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Prognostic Significance of Left Anterior Hemiblock in Patients With Suspected Coronary Artery Disease

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OBJECTIVES This study was designed to assess the functional and prognostic significance of left anterior hemiblock (LAHB) in patients with no history of myocardial infarction referred for dobutamine stress echocardiography (DSE).

BACKGROUND The significance of isolated LAHB in patients with suspected coronary artery disease (CAD) is unclear.

METHODS We studied 1,187 patients with suspected CAD and no history of myocardial infarction who underwent DSE and were followed for occurrence of cardiac death.

RESULTS Left anterior hemiblock was detected on baseline electrocardiogram in 159 patients (13%). Ischemia occurred more frequently in patients with LAHB (43% vs. 33%, $p = 0.02$). During a mean follow-up of 5.0 ± 2.5 years, 125 patients (11%) died of cardiac causes. The annual cardiac death rate was 4.9% in patients with LAHB and 1.9% for patients without ($p < 0.0001$). Patients with both LAHB and an abnormal DSE had the highest annual cardiac death rate (6.3%). In a Cox multivariable analysis, independent predictors of cardiac death were age, smoking, history of heart failure, diabetes, and ischemia. Left anterior hemiblock was independently associated with increased risk of cardiac death among patients with normal DSE (hazard ratio 1.8, 95% confidence interval 1.1 to 3.8) and in patients with abnormal DSE (hazard ratio 1.7, 95% confidence interval 1.1 to 2.7).

CONCLUSIONS In patients with suspected CAD referred for stress testing, LAHB is associated with increased risk of cardiac death. This risk is persistent after adjustment for major clinical data and abnormalities on the stress echocardiogram. Therefore, isolated LAHB should not be considered a benign electrocardiographic abnormality in these patients. (J Am Coll Cardiol 2005;46:858–63) © 2005 by the American College of Cardiology Foundation

Data on prognostic significance of left anterior hemiblock (LAHB) are scarce, particularly among patients without a history of myocardial infarction (MI) (1). In subjects with no evidence of cardiac disease, some investigators found a marginally increased incidence of coronary artery disease (CAD) in patients with LAHB (2). No association has been found between the presence of LAHB and an increased risk of cardiac death (3–5). However, published data are limited by small numbers of patients and short-term follow-up. Left anterior hemiblock is a well-recognized complication that occurs in 3% to 5% of patients after acute MI. In these patients, LAHB is believed to be due to ischemic injury of the anterior fascicle of the left conduction system. In patients with acute inferior MI, LAHB was associated with a larger infarct extension and left anterior descending CAD (6). However, its occurrence in patients after acute MI has not been related to a higher mortality rate (7–10). We sought to study the association of LAHB with abnormalities on the stress echocardiogram and prognosis in patients with suspected CAD who have no history of MI.

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METHODS

Patients. The study population consisted of consecutive patients with suspected CAD referred for dobutamine stress echocardiography (DSE) at the Thoraxcenter, Rotterdam, the Netherlands. Exclusion criteria were history of MI, cardiac pacemaker, pathologic Q-wave, and complete left or right bundle branch block on the baseline electrocardiogram (ECG). Criteria were fulfilled in 1,199 patients. Follow-up was successful in 1,187 patients (99%), who represented the final population of the study. Patients were divided into two groups according to the presence or absence of LAHB on the baseline ECG. Criteria for LAHB were leftward QRS axis of −30° to −90°, with rS patterns in leads II, III, and aVF, and Q waves in aVL (11,12). Left ventricular hypertrophy (LVH) was defined using the Cornell voltage-duration product, which was calculated as follows: RaVL ± SV3 (with 6 mm added in women) × QRS complex duration. A threshold of 2,440 mm × ms was used to identify LVH (13,14). The duration of the QRS complex was electronically measured by a 12-lead ECG performed in the resting supine position (Escribe v15.25, Mortara Instruments, Milwaukee, Wisconsin). QRS complex detection by this system used a filter and template matching technique to identify a median QRS complex, which was then aligned in all leads simultaneously. QRS complex duration was measured from the earliest onset in any lead to the latest

