Extension of Hospital Services into the Community

Former inpatients, day-patients and outpatients, poorly motivated and experiencing adjustment difficulties at home, can be helped by the hospital’s domiciliary workers—the psychiatric nurse, the psychiatric social worker and the occupational therapist, all working in collaboration with the psychiatrist.

The hospital must necessarily have a close link with other community workers and organizations; for example, with district nurses, health visitors, the clergy, school doctors, school psychologists, school nurses, probation officers, and with organizations dealing with alcoholism, child welfare, marriage guidance, the aged, cripple care, the deaf, the blind, and so on. The domiciliary workers of the hospital are in a good position to provide this kind of liaison. The psychiatric nurse can help the patient where psychiatric nursing skills are necessary after discharge from hospital (as well as at the pre-admission level). At the supportive level the psychiatric nurse as well as the psychiatric social worker can, through counselling, help both patient and relatives at pre- and post-hospital phases; furthermore, by providing basic mental health education, they can help families toward understanding and coping with the rehabilitee's symptoms.

The occupational therapist can organize the resources in the home so as to develop a sensible programme for daily living activities, devising work and recreation and affording encouragement and support.

TEMPORARY TRANSVENOUS ENDOCARDIAL PACING IN ACUTE MYOCARDIAL INFARCTION

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Myocardial infarction is the commonest disease encountered in Caucasian communities. It strikes the leaders of society at the peak of their productive life with sudden death or permanent disability. Death is often a result of extensive myocardial necrosis, but many patients die of remediable arrhythmias. If these potentially malignant arrhythmias are treated effectively it is possible to reduce the mortality. Complete heart block occurs in 3-8% of patients with acute myocardial infarction if they are monitored continuously; the mortality ranges from 40 to 100%. Careful postmortem studies have shown that complete heart block may be produced by varying degrees of myocardial damage. The critical area of the atrioventricular node may be the site of extensive infarction producing disruption of A-V conduction or there may be only oedema of this region. Extension disruption is usually associated with a large transmural infarct, whereas transient oedema with paralysis of the node occurs in diaphragmatic infarction with occlusion of the right coronary artery and its branches to the A-V node.

In individual patients the outcome is difficult to predict. Temporary transvenous pacing is now well established as a simple, safe and effective method for treating acute heart block. We have therefore paced an unselected group of patients with acute myocardial infarction who developed complete or high-grade partial heart block.

This report describes 11 such patients who were treated during a 13-month period May 1965 - June 1966. It will be shown that temporary artificial pacing is invaluable and often life-saving in a variety of clinical situations.

METHODS

Patients who sustained a recent myocardial infarction and who developed complete heart block were treated by temporary transvenous endocardial pacing if an experienced physician and suitable laboratory facilities were available. This does not include all patients who were admitted to Groote Schuur Hospital with myocardial infarction and who subsequently developed complete heart block.

A C52-5° bipolar pacemaking electrode wire was passed from an antecubital or from the external jugular vein and was guided and wedged in the trabeculae at the apex of the right ventricle under fluoroscopic control. Pacing was undertaken with a battery-operated external generator with a variable rate and power output. A rate of 80 to 90/min. was chosen, since this degree of tachycardia, probably, was beneficial in a patient with an injured myocardium and a heart deprived of its normal atrial transport function. Patients were then returned to the ward and monitored on a cardioscope. Pacing was continued until electrocardiographic interference between the sinus node and pacemaker indicated a return to sinus rhythm. Pacing was stopped 12 hours later and the wire removed after a further 48 hours if sinus rhythm persisted.

The patients received acute coronary care in the ward. They were nursed in an oxygen tent. A head-down position was used if they were severely shocked. Digitalis, Mersalyl and frusemide were given for heart failure. Shock, hypotension and bradycardia were treated with a continuous intravenous infusion of isoprenaline. The infusion was continued while the pacemaker was inserted, and later if hypotension persisted. Patients were weaned off the isoprenaline support over 24 hours. The best clinical guides of a good response were a warm skin, and a constant urinary output. Subnormal body temperature was a bad prognostic sign.

All patients were given steroids (intravenous hydrocortisone, 100 mg. 6-hourly). Prophylactic antibiotics (cloxacillin) were also administered.

