Electrocardiography

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ELECTROCARDIOGRAPHY

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ELECTROCARDIOGRAPHY

"Graphic records have now an established place in the studies of the heart. That place is to supplement clinical examination. While it is true that no examination of the heart at the present time can be considered fully complete unless it includes graphic study, it is equally true that a clinically satisfactory diagnosis can, in the majority of patients, be reached without graphic study; hence, cardiography is not the 'sine qua non' in diagnosis. Heart records can only supplement, they can never supplant, the painstaking clinical examinations of today which have evolved from the careful observations of generations past. One of the valuable contributions of cardiography to medicine lies in the fact that familiarity with graphic methods quite invariably sharpens the perception of the physician so trained and aids in the clinical recognition of heart affections."

S. Calvin Smith.
THE IMPULSE MECHANISM OF THE HEART

HISTORY: - An electrocardiogram is a record of the pathway taken by the excitation wave of a heart cycle as it courses along the conduction system from its origin in the sino-auricular node to the terminal Purkinje fibers. Hence, a conception of electrocardiography should have as its basis the anatomy and physiology of the impulse mechanism.

As early as 1773, we find John Walsh and John Hunter (1) conducting experiments in animal electricity with the torpedo or electric ray fish. History mentions that the Greeks and Romans used these fish for therapeutic measures indicating that electrical phenomena were recognized even before the time of Walsh and Hunter. However, the story of electrocardiography really begins some twenty years later with the epoch making experiments of Luigi Galvani on muscle nerve preparations. Galvani (2) in 1790, while preparing frog legs for experimental purposes, hung several upon copper hooks and suspended his hooks upon an iron wire. Noticing the twitchings of the leg muscles as he hung the hooks, led him to devise what he called his "metallic arc" of two different metals. When one metal was connected to the nerve and the other to
the muscle of his frog legs, a contraction of the latter resulted. In reality, Galvani had constructed a minute electric cell and had demonstrated that an electrical stimulus would cause muscular contraction. This shrewd observation opened the field for future physiological research.

Many experiments followed Galvani's discovery both upon intact muscles and excised muscle nerve preparations. At this time, the heart was considered in the same category as the skeletal muscles. The contractions of the organ were explained by the "neurogenic theory", that is, being under direct control of the central nervous system with which it had connection through its nerve trunks.

Johannes E. Purkinje (3) perfected his microtome in 1839 and was the first to describe certain peculiar muscle cells throughout the cardiac wall. These fibers today bear his name. The innervation of the heart was investigated by Henle in 1841, and by Friedrich Bidder (3) in 1852 with the discovery of the gaulgionic cells at the junction of the auricle and ventricle. Having the nerve trunk to the heart well established, vagus, the gaulgia of Bidder seemingly fortified the neurogenic theory of cardiac contraction.

However, in 1856 Kolliker and Müller (4) working independently of each other, proved almost simultaneously
that the heart beat was accompanied by an intrinsic electrical discharge. They laid the nerve of a muscle nerve preparation upon the beating ventricle of an exised mammalian heart and observed that with each ventricular contraction, the muscle of their preparation was activated. Thus a heart completely removed from the animals body was stimulating the frog leg, proving that the heart contained its own impulse mechanism and was entirely independent of the central nervous system for such, "myogenic theory" (5).

Having established the proof that the heart's stimuli were autonomic in nature, the next step was to discover the origin and mode of spread of the impulse to the various portions of the cardiac musculature. Hermon Stannius (6) had shown in 1852 that ligatures placed at the junction of the auricle and sinus venosus brought the heart to a standstill, while a second ligature, applied to the auriculo ventricular groove, caused the ventricle to beat again. According to the neurogenic theory then accepted, the effect of the ligatures was supposed to be due to inhibition of the ganglia of Bidder. But after the demonstrations of Kolliker and Muller in 1856, this theory had to be abandoned leaving the phenomenon of Stannius's ligatures unsolved.
Albert von Bezold demonstrated the accelerator nerves of the heart and their origin in the spinal cord in 1862. Much experimentation followed and Gaskell (7) published his great memoir on the musculature and innervation of the heart in 1881. In this he showed that the motor influences from the nerve ganglia in the sinus venosus, Bidder's ganglia, influenced the rhythm, rate, and force of the heart but did not originate its movements or beat. This he explained as being due to the automatic rhythmic contractile power of the heart muscle itself and to the peristaltic contraction wave which proceeds from sinus venosus to bulbus arteriosus and from muscle fiber to muscle fiber. By 1883, Gaskell and Englemann had proved, using criss-cross incisions on excised hearts, that cardiac impulses are conducted by muscular pathways. In 1892, Stanley Kent, and in 1893 Wilhelm His, Jr., discovered a narrow band of muscle, an embryonic rest between the auricles and ventricles. This is now known as the auriculo-ventricular bundle of His. More recently, Arthur Keith and M. Flack, 1907, discovered a tissue rest of fine, pale, faintly striated fibers in the heart wall in the region of the superior vena cava. This tissue is richly supplied with arterioles and is intimately connected with the Purkinje fibers and nerve
Conduction System of Heart

Showing approximate times of spread of impulse over auricles and ventricles.

Wiggers
terminals. From experimental data, this bit of tissue has been called the "pacemaker of the heart", the sino-auricular node. In 1908, S. Tawara (8) traced out the ramifications from the His bundle and discovered another muscular node in close relation to it, the atrio-ventricular node.

These histological findings have been supported by a mass of clinical and experimental data which brings us to the present concept of the impulse-conduction system of the heart. This concept has the impulse arising in the sino-auricular node, according to Heatherley (9), as part of the energy liberated by the breaking down of a highly complicated molecule which is produced most freely by cells in the sino-auricular node and in a lesser degree by all of the other heart muscle cells. Part of this energy takes the form of an electric disturbance which spreads as a wave preceding the wave of contraction. We find this electrical wave spreading from this node in all directions over the auricular musculature and activating that organ; entering the auriculo-ventricular node, passing to the bundle of His, over its right and left branches, out to their minute terminals, whose ends are the Purkinje fibers; and finally activating the cardiac musculature of the ventricle which lies in close relationship with the fibers of Purkinje.
Development of The Heart

Fig. 1 - Model of Early Human Heart, Ventral View. About 11 somite age.

(Arey after His)

Fig. 2 - Model of Human Heart. 10mm. Embryo.

(Arey after His)
EMBRYOLOGY: - To thoroughly comprehend the anatomy of
the heart, it is necessary to review briefly the embryolog-ical development of that organ. Arey (10) in his
text, Developmental Anatomy, describes the heart as arising from two lateral halves in the blastoderm which swing
together and secondarily unite. Even before the fusion
takes place, two constrictions are evident marking the
future regions of atrium, ventricle, and bulbus. Soon
a fourth division, the sinus venosus, arises by construc-
tion from the hind end of the atrium. The cardiac tube
grows more rapidly than the pericardial cavity which
throws the tube into a bend and finally a spiralled S
shape results. Subsequent growth places the atria cephalad
and the ventricles caudal. Overgrowth and differentia-
tion of parts results in a rapid development of ventricles,
auricles, etc., as seen in the adult heart.

