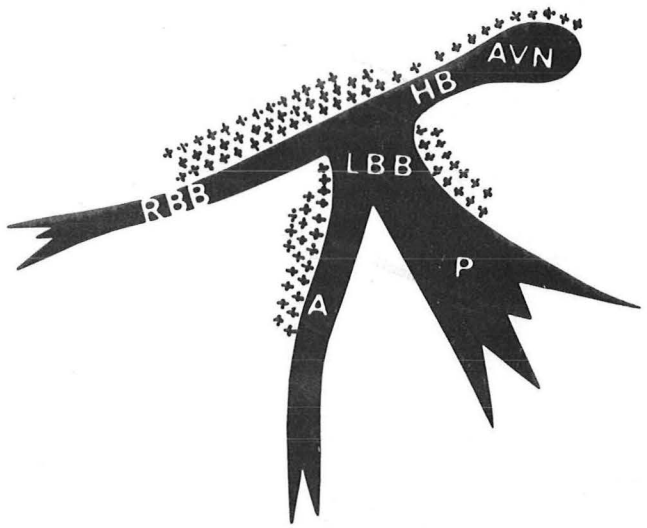
PARKLAND MEMORIAL HOSPITAL

MEDICAL GRAND ROUNDS

FEBRUARY 11, 1971

THE HEMIBLOCKS



NIGHT THOUGHTS

"'Tis as the general pulse
Of life stood still, and Nature made a pause;
An awful pause! prophetic of her end."

Edward Young

CONTENTS

THE HEMIBLOCKS - CLINICAL IMPLICATIONS AND MANAGEMENT

INTRODUCTION

"Every great damage which abides in the solid substance of the heart makes the pulse weaker . . .

"A cessation for the space of one pulsation is of all intermissions the most moderate, for many both of old persons and children have recovered after it, but not one young person. And those whose pulses have stopped for the space of two or more pulsations have infallibly died."

Claudius Galen, 130 A.D.

Disturbances in the conduction system of the heart of minor anatomical nature, can result in major physiological events. New techniques of study of the conduction system, such as His bundle potentials (44, 46), have resulted in a broadening of our knowledge of conduction patterns in the heart and especially have fused previous anatomical studies of the conduction system with pathophysiological events occurring in patients and expressed in the electrocardiogram. The dedication of Mauricio Rosenbaum (1, 2) to the elucidation of the "Hemiblocks" has, in a major way, been responsible for a recent evaluation of conduction disturbances, their clinical implications, and their management with pacemakers. Several clear-cut diseases involving the conduction system have been defined, such as Lev's disease (58-60) and Lenegre's disease (51-57), while others are a result of generalized vascular disease of the heart. Each etiologic agent has a different prognosis and management and will be discussed in detail in this review.

ANATOMY OF THE CONDUCTION SYSTEM

A review of the anatomy of the conduction system and its blood supply is warranted prior to the definition of specific disturbances (15-26).

The intraventricular conduction system begins with the A-V node which is located in proximity to the membranous ventricular system, almost within the right atrium at the junction of the supporting tissue of the mitral valve and just below the posterior cusp of the aortic valve. Thus, the A-V node may be involved with pathology of the aortic valve, mitral valve, atrial, and ventricular septum.

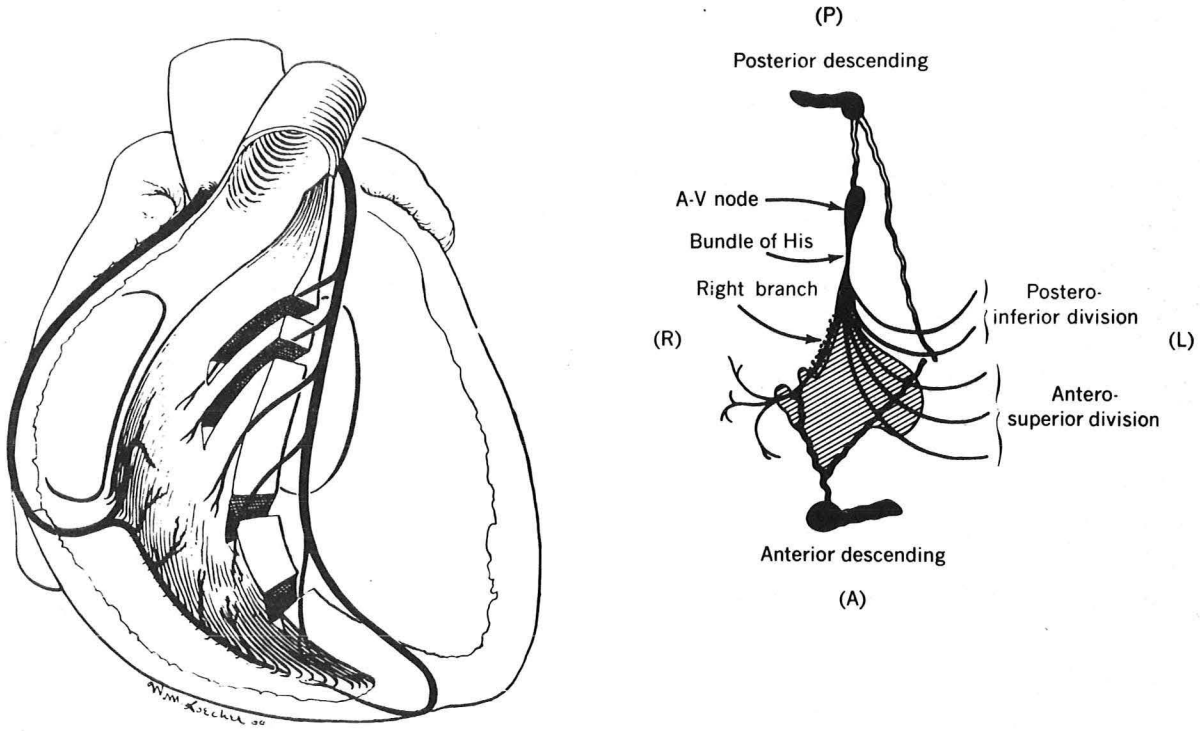
The bundle of His connects the A-V node with the bundle branches. It passes through the central fibrous body and runs close to the membranous septum. It is in proximity to the mitral and tricuspid valvular rings and may be involved in the calcification process of these rings resulting in heart block. The bundle of His branches into a segment which forms the right bundle and into two divisions which form the left bundle. The two divisions which compose the left bundle are the anterior (superior) division, the smaller and more superficial of the two, and the posterior (inferior) division, the larger and more deeply penetrating. The right bundle and the anterior division of the left bundle continue together for a short distance before separating while the posterior division of the left bundle separates early and penetrates the septum rapidly. This is of clinical importance since disruption of the superficial portion of the His bundle may result in simultaneous injury to the right bundle and the anterior division of the left bundle resulting in the EKG pattern of right bundle branch block and left anterior hemiblock (RBBB and LAH)(1). This pattern has been produced experimentally in animals with a lesion produced at the point of common pathway of both the right bundle and the anterior division of the left bundle (39-43; 67-91).

The right bundle traverses beneath the tricuspid valve superficially along the right side of the ventricular septum. It divides into 3 main segments which are anatomically variable, partially explaining the diversity seen electrocardiographically in RBBB patterns. One segment follows the membranous septum while another traverses the muscular ventricular septum, the third segment runs within the moderator band to reach the base of the anterior papillary muscle of the right ventricle. The right bundle is mostly superficial and subjective to injury resulting in delayed conduction or block with right ventricular dilatation, disease processes involving the tricuspid valve, or surgical replacement of the tricuspid valve.

The left bundle is composed of a series of fibers originating from the bundle of His; the first fibers constitute the posterior part of the left bundle and the last fibers form the anterior part. The main left bundle emerges in the subendocardium at the angle formed by the noncoronary and right coronary aortic cusps, where it can be easily damaged by disease processes involving the aortic valve producing "divisional" or main left bundle branch block. The main bundle is short and promptly separates into its two divisions (fascicles), the anterior and posterior. The anterior division traverses obliquely across the outflow tract of the left ventricle (L.V.) towards the base of the anterior papillary muscle. This division is often damaged by L.V. outflow tract lesions, hypertension and aortic valve lesions.

The posterior division of the left bundle (L.B.), the largest segment of the L.B., turns posteriorly and rapidly reaches the base of the posterior papillary muscle and is better protected from injury since it is deep within the septal musculature and traverses the inflow tract of the L.V., a less turbulent area.

BLOOD SUPPLY TO THE CONDUCTION SYSTEM



Figures Illustrating The Blood Supply To The Septum

Left Figure: Right coronary artery on the left supplies the posterior septum. Left anterior descending coronary on the right supplies the anterior septum(24).
Right Figure: Conduction system blood supply.

P = posterior; A = anterior; R = right; L = left; posterior descending = branch of the right coronary artery; anterior descending = branch of the left coronary artery. Note the dual blood supply to the posterior division (fascicle) of the left bundle(22).

A-V node and bundle of His; A-V nodal artery from the right coronary artery (90%)(22, 23).

Right Bundle: 1st segment - (Right) A-V nodal artery
2nd and 3rd segment - (Left) anterior descending coronary artery

Left Bundle: Anterior division - (Left) anterior descending
Posterior division - (Left) anterior descending and (Right) posterior descending coronary arteries.

TRIFASCICULAR NATURE OF THE CONDUCTION SYSTEM

It is apparent that the ventricular conduction system is composed of 3 components; right bundle, left anterior division, and left posterior division. A block in any one of these three fascicles is accompanied by a characteristic change in ventricular activation and in the QRS configuration. Blocks in two of the three fascicles will result in three different combinations of intraventricular block (1, 2, 14). These are found experimentally, as well as clinically (1, 2): Right bundle branch block with left anterior hemiblock; right bundle branch block with left posterior hemiblock; and left bundle branch block. Complete interruption in all three fascicles results in complete heart block.

If conduction is interrupted in one or two of the three fascicles, a new group of electrocardiographic syndromes result, characterized by multiple different patterns of ventricular activation, associated with varying degrees of A-V block and are termed trifascicular blocks (1, 2, 9, 10, 14, 78, 88).

TYPES OF CONDUCTION BLOCKS

1. Bundle Branch Blocks
   a. RBBB
   b. LBBB
2. Segmental (Fascicular) Blocks
   a. Right bundle branch block, several varieties (inconsistent)
   b. Left anterior hemiblock
   c. Left posterior hemiblock
3. Arborization Blocks, Reticular or Purkinjean Blocks within the Purkinje System
4. Parietal Blocks within the ventricular wall

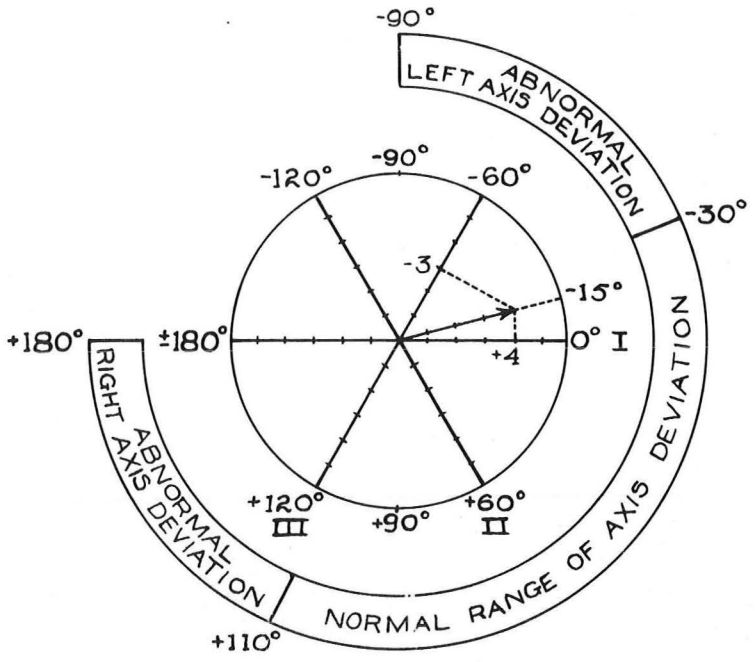
Arborization blocks and parietal blocks are almost always a result of an extensive underlying lesion and, therefore, are usually accompanied with other electrocardiographic findings, i.e., peri-infarction block, rather than simply pure conduction disturbances as may occur with bundle branch blocks and segmental blocks (27-38).

ANATOMIC AND PHYSIOLOGIC VULNERABILITY

	Length	Width
RBB	45-50 mm	1 mm
Anterior LBB	25 mm	3 mm
Posterior LBB	20 mm	6 mm
Main LBB	10 mm	4-10 mm

The anatomical vulnerability to injury is probably related inversely to the width of the fascicle, the narrowest is most vulnerable while the physiological vulnerability (delayed repolarization) is probably related to the length of the fascicle, i.e., the longest is most susceptible to delayed repolarization (1).

Clinical physiological vulnerability, i.e., ventricular aberration of the QRS, does approximate this hypothesis in decreasing frequency of occurrence: RBBB, LAH, LPH and LBBB (1). The clinical anatomical occurrence of block is less confirmatory of this hypothesis of decreasing occurrence: LAH, RBBB, LBBB and LPH (1).



ELECTROCARDIOGRAPHIC DIAGNOSIS OF LEFT ANTERIOR HEMIBLOCK

The three main signs of "pure" left anterior hemiblock are (1, 14):

1) A QRS axis around - 60°

2) A Q1-S3 pattern

3) Normal to slightly prolonged QRS

A more detailed analysis of EKG changes in the standard limb resulting from LAH are listed below (1, 14):

1) LAH shifts from the main QRS forces superiorly and to the left, causing deep S waves in leads II, III and aVF. The depth of these S waves depends, among other factors, on the "degree" of LAH.

2) LAH shifts the first 0.02 sec. QRS forces inferiorly and to the right, to about +120° in the frontal plane. This change is responsible for the occurrence of a small Q wave in lead I and a small R wave in leads II and III. If these initial forces are originally directed toward the right, as happens in many horizontal hearts, LAH will shift them only inferiorly.

3) The changes described in 1) and 2) together are responsible for the generation of a Q1S3 pattern, which simulates counterclockwise rotation of the heart about its longitudinal axis.

4) LAH typically shifts the QRS axis direction to -60°. In some cases, however, the AQRS shift may go no further than -30°.

5) All these changes occur with a QRS widening no greater than 0.02 sec. However, in the presence of myocardial infarction, particularly when the fibers connecting the anterior and posterior left ventricular Purkinje networks are damaged, LAH may be indirectly responsible for a more substantial QRS widening.

6) The most characteristic changes of LAH occur in the limb leads.

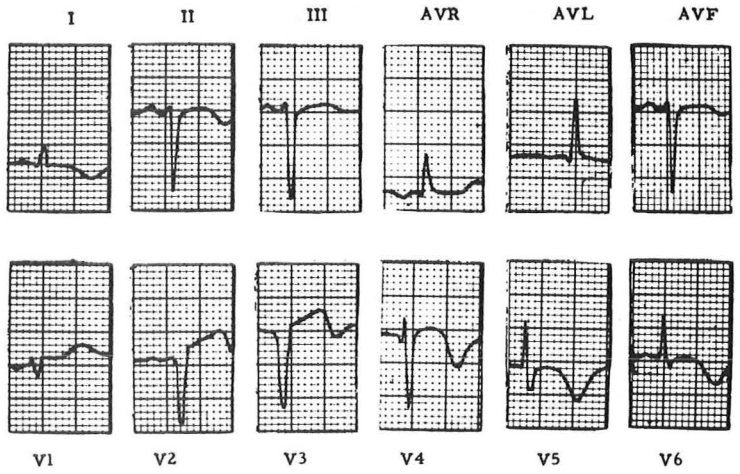
7) LAH may imitate left ventricular hypertrophy or a lateral infarct in aVL. In contradistinction, it may conceal signs of left ventricular hypertrophy or lateral ischemia in the left precordial leads. LAH may also mask signs of inferior ischemia and infarction.

