# The Role of Arrhythmia and Left Ventricular Dysfunction in Patients with Acute Myocardial Infarction and Bundle Branch Block

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**SUMMARY**

To determine the immediate and remote prognostic significance of bundle branch block (BBB) associated with acute myocardial infarction (AMI), 40 patients with AMI and BBB were studied. One hundred forty-four patients with AMI but without BBB were evaluated during the in-hospital phase and 45 of them were taken as a control group. These patients were followed for an average of 15 months (3–28 months). Arrhythmias and left ventricular function were investigated with 24-hr Holter monitoring and echocardiography, respectively.

The hospital mortality was significantly greater in patients with BBB than in the control group (32.5% vs 10.4%, $p<0.001$). The main cause of mortality was pump failure in the group with BBB (76.9%) and ventricular fibrillation in the control group (53.3%). The peak creatine kinase level was significantly higher in the group with BBB than in the control group (2094.8±288.4 IU/L vs 416.7±30.5 IU/L, $p<0.001$).

In patients with BBB prophylactic temporary pacemaker insertion was not found to improve the hospital mortality rate. In the hospital phase, although 32% of the patients with BBB had complicated arrhythmias (multiform, paired VPB, runs, R-on-T) the cause of death in 10 of the 13 patients who died was pump failure but not arrhythmia. In patients with BBB the wall motion index and the number of patients who had a left ventricular aneurysm were greater than in patients without BBB (9.5±0.9 vs 6.3±0.6, $p<0.01$ and 52.0% vs 14.3%, $p<0.01$, respectively).

In patients with BBB follow-up mortality (12.0%) was lower than hospital mortality (32.5%). During the follow-up period there was no significant difference between patients with BBB and those without with regard to complicated arrhythmias (14.8% vs 15.6%).

These results indicate that the main cause of poor prognosis during the

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hospital period in patients with AMI and BBB was not arrhythmia or conduction disturbance but severe pump failure due to extensive myocardial necrosis. Prophylactic temporary pacemaker insertion did not improve the hospital mortality rate of these patients, and patients with AMI and BBB who survive the in-hospital phase after infarction have a good prognosis during the following 15 months. (Jpn Heart J **34**: 145–157, 1993.)

**Key Words:**
Acute myocardial infarction Bundle branch block Temporary pacemaker Ventricular arrhythmia Left ventricular function Prognosis

THE influence of bundle branch block (BBB) on survival in patients with acute myocardial infarction (AMI) and the question of whether or not to pace these patients prophylactically in the acute phase remains unresolved.[1)–9)]

In this study we investigated the early and late prognosis and the role of prophylactic temporary pacemaker use on in-hospital mortality in patients with acute Q-wave myocardial infarction and transient or persistent BBB. Therefore in these patients we studied ventricular function, the incidence and types of arrhythmias, the incidence of nonfatal myocardial infarction and mortality.

## Methods

We examined 312 patients with AMI who were consecutively admitted to our coronary care unit (CCU) between January, 1987—March, 1989. Of the 312 patients with AMI, 40 had transient or persistent BBB. These 40 patients were our study group. As a control group 144 patients with AMI but without BBB who were consecutively admitted to the CCU were evaluated during their in-hospital phase. Forty-five of them who were randomly chosen were followed up.

The types of BBB in the 40 patients were as follows: right bundle branch block (RBBB): 23 patients; RBBB+left anterior fascicular block (LAFB): 7 patients; RBBB+left posterior fascicular block (LPFB): 2 patients; left bundle branch block (LBBB): 4 patients; alternating bundle branch block: 1 patient; and isolated LAFB: 3 patients (Table I). BBB was considered transient if it was no longer present at the time of discharge from the hospital. Patients with nonspecific intraventricular conduction defect, BBB occurring only during the terminal phases of cardiogenic shock and isolated left axis deviation in association with inferior myocardial infarction were excluded.

