The Concept of "Masquerading" Bundle-Branch Block
An Electrocardiographic-Pathologic Correlation

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This is a clinicopathologic study of 2 patients who electrocardiographically presented so-called "masquerading" bundle-branch block. The conduction systems and the entire hearts of these patients were studied pathologically by methods especially devised by Lev for electrocardiographic correlation. This report is part of a long-term project attempting to ascertain the anatomic substrate of electrocardiographic abnormalities.

The electrocardiographic complex referred to as "masquerading bundle-branch block," consisting of the pattern of left bundle-branch block (LBBB) in the limb leads and right bundle-branch block (RBBB) in the unipolar precordial leads, has generated considerable speculation and interest relating to possible mechanisms responsible for its production. It has been designated "masquerading" by Richman and Wolff because they thought that even though the precordial leads suggested RBBB according to Wilson's criteria (right precordial leads showing late R or R' deflections, regardless of the morphology of the limb leads), this complex was actually the result of LBBB. This discrepancy was believed to be the result of the probable transmission of high septal potentials through the infarcted free right ventricular wall. These authors described 4 such cases in which the vectorcardiograms were interpreted as LBBB. The vectorcardiograms obtained in their cases differed markedly from those usually seen in LBBB except for the initial vectors, which were thought to be typically suggestive of LBBB. The marked difference in the remainder of the vectorcardiogram was attributed to the complication of septal infarction. A postmortem examination was reported by these authors on a case in which extensive infarction of the interventricular septum and the posterolateral wall of the left ventricle was found. No histologic study of the conduction system was mentioned.

Sodi-Pallares and Rodríguez in an earlier study on activation of the interventricular septum and the clinical evaluation of septal damage described this same electrocardiographic complex. In an effort to clarify this discrepancy between limb and precordial leads, they studied additional leads taken around the thorax at different levels searching for the electrocardiographic evidence of LBBB in these leads to conform with what the limb leads showed. In some of their cases this was found in leads taken in higher interspaces than the conventional chest leads. However, even here the precordial leads showed delayed intrinsicoid deflections over both the right and left precordial regions. The explanation offered was that the infarct had involved the lower portion of the interventricular septum and invaded the adjacent right and left free ventricular walls. The infarct then would extend in the septum to portions of the septum formed by the right ventricle and would only partially involve the portion formed by the left ventricle. The left intraventricular cavity potential would be that found in the presence of LBBB with the pattern varying, depending upon the orientation of the precordial elec-
trodes to the infarcted portions of the septum and left ventricle, producing an RS complex over infarcted areas and a broad notched R over portions of the left ventricle which were not infarcted or were not oriented toward the infarcted septum. Leads over the right ventricle would show a qR, qR′, or R′ complex, depending on their orientation to different portions of the septum. Despite the late R over the right precordial leads, these investigators thought that the lesion represented by the electrocardiogram was referable to LBBB rather than RBBB or bilateral BBB. In a subsequent paper from the same group (Rodriguez, Anselmi, and Sodi-Pallares), the possibility of an alteration of conduction in both bundle branches is mentioned. This explanation, however, is discarded in favor of one explaining the findings on the basis of LBBB alone.

In a review of the literature, we could find no postmortem studies in these cases with detailed examination of the conduction system and a correlation of these findings with multiple unipolar precordial leads in addition to standard limb leads. We therefore studied 2 of our cases of the same type heretofore described, which had come to postmortem examination, utilizing the method of study of the conduction system and of the entire heart devised by Lev and his associates. Vectorcardiograms were not done on these patients.

MATERIALS AND METHODS

The electrocardiograms were taken with the Sanborn direct-writing electrocardiograph. Conventional standard limb leads, augmental unipolar extremity leads, and unipolar chest leads were routinely taken on both patients.

