QRS-T Patterns in Multiple Precordial Leads That May Be Mistaken for Myocardial Infarction

III. Bundle Branch Block

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The electrocardiograms of patients in whom myocardial infarction was excluded at autopsy are presented to bring out (1) the following features of left bundle branch block suggesting anteroseptal infarction—QS or qS patterns in leads from the right precordium or localized to intermediate leads and/or cove plane inversion of the T waves near the transitional zone; (2) the following features of right bundle branch block suggesting infarction—pseudo Q waves, elevated RS-T segments and inverted T waves in right precordial leads, prominent Q waves in leads from the left precordium.

I T IS WELL known that bundle branch block may accompany myocardial infarction or may occur independently. The diagnosis of associated myocardial infarction and bundle branch block has been considered, but little attention has been given to the electrocardiographic features of uncomplicated bundle branch block that may be mistaken for those of myocardial infarction. These features will be brought out through the presentation of four cases of left bundle branch block and seven cases of right bundle branch block, in which myocardial infarction was diagnosed or at least considered from the electrocardiographic findings during life, but was subsequently excluded at autopsy. The method of electrocardiographic and pathologic study is similar to that described in a preceding article.

LEFT BUNDLE BRANCH BLOCK

A diagnosis of left bundle branch block is based upon the following criteria: (1) a QRS interval of 0.12 second or longer in complexes derived from impulses of sinoauricular origin and associated with a P-R interval of at least 0.12 second*; (2) an initial upstroke in all leads facing the epicardial surface of the left ventricle, due to early positivity of the left ventricular cavity; (3) a broad, slurred, or notched R wave with abnormally delayed intrinscoid deflection in the same leads, indicating late arrival of the impulse at the epicardial surface of the left ventricle. The QRS complexes in left ventricular leads are characterized by: depression of the RS-T junction, upward convexity of the RS-T segment, and sharp inversion of the T wave.

The presence of the foregoing QRS-T pattern in V4, V5 and V6, in V1 and V2, or even in V6 alone should lead to a diagnosis of left bundle branch and should not be mistakenly interpreted as due to myocardial infarction. In fact, the diagnostic error likely to be made in the interpretation of left ventricular leads in the presence of left bundle branch block consists in failure to detect coexistent anterolateral infarction. The initial R wave produced in left ventricular leads by septal activation from impulses distributed through the right Purkinje network persists when left bundle branch block is complicated by anterolateral infarction and thus prevents the registration of abnormal Q patterns.

In the presence of left bundle branch block, the impulse arrives early in the right side of the septum and free wall of the right ventricle and late in the free wall of the left ventricle. The late activation of the outer wall of the left ventricle...
ventricle is always manifested by a deep, broad, slurred S wave in leads facing the epicardial surface of the right ventricle and atrium. The initial phase of the QRS complex in these leads depends upon the interplay of negative potentials, transmitted from the endocardial surface of the right side of the septum, and positive potentials produced by activation of the free wall of the right ventricle and perhaps positive potentials developing in the left ventricular cavity and transmitted via the left and right atria to the right precordium. As a consequence, the QRS pattern in leads over the right atrium and ventricle in patients with uncomplicated left bundle branch block may take one of three forms: (1) an rS complex, consisting of a small, narrow initial r wave due to early preponderance of positive potentials coming from activation of the free wall of the right ventricle and a deep, broad, slurred S wave due to activation of the free wall of the left ventricle; (2) a triphasic qR complex; the q wave representing initial negativity of the right ventricular cavity from septal activation, the r wave reflecting passage of the impulse through the free wall of the right ventricle; (3) a deep, broad, slurred QS deflection. The r wave may be submerged and obscured in this monophasic downstroke because of complete dominance of negative potentials transmitted from the endocardial surface of the right side of the septum over positive potentials coming from activation of the free wall of the right ventricle, or the r wave may be represented by an equivalent in the form of notching or coarse slurring near the onset of the QS deflection.

Both the QS and the qR S patterns, which may be recorded in leads over the right atrium and ventricle in uncomplicated left bundle branch block, resemble the findings obtained in left ventricular leads as a result of transmural infarction. Furthermore, these QS or qR S deflections are generally followed by abnormal elevation of the RS-T junction and a tall upright T wave. The RS-T segment may exhibit straightening or upward convexity instead of the usual upward concavity, especially when digitalis has been given. The combination of an abnormally elevated RS-T junction and a straightened or convex RS-T segment is strongly suggestive of the RS-T pattern found in the stage of injury associated with infarction of the outer wall of the left ventricle. In the presence of right ventricular dilatation, coupled with clockwise rotation of the heart, the potential variations of the epicardial surface of the right ventricle may be transmitted as far to the left as the anterior axillary line and occasionally even to the midaxillary line. The registration of a broad, slurred QS deflection and an abnormally elevated, convex, or straightened RS-T segment in Leads V, V, and V, which ordinarily reflect the potential variations of the left ventricle, may lead to an erroneous diagnosis of anterolateral infarction unless due consideration is given to the presence of left bundle branch block and the displacement of the transitional zone far to the left.

Another diagnostic error may arise from the fact that leads over the right atrium and ventricle in uncomplicated left bundle branch block do not necessarily show either the expected increase in the initial R wave as the electrode is moved towards the left or the uniform QS or rS pattern occurring as a recognized variant. A distinct rS deflection (or rarely a qR S complex) may be recorded in Lead V or V and a QS deflection in one or more leads farther to the left in the presence of uncomplicated left bundle branch block and may lead to an erroneous diagnosis of anterior infarction. The distinct R wave in Lead V and/or V may be correlated with an electrode position over the tricuspid orifice favorable to the reception of positive potentials coming from the relatively thick base of the right ventricle and perhaps from the left ventricular cavity via the left and right atria, whereas the decrease or disappearance of the R wave in V and V may be correlated with an electrode position over the relatively thin apex of the right ventricle and nearer the right side of the septum, which transmits opposing negative potentials.

