The Electrocardiogram in Ventricular Hypertrophy and Bundle-Branch Block

A Panel Discussion

By Charles E. Kossmann, M.D., Moderator, Howard B. Burchell, M.D., Raymond D. Pruitt, M.D., and Ralph C. Scott, M.D.

DR. KOSMANN: Our panel has been assigned the task of unravelling for you, to the best of our ability, the dual problems of the electrocardiogram in ventricular hypertrophy and in bundle-branch block. For the achievement of this formidable assignment we have been allotted exactly 45 minutes. That we will succeed in clarifying a subject that was destined, almost from its inception a half century ago, to be characterized by repeated experimental error, fanciful speculation, and whimsical nomenclature is not likely, or more correctly, is not possible. But perhaps our deliberations will show at least that there are inadequate facts to justify making certain electrocardiographic interpretations, and drawing inevitable anatomic conclusions therefrom as is commonly practiced in the clinic.

Historically the matter of bundle-branch block got off on the wrong foot, or more exactly, on the wrong side. After Eppinger and Rothberger1 introduced the concept in 1910, Lewis and Rothschild2 performed experiments on dogs and defined the criteria for left and right bundle-branch block. Roughly 15 years later the classic experiments of Barker, Macleod, Alexander, and Wilson3 on an exposed human heart demonstrated that the usually meticulous Lewis, probably due to the unusual thoracic position of the dog's heart in his experiments, had mistakenly reversed the sides of the block in his conclusions.

With the advent of chest leads and practical methods for recording vectorcardiograms the quest for greater refinement in the diagnosis of intraventricular block of all types, as well as in the diagnosis of enlargement not only of an entire chamber but of parts of it, has gone on apace. In fact, almost no academic institution with even a passing interest in cardiac disease has failed to assign at some time one of its young men to investigate these intriguing but stubbornly unyielding problems.

Perhaps it will be of some value to point out first some of the numerous variables that must be considered in any intelligent discussion, or in any further investigation of the form of the electrocardiogram in ventricular hypertrophy and in bundle-branch block.

I. Hypertrophy

A. Extracardiac—variable normal position of heart,4 distortion of position by disease (effusion, thoracic deformity, pneumothorax)

B. Intracardiac

1. Anatomic

   a. Dilatation—internal medium and electric images5

   b. Hypertrophy

      (1) Degree

      (2) Location

         (a) Outflow tract (R' in leads V1 and V2 in right ventricular hypertrophy)

         (b) Inflow tract (deep S wave in leads V1 to V3 in right ventricular hypertrophy)

         (c) Free wall

         (d) Crista supraventricularis (right ventricle)

         (e) Combinations

         (f) Panventricular

         (g) Concentric or eccentric

      (3) With contralateral ventricle

      (4) With intrinsic myocardial disease

2. Physiologic (dynamic)

   a. Flow—diastolic overload (volume overload), long diastolic fiber length6
b. Pressure—systolic overload (pressure overload)
c. Combinations
d. Underload—tricuspid atresia
e. Effects of surgical correction of abnormal pressures and flows

C. Block vs. hypertrophy—simulation of one by the other

D. Nomenclature—the undisciplined procedure of using anatomic terms in describing electrocardiographic configurations leads not only to erroneous diagnoses but has probably impeded productive thinking.

II. Bundle-branch block
A. Nomenclature—bundle-branch block used loosely to include all types of intraventricular block
B. Possible locations
1. Bundle branch
   a. Major subdivision of branch (particularly of left)
2. The Purkinje system
3. The transitional cells
4. Myocardium itself (hyperkalemia), including “mural” or “parietal” block
C. With hypertrophy
D. With widespread intrinsic disease
E. Block vs. sequence—variable normal sequence of ventricular excitation apparently has not been properly weighted, leading to acceptance of form of QRS as a criterion of intraventricular block without regard to duration
F. Surgical production—may be useful, where it occurs inadvertently, to answer certain questions.

These are just a few of the aspects of the problem that have made it a difficult one to resolve.

With the hope of achieving some meeting of the minds relative to the electrocardiogram in ventricular hypertrophy and in bundle-branch block we will proceed to a series of questions to be put to our panelists.

Dr. Scott, what in your opinion, are the most useful electrocardiographic criteria that would lead to an accurate inference that there is (a) left ventricular hypertrophy, (b) right ventricular hypertrophy?

Dr. Scott: In our experience the diagnosis of left ventricular hypertrophy (LVH) is most accurately made when there is a combination of high voltage in the left precordial and in the limb leads, a delay in the onset of the intrinsicoid deflection, and secondary S-T segment and T-wave abnormalities in left precordial leads.

The voltage criteria we employ in adults are R\text{V}_5, v_6 > 26 \text{ mm.}, S\text{V}_1 + R\text{V}_5, v_6 > 35 \text{ mm.} (> 40 \text{ mm. in men 20 to 25 years of age}), R\text{A}V_L > 11 \text{ mm.}, R + S_m > 25 \text{ mm.}, maximum R + maximum S in precordial leads > 45 \text{ mm.} Some workers have insisted that the high voltage be present in lead V_6 as well as in lead V_5. We agree that this makes the likelihood greater of LVH being present but at the same time by insisting on this criterion, certain cases of anatomic LVH may be missed.