8) LAH produces a typical vectorcardiographic frontal loop with small initial forces directed inferiorly and to the right, main forces oriented superiorly and to the left, and a wide open counterclockwise-rotated loop.

The changes produced in the precordial leads are usually those of:

1) V_1 - V_3 unchanged
2) V_4 - V_6 deep S wave and absence of normal Q waves.

LAD FROM LEFT ANTERIOR HEMIBLOCK



ETIOLOGIES OF LAH

Patients >40 years of age

1) Anteroseptal or anterolateral myocardial infarction
2) Diffuse myocardial fibrosis
3) Anterior coronary artery obstruction
4) Lev's disease (sclerosis of the left cardiac skeleton)
5) Lenegre's disease
6) Hypertension

Patients <40 years of age

1) Aortic valve disease
2) Cardiomyopathies
3) Congenital defect in the conduction system
4) Left ventricular dilatation

It is postulated that LAH (i.e., LAD >-45°) with a few exceptions has a clinical significance similar to those currently ascribed to LBBB, i.e., coronary artery disease, probably a result of myocardial infarction (67-91).

OTHER CAUSES OF LAD

The other major causes of LAD are 1) left ventricular hypertrophy, and 2) anatomically horizontal heart and/or counterclockwise rotation (61-66). However, the axis shift occurring with these conditions rarely reaches -60° (<1.5%)(34). Therefore, if one assumes all axis shifts >-45° are due to LAH, the error will be small (1, 14) excluded, of course, would be the case of complete LBBB and some cases of inferior myocardial infarction. The upper limits of axis shifts with LAH have been found to be -80° (1, 14).

ELECTROCARDIOGRAPHIC DIAGNOSIS OF RBBB WITH LAH

LAH is suggested with RBBB when: (See EKG page 16)

1) QRS axis is between -60° and -120°
2) Q1-S3 pattern
3) RBBB pattern

"In RBBB, the additional presence of LAH should be considered whenever the AQRS is oriented superiorly between -60° and -120°, with a Q1-S3 pattern. The diagnosis is further supported when the first half of the QRS forces points at about -60°, with a very small r/S relationship in lead II. In pure, uncomplicated RBBB with LAH, three main directions of the electrical forces in the frontal plane are recognized. During the first 0.02 sec, the forces point inferiorly and to the right, close to +120°, producing a small Q wave in lead I and a small R wave in II and III. In the next 0.04 sec, the forces are directly superiorly and to the left, at about -60°. These two initial vectors are due to the LAH. The terminal forces, strictly ascribable to the RBBB, point toward the right, around 180°." (1, 14).

ETIOLOGY OF RBBB WITH LAH

See preceding section under "Etiologies of LAH". This is essentially the same.

RBBB with LAH, with a few exceptions, has a clinical significance similar to those currently ascribed to LBBB, i.e., coronary artery disease, usually a result of anterior myocardial infarction, since the left anterior descending coronary artery supplies both the right bundle and the left anterior division within the anterior portion of the ventricular septum.

RBBB with LAH may be followed by complete heart block (trifascicular block), especially if a previous inferior myocardial infarction has occurred since the dual blood supply to the main left bundle and the left posterior division may have been compromized.

ELECTROCARDIOGRAPHIC DIAGNOSIS OF RBBB WITH LEFT POSTERIOR HEMIBLOCK

Primary signs of LPH with RBBB:

1) QRS axis around +120°

2) S1-Q3 pattern

3) Tall R waves in II and III

Provided:

1) No RVH is present

2) No vertical heart or emphysema

3) Forces of first half of QRS are also toward +120°

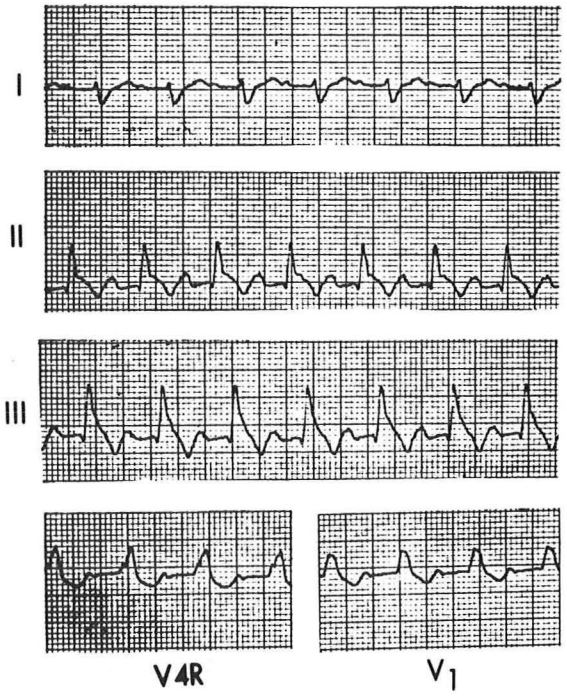
A more detailed analysis of this complex electrocardiographic pattern is given below (1, 14).

"The diagnosis of RBBB with LPH should be considered whenever RBBB is associated with a QRS axis direction around +120° and an S1-Q3 pattern, *provided* right ventricular hypertrophy or a vertical heart can be excluded, and *provided* the forces of the first half of the QRS complex are also directed toward +120°. The diagnosis is strongly supported by tall R waves in leads II and III, and by A-V conduction disturbances."

"In RBBB with LPH, the first 0.02 sec QRS forces are directed at approximately -45° in the frontal plane. These forces, due to the LPH, are caused by early activation of the anterolateral wall of the left ventricle in a superior and leftward direction, and are responsible for a small Q wave in leads II and III and a small R wave in lead I. The middle forces, between 0.02 and 0.08 sec, are directed at approximately +120°. These forces, also due to the LPH, are caused by delayed activation of the posteroinferior wall of the left ventricle in an inferior and slightly rightward direction and are responsible for the first part of the S wave in lead I, and for the R waves in leads II and III. The terminal forces, or at least 0.04 to 0.06 sec, are oriented between +150° and +180°, are caused by the RBBB, and are responsible for the final part of S_1, R_2 and R_3. The two dominant forces, those depending on RBBB (at +150°) and on the LPH (at +120°) are additive, resulting in the tall R waves in leads II and III described earlier."

"RBBB may be accompanied by a QRS axis around +120° and an S1-Q3 pattern under several circumstances: (1) when LPH co-exists; (2) in right ventricular hypertrophy; (3) with a vertical heart due to a slender body build or emphysema; and (4) if the previous QRS lies between +30 and +60°, RBBB may shift the QRS axis direction to about +120°. In this case, however, the forces of the first half of the QRS maintain their original direction and an S1-Q3 pattern is not necessarily present. (5) Occasionally, an extensive lateral infarction may elicit an QRS axis direction of about +120°. In this particular case, the cause of the axis shift is readily apparent, an S1-Q3 pattern is unlikely, and the QRS voltage in the limb leads is characteristically small. This listing explains why the criteria for the diagnosis of RBBB with LPH must be necessarily complex."

POSTERIOR HEMIBLOCK AND RBBB



ETIOLOGY OF RBBB WITH LPH (67-91)

The significance of this EKG pattern revolves around the high incidence of complete heart block developing in these patients. In Rosenbaum's group of 30 patients, 83% developed some form of A-V block with two-thirds developing complete A-V block. Sixty per cent of these patients developed Adams-Stokes seizures.

The resultant complete block in these patients is presumed to be due to the development of block in the anterior division of the left bundle to complete the trifascicular block pattern. The inference fits deductively since (1) LPH is much less common than LAH, (2) RBBB with LPH is less common than RBBB with LAH, and (3) LPH is almost invariably associated with RBBB.

This is anatomically substantiated since the posterior division of the LBB is the least vulnerable segment of the whole intraventricular conduction system. As a consequence, when the lesions are sufficiently extensive to alter conduction in the posterior division, it is almost axiomatic that the RBB, or the anterior division of the LBB, or both, are also involved.

Rosenbaum feels that "RBBB with pronounced 'right axis deviation' may be divided into the following two groups: (1) that which occurs with right ventricular hypertrophy, a slender body build or emphysema; and (2) that which is associated with LPH. The latter constitutes the single QRS pattern most consistently heralding the development of complete heart block."

ELECTROCARDIOGRAPHIC DIAGNOSIS OF LEFT POSTERIOR HEMIBLOCK (LPH)

The electrocardiographic diagnosis of left posterior hemiblock evolved from the analysis of RBBB with LAH and intermittent left posterior hemiblocks and eventually experimental production of LPH (1, 14). In its pure form, LPH is very uncommon probably because of the short wide anatomy of the left posterior division and because of the dual blood supply to the division supplied deep within the septum.

Electrocardiographic criteria for diagnosis are suggested:

1) QRS axis of about +120° (right axis deviation)

2) S1-Q3 pattern

3) Normal QRS duration

Excluded must be:

1) Vertical heart

2) Right ventricular hypertrophy

3) Lateral wall myocardial infarction

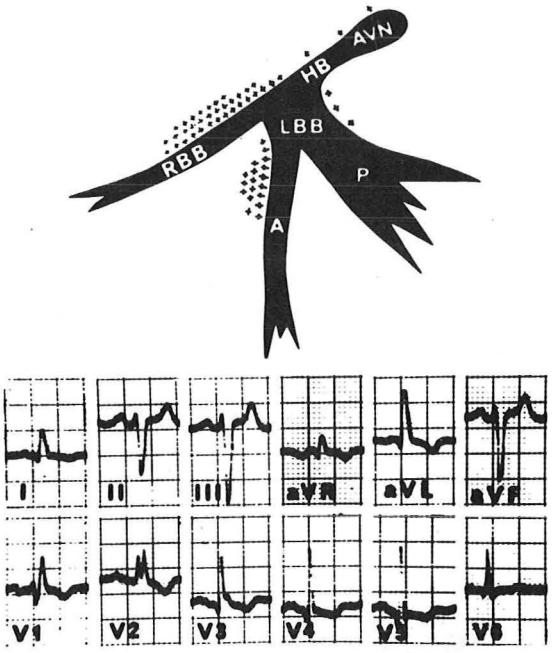
It is evident that LPH is not a pure electrocardiographic diagnosis but must be a clinical-electrocardiographic diagnosis. It occurs but rarely without RBBB and is considered in more detail in that section.

LPH carries a worse prognosis than pure LAH since the posterior division usually is the most "protected" of the fascicles of the bundles and requires rather extensive disease to result in its blockage. This implies that the other fascicles left anterior division and RBB may easily be involved in the same disease process.

LENEGRE'S DISEASE (PRIMARY CONDUCTION SYSTEM DEGENERATION)

Lenegre, in 1964, reported 11 cases of sclerodegenerative disease involving both bundles with no evidence of myocardial disease or coronary artery disease (26). Ten of these eleven patients developed high degrees of heart block. Davis and Harris in 1969 reported an excellent pathological study of similar patients with primary conduction system degeneration and heart blocks (55). Typically, these patients are middle-aged to elderly patients (40-60) who develop high degree of A-V block or Adams-Stokes seizures in the absence of other findings of heart disease. Electrocardiographically, these patients may initially present with RBBB and LAH or pure LAH and gradually progress to trifascicular block. Rarely, a RBBB and LPH pattern can be found (51-57).

LENEGRE'S DISEASE



An example of and EKG from a patient with RBBB and LAH as a result of Lenegre's disease involving the right bundle and anterior division of the left (1).

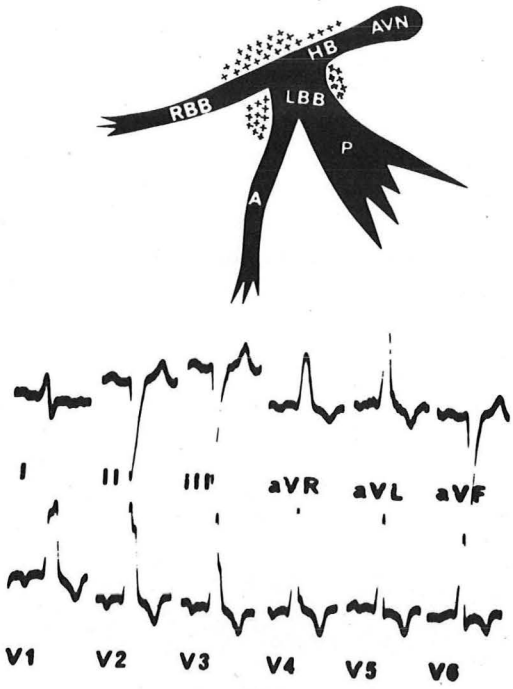
LEV'S DISEASE: "SCLEROSIS OF THE LEFT CARDIAC SKELETON" PRODUCING RBBB WITH LAH

Lev, in 1964, described a process involving the conduction system which may cause conduction disturbances but rarely leads to complete heart block (17, 18).

Lev states: "Normally, with advancing age, there is progressive fibrosis and calcification of the mitral annulus, the central fibrous body, the pars membranacea, the base of the aorta and the summit of the muscular ventricular septum. The maximal location of the degerative changes varies from person to person. We designate this *sclerosis of the left side of the cardiac skeleton*. In my material, the process is first seen at the age of about 40 years. It may be related to the wear and tear of these structures due to the pull of the left ventricular musculature, the mitral and aortic valves and the base of the aorta"(17). This probably represents a mechanical wear and tear phenomenon with age.

This is probably the most common cause of RBBB with LAH seen in elderly people (>60) and is usually not associated with other significant heart disease. Uncommonly, do they develop complete heart block. It is rarely rapidly progressive and rarely involves the left posterior division (58-60).

LEV'S DISEASE



An example of an EKG from a patient with hypertension and no coronary artery disease resulting in RBBB with LAH (1).

THE TRIFASCICULAR BLOCKS

TYPES OF VENTRICULAR CONDUCTION BLOCKS

I. A-V Nodal

II. His Bundle

III. Arborization (Purkinjean) Blocks

IV. Parietal Blocks

V. Segmental (Fascicular)

SEGMENTAL (FASCICULAR) (1, 2, 14)

Several types of fascicular blocks may occur due to the trifascicular nature of the ventricular conduction system. These may be complete or incomplete blocks resulting in a series of electrocardiographic findings (1, 2, 14, 87-91).

Generally, fascicular blocks may be classified as:

1) Monofascicular (A-V nodal or His bundle)

2) Bifascicular (RBBB and LBBB) ("Bilateral BBB")

   a) Complete

   b) Incomplete

3) Trifascicular (RBBB, LAH, LPH)

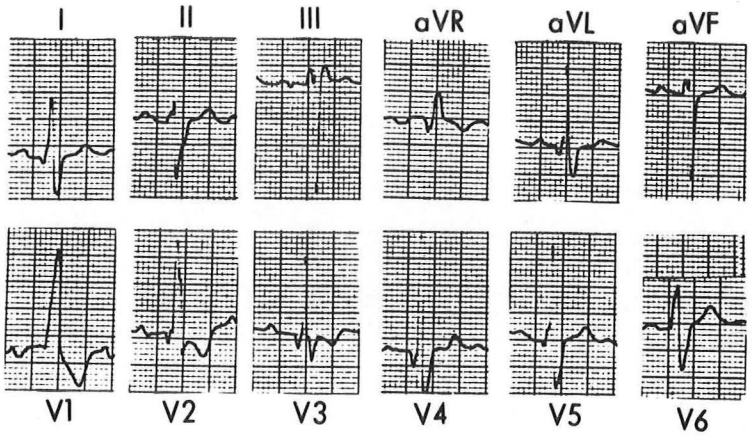
   a) Incomplete

   b) Complete

(These may be intermittent [physiologic] or permanent.)

