American Heart Journal

VOL. 28 SEPTEMBER, 1944 NO. 3

Original Communications

THE Q_1 DEFLECTION OF THE ELECTROCARDIOGRAM IN BUNDLE BRANCH BLOCK AND AXIS DEVIATION

WILLIAM A. SODEMAN,* M.D., FRANKLIN D. JOHNSTON, M.D., AND FRANK N. WILSON, M.D.
ANN ARBOR, MICH.

IT IS the purpose of this article to present and discuss observations on the incidence, in standard Lead I, of QRS complexes which display an initial downward deflection, or Q wave.

At the beginning of this study, we examined the tracings taken in 169 cases of bundle branch block. In all of these, the QRS interval measured 0.12 second, or more, and pronounced slurring or notching of the broadest QRS component was present. In 92 cases, there was no S wave in Lead I; these were classified as left branch block. The remaining 77 cases, in which there was a conspicuous S deflection in Lead I, were considered characteristic of right branch block. In 84, or 91.3 per cent, of the cases of left branch block, the first and only QRS component in Lead I was a broad R wave; in the remaining 8 cases (8.7 per cent), a small initial downstroke preceded this deflection (Table I). In the same group of cases, there were 33, or 35.9 per cent, in which the QRS complex of Lead III began with a downstroke, and these included one of the 8 which displayed a Q wave in Lead I. A Q_1 deflection was present in 34, or 44.2 per cent, and absent in 43, or 55.9 per cent, of the cases of right branch block. The QRS complex of Lead III began with a downstroke in 36.4 per cent of the cases of this group.

These observations on the incidence of Q_1 in bundle branch block were confirmed by examination of the tracings obtained in a group of cases of bundle branch block in which the conduction defect was present on one examination, but absent on an earlier or later occasion.

From the Department of Internal Medicine, University of Michigan Medical School.

Many of the observations upon which this article is based were made with the aid of grants to F. N. Wilson from the Horace H. Rackham School of Graduate Studies.

Received for publication Aug. 1, 1943.

*Commonwealth Fellow.

TABLE I

THE FREQUENCY OF Q_1 AND Q_3 IN THE ELECTROCARDIOGRAMS OF 169 PATIENTS WITH BUNDLE BRANCH BLOCK

	LEFT BUNDLE BRANCH BLOCK		RIGHT BUNDLE BRANCH BLOCK	
	NUMBER OF PATIENTS	PERCENTAGE	NUMBER OF PATIENTS	PERCENTAGE
Q_1 present	8	8.7	34	44.2
Q_1 absent	84	91.3	43	55.9
Q_3 present	33	35.9	28	36.4
Q_3 absent	58	64.1	49	63.6
Q_1 and Q_3 present	1	1.1	11	14.3
Q_1 and Q_3 absent	52	56.5	26	33.8
Total	92		77	

TABLE II

THE EFFECT OF DEVELOPMENT OF BUNDLE BRANCH BLOCK ON THE Q WAVE IN LEAD I IN 102 PATIENTS SHOWING, AT ONE TIME, BUNDLE BRANCH BLOCK, AND, AT ANOTHER, NORMAL CONDUCTION

	LEFT BUNDLE BRANCH BLOCK		RIGHT BUNDLE BRANCH BLOCK	
	NUMBER OF PATIENTS	PERCENTAGE	NUMBER OF PATIENTS	PERCENTAGE
Q_1 present. Normal conduction	20	25.3	8	34.8
Q_1 absent. Normal conduction	59	74.7	15	65.2
Q_1 present. Bundle branch block	4	5.1	8	34.8
Q_1 absent. Bundle branch block	75	94.9	15	65.2
Q_1 absent, with and without block	57	72.1	14	60.9
Q_1 disappeared, with block	18	22.8 (of total) 90.0 (of those with Q_1)	1	4.3 (of total) 12.5 (of those with Q_1)
Q_1 appeared, with block	2	2.5 (of total) 3.4 (of those without Q_1)	1	4.3 (of total) 6.6 (of those without Q_1)
Q_1 with and without block	2	2.5 (of total) 10.0 (of those with Q_1)	7	30.4 (of total) 87.5 (of those with Q_1)
Total	79		23	

In our own files, we found 9 cases of this kind in which the block was on the left side, and 10 cases in which it was on the right side. By searching the literature, we collected 70 additional cases of the first sort and 13 of the second.[1-55] We did not include in this series any cases of the Wolff-Parkinson-White syndrome, nor any cases in which the QRS interval measured less than 0.12 second when the block was present, or more than 0.10 when it was absent. The incidence of Q_1 and other data relating to the 79 cases of left and 23 cases of right branch block assembled in this way are given in Table II. In 20 of the 79 cases of the first group, the QRS complex of Lead I began with a downstroke when intraventricular conduction was normal, and in all but two of these it began with an upstroke when left branch block was

TABLE III

INCIDENCE OF Q_1 IN PATIENTS WITH LEFT BUNDLE BRANCH BLOCK, RIGHT BUNDLE BRANCH BLOCK, LEFT AXIS DEVIATION, AND RIGHT AXIS DEVIATION

	LEFT BUNDLE BRANCH BLOCK		RIGHT BUNDLE BRANCH BLOCK		LEFT AXIS DEVIATION		RIGHT AXIS DEVIATION	
	NUMBER OF PATIENTS	PERCENTAGE	NUMBER OF PATIENTS	PERCENTAGE	NUMBER OF PATIENTS	PERCENTAGE	NUMBER OF PATIENTS	PERCENTAGE
Q_1 present	13	7.3	41	41	175	58.3	6	6
Q_1 absent	164	92.7	59	59	125	41.7	94	94
Total	177		100		300		100	

