

## Bundle-Branch Block in a General Male Population The Study of Men Born 1913

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**Background**—Interest in bundle-branch block has focused primarily on its role as a predictor of mortality and coexisting cardiovascular diseases. Previous studies of prevalence, correlation to cardiovascular disease, and mortality have produced conflicting results.

**Methods and Results**—We studied a random-sampled population of 855 men who were 50 years old in 1963 and followed them up for 30 years with repeated examinations. Men who developed bundle-branch block were studied with regard to cumulative incidence, relationship with cardiovascular disease/risk factors, and survival. The prevalence of bundle-branch block increases from 1% at age 50 years to 17% at age 80 years, resulting in a cumulative incidence of 18%. No significant relationship with ischemic heart disease or mortality was found. Men who would develop bundle-branch block had a bigger heart volume at age 50 years and developed diabetes mellitus and congestive heart disease during follow-up more often than control subjects.

**Conclusions**—Bundle-branch block correlates strongly to age and is common in elderly men. Our results support the theory that bundle-branch block is a marker of a slowly progressing degenerative disease that also affects the myocardium. (*Circulation*. 1998;98:2494-2500.)

**Key Words:** bundle-branch block ■ epidemiology ■ population ■ survival

In the literature, interest in bundle-branch block has focused primarily on its role as a predictor of mortality and coexisting cardiovascular diseases. The epidemiological data were derived mostly from hospitalized patients,<sup>1-8</sup> thus being confounded by the reason for performing a standard 12-lead ECG. Others have studied healthy populations on a routine check-up basis<sup>9-12</sup> and were often confounded by age. In the Framingham Heart study, it was concluded that people with acquired bundle-branch block were more likely to have or to subsequently develop advanced cardiovascular manifestations, especially men with left bundle-branch block.<sup>13</sup> In the Reykjavik studies, no increased death rate due to coronary artery disease or hypertension was observed among people with left bundle-branch block.<sup>14</sup> In people <60 years old with right bundle-branch block, there was a positive correlation to hypertension.<sup>15</sup> Several studies have found increased mortality in patients with bundle-branch block and concomitant cardiovascular disease.<sup>1,3,8,16</sup> In patients with acute myocardial infarction, the presence of bundle-branch block is a marker of worse outcome<sup>17,18</sup> that persists in the modern era of thrombolytic therapy.<sup>19</sup> Among patients with chronic coronary artery disease, bundle-branch block has been shown to be a strong predictor of mortality, independent of the degree of heart failure and extent of coronary disease.<sup>16</sup>

Whether it is the pathogenesis and morphology of the bundle-branch block itself or the relationship or combination with ischemic heart disease that has an impact on mortality is unclear.

As far as we know, no previous study has recorded 12-lead ECGs in a random sample of 50-year-old men with a 30-year follow-up. The aim of our study was to describe the cumulative incidence of bundle-branch block and its relationship with cardiovascular disease, risk factors, and prognosis.

### Methods

#### Study Population

The Study of Men Born in 1913 is a longitudinal prospective study of men born in 1913 and living in the city of Göteborg on the west coast of Sweden.<sup>20</sup> In 1963, Göteborg had ≈500 000 inhabitants. All residents in Sweden have a unique national 10-digit registration number based on their date of birth. The County Census Bureau is required by law to keep registration numbers, names, and addresses up to date in an official computerized register. In 1963, a sample was drawn from the population register consisting of all men born in 1913 on a day divisible by 3 (ie, day 3, 6, 9, etc, of each month) and living in the city of Göteborg. These criteria were fulfilled by 973 men, 855 (88%) of whom agreed to participate in a health examination. From the baseline examination in 1963, when all the men were 50 years old, 855 men have been followed up for 30 years with repeated examinations (in 1967, 1973, 1980, 1988, and 1993). Those who did not participate in the examinations were asked to fill out questionnaires or participate in telephone interviews. Information about hospitalization, medication, and morbidity since the

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TABLE 1. Study Population

Year of Examination	Possible Participants (n)	Dead or Lost to Follow-Up* (n)	No ECG Available (n)	In the Study With ECG Available (n)
1963	855	...	1	854 (99.9%)
1980	697	158	130	567 (81%)
1988	518	337	123	395 (76%)
1993	361	494	149	212 (59%)

Possible participants indicates all men alive and not lost to clinical follow-up; dead or lost to follow-up, cumulative numbers are shown; and no ECG available indicates at that particular examination.

\*A total of 13 patients (1.5%) were lost to clinical follow-up over the 30-year follow-up period.

previous examination was obtained at each examination. Death certificates, autopsy reports, and medical records were studied for those who died. The participants and nonparticipants have previously been described in detail (References 20 to 23). Systematic 12-lead ECG recordings were made in 1963, 1980, 1988, and 1993 and form the basis of the present study (Table 1).

