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Prognostic significance of right bundle branch block in patients with acute inferior myocardial infarction

Jun Iwasaki[*]   Kunihisa Kono[†]   Yusuke Katayama[‡]

Natsuki Takahashi[**]   Kazufumi Takeuchi[††]   Machiko Tanakaya[‡‡]

Kazuhiro Osawa[§]   Teruo Shiraki[¶]   Daiji Saito[||]

[*]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[†]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[‡]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[**]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[††]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[‡‡]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[§]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[¶]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,
[||]Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization,

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Abstract

There is little information available concerning the influence of right bundle branch block (RBBB) on the prognosis of patients with inferior myocardial infarction (MI). In this study we evaluated the influence of RBBB on the short-term prognosis of patients with inferior MI. Our study subjects were 1,265 hospitalized patients with Q wave MI. Patients were divided into 4 groups based on the presence or absence of RBBB and on the location of the infarction. RBBB was classified into 4 categories according to the timing of its appearance and its duration as new permanent, transient, old and age indeterminate. In-hospital death and pulmonary congestion were observed more frequently in patients with RBBB than in those without RBBB. Moreover, in inferior MI as in anterior MI, in-hospital death and pulmonary congestion occurred more frequently in new permanent RBBB patients than in patients with other types of RBBB. Multivariate regression analysis reveals that new permanent RBBB was a strong independent predictor for an adverse short-term prognosis in patients with inferior MI, as well as in patients with anterior MI. New permanent RBBB during inferior MI is a strong independent predictor for increased in-hospital mortality, regardless of the infarction location.

KEYWORDS: right bundle branch block, inferior infarction, in-hospital death, new permanent RBBB

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Original Article

Prognostic Significance of Right Bundle Branch Block in Patients with Acute Inferior Myocardial Infarction

Jun Iwasaki*, Kunihisa Kono, Yusuke Katayama, Natsuki Takahashi,
Kazufumi Takeuchi, Machiko Tanakaya, Kazuhiro Osawa,
Teruo Shiraki, and Daiji Saito

Department of Cardiology, Iwakuni Clinical Center, National Hospital Organization, Iwakuni, Yamaguchi 740–8510, Japan

There is little information available concerning the influence of right bundle branch block (RBBB) on the prognosis of patients with inferior myocardial infarction (MI). In this study we evaluated the influence of RBBB on the short-term prognosis of patients with inferior MI. Our study subjects were 1,265 hospitalized patients with Q wave MI. Patients were divided into 4 groups based on the presence or absence of RBBB and on the location of the infarction. RBBB was classified into 4 categories according to the timing of its appearance and its duration as new permanent, transient, old and age indeterminate. In-hospital death and pulmonary congestion were observed more frequently in patients with RBBB than in those without RBBB. Moreover, in inferior MI as in anterior MI, in-hospital death and pulmonary congestion occurred more frequently in new permanent RBBB patients than in patients with other types of RBBB. Multivariate regression analysis reveals that new permanent RBBB was a strong independent predictor for an adverse short-term prognosis in patients with inferior MI, as well as in patients with anterior MI. New permanent RBBB during inferior MI is a strong independent predictor for increased in-hospital mortality, regardless of the infarction location.

Key words: right bundle branch block, inferior infarction, in-hospital death, new permanent RBBB

Right bundle branch block (RBBB) appeared to be an independent risk factor in patients with acute anterior myocardial infarction (MI). Considering the anatomy and vascular supply of the conduction system, RBBB is usually the manifestation of large anterior MI that are often accompanied by heart failure and atrioventricular (AV) conduction block [1, 2], but the mechanism by which it represents an independent risk factor is still unclear. Furthermore, RBBB appears not only in patients with anterior myocardial infarction but is also observed frequently in acute MI of other locations, especially of the left ventricular inferior wall. There have been few investigations available that address the influence of RBBB complicated by inferior MI on short-term prognosis [3, 4]. The aim of the present study was to determine the current incidence and significance of RBBB complicated by MI involving the left ventricular inferior wall.