The myocardium is at first continuous over the entire
surface of the heart but soon becomes divided by connect-
tive tissue at the atrio-ventricular canal and leaves only
a bridge there. This connecting strand of modified mus-
cle tissue is located behind the posterior endocardial
cushion and is called the atrio-ventricular bundle, Bun-
dle of His. By analogy with other vertebrates, it is
supposed that the human heart begins to beat during the
fourth week when the embryo is two millimeters or less
in length. At that time the rhythmic contractions are purely muscular phenomena since the nerves first invade the heart several weeks later.

HISTOLOGY: - In mammals, microscopical examination has found special muscular elements in those portions of the heart muscle in which the contractile impulse originates. On the boundary between the right atrium and the superior vena cava, in the region of the sulcus terminalis, is a special area one centimeter in length and from three to five millimeters in width. Although not sharply outlined, it can be seen with the naked eye. It consists of a very dense network of twisted, atypical muscle fibers. These differ from the surrounding cardiac muscle fibers in their thinness, richness in sarcoplasm, few cross-striations, and often by their considerable content of glycogen. Between these fibers is a dense interstitial connective tissue. The fibers themselves are thought by most investigators to continue everywhere directly into the circular, cross-striped fibers in the wall of the vena cava, as well as into those of the atrial myocardium. This area is called the sino-auricular node; in its atypical muscle fibers is the seat of the autonomic origin of the rhythmic, contractile impulse of the heart. It is also the first
The Spread of The Excitation Wave
In The Auricle. Concentric Waves
Follow The Chief Muscle Bands.
(After Smith)

The Conduction System of The Heart.
(After Smith)
part of the conducting system.

The question now arises as to how the impulse travels to other parts of the heart. Does it travel along the muscle fibers or has it special nerve tracts?

Maximow (11) states that in the mammalian heart, including man, the musculature of the atria is apparently separated from that of the ventricles by the fibrous rings of the atrio-ventricular openings and the trigona fibrosa. However, there is a muscular connection between these chambers in the form of the conduction system formed by the Purkinje fibers. Throughout the atria and most of the ventricles there is a dense, interlacing network of atypical cardiac muscle fibers first described by Purkinje in 1839. These peculiar fibers are aggregated into two-node-like masses; one the above mentioned sino-atrial node, the other the atrio-ventricular node. The structure of the fibers is the same in both the network and the two nodes. The sino-atrial node has been described above.

The atrio-ventricular node in man is a flat, white structure about six millimeters long and two to three millimeters wide. It is located in the posterior lower part of the septum atriorum under the posterior aortic valve. The node consists of peculiar muscle fibers rich in sarcoplasm and poor in fibrils. These ramify and
interlace in all directions, forming a tangled dense network whose meshes are filled with connective tissue. Toward the atrium, these fibers pass into or between the usual myocardial fibers. Accordingly the boundary of the node is very indistinct over much of its periphery. Toward the ventricles the substance of the node contracts abruptly into a sharply defined shaft about one centimeter long. This is the atrio-ventricular bundle. It is located in the dense connective tissue of the trigonum fibrosum and continues into the interventricular septum where it divides into two branches. The first branch, a cylindrical bundle one to two millimeters thick, runs downward along the posterior surface of the septum and is located in part directly under the endocardium of the right ventricle. It proceeds along the interventricular septum to the beginning of the trabecule septomarginalis where it splits into many branches. These spread along the entire internal surface of the right ventricle and along the papillary muscles of the trabeculae carnae. All of these anastomosing and splitting branches form a network with rounded meshes whose threads mix with the substance of the myocardium and become invisible.

The left branch is a rather wide, flat band which comes forward under the endocardium of the left ventri-
cle in the upper portion of the interventricular septum, under the interior edge of the posterior cusps of the aortic valve. At first it divides into two main branches at the border between the upper and middle threads of the septum. Then it separates as in the right ventricle into numerous, anastomosing, interlacing, thin threads which run in various directions and are last to view in the myocardium.

This system of conducting fibers, even up to the terminal ramifications in the ventricles, is covered with a connective tissue membrane which separates it from the remaining muscular mass of the heart. In the region of the main stem of the His bundle and its two immediate branches, this membrane is so thick and dense that it can be dissected out microscopically as a separate distinct sheath. Due to its loose connection with the conducting fibers, it has been possible to inject this sheath thereby demonstrating its continuity from the ariculo-ventricular node to its terminals in the myocardium.

Hence, we have a definite conducting system in the heart composed of modified muscular tissue. It has been repeatedly demonstrated histologically, confirmed by post mortem examinations, and proved by physiological experimentation that this system serves for the autonomic origin
of the impulse as well as for its distribution to the entire myocardium.
BRIEF SKETCH OF ELECTROCARDIOGRAPHY

HISTORY: - Electrocardiography is based upon the fundamental physical fact that a current flows from a point of greater to one of lesser potential. The impulse, arising in the sino-auricular node, presumably a result of a biochemical phenomenon, flows along the conducting system toward the terminals which are points of lesser potential than the node. It is the record of the course taken by the impulse in its passage along the conducting system that we call the electrocardiogram.

Electrical phenomena in animals had long been familiar to the Greeks and Romans who used them for therapy. But the first attempt to explain these biological phenomena is found in the experiments in electrophysiology by John Hunter (1) in 1773 upon the electric ray fish. The first real advance, the birth of systematic ordered physiology goes to Luigi Galvani (2) who in 1790 demonstrated a frog nerve-muscle preparation activated with a minute electrical cell. The flow of electrical currents from points of higher potential to those of lower was first observed by Nobili (12) in 1824 shortly after his invention of the galvanometer. This instrument armed the laboratory worker with an indispensable piece of equipment for research in muscle physiology. The next
step forward was taken by Carlo Matteucci in 1842 (13) when he demonstrated the rheoscopic frog effect. He placed the sciatic nerve of one frog leg upon the exposed muscle of the opposite leg. By stimulating the sciatic nerve of the second leg, he caused a contraction in both legs. Thus he showed that the first leg had not contracted from a stimulus applied directly to its nerve but from a current of action transmitted through the second in consequence of its contraction. Finally came Kolliker and Müller's (4) discovery in 1856, that the beating excised heart produces impulses sufficiently strong to cause contraction of a rheoscopic frog leg. This proved the autonomic origin of the impulse.

In 1878, Sanderson and Page (13) demonstrated currents produced by the heart's action with a Lippmann capillary electrometer. This instrument is a very sensitive column of mercury which registers minute differences in potential. However, the inertia of the mercury column is too great to give the accurate contour to the waves caused by the electrical discharge; the equipment was extremely temperamental, failing without apparent reason at the most inopportune moments; and the heart had to be contacted directly which excluded it from any possible clinical application.

