Left ventricular hypertrophy in presence of complete left bundle-branch block*

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SUMMARY Electrocardiograms of 2500 consecutive patients who died in hospital were reviewed in order to study the relation of complete left bundle-branch block to pathological determinants of anatomical left ventricular hypertrophy. Of these, 43 patients demonstrated complete left bundle-branch block and all but two had anatomical left ventricular hypertrophy at necropsy. Mean values of total heart weight and percentage total heart weight of normal were significantly higher in this group of 43 patients with complete left bundle-branch block than in a control necropsy group of 300 patients with anatomical left ventricular hypertrophy but without complete left bundle-branch block. Left ventricular wall thickness did not correlate with other measures of left ventricular muscle mass. Commonly used electrocardiographic voltage criteria for left ventricular hypertrophy identified approximately one-half of the 41 patients with left bundle-branch block and anatomical left ventricular hypertrophy. The degree of electrocardiographic voltage abnormality correlated modestly with total heart weight and percentage total heart weight of normal. A group of 25 patients was studied in whom electrocardiograms were available before and after complete left bundle-branch block. Normal voltage before left bundle-branch block tended to remain normal, while abnormally high voltage tended to fall after complete left bundle-branch block developed. Complete left bundle-branch block in a necropsy population characterised a group with extreme anatomical left ventricular hypertrophy. The sensitivity of the usual electrocardiographic left ventricular hypertrophy voltage criteria in the presence of complete left bundle-branch block is only slightly less than in patients with left ventricular hypertrophy alone. The degree of electrocardiographic voltage abnormality with complete left bundle-branch block shows modest correlation with degree of anatomical left ventricular hypertrophy.

Opinions vary as to whether or not the electrocardiographic diagnosis of left ventricular hypertrophy can be made when complete left bundle-branch block coexists.¹⁻³ Few data have been reported on the subject, and reports suffer somewhat from lack of clear definition of methods and terminology. The issue may be of more than academic interest when one considers that left ventricular hypertrophy may be missed by routine chest x-ray film⁴ and physical examination.⁵ Though other clinical modalities, that is angiography and echocardiography are also available for assessment of left ventricular mass, the cornerstones

for the diagnosis of left ventricular hypertrophy remain the chest x-ray film, physical examination, and electrocardiogram in most routine clinical settings.

The purpose of this report is to explore further how well currently employed electrocardiographic criteria for the diagnosis of left ventricular hypertrophy can be applied to a group of patients at necropsy who had complete left bundle-branch block. We also examine the electrocardiograms of a group of patients before and after the development of complete left bundle-branch block assessing its effect on the QRS voltage.

Methods

Electrocardiograms of 2500 consecutive patients who died at Strong Memorial Hospital in Rochester, New York, during the years 1972 to 1976 were

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reviewed. Records were selected for study if the patient was 21 years or older and if complete left bundle-branch block was recorded within six months of death. Patients who developed complete left bundle-branch block as a terminal event (within 24 hours of death) were excluded. All the following criteria were required for the diagnosis of complete left bundle-branch block: (1) documented atrioventricular conduction, (2) absence of Wolff-Parkinson-White syndrome, (3) QRS duration in any lead ≥ 120 ms, (4) intrinsicoid deflection ≥ 50 ms in the left heart leads, (5) deformity of the R wave in the left heart leads consisting of slurred, notched, flat, or bifid R wave, (6) major fraction of the QRS duration in the left heart leads occupied by the R wave, (7) absence of significant Q waves in any left heart lead,⁸ (8) absence of a large S wave ($\geq 20\%$ of the total QRS deflection) in any left heart lead, and (9) ST segment depression and/or T wave inversion in the left heart leads.