## ECG

Standard 12-lead ECGs were recorded with the patients at rest in the supine position. Paper speed was 50 mm/s, and calibration was 1 mV:10 mm. All 12-lead ECGs were read by one of the authors (P.E.), who was blinded to all data, and were classified as to whether bundle-branch block was present or not.

Left bundle-branch block was defined as (1) QRS duration  $\geq 120$  ms, (2) PQ interval  $>120$  ms, (3) predominantly upright complexes with slurred R waves in leads I,  $V_5$ , and  $V_6$ , and (4) QS or rS pattern in  $V_1$ .

Right bundle-branch block was defined as (1) QRS duration  $\geq 120$  ms, (2) PQ interval  $>120$  ms, (3) rSR' in lead  $V_1$  or  $V_2$ , and (4) S waves in lead I and either lead  $V_5$  or  $V_6$ . They were further classified according to the QRS axis in the presence of right bundle-branch block, in which an axis  $<-30^\circ$  or  $>+90^\circ$  indicated a possibility of a bifascicular block (concomitant left anterior hemiblock or left posterior hemiblock, respectively).<sup>24</sup>

If atrial fibrillation was present, the ECG was still included as bundle-branch block even if the criterion of PQ interval  $>120$  ms could not be fulfilled. Left ventricular hypertrophy was defined as Sokolow-Lyon criterion  $>3.5$  mV<sup>25</sup> and was measured only in men with QRS complex  $<120$  ms before they developed bundle-branch block.

## Baseline Examinations in 1963

Participants were examined in the morning after an overnight fast. Body weight was measured with a balance scale to the nearest 0.1 kg with the men wearing light indoor clothing. Height was measured to the nearest centimeter. Body mass index was calculated as weight (kg) divided by the square of height ( $m^2$ ).

Blood pressure was recorded in the right arm, with the participant seated after a 5-minute interview. A mercury sphygmomanometer with a cuff size of 12 $\times$ 23 cm was used. All blood pressures were measured by the same observer to the nearest 2 mm Hg.

Chest radiographs were taken during inspiration in the frontal, left lateral, and 2 oblique projections. Radiographs were interpreted by 2 experienced radiologists who had no other information about the participants. Absolute heart volume (mL) was measured on the radiograph according to Jonsell.<sup>26</sup>

Blood samples were drawn from an antebachial vein for determination of serum cholesterol, serum triglycerides, and blood glucose. Information on smoking habits was obtained by questionnaire.

## Follow-Up

From the baseline examination in 1963, 855 men were followed up for a mean of  $30.5 \pm 0.5$  ( $\pm$ range) years, with repeated examinations in 1967, 1973, 1980, 1988, and 1993.

Of the men still alive in 1993, 232 attended the examination. Of the men who did not participate in 1993, 67 were interviewed by telephone, 29 answered a questionnaire, and medical records were studied for another 32. Six of the men had left the country and were unavailable to follow-up. Another 7 men were unavailable for end-point registration because they had moved out of the area and their medical records could not be found. Among these 7 men, 1 had right bundle-branch block and died in 1995. He has been included in the calculation of the survival data. Thus, the clinical follow-up rate during the 30-year follow-up period was 98%.

Morbidity data for coronary heart disease and stroke were obtained by interview, from death certificates, from the Myocardial Infarction Register,<sup>27</sup> and from the Stroke Register<sup>28</sup> covering the city of Göteborg. The criteria for stroke were hospital admission with the diagnosis of stroke or a fresh cerebral thrombosis or hemorrhage at postmortem examination.

The criteria for ischemic heart disease during follow-up were myocardial infarction defined by the Swedish Society of Cardiology, postmortem findings of fresh coronary heart disease, or hospitalization due to angina pectoris/unstable angina pectoris and suspected acute myocardial infarction. The end point congestive heart failure was defined as hospitalization for heart failure or outpatient treatment for heart failure for at least 3 months. Diabetes mellitus was defined as known diabetes mellitus under treatment or fasting blood glucose  $\geq 6.7$  mmol/L.

## Statistics

The study group consisting of bundle-branch block was compared with the rest of the population with available ECGs (Table 1).

Nonparametric tests were used. For difference between groups, the Wilcoxon rank sum test was used for continuous variables. Differences in proportions were analyzed with the  $\chi^2$  test. A life-table method according to Kaplan-Meier<sup>29</sup> was used to calculate the survival curves and the cumulative incidence for bundle-branch block. The cumulative incidence was based on those still alive and available to follow up with regard to ECGs. Men with bundle-branch block were not considered at risk the next year.