An unusual respiratory variation that might raise the question of myocardial infarction was encountered in a case of left bundle branch block reported by Lapin and Sprague. This
was characterized by the registration of a broad, notched QS deflection in transitional lead V₆ during inspiration and a broad, notched R wave in the same lead during expiration. This was attributable to sufficient cardiac rotation in a clockwise direction during inspiration so that the electrode received chiefly the potential variations of the epicardial surface of the right ventricle and sufficient rotation in a counterclockwise direction during expiration so that the record was then dominated by the potential variations of the epicardial surface of the left ventricle. Another extraordinary respiratory variation was observed in Leads V₆ and aV_L of the same patient. This was characterized by the inspiratory registration of an abnormal Q wave followed by a broad, notched R wave, strongly suggestive of the pattern associated with an anterolateral infarct of dense distribution in the subendocardial layer and patchy in the remainder of the wall. However, the inscription of a broad, notched, initial R wave of greater amplitude in the same leads during expiration indicated that the QR pattern was a transitional zonal phenomenon in a case of left bundle branch block. The initial Q wave was probably transmitted from the epicardial surface of the right ventricle and the subsequent R wave was undoubtedly transmitted from the epicardial surface of the left ventricle. This might have occurred as a result of an electrode position that straddled the intraventricular septum, but was more likely due to a shift in cardiac position during the inscription of the QRS as a result of contraction of the right ventricle ahead of the left.

Another pattern occasionally recorded at the transitional zone in left bundle branch block, but likely to be mistaken for that due to myocardial infarction, consists in a QS deflection, accompanied either by elevation of the RS-T junction and inversion of the terminal portion of the T wave or by an isoelectric RS-T segment and deeply inverted T wave. The QS deflection and the elevated RS-T segment resemble those in leads farther to the right and represent predominant transmission of the potential variations of the epicardial surface of the right ventricle during the first part of the cycle. The sharp inversion of the terminal portion of the T or the entire wave resembles that in leads farther to the left and represents predominant transmission of the potential variations of the epicardial surface of the left ventricle during the last part of the cycle. Such a change may be observed as a result of counterclockwise rotation of the heart during left ventricular con-

Figure 1.—Serial electrocardiograms in Case 28.
traction which occurs between the inscription of the QRS and T waves.

A case similar to that of Lapin and Sprague has not as yet been encountered in our autopsy material. The other features of uncomplicated left bundle branch block that may be mistaken for those of myocardial infarction are illustrated in figs. 1, 2, and 3, representing the electrocardiograms in Cases 28 through 31, inclusive.

**Broad Slurred QS Deflections in the First Four, Five, or Six Precordial Leads**

This may occur in association with left bundle branch block as a result of an associated
The inadequacy of the six customary precordial leads for diagnostic purposes was indicated by the lack of contrasting QRS-T patterns in V₁ and V₄ and led to the registration of V₇ on both occasions. A broad, slurred R wave of 0.12 second’s duration was recorded in V₇ and was incorrectly interpreted as evidence of delayed conduction through the uninfarcted posterolateral wall of the left ventricle. Upon restudy of these two tracings, it is evident that the findings in V₇ and aV₁ were compatible with those recorded over the left ventricle in left bundle branch block and the findings in V₁ through V₆ were in keeping with those recorded over a markedly dilated right ventricle in the absence of infarction. Since the P wave was inverted in V₁ and upright in V₆, the electrode in the latter position was apparently in the vicinity of the tricuspid orifice. The initial r in V₁ could have been derived from activation of a relatively thick ring of muscle at the tricuspid orifice, whereas the initial downstroke in the next four leads could be explained by preponderance of negative potentials transmitted from the right side of the septum over weak positive potentials coming from activation of a dilated, thin-walled apex of the right ventricle. The lower voltage in V₆ than in V₁ or V₇ should have indicated that V₆ was a transitional lead. The deep Q wave in V₆, therefore, could have represented negative potentials transmitted from the epicardial surface of the right ventricle, whereas the late R wave on February 16 could have been derived from activation of the adjoining left ventricle. The QS deflection in aV₁ could have been a manifestation of horizontal position in which the potential variations of the epicardial surface of the right ventricle are transmitted to the left leg. Thus, all findings on February 12 and February 16 could be explained by a diagnosis of left bundle branch block with marked right ventricular dilatation, but without infarction.

On March 16, a broad, slurred R wave was inscribed in V₁ similar to that previously recorded in V₇. This change was in keeping with reduction in cardiac size noted on physical and roentgen-ray examination, permitting reference of left ventricular potentials to midaxilla. The initial r wave had disappeared from V₂ and a QS deflection, elevated RS-T junction, and tall, erect T wave were recorded in the first four precordial leads. Lead V₅ displayed a triphasic qRS complex, consisting of a 1-mm. q, 2-mm. r, and 8-mm. S wave, compatible with the pattern sometimes found in left ventricular leads in the presence of patchy transmural infarction or in right ventricular leads in the presence of septal infarction. However, the presence of a broad, slurred R wave in V₄ and aV₁, indicative of left bundle branch block, should have suggested that the pattern in V₅ represented a variant occasionally found in right ventricular or transitional leads. The q wave could have represented negative potentials transmitted from the right side of the septum, the slurred r wave could have been derived from activation of
the adjacent anteroseptal walls of the right and left ventricles, the deep s wave from activation of the lateral and posterior walls of the left ventricle. Hence the findings in the first five precordial leads on March 16 could be explained by right ventricular dilatation in the presence of left bundle branch block, but without infarction.

The patient had further attacks of sudden weakness and dyspnea on April 7 and April 11, the latter ending fatally. Death was due to pulmonary embolism and autopsy revealed multiple, old, organized pulmonary infarcts, which were apparently responsible for the attacks of weakness and dyspnea leading to admission to the hospital. At autopsy the heart weighed 530 grams, which represented marked hypertrophy in view of a body weight of only 40 kilograms. The increased cardiac weight was due chiefly to left ventricular hypertrophy, believed secondary to hypertension because of the presence of advanced renal arteriolosclerosis. There was marked dilatation of the right ventricle, sufficient to explain the displacement of the transitional zone to the left. The coronary vessels were of normal caliber and no evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks. There was patchy fibrosis, which may have been responsible for the conduction defect. In view of the pathologic findings, the abnormalities in the electrocardiogram were evidently the result of left bundle branch block complicated by marked dilatation of the right ventricle.