Each heart at autopsy was opened in the traditional manner along the course of the circulation, with the exception that the initial cut passed from the inferior vena cava through the right atrial appendage, thus sparing the sinoatrial (SA) node. The heart was then fixed in neutral formalin for 2 to 7 days, at the end of which time all chambers were photographed. The SA node and its approaches were then removed for serial sectioning, as previously published, and every twentieth section was retained. A second block was made from the right atrial appendage and the superior wall of the right atrium. This block contained the ramus ostii cavae superiores, the blood supply to the SA node. This block was cut serially and every twentieth section retained. The anterior and inferior walls of both atria and ventricles were then removed from the atrial and ventricular septa. The atrioventricular (AV) node, bundle, and bundle branches up to the region of the papillary muscles were then included in 5 blocks. The AV node and its approaches (including the blood supply—the ramus septi fibrosi) and the penetrating portion of the bundle of His were present in the first block and were sectioned serially, every twentieth section being retained. The branching portion of the bundle of His and the beginning of the right and left branches were present in the second block and were sectioned serially, every fortieth section was retained. The remainder of the bundle branches up to the level of the papillary muscles of the left ventricle and the moderator band were found in the third, fourth, and fifth blocks and were sectioned serially, every fortieth section being retained. The remainder of the heart—the atrial septum, the most anterior part of the ventricular septum, the posterior ventricular septum, and the anterior and posterior walls of both atria and ventricles—were completely cut into blocks, and 2 sections were made from each block. Sections were cut at 6 or 7 microns. All sections were stained alternately with hematoxylin-eosin and Weigert-van Gieson stains. The number of sections thus obtained were 1,081 in case 1 and 854 in case 2.

CASE REPORTS

Case 1. B. S., a 75-year-old white man was admitted to the hospital on November 28, 1954, for congestive heart failure. He was a known hypertensive patient for many years and for over 2 years had experienced angina. He denied knowledge and symptoms of a definite coronary thrombosis in the past. Prior to admission his angina increased in both frequency and severity with concomitant increase in his congestive heart failure and with an episode of protracted chest pain. Following admission to the hospital his course persistently worsened. Peripheral vascular collapse did not develop but he remained in congestive failure. His course was marked by cerebral vascular insufficiency. Treatment consisted of digitalis, oxygen, diuretics, and other supportive measures. Quinidine was employed because of frequent premature ventricular contractions. He suddenly died on December 2, 1954, the fourth hospital day.

The laboratory data were nonecontributory except for 3+ albuminuria, a specific gravity of 1.010, and negative serologic tests.

Electrocardiographic Examination. Two electrocardiograms were available for study, one taken 1 month prior to admission and another 1 day after
THE CONCEPT OF "MASQUERADING" BUNDLE-BRANCH BLOCK

admission. An electrocardiogram taken on October 26, 1954 (fig. 1), showed atrial fibrillation with ventricular premature contractions. The standard limb and unipolar extremity leads showed a LBBB configuration, whereas the precordial leads showed RBBB. The intrinsicoid deflection in V₆ was 0.12 second, and 0.06 second in aVL. The QRS interval was 0.12 second. (There was infarction of the septum, anterior and posterior walls.) No true left ventricular leads were evident in the usual precordial locations and, unfortunately, no exploratory leads were taken. These electrocardiographic findings are consistent with RBBB, left ventricular hypertrophy, infarction of the septum and free walls of the left ventricle, and possibly right ventricular hypertrophy. An alternative interpretation is "masquerading" BBB.

An electrocardiogram taken on November 29, 1954, showed atrial fibrillation with ventricular premature contractions and LBBB pattern in standard limb and unipolar extremity leads. The QRS interval was 0.12 second. The intrinsicoid deflection in the right precordial leads was 0.12 and 0.06 second in aVL. The interpretation is the same as for the previous electrocardiogram.

Postmortem Examination. Aside from the findings in the heart, the pathologic diagnoses were (1) arteriolar nephrosclerosis with arteriolar necrosis, (2) generalized arteriolar sclerosis with early necrosis, (3) chronic passive hyperemia of the lungs, liver and spleen, (4) bilateral bronchopneumonia, lower lobes, and (5) chronic cholecystitis with cholelithiasis.

Heart. Gross Examination. The heart weighed 630 Gm. The epicardium was somewhat puckered over the posterior wall of the left ventricle. The myocardium in this region was somewhat firmer than normal, and white streaking permeated this

![Fig. 1. Case 1. 10/26/54. Atrial fibrillation with premature ventricular contractions. LBBB in standard and unipolar extremity leads and RBBB in unipolar precordial leads. Intrinsicoid deflection is 0.12 second in V₆, 0.06 in aVL. QRS duration is 0.12 second. Anteroseptal and posterior myocardial infarction.](http://circ.ahajournals.org/)

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subepicardial and perivascular fibrosis with a dispersion of small microscopic scars and occasional organizing infarcts. These changes were most marked in the posterior wall. The posterior part of the ventricular septum was similarly markedly involved with a lesser involvement of the anterior part of the septum. The free walls of the right ventricle showed only occasional small scars with minimal fibrosis.

