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CLINICAL ASPECTS OF THE ELECTROCARDIOGRAM

FRED FRICKE
CLINICAL ASPECTS OF THE ELECTROCARDIOGRAM

The electrocardiograph is an instrument of comparatively recent invention for use in the study of heart cases. Its purpose is to photograph the changes in electrical potential which take place in the heart during activity. It was known as early as 1856 that an electric current was produced by the contracting heart muscle (1), and in 1885 Kolliker and Muller (2) showed the presence of action currents in heart muscle by laying the nerve of a nerve-muscle preparation across a contracting heart, and observing the activity of the nerve muscle preparation. Two years later Waller demonstrated by means of a mercury galvanometer that the current produced by the active heart muscle could be lead from the surface of the body if a satisfactorily sensitive instrument was available to record it. Einthoven, of Amsterdam, made the recording of these heart currents possible by the invention of the exceedingly sensitive string galvanometer, which was first described by him in 1903. Sir Thomas Lewis (3) was the first person to use the electrocardiograph for clinical purposes, and published the results of his studies in 1906 and 1908. The use of the instrument was then rapidly taken up abroad, and was introduced in this country between 1910 and 1913.
The electrocardiograph (4) which employs Einthoven's string galvanometer consists of an electromagnet activated by a storage battery of five or six amperes which gives about ten volts. The "string" which is suspended between the poles of this electromagnet consists of a filament of platinum or silvered quartz about half as thick as the diameter of a red blood cell or two to four microns. When the current from the patient's heart passes along this string at right angles to the lines of force of the electromagnetic field the string will be deflected to one side or the other depending upon the direction of the current which is passing thru it. By means of an arc light passing thru a system of condensing lenses the shadow of the string is magnified, focused, and printed on a motor-driven plate or film. This photographic record of the heart action is known as an electrocardiogram. The string can be adjusted to a proper tension so that one centimeter deviation of the shadow will be caused by one millivolt of current. A single dry cell battery is used to counter-balance the skin currents such as those set up by glandular activity. Vertical lines are photographed on the plate to determine the time. This is usually done by means of a spoked wheel which revolves
at a constant known rate. Horizontal lines are also printed on the film so that the height of the shadows can be determined. The electrodes used to connect the patient with the electrocardiograph may be German silver, zinc or lead plates, or vessels filled with salt solution.

More recently the Victor electrocardiograph (5) has been placed upon the market and is now used quite extensively in place of the string galvanometer of Einthoven, it has less sensitive torsion galvanometer, consisting of a fixed U magnet with an enameled iron vane suspended by a phosphor-bronze ribbon, with a mirror attached to the vane which reflects the beam of light on to the photographic plate. This part of the electrocardiograph is known as an oscillograph. To compensate for the lesser sensitivity of this instrument amplifying tubes are used which multiply the heart current about three thousand times.

When the body is at rest the heart is practically the only active muscle present, so any current that is produced must come from the heart muscle (6). Therefore, electrodes placed anywhere on the body with the heart between them will register the electrical activity of the heart. In order to
make the work more uniform special parts of the body were used for attachment of the electrodes. These were the right and left arms which constitute lead I, the right arm and left leg which lead II, and the left arm and left leg which is lead III. Three different leads are used because each one gives a different result, since the total current of the heart is in the same direction for each lead, but the receiving apparatus is in a different relation to this current. Therefore, under abnormal conditions one lead may show certain characteristics not present in either of the other leads.

The action currents of the heart (1) which the electrocardiograph records are produced because the muscle fibers of the heart are not always all contracting at the same time, and the actively contracting muscle fibers acquire an electrical potential difference from that of the non-contracting fibers. Therefore, as long as there is a difference in activity of the heart muscle fibers there will be a difference in potential, and an electric current will be produced. However, if all of the muscle fibers are contracting at the same time they will all have the same potential and no current will be present. Since the electrocardiogram of
a given heart is normally the same during each cycle it can be assumed that the contraction always starts in the same place and travels over the same route, producing the same difference of potential. From experimental work the normal starting point (7) for the heart muscle contraction has proven to be a mass of speriolized muscle tissue in the wall of the right auricle near the superior vena cava, known as the sino-auricular node or S-A node. From this point the impulse, which precedes and causes the contraction, passes thru the auricular muscle, the auriculo-ventricular or A-V bundle or bundle of His, the right and left branches of this bundle, and finally to the entire ventricular wall thru the fine terminations of these branches, which are known as the fibers or corpuscles of Purkinje.