TABLE IV

Q_1 RELATIONSHIPS IN PATIENTS WITH LEFT BUNDLE BRANCH BLOCK AND LEFT AXIS DEVIATION (CONSECUTIVE CASES)

TYPE OF ELECTROCARDIOGRAM	Q_1 PRESENT	Q_1 ABSENT	TOTAL
Left axis deviation			
Index 24 or less	51	49	100
Index 25 or more	62	38	100
Normal T waves	69	31	100
Inverted T_1 or T_3 and T_2	55	45	100
Questionable left bundle branch block (QRS = 0.10 to 0.12)	46 (42.6%)	62 (57.4%)	108
Left bundle branch block	8 (8.7%)	84 (91.3%)	92

present. In the other 59 cases, the QRS complex of Lead I began with an upstroke when intraventricular conduction was normal; in 2 of these it displayed a Q wave when the left limb of the His bundle was blocked. As in the cases of left bundle branch block previously analyzed, the incidence of a Q_1 deflection was very low; it occurred in the presence of block in only 4 cases, or 5.1 per cent of the group. There was a Q_1 deflection with normal intraventricular conduction in 8 of the 23 cases of the second group, which is rather small for statistical purposes; in 7 of these, this deflection was likewise present when the right bundle branch failed to conduct. In the remaining 15 cases, Q_1 was absent with normal intraventricular conduction; in one of these, this deflection appeared when the right branch of the bundle was blocked.

The foregoing observations led us to compare the incidence of Q_1 in simple axis deviation with its incidence in bundle branch block. The ventricular complexes which depict simple left axis deviation and those which represent left branch block are often very similar in general contour, but our observations indicate that the electrocardiographic changes in these two conditions are fundamentally different in origin (Table III). With regard to the incidence of Q_1, left branch block is very different from simple left axis deviation (Table III). The relatively great frequency of a conspicuous Q_1 in simple left axis deviation, as compared to left branch block, is not materially affected by the criteria employed in the selection of examples of the former (Table IV). A Q_1 deflection was present in 51 of 100 cases of simple

TABLE V

Q-WAVE RELATIONSHIPS IN PATIENTS WITH RIGHT BUNDLE BRANCH BLOCK AND RIGHT AXIS DEVIATION

TYPE OF ELECTROCARDIOGRAM	Q_1 PRESENT		Q_3 PRESENT		Q_1 AND Q_3 PRESENT		NO Q		TOTAL
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE	
Right axis deviation	6	6.0	81	81.0	2	2.0	15	15.0	100
Right bundle branch block	34	44.2	28	36.4	11	14.3	26	33.8	77

left axis deviation in which the axis deviation index $(R_1 + S_3) - (R_3 + S_1)$, was 24 or less, and in 62 of 100 cases of left axis deviation in which this index exceeded 24. This deflection was present in 69 of a series of 100 consecutive cases of simple left axis deviation with normal T waves, and in 55 of 100 consecutive cases of the same kind in which the T waves were inverted in Lead I or in Leads I and II. It is of particular interest that Q_1 was present in 42.6 per cent of a series of 108 cases, all that could be found in a file of 8,000 electrocardiograms, in which definite left axis deviation was associated with a QRS interval of 0.10 to 0.12 second. With respect to the incidence of this deflection, electrocardiograms of this kind resemble those which depict simple left axis deviation, and are quite unlike those which represent left bundle branch block.

In simple right axis deviation, the frequency of Q_1 is very small, about the same as in left branch block, whereas, in right branch block, the frequency of this deflection is not very different from its frequency in left axis deviation (Tables I and V). The incidence of Q_3 is very high in right axis deviation and relatively low in right bundle branch block.

DISCUSSION

The incidence of Q_1 in bundle branch block has received little attention in the literature. Many years ago, Willius[56] recorded the size of the different QRS components in 99 examples of left branch block. His tables show that Q_1 was present in only two of his cases, but he did not comment upon this infrequency. In 1916, Lewis[57] was under the impression that Q_1 was usually present in bundle branch block of the common type; in 1924 he spoke of it as appearing to a variable extent.[58] In 1931, Wilson, Macleod, and Barker[59] stated that, in left branch block, Q is almost always present in Lead I and absent in Lead III. This statement was evidently based upon an impression, rather than upon the examination of an adequate series of cases.

With regard to the frequency of a Q_1 deflection, the curves that represent canine branch block are quite different from those that represent human branch block. In 6 of Lewis' experiments on dogs, Q_1 became larger; in two, it disappeared; and, in one, it persisted un-

changed when the left bundle branch was cut. In the remainder, it was absent both before and after section of this tract. A Q_1 deflection was present in four and absent in two of six examples of canine left branch block studied by Wilson and Herrmann.[60]

The rarity of Q_1 in human left branch block and the pronounced tendency for this deflection to disappear when left branch block develops clearly indicate that, in the vast majority of human electrocardiograms, it represents electrical forces originating in left ventricular muscle, or at least in muscle which receives the excitatory impulse by way of the left Purkinje plexus. The relatively high frequency of Q_1 in right branch block, and the tendency for this deflection to persist when normal conduction gives place to this disturbance, point in the same direction. Lewis, believing that the incidence of Q_1 was high in bundle branch block of the common type and, at the same time, that this kind of block was due to a conduction defect in the right bundle branch, arrived at the correct conclusion—that this deflection was contributed to the human bicardiogram by the levocardiogram. This is an instance in which the conclusion was valid even though the premises were erroneous.