We also believe that it is a mistake to use the depth of the S wave in lead V_2 rather than in lead V_1 in the expression of S\text{V}_1 + R\text{V}_5, v_6 > 35 \text{ mm.} because this will greatly increase the incidence of false positive diagnosis.

R + S_m > 25 \text{ mm.} is less frequently encountered in LVH than in high voltage in the precordial leads, but when present it is a reliable sign. We have only occasionally encountered it as a false positive.

We have evaluated the accuracy of 11 sets of voltage criteria for LVH in 71 unselected cases intensively studied at necropsy. The right and left ventricles were separated and individually weighed. There were 37 cases of isolated or dominant LVH and 34 cases of no ventricular hypertrophy. The most useful criteria are those that give the highest number of positive diagnoses with the fewest false positive diagnoses (table 1).

In view of Simonson’s recent excellent monograph on the normal ranges in electrocardiography, certain revisions in our voltage criteria may be indicated. On the basis of his upper (97.5 percentile) limits, it would appear that the following voltages are abnormal: R\text{V}_6 > 20 \text{ mm.}; S\text{V}_1 + R\text{V}_5 > 33 \text{ mm.} in women; S\text{V}_1 + R\text{V}_5 > 36 \text{ mm.} in men over 30; S\text{V}_1 + R\text{V}_5 > 44 \text{ mm.} in men 20 to 29 years of age.

Delay in the onset of the intrinsicoid deflection has been found in only about 30 per cent of our cases of autopsy-proved LVH and seldom has it been the only criterion present.

Because of the multiplicity of factors that

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may affect the S-T segment and T wave, we seldom make a diagnosis of LVH on these changes alone.

The criteria we employ for the diagnosis of LVH in infancy and childhood are based primarily upon R waves in the left precordial leads and S waves in the right precordial leads greater than the maximum normal for age. It should be pointed out that T-wave inversion in the left chest leads in infants and children may occasionally be the only manifestation of LVH. Tall R waves accompanied by deep Q waves and tall T waves in leads II, III, aVF (as well as in the left chest leads), especially in infants and children, may also indicate LVH (left ventricular diastolic overloading).

The electrocardiographic diagnosis of right ventricular hypertrophy (RVH) leans heavily upon abnormal right axis deviation (RAD) and high voltage in lead V1 (V3R, V4R). The criteria we employ for the diagnosis of RVH in the adult are RAD of +110° or greater; R/S ratio > 1 in lead V1 (V3R, V4R); R/S ratio < 1 in lead V6 (V6); qR, rR, R, Rs, or rSR’ in lead V1 (V3R, V4R). We interpret the rSR’ pattern to represent hypertrophy of the right ventricular outflow tract unless the R’ is broad and accompanied by broad S waves in leads I, V5, and V6. In the latter case this indicates terminal slowing of inscription of QRS and is thought more likely to be due to incomplete right bundle-branch block. In the presence of incomplete right bundle-branch block tall secondary R waves (> 10 mm.) may, but do not necessarily, indicate concomitant RVH. Small r and deep S waves extending across the precordium may indicate dilatation or hypertrophy of the trabecular region or inflow tract of the right ventricle. This pattern, of course, must be differentiated from that encountered in pulmonary emphysema and front anterior or anterolateral myocardial infarction. Deep S waves in leads V1, V5, or V6 have been described in RVH and have been ascribed to posterior and rightward deviation of the QRS loop in the horizontal plane.

The electrocardiographic criteria we employ for the diagnosis of RVH in infancy and childhood may be listed as follows: RAD of +120° or more after 3 months of age; R/S
ratio in lead V1 (V4R) > maximum normal for age; R wave voltage in lead V1 (V4R) > maximum normal for age; SV6 > maximum normal for age; qR in lead V1 (V4R); positive T wave in lead V1 (V4R) after the first 48 hours of life with R/S ratio > 1. We are currently engaged in an electrocardiographic-pathologic correlation study of 180 infants and children. Unfortunately this study is not yet completed and we cannot assess the sensitivity and specificity of these criteria.

Dr. Koossmann: Dr. Burchell, what is the significance of high voltage per se in the precordial leads as an indication of hypertrophy?

Dr. Burchell: In the way of introduction to this question, it may be emphasized that the interpretation of changes in the electrocardiogram that are helpful as indications of ventricular hypertrophy are quite different for the clinician at the bedside, where the tracing is used in conjunction with all the other evidence, than for the pure electrocardiographer, who would like to have exact criteria and to know the statistical probabilities of rightness and wrongness of a report based on the electrocardiogram alone. Insurance medical directors and the surgeons of the Armed Forces would also find the latter information most valuable. For those engaged in adapting machine analyses to electrocardiographic recordings, standards for abnormality are essential, if diagnoses are to be printed out directly.

