Electrophysiological factors of left bundle-branch block^{*}

PETER A BARRETT,^{*} IWAO YAMAGUCHI, JAY L JORDAN,
WILLIAM J MANDEL[†]

From the Division of Cardiology, Department of Medicine, Cedars-Sinai Medical Center, and the Department of Medicine, UCLA School of Medicine, Los Angeles, California, USA

SUMMARY Left bundle-branch block is rarely an isolated disorder of conduction, additional disorders being found in 29 of 30 patients studied by intracardiac stimulation techniques. These included disorders of sinus node function (prolonged maximum sinus node recovery time (corrected) in 23%, prolonged sinuatrial conduction time in three of eight patients), atrioventricular node function (prolonged AH interval in 33%, prolonged effective and functional refractory periods in 37% and 74%, respectively), "His bundle to right bundle branch" conduction (prolonged HV interval in 53%), and ventriculoatrial conduction (absent in 62%). It is postulated that at least half of the cases of left bundle-branch block were incomplete, even though the duration of the QRS complex exceeded 120 ms, because of (further) leftward deviation of the mean frontal QRS axis with sufficiently premature atrial extrastimuli. Block may be complete or incomplete in left bundle-branch block with left axis deviation of -30° or more on the standard electrocardiogram.

The appearance of left bundle-branch block on the standard electrocardiogram indicates a disorder of conduction in part of the His-Purkinje system. Adequate assessment of conduction across the remaining His bundle to right bundle-branch cannot be made, however, as the PR interval also represents intra-atrial and atrioventricular nodal conduction time.¹⁻³ In addition, there may be disorders of conduction at other sites which are not immediately apparent, but which may be relevant to the patient's outcome.

Study of the "sick sinus syndrome"⁴ has made it clear that patients with manifest sinus node disorder may frequently have more generalised disturbances.⁵ Such a diffuse nature of the disorder may not be the sole property of the "sick sinus syndrome" and may be present in other apparently isolated conduction disorders. This study was designed to test the existence of a parallel diffuse conduction disorder in patients with left bundle-branch block.

Much controversy has existed, moreover, in

regard to the determinants of the mean frontal QRS axis in left bundle-branch block,⁶⁻⁸ ever since the introduction of the concept of bundle-branch block and the confusion between left and right bundle-branch block.^{9 10} An assessment of these determinants formed a second part of this study.

Subjects and methods

Thirty patients with left bundle-branch block were studied. Twenty-two (73%) were men. The ages of the patients ranged from 41 to 82 years, with a mean of 63 years. Seventeen (57%), 16 of whom were men, were diagnosed as having coronary heart disease, on the basis of angina or a past history of myocardial infarction, the latter being present in 11 patients. Eight of these 17 patients underwent coronary arteriography on a separate occasion. Significant disease (more than 70% stenosis) was present in the right coronary artery alone in one case, the left anterior descending coronary artery alone in two cases, and involved both the right and left systems in five cases. Six of the remaining 13 patients also underwent coronary arteriography and none had significant coronary artery disease. Fourteen patients (47%) had a past history of hypertension requiring anti-hypertensive medication 15 (50%) of clinical

^{*} Supported in part by a NIH grant.

[†] Present address: The St George Hospital, Kogarah, Sydney 2217, Australia.

[‡] Milly Factor Clinical Investigator of the Western Cardiac Foundation.

Received for publication 26 November 1980

cardiac failure, and 16 (53%) of syncope. Five patients (17%) had left bundle-branch block without clinical coronary heart disease, hypertension, or cardiac failure.

Electrophysiological study was performed in the fasting state, after obtaining informed consent. The patients had not received digoxin or antiarrhythmic medication for at least 48 hours before study. All patients were in sinus rhythm. The PR interval and QRS complex duration were determined from the standard electrocardiogram. The mean frontal QRS axis was determined from the most equiphasic QRS complex, in terms of net amplitude, as opposed to area.

Intracardiac electrograms were obtained by means of standard techniques.¹¹ The electrograms were recorded on a multichannel photographic recorder, at a paper speed of 100 mm/s (Electronics for Medicine, Model DR-12). Standard electrocardiographic leads were recorded, with high right atrial and His bundle electrograms.