Although it is clear that Q_1 usually represents electrical forces produced by the activation of left ventricular muscle, direct evidence as to the exact manner of its origin is not available. In experiments on dogs, endocardial readings have been employed in an attempt to locate the regions of ventricular muscle which are first to pass into the active state, but the data obtained in this way are of comparatively little value for the purpose of ascertaining the origin of the earliest QRS component of the human electrocardiogram. In the first place, the incidence of Q_1 is by no means the same in canine as in human curves, and, in the second, endocardial readings are not entirely trustworthy, as Wilson, Macleod, and Barker have pointed out. Even if the ventricular point which is activated earliest were known, we could not feel certain that Q_1 is written by events occurring in its neighborhood, for the electrical forces developed in this region may be overbalanced by the more rapid development of opposing forces somewhere else before they become large enough to produce a potential difference between the distant electrodes on the two arms.

The activation of left ventricular muscle may give rise to a Q_1 deflection by producing initial positivity of the right arm, initial negativity of the left arm, or both. In normal subjects, initial negativity of the anterolateral surface of the left ventricle often produces a Q deflection in leads from the left side of the precordium, and is frequently transmitted to the left arm as well. This negativity is transmitted to the epicardial surface from the ventricular cavity when the subendocardial muscle of the anterolateral wall enters the active state later, or produces electrical forces of less magnitude than the

subendocardial muscle on the opposite side of the left ventricle. It may, therefore, be ascribed to unbalanced forces produced by the spread of the excitatory impulse into the septum from the left Purkinje plexus. These same forces and, also, those generated by activation of the free wall of the right ventricle from within outward produce initial positivity of the epicardial surface on the right side of the heart, which gives rise to small R waves in leads from the right side of the precordium and is sometimes transmitted to the right arm. The absence of Q_1 in the vast majority of the cases of human left branch block appears, therefore, to be due to the absence of electrical forces normally produced by the activation of septal muscle from left to right.

What is the significance of Q_1 in the small percentage of cases of human left branch block in which it occurs? In left branch block, no left ventricular muscle is undergoing activation at the beginning of the QRS interval, and, if a Q deflection is present, it must be ascribed to forces of right ventricular origin. Under certain circumstances the initial positivity of the surface of the right ventricle due to the outward spread of the impulse through its free wall, which usually gives rise to small R waves in leads from the right side of the precordium in left branch block, may be transmitted to the right arm and thus give rise to a Q_1 deflection. It is apparent that this often happens in the dog and seldom happens in man. In the former, the long axis of the heart is much more nearly in line with the long axis of the body, and this may account for the difference in the frequency of Q_1 between canine and human left branch block. Rotation and elevation of the heart after section of the left bundle branch have caused a Q_1 deflection to appear in experiments on the dog, but not in experiments on the monkey, an animal in which the heart, with regard to its position, is more like that of man.[61] No peculiarities in the position of the heart were noted in the thirteen cases of left branch block in our series in which a Q_1 deflection was present. Both in the dog and in man, the presence of a Q_1 deflection, when the left branch of the His bundle is blocked, may, of course, depend upon some factor other than the position of the heart. A possibility that must be considered is that it is due to some peculiarity of the Purkinje system or the architecture of the subdivisions of the bundle branches, and consequently of the order of ventricular activation. In two instances, it was noted that a Q_1 deflection was present both before and after the development of left branch block, and had the same contour in both tracings. There exists, then, the possibility that in some instances the distribution of the conducting tracts is such as to lead to more rapid or earlier activation of those parts of the right ventricular muscle which produce forces of the kind that give rise to a Q_1 deflection, and that under these circumstances this deflection occurs and displays the same form in both bicardiogram and dextrocardiogram. This would account for the rare cases in which Q_1 disappears when right branch block develops.

In order to ascertain whether the presence of a Q_1 deflection in left bundle branch block has any diagnostic significance, we reviewed the histories of the thirteen patients in our own series and twenty-four cases found in the literature which presented this combination. An autopsy was performed in only two of our own cases. One of these was that of a man, aged 51 years, and in this instance the right coronary artery was occluded and the posterior ventricular wall was infarcted; the ventricular septum was not involved. In the second case, that of a man aged 56 years, there were pronounced cardiac hypertrophy, moderate coronary sclerosis, and slight fibrosis and patchy fatty degenerative infiltration of the myocardium. No macroscopic, circumscribed, septal lesions were found. In the remaining eleven cases, the following clinical diagnoses were made: arteriosclerotic heart disease with questionable coronary occlusion in two; coronary occlusion in five, in one of which left branch block antedated the symptoms pointing to infarction; arteriosclerotic heart disease with congestive failure in two; and rheumatic heart disease with mitral stenosis and aortic insufficiency in two.

The data relating to the conditions present in the twenty-four cases found in the literature are meager.[8, 22, 25, 32, 36, 41, 54, 56, 57, 62-70] In eight instances, no details of any kind were given as to the nature of the cardiac lesions. One patient had arteriosclerotic heart disease, cardiac enlargement, aortic insufficiency, and congestive failure. This patient died, but was not autopsied. Another had pericarditis with effusion and recovered; a third was said to have myocardial degeneration; a fourth, auricular fibrillation with congestive failure; a fifth, mitral stenosis; a sixth, aortic insufficiency; a seventh, diphtheria; an eighth, arteriosclerotic heart disease with failure. The ninth and tenth were reported as cases of coronary thrombosis in which the clinical diagnosis was confirmed by electrocardiographic examination; both of these patients recovered.