The electrocardiogram of a regularly beating heart (8) whose impulses rise at and travel over the usual route described above will present certain characteristics to be looked for in all electrocardiograms. The record of each cycle in any given lead for a single patient will appear exactly the same as every other cycle record in that lead. Minor variations may always be expected in records from different leads in the same patient, or in the same lead from different patients.
There are normally three upward curves to each heart cycle, and two very small downward curves of less amplitude and less constancy. The first upward curve is known as the P-wave, and represent the electrical disturbance associated with auricular systole. It is a rounded elevation varying in height from bare perceptibility to two millimeters. The second upward deflection is known as the R-wave, and consists of a sharply pointed or spiked deflection, from seven to sixteen millimeters, in height in the lead in which it is greatest. The R-wave is usually highest in lead II, always being equal to the sum of the R-waves in leads II and III. Th Q and S waves, if present, appear as sudden downward deflections of small amplitude just before and after the R-wave respectively. No pathological significance is attached to the absence of either or both of these waves. The Q, R, and S waves are usually spoken of as the QRS interval or complex, and represents the beginning of ventricular activity. The S-wave, after a period of inactivity, is followed by an upward deflection usually not over five millimeters in height, and with a comparatively rounded apex. This wave represents the end of ventricular systole.
the difference of potential resulting from the cessation of activity of the muscle, which does not all cease contracting at the same time. The distance between the S and T waves where the line is straight, which is known as an isoelectric period, is probably due to the entire ventricle being in contraction and, therefore, no electrical reaction is produced. The period from the beginning of the Q, R, S interval to the end of the T-wave represents Ventricular systole. The isoelectric period following the T-wave to the next P-wave occurs while the heart is completely inactive or during diastole. A very slight upward deflection known as the U-wave is sometimes seen during this period, but it is very inconstant and no significance is attached to its presence or absence. The period from the beginning of one P-wave to the beginning of the next P-wave represents a complete heart cycle. The space from the beginning of the P-wave to the beginning of the Q, R, S complex is known as the P-R interval, and normally is not over 0.20 of a second in duration. The duration of the Q, R, S interval is also of considerable importance, 0.10 of a second being the upper limit of normal. In interpreting an electrocardiogram direction, amplitude, duration, and time relations of the various
waves and intervals are important factors to note. The R-wave is related to the condition of the ventricular muscle, but often increases in amplitude after acute illnesses or with the over-activity of exophthalmic goiter, and decreases following prolonged severe physical exertion. The T-wave may be extra large or occasionally inverted in lead III in the normal record (9), but is always upwardly directed in leads I and II.

Abnormal electrocardiograms vary considerably in their appearance, and significance, depending upon the condition causing the abnormality. An arbitrary classification (7) of disorders in which the electrocardiograph is a useful aid will be given to simplify the matter of describing them. I Disorders of Rhythm. II Disorders of Rate. III Disorders of Conduction. IV Myocardial Disturbances. V Congenital Abnormalities.

One of the most common and simplest of the disorders of rhythm is sinus arrhythmia (1). It is a normal change in the pulse rate, and the variations being synchronous with the respiratory phases due to vagus pressure. The pulse rate increases with inspiration and decreases during expiration. It is seen in practically all children and in many adults. The electrocardiographic tracings are perfectly normal except for the regular variation in the rate produced (page 9)
by variations in the length of the diastole, or S-T interval. It is termed sinus arrhythmia because the impulse arises in the sinus or sino-auricular node. It is not a pathological condition, it produces no symptoms, and therefore, it requires no treatment.

The occurrence of extrasystoles (7) is also a common heart disorder, and is quite easily recognized both clinically, and by means of the electrocardiogram. Extrasystoles may be divided into three groups, namely, Auricular, Ventricular, and Nodal. An auricular consists of a premature impulse arising in the auricle and causing a complete heart beat. It is followed by a pause that is longer than normal, which is known as a compensatory pause. The electrocardiogram of this condition shows the T-P interval preceding the premature beat shorter than normal, and a prolonged T-P interval following the early contraction. The P-wave of the premature contraction may be normal, lost in the preceding T-wave, exaggerated, flat, or inverted, depending upon the prematurity and the point of origin of the impulse within the auricle. The interval of the heart cycle preceding the auricular extrasystole plus the premature beat is slightly shorter than
two normal beats.

