Electrocardiography: Its Clinical Application

By GEORGE V. PARSONS, '40

IN recent years there has been great progress in electrocardiography. It has advanced from a method of physiological research to an indispensable aid in the diagnosis of certain cardiac disorders, such as myocardial infarction and arrhythmia. It ranks second only to history taking and physical examination, often surpassing these in importance. Although a thorough knowledge of the large amount of material accumulated is only possible to cardiologists, yet there is a number of facts, the value of which can be easily appreciated if one has the opportunity of carrying out or witnessing studies on patients under his care.

In 1856 Kolliker and Muller discovered that electrical changes accompanied each heart beat, but it was not until 1887 that Waller demonstrated that changes in potential at the surface of the human body were sufficient to produce records of the heart beat. The recording instrument then in use, the capillary electrometer, was inaccurate and curves obtained had to be corrected. Despite this disadvantage, progress was made in the analysis of electrocardiograms. In 1903 Einthoven introduced the string galvanometer, then electrocardiography came within the reach of clinical medicine. The study of the arrhythmias, in which Lewis played a prominent part, first held the field, but, as time went on, more attention was applied to changes in the ventricular complexes, until today there are established the diagnostic signs of coronary thrombosis, and evidence highly suggestive of other disturbances in coronary circulation. In addition to the three standard limb leads, precordial leads have been introduced and in some clinics have become a routine, while oesophageal leads are gaining a place for valuable information they may give.

Exact information is available in the study of arrhythmias, and some of these cannot be diagnosed without the aid of electrocardiograms. In bradycardia, the distinction between S-A block, A-V nodal rhythm, and A-V heart block is impossible unless the auricular sound or auricular pulsations in the jugular veins can be identified with certainty. Auricular fibrillation rarely needs electrocardiographic confirmation, but such a study is necessary to show the presence, in addition, of bundle branch block, or sometimes coronary artery disease. The effect of digitalis therapy may be followed closely in this way, and if the rhythm returns to normal, any degree of A-V heart block can be determined.

Bundle branch block has been found to be of three types; functional, transient, and permanent. So-called functional block occurs in young individuals prone to attacks of paroxysmal tachycardia and is associated with a short P-R interval. It is usually transient and a normal rhythm is sometimes established by inhibition of vagal tone. The Q-R-S complex

shows delayed conduction to be in the right branch of the bundle of His. The transient type occurs in older persons suffering from cardiac disease, and is most likely to occur during periods of decompensation, or following coronary thrombosis. Permanent bundle branch block is the most common and more prevalent than previously suspected. Bayley[1] in 172 cases found block of the left bundle in 103, and of the right bundle in 70. Of the 156 case histories available, 136 showed hypertension or arteriosclerosis. In only one case was block suspected on physical signs. The changes in the Q-R-S complex in what was considered left bundle branch block are now interpreted as indicating right bundle branch block and vice versa. The complexes vary considerably but the only distinguishing sign, in the limb leads, is the presence of a conspicuous S wave in lead I in right bundle block.[1, 19.]

Herrick,[8] in 1919, was the first to co-relate electrocardiographic signs with coronary artery thrombosis; about a year later Pardee[15] published his classical signs of cardiac infarction; Parkinson and Bedford[14] first differentiated the T_1 and T_3 types of electrocardiograms found in this disease. T_1 type is said to be found in infarction of the anterior or lateral wall of the left ventricle, while the T_3 type is found when the necrosis is situated in the diaphragmatic wall of the heart. The former shows first a displacement upward of the RS-T segment in lead I with a corresponding junction in lead I is negative, and positive in lead III. T_1 is upright while line is the rule, though all evidence of the T wave may be absent during the first day or two. T_3 is always upright. In the T_3 type, the RS-T junction in lead I is negative, and positive in lead III, T_1 is upright while T_3 is inverted. Lead II in each type follows the configuration of lead III more frequently than that of lead II. The changes in the RS-T junction pass off within two to three weeks, but the changes in the T waves become more marked and are fully developed by this time. They persist several months, showing some change toward normal in about two to three months. Completely upright T waves do not occur for a longer time although they may be normal in a shorter interval or may last several years.

The common changes in the Q-R-S complex are lowering of the voltage and some widening. Recently, importance has been attached to the appearance of an initial downward deflection on Q wave in the leads that have the T waves inverted. As these changes frequently outlast changes in the T waves, the diagnosis of old cardiac infraction may be made more certain by electrocardiograms. Durant[6], under the direction of Wilson, has carried out a number of studies along these lines. He found (in the absence of right ventricular preponderance) when Q_3 was 50% of the largest Q-R-S deflection and Q_2 at least equal to R_2, that 82% gave evidence of coronary disease; if, in addition, T_2 and T_3 were inverted the percentage was 100. When Q_1 was 20% of the greatest R and at least 1mm. with no R_1 74% had coronary disease; when T_1 was inverted, with these changes, the percentage increased to 93. Wallace[17] found that Q_3 greater than 5 mm. was

suggestive of serious myocardial disease and that the size of the Q wave was more significant than its size in relation to other parts of the Q-R-S complex. Pardee[16] found Q_3 waves in only two of 277 normal electrocardiograms, 73% of his cases with Q_3 50% of the greater R was suffering from angina pectoris. Q-R-S complexes in lead II, not greater than 5mm. and M or W, shaped throughout conforming to the other criteria laid down by Edeiken and Wolferth[7], are evidence of serious heart involvement, and may develop after coronary thrombosis. They found that a poor prognosis may be given in patients showing this type of tracing. Parkinson's electrocardiograms published in Heart in 1928 show several such type of lead III[4].