The solution to the lead difficulty was found in
1887 by Waller (14) who demonstrated that satisfactory records could be obtained with the capillary electrometer by placing zinc plates on the external surface of the chest wall. He connected a pair of zinc electrodes, moistened with salt solution, and held in place by chamois skin, to the front and back of the chest and made the necessary connections with his Lippman electrometer. A stylus device, attached to the mercury column of the electrometer, made contact with a smoked drum and recorded the movements of the mercury. Waller, likewise, incorporated a time device and a simultaneous cardiographic apparatus in his set-up. From his records thus obtained, he concluded that the electrometric readings began a little before the corresponding cardiographic wave. He also took leads from the hands and feet with good results.

The final step in the evolution of the means of detecting and graphically recording changes in electropotential during cardiac activity were made by Adler in 1897 and Einthoven in 1903 (15). The invention of the string galvanometer by the former made quantitative measurement of electropotential possible. Einthoven took Adler's galvanometer and added a reflecting system to the string. Then he projected his string shadow upon a moving photographic film. This arrangement with a few
minor refinements is the basis of the electrocardiograph used today.

**EQUIPMENT:** An electrocardiograph may be defined as a complete assemblage of physical apparatus required to take records of the minute electrical currents generated with each cardiac cycle (16). This equipment usually consists of the following: a recording galvanometer, an optical system, a recording camera, a time recording device, appropriate electrodes, and a current control unit or control box.

The galvanometer most commonly employed is the string instrument of Einthoven. In this type of galvanometer a very delicate fiber of quartz, coated with silver or gold to make it a conductor, is stretched vertically between the poles of powerful electromagnets. The tension of the string can be varied by means of a micrometer adjustment attached to its upper connection. When a current passes through such a string placed in a magnetic field, it deflects in a direction which is at right angles to both the plane of the field and the axis of the string. This is due to the interaction of the magnetic field produced by the current in the string and that of the fixed magnet, tending to strengthen the field on one side of the string while weakening that on the other. The direction of movement is determined by
the direction of current flow through the string. By connecting the body to the galvanometer by means of suitable electrodes, the heart action current can thus be passed through the string causing its deflection.

The optical system on most modern sets of equipment consists of a series of lenses very much like a microscope. A brilliant spot of light is focused upon the string very similarly as does the condenser on a microscope center the light on a slide. An adjustable objective is placed on the opposite side of the string and an appropriate ocular farther out. By focusing the objective, an enlarged image of the string can be projected upon a screen or sensitive film. When the latter is used, a photograph of the string can be taken.

The photographic camera consists of a mechanism for moving a plate, film, or bromide paper at an even rate behind a narrow slot, the size of which is controlled by a shutter. A small portion of the projected string is allowed to pass through this slot and is again focused by a cylindrical lens to a fine line. Most machines carry a transparent metric scale of ruled lines behind the lens or etched upon it to give ordinate lines on the record.

Time intervals are recorded upon the record by various devices; tuning forks, spoked wheels, clock work,
etc., have all served the purpose. All of these merely interrupt the stream of light at regular intervals in the corner of the slot and thus inscribe regular intervals upon the record.

Electrodes usually are of German silver, zinc or lead all of which can be easily moulded to fit the contour of the limbs. They are covered with absorbent material, wet with saline, and applied with bandages or straps.

The control box consists of a series of switches and resistances. Each box differs with the manufacturer's design of machine. In general their function is to select various leads; to protect, through shunts, the string; to provide a rheostat for introducing a compensatory current to offset the current of glandular activity, skin current, etc.; and to provide a system for standardization or comparison.

This constitutes the modern electrocardiographic equipment almost exclusively used today. More recent developments will be discussed later, but, since this equipment is as yet in the experimental stage, it is omitted from standard equipment.
Normal Electrocardiogram

Normal Electrocardiogram showing three leads.
Note that lead II equals I and III. Leads in pairs:
Right arm - Left arm = I
Right arm - Left leg = II
Left arm - Left leg = III

(After Willius)
THE NORMAL ELECTROCARDIOGRAM

LEADS AND THREE RECORDS: As the electrocardiogram depicts the path of the excitation wave of the heart as it passes from the sino-aortic node over the conducting system to the myocardium, the electrocardiograph must first register and then record this impulse or minute electrical discharge. To perform these requirements the above described pieces of equipment are used in the following manner:

Waller demonstrated in 1887 that the heart current could be tapped from the surface and as a consequence three leads are conventionally used. Generally a zinc plate is placed over the two arms and left leg having a gauze pad soaked in saline beneath. These plates are secured into position by bandage or straps. These leads have been arbitrarily named I, II, and III. Lead I is the combination of right arm and left arm; lead II the right arm and left leg; while lead III is left arm and left leg (17). There has been some discussion of late upon a fourth lead but we shall pass over that for the present.

These metal sheets are connected to the string galvanometer by wires soldered to their surfaces. The electrical wave of the heart passes through the body,
into the zinc plates, over the lead wires, and into the galvanometer where its passage through the string causes a deflection.

To record the deflections, it is necessary to illuminate the quartz fiber string with electric light, the rays of which are centered and condensed on the string. As this quartz fiber is only twelve one hundred thousandths of an inch in diameter, half as thick as a red blood corpuscle, it is necessary to magnify its shadow with lenses. This enlarged shadow is projected upon the face of a camera having a motor which feeds sensitized film or paper past the lens at a regular and stated speed of one inch per second. The photographic reproduction of the movements of the magnified string shadow, thus made, is called an electrocardiogram.

MEASURING DEVICES:— The electrocardiogram must have scale divisions on it so that one can measure the height and width of the various summits and depressions which are written by the excitation wave of the heart, in order to determine whether the waves lie within the accepted limits of normal. These scales are secured by placing a ruled plate of glass behind the lens of the camera to give the ordinates and by a rapidly rotating smoked wheel which revolves simultaneously with a vibrat-
Electrocardiogram and Heart Cycle

<table>
<thead>
<tr>
<th>Auricular Systole</th>
<th>Ventricular Systole</th>
<th>Aur-Vent. Diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.17 sec.</td>
<td>0.38 sec.</td>
<td>0.48 sec.</td>
</tr>
</tbody>
</table>

Note: the P wave beginning just before Auricular systole.
QRS complex preceding Ventricular Systole.
PR interval approx. 0.2 sec - ST segment 0.28 sec.

(Schematic)
ing tuning fork. Having these lines and knowing the strings' response, accurate measurements can be taken for comparison with the normal (18). Before the record is taken, the string is adjusted to give a deflection of one millimeter per millivolt input.

WAVES AND TERMINOLOGY: The type and form of record obtained when a patient undergoes an electrocardiogram as explained above is dependent partly upon the lead employed; two leads do not give identical records (19). Hence, three separate records are obtained in the classical electrocardiographic study, and, as will be pointed out later, a fourth lead has been used by some investigators along with the three conventional leads.