**Progressive Diminution of the Initial R Wave, Then Replacement by a qRS Complex as the Electrode is Moved Towards the Left**

These findings arouse the suspicion of myocardial infarction, but may occur in association with uncomplicated left bundle branch block, as exemplified by Case 29.

**Case 29.**—The patient, a man, age 73 years, showed evidence of congestive failure secondary to hypertension and uremia associated with prostatic obstruction.

The electrocardiogram reproduced in Fig. 2A was obtained after digitalization and approximately twelve hours before death. A diagnosis of left bundle branch block was easily established by the QRS interval of 0.14 second together with the broad, notched initial R wave in Leads V5 and V6. The extreme depression of the RS-T junction in these leads and the abrupt return of the string to the isoelectric line to complete the T wave were extraordinary and raised the question of subendocardial ischemia. However, the short Q-T interval and the contour of the ascending limb of the T wave indicated that overdigitalization was largely, if not entirely, responsible.

The first three precordial leads displayed an rS deflection and Lead V1 showed a qRS complex. The suspicion of myocardial infarction was aroused by: (1) the decrease in the initial r wave from 2.0 mm. in V1 to 1.0 mm. in V2 and to 0.5 mm. in V3; (2) the replacement of the initial r by a 2.5-mm. q wave in V1; (3) the marked elevation and domelike contour of the RS-T segments in the first three precordial leads. The deep, broad S wave in V1, V2, and V3 indicated that the electrode was over the right side of the heart, and the marked upward displacement and rounding of the RS-T segments in these leads could have been produced by digitalis effects superimposed upon those of uncomplicated left bundle branch block. The relatively large initial R wave in V1 could be accounted for by placement of the electrode over the right atrium in a position to receive positive potentials produced by activation of the relatively thick base of the right ventricle. The progressive reduction in amplitude of the initial r wave in the first three precordial leads could be explained by shift of the electrode from the vicinity of the tricuspid ring to points over the thin apex of the right ventricle, where it was in a position to receive relatively weak positive potentials coming from activation of the free wall of the right ventricle and simultaneous negative potentials transmitted from the endocardial surface of the right side of the septum. The qRS pattern in V1 was in keeping with an electrode position that straddled the septum. The small q could have represented normal initial negativity transmitted from the subjacent endocardial surface of the right side of the septum; the timing and amplitude of the succeeding r wave indicated transmission from the adjoining anteroseptal wall of the left ventricle and the final S wave was derived from activation of lateral and posterior walls of the left ventricle. Thus, all atypical features of this electrocardiogram could be explained without postulating complicating infarction.

At autopsy the heart weighed 700 grams and showed marked left ventricular hypertrophy, but no evidence of myocardial infarction. The coronary vessels were of normal caliber and showed only mild sclerosis.

**An rS Deflection in Lead V1 and/or V2 and a QS in Leads Farther to the Left**

This may occur in association with uncomplicated left bundle branch block and may lead to an erroneous diagnosis of myocardial infarction. This finding was encountered in the first two tracings of Case 28, discussed above, and will be exemplified further by Case 30.

**Case 30.**—The patient was a man, age 66 years, who had been incapacitated because of congestive failure for five months, in spite of the administration of digitoxin and diuretics by his family physician. He was admitted to the hospital with congestive failure complicated by shock associated with unobtainable blood pressure.
The routine electrocardiogram reproduced in figure 2B was obtained on the first hospital day and the additional high precordial leads were taken three days later. Digitoxin was continued in doses of 0.2 mg. daily throughout this period. The P-R interval was lengthened to 0.24 second and the QRS interval to 0.16 second. The presence of left bundle branch block was indicated by the broad, slurred initial R waves in left ventricular leads V₄ and aVL. An upright T wave was recorded in V₄, instead of the inverted T wave usually found in left ventricular leads in the presence of left bundle branch block. Concordant T waves represent an occasional finding in uncomplicated bundle branch block and do not, in themselves, constitute evidence of myocardial infarction.

The atypical features of the tracing that raised the question of infarction consisted in the registration of a distinct initial r wave in V₁ and a QS complex with abnormally elevated, convex RS-T segments in V₂ and V₃. The diphasic P wave in V₁ indicated that the electrode was over the right atrium, whereas the upright P waves in V₂ and V₃ suggested that those positions were over the right ventricle. The initial R wave in V₁ was presumably derived from activation of the relatively thick ring of muscle at the tricuspid orifice. The disappearance of the R wave in V₂ and V₃ as the electrode approached the septum could be explained by preponderance of negative potentials transmitted from the right side of the septum over weaker positive potentials produced by activation of the thinner apical portion of the right ventricle.

High precordial leads were taken at the intersections of a horizontal line through the junction of third intercostal space and sternum with vertical lines through the V₃, V₄, V₅, and V₆ positions. The records taken high in the anterior axillary and mid-axillary lines resembled those taken in the customary V₃, V₄, and V₅ positions, respectively. The electrode in both the high and the standard V₃ and V₄ positions was apparently to the right of the septum, as judged by the relatively deep and broad S waves. The tracing in the high V₃ position displayed a triphasic qrS complex, consisting of a q wave 1.5 mm. deep, an r wave that barely crossed the isoelectric line and an S wave 11 mm. deep, followed by an abnormally elevated, dome-shaped RS-T segment. The contour of both the qrS and the RS-T complexes in high V₃ was strongly suggestive of infarction. However, in view of the left bundle branch block a qrS of this type could be recorded in a right ventricular lead in the absence of infarction. The q wave reflected initial negativity of the right ventricular cavity, as a result of reversal in the vector associated with septal activation; the r wave was produced by passage of the impulse through the free wall of the right ventricle. The elevated domelike RS-T segment was the reciprocal of the depressed V-shaped segment in left ventricular Lead aVL and was attributable to digitalis effect. Hence the findings in both the customary and high precordial leads could be explained by uncomplicated left bundle branch block.

The patient died in congestive failure on the fifth hospital day. At autopsy the heart weighed 607 grams and showed marked left ventricular hypertrophy of hypertensive origin, and secondary right ventricular dilatation. The coronary vessels were of normal caliber and revealed only mild atherosclerosis. No evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks.