In all patients the diagnosis of AMI was based on a typical history of chest pain associated with the appearance of new diagnostic Q waves and characteristic changes in serum enzymes. The class of heart failure for each patient was obtained by physical examination as defined by Killip and Kimball.[10)] Creatine

kinase (CK) enzyme determinations were continued until it returned to normal values (22–269 IU/L). The size of infarction was estimated by measuring the peak CK enzyme value.[11]

The timing of the onset of BBB was determined by review of old, admission and serial electrocardiograms (ECGs). The following terminology was used: *Old*: block present on an ECG antedating infarction; *Indeterminant Onset*: block present on the first ECG with infarction but with no old ECG available for comparison; *New*: block developing in the hospital documented by serial tracings or block present on the first ECG with infarction but absent on ECGs taken within the prior 3 months.

Patients stayed in the hospital for 2–4 weeks. All patients in the CCU were monitored continuously during the first 3 days. Twelve-lead ECGs were taken at the time of admission, at least daily during the hospital period and whenever a change in the QRS pattern was noticed on the monitor.

In the first few days after admission echocardiographic studies were performed. Echocardiograms were obtained with a Hewlett Packard model 77020A using a 3.5 MHz transducer. Images were recorded on videotape for subsequent analysis. For purposes of analysis, the ventricle was divided into 11 segments.[12] Each segment was evaluated for the presence and degree of asynergy. Wall motion of each segment was characterized as normal, hypokinetic, akinetic or dyskinetic. To grade the severity of segmental abnormality, each segment was assigned a numerical score based upon the type of wall motion noted. These scores were assigned as follows: normal, 0; hypokinesis, 1; akinesis, 2; and dyskinesis, 3. In order to obtain a measure of overall left ventricular function, a wall motion index was then obtained by summing the scores for each of the 11 segments.[13]

To evaluate the occurrence of ventricular premature beats (VPBs) and conduction disturbances, 24-hour Holter recordings were obtained after a continuous monitoring period. Recordings were made on a double channel Del Mar Avionics model 449B tape recorder and the tapes were then scanned on a Del Mar Avionics model 9000A Cardio Scanner. For the classification of the VPBs the following grading system was used: grade 0, no VPB; grade 1, less than 30 VPB per hour; grade 2, 30 or more VPB per hour; grade 3, multiform VPB; grade 4a, couplets; grade 4b, salvos; grade 5, early VPB.[14] Grades 0, 1 and 2 were characterized as noncomplicated arrhythmias, and grades 3, 4 and 5 as complicated arrhythmias.[15]

Patients who were discharged from the hospital were followed for 3–28 months (average of 15 months). Twenty-four-hour Holter recordings, echocardiographic studies, and clinical evaluation were repeated at 2, 6, 12, 18 and 24 months after initial admission in the both groups.

In the study group no patients underwent thrombolytic therapy, percutaneous transluminal coronary angioplasty or coronary artery bypass surgery.

For statistical analysis Student's t-test, chi-square test, Fisher's exact test and t-test were used. The results are expressed as means±standard errors. P values of $<0.05$ were considered significant.

## Results

### Hospital Period

#### Study Group

The 40 patients studied were 32 men (80%) and 8 women (20%). Their ages ranged from 32–82 years with a mean age of 58±1.9 years. The infarctions were anterior in 32 patients (80%), inferior in 6 (15%) and anterior+inferior in 2 (5%). Eight patients (20%) had previous myocardial infarctions.

*Bundle branch block:* The incidence of BBB in AMI was 12.8%. Table I shows the incidence of the various types of BBB and their timing of onset. In the 23 patients with isolated RBBB, the block was transient in 7 patients and persistent in 16 patients.

*Atrioventricular block:* Atrioventricular (AV) block developed in 8 patients (20%). First degree AV block was detected in 2 patients with inferior AMI. In one of these patients this block was transient. This patient also had transient Type I 2° AV block. In the remaining 6 patients complete AV block (CAVB) developed. Clinical characteristics of these patients are seen in Table II.