The remaining six patients died and were subjected to autopsy. The ventricular septum was involved in all. In one instance, there was occlusion of the anterior descending coronary artery, with infarction involving the anterior and part of the posterior wall of the left ventricle, the septum, and the apex. The interventricular septum was almost completely infarcted and in a state of liquefaction necrosis.[54] In another case there was thrombosis of an artery supplying the septum, with myocardial infarction and serofibrinous pericarditis.[66] In a third there was anterior infarction involving the septum.[32] The fourth was one with multiple infarcts; the lower, anterior part of the interventricular septum was involved.[70] In the fifth, described in the same report[70] as the fourth, a diagnosis of hypertensive heart disease with congestive failure was made, and moderate sclerosis of the coronary arteries was found post mortem. Histologic studies disclosed small scars in the myocardium, including a few in the septum, but the bulk

of the heart muscle appeared to be in good condition. In the sixth and last case,[63] the QRS interval measured only 0.107 second, but the precordial electrocardiogram indicated that activation of the left ventricle was delayed. At autopsy there was no cardiac hypertrophy; a healed infarct was found. It involved the entire apex, the apical four-fifths of the anterior and lateral walls of the left ventricle, and apical four-fifths of the anterior two-thirds of the interventricular septum.

From these few data, no very definite conclusions can be drawn. The heart was examined post mortem in only eight of the thirty-seven cases of left branch block with a Q_1 deflection which we were able to collect. It may be significant that, in six of these, myocardial infarction had occurred, and that, in five of the six and in one additional case, septal lesions were present. On the other hand, the cardiac abnormalities diagnosed clinically in many of the remaining twenty-nine cases in which there was no autopsy are not of a kind in which septal involvement would be expected. Even when bundle branch block is found after the occurrence of symptoms and physical signs characteristic of coronary thrombosis, one cannot feel certain that a large amount of the ordinary muscle of the ventricular septum has been infarcted. We know, however, that, in dogs, ligation of the septal artery, a large vessel not present in man, produces infarction of the basal part of the ventricular septum and often induces right bundle branch block or complete atrioventricular block.[71] Whether it ever induces left branch block alone is not certain. Right branch block produced in this way is sometimes, although not always, represented by ventricular complexes quite different in form from those obtained after section of the right branch of the His bundle.

On theoretical grounds, one might expect that, in left branch block, damage to the ventricular septum would lead to the appearance of a Q deflection in Lead I. In uncomplicated left branch block, the cavity of the right ventricle is negative throughout the QRS interval, but the cavity of the left is initially positive because of the direction of the electrical forces produced by activation of the septal muscle from right to left. This initial positivity is transmitted through the still inactive free wall of the left ventricle to the outer surface of this chamber and to the adjacent parts of the body, including the left side of the precordium, the left axilla, and, when the heart is in a relatively horizontal position, as in most patients with left branch block, to the left arm. Under these circumstances, the QRS complex of leads from the left side of the precordium display no Q deflection, and those of Lead I are of the same form. When the septum is extensively damaged, the electrical forces produced by its activation are reduced or abolished, and the initial negativity of the right ventricular cavity is transmitted to the left, and, consequently, to those regions on the left side of the body that are initially positive in left branch block when the septal

muscle is healthy. When this happens, Q deflections occur in leads from the left side of the precordium. They may be expected in Lead I also, although, in one case of this sort on record, large Q waves were present in leads from the left side of the precordium, but not in Lead I.[32]

From the data available, we cannot be sure that the mechanism in question gave rise to the Q_1 deflection in the cases of left branch block with septal lesions under consideration. In 2 of these, this deflection appeared after symptoms characteristic of coronary thrombosis had occurred, which suggests that infarction of the septum produced it. In another case, however, the Q_1 deflection antedated the coronary accident. The final decision as to whether there is a pronounced positive correlation between the presence of a Q_1 deflection in left branch block and septal involvement must wait until more extensive studies have been carried out. In the meantime, it is desirable that a full set of precordial leads be taken whenever a Q_1 deflection is encountered in tracings otherwise characteristic of left branch block, not only for the purpose of ascertaining whether left branch block is really present, but also to find out whether a Q deflection is present in leads from the left side of the precordium and left axilla.

It must be remembered that bundle branch block in man is almost always complicated by other cardiac abnormalities. The form of the electrocardiogram is determined not only by the failure of one bundle branch to conduct, but by extensive lesions of the ordinary ventricular muscle, as in infarction, and by involvement of other conducting tracts or the Purkinje network. Wilson and Herrmann[60] severed minor and major subdivisions of the left bundle branch in their experiments on dogs. In one instance, a cut on the left side of the septum, after the right branch of the His bundle had been divided, led to the appearance of a prominent Q_1 deflection. The possibility that the presence of Q_1 in left branch block is sometimes due to a combination of conduction defects must, therefore, be borne in mind.

The similarity in general contour between the ventricular electrocardiograms obtained in preponderant hypertrophy of the left ventricle and those characteristic of bundle branch block of the more common type has attracted attention for a great many years. Lewis, believing that the right branch of the His bundle was the one affected in bundle branch block of this type, brought forward a considerable body of evidence in support of the view that the ventricular complex was dominated by the levocardiogram in both conditions. Now that the block is known to be on the left side instead of the right, this view is no longer tenable.

The similarity in question involves the position of the mean electrical axis, the direction of rotation of the instantaneous electrical axis, the direction and sequence in time of the major QRS deflections of the different limb leads, and the form of the T waves, which are almost always inverted in Lead I in left bundle branch block and are very

often inverted in this lead in left ventricular hypertrophy. In many instances of great hypertrophy of the left ventricle, the QRS interval is increased to between 0.10 and 0.12 second, and, under such circumstances, it may be difficult to ascertain whether the electrocardiographic abnormalities are due to left ventricular hypertrophy alone or to incomplete left branch block.