The electrocardiogram is the best single method available to us clinically as an indicator of predominant hypertrophy of either ventricular chamber. In a physiologic sense the adult mammal has a relative left ventricular hypertrophy, a concept embodied years ago in the term, left ventricular predominance. In the dog particularly, scalar electrocardiograms have a form that, from human standards, might be ascribed to left ventricular hypertrophy [systolic (or pressure) overloading type] in a vertical heart. In addition, one may at times record electrocardiograms from the lower esophagus or upper stomach in normal man that suggest left ventricular hypertrophy, including a negative T wave. In the infant, there is normally relative right ventricular hypertrophy, and thus there is a different base from which to judge an electrocardiogram than in the adult.

As a historic aside, it may be noted that Fahr (for whom an Eightieth Birthday Festschrift has been prepared this year—in Journal Lancet 82: February, 1962) proposed methods in 1920 for demonstrating the sequential vectors as projected on the frontal plane in persons with right and left ventricular hypertrophy. From theoretic approaches he also concluded that the electrocardiographic diagnosis at that time of right and left bundle-branch block was reversed.

In the diagnosis of congenital defects, the information contributed by the electrocardiogram as to the size and thickness of either ventricle, and as a corollary, its dynamic function, is singularly more imposing than generally observed in the area of acquired heart disease.

As to the significance of high voltage per se, Dr. Scott has outlined this in detail. It is to be emphasized that it is just one item that is often helpful. The duration of the QRS, and the spatial orientation of QRS and T in the frontal and horizontal planes may be often more important. In the past decade, however, the value of high voltage of tracings, and herein one refers to precordial leads, may have been downgraded to the extent that it is not utilized to its full advantage as a clue to hypertrophy. In infancy and early childhood the relative voltages of the R in leads V1 and V5 will give a good indication of the comparative thicknesses of the two ventricles; when the R voltage is in excess of 3.5 mv., hypertrophy of the ventricle underlying that lead is to be expected.

One criterion for left ventricular hypertrophy has been Sv5 + Rv5 in excess of 3.5 mv. While a reasonable correlation might be expected, this summation has not been particularly useful in my experience, but such a value (3.5 mv.) in either Sv5 or V5 or Rv5 or V5 has been impressive in clinical practice. In particular, the depth (high voltage) of Sv5 is important as an indicator of probable left
ventricular hypertrophy in adults and if one visualizes this lead with reversed polarity, which potentials should exist posteriorly, a more conventional pattern of hypertrophy may be conceived. This feature may be the more impressive when lead V₆ reveals an rS with a mild S-T segment elevation slanting upward to a prominent T wave and not uncommonly followed by a noticeable U wave.

Eventually such impressions as these will need documentation by hard facts—data from large numbers of persons with autopsy protocols—or else be properly discounted.

**Dr. Kossmann:** Would you go on, Dr. Burchell, and tell us whether there is an adequate electrophysiologic explanation for the electrocardiographic findings with hypertrophy?

**Dr. Burchell:** Electrophysiologic explanations may be offered for the changes in voltage that may occur with hypertrophy. Potentials recorded from the surface of the heart are often roughly five-fold what may be recorded from the chest wall overlying the same area of the organ. This emphasizes the distance (or proximity) factor as the enlarged heart may be more closely applied to the chest wall. It is recognized that the distance of the electrode to the heart may be modified by other factors, as obesity, pulmonary disease, and thoracic malformation, and these features undoubtedly forestall any good correlation between hypertrophy and chest wall voltage.

While the basic concept of the existence, at any instant, of a single summated cardiac dipole is held as a starting point for all electrocardiographic interpretations, it is also held that there are overwhelming proximity effects of the enlarged and hypertrophied heart and indeed specifically of potentials arising in one or the other enlarged and hypertrophied ventricle may dominate the recorded potential at the chest wall.

The terms enlargement and hypertrophy are sometimes used interchangeably, which in practice is usually valid, but, certainly hypertrophy can occur before external dimensions can be appreciably increased as judged by our crude clinical methods (e.g., aortic stenosis) and certainly dilatation can occur before appreciable hypertrophy (e.g., acute arteriovenous fistula). As a generalization, hypertrophy will be more clearly reflected in the electrocardiogram when there is concomitant enlargement. The size of the ventricle will influence the electric field and an increased radius of curvature will allow a more perpendicular excitation front to face the exploring electrode and a larger voltage to be recorded. This effect is the offered explanation in part when one has recorded increased voltages in arteriovenous fistula, and decreased voltages in animals in shock with small hearts.

A third factor that may be operative could be such sufficient delay in the completion of the excitatory process in the hypertrophied ventricle, to allow its last potentials to be unopposed by those of opposite polarity arising in other portions of the heart. This factor undoubtedly is present and operative in some cases of intraventricular conduction defects. Classic right and left bundle-branch block give rise to minor voltage changes and when excitation delays occur in a hypertrophied ventricle, the terminal portion of the QRS may be expected theoretically to have increased voltage and, practically, modest increases are seen. One may state that in a hypertrophied left ventricle the recorded electrocardiograms are predominantly potentials originating in the left free wall of this ventricle.

It is common experience to see low voltage precordial electrocardiograms in some persons with enlarged hearts with hypertrophy, for example in marked scarring of the myocardium from infarction or in amyloid disease, and the obvious explanation is that the excitation front is disorganized and there is internal neutralization of the summated instantaneous vector from the multiple dipoles that are oriented irregularly in the excitation area.