Abbreviations and Acronyms

CAD = coronary artery disease
CI = confidence interval
DSE = dobutamine stress echocardiography
ECG = electrocardiogram
HR = hazard ratio
LAHB = left anterior hemiblock
LVH = left ventricular hypertrophy
MI = myocardial infarction

detection in any lead. The protocol was approved by the hospital ethics committee. All patients gave informed consent before the test. Clinical characteristics and indications for testing were entered in a computerized database before DSE and were defined as previously described (15).

Dobutamine stress test. Dobutamine-atropine stress testing was performed according to a standard protocol as previously reported (16). After a baseline echocardiogram, dobutamine was administered intravenously, starting at a dose of 5 to 10 μg/kg/min for 3 min. Incremental dobutamine doses of 10 μg/kg/min were given at 3-min intervals up to a maximum dose of 40 μg/kg/min. If the test end point was not reached at a dobutamine dose of 40 μg/kg/min, atropine (up to 2 mg) was given intravenously. Blood pressure, heart rate, and electrocardiography were constantly monitored. Test end points were achievement of target heart rate (85% of maximum age- and gender-predicted heart rate), maximal dose of dobutamine and atropine, extensive new wall motion abnormalities, >2 mV downsloping ST-segment depression measured 80 ms after the J point compared with baseline, hypertension (blood pressure >240/120 mm Hg), a decrease in systolic blood pressure of >40 mm Hg compared with at rest, significant arrhythmias, or any intolerable adverse effect considered to be the result of dobutamine or atropine. An intravenous beta-blocker (metoprolol 1 to 5 mg) was available to reverse the adverse effects of dobutamine/atropine.

Echocardiographic imaging and interpretation. Two-dimensional echocardiographic images were acquired at rest, during dobutamine stress, and recovery using the standard views. The echocardiograms were recorded in a quad-screen format. The interpretation of DSE studies was performed offline from cine loops by two experienced observers blinded to the clinical and electrocardiographic data. In case of disagreement, a consensus decision was reached with a third observer. Regional function was scored using a 16-segment, 5-point scoring model (1 = normal, 2 = mild hypokinesia, 3 = severe hypokinesia, 4 = akinesia, 5 = dyskinesia). Ischemia was defined as new or worsened wall motion abnormalities during stress indicated by an increase of wall motion score ≥1 grade in ≥1 segment. A biphasic response in an akinetic or severely hypokinetic segment was considered as an ischemic response. An abnormal DSE was defined as resting or inducible wall motion abnormalities. The wall motion score index was calculated by dividing the sum of regional wall motion scores by the total number of interpreted segments.

Follow-up. Follow-up data collection was performed by contacting the patient's general practitioner and by review of hospital records. The date of the last review or consultation was used to calculate follow-up time. Follow-up events noted were overall mortality and hard cardiac events (nonfatal MI and cardiac death). Cardiac death was defined as a death caused by acute MI, significant cardiac arrhythmias, or refractory congestive heart failure. Sudden death occurring without another explanation was considered as cardiac death. Nonfatal MI was defined based on criteria of typical chest pain, elevated cardiac enzyme levels, and typical changes on electrocardiography. Myocardial revascularization procedures were also noted.

Statistical analysis. Continuous data were expressed as mean value ± SD. The Student *t* test was used to analyze continuous data. Differences between proportions were compared using the chi-square test. The following major clinical and stress test data were used in a Cox regression model to identify independent predictors of cardiac events: age, gender, smoking, hypertension, diabetes mellitus, history of heart failure, angina, LAHB, QRS duration, LVH on baseline ECG, ST-segment depression during stress, peak stress rate pressure product, peak wall motion score index, and myocardial ischemia on DSE. The risk of a variable was expressed as a hazard ratio (HR) with a corresponding 95% confidence interval (CI). The probability of survival was calculated using the Kaplan-Meier method, and survival curves were compared using the log-rank test. A p value <0.05 was considered statistically significant.