## Results

During 30 years of follow-up, 82 patients (9.6%) with bundle-branch block were found. The vast majority (86%) had acquired the condition after the age of 50 years. In 2 patients, the bundle-branch blocks were found to be intermittent. One patient developed right bundle-branch block that changed to left bundle-branch block. He has been considered as having right bundle-branch block in all the analyses. ECG signs of left ventricular hypertrophy before bundle-branch block developed were seen in 26% of the patients who

**TABLE 2. Description of All Cases of Bundle-Branch Block Over 30 Years of Follow-Up**

	LBBB (n=22)	RBBB (n=60)	P
Mean age at diagnosis, y	71±10	69±8	NS
QRS duration, ms	141±19	140±15	NS
Known acquired, n (%)	19 (86)	52 (87)	NS
Prior signs of LVH, n (%)	5 (26)	3 (6)	<0.01
Prior incomplete BBB, n (%)	3 (16)	14 (27)	NS
Additional AV block, n (%)	1 (5)	3 (5)	NS
Atrial fibrillation, n (%)	2 (9)	8 (14)	NS
Known intermittent, n (%)	1 (5)	1 (2)	NS
Possible bifascicular, n (%)	...	28 (47)	

(LAH=27, LPH=1)

LBBB indicates left bundle-branch block; RBBB, right bundle-branch block; LVH, left ventricular hypertrophy on ECG before bundle-branch block developed; incomplete BBB, intraventricular conduction delay present before development of complete bundle-branch block; additional AV block, concomitant atrioventricular block, ie, AV block I, II, or III; LAH, left anterior hemiblock; and LPH, left posterior hemiblock.

acquired left bundle-branch block compared with 6% for right bundle-branch block (Table 2).

The prevalence of bundle-branch block, both right and left, increased with age. Right bundle-branch blocks were more common than left bundle-branch blocks (Table 3). At age 75 years, the prevalence of right bundle-branch block was >4 times that of left bundle-branch block. The cumulative incidence rate for all bundle-branch blocks at age 80 years was 18.1% (left bundle-branch block, 6.5% and right bundle-branch block, 12.9%) (Figure 1).

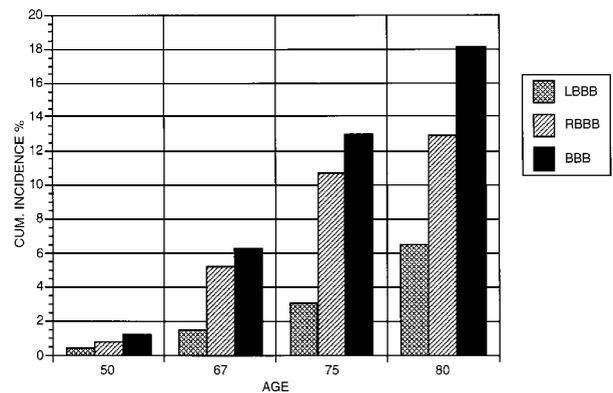
All patients who were alive and had an ECG taken at the age of 67, 75, and 80 years were analyzed separately for differences in coronary risk factors at age 50 years as well as being given a diagnosis of myocardial infarction, ischemic heart disease, congestive heart failure, or diabetes mellitus during follow-up (Tables 4, 5, and 6).

In survivors, at age 67, 75, and 80 years, there were no significant differences in risk factor profile at age 50 between

**TABLE 3. Prevalence of Bundle-Branch Block at 50, 67, 75, and 80 Years of Age**

	Age, y			
	50 n (%)	67 n (%)	75 n (%)	80 n (%)
ECG available	854	567	395	212
All bundle-branch block	10 (1.2)	36 (6.3)	48 (12.2)	36 (17)
New cases		32 (89)	28 (58)	12 (33)
Left bundle-branch block	3 (0.4)	8 (1.4)	9 (2.3)	12 (5.7)
New cases		6 (75)	6 (67)	7 (58)
Right bundle-branch block	7 (0.8)	28 (4.9)	39 (9.9)	24 (11.3)
New cases		26 (93)	22 (56)	5 (21)

ECG available refers to Table 1. New cases indicate the first time of diagnosis of bundle-branch block. Men with previous diagnosis of bundle-branch block alive but without available ECG at that particular examination were not included. The percentage is calculated as share of the prevalence accounted for by new cases.

**Figure 1.** Cumulative incidence of bundle-branch block (BBB; L, left; R, right) in men from 50 to 80 years old.

those who subsequently developed bundle-branch block and those who did not, and there was no significant difference in having myocardial infarction or a diagnosis of ischemic heart disease during follow-up. The heart volume at age 50 years was consistently larger among those who developed bundle-branch block (the majority had not developed bundle-branch block at the time of their radiographs in 1963) compared with control subjects. A diagnosis of congestive heart failure during follow-up was significantly more common among those with bundle-branch block. The biggest difference was found in survivors at age 67 years, among whom 36% of those with bundle-branch block developed congestive heart failure, compared with 14% of the control subjects (Table 4). Diabetes mellitus was also more common among men who developed bundle-branch block.

