N. and A.S.) measurements were not made in heart block. In 6 patients
there was a progressive rise in cardiac index with increasing ventricular rate.
The maximal cardiac output response occurred at rates above 100/min in all
patients who were paced at rates above this level. In 2 patients the cardiac
index fell as the rate was increased from 107/min to 125/min (G.C.) and 115/min
to 130/min (J.D.). Although the maximal response occurred above 100/min, an
adequate increase in cardiac index had occurred with rates between 80 to 90/min
in most patients. In one patient (J.M.) who had severe cardiogenic shock and whose clinical condition appeared to be deteriorating steadily, the cardiac index increased slightly from 1.21 L/min/sq.m to 1.44 L/min/sq.m with pacing at 83/min, but fell to 0.99 L/min/sq.m when the rate was increased to 104/min (figure 4).

The results of haemodynamic measurements repeated between 2 and 10 days after the onset of CHB are presented in figure 5.

Mean Systemic Arterial Blood Pressure

The mean systemic blood pressure was measured in heart block in all 13 patients and averaged 80 ± 14.7 mm Hg. Pacing at maximal output rates increased it to an average of 96 ± 17.3 mm Hg (p < 0.001) (table 2, figure 1). The group of patients with cardiac indices of less than 2.50 L/min/sq.m in heart block had an average mean pressure of 82 mm Hg which increased with pacing at maximal output rates to 98 mm Hg. The group of patients with higher cardiac indices had an identical average pressure in heart block of 82 mm Hg which increased with pacing at maximal output rates to 97 mm Hg. However, if the initial blood pressure was low, there was a proportionately greater increase with pacing than if it was initially high. The average increase for patients with pressures of 80 mm Hg or less during heart block was 32% compared with 18% for those with higher initial pressures. Blood pressure fell with pacing at maximal measured output rates despite increases in cardiac index in 2 patients. Patient W.H. studied again on the third day of heart block had developed 2:1 block with a relatively high cardiac index, while patient M.P. was paced at 120/min to suppress a nodal tachycardia which was alternating with CHB (table 2).

In 8 patients blood pressure was measured in heart block and during pacing at 3 or more rates (figure 3b). Blood pressure rose progressively in all but
2 patients (C.O. and G.R.). In these 2 patients there was an initial fall with pacing at 65/min and 75/min respectively before it rose with higher rates. In G.R. this was associated with a fall in cardiac index, but in the case of C.O., cardiac index had risen. In patient J.D. (figure 6) blood pressure rose when the rate was increased from 115/min to 130/min although cardiac index fell. With these exceptions, changes in blood pressure tended to parallel changes in cardiac index produced by pacing. There was no consistent relationship, however, between the absolute levels of cardiac index and the patient's blood pressure on the first or subsequent days of heart block or after return to sinus rhythm (figure 5).

**Stroke Index**

The mean stroke index in the 11 patients in whom measurements were made in heart block was $41 \pm 15.8$ ml/sq.m. With pacing at maximal output levels this decreased by 25% to $31 \pm 8.9$ ml/sq.m ($p<0.005$) (table 2, figure 1). In 6 of the 16 instances in which measurements were made in both heart block and during pacing in these 11 patients, the stroke index increased or fell only slightly with pacing despite increases in heart rate of between 15 to 46/min (table 2). Patient M.F. represents a special case. She had CHB with an idio-ventricular rate of 42/min alternating with brief periods of nodal tachycardia at 115/min and periods of ventricular asystole. Her average ventricular rate was 98/min. Pacing at 120/min suppressed the nodal tachycardia and resulted in an increase in stroke index of 6 ml/sq.m.

In 7 of the 8 patients paced at 3 or more rates, stroke index fell progressively with increases of heart rate. In the case of patient C.C., however,
stroke index increased with pacing at 87/min and 107/min and only fell when the rate was increased to 125/min when the cardiac index also fell (figure 3c). Figure 5 shows the changes in stroke index on subsequent days. Despite relatively large increases in heart rate, stroke index either had changed very little or had increased.