The AH and HV intervals were measured during sinus rhythm.

High right atrial pacing¹² was performed at increasing rates, with increments of 20 beats per minute, until failure of 1:1 atrioventricular conduction. The shortest cycle length of atrial pacing with persistent 1:1 atrioventricular node conduction was used as a measure of the atrioventricular node effective refractory period. At each rate the sinus node recovery time¹³ was measured after cessation of atrial pacing for 60 seconds. The maximum sinus node recovery time (corrected) was obtained by subtracting the basic sinus cycle length from the maximum sinus node recovery time.

The atrioventricular node effective refractory period was also determined by means of the atrial extrastimulus technique,¹⁴ as well as the atrioventricular node functional refractory period. Programmed premature atrial extrastimuli were introduced during atrial pacing at a rate slightly in excess of the sinus rate. The sinuatrial conduction time was determined when atrial extrastimuli were also introduced during sinus rhythm.¹⁵ The QRS complex of conducted atrial extrastimuli was examined for further leftward deviation of the mean frontal QRS axis.¹⁶

Both techniques were used for the estimation of the atrioventricular node effective refractory period because each is commonly used alone, and because each measures the atrioventricular node effective refractory period under different conditions.

Right ventricular apical pacing¹⁷ was performed at increasing rates, with increments of 20 beats per minute, until failure of 1:1 ventriculoatrial conduction. The shortest cycle length of ventricular

pacing with persistent 1:1 ventriculoatrial conduction was used as a measure of the ventriculoatrial effective refractory period.

The ventriculoatrial effective refractory period was also determined by means of the ventricular extrastimulus technique,¹⁷ with introduction of programmed premature ventricular extrastimuli during ventricular pacing at a rate slightly in excess of the sinus rate, when this was associated with 1:1 ventriculoatrial conduction.

Normal values for each of the variables measured were taken as those which apply in our laboratory. Methods of statistical analysis are indicated in the text.

Results

Twenty-nine of the 30 patients (97%) were shown to have one or more additional disorders at other sites. Of possible additional disorders of sinus and atrioventricular node function, His bundle to right bundle-branch conduction, and ventriculoatrial conduction, nine patients had a single additional disorder, 13 had two, four had three, and three had four additional disorders.

SINUS NODE FUNCTION (Fig. 1)

The maximum sinus node recovery time (corrected) ranged from 87 to 1639 ms, with a mean of 355 ms, in 26 patients studied. It was prolonged

Fig. 1 Sinus node function—maximum sinus node recovery time (corrected) (SANRTC), and sinuatrial conduction time (SACT). The broken lines, in this and the other figures, indicate the upper limit of normal. Mean $\pm$ standard error of the mean are also indicated.

(more than 450 ms) in six of these (23%). The sinuatrial conduction time ranged from 62 to 140 ms (mean 99 ms) in eight patients studied. It was prolonged (more than 120 ms) in three of these (38%).

ATRIOVENTRICULAR CONDUCTION TIMES

(Fig. 2)

The PR interval ranged from 140 to 340 ms (mean 199 ms) in the whole group. It was prolonged (more than 200 ms) in eight patients (27%). The AH interval ranged from 50 to 195 ms (mean 115 ms). It was prolonged (more than 120 ms) in 10 patients (33%). The HV interval ranged from 35 to 120 ms (mean 62 ms), being prolonged (more than 55 ms) in 16 patients (53%).

Of the eight patients with a prolonged PR interval, the AH and HV intervals were prolonged in seven and five patients, respectively. Similarly, of the 10 patients with a prolonged AH interval, the PR interval was prolonged in seven, but of the 16 patients with a prolonged HV interval, the PR interval was prolonged in only five patients.

Fig. 2 Atrioventricular conduction times during sinus rhythm, measured from the standard electrocardiogram (PR interval) and the His bundle electrogram (AH and HV intervals).

Fig. 3 Atrioventricular node effective (E) and functional (F) refractory periods (RP), as determined by atrial pacing and atrial extrastimulation.