In ventricular extrasystoles the premature impulse arises at some point within the ventricle itself. It is not influenced by the P-wave, nor is the normal auricular activity interfered with. The P-wave may be found just before the extrasystole occurs, or may be lost in Q, R, S complex of the premature beat, but it is always just where it would be normally if no extrasystole had occurred. The ventricular complex, however, is distorted because the impulse arises below the normal level. The Q, R, S interval is increased in duration over 0.20 seconds, the largest deflection is increased in amplitude, and there is usually thickening or slurring of the limbs. The T-wave is exaggerated both as to time and amplitude, and it points in the direction opposite that of the greatest deflection of the Q, R, S Group. Since the entire Q, R, S, and T complex is not increased in length the S-T interval is practically absent. The ventricle from which the impulse for the extrasystole arises can usually be determined by the directions of the greatest deflections of the Q, R, S groups in each of the three leads. If the impulse starts in the right ventricle the greatest deflection of the
Q, R, S group is downward in lead I and upward in leads II and III. If the impulse arises in the left ventricle the greatest deflection of the Q, R, S group is upward in lead I and downward in leads II and III.

Nodal extrasystoles are those arising in the auricular-ventricular node. Since this node is at the junction of the auricles and ventricles the P-wave and Q, R, S group will occur practically simultaneously. The portion of the node from which the impulse arises determines the exact time relationship of the auricular and ventricular curves. If the impulse arises in the upper part of the node the auricular contraction will just precede the ventricular contraction. If the impulse arises near the middle of the node the auricles and ventricles will contract together and the P-wave will be lost in the Q, R, S group. If the lower portion of the node gives rise to the impulse the ventricle will contract first and the P-wave will be found just after the Q, R, S complex. In any of these conditions of nodal extrasystoles the impulse arises above the ventricles. Therefore, the Q, R, S complex is always normal except when distorted by a synchronous P wave. The P-wave if found, is usually inverted or distorted in some way because
the impulse comes from the lower instead of the upper part of the auricles. Infrequently the auricular-ventricular node replaces the sinus node completely as the point of origin for the cardiac impulses (10). This condition is known as nodal rhythm, and if persistent indicates destruction of the sino-auricular node and may, therefore, be considered as evidence of considerable myocardial damage. The electrocardiogram in this condition shows a slow regular pulse of 35 to 50 beats per minute with all of the cycles showing one of the three types of abnormality just described as nodal extrasystoles.

Extrasystoles may occur in normal hearts as the result of some stimulus such as coffee, alcohol, drugs, etc. In these conditions they are usually infrequent and not associated with any other abnormalities. If they occur frequently, in an irregular manner, or associated with other abnormalities they are usually due to some organic heart disease, and treatment should be directed toward the removal of the underlying pathology (11).

A condition in which the electrocardiograph is a valuable aid in diagnosis is auricular flutter. In this condition the impulses arise at a greatly increased rate,
varying from 230 to 350 per minute. Their origin is some point in the auricle near the superior vena cava, but outside the sino-auricular node, and they travel in a circular course about the vena cavae. The P-waves are usually diphasic, especially in lead II, because of the unusual course of the impulse. The ventricular complexes are normal because the impulses arise above the auricular-ventricular node. There is practically always some block associated with auricular flutter, the most frequent type being 2:1 block, producing a ventricular rate one-half that of the auricle. A 3:1 block, 4:1 block, etc., are occasionally seen also, and infrequently the ratio of the auricular beats to the ventricular beats vary, producing what has been termed impure flutter (13). Very rarely each auricular impulse reaches the ventricle, and the ventricle beats as rapidly as the auricle. This excessively rapid ventricular rate warrants a poor prognosis since heart failure is likely to follow this over-exertion. The etiology of auricular flutter (11) is not known, and associated cardiac pathology is not constant. The treatment, however, is often very successful, even if the condition has lasted for months, as not infrequently happens. Digitalis is the drug of choice, and at first it slows the ventricular rate, but soon produces
auricular fibrillation. If the drugs is not discontinued the rate very often returns to normal. Quinidin may be used in the same way alone or with digitalis.

Auricular fibrillation (13) is a more frequent condition which resembles flutter slightly. The impulse arises in the auricular muscle and takes a circular course, but unlike flutter the course is not constant, and it is supposed that impulses also pass off at a tangent to the circular path. The rate of these impulses although irregular due to their varied courses vary from 350 to 500 or more per minute. These impulses occur so rapidly that the auricle does not have time to respond to each one and remains in a state of fibrillation. The impulses which reach the ventricle are variable as to number, and markedly irregular. The most characteristic feature of the electrocardiogram of the fibrillating patient are the irregularity of the ventricular complexes and the absence of normal P-waves. The ventricular complexes may be normal or the R-waves may vary in height from cycle to cycle. The auricular activity may be manifest on the electrocardiogram by fine twitchings seen during ventricular diastole and known as fibrillary or F-waves.
Auricular fibrillation is generally found with organic heart disease, especially rheumatic or mitral disease and toxic goiter. The prognosis of this condition with mitral disease is not favorable as a rule, although if there is little other evidence of muscle damage the patient may live for some time if properly treated. Auricular fibrillation associated with thyroid disease is less serious, but its presence increases the seriousness of the thyroid condition (12). The habits of the patient (11) must be regulated, and digitalis is the chief drug to lessen the rate of the over-marked ventricle. It does this by impeding the passage of the impulse from the auricle to the ventricle, or increasing the heart block. Quinidin is also very popular in the treatment of this condition, and often gives excellent results.

