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Right Bundle-Branch Block in Coronary Artery Disease: A Hemodynamic and Angiographic Study

R. P. DABIZZI, M.D., L. AIAZZI, M.D., G. A. BARLETTA, M.D., G. TEODORI, M.D.

Department of Medicine, Division of Cardiology, University of Florence, Florence, Italy

Summary: Thirty-four patients with right bundle-branch block (RBBB) and coronary artery disease (CAD) (RBBB was not pre-existent to clinical development of CAD) and 52 consecutive CAD patients without conduction disturbances were studied and compared to verify whether the presence of RBBB implies more severe and extensive left ventricular myocardial damage as well as more severe CAD. The two groups did not differ either in age or in New York Heart Association functional class. The incidence or location of previous myocardial infarction (MI) was not different in the two groups. No significant differences were found in left ventricular volumes or ejection fraction. Higher end-diastolic left ventricular pressure and more severe and diffuse left ventricular wall asynergy were present in RBBB patients. At coronary arteriography, more severe involvement of the right coronary artery in CAD patients without conduction disturbances was the only significant finding. The group of patients with CAD and RBBB without MI showed significantly less involvement of the left anterior descending coronary artery and significantly more severe damage of the inferior wall of the left ventricle than the group with CAD without RBBB and MI. Patients with inferior wall MI and RBBB had more severe asynergy of the posterobasal region of the left ventricle than did patients with inferior wall MI without RBBB. The group of patients with anterior wall MI and RBBB had a higher left ventricular end-diastolic pressure, a lower left ventricular ejection fraction, and a greater extent of myocardial damage compared to similar patients of the control group. The groups with MI and RBBB had the same Gensini's score as similar groups without RBBB. The hemodynamic, angiographic, and ventriculographic findings in 14 of the 34 RBBB patients who had also presented an abnormal QRS frontal axis deviation showed no significant differences in comparison both with the CAD control group and the remaining RBBB patients with normal QRS frontal axis. These data support the hypothesis that conduction defects and diffuse left ventricular damage do not emanate from anatomical coronary lesions.

Key words: bifascicular block, coronary artery disease, regional wall motion, right bundle-branch block

Introduction

Though the appearance of complete right bundle-branch block (RBBB) during coronary artery disease (CAD) is frequently an ominous sign,[1-4] many questions remain as to its overall clinical implications.

The development of RBBB during acute myocardial infarction has been shown to be associated with a high incidence of pump failure syndrome.[1,3,4-7] The high mortality rate seems to be due to extensive myocardial damage rather than to the conduction disorder itself.[1-3]

However, the significance of complete RBBB in patients with chronic CAD is not clear. In patients surviving their most recent infarction who are New York Heart Association functional class I or II, the presence of RBBB does not seem to contribute to the higher risk of mortality.[8] Other studies have demonstrated that hospital survivors of myocardial infarction complicated by RBBB have a higher mortality rate during the first year of follow-up than control subjects without conduction disturbances due to the extent of myocardial damage.[9-11]

Address for reprints:

Prof. Roberto Piero Dabizzi
Fisiopatologia Cardiovascolare
Istituto di Clinica Medica-Universita' di Firenze
Viale Morgagni 85
50134 Florence, Italy

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In the present study, we compared the data of 34 subjects with RBBB and CAD with those of 52 consecutive patients with ischemic heart disease without conduction disturbances. All of them underwent coronary arteriography, left ventricular angiography, and hemodynamic studies.

Our aim was to investigate whether the presence of RBBB implies severe CAD and more extensive myocardial damage.

Material and Methods

Among the subjects who underwent cardiac catheterization, coronary arteriography, and left ventricular angiography between 1980 and 1985 for investigation of chest pain suggestive of ischemic heart disease, we identified 34 patients (32 male, 2 female, age range 34–66, mean age 52) with electrocardiographic pattern of RBBB according to the NYHA criteria. In all patients conduction disturbance did not predate the clinical development of the CAD. All 34 patients had rest and/or effort angina; 19 subjects had had a previous myocardial infarction; moderate arterial hypertension was present in 3 cases.

These patients were compared with 52 consecutive patients admitted during 1984 and 1985 with ischemic heart disease without conduction disturbance. In the latter group, 49 patients were male and 3 female; age range was 34–71 (mean age 55). Thirty-seven subjects had had a previous myocardial infarction; moderate arterial hypertension was present in 8 cases. At admission, comparison between the two groups showed no significant difference in the NYHA functional class. All 52 subjects underwent coronary arteriography for angina at rest and/or effort angina.