Materials and Methods

Study patients. This study was retrospectively conducted using the data obtained from 1,265 patients

who were admitted to Iwakuni Clinical Center, National Hospital Organization, from January 1, 1997 to December 31, 2006 due to acute anterior or inferior myocardial infarction within 48h after the onset of symptoms.

RBBB was defined using standard electrocardiographic (ECG) criteria as 1) a QRS duration $\geq$ 120 msec, 2) the presence of an rSR' pattern of QRS in lead V1, 3) a PQ interval > 120 msec, and 4) a S wave in lead I and either lead V5 or V6. RBBB was further classified according to the time of appearance. "New" was when RBBB appeared after admission or was present on admission but was not observed on an ECG within the previous 6 months. New RBBBs were further classified into 2 groups according to the duration of RBBB as "transient" if the RBBB disappeared during the stay in the hospital or "new permanent" if the RBBB was present when the patient either died or was discharged. "Old RBBB" were those present on admission and documented on a previous ECG. If the RBBB was present on admission and no previous ECG was available, we classified the RBBB as "age indeterminate".

The diagnosis of MI was established by the presence of 2 of the following 3 criteria: 1) elevation of serum creatine kinase (CK) to more than twice the upper limit of normal values, 2) characteristic chest pain lasting at least 30 min. or 3) ECG ST-T changes with evolution of an abnormal Q wave. Clinical history, physical examination, laboratory data, the standard 12-lead ECG and chest roentgenograms were obtained on admission. ECG leads CM5 and NASA were continuously monitored for at least the following 2 days. After admission, the standard 12-lead ECG was recorded every day for 3 days and then every 3 days for a week, and a chest x-ray was obtained on the 2nd day after admission and when necessary thereafter. Laboratory examination was followed on the 2nd, 3rd and 6th days.

The patients were divided into 2 groups based on the location of infarction using standard 12-lead ECG. When an abnormal Q wave was observed in any of leads V1 through V4 with a characteristic ST elevation, the infarction was defined as anterior. If the abnormal Q wave was not in any of these anterior chest-leads and was found in leads Ⅱ, Ⅲ and/or a ventricular fibrillation with significant ST elevation, the infarction was labeled an "inferior infarction". The

patient in whom an abnormal Q wave was observed that was located in neither the anterior nor the inferior leads was excluded from this study. Patients with non-Q wave infarction were also excluded from this study, because it was difficult to localize the infarction by ECG. Pulmonary congestion due to left ventricular failure was categorized according to Killip's classification, and the affected patients were then further categorized as with (Killip's criteria ≥ 2) or without (Killip's criteria $= 1$) congestion.

In this study, in-hospital death included patients who died during admission due to cardiac disorder and patients who showed cardiac arrest on admission but whose symptoms and ECG recorded before arriving at our hospital demonstrated typical acute myocardial infarction. The laboratory data on admission were adopted for analysis. Hypertension was considered to be present if there was an apparent history of high blood pressure regardless of medication, or if physical examination showed elevated systolic and/or diastolic blood pressure (persistently $\geq 140/90$ mmHg).

Data analysis.   SAS software (SAS Institute Inc., Cary, NC, USA) was used for data analysis. Analysis of variance and then Bonferroni's modified *t-test* were applied to compare group means for continuous variables, and the chi-square test with Yates' correction was used to compare the incidence of discrete variables. Multivariate logistic regression analysis was performed to evaluate the independent importance of the variables for in-hospital death.

The research protocol was approved by the ethical committee of Iwakuni Clinical Center.