During the early days after coronary thrombosis, changes occur quickly, and in this stage normal electrocardiograms may be found. Holland and Levine[9] demonstrated a number of cases showing no changes a few hours to seven days after the accident, but typical changes later. One case showed return to normal on the fourth day when changes were significant on the second day. They stressed the importance of repeated studies to reduce this small percentage of error when electrocardiograms are normal, or show only slight changes in the early days following coronary thrombosis.

Precordial leads are important in the diagnosis of cardiac infarction when limb leads are equivocal. The technique consists in placing an electrode over the apex and pairing it with another on one of the limbs, or with high resistance electrodes on all the limbs. Depending upon the connections with the electrometer, the initial deflection may be upright with an upright T wave, or downward with an inverted T wave in the normal person. Changes in the direction of the initial deflection with reversal of the T wave, and a large initial deflection with increased size of of and sharp T wave are said to occur with anterior and posterior wall infarction respectively. These leads also aid in the diagnosis of bundle branch block and hypertrophy. The precordial lead, or lead IV, is taken simultaneously with the standard lead I. Abnormal delay of the onset of the intrinsic deflection, or downstroke of the R wave, relative to the same deflection in lead I, (provided R is upright) indicates delay in conduction of the impulse from the endocardial to the epicardial surfaces. Leads taken from the right side of the heart in right bundle branch block block or right ventricular hypertrophy show this abnormality, while in leads from the left side this does not occur. The reverse is seen in left bundle branch block and left hypertrophy.

Leads with an electrode in the oesophagus making contact with the auricles, or with the posterior surface of the left ventricle, are sometimes useful in elucidating obscure points. P waves are prominently brought out, and in posterior wall infarction, Q-R-S complexes and T waves may show changes not appearing elsewhere. The tracings are similar to those obtained in precordial leads in anterior wall infarction.

Clinical findings should not be depreciated for the electrocardiogram. Neither should the significance of the latter be underestimated. The one

adds to the value of the other. Cardiac efficiency is determined by clinical means, but changes in the myocardium and the conducting system of the heart may be diagnosed, and their course followed with greater accuracy by the electrocardiogram than by any other method.

This study was undertaken through the kind courtesy of Professor Kennth A. MacKenzie. His constant encouragement and helpful criticism are gratefully remembered by the writer.

REFERENCES

1. BAYLEY, R. H.—The Frequence and Significance of Right Bundle Branch Block. Am. J. Med. Sciences, 188: 236, 1934.
2. BOHNING, A. & KATZ, L. M.—The Four Lead Electrocardiogram in Coronary Occulsion. Am. J. Med. Sciences, 189: 833, 1935.
3. BROWN, W. H.—A Study of the Oesophageal Lead in Clinical Electrocardiography. Am. Ht. J., 12: 1 and 307, 1936.
4. CHEN-LANG TUNG—Functional Bundle Branch Block, Am. Ht. J., 11: 89, 1936.
5. COHN, A. E. et al—Influence of Digitalis on T Waves of the Human Electrocardiogram. J. Expt. Med., 21: 593, 1915.
6. DURANT, T. M.—The Initial Deflection of the Electrocardiogram in Coronary Disease. Am. J. Med. Sciences, 188: 225, 1934.
7. EDEIKEN, J & WOLFERTH, C. C.—Clinical Significance of M and W Shaped Q-R-S Complexes in Lead ii of the Electrocardiogram. Am. J. Med. Sciences, 188: 842, 1934.
8. HERRICK, J. B.—Thrombosis of the Coronary Arteries. J. Am. Med. Assoc., LXXII: 387, 1919.
9. HOLLAND, C. W. & LEVINE, S. A.—Limitation of the Electrocardiogram as an Aid in the Diagnosis of Coronary Occlusion. New England J. of Med., 206: 545, 1932.
10. HAMILTON, J. G. M. & NYBOER, J.—An Electrocardiographic Study Using Oesophageal and Precordial Leads. Am. Ht. J., 15: 414, 1938.
11. KOUTZ, W. B. et al—The Electrocardiogram in Revived Perfused Human Hearts. Am. Ht. J., 10: 605, 1935.
12. KUNTZ, C. M.—Transient Complete Bundle Branch Block. Am. Ht. J., 11: 212, 1936.
13. LEWIS, T.—The Mechanism and Graphic Registration of the Heart Beat. Shaw & Sons Ltd., London, 3rd Ed. 1925.
14. PARKINSON, J. & BEDFORD, C. D.—Successive Changes in the Electrocardiogram after Cardiac Infarction. Heart, XIV: 195, 1928.
15. PARDEE, H. E. B.—An Electrocardiographic Sign of Coronary Artery Obstruction. Arch. Int. Med., 26: 244, 1920.
16. IBID—The Significance of an Electrocardiogram with Changes in Q in Lead iii. Arch. Int. Med., 46:470, 1930.
17. WALLACE, A. W.—The Q Wave in the Electrocardiogram. Am. J. Med. Sciences, 188:498, 1934.
18. WHITE, P. D.—Heart Disease. The MacMillan Co., New York, 2nd Ed. 1937.
19. WILSON, F. N.—Recent Progress in Electrocardiography. Recording and Statistical Co., New York, 1938.
20. WOLFF, L., PARKINSON, J., and WHITE, P. D.—Bundle Branch Block with Short P-R Interval in Healthy Young People Prone to Paroxysmal Tachycardia. Am. Ht. J., 5:685, 1930.
21. WOOD,F. C. & WOLFERTH, C. C.—Huge T Waves in Precordial Leads. Am. Ht. J., 9:706, 1934.
22. Standardisation of Precordial Leads. Am. Ht. J., 15:107, 1938.

To Doctor Ted Thorne, of the interne staff of the Victory General Hospital our sympathy in his recent bereavement.