RESULTS

Clinical and stress test data. Clinical characteristics are presented in Table 1. Patients with LAHB were older and more often had a history of hypertension and heart failure and had a larger QRS complex duration. Dobutamine stress data are presented in Table 2. Peak heart rate was lower in patients with LAHB, reflecting the lower maximal predicted heart rate due to older age in patients with than without LAHB. Ischemia (new or worsened wall motion abnormalities) occurred more frequently in patients with than without LAHB. Resting wall motion score index was comparable between the two groups, whereas the peak wall motion score index was significantly higher in patients with LAHB. Regional distributions of stress wall motion abnormalities in patients with and without LAHB are presented in Figure 1. Patients with LAHB had a higher incidence of abnormalities in the anterior, apical, and lateral walls compared with patients without LAHB.

Follow-up data. During a mean follow-up of 5.0 ± 2.5 years, 283 patients (24%) died, 125 (11%) from cardiac causes. Nonfatal MI occurred in 54 patients (5%); revascularization was performed in 177 (15%) patients. Patients

Table 1. Clinical Characteristics of Patients With and Without LAHB

	LAHB		
	Yes (n = 159)	No (n = 1,028)	p Value
Age (yrs, standard deviation)	66 ± 11	60 ± 13	<0.0001
Men	95 (60%)	581 (56%)	0.5
Hypertension	70 (44%)	363 (35%)	0.04
Hypercholesterolemia	32 (20%)	275 (27%)	0.09
Smoking	40 (25%)	252 (24%)	0.9
Diabetes mellitus	15 (9%)	140 (14%)	0.2
History of typical angina	25 (16%)	215 (21%)	0.2
Atypical chest pain	33 (21%)	281 (27%)	0.1
History of heart failure	16 (10%)	56 (5%)	0.04
LVH by ECG criteria	14 (9%)	90 (9%)	1.0
QRS duration	109 ± 26	95 ± 14	<0.0001
Beta-blockers	52 (33%)	338 (33%)	1.0
Calcium channel blockers	45 (28%)	262 (25%)	0.5
ACE inhibitors	44 (28%)	212 (21%)	0.06
Nitrates	32 (20%)	167 (16%)	0.3

ACE = angiotensin-converting enzyme; ECG = electrocardiogram; LAHB = left anterior hemiblock; LVH = left ventricular hypertrophy.

with LAHB had a higher incidence of overall mortality (53 [33%] vs. 230 [22%], p = 0.004) and cardiac mortality (32 [20%] vs. 93 [9%], p < 0.0001) compared with patients without LAHB. Nonfatal MI occurred in 11 patients (7%) with LAHB and in 43 patients (4%) without (p = 0.1). Among patients with normal DSE, MI occurred in 4 of 69 patients (6%) with LAHB and in 19 of 497 patients (4%) without LAHB (p = 0.6). Six of 69 patients (9%) with LAHB and a normal DSE underwent subsequent coronary angiography during follow-up. Coronary artery disease was detected in four of these patients. Cardiac death occurred in 11 of 69 patients (16%) with LAHB and a normal DSE and in 33 of 497 patients (7%) without LAHB and a normal DSE (p = 0.01).