The 43 patients that met the above criteria were subdivided into (a) a group of 32 patients with persistent complete left bundle-branch block (present for at least six months without intervening normal conduction) or left bundle-branch block of indeterminate onset and (b) a group of 11 patients in whom the onset of complete left bundle-branch block occurred within six months of death or in whom it was intermittent. The former group of 32 patients is termed persistent complete left bundle-branch block for the sake of conciseness, while the latter group of 11 patients is termed intermittent left bundle-branch block, though, strictly speaking, some of these simply represent recent onset left bundle-branch block.

As a control group, the electrocardiograms and findings at necropsy of all patients age 21 years or more who came to necropsy during the first three months of each year during the study period 1972–76 were reviewed.

The total heart weight, left ventricular wall thickness, body length, and sex were recorded at necropsy. Normal predicted total heart weight was defined according to the tables of Zeek,⁷ and any heart weight two standard deviations above the mean of normal was regarded as anatomical left ventricular hypertrophy. Percentage total heart weight of normal was calculated for each patient at necropsy by dividing the recorded total heart weight by the predicted value. This measurement allows correction of total heart weight for sex and body length.

One investigator coded by number all electrocardiograms without knowledge of the results of the necropsies. The following electrocardiographic measurements (paper speed 25 mm/s, sensitivity 1 mV/10 mm) were then recorded by blinded observers: (1) height (mV of R waves in leads I, II, II, aVL, aVF, and V1 to V6; (2) depth (mV) of S waves in leads I, II, III, aVL, aVF, V1 to V6; (3) intrinsicoid deflection (ms) from the left heart leads; (4) maximum QRS width (ms) from any lead; and (5) mean QRS axis. Electrocardiographic voltage criteria for left ventricular hypertrophy were reviewed, and those criteria shown by Romhilt and co-workers⁸ to have a higher predictive value for left ventricular hypertrophy were tested. Electrocardiogram and necropsy data were transferred to IBM punch cards for computerised statistical analysis.

Electrocardiographic variables were compared with pathological determinants of left ventricular hypertrophy using standard correlational analyses. Student *t* tests (two-sided) were used to compare pathological determinants of left ventricular hypertrophy in the various subgroups of patients at necropsy. McNemar's test (exact version) was employed to determine statistically significant change in electrocardiographic variables before and after development of complete left bundle-branch

Table 1 Identification of patient groups

Patient groups		Number of patients			Mean age (y)
		Total	Male	Female	
Total patient records reviewed		2500	—	—	—
Patients with complete LBBB		90	—	—	—
Necropsy patients with complete LBBB	{ Patients with persistent* LBBB	32	24	8	66.4
	{ Patients with intermittent* LBBB	11	8	3	63.5
Patients with intermittent* LBBB	{ Necropsy	11	8	3	63.5
	{ No necropsy	14	7	7	62.9
Control† necropsy population	{ Anatomical LVH‡ LBBB	11	7	4	63.3
	{ Anatomical LVH‡ No LBBB	300	160	140	66.1
	{ No anatomical LVH‡ LBBB	1	1	0	65.0
	{ No anatomical LVH‡ No LBBB	168	101	67	58.3

* See text for definition of persistent and intermittent LBBB. † Control (base) necropsy population defined in text. ‡ Anatomical LVH is described in text. LBBB, left bundle-branch block; LVH, left ventricular hypertrophy.

block in a group of 25 patients in whom both types of electrocardiograms were available within six months of each other.

Results

The sex and mean age of the patient groups are shown in Table 1. Forty-three of 90 patients (47.7%) who died with complete left bundle-branch block came to necropsy. The average percentage of necropsy examinations for the entire hospital population during this period of study was 53.2.

Table 2 *Clinicopathological findings in 43 patients with complete left bundle-branch block and necropsy*

Diagnosis	Number	Per cent
Old or recent myocardial infarction*	31	72.1
Aortic valve disease†	4	9.3
Cardiomyopathy	4	9.3
Normal heart	2	4.6
Pulmonary embolism	1	2.3
Cor pulmonale	1	2.3
Total	43	100.0

* Significant hypertension was additionally present in five of these patients. † An additional five patients had significant aortic valve disease in association with old or recent myocardial infarction and are included in the latter category.