INCOMPLETE TRIFASCICULAR BLOCK

(RBBB AND LAH AND 1° BLOCK)



This is an example of the most common type of incomplete trifascicular block.

Nine types of complete fascicular blocks for all practical purposes are found (1, 14):

1) Left anterior hemiblock (LAH), or block in the anterior division of the LBB.

2) Left posterior hemiblock (LPH), or block in the posterior division.

3) Divisional left bundle branch block (LBBB), or simultaneous block in both divisions of the LBB.

4) Predivisional LBB, or block in the main LB.

5) Right bundle branch block (RBBB).

6) RBBB with LAH.

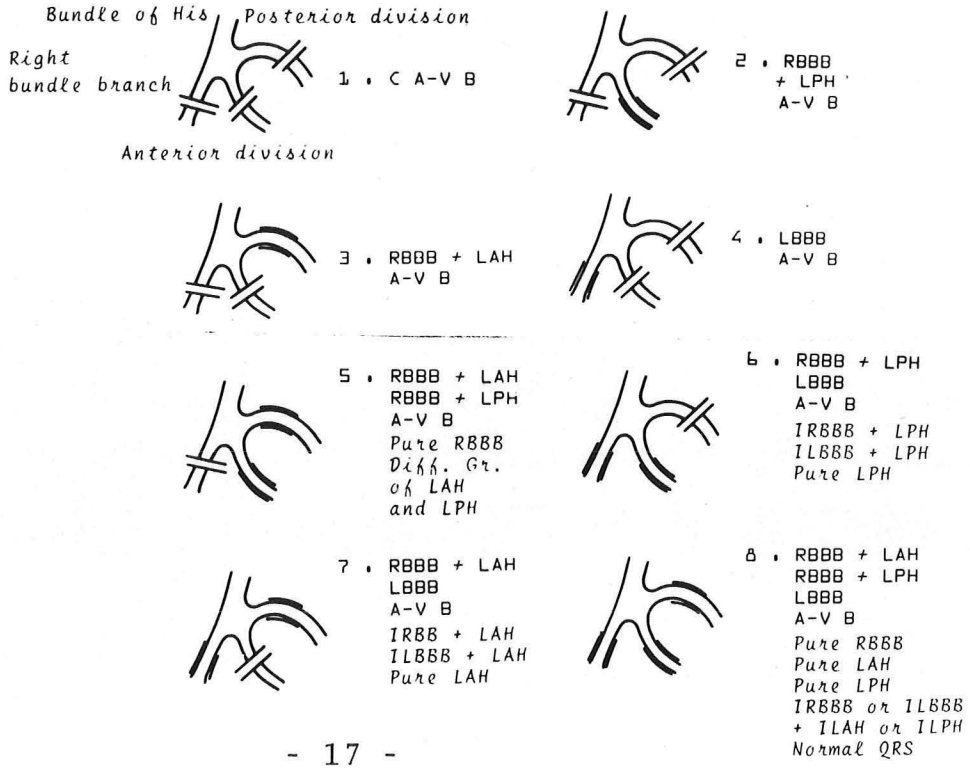
7) RBBB with LPH.

8) Bilateral bundle branch block (BBBB), or simultaneous block in the two main bundle branches.

9) Trifascicular block, or simultaneous block in the RBB and in the two divisions of the LBB.

Incomplete fascicular blocks are diagnostically drawn below (1). Complete blocks are illustrated by perpendicular bars across the fascicle while incomplete or intermittent blocks are illustrated by parallel bars. C= complete; I - incomplete; AVB = complete atrioventricular block; Diff. Gr. = different grades of block. The heavy type letters indicate the more likely occurrences.

TRIFASCICULAR BLOCKS



GENERAL CLINICAL SIGNIFICANCE WITH CONSIDERATIONS FOR PACING IN RBBB WITH LAH

Involvement of both the right bundle branch and the anterior division of the left bundle denotes a reasonable involvement of the conduction system in a disease process. By implication, this means one or more of the following must be present:

1) Major degenerative disease of the conduction system (Lenegre's or Lev's disease).

2) At least one major coronary vessel disease (LAD), or more.

3) Generalized cardiomyopathy as with alcoholic or chagasic myocarditis.

Most patients with this EKG pattern over 50 years of age have coronary artery disease as the etiology for this conduction defect.

Those patients without known heart disease and 60-65 years of age or older may be considered to have Lev's disease with a predictably benign course. The majority of the remainder of the patients, however, are subject to progressive disease resulting in a propensity to develop higher grades of heart block.

The development of A-V conduction disturbances in patients with RBBB and LAH has been reported to be as high as 16% (2). Complete heart block develops in 5-10% of these patients; some followed for as long as 10 years (2, 4, 35).

On the other hand, it has been shown that chronic complete heart block is preceded by RBBB and LAH in 40-51% of the cases (35, 78).

As might be expected, the majority of those patients developing or passing through a form of second degree block develop a Mobitz Type II block (79, 80, 81).

The new technique of His bundle potential recordings in man may well differentiate the true trifascicular nature of these blocks (44, 45, 46).

The indication for permanent pacing is obvious in patients with RBBB and LAH with an associated A-V block unrelated to drug therapy, or in the setting of symptoms such as dizzy spells or Adams-Stokes seizures (1).

In those patients without symptoms or signs of A-V block other than RBBB and LAD, the indications for pacing are not defined, especially in those patients presenting without evidence of a recent myocardial infarction (1, 92-100).

Predictably, 5-10% of these patients will eventually develop complete heart block, but this may be as long as 10-15 years after onset of the conduction defect. A few guidelines may be established for permanent pacing in patients with RBBB and LAH (1, 92-100).

INDICATIONS FOR PACING IN RBBB AND LAH

1) Symptoms of Stokes-Adams attack

2) Patients <60 years of age (Lenegre's disease)

3) Signs of trifascicular involvement

   a) ↑P-R

   b) Mobitz Type II block or Wenckebach

   c) Third degree block

   d) Intermittent LPH

4) Development with an acute anterior myocardial infarction

5) Chronic RBBB and LAH, plus recent inferior myocardial infarction

Additional procedures may be helpful in chronic RBBB and LAH to elucidate the possibility of imminent trifascicular block:

1) His bundle recordings

2) Atrial pacing at rapid rates to uncover bundle fatigue

In congenital heart disease, RBBB and LAH are common in endocardial cushion defects (47-50). This is not surprising since the conduction system is juxtaposition to the defect in development. The prognosis for block here does not warrant pacing, except perhaps temporarily during surgical repair.

GENERAL CLINICAL SIGNIFICANCE WITH CONSIDERATIONS FOR PACING IN RBBB WITH LPH

Involvement of both the right bundle and the large posterior division of the left bundle by a disease process means a large segment of the conduction system must be involved. This implies that one or more of the following must be present:

1) At least 2 coronary vessel disease

2) Diffuse septal disease

3) Lesion of the main left bundle or bundle of His

4) Generalized cardiomyopathy

5) Primary conduction system disease (Lenegre's disease)

The etiology of disease resulting in RBBB with LPH is most likely ASHD (28%); however, a larger number of these patients do not have a clear-cut etiology (31%) and by implication may have primary conduction degenerative disease (1, 2). In Argentina, 35% were noted to be due to Chagas disease (1, 2).

Clinically, these patients have a high propensity to develop A-V conduction disturbance (83%) (1, 2, 10) with 66% developing complete heart block and 59% developing Adams-Stokes seizures.

This finding suggests that permanent pacing should be accomplished in all patients with this EKG pattern, at least those that are symptomatic and those over 40 years of age without evidence of cardiomyopathy.

INCIDENCE OF CONDUCTION DISTURBANCES

The incidence of conduction disturbances and their propensity to develop blocks are listed below (1, 4, 9, 10, 42):

INCIDENCE OF CONDUCTION DISTURBANCES

LAH	4.6%
RBBB	3.2%
LBBB	1%
RBBB and LAH	1%
RBBB and LPH	Unusual
LPH	Rare

INCIDENCE OF DEVELOPMENT OF COMPLETE A-V BLOCK

All BBB	5-10%
RBBB and LAH	10-16%
RBBB and LPH	21-75%

CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION

The development of bundle branch block (BBB) in the setting of acute myocardial infarction is associated with a high mortality rate and is a sign of poor prognosis. The incidence of complete bundle branch block in monitored patients with myocardial infarction ranges between 5 and 15% with an average of 10% (87, 107, 94, 6, 96, 42). The mortality associated with these BBB exceeds the mortality from complete heart block in the same group of patients. The mortality from BBB averages 56% with an average mortality in the same group of patients with myocardial infarction and no BBB of 26%. The majority of these deaths is due to pump failure with the minority resulting from fibrillation and even less from asystole (101-107) indicating that pacemakers may be of little value in this group. The development of BBB is often an indicator of massive infarction which probably explains the high mortality figures. The prognosis in terms of longevity in patients with permanent BBB and especially LBBB who recover from their initial insult is guarded and, in fact, worse than those patients that develop permanent heart block. The average longevity of patients with BBB is 4.5 years (105). This prognosis as pointed out by Paul Wood is probably that of the heart disease which produced the conduction defect.

The incidence of RBBB is roughly twice the incidence of LBBB (7% vs 4%) in patients with myocardial infarction with a mortality of about 60% with RBBB and 48% with LBBB; these probably are not statistically significantly different (87, 94).

With the recent interest in hemiblocks associated with myocardial infarction, a few reports have appeared relating mortality to bifascicular and trifascicular blocks which are of interest and will be necessary to determine the indications for pacemakers in this setting (6, 96).

Godman (96) reports 68 of 806 patients monitored with acute myocardial infarction who developed conduction defects:

	Incidence		
	Of Total Group	Of Conduction Defects	% Developing 3° Block
Pure RBBB or LBBB	6%	69%	13%
RBBB + LAH	1.2%	16%	55%
Incomplete Trifascicular Block	1.2%	15%	90%

Prophylactic pacing in his last 31 patients did not significantly change the mortality; however, these were not permanently paced which may have prevented one sudden death, and only 9 patients developed complete block. The over-all mortality in this group was 56%.

Scanlon (6) reported a similar series of 28 patients with the following results:

	Incidence Of Conduction Defects	% Developing 3° Block	Mortality
RBBB + LAH	21%	27%	36%
RBBB + LPH	22%	0	33%

They suggested the use of a pacemaker in these patients.

From these data it is clear that the mortality is high with bifascicular block and a large per cent of these patients develop complete heart block. However, no significant statistics are yet available showing that this mortality can be changed with prophylactic insertion of temporary or permanent pacemakers. This will follow with time and a larger series of patients reported.

PMH PATIENT REVIEW

Considerable interest has been shown during the last few years in conduction disturbances following acute myocardial infarction, and this has been stimulated by the study of arrhythmias during continuous electrocardiographic monitoring, and the treatment of atrioventricular (A-V) block with endocardial pacing. There has been much discussion on the role of pacing in acute infarction, and a number of workers have agreed that all patients with second or third degree A-V block should be paced, or should have a pacing electrode inserted into the right ventricle and attached to a demand pacemaker. Less attention has been paid to the prognostic significance of bundle branch block and its relation to A-V block, although its incidence and natural history have been described.

It has been found that A-V block in posterior infarction often progressed from first to second degree and occasionally to third degree, Stokes-Adams attacks were uncommon, the prognosis from the A-V block itself was good, and when complete heart block occurred, the QRS complexes were usually of normal duration, indicating a high ventricular pacemaker. In anterior infarction, on the other hand, A-V block was usually complete, QRS duration was prolonged, attacks of asystole (Stokes-Adams attacks) were common, the prognosis was poor, and the warning of complete heart block in anterior infarction was not first degree heart block but right bundle branch block (RBBB). It was thought that these findings indicated that it was not necessary to treat most patients with posterior infarction and A-V block with endocardial pacing. It might be advantageous, however, to insert a pacemaker in patients with anterior infarction and RBBB, even if no A-V block was present.

A review of patients in our medical intensive care unit has recently been undertaken by Dr. James Atkins to further elucidate the prognostic role of hemiblock and bundle branch block in patients admitted to that unit and to determine the role of cardiac pacemakers in the management of these problems (100).

The electrocardiograms and charts were reviewed of all patients admitted to the cardiac portion of the medical intensive care unit at Parkland Memorial Hospital from January 1, 1969, to September 15, 1970; and the incidence and complications of conduction defects were compared with those patients without conduction defects (100).

There were 815 cardiac admissions during the 20 months of the study. Four hundred twenty-five (425) of these admissions proved to have acute myocardial infarctions as shown by either diagnostic electrocardiographic changes or suggestive electrocardiographic changes and compatible enzyme rises. Conduction blocks occurred in 77 patients or 18% of patients with acute myocardial infarction.

CONDUCTION BLOCKS IN THE CCU

INCIDENCE OF CONDUCTION BLOCK

Total Admissions: 815 patients

Acute Myocardial Infarction:

425 Patients — 18%

Others:

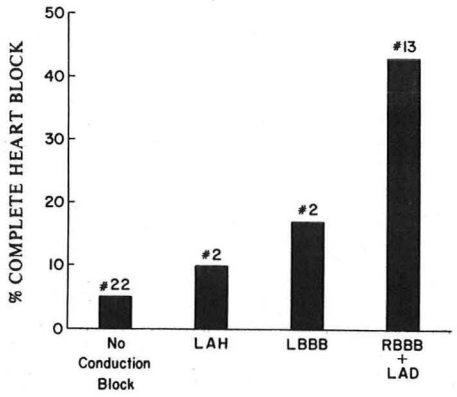
390 Patients — 12%

CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION
(77 PATIENTS)

TYPE OF BLOCK	PATIENTS
Left Anterior Hemiblock (LAH)	20
Left Bundle Branch Block (LBBB)	18
Right Bundle Branch Block-Left Axis (RBBB-LAD)	30
Other Blocks	9

The remaining patients had either right bundle branch block or left posterior hemiblock.

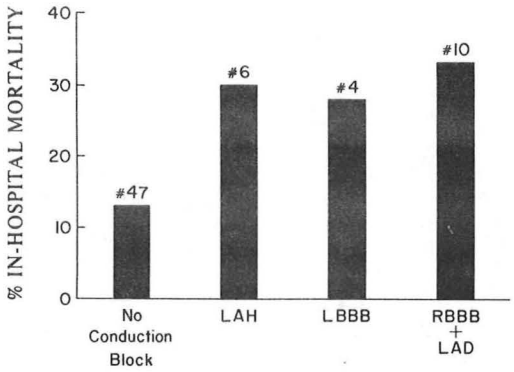
COMPLETE HEART BLOCK IN MYOCARDIAL INFARCTION



Refers to number of patients.

The incidence of advanced heart block with acute myocardial infarction differed with the presence and type of conduction block. Patients with no conduction block had a 5% incidence of heart block, patients with left anterior hemiblock had a 10% incidence, patients with left bundle branch block had a 17% incidence, while patients with right bundle branch block and left axis deviation had a 43% incidence. The incidence of heart block in patients with right bundle branch block and left axis deviation was significantly greater than the others. In the patients with right bundle branch block and left axis deviation, the onset of complete heart block was often sudden without any forewarning. One of the patients in this group expired with the initial onset of heart block despite immediate resuscitative attempts; two more patients survived following immediate fortuitous resuscitation. This high incidence of sudden complete heart block in patients with right bundle branch block and left axis deviation with acute myocardial infarction suggests that prophylactic pacing in this group could prevent sudden death.

