British Heart Journal, 1971, 33, 595–600.

Conservative treatment of AV block in acute myocardial infarction
Results in 105 consecutive patients

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Four years' results of a prospective study on treatment of AV block in acute myocardial infarction are reported. Second and third degree AV block occurred in 105 of 1665 patients. Isoprenaline intravenously was used when clinically necessary and pacemaker only if isoprenaline failed to improve heart rate. Forty-four patients received no specific therapy, 47 received isoprenaline only, and 14 transvenous pacemaker therapy. Few complications of isoprenaline therapy were observed, which is ascribed to relatively small doses and careful adjustment if necessary. The overall mortality was 47 per cent. Though the results did not differ significantly from those in compiled reports on routine use of pacemaker they show that AV block in acute myocardial infarction carries a high mortality despite conventional drug therapy. An analysis of the causes showed that associated complications of a large infarction are probably often implicated, but also that refined pacemaker methods combined with drugs may offer hope of better results.

Acute myocardial infarction complicated by AV block carries a high mortality (Epstein *et al.*, 1966). Most authors (Gregory and Grace, 1968; Kimball and Killip, 1968; Sutton, Chatterjee, and Leatham, 1968; Beregovich *et al.*, 1969; Grendahl and Sivertssen, 1969) suggest routine use of temporary pacemaker in second or third degree AV block. Scott *et al.* (1967) used either isoprenaline or pacemaker and recommended pacemaker when there was congestive heart failure, shock, or syncope.

Our purpose is to report on a prospective study on four years' use of isoprenaline when clinically necessary and pacemaker if isoprenaline failed. This approach was chosen as we were not convinced that the routine use of the pacemaker was preferable, particularly in a hospital without university facilities, even if pacemaker therapy of chronic AV block were currently applied.

Patients and methods

Table 1 shows that 105 of 1665 patients with acute myocardial infarction admitted from January 1966 to March 1970 had second or third degree AV block, a mean frequency of 6·3 per cent. The increasing numbers of block are probably due to better monitoring, after a four-bed coronary care unit was established during 1967–1968. Our medical department has 200 beds, however, and monitoring was only possible in about a third of the patients with acute infarction. Block of short duration after resuscitation may be due to metabolic acidosis (Stewart *et al.*, 1965) and has been excluded. Otherwise, all patients were included, as well as those who died before therapy could be given. The mean age was 66 years. The time interval from the onset of pain to admission was a quarter of an hour to 4 days, on average 13 hours, and most often 6 to 8 hours. Shock (peripheral vasoconstriction, cerebral symptoms, a systolic blood pressure less than 90, and oliguria) and/or congestive heart failure (pulmonary oedema) occurred in 69 patients.

An intravenous 5 per cent glucose drip was established. If heart rate fell below 50, with hypotension, shock, congestive heart failure, ventricular arrhythmias, or Adams-Stokes attacks, isoprenaline was given as well, 4 mg/l., 1 μg/min. The dose was carefully increased if necessary and varied from 1 to 16, usually 1 to 2, and rarely more than 3 μg/min. The duration of therapy was 1 to 14 days, usually 4 days. Isoprenaline alone was given to 47 patients.

If isoprenaline failed to improve heart rate, an Elema unipolar or a Medtronic bipolar electrode was inserted transvenously under fluoroscopy to the right ventricle. An external fixed rate pacemaker was used in 6 patients, and later a QRS inhibited Medtronic pacemaker in 8. The electrodes were kept in place for three weeks in the surviving patients. Nine patients died so rapidly that there was no time for effective therapy. In 3

Received 8 October 1970.

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of them a transthoracal pacemaker was attempted with electrical but no mechanical heart activity.

Treatment of congestive heart failure consisted of low salt diet, bed rest, and diuretics, and did not include digitalis routinely unless a satisfactory heart rate (above 70) was achieved. Intravenous lignocaine was used in addition to isoprenaline or pacemaker if frequent ventricular extrasystoles developed.

Results

The overall mortality was 47·6 per cent, while 21 of 30 women died compared to 29 of 75 men. The mean age, however, in the former was 72 years and 63 years in the latter. Fig. 1 shows that mortality increased with age. Among 71 patients with first infarcts 39 per cent died as against 65 per cent of 34 patients with previous infarcts.

Second degree AV block occurred in 32 patients, 25 with Mobitz's type 1 (Wenckebach) had a mortality of 28 per cent, and 7 with Mobitz's type II all died. Third degree block was found in 73 patients. The mortality in 38 with a QRS complex less than 0·12 sec was 29 per cent, and in 35 with QRS over 0·12, 71 per cent. Gradual development from first, second, to third degree block with narrow QRS was associated with a good prognosis: of 17 patients only 1 died from ventricular fibrillation on the fifth day.

