With the compliments of
the author.

THE

DIAGNOSIS AND TREATMENT

OF

ABNORMALITIES

OF

MYOCARDIAL FUNCTION

With special reference to the use of

GRAPHIC METHODS

BY

T. STUART HART, A.M., M.D.

Assistant Professor of Clinical Medicine in the College of Physicians and Surgeons, Columbia University. Visiting Physician to the Presbyterian Hospital in the City of New York.

Illustrated with 248 Engravings,
240 of which are Original

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TO

DOCTOR WALTER B. JAMES

A PIONEER IN
THE APPLICATION OF
METHODS OF PRECISION
TO THE SOLUTION OF
CLINICAL PROBLEMS
THIS BOOK IS INSCRIBED
AS A TOKEN OF
ESTEEM AND AFFECTION

FOREWORD

In very recent years there has been added to clinical medicine a new chapter on the functional activity of the heart muscle. These pages have been written in a language almost wholly new and in characters hitherto unfamiliar.

In conducting a series of lectures and laboratory exercises on cardiac pathology during the past five years in Columbia University it has become increasingly clear to the writer, that there was needed a manual which should present the knowledge more recently acquired in this field in a simple and condensed form to meet the requirements of the student and practitioner.

The attempt has been made to approach the subject from the clinical side and to lay stress on the features which are of practical importance to those whose advice is sought on these questions of disordered cardiac activity. We have endeavored to use as simple a vocabulary as possible and yet to introduce the terms which in a brief period must be the daily companions of the physician who would be abreast of the times.

If it should seem that we have consumed too considerable a portion of our space in a presentation and discussion of graphic records it is because our new knowledge of myocardial function has been to a large extent obtained by these agencies, and we believe that these afford the easiest and simplest approach to a clear comprehension of the views at present held. Today's literature is teeming with these pictures of heart activity and a familiarity with them will make accessible the valuable additions which are daily being made to our knowledge in the fields of myocardial pathology and therapeutics.

It is not presumed that every practitioner will have the time or facilities to make graphic records of those of his patients in whom he suspects a myocardial defect, but it is hoped that these pages may be of service in inducing a closer study of the signs which are obtainable by the more common methods and may help in the interpretation of the facts thus secured.

The theories of myocardial function as generally accepted at the present time have been briefly outlined together with a few of the more important facts upon which these hypotheses rest. Many points which are still in controversy have been touched upon, but an effort has been made to avoid an extended argument on those subjects which can only be decided by further investigation.

The literature has become voluminous and the papers, clinical and experimental, bearing on this subject are of very great number.

No attempt has been made to present a complete bibliography, but references have been given to many of the recent important papers and from these the student who is more deeply interested may secure further leads to special lines of investigation which may be of use to him.

Perhaps, in no other field, is clinical medicine a greater debtor to the combined efforts of the physicist, physiologist and the pathologist than the one presented in these pages. It is a brilliant illustration of the practical results which may be obtained by the coöperation of those working in the different sciences. The discoveries of pure physiology made accessible by the physicist and verified by the pathologist have been pressed into the service of humanity by the master clinician. The practitioner is under deep obligation to the many investigators who have contributed to this result and whose names will be found on every page of this book. For their ingenuity in devising methods and their application to clinical medicine the services of James Mackenzie and Willem Einthoven will always remain conspicuous.

The investigations of the writer have been conducted in the Laboratory of Physical Pathology and the Wards of the Presbyterian Hospital and he is deeply in debt to his colleagues in the hospital for placing interesting cases at his disposal for observation and study, and to others in several of the departments of Columbia University for counsel and assistance.

He is particularly under obligation to Dr. Walter B. James, Clinical Professor of Medicine, the founder of the laboratory devoted to the study of the heart by physical methods in the Presbyterian Hospital; to Dr. Warfield T. Longcope, Professor of Medicine and Director of the Medical Service of the Hospital; to Dr. Horatio B. Williams, of the Department of Physiology, who at all times has ungrudgingly placed his rare attainments as a physicist and physiologist at the disposal of the author; and to his one-time associate Dr. Francis Fraser, an acute observer and skilled clinician.