### Survival and Mortality

Survival curves for men with and without bundle-branch block were calculated separately at age 50, 67, and 75 years to avoid survival bias (Figures 2, 3, and 4, respectively). On all 3 curves, men with bundle-branch block showed a non-significant trend toward a higher mortality. Among men who died without being known to have bundle-branch block, 262 of 446 deaths (59%) were diagnosed as being cardiovascular, compared with 23 of 35 (66%) in patients with bundle-branch block ( $P=NS$ ). Among those who died of cardiovascular causes without being known to have bundle-branch block, 73 of 262 (28%) had a prior diagnosis of chronic congestive heart failure, compared with 14 of 23 (61%) in patients with bundle-branch block ( $P<0.01$ ).

### Discussion

In a large screening program by Fahy et al,<sup>10</sup> the prevalence of bundle-branch block was only 0.28%; in the male population >64 years old the prevalence was 1.6%. The Reykjavik study showed a prevalence of 0.43% for left bundle-branch block among middle-aged men.<sup>14</sup> Among men 75 to 79 years old, right bundle-branch block was found in 4.1%.<sup>15</sup> In a retirement community, a prevalence of bundle-branch block of 5.1% was found in the male population  $\geq 52$  years old.<sup>12</sup> The Tecumseh study showed the prevalence of bundle-branch block to be 2.4% in men >50 years old.<sup>30</sup> In the study by Kreger et al,<sup>31</sup> complete intraventricular block, defined as

**TABLE 4. Baseline Risk Factors and End Points During Follow-Up in Survivors at Age 67 Years With and Without Bundle Branch Block**

	RBBB (n=28)	LBBB (n=8)	BBB (n=36)	No BBB (n=531)
At age 50 y, mean				
Systolic blood pressure, mm Hg	135	144	137	137
Diastolic blood pressure, mm Hg	92	92	92	91
Blood glucose, mmol/L	4.4	5.3	4.7	4.6
Serum cholesterol, mmol/L	6.4	6.3	6.4	6.4
Serum triglycerides, mmol/L	1.3	1.1	1.3	1.2
Body mass index, kg/m <sup>2</sup>	25.6	25.1	25.5	24.8
Heart volume, mL	796*	785	794*	746
Never smoked, n (%)	5 (18)	2 (25)	7 (19)	149 (28)
During follow-up, n (%)				
Diabetes	8 (29)	3 (38)	11 (31)*	88 (17)
Ischemic heart disease	8 (29)	3 (38)	11 (31)	142 (27)
Myocardial infarction	8 (29)	2 (25)	10 (28)	111 (21)
Congestive heart failure	10 (36)†	3 (38)	13 (36)†	72 (14)

RBBB indicates right bundle-branch block; LBBB, left bundle-branch block; BBB, all bundle-branch block; and No BBB, no bundle-branch block.

\* $P < 0.05$ , † $P < 0.01$  vs no BBB.

QRS  $\geq 0.12$  second, was strongly dependent on age, with a prevalence of 11% in men in the 8th and 9th decades, and was twice as common in men as in women. Our results, as shown in Table 3, explain the wide prevalence range noted in earlier trials and show that bundle-branch block is highly age-dependent, going from uncommon (1.2%) at age 50 to becoming common (17%) at age 80 in the same population.

#### Relationship With Coronary Heart Disease

In the Framingham Heart Study,<sup>13</sup> univariate analysis showed an increased risk of subsequent development of coronary heart disease or congestive heart failure in patients who

developed bundle-branch block. When adjusted for age, this difference was not significant. Froelicher et al,<sup>32</sup> using coronary angiography, examined 75 asymptomatic male aircrew members with bundle-branch block and found significant stenoses in 16 (22%) of the men but no causal correlation to the length of the left main coronary artery and numbers of septal perforators in left bundle-branch block, as stated earlier in a study by Herbert.<sup>33</sup> Patients with chest pain and right bundle-branch block were angiographically studied by Haft et al,<sup>34</sup> and no difference in severity or extension in coronary artery disease was seen compared with control subjects. From the Coronary Artery Surgery Study,<sup>16</sup> with >15 000 patients

**TABLE 5. Baseline Risk Factors and End Points During Follow-Up in Survivors at Age 75 Years With and Without Bundle-Branch Block**