Left ventricular angiography and selective coronary angiography were carried out using Sones' or Judkins' technique. Before coronary arteriography, left ventricular angiography in the right anterior oblique projection was obtained in each subject at the end of hemodynamic study, by injection of about 45 ml of contrast medium (Urographin 60%). Selective coronary angiography was performed by means of cineangiograms recorded in multiple projections. Film was exposed at 50 frames/s on 35 mm film using an Angioskope Siemens system. The end-diastolic and end-systolic ventricular silhouettes (extrasystolic and postextrasystolic cycles were not analyzed) as well as the magnification grid were digitized by means of a Kontron Cardio 200 computer. Left ventricular volumes and ejection fraction were evaluated by applying Simpson's rule. In each silhouette the apex and the midaortic valve point were identified in order to define the long axis, so that the baricentrum was automatically calculated. Our program evaluated the shifting of the reference points during systole. Thirty-six radial lines originating from the baricentrum were drawn from the posterior to the anterior edge of the ventricular silhouette; then radii percent systolic shortening was calculated. Radii from 1 to 6 represent the left ventricle posterobasal portion, from 7 to 16 the diaphragmatic region, from 17 to 20 the apical region, from 22 to 29 the anterolateral region, and from 30 to 36 the anterobasal region. Calculation of the percent shortening of the radii was performed by the floating method (i.e., by alignment of silhouettes on the baricentrum). We used Gensini's score[12] to estimate the degree of coronary artery involvement because it takes into account both the location and entity of coronary lesions and the presence of collateral vessels.

The differences between the groups were tested using Student's *t*-test for unpaired data or chi-square test when necessary.

Results

In 19 patients with RBBB, electrocardiographic analysis showed previous myocardial infarction (14 anterior, 4 inferior, and 1 lateral). Fifteen had no electrocardiographic evidence of previous myocardial infarction. The evaluation of QRS frontal axis distribution (according to the American Heart Association criteria),[13] in patients with RBBB, showed 20 cases with normal or slightly leftward axis (from +100 to −45 degrees), 11 with abnormal left-axis deviation (greater than −45 degrees), and 3 with right-axis deviation (from +100 to +120 degrees). No significant difference was found comparing the incidence or the location of previous myocardial infarction in patients with RBBB with that of the 52 subjects without conduction defects. In fact, 15 patients in the latter group had no electrocardiographic evidence of previous myocardial infarction. Of the subjects who had had myocardial infarction, 20 had anterior, 11 inferior, and 6 a lateral myocardial infarction.

The hemodynamic data of patients with RBBB and those designated as control subjects are reported in Tables I and II. In 26 patients with RBBB, left ventricular end-diastolic pressure was found to be abnormal (more than 15 mmHg); in 24 patients the end-diastolic volume was found to be increased (more than 100 ml); and in 16 patients the ejection fraction was less than 0.50. A higher value of left ventricular end-diastolic pressure ($p < 0.05$) was found in CAD patients with RBBB in comparison with the 52 patients without conduction defects. No significant differences between the two groups were found regarding left ventricular volume and ejection fraction.

In the RBBB group, coronary angiography showed a left dominance pattern in 3 patients, a balanced pattern in 2 patients, and a right dominance in the other patients. This distribution was not significantly different from that of the control CAD patients (5 with left dominance, 4 with balanced pattern, 43 with right dominance).

Selective coronary angiography showed evident stenosis in all 34 subjects with RBBB. Twenty patients had involvement of three coronary arteries and 9 patients had double-vessel disease (the left anterior descending artery and the right coronary artery in 5 patients, the left cir-

TABLE I Hemodynamic data of the 34 CAD patients with RBBB and of the 52 CAD patients without RBBB

	n	Age	LVEDP	LVEDVI	LVESVI	LVEF
CAD with RBBB	34	52±9	21.09[a]±7.37	126.94±38.7	67.97±39.47	.49±.17
CAD without RBBB	52	55±8	15.73±8.43	125.47±39.09	61.35±34.14	.54±.15

[a]$p<0.01$.

Abbreviations: CAD=coronary artery disease; RBBB=right bundle-branch block; LVEDP=left ventricular end-diastolic pressure (mmHg); LVEDI=left ventricular end-diastolic volume index (ml/m²); LVESVI=left ventricular end-systolic volume (ml/m²); LVEF=left ventricular ejection fraction. [a]$p<0.01$.