Results

Among 1,265 patients recruited for this study, 38 patients had ECG findings of intraventricular conduction block other than RBBB and were not considered for further analysis. RBBB was documented in 145 patients (11.5%). The remaining 1,082 patients, documenting normal intraventricular conduction, served as controls. There were 99 cases (68.3%) with newly developed RBBB, of which 41 (41.4%) were categorized as new permanent RBBB and 58 (58.6%) were categorized as transient RBBB. Of the transient RBBB cases, 21 RBBBs lasted < 1h. Old RBBB was observed in 20 patients (13.8%), and RBBB in the remaining 26 patients (17.9%) was classified as age

indeterminate (Fig. 1). With respect to the location of infarction, of the 544 patients who had anterior MI, 73 had RBBB. Six hundred and seventy three patients had inferior MIs, of which 72 were documented as RBBB.

Coronary artery involvement. Coronary arteriograms on admission demonstrated that segment 1 or 2 of the right coronary artery was a culprit lesion in 30 (73.2%) of 41 patients with new permanent RBBB and in 36 (69.2%) of 52 patients with transient RBBB in inferior infarction, while 43.1% of 1,010 patients were without RBBB. The involvement of this coronary segment was significantly higher in frequency in patients with new permanent or transient RBBB than in patients with normal intraventricular conduction (43.1%, $p < 0.01$). Early coronary reperfusion by thrombolysis, percutaneous coronary balloon angioplasty and/or coronary stenting was intended in 905 patients with normal conduction and in 125 patients with RBBB, and was successfully achieved (TIMI 3) in 873 (96%) and 118 (94%) patients, respectively. The rate of successful coronary reperfusion achieved was not significantly different between patients with RBBB and those with normal intraventricular conduction.

Clinical characteristics. The clinical characteristics of the study patients with and without RBBB are represented in Table 1. The mean age and gender distribution were not different between the patients with and without RBBB. The incidence of inferior wall involvement was similar between the 2 groups. Pulmonary congestion (Killip $\geq$ 2) was documented

frequently in the patients with RBBB compared to those with normal conduction (34.5% vs. 17.8%, $p < 0.001$). A total of 112 patients died during their stay in the hospital. Of the 112, 101 patients died because of cardiogenic causes, including 49 from congestive heart failure, 28 from cardiogenic shock, 9 from cardiac rupture, 8 from sudden death and 7 from cardiopulmonary arrest on arrival with successful cardiac resuscitation prior to arrival. Two patients died due to cerebral infarction, and 1 died due to a dissecting aneurysm of the thoracic aorta complicated with myocardial infarction. Six patients were lost due to pneumonia, and 2 were lost due to sepsis. In-hospital death occurred more frequently in the RBBB patients than in those with normal conduction (20.7% vs. 7.6%, $p < 0.001$). A prevalence of hypertension, diabetes mellitus, cerebrovascular accidents or previous myocardial infarction in patients with RBBB was not significantly different from that in patients with normal conduction. Complete AV block appeared in 2 patients during the hospital course: one patient with inferior infarction was treated with temporary pacing, and the other who showed anterior infarction needed permanent cardiac pacing. Both patients fully recovered and were discharged.

Prognosis of patients with RBBB. With respect to the type of RBBB, successful coronary reperfusion of all RBBB groups varied from 91% to 96%, and the rates were not different among the groups. New permanent RBBB was associated with a greater incidence of pulmonary congestion (68.3%) than were old RBBB (40.0%, $p = 0.035$), age indeter-
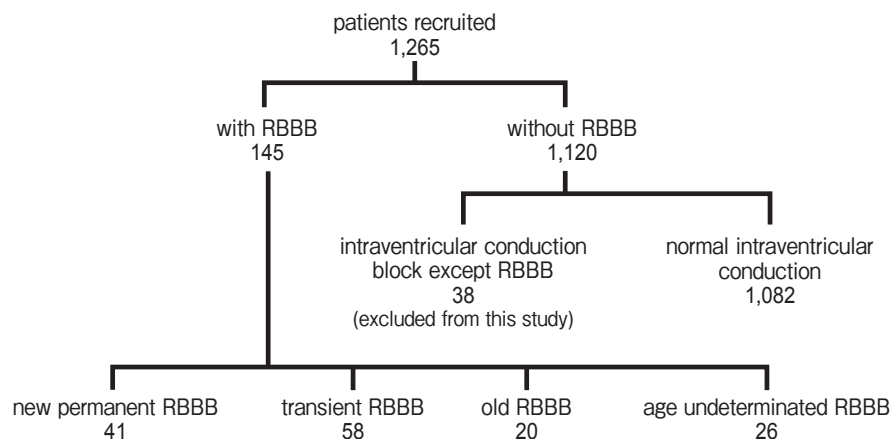