**Systemic Vascular Resistance**

The average systemic vascular resistance for the group as a whole did not change significantly with pacing at maximal output rates (table 2). However, the patients with cardiac indices of less than 2.50 L/min/sq.m had an average resistance of 3468 ± 873 dyne sec cm⁻⁵/sq.m which fell significantly with pacing at maximal output rates to 2956 ± 641 dyne sec cm⁻⁵/sq.m (p<0.05). In contrast, the patients with cardiac indices of greater than 2.50 L/min/sq.m had a much lower resistance in heart block of 1737 ± 300 dyne sec cm⁻⁵/sq.m (p<0.005), and pacing at maximal output rates increased this insignificantly.

In 3 of the 6 patients studied on subsequent days systemic vascular resistance fell as cardiac index increased (figure 5).

**Pulmonary Arterial Pressure**

The mean pulmonary arterial pressure was elevated above 20mm Hg in 6 of the 12 patients in whom it was measured in heart block (table 2). There was no significant difference in pressures between the patients with cardiac indices in heart block of less than 2.50 L/min/sq.m and those with higher indices. Pacing had no significant effect on the pulmonary arterial pressure whether normal or elevated and there was no significant change in pressure with pacing at different rates.
Right Atrial Pressure

The mean right atrial pressure was elevated above 6 mm Hg in 8 of the 9 patients in whom it was measured in heart block (table 2). The mean atrial pressure averaged $12 \pm 4.3$ mm Hg in 5 patients with elevated pulmonary arterial pressures compared with $8 \pm 4.6$ mm Hg in 4 with normal pulmonary arterial pressures. The elevation of right atrial pressure was always associated with a corresponding elevation in right ventricular end-diastolic pressure. Pacing had no significant effect on right atrial pressure (table 2).

Left Ventricular External Work

Left ventricular external work averaged $0.94 \pm 0.43$ Kg-m/min/sq.m in the 7 patients in whom it was measured in heart block and increased with pacing at maximal output rates to $2.91 \pm 1.90$ Kg-m/min/sq.m ($p<0.02$) (table 2).

Tension Time Index

The tension time index was measured in 9 patients. It increased from a mean of $1308 \pm 528$ mm Hg sec/min in heart block to $6782 \pm 3613$ mm Hg sec/min with pacing at maximal output rates ($p<0.001$) (table 2). The progressive rise in tension time index with increasing rates is shown in figure 7.

DISCUSSION

There is general agreement that ventricular asystole is an indication for pacing in CHB complicating acute myocardial infarction (Faulk and Hurst 1966, Parsonnet et al. 1967, Lassers and Julian 1968). However, the role of pacing in the management of bradycardia is less easy to define (Epstein et al. 1966, Lassers and Julian 1968). This is partly because an alternative treatment in
the form of isoprenaline is available, and partly because there have been no objective measurements of the haemodynamic effects of pacing in this situation. The present study was undertaken to determine the circulatory changes associated with CHB in acute infarction and to evaluate the effect of pacing on these changes.

The cardiac output in CHB complicating acute myocardial infarction was usually, but not invariably, severely reduced. In contrast to chronic CHB (Stack et al. 1958, Bevegard 1962, Escher et al. 1964, Judge et al. 1964, McGregor and Klassen 1964), most patients were unable to increase stroke volume because of depression of myocardial function and could not, therefore, compensate for the bradycardia and maintain an adequate flow. The patient's mental state and skin circulation provided the most useful clinical information about the adequacy of the cardiac output in heart block. Thus 7 patients who had varying degrees of inattention, confusion, restlessness and poor skin circulation had cardiac indices of less than 2.03 L/min/sq.m. In contrast, 4 patients who had nearly normal mental function and good skin circulation had cardiac indices between 2.22 and 3.13 L/min/sq.m. Heart rate was not a reliable guide to the adequacy of the cardiac output. Although patients with rates of less than 45/min usually had low cardiac indices, low values were also found in several patients with rates between 50 and 61/min. Similarly, blood pressure was unreliable in predicting cardiac output. There were, on the one hand, patients with low blood pressures but well maintained cardiac indices, and on the other, patients with relatively well maintained pressures but very low cardiac indices.