TABLE II Hemodynamic data of the 20 patients with RBBB and of the 14 patients with bifascicular block

	n	Age	LVEDP	LVEDVI	LVESVI	LVEF
RBBB with LH	14	54±8	22.50±8.34	127.64±41.72	69.21±40.39	.48±.16
RBBB without LH	20	50±9	20.10±6.43	126.75±36.88	67.10±38.79	.48±.20

Abbreviations: RBBB=right bundle-branch block; LH=left hemiblock; LVEDP=left ventricular end-diastolic pressure (mmHg); LVEDVI=left ventricular end-diastolic volume index (ml/m²); LVESVI=left ventricular end-systolic volume index (ml/m²); LVEF=left ventricular ejection fraction.

cumflex artery and the left descending coronary artery in 4 patients). Single-vessel disease (in all the anterior descending artery) was present in 5 patients.

Stenosis was present in the left main coronary artery in 5 patients; in 3 patients there was subocclusive stenosis (99%), and 2 patients had mild stenosis (50–75%).

Involvement of the anterior descending artery was significant in all patients: 5 patients had 50–75% narrowing, 24 had 75–99% narrowing, and 5 patients had complete occlusion.

Left circumflex artery showed a 50–75% diameter narrowing in 6 patients, a 75–99% narrowing in 11 patients, and complete occlusion in 3 patients.

First diagonal branch involvement was evident by the presence of major lesions (>90%) in 6 cases, and by minor lesions (50–75%) in 14 cases.

On the marginal branch, severe stenosis (75–99%) was found in 7 patients, mild stenosis (50–75%) in 13 patients, and complete occlusion in 2 patients.

The right coronary artery was occluded in 6 patients and showed a 75–99% narrowing in 6 patients, and a 50–75% narrowing in 13 patients.

No significant differences were found in the incidence of one-, two-, and three-vessel disease or in the vessels involved with respect to CAD control group. In the CAD group without RBBB, 29 patients had three-vessel disease, 16 double-vessel disease (left anterior descending artery and right coronary artery in 12, left circumflex artery and left descending coronary artery in 4 patients). Single-vessel disease was present in 7 subjects: 4 in the anterior descending artery, 3 in the right coronary artery.

Using Gensini's score, which takes into account both the location and entity of coronary lesions and the collateral vessels present, greater involvement of the right coronary artery in CAD patients than in RBBB patients was noted. No other significant differences were found (Tables III and IV).

More severe abnormalities of left ventricular wall motion were observed in patients with RBBB. Only seven of those subjects had normal contraction patterns. The site and degree of wall motion abnormalities in the RBBB patients and in CAD patients is shown in Table V.

In RBBB patients, quantitative wall motion analysis showed a minor radii shortening of the anterobasal, apical, and diaphragmatic regions of the left ventricle (Figs. 1 and 2).

We compared the group of patients with CAD and RBBB without myocardial infarction with the group of patients with CAD without either myocardial infarction or RBBB. The former group had a significantly lower involvement of the coronary arteries (left anterior descending coronary artery and right coronary artery) (Fig. 3A) and significantly more severe damage of the inferior wall of the left ventricle (a significant reduction in the shortening of radii from 7 to 13) (Fig. 4A) than the second group. No significant differences between the two groups were found regarding left ventricular end-diastolic pressure, left ventricular volumes, and ejection fraction.

A further comparison was performed between the patients with RBBB and myocardial infarction and the control group without conduction disturbances and with myocardial infarction in the same site. The patients with inferior wall myocardial infarction and RBBB had more severe asynergy of the posterobasal portion of the left ventricle (radii from 3 to 6) than did the control subjects (Fig. 3B). Gensini's score (Fig. 4B) and the hemodynamic

TABLE III Gensini's score of the 34 CAD patients with RBBB and of the 52 CAD patients without RBBB

	Total	LCMA	LAD	DB	LCF	MB	RCA
CAD with RBBB	69.00±42.60	5.88±18.96	32.62±26.93	2.65±2.90	14.85±22.74	4.82±7.82	8.15[a]±8.89
CAD without RBBB	74.02±44.61	6.53±17.15	33.10±24.65	4.92±8.88	9.84±19.70	3.82±6.53	15.96±12.33

[a]$p<0.01$.

Abbreviations: CAD=coronary artery disease; RBBB=right bundle-branch block; LCMA=left main coronary artery; LAD=left anterior descending artery; DB=first diagonal branch; LCF=left circumflex coronary artery; MB=marginal branch; RCA=right coronary artery.