**Triphasic qrS Pattern in One or More Precordial Leads Just to the Right of the Transitional Zone**

A triphasic qrS complex, consisting of small, narrow q and r deflections and a deep, broad S wave, is occasionally recorded in right ventricular leads in association with uncomplicated left bundle branch block, as illustrated by Lead V₃ of the last tracing in Case 28 and by high Leads V₃ and V₄ of Case 30. The origin and significance of this pattern have been discussed above and also in connection with the report of Case 99 of a previous manuscript.

**QS or W-Shaped Complexes Together With Cove-Plane Inversion of the T Waves in Intermediate Leads Near the Transitional Zone**

Findings of this type in Leads V₃ and V₄ of Case 31, figure 3, led to an electrocardiographic diagnosis of anteroseptal infarction that could not be substantiated at autopsy.

**Case 31.—The patient, a housewife, age 52 years, was known to have had diabetes and hypertension for one year. On June 3, 1944, she was awakened from a sound sleep by a severe choking sensation with accompanying orthopnea, which lasted for approximately one hour. Paroxysmal dyspnea recurred on the two succeeding nights and was not accompanied by pain. She was admitted to the hospital on June 6 with marked pulmonary edema and slight peripheral edema, and received a total of 1.0 Gm. digitalis leaf up until June 13, at which time the drug was discontinued. Compensation was rapidly restored and the hospital course was uneventful. There was no fever, leukocytosis, or elevation in sedimentation rate.**

The electrocardiograms reproduced in figure 3 were selected from a series taken over a period of six months. The QRS-T patterns in five electrocardiograms taken after August 1 corresponded closely and consequently only the final tracing, on December 6, is included in the illustration. Left
bundle branch block was present throughout, as indicated by (1) a QRS duration of 0.12 second or more, (2) a broad, slurred initial R wave in Leads V2 and V4. A deep, broad QS deflection, elevated RS-T segment, and tall upright T wave were consistently recorded in the first two precordial leads. A similar QS deflection was also present in Lead V1 and was accompanied by an elevated domelike RS-T segment and sharply inverted T wave in June, and by a normal RS-T complex after the first of August. The tracings taken at position V4 in June revealed (1) a triphasic QRS complex, consisting of a deep Q wave, a small late r wave, which sometimes failed to cross the isoelectric line, and a relatively small S wave; (2) an isoelectric RS-T segment and sharply inverted T wave. In later tracings, Lead V4 displayed either a minute r and deep broad S wave or a QS deflection like that at V2, accompanied by an upright T wave. On the basis of the QRS-T pattern in Leads V3 and V4 in June, a diagnosis of recent anterior septal infarction was made. The change in RS-T complexes during the ten-day interval between the first two tracings was not as great as would be expected in the presence of recent infarction, but the subsequent change in QRS pattern in V4, along with the evolution to a normal upright T wave in V2 and V4, tended to overcome this objection and to support the diagnosis of infarction.

The patient remained under close medical supervision. Digitalization was carried out in July and was maintained for the rest of her life. She had no further attacks of paroxysmal dyspnea and no chest pain. She was readmitted to the hospital on Oct. 23, 1944, with increasing nervousness, heat intolerance, and weight loss of one month's duration. The basal metabolism rate ranged from +38 to +41 and thioracil was instituted. A satisfactory clinical response was obtained until Jan. 17, 1945, when the white blood count dropped to 3900 per cu. mm. of blood. Thioracil was stopped, but the white count continued to fall and the patient died of agranulocytosis ten days later.

At autopsy the heart weighed 347 grams and showed slight left ventricular hypertrophy. The coronary tree injected well and the vessels were of normal caliber and showed only minimal atherosclerosis. Meticulous gross examination revealed no evidence of myocardial infarction. There was a small patch of healed epicarditis at the left apex, occupying an area 2.5 cm. in diameter. This lesion was well healed and did not extend into the myocardium either on gross or microscopic examination. Many microscopic blocks were studied, but no evidence of infarction was found. The possibility that anterior septal infarction had occurred in June 1944 and had healed completely except for an epicardial scar could not be ruled out, but was deemed unlikely because of the normal histologic appearance of the myocardium.

Upon restudy of the electrocardiograms, it would appear that the abnormalities in V3 and V4, which had been interpreted as evidence of anteroseptal infarction, could have been the result of admixture of right and left ventricular potentials at the transitional zone. The deep QS deflections, elevated RS-T segments, and upright T waves in V3 and V4 were representative of the pattern sometimes recorded over the normal right ventricle in the presence of left bundle branch block. The resemblance of the QRS deflection and elevated RS-T segment in V4 to those in V1 and V2 suggested that the electrode lay to the right of the septum during the inscription of this portion of the ventricular complex, whereas the resemblance of the inverted terminus of the T wave to that in leads farther to the left suggested that the heart may have rotated sufficiently during the intervening mechanical systole to bring the left ventricle beneath the electrode.

The QRS pattern in V4 could also be explained as a manifestation of the transitional zone associated with an electrode position straddling the septum. The Q wave corresponded to the first portion of the QS in the first three precordial leads and was probably transmitted through the anteroseptal wall of the right ventricle. The R wave corresponded to the expected time of activation of the anteroseptal wall of the left ventricle and was probably derived in this manner. The T wave simulated those in left ventricular Leads V3 and V4. In left bundle branch block, contraction of the right ventricle may begin before completion of activation of the left and might cause cardiac rotation during inscription of the QRS complex. This may constitute the mechanism for the predominant transmission of the first portion of the QRS from the right ventricle and the last portion from the left ventricle in a lead overlying the septum.

The QRS-T patterns in V4 and V5 could therefore have represented a transitional zonal effect in left bundle branch block uncomplicated by myocardial infarction, ischemia, or pericarditis. Whether or not the pericarditis found at autopsy developed in June and contributed to the T-wave inversion is a matter for conjecture. The fact that the T waves in V3 and V4 subsequently became upright does not necessarily indicate that either epicarditis or ischemia was responsible for the inversion. The change to upright T waves in V3 and V4 was believed secondary to the reversal in the T wave in left ventricular lead V5. Discordant T waves are occasionally replaced by concordant T waves for no discoverable reason in uncomplicated left bundle branch block.