*USCI Catheter Co.
CASE REPORTS

Group 1. Patients with Transient Complete Heart Block in whom Temporary Pacing was Life Saving

(a) Diaphragmatic Infarction

Case 1. E.C., a 60-year-old White male building contractor, was admitted with a 24-hour history of epigastric pain, thought to be due to a perforated peptic ulcer. He then had 3 convulsive episodes and was hypotensive in complete heart block, with episodes of ventricular tachycardia and prolonged asystole. The electrocardiogram confirmed the presence of diaphragmatic infarction. An isoprenaline infusion improved his heart rate and cardiogenic shock. Complete asystole occurred as the external jugular vein was isolated. This responded to external cardiac massage and artificial pacing. Sinus rhythm returned an hour later. The patient has recovered and is now alive and well.

Case 2. J.S., a 52-year-old White male orchestral musician, had been a diabetic since the age of 14 years. He noticed severe precordial pain, felt weak and dizzy and became shocked with a systolic blood pressure of 70 mm.Hg. A metaraminol infusion was started. The electrocardiogram showed a recent diaphragmatic myocardial infarct, with atrial standstill, marked arrhythmia and a ventricular rate of 40/min. Presumably the atrial standstill was a result of ischaemia of the sinus node. A pacemaking wire was inserted; the blood pressure increased to 100 mm.Hg and the patient improved. Sinus rhythm returned 24 hours later. He recovered rapidly and is now at work again.

Case 3. L.C., a 67-year-old retired White clerk, had a three-year history of mild diabetes. One year previously he had a left hemiplegia with complete recovery. He collapsed suddenly with breathlessness and hypotension. A metaraminol infusion was started. The electrocardiogram showed a diaphragmatic infarct. Later there was marked sinus bradycardia with arrhythmia, ventricular premature systoles and paroxysms of atrial tachycardia. He became cold and clammy with intense peripheral vasoconstriction and cyanosis; his blood pressure was 40 mm.Hg systolic; muffled heart sounds and a quadruple rhythm were present. The blood pressure improved on an infusion of isoprenaline. A long PR interval was present on admission; 2nd degree and finally complete heart block then developed, although isoprenaline infusion maintained the ventricular rate at 60/min. A pacemaking wire was inserted and his clinical condition improved dramatically when the heart rate had been increased to 90/min. Sinus rhythm returned after 48 hours. The patient is well and leading a normal life.

In these 3 patients temporary artificial pacemaking was a life-saving procedure during the period of complete heart block. The slow heart rate was unable to maintain a satisfactory cardiac output and pacing improved the output until sinus rhythm returned.

(b) Infarction of Other Sites

Case 4. E.S., a 54-year-old Coloured male motor mechanic, had an extensive anteroseptal infarct. Initially he was shocked and in heart failure. Two days later he developed complete heart block with a ventricular rate of 50/min. He was pale, cold and grey in gross cardiac failure and with a blood pressure of 90/60 mm.Hg. ECG showed anteroseptal infarction with lateral and diaphragmatic extension, and right bundle-branch and complete heart block. Once satisfactory pacing was achieved the patient's mental state improved and his blood pressure increased to 90/70 mm.Hg. Pacing and diuretic therapy produced a good diuresis. Sinus rhythm returned 12 hours later, but evidence of massive infarction remained. Tachycardia (140/min.) and tachyphoea persisted for a week in spite of adequate dietetics.

The patient sustained a large myocardial infarct. Further extension produced transient heart block and the slow ventricular rate precipitated cardiogenic shock. He improved with satisfactory pacing. He is now well and working.

Case 5. W.W., a 55-year-old White male, was admitted to hospital after 2 episodes of syncope. On admission, he was in complete heart block with a pulse rate of 30/min. ECG showed complete right bundle-branch block with high lateral myocardial infarction. There were intermittent episodes of ventricular asystole, each attack responded to thumping of the chest. A transvenous endocardial pacemaking wire was inserted to prevent the episodes of ventricular asystole since oral isoprenaline, ephedrine and atropine failed.

The patient returned to sinus rhythm after a few hours and further episodes did not occur. He is now well without a pacemaker or other treatment.