**Dr. Kossmann:** Dr. Pruitt, do you think it possible to distinguish ventricular dilatation from hypertrophy on the electrocardiogram?

**Dr. Pruitt:** I believe the facts suggest that
an electrocardiographic distinction between the consequences of essentially pure and extreme ventricular hypertrophy and pure and extreme ventricular dilatation is possible at a high level of accuracy. Our frustration stems from the rare occurrence of either hypertrophy or dilatation in pure state.

Pure dilatation should ensue when a ventricular chamber maintains an exceptionally high stroke volume against an inconsequential resistance to flow. Such a situation is approximated in patients having a large atrial septal defect and a normal pulmonary vascular resistance. The consistent relation between this congenital defect and its electrocardiographic expression is established, though the precise cause of that distinctive ventricular complex evades definition. Pure ventricular dilatation of the left or systemic ventricle is rendered impossible by the etiologic requirement of an inconsequential peripheral resistance enduring over a period of months or years.

Pure hypertrophy should ensue when a ventricular chamber maintains a normal stroke volume against an exceptionally high resistance to flow, and accomplishes this work without sacrifice of myocardial nutrition or efficiency. These requirements can be met by the ventricle supplying either the pulmonary or systemic circulation, and the corresponding clinical state is encountered in young patients having severe pulmonic or aortic stenosis. The electrocardiographic expressions of pure and severe right ventricular hypertrophy are quite distinct from those of pure and severe right ventricular dilatation. The expressions of pure and severe left ventricular hypertrophy, on the other hand, cannot be set in clear distinction from those of (a) pure dilatation, which is a more or less hypothetical condition, (b) dilatation combined with hypertrophy, which is an inherently complex condition, or (c) a normal left ventricle, which is, though normal, in a state of physiologic hypertrophy as compared with its right-sided counterpart.

Between the extremes of pure and severe ventricular hypertrophy on the one hand and pure and severe dilatation on the other, lie a multitude of states representing gradations in severity and combinations of forms, i.e., hypertrophy and dilatation. The impure variants at differing levels of severity constitute the majority of situations encountered by the clinician, and justifiably frustrate an overzealous attempt to categorize the usual electrocardiogram as indicative of hypertrophy to the exclusion of dilatation or dilatation without hypertrophy.

In summary, basic physiologic consideration would lead to the surmise that the electrocardiographic distinction between ventricular hypertrophy and ventricular dilatation would be most clearly defined when these changes in relatively pure and severe form affected the right ventricle; that left ventricular changes would present a complex and confused electrocardiographic expression, as would likewise the consequences of combined hypertrophy and dilatation affecting either ventricle. Since diseases producing pure and severe right-sided hypertrophy or dilatation are rare, so also would be a clearly discernible distinction between the electrocardiographic effects of hypertrophy and those of dilatation. Experience validates these surmises.

Dr. KoSSMann: Dr. Scott, how accurate and inclusive are the diagnostic criteria of hypertrophy you gave earlier? What are some of the causes of false positive and false negative diagnoses?

Dr. Scott: We have studied the accuracy of the electrocardiographic criteria in the diagnosis of ventricular hypertrophy chiefly in autopsy-controlled studies. It should be emphasized that because of the high incidence of abnormally heavy hearts occurring in an unselected autopsy population, this may result in what appears to be a greater accuracy in the electrocardiographic diagnosis of ventricular hypertrophy than may actually be the case.

It is well recognized that minimal or borderline cases of left ventricular hypertrophy may be missed by all electrocardiographic criteria. High voltage in the precordial leads is the most sensitive criterion for the diag-
nosis of isolated or dominant LVH but at the same time may lead to a considerable number of false positive diagnoses. Increased voltage in the limb leads is less sensitive but more specific, since it is less prone to result in a false positive diagnosis.

Delay in the onset of the intrinsicoid deflection in the left precordial leads occurs less frequently than does high voltage and seldom does it occur as the only criterion, but when present it adds to the specificity of the diagnosis of LVH. Delay in the onset of the intrinsicoid deflection occasionally may result in a false positive diagnosis; this may be due to incomplete left bundle-branch block.

With use of one or more electrocardiographic criteria, perhaps as many as 85 per cent of proved cases of LVH may be correctly diagnosed.

Causes of false positive diagnoses of LVH include such diverse conditions as overstandardization of the record, slender body build, emaciation, and the application of adult criteria to the younger age groups. The fallacy of diagnosing LVH on changes in the S-T segment and T wave alone has been pointed out earlier.

Causes of false negative diagnoses of LVH may include such conditions as overstandardization of the record, congestive heart failure, pleural effusion, anasarca, pericardial effusion, pulmonary emphysema, left pneumothorax, myocardial infarction, and obesity.

We have also found that right bundle-branch block, as well as coexisting RVH, may mask the diagnosis of LVH. While anatomic LVH is usually found in the presence of left bundle-branch block, the conventional electrocardiographic criteria for the diagnosis of LVH are not valid.

The electrocardiographic diagnosis of RVH is less reliable than that of LVH. It is well known that considerable anatomic RVH may be present without any electrocardiographic evidence and even with relatively normal total heart weight.