Disturbances of rate can be very accurately diagnosed by means of the electrocardiogram (14). If a bradycardia or tachycardia is present the electrocardiograph will determine whether it is due to a disturbed conduction mechanism within the heart itself, or to some external cause. In the first instance some abnormalities in one or another of the waves will be present, while in the latter case the cycles will be
normal but either farther apart or closer together than usual.

The most important pathological disturbance of the heart rate is the condition known as paroxysmal tachycardia (7). It consists of a sudden increase in rate, and a sudden return to normal, the attack lasting from a few seconds to several days or even two weeks. The shorter intervals are more common, a few hours being the most frequent length. The impulses producing this condition are ectopic in origin, that is, they do not arise at the sino-auricular node. Auricular ectopic impulses are most frequently responsible for the paroxysms, but they may also arise in the auriculo-ventricular node or in the ventricle. Since each beat during the attack is an extrasystole the electrocardiographic curves will simulate those described earlier for extrasystoles. The rapid rate will naturally place the heart cycles very close together, and the origin of the impulse may be hard to determine unless the beginning or the end of an attack is included in the record.

The etiology of this condition is very indefinite. In one series of cases (11) approximately 22% were associated with mitral stenosis, 22% with symptoms of myocardial degeneration, 6% with arterial disease including angina pectoris,
6% with renal disease and cardiac enlargement, 2% with thoracic aneurism, 2% with early pulmonary tuberculosis, and 56% without any other symptomatology. The symptoms associated with paroxysmal tachycardia vary considerably depending upon the length of the attack and the heart rate during the paroxysm. If the attack is of very short duration the patient may not be aware of it at all, whereas, in severe attacks complete incapacitation may be experienced due to precordial pain, palpitation, exhaustion, coldness, sweating and gastrointestinal symptoms. The prognosis of this condition, while it should be guarded, is not very grave. Death has been reported due to congestive heart failure during an attack, but the great majority of cases recover. Complete freedom from recurrent attacks cannot be promised, although in young patients the prospects for it are fair. No treatment has yet been found which constantly gives good results, although a great many things have been tried.

The electrocardiogram is very helpful in the classification and diagnosis of the disorders of conduction, or heart block. A rather infrequent type of this condition is known as sino-auricular block or dropped beats (14). The impulse arising at the sino-auricular fails to get to the auricular muscle or fails to arise at all. Therefore, the
auricular muscle does not contract nor does the ventricle contract. Therefore, there are no curves on the electrocardiogram for a short period. The interval between the two P-waves which includes the isoelectric period is slightly less than two normal cycles. The etiology of dropped beats is unknown, although they occur more frequently when the heart rate is rapid. They produce no symptoms and no treatment is required. A clinical diagnosis of dropped beats is frequently made from the compensatory pause following extrasystoles.

A much more common conduction disorder is auriculo-ventricular heart block (7). It consists of some interference with conduction at the auriculo-ventricular node or bundle of His, and may be divided into incomplete and complete block. The incomplete or partial block may be only severe enough to slow the impulse, which is demonstrated on the electrocardiogram by a prolonged P-R interval of more than 0.20 seconds. If the condition is slightly worse an occasional impulse may fail to reach the ventricle, and the Q, R, S, T complex will be absent after the P-wave. More commonly the failure of the impulse to reach the ventricle occurs regularly. The ratio of impulses present (auricular contractions) to those which
reach the ventricle may be 2:1, 3:1, 4:1, etc., the condition then being termed 2:1 block, 3:1 block, etc. These more severe types of incomplete block are usually associated with auricular flutter. The lesser incomplete block may result from drugs such as digitalis and morphin, or from the toxic condition accompanying such diseases as pneumonia, diphtheria, and rheumatism. The cardiac condition itself is not harmful, and treatment should be directed toward the removal of the cause.

