

who are less fortunate. Some persons become exceedingly interested in our bureau and have given freely and gladly to its support.

The technic of expression has been discussed by every one of the essayists and I am sure if Dr. Faison will interview any one of these men they will be glad to have a private conference with him.

ELECTROCARDIOGRAPHIC CHANGES ASSOCIATED WITH MYOCARDIAL INVOLVEMENT

WITH SPECIAL REFERENCE TO PROGNOSIS *

BERNARD S. OPPENHEIMER, M.D.

AND

MARCUS A. ROTHSCILD, M.D.

NEW YORK

Theoretical considerations have led us to investigate a certain type of electrocardiogram with a view to determining its relationship to myocardial involvement. We were struck by the fact that the electrocardiograms interpreted as due to bundle branch lesions in human beings differed considerably from those resulting from experimental bundle branch lesions in animals. Moreover, pathologically the occurrence of a lesion confined to either of the main bundle branches is a rarity. In fact there is scarcely a single absolutely convincing reported case, confirmed by pathologic examination, of bundle branch block. On the contrary, pathologic examination in cases diagnosed electrocardiographically as bundle branch block has for the most part failed to show definite bundle branch lesions.

However, it is common pathologic experience to find lesions in the lower third of the septum involving the wall of the left ventricle and, as will be shown, such lesions are the usual findings in cases diagnosed as bundle branch block.¹ Anatomically these lesions involve not only heart muscle but the arborizations of the conducting tissue (Purkinje fibers) as well.

THEORETICAL CONSIDERATIONS

The normal electrocardiogram is to be considered the result of the passage of an impulse at a normal velocity through the usual channels, that is, node of Tawara, main stem, bundle branches and arborizations, which consist of the so-called Purkinje fibers. The latter form a network covering practically the entire endocardial surface of the ventricles. The velocity of the impulse through Purkinje fibers is at least ten times faster than its rate through ordinary ventricular musculature. The impulse reaches the ventricle normally through the Purkinje fibers, stimulating the ventricular walls practically as a whole.²

It is conceivable that the passage of this impulse may be hindered at any point in the conducting system. An experimental injury of either right or left main branch may interrupt completely the passage of the impulse over normal channels to the corresponding ventricle, the spread then occurring through ordinary muscular connections. This gives a characteristic electrocardiogram differing in many respects from

those to which we shall devote our attention. But the theoretical possibilities are not limited to a complete block of one or both bundle branches. A lesion only partly involving either bundle branch, or an extensive lesion of the arborizations of a branch such as occurs in human pathology, would cause a delay in the transmission of the excitation wave to the area supplied by the damaged conduction fibers. Such a lesion, if sufficiently extensive, should give observable changes in the electrocardiogram, and it is probable that these changes will be distinguishable from the changes that are produced by most other cardiac abnormalities.

These *a priori* considerations lead us to believe that there is an electrocardiogram that is characteristic of lesions involving part of a bundle or its arborizations. The agreement of pathologic and clinical experience with theoretical considerations supports our belief that there is a more than accidental relationship between the two.

Many of the cases we shall discuss cannot properly be termed bundle branch block and therefore we are introducing the term intraventricular block. By intraventricular block we mean any delay in conduction below the main stem of the bundle of His. Intraventricular block includes: (1) bundle branch block, and (2) arborization or Purkinje block, by which we mean interference with the conduction beyond the two chief branches of the bundle of His.

This disturbance of conduction may be permanent or temporary. The permanent changes we consider indicative of a definite pathologic lesion, generally myodegeneration. There are cases in which the electrocardiogram is temporarily of the type to be described.³ Experimentally the changes can be produced by morphin, asphyxia, etc. Decompensation and agonal states do not as a rule produce these changes.

The criteria in the electrocardiograms that we have used are in general as follows:

1. Abnormal prolongation of the time interval of the QRS group beyond the normal limit of 0.1 second. This prolongation is most manifest in a widening of the R wave, so that its foot points are abnormally separated. The R wave no longer has its slender, tall, spike-like appearance, but is broader and sometimes blunter than normally.

2. Notching of the R wave. This notching may appear on the ascending or descending limb, on both limbs, or at the peak. It may be multiple, and its degree and location may vary slightly from beat to beat. In arrhythmias, the shorter the preceding interval, the more pronounced is the evidence of disturbed intraventricular conduction.