Over-all electrocardiographic accuracy in diagnosing RVH has ranged from 23 to 100 per cent in various autopsy-controlled series.

The diagnosis of RVH is most accurate in cases of congenital heart disease, moderately accurate in mitral stenosis and cor pulmonale, and commonly missed in cases of right ventricular enlargement secondary to left heart failure.

The electrocardiographic criteria that depend upon both rightward as well as anteriorly directed forces are more accurate and specific than either class of criteria alone.

A variety of conditions may result in a false positive diagnosis of RVH. Strictly posterior myocardial infarcts may produce tall R waves in the right precordial leads; sequential S-T segment depression followed by tall T waves in these leads will help distinguish these cases from RVH. The Wolff-Parkinson-White syndrome (type A) may superficially resemble RVH. Emphysema (without cor pulmonale) may result in electrocardiographic changes (rS patterns in all precordial leads) that mimic RVH. Anterolateral myocardial infarction may produce abnormal RAD as well as an rS pattern in left precordial leads. Abnormal RAD may rarely occur in normal hearts and in LVH. Displacement of the heart to the left may paradoxically cause RAD.

Such conditions as dextrocardia, dextroposition, and dextrorotation may erroneously be diagnosed as RVH. Misplacement of the right precordial electrodes, displacement of the transition zone to the right, and utilization of adult criteria in infants and children also may result in a false positive diagnosis of RVH.

Concomitant LVH commonly masks RVH. The difficulty in distinguishing between the pattern of hypertrophy of the right ventricular outflow tract and incomplete right bundle-branch block has already been noted. Left bundle-branch block, although uncommonly associated with RVH, will usually mask the latter diagnosis.

DR. KOSSMANN: Dr. Burchell, how accurate is the electrocardiographic recognition of ventricular hypertrophy in patients with congenital heart disease? Consider ventricular
septal defect, patent ductus arteriosus, and aortic and pulmonic stenosis.

Dr. Burchell: In the presence of severe outlet obstruction of either ventricle with the ventricular septum intact the electrocardiogram will accurately reflect hypertrophy of the obstructed ventricle. In infancy it will modify through delay or acceleration the normal involution of the tracing, dependent upon the ventricle obstructed. When both are obstructed, the nature of the hypertrophy may be obscured. With mild stenosis (gradient < 30 mm.) the electrocardiogram is expected to be normal; with moderate obstructions (gradient 30 to 70 mm. Hg), the tracing may be questionably abnormal. The electrocardiographic manifestations often fit the pattern of systolic or pressure overloading with a Q wave followed by a high R wave and negative T wave in the precordial lead overlying the hypertrophied ventricle. In a vectorial analysis, in right ventricular hypertrophy, the mean QRS is oriented to the right, anteriorly, and superiorly and the T axis in the opposite direction (as a noteworthy exception sometimes the T axis is in the same direction in young infants manifested as a positive T wave in lead V₁). In left ventricular hypertrophy the QRS is directed to the left, downward, and backward with the T vector characteristically oriented in the opposite direction.

With hypertrophy related to large flows (diastolic or volume overloading) records are usually indicative of the enlargement and hypertrophy, and reflect the hemodynamic state reasonably well. For the right side one commonly sees the rsR in right precordial leads but the degree of hypertrophy of the wall is not accurately portrayed. In left ventricular enlargement, the trend to left axis deviation and prominent voltage of a qR and of an upright T in the left precordial leads are characteristic. Pressure and volume overloading of the ventricles is a useful concept in one’s electrocardiographic approach in congenital heart defects but patterns so delineated will merge one into the other. If one thinks in the frame of reference of wall tension, it may be helpful, in that with an increasing radius with ventricular dilatation, systolic tension will increase for the same intraventricular pressure (LaPlace’s law). This approach may be helpful in the explanation of a pressure overloading type of record in a case of a chronically enlarged (“volume overloaded”) ventricle.

Dr. Kossmann: Dr. Scott, what are your thoughts on the mechanisms involved in complete left bundle-branch block?

Dr. Scott: There is a profound lack of agreement on the mechanisms involved in complete left bundle-branch block. There are several schools of thought and I shall attempt to review these concisely.

One school believes that there is a slow uniform spread of activation across the interventricular septum from right to left. Once the impulse has reached the endocardial surface of the left side of the septum, it regains entrance into the left bundle below the site of block and the free wall of the left ventricle is activated in a normal manner both as to direction and sequence through the Purkinje network. The QRS prolongation is thought to be due to the slow activation of the interventricular septum.

The second group also holds that there is a slow uniform spread of activation across the interventricular septum from right to left. However, the impulse never regains entrance into the left bundle. Instead, the impulse spreads through the septum and medial half of the left ventricular wall by muscle conduction. In the lateral portion of the free wall of the left ventricle, the endocardial surface is thought to be activated by the Purkinje network. These workers believe that the QRS prolongation is due to the anomalous activation both of the septum and of the free left ventricular wall. Smith and associates believe that the delay in activation of the free left ventricular wall is a result of conduction through a lengthened Purkinje pathway.