Luten and Grove[35] and Hyman and Parsonnet[25] championed the view that pronounced left axis deviation with inversion of the T deflections in Lead I and upright T waves in Lead III is due to incomplete branch block, even when the QRS interval is not distinctly increased. Luten and Grove stressed the point that this conception was the only one that satisfactorily explained both the axis deviation and the form of the T waves. The anatomic arguments which they advanced to support it are no longer valid because they were based on the erroneous ideas concerning the diagnosis of right and left branch block which were current at the time their paper was written. In 1920, Fahr[73] put forward the hypothesis that the form of the ventricular complex in preponderant enlargement of the left ventricle is due to an increase in the length of the subdivisions of the left branch of the His bundle, and a consequent delay in the activation of the muscle of the left ventricle as compared to that of the right. At the same time he asserted that the classical views as to the location of the conduction defect in the two varieties of bundle branch block were erroneous, and that what had been considered right was really left branch block, and vice versa. Fahr's contention is clearly in accord with the observations of Luten and Grove and Hymen and Parsonnet, and supports the view that left axis deviation accompanied by inversion of the T waves in Lead I is the first stage, so to speak, in the development of left branch block; it also offers an alternative explanation of the tendency toward an increase in the QRS interval in left ventricular hypertrophy, attributed by Lewis to the increased thickness of the left ventricular wall.

The views regarding the cause of left axis deviation and inversion of the T deflections in Lead I in preponderant enlargement of the left ventricle held by Fahr are nearly, although not exactly, equivalent to the idea that these electrocardiographic changes are the result of incomplete left bundle branch block. Now, it is obvious that the initial QRS components in incomplete left branch block must be identical in form with those present in complete left branch block. In both cases these components represent the earliest phases of the dextrocardiogram, and there can be no reason why this should begin with a downward deflection in the one case and not in the other. It is for this reason that we have compared the incidence of Q_1 in axis deviation with its incidence in bundle branch block (Table IV). As we have already pointed out, this deflection is present in about one-half the cases of left axis deviation, and its frequency is nearly the same, regardless of whether we confine our attention to cases in which the

axis deviation index is very large or to cases in which it is only moderately increased, to cases in which the T waves are normal or to those in which the T waves are inverted in Lead I, or to cases in which the QRS interval lies within the accepted normal range or to those in which it measures between 0.10 and 0.12 second. On the other hand, Q_1 occurs in less than one-tenth of the cases of left bundle branch block. Contrary to what would be expected if left axis deviation were due to slow conduction of the cardiac impulse through the left limb of the His bundle, there is no tendency for the incidence of Q_1 to fall as the axis deviation index rises, or as the QRS interval lengthens. In order to obtain additional data bearing upon this problem, we reviewed all of the electrocardiograms taken in this laboratory over a period of three years with reference to the number of cases of left axis deviation in which Q_1 was the largest Q wave present in any of the limb leads; this information had been routinely coded. There were 1,199 cases of left axis deviation, and Q was largest in Lead I in 566, or 47.2 per cent of the total; Q was also largest in Lead I in 39 per cent of the 588 classified as showing slight left axis deviation, 54 per cent of the 611 classified as showing pronounced left axis deviation, and 40.4 per cent of 304 cases in which the T waves were inverted in Lead I and no digitalis had been given. In a review of curves of this last type, it was often noted, when a series of curves had been taken, that inversion of the T waves developed with the passage of time without any accompanying change in the contour of the QRS complex. These data show clearly that there is no justification for considering incomplete left branch block the sole, or even a common, cause of left axis deviation alone, or of left axis deviation associated with inversion of the T deflections in Lead I. For, if it were, we should certainly expect the incidence of Q_1 in left axis deviation to approach its incidence in complete left branch block as the form of the ventricular complex became more abnormal with respect to the value of the axis deviation index, the form of the T waves, or the length of the QRS interval.

We do not, of course, deny that left axis deviation, whether or not it is accompanied by inversion of T in Lead I, by an increase in the QRS interval, or by both, is sometimes due to incomplete left bundle branch block. When a Q deflection is present in Lead I, however, this is very unlikely, because the incidence of this deflection cannot be greater in incomplete than in complete left bundle branch block. When Q_1 is absent, the estimation of the probability that incomplete left branch block is or is not present is much more difficult. The probability that it is present is no doubt greater when the QRS interval measures between 0.10 and 0.12 second than when it is shorter. Since the incidence of Q_1 reached 42.6 per cent in the group of 108 cases of left axis deviation in which the QRS interval was more than 0.10 and less than 0.12 second in length and was no greater in those in which T_1 was inverted than in those in which it was upright, it seems prob-

able, however, that only a small percentage of the curves of this kind, in which Q_1 is absent, represent a conduction defect in the left limb of the His bundle.

In Lead III, the QRS complex begins with a downward deflection (Q or QS) in about one-third of the cases of left bundle branch block, and in approximately the same percentage of the cases of right branch block. In left branch block this deflection is not followed by a positive component, and should, therefore, be called QS instead of Q. Its presence may be due either to initial positivity of the left arm, to initial negativity of the left leg, or to both. The former is exceedingly common in left branch block because the initial positivity of the cavity of the left ventricle due to the spread of the cardiac impulse through the septal muscle from right to left is usually transmitted to the left arm. Were it not for the circumstance that the left leg is also initially positive in the majority of cases, because the initial positivity of the right ventricular surface due to the spread of the impulse through the free wall of the right ventricle is transmitted to it, a QS deflection would occur in Lead III almost as frequently as Q is absent in Lead I, and for the same reason. In a considerable percentage of the cases of left bundle branch block, the surface of the right ventricle is initially negative, as is shown by the absence of an R deflection in leads from the right side of the precordium, and in many of these this initial negativity is, no doubt, transmitted to the left leg and contributes to the frequency of a QS deflection in Lead III. It should be pointed out that these relations hold when the heart is in a relatively horizontal position. When the heart is relatively vertical, the potential of the left leg is like that at the left, instead of like that at the right, ventricular surface. In the dog, the long axis of the heart is nearly in line with the long axis of the trunk, and Q, or QS, deflections are very rare in Lead III in canine left branch block. In canine right branch block, on the other hand, Q_3 is present more often than absent.