TABLE IV Gensini's score of the 20 patients with RBBB and of the 14 patients with bifascicular block

	Total	LCMA	LAD	DB	LCF	MB	RCA
RBBB with LH	64.12±34.05	0.00±0.00	41.14±26.43	2.57±3.16	6.43±7.66	5.57±7.86	8.86±11.15
RBBB without LH	71.35±50.22	10.00±23.67	26.65±25.65	2.70±2.70	20.25±27.73	4.30±7.75	7.65±6.84

Abbreviations: RBBB=right bundle-branch block; LH=left hemiblock; LCMA=left main coronary artery; LAD=left anterior descending artery; DB=first diagonal branch; LCF=left circumflex coronary artery; MB=marginal branch; RCA=right coronary artery.

parameters were similar in both groups. Patients with anterior wall myocardial infarction and RBBB had a higher value of left ventricular end-diastolic pressure (27.9±6.0 vs. 18.3±11.0 mmHg, $p<0.01$) and a lower value of left ventricular ejection fraction (0.34±0.11 vs. 0.48±0.19, $p<0.05$) in comparison with the similar group of patients of the control group. In the former group there was a greater extent of myocardial damage (a significant reduction in the shortening of radii from 6 to 24 and from 29 to 35) (Fig. 3C). Using Gensini's score, no differences were present between the two groups (Fig. 4C).

Patients with RBBB were subdivided into two subsets: those with normal QRS frontal axis and those with abnormal left- or right-axis deviation. Hemodynamic and angiographic data did not show any significant difference between them. Moreover, the group with RBBB and abnormal axis deviation presented the same hemodynamic (Table II) and angiographic (Table IV and Fig. 2) pattern as the RBBB total group.

Discussion

Right bundle-branch block, as a complication of acute myocardial infarction, is associated with higher hospital mortality caused by heart failure due to extensive left ven-

TABLE V Qualitative wall motion analysis[a]

	Posterobasal region	Diaphragmatic region	Apical region	Anterolateral region	Anterobasal region	Total
Normokinesis						
RBBB	26	16	12	13	20	87
Controls	46	26	33	27	38	170
Hypokinesis						
RBBB	3	8	10	8	8	37
Controls	5	17	14	19	13	68
Akinesis						
RBBB	2	4	4	5	2	17
Controls	0	3	2	2	0	7
Dyskinesis						
RBBB	3	6	8	8	4	29
Controls	1	6	3	3	1	14

[a]The distribution of asynergy degree of the two groups was significantly different ($p<0.001$).

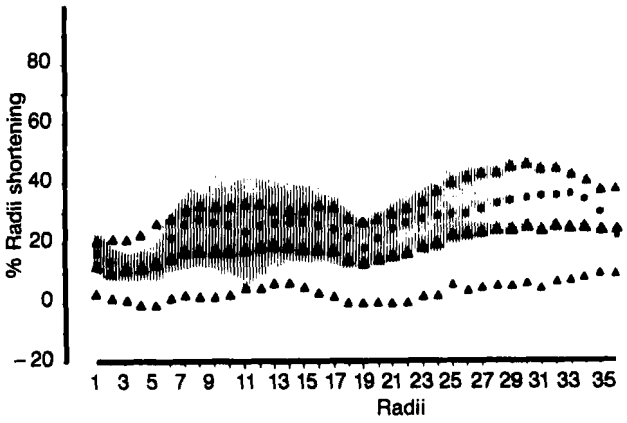

FIG. 1 Computerized wall motion analysis of the 34 patients with right bundle-branch block (RBBB, ▲) and of the 52 patients with CAD without conduction abnormalities (controls, ●). Radii are numbered from the posterobasal region to the anterobasal region.

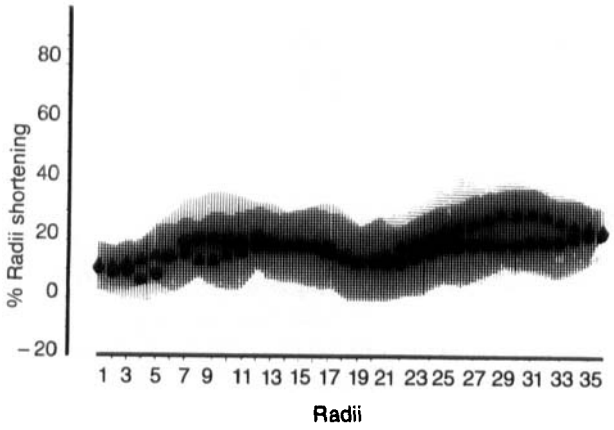

FIG. 2 Computerized wall motion analysis of the 20 patients with RBBB and of the 14 patients with bifascicular block. Radii are numbered from the posterobasal region to the anterolateral region. RBBB = right bundle-branch block; LH = left hemiblock. ● = RBBB with LH; ▲ = RBBB without LH.