Of the patients with complete left bundle-branch block, 41 of 43 (95.3%) had evidence of anatomical left ventricular hypertrophy on the basis of total heart weight and percentage total heart weight of normal. Left ventricular wall thickness showed no correlation with total heart weight ($r = -0.05$) and percentage total heart weight of normal ($r = -0.05$).

The control necropsy group consisted of 480 patients, 311 (64.8%) of whom were found to have anatomical left ventricular hypertrophy; 12 of 480 (2.5%) also showed complete left bundle-branch block within six months of death. Only one of the 169 control patients without left ventricular hypertrophy had left bundle-branch block, while 11 of the 311 control patients with left ventricular hypertrophy had left bundle-branch block.

Table 2 summarises the clinical information of 43 patients with complete left bundle-branch block at necropsy.

Table 3 summarises the comparison of mean relative heart weights in the groups of patients mentioned. The entire group of 43 patients with left bundle-branch block and the subgroup of 32 patients with persistent left bundle-branch block both demonstrate a significantly higher mean relative heart weight than do the subgroup of 11 patients with intermittent left bundle-branch block and the group of 300 control patients with anatomi-

Table 3 *Comparison of percentage total heart weight of normal in groups of patients with and without complete left bundle-branch block*

Group	Number of patients	Percentage total heart weight of normal $\pm$ SD
Complete LBBB	43	200.4 $\pm$ 49.9*
Persistent* LBBB	32	209.7 $\pm$ 54.3*
Intermittent† LBBB	11	175.6 $\pm$ 37.8
LVH but no LBBB (control)	300	169.1 $\pm$ 36.2

* Significantly higher than in the last two groups ($P < 0.05$).

† See text for definition of persistent and intermittent LBBB. LBBB, left bundle-branch block; LVH, left ventricular hypertrophy; SD, standard deviation.

cal left ventricular hypertrophy but no left bundle-branch block ($P < 0.05$).

Table 4 shows the results of application of currently employed electrocardiographic voltage criteria for left ventricular hypertrophy to the group of 41 patients with complete left bundle-branch block and anatomical left ventricular hypertrophy. A comparison of the expected frequency of abnormality (on the basis of Romhilt *et al.*'s⁸ data for patients with left ventricular hypertrophy but no left bundle-branch block with observed frequency of abnormality is shown. The data reveal only a minor loss of sensitivity of these criteria in our group of patients with complete left bundle-branch block.

Table 4 *Frequency of abnormality of left ventricular hypertrophy voltage criteria in 41 patients with complete left bundle-branch block and anatomical left ventricular hypertrophy*

Voltage criteria*	Normal* value (mm)	Observed frequency of abnormality (%)	Expected frequency* of abnormality (%)
RaVL	≤ 7.5	46.3	22.5
(RI + SIII) - (RIII + SI)	< 17.0	26.8	17.5
SV1 or SV2 + RV5	< 35.0	51.2	56.3
SV1 or SV2 + RV5 or RV6	≤ 35.0	53.7	55.6
R + S†	≤ 40.0	48.8	55.0
RS‡	≤ 35.0	36.6	40.6
SV1 or SV2	< 30.0	34.1	—
SV1 or SV2 or SV3	≤ 30.0	34.1	—
RV5 or RV6	< 30.0	4.9	—
SV1 or SV2 or SV3 or RV5 or RV6	< 40.0	48.8	—

* From data by Romhilt *et al.*⁸ in patients with anatomical left ventricular hypertrophy but no left bundle-branch block.

† Sum of largest R wave and largest S wave in unipolar precordial leads. ‡ Largest total RS deflection in any unipolar lead.