Fig. 1   Flow chart for patients analyzed in this study. RBBB, right bundle branch block.

Table 1   Clinical characteristics of patients with and without RBBB

	RBBB (+) (n = 145)	RBBB (−) (n = 1,082)	P
Gender (M/F)	106/39	752/330	0.368
Age (age)	67 (12)	70 (12)	0.0053
Infarction site (no.)			
anterior	73	471	
inferior	72	611	0.144
Pulmonary congestion (no.)			
yes	60	187	
no	85	895	<0.001
In-hospital death (no.)			
dead	30	82	
alive	115	1,000	<0.001
Peak CK (IU/ml)	1,368 (1,582)	1,438 (1,504)	0.658
TC (mg/dl)	182 (43)	193 (39)	0.0013
HDLc (mg/dl)	43 (11)	45 (13)	0.259
LDLc (mg/dl)	118 (35)	128 (36)	0.023
HDLc/LDLc	0.27 (0.11)	0.27 (0.14)	0.972
TG (mg/dl)	99 (59)	107 (57)	0.265
Uric acid (mg/dl)	5.4 (1.8)	5.3 (1.9)	0.665
Smoking (no.)			
yes	76	553	
no	69	529	0.836
Hypertension			
yes	57	472	
no	88	610	0.371
Diabetes (no.)			
yes	28	240	
no	117	842	0.497
CVA (no.)			
yes	14	158	
no	131	926	0.140
Previous MI (no.)			
yes	26	156	
no	119	956	0.321
≦6h (no.)	94	682	0.710

no., number of patients; CK, creatine kinase; TC, total cholesterol; HDLc, high density lipoprotein cholesterol; LDLC, low density lipoprotein cholesterol; TG, triglyceride; CVA, cerebrovascular accident; MI, myocardial infarction; ≦6h, patients arriving within 6h from the onset of the chest pain. Data are expressed by mean (SD).

minate RBBB (34.6%, $p < 0.001$) or normal conduction (29.1%, $p < 0.01$). There were more frequent in-hospital deaths in patients with new permanent RBBB (41.5%) than in patients with transient (13.8%, $p < 0.01$), old (10.0%, $p < 0.01$) or age indeterminate RBBB (11.5%, $p < 0.01$) or normal intraventricular conduction (7.6%, $p < 0.01$), but no significant difference of mortality rate was observed

among the latter 4 groups. When the data of the transient RBBB and the new permanent RBBB were summed as "new RBBB" and compared with those of old RBBB, no measurements analyzed in this study showed a significant difference between these two RBBB groups.

Table 2 summarizes the measurements of inferior and anterior myocardial infarction in the setting of the presence or absence of RBBB. RBBB occurred in 10.7% of patients with inferior infarction and in 13.4% of patients with anterior infarction. The incidence was not statistically different ($p = 0.15$). In-hospital death in overall inferior MI patients with RBBB occurred in 15.2% of patients, which was significantly higher than that of patients with normal conduction in inferior MI (6.7%, $p = 0.012$). For patients with RBBB, pulmonary congestion was observed less frequently in inferior than anterior MI. Peak CK values of the 4 patient groups were around 1,300–1,600 IU/L, with wide variations, and were not significantly different among the groups. Patients with new permanent RBBB had higher peak CK values than did patients with other types of RBBB, but the difference did not reach the significant level {mean (SD) 2,185 (1,228) IU/L vs. 1,296 (1,195) IU/L, $p = 0.06$}.