ATRIOVENTRICULAR NODE REFRACTORY PERIODS (Fig. 3)

In 29 patients studied the atrioventricular node effective refractory period, as determined by the method of incremental atrial pacing, ranged from 273 to 857 ms (mean 441 ms). It was prolonged (more than 445 ms) in 12 of these 29 patients (41%).

As determined by the atrial extrastimulus technique, the atrioventricular node effective refractory period ranged from 205 to 690 ms (mean 314 ms) in 27 patients studied. It was prolonged (more than 320 ms) in 10 of these (37%). The atrioventricular node functional refractory period ranged from 350 to 760 ms (mean 448 ms). It was prolonged (more than 400 ms) in 20 patients (74%).

VENTRICULOATRIAL EFFECTIVE REFRACTORY PERIODS

In 13 of 21 patients studied (62%) 1:1 ventriculoatrial conduction was absent at all rates during

ventricular pacing. In the eight remaining patients, the ventriculoatrial effective refractory period, as determined by the method of incremental ventricular pacing, ranged from 400 to 750 ms (mean 593 ms). The ventricular extrastimulus technique was used to estimate the ventriculoatrial effective refractory period in six of these eight patients with intact 1:1 ventriculoatrial conduction. It ranged from 360 to 615 ms (mean 456 ms).

IDIOPATHIC LEFT BUNDLE-BRANCH BLOCK

Of the five patients with idiopathic left bundle-branch block, the HV interval was prolonged in three, the atrioventricular node effective and functional refractory periods in one and two, respectively, 1:1 ventriculoatrial conduction was absent in two, and sinuatrial conduction time was prolonged in one of the three cases in which it was estimated.

INTRAVENTRICULAR CONDUCTION

The mean frontal QRS axis ranged from 80° to -70° , and was -30° , or further leftward, in 15 patients (50%).

During the atrial extrastimulus technique, with progressively premature atrial extrastimuli, leftward deviation of the mean frontal QRS axis (Fig. 4) developed and persisted in 12 of 25 patients studied (48%). It occurred at His coupling (H_1H_2) intervals ranging from 410 to 575 ms (mean 483 ms). In these patients, the atrioventricular node functional refractory period (the minimum

Fig. 4 Leftward deviation of the mean frontal QRS axis with sufficiently premature atrial extrastimuli. At a coupling interval (A_1S_2) of 565 ms, the mean frontal QRS axis remains 30° . When A_1S_2 is decreased to 410 ms, the QRS axis becomes -30° . HBE, His bundle electrogram; S_2 , stimulus artefact; I, II, III, standard electrocardiographic leads.

Fig. 5 Lack of relation between the mean frontal QRS axis and the presence of coronary heart disease, hypertension, cardiac failure, syncope, or leftward deviation of the mean frontal QRS axis (LAD) with atrial extrastimuli.

H_1H_2 interval) ranged from 350 to 500 ms (mean 412 ms). In the remaining 13 patients, where there was no change in the QRS axis with premature atrial extrastimuli, the atrioventricular node functional refractory period was generally longer, ranging from 355 to 760 ms (mean 482 ms).

There was no statistically significant association ($p > 0.05$, χ^2) between a mean frontal QRS axis of -30° or further leftward, on the standard electrocardiogram, and the presence of coronary heart disease, hypertension, cardiac failure, syncope, or the development of leftward deviation of the QRS axis with premature atrial extrastimuli (Fig. 5). In particular, further leftward deviation of the QRS axis with premature atrial extrastimuli occurred in six of 14 patients (43%) with left axis deviation of the QRS complex, of -30° or more, on the standard electrocardiogram.

Similarly, there was no statistical correlation (Spearman's test for rank-difference correlation) between the mean frontal QRS axis and the duration of the QRS complex ($r = -0.1$), or the HV interval ($r = -0.2$) (Fig. 6).

Discussion

In the study of the sick sinus syndrome⁴ it has become apparent that a range of disorders may

exist at other sites in the heart.⁵ It would not be unusual, therefore, if a similar situation applied in the case of left bundle-branch block. It may be that many manifest disorders of conduction are associated with unsuspected disorders at other sites. As regards left bundle-branch block, there have been several reports¹⁸⁻²⁰ indicating the frequency of associated conduction delay in the atrioventricular node and His bundle to right bundle-branch (prolonged AH and HV intervals, respectively), but there has been no systematic examination of sinus node function, anterograde and retrograde atrioventricular conduction, and intraventricular conduction in these patients.