Pathology of the Conduction System. The ramus ostii cavae superioris showed no change. The ramus septi fibroso showed slight narrowing. The SA node showed degeneration of occasional isolated fibers. At the approaches to the SA node, the posterior crest and other atrial musculature presented slight to moderate fibrosis with an occasional small scar. The approaches to the AV node showed slight to moderate fibrosis. The AV node presented no change. The penetrating portion of the bundle of His presented a small scar near its termination. The branching portion of the bundle of His at its bifurcation showed elastosis and compression by a calcified mass in the adjacent ventricular musculature (fig. 2). Right bundle branch (fig. 3C, D): Close to its origin, there was marked fibrosis which diminished somewhat up to the region of the muscle of Lancisi. In addition, this segment showed some acute degenerative changes. Distal to the muscle of Lancisi the fibrosis became more marked, amounting at 1 point to about four-fifths replacement of the bundle branch. The process again diminished to be followed by almost complete replacement by connective tissue. The most distal portion and the divisions of the moderator bundle were not available for study. Left bundle branch (fig. 3C, D): There was calcification of the septum adjacent to, but not involving, the beginning of the LBB. Slight fibrosis was focally present throughout. In addition, in the upper portion there were mild acute degenerative changes with a scattered infiltration of lymphoid cells. In the distal portion there was an increase in the extent of the acute degenerative changes, accompanied by fatty infiltration and a fine dispersion of round cells.

Pathologic Diagnoses of the Heart. (1) Marked hypertrophy of the right and left ventricles, (2) arteriosclerosis of the myocardium with superimposed mild acute vascular degeneration, (3) diffuse fibrosis with small scars of the myocardium, with maximal involvement of the posterior part of the ventricular septum and posterior wall of the left ventricle, (4) elastosis and compression of the common bundle at its bifurcation, (5) marked fibrosis of the RBB and slight fibrosis of the LBB, and (6) focal acute degenerative changes of the ventricular myocardium and the right and left bundle branches.

Electrocardiographic-Pathologic Correlation. In
a patient with hypertensive and arteriosclerotic heart disease, electrocardiographic findings suggested septal infarction with involvement of the free walls of the left ventricle, and there was an electrocardiographic pattern of LBBB in the limb leads and RBBB pattern in the precordial leads. Autopsy findings revealed hypertensive and arteriosclerotic heart disease with generalized fibrosis and small scars involving maximally the posteroseptal wall of the left ventricle, elastosis and compression of the atrioventricular bundle at its bifurcation, marked fibrosis of the RBB, and slight fibrosis of the LBB.

Case 2. S. A., a 61-year-old male carpenter, was initially hospitalized on November 14, 1949, because of exertional dyspnea of 6 months’ duration.

**Fig. 4** Top. Case 2. 12/5/49. Normal sinus rhythm. Left ventricular hypertrophy. QRS duration 0.10 second, P-R 0.19 second.

**Fig. 5** Bottom. Case 2. 12/4/51. Normal sinus rhythm. QRS 0.13 second. Q wave has disappeared in L1 and aVL. Change from previous tracing demonstrating intraventricular conduction defect of LBBB configuration.
On admission, his blood pressure was 230/120 and there were findings of congestive heart failure. He was discharged 1 month after admission, on digitalis, after diagnoses were made of arteriolonephrosclerosis, hypertensive heart disease with left ventricular hypertrophy and congestive heart failure. The patient was readmitted on April 28, 1951, because of recurrent cardiac insufficiency. There was no interim history of precordial distress. The blood pressure was 250/120 and the physical findings included those of right- and left-sided heart failure. He was treated as in his previous admission, losing 22 lb. in 2 months. The patient had 2 subsequent hospital admissions on November 11, 1951, and July 2, 1954, for recurrent symptoms of congestive failure with clinical evidence of severe failure and moderately severe hypertension. The heart showed progressive enlargement to an extremely large size. His fifth and final admission on October 1, 1954, was for intermittent epigastric pain associated with vomiting. On examination, in addition to severe congestive failure, he showed evidence of a perforated abdominal viscus and massive atelectasis of the right lung. Because he was a poor surgical risk, he was treated with Wangensteen suction and a Cofflator. The right lung re-expanded, but the patient lapsed into coma and died on the sixth hospital day. No history of angina could be obtained after repeated questioning.