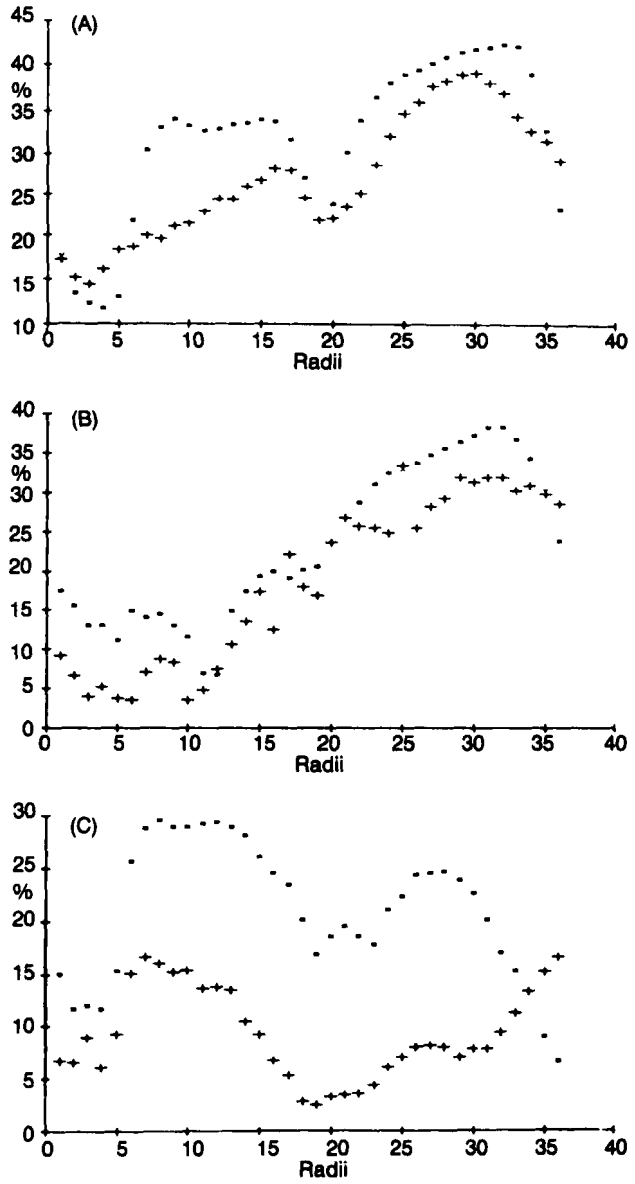

FIG. 3 Computerized wall motion analysis of the three subsets: (A) without myocardial infarction, (B) with inferior myocardial infarction, (C) with anterior myocardial infarction in the two groups: that with RBBB (+) and that without (−) conduction abnormalities. Radii are numbered from the posterobasal region to the anterobasal region. The x axis shows the percent radii shortening.

tricular myocardial necrosis. Some studies have also shown an ominous prognosis in patients with previous myocardial infarction complicated by RBBB.[9] This kind of patient has a tendency toward severe atrioventricular (AV) block.[1,14] Since some studies have shown that CAD subjects with bifascicular block often die of congestive heart failure, it may be useful to identify parameters which are associated with higher risk due to this complication.

Our data show severe and extensive myocardial damage in patients with RBBB and CAD and suggest that this damage may determine the poor prognosis and the development of cardiac failure. Moreover, the reported data show that in patients with CAD complicated by RBBB, serious hemodynamic derangement and severe left ventricular asynergy were present without necessarily more diffuse and severe coronary artery involvement.