3. Low voltage as expressed by a low amplitude of the waves in all three leads. This change is not uniformly present, but when it occurs it helps to differentiate this type from the electrocardiograms typical of bundle branch block.

4. Absence of the typical diphasic curves with huge T' waves found in experimental bundle branch block.

CLINICAL OBSERVATIONS

In the course of the past few years there have been observed sixty-two cases whose electrocardiograms indicated an intraventricular block. Four of these cases showed electrocardiograms more or less typical of bundle branch block. The other fifty-eight cases observed presented in their electrocardiograms the

* From the Electrocardiographic Laboratory, Mount Sinai Hospital.

* Read before the Section on Practice of Medicine at the Sixty-Fifth Annual Session of the American Medical Association, New York, June, 1917.

1. Carter has reported a series of cases diagnosed as bundle branch block most of which we believe to be cases similar to those which we are about to describe.

2. Lewis and Rothschild: The Excitatory Process in the Dog's Heart, Part II, Ventricles, Phil. Tr. Roy. Soc. London, Series B, 206, 181-226.

3. G. Canby Robinson has reported several such cases.

above mentioned criteria and were consequently interpreted as arborization block. The sixty-two cases presented the clinical pictures of cardiovascular renal disease, atherosclerosis, coronary artery disease, angina pectoris, myocarditis, syphilis, acute articular rheumatism, "grip," diabetes, gout, etc.

The ages of these patients summarized according to decades can be seen in the accompanying table:

AGES OF PATIENTS ACCORDING TO DECADES *

Age	Number
From 20 to 30 years	6
From 30 to 40 years	4
From 40 to 50 years	8
From 50 to 60 years	20
From 60 to 70 years	19
70 years or over	5
	<hr/> 62

* Average age 54.

An analysis according to sex shows that forty-eight were men and fourteen women.

Angina Pectoris.—Twenty-seven of the patients had definite attacks of angina pectoris, fifteen complained of precordial distress, and twenty gave no history of either symptom.

Blood Pressure.—It is interesting but not surprising that of the forty-eight patients on whom we had blood pressure determinations, twenty-five had a systolic pressure below 140 mm. of mercury; five between 140 and 160 mm.; seven between 160 and 180 mm., and eleven between 180 and 220 mm.

DISTURBANCES OF CARDIAC MECHANISM ASSOCIATED WITH INTRAVENTRICULAR BLOCK

1. There were thirty-five cases showing no arrhythmia; of these, twenty-four showed a left ventricular predominance according to the electrocardiogram, and one a right ventricular predominance. It is worth pointing out in this connection that in the presence of marked intraventricular conduction defects, the ordinary electrocardiographic criteria for hypertrophy may not hold.

2. Twelve cases showed auricular fibrillation.

3. Two cases showed auricular flutter.

4. Ten cases showed ventricular extrasystoles alone, and two others showed ventricular extrasystoles in combination with auricular fibrillation.

5. Five cases showed varying ventricular complexes.

Prognosis.—Special emphasis should be laid on the serious prognosis in patients showing electrocardiograms indicative of intraventricular block. Of the fifty-eight patients showing arborization block, twenty-two are dead, twenty-four are alive (and of these four are in a precarious condition) and twelve could not be traced. Of the patients whose fate is known, the mortality has been 48 per cent. within two years. It should be stated that only one of our patients is apparently well, but he has been under observation only five months. Of the four patients with more or less complete bundle branch block, three are dead, and one is in a precarious condition.

There have been twenty-two other patients suffering from angina that have not shown these electrocardiographic changes; of these, only one is known to be dead, seventeen are known to be alive, and sixteen of these seventeen are doing well or fairly well, and one, an old man of 77, is in a precarious condition. The average age of this group is 52 years.

On physical examination two signs have been especially noted: (1) a muffled, poor or practically absent

first heart sound, and (2) a gallop rhythm. One is often struck by the fact that the heart is hypertrophied, but that the first sound, instead of being booming, has a poor or muffled quality.

ASSOCIATION OF ATRIOVENTRICULAR AND INTRAVENTRICULAR BLOCK

Intraventricular block is frequently associated with various grades of atrioventricular (auriculoventricular) block. These are not included in our analysis of sixty-two cases. Of eighteen patients showing various grades of heart block, thirteen also presented evidence of intraventricular block. Only three of the eighteen patients are known to be dead, but these three had the combination of atrioventricular and intraventricular block.