Pertinent laboratory data during the long period of observation included a positive blood Kahn test (less than 10 units), hyposthenuria, and moderate retention of urea nitrogen. A benzodioxane test was negative for evidence of pheochromocytoma.

Electrocardiographic Examination. An electrocardiogram taken December 5, 1949 (fig. 4), on the first hospital admission showed a normal sinus rhythm and suggestive evidence of left ventricular hypertrophy. The QRS interval was 0.10 second and the P-R interval was 0.19 second.

An electrocardiogram taken on December 4, 1951 (fig. 5), on the third hospital admission, showed a normal sinus rhythm, P-R interval of 0.20 and QRS of 0.13 second. The Q wave had disappeared in lead I and aV_{6}. A change from the previous tracing was noted, with development of an intraventricular conduction defect of LBBB configuration.

An electrocardiogram taken July 2, 1954, on the fourth hospital admission, showed a normal sinus rhythm, P-R interval of 0.20 and QRS of 0.13 second. RBBB configuration was now present in the right precordial leads and LBBB configuration in the standard and unipolar extremity leads. The intrinsicoid deflection was 0.12 second in V_{1} and V_{6}.
An electrocardiogram taken July 6, 1954 (fig. 6), showed a normal sinus rhythm, P-R interval of 0.19 second and QRS of 0.14 second. RBBB was present in right precordial leads and LBBB in standard and unipolar extremity leads. The intrinsoid deflection was 0.11 second in V₁ and 0.12 second (second peak) in V₄. There was no diagnostic electrocardiographic evidence of myocardial infarction.

Postmortem Examination. Aside from the findings in the heart, the pathologic diagnoses were (1) chronic passive hyperemia of the lungs with pulmonary edema, (2) chronic ulcer of the stomach with perforation, (3) fatty metamorphosis of the liver with periportal fibrosis, (4) congenital absence of the right kidney, (5) arteriolsclerosis of the left kidney with nephrosis, (6) ascites, (7) interus, and (8) cyanosis.

Heart. Gross Examination. The heart weighed 950 Gm. Both ventricles were markedly hypertrophied, the left, however, dominantly so. Both atria shared in the hypertrophy. The anterior descending coronary artery coursed deep into the ventricular myocardium. There was only minimal sclerosis of the coronary arteries, and no narrowing.

Microscopic Examination. General Pathology. There was diffuse arteriolsclerosis with acute vascular degeneration, with associated degenerative and inflammatory changes in collagen and fat tissue.

Pathology of Individual Chambers and Walls. The myocardium of the free walls of the atria and the atrial septum showed diffuse fibrosis, which was more marked on the right side. The myocardium of the free walls of the left ventricle, the ventricular septum and the anterior wall of the right ventricle showed marked fibrosis with microscopic scars. This was more marked in the inner two-thirds of the walls. Subepicardially, there were also acute degenerative changes in the myocardial fibers. The posterior wall of the right ventricle showed similar slight changes.

Pathology of the Conduction System. There was no narrowing of the ramus ostii cavae superioris. The SA node showed no change. The approaches to the SA node showed considerable fibrosis, with acute degenerative changes of the myocardial fibers. The approaches to the AV node showed acute degeneration of the fat tissue with focal infiltration of macrophages. The AV node was normal. The penetrating portion of the AV bundle showed the above-mentioned changes in the associated fat and collagen. The branching portion of the bundle showed marked elastosis and what appeared to be compression at the bifurcation. Right bundle branch (fig. 7A, B): At the origin, there was slight fibrosis. This became progressively more marked at the muscle of Lancisi, so that at one point it was considerably replaced by connective tissue. In addition, focal acute degenerative changes were noted in this portion. Distal to the muscle of Lancisi, the bundle branch lay in a scar where at one point it was markedly, but not completely, replaced. It then was the seat of moderate fibrosis, which was present until its termination. Here it was surrounded by collagen and fat showing degenerative changes. The most distal part of the moderator band and its branches were not available for study. Left bundle branch (fig. 7C, D): There were fibrosis and compression at the beginning of the LBB, which persisted focally more distally. In addition, there were collagen and fat changes around the fasciculi, with focal degeneration of some of the bundle fibers.