The writer takes this opportunity to express his thanks to the Editor of *The Archives of Diagnosis*, Dr. Heinrich Stern, for the permission to utilize matter and illustrations which have appeared in that journal, and his appreciation of the many courtesies of his publishers, Messrs. Rebman and Company, extended while the volume has been passing through the press.

T. STUART HART.

160 West Fifty-ninth Street, New York City.

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CHAPTER I

Introduction

The diagnosis of cardiac abnormalities requires a knowledge of three elements: (1) Etiological, (2) Anatomical and (3) Functional. In the past while all these features have been considered, the greater stress has been laid on the anatomical diagnosis, etiology has taken a somewhat less conspicuous place and function has perhaps received the least attention. Again until recent years attention has been most closely directed to the anatomical abnormalities of the valves, the endocardium and the pericardium, while the myocardium has received relatively little clinical study.

The explanation of such a development is perfectly simple; our methods of examining the heart mainly by the employment of physical signs were such as to lend themselves particularly to the elucidation of the conditions of the endocardium and the pericardium. Reasoning from our physical signs we were fairly sure to correctly interpret the kind and extent of the damage to the valves and the pericardium; beyond determining the presence or absence of hypertrophy and dilatation our anatomical diagnosis of the condition of the myocardium was little more than a shrewd guess.

The methods of cardiac examination which have been so rapidly developed in the past decade have afforded us the means of studying the heart from an entirely new standpoint.

The polygraph and the electrocardiograph have put us in the way of studying many cardiac conditions which had hitherto been unrecognized. These instruments afford us records of the functional activities of the heart; in the main they are records of myocardial function. Such studies often permit us to draw inferences in regard to the anatomical condition of the heart muscle. They have already helped us materially in formulating our prognosis, and our accuracy in this regard should be greatly improved as time passes and we are able to correlate our findings with the events which follow as the years in which such observations are made increase in numbers. They have furnished information which has greatly modified our methods of treatment; they should be very useful in the

study of the effect of drugs on myocardial function. Such records are extremely valuable in that they register graphically the functional condition and can be preserved for future reference and comparison with later observations, unclouded by the haze with which time is so apt to obscure the evidence obtained by the eye, ear and finger, even though these are reinforced by carefully written notes.

Perhaps the most important contribution which these later methods have made to the average clinician is that they have made his powers of observation more acute; they have given new meaning to the old physical signs and, with a knowledge of what the polygram and the electrocardiogram have disclosed, he is able to detect and interpret physical signs which hitherto went unrecognized or were without meaning.

Every patient with abnormal cardiac function cannot be brought within the sphere of the electrical attachments of an electrocardiograph. The electrocardiograph is an expensive laboratory instrument suited to the facilities of the large hospitals or the office of the consultant and cannot be included in the armentarium of the average general practitioner. Even the polygraph, although portable and not particularly expensive either in its first cost or upkeep, is an instrument which requires a certain amount of training and a very large expenditure of time for its successful operation.

It is therefore comforting to know that if one sufficiently familiarizes himself with the kind of evidence which these instruments afford, and the nature of the cardiac abnormalities upon which they throw light, he should by carefully cultivating his powers of observation be able to detect on physical examination the signs which in 90 per cent. of all cases will allow him to make as correct a diagnosis and apply as well a directed course of treatment as he could if his observations were reinforced by the most elaborate records.

CHAPTER II

Anatomy

In order to elucidate our conception of the functional activities of the myocardium it will be well for us to briefly review a few anatomical facts.