Table 2 shows the site of infarction. Adams-Stokes attacks occurred in 19 of 42 patients with bundle-branch block, being the first sign of AV block in 12. Among 63 patients with narrow QRS only 7 had syncope which was never the initial sign.

Fig. 2 shows that mortality was highest when block developed early. AV block diagnosed after two days was most often of Wenckebach's type associated with inferior infarction and narrow QRS complex, or occurred in anterior infarction with bundle-branch block. There was no relation between mortality and initial heart rate.

Among 69 patients with shock and/or congestive failure 41 died as against only 9 of the remaining 36. Shock was present in 33 patients, 23 having in addition pulmonary oedema. Only one of the latter survived, a woman of 56 who was treated with isoprenaline. AV block disappeared after 3 days, but for another 10 days her blood pressure rapidly fell if isoprenaline was temporarily discontinued. On admission 14 patients were in shock and AV block may have been secondary.

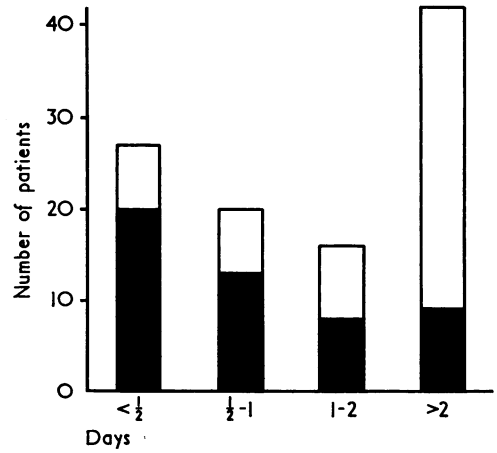

FIG. 2 *Time from onset of infarction to development of AV block. Number of patients dying is indicated by the shaded areas.*

FIG. 1 *Age distribution. Number of patients dying is indicated by the shaded areas.*

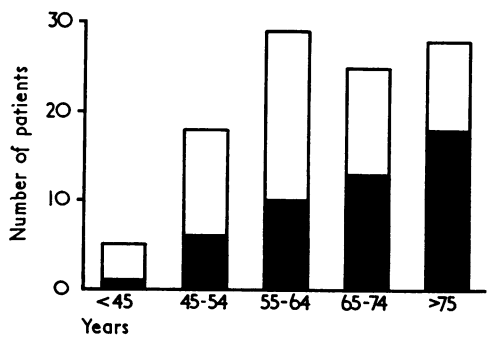

TABLE 1 *Frequency of second and third degree AV block and acute myocardial infarction*

	1966	*1967*	*1968*	*1969*	*1970*	*Total*
No. of infarcts	388	369	404	426	78	1665
No. with AV block	16	21	24	37	7	105
Per cent	4·1	5·7	5·9	8·7	9	6·3

TABLE 2 *Site of infarction, Adams-Stokes attacks, and mortality*

Site of infarction	*No. of patients*	*Adams-Stokes attacks*	*No. of deaths*
Inferior	62	11 (7)	23
Anterior	28	12 (9)	19
Anterior and inferior	5	1 (1)	4
Unknown	10	2 (1)	4
Total	105	26 (18)	50

Survivals in parentheses.

Eleven of them died during the first few hours, one on the 2nd and one on the 30th day, while one survived. In 7 patients AV block occurred after admission and after shock had developed. All of them died within a few hours. Of the 4 patients whose AV block occurred after ventricular fibrillation and persisted from hours to days, only 1 survived, 2 died from ruptured myocardium, and 1 from congestive heart failure.

Ventricular tachycardia occurred in 7 patients, only 1 having block and 6 sinus rhythm. Ventricular fibrillation occurred in 16 patients (5 during the first two days, 6 others in the first week, and 5 from 2 to 4 weeks after onset of symptoms). Five had block and 11 had reverted to sinus rhythm. Five of them received isoprenaline with addition of lignocaine if frequent extrasystoles were present. Only one had another attack of fibrillation which did not recur after increasing the lignocaine dose.

As seen in Table 3, shock and/or heart failure were the most frequent causes of death. It is noted that 14 patients died suddenly, 6 in ventricular fibrillation and 5, with previous bundle-branch block, of asystole, while 3 patients were not monitored. Necropsy in 30 patients showed ruptured myocardium in 5.