The third school is that of Medrano and associates. They maintain that there is an electrical partition of the septum and that there is a “barrier” between the right septal mass and the left septal mass. There is a delay...
here of from 0.02 second to 0.06-0.07 second. Once the activation wave has jumped this barrier, there is also a delay in the spread of the impulse from right to left through the left septal mass. These workers have found that once the impulse has reached the left septal surface, the activation of this surface and the free left ventricular wall proceeds through the Purkinje network at a normal rate and in a normal direction. These workers believe the QRS prolongation is due not only primarily to the delay at the "barrier" between the right and left septal masses but also to some delay in spread through the left septal mass itself.

Most workers do agree that in complete left bundle-branch block the first portion of the interventricular septum to be activated is the right septal surface in the lower third in the region of the anterior papillary muscle. The remainder of the right septal surface is then activated in a normal manner from below upward.

**Dr. Kossmann:** What is your concept, Dr. Burchell, of incomplete left bundle-branch block, and what is the anatomic implication or other meaning of the diagnosis?

**Dr. Burchell:** The term, incomplete or partial left bundle-branch block, is one which many of us have used for tracings wherein there is left excitation delay with absence of a Q wave in the left precordial leads and sometimes absence of R in right precordial leads. The appellation introduces a concept that is hard to defend, namely, an incomplete block. In relation to the terminology usually used in atrioventricular conduction: is there truly first-, second-, and third-degree left bundle-branch block? It is possible that first-degree left bundle-branch block exists but tracings purported to show this may be better explained by blocks or delays in branches of the bundle. Certainly second-degree left bundle-branch block occurs and tracings showing alternating normal and left bundle-branch block complexes (2:1 left bundle-branch block) are not uncommon. It is unusual to see tracings suggesting 1° left bundle-branch block alternating with sequences of 3° left bundle-branch block but they do occur. From the classic animal experiments one might conclude that the simple demonstration of an initial R wave in the left ventricular cavity might be adequate evidence of a left bundle-branch conduction defect but the situation is more complicated than such a simple experimental approach could answer. At the present time the term incomplete left bundle-branch block might be shelved and the term intraventricular conduction defect substituted. A considerable number of cases without a Q wave in any extremity lead or left precordial leads will have septal scarring —and this may be suspected when such tracings are seen.

**Dr. Pruitt:** May I introduce one additional observation which suggests that the phenomenon of incomplete bundle-branch block may

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occur? In figure 1 are records obtained with an exploring needle resting on the right bundle-branch in a calf's heart, the left bundle of which had been severed. Frame 1 shows a normal biphasic action potential of the right bundle-branch interposed between P and QRS deflections. Frame 2 shows the action spike from this same electrode separated into two monophasic positive components, the first taller than the second. In frame 3, the initial spike was not followed by the smaller second spike and ventricular excitation also failed to occur. In frame 4, the interval between the two spikes had been reduced and, in frame 5, they were partially fused. Inasmuch as the delay resulting from this slowing in the right bundle ranged from a few milliseconds to 80 milliseconds, similar delays, imposed in the presence of a normally functioning left bundle, would produce a variety of complexes ranging from first-degree to complete right bundle-branch block.

**Dr. Kossmann:** Dr. Pruitt, what is the nature and incidence of intraventricular block other than bundle-branch block?

**Dr. Pruitt:** The answer to this question, even after years of controversy, remains unsettled. A stimulating view is that of Grant, who stated: "It is believed that in the vast majority of cases of marked left axis deviation, a left ventricular parietal block is responsible, resulting from a disturbance of conduction in the anterior division of the left bundle." If Grant's belief is correct, then intraventricular block other than bundle-branch block is indeed a relatively common phenomenon. That a conduction disturbance, as distinguished from a single increase in thickness or dilatation of ventricular wall, plays a role in the development of left axis deviation and other electrocardiographic changes ascribed to left ventricular hypertrophy, is an attractive hypothesis.

But what is the precise nature of this conduction disturbance? Is the postulated aberration in conduction located in the left bundle branch, in one of its two principal subdivisions of that branch, or in the subendocardium itself? Experience with extensive subendocardial lesions produced in canine hearts has led me to take most seriously the synergetic nature of the ventricular myocardium and to regard with skepticism any thesis that ascribes major QRS changes to minor lesions in the parieties of the heart. This is not to deny the possibility that such minor lesions in the bundle branches themselves may influence the QRS profoundly and variably, that variability stemming from the specific measure of delay in transmission produced by these minor lesions.

In figure 2 are illustrated the effects of placing a solution of cocaine in the left ventricular cavity of the isolated perfused canine heart. In each strip of the tracing, the upper sequence of complexes was derived from an exploring electrode placed in the left ventricular cavity, and the lower sequence of complexes from an exploring electrode located on the surface of the lateral wall of this left ventricle. The four electrocardiographic strips formed a continuous record. Between the first and the last deflections in the entire run are included a spectrum of ventricular complexes ranging from normal conduction to complete left bundle-branch block. Unfortunately, these data do not permit identification of the site of the lesion responsible for progressive aberration in left ventricular excitation. The left bundle branch, the divisions thereof, and the subendocardial myocardium were exposed to the traumatizing agent. Within the totality of this left ventricular conduction system resides the potentiality for producing this entire spectrum of aberrant QRS forms. Which component or components played the determining role in this experiment remains an unanswered question. A correct answer would, I believe, resolve a fair share of the confusion regarding the site and significance of bundle-branch lesions in the production of aberrant QRS deflections other than those of complete bundle-branch block.