The embryonic heart of the vertebrates first appears as a tube, at the posterior portion of which the veins coalesce to form a cavity which is known as the sinus venosus. In the course of development the tube is bent upon itself and from its wall a series of chambers are formed which ultimately become the auricles and ventricles. These features are more clearly seen in some of the lower vertebrates. In the higher vertebrates the separation of these chambers becomes less distinct; the sinus venosus disappears as a distinct structure and is fused with the tissues of the superior and inferior cavæ and that portion of the right auricle which lies between the termination of these veins.

Recent histological studies have afforded facts of peculiar interest and lend support to the theory of myocardial function, which is today pretty generally accepted. The study of serial sections of the mammalian heart has served to demonstrate certain structures which up to this time had been unrecognized. Keith and Flack have described a collection of muscle cells of such structure as to distinguish them from the surrounding tissue lying near the junction of the superior cava with the right auricle and extending along the sulcus terminalis for a distance of about 2 c.m. (in man). These cells are fusiform, striated, have elongated nuclei and are embedded in dense connective tissue; they have a special arterial supply and intermingled with them are some nerve cells and nerve fibers which connect with the vagal and sympathetic nerve trunks. This specialized tissue is known as the *sino-auricular node* and is believed by Keith and Flack to be a remnant of the original sinus tissue. Similar isolated masses, which are believed to be remnants of the primitive canal as it passed through the auricle, have been found near the coronary sinus, in the auricular septum, in the valve of Eustachius and at the mouths of the pulmonary veins.

A differentiated mass of tissue similar in structure to the sino-auricular node and known as the *auriculo-ventricular node*, was first described by Tawara. It is situated low down in the auricular tissue at the right posterior edge of the septum; at the anterior end of the auriculo-ventricular node these specialized muscle cells become more parallel in arrangement and form a narrow band ensheathed in a fibrous canal. This structure is known as the *bundle of His*. It runs forward and to the left in the central fibrous portion of the heart to the membranous septum of the ventricles; at a point a little in front of the anterior end of the attachment of the median segment of the tricuspid valve the bundle divides into two branches; the left branch immediately passes through the membranous septum and is continued downward along the septum beneath the endocardium of the left ventricle; branches are given off all through its course in the septum; the principal branches going to the papillary muscles of the mitral valve; the right branch of the bundle is directed downward beneath the endocardium of the right ventricle to the papillary muscles where subdivisions begin to be given off from the main trunk.

The subdivisions of the conducting system are continued into that complex network lying beneath the endocardium of both ventricles known as *Purkinje's fibers* and these in turn make direct connection with the muscle fibers of the ventricles.