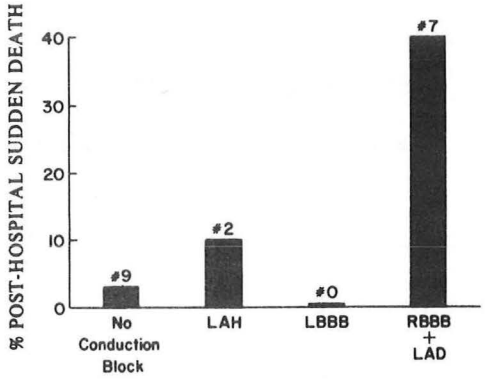
IN-HOSPITAL MORTALITY IN MYOCARDIAL INFARCTION



Refers to number of patients.

The in-hospital mortality of patients with a conduction block and acute myocardial infarction was approximately twice the no block group. The no conduction block group had an in-hospital mortality of 13% while the conduction blocks had approximately a 30% mortality. Of the 10 deaths with right bundle branch block left axis deviation, 7 patients died of pump failure, 1 of sudden complete heart block and 2 more died suddenly of unknown causes. It is evident that in our series the presence of a conduction block carries a significantly greater risk of mortality.

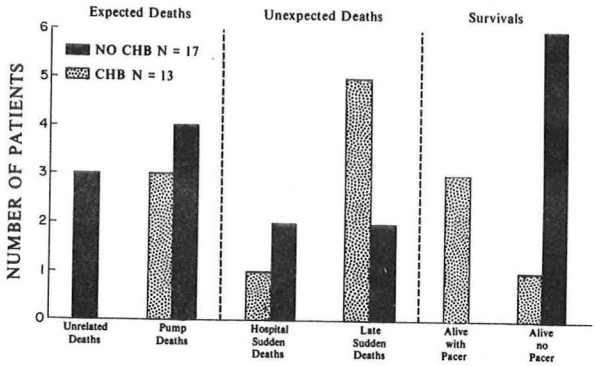
POST-HOSPITAL SUDDEN DEATH AFTER MYOCARDIAL INFARCTION MEAN FOLLOWUP - 6 MONTHS



Refers to number of patients.

A post-hospital follow-up period averaged 6 months. Importantly was that 43% of all deaths occurred in those patients with RBBB and LAD, while only 7% of the total number of patients had this conduction defect. Most of the deaths were sudden deaths suggesting heart block or fibrillation

FOLLOWUP OF RBBB-LAD IN MYOCARDIAL INFARCTION
(30 PATIENTS)



The follow-up of the 30 patients developing RBBB and LAH is of interest. Forty-three per cent (43%) (13) of these patients died in the hospital; seven with pump failure and 3 suddenly and 3 of causes other than cardiac, suggesting that 23% (3) of the deaths (10% of the total with conduction blocks) might have been prevented with a prophylactic pacemaker during hospitalization. The numbers are too few to consider any relationship to heart block in this group.

Of interest is the late sudden death group. Seventeen patients were discharged with RBBB and LAH. Of these, 40% (7) died suddenly and 5 of the 7 had had complete trifascicular block during the initial onset of their infarction with a return to RBBB and LAH. The 3 patients discharged with permanent pacemakers had no deaths, suggesting that permanent pacemakers may be indicated in all patients with RBBB and LAH with transient complete trifascicular (3°) block in association with an acute myocardial infarction. Perhaps all patients with RBBB and LAH due to an acute myocardial infarction should be permanently paced.

SUGGESTED INDICATIONS FOR PACING IN ACUTE M.I. (92-100a)

A. Sinus bradycardia with

   1. CHF

   2. Syncope (↓CNS perfusion)

   3. Angina

   4. Escape ventricular rhythms

B. Overdrive for recurrent tachyarrhythmias

C. Second degree block

1. Mobitz Type II

2. Wenckebach with anterior M.I.

3. ? Wenckebach with inferior M.I.

D. Complete A-V block

E. Incomplete trifascicular block

1. RBBB + LAH + ↑PR

2. RBBB + LPH + ↑PR

3. LBBB + ↑PR

4. Alternating RBBB with LBBB

5. Alternating RBBB with LAH and LPH

F. "Bifascicular" blocks

1. RBBB + LAH with transmural anterior M.I.

2. Chronic RBBB + LAH with acute inferior M.I.

3. RBBB + LPH

G. ? Indications

1. All RBBB + LAH

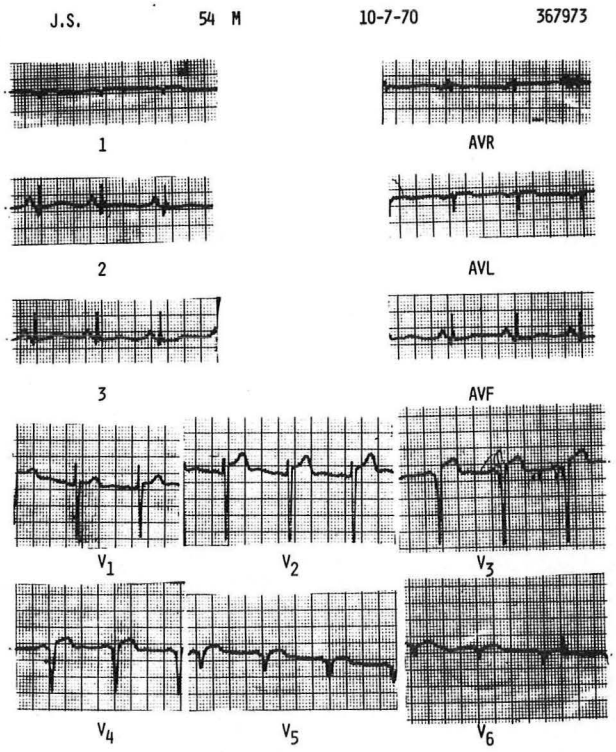
2. RBBB with acute transmural anterior M.I.

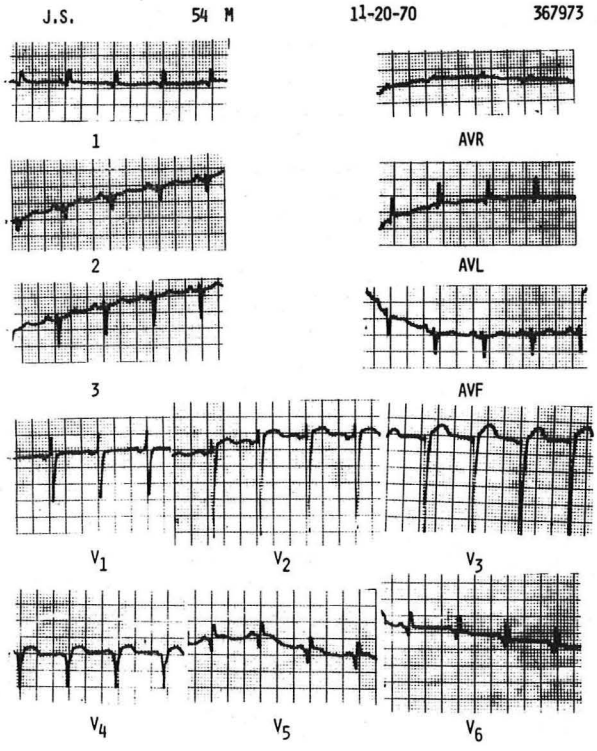
1. Surgical Production of LAH

[redacted] [redacted] 54 y/o Male [redacted]/70

This patient had an anterior myocardial infarction in June of 1969 and an inferior myocardial infarction in [redacted], 1969, with congestive heart failure. He remained in chronic pulmonary edema despite good medical management. His physical findings and EKG suggested a ventricular aneurysm and a cardiac catheterization on [redacted] 1970, revealed a large paradoxical left ventricular aneurysm with a totally occluded LAD coronary artery and an 85% obstruction in the distal right coronary artery. His electrocardiogram at this time suggested an old lateral and inferior wall infarction with right axis deviation due to the old myocardial infarction. He underwent cardiac surgery on [redacted], 1970, with a resection of his ventricular aneurysm and a venous bypass graft to his distal right coronary artery. Immediately postoperatively and in a follow-up visit in the clinic on [redacted] 1970, his EKG revealed a LAH with an axis shift from his previous right axis to left axis deviation.

This represents a left anterior hemiblock developing as a result of left ventricular surgery and probable damage to the left anterior division of the left bundle.



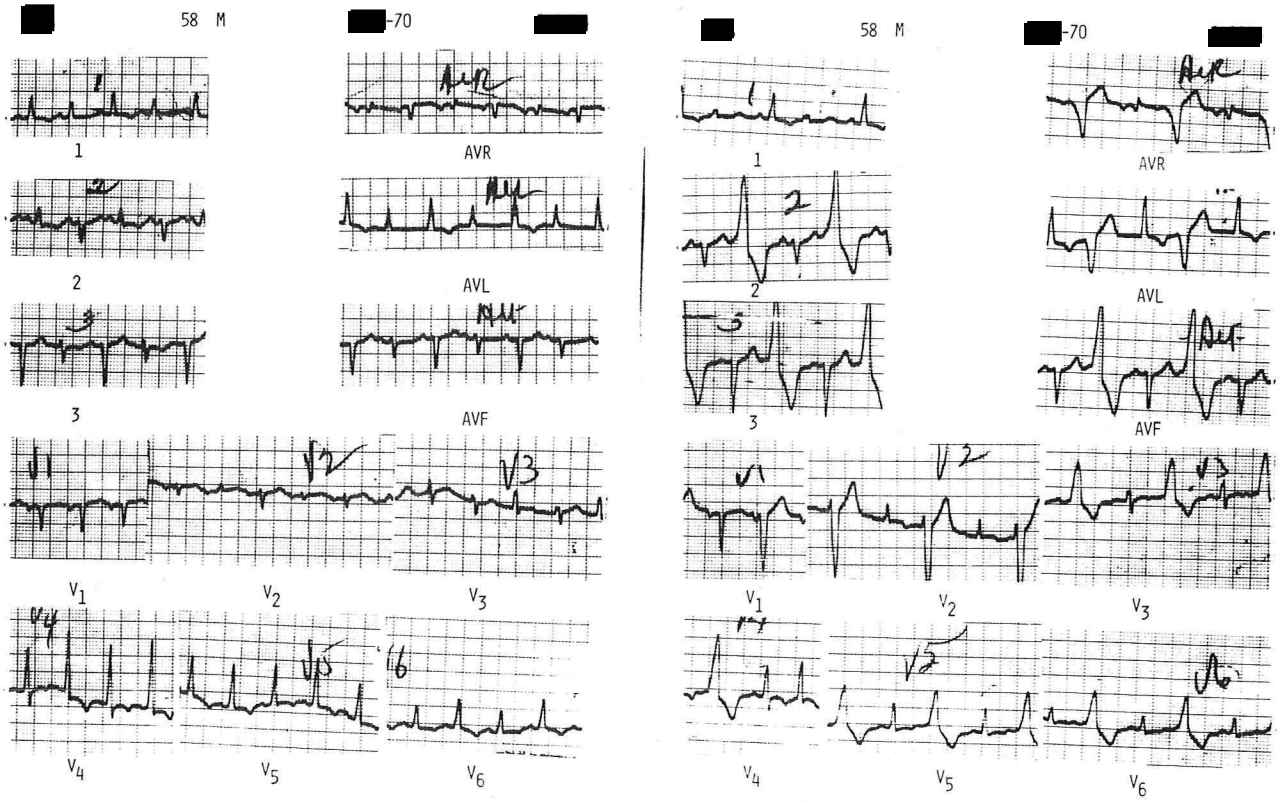


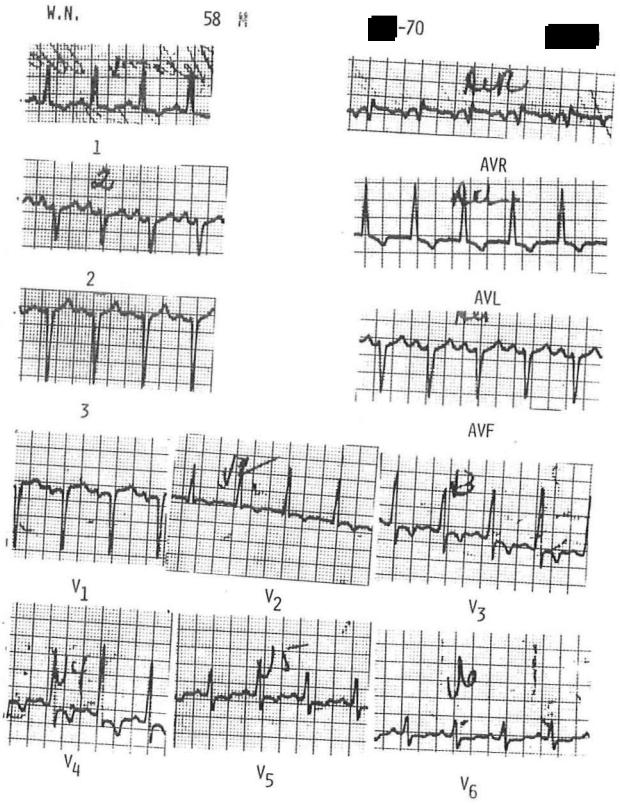
2. Intermittent LAH to Permanent LAH

[redacted] [redacted] [redacted] 58 y/o Male [redacted]/70

Alcoholic patient with a 2 month history of increasing CHF with progressive DOE and PND followed by peripheral edema. He was admitted in pulmonary edema with anasarca and responded to the usual management for CHF. SGOT's were 52 and 37 on the first 2 consecutive days of admission. His EKG's show alternating LAH on [redacted]/70; LAH with bigeminy on [redacted]/70 and anterolateral ischemia; LAH with anterolateral ischemia on [redacted]/70. His current EKG still shows permanent LAH with no ischemic changes.

This is an example of anterior wall ischemia and probable infarction resulting in LAH.



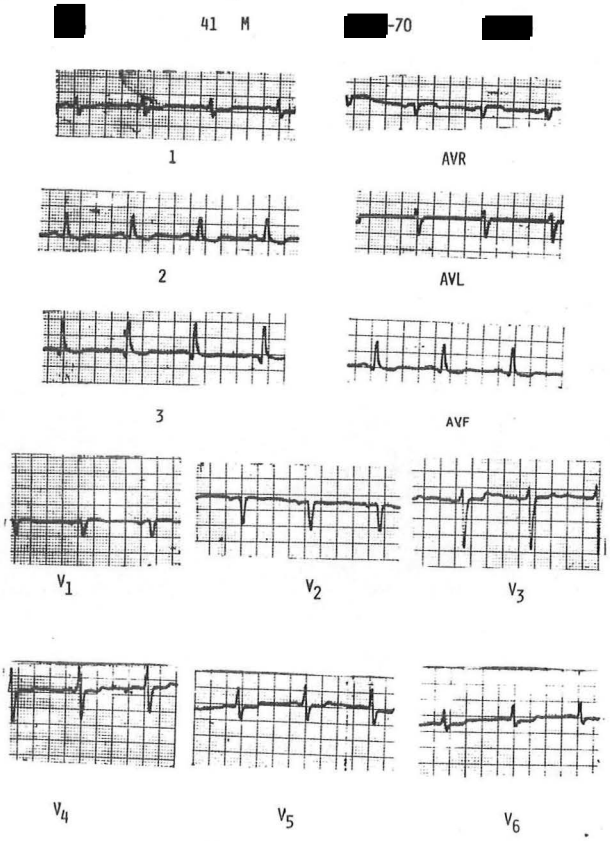


3. LPH

[redacted] [redacted] 41 y/o Male [redacted]/70

The patient had two previous inferior myocardial infarctions in 1967 and 1968. He had severe postinfarction angina and coronary angiograms revealed severe three vessel disease. He underwent venous bypass grafting 18 months ago without difficulty. He has had no changes in his EKG since his previous myocardial infarction which shows right axis deviation.