PATHOLOGY

There have been necropsies in fourteen of the twenty-five known fatal cases. In eleven cases there has been an opportunity to examine the hearts grossly, as well as microscopically. In one case we secured only the septum including the atrioventricular bundle, and in two others, we have the necropsy reports and a single slide of heart muscle. Serial sections are being prepared.

1. At the present time we can report that eight of the cases showed coronary artery sclerosis with closure of the anterior descending branch of the left coronary. This artery gives off septal branches which supply the anterior part of the septum (the bundle of His and its two main divisions are supplied chiefly by branches from the right coronary artery). Four other cases showed a nodular sclerosis of the coronary arteries, but no actual occlusion.

2. Thirteen of the fourteen patients had a widely disseminated patchy sclerosis of the myocardium. Of the two that did not have coronary disease, one was a young girl aged 20, who had no known cause for the interstitial myocarditis except a recent "grip," the other was aged 30 and died of cardiac insufficiency resulting from valvular defects.

3. The pathologic changes, especially the sclerosis, predominate in the endocardial and subendocardial layers, that is, in the region of the so-called Purkinje network, as compared with the outer two thirds of the ventricular musculature.

4. In general, the changes were grossly more marked in the left ventricle than in the right.

Experimental work has been begun in collaboration with Dr. H. B. Williams, and at present it can only be stated that injury of the main branches or of the arborization produces changes in the electrocardiogram. It has already been shown by Eppinger and Rothberger* that extensive experimental injuries to the external layers of the ventricular wall do not result in changes in the electrocardiogram.

CONCLUSIONS

1. There has been a discrepancy between previous electrocardiographic interpretation and pathologic findings.

2. Theoretical considerations and pathologic findings point to the existence of a hitherto undescribed type of disturbance which we have called arborization block.

3. We venture to state, therefore, that there is a definite clinical condition to be known as arborization

* Eppinger and Rothberger: Wien. klin. Wchnschr., 1909, 22, 1091-1098.

block; that this condition can be diagnosed by the presence of a definite and permanent type of electrocardiogram; and that the condition has a very serious prognosis.

125 West Eighty-Sixth Street—930 Park Avenue.

ABSTRACT OF DISCUSSION

DR. H. B. WILLIAMS, New York: I have followed the work of Drs. Oppenheimer and Rothschild with interest because I have seen a number of cases of the same nature, but have been unable to secure necropsies in many of them. Dr. Rothschild has properly emphasized the fact that one cannot make a diagnosis of lesions of one main branch of the bundle of His when there is marked hypertrophy of a ventricle. The lesion referred to has often been made on the basis of the electrocardiogram without sufficient justification. I have seen but three cases in the last seven years in which I have felt reasonably secure in diagnosing this lesion.

The peculiar notched and deformed electrocardiograms which Dr. Rothschild has shown I have learned to look on as calling for a serious prognosis. Of a considerable number of such patients seen by me prior to 1915, only two are still living. One sometimes sees this type of electrocardiogram, however, in patients who get along tolerably well for a time. We should, therefore, make use of all the other available clinical evidence before making a decision.

DR. E. LIBMAN, New York: Apart from neuroses, aneurysm and pericarditis, the most common causes of severe attacks of cardiac pain are the following: hypertension, atherosclerosis of the arch with narrowing of the orifices of the coronary arteries or of their lumen, diffuse arterial disease in the coronary system, thrombosis of the coronary arteries and syphilitic lesions at the aortic valve or the root of the aorta. The cases with the classical picture of angina pectoris are mostly cases of coronary thrombosis. If one has had sufficient postmortem experience, it is very often not difficult to say whether there is a thrombosis of the coronary arteries, and at times it is possible to suspect whether the right or the left coronary artery is thrombosed. There are cases in which it is very difficult to say whether the patient is suffering from a gastric condition, such as ulcer, with reflex pain in the cardiac area, or whether the patient has coronary artery disease, or possibly both. In all such doubtful cases the electrocardiographic findings, as described by Drs. Oppenheimer and Rothschild, ought to be of value.