*Heart failure:* Fifteen of 40 patients (37.5%) never developed heart failure (Killip Class I). Fourteen patients (35.0%) had mild heart failure (Class II), 1 patient (2.5%) developed pulmonary edema (Class III), and 10 patients (25.0%) developed cardiogenic shock (Class IV). The association of class of heart failure

Table I. Incidence and Timing of Onset of Bundle Branch Block in 40 Patients with Myocardial Infarction

| Type of BBB | Onset of BBB | | | Total (%) |
|---|---|---|---|---|
| | Old | Indeterminant | New | |
| RBBB | 2 | 8 | 13 | 23 (57.5) |
| RBBB+LAFB | — | 5 | 2 | 7 (17.5) |
| RBBB+LPFB | — | — | 2 | 2 ( 5.0) |
| LBBB | 1 | 1 | 2 | 4 (10.0) |
| ABBB | — | — | 1 | 1 ( 2.5) |
| LAFB | 1 | 1 | 1 | 3 ( 7.5) |
| Total (%) | 4 (10.0) | 15 (37.5) | 21 (52.5) | 40 (100) |

BBB = bundle branch block; RBBB = right bundle branch block; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block; LBBB = left bundle branch block; ABBB = alternating bundle branch block.

Table II. Clinical Characteristics of the Patients with Complete Atrioventricular Block

| Case No. | Site of AMI | Associated BBB | CAVB | | Status | Cause of Death |
|---|---|---|---|---|---|---|
| | | | Timing of Onset | Duration | | |
| 1* | A | New persistent RBBB | 4th day | 2 hours | Died in hospital | HF |
| 2 | I | Ind onset persistent RBBB | 5th day | 1 day | Alive 9 months | — |
| 3* | A | New persistent RBBB+LAFB | 2nd day | 30 min | Died in hospital | HF |
| 4* | A | New persistent RBBB+LAFB | 2nd day | 30 min | Died in hospital | HF |
| 5 | A | Ind onset transient RBBB+LAFB | Present on admission | 1 day | Died 20 months | HF |
| 6 | A | New ABBB | Present on admission | 6 hours | Died in hospital | HF |

* In these patients CAVB continued till their death.

CAVB = complete atrioventricular block; A = anterior; I = inferior; Ind = indeterminant; HF = heart failure. Other abbreviations: See Table I.

Table III. In Hospital Mortality and Causes of Death in Study and Control Groups

| | Control Group ($n$ : 144) | | Study Group ($n$ : 40) | | P |
|---|---|---|---|---|---|
| | Patients dying | Mortality (%) | Patients dying | Mortality (%) | |
| Cause of Death | | | | | |
| HF | 6 | 40.0 | 10 | 76.9 | <0.05 |
| VF | 8 | 53.3 | 2 | 15.4 | <0.05 |
| CAVB | 1 | 6.7 | — | | |
| Other* | — | | 1 | 7.7 | |
| Total Deaths† | 15 (10.4%) | | 13 (32.5%) | | <0.001 |

* Cerebrovascular accident.

† Figures in parenthesis refer to % of patients dying in each group.

HF = heart failure; VF = ventricular fibrillation; CAVB = complete atrioventricular block.

and BBB type was as follows:

Killip Class I (15 patients): 5 transient RBBB, 6 persistent RBBB, 1 persistent LBBB, 1 persistent LAFB, 1 transient RBBB+LAFB, and 1 persistent RBBB+LAFB.

Killip Class II (14 patients): 2 transient RBBB, 5 persistent RBBB, 2 transient LBBB, 2 persistent LAFB, 1 transient RBBB+persistent LAFB, 1 persistent RBBB+LAFB, and 1 persistent RBBB+transient LPFB.

Killip Class III and IV (11 patients): 5 persistent RBBB, 1 persistent LBBB, 3 persistent RBBB+LAFB, 1 persistent RBBB+LPFB, and 1 alternating BBB. Of these 11 patients, 7 (63.6%) had new onset BBB.