This is an example of left posterior hemiblock resulting from ASHD.

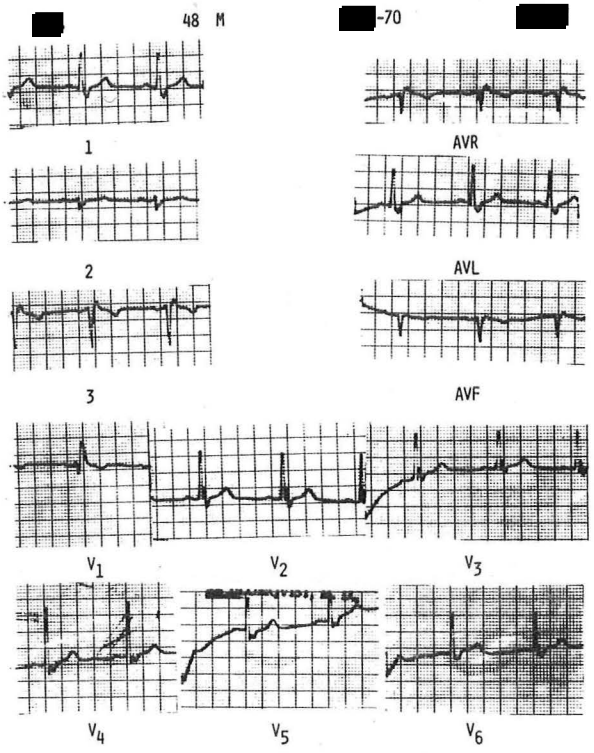


4. Long Standing RBBB with LAH

[redacted] [redacted] 48 y/o Male [redacted]/70

This patient had hypertension (150/105 average) documented since 1962 and an EKG revealing RBBB and LAH. The patient complained of nonspecific chest pain but was treated for angina. A graded exercise test was accomplished with a good workload and no evidence of ischemia or angina. Cardiac catheterization was accomplished which revealed normal large coronary arteries and a dilated aortic root thought possibly to be due to cystic medial necrosis of the aorta.

This is an example of a patient with prolonged RBBB and LAH not associated with coronary artery disease and possibly secondary to fibrosis of the conduction system from prolonged hypertension.

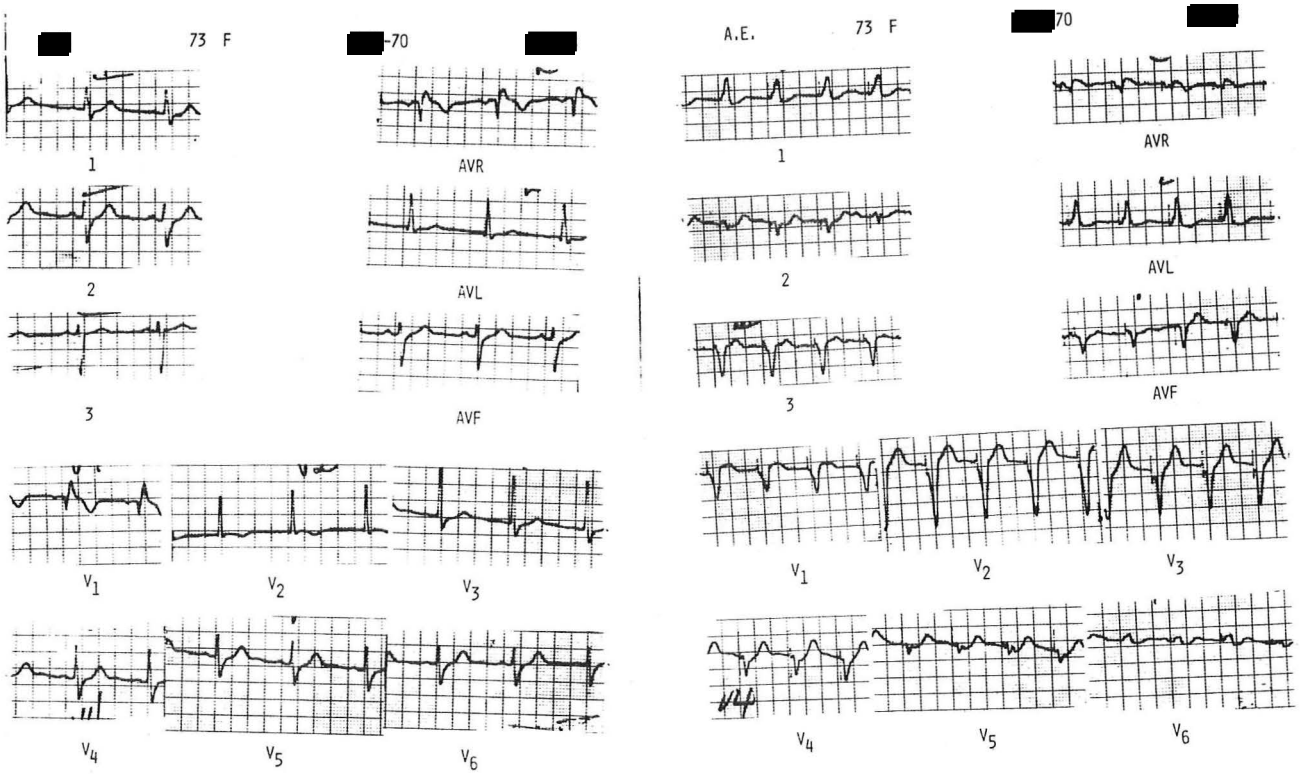


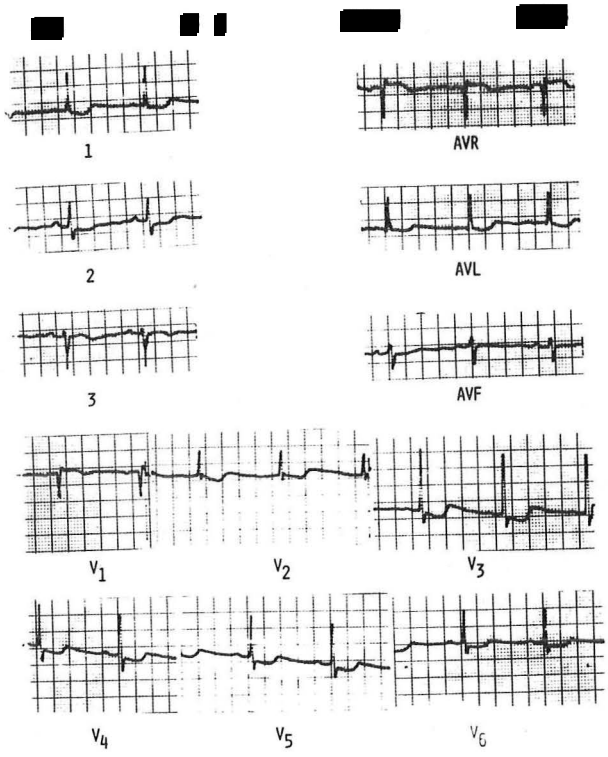
5. RBBB and LAH Progressing to Transient Trifascicular Block With An Acute Myocardial Infarction

[redacted] [redacted] 73 y/o Female [redacted]/70

This patient was admitted on [redacted] 1970, with an anterior myocardial infarction with an EKG showing LAH with RBBB. On her second hospital day she developed asystole followed by a third degree block; a pacemaker was inserted. She reverted to normal sinus rhythm within 48 hours. She later developed atrial flutter requiring cardioversion. She subsequently did well and her EKG reverted to normal conduction.

This represents a RBBB with LAH due to an acute anterior myocardial infarction possibly progressing to trifascicular block and subsequently, with healing of the infarction, a return to no major conduction defects.



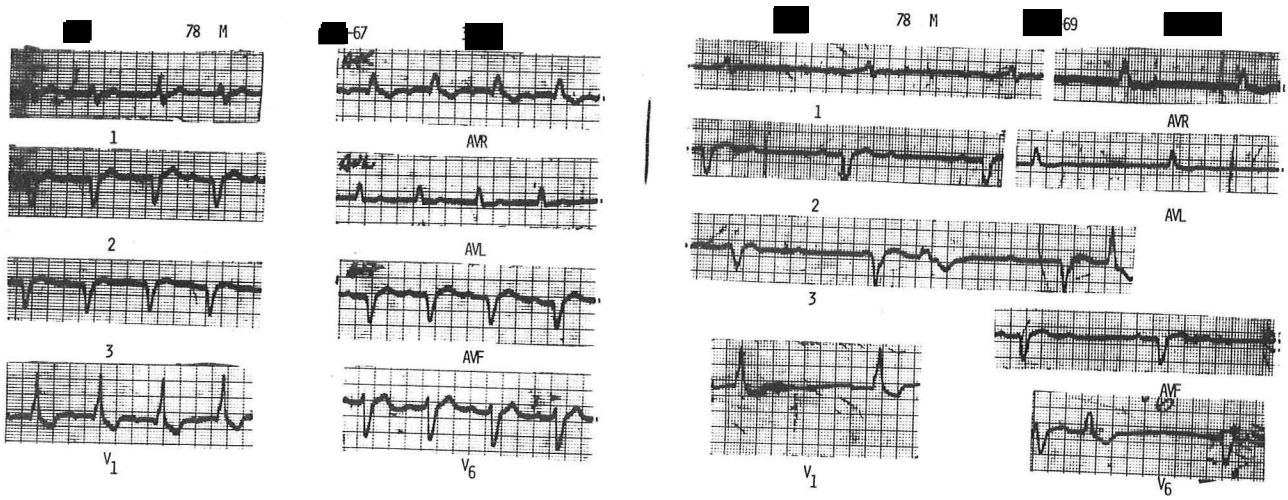


6. Incomplete Trifascicular Block (1°, RBBB and LAH) Developing Complete Trifascicular Block

[redacted] [redacted] 78 y/o Male [redacted]/69 (death)

This patient gave a history of 10 years of Adams-Stokes attacks. He was first admitted on [redacted], 1967, with CHF and an EKG showing RBBB with LAH, 1° heart block and an old anterior myocardial infarction. He refused a pacemaker, but did respond to medical treatment for his CHF. He was readmitted [redacted]/69, complaining of anterior chest pain. His EKG showed RBBB, LAH, Mobitz Type II block and PVC's. A temporary pacemaker was inserted, followed by a permanent pacemaker. He reverted his EKG back to an incomplete trifascicular block (1°, RBBB and LAH). He was discharged; however, he was readmitted [redacted]/69, with sepsis in his pacer site. The pacemaker was removed and not replaced. His EKG's showed a stable incomplete trifascicular block pattern. He returned to the emergency room on [redacted]/69 in shock with a 3° block and expired.

This is an example of an incomplete trifascicular block progressing intermittently to a complete trifascicular block over two or more years.

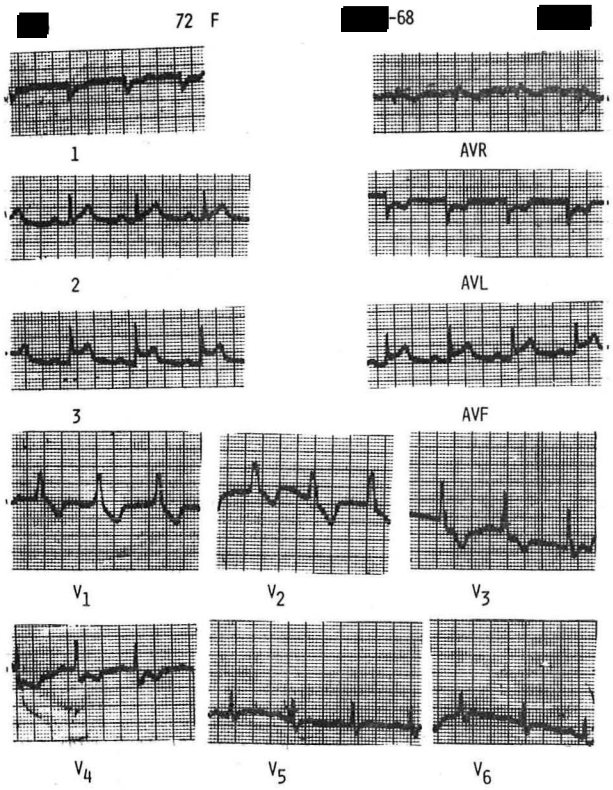


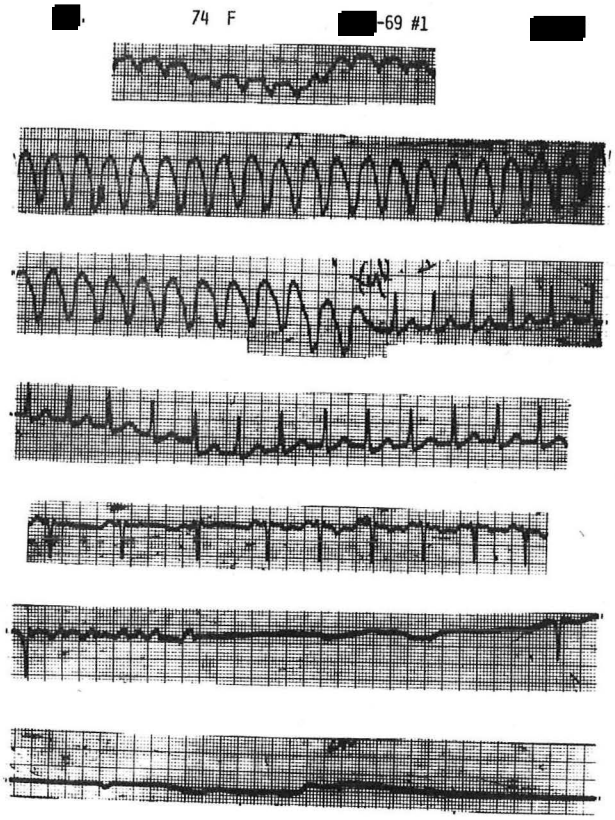
7. RBBB and LPH With Recurrent Myocardial Infarction Resulting In Transient Trifascicular Block

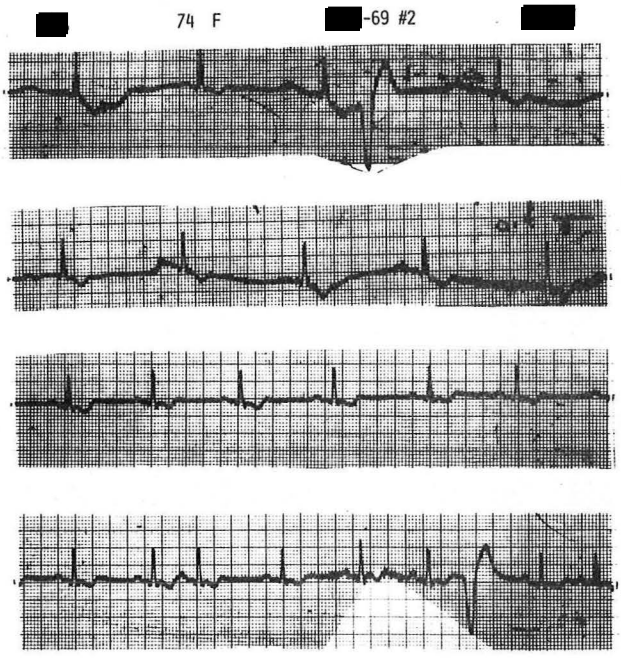
[redacted] [redacted] 74 y/o Female [redacted]/69