Pathologic Diagnoses of the Heart. (1) Massive hypertrophy of the heart (left ventricle maximal, right ventricle marked, both atria marked), (2) intramyocardial course of the left anterior descending coronary artery, (3) marked arteriolsclerosis with acute vascular and perivascular degeneration, (4) marked fibrosis of the myocardium with small scars involving the septum, the anterior and posterior walls of the left ventricle, and the anterior wall of the right ventricle maximally, the right atrium moderately, and the left atrium, atrial septum, and the inferior wall of the right ventricle slightly, (5) compression and elastosis of the bundle of His at its bifurcation, and marked fibrosis of the RBB with moderate fibrosis of the LBB, (6) focal acute degenerative changes of the right and left bundle branches.

Electrocardiographic-Pathologic Correlation. In a patient with syphilis, a peptic ulcer and hypertensive heart disease, the electrocardiographic diagnosis was left ventricular hypertrophy, and there was an electrocardiographic pattern of LBBB in the limb leads and delayed intrinsoid deflections over both the right and left chest leads. Autopsy findings revealed hypertensive and arteriosclerotic heart disease with massive left ventricular hypertrophy and marked right ventricular hypertrophy, marked fibrosis with small scars of the entire left ventricle and anterior wall of the right ventricle, with less marked involvement of the inferior wall of the right ventricle, elastosis of the atrioventricular bundle at its bifurcation, marked fibrosis of the RBB, and moderate fibrosis of the LBB.

Discussion

The electrocardiograms in our cases disclosed LBBB in the limb leads and RBBB in the precordial leads. Case 2, which showed LBBB in the extremity leads and delayed intrinsoid deflections over the right and left
precordial regions, points up the value of exploring the left precordial area in cases of this type in search for left ventricular potentials. In these cases the usual chest leads may

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**Fig. 7.** Case 2. Bundle branches, showing marked fibrosis of RBB, and zone of marked fibrosis of LBB, in addition to acute degenerative changes of RBB. A, RBB, intramyocardial portion, proximal. Hematoxylin-eosin stain. × 170. B, RBB, intramyocardial portion, distal. Weigert-van Gieson stain. × 170. C, LBB in the upper portion. Hematoxylin-eosin stain. × 51. D, LBB. Hematoxylin-eosin stain. × 170. Arrows point to the respective bundles.
be oriented to an infarcted portion of the left ventricle and septum, and could reflect transeptal potentials originating in the right ventricular cavity, thereby failing to reflect the depolarization process in the left ventricle.

The pathologic lesions that were found to be common to our cases are (table 1) (1) incomplete, widespread damage to both the right and left bundle branches, (2) marked damage to the septal myocardium and the free walls of the left ventricle, and (3) marked hypertrophy of both ventricles. In an attempt to correlate the electrocardiographic findings of "masquerading" BBB with the pathologic findings, we must consider first those electrocardiographic changes that have been found to be associated with bilateral bundle-branch lesions at autopsy.11-22 These are LBBB alone, RBBB alone, bilateral partial BBB, "unstable" BBB, and complete heart block. In LBBB, the RBB is normal or shows fewer changes than the opposite branch. In RBBB, the LBB is normal or presents fewer changes than the opposite branch. In bilateral and "unstable" BBB, the lesions are severe in both branches.