In 4 patients with old BBB heart failure had not developed. On the contrary, pulmonary edema or cardiogenic shock developed in 26.7% of patients with indeterminant onset BBB and in 33.3% of patients with newly developed BBB. Pulmonary edema or cardiogenic shock was seen in 34.3% of patients with

Table IV. Clinical Characteristics of the Patients Who Had RBBB with and without Prophylactic Temporary Pacemaker

| | New or Indeterminant Onset | | | |
|---|---|---|---|---|
| | Persistent RBBB | | RBBB + LAFB or LPFB | |
| | Paced | No paced | Paced | No paced |
| No. of Patients | 7 | 7 | 9 | — |
| CAVB | 2 | — | 3 | |
| Site of AMI | | | | |
| Anterior | 6* | 5 | 9 | |
| Inferior | 1 | 2 | — | |
| Cause of Death | | | | |
| HF | 2 | 3 | 4 | |
| VF | 1 | — | — | |
| Total Deaths† | 3 (42.9%) | 3 (42.9%) | 4 (44.4%) | |

* Of these patients one had anterior+inferior AMI.
† Figures in parenthesis refer to % of patients dying in each group.
CAVB = complete atrioventricular block; HF = heart failure; VF = ventricular fibrillation.
Other abbreviations: See Table I.

Table V. Determinants of Hospital Mortality in Patients with Acute Myocardial Infarction and Bundle Branch Block

| Determinants | | No. of Patients | Patients Dying | Mortality (%) |
|---|---|---|---|---|
| Previous MI | Present | 8 | 3 | 37.5 |
| | Absent | 32 | 10 | 31.3 |
| Site of AMI | Anterior | 32 | 11 | 34.4 |
| | Inferior | 6 | — | 0 |
| | Anterior+inferior | 2 | 2 | 100 |
| Type BBB | Transient RBBB | 7 | 1 | 14.3 |
| | Persistent RBBB | 16 | 6 | 37.5 |
| | RBBB+LAFB | 7 | 3 | 42.9 |
| | RBBB+LPFB | 2 | 1 | 50.0 |
| | LBBB | 4 | 1 | 25.0 |
| | ABBB | 1 | 1 | 100.0 |
| | LAFB | 3 | — | 0 |
| Onset BBB | Old | 4 | — | 0 |
| | Indeterminant | 15 | 4 | 26.7 |
| | New | 21 | 9 | 42.9 |
| Duration BBB | Transient | 10 | 1 | 10.0 |
| | Persistent | 30 | 12 | 40.0 |
| Heart Failure | Killip Class I | 15 | 2 | 13.3 |
| | Killip Class II | 14 | 1 | 7.1 |
| | Killip Class III | 1 | — | 0 |
| | Killip Class IV | 10 | 10 | 100.0 |
| CAVB | Present | 6 | 4 | 66.7 |
| | Absent | 34 | 9 | 26.5 |

MI = myocardial infarction; CAVB = complete atrioventricular block.
Other abbreviations = See Table I.

anterior AMI, while there was no heart failure in patients with inferior AMI.

*Hospital mortality:* Thirteen patients died in hospital within 12 days after admission. The causes of deaths of these patients are shown in Table III. CAVB developed in 4 of these 13 patients. In spite of prophylactic temporary pacemaker insertion before development of CAVB, these 4 patients died from cardiogenic shock.

In the patients with new or indeterminant onset RBBB, prophylactic temporary pacemaker insertion did not influence the hospital mortality rate (Table IV).

Table V summarizes hospital mortality in different subsets of patients.

When patients with new RBBB were analyzed separately, 4 of 7 patients (57%) with AMI+new RBBB and 3 of 4 patients (75%) with new RBBB+LAFB or RBBB+LPFB died in hospital from pump failure.

**Control Group**

The control group included 41 men (91%) and 4 women (9%). Their ages ranged from 29–71 years with a mean age of 55±1.3 years. The average ages of the 2 groups were not significantly different. AMI locations were anterior in 57.6% and inferior in 42.4%. The incidence of anterior AMI was higher in the study group than in the control group (80.0% vs 57.6%, $p<0.01$). In the control group hospital mortality was 10.4% (Table III).

The average peak CK level was significantly higher in the study group than in the control group (2094.8±288.4 IU/L vs 416.7±30.5 IU/L, $p<0.001$).

Follow-Up

In the study group 2 patients were lost to follow-up. The remaining 25 patients were followed for an average of 15.1±1.3 months (3–26 months). In the control group 45 patients were followed for an average of 14.6±1.1 months (5–28 months). The follow-up period was not significantly different between the 2 groups.