	RBBB (n=39)	LBBB (n=9)	BBB (n=48)	No BBB (n=347)
At age 50 y, mean				
Systolic blood pressure, mm Hg	139	142	140	136
Diastolic blood pressure, mm Hg	93	94	93	90
Blood glucose, mmol/L	4.6	4.7	4.6	4.6
Serum cholesterol, mmol/L	6.3	6.1	6.3	6.4
Serum triglycerides, mmol/L	1.4	1.1	1.3	1.2
Body mass index, kg/m <sup>2</sup>	25.1	24.0	24.9	24.6
Heart volume, mL	775	876*	790†	738
Never smoked, n (%)	12 (31)	0 (0)*	12 (25)	107 (31)
During follow-up, n (%)				
Diabetes	9 (23)	4 (44)*	13 (27)	58 (17)
Ischemic heart disease	9 (23)	3 (33)	12 (25)	70 (20)
Myocardial infarction	8 (21)	2 (22)	10 (21)	58 (17)
Congestive heart failure	7 (18)	1 (11)	8 (17)	33 (10)

Abbreviations as in Table 4.

\* $P < 0.05$ , † $P < 0.01$  vs no BBB.

**TABLE 6. Baseline Risk Factors and End Points During Follow-Up in Survivors at Age 80 Years With and Without Bundle-Branch Block**

	RBBB (n=24)	LBBB (n=12)	BBB (n=36)	No BBB (n=176)
At age 50 y (1963), mean				
Systolic blood pressure, mm Hg	134	144	137	135
Diastolic blood pressure, mm Hg	91	96	93	89
Blood glucose, mmol/L	4.6	4.6	4.6	4.6
Serum cholesterol, mmol/L	6.1	5.9	6.1	6.3
Serum triglycerides, mmol/L	1.2	1.0	1.1	1.2
Body mass index, kg/m <sup>2</sup>	24.4	25.8	24.9	24.5
Heart volume, mL	740	830	768	737
Never smoked, n (%)	10 (42)	5 (42)	15 (42)	53 (30)
During follow-up, n (%)				
Diabetes	5 (21)	5 (42)*	10 (28)	28 (16)
Ischemic heart disease	3 (12)	1 (8)	4 (11)	19 (11)
Myocardial infarction	3 (12)	1 (8)	4 (11)	16 (9)
Congestive heart failure	4 (17)	2 (17)	6 (17)*	9 (5)

Abbreviations as in Table 4.  
\**P*<0.05 vs no BBB.

with chronic coronary artery disease, 522 patients with bundle-branch block were identified. No particular location of coronary stenosis or left ventricular wall motion abnormalities predominated, indicating that the bundle-branch block was the result of infarction of the proximal conduction system.

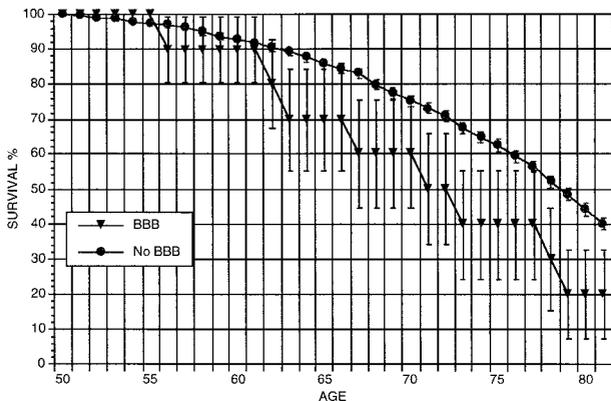
In our study, with regard to risk factors for coronary heart disease at age 50 years, there was no difference between those who developed bundle-branch block and those who did not, except for diabetes mellitus, which was more common in men with bundle-branch block. The risk of having or developing ischemic heart disease/myocardial infarction in the future was not higher in the bundle-branch block population. In our population, coronary heart disease did not seem to play any major role in the development of bundle-branch block; instead, our study supports the theory that bundle-branch block is a progressive degenerative disease that affects not only the conduction system but also the myocardium itself, as shown by a larger heart volume at age 50 years in those who

developed bundle-branch block at follow-up and a significantly higher incidence of the diagnosis of congestive heart failure during follow-up (Tables 4 to 6).