**Dr. Kossmann:** Would you elaborate further, Dr. Pruitt, on the dilemma of intraventricular block and myocardial infarction, including "peri-infarction" block?

**Dr. Pruitt:** Actually the evidence on cer-
As recording of strip 1 began, 5 ml. of a 5-per cent solution of cocaine were introduced into the left ventricular cavity. Strips 2, 3, and 4 represent an uninterrupted record of the changes in form of complexes from a left ventricular cavity lead (above) and a lead from the anterior surface of the left ventricle (below). Sensitivity, upper complexes: N/15; lower complexes: N/7.5 (From Pruitt, R. D., Essex, H. E., and Burchell, H. B., Circulation 3: 418, 1951.)

tain aspects of this dilemma impresses me as sufficient to justify a reasonably precise conclusion. Large subendocardial infarcts of the lateroposterior wall of the left ventricle produce a distinctive deformity in QRS. In figure 3 are examples of such complexes. Characteristic are the qR deflections in leads II and III and predictably in aV_R, together with the deep and wide S waves in leads I and V_5. The QRS interval is 0.12 second. Such a tracing is atypical either for right or left bundle-branch block and provides evidence of delayed excitation of the posterobasal aspect of the left ventricle. Esophageal leads in such instances may produce supportive evidence of this conclusion. On the left in figure 3 are
shown two ventricular slices, from the heart in this case, viewed from above (i.e., left ventricle on left, anterior surface above). The more apical of the two slices is the upper. A large subendocardial scar had produced thinning of the lateroposterior wall of the left ventricle. A layer of epicardially disposed myocardium remains as does the full thickness of basal ventricular wall.

These findings support the judgment that the characteristic QRS changes of postinfarction block result from an aberrant spread of excitation through preserved myocardium beneath or retrograde to which the subendocardial pathways of rapid transmission have been involved by the infarct. Similar deformities can be produced in experimental procedures on canine hearts wherein the subendocardium has been destroyed.16

This much of the story of intraventricular block of the postinfarction type can be presented with confidence that the supporting evidence is substantial. When infarction involves the anteroseptal myocardium and the QRS interval is prolonged, the late R waves occurring in left precordial leads, then distinction between postinfarction block on the one hand and left bundle-branch block attended by infarction on the other, may become difficult or impossible. In such instances, descriptive terms of evasive character may be justified as, for example, myocardial infarction attended by intraventricular block of the left bundle-branch type.

Dr. Kossmann: How do you, Dr. Scott, differentiate the electrocardiogram of so-called peri-infarction block from the tracings displaying evidence of left bundle-branch block or of simple left ventricular hypertrophy?

Dr. Scott: Grant19 has popularized the term "peri-infarction block" and has listed certain specific criteria for its diagnosis. He has subdivided peri-infarction block into anterolateral and diaphragmatic. We need be concerned here only with anterolateral peri-infarction block. His criteria for this diagnosis are as follows: (1) the initial 0.04-second vector points inferiorly or rightward (away from the anterolateral infarct) pro-

Figure 3

Electrocardiogram in postinfarction block, the lesions involving the lateral and posterior portions of the free wall of the left ventricle.
HYPERTROPHY AND BUNDLE-BRANCH BLOCK

Reducing Q waves in leads aVL (and I) and broad R waves in III; (2) the terminal 0.04 second QRS force points markedly leftward (toward the anterolateral infarct) producing abnormal left axis deviation (LAD); (3) the angle between the initial and terminal 0.04-second forces is 110° or greater; (4) the QRS shows little or no prolongation.

The initial vector abnormality is thought to be due to the loss of electrical activity in the subendocardial layers of the infarcted area. The terminal vector abnormality is attributed to damage to the anterior or superior division of the left bundle so that the excitation must spread over the inferior division upward resulting in LAD of the terminal forces.

In certain cases of suspected peri-infarction block there may be a diagnostically wide angle between the initial and terminal forces, yet the initial forces are not directed sufficiently rightwardly to produce Q waves in I or aVL.

Left ventricular hypertrophy is not uncommonly associated with LAD, which has been attributed to interstitial fibrosis resulting in so-called parietal block. The initial 0.04-second vector is not abnormally directed in parietal block and the angle between the initial and terminal vectors is usually less than 60°. More recently Grant20 has encountered cases of uncomplicated LVH with criteria identical to those of peri-infarction block.

When QRS prolongation develops in anterolateral peri-infarction block, it resembles left bundle-branch block. Grant has stated in his earlier publications that these may be distinguished by the angle between the initial and the terminal 0.04-second forces. In left bundle-branch block the angle is usually less than 45° while in peri-infarction block with QRS prolongation the angle is usually 100 to 110° or greater. Abnormal LAD also is less common in left bundle-branch block than in peri-infarction block. More recently, however, Grant has stated that the criteria for the diagnosis of peri-infarction block are less secure in the presence of QRS prolongation.20

We have applied Grant's criteria to our cases for the past several years and have some interesting observations to report as far as autopsy correlation is concerned.