In the kind of branch block curves that closely resemble those obtained in preponderant hypertrophy of the right ventricle with regard to the direction and relative size of the ventricular deflections of the standard limb leads, a Q_1 component very rarely occurs. Curves of this kind, which were at one time considered characteristic of left branch block, are very uncommon. In the great majority of the cases in which they occur, the precordial electrocardiogram is in every way typical of right branch block; in some instances, however, it indicates that the conduction defect is on the left side. Of the seventy-seven cases classified as right branch block in Table I, there were only seven in which the electrocardiograms were of this kind. In the other seventy cases, the QRS complex of Lead I displayed a narrow R wave which often attained a voltage equal to, or greater than, that of the broad, notched, or slurred S wave which followed it. The high incidence of Q_1 in right branch block is mainly due to the frequency of

this deflection in electrocardiograms of this type. When the heart is in a relatively horizontal position, as is usually the case when the patient has left axis deviation or bundle branch block, Q_1 is almost always of left ventricular origin, and its presence or absence is determined by the character of the potential variations at the surface of the left ventricle at the beginning of the QRS interval. In left branch block, it is rare because the potential of the left ventricular surface, and consequently of the left arm, is initially positive. In left axis deviation, it is present when these regions are initially negative and absent when they are initially positive. Since right bundle branch block does not materially affect the potential at the surface of the left ventricle early in the QRS interval, Q_1 persists, unchanged in form, or remains absent, as the case may be, when right branch block develops (Table II).

The rarity of Q_1 in right axis deviation cannot be explained in an entirely satisfactory manner at the present time. In normal persons who display this electrocardiographic peculiarity, the heart is usually in a vertical position. For this reason, initial negativity of the left ventricular surface is transmitted to the left leg, and produces Q deflections in Leads II and III instead of in Lead I. The potential of the left arm resembles that of the right ventricular surface, which is initially positive. In right ventricular hypertrophy the situation is different; the enlarged heart is ordinarily transversely placed. Usually, unlike right bundle branch block, right ventricular hypertrophy has a profound effect upon the potential at the surface of the left ventricle at the very beginning of the QRS interval. This condition is represented in the precordial electrocardiogram by tall R waves, often preceded by Q waves in leads from the right side of the precordium and by small R waves, followed by deep S waves, in leads from the left side of the precordium.[74] In right ventricular hypertrophy, therefore, Q deflections are of right ventricular origin, and depend upon the occurrence of initial negativity at the surface of the right ventricle. Since this initial negativity, when it occurs, is transmitted to the left leg and not to the left arm, which undergoes potential variations like those at the left ventricular surface, Q deflections, when present, appear in Leads II and III and not in Lead I. The data available at the present time afford no satisfactory explanation of the tendency for right ventricular hypertrophy to abolish initial negativity at the left ventricular surface or to induce initial negativity at the right ventricular surface. The solution of this problem must, therefore, be left to the future.

SUMMARY

An initial downward, or Q, deflection in Lead I is very uncommon in human left branch block. When this component occurs in an electrocardiogram otherwise characteristic of this conduction defect, a lesion

of the ordinary muscle of the ventricular septum should be suspected, and a full set of precordial leads should be taken.

A Q deflection in Lead I occurs in about one-half of all cases of left axis deviation, regardless of the criteria employed in selecting examples of this electrocardiographic abnormality. Left axis deviation accompanied by inversion of the T waves in Lead I may sometimes be due to incomplete left bundle branch block when Q_1 is absent, but it is almost never due to this cause when this deflection is present.

The incidence of Q_1 in right branch block is similar to its incidence in left axis deviation. In right axis deviation, this deflection is extremely rare.