**Right Bundle Branch Block**

Uncomplicated right bundle branch block is manifested by 1, 6, 9: (1) a QRS interval of 0.12 second or longer in complexes derived from an impulse of sinoauricular origin and associated
with a P-R interval of at least 0.12 second; (2) an initial upstroke in all leads facing the epicardial surface of the right ventricle, due to early positivity of the right ventricular cavity, due to (3) a late secondary upstroke in the same leads, due to delayed activation of the free wall of the right ventricle. The intervening space between these two R waves may be taken up by coarse slurring or by a downstroke that may stop above the isoelectric line to form a notch or carry below to form an S wave. This downstroke reflects negative potentials transmitted to the right precordium during the brief interim between activation of the septum and the free wall of the right ventricle. The QRS complexes in right ventricular leads are characteristically followed by: depression of the RS-T junction, upward convexity of the RS-T segment, and sharp inversion of the T wave.

Right bundle branch block secondary to septal infarction is distinguished from the uncomplicated variety by the direction of the initial phase of the QRS complex in leads from the right precordium. An abnormal Q wave is characteristically found in these leads in the presence of septal infarction, whereas an initial upstroke should be detectable in uncomplicated right bundle branch block. When recent septal infarction is responsible for the conduction defect, right precordial leads generally show elevation of the RS-T junction and cove inversion of the T wave, in contrast to the RS-T depression usually associated with uncomplicated right bundle branch block.

The following variants may occur in the pattern of uncomplicated right bundle branch block and may lead to an erroneous diagnosis of septal infarction: (1) pseudo Q waves in leads from the right precordium and/or transitional zone; (2) elevation of the RS-T junction and abnormal inversion of the T wave in leads from the right precordium; (3) reduction in the R wave and deep inversion of the T wave in intermediate leads; (4) concordant T waves; (5) prominent Q waves in leads from the left precordium. These features are collectively illustrated by the electrocardiograms in figure 4, obtained in Cases 32 to 38, inclusive.

**Pseudo Q Waves in Leads from the Right Precordium and/or Transitional Zone**

The initial R wave transmitted to the right precordium as the result of septal activation in association with uncomplicated right bundle branch block progressively diminishes with increasing distance from the septum and may be barely detectable in leads over or beyond the right atrium. If a minute initial R wave is overlooked, the succeeding downstroke may be misinterpreted as a Q wave and an erroneous diagnosis of septal infarction may be made. The problem is exemplified by Cases 32 and 33. The electrocardiograms are reproduced in figures 4A and B.

**Case 32.**—The patient, a man, age 51 years, had had recurrent asthmatic bronchitis over a period of four years and was admitted to the hospital with left-sided bronchopneumonia and pleurisy. The electrocardiogram reproduced in figure 4A was obtained during the febrile period, after partial digitalization. At first glance, there appeared to be an initial downstroke and late R wave in V1 and V2 and a deep, notched QS complex in V3. If this had constituted a correct identification of the components of the QRS complex, a diagnosis of septal infarction would have been justifiable. However, close scrutiny revealed a small but distinct initial r wave, averaging 0.5 mm. in amplitude, in each of the three leads. This initial r wave was a manifestation of septal activation and the succeeding downstroke was therefore an S wave, commonly found in association with uncomplicated right bundle branch block.

In searching for an explanation for the exceptionally small initial r wave recorded in right precordial leads V1 and V2, an unusually high position of the electrode was suggested by the following observations: (1) emphysematous depression of the diaphragms, which tended to lower the heart in reference to fixed points on the thoracic cage; (2) inverted P waves in the presence of sinus rhythm, recorded not only in right ventricular leads V1 and V2, but also in transitional lead V3 and in left ventricular lead V5. The configuration of the P waves suggested that the electrode lay over the atria and was therefore in a position to receive potential variations of the two ventricular cavities. Simultaneous transmission of negative potentials through the mitral orifice to supraventricular positions V1, V2, and V3 nearly succeeded in neutralizing positive potentials coming from the right ventricular cavity, thereby accounting for the unusually small initial r wave.

A noteworthy feature of the last four precordial leads was the 1- to 3-mm. elevation of the RS-T
junctions and the straightening of the RS-T segments. Although digitalis tends to straighten the RS-T segments and displace them in a direction opposite to the main deflection of the QRS complex, it was not responsible for the RS-T abnormalities in this case because of: (1) absence of Q-T shortening; (2) presence of a similar degree of RS-T segment elevation in a tracing made one week previously, before the administration of glycosides. The contour of the elevated RS-T segments in $V_4$, $V_5$, and $V_6$ was strongly suggestive of a lesion of the subepicardial layer of myocardium and aroused the suspicion of infarction and pericarditis. The possibility of transmural infarction could be virtually excluded by the absence of Q waves, but the question remained as to whether the subepicardial lesion was the result of superficial infarction or pericarditis.

Death occurred five days after the electrocardiogram was made. At autopsy the heart weighed 387 grams and showed moderate right ventricular hyper-
tropho secondary to chronic obstructive pulmonary emphysema. There was no evidence of coronary narrowing or myocardial infarction. No lesion other than right ventricular hypertrophy was found to account for the right bundle branch block, but the possibility of microscopic degeneration or fibrosis in the right branch of the bundle of His was not excluded, because of failure to take serial microscopic sections in this area. A subacute epicarditis was found over the anterolateral aspect of the left ventricle and extended sufficiently into the subepicardial layer of myocardium to explain the RS-T elevation in V₆, V₇, and V₈.

**Elevation of the RS-T Junction and/or Abnormal Inversion of the T Wave in Leads from the Right Precordium**

RS-T patterns of this type in association with right bundle branch block are strongly suggestive of septal infarction, but may occur independently, as exemplified by Cases 33 and 34. Electrocardiograms in these cases are reproduced in figs. 4B and C.