The cases in my own service at the hospital in which these findings were present were all cases in which I had assumed that the patient was suffering from organic heart disease of the coronary arteries—with the exception of one case of aortic insufficiency in a young man; this case did not come to necropsy, and it is therefore not known whether or not the patient had any disease of the myocardium. Complete closure of the coronary artery may occur very insidiously, and the patients make no complaint of any cardiac symptoms. Some of these patients can be recognized by a peculiar color of the face, an earthy color overcast by a leaden tint. In such cases an electrocardiographic examination might be of value in confirming the diagnosis. The diagnosis in such cases is particularly important if the question of operation on some part of the body has arisen. Such patients are apt to succumb within twenty-four to forty-eight hours after operation, although they have had no cardiac symptoms before.

DR. JAMES B. HERRICK, Chicago: I wish to add a word of commendation of this excellent work which has been done in the attempt to correlate the pathologic findings with the findings of the electrocardiograph. In Chicago we have been trying to get at this problem by experimental work on dogs, producing myocardial lesions by ligating branches of the coronary arteries. I think we will reach conclusions much like those presented to us today. Already we have begun to recognize in man curves which we have found in dogs which were operated on. In a man who had, as I believe, coronary thrombosis, the cardiogram was like that which we got in dogs by ligating the left branch of the coronary artery. The changes in the cardiogram described by Dr. Rothschild seem

to be associated with very definite clinical evidences of myocardial weakness. A large percentage of the patients who have shown anything like that have died in a short time.

DR. M. A. ROTHSCHILD, New York: I should like to emphasize one point again. In the twenty-two cases of classical angina that we have included in our series that did not show the electrocardiographic criteria of arborization block, we have been able to follow seventeen patients, and only one of the seventeen is dead. In the other sixty-two cases, with or without angina, whose electrocardiograms presented the criteria of arborization block, the mortality has been approximately 50 per cent. in two years. In our experience, therefore, the electrocardiogram gives valuable evidence in regard to prognosis, especially in those doubtful cases of angina.

HOOKWORM INFECTION AS A MEDICO-MILITARY CONSIDERATION

RECENT EXPERIENCES WITH SOUTHERN TROOPS
DURING MOBILIZATION ON THE
MEXICAN BORDER *

GEORGE B. FOSTER, JR., M.D., DR.P.H.

AND

CHARLES G. SINCLAIR, M.D.

Captain and First Lieutenant, Respectively, Medical Corps, United States Army

ATLANTA, GA.

Following the call of the President, June 18, 1916, approximately 10,000 men were mobilized at Nogales, Ariz. These troops, with the exception of 1,200 regulars, were composed of organizations of the National Guard from northern, eastern and Pacific states. They had very little sickness. The morbidity rates for August, September and the first three weeks in October varied from 14 to 35 per thousand for the regulars and from 7 to 29 per thousand for the National Guard. The average morbidity rate for regulars was 23.1 per thousand while that of the National Guard was somewhat lower, 17.9 per thousand.

During the last week in October, 1916, a brigade of the Alabama National Guard was mobilized at Nogales. This command consisted of headquarters, three regiments of infantry, an ambulance company, a field hospital and a company of Signal Corps troops. Shortly after the arrival of these troops the morbidity

TABLE 1.—INCIDENCE OF PNEUMONIA, NOGALES DISTRICT

Organization	No. Cases	No. Deaths	Per Cent. Mortality
First Alabama Infantry	63	17	26.9
Second Alabama Infantry	28	4	14.3
Fourth Alabama Infantry	28	6	21.4
First Alabama Ambulance Company	2	0	
Alabama Signal Corps (one company).....	2	0	
Total	123	27	21.9
Medical Department, U. S. Army.....	3	0	
Twelfth U. S. Infantry	8	1	12.5
Battery D, Sixth U. S. Field Artillery	1	1	100
First U. S. Cavalry (two troops)	1	0	
Battery C, First California Field Artillery.....	1	0	
Second Squadron, First Utah Cavalry	2	0	
Second Idaho Infantry	1	0	
Total	17	2	11.6

rates began to soar. During November, acute respiratory infections—colds, grip and bronchitis—reached epidemic proportions, and the base hospital was taxed to its full capacity. In December, pneumonia and measles became epidemic and continued until March.

* From Department Laboratory No. 3, Southern Department, U. S. Army, Nogales, Ariz.