The duration of block in the 78 patients who reverted to sinus rhythm was a few hours to 2 days in 39, 3 to 7 days in 31, and more than one week in 8. In 4 patients AV block recurred after 2, 2, and 8 days, and in the fourth, several times for up to 6 weeks. Three patients were discharged from hospital with total AV block. A permanent pacemaker was implanted in one while the two others using isoprenaline sublingually were free of symptoms.

Treatment groups Forty-four patients received *no specific therapy*, 35 because of lack of symptoms and 9 because of rapid death. Among the former, 8 died, 4 of congestive failure after 15 to 31 days, 1 had a fresh infarct on the 25th day, 1 had ventricular fibrillation on the 5th day, and 2 without monitoring died suddenly on the 6th and 9th days. Sinus rhythm was present in 7 and persisting AV block in 1 patient.

TABLE 3 *Causes of death*

	Total	*QRS* <0·12	*QRS* >0·12
Shock/heart failure	30	9	21
Ventricular fibrillation	6	4	2
Asystole	5	0	5
'Sudden death'*	3	1	2
Rupture	5	3	2
Other	1	1	0
Total	50	18	32

* Not monitored.

Isoprenaline caused an increase in heart rate/blood pressure and/or terminated further Adams-Stokes attacks in 38 patients, an increase in heart rate but only a temporary increase in blood pressure in 12, a slight rise in heart rate only in 8, and in 2 had to be terminated because of increasing ventricular extrasystoles. The average heart rate was 73, being less than 70 in only 4 patients (58, 60, 64, 64). In 5 patients immediate conversion to sinus rhythm occurred.

Complications in the *isoprenaline treated* group were few. Among 8 patients with ventricular fibrillation in this group, 4 had the arrhythmia before AV block and before isoprenaline was given, and in 2 it occurred after isoprenaline had been discontinued (for 2 and 5 days). Fibrillation during isoprenaline therapy occurred in 3 patients, in 2 while the dose was rapidly increased because of falling blood pressure. One of the latter had had fibrillation before AV block. Cardioversion was successful in both, and isoprenaline drip was continued with addition of lignocaine without further complications. The third patient had shock and died in asystole after defibrillation.

The transvenous *pacemaker* was used because of further Adams-Stokes attacks in 3 patients, persistent hypotension, or congestive heart failure with bradycardia or alternating bradycardia/tachycardia in 8, and increase of ventricular extrasystoles during isoprenaline in 2. One was given a pacemaker without attempting isoprenaline.

Complications during pacemaker therapy In one patient ventricular fibrillation occurred repeatedly as soon as the electrode entered the right ventricle. It was therefore placed in the right atrium and pacing was not established. On the fifth day he died of recurrent fibrillation, necropsy showing that the electrode tip had entered the right ventricle. Mechanical irritation might therefore have been the cause of death. This was the single patient who died among the 17 with inferior infarction, normal QRS, and gradual development of AV block. A second patient with a demand pacemaker was in good condition with sinus rhythm until on the eighth day she developed increasing ventricular extrasystoles, and despite treatment with lignocaine, died of

recurrent ventricular tachycardia/fibrillation. Turning the pacemaker off or increasing the rate up to 120 a minute had no effect. Electrocardiogram strips taken when ventricular tachycardia developed did not suggest interference which may also occur with a demand pacemaker by low endocardial potentials. Mechanical irritation must therefore also be kept in mind in this case. In 2 patients with fixed rate pacemaker ventricular fibrillation developed after 7 and 18 days, in the latter preceded by interference phenomena. The first patient was converted to sinus rhythm but died two hours later with asystole. The pacemaker system was intact. In one patient pacemaker failure developed after one day and the patient died rapidly in shock. The electrode was found to be defective.

The causes of death in the various treatment groups are set out in Table 4. Ventricular fibrillation was relatively frequent in patients treated with pacemaker. From Table 5 it is apparent that the few survivors among those having bundle-branch block were approximately equally distributed between the three treatment groups. Fig. 3 shows that a large proportion of patients dying after the first 2 days had reverted to sinus rhythm, which indicates that complications other than AV block influence mortality.

Late results A second infarction occurred in 8 of the surviving patients. Temporary AV block reappeared in one, a second died with asystole without preceding AV block, and 6 patients had sinus rhythm.