Pacing was used in this patient to prevent asystole and episodes of syncope.

Group 2. Survivors with Persistent Heart Block

Case 6. C.S., a 59-year-old White male commercial traveller, had two episodes of syncope. This was followed by retrosternal pain, dyspnoea, sweating and heart failure. The electrocardiogram showed complete heart block with a ventricular rate of 50/min. and a recent diaphragmatic infarct. A temporary pacemaking wire was inserted. The patient improved. Three days later 2:1 heart block was observed and sinus rhythm returned after a further 2 days. He was discharged home after 3 weeks. One month later he became short of breath and felt giddy while walking. He was in complete heart block again and developed frank cardiac failure. A permanent transvenous endocardial pacemaking wire with an implanted auxiliary generator was inserted. Eight months later the unit failed when the wire fractured in the neck. A new endocardial pacemaking wire was inserted via the jugular vein, passed subcutaneously and exteriorized in the flank before inserting a new generator. While under surveillance in hospital he had a fresh anteroseptal infarct. This was masked on ECG by the presence of a left bundle-branch block pattern produced by right ventricular stimulation for pacing. The new permanent generator was implanted after 3 weeks, but during this interval the patient returned to sinus rhythm for a short period. The patient is now working.

Here the temporary pacemaking wire was inserted to control heart failure and prevent Stokes-Adams attacks. Later when complete block returned a permanent unit was implanted to treat heart failure.

Case 7. H.G., a 63-year-old White male, a ship's captain, was a known hypertensive subject. He developed precordial pain, and was found to have endocarditis. A diaphragmatic myocardial infarct with partial heart block. There were no further symptoms and 2 days later atrial flutter occurred with right bundle-branch block, complete heart block and a ventricular rate of 40/min. He was mildly shocked, but not hypotensive. Heart failure developed. A transvenous endocardial pacemaking wire was inserted and temporary pacing continued for a week. Interference was observed on the electrocardiogram and pacing discontinued. A ventricular rate of 60/min. was now present. Cardioversion restored a normal atrial rhythm, but complete heart block persisted with a ventricular rate of 60/min. He was discharged home in complete heart block since he was not disabled with the relatively rapid heart rate and wished to return to England.

In this patient artificial pacing improved heart failure.

Group 3. Prophylaxis in the Presence of Partial Heart Block

Case 8. D.M., a 40-year-old White male artist, complained of 3 episodes of retrosternal pain with extreme shortness of breath. He was dyspnoeic, in heart failure, and though hypertensive previously, his blood pressure had fallen to 135/85 mm.Hg. The electrocardiogram showed a recent diaphragmatic myocardial infarct with 2:1 heart block. A pacemaking wire was inserted, but the cardiac rhythm changed to partial heart block with Wenckebach phenomenon. Pacing was not required.

The heart failure responded to digoxin and Mersalyl. Normal atrioventricular conduction was restored and the wire was removed 48 hours later. The patient is now back at work, in sinus rhythm, but has a persistent third heart sound and is still hypertensive (160/120 mm.Hg).
In this patient satisfactory conduction was present during the period of heart block and the pacemaking wire was a useful prophylactic measure.

**Group 4. Deaths**

**(a) Diaphragmatic Infarction**

*Case 9.* E.B., a 54-year-old White housewife, had severe chronic bronchitis. She complained of precordial pain and collapsed. She was in gross cardiac failure; blood pressure 120/90 mm Hg and with cold, clubbed extremities and a heart rate of 40/min. An electrocardiogram showed complete heart block with recent diaphragmatic infarction and lateral extension. A pacemaking wire was inserted. Her clinical condition improved, but the pacemaker failed after 4 hours and the position of the tip of the electrode wire had to be altered. After this she remained hypotensive and needed larger doses of intravenous isoprenaline. Acute dilatation of the stomach occurred and she died.

Necropsy showed that death was due to extensive diaphragmatic infarction also involving the entire ventricular septum. The infarcted area had probably extended when pacing failed, suggesting that the infarcted segment was insensitive to endocardial stimulation. Death occurred from extensive death of heart muscle.