Prognosis of patients with new permanent RBBB.   New permanent RBBB was observed in 19 patients (2.8%) with inferior infarction and in 22 (4.0%) who had anterior infarction. The incidence of this type of RBBB was not different between the inferior and anterior infarction groups. Five patients (26.3%) with new permanent RBBB complicated by inferior infarction died during their stay in the hospital, while in-hospital death occurred in 6 (11.3%) inferior MI patients with transient, old or age indeterminate RBBB (Table 3). The incidence of in-hospital death in patients with inferior MI with new permanent RBBB was significantly higher ($p < 0.01$) than that in patients with other types of RBBB. Table 4 represents the direct causes of death in patients with RBBB. Pump failure (congestive heart failure and cardiogenic shock) was a major cause of death in patients with any type of RBBB. The incidence of pump failure death was significantly higher in patients with RBBB than in those with normal intraventricular conduction (90.0% vs. 61.0%, $p = 0.0033$).

Multivariate logistic regression analysis for the prediction of in-hospital death was conducted using the

Table 2 Clinical characteristics of patients with anterior and inferior myocardial infarction complicated by RBBB

	Anterior infarction (n = 544)		Inferior infarction (n = 673)	
	RBBB (+)	RBBB (−)	RBBB (+)	RBBB (−)
Gender (M/F)	52/21	314/157	54/18	438/173
Age (age)	70 (12)	65 (12)	69 (12)	67 (12)
Pulmonary congestion (no.)				
yes	41	102	19	85
no	32	369	53	526
In-hospital death (no.)				
dead	19	41	11	41
alive	54	420	61	570
Peak CK (IU/ml)	1,495 (1,811)	1,569 (1,840)	1,466 (1,322)	1,336 (1,412)
TC (mg/dl)	181 (24)	197 (38)	182 (24)	191 (40)
HDLc (mg/dl)	44 (12)	46 (13)	43 (10)	43 (13)
LDLc (mg/dl)	118 (36)	131 (36)	118 (36)	126 (37)
HDLc/LDLc	0.26 (0.09)	0.27 (0.13)	0.27 (0.13)	0.26 (0.14)
TG (mg/dl)	94 (51)	105 (53)	106 (62)	107 (58)
Uric acid (mg/dl)	5.7 (1.9)	5.1 (2.0)	5.2 (1.6)	5.5 (1.8)
Smoking (no.)				
yes	42	237	34	317
no	31	234	38	294
Hypertension				
yes	20	189	35	267
no	53	282	37	314
Diabetes (no.)				
yes	11	106	14	117
no	62	365	58	494
CVA (no.)				
yes	7	75	7	63
no	66	396	65	548
Previous MI (no.)				
yes	10	57	14	75
no	63	414	58	536
≦6h (no.)	46	297	48	385

no., number of patients; CK, creatine kinase; TC, total cholesterol; HDLc, high density lipoprotein cholesterol; LDLc, low density lipoprotein cholesterol; TG, triglyceride; CVA, cerebrovascular accident; MI, myocardial infarction; ≦6h, patients arriving within 6h from the onset of the chest pain. Data area expressed by mean (SD).

overall data (Table 5). Of 17 variables, 4 measurements were independent factors that were associated with in-hospital mortality: age, location of infarction, presence of pulmonary congestion and hypertension. When all RBBBs were grouped together, RBBB was not a significant predictor for in-hospital mortality (relative risk = 1.69, $p = 0.108$). When we used the data of new permanent RBBB instead of all RBBBs for the logistic regression model, the relative risk of new permanent RBBB for in-hospital death increased to a significant level. The presence of pulmonary congestion demonstrated the strongest relation to in-hospital death (relative risk = 11.58, $p < 0.0001$), and new

permanent RBBB was the second strongest predictor (relative risk = 5.45, $p = 0.0007$) among the variables used in this study.