Complete heart block is a condition in which there is complete blocking of the passage of impulses from auricle to ventricle. An independent impulse then arises within the ventricle itself, the rate of these impulses usually being slower than that of the auricle, and entirely independent of it. The ventricular rate may be below fifty with an auricular rate of over one hundred. The picture of complete block presented by the electrocardiogram consists of normal regularly recurring P-waves and a smaller number of normal, regular recurring Q, R, S, T complexes, with no definite time relations between the two. Complete heart block (13) may be the result of more advanced toxic conditions
similar to those causing partial heart-block, namely, drugs or certain infectious diseases. Among the organic lesions which might be responsible for complete heart block are atherosclerosis, gumma, calcified areas, infarcts, and coronary disease. The symptoms and treatment will depend upon the etiological factor.

A block of either one of the main branches of the bundle of His will produce a quite characteristic electrocardiographic tracing. The P-wave will be normal, but there will be a prolonged Q, R, S interval of more than 0.10 seconds, there will be notching or slurring (thickening of the limbs) of the major deflections of the Q, R, S group, and a T-wave directed opposite to the largest deflection of the Q, R, S group. If the block is complete these abnormalities will be marked in all three leads. If the block is only a portion, or appears farther from the bundle of His on one of the small branches (arborization block) the finding on the electrocardiogram will not be as extensive. The ventricle in which the block occurs can usually be determined by the directions of the largest deflections of the Q, R, S complex. In right-sided block the largest deflection will be downward in lead
III, and in left-sided block the largest deflection will be downward in lead I. The etiological factor of bundle branch or arborization block is more frequently coronary disturbance, but its significance is similar to that of other types of heart block.

A discussion of myocardial disturbances may be divided into preponderance, myocardial impairment, and coronary artery obstruction. If one side of the heart becomes abnormally large the direction of the current generated by the muscle contraction will be changed (7), and definite changes will be seen on the electrocardiogram. If the right ventricle is enlarged the main deflection of the Q, R, S group in lead I will be downward, and with enlargement of the left side of the heart. The greatest deflection of the Q, R, S group will be downward in lead III. More recent investigation (14, 15) has shown that these abnormalities are not always due to enlargement of one side of the heart, but may occur in normal hearts. Electrocardiographic tracings characteristic of right-sided preponderance may occur in long, narrow individuals in whom the apex of the heart is more toward the right; and records simulating left-sided preponderance may be produced from short, stocky individuals whose hearts are more transverse
within the chest than usual.

Myocardial impairment per se cannot be diagnosed by the electrocardiograph. It is suggested, however, by various abnormalities (16) of the ventricular complex, such as a widened Q, R, S interval, notched or slurred deflections, and various T-wave changes, especially a negative T-wave. Low voltage or small deflections of the waves was at one time considered significant of poor muscle function, but this relationship has not proven constant (14, 17).

An indirect evidence of myocardial damage is seen in the electrocardiographic records suggesting coronary artery obstruction (18). The Q, R, S Group is notched in at least two leads, left ventricular preponderance is usually shown, the T-wave starts from above the zero line and quickly turns away from the starting point without the normal isoelectric period (the Pardee curve), and the T-wave is usually directed downward in lead II and one other lead. The history symptoms and other clinical findings must be considered along with the interpretation of the electrocardiogram.

The only congenital condition (14) which gives a constant, significant electrocardiogram is dextrocardia with a change in the relative positions of the heart cavities.
In this condition the waves in lead I are all pointing in the opposite direction from the normal. They appear as mirror-writing on the record. If the relationship of the cavities is not disturbed in a dextrocardia the electrocardiogram will be normal.

The electrocardiogram is not of any appreciable value in the diagnosis of value lesions. It may show preponderance of one of the ventricles, but this evidence is not always reliable, and the x ray or fluoroscope will diagnose enlargement more accurately and more easily (14).

From a purely prognostic standpoint it was found by careful tabulation of cases (13) that a negative T-wave in the electrocardiogram is very important. As the close of this paper a few of the figures on negative T-waves will be given. Of 347 patients with mitral stenosis but without negative T-waves 37% were dead (cardiac deaths) within 16 months, while of 43 patients with mitral stenosis, but with negative T-waves 63% were dead within 12 months. Of 200 patients with rheumatic aortic insufficiency without negative T-waves 39% were dead within 16 months, while of 62 patients with rheumatic aortic insufficiency plus negative T-waves
56% were dead within 15 months. Of 137 patients with syphilitic aortic insufficiency without negative T-waves 46% were dead within 16 months, while of 42 patients with syphilitic aortic insufficiency with negative T-waves 76% were dead in 11 months.
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