Alternating and Co-existing Block in the Divisions of the Left Bundle Branch*

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The tracings of two patients with acute myocardial infarction showing complete right bundle branch block and alternating block in the divisions of the left branch are presented. Both developed a type II (Mobitz) block which was attributed to a simultaneous interruption of conduction in the three main intraventricular tracts (trifascicular block). Left divisional blocks produced specific changes in the initial QRS vectors. Septal activation was considered to be a function of both divisions, rather than of the left bundle branch, a view departing from conventional electrocardiographic theory.

Histopathologic studies of the intraventricular conduction system have shown that the left branch usually subdivides into two major groups of fibers soon after its emergence on the left septal surface. According to some authors, interruption of these subdivisions produces specific electrocardiographic abnormalities in the human heart. The clinical diagnosis of block in the anterosuperior division of the left branch (BSDBL) was first suggested by Wilson et al in 1934. More recently, Grant, Pryor and Blount, Rosenbaum et al, and others have described in more detail the electrovectorcardiographic changes attributed to this conduction disturbance. In contrast, there have been very few reports dealing with the criteria required to diagnose blocks in the posteroinferior division of the left branch (BIDLB). Doubts concerning the existence of left divisional blocks as specific entities arose from the well-known experimental difficulties in producing the expected vectorial changes after section of these structures. This contrasts with the ease with which block in the trunk of the left branch (LBBB) can be produced in dogs. Recently, Watt et al have shown that the apparent inconsistencies between man and dogs are species-related and that the clinical and experimental results coincide if primates are used instead of other animals.

The purpose of this report is to present two cases in which BSDBL and BIDLB alternated and coexisted with complete right bundle branch block (CRBBB). It is postulated that complete A-V block was probably related to the simultaneous occurrence of BSDBL, BIDLB and RBBB, ("trifascicular" block).

Criteria

The following criteria were used in the diagnosis of blocks in the divisions of the left branch:

Block in the Superior Division of the Left Branch (BSDBL)

a) Electrical axis between -30° and -90°
b) Inferiorly oriented initial vectors
c) Counterclockwise rotation of the frontal plane QRS loop
d) QRS duration within normal range, or slightly prolonged
e) The presence of anterior wall myocardial infarction and of RBBB does not interfere with this diagnosis
f) Exclusion of other causes of abnormal left axis deviation (ALAD) such as inferior wall myocardial infarction, pulmonary emphysema, Wolff-Parkinson-White syndrome, right ventricular apical pacing and hyperkalemia.

Since clinical, radiologic or pathologic information is not usually required to diagnose these other causes of ALAD it is evident that BSDBL can be suggested by the electrocardiogram alone.

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Block in the Inferior Division of the Left Bundle (BIDLB)\textsuperscript{1,11,13,15-17}

a) Electrical axis between $+80^\circ$ and $+120^\circ$

b) Terminal vectors to the right, but not necessarily anteriorly

c) CW rotation of frontal plane QRS loop

d) The presence of inferior wall or anteroseptal myocardial infarction and of RBBB does not interfere with this diagnosis.

e) Exclusion of other factors which can produce similarly oriented vectors: 1) extensive lateral myocardial infarction; 2) right ventricular hypertrophy; 3) pulmonary diseases; 4) extremely vertical hearts.

The last three conditions might require additional clinical, radiologic or pathologic data to establish the correct diagnosis. Hence, in contrast with what occurs in the presence of BSDLB, the electrocardiogram by itself does not offer enough information for the diagnosis of BIDLB.