| SA node | Blood supply to SA node | Approaches to SA node | Right atrium | Left atrium | Approaches to AV node | AV node | Blood supply to AV node | AV bundle, penetrating | AV bundle, branching | Right bundle branch | Left bundle branch | Left ventricle, anterior wall | Left ventricle, posterior wall | Right ventricle, anterior wall | Right ventricle, posterior wall | Atrial septum | Ventricular septum, anterior | Ventricular septum, posterior | Perforating arteries | Hypertrophy, left ventricle | Hypertrophy, right ventricle |
|---------|-------------------------|----------------------|-------------|------------|-----------------------|--------|------------------------|-----------------------|---------------------|---------------------|-----------------|---------------------|----------------------------|-----------------|---------------------|---------------------|-----------------|---------------------|---------------------|----------|
|         |                         |                      |             |            |                       |        |                        |                       |                    |                     |                 |                     |                             |                 |                      |                     |                |                     |                     | Case 1 |                  |                      |          |
|         |                         |                      |             |            |                       |        |                        |                       |                    |                     |                 |                     |                             |                 |                      |                     |                |                     |                     |         |                  |                      |          |
| Case 1  | ±                       |                      |             | ±          | ±                    | +2     | ±                      | ±                    | ±                   | ±                   | +3               | +3                 |                             |                 | ±                   | +3                   | +4          | +4                 | ±                   | ±               |
| Case 2  |                        |                      |             | ±          |                      | ±      |                        | ±                    | ±                   | ±                   |                  | ±                  |                             | ±               | ±                   | ±                    | ±            | ±                  | ±                    | ±              |

In our cases, the lesions were considerable in both branches, and perhaps more severe in the right than in the left bundle branch. However, we believe that it is difficult to equate lesions in two so completely different anatomic structures as the left and right bundle branches. Therefore it is unsafe to speak of preponderance of change on either side unless the difference is very marked. Since this degree of difference was not present in our cases, we can only say that we have considerable, but incomplete, lesions in both bundle branches. Hence, from the point of view of lesions in the conduction system, our cases fall more into the category of bilateral or "unstable" BBB, rather than in those of right or left BBB alone. However, our findings do not rule out the latter 2 possibilities from the physiologic or electric point of view.

We are, of course, cognizant of the fact that factors other than changes in the conduction system may be related to the electrocardiographic patterns in BBB.23-28 Thus, the findings of hypertrophy of both ventricles, and the marked pathologic change in the septum in our cases, may prove to be of significance. It is to be noted that our study does not include examination of the nerves of the conduction system and of the heart, which may have a bearing on this problem.22 Thus, a definitive statement as to the pathologic basis of this pattern must await completion of further studies of the anatomic base of right and left BBB, and of right and left ventricular hypertrophy, and of alterations in the conduction system without electrocardiographic evidence of conduction disturbances.

It is of interest that both of our cases were associated with ischemic changes related to arteriolar narrowing only. BBB and bundle-branch lesions associated with coronary arteriosclerotic heart disease have previously been described by Evans and Turnbull,23 and merit re-emphasis.

Hence we subscribe to the thesis that "masquerading" BBB is a type of bilateral BBB. Recently, Rosenbaum and Lepeschkin24 have dealt with the problem of bilateral BBB from the clinical, electrocardiographic, and
THE CONCEPT OF "MASQUERADING" BUNDLE-BRANCH BLOCK

physiologic points of view. They stated that a definite diagnosis of bilateral BBB can be made from electrocardiograms in which changes in the comparative degree of the block in the 2 branches allow the patterns of RBBB and LBBB to appear alternately, intermittently, or simultaneously in the same patient. They reiterate the contention that true partial bilateral BBB must prolong the atrioventricular conduction time. The intrinsicoid deflection is delayed only over the ventricle corresponding to the branch showing the greater degree of block and is within normal limits over the opposite chamber. The form of the QRS in bilateral BBB is determined by the branch that shows the greater degree of block, with the delayed conduction in the other branch not affecting the shape of the QRS, but acting by prolonging atrioventricular conduction. Where prolonged atrioventricular conduction does not occur, they believe the bundle branch on one side is associated with intraparietal block of the contralateral ventricle. Accordingly, they would interpret case 2 as unilateral BBB with contralateral intraparietal block, while case 1, in which atrial fibrillation was present, and the time of atrioventricular conduction hence unknown, they would interpret as bilateral partial BBB. Despite the absence of disturbances of atrioventricular conduction, we prefer our interpretation of partial bilateral BBB, because of the considerable pathologic changes in both bundle branches. However, here again further work must be done on the anatomic substrate of electrocardiographic disturbances to resolve this point.

The concept of Richman and Wolff\(^1\) that LBBB alone exists in these cases is not ruled out by our pathologic findings as detailed above, but it is difficult to reconcile with the marked changes in the RBB. The explanation of Sodi-Pallares and Rodríguez that the electrocardiographic pattern is related to infarction involving the lower portion of the interventricular septum and the adjacent free walls of both right and left ventricles, and dominantly affecting the right ventricular portion of the septum, is not completely borne out by our pathologic findings. As pointed out above, the free walls of the right ventricle were significantly affected in only 1 of our cases.