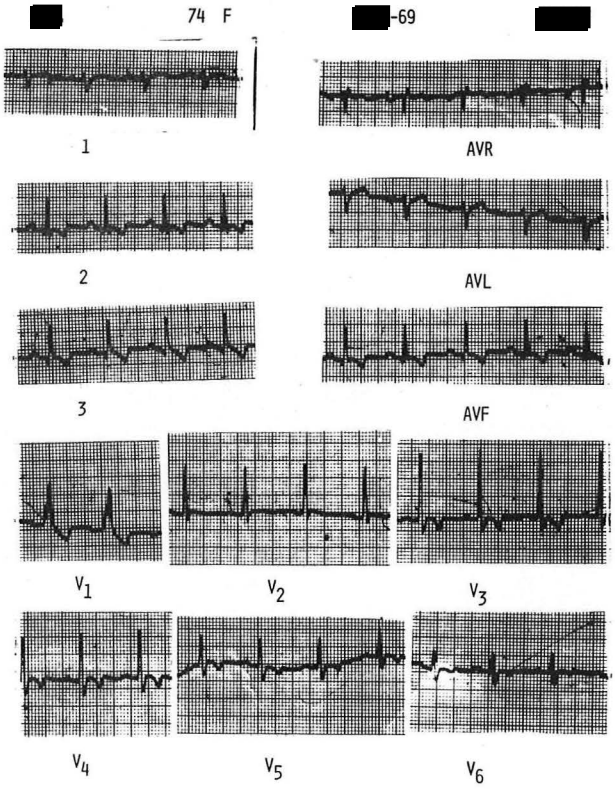
This patient developed an inferior myocardial infarction on [redacted] 1968, resulting in a permanent EKG pattern of RBBB and LPH. She subsequently developed a recurrent inferior myocardial infarction on [redacted]/69, resulting in trifascicular block transiently.

This represents a transient trifascicular block following a recurrent myocardial infarction. This block was probably due to damage to the only remaining fascicle (left anterior fascicle).







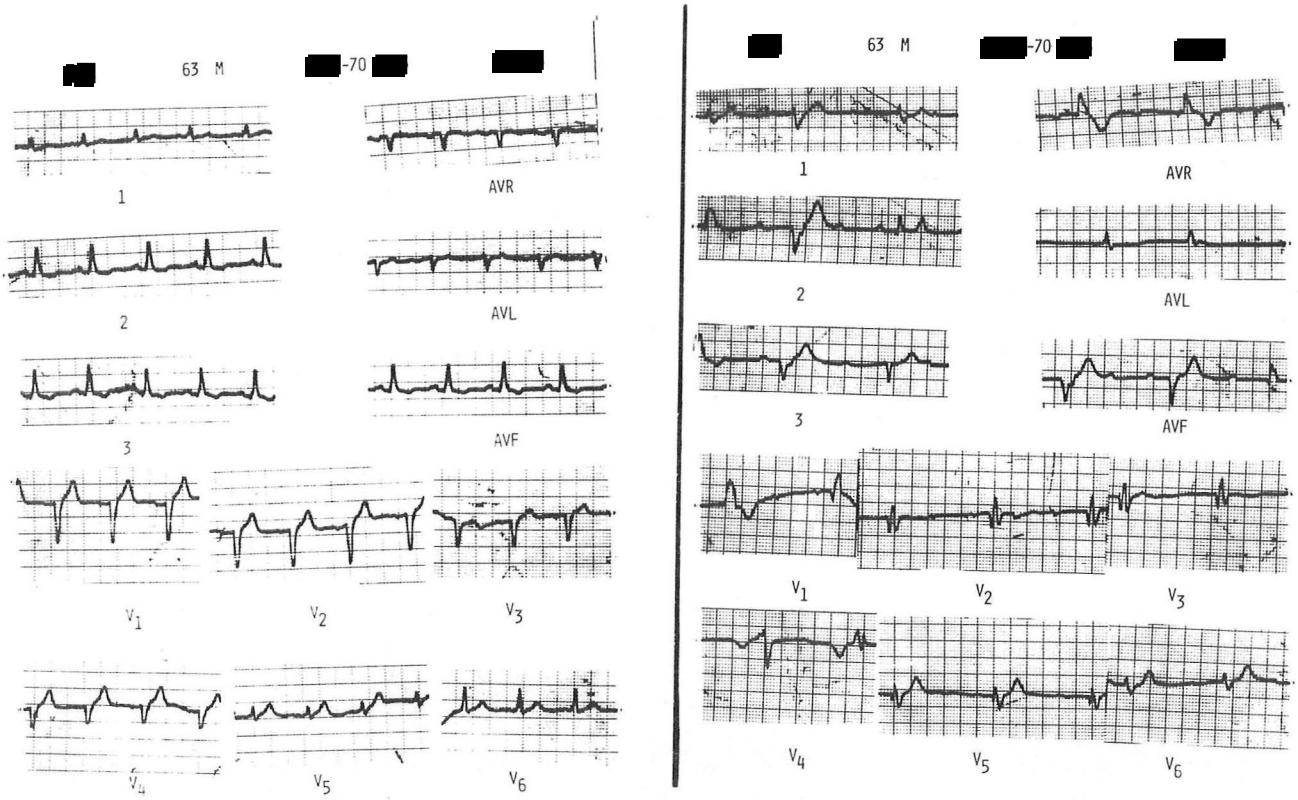


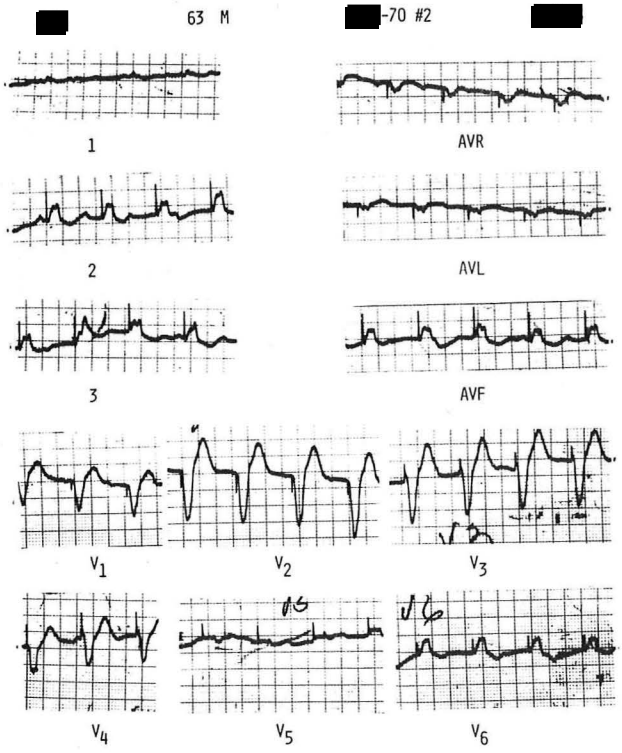
8. Trifascicular Block To LAH To Normal Axis To Sudden Death

[redacted] [redacted] [redacted] 63 y/o Male [redacted]/70

A patient with diabetes and two previous myocardial infarctions (anterior and inferior) in 1957 and 1968 who developed acute onset of substernal chest pain with symptoms of CHF. His admission electrocardiogram showed a slight axis shift (within normal limits) and previously demonstrated Q waves in V_1 - V_4. The second day of admission he developed complete heart block requiring a pacemaker. He reverted to sinus rhythm within 24 hours but his EKG [redacted]/70) revealed LAH and prolonged P-R suggesting ischemia of the conduction system. He improved and was doing well. He was ambulatory and free of CHF or arrhythmias. His EKG on [redacted]/70 revealed a normal axis with resolving changes of an acute myocardial infarction and an old myocardial infarction. On [redacted]/70 he suddenly developed syncope. Resuscitative measures were carried out for 5 hours but without success.

This is an example of a patient with trifascicular block with an acute infarction reverting back to normal conduction, followed by a cardiac arrest, possibly due to reoccurrence of his trifascicular block.

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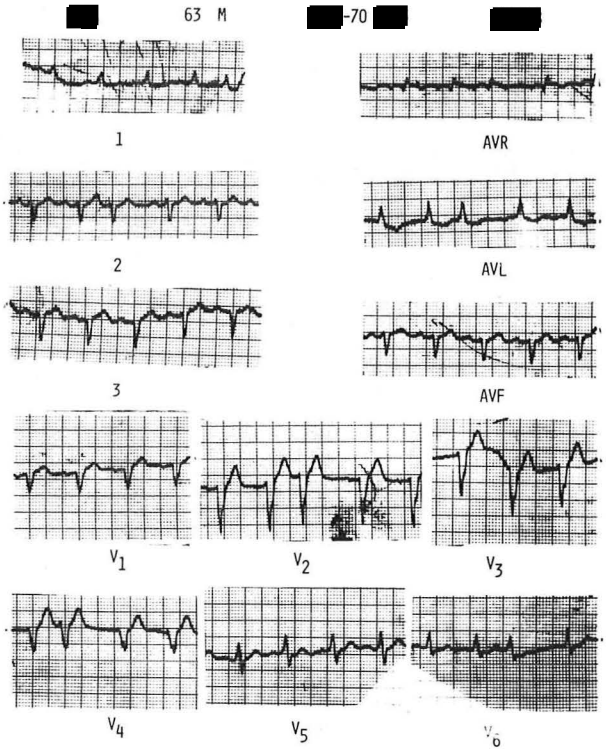
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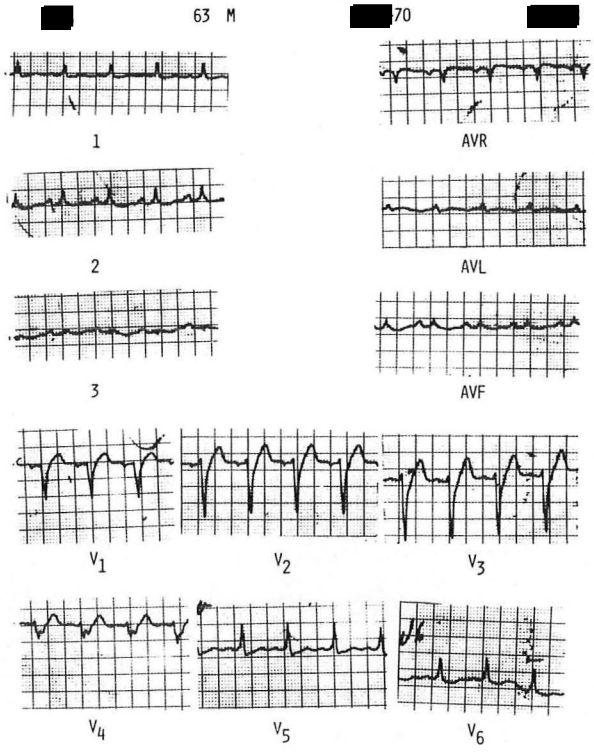
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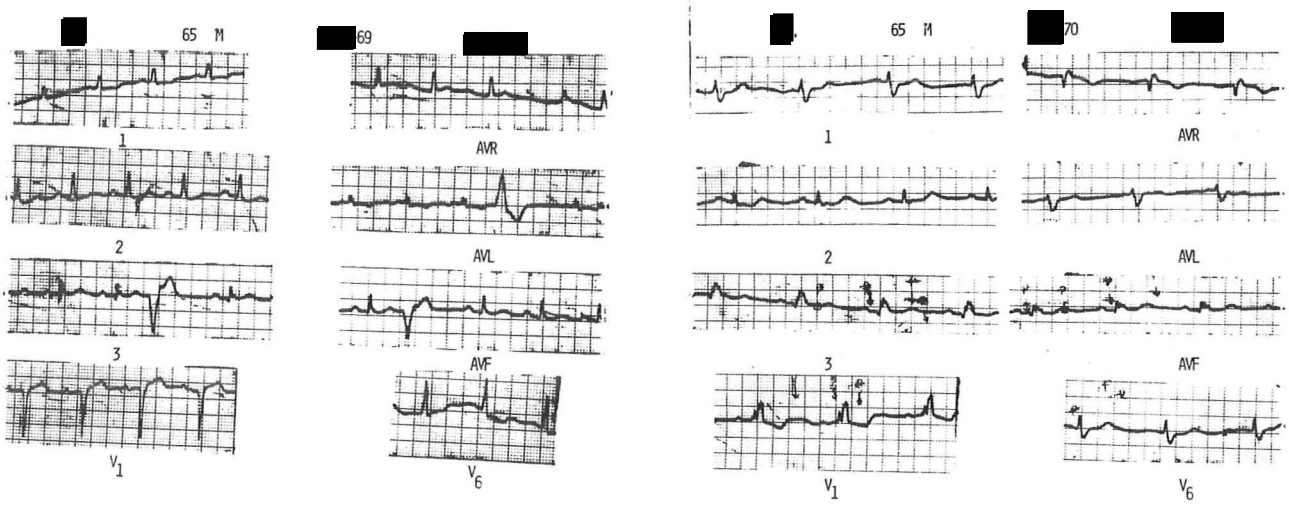
9. "Asymptomatic" Incomplete Trifascicular Block

[redacted] [redacted] 65 y/o Male [redacted]/69

The patient developed CHF in 1965 and atrial flutter. He converted on quinidine and was maintained on digitalis. He was readmitted on [redacted]/69 with progressive CHF and again cardioverted and given digitalis and diuretics. His EKG on [redacted]/69 showed no evidence of conduction block. He has been followed in clinic with occasional anginal episodes relieved by nitroglycerine and Isordil. An EKG obtained in the clinic on [redacted]/70 as part of a routine follow-up, without pertinent symptoms, revealed bifascicular block (RBBB with LPH) and a slightly prolonged P-R interval suggesting incomplete trifascicular block occurring without evidence of infarction. His follow-up to date has been uneventful.

This represents incomplete trifascicular block (RBBB with LPH and prolonged P-R) developing without symptoms of infarction in a patient with ischemic heart disease. No evidence to date of complete block has developed.