*Case 10.* P. de V., a 50-year-old White executive, developed acute retrosternal chest pain while fishing. The pain recurred later and he became hypotensive and shocked. An electrocardiogram showed diaphragmatic infarction with complete heart block. A transvenous endocardial pacemaking wire was inserted and the patient's clinical condition improved dramatically; he became talkative and his sweating disappeared. Two hours later he developed sudden ventricular fibrillation. Adequate facilities for resuscitation were not available and he died because of delay in providing appropriate treatment.

**(b) Infarction in Other Sites**

*Case 11.* F.S., a retired, 72-year-old, Coloured male lorry driver, felt weak for 7 days and then collapsed. He was shocked, and the femoral, but not the brachial and radial pulses were palpable; the heart sounds were distant. The electrocardiogram showed complete heart block with a ventricular rate of 57/min., left axis deviation and left bundle-branch block. A ventricular ectopic beat in V2 showed the q wave and ST elevation of anteroseptal infarction.

An isoprenaline infusion was started and a transvenous endocardial pacemaking wire inserted. With a paced ventricular rate of 90/min., the previously stuporous patient was able to reply to questions and take an interest in his surroundings. The radial and brachial pulses remained impalpable and the hypotension and shock persisted although intravenous isoprenaline, digoxin and hydrocortisone were given and cerebrovascular perfusion was adequate.

He died suddenly 4 hours after pacemaker insertion, presumably as a result of sustained hypotension with poor coronary perfusion. The ventricular response to pacing faded over a period of 5 minutes. At necropsy, there was extensive diaphragmatic, septal and anterior myocardial infarction.

Initially this patient had an anteroseptal infarct with obstruction to the anterior descending branch of the left coronary artery. A few days later the right coronary artery was obstructed, producing a diaphragmatic infarct and complete heart block. Poor left ventricular function and a slow heart rate were responsible for severe cardiogenic shock. Pacing improved the cardiac output, but this was still inadequate and he died. This patient demonstrated that if artificial pacing and isoprenaline were unable to maintain a satisfactory cardiac output extensive muscle necrosis probably was present and a poor prognosis could be predicted.

**Discussion**

The clinical results in our patients were gratifying. Three of 10 patients in complete heart block died; a mortality rate of 30%. The effectiveness of a new form of treatment can be assessed only if the natural history of the condition untreated is known. In uncomplicated, acute myocardial infarction, the mortality varies from 20 to 30%. The mortality is increased by the presence of associated heart block and the prognosis in 6 groups of patients are given in Table I. The mortality ranged from 30 to 70% and Mintz and Katz reported a figure of 100%. Paulk and Hurst had a mortality rate of 44%. The most important factor in anticipating the outcome is the nature of the underlying pathology in the pathogenesis of block. The clinical implications and prognosis vary considerably and a suggested classification is given in Table II.

**Table I. Clinical Implications of the Pathogenesis of Complete Heart Block in Acute Myocardial Infarction**

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Prognosis</th>
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</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Good</td>
</tr>
<tr>
<td>1. Obstruction of right coronary artery or branch to atrioventricular node</td>
<td>Poor</td>
</tr>
<tr>
<td>2. Extensive infarction with disruption of the interventricular septum</td>
<td>Good</td>
</tr>
<tr>
<td>Subacute</td>
<td>Good oedema of the A-V node or conducting tissue</td>
</tr>
<tr>
<td>1. Ischaemia with reactionary hypoxic death of conducting tissue</td>
<td>Poor</td>
</tr>
<tr>
<td>2. Persistent hypotension or Bad shock with subnormal perfusion or hypoxia of the junctional tissues</td>
<td>Good</td>
</tr>
<tr>
<td>Chronic</td>
<td>Good tissue only with subsequent reactionary fibrosis</td>
</tr>
</tbody>
</table>