**Case 33.**—The patient, a man, 74 years of age, suffered acute left ventricular failure following a ureterosigmoid transplant necessitated by carcinoma of the bladder. The electrocardiogram reproduced in figure 4B was obtained after the intravenous administration of 0.8 mg. Cedilanid. The rhythm was regular throughout, but the rate increased from 130 during the inscription of the limb leads to 150 during the registration of the precordial leads. This observation, together with the widened QRS complexes in the first four precordial leads and the apparent normal duration of the QRS in the remaining leads, aroused the suspicion of a transient episode of ventricular tachycardia, beginning between the registration of Leads aVF and V₆, and terminating between the inscription of Leads V₃ and V₆. From a more careful study of the tracing, the following evidence was elicited, indicating that a sinus tachycardia was present throughout: (1) distinct upright P waves were found in V₁ and V₃, occupying a constant position in reference to the T waves and preceding the QRS complex by the same P-R interval as in the limb leads; (2) the cardiac rate and Q-T interval were the same in V₁ and V₆, where the QRS complexes were widened, as in V₃ and V₆, where the QRS complexes appeared to be of normal duration. The discrepancy in apparent QRS interval was thus due to the fact that the last portion of the QRS was isoelectric or nearly isoelectric in V₃, V₆, and the limb leads. Measurements with the dividers indicated that the depressed segment following the R wave in Leads V₆, aVF, and I and the elevated segment following the downstroke in Leads aVR, aVF, and III were part of the QRS complex, and that the RS-T segment in these leads was isoelectric and fused with the QRS complex.

From the general resemblance of the QRS complexes in the first four precordial leads it was concluded that they represented predominantly the potential variations of the right side of the septum and epicardial surface of the right ventricle. The broad R wave and late intrinsicoid deflection in these leads, together with the broad S wave in left ventricular leads V₆ and aV₆, indicated that the sinus tachycardia was complicated by right bundle branch block.

Unless careful inspection were made of the QRS complexes in the first three precordial leads, the downstroke might be misinterpreted as a Q wave and mistaken as evidence of myocardial infarction. However, close scrutiny revealed a small but distinct initial R wave in each lead. Still more confusing, however, was the apparent RS-T displacement in V₁ and V₃. It might seem, from first glance at V₁, that the RS-T junction was located at the peak of the upstroke, 6 mm. above the isoelectric line. In that event, the precipitous descent to an inverted T wave would be strongly suggestive of recent septal infarction. However, the contour of the tracing in this precipitous descent of the string was more in keeping with a portion of the QRS rather than a portion of the RS-T complex. This led to an attempt to ascertain the termination of the QRS complex in V₁ by interpolation of measurements of the QRS interval made in V₆, where the beginning and end of the complex were quite distinct. On the assumption of a similar QRS interval in V₁ and V₆, to that in V₃, the RS-T junction in both leads was located at or slightly below the isoelectric line. Thus, from careful study, it was concluded that both the QRS and RS-T patterns could be accounted for on a basis of uncomplicated right bundle branch block.

Death occurred sixteen hours after the electrocardiogram was made. At autopsy the heart weighed 900 grams and showed a normal ventricular ratio. There was moderate coronary sclerosis, but no narrowing or occlusion. There was no evidence of myocardial infarction. The right bundle branch block was believed secondary to the acute left ventricular failure, but serial sections of the bundle of His were not made to exclude the possibility of a localized lesion.

Another type of RS-T pattern that might be mistaken for that of myocardial infarction is illustrated in figure 4C, which represents the electrocardiogram in Case 34.

**Case 34.**—The patient was an 18 year old girl with chronic glomerulonephritis. The electrocardiogram was obtained shortly before death, which was due to uremia. Blood potassium was not deter-
mined, but was probably elevated in view of marked oliguria and a creatinine level of 18.6 mg. per cent.

A diagnosis of right bundle branch block was readily established from the broad, double-peaked R wave in Leads V₁ and V₂. The possibility of recent septal infarction was suggested by the elevated RS-T junctions and sharply inverted T waves in V₁ and aV₆, and the possibility of continuation of the lesion into the subendocardial layer of the anterolateral wall of the left ventricle was suggested by the RS-T depression in Leads V₅, V₆, and V₇. The absence of Q waves militated against infarction and necessitated search for another explanation for the RS-T pattern. The narrow base and sharp peak of the T waves in V₂ and V₆ were strongly suggestive of hyperpotassemia and the exceptionally deep and sharply inverted T waves in the first three precordial leads could also be explained on the same basis.

At autopsy the heart weighed 350 grams. Left ventricular hypertrophy was present, but gross and microscopic examination revealed no other abnormality.

Reduction of the R Wave and Deep Inversion of the T Wave in Intermediate Leads

Patterns of this type may arouse the suspicion of anteroseptal infarction, but may occur as a transitional zonal phenomenon in association with uncomplicated right bundle branch block, as exemplified by Case 35.

Case 35.—The patient, a woman, 58 years of age, was admitted to the hospital with a typical history of hyperthyroidism of three years’ duration, complicated by congestive failure of three weeks’ duration. The electrocardiogram reproduced in figure 4D was obtained on the fourth hospital day after digitalization. Fine auricular fibrillation was present.

Lead V₅ displayed an rsR’ complex typical of right bundle branch block, followed by the expected RS-T depression and inversion of the T wave. A glance at V₁ would disclose a comparable late R wave and RS-T complex, but the minute initial r might escape detection and thus lead to the misinterpretation of the downstroke as a Q wave and an erroneous diagnosis of septal infarction. Careful scrutiny of a number of cycles revealed a small but distinct initial r wave, ranging from 0.25 to 0.5 mm. in amplitude. The smaller initial r wave in V₁ than in V₅ was presumably due to the fact that the electrode was farther from the right side of the septum, but closer to the right atrium, where it might be in a position to receive negative left ventricular cavity potentials transmitted through the left atrium.

A more confusing pattern was recorded in Lead V₃. At first glance, the QRS might be classed as a W-shaped complex and thus be mistaken as evidence of infarction. However, measurements of the ORS interval indicated that the preliminary oscillations were part of the complex, constituting an initial R, preceding a broad, notched S wave. The fact that the R wave in V₃ was much smaller than the R’ in V₂ and the R wave in V₄ could be ascribed to the transitional zone, but the fact that the inverted T wave in V₃ was significantly deeper and different in shape from that in V₂ and V₄ was not so readily explained. The possibility of anteroseptal infarction was originally entertained from the RS-T pattern in V₃, but was later discarded after subsequent tracings, taken from slightly different points, showed that the inverted T wave was also a localized phenomenon, referable to the transitional zone.