During the follow-up period 3 patients (12%) in the study group died and 5 patients (11%) in the control group died. Thus, no significant differences were found in late mortality between the patients with AMI+BBB and the patients with AMI but without BBB.

*Echocardiography:* There were 25 patients in the study group and 28 patients in the control group who had echocardiograms that were acceptable for analysis. In the study group a total of 61 echocardiographic studies and in the control group a total of 60 echocardiographic studies were performed ($p>0.05$). Results of echocardiographic analysis between the 2 groups are shown in Table VI.

Table VI. Comparison of Echocardiographic Analysis between Study and Control Groups

| | Study Group ($n$: 25) | Control Group ($n$: 28) | P |
|---|---|---|---|
| Wall motion index ($\bar{x} \pm S_{\bar{x}}$) | 9.5 ± 0.9 | 6.3 ± 0.6 | <0.01 |
| LVEDD (cm) ($\bar{x} \pm S_{\bar{x}}$) | 6.2 ± 0.1 | 5.9 ± 0.1 | >0.05 |
| Fractional Shortening (%) ($\bar{x} \pm S_{\bar{x}}$) | 22.0 ± 0.7 | 25.6 ± 0.7 | <0.001 |
| LV aneurysm (%) | 52.0 | 14.3 | <0.01 |

LVEDD = left ventricular end-diastolic dimension; LV = left ventricle.

Table VII. Results of 24-Hour Holter Monitoring in Study and Control Groups

| | Study Group ($n$: 27) | | Control Group ($n$: 45) | |
|---|---|---|---|---|
| | No. of Patients | Death at Follow-up | No. of Patients | Death at Follow-up |
| Non-complicated arrhythmia | | | | |
| Grade 0 | 11 | 2 | 24 | 1 |
| Grade 1 | 12 | 1 | 11 | |
| Grade 2 | — | | 3 | |
| Complicated arrhythmia | | | | |
| Grade 3 | — | | — | |
| Grade 4a | 3 | | 4 | 1 |
| Grade 4b | — | | — | |
| Grade 5 | 1 | | 3 | 3 |

The study group had a significantly lower number of normal segments and a significantly higher number of hypokinetic segments than those found in the control group (44.7% vs 63.6%, $p<0.001$ and 29.1% vs 17.9%, $p<0.01$, respectively). Akinetic or dyskinetic segments were also more common in the study group compared with the control group, but there was no significant difference. In the study group the number of asynergic segments was significantly higher in the anterior ($p<0.05$), inferior ($p<0.001$) and interventricular septum ($p<0.01$) than that found in control group.

*Holter monitoring:* Sixty-two Holter recordings were obtained in the study group and 111 in the control group ($p>0.05$). Results are shown in Table VII.

With regard to complicated arrhythmia, there was no significant difference between patients with BBB and those without (14.8% vs 15.6%).

At follow-up no new AMI developed in the study group. Also in this group no new conduction disturbances developed. All patients who died in this group had anterior myocardial infarction and the cause of death was pump failure.

## Discussion

In patients with AMI the presence of BBB has been noted to be associated with a higher mortality and a high incidence of progression to high-degree AV

block.[3)-9),16)-33)] The incidence of BBB in acute infarction varies from 8% to 28.8%[16),20),26),29)]; in our series it was 12.8% for 312 patients.

Few studies have separated those patients who develop a conduction defect as a consequence of their infarction from those whose conduction abnormality preceded the infarction.[7),23),29),33),34)] However, in those patients where it has been possible to define the onset of the conduction defect in relation to the acute infarction, approximately 50% have been of acute onset.[2),7),29)] In our study, in 21 patients (52.5%) BBB was of new onset and in 4 patients (10%) BBB preceded the infarction.