Heart block may occur in diaphragmatic infarction as a result of obstruction to the right coronary artery or the branch to the A-V nodal tissue with ischaemia or reactionary oedema of the region; often there is death of a small area of muscle only and the prognosis is excellent. In anterior infarction the A-V node is rarely affected and complete heart block is associated with extensive disruption of the ventricular septum. The prognosis is grave. Our patients show that there are notable exceptions. One patient with a diaphragmatic infarct died, and at postmortem the extensive muscle damage involving the diaphragmatic surface of the left ventricle and the major portion of the ventricular septum. Two patients with extensive anterior infarction survived. Thus complete heart block associated with diaphragmatic infarction can occur with extensive muscle damage, while patients with extensive anterior infarction can be salvaged. Heart block may also be an agonal event.
Complete heart block, therefore, is a potentially dangerous complication of myocardial infarction. Cardiac function is already compromised by an area of dead muscle with surrounding oedema. Although the heart rate is often 50-60 beats per minute, bradycardia, and lack of an efficient atrial transport mechanism, further impairs cardiac function. In addition the heart may be unable to increase its stroke volume suddenly to compensate for the slow heart rate, a clinical phenomenon frequently observed when elderly patients first develop complete heart block. Moreover they are liable to sudden ventricular asystole or fibrillation. Our patients highlight the deleterious effects of complete block; cardiac failure occurred in 10, hypotension and cardiogenic shock in 7, and episodes of syncope in 3 cases.

In the majority of patients complete block is a transient event occurring at the onset of fresh infarction or 24 - 48 hours later. Only three of our patients were in complete block when first seen; the remainder developed block after 24 - 48 hours and more than half had antecedent first and second degree block. The heart block was of short duration (30 mins. to 96 hours) and complete recovery occurred in all but 2 patients. During this period of block, there was always clinical evidence of a low cardiac output and this improved with artificial pacing. One patient with persistent complete block required long-term artificial pacing.

Paulk and Hurst have reviewed the potential hazards and difficulties of artificial pacing in this group of patients; many were seriously ill at the time of insertion of the pacemaking wire electrode. In our own limited experience we have had no major problems. Acutely ill patients were moved to the catheterization laboratory for pacemaker insertion. In the presence of shock or heart failure they were given additional intravenous isoprenaline infusions. In 9 of the patients, the pacemaker wire was passed from an antecubital vein and in two from the external jugular vein. There were no technical difficulties. Ventricular premature systoles occurred when the wire was passed into the right ventricle. At this stage two patients developed asystole which responded to a thump on the chest. Once artificial pacing was started, the response to the increase in heart rate was dramatic.

Late complications of pacing were rare. Sepsis did not occur, but all the patients were given prophylactic antibiotics. Pacing failed in one patient when an area of myocardium became insensitive. In the other patient ventricular fibrillation occurred. Unfortunately resuscitation was delayed and this patient died. Fibrillation, probably, was due to an inherent irritability of the ventricle and not a complication of artificial pacing.

We have preferred a heart rate of 80 - 90 per minute on an empirical basis assuming that the heart with compromised function needed a slight tachycardia. Steroids were also given to all patients in view of previous enthusiastic reports. Natural recovery frequently occurred without treatment and block soon disappeared if the patient survived the acute infarct. This interval was synchronous with clearing of the inflammatory reaction at the site of infarction. They suggested that the transient block frequently was due to inflammatory reaction and not to actual infarction. Steroids reduced the duration of block by improving the inflammatory reaction. This occurred in our patients, but still did not avoid the short period of block which required artificial pacing.

Artificial pacing, therefore, was invaluable in treating patients with acute myocardial infarction and complete heart block. This was often associated with hypotension, cardiogenic shock and heart failure. In the majority of patients the heart block was transient and if the patient survived, the ultimate prognosis was good. Our patients showed a dramatic response to an artificial increase in heart rate. If the clinical response to pacing was poor—persistent hypotension or lack of response to an infusion of isoprenaline, subnormal temperature, cold skin, peripheral cyanosis and oliguria—a fatal outcome could be predicted. There were no serious complications of artificial pacing and the procedure was well tolerated.

It is now our policy to insert a pacemaker electrode wire in two groups of patients with acute myocardial infarction. The first group consists of patients who are admitted to hospital with complete or high-grade partial heart block or who develop this while under observation. The second group consists of patients with partial heart block since they often develop complete block later. A prophylactic pacemaking wire is inserted, but pacing is withheld unless complete or high-grade partial block develops.