We can divide our material (with abnormal LAD and wide angle between initial and terminal forces) into cases with normal QRS duration and those with prolonged QRS; cases with normal direction of initial 0.04-second forces and those with abnormal direction of initial forces; cases with necropsy evidence of infarction and cases without infarction.

To summarize briefly, almost all of these cases have had LVH at autopsy. Only about 25 per cent, however, have met the conventional voltage criteria for LVH. Approximately half of our cases have had infarction. Of those with infarction, about two thirds have involved the anterolateral left ventricular wall. Of considerable interest has been the high frequency (80 per cent) of involvement of the interventricular septum by infarction, frequently the anterior portion. In some cases the septum has been involved without anterolateral infarction. This raises the interesting possibility that the superior division of the left bundle was damaged near its origin in the septum rather than more peripherally in the free left ventricular wall.

In our cases with infarction, we have encountered examples both of normal (40 per cent) and of abnormal (60 per cent) direction of the initial 0.04-second vector.

In our cases of peri-infarction-block patterns without infarction at necropsy, we have found a surprisingly high (80 per cent) incidence of abnormally directed initial forces. In almost all of our cases without infarction there has been moderate to marked fibrosis of the left ventricular wall or interventricular septum. We have encountered some cases with septal fibrosis and no demonstrable fibrosis in the free left ventricular wall. This again raises the possibility that the superior division of the left bundle may be damaged in such cases in the region of the anterior portion of the septum rather than more peripherally.

In our experience, on the basis of autopsy-
controlled studies, when the pattern of anterolateral peri-infarction block occurs in the electrocardiogram, there is only about a 50 per cent chance that there is actually an anterolateral myocardial infarction present. Fibrosis of the interventricular septum or free left ventricular wall is frequently encountered in the absence of infarction. Anatomic LVH is almost always present, although it can be diagnosed by conventional voltage criteria in only about 25 per cent of the cases.

**Dr. Kossmann:** Any further remarks on peri-infarction block?

**Dr. Burche]ell:** Dr. Kossmann has implied that the term bundle-branch block is used loosely, and I agree. The term I prefer is intraventricular block in that it is an inclusive one that invites theoretic and experimental work, does not tie one to a priori conclusions, and indeed exposes one's ignorance of the exact disturbance present. Likewise I have preferred the term postinfarction block rather than peri-infarction block, the former being used in its widest temporal sense, meaning a conduction aberration appearing after an infarction. In this area, the work of Van Dam and Durret\(^2\) indicating delays of conduction through an old infarction scar is of pertinent interest.

Postinfarction intraventricular conduction defects need not increase the total duration of QRS beyond normal limits, as many have recognized. Such aberrations may be apparent in scalar leads as the wide splintered Q\(_3\) associated with posterior myocardial scars, often with a wide R (> 0.02 second) of more than 1 mv. in right precordial leads, or as a wide low voltage S wave in left precordial leads, which may be associated with a broad R wave in esophageal leads. A further example is the left axis deviation of the late QRS vectors in the frontal projection—what has been called the R\(_r\)-R\(_s\)-R\(_s\)_ pattern—and which probably constitutes the most common difficult problem in the interpretation of routine electrocardiographic records. The validity of the abnormality is recognized, its occurrence after infarction admitted, its frequent association with hypertrophy recognized, but its exact significance as the only abnormality in a nonsymptomatic older patient is not established. While such tracings are usually stable and permanent, we have observed such abnormalities to occur as a transient phenomenon after exercise (as indeed did Wilson). Also such changes may appear after transventricular surgical approaches to the aortic valve. Aberrations in ventricular excitation of this general pattern have been recently reported in many types of heart disease such as Chagas' myocarditis and endocardial sclerosis.

**Dr. Kossmann:** Our time is up. I would like to thank each of the panelists for sharing with us his own special knowledge and experimental data on the continuing problem of the electrocardiogram in intraventricular block and in ventricular enlargement.

**References**

The Effects of Atmospheric Electricity on Muscular Motion

Having discovered the effects of artificial electricity on muscular contractions which we have thus far explained, there was nothing we would sooner do than to investigate whether atmospheric electricity, as it is called, would afford the same phenomena, or not: whether, for example, by employing the same devices, the passage of lightning, as of sparks, would excite muscular contractions.

Therefore we erected, in the fresh air, in a lofty part of the house, a long and suitable conductor, namely an iron wire, and insulated it, and to it, when a storm arose in the sky, attached by their nerves either prepared frogs, or prepared legs of warm animals.

The thing went according to our desire, just as in artificial electricity; for as often as the lightning broke out, at the same moment of time all the muscles fell into violent and multiple contractions, so that, just as the splendor and flash of the lightning are wont, so the muscular motions and contractions of those animals preceded the thunders, and, as it were, warned of them.—LUIGI GALVANI. *Commentary on the Effect of Electricity on Muscular Motion*. Translated by ROBERT MONTRAVILLE GREEN, M.D. Cambridge, Massachusetts, Elizabeth Licht, Publisher, 1953, p. 36.
The Electrocardiogram in Ventricular Hypertrophy and Bundle-Branch Block: A Panel Discussion
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