10. RBBB to RBBB and LPH to Trifascicular Block

[redacted] [redacted] 88 y/o Male [redacted]/69

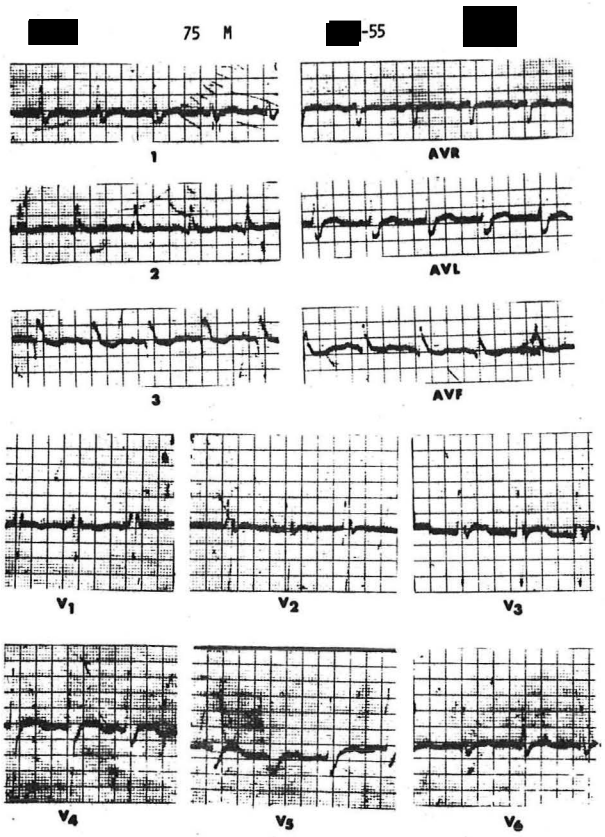
This patient had documented hypertension and angina with a RBBB pattern on his EKG since 1955. He was first admitted on [redacted], 1969, in CHF and complete heart block with a probable nodal rhythm. He refused a pacemaker. He returned on [redacted]/69 again in CHF and still in complete heart block. He accepted a permanent pacemaker. His EKG's revealed:

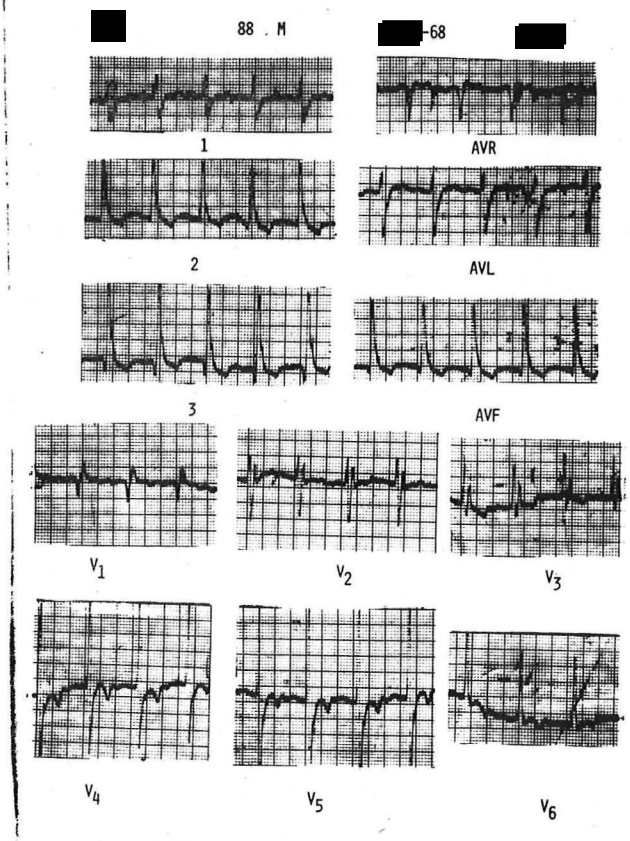
RBBB with a normal axis [redacted]/55

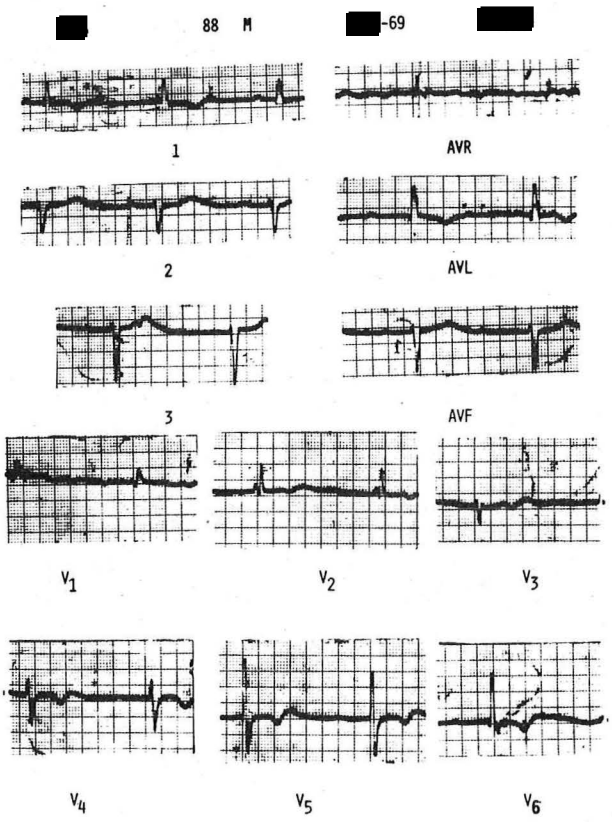
RBBB and LPH and prolonged P-R (incomplete trifascicular block) [redacted]/68

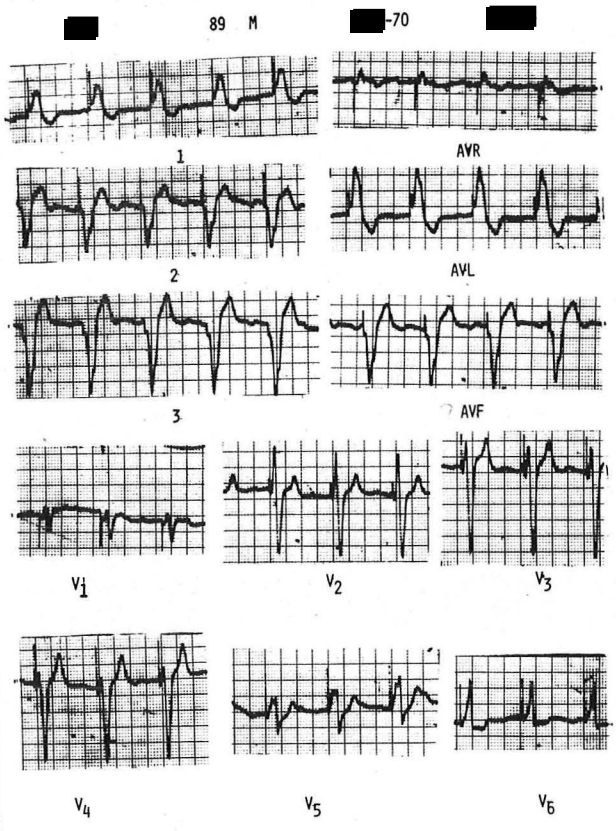
Complete trifascicular block [redacted]/69

This represents a long standing RBBB progressing to incomplete trifascicular block and then to complete trifascicular block.







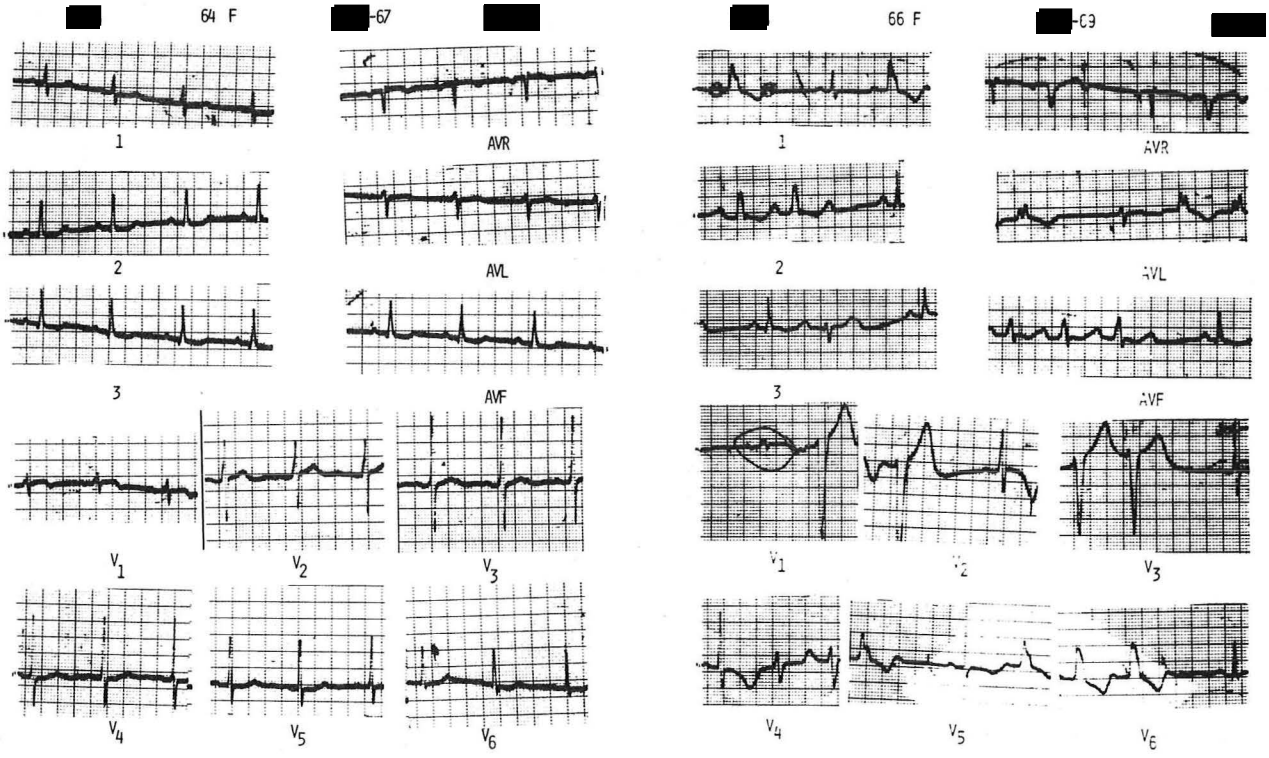


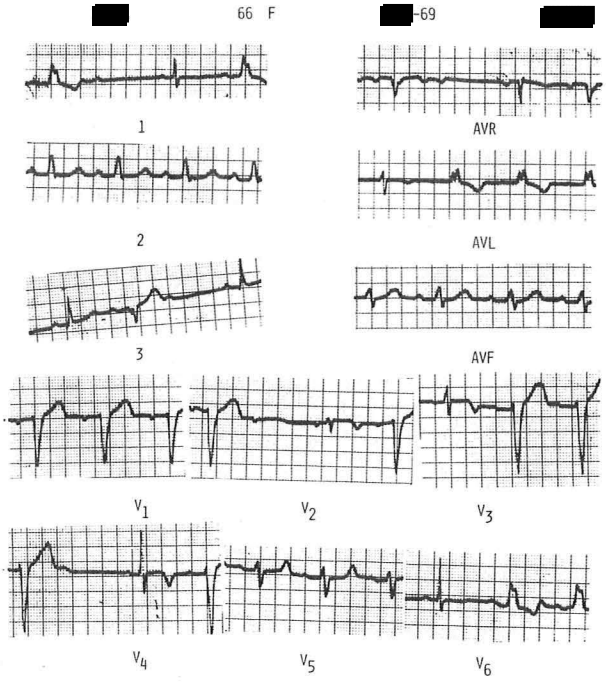
11. Intermittent "Bilateral Bundle Branch Block" and Trifascicular Block.

[redacted] [redacted] 66 y/o Female [redacted]/69

This lady was first seen in 1967 with an acute episode of CHF and supraventricular tachycardia. She responded well to treatment but a transient LBBB was present which reverted. She reappeared on [redacted]/69 with recurrent CHF and a heart block. Her electrocardiogram at this time revealed "Bilateral Bundle Branch Block" alternating between RBBB and LBBB and a prolonged P-R with associated Mobitz Type II 2° block. This represents intermittent trifascicular block. A temporary pacemaker, followed by a permanent pacemaker was inserted. The etiology of the block was uncertain but thought perhaps to be 2° to Mellaril toxicity. She intermittently alternated between pacing and LBBB with 1° block. A follow-up clinic visit on [redacted]/70 showed a return to NSR with normal axis and normal P-R interval. Another clinic visit on [redacted]/70 showed a return to LBBB.

This case represents an intermittent bilateral bundle branch block (bifascicular) and trifascicular block.




[redacted] 66 F [redacted]-69 [redacted]
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V4
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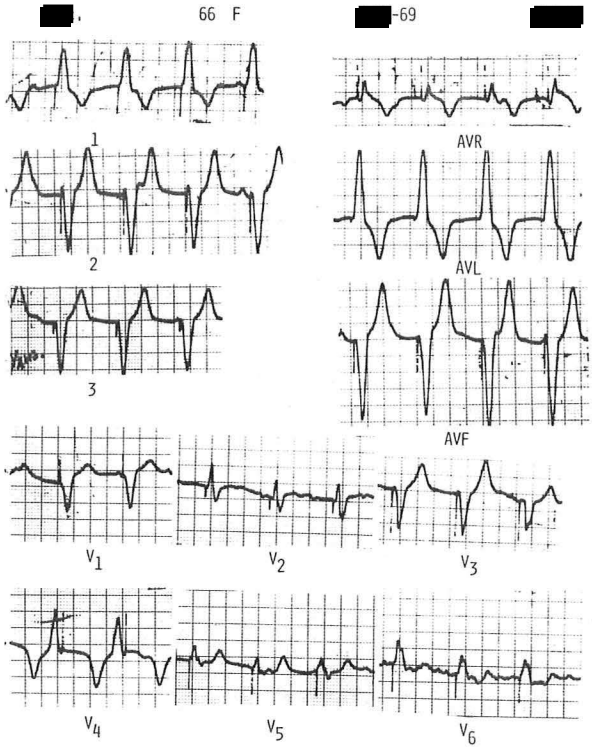
[redacted] 66 F [redacted]-69 [redacted]
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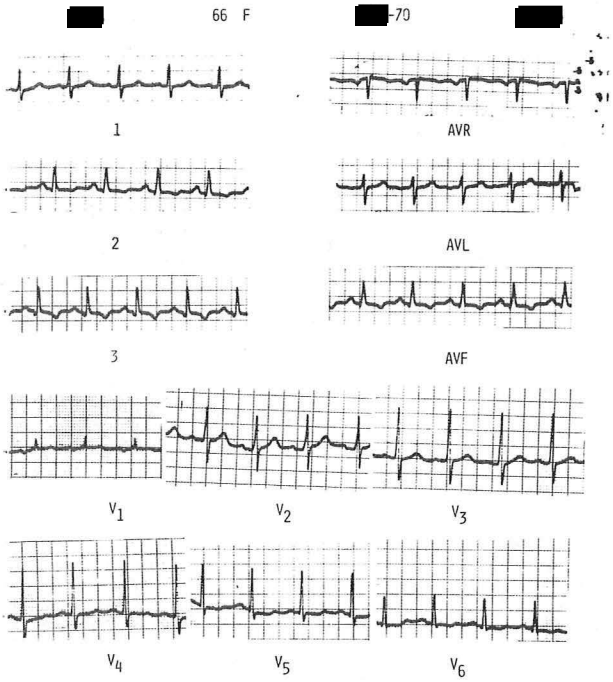
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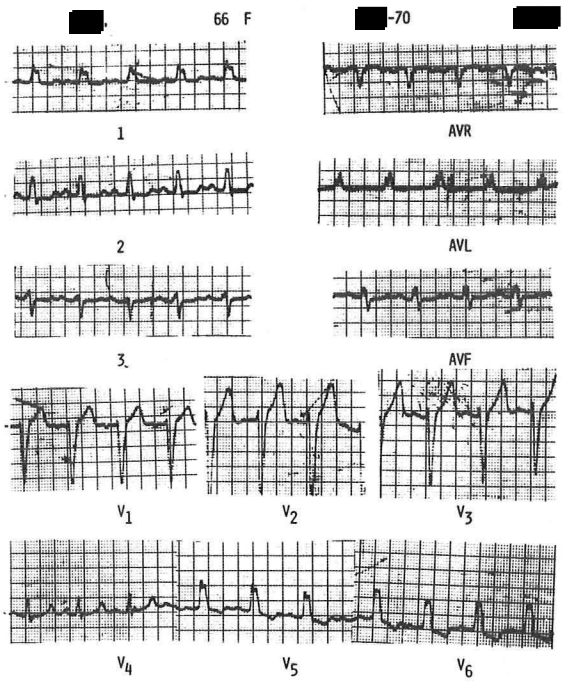
[redacted] 66 F [redacted]-70 [redacted]
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BIBLIOGRAPHY

General Review and Pertinent Background

1. Rosenbaum, M. B., Elizari, M. V. and Lazzari, J. O.: The Hemiblocks. Tampa tracings, 1970.

2. Rosenbaum, M. B., et al. Los Hemibloqueos. 1968, Ed. Paidos, Buenos Aires.

3. Hoffman, B. F., and Cranefield, P. F.: Electrophysiology of the heart. McGraw-Hill Book Company, Inc. 1960.

4. Scanlon, P. J., Pryor, R., and Blount, S. G., Jr.: Right bundle-branch block associated with left superior or inferior intraventricular block. Circ. 42:1123, 1970.