From electrocardiograms upon apparently normal hearts, a more or less constant record is obtained. The waves or deflections of the tracings have been arbitrarily designated P, Q, R, S, and T. If records are taken simultaneously in leads I and III, the algebraic sum of these waves will yield lead II. This was stated by Einthoven in his rule that lead I plus III equalled II (20). Hence, variations may occur in one of the three leads and be totally absent from another. This makes obvious the point that all three leads should be carefully examined for variations from the normal.
In man, according to Willius (21), the electrocardiogram is composed of an auricular and a ventricular component; the former being represented by the P wave; the latter by the Q R S and T waves. The P wave appears on the record as a smoothly rounded wave just preceding the sharp R wave. It reaches its height comparatively slowly and drops to the baseline to remain there until the ventricular complex arrives. The Q and S deflections, when present, are short abrupt peaks directed downward or negative to the P and R waves. They blend with the ascending and descending waves of the R respectively. The R wave is usually tall and has an amplitude of deflection of from ten to fifteen millivolts. It is a very rapid deflection of abrupt contour. Then follows a period of rest with the T wave appearing after the Q R S deflection. It is a rounded wave similar to the P wave and generally of an amplitude of three to seven millivolts. The T wave is sometimes followed by a small blunt wave designated as U.

As a normal tracing, lead II will be discussed for the meaning of the various waves. Numerous experiments as well as autopsy findings upon cardiac patients have been correlated with the electrocardiographic records to indicate that the P wave occurs in relation to auricular systole and probably indicates the conduction path
of the impulse through the auricular muscle (22). This statement is fortified by the fact that the P wave is absent in cases of auricular fibrillation when dynamic contraction of the auricle is known to have ceased.

The Q R S complex is definitely ventricular; its form and direction are determined by the course taken by the wave of excitation through the ventricular muscle, and its duration by the time consumed by the wave in completing such passage (23). The course traversed by the wave of excitation through the ventricular musculature has been determined both by dissection and experimental physiology to be a special type of modified muscle tissue, the Bundle of His, which constitutes the conducting system to the ventricles. It has been shown that the rate of conduction through this system is ten times that of ordinary cardiac muscle (24).

The Q R S complex is followed by a straight line which indicates a fairly uniform equalization of electric effects in all portions of the ventricle.

The T wave is still without a definite explanation (25). It is clearly an indication of a ventricular event and is perhaps an expression of a relaxation or decline of the state of excitation in the ventricular muscle (26). Numerous theories have been offered as an explanation of this phenomenon but none are quite con-
clusive. The wave, however, has considerable prognostic significance and explanation of its origin may in the future increase its prognostic value.

Likewise, the meaning of the inconstant U wave, sometimes found following the T wave, has eluded explanation to date (26).
INDICATIONS FOR ELECTROCARDIOGRAPHIC STUDY

According to C. Black (27) in a recent article in the Medicine Klinic, the following conditions are indications for electrocardiographic study:

1. In diagnosis, prognosis, and therapy in various heart diseases.
2. Coronary occlusion, myocardial infarct.
3. Chronic degenerative myocarditis.
4. Acute myocarditis.
5. Pericarditis.
6. Arhythmia.
7. Digitalis intoxication.
8. Myocardial affections preceding:
   a. Operative procedures.
   b. Antiluetic therapy, salvarsan.
   c. For any other procedures embarrassing the heart.
Sinus Arhythmia

---

Sinus arhythmia. No change occurs in the character of waves, nor in their relationship to each other, but a phasic variation in the intervals between the R waves does occur.

Willius
REVIEW OF CLINICAL ELECTROCARDIOGRAPHY

Any method which promises to make us keener in detecting what is wrong with the sick man and in estimating the effect of such ailment upon his health and welfare deserves at least a fair trial. If it stands the trial and is available to the average patient, it becomes another effective weapon in our fight against disease (28). The electrocardiograph has stood this test and is now available to the average hospital patient. No heart can be considered completely analyzed at the present time until it has been given the advantage of electrical study.

The electrocardiograph reaches its clinical acme in the study of cardiac arrhythmias but is, nevertheless, very valuable in other heart disorders. Sinus arrhythmia, premature contraction, paroxysmal tachycardia, auricular flutter, auricular fibrillation, complete and partial heart block, ventricular preponderance, bundle branch block, and myocardial infarction will be considered and typical electrographic tracings presented as given by Willius (29).

SINUS ARHYTHMIA:—The simplest disturbance in cardiac rhythm is known as sinus, or respiratory,
Premature Contractions

Premature contractions of both right and left ventricles RVX - LVX. Note the pause following.

(Willius)
arhythmia. It is a benign physiological phenomenon generally occurring in children. Wiggers (30) states that careful examination of the electrocardiogram shows more or less variation from beat to beat with a tendency for cycles to shorten during inspiration and to increase during expiration. It indicates that the discharging respiratory center, located near the vagus center in the medulla, sends inhibitory impulses to the latter, causing its release and a consequent brief acceleration of the heart. He found this condition present in a large number of normal young men whom he examined and states that the condition is of no significance whatever.

PREMATURE CONTRACTIONS: - Premature contractions, or extra-systoles, are one of the most common causes of cardiac arrhythmia of adult life. Experimental study upon exposed mammalian hearts with electric shocks has proved that the heart's response to stimuli is not proportionate, but is always maximal (31) conforming to the all or none law of Bowdilick. Thus, any stimulus of sufficient intensity can cause a contraction provided the muscle is not in a refractory state, i.e. unable to respond due to a period of recovery following each contraction. These conditions give the typical tracing as shown: and early stimulus causes a contraction before the expected regular
contraction initiated from the S-A node, the heart then becomes refractory and in consequence does not respond when the legitimate impulse arrives from the pacemaker. Hence, we find a pause following each extra systole, the compensatory pause, after which normal rhythm is established.

Premature contractions may arise from any position of the ventricles, auricles, or auriculo-ventricular junctional tissue. They are in reality ectopic contractions which arise from foci of greater irritability than the surrounding tissue. They may appear regularly, interrupting the rhythm at definitely recurrent intervals or their appearance may be wholly haphazard. Their occurrence every second beat is referred to as "coupled beats". However, more often they occur with such a lack of uniformity that the rhythm is almost totally irregular.

The presence of premature contractions does not indicate organic heart disease (32). Premature contractions are frequently observed in perfectly healthy individuals and are undoubtedly the expression of increased cardiac irritability which may be the result of cardiac neurosis, excessive use of tobacco, tea, coffee, stimulating drugs, and emotional stress. It is true that premature contractions may occur in patients with serious heart disease but the diagnosis of cardiac pathology should not
Paroxysmal Tachycardia.

Ventricular tachycardia interrupted by a normal heart cycle.
be made upon the presence of extra-systoles alone. The site from which these aberrant impulses arise does not seem to affect the prognosis.

PAROXYSMAL TACHYCARDIA: - In the strict sense of the term, this condition comprises three forms of cardiac acceleration which arise from the production of abnormal stimuli in the ventricles, the auricles, or the auriculo-ventricular junctional tissues. The origin of the impulse in these forms of tachycardia is ectopic; that is, in some area of the heart away from the sino-auricular node. Tachycardia results whenever any of these centers discharges periodically and at a rate greater than that of the sino-auricular node, thus becoming the pacemaker of the heart (33).

