Intermittent Left Bundle Branch Block: Anatomic Substrate as Reflected in the Electrocardiogram During Normal Conduction

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SUMMARY Examination of ECGs in patients with intermittent left bundle branch block (LBBB) may provide insight into mechanisms and cause of LBBB. In this study, we obtained ECG files of patients with intermittent LBBB by mail solicitation of cardiologists. The group suitable for analysis included 275 patients in whom both LBBB was documented and at least one 12-lead ECG was available which demonstrated absence of LBBB (ALBBB) after the first LBBB ECG. ALBBB ECGs revealed normal QRS in 151 patients (55%), and abnormal QRS in 124 (45%). These 124 had one or more of the following: left ventricular hypertrophy — 53 patients (19% of total group), myocardial infarction (MI) — 53 patients (19% of total group) and conduction disturbance (QRS > 0.10 sec and/or axis of -30° to -90°) — 52 patients (19% of total group). Sixty-three MIs were localized in the 53 patients; 31 (49%) were anteroseptal, seven (11%) were anterior, 17 (27%) were inferior and eight (13%) were lateral (p < 0.10). The 52 patients with conduction defects had the following: left anterior hemiblock — 32 patients (62% of conduction defects, 12% of total group), incomplete LBBB — 19 patients (31% of conduction defects, 7% of total group), right bundle branch block — one patient (2% of conduction defect, 0.1% of total group).

More than 50% of the patients with intermittent LBBB did not have abnormal QRS, suggesting an underlying cause of LBBB. If underlying infarction is present, it is most commonly anteroseptal, implying disease of the left anterior descending coronary artery.

The site of most intermittent LBBB appears predivisional (His or main left bundle), since preexisting left-sided unifascicular block is infrequent.

BY DEFINITION, PATIENTS with intermittent left bundle branch block (LBBB) have periods of conduction with LBBB and periods of conduction without LBBB. Examination of ECGs during absence of LBBB allows application of standard electrocardiographic criteria to diagnose fascicular conduction disturbances, hypertrophy and myocardial infarction. Application of standard electrocardiographic criteria during periods of normal conduction thus allows delineation, to a certain extent, of the underlying pathological substrate of intermittent LBBB.

In this series, we systematically analyzed electrocardiographic findings during normal conduction in patients with intermittent LBBB in an attempt to delineate the underlying pathologic substrate. In addition, we provide some insight into the pathophysiology of both intermittent and established LBBB.

Materials and Methods

Electrocardiographic files (inpatient and outpatient) from patients with intermittent LBBB were obtained by: 1) mail correspondence with cardiologists in 12 selected states (244 files); 2) use of coded electrocardiographic files located at the University of Minnesota Laboratory of Physiologic Hygiene (25 files); and 3) screening of inpatient and outpatient ECGs by members of the Cardiology Section, University of Illinois Hospital (six files). In this study, the following had to be available for each patient: 1) an ECG demonstrating complete LBBB, and 2) a subsequent ECG showing absence of LBBB (normal conduction). Standard criteria were used to document LBBB and were as follows: 1) QRS duration ≥ 0.12 second and 2) presence of notched M-shaped complexes in left precordial leads. Patients with intermittent atypical LBBB (q or S waves in V₅) were included. Two hundred seventy-five patient files were suitable for analysis. None of the patients had functional intermittent LBBB reflecting known hyperkalemia, drug intoxication or post-cardiac arrest.

The time interval between the LBBB and normal conduction tracings ranged from 0-2640 days; 154 (56%) of the 275 pairs of tracings (LBBB and normal conduction) had a time interval of shorter than 30 days. The age and sex of the patients were available in 231 (84%) of the 275 files, 122 (53%) were male and 109 (47%) were female with a mean ± sd age of 61.2 ± 14.8 years (range 10-91 years).

Criteria Applied to ECGs With Normal Conduction

For this study, we examined the ECGs with normal conduction to determine the presence of QRS abnormalities and T-wave changes. The QRS abnormalities included left ventricular hypertrophy, myocardial infarction and conduction disturbances. Left ventricular hypertrophy was diagnosed if one or more of the following voltage criteria were present: 1) S wave in V₁ + R wave in V₅ or V₆ > 35 mm, 2) R wave in lead aVₑ

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The presence and location of myocardial infarction were determined when significant q waves were present (≥ 0.04 seconds and/or 25% of R-wave height). Infarctions were localized as follows: anteroseptal q waves in V1 and V2 to V4, anterior q waves in V2 and V3 to V5, inferior q waves in II, III, and aVF, and lateral q waves in I and aVF and/or V6 and V5.