Since neither of our cases showed complete destruction of either the right or left bundle branches, it would appear, from the standpoint of anatomic continuity, that both bundle branches could still function. This, however, does not presume to indicate the physiologic state or degree of responsiveness of the bundles. Unfortunately, we did not obtain vectorcardiograms on these patients, nor simultaneous chest leads, so that we cannot decisively indicate the direction of the initial electric forces and the dominant path followed by the electric impulse in the myocardium.

Several theoretic possibilities should be considered. Bilateral partial delay in both branches can produce the following electrocardiographic effects: 1. The pre-existing P-R interval can be lengthened without showing any evidence of RBBB or LBBB if the degree of delay in both branches is not too divergent. In this situation the bilateral partial BBB will be manifested by the prolonged atrioventricular conduction time, and the presence and extent of ventricular myocardial damage might be otherwise obscured. 2. If the block in the RBB is "complete" or almost complete, the electrocardiogram may show the pattern of RBBB, the LBBB manifesting itself in terms of lengthening of the pre-existing P-R interval. It is to be noted that in case 2, with a P-R of 0.20 second, there were considerable lesions of both bundle branches. It is also to be noted that in this case the P-R interval showed an insignificant increase from 0.19 to 0.20 second over the course of 4 years, whereas the serial electrocardiograms showed progressive development of LBBB in the limb leads and delayed intrinsicoid deflections in both right and left precordial leads. 3. A pattern of complete LBBB might occur when only partial block exists, due to the greater delay in activation of one ventricle while there is significantly less delay in the conducting system of the other ventricle. 4. Mahaim's paraspecific fibers\(^13\,30-32\) arising from the atrio-
ventricular bundle and the LBB, might be functional or become so as a result of irritative disease processes. If these fibers would permit the wave of activation to bypass the bundle branches, then the electrocardiogram or the vectorcardiogram could not specifically determine the physiologic responsiveness of the right and left bundle branches and the route of activation. 5. The concept accepted by Rosenbaum and Lepeschkin\textsuperscript{29} that RBBB plus focal or parietal block in the left ventricle can produce an electrocardiographic pattern indistinguishable from partial bilateral BBB may pertain in some cases.

It can be seen from the foregoing that the determination of the predominant pathway of activation in the cases considered in this report cannot be derived from the anatomic findings in our present state of knowledge. What requires reemphasis, is that this type of electrocardiographic complex signifies extensive myocardial damage, and with it bilateral involvement of the bundle branches. This study serves to point out the need for complete examination of all parts of the conducting system and the myocardium in order to establish a firm anatomic base for clinical electrocardiography.

**Summary**

1. Two cases having the features of "masquerading" bundle-branch block were subjected to careful histologic study of the entire heart, including the conduction system. In each instance, bilateral bundle-branch lesions of considerable intensity, which did not completely disrupt the continuity of the branches, were demonstrated.

2. Each case revealed extensive destruction of the interventricular septum, the free walls of the left ventricle, and marked bilateral ventricular hypertrophy.

3. In both of our cases the vascular changes in the heart were those of diffuse arteriosclerosis.

4. It is suggested that this electrocardiograph complex is the result of partial bilateral bundle-branch block.

5. It is further suggested that the concept of "masquerading" bundle-branch block be discarded.

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**Summario in Interlingua**

1. Duo casos con le aspectos de "mascarada" de bloco de branca esseva subjicite a un meticolose studio histologic del corde integre, inclusive le sistema de conductio. Esseva demonstrate in ambe casos lesiones bilateral de branca de fasce que habeva atingite un intensitate considerabile sed que non disrumpeva le continuitate del branca completesmente.

2. Ambe casos revelava extense grados de destruction del septo interventricular e del pariete libere del ventriculo sinistre e marcate grados de bilateral hypertrophia ventricular.

3. In ambe nostre casos le alterationes vascular in le corde esseva le alterationes de diffuse arterioloosclerosis.

4. Es suggerite que iste complexo electrocardiographic es le risultato del bilateral bloco de branca.

5. Es etiam suggerite que le concepto del "mascarada" de bloco de branca sia abolite.

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