The average incidence of type II 2° AV block or CAVB is 24% (8–47%).[7)] The frequency of CAVB has been related to the type of the associated BBB. Although reported statistics vary, approximately 23% of patients with isolated RBBB, 34% of patients with RBBB+LAFB, 31% of patients with RBBB+LPFB, and 16% of patients with LBBB progress to CAVB during the acute stage of infarction.[2)] In our patients these rates were 42.9% in RBBB+LAFB and 12.5% in isolated RBBB. Hindman et al reported that the risk of progression to high degree AV block was significantly higher in patients with new or indeterminant onset bilateral BBB (RBBB+LAFB, RBBB+LPFB, or alternating BBB).[28)] Among our 6 patients who had developed CAVB, 4 of them had this criterion.

When BBB complicates AMI, the site of infarction is usually anteroseptal.[34)] In our study 80% of the infarctions were anterior and the in-hospital mortality rate in this group was 34.4%. The overall mortality rate of patients with acute infarction and BBB is greater than that of patients with AMI without BBB (28% vs 12%).[7)] The degree of increase in mortality rate is related to the particular conduction defect present. The mortality rate of patients with isolated RBBB is approximately 44%, RBBB+LAFB is 38%, RBBB+LPFB is 48% and LBBB is 29%.[2)] A higher mortality rate was found in our patients who had persistent RBBB and RBBB+LAFB or LPFB.

Several groups have found that patients with transient BBB secondary to acute infarction have a much better prognosis than patients with acute, persisting defects.[5),6),8),9),17)] In our patients with transient BBB hospital mortality rate was 10%. This rate is similar to that in patients who had never demonstrated BBB (10.4%).

Several series have attempted to separate patients with conduction defects present prior to infarction from those developing defects acutely as a result of the infarction; results indicate that absolute mortality rates in the two settings are similar.[5),7),23),29)] However, in evaluating specific lesions, new defects involving the right bundle (isolated RBBB, RBBB+LAFB or RBBB+LPFB) were associated with a higher mortality rate than the mortality rate associated with preexisting defects.[7),9),34)] In this study, of 11 patients with new onset persistent RBBB,

RBBB+LAFB or RBBB+LPFB, 7 died during the in-hospital period (63.6%). Two patients who had old RBBB were alive during the 14 months of follow-up.

Most of the studies reported that hospital mortality was directly related to the degree of heart failure. Myocardial infarctions with BBB without pump failure (Killip Class I) have a hospital mortality rate of 6% to 8%, while the concomitant presence of severe pump failure (Killip Class III or IV) carries a mortality rate exceeding 50%.[4),5),7),19),27)] According to these investigators the primary cause of death in these patients is pump failure or cardiogenic shock and not electrical instability as presented by CAVB or asystole. However, another group has claimed that the majority of deaths were due to CAVB, asystole, or ventricular arrhythmia, not pump failure.[6),8),16),29)] Like the former investigators we also had found a close association between deaths and pump failure. The in-hospital mortality rate of our patients in Killip Class III+IV was 90.9% while patients in Killip Class I+II was 10.3%, and 76.9% of the hospital deaths were the result of pump failure.

We found that peak creatine kinase level, which reflects the degree of myocardial necrosis, was significantly higher in patients with BBB than in the control group.

Echocardiographic studies showed that left ventricular dysfunction in patients with AMI and BBB was greater than in patients with AMI unaccompanied by BBB. There was no significant difference between patients with BBB and those without with regard to complicated arrhythmias. All these data support the conclusion that the cause of poor prognosis for most patients with BBB was pump dysfunction because of the extent of myocardial damage, rather than the result of a conduction disturbance.

It has been known from autopsy studies that in most patients with AMI and BBB, the left anterior descending artery or the left main coronary artery before its bifurcation was occluded or grossly narrowed by atheroma or thrombus. Infarction was extensive and usually involved the anterior part of the left ventricular myocardium and interventricular septum.[5),6),22)] Similarly, in our patients with BBB the number of asynergic segments was significantly higher in the anterior wall and interventricular septum than that found in patients without BBB. In addition, all aneurysms that were found in patients with BBB were located in the anterior wall of the left ventricle.