Limiting the data to inferior infarction for logistic regression analysis, only pulmonary congestion and new permanent RBBB were significant independent risk factors for predicting adverse in-hospital prognosis (Table 6). Unlike anterior MI in which pulmonary congestion, new permanent RBBB, age and hypertension were all significant predictors of in-hospital mortality, age and hypertension were not statistically significant as predictors for in-hospital mortality. The relative risks of pulmonary congestion and new perma-

nent RBBB were 7.25 ($p = 0.0002$) and 5.79 ($p = 0.033$), respectively.

Discussion

Relation of RBBB to coronary artery distribution.   The overall rate of RBBB in our study was 10.1%, which is within the range of previous investigations [5–8]. Previous reports documented that new RBBB in patients with MI is more frequently associated with anterior wall infarction than with infarction of other locations [8, 9]. This tendency was observed in the present study, but the difference was below the statistically significant level ($p = 0.15$). The lower area of the interventricular septum comprises the greater mass of the septum, including most of the 2 main bundle branches. This area is supplied

Table 3   Mortality in patients with each type of RBBB

	No. of patients	No. of death	Mortality rate (%)
New permanent RBBB	41	17	41.5#
Anterior infarction	22	12	54.5
Inferior infarction	19	5	26.3*
Transient RBBB	58	8	13.8
Anterior infarction	30	5	16.7
Inferior infarction	28	3	10.7
Old RBBB	20	2	10.0
Anterior infarction	11	1	9.1
Inferior infarction	9	1	11.1
Age undeterminate RBBB	26	3	11.5
Anterior infarction	10	1	10.0
Inferior infarction	16	2	12.5

RBBB = right bundle branch block.
Significantly different * from anterior infarction with new permanent RBBB, and # from infarction with transient, old and indeterminate age RBBB at level of $p < 0.01$.

Table 4   Direct causes of death in patients with RBBB

Type of RBBB	CHF	shock	LV rupture	pneumonia
New permanent (n = 17)	10	5	1	1
Transient (n = 8)	4	3	1	0
Old (n = 2)	2	0	0	0
Age undetermined (n = 3)	2	1	0	0
Total (n = 30)	18	9	2	1

CHF, congestive heart failure; LV rupture, left ventricular rupture.

Table 5   Relative risk of variables for in-hospital mortality in all study patients

	Relative Risk	95% CI	P
Gender (male)	1.18	0.63 – 2.18	0.61
Age	1.06	1.03 – 1.09	0.003
New permanent RBBB	5.45	2.05 –14.44	0.0007
Pulmonary congestion	11.58	5.72 –23.43	< 0.0001
Peak creatine kinase	1.02	0.45 – 2.01	0.95
Total cholesterol	1.01	0.81 – 1.26	0.93
HDLc	1.00	0.80 – 1.25	0.97
LDLc	1.11	0.81 – 1.26	0.93
HDLc/LDLc	0.16	0.004– 7.39	0.35
Trigyceride	0.99	0.95 – 1.04	0.92
Uric acid	1.15	1.03 – 1.29	0.16
Smoking	1.06	0.60 – 1.85	0.85
Hypertension	0.45	0.26 – 0.78	0.004
Diabetes mellitus	0.57	0.28 – 1.15	0.12
CVA	1.35	0.71 – 2.59	0.36
Previous MI	1.01	0.49 – 2.06	0.99
$\leqq$ 6 h	0.78	0.47 – 1.32	0.36

RBBB, right bundle branch block; HDLc, high density lipoprotein cholesterol; LDLc, low density lipoprotein cholesterol; CVA, cerebrovascular accident; MI, myocardial infarction; $\leqq$ 6 h, patients arriving within 6 h from the onset of chest pain. 95% CI, 95% confidence interval.