Paroxysmal tachycardia is characterized by the sudden inception of an extremely rapid rhythm, of variable duration, and usually of abrupt termination. The condition as it occurs in man is not yet fully understood, according to Willius (34). The cause, he states is probably best attributed to "increased cardiac irritability". Clinical observations of interest but without scientific confirmation are the frequency with which such attacks are precipitated by some emotional disturbance, their appearance in individuals of an unstable nervous make-up,
Auricular Flutter.

Auricular Rate 200, Ventricular 80.

(Willius)
and the failure to discover any organic basis for such. These facts tend to support the neurogenic or functional theory as to the etiology of the condition. The prognosis depends upon the presence or absence of concurrent cardiac disease. The tachycardia is in itself quite benign and easily controlled with small doses of quinidine.

**AURICULAR FLUTTER:** Auricular flutter is an acceleration of the auricular rate over 200 per minute. It may occur as a fairly permanent disturbance of cardiac action or it may occur as a distinctly paroxysmal disorder, with an abrupt onset and a correspondingly abrupt termination.

The disturbed mechanism responsible for auricular flutter has been worked out experimentally by Lewis (35) by applying rapid, rhythmic induction shocks at varying rates to the auricles. It was found that the spread of the wave of excitation through the auricular mass was very regular and that the activation of the same portion of the auricular tissue occurred during the whole cycle. Once the mechanism was started, its continuation was entirely independent of the original stimulus and the flutter was maintained by a wave continuously traveling through the auricular mass and returning to its point of inception. The wave constituted a continuous circulating
Auricular Fibrillation.

Ventricular Rate 190, Completely Irregular.
mechanism called by Lewis a "circus movement".

Auricular flutter occurs in many different heart diseases and is not characteristic of any particular one. It is probably most frequently observed in cases of mitral stenosis, especially when the lesion is well advanced, after considerable enlargement of the heart has occurred, and particularly when heart failure is present. In these cases it is prone to exist as a permanent mechanism until corrected by treatment. It is a rather common occurrence in exophthalmic goiter, in which it usually appears as a paroxysmal phenomenon and very often terminates spontaneously. Flutter is not uncommon in hypertensive heart disease, during the state of failure, and is likely to alternate with auricular fibrillation.

It has been observed in adhesive pericarditis, coronary disease, and congenital heart disease.

AURICULAR FIBRILLATION: - This is essentially the same condition as flutter, that is, a circus movement, except that the circulating wave does not pursue a fixed course as in flutter but undergoes a change in its plane of activity from time to time. This produces a completely irregular rhythm so marked as to be palpable at the radial pulse. This condition constitutes about fifty percent of the cases of persistant cardiac arrhythmias but may be
Heart Block...

Complete heart block
transient or paroxysmal in nature. It occurs in all those cases listed above in which auricular flutter is seen, the most common being mitral stenosis.

HEART BLOCK: - Willius defines heart block as any condition which interferes with conduction of the impulse from auricles to ventricles through the auriculo-ventricular bundle. Three types of heart block exist; complete, partial and delayed conduction.

1. Complete block is characterized by an extremely slow ventricular rate, usually around thirty beats each minute. Since there are no impulses arriving through the normal conducting system, the ventricle sets up its own pacemaker, idioventricular rhythm, in some part of the ventricular muscle mass. The auricular rate may be normal or even increased.

Complete heart block in man results from various causes, according to Willius. These are chiefly due to lesions of the atrio-ventricular node and bundle; from asphyxia; from drugs such as digitalis, strophanthus, and squill, and occasionally from vagal influences. The most common lesion involving the bundle conducting system results from arterio sclerosis. Thrombosis of the coronary arteries supplying the A-V node and bundle cause difficulty in conduction although a gross lesion is not
present. Other lesions are gumma, areas of calcification, infarction of septum, ulcerative endocarditis, and various types of cellular infiltration and degeneration.

2. Partial block differs from complete block in that some auricular impulses are conducted through the conducting system to provoke ventricular response. Partial block with a two to one relationship is the type most frequently seen. Partial block may exist as a temporary type and is often associated with complete block. Probably it is frequently the forerunner of established complete block. The causes and clinical significance of partial and complete heart block are similar.

3. Delayed auriculo-ventricular conduction is a condition in which the impulse is slowed in its passage through the A-V node and His bundle. It may result from disease of the node or bundle, from asphyxia, from certain drugs, and from vagal influences. The condition appears in the graph as a lengthened P-R interval above the accepted normal of thirteen one hundredths to twenty-one one hundredths seconds. It may also be due to causes similar to those giving complete or partial block. During recent years, delayed conduction has been observed in cases of rheumatic fever. It may be caused by vagal influence and cases in which demonstrable pathology is not apparent, a subcutaneous injection of the
Ventricular Preponderance.

Depressed S wave. Right Ventricular Preponderance.
physiologic dose of atropin should be given to rule out this condition.

VENTRICULAR PREPONDERANCE: - Einthoven was the first to associate certain variations in direction of the initial ventricular complexes with hypertrophy of the left or right ventricle (36). He described exaggeration of the R wave in lead I and of the S wave in lead III as indicative of left ventricular hypertrophy. Conversely, he associated exaggeration of the S wave in lead I and of the R in lead III with hypertrophy of the right ventricle. These rules may hold but often there is found the exception where the electro-cardiogram and clinical findings do not coincide. This discrepancy arises when the terms "preponderance" and "hypertrophy" become confused. Ventricular preponderance, as seen in the electrocardiogram, is the expression of the electrical effects of one ventricle in relation to the other. It is true that an hypertrophied ventricle gives a greater response than its fellow but the electrical balance of the two chambers can be disturbed in a heart of normal size and weight. The position of the heart in the chest will effect the record, hence, uniform position must be insisted upon. Bundle-branch block often gives a similar tracing but is accompanied by other changes which make differentiation read-
Coronary Thrombosis.

Inverted T Wave Lead I.

Bundle Branch Block.

Complete right bundle branch block with notched R wave and inverted T. Note spread of QRS complex.
ily possible.

**CORONARY THROMBOSIS:** Myocardial infarction, due generally to coronary thrombosis, gives a typical record at various periods following the accident. Frequent tracings are necessary to detect the changes which occur in the electrocardiogram in this condition. At times, changes appear within thirty minutes after the occlusion while in others thirty-six to forty-eight hours may need elapse before changes are noted. The most constant findings are inversion of and high take off of the T wave. Depending upon the area in which the infarct is located, various conditions may appear such as bundle-branch block if the conducting system be included.

If the patient recovers, the electrocardiogram often returns to normal within six months to a year (37).