In patients who developed CAVB in association with BBB, a high mortality rate of approximately 85% has been noted.[1)] Because of the high incidence of CAVB in patients with acute infarction and BBB, prophylactic ventricular pacing has been utilized in an effort to decrease the mortality rate in these patients. While all authors are in agreement regarding the necessity of temporary pacing in patients with CAVB and BBB, the use of prophylactic pacing for BBB without

CAVB is much more controversial. In a retrospective multicenter study Hindman et al have helped to identify patients who might benefit from prophylactic pacemaker therapy. The authors recommended that temporary pacing be utilized in a high-risk subgroup which included patients who demonstrate new or indeterminant onset bilateral BBB.[28] In the present study, of the 6 patients who had developed CAVB, 4 were in the high-risk subgroup. Although in all these patients prophylactic temporary pacemakers were inserted before the development of CAVB, 4 patients died from pump failure during the in-hospital period. Again, in those patients with new or indeterminant onset RBBB prophylactic temporary pacemaker insertion did not influence the hospital mortality rate. As a result, in patients with BBB, prophylactic temporary pacemaker insertion was not found to be effective in improving their hospital mortality rate.

Some investigators stated that hospital survivors of AMI complicated by BBB have an increased risk of dying during the follow-up period after infarction compared to patients who survive AMI not complicated by BBB.[7),30] Our follow-up observations appear to be in contrast to this result. The results of our study indicate a good prognosis during the 15 months following AMI. Other studies also reported that the prognosis appears to be good for patients who survive the acute phase.[4),32]

## Conclusions

1. AMI+BBB is associated with a high mortality rate during the in-hospital period.
2. The main cause of poor in-hospital prognosis in patients with AMI and BBB was not arrhythmia or conduction disturbance, but rather severe pump failure due to extensive myocardial necrosis.
3. Prophylactic temporary pacemaker insertion was not found to be effective in improving the hospital mortality rate of these patients.
4. Patients with AMI and BBB who survive the in-hospital phase after infarction have a good prognosis during the 15 months of follow-up.

## References

1. Roos JC, Dunning AJ: Bundle branch block in acute myocardial infarction. Eur J Cardiol **6**: 403, 1978
2. Klein RC, Vera Z, Mason DT: Intraventricular conduction defects in acute myocardial infarction: incidence, prognosis and therapy. Am Heart J **108**: 1007, 1984
3. Jones ME, Terry G, Kenmure ACF: Frequency and significance of conduction defects in acute myocardial infarction. Am Heart J **94**: 163, 1977
4. Waters DD, Mizgala HF: Long-term prognosis of patients with incomplete bilateral bundle branch block complicating acute myocardial infarction. Am J Cardiol **34**: 1, 1974
5. Col JJ, Weinberg SL: The incidence and mortality of intraventricular conduction defects in acute