CASE REPORTS

CASE 1

Figures 1–3 were obtained from a 62-year-old patient with acute myocardial infarction. The control electrocardiogram recorded one month before admission is presented in Figure 1. It shows uncomplicated CRBBB with an $hQRS$ close to $+30^\circ$. One day after an episode of severe substernal pain (Fig 2), there is unquestionable evidence of acute anteroseptal myocardial infarction associated with CRBBB. Wide Q waves and abnormal ST segments are now present in leads V\textsubscript{1}–V\textsubscript{3}. The electrical axis points to $-30^\circ$, since lead II shows diphasic QRS complexes associated with predominant negative deflections in leads III and aVF. Inferiorly oriented initial vectors were seen in the frontal plane vectorcardiogram (not shown). This finding correlated well with the small r in aVF. Therefore, the left axis shift was attributed to BSDLB and not to extensive inferior wall necrosis. Variations of intraventricular conduction, always accompanying CRBBB, occur in Figure 3. At times these changes appeared during alternation of sinus and ectopic atrial beats. The first QRS complex (S) in leads I, II, III and aVF shows abnormal left axis deviation ($-40^\circ$) due to BSDLB. In contrast, the electrical axis of the second ventricular deflection in the same leads (I) has shifted to $+110^\circ$ showing all the characteristics of the so-called rare\textsuperscript{18} or classic\textsuperscript{19} type of CRBBB. Moreover, an inferior wall infarction can now be diagnosed from these beats. Right axis deviation was attributed to the presence of BIDLB. Changes in the maximal deflections described in the standard and unipolar extremity leads are due to block in the divisions of the left branch. They did not masquerade either the CRBBB or the anteroseptal myocardial infarction (lead V\textsubscript{1}). Intermittent complete A-V block was recorded from the bedside monitor several hours later (Fig 3, lower strip). This arrhythmia, which followed a premature atrial contraction, disappeared transiently toward the end of the tracing. In other moments, classic Mobitz II block appeared before insertion of a temporary pacemaker. If the patient had CRBBB co-existing with intermittent BSDLB and BIDLB, it is tempting to assume that the A-V block was really due to...

**FIGURE 1.** (Case 1) Control tracing showing complete right bundle branch block (CRBBB) with normal axis.

**FIGURE 2.** (Case 2) Simultaneous, co-existing CRBBB and BSDLB after an acute myocardial infarction.

**FIGURE 3.** CRBBB and BSDLB alternating with CRBBB and BIDLB (top row) A-V block, presumably due to simultaneous co-existing CRBBB, BSDLB, and BIDLB ("trifascicular" block) appears in the bottom strip.
an unusual form of bilateral bundle branch block resulting from the simultaneous interruption of the right branch, and of both divisions of the left branch ("trifascicular block").

**CASE 2**

The electrocardiograms shown in Figures 4-6 were obtained from a 70-year-old patient admitted with the diagnosis of acute myocardial infarction. The first tracing (Fig 4) shows sinus rhythm with an AQRS of $+35^\circ$. The P-R interval measures 0.16 sec. There is an overall tendency toward low voltage. Note the presence of small $q$ waves in leads II, III and aVF. The classic image of recent anterior wall infarction appears in the chest leads. The AQRS is close to $-30^\circ$ in the tracing taken one day later (Fig 5). An initial $r$ wave is now seen in leads II, III and aVF. The QRS changes are suggestive of BSDLB. In addition, the wide S wave in the latter leads and the $R'$ in aVR and right chest leads suggests RBBB. There has been further evolution of the infarction. The P-R interval still measures 0.16 seconds. The tracing taken one day later (48 hours after admission) shows a change in the axis (Fig 6). The AQRS now points to $+115^\circ$. At this moment, right axis deviation occurring in the absence of pulmonary disease can be attributed to BIDLB. A $q$ wave has now appeared in leads II, III and aVF. RBBB is still present. A few hours later, the patient developed advanced A-V block and Adams-Stokes attacks. Non-conducted P waves were not preceded by previous P-R prolongation indicating that the block was of the Mobitz II type. The presence of RBBB and alternating BSDLB and BIDLB suggests that the impedance to A-V conduction resulted from a simultaneous involvement of the three main intraventricular tracts (trifascicular blocks).

**DISCUSSION**

In Figures 1-6, the changes appeared from day to day and even from beat to beat indicating that they were not due to positional or pulmonary factors, but to variations of intraventricular conduction of cardiac origin. Pericardial effusion (which is a cause of alternating QRS complexes) was not present in either patient. There was no evidence of digitalis toxicity or electrolyte imbalance.

According to Sodi Pallares et al.\textsuperscript{20} the first part of the ventricles to be activated is the middle part of the left septal surface. Thereafter, the stimulus proceeds from left to right in an anterior direction.
These authors postulated that the orientation of the early QRS vectors depended on the integrity of the left bundle branch. This concept is accepted by the majority of electrophysiologists. If the left branch is blocked, septal activation will be reversed, since in this instance, the right side of the septum will be activated before the left. In contrast, normal depolarization of the left ventricular wall results from the simultaneous spread of excitation through both divisions of the left branch. The electrical axis is a consequence of the interplay of propagation through both divisions and depends on the integrity of these structures. Of course, certain extracardiac conditions such as abdominal problems, lung pathology and pericardial effusion20 can also change the QRS axis.