The present study demonstrates that cardiac output can be increased in almost all instances by increasing rate by artificial pacing. This response was accompanied by an increase in systemic blood pressure which was progressive
with increasing rates. In those patients in whom the initial cardiac index was low there was a considerable improvement in mental state and skin circulation. The fact that the clinical benefit achieved by pacing continued for many hours suggests that the haemodynamic improvements demonstrated in the acute studies were also maintained. When pacing rates were increased, cardiac index continued to rise until rates between 102 and 120/min were reached. In 2 patients who were studied at 125/min and 130/min respectively, cardiac index had begun to fall. In one patient with cardiogenic shock, cardiac index fell when the rate was increased to 104/min. The rate associated with the maximal output ("optimum rate") was found, therefore, with one exception to be between 102 and 120/min, but as faster rates were not tested in most patients, it might have been higher. This range of optimum rates is considerably higher than the range of 60 to 80/min which has been reported in chronic CHB (Benchimol et al. 1964, Escher et al. 1964, Judge et al. 1964, Segel et al 1964, Sanet and Bernstein 1966, Sowton 1967).

The effect on myocardial function of increasing cardiac output and systemic blood pressure by pacing has not been established. In the patients in whom the stroke index increased or stayed the same in spite of increased rate, it is reasonable to assume that myocardial function was good or had been improved by pacing and that the bradycardia per se had been the major factor in the previously low cardiac index and blood pressure. In most patients, however, stroke index fell in response to pacing in spite of still subnormal cardiac indices and in these patients myocardial performance was probably abnormal. In all instances in which measurements were repeated after the first day of CHB, stroke index had either increased or changed only slightly despite relatively large increases in heart rate, suggesting that myocardial function was
improving with recovery from the infarct.

The loss of atrial transport function which accompanies CHB could be another factor responsible for the inadequate stroke output with increasing rates. In the present study a regular increase and decrease in systemic arterial pressure occurred as atrial systole moved in and out of phase with ventricular diastole. This is almost certainly attributable to an increase and decrease in stroke volume as the result of varying ventricular filling and is also often seen in chronic CHB (Escher et al. 1964, Judge et al. 1964, Samet and Bernstein 1966). In the patients with acute myocardial infarction, these pressure changes were of relatively small magnitude. Moreover, Gillespie et al. (1967) have shown that the augmentation of stroke volume due to optimally timed atrial systole is of much less importance in the diseased than in the normal heart. Although the temporary value of atrial synchronised pacing has been demonstrated in chronic CHB (Samet & Bernstein 1966), this remains to be demonstrated for CHB complicating acute myocardial infarction.

Five patients in the present study had mild elevation of the pulmonary arterial pressure (mean pressure 20 to 31 mm Hg) with marked elevation of the right atrial pressure (mean pressure 12 to 19 mm Hg) and corresponding elevations of the right ventricular end-diastolic pressures. These levels could not be due to volume overload or to the mild pressure overload of the right ventricle and were, therefore, probably due to failure of the right ventricular myocardium secondary to ischaemia. These patients all had inferior myocardial infarctions which are usually due to right coronary artery occlusion (Janes 1962). Since the right coronary coronary artery supplies most of the right ventricle as well as part of the left in most hearts (Hudson 1965), the
predominant involvement of right ventricular myocardium by the occlusion could account for the predominant right ventricular failure.

Although pacing may result in substantial improvement in cardiac output and blood pressure, this is accompanied by a considerable rise in the tension time index implying a corresponding increase in myocardial oxygen requirements (Sarnoff et al. 1958, Rolett et al. 1965). In the normal heart a rise in oxygen demand is met by an increase in coronary flow (Katz and Shaffer, 1966). However, when coronary flow is restricted this may limit myocardial oxygen uptake (Kahler et al. 1963, Weisberg et al. 1963, Bacaner et al. 1965) and "set a limit to the ability of the heart to speed up its rate without suffering a further decline in work capacity" (Katz and Shaffer 1966). This contention is supported by the observation in chronic CHB that a tachycardia induced by pacing whether or not it results in an increase in cardiac output, may produce a decrease in actual myocardial oxygen consumption and an increase in excess myocardial lactate production -- indicating an increase in anaerobic myocardial metabolism (Gregory et al. 1967). In addition it is evidenced clinically by the occasional patient who develops angina with pacing and may explain in part the inadequate stroke output response seen in most cases. On the other hand, it is possible that in some patients pacing may have a favourable effect on myocardial oxygen requirements by decreasing ventricular end-diastolic volume and, therefore, reducing myocardial tension (Sarnoff et al. 1958, Rodbard et al. 1959).