myocardial infarction. Am J Cardiol **29**: 344, 1972
6. Norris RM, Croxson MS: Bundle branch block in acute myocardial infarction. Am Heart J **79**: 728, 1970
7. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, Hutter AH, Yeatman L, Rubenfire M, Pujura C, Rubin M, Morris JJ: The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. Circulation **58**: 679, 1978
8. Lichstein E, Gupta PK, Chadda KD: Long-term survival of patients with incomplete bundle branch block complicating acute myocardial infarction. Br Heart J **83**: 924, 1975
9. Ricou F, Nicod P, Gilpin E, Henning H, Ross J: Influence of right bundle branch block on short- and long-term survival after acute anterior myocardial infarction. J Am Coll Cardiol **17**: 858, 1991
10. Killip T, Kimball JT: Treatment of myocardial infarction in a coronary care unit: a two-year experience with 250 patients. Am J Cardiol **20**: 457, 1967
11. Herlitz J, Hjalmarson A, Waldenstrom J: Relationship between different enzymatic estimations of the infarct size. Acta Cardiol **40**: 217, 1985
12. Gibson RS, Bishop HL, Stamm RB, Crampton RS, Beller GA, Martin RP: Value of early two dimensional echocardiography in patients with acute myocardial infarction. Am J Cardiol **49**: 1110, 1982
13. Heger JJ, Weyman AE, Wann LS, Rogers EW, Dillon JC, Feigenbaum H: Cross-sectional echocardiographic analysis of the extent of left ventricular asynergy in acute myocardial infarction. Circulation **61**: 1113, 1980
14. Lown B, Wolf M: Approaches to sudden death from coronary heart disease. Circulation **44**: 130, 1971
15. Schulze RA, Humphries JO, Griffith LSC, Ducci H, Achuff S, Baird MG, Mellits ED, Pitt B: Left ventricular and coronary angiographic anatomy: relationship to ventricular irritability in the late hospital phase of acute myocardial infarction. Circulation **55**: 839, 1977
16. Scheidt S, Killip T: Bundle branch block complicating acute myocardial infarction. JAMA **222**: 919, 1972
17. Lie KI, Wellens HJ, Schuilenburg RM, Becker AE, Durrer D: Factors influencing prognosis of bundle branch block complicating acute anteroseptal infarction. Circulation **50**: 935, 1974
18. Fenig S, Lichstein E: Incomplete bilateral bundle branch block and A-V block complicating acute anterior wall myocardial infarction. Am Heart J **84**: 38, 1972
19. Ritter WS, Atkins JM, Blomqvist CG, Mullins CB: Permanent pacing in patients with transient trifascicular block during acute myocardial infarction. Am J Cardiol **38**: 205, 1976
20. Scheinman M, Brenman B: Clinical and anatomic implications of intraventricular conduction blocks in acute myocardial infarction. Circulation **46**: 753, 1972
21. Rizzon P, Rossi L, Baissus C, Demoulin JC, DiBiase M: Left posterior hemiblock in acute myocardial infarction. Br Heart J **37**: 711, 1975
22. Roos JC, Dunning AJ: Right bundle branch block and left axis deviation in acute myocardial infarction. Br Heart J **32**: 847, 1970
23. Nimetz AA, Shubrooks SJ, Hutter AM, DeSanctis RW: The significance of bundle branch block during acute myocardial infarction. Am Heart J **90**: 439, 1975
24. Basualdo CAE, Haraphongse M, Rossal RE: Intraventricular blocks in acute myocardial infarction. Chest **67**: 75, 1975
25. Scanlon PJ, Pryor R, Blount SG: Right bundle branch block associated with left superior or inferior intraventricular block. Circulation **42**: 1135, 1970
26. Godman MJ, Lassers BW, Julian DG: Complete bundle branch block complicating acute myocardial infarction. N Engl J Med **282**: 237, 1970
27. Atkins JM, Leshin SJ, Blomqvist G, Mullins CB: Ventricular conduction blocks and sudden death in acute myocardial infarction. N Engl J Med **288**: 281, 1973
28. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, Hutter AH, Yeatman L, Rubenfire M, Pujura C, Rubin M, Morris JJ: The clinical significance of bundle branch block complicating acute myocardial infarction. 2. Indications for temporary and permanent pacemaker insertion. Circulation **58**: 689, 1978
29. Gann D, Balachandran PK, Sherif NE, Samet P: Prognostic significance of chronic versus acute

bundle branch block in acute myocardial infarction. Chest **67**: 298, 1975

30. Waugh RA, Wagner GS, Haney TL, Rosati RA, Morris JJ: Immediate and remote prognostic significance of fascicular block during acute myocardial infarction. Circulation **47**: 765, 1973
31. Ginks WR, Sutton R, Oh W, Leatham A: Long-term prognosis after acute anterior infarction with atrioventricular block. Br Heart J **39**: 186, 1977
32. Hauer RNW, Lie KI, Liem KL, Durrer D: Long-term prognosis in patients with bundle branch block complicating acute anteroseptal infarction. Am J Cardiol **49**: 1581, 1982
33. Hollander G, Nadiminti V, Lichstein E, Greengart A, Sanders M: Bundle branch block in acute myocardial infarction. Am Heart J **105**: 738, 1983
34. Lie KI, Wellens HJ, Schuilenburg RM: Bundle branch block and acute myocardial infarction. *in* The Conduction System of the Heart: Structure, Function and Clinical Implications, ed by Wellens HJ, Lie KI, Janse MS, Leiden HE, Lea and Febiger, Philadelphia, p 662, 1976