FIG. 3 *Time interval from admission to death in 50 patients. Number of patients reverting to sinus rhythm is indicated by the hatched areas.*

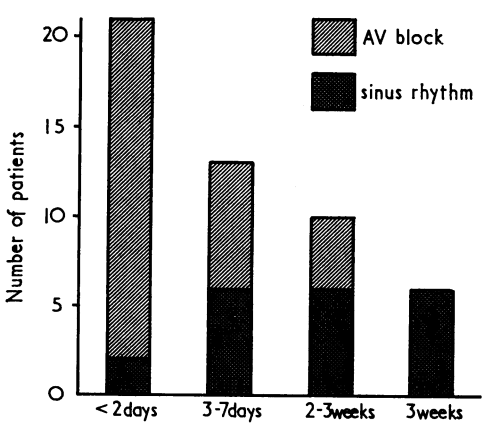

TABLE 4 *Causes of death in various treatment groups*

	Total	*No special treatment (44 patients)*	*Isoprenaline (47 patients)*	*Pacemaker (14 patients)*
Shock/heart failure	30	10	17	3
Ventricular fibrillation	6	2	1	3
Asystole	5	2	1	2
'Sudden death'*	3	2	1	0
Other	6	1	4	1
Total	50	17	24	9

* Not monitored.

TABLE 5 *Patients with bundle-branch block*

	Total	*No specific treatment*	*Isoprenaline*	*Pacemaker*
Inferior infarction	12 (2)	6 (1)	5 (0)	1 (1)
Anterior infarction	19 (3)	4 (1)	7 (1)	8 (1)
Anterior and inferior	5 (1)	2 (1)	2 (0)	1 (0)
Site unknown	6 (4)	2 (1)	4 (3)	
Total	42 (10)	14 (4)	18 (4)	10 (2)

Survivals in parentheses.

Comments

Our study shows the well-known high mortality associated with bundle-branch block, advanced age, previous infarcts, shock, and congestive heart failure. Necropsy in patients with inferior infarction and bundle-branch block often showed involvement also of the septum and sometimes of the anterior wall. The prognosis in patients with inferior infarction and narrow QRS complexes was good. Few had Adams-Stokes attacks and none died in asystole. Our results support those of Scott *et al.* (1967) who found a higher mortality when AV block occurred early after onset of symptoms, while Sutton *et al.* (1968) found that the time interval was of no importance. Adgey *et al.* (1968) described a high frequency of early, transitory bradycardia/AV block, sometimes disappearing after atropine, suggesting a vagus mechanism as the most important factor. It is noted that most of our patients were admitted several hours after the initial symptoms of infarction and that AV block in inferior infarction usually occurred after 2 to 3 days in hospital.

A comparison with reports on routine pacing is difficult, often due to incomplete information on the various clinical factors. We have, however, compiled 11 reports on routine pacemaker therapy including 334 patients (Epstein *et al.*, 1966; Harris and Bluestone, 1966; Paulk and Hurst, 1966; Parsonnet *et*

al., 1967; Scott *et al.*, 1967; Gregory and Grace, 1968; Lassers and Julian, 1968; Sutton *et al.*, 1968; Beregovich *et al.*, 1969; Grendahl and Sivertssen, 1969; Godman, Lassers, and Julian, 1970). Sex and age were comparable with our material. The mean mortality was 44 per cent. Information on bundle-branch block was given in 231 patients. The mortality in 98 with bundle-branch block was 72 per cent, and in 133 with narrow QRS, 25·5 per cent. Few authors state whether they also included patients who died before therapy could be given. Lassers and Julian (1968) included all patients and found a mortality of 47 per cent. Excluding the above cases, Beregovich *et al.* (1969) found a mortality of 36 per cent in 25 patients. There appears to be no significant difference between our results and compiled results on the routine use of the pacemaker. Some newer reports (Skjaeggestad *et al.*, 1970) suggest a somewhat lower mortality (38%) by extensive use of demand pacemaker. Chatterjee, Harris, and Leatham (1969) reported a strikingly low mortality of 11 per cent in 27 patients by the combination of demand pacemaker and the liberal use of suppressant drugs.

We have analysed the causes of death in our patients to find if better results could have been obtained. The reasons for using a pacemaker are to prevent asystole and to increase heart rate and thus improve circulation or prevent ventricular arrhythmias. A pacemaker is possibly the best method to avoid asystole. Isoprenaline, however, was successful in preventing further syncopes in 14 of the 19 who had recurrent Adams-Stokes attacks. Of the remaining 5, one reverted to sinus rhythm with a rate of 80 a minute, but was still in deep shock and died in asystole, 3 were treated with transvenous pacemaker (one of them survived), and in the fifth a transthoracal pacemaker was ineffectual.