5. Lasser, R. P., Haft, J. I., and Friedberg: Relationship of right bundle-branch block and marked left axis deviation (with left parietal or peri-infarction block) to complete heart block and syncope. Circ. 37:429, 1968.

6. Scanlon, P. J., Pryor, R., and Blount, S. G., Jr.: Right bundle-branch block associated with left superior or inferior intraventricular block. Circ. 42:1135, 1970.

7. Fernandez, F., Scebat, L., and Lenegre, J.: Electrocardiographic study of left intraventricular hemiblock in man during selective coronary arteriography. Am. J. Cardiol. 26:1, 1970.

8. Harper, J. R., et al.: Coronary artery disease and major conduction disturbances. A pathological study designated to correlate vascular and conduction system abnormalities with electrocardiogram. Am. Heart J. 77:411, 1969.

9. Rosenbaum, M. B.: Types of right bundle branch block and their clinical significance. J. Electrocardiology 2(2):197, 1969.

10. Rosenbaum, M. B.: Types of right bundle branch block and their clinical significance. J. Electrocardiology 1(2):221, 1968.

11. McNally, E. M., and Benchimol, A.: Medical and physiological considerations in the use of artificial pacing. Part I. Am. Heart J. 75:380, 1968.

12. Castellanos, A., Lemberg, L., Arcebal, A. G., and Claxton, B. W.: Post-infarction conduction disturbances: A self teaching program. Dis. Chest. 56:421, 1969.

13. Castellanos, A., Lemberg, L., Arebal, A. G., Berkovits, B. V., and Claxton, B. W.: Pacing in acute myocardial infarction: A programmed introduction. Chest 58: No. 2, 1970.

14. Rosenbaum, M. B.: Types of right bundle branch block and their clinical significance. J. Electrocardiology 1(2):221, 1968.

Anatomy Of The Conduction System

15. Lev, M., et al.: A method for the histopathologic study of the atrioventricular node, bundle and bundle branches in the human heart. Circ. 4:863, 1951.

16. Lev, M., and Lerner, R.: The theory of Kent: A histologic study of the normal atrioventricular communications of the human heart. Circ. 1:176, 1955.

17. Lev, M.: Anatomic basis for atrioventricular block. Am. J. Med. 37:742, 1964.

18. Lev, M.: The normal anatomy of the conduction system in man and its pathology in atrioventricular block. Ann. New York Acad. Sci. 111:817, 1964.

19. Hudson, R. E. B.: The human conducting system and its examination. J. Clin. Path. 16:492, 1963.

20. Hudson, R. E. B.: Cardiovascular Pathology, Vol I, Chapter 2: The Conduction System. Edwards Arnold Pub., London, 1965.

21. *loc. cit.* 1.

22. James, T. N.: Anatomy of the coronary arteries in health and disease. Circ. 32:1020, 1965.

23. James, T. N., and Burch, G. E.: The blood supply of the human interventricular septum. Circ. 17:391, 1958.

24. Mahaim, I.: Les Maladies Organiques du Faisceau de His-Tawara. Masson et Cie., Paris, 1931.

25. Kistin, A. D.: Observations on the anatomy of the atrioventricular bundle of His and the questions of other muscular atrioventricular connections in normal human hearts. Am. Heart J. 37:849, 1949.

26. Lenegre, J.: Etiology and pathology of bilateral bundle branch block in relation to complete heart block. Progress Card. Dis. 6:409, 1964.

Types of Conduction Blocks

27. Smith, L. A., et al.: Studies on the mechanism of ventricular activity. IV. Ventricular excitation in segmental and diffuse types of experimental bundle branch block. Circ. Res. 2:221, 1954.

28. Oppenheimber, B. S., and Rothschild, M. A.: Abnormalities in the QRS group of the electrocardiogram associated with myocardial involvement. Proc. Soc. Exper. Biol. and Med. 14:57, 1916.

29. Segers, M.: The different types of intraventricular block. Am. Heart J. 37:92, 1949.

30. First, S. R., et al.: Peri-infarction block; electrocardiographic abnormality occasionally resembling bundle branch block and local ventricular block of other types. Circ. 2:31, 1950.

31. Burchell, H. B., and Pruitt, R. D.: The value of the esophageal electrocardiogram in the elucidation of postinfarction intraventricular block. Am. Heart J. 42:81,1951.

32. Grant, R. P.: Spatial vector electrocardiography. A method for calculating the spatial electrical vectors of the heart from conventional leads. Circ. 2:676, 1950.

33. Grant, R. P., and Estes, E. H., Jr.: Spatial Vector electrocardiography. Clinical electrocardiographic interpretation. The Blakinston Co., 1952.

34. Pryor, R., and Blount, S. G., Jr.: The clinical significance of true left axis deviation. Left intraventricular blocks. Am. Heart J. 72:391, 1966.

35. Lasser, R. P., et al.: Relationship of right bundle branch block and marked left axis deviation (with left parietal or peri-infarction block) to complete heart block and syncope. Circ. 37:429, 1968.

36. *loc. cit.* 1.

37. *loc. cit.* 2.

38. Rosenbaum, M. B., et al.: Intraventricular trifascicular blocks. Review of the literature and classification. Am. Heart J. 78:450, 1969.

Experimental Blocks and His Bundle Recordings

39. 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.

40. Watt, T. B., Jr., and Pruitt, R. D.: Electrocardiographic findings associated with experimental arborization block in dogs. Am. Heart J. 69:642, 1965.

41. Watt, T. B., Jr., et al.: Left axis deviation induced experimentally in a primate heart. Am. Heart J. 70:381, 1965.

42. Watt, T. B., Jr., and Pruitt, R. D.: Character, cause and consequence of combined left axis deviation and right bundle branch block in the human electrocardiogram. Am. Heart J. 77:460, 1969.

43. Hugenholtz, P. G., et al.: Electrocardiographic and vectorcardiographic changes after cocain block of selected points in the canine left bundle branch system. (Abstract) Circ. 28:740, 1963.

44. Damato, A. N., et al.: A study of heart block in man using His bundle recordings. Circ. 39:297, 1969.

45. Damato, A. N., et al.: Recording of specialized conducting fibers (A-V nodal, His bundle, and right bundle branch) in man using electrode catheter technique. Circ. 39:435, 1969.

46. Scherlag, B. J., et al.: Catheter technique for recording His bundle activity in man. Circ. 39:13, 1969.

Blocks in Congenital Heart Disease

47. *loc. cit.* 1.

48. Burchell, H. B., et al.: The electrocardiogram of patients with atrioventricular cushion defect (defects of the atrioventricular canal). Am. J. Cardiol. 6:575, 1960.

49. Durrer, D., et al.: The genesis of the electrocardiogram of patients with ostium primum defects (ventral atrial septal defects). Am. Heart J. 71:642, 1966.

50. Kulbertus, H. E., et al.: Electrocardiographic correlation of anatomic and haemodynamic data in ostium primum atrial septal defects. Brit. Heart J. 30:464, 1968.

Lenegre's Disease

51. *loc. cit.* 26.

52. Lenegre, J.: Bilateral bundle branch block. Cardiologia 48:134, 1966.

53. Johansson, B. W.: Complete heart block. A clinical hemodynamic and pharmacological study in patients with and without an artificial pacemaker. Acta Med. Scandinav. 180, Suppl. 451, 1966.

54. Portal, R. W., et al.: Artificial pacing for heart block. Lancet 2:1369, 1962.

55. Davies, M., and Harris, A.: Pathological basis of primary heart block. Brit. Heart J. 31:219, 1969.

56. Davies, M.: Histological study of the conduction system in complete heart block. J. Path. Bact. 94:351, 1967.

57. Yater, W. M., et al.: Auriculoventricular heart block due to bilateral bundle branch lesions. Arch. Int. Med. 57:132, 1936.

Lev's Disease

58. *loc. cit.* 17.

59. *loc. cit.* 18.

60. Sugiura, M., et al: Histological studies on the conduction system in 14 cases of right bundle branch block associate with left axis deviation. Japanese Heart J. 10:121, 1969.

Left Axis Deviation

61. Ashman, R., and Hull, E.: Essentials of Electrocardiography. The MacMillan Co., 1937.

62. Grant, R. P.: Left axis deviation: An electrocardiographic-pathologic correlation. Circ. 14:233, 1956.

63. Durrer, D.: Electrical aspects of human cardiac activity: A clinical-physiological approach to excitation and stimulation. Cardiovasc. Res. 2:1, 1968.

64. Burch, G., and Winsor, T.: A primer of electrocardiography. Lea and Febiger, 1945.

65. Grant, R. P.: Peri-infarction block. Progress Card. Dis. 2: 237, 1959.

66. Libanoff, A. J.: Marked left axis deviation. Parietal and peri-infarction block. Am. J. Cardiol. 14:339, 1964.

Clinical Significance and Etiology Of Fascicular Blocks

67. Oliveira, E., and Gomez-Patino, N.: Falcemic cardiopathy. Report of a case. Am. J. Cardiol. 11:686, 1963.

68. Unger, P. N., et al.: The concept of "masquerading" bundle branch block. An electrocardiographic-pathologic correlation. Circ. 17:397, 1958.

69. Davies, H., and Evans, W.: The significance of deep S waves in leads II and III. Brit. Heart J. 22:551, 1960.

70. Eliot, R. S., et al.: Cardiac amyloidosis. Circ. 23:613, 1961.

71. Eliot, R. S., et al.: The clinical significance of uncomplicated marked left axis deviation in men without known disease. Am. J. Cardiol. 12:767, 1963.

72. Evans, W.: Alcoholic myocardiopathy. Progress Card. Dis. 7:151, 1964.

73. Lamb, L. E., et al.: Intermittent right bundle branch block without apparent heart disease. Am. J. Cardiol. 4:302, 1959.

74. Lamb, L. E., et al.: Electrocardiographic findings in 67,375 asymptomatic subjects. V. Left bundle branch block. Am. J. Cardiol. 6:130,1960.

75. Kirklin, J. W., and Mankin, H. T.: Open operation in the treatment of calcific aortic stenosis. Circ. 21:578, 1960.

76. Eliot, R. S., et al.: Prognostic significance of anterolateral peri-infarction block in surgery for aortic regurgitation. Am. J. Cardiol. 16:67, 1965.

77. Richman, J. L., and Wolff, L.: Left bundle branch block masquerading as right bundle branch block. Am. Heart J. 47:383, 1954.

78. Rothfeld, E. L., et al.: The electrocardiographic syndrome of superior axis and right bundle branch block. Dis. Chest 55:306, 1969.

79. Watanabe, Y., and Dreifus, L. S.: Second degree atrioventricular block. Cardiovasc. Res. 1:150, 1967.

80. Watanabe, Y., and Dreifus, L. S.; New concepts in the genesis of cardiac arrhythmias. Am. Heart J. 76:114, 1968.

81. Langendorf, R., and Pick, A.: Atrioventricular block, Type II (Mobitz). Its nature and clinical significance. Editorial. Circ. 38:819, 1968.

82. *loc. cit.* 68.

83. *loc. cit.* 1.

84. Bauer, G. E., et al.: Bundle branch block in acute myocardial infarction. Brit. Heart J. 27:724, 1965.

85. Stock, R. J., and Macken, D. L.: Observations on heart block during continuous electrocardiographic monitoring in myocardial infarction. Circ. 38:993, 1968.

86. Lopez, J. F.: Electrocardiographic findings in patients with complete heart block. Brit. Heart J. 30:20, 1968.

87. Norris, R. M.: Heart block in posterior and anterior myocardial infarction. Brit. Heart J. 31:352, 1969.

88. Saltzman, P., et al.: Right bundle branch block with left axis deviation. Brit. Heart J. 28:703, 1966.

89. Rosenbaum, M. B., and Lepeschkin, E.: Bilateral bundle branch block. Am. Heart J. 50:38, 1955.

90. Toyama, S.: Intraventricular block. Jap. Circ. J. 30:144, 1966.

91. Lepeschkin, E.: Modern electrocardiography. Vol. I. The P-Q-R-S-T-U Complex. The Williams and Wilkins Co., 1951.

Pacing In Fascicular Blocks

92. *loc. cit.* 11.

93. *loc. cit.* 1.

94. *loc. cit.* 8.

95. Lopez, J. F., Mori, M., and Baltzan, B. L.: Myocardial infarction and complete heart block. Canad. Med. Assoc. J. 102:No. 7, April, 1970.

96. Godman, M. J., Lassers, B. W., and Julian, D. G.: Complete bundle-branch block complicating acute myocardial infarction. N. Eng. J. Med. 282:237, 1970.

97. *loc. cit.* 85.

98. *loc. cit.* 12

99. *loc. cit.* 13.

100. Atkins, J. M., Leshin, S. J., Blomqvist, G., and Mullins, C. B.: Prognosis of right bundle branch block and left anterior hemiblock: A new indication for permanent pacing. (Abstract). Presented at American College of Cardiology, February, 1971.

100a. Kulbertus, H., and Collignon, P.: Association of right bundle branch block with left superior or inferior intraventricular block. Its relation to complete heart block and Adams-Stokes syndrome. Brit. Heart J. 31:435, 1969.

Myocardial Infarction With Blocks

101. *loc. cit.* 1, 8, 11, 72, 13, 87, 95, 96

102. Shadaksharappa, K. S., et al.: Recognition and significance of intraventricular block due to myocardial infarction (peri-infarction). Circ. 37:20, 1968.

103. Sutton, R., and Davies, M.: The conduction system in acute myocardial infarction complicated by heart block. Circ. 38: 987, 1968.

104. Schloff, L. D., Adler, L., Donoso, E., and Friedberg, C. K.: Bilateral bundle-branch block. Clinical and electrocardiographic aspects. Circ. 35:790, 1967.

105. Campbell, M.: The outlook with bundle-branch block. Brit. Heart J. 31:575, 1969.

106. Corne, R. A., et al.: Peri-infarction block: postmyocardial-infarction intraventricular conduction disturbance. Am. Heart J. 70:150, 1965.

107. Bauer, G. E., Julian, D. G., and Valentine, P. A.: Bundle-branch block in acute myocardial infarction. Brit. Heart J. 27:274, 1965.

In Hamlet,

> *"My pulse, as yours, doth temperately keep time,*
> *And makes as healthful music. It is not madness*
> *That I have uttered; bring me to the test."*