CLINICAL FEATURES

In our study, 73 per cent of the 30 patients were men, and coronary heart disease was common, being present in 57 per cent of the whole group.

Fig. 6 Lack of relation between the mean frontal QRS axis and the QRS complex duration or the HV interval.

At least in part this reflects patient selection, as many were primarily being investigated for coronary heart disease. The common association between left bundle-branch block and hypertension and cardiac failure, present in 47 per cent and 50 per cent of the patients, respectively, is consistent with previous reports.^{10 18 21} In Blondeau's large series,²¹ 60 per cent of 608 patients with left bundle-branch block were men, coronary heart disease was present in 31 per cent, and hypertension in 32 per cent. In both his series and ours, left bundle-branch block was idiopathic in 17 per cent. In terms of aetiology, therefore, our sample of patients appears to be fairly representative of the total population of patients with left bundle-branch block.

ASSOCIATED CONDUCTION DISORDERS

In addition to left bundle-branch block, all except one of our patients were shown to have at least one other disorder, of sinus or atrioventricular node function, His bundle to right bundle-branch conduction, or ventriculoatrial conduction. The maximum sinus node recovery time (corrected) was prolonged in 23 per cent of the patients, and the sinuatrial conduction time in three of eight patients studied. Atrioventricular node function was commonly abnormal. First degree atrioventricular node block (prolonged AH interval) was present in 33 per cent of the patients. The atrioventricular node effective refractory period was prolonged in 37 to 41 per cent, depending on the method of determination, and the atrioventricular node functional refractory period in 74 per cent. His bundle to right bundle-branch conduction was also frequently affected, first degree block (prolonged HV interval) being present in 53 per cent of the patients. Though of uncertain significance, absence of ventriculoatrial conduction was very common, being present in 62 per cent of the patients. Even though our patients were somewhat selected, in that a history of syncope was present in 53 per cent, such a prevalence and range of associated disorders were unexpected. Should syncope develop in patients with left bundle-branch block, in some of these it may be on the basis of sinus or atrioventricular node disorder, as opposed to high grade atrioventricular block in the His-Purkinje system.

Moreover, it is difficult to determine from the standard electrocardiogram which patients have an additional disorder of conduction in the remaining His bundle to right bundle-branch. In our patients, first degree atrioventricular block correlated more closely with first degree atrioventricular node block, as opposed to first degree His bundle to right bundle-branch block.

INTRAVENTRICULAR CONDUCTION

As there was no association between left axis deviation of the QRS complex on the standard electrocardiogram and the presence of coronary heart disease, hypertension, or cardiac failure, left axis deviation in left bundle-branch block in our patients did not appear to be primarily related to left ventricular function. Similarly, as the QRS axis did not correlate with the HV interval or the duration of the QRS complex, it did not appear to be primarily related to His bundle to right bundle-branch conduction time or total ventricular activation time.

Haft *et al.*²² similarly reported no significant difference in the prevalence of coronary heart disease or left ventricular dysfunction in patients with left bundle-branch block and a normal or leftward QRS axis. Swiryn *et al.*²³ found no association between the mean frontal QRS axis in left bundle-branch block and the presence of myocardial infarction or left ventricular hypertrophy. In studying patients with left bundle-branch block and a normal, intermediate, or prolonged HV interval, Rosen *et al.*¹⁸ reported no significant difference in the mean frontal QRS axis, and Lichstein *et al.*²⁴ found no difference in the QRS complex duration in patients with left bundle-branch block and a normal or leftward QRS axis.

Dhingra *et al.*²⁰ however, while reporting no difference in the prevalence of hypertension in patients with left bundle-branch block and a normal or leftward QRS axis, noted an increased prevalence of coronary heart disease and related myocardial dysfunction in the group with left axis deviation, and also of HV interval prolongation, previously reported by Spurrell *et al.*²⁵ No doubt these discrepancies reflect patient selection, and it is possible that other factors in the determination of the mean frontal QRS axis in left bundle-branch block are more basic.