The following conduction defects were diagnosed: left anterior hemiblock, incomplete LBBB, right bundle branch block and nonspecific conduction disturbance. Left anterior hemiblock was diagnosed if the QRS axis was between −30° and −90° (QRS duration of 0.06–0.11 seconds). Incomplete LBBB was diagnosed if the QRS was 0.10 or 0.11 seconds long and had absent q waves in leads V5 and V6 and slurring or notching of QRS complexes in the left precordium.

All tracings with incomplete LBBB had a QRS axis greater than −30°. Right bundle branch block was diagnosed when QRS duration was ≥ 0.12 seconds and rSR' or rR' was present in lead V1. Non-specific conduction defect was diagnosed if the QRS had a duration of 0.10 or 0.11 seconds, but did not fulfill the criteria for left anterior hemiblock, incomplete LBBB or right bundle branch block. Left ventricular hypertrophy or anteroseptal myocardial infarction were not diagnosed in patients who had evidence of incomplete LBBB.

T-wave inversions were quantified during normal conduction as recently described. These were quantified only in the tracings that did not show QRS evidence of myocardial infarction, left ventricular hypertrophy or conduction disturbance. The tracings with T-wave inversions were grouped with respect to location of the inversions as follows: 1) precordial leads only, 2) standard leads only, and 3) precordial and standard leads.

The findings were recorded and subsequently transcribed to keypunch cards. The data was then entered into a Digital Equipment Corporation, Model PDP 11/45 computer located at the Arthur Goldberg Research Center, University of Illinois, Chicago. The INGRESS data base system was used for data retrieval and the chi-square method was used for statistical analysis.

Results

QRS Abnormalities

During normal conduction, normal QRS morphology was noted in 151 patients (55%) and 124 patients (45%) had abnormal QRS morphology. These 124 tracings showed one or more of the following QRS abnormalities: left ventricular hypertrophy — 53 (19% of total group), myocardial infarction — 53 (19% of total group) and conduction disturbance — 52 (19% of total group).

Sixty-three electrocardiographic locations of myocardial infarction were present in the 53 myocardial infarction tracings; 31 (49%) were anteroseptal, seven (11%) anterior, 17 (27%) inferior, and eight (13%) lateral. The incidence of anteroseptal myocardial infarction was significantly higher than the incidence of anterior and inferior or lateral myocardial infarction (p < 0.01). There were no other significant differences in sites of myocardial infarction.

The distribution of conduction defects in the 52 ECGs that showed conduction disturbances included 32 (62% of conduction disturbances, 12% of the total group) with left anterior hemiblock, 16 (31% of conduction disturbances, 6% of the total group) with incomplete LBBB, one (2% of conduction disturbances, 0.4% of the total group) with right bundle branch block, and three (1% of conduction disturbances, 1% of the total group) with nonspecific conduction disturbance. Two (67%) of the three ECGs with nonspecific conduction disturbance showed QRS evidence of left ventricular hypertrophy, and the other one (33%) exhibited QRS evidence of myocardial infarction.

T-Wave Inversions

Examination of the T waves in the 151 patients with normal QRS morphology yielded 79 (52%) with T-wave inversions and 72 (48%) without T-wave inversions. The T-wave inversions had the following distribution with respect to location: precordial leads only — 50 (63%); standard leads only — 21 (27%); and both standard and precordial leads — eight (10%).

Discussion

Most patients with intermittent LBBB develop established LBBB. Thus, our findings presumably relate to a patient population with early disease of the left bundle branch system (early relative to development of established LBBB). Since most of our tracings were obtained by mail correspondence with cardiologists, no bias would appear to be reflected in selecting patients with particular underlying disease processes or a particular level of severity of underlying cardiac disease.

Many studies of patients with established LBBB that used clinical and pathologic data have provided insight into the underlying cardiac pathology in these patients. Although some studies have presented small numbers of patients with established LBBB and no apparent associated cardiac disease, many more have demonstrated the common association of coronary artery disease or hypertensive heart disease with established LBBB. Lewis et al. found that 76-85% of documented cases of LBBB in the literature before their study had associated coronary artery disease and/or hypertensive heart disease. The severity of cardiac disease in patients with established LBBB is shown by the poor prognosis reported in many patients with the conduction defect.