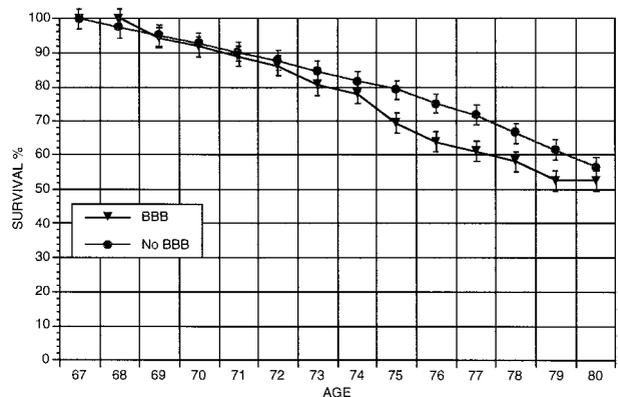
**Mortality**

Considerable information in the literature indicates that patients with bundle-branch block, either right or left, may have normal longevity.<sup>9-11,35-37</sup> In the Framingham Study, an increased mortality from cardiovascular disease was seen in people with bundle-branch block. However, total mortality was not described.<sup>13</sup>

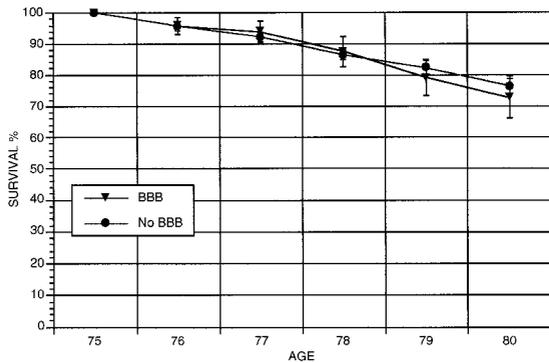
Despite the high prevalence of coronary heart disease in patients with bundle-branch block, the onset of chronic bundle-branch block is only rarely accompanied by clinically recognized myocardial infarction. More often, bundle-branch block is discovered as an incidental accompaniment to chronic coronary artery disease. In studies of myocardial infarction/ischemic heart disease, bundle-branch block has been shown to be a strong predictor of high mortality at



**Figure 2.** Survival curve for 50-year-old men followed up for 30 years. Men with bundle-branch block (BBB) at age 50 years (n=10) vs no bundle-branch block. Bars indicate SE, *P*=NS.



**Figure 3.** Survival curve for 67-year-old men followed up for 13 years. Men with bundle-branch block (BBB) at age 67 years (n=36) vs no bundle-branch block. Bars indicate SE, *P*=NS.



**Figure 4.** Survival curve for 75-year-old men followed up for 5 years. Men with bundle-branch block (BBB) at age 75 years (n=48) vs no bundle-branch block. Bars indicate SE,  $P=NS$ .

follow-up.<sup>16,17,19,38-40</sup> Bundle-branch block has been reported to be present in 13% of patients with acute myocardial infarction.<sup>38,41-43</sup> In our study, no increased mortality was seen in men with bundle-branch block at follow-up and no difference in the incidence of ischemic heart disease or death due to cardiovascular disease.

Coronary heart disease does not seem to play any major role in the pathogenesis of bundle-branch block. Rather, in our own study and those of others, bundle-branch block gives the impression of being a marker of a slowly progressing degenerative disease affecting not only the conduction system but also the myocardium.<sup>44-46</sup> A slow increase in cumulative mortality over time would then be expected but would be detected only in large populations followed up for a long period of time.

The marked increase in mortality in patients with bundle-branch block is seen only in combination with ischemic heart disease. In bundle-branch block, the depolarization phase is by definition prolonged. Furthermore, the prolongation of the vulnerable repolarization phase in combination with an increased number of premature ventricular beats (secondary to ischemic heart disease) would expose the patient to an increased risk of sudden ventricular tachyarrhythmias.

This theory is supported by electrophysiological studies of patients with bifascicular block, in whom sustained monomorphic ventricular tachycardia was induced exclusively in patients with a previous myocardial infarction.<sup>47</sup> Furthermore, McAnulty et al<sup>3</sup> followed up 554 patients with bundle-branch block and noticed an increased risk of sudden death due not to bradyarrhythmias but rather to tachyarrhythmias and myocardial infarction.

Another explanation of the high mortality from acute myocardial infarction could be a degenerative cardiomyopathy less able to compensate for a sudden loss of functional myocardium during the course of an acute myocardial infarction.

### Limitations of the Study

We looked at ECG recordings on only 4 occasions during a follow-up period of 30 years. In men who died who were not regarded as having bundle-branch block, we do not know whether they would have developed bundle-branch block before death. If that number is substantial, our results under-

estimate the cumulative incidence and mortality of bundle-branch block. Because both bundle-branch block and ischemic heart disease differ in a number of ways between sexes, our results cannot be extrapolated to women.

### Conclusions

In a prospective population sample of men at age 50 years who were then followed up for 30 years, bundle-branch block was found to be common in elderly men and to increase with age. No correlation to risk factors for coronary heart disease at age 50 years, incidence of myocardial infarction during follow-up, or cardiovascular deaths was found. The results support the theory that bundle-branch block is a marker of a progressive degenerative disease that also affects the myocardium.