Table 6   Relative risk of clinical variables for in-hospital mortality in patients with inferior myocardial infarction

	Relative Risk	95% CI	P
Gender	1.57	0.54– 4.58	0.41
Age	1.04	0.99– 1.09	0.092
New permanent RBBB	5.79	1.15–29.13	0.033
Pulmonary congestion	7.13	2.47–20.56	0.0003
Peak creatine kinase	1.00	0.97– 1.20	0.59
Total cholesterol	0.81	0.44– 1.48	0.49
HDLc	1.27	0.69– 2.33	0.44
LDLc	1.24	0.68– 2.24	0.49
HDLc/LDLc	0.91	0.11– 5.02	0.37
Triglyceride	1.04	0.93– 1.18	0.48
Uric acid	1.09	0.88– 1.34	0.42
Smoking	1.23	0.84– 2.51	0.86
Hypertension	0.60	0.24– 1.48	0.26
Diabetes	0.43	0.11– 1.70	0.23
CVA	2.32	0.80– 6.74	0.12
Previous MI	1.19	0.39– 3.65	0.76
$\leqq$ 6 h	1.34	0.56– 3.20	0.51

RBBB, right bundle branch block; HDLc, high density lipoprotein cholesterol; LDLc, low density lipoprotein cholesterol; CVA, cerebrovascular accidents; MI, myocardial infarction; $\leqq$ 6 h, patients arriving within 6 h from the onset of chest pain; 95% CI, 95% confidence interval.

mainly by penetrating branches from the left anterior descending coronary artery [10]. Therefore, it is reasonable to assume that impaired circulation through this vessel may cause dysfunction of the right bundle branch. In most cases of inferior MI, the right coronary artery was the culprit. In the study of James and Burch [10], the right coronary artery in more than 90% of the hearts perfused the upper portion of the interventricular septum, which includes the AV node, the bundle of His and the upper segments of the 2 main bundle branches. Based on this arbitrary division of the blood supply we suppose that occlusion of the right coronary artery may be associated with disturbance of AV conduction (various degrees of AV block) rather than bundle branch block. Thus, interruption of the blood supply to the right bundle by occlusion of the right coronary artery was not thought to be a cause of RBBB. Chronic damage of the right bundle branch appeared to be the pathologic basis of RBBB [10] in the absence of infarction. The interruption of blood flow at the proximal right coronary artery involves the right ventricular wall, resulting in right ventricular dilatation. Mechanical stretching along with a chronically damaged right bundle branch may also play a role in the development of RBBB [11]. The findings of more frequent involvement of the proximal right coronary artery (segment 1 or 2) in patients with new permanent RBBB than in patients with normal intraventricular conduction would support the concept that right ventricular distension could be responsible for newly developed RBBB. Therefore, the new appearance of RBBB during inferior MI could be interpreted as an important ECG sign of right ventricular enlargement.

Mechanism of adverse effects of RBBB. With respect to short-term mortality, the prognosis of patients who develop bundle branch block depends on the underlying cause. As reported previously, the occurrence of new permanent RBBB indicates a poor prognosis in patients with anterior MI [8, 12–14]. The increased in-hospital mortality in patients with new permanent RBBB associated with MI was also confirmed in our study population, which agrees with the data presented by Ricou *et al.* [15] and Hod *et al.* [5]. Of several explanations proposed, sudden cardiac death due to complete AV block was a possible explanation for the increased mortality in patients with new permanent RBBB during MI. However, the increased in-hospital mortality in our patients could not be explained by the sudden development of a high degree of AV block, since none of the patients with RBBB died of documented complete AV block during their stay in our hospital. The increased mortality in patients with new permanent RBBB may be due to more extensive involvement of the ventricular wall by infarction, since pulmonary congestion was observed more frequently in patients with new permanent RBBB than in patients in the other patient groups. This possibility was supported by the findings that peak CK tended to be higher in patients with new permanent RBBB than in those with other types of RBBB. In our hospital, we do not measure cardiac output and left ventriculography before coronary angiography of AMI as a general rule. Although few data on cardiac output and left ventricular ejection fraction were available for this study, the indexes of cardiac function could deteriorate in patients with new permanent RBBB, because a higher prevalence rate of pulmonary congestion was observed in this patient group. However, multivariate logistic regression analysis revealed that the presence of new permanent RBBB in the setting of MI was a strong predictor for increased in-hospital mortality independent of pulmonary congestion. The results suggested that an effect of the conduction disturbance on short-term mortality may not come only from extensive left ventricular impairment.