REFERENCES

1. Bach, F.: On the Clinical Significance of Right Bundle Branch Block, Quart. J. Med. **23**: 261, 1930.
2. Baker, B. M.: The Effect of Cardiac Rate and the Inhalation of Oxygen on Transient Bundle Branch Block, Arch. Int. Med. **45**: 814, 1930.
3. Bishop, L. F., Jr.: Transient Recurrent Complete Left Bundle Branch Block, AM. HEART J. **15**: 354, 1938.
4. Bohnengel, C.: Paroxysmal Bundle Branch Block Associated With Physiologic Changes in a Patient With Organic Heart Disease, AM. HEART J. **16**: 587, 1938.
5. Bousfield, G.: Angina Pectoris: Changes in Electrocardiogram During Paroxysm, Lancet **2**: 457, 1918.
6. Campbell, M., and Suzman, S. S.: Simultaneous Disappearance of Gallop Rhythm and Bundle Branch Block, Lancet **1**: 985, 1932.
7. Carr, F. B.: Functional Bundle Branch Block, New England J. Med. **209**: 1101, 1933.
8. Carter, E. P.: Clinical Observations on Defective Conduction in the Branches of the Auriculo-Ventricular Bundle, Arch. Int. Med. **13**: 803, 1914.
9. Comeau, W. J., Hamilton, J. G. M., and White, P. D.: Paroxysmal Bundle Branch Block Associated With Heart Disease, AM. HEART J. **15**: 276, 1938.
10. Cowan, J., and Bramwell, J. C.: The Clinical Aspects of Bundle Branch Block, Quart. J. Med. **19**: 95, 1925.
11. Cutts, F. B., and Roberts, C. P.: Two-to-One and Three-to-One Left Bundle Branch Block in the Presence of Auricular Flutter, AM. HEART J. **15**: 501, 1938.
12. Digilio, V. A.: Reversible Bundle Branch Block in a Case of Toxic Goiter, AM. HEART J. **15**: 116, 1938.
13. Donath, F.: Über inkonstanten Schenkelblock und über die Wirkung von Corphyllamin auf das Herz, Ztschr. f. klin. Med. **132**: 802, 1937.
14. Dressler, W.: Atlas der klinischen Elektrokardiographie, Berlin und Wien, 1933, Urban & Schwarzenberg, Figs. 42 and 45.
15. Eidlow, S.: Transient, Recurrent Bundle Branch Block, Canad. M. A. J. **37**: 240, 1937.
16. Elliot, A. H., and Nuzum, F. R.: Bundle Branch Block With Periods of Normal Intraventricular Conduction: Report of an Unusual Case, AM. HEART J. **7**: 680, 1932.
17. Freundlich, J.: Über die Beeinflussung intraventrikularer Leitungsstörung durch den Carotisdruck, Deutsches Arch. f. klin. Med. **168**: 360, 1930.
18. Gilchrist, A. R., and Ritchie, W. J.: The Ventricular Complexes in Myocardial Infarction and Fibrosis, Quart. J. Med. **23**: 273, 1930.
19. Hart, T. S.: Block of the Branches of the Bundle of His, Arch. Int. Med. **35**: 115, 1925.
20. Herrmann, G., and Ashman, R.: Partial Bundle Branch Block: A Theoretical Consideration of Transient Normal Intraventricular Conduction in the Presence of Apparently Complete Bundle Branch Block, AM. HEART J. **6**: 375, 1931.
21. Hewlett, A. W.: A Case Showing Bundle Branch Block With Extrasystoles Originating in the Ventricular Septum, Heart **9**: 1, 1921.
22. Hill, I. G. W.: Bundle Branch Block: A Clinical and Histological Study. Quart J. Med. **24**: 15, 1930.

23. Horine, E. F.: Prognosis in Rheumatic, Hypertensive, and Syphilitic Heart Disease, AM. HEART J. 1: 617, 1926.
24. Hubert, G.: Beobachtungen über einen Fall von ausheilendem rechteseitingem Schenkelblock, Ztschr. f. Kreislaufforsch. 27: 449, 1935.
25. Hyman, A. S., and Parsonnet, A. E.: Bundle Branch Block. The Phenomenon of Its Development in Relation to Axis Deviation of the Heart, Arch. Int. Med. 45: 868, 1930.
26. Kelly, L. W.: Two-to-One Right Bundle Branch Block, AM. HEART J. 6: 285, 1930.
27. Keppel, C.: Über die Bedeutung der Elektrokardiographie für die Beurteilung von Diphtheriekranken, Jahrb. f. Kinderh. 147: 171, 1936.
28. Korth, C., and Schrumpf, W.: Über Interferenzdissoziation in Elektrokardiogramm, Deutsches Arch. f. klin. Med. 179: 321, 1936.
29. Krumbhaar, E. B.: Transient Heart Block—Electrocardiographic Studies, Arch. Int. Med. 19: 750, 1917.
30. Kurtz, C. M.: Transient Complete Bundle Branch Block, AM. HEART J. 11: 212, 1936.
31. Leinbach, R. F., and White, P. D.: Two-to-One Right Bundle Branch Block, AM. HEART J. 3: 422, 1928.
32. Levine, S. A., and Brown, C. L.: Coronary Thrombosis: Its Various Clinical Features, Medicine 8: 245, 1929.
33. Lewis, T.: Certain Physical Signs of Myocardial Involvement, Brit. M. J. 1: 484, 1913.
34. Lewis, T.: Auricular Flutter Continuing for 24 Years, Brit. M. J. 1: 1248, 1937.
35. Luten, D., and Grove, E.: The Incidence and Significance of Electrocardiograms Showing the Features of Left Axis Deviation and QRS of Normal Duration With Inverted T_1 and Upright T_3, AM. HEART J. 4: 431, 1929.
36. Master, A. M., Dack, S., and Jaffe, H. L.: Bundle Branch and Intraventricular Block in Acute Coronary Artery Occlusion, AM. HEART J. 16: 283, 1938.
37. Morris, R. S., and McGuire, J.: Transient Complete Bundle Branch Block, Am. J. M. Sc. 184: 202, 1932.
38. Perry, C. B.: Observed Onset of Bundle Branch Block With Coronary Thrombosis 45 Hours Later, AM. HEART J. 9: 677, 1934.
39. Pezzi, C.: Quelques remarques sur les troubles de la conductibilite intraventriculaire enregistres par l'electrocardiogramme, Arch. d. mal du cœur 18: 753, 1925.
40. Pick, A.: Beitrag zur Frage des atypischen Schenkelblocks, Ztschr. f. klin. Med. 129: 719, 1936.
41. Robinson, G. C.: The Relation of Changes in the Form of the Ventricular Complex of the Electrocardiogram to Functional Changes in the Heart, Arch. Int. Med. 18: 830, 1916.
42. Schwab, R.: Klinische und elektrokardiographische Beobachtungen über vorübergehenden Schenkel—under Verzweigungsblock, Deutsches Arch. f. klin. Med. 180: 664, 1937.
43. Slater, S. R.: Partial Bundle Branch Block. A Case of Three-to-One and Four-to-One Block, AM. HEART J. 5: 617, 1930.
44. Stecher, R. M.: Electrocardiographic Changes in Diphtheria, AM. HEART J. 4: 715, 1929.
45. Stenström, N.: An Experimental and Clinical Study of Incomplete Bundle Branch Block, Acta Med. Scandinav. 60: 552, 1924.
46. Stenström, N.: Further Experience on Incomplete Bundle Branch Block in Man, Acta Med. Scandinav. 67: 353, 1927.
47. Tung, C. L., and Cheer, S. N.: A Correlation of Clinical and Electrocardiographic Findings in Human Bundle Branch Block, Chinese M. J. 47: 15, 1933.
48. Viko, L. E., Marvin, H. M., and White, P. D.: A Clinical Report on the Use of Quinidine Sulphate, Arch. Int. Med. 31: 345, 1923.
49. von Mentzingen, A.: Über einen Fall von funktionellen Verzweigungblock, Klin. Wchnschr. 13: 1158, 1934.
50. Willius, F. A.: The Progression of Myocardial Disease as Recorded by Serial Electrocardiograms, M. Clin. North America 16: 1493, 1933.
51. Willius, F. A., and Anderson, M. J.: Transient Recurrent Complete Bundle Branch Block, AM. HEART J. 10: 248, 1934.
52. Willius, F. A., and Keith, N. M.: Intermittent Incomplete Bundle Branch Block, AM. HEART J. 2: 255, 1927.
53. Wolferth, C. C., and Margolies, A.: Asynchronism on Contraction of the Ventricles in the So-called Common Type of Bundle Branch Block: Its Bearing on the Determination of the Side of the Significant Lesion and on the Mechanism of Split First and Second Heart Sounds, AM. HEART J. 10: 425, 1935.