**BUNDLE BRANCH BLOCK:** Much uncertainty and difference of opinion have existed regarding the meaning of changes in form and delay in execution of the QRS complex. Recent investigators (38), however, have given a much clearer idea concerning these graphic changes. It has been shown that the form of the QRS complex is dependent upon the course of the excitation wave through the
ventricular musculature, i.e., along the His bundle and its branches. The duration of the complex depends upon the time taken by the excitation wave in completing its course over the Bundle of His and branches. Experimental work upon dogs in which the bundle is completely divided and human tracings correlated with autopsy findings have proved that the increased duration of QRS complex is the most reliable indication of complete bundle branch block, although the degree of the deflections of amplitude cannot be overlooked. The QRS interval usually ranges between twelve and eighteen hundredths seconds. Increased length of QRS is generally accompanied by exaggerated amplitude and inverted T wave. In complete right bundle branch block, the QRS complex is up in lead I and inverted T wave; the reverse is found in lead III. Left bundle branch block is exactly opposite to this. Considerable notching of the QRS complex is generally seen.

Bundle branch block may be only partial and may be due to nutritional defects as in coronary disease. It may occur in heart failure from many causes. It also may result from fatigue as in a greatly hypertrophied heart with impaired conduction. The most common causes, however, are coronary disease and hypertension.

Bundle branch block is a very significant finding
and furnishes reliable evidence toward prognosis. Sixty-five percent die of heart disease within thirteen months after the appearance of such changes (39).

CURRENT LITERATURE:—Thus we see that the common cardiac irregularities can be readily analyzed by the electrocardiograph. And it is in the diagnosis of these disturbances in rhythm that the electrocardiograph finds its chief field of usefulness. However, the physician of today finds the heart records of value in other cardiac conditions as well.

Walker (40) states that the electrocardiograph has a very definite place in medicine. He accordingly recommends graphic study in cardiac arrhythmias; preoperatively to judge the degree of cardiac power and integrity; to detect ventricular imbalance, preponderance; in the control of digitalis therapy; in the prognosis of many diseases; as a guide in the convalescence in acute fevers; and as an instrument for research.

With these indications in mind, many investigations have been conducted both upon the experimental animal and man with the compilation of valuable data, all of which adds to our understanding of the heart and its diseases. The clinical significance of changes in the P wave have been studied and reviewed by Goddard and Smith. Smith (41)
states that the P wave is normally upright, rounded, and measures from two to four millimeters in height. Its base measures around one-tenth second and occupies about one-third of auricular systole. It is occasionally followed by a slight dip; the auricular T wave is generally obscured or obliterated by the QRS complex. Abnormalities in this wave are seen in extra systole arrhythmia, which is not clinically of prognostic significance; auricular and ventricular paroxysmal tachycardia; and auricular fibrillation and flutter. A serrated P wave is often seen in flutter and a normal wave in fibrillation. Gerandel (42) states that an inverted P wave is interpreted as indicating a systole of the auricle due to an abnormal stimulus passing up from the node of Tawara to the auricle. Goddard (43) found the most important changes in the P wave in mitral stenosis, manifested by an increase in height and broadening of the base of the wave. The T wave is normally higher than the P, hence, the P-T ratio is less than one. When this relation is found greater than one, auricular hypertrophy is suggested. An increased height of the P wave occurred in fifty-three percent of his seven hundred cases. However, he states that there are probably other factors which influence the height of the P wave. Section of the vagus nerve will produce a heightened P wave in dogs. He concludes that
the dogmatic statement that all cases of heightened P wave mean auricular hypertrophy and that all cases of mitral stenosis will show a high P wave is open to great chances of error. As stated previously, the electrocardiogram must be used in conjunction with clinical findings in all cases.

The Q R S complex is discussed by Willius (44) in a recent article in the Proceedings at Mayo Clinic. He said that the electrocardiogram is the graphic inscription of the changes in electric potential occurring in the heart muscle preceding cardiac activity. The Q R S complex is the initial phenomenon of ventricular activity. It lasts one-tenth second and is the record of the transmission of the cardiac impulse through the ventricular conducting system: the A-V node, bundle of His, and the branches. Interference with this conduction is called block and is either complete or incomplete. Coronary sclerosis is the most common cause of block with syphilis and rheumatic myocarditis second and third respectively. Bundle branch block is manifested in the electrocardiogram by notched, irregular, and widened Q R S complexes.

Abnormalities in the T wave are reviewed by Barnes (45) who describes the T wave as the record of the summation of the electro-potential forces developed by the
fractionate components of the ventricular components of the ventricular muscle in the terminal phases of excitation and contraction; or the algebraic summation of electrical forces acting in opposite directions, of the right and left ventricles. Any condition which interferes with either side and not the other will produce T wave changes. He lists as the most common causes of T wave abnormalities, strain on the left ventricle as in hypertension, delay in conduction in one bundle branch causing one ventricle to contract ahead of the other, and acute coronary occlusion. Changes can also occur as a result of the action of toxins or by purely mechanical means. These changes may or may not be serious to the individual. Graybiel and White (46) found the T wave inverted in leads II and III in cases of neurocirculatory asthenia and thyrotoxicosis. They emphasized that caution should be exercised in diagnosis with only inverted T waves and no other findings as a basis. Likewise, Huréthal (47) has sited that two cases in his knowledge had inverted T waves before a coronary attack, were upright immediately following the attack, and later again became inverted. He recommends early electrocardiograms and reliance upon clinical measures in such emergencies. Pardee (48) states that an electrocardiogram with a notched Q R S complex, ventricular preponderance, and
changes in the T wave, with clinical findings indicate marked myocardial damage as produced by acute coronary occlusion. These findings were investigated and confirmed by surgical ligation of the coronary arteries in the dog by Fowler (49). He tied the small branches of both the left and right coronary arteries and took electrocardiograms immediately following. In each case, a definite alteration of the T wave was produced. He also took tracings following the opening of the pericardium without tying a vessel and found that this also produced definite T wave changes. Hence, he concluded that, since a definite myocardial lesion could be demonstrated following each procedure, changes in the T wave as seen in the electrocardiogram are indicative of a myocardial lesion. Christian (50) states that if repeated electrocardiograms are made in myocardial infarction, besides certain arrhythmias changes in the ventricular complex in all leads may be found. When they do occur he thinks they are almost diagnostic of cardiac infarction. These changes may appear in the T wave as high take-off of the first part of the wave and inversion with a deep V shape to the T wave. These findings substantiate our clinical application of the electrocardiogram to heart cases and seems to make firm the dogmatic statement that altered T waves with obvious clinical symptoms mean myocardial damage generally
due to coronary occlusion.

A study of the S-T interval has been made by Buchanan (31) of the Mayo Clinic. In one thousand and twenty-eight cases, he found that lesions of the conduction system have no distinctive influence upon the length of the S-T interval and that no anatomical variation in the heart per se can cause any alteration in the S-T length. From his study of more than one thousand cases, he concluded that if the horizontal portion of the S-T interval be the expression of an iso-electric state, and if the T wave be the expression of preponderance of contraction on one side of the line of equipotential, it follows that only some factor which either accelerates or retards the rate of change of electropotential can cause a variation in length of the S-T segment. The change whenever present is no doubt electro-chemical in nature rather than histo-pathologic.