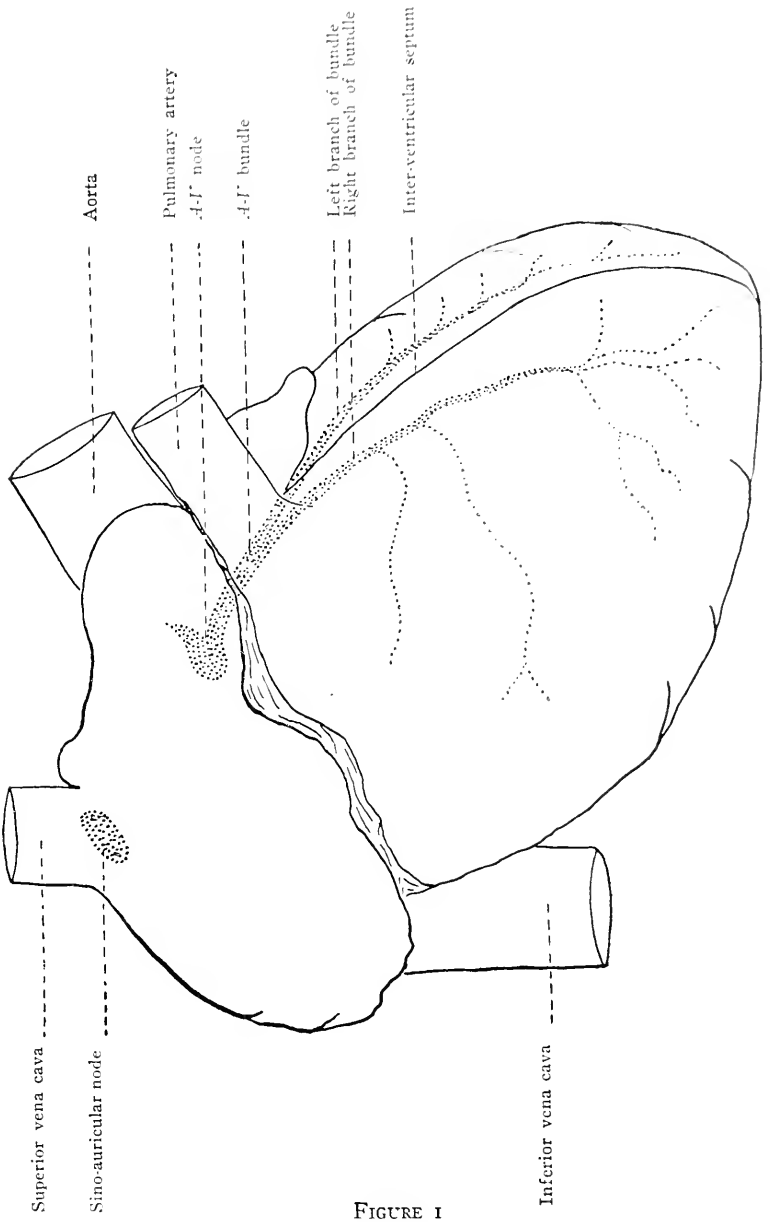

FIGURE 1

Diagram of the specialized conducting system of the heart.

CHAPTER III

Physiology

As an introduction to the study of certain types of cardiac function, it will be well for us to consider briefly the theories which are at present in vogue as to the mechanism of cardiac activity.

THE NEUROGENIC THEORY

Up to a few years ago it was generally held that the origin and regulation of the heart beat must be located in some part of the nervous system. The discovery that the contraction of the voluntary muscles had its origin in the central nervous system made it seem probable that the contraction of the heart was the result of a stimulus arising in a nerve center which was conveyed to the heart by nerve fibers, thus a musculo-motor nerve center was assumed to exist and efforts were made to locate it. When it was discovered that the excised heart of many animals, under suitable conditions, was able to continue its rhythmical action for a considerable period, it seemed necessary to assume that this motor center was located in the nervous substance embedded in the cardiac tissue. Further, since it was recognized that the heart muscle did not contract as a whole, but that the activity was first seen at the sinus and thence spread successively over the auricles and ventricles, it was supposed that the nerve center originating this stimulus was located in the wall of the sinus and that from this point the impulses were carried by the intrinsic nerves of the heart to muscle fibers of its successive chambers. The influence of the vagus and accelerator nerves was recognized and their activity was believed to heighten and depress the automatic nerve center which originated the rhythmic contraction of the heart.

Briefly, the "neurogenic theory" is as follows: The initiation of the rhythmic activity of the heart resides in an intra-cardial nerve center, the muscle fibers respond to stimuli originating in this center, the activities of this nerve center are modified by positive and negative influences conveyed to it by the vagus and sympathetic nerves, thus permitting an adjustment of the activity of the heart in ac-

cordance with the needs of the body at any particular moment by a reflex mechanism.

The important point in which this theory differs from the one at present generally accepted is that it ascribes to the muscle cells an entirely passive rôle, making their activity directly dependent on the activity of the intra-cardial nervous tissue.

THE MYOGENIC THEORY

As a result of physiological investigations of the last two decades the theory of the neurogenic origin of the heart action has been opposed by the view that the source of the rhythmical movement is to be found in the heart muscle itself. This is known as the "myogenic theory." This theory has been formulated to explain and harmonize the facts discovered by the older investigators, such as Bowditch, Traube and Ludwig, and the newer facts brought to light especially by the researches of Gaskell and Engelmann.