54. Wolferth, C. C., Wood, F. C., and Bellet, S.: Acute Cardiac Infarction Involving Anterior and Posterior Surfaces of Left Ventricle, Arch. Int. Med. **56**: 77, 1935.
55. Wood, F. C., Jeffers, W. A., and Wolferth, C. C.: Follow-Up Study of 64 Patients With a Right Bundle Branch Conduction Defect, AM. HEART J. **10**: 1056, 1935.
56. Willius, F. A.: Clinical Features of Cases Exhibiting Electrocardiograms Conforming to Those of Experimental Complete Bundle Branch Block, AM. HEART J. **1**: 576, 1926.
57. Lewis, T.: The Spread of the Excitatory Process in the Vertebrate Heart, Phil. Trans. Roy. Soc. **207B**: 221, 1916, Part IV.
58. Lewis, T.: Clinical Electrocardiography, Ed. 3, London, 1924, Shaw & Sons.
59. Wilson, F. N., Macleod, A. G., and Barker, P. S.: The Interpretation of the Initial Deflections of the Ventricular Complex of the Electrocardiogram, AM. HEART J. **6**: 637, 1931.
60. Wilson, F. N., and Herrmann, G. R.: An Experimental Study of Incomplete Bundle Branch Block and of the Refractory Period of the Heart of the Dog, Heart **8**: 229, 1921.
61. Foster, P. C.: The Relation of the Position of the Heart to the Initial Ventricular Deflections in Experimental Bundle Branch Block, AM. HEART J. **10**: 1042, 1935.
62. Katz, L. N., Landt, H., and Bohning, A.: The Delay in the Onset of Ejection of the Left Ventricle in Bundle Branch Block, AM. HEART J. **10**: 681, 1935.
63. Kossmann, C.: The Precordial Electrocardiogram in Myocardial Infarction. Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of Doctor of Medical Science at New York University, April 1, 1938.
64. Kountz, W. B., Prinzmetal, M., and Smith, J. R.: The Effect of Position of the Heart on the Electrocardiogram. II. Observations Upon the Electrocardiogram Obtained From a Dog's Heart Placed in the Human Pericardial Cavity, AM. HEART J. **10**: 614, 1935.
65. Maher, C. C.: Electrocardiography, Baltimore, 1934, William Wood & Company.
66. Saphir, O., Prest, W. S., Hamburger, W. W., and Katz, L. N.: Coronary Arteriosclerosis, Coronary Thrombosis, and the Resulting Myocardial Changes, AM. HEART J. **10**: 567, 1935.
67. Talley, J. E., and Reed, O. K.: A Study of Twenty-Eight Cases of Bundle Branch Block, AM. HEART J. **1**: 262, 1926.
68. Van Nieuwenhuizen, C. L. E., en Matthijssen, E.: Het Electrocardiogram in vier afleidingen bij bundel-en vertakkingsblock, Nederl. tijdschr. v. geneesk. **81**: 5308, 1937.
69. Wenckebach, K. E., and Winterberg, H.: Die unregelmässige Herztätigkeit, Tafelband, Leipzig, 1927, Wilhelm Engelmann.
70. Yater, W. M.: Pathogenesis of Bundle Branch Block, Arch. Int. Med. **62**: 1, 1938.
71. Wilson, F. N., Hill, I. G. W., and Johnston, F. D.: The Form of the Electrocardiogram in Experimental Myocardial Infarction, AM. HEART J. **9**: 596, 1934.
72. Wilson, F. N.: In Diseases of the Coronary Arteries and Cardiac Pain, New York, 1936, The Macmillan Co. Edited by Robert L. Levy.
73. Fahr, G.: An Analysis of the Spread of the Excitation Wave in the Human Ventricle, Arch. Int. Med. **25**: 146, 1920.
74. Wilson, F. N., Johnston, F. D., Cotrim, N., and Rosenbaum, F. F.: Relations Between the Potential Variations of the Ventricular Surface and the Form of the Ventricular Electrocardiogram in Leads From the Precordium and the Extremities, Tr. A. Am. Physicians **56**: 258, 1941.