The occurrence of leftward deviation of the QRS axis with sufficiently premature atrial extrastimuli,¹⁶ present in 48 per cent of our patients, suggests the development of refractoriness in part or all of the left bundle-branch, that is, that left bundle-branch block was previously incomplete. This suggests, therefore, that a normal QRS axis in left bundle-branch block indicates that the latter is incomplete. It is possible, however, that other factors, such as lateral wall myocardial infarction in the presence of complete left bundle-branch block, could also result in a normal QRS axis.

Failure of premature atrial extrastimuli to result in leftward deviation of the QRS axis, in 52 per cent of the patients, initially suggested that, in these cases, left bundle-branch block was already

complete, in spite of the fact that the QRS axis was not necessarily leftward. In these patients, however, the atrioventricular node functional refractory period was generally prolonged, and so in many cases may not have allowed His coupling (H_1H_2) intervals short enough to result in leftward axis deviation of the QRS complex. Hence, the number of cases where left bundle-branch block was in fact incomplete may have been considerably greater than 48 per cent, even though the common,^{10 26 27} but rather arbitrary, criterion for "complete" bundle-branch block, of a QRS complex duration equal to or exceeding 120 ms, was satisfied.

There are anatomical considerations which support the view that when left bundle-branch block is complete, the QRS axis should be leftward and superior. In this situation, the cardiac impulse is conducted solely across the right bundle-branch and initially activates the ventricular myocardium in the area of the anterior papillary muscle of the right ventricle.^{10 28 29} Subsequent left ventricular activation results from spread of the activation front from this area. The mean direction of such spread, therefore, may be expected to be leftward, superior, and posterior,^{10 26} such as occurs in pacing from the apex of the right ventricle.³⁰

On the standard electrocardiogram, a QRS axis of -30° , or further leftward, is common in left bundle-branch block^{20 21 31} and was present in 50 per cent of our patients. In these cases, left bundle-branch block may not necessarily be complete, however, as further leftward deviation of the QRS axis with premature atrial extrastimuli occurred in 43 per cent of such patients. To determine which of the remainder are also in fact incomplete, the duration of the QRS complex is of limited value, as it depends on other factors, such as right-to-left transseptal conduction time.^{26 32} The atrial extrastimuli technique, with observation of leftward deviation of the mean frontal QRS axis with sufficiently premature atrial extrastimuli, could be adapted so that extrastimuli are produced at the area of the His bundle, thereby overcoming the limitation of the frequently prolonged atrioventricular node functional refractory period. The technique would then be limited only by the local cardiac muscle functional refractory period.

In Swiryn's study,²³ 23 per cent of 79 patients with left axis deviation during left bundle-branch block had known disorder of conduction in the left anterior fascicle of the left bundle-branch, because left anterior hemiblock was present when left bundle-branch block was absent. Such was the case, however, in only 5 per cent of 152 patients with a normal axis during left bundle-branch

block. A similar discrepancy was noted by Lichstein *et al.*²⁴ It is possible that in left bundle-branch block with left axis deviation, left bundle-branch block may be incomplete with greater delay in impulse conduction across the left anterior fascicle (in its pre-divisional or post-divisional course) as compared with the posterior fascicle. Posterior greater than anterior fascicular delay could result in incomplete left bundle-branch block with a normal axis. Some evidence for this is available.³³ The duration of the QRS complex could be similar in either case.

Although this concept was not able to be examined in this study, it would explain the lack of correlation between the mean frontal QRS axis and the duration of the QRS complex, and the other features examined, a "final" left axis deviation and a "final" QRS complex duration not being reached until left bundle-branch block is complete. Schamroth and Bradlow³⁴ showed progressive widening of the QRS complex in incomplete left bundle-branch block as it becomes "more complete", with some incomplete forms having a QRS duration greater than 120 ms. Though only one lead was available, examination of their tracings does suggest a concomitant change in QRS axis as left bundle-branch block becomes complete.

As shown in this study, however, whether left bundle-branch block is complete or incomplete, additional conduction disorders at other sites in the heart are common, and these may independently influence prognosis.

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Requests for reprints to Dr William J Mandel, Cedars-Sinai Medical Center, Box 48750, Los Angeles, California 90048, USA.