Table 2. Dobutamine Stress Hemodynamic and Echocardiographic Data

	LAHB		
	Yes (n = 159)	No (n = 1,028)	p Value
Heart rate at rest (beats/min)	72 ± 13	74 ± 14	0.2
Heart rate at peak (beats/min)	126 ± 16	130 ± 17	0.002
Patients who achieved target heart rate	140 (88%)	888 (86%)	0.6
Rest systolic blood pressure (mm Hg)	136 ± 16	133 ± 23	0.2
Peak systolic blood pressure (mm Hg)	140 ± 24	138 ± 25	0.5
Rest rate pressure product	9,914 ± 2,648	9,877 ± 2,501	0.9
Peak rate pressure product	16,796 ± 4,262	17,506 ± 4,299	0.07
Maximal dobutamine dose (μg/kg/min)	33 ± 9	34 ± 8	0.1
Atropine use, patients	54 (6%)	391 (9%)	0.4
ST-segment depression	9 (6%)	98 (9%)	0.2
Resting wall motion score index	1.1 ± 0.2	1.1 ± 0.3	0.2
Peak wall motion score index	1.3 ± 0.5	1.2 ± 0.4	0.02
Normal DSE, patients	69 (43%)	497 (48%)	0.3
Fixed abnormalities, patients	21 (13%)	188 (18%)	0.1
New or worsening wall motion abnormalities, patients	69 (43%)	343 (33%)	0.02

DSE = dobutamine stress echocardiography; LAHB = left anterior hemiblock.

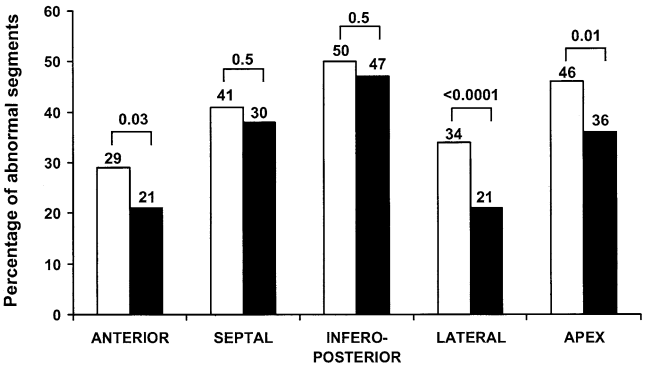

Figure 1. Regional distribution of abnormalities on the dobutamine stress echocardiogram in patients with and without left anterior hemiblock (LAHB). **White bars** = LAHB; **black bars** = no LAHB.

Tables 3 and 4 present predictors of cardiac death and all-cause mortality, respectively.

Age, smoking, history of heart failure, diabetes, LAHB, and myocardial ischemia during DSE were independent predictors of cardiac death. Age, gender, smoking, history of heart failure, LVH, LAHB, and myocardial ischemia during DSE were independent predictors of all-cause mortality. Left anterior hemiblock was independently associated with increased risk of cardiac death among patients with normal DSE (HR 1.8, 95% CI 1.1 to 3.8) as well as in patients with abnormal DSE (HR 1.7, 95% CI 1.1 to 2.7). The annual cardiac death rate was 1.9% for patients without LAHB and 4.9% for patients with LAHB (p < 0.0001). The hard event rate was 2.5% for patients without LAHB and 5.9% for patients with LAHB (p < 0.0001).

Kaplan-Meier survival curves in patients with normal

Table 3. Univariate and Multivariate Association of Clinical, Electrocardiographic, and Echocardiographic Data With Cardiac Death

Parameter	Univariate Hazard Ratio (95% CI)	Multivariate Hazard Ratio (95% CI)
Age	1.06 (1.04–1.07)	1.05 (1.03–1.08)
Gender	1.9 (1.4–2.6)	1.4 (0.9–2.1)
Smoking	2.1 (1.6–2.8)	2.0 (1.3–3.1)
Hypertension	0.9 (0.7–1.3)	1.2 (0.7–1.8)
History of heart failure	4.2 (2.5–6.8)	1.8 (1.1–3.3)
Diabetes mellitus	1.4 (0.9–2.1)	1.4 (1.1–2.4)
History of typical angina	0.8 (0.5–1.2)	0.4 (0.2–1.0)
LAHB	2.5 (1.6–4.0)	1.7 (1.1–2.8)
QRS duration (ms)	4.8 (2.1–11)	1.16 (0.3–4.2)
LVH by ECG criteria	1.6 (0.9–2.8)	1.3 (0.7–2.5)
ST-segment depression	0.8 (0.4–1.4)	1.0 (0.5–2.1)
Peak rate pressure product	0.97 (0.94–1.00)	0.01 (0.95–1.07)
Peak wall motion score index	3.04 (2.39–3.86)	2.13 (0.94–4.85)
Myocardial ischemia	1.9 (1.2–2.7)	1.8 (1.1–2.9)

CI = confidence interval. Other abbreviations as in Table 1.