Death occurred three weeks later from intercurrent pneumonia. At autopsy the heart weighed 440 grams. Left ventricular hypertrophy was found and was attributed to thyrotoxicosis after exclusion of other recognized causes. The coronary arteries were of normal caliber and no evidence of myocardial infarction was found. The septum appeared normal on gross and routine microscopic examination, but serial sections in the vicinity of the bundle of His were not made.

Concordant T Waves

Since it is generally recognized that concordant T waves may occur in association with uncomplicated bundle branch block, Case 36 is included more because of the clinical than the electrocardiographic problem that was presented.

Case 36.—The patient, a hypertensive man 65 years of age, was suddenly seized with severe retrosternal constriction and dyspnea and was brought to the hospital in shock. The blood pressure was 60/40 in the left arm and the pulse rate was 50 in the left wrist; neither blood pressure nor pulse rate was obtainable on the right. No cardiac glycosides were given.

The electrocardiogram obtained eight hours after admission is reproduced in figure 4E. A sinus arrhythmia was present with cycle lengths ranging from 1.0 to 1.24 seconds. The QRS interval of 0.13 second and the double peaking of the R waves in right ventricular leads V₁, V₂, and V₃, and V₇, were indicative of right bundle branch block. As the electrode was moved from the V₁ position near the relatively thick base of the right ventricle to the V₃ position over the relatively thin apex of the right ventricle and close to the septum, the initial R wave showed the progressive increase in amplitude expected of a deflection of septal derivation, whereas the R’ wave showed a concomitant decrease, commensurate with the diminishing mass of the free wall of the right ventricle. The progressive decrease in the intervening downstroke was also in keeping with its origin from negative left ventricular cavity po-
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potentials, transmitted through the left and right atrium to the right precordium. The Q wave recorded in left ventricular leads V₃, V₄, and V₅ was considered normal, both as to duration and relative amplitude. The slight delay in onset of the intrinsioic deflection in V₃ and V₄ and the relatively tall R waves in these leads, in the face of right bundle branch block, suggested left ventricular hypertrophy. The Q-T interval of 0.32 second was abnormally long, even for the slow rate.

The T wave was upright and concordant with the double-peaked R wave in the first three precordial leads and, like the initial R wave of septal origin, showed a progressive increase in amplitude as the electrode was moved from the V₁ to the V₃ position. Tall, erect T waves of this configuration in V₂ and V₃ arouse the suspicion of posterolateral infarction, but no evidence of such was found in Lead aV₃ or V₄. The inverted T waves in V₂ and V₃ were concordant with the broad, slurred portion of the QRS complex. In summary, the electrocardiogram showed evidence of right bundle branch block and probable left ventricular hypertrophy. The only atypical feature was the concordance of the T waves and this was compatible with uncomplicated bundle branch block. Thus, the electrocardiogram was abnormal, but failed to confirm the suspicion of myocardial infarction engendered by the clinical history.

Death occurred eighteen hours after the electrocardiogram was made and autopsy revealed a dissecting aneurysm of the aorta. The heart weighed 548 grams and showed left ventricular hypertrophy. There was moderate coronary sclerosis, but no narrowing or occlusion and no evidence of myocardial infarction. Thus, a correctly interpreted electrocardiogram in this case furnished valuable negative evidence against myocardial infarction and pointed towards some other cause for the acute retrosternal constriction.

Q Waves in Leads from the Left Precordium

These are not uncommon in association with right bundle branch block, in the absence of myocardial infarction. The duration of these Q waves and the relative amplitude in reference to the succeeding R waves may be sufficiently great, especially in the presence of left ventricular hypertrophy, to raise the question of subendocardial anterolateral infarction. The problem is exemplified by Cases 37 and 38. The electrocardiograms are reproduced in figures 4F and G.

Case 37.—The patient, a man 67 years of age, had had increasing dyspnea over a period of one year and was admitted to the hospital with severe orthopnea with extensive peripheral edema. There were physical signs of aneurysm of the ascending aorta, complicated by atelectasis of the right upper lobe. Digitalization had been carried out prior to the recording of electrocardiographic tracings.

The presence of right bundle branch block was established by the QRS interval of 0.12 second together with the broad double-peaked R waves in right ventricular leads V₃ and V₄. The excessive RS-T depression and straightening of the RS-T segments in these leads were attributable to superimposed digitalis effects. The transitional zone was situated between the V₂ and the V₃ positions and the potential variations of the epicardial surface of the anterolateral wall of the left ventricle had the predominant effect on the QRS-T pattern in the last four precordial leads. The slight delay in onset of the intrinsioic deflection in these leads together with the relatively tall R waves, in the face of right bundle branch block, were interpreted as evidence of left ventricular hypertrophy. The presence of Q waves 4 mm. in depth and 25 per cent of the succeeding R waves in V₃ and V₄ raised the question of infarction of the subendocardial layer of the anterolateral wall and the Q/R ratio of 25 per cent in aV₃ suggested the possibility of extension into the subendocardial portion of the posterior wall. Inasmuch as the time interval from onset to nadir of the Q waves did not exceed 0.02 second in any lead, and in view of the fact that the Q/R ratios were borderline, but not definitely abnormal, it was concluded that the Q waves in all leads could be accounted for by left ventricular dilatation and hypertrophy in the absence of infarction.

On the day after the electrocardiographic tracings were made, death occurred due to massive pulmonary hemorrhage. Autopsy revealed a large aneurysm that obstructed and eroded the rightstem bronchus and compressed the pulmonary artery. The heart weighed 448 grams and showed moderate left ventricular hypertrophy. There was no evidence of coronary narrowing or myocardial infarction.

Case 38.—The patient was a disoriented man, 52 years of age, who was brought to the hospital at midnight with extreme orthopnea and cyanosis, but only moderate peripheral edema. He had had a chronic cough for several years and had been treated for recurrent congestive failure by his family physician during the preceding six months. The chest was barrel-shaped, but auscultation revealed crepitant rales throughout both lung fields. These rales were interpreted as evidence of acute pulmonary edema due to left ventricular failure. Despite the lack of definite information as to the amount of digitalis taken prior to admission, Cedilanid was given intravenously in three divided doses during the night to a total of 1.6 milligrams.