In contrast to Sodi Pallares' classic views, Rosenbaum et al11 believe that septal depolarization is not a function of the left bundle branch, but of its divisions. In fact Scher21 pointed out that activation reached two sites of the left septal surface almost simultaneously, rather than one single area, as postulated by Sodi Pallares et al. Accordingly to Scher, these sites corresponded to the distal portions of the superior and inferior divisions of the left branch. The normal left-to-right vector representing activation of the septum would therefore be the result of two vectors, one pointing posteriorly and to the right and the other, oriented anteriorly and to the left.47 For instance, in non-complicated BSDLB, excitation will proceed through the intact inferior division in a posteroinferior and slightly rightwards direction. The initial vectors will have a similar orientation. An r wave will be recorded in the inferior leads (Fig 2, 3, and 5) whereas, a negative deflection will be seen in V6 and (high) V2.

In the presence of BIDLB, excitation proceeds through the intact superior division in an anterosuperior (Fig 3 and 6) and slightly leftward direction. Abnormalities of the initial vectors in the standard leads (frontal plane) due to block in the divisions of the left branch are well illustrated in Figures 2, 3, 5 and 6. However, changes in the chest leads are more difficult to analyze due to the presence of septal infarction, a process which by itself can re-orient the initial vectors in their anteroposterior orientation.18

Rosenbaum et al11 also found that the appearance of BSDLB did not change septal activation when the electrocardiographic position of the heart was horizontal. In vertical hearts with absent q waves in leads I and aVL, BSDLB produced an initial negative deflection in these leads. A reverse phenomenon occurred after BIDLB. Variations in the direction of the initial vectors were seen in horizontal hearts but not in patients with a vertical position.

The causes of the infrequency with which BIDLB is diagnosed have been analyzed by Rosenbaum et al.11 They emphasized that until recently, there had been serious doubt as to whether blocks in the subdivisions of the left branch could produce specific electrocardiographic abnormalities. Proof of the anatomic existence of divisional blocks was first presented by Tawara in 1906.2 Later, other investigators tried to section these structures, but consistently failed to produce characteristic changes in canine hearts.11 Wilson and co-workers,29 among others, were confronted with this problem. Recently, Watt et al4 showed that the predicted ab-
normalities would occur if the baboon was used instead of the dog.

Anatomic studies have revealed certain differences between both divisions. The superior is usually (although not invariably) longer and narrower in cattle, dogs, and pigs.11 Rosenbaum et al quoted the following average measurements for the human heart: superior division, length 35 mm and width 3 mm; inferior division, length 30 mm and width 6 mm. In general, these figures indicate that smaller lesions are required to block the former than to interrupt the inferior division.

The greater vulnerability of the superior division can also be attributed to its blood supply. The latter depends mainly on the perforating vessels emerging from the anterior descending branch of the left coronary artery.12,24-27 On the other hand, the inferior division receives blood from both major coronary vessels, through the perforating branches of the anterior and posterior descending arteries.12,24-28 The frequency with which CRBBB co-exists with BSDLB is understandable since the blood supply to these structures depends on vessels arising from the same [anterior descending] artery.

Finally, other factors also account for the relatively higher frequency of BSDLB. Because of its early origin and its location in the inflow tract of the left ventricle, the inferior division is more protected from certain processes which frequently involve the outflow tract of left ventricle.11 On the other hand, the superior division is more readily affected in the presence of sclerosis of the left side of the cardiac skeleton29 and by localized areas of fibrosis produced by hemodynamic factors secondary to aortic valvular disease or arterial hypertension.11 Moreover, studies are necessary to evaluate the extent of damage to the divisions of the left branch produced by the peculiar types of cardiomyopathy described by Lenegre and Moreau.30