### Acknowledgments

This study was supported by grants from the Swedish Heart and Lung Foundation, the Swedish Medical Research Council (B97-27X-06276-15A), King Gustav V and Queen Victoria's Foundation, Göteborg Medical Association, and Göteborg University.

### References

- McAnulty HJ, Kauffmann S, Murphy E, Kasselbaum GD, Rahimtoola HS. Survival in patients with intraventricular conduction defects. *Arch Intern Med.* 1978;138:30-35.
- McAnulty HJ, Rahimtoola HS, Murphy SE, Kauffman S, Ritzmann WL, Kanarek P, DeMots H. A prospective study of sudden death in "high-risk" bundle-branch block. *N Engl J Med.* 1978;299:209-215.
- McAnulty HJ, Rahimtoola HS, Murphy E, DeMots H, Ritzmann L, Kanarek EP, Kauffman S. Natural history of "high-risk" bundle-branch block. *N Engl J Med.* 1982;307:137-143.
- Johnson PR, Messer LA, Shreenivas, White DP. Prognosis in bundle-branch block, II: factors influencing the survival period in left bundle-branch block. *Am Heart J.* 1951;41:225-238.
- Messer LA, Johnson PR, Shreenivas, White DP. Prognosis in bundle-branch block, III: a comparison of right and left bundle-branch block with a note on the relative incidence of each. *Am Heart J.* 1951;41:239-245.
- Scanlon JP, Pryor R, Blount SG. Right bundle-branch block associated with left superior or inferior intraventricular block. *Circulation.* 1970;42:1123-1133.
- Shreenivas, Messer LA, Johnson PR, White DP. Prognosis in bundle-branch block, I: factors influencing the survival period in right bundle-branch block. *Am Heart J.* 1950;40:891-902.
- Smith S, Hayes LW. The prognosis of complete left bundle-branch block. *Am Heart J.* 1965;70:157-159.
- Rotman M, Triebwasser HJ. A clinical and follow-up study of right and left bundle-branch block. *Circulation.* 1975;51:477-484.
- Fahy JG, Pinski LS, Miller PD, McCabe N, Pye C, Walsh JM, Robinson K. Natural history of isolated bundle-branch block. *Am J Cardiol.* 1996; 77:1185-1190.
- Rodstein M, Gubner R, Mills PJ, Lovell FJ, Ungerleider E. A mortality study in bundle-branch block. *Arch Intern Med.* 1951;87:663-668.
- Edmands RE. An epidemiological assessment of bundle-branch block. *Circulation.* 1966;34:1081-1087.
- Schneider FJ, Thomas EH, Sorlie P, Kreger EB, McNamara MP, Kannel BW. Comparative features of newly acquired left and right bundle-branch block. *Am J Cardiol.* 1981;47:931-940.
- Hardarson T, Arnarson A, Eliasson JG, Palsson K, Eyjolfsson K, Sigfusson N. Left bundle-branch block: prevalence, incidence, follow-up and outcome. *Eur Heart J.* 1987;8:1075-1079.
- Thrainsdottir I, Hardarson T, Thorgerirsson G, Sigvaldason G, Sigfusson N. The epidemiology of right bundle-branch block and its association with cardiovascular morbidity: the Reykjavik Study. *Eur Heart J.* 1993; 14:1590-1596.
- Freedman R, Alderman E, Sheffield T, Saporito M, Fisher L. Coronary Artery Surgery Study (CASS). Bundle-branch block in patients with chronic coronary artery disease: angiographic correlates and prognostic significance. *J Am Coll Cardiol.* 1987;10:73-80.