In clinical practice, therefore, the value of improving the systemic circulation by pacing must be balanced against this increased work and oxygen
requirement in deciding which patients should be paced and the best rate for pacing. Patients with normal mental function and good skin circulation usually have adequately maintained cardiac outputs and do not require pacing (Lassers and Julian 1968). When there is depressed mental function and poor skin circulation, however, severe reduction of the cardiac output is invariable and pacing is indicated. In determining the rate at which to pace, it is reasonable to aim for the lower limits of normal for cardiac output. It may be assumed that the cardiac output has reached adequate levels when the blood pressure has done so if this is accompanied by improved mental function and by evidence of increased skin circulation. The provocation of angina or dyspnoea is an indication that the optimum rate has been exceeded. In practice this rate can be determined by clinical trial -- in most cases it will prove to be between 80 to 90/min.

**********
REFERENCES


TABLE 1

CLINICAL DETAILS

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<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
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<th>Rhythm</th>
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<th>Duration of Advanced* Heart Block (hours)</th>
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RVF: Jugular venous pressure elevated above clavicle at 45°
X-ray: Radiographic evidence of pulmonary venous congestion
*Advanced Heart Block: Second and third degree block
NT: Radiograph not taken on day of study
PM: No X-ray taken, but post-mortem evidence of pulmonary congestion

CHB: Complete heart block
SR: Sinus rhythm
AF: Atrial fibrillation
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HB = Heart Block  
PAC = Paced  
* = Rate associated with maximal cardiac index  
RA = Right Atrial  
PA = Pulmonary Arterial  
Q = Clinically not elevated  
+ = Clinically elevated  
(Figures in brackets not included in mean values as complete measurements were not made)
FIGURES
HAEMODYNAMIC CHANGES IN COMPLETE HEART BLOCK
COMPLICATING ACUTE MYOCARDIAL INFARCTION

CARDIAC INDEX
L/min./sq.m

STROKE INDEX
mL/sq.m

MEAN SYSTEMIC ARTERIAL B.P.
mm. Hg.

TENSION TIME INDEX
mm. Hg. sec./min.

Fig. 1—— Effect of pacing at rate associated with maximal measured cardiac output in 11 patients with complete heart block complicating acute myocardial infarction. (mean and standard deviation)
HAEMODYNAMIC CHANGES IN COMPLETE HEART BLOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION

CARDIAC INDEX
1/min./sq.m

STROKE INDEX
ml./sq.m

MEAN SYSTEMIC ARTERIAL B.P. mm.Hg

Fig. 2—Haemodynamic effects of pacing at 83/min for 65 minutes in a patient with complete heart block complicating acute myocardial infarction.
Fig. 3a—Effect of increasing ventricular rate by pacing on cardiac index in complete heart block complicating acute myocardial infarction.
Fig. 3b—Effect of increasing ventricular rate by pacing on mean systemic arterial blood pressure in complete heart block complicating acute myocardial infarction.
Fig. 3c-- Effect of increasing ventricular rate by pacing on stroke index in complete heart block complicating acute myocardial infarction.
Fig. 4—Haemodynamic effects of increasing ventricular rate by pacing in a patient with complete heart block and cardiogenic shock complicating acute myocardial infarction.
Fig. 5—Haemodynamic changes on the first and subsequent days of heart block and after return to sinus rhythm in patients with complete heart block complicating acute myocardial infarction.
Fig. 6-- Haemodynamic effects of increasing ventricular rate by pacing in a patient with complete heart block complicating acute myocardial infarction.
COMPLETE HEART BLOCK COMPICATING ACUTE MYOCARDIAL INFARCTION

EFFECT OF ALTERATION OF VENTRICULAR RATE ON TENSION TIME INDEX

Fig. 7—Effect of increasing ventricular rate by pacing on the tension time index in complete heart block complicating acute myocardial infarction.
ACKNOWLEDGEMENTS

The author wishes to thank Dr. J.L. Anderton without whose help these studies could not have been performed; Dr. M. George and Dr. A.L. Hair for their assistance; and Dr. D.G. Julian, Professor K.W. Donald, Dr. R.M. Marquis and Dr. M.F. Oliver for their advice and criticism. I am also grateful to the Physicians and the medical and nursing staff of the Coronary Care Unit, Royal Infirmary, Edinburgh for their interest and co-operation, and to the technical staff of the University Department of Medicine for their assistance. The British Heart Foundation provided funds for research equipment and Dr. J.L. Anderton was supported by a grant from the Scottish Hospitals Endowment Research Trust.