The valves of the heart can be investigated directly by the electrocardiograph through their affect upon the various chambers of that organ. Pardee (52) states that auricular hypertrophy is manifested by heightened P wave and changes in the R wave indicate ventricular hypertrophy; enlargements produced by well known valve dysfunction. The alteration in these waves is due to a change in tissue mass, the normal ratio between right
and left ventricle being about one to two. Thus, hypertrophy with the altered waves, can be readily differentiated from dilation in which no increase in total tissue mass is present. White (53) working on the same subject, concludes from observations made upon four thousand cases at the Massachusetts General Hospital, that mitral and pulmonic stenosis produce abnormalities seen as right axis deviation while aortic regurgitation and arterial hypertension give left axis deviation. He likewise noted an exaggeration in the auricular complex in pulmonic and mitral stenosis. Anderson (54) at Nebraska University Hospital, found cases with generalized arterio-sclerosis and hypertension showing left axis deviation and coronary type of T wave, while those with mitral stenosis showed high and notched P waves and inverted T waves. So the preponderance of evidence seems to point to the conclusion that any condition of the heart's valves which tends to throw a strain upon its chamber, will eventually produce hypertrophy of that chamber. This hypertrophy is reflected in the electrocardiogram as an over response, either a heightened or inverted wave.

Rheumatic fever is one of the most common serious diseases of the heart. Reid (55) states that in the young patient, the symptoms noted clinically are essent-
ially those of rheumatic fever and not those referable to the heart. The rheumatic symptoms clear up readily under salicylate medication but, as shown by repeated electrocardiographic tracings, the cardiac pathology may even progress while under salicylate treatment. The heart changes often appear before those of rheumatism become evident. Rheumatic fever is exudative in its arthritic role but proliferative in the heart. The development of the myocardial lesions, Aschoff bodies, may be responsible for one of the following electrical changes depending upon its anatomical location in the heart: 1. Increase in A-V conduction time, ninety-two percent; 2. Alteration in QRS complex, eighty percent; 3. Changes in cardiac rhythm, thirty-four percent. Benedict (56) has observed one hundred and twenty-one patients with acute rheumatic fever and found a prolonged A-V conduction time and lengthened P-R interval in their electrocardiograms. He thinks that the graphic tracings may be the only indication of the activity of the process. The conclusions from findings of each of these men are in strict harmony, namely that pathology in the heart is generally evident both before clinical symptoms are present and after they have disappeared, either as a result of spontaneous resolution or from the effects of medication. Hence, the electrocardiograph is an indispensable instru-
ment to the clinician in his management of a rheumatic fever case both for prognosis and treatment.

Digitalis is the most common cardiac medicament used today. Its dosage is variable but clinical use has taught us certain rules by which we may know when a patient is thoroughly digitalized. These signs and symptoms are vomiting, pulse below sixty beats per minute, or the appearance of diuresis. The electrocardiogram of a digitalized heart is similar to that of heart block. Frequent tracings allow the physician to observe the depth and effect of his medication. According to East and Bain (57) changes in the T wave, and in the S-T or R-T interval may appear as soon as two hours after the beginning of oral administration of digitalis. Usually there is a depression of the R-T or S-T phase, with flattening or inversion of the T wave. These generally appear first in lead III. When large toxic doses are given, there is slurring and widening of QRS with decreased height of R wave. These changes may persist for some days to two weeks after discontinuing the administration of the drug.

The question of maintenance dose of digitalis was investigated by Bromer (58) who administered digitalis by the Eggleston method and took heart tracings at the same time. He adjusted the dose and observed the cardiac response and concluded that the average maintenance dose
was around twenty-five minims per twenty-four hours, or about one minim per hour. He noted his first electrocardiographic changes on about one-half the Eggleston dose. From these experiments we see that the electrocardiograph can be used to a nice advantage in guiding therapy as well as in diagnosis.

But probably the most fertile field for electrocardiography and the one yet to be completely cultivated, is that of prognosis. Here we find spectacular results forthcoming; cases appearing from the surface as being very threatening to the individual's life revealed as harmless, while others of a more serious insidious nature uncovered. Bloedorn and Roberts (59) refer to the inverted T wave in leads I and II as bad prognostic signs. Anderson (54) states that the highest hospital mortality rate occurs in patients with a notched broadened QRS complex and an inverted T wave in lead I. This means ventricular myocardial damage and the expectancy for life is only six months to one year. Less serious prognosis is merited in cases of coronary disease where an acute accident has produced only an inversion of the T wave. Often these waves are seen to return to normal in six months but there is no means for prognosticating when another and possibly more serious occlusion will take place. Then those cases of cardiac irregularity,
namely sinus arhythmia and extra-systole, which cause the patient so much anxiety can be definitely diagnosed and demonstrated to the patient with a complete reassurance as to their harmless nature. Thus we see the electrocardiograph become an instrument for separating those malignant cardiac conditions from the benign disorders which would certainly produce a great many psychic heart invalids if passed unrecognized.
REGENT DEVELOPMENTS IN ELECTROCARDIOGRAPHY

ERRORS IN STRING GALVANOMETER: - To increase the sensitivity of the string galvanometer, Forbes and Thatcher (60) devised a hook-up of electron tubes with the classical string galvanometer. A "D tube" made by Western Electric Company was used in the amplifier. It consisted of a vacuum tube with a hot filament cathode, metal plate anode, and auxiliary electrode in the form of a grid between these two. The electron flow was from plate to filament; the grid potential controls the plate filament current, thus producing the amplification. This amplifier hooked in series with the string galvanometer and suitable condensers allows magnification of minute electrical currents too small to be recorded by the string galvanometer alone. This equipment was a definite improvement over the string galvanometer alone, as it gave much more accurate tracings than before. It is essentially the equipment used in most hospitals today, of which the Victor is an example.

There are numerous machines on the market today, many of them sufficiently compact to be classed as portable, which will produce good electrocardiograms. However, Reid (61) points out that the medical profession, not being as a group carefully trained electrical engi-
neers, has accepted or taken for granted that the string galvanometer is the ideal instrument for detecting and recording the electrical potential variations occurring in the heart cycle. This assumption, he says, is erroneous and is not in the least concurred in by electrical engineers; these men use the cathode ray oscillograph almost exclusively for recording such minute variations in electrical potential. He cites the work of Caldwell (62) at the Department of Electrical Engineering of the Massachusetts Institute of Technology. Caldwell says that his experiments show that the wave forms recorded by the string galvanometer become distorted when frequencies which in excess of twenty cycles per second are present. A wave, the QRS complex from a normal electrocardiogram, was subjected to what is known as harmonic analysis, a form of mathematical analysis, and it was found that the frequency of the components involved greatly exceeded twenty cycles per second. And since the relative amplitudes show that these higher harmonics are of great importance in determining the wave form, any failure to respond equally well at high and low frequencies must result in distortion of the wave. The second source of error in the string galvanometer lies in the presence of body resistance in the measuring circuit. Under average conditions, the body resistance is
around twenty-five hundred ohms between terminals such as are generally used in electrocardiographic studies. The string galvanometer resistance is generally of the same order of magnitude. Hence, the current which flows in the galvanometer is controlled as much by the internal resistance of the body as by the external resistance of the measuring circuit. Although the galvanometer resistance is substantially constant, the body resistance may vary due to muscle action, glandular activity, and what not, and thus alter the shape of the wave on the record. Both of these defects, i.e., failure to respond to high and low frequencies and resistance difficulties, are eliminated by the cathode ray oscillograph.