Asystole occurred despite pacing in 2 patients. The first died suddenly on the 21st day, necropsy showing anterior wall infarction and a fresh thrombus in the right coronary artery. The second developed asystole 2 hours after successful conversion of ventricular fibrillation. Necropsy showed extensive infarction. The electrode tip in both was found intact in the right ventricular apex. Asystole was frequent and early in patients with block of Mobitz type 2, but attempts to reduce the mortality with a prophylactic pacemaker in sinus rhythm and bundle-branch block have not shown less mortality when AV block develops (Godman *et al.*, 1970). Our main impression is that isoprenaline can usually prevent Adams-Stokes attacks. In 2 patients who were not monitored and died suddenly the use of a pacemaker might have given better results.

Of the 30 patients who died of shock or failure, 13 had AV block and had been treated with isoprenaline; the remaining 7 had pacemakers or were in sinus rhythm. In some of them a further increase in heart rate than that obtained by isoprenaline in moderate doses might have been beneficial. Pacing might have relieved circulatory failure and thereby reduced a possible extension of the infarction. Pacing would also have permitted a more liberal use of digitalis. Bruce *et al.* (1965) described clinical improvement in patients with shock or failure by increasing the pacing rate above 70 to 80. The optimal heart rate, however, may vary with the state of ventricular function (Siddons and Sowton, 1967), and is difficult to ascertain in these conditions. Lassers *et al.* (1968) found maximal cardiac output at a pacing rate of 90 to 100 in acute infarction with AV block. But a lower rate may be 'optimal', giving sufficient increase in cardiac output/blood pressure without too high an increase in myocardial oxygen demand with possible detrimental effect. On the other hand, increased cardiac output/blood pressure is beneficial in augmenting myocardial blood supply. Similar reasoning applies to isoprenaline which in addition has an ionotrophic effect. High doses which increase oxygen demand should probably be avoided. One patient received a very large amount, 40 μg/min, because of frequent Adams-Stokes attacks with convulsions which delayed pacemaker electrode insertion. The procedure was ultimately accomplished, but death from shock and asystole occurred after one and a half days. Necropsy showed extensive inferior, septal, and partly anterior infarction with multiple small haemorrhages, possibly due to the very large dose of isoprenaline and/or to repeated convulsions.

Does prolonged isoprenaline therapy, even in small doses, have an unfavourable effect? A quite high percentage of our patients died with congestive heart failure. But the average isoprenaline dose and increase in heart rate were approximately the same in those who survived. Furthermore, deaths due to failure were least frequent in the isoprenaline treated group.

Morse, Danzig, and Swan (1967) reported a favourable effect of isoprenaline in cardiogenic shock, while Gunnar *et al.* (1967) described a fall of blood pressure. The latter used larger doses, 1–7 μg/min, than the former, 0·5–3 μg/min. Our doses of isoprenaline were low, and falling blood pressure was not ob-

served. On the contrary, shock in 2 patients persisting after pacemaker therapy disappeared when isoprenaline was added.

Sutton *et al.* (1968) suggest a high frequency of ventricular fibrillation in patients with AV block, which accords with our study (15%). Scott *et al.* (1967) rarely observed fibrillation after the first two days while two-thirds of our patients had their attacks after the first two days. Ventricular fibrillation in our patients was usually unrelated to the presence of AV block or therapy. In our patients treated with isoprenaline, there was no increased frequency of ventricular fibrillation. Grendahl and Sivertssen (1969) reported occasional ventricular fibrillation with fixed rate, but rarely by using a demand pacemaker. Chatterjee *et al.* (1969) noted a high frequency of ventricular arrhythmias even with demand pacing but reduced the risk considerably by adding suppressant drugs.

Conclusions

Advanced AV block in myocardial infarction carries a high mortality despite a treatment programme using isoprenaline in cases of circulatory symptoms and a pacemaker if isoprenaline fails. Our results did not differ from compiled reports on routine pacemaker therapy, and the high mortality may at least partly be explained by associated complications of a massive infarction, e.g. power failure and ruptured myocardium.

Analysis of the results shows that patients with inferior infarction, narrow QRS segment, and gradual development of AV block are not likely to need pacing. The value of low doses of isoprenaline in relieving symptoms such as syncope and circulatory failure has been shown. Pacemaker therapy is not without complications, e.g. mechanical irritation by the electrode. However, by analysing our causes of death and comparing our results with those of others, there appears to be scope for some improvement, e.g. by refined pacemaker technique combined with a liberal use of drugs to combat arrhythmias and congestive failure.

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