An alternative explanation for the adverse short-term prognosis was dyssynchrony of the ventricles. Some have proposed that intraventricular conduction delay may further impair the ability of the failing heart to eject blood [16, 17]. The finding of an intraventricular conduction disturbance has been associated with clinical instability and an increased risk of death in patients with heart failure [18, 19]. However, such dyssynchrony of the ventricles cannot fully explain the increased risk of in-hospital death, because the patients with old or age indeterminate RBBBs complicated by MI were documented as being at low risk, as were the patients without RBBB. In addition, the beneficial effect of cardiac resynchronization on cardiac function was limited in the heart with left bundle branch block (LBBB) but not RBBB [20].

Location of infarction and cardiac mortality. With respect to the location of infarction, the present

study documented that new permanent RBBB adversely affected the short-term prognosis of patients with inferior infarction, the same having been observed in patients with anterior infarction. Post-mortem studies revealed that there is right ventricular involvement in 19% to 51% of patients with acute inferior MI [21-24]. Zehender and associates [25] reported that right ventricular infarction was a strong, independent predictor of major complications and in-hospital mortality. The relative risk was 7.7 for in-hospital mortality and 4.7 for major in-hospital complications. Although our study did not intend to provide conclusions about the right ventricular infarction in inferior MI patients, the right ventricular involvement could occur in a certain percentage of the patients with new permanent RBBB. The high incident rate of pump failure death in our study patients would support the contribution of right ventricular dysfunction to the relatively poor prognosis in patients with RBBB. Thus, it may be reasonable to assume that right ventricular dysfunction would be present in association with acute inferior infarction and would worsen the short-term prognosis of our patients.

In contrast to patients with new permanent RBBB, the incidence of in-hospital death in patients with transient RBBB was low, similar to that of patients with normal conduction. Experimental and clinical evidence suggests that transient conduction disturbances may relate to extensive but reversible ischemia and inflammatory responses surrounding the specialized conduction tissue [16-29]. Rapid resolution of RBBB in MI has been described [8, 15] and more recently has been linked with thrombolytic therapy [28-30]. Early coronary reperfusion could interrupt the advance of myocardial ischemia and salvage the conduction system from extensive dysfunction. In addition, recovery from excessive stretching of the right bundle branch by improved hemodynamics in the right ventricle could play a role in the resolution of RBBB.

Based on our study results, we conclude that RBBB newly developed and persisting during MI suggests poor short-term prognosis in patients with inferior infarction as well as in those with anterior infarction.

Study limitations.   Some asymptomatic, short-lived RBBBs may have been underestimated, since their recognition depends primarily on the careful attention of the CCU nurse and the sensitivity of the electrical alarm in response to the changes of the QRS pattern during ECG monitoring. Furthermore, the number of RBBBs, especially new permanent RBBBs, in this study was too small to apply for general consideration. As is well known, the management of MI has dramatically changed, and the prognosis of MI, even in patients with new permanent RBBB, has been improved year by year. Our analysis was conducted in patients treated over a period 10 years, and the 10-year period may be too long to allow us to deal meaningfully with the data as one group. The short-term prognosis in the recent several years could be different from that of the earliest years. Large-scale studies on patients in whom myocardial infarction developed within the latest several years are needed.

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