With these factors in mind, Caldwell, Oler, and Peters (62) designed an amplifier to be combined with a cathode ray oscillograph which would produce a more nearly accurate wave form than that obtained with the string galvanometer. Omitting the details of the amplifier circuit and a technical discussion of the oscillograph, it may be said that their equipment consisted of a four stage resistance coupled type amplifier which delivered to a standard type three-element Westinghouse oscillograph. Records taken simultaneously upon this equipment and upon the conventional string galvanometer showed striking differences. This equipment produced satisfactory records of
wave forms containing frequency components up to two hundred and fifty cycles per second or five times the frequency range of the galvanometer. Furthermore, the records obtained showed better detail in the individual waves which allows more accurate analysis and timing measurements.

More recently Robertson (63) has described a similar piece of apparatus in the Proceedings of the Royal Society of Medicine. His equipment is essentially the same as that used by Caldwell et al with the exception of the employment of an English made oscillograph. In brief, the equipment consists of a cathode ray oscillograph, a time base, and a four stage amplifier. The chief part of the oscillograph is the cathode ray tube which produces the spot on the screen as follows: Inside the tube is a filament or cathode at the top of the arch of which is a blob of barium oxide which emits electrons profusely when the filament is heated by passage of the filament current. The electrons are attracted to the anode, or "gun", which is at high positive potential with respect to the filament. At the same time, the electrons are concentrated into a sheath by the negative bias applied to the cylinder. Most of the electrons emitted from the filament will pass, therefore, through the small hole in the center of the anode, constituting the cathode ray,
or electron jet which is a beam of minute particles of negative charge. This cathode ray then passes successively between two pairs of plates, each pair set at right angles to the other pair. If, therefore, one plate of a pair is at a positive potential compared to its fellow, the ray will be deflected towards it by its attraction for the negatively charged electrons constituting the ray. Deflection of the cathode stream is, therefore, possible in two axes at right angles to one another, corresponding to the disposition of the two pairs of plates.

After emerging from the fields of the deflecting plates, the cathode ray moves in a straight line to the end of the cathode ray tube, where it impinges on the fluorescent screen and gives a visible so-called spot. The fluorescent screen may be of different materials; calcium tungstate gives a brilliant blue spot of intense actinic value and is called a "cold screen", while one of zinc sulphide gives a green spot with an extremely long afterglow. A combination of these screens is had upon the more expensive tubes and produces very excellent photographic results.

For the purpose of electrocardiography, a standard amplifier with a gain of from eight to one hundred and twenty thousand, representing a movement of the spot on the screen of from four to six centimeters per millivolt
input, is adequate. Such a high gain amplifier must be absolutely steady for this work. Furthermore it is advantageous if no compensation for skin potentials is required.

A time base is incorporated and is arranged to give a straight traverse of the spot from left to right across the screen at a rate which is nearly uniform and which can be varied at the will of the operator. It is a repeating time base, as the spot flicks back at the end of its traverse, and automatically begins the migration again. This effect is produced by slowly charging a condenser through a resistance and then quickly discharging it across a neon discharge tube with a definitely set striking voltage. This is connected to the horizontal set of plates so that a gradual change in potential is produced on the cathode ray tube plate thus drawing the electron beam slowly across the screen and rapidly flicking back as the neon tube discharges. This is called a linear time base.

Conventional metal electrodes are strapped to the patients' limbs and these are connected with the amplifier by suitable lead wires. A jack connects the amplifier with the time base, and another jack the time base with the oscillograph. A camera, very similar to that used on the string galvanometer, records the movement
of the spot on the fluorescent screen which is the electrocardiogram.

Robertson has obtained electrocardiograms with this equipment which appear very similar to those reported by Caldwell. However, he admits several disadvantages in this method along with the advantages. Of the latter, he thinks the oscillograph superior to the string galvanometer first because it is impossible to damage the equipment by overload voltages as there is no mechanical part such as the string to dislocate; second, the moving part, the electrons, are of such minute size that there is no inertia in changing directions as at the summit of a wave; third, the oscillograph embodies two planes of motion, horizontal and vertical, allowing examination on the fluorescent screen of curves with a suitable time base motion; and fourth, an oscillograph with a screen with long after-glow will make it possible to examine the electrocardiogram directly, thus eliminating the delay encountered in developing the records.

However, he points out several technical difficulties which must be solved before the oscillograph-electrocardiograph is perfected. The most troublesome of these is the fuzzy line produced by the beam of light upon the photographic film. The photographic record of the movements of the spot on the cathode ray tube, i.e., the
electrocardiogram is produced by the exposure of the film to the actinic rays of the spot produced by the focusing of the stream of electrons upon the fluorescent screen of the oscillograph tube. This is a recording of direct light whereas the record of the string galvanometer is that of a shadow. The actinic rays tend to aborize or spread and this spreading of the light rays tends to produce shadows and auras upon the film giving a fuzzy, poorly defined line. This difficulty, however, does not seem impossible of elimination and we can expect the appearance of a revolutionary type electrocardiograph in the near future.

At the present time, research is being conducted in the Department of Physiology, University of Nebraska College of Medicine (64) upon an apparatus for recording simultaneous electrocardiograms and heart tones. The perfection of this equipment should add greatly to our store of knowledge concerning the relationship of the cardiac impulse to the physiology of the heart's contraction. Such records will also be extremely valuable in studying the various heart murmurs. A report of this equipment will undoubtably be published in the near future.
"It is to be expected that, just as formerly the string galvanometer gave rise to a new epoch in bio-electrical research, so the cathode ray oscillograph will soon discover its proper field of application in biology and medicine on the ground of its superiority as a measuring instrument. As is always the way with every new and good method of measurement, this will not remain without influence on our exact knowledge of biological processes." (Translated from the preface by Professor Therring to "Kathodenstrahlaszillographie in Biologie und Medizin" by W. Holzer.)

Only a little over one hundred and thirty years have elapsed since man first became curious about the electrical phenomena which he observed in the animals about him. Scarcely thirty years have passed since man conquered this electrical mystery in a manner sufficient to warrant its general clinical application. But in those thirty some odd years, our conception of the heart and its physiology has undergone a complete metamorphosis, until today with the equipment at hand we can detect the most minute variations in the activity of the myocardium. Who knows what the next thirty years will reveal? With the amplifier-oscillograph-electrocardiograph, the electrocardiogram can be magnified to a point
comparable with that of the oil immersion lens of the microscope; simultaneous electrocardiograms and heart tones can correlate the electrical and mechanical phases of the cardiac cycle to an inestimable fraction of a second. Details in the heart record will be revealed which have heretofore been unknown, to be added to our present store of knowledge of cardiac activity. The physician of the future will inherit these advances; he will be armed with a weapon much sharper than any possessed by his fore-fathers; he should be, with their aid, much keener in his battle against disease.


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