In our study, patients with RBBB and CAD showed a significantly higher left ventricular end-diastolic pressure than patients without conduction disturbances. Higher left ventricular end-diastolic pressure was associated with left

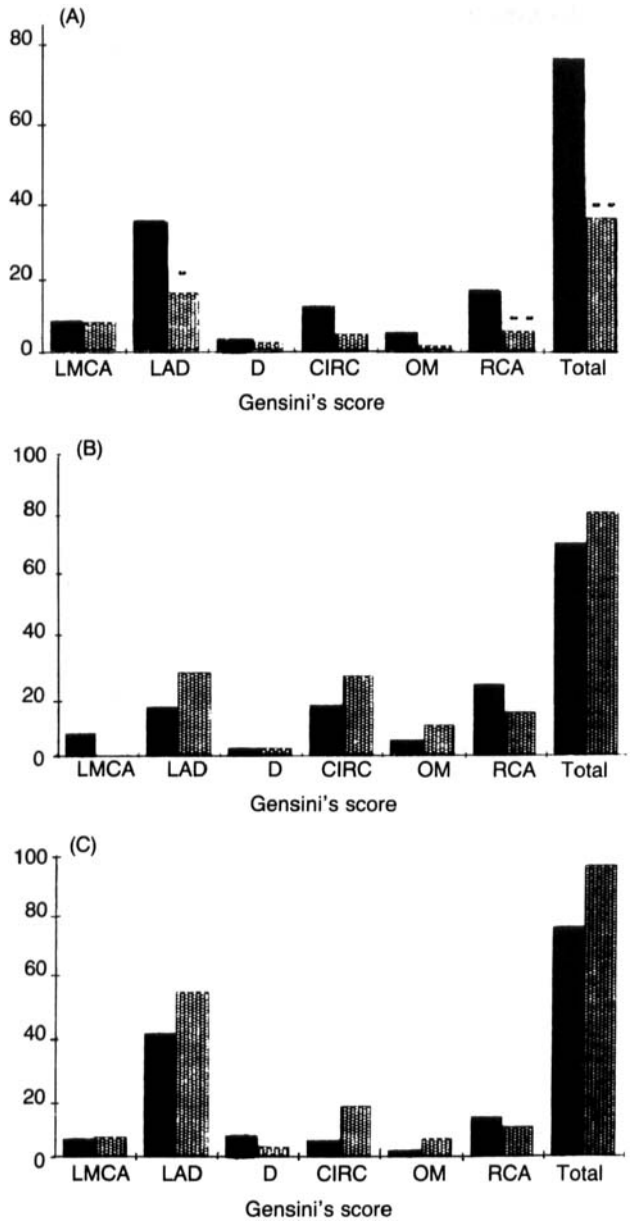

FIG. 4 Gensini's score of the three subsets: (A) without myocardial infarction, (B) with inferior myocardial infarction, (C) with anterior myocardial infarction in the two groups: that with (■) RBBB and that without (▣) conduction abnormalities. CIRC = left circumflex artery, D = first diagonal branch, LAD = left anterior descending artery, LMCA = left main coronary artery, OM = marginal branch, RCA = right coronary artery, * = $p < 0.05$, ** = $p < 0.005$.

ventricular end-diastolic volume similar to that found in control CAD patients and suggested more severe derangement of the left ventricle with diffuse areas of fibrosis. This was particularly evident in the subset of patients with anterior myocardial infarction and RBBB.

Patients with RBBB and CAD had a higher frequency of severe dysfunction in left ventricular wall motion with more diffuse regional involvement than CAD control patients. This was also confirmed in the subsets of patients with or without myocardial infarction.

The extent of coronary artery disease did not differ between the two groups except for a lesser involvement of the anterior descending coronary artery and the right coronary artery in CAD patients without myocardial infarction and RBBB. This finding could explain the slight prevalence of inferior myocardial infarction in the CAD control group (30 vs. 21%, not statistically significant), but it is insufficient to explain the greater wall motion derangement in RBBB patients. We did not find specific coronary lesions in RBBB patients nor, in contrast to other studies,[15] more severe involvement of coronary arteries in subjects with bifascicular block when compared with the control group without conduction defects.

Also Harper *et al.*[16] found poor correlations between chronic coronary artery disease and specific conduction abnormality.

Moreover, the analysis performed in the subset without myocardial infarction showed that the patients with RBBB had a lesser coronary involvement and a worse regional wall motion. Our data do not explain the localization of the regional damage to the inferior region in these patients. The inferior region is also involved in the subset with anterior and inferior myocardial infarction. In agreement with Haft *et al.*,[17] we think that conduction defects and diffuse left ventricular damage in CAD patients may occur during prolonged ischemia by total occlusion with subsequent partial recanalization. The right bundle branch, usually small in size, is supplied primarily by the left descending coronary artery and secondly by the right coronary artery.[18] Only later, when the damage of the cardiac muscle and of the conduction pathways is irreversible, collateral vessels may develop. This could explain why the control patients with more severe involvement of the right coronary artery had minor contraction abnormalities.

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