The electrocardiogram reproduced in figure 4G was obtained one-half hour before death. The cardiac rhythm appeared regular to auscultation.
both before and after the administration of Cedral and, but the electrocardiogram revealed auricular fibrillation with slightly irregular ventricular rhythm, cycle lengths varying from 0.40 second to 0.52 second. The ventricular complexes were uniform in contour in any given lead. The electrocardiogram was therefore interpreted on the assumption that the impulses responsible for the QRS-T complexes reached the ventricles through the A-V node.

Close scrutiny of Leads V1 and V2 revealed a minute initial R wave, a small downstroke, and a prominent slurred R' deflection, which attained a peak 0.08 second after the onset of the QRS complex. These findings together with a QRS interval of 0.12 second led to a diagnosis of right bundle branch block. The striking features of this case were the qRs complexes and abnormally elevated, dome-like RS-T segments in V1 and V2, and the Q deflection and rounded RS-T segment in V3 and aVL. These findings were attributed to recent infarction of the anterolateral wall of the left ventricle.

Autopsy revealed far-advanced obstructive emphysema and pulmonary arteriosclerosis, with a complicating acute suppurative bronchiolitis to account for the diffuse crepitant râles. The heart weighed 421 grams and showed evidence of marked dilatation and hypertrophy of the right ventricle. The hypertrophy was so marked that the weight of the right ventricular segment exceeded that of the left, as indicated by a ratio of 0.74. The coronary tree was of normal caliber. No evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks taken around the circumference of the ventricle at different levels.

After the necropsy was completed, reconsideration was given to the possibility that a ventricular tachycardia, arising from a focus in the left apex, might have been responsible for the bizarre QRS-T pattern. A QS- or W-shaped complex may be recorded through a lead over the site of origin of the ectopic impulse, but would not be expected over so wide an area as that covered by the V1, V2, and V5 positions. Furthermore, the first portion of the QRS in V1 and V2 (i.e., the rs deflection) was atypical of the pattern found in right ventricular leads in ectopic beats arising in the left ventricle. Hence restudy of the tracing affirmed the previous conclusion that the impulses responsible for the QRS-T complexes reached the ventricle by way of the A-V node.

Activation of the free wall of the markedly hypotrophied right ventricle was believed responsible for the prominent R' wave in V1 and V2 and for the deep S wave in V4, and V6. Predominant activation of the septum by impulses spreading from left to right apparently led to the insertion of the initial R wave in V1 and V2 and to the Q wave in V4 and V6. Since the initial r wave in V1 and V2 was normal in duration and very low in voltage, whereas the final R' was very broad and slurred, the conduction defect responsible for the lengthening of the QRS interval was probably located in the free wall of the right ventricle rather than in the septum. Activation of the anterior wall of the left ventricle was believed responsible for the small R wave in V4 and V6 and the small S wave in V1 and V2. Opposing negative potentials coming from activation of the right ventricle and perhaps the posterior wall of the left ventricle probably accounted for the smallness and brevity of the r wave in V1 and V2. The duration of the Q deflection in V4 corresponded with the time interval from the end of the R to the termination of the QRS complex in V1 and V2. Thus, the Q deflection in V4 was actually an S wave derived from activation of the right ventricle and perhaps in part from the posterior wall of the left ventricle. The potentials responsible for the antecedent qr deflection registered in V1 and V2 were apparently lost during the course of transmission through an emphysematous lung to position V4.

Dome-like elevation of the RS-T segment following a QS or a qRs complex may occur not only as a manifestation of myocardial infarction, but also from overdigitalization. The latter alternative was apparently responsible for the RS-T displacement in V1, V2 and V3 and should have been suspected during life for the following reasons: (1) a full dose of Cedilanid had been given to a patient who had been receiving digitalis in unknown amounts; (2) the Q-T interval ranged from 0.23 second to 0.25 second and was slightly shorter than the Ashman and Hull standards of 0.25 to 0.28 second for the cycle lengths represented. The absence of typical digitalis effects in other leads is left unexplained.

This case serves as another illustration of how closely patterned associated with right ventricular dilatation and hypertrophy mimic those due to myocardial infarction. An erroneous diagnosis of myocardial infarction was made by all members of the department who studied the tracing prior to knowledge of the pathologic findings. Experience with this case indicates that caution must be exercised in the interpretation of findings in leads from the left precordium when leads from the right precordium show evidence of right bundle branch block or right ventricular hypertrophy.

**Summary**

The differential diagnosis between uncomplicated bundle branch block and that associated with myocardial infarction has been brought out through a detailed analysis of the electrocardiograms in eleven cases. These cases were selected because of (1) QRS-T patterns considered diagnostic, or at least suggestive, of myocardial infarction; (2) subsequent exclusion of myocardial infarction at autopsy.

Left bundle branch block was present in four
of the cases and was manifested by one or more of the following signs that raised the question of myocardial infarction or led to an erroneous diagnosis during life: (1) broad, slurred Q8 deflections in the six customary precordial leads (V1 through V6); (2) a triphasic qRS pattern, consisting of small, narrow q and r deflections and a deep, broad S wave, in one or more precordial leads just to the right of the transitional zone; (3) registration of an rS complex in Lead V1 and/or V2 and a Q8 deflection in one or more leads farther to the left; (4) progressive diminution of the initial R wave, then replacement by a qRS deflection as the electrode was moved towards the left; (5) cove-plane inversion of the T wave associated with Q8- or W-shaped complexes in leads near the transitional zone.

Right bundle branch block was present in the other seven cases and was manifested by one or more of the following signs likely to be mistaken for those of myocardial infarction: (1) pseudo Q waves in leads from the right precordium and/or transitional zone; (2) elevation of the RS-T junction and abnormal inversion of the T wave in leads from the right precordium; (3) reduction in the R wave together with deep inversion of the T wave in intermediate leads; (4) concordant T waves; (5) prominent Q waves in leads from the left precordium.

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