Fernandez et al31 studied the variations of intraventricular conduction appearing during coronary angiography. They observed, among other findings, that right axis deviation appeared after right coronary injections, whereas left coronary injections produced left axis deviation. These changes were attributed to transient block in the divisions of the left branch. The functional importance of these conduction defects is of such magnitude that some authors have suggested that it is more proper to think in terms of three, rather than of two, bundle branches.11,32 The image of “complete” left bundle branch block can result from the simultaneous occurrence of BSDLB and BIDLB.11 Bilateral bundle branch block is a recognized cause of complete A-V block,38 but the latter will also occur whenever CRBBB co-exists with a simultaneous block in both divisions of the left branch.11 CRBBB with BSDLB is a recognized form of incomplete bilateral bundle branch block. The clinical importance of this double conduction disturbance was stressed by Lasser et al14 who studied the patterns of intraventricular conduction in patients with documented established or intermittent complete A-V block. Records obtained prior to, after recovery from, or during advanced A-V block at one time or another revealed the association of CRBBB and BSDLB in 59 percent of the cases studied.

Tracings from patients with left divisional blocks co-existing with CRBBB which developed A-V block presumably due to simultaneous interruption of these three anatomic structures (“trifascicular” blocks)11 have been presented by Rosenbaum et al,11 Katz et al,35 Dressler,36 Winterberg,37 and Grassberger38 (Table 1). These reports represent the clinical counterpart of the experimental work first attempted by Rothberger and Winterberg in 1917.39 The latter authors were able to produce complete (“trifascicular”) A-V block in dogs after sectioning the conducting tracts in the following order: a) the right branch, superior division and inferior division; superior division, anterior division and right branch. The importance of these observations is enhanced by the fact that the exact electrocardiographic characteristics of bundle branch blocks were not known at that time.39

The findings in Fig 1-6 suggest that left divisional blocks can indeed occur in acute myocardial infarction. Prospective studies by means of continuous electrocardiographic monitoring of patients in coronary care units will help in assessing the frequency and importance of “trifascicular” blocks in the genesis of A-V block occurring during the early stages of this disease.

CRITIQUE OF NOMENCLATURE

Many terms have been applied to the conduction disturbances resulting from block in the divisions of the left branch. It is interesting to note that Grant,8,10 who was the first to emphasize the electrocardiographic diagnosis and significance of BSDLB, considered that “peri-infarction” block was the most appropriate term for this conduction disturbance. However, more recent studies suggested that not all patients with BSDLB have an underlying myocardial infarction.40,41 Moreover, Grant used “peri-infarction” block in a different context than the authors who first described the latter.42-44 Wilson et al, and First et al, conceived “peri-infarction” block as a conduction disturbance not related to a lesion in the bundle branches or their divi-
vious.12-14 Pryor and Blount1 applied the term "peri-
infarction" block only when they recognized
that left divisional blocks co-existed with myocardial infarction. Otherwise they referred to them as inferior, or superior, "intraventricular" blocks. On the other hand, Watt et al.15,16,17 have performed interesting experimental studies dealing with left divisional blocks, believe that the use of anterior or posterior "arborization" block is more appropriate. The most exhaustive study on this sub-
ject has been made by Rosenbaum et al who published a 742 page text stressing the importance of "hemi" blocks.17

All authors mentioned above accepted, irrespec-
tive of the terminology employed, that the genesis of the electrocardiographic abnormalities under consideration was a block in the divisions of the left branch. It is suggested that concepts are more important than semantics, so that it does not matter what nomenclature is used (although admittedly this may be at times confusing) as long as it is understood that the basic mechanism of the patterns stressed in this report is an interruption of conduction through the divisions of the left branch.

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THE CONTRACTILITY OF THE SMALL PULMONARY ARTERIES

In the lung which has been fixed within the thorax as soon as possible after death by perfusion with formalin-alcohol, there are two extreme forms of small arteries which are linked by all of the transitional forms. Since both contracted and wide open small arteries are found, it may be assumed that the various degrees of contraction had existed prior to fixation, presumably in vivo. There is a possibility that individual parts of the lung are temporarily more weakly or strongly vascularized just as an intermittent functioning is known for the glomeruli of the kidneys. In addition to the contractility of the small arteries, sphincter-like muscle bands at the origin of the precapillaries and arterioles from the small arteries are concerned in the regulation of the flow of blood. The contractility depicted for the arteries of the lung seems to provide an explanation for the vascular blockade which Mauthner and Pick describe in peptone shock and similar conditions, as well as for the dissimlar vascularization of individual parts of the lung to which Schoen refers.

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