17. Col JJ, Weinberg SL. The incidence and mortality of intraventricular conduction defects in acute myocardial infarction. *Am J Cardiol.* 1972; 29:344–350.
18. Newby KH, Pisano E, Krucoff MW, Green C, Natale A. Incidence and clinical relevance of the occurrence of bundle-branch block in patients treated with thrombolytic therapy. *Circulation.* 1996;94:2424–2428.
19. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of over 1000 patients. *Lancet.* 1994;343:311–322.
20. Tibblin G. A population study of 50-year-old men: an analysis of the non-participant group. *Acta Med Scand.* 1965;178:453–458.
21. Tibblin G. High blood pressure in men aged 50. *Acta Med Scand.* 1967;470:1–84.
22. Eriksson H, Svärdsudd K, Larsson B, Ohlsson L-O, Welin L, Tibblin G, Wilhelmsson L. Dyspnea in a cross sectional and longitudinal study of middle aged men. The Study of Men Born 1913. *Eur Heart J.* 1987;8: 1015–1023.
23. Hansson PO, Wilhelmson L, Tibblin G, Eriksson H. Deep vein thrombosis and pulmonary embolism in the general population. *Arch Intern Med.* 1997;157:1665–1670.
24. Schneider JF, Thomas H, Kreger BE, McNamara PM, Sorlie P, Kannel WB. Newly acquired right bundle-branch block: the Framingham Study. *Ann Intern Med.* 1980;92:37–44.
25. Sokolow M, Lyon TP. The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J.* 1949;37:161–186.
26. Jonsell SA. A method for the determination of the heart size by teleroentgenography. *Acta Radiol.* 1939;20:325–340.
27. Elmfeldt D, Wilhelmson L, Tibblin G, Vedin JA, Wilhelmsson C-E, Bengtsson C. Registration of myocardial infarction in the city of Göteborg, Sweden. *J Chronic Dis.* 1975;28:173–186.
28. Harmsen P, Tibblin G. A stroke register in Göteborg, Sweden. *Acta Med Scand.* 1972;191:463–470.
29. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc.* 1958;53:457–481.
30. Ostrander LD, Brandt RL, Kjelsberg MO, Epstein FH. Electrocardiographic findings among the adult population of a total natural community, Tecumseh, Michigan. *Circulation.* 1965;31:888–898.
31. Kreger BE, Anderson KM, Kannel WB. Prevalence of intraventricular block in the general population: the Framingham Study. *Am Heart J.* 1989;117:903–910.
32. Froelicher VF, Thompson AJ, Wolthuis R, Fuchs R, Balusek R, Longo MR, Triebwasser JH, Lancaster MC. Angiographic findings in asymptomatic aircrewmembers with electrocardiographic abnormalities. *Am J Cardiol.* 1977;39:32–38.
33. Herbert WH. Left bundle-branch block and coronary artery disease. *J Electrocardiol.* 1975;8:317–324.
34. Haft JJ, DeMaio SJ, Bartoszyk OB. Coronary arteriographic findings in symptomatic right bundle-branch block. *Am J Cardiol.* 1984;53:770–773.
35. Schreenivas, Messer AL, Johnson RP, White PD. Prognosis in bundle-branch block. *Am Heart J.* 1950;40:891–902.
36. Langley RW, Reed JC, Utz DC. Bundle-branch block: a review of 100 cases. *Am Heart J.* 1947;33:730.
37. Smith RF, Jackson DH, Harthorne JW, Sanders CA. Acquired bundle-branch block in a healthy population. *Am Heart J.* 1970;80:746–751.
38. Hindman MC, Wagner GS, JaRo M, Atkins JM, Scheinman MM, DeSanctis RW, Hutter AH Jr, Yeatman L, Rubenfire M, Pujara C, Rubin M, Morris JJ. The clinical significance of bundle-branch block complicating acute myocardial infarction, I: clinical characteristics, hospital mortality, and one-year follow-up. *Circulation.* 1978;58:679–688.
39. Eriksson P, Andersen K, Swedberg K, Dellborg M. Vectorcardiographic monitoring of patients with acute myocardial infarction and chronic bundle-branch block. *Eur Heart J.* 1997;18:1288–1295.
40. Piérard LA, Dubois C, Albert A, Smeets JP, Kulbertus HE. Prediction of mortality after myocardial infarction by simple clinical variables recorded during hospitalization. *Clin Cardiol.* 1989;12:500–504.
41. Mullins CB, Atkins JM. Prognosis and management of ventricular conduction blocks in acute myocardial infarction. *Mod Concepts Cardiovasc Dis.* 1976;45:129–133.
42. Killip T, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two year experience with 250 patients. *Am J Cardiol.* 1967; 20:457–464.
43. Bigger JT, Dresdale RJ, Heissenbuttel RH, Welch FM, Wit AL. Ventricular arrhythmias in ischemic heart disease: mechanism, prevalence, significance and management. *Prog Cardiovasc Dis.* 1977;19:255–300.
44. Lenegre J. Etiology and pathology of bilateral bundle-branch block in relation to complete heart block. *Prog Cardiovasc Dis.* 1964;6:409–444.
45. Davies M, Harris A. Pathological basis of primary heart block. *Br Heart J.* 1969;31:219–226.
46. Kuhn H, Breithart G, Knieriem HJ, Köhler E, Lösse B, Seipel L, Loogen F. Prognosis and possible presymptomatic manifestations of congestive cardiomyopathy. *Postgrad Med J.* 1978;54:451–459.
47. Englund A, Bergfeldt L, Rehnqvist N, Åström H, Rosenqvist M. Diagnostic value of programmed ventricular stimulation in patients with bifascicular block: a prospective study of patients with and without syncope. *J Am Coll Cardiol.* 1995;26:1508–1515.

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*Circulation*. 1998;98:2494-2500  
doi: 10.1161/01.CIR.98.22.2494

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231  
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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