According to this theory, the stimulus arises not in a nervous center, but in the muscle cells of the heart and is conveyed to the successive portions of the heart not by the nerve fibriles, but by the cells of the contractile tissue itself.

The "myogenic theory" is of the highest importance in analyzing and explaining the mechanism of the activity of the heart, and affords us a practical working hypothesis of great value in the study and treatment of pathological conditions of the myocardium.

It will be impossible for us to present here the detailed evidence upon which this theory is based. This can be obtained from the critical reviews, such as have been written by Biederman and Langendorff (*Ergebnisse der Physiologie*), and from the original papers, especially those of Engelmann.

For our purpose it will be sufficient to review briefly a few of the main facts upon which this theory rests.

The muscle cells of the wall of the heart have been found to possess five properties, which, while interdependent to a certain extent, are sufficiently distinct to permit of separate study and description.

These five properties are:

1. Stimulus production.
2. Stimulus conduction.

3. Excitability.
4. Contractility.
5. Tonicity.

The coöperation of the first four of these properties are the means through which the rhythmical movements of the heart are initiated and maintained, and are efficient even when the heart is removed from the body. In the intact animal its needs at any particular moment are met by a reflex regulation through the nerves. The influences thus brought to the heart may affect any one of these five properties, and these nerve influences may have a positive or negative effect, heightening or depressing one or more of these five properties of cardiac tissue.

STIMULUS PRODUCTION

The nature of the stimulus which is automatically found in the cardiac muscle cells is not as yet definitely settled. There is, however, considerable evidence which indicates that it consists of a stimulus-material, a chemical compound the constituents of which are not as yet determined, but which has definite affinities and is governed by physico-chemical laws the nature of which we are just beginning to unravel. It seems that this stimulus-material is being continuously manufactured and consists of molecules which increase in size and complexity until a point is reached where its mere complexity renders it an unstable compound, and it is, therefore, suddenly decomposed into its constituent ions. All the stimulus-material in existence at this particular moment is destroyed by this sudden dissociation, but immediately its manufacture is recommenced. This continuous formation of stimulus-material, with its automatic periods of dissociation, constitutes the basis of the rhythmic stimulation of the heart.

This property, the manufacture of stimulus-material, is a function of the muscle cells of all parts of the heart, but is most highly developed in the region of the junction of the superior cava of the right auricle (the homologue of the sinus venosus of the lower animals), hence the stimulus arising here sets the pace, under normal conditions, for the rhythmic activity of the heart, the stimulus passes from the pacemaker to the successive portions of the heart, and as each muscle cell is stimulated all the stimulus-material pres-

ent in the cell at that particular moment is dissociated into its constituent ions.

Under certain pathological conditions parts of the heart other than the area at the roots of the great veins may have this property (the construction of stimulus-material) heightened, and these parts may, through this process, become the pacemaker for the whole heart.

The property of the formation of stimulus-material may be heightened or depressed by the influences brought to the muscle cells by nerve influences and probably also by variations in the chemical constitution of the blood supply. Those influences which accelerate this process are known as positive chronotropic, those which depress are known as negative chronotropic.

EXCITABILITY

is that property of the cardiac muscle by virtue of which it responds to stimuli. It is probably, as Engelmann's experiments show, quite distinct from the properties of stimulus production, conduction and contractility, and is dependent upon molecules entirely different from those upon which these other depend. Excitability may be heightened or depressed quite aside from the positive or negative changes which may occur in the other fundamental properties. Excitability is measured, not by the amount of the reaction resulting from a stimulus, but by the strength of the smallest stimulus that is sufficient to produce a contraction. Thus when a very small stimulus is effective in producing a contraction the degree of excitability is high, when a contraction can be produced only by a relatively large stimulus the degree of excitability is low. Each contraction of the heart temporarily destroys the irritability of its muscle cells. During systole and for a short time after it the heart cannot be excited even by the strongest stimuli. After the systole the property of excitability gradually increases, and smaller and smaller stimuli are effective in producing a contraction. When excitability is heightened, it is assumed that this is due to a more rapid formation of irritable-material; when it is lowered, it is probable that the irritable-material is formed less rapidly.