Table 4. Univariate and Multivariate Association of Clinical, Electrocardiographic, and Echocardiographic Data With All-Cause Mortality

Parameter	Univariate Hazard Ratio (95% CI)	Multivariate Hazard Ratio (95% CI)
Age	1.03 (1.02–1.04)	1.04 (1.02–1.06)
Gender	1.9 (1.5–2.4)	1.9 (1.4–2.7)
Smoking	1.6 (1.3–2.1)	1.7 (1.3–2.3)
Hypertension	0.9 (0.8–1.2)	1.1 (0.9–1.5)
History of heart failure	3.8 (2.5–5.9)	1.9 (1.3–3.0)
Diabetes mellitus	1.1 (0.8–1.6)	1.3 (0.9–2.0)
History of typical angina	0.8 (0.6–1.1)	0.8 (0.5–1.2)
LAHB	1.7 (1.2–2.5)	1.4 (1.1–2.0)
QRS duration (ms)	0.64 (0.3–1.6)	0.71 (0.3–1.6)
LVH by ECG criteria	1.8 (1.5–2.6)	1.5 (1.1–2.3)
ST-segment depression	0.6 (0.4–1.0)	0.9 (0.6–1.6)
Peak rate pressure product	0.95 (0.92–0.99)	0.93 (0.90–0.97)
Peak wall motion score index	1.39 (0.76–2.59)	1.38 (0.75–2.57)
Myocardial ischemia	1.8 (1.2–2.6)	1.5 (1.1–2.2)

Abbreviations as in Table 3.

DSE (Fig. 2A) and in patients with abnormal DSE (Fig. 2B) demonstrated a lower event-free survival among patients with LAHB in both groups. The annual cardiac event rates, according to a combination of LAHB and abnormalities on DSE, are presented in Figure 3. The presence of LAHB in both groups of patients with normal and abnormal DSE identified a significantly higher risk population for cardiac death and hard cardiac events. Patients with both

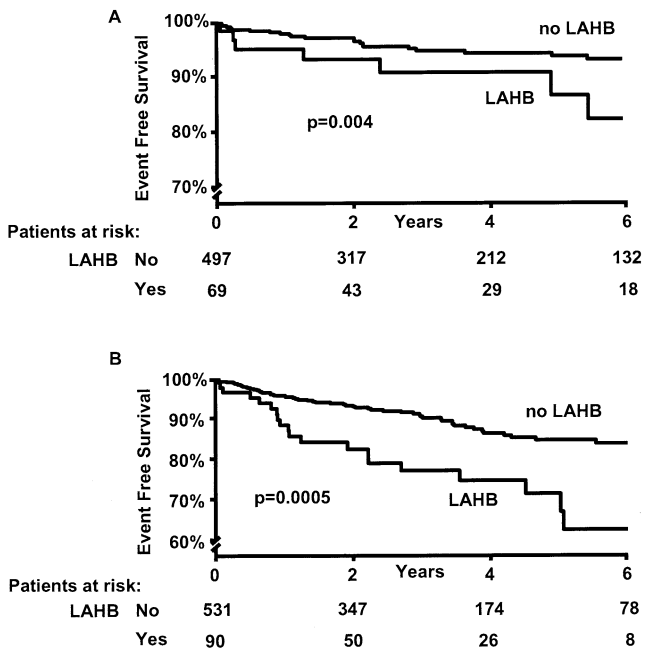

Figure 2. Kaplan-Meier survival curves (end point of cardiac death) in the presence and in the absence of left anterior hemiblock (LAHB) in patients with normal dobutamine stress echocardiogram **(A)** and in patients with abnormal dobutamine stress echocardiogram **(B)**.