We have also had experience with a third group of patients who were not included in this report. These patients were moribund after a prolonged period of hypotension or had undergone successful defibrillation. Slow nodal rhythm was usually present and the outcome was fatal in spite of heroic resuscitation and artificial pacing. We now regard this as a 'dying-heart syndrome', and the slow nodal rhythm as a manifestation of extensive myocardial damage after exposure to prolonged hypotension, hypoxia and acidosis. Until suitable artificial circulatory support is available, poor results can be anticipated from pacing these patients.

SUMMARY

Temporary transvenous endocardial pacing was undertaken in 11 patients in whom complete or high-grade partial heart block was associated with acute myocardial infarction. Seven had diaphragmatic infarction. In 8, pacing was undertaken as an acute emergency life-saving procedure. Seven were acutely shocked and hypotensive and 10 were in heart failure at the time of pacemaker insertion.

There were no technical problems associated with pacemaker insertion. One patient stopped pacing due to extension of the area of infarction and stable pacing was achieved when the wire was repositioned.

In all the patients dramatic clinical improvement occurred when the heart rate was increased by artificial pacing. Three patients died from extensive myocardial infarction. It is unlikely that artificial pacing contributed towards death. Two survivors have persistent complete heart block and one has needed permanent artificial pacing.

The technique of pacemaker insertion is simple and life-saving for patients with acute myocardial infarction in whom transient bradycardia may cause death.
INCIDENCE OF DIABETES MELLIITUS IN THE PERI-URBAN BANTU:
ANTE-NATAL SURVEYS

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In studies of glycosuria among the rural South African Bantu, it is generally agreed that the incidence of glycosuria is rare when compared with White groups. Assessments made among the Bantu in Southern Rhodesia and the former territory of Nyasaland confirm this view (Table I).

Many surveys have been published which appear to indicate that, as the Bantu moves into the White man's environment and consumes Westernized foods, the prevalence of diabetes rises and in time may approach that of Whites.

Campbell found a great increase in diabetes in the Zulu attending his diabetic clinic, which he attributed to the extra sugar consumption of these workers who had moved from rural surroundings to work in the cane-sugar areas of Natal.

The statistical evidence from various surveys in Bantu urban areas is confusing. The figures provided by hospital diabetic clinics or outpatient departments are obtained from variable populations, as the areas served by a hospital cannot be clearly defined, and furthermore, cases with the disease, referred by general practitioners and clinics, would tend to increase the incidence.

To obviate these difficulties, in the absence of universal surveys, the sample survey of the antenatal rate of glycosuria would give some indication of the incidence of this disease. In such an assessment both the population type and the numerical value of the sample is fixed and the age-group defined within limits. Extrapolation to the whole population is of course invalid as the number of older diabetics cannot be assessed. Here again, complex factors other than diet play a part in the aetiology.

There is evidence to suggest that pregnancy evokes diabetes. However, Bland, in an antenatal survey carried out in Southern Rhodesia, found only one diabetic in a sample of 15,521. At Queen Charlotte's Maternity Hospital in London between 1944 and 1953, 0.005% of the antenatal cases were found to be suffering from diabetes—a figure below that of the general population. These statistics do not show the pre-diabetic state as there is no evidence that blood-sugar estimations were done on the whole sample.

Two independent studies were carried out, one at Meadowlands and one at the Moroka Clinic.

1. THE MEADOWLANDS SURVEY—ANTE-NATAL CASES

Meadowlands is a Bantu peri-urban community. The present population consists of the following ethnic groups: Sotho, Nguni (Zulu, Xosa, Swazi), Venda and Shangaan. The Sotho and Nguni are rapidly approaching Western standards of dress, diet and ways of life. Many of the women attending this antenatal clinic are employed in the domestic service of Whites where, in part at least, they partake of Western-type food. The Venda and Shangaan, especially the women, are still very primitive in their outlook and customs and have not adapted themselves to urban society. Many women spend periods in urban areas, often for the purpose of having a confinement, and then return to their homes where they live under the most primitive conditions.

Subjects and Methods

A total of 1,716 antenatal patients were investigated for glycosuria during pregnancy, their ages varying between 15 and 49 years, the average patient attending the clinic at least 4 times during gestation; in most cases the patients were followed-up to term. Postprandial urine specimens were tested by trained Bantu nurses using glucose oxidase paper (Tes-tape). If glycosuria was discovered a postpran-