Table 5 shows the correlation of three voltage criteria (two of which were the most sensitive as found in this study) with the total heart weight, percentage total heart weight of normal, and left ventricular wall thickness of the 41 patients with

Table 5 Correlation of electrocardiographic variables with measures of anatomical left ventricular hypertrophy in 41 patients with complete left bundle-branch block and anatomical left ventricular hypertrophy

Electrocardiogram variable	Correlation with		
	Absolute heart weight	Relative heart weight (percentage of normal)	Left ventricular wall thickness
RaVL	0.375*	0.370*	0.212
SV1 or SV2 + RV6	0.346*	0.359*	0.218
SV1 or SV2 + RV5 or RV6	0.373*	0.407*	0.184
Max. QRS width	0.014	0.008	-0.211
Intrinsicoid deflection	0.155	0.216	0.090

* Significant at 5 per cent level.

complete left bundle-branch block and anatomical left ventricular hypertrophy. Left ventricular wall thickness showed no significant correlation with any of the electrocardiographic variables listed. All three voltage criteria correlated modestly with the measures of absolute and relative heart weight. There was no significant correlation of maximum QRS width or intrinsicoid deflection to any of the measurements of anatomical left ventricular hypertrophy.

Fourteen of the 43 patients with complete left bundle-branch block and necropsy showed a normal QRS axis, that is a mean QRS axis between -29 and $+119$ degrees, on their pre-mortem electrocardiograms, while 29 showed left axis deviation, that is a mean QRS axis equal to or greater than -30 degrees. There was no correlation of mean QRS axis with total heart weight ($r = -0.07$) or percentage total heart weight of normal ($r = -0.05$) in the entire group of 43 patients.

Analysis of the 25 patients with electrocardiograms recorded before and after the development of complete left bundle-branch block within six months of each other disclosed that the most sensitive voltage criteria found in our study (SV1 or SV2 + RV5, SV1 or SV2 + RV5 or RV6, and R + S) when abnormal before left bundle-branch block tended to become normal after the development of left bundle-branch block ($P < 0.05$). When these three voltage criteria were normal before left bundle-branch block, they tended to remain normal after left bundle-branch block developed (Fig.). No clear pattern of change was noted in the other electrocardiographic criteria tested.

Discussion

The high incidence of anatomical left ventricular hypertrophy in 43 patients with complete left bundle-branch block (95.3%) is not surprising

since almost all our patients with left bundle-branch block had other associated cardiovascular disease. The strong association of anatomical left ventricular hypertrophy and other cardiovascular disease (especially hypertension and coronary artery disease) in patients with complete left bundle-branch block has been noted by others.⁹⁻¹¹ The severity of anatomical left ventricular hypertrophy in our population of 43 patients was unexpected. Mean heart weights and relative heart weights were twice normal predicted values. Furthermore, the mean heart weight of the 43 patients with complete left bundle-branch block was significantly higher ($P < 0.05$) than the mean heart weight of the 300 control necropsy patients with anatomical left ventricular hypertrophy but without complete left bundle-branch block. Interestingly, the mean heart weight in the subgroup of 11 patients with intermittent and recent onset left bundle-branch block was significantly lower ($P < 0.05$) than in the subgroup of 32 patients with persistent and indeterminate onset left bundle-branch block. This suggests, though does not prove, a progression of anatomical left ventricular hypertrophy in association with the development of complete left bundle-branch block.

Although sensitivity of currently employed electrocardiographic voltage criteria for left ventricular hypertrophy is relatively low when applied to our population of 41 patients with complete left

Fig. Change in electrocardiographic voltage in 25 patients before and after development of complete left bundle-branch block.