Less information is available concerning patients with intermittent LBBB. In a comprehensive study done in 1938, Comeau et al. evaluated 60 patients with intermittent LBBB and found that 42 had evidence of hypertensive heart disease or coronary artery dis-
The electrocardiographic substrate of intermittent LBBB has been examined in two previous studies. Bauer has analyzed tracings with normal conduction from 12 patients with intermittent LBBB and found that three had normal ECGs and the others had left ventricular hypertrophy or myocardial infarction. More recently, Krikler and Lefevre reported two patients with intermittent LBBB who had normal tracings during normal conduction.

In the present study, analysis of the QRS complexes with normal conduction revealed that greater than 50% of the total group did not have an identifiable QRS abnormality. None of the specific diagnostic categories, such as left ventricular hypertrophy, myocardial infarction and conduction disturbance, was present in greater than 20% of the group. The small number of patients with left ventricular hypertrophy or myocardial infarction was particularly surprising, since there is a frequent association of hypertensive heart disease and coronary artery disease with established LBBB. One explanation for this apparent discrepancy is that the ECG with normal conduction in patients with intermittent LBBB is insensitive to underlying cardiac disease. However, this seems unlikely, since patients with hypertensive heart disease and coronary artery disease often have QRS abnormalities. A more likely explanation is that patients with intermittent LBBB have less severe cardiac disease than patients with established LBBB. Intermittent LBBB appears to reflect early disease of the left bundle branch system, and our data suggest that associated cardiac disease in these patients is at an early or less severe stage compared with patients who have established LBBB.

Only 20% of our patients had myocardial infarction, and the infarcts were present in diverse locations with varying frequencies. Previous workers have described an association of acute anterior or anteroseptal myocardial infarction with LBBB. This association presumably reflects the fact that the left anterior descending system is the dominant supply for the ventricular system, and thus the left bundle branch. Our data suggest a cause-and-effect relationship between intermittent LBBB and coronary artery disease in our patients, since most of these patients had anterior or anteroseptal localization of infarction. The relatively high incidence of infarctions at other locations in our patients suggests that the above anatomical explanation may be oversimplified. Explanations for our findings of other locations of infarct could include the following: 1) In keeping with recent findings of associated acute inferior wall myocardial infarction and LBBB, the blood supply to the left bundle branch originates from both right and left systems, the right supplying the proximal left bundle and His bundle; or 2) in some of our patients with intermittent LBBB, there is no cause-and-effect relationship between coronary artery disease and conduction defect.

In this series, only 6% had classical incomplete LBBB during periods of normal conduction. This finding suggests that LBBB in patients with intermittent LBBB is an "all or none" phenomenon. This is consistent with previous electrocardiographic findings concerning transition of normal conduction to rate-dependent LBBB, which usually occurs as a type II phenomenon.

If one considers the left bundle branch as bifascicular (anterior and posterior fascicles) with a proximal portion being predivisional (including a longitudinally dissociated portion of the distal His bundle), the site of intermittent LBBB could be either pre- or post-divisional. In the 12% of our patients with left anterior hemiblock, intermittent LBBB could reflect intermittent failure of the left posterior fascicle. In the remaining 88% of patients, the site of intermittent LBBB is probably predivisional (His bundle or proximal left bundle branch), since there probably would not be simultaneous failure of anterior and posterior divisions, and it is more likely that intermittent failure of a more proximally located structure is the site of block.

Recent studies by Denes et al. and Engel et al. have described characteristic precordial T-wave inversions in patients with intermittent LBBB during normal conduction. These T-wave inversions did not appear to reflect the presence of ischemic heart disease, since coronary arteriograms demonstrated the frequent absence of large-vessel obstructive coronary disease in patients with these T-wave changes during periods of normal conduction. It was suggested that these T-wave changes reflected an abnormality of repolarization related to previous occurrence of LBBB pattern. These changes closely resembled the T-wave inversions seen after cessation of right ventricular pacing (also LBBB pattern). How the heart translates an abnormality of depolarization (LBBB) into a subsequent abnormality of repolarization was unknown. Our study does not clarify the mechanism of this phenomenon, but does document the high incidence (50%) of characteristic T-wave changes during normal conduction in patients with intermittent LBBB.

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