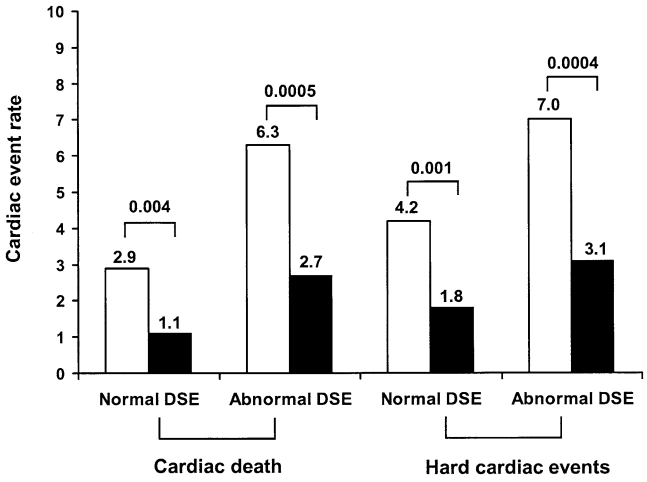

Figure 3. Annual cardiac event rates according to combinations of electrocardiographic and stress echocardiographic findings. **White bars** = LAHB; **black bars** = no LAHB. DSE = dobutamine stress echocardiography; LAHB = left anterior hemiblock.

LAHB and abnormal DSE were at the highest risk of cardiac death.

DISCUSSION

In this study, we assessed the functional and prognostic significance of LAHB on a resting 12-lead ECG in patients with suspected CAD who had no history or electrocardiographic evidence of previous MI. Left anterior hemiblock was detected in 159 patients (13%). Patients with LAHB had 2.5-fold higher incidence of cardiac death and 1.5-fold higher incidence of all-cause mortality as compared with patients without LAHB during a mean follow-up of five years. The associated risk of death was independent of clinical parameters, QRS duration, LVH on the ECG, and abnormalities on the DSE. The higher incidence of cardiac death in association with LAHB was observed among patients with normal as well as patients with abnormal DSE. The combination of LAHB and abnormal DSE identified patients with the highest incidence of cardiac death and hard cardiac events.

Echocardiographic data and prognosis. Patients with LAHB had a 10% higher incidence of ischemia on DSE. This suggests that LAHB is associated with a higher prevalence of significant CAD. However, this modest difference alone does not explain the large difference in cardiac mortality, particularly with the similar resting wall motion score index in both groups and the fact that the Cox model adjusted for myocardial ischemia. The reason for the independent association of LAHB with cardiac mortality after adjustment for DSE data is unclear. It is possible that LAHB is associated with ultrastructural myocardial damage that cannot be grossly identified by echocardiographic imaging. Because of the small size of the left anterior fascicle, it is vulnerable to blockage in the presence of trivial ischemia in the region of its course. Another explanation could be the higher propensity for fatal arrhythmias in the

presence of a substrate of myocardial dysfunction, LAHB, and triggering ischemia. It is also possible that patients with LAHB have a higher rate of false-negative DSE. However, because of the very small number of patients who underwent coronary angiography during follow-up, a lower sensitivity of DSE in patients with LAHB remains speculative.

Left anterior hemiblock and LVH were independent predictors of all-cause mortality, whereas QRS complex duration was not. The lack of an independent association of QRS complex duration with outcome may be explained by exclusion of patients with complete bundle branch block and patients with previous MI, thereby including a population with relatively preserved left ventricular function.