bundle-branch block and anatomical left ventricular hypertrophy (26.8~53.7%), the sensitivity is only slightly less than when the same criteria are applied to a group of patients with anatomical left ventricular hypertrophy but without left bundle-branch block (17.5~56.3%) according to Romhilt *et al.*'s⁸ data. Pathological criteria for anatomical left ventricular hypertrophy in our study differ from those used by Romhilt *et al.*⁸ who used weight of the isolated left ventricle as the criterion for anatomical left ventricular hypertrophy while we used total heart weight and total heart weight corrected for sex and body length as suggested by Zeek.⁷ Perhaps the sensitivity of the electrocardiographic voltage criteria we tested would have differed had we employed the same pathological method as Romhilt's group. Weight of the separated left ventricle appears to be a superior pathological measure of anatomical left ventricular hypertrophy than total heart weight even if the total heart weight is corrected for sex and body length, though most necropsy laboratories (including ours) do not employ this technique.¹²⁻¹³ Pathologically, left ventricular wall thickness has not been shown to correlate well with any other indices of left ventricular muscle mass,¹²⁻¹⁴ and the absence of correlation of this measurement in our study with electrocardiographic and other pathological criteria of left ventricular hypertrophy supports this observation. Our data regarding the sensitivity of various electrocardiographic voltage criteria for left ventricular hypertrophy in the presence of complete left bundle-branch block tend to parallel the recent findings of Cokkinos *et al.*³ who showed good correlation of pre- and post-left bundle-branch block Sokolow index values in a group of 40 patients with intermittent complete left bundle-branch block.

Interestingly, if one employs our most sensitive electrocardiographic voltage criteria as shown in the Fig. to the group of 25 patients with pre- and post-left bundle-branch block electrocardiograms, patients without electrocardiographic voltage criteria for left ventricular hypertrophy before the development of complete left bundle-branch block tend to retain normal voltage after complete left bundle-branch block develops while those with abnormally high voltage before left bundle-branch block tend to lose it after complete left bundle-branch block develops. This observation, plus the fact that our most sensitive electrocardiographic voltage criteria correlate modestly with total heart weight and percentage total heart weight of normal in 41 patients with both anatomical left ventricular hypertrophy and complete left bundle-branch

block, suggest that when abnormal electrocardiographic voltage remains in a patient with complete left bundle-branch block, anatomical left ventricular hypertrophy must certainly be suspected. The mere presence of complete left bundle-branch block in our population is associated with extreme left ventricular hypertrophy in nearly all (95.3%) patients. Perhaps, though not very sensitive, the presence of complete left bundle-branch block may be a specific marker of underlying anatomical left ventricular hypertrophy as judged by our study and that of Scott's.¹⁵ Exact specificity of the electrocardiographic variables for left ventricular hypertrophy in the presence of complete left bundle-branch block cannot be accurately gauged by this study since only two of 43 patients with complete left bundle-branch block were found not to have anatomical left bundle-branch block.

We were unable to show a difference in total heart weight and percentage total heart weight of normal between patients with complete left bundle-branch block and normal QRS axis and patients with complete left bundle-branch block and left axis deviation. However, recent data¹⁰ suggest that patients with both complete left bundle-branch block and left axis deviation experience more severe cardiac diseases, with more deaths from cardiovascular events than do patients with complete left bundle-branch block but with normal QRS axis.

The population sampled by our study differs considerably from the population of all persons with left bundle-branch block, and caution must be exercised in extending these findings to that population. A group of generally younger patients has been described¹⁶⁻¹⁷ who have complete left bundle-branch block but no other evidence of heart disease. The magnitude and status of this group are of principal interest. Since such patients were rarely encountered (1/12 of those with complete left bundle-branch block) in our control necropsy population of 480 patients, it appears that they either comprise a very small portion of the population of persons with left bundle-branch block or that they develop anatomical left ventricular hypertrophy at a higher rate than persons without left bundle-branch block. Perhaps some of the population of young persons with asymptomatic complete left bundle-branch block do indeed have a degree of anatomical left ventricular hypertrophy not detectable clinically, or longer follow-up of these persons would eventually reveal that anatomical left ventricular hypertrophy develops as a result of previously undetected associated heart disease.

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