Regional abnormalities with LAHB. The occurrence of LAHB in association with myocardial ischemia is thought to be related to left anterior descending CAD (17,18). However, there are no previous reports on tomographic localization of functional abnormalities in these patients. Analysis for regional distribution of stress wall motion abnormalities in our study showed a higher incidence of abnormalities in the anterior and apical regions in patients with compared with patients without LAHB. This is in concordance with anatomic studies showing that the left anterior division of the left bundle branch block runs across the septal surface toward the antero-lateral aspect of the free wall of the left ventricle and is supplied by the left anterior descending coronary artery. The higher incidence of abnormalities in the lateral wall among patients with LAHB, with a similar incidence of abnormalities in the posterior and inferior walls, suggests that abnormalities noted in the lateral wall are due to diagonal rather than left circumflex CAD in some of these patients.

Previous studies. Previous data on the correlation between LAHB and cardiac disease in the general population are scarce (2–4). Corne et al. (2) found a relation with systemic hypertension and cardiac disease in 390 men ages 30 years and over with LAHB compared with an age- and gender-matched control group. Studies of the prognostic significance of isolated LAHB in the general population or in subjects without clinical evidence of cardiac disease are limited, but have reported no effect of LAHB on mortality (3,4). Yano et al. (3) followed 70 clinically normal subjects with LAHB for periods ranging from three to six years and found no increase in the mortality risk ratio compared with normal control patients. Ostrander (4) analyzed data from the Tecumseh Communities Health Study and found that 102 subjects with left axis deviation and no other findings suggestive of heart disease had no excess of morbidity or mortality during an average observation of four years.

Left anterior hemiblock is not uncommonly observed in patients with acute MI. Development of LAHB during inferior acute MI has been correlated with significant stenosis in left anterior descending coronary artery and multivessel disease (6–19). Assali et al. (6) studied 87 patients with a first inferior wall acute MI. Seventeen (19%) developed a LAHB. Significant stenosis of the left anterior descending artery was found in 82% of patients with LAHB and in 21% of patients without LAHB ($p = 0.001$). By logistic regression analysis, the odds ratio for development of LAHB was 27.5 ($p = 0.0001$) for left anterior descending artery stenosis compared with 2.9 ($p = 0.05$) and 0.3 ($p = 0.3$) for circumflex and right coronary artery, respectively. However, LAHB after acute MI has not been found to be related to an increased mortality (7–10).

Information regarding the prognostic significance of LAHB in patients referred for stress test is limited. In the study by Cortigiani et al. (20), the prognostic implication of intraventricular conduction defects in patients with suspected CAD referred for pharmacologic stress echocardiography was evaluated during a mean follow-up of three years. Out of a total of 1,230 patients, 173 had complete left bundle branch block, 98 isolated right bundle branch block, 43 right bundle branch block plus LAHB, and 106 isolated LAHB. The presence of right bundle branch block together with LAHB was associated with a poor prognosis and was an independent predictor of mortality. The presence of isolated LAHB was not associated with all-cause mortality in a multivariate analysis.

Study limitations. Left ventricular mass was not evaluated because the study was not prospectively designed to obtain the required measurements, which may limit the feasibility of these calculations. Left ventricular hypertrophy was evaluated by electrocardiographic criteria, with possible underestimation of prevalence of LVH if defined by echocardiographic criteria (21). Nevertheless, LVH as defined by electrocardiography was an independent predictor of mortality in this study, despite possible underestimation of the true incidence of LVH. The ECG has been generally used as an acceptable method to define LVH in outcome trials (22). Finally, this study could not determine whether the increased risk in association with LAHB is related to a lower sensitivity of DSE in patients with LAHB, because only a small number of patients underwent coronary angiography. Further studies are needed to assess the diagnostic performance of DSE in patients with LAHB.

Conclusions. Isolated LAHB is an independent predictor of total and cardiac mortality in patients with suspected CAD who have no history of MI. The increased risk of mortality is persistent after adjustment for clinical data, left ventricular function, and inducible ischemia on DSE. Among patients with a normal DSE, the presence of LAHB is associated with a worse prognosis. Patients with both LAHB and abnormal DSE had the worst outcome. Therefore, isolated LAHB should not be considered a benign electrocardiographic abnormality in these patients and should be considered along with other clinical and stress test data in estimating the risk of cardiac events.

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