Nervous and nutritional influences which increase and diminish

irritability have been named by Engelmann positive and negative bathmotropic.

Increase in excitability tends to shorten the cardiac cycle, thus increasing the rate and making the heart susceptible to smaller stimuli. Whether they be intrinsic or extrinsic, such a change is probably an important predisposing element in the production of extrasystoles.

A decrease in excitability will tend to lengthen the cardiac cycle and, hence, will slow the heart.

CONDUCTIVITY

Formerly it was believed that the conduction of stimuli to successive portions of the heart was a function which belonged exclusively to the intrinsic nerves of the heart wall. To-day the evidence is very strong that the function of conducting stimuli is a property of the muscle cells of the heart wall. Without attempting to give all the evidence upon which the latter assumption rests, we may mention the following facts: Morphological and embryological evidence lend probability to the theory that the heart muscle cells are capable of transmitting stimuli from muscle cell to muscle cell. Experimentally the wave of conduction can be made to start from any point in the wall of the heart and pass in a direction opposite to that which it normally takes. This would be exceedingly difficult to explain on the assumption that conduction is dependent on a reflex nervous mechanism, while the hypothesis which assigns this to the muscle cell renders this phenomenon quite intelligible.

The rate of conduction in the heart is relatively slow. In the frog's heart (according to Engelmann) it is three hundred times slower than in motor nerves, and the rate of conduction from auricle to ventricle, where connection is made by a very narrow strip of muscle, the conduction is even slower.

Conductivity is temporarily destroyed during the systole of the heart, but returns gradually after each contraction; hence, this property is believed to be dependent upon molecules which on stimulation are broken down into their constituent ions, whence they are gradually rebuilt, becoming less stable as the molecule increases in size.

Conductivity can be heightened and depressed by nervous influences by the application of heat and cold and the employment of chemicals which modify the normal metabolism. It is slowed or abolished by mechanically narrowing the muscle mass. Such influences are termed positive and negative dromotropic effects.

CONTRACTILITY

is that property by virtue of which the muscle cells become shortened, thus narrowing the chambers of the heart and emptying them of their contents. Since, in accordance with the law established by Bowditch, the contractions of the heart are always maximal, i.e., if it contracts at all, it contracts with all the power of which it is capable at the particular moment. The size of the contraction is a measure of its contractility.

The property of contractility, as is the case with the other fundamental properties, is destroyed for the time being by the contraction of the muscle cell, and, as is the case with the others, this function is gradually restored during systole. The contractile power, therefore, varies with the length of the diastole; that is to say, the longer the period allowed for recuperation, the greater will be the power of the succeeding contraction.

Nervous and nutritional influences which heighten or depress contractility are termed positive and negative inotropic effects, respectively.

While the functions of stimulus production, excitability, conductivity and contractility can be shown to be distinct properties of cardiac muscle, their interdependence is well illustrated by the effect which a modification of one or more of these may have upon contractility. For example, any considerable increase in the properties of stimulus formation and excitability have a negative inotropic effect and contractions are less powerful, while a depression of stimulus production and excitability have a positive inotropic effect and contractility is increased.

TONICITY

Every muscle normally possesses a certain tone; that is to say, even when it is not contracting it maintains a position which is

somewhat short of complete relaxation. The muscle of the heart is no exception to this general rule.

Tonus, while related to the other fundamental properties of heart muscle, is probably quite distinct from them; for example, Porter* has shown that, unlike the other fundamental properties, "tonus contractions" are proportional to the strength of the stimulus employed and have no refractory period. In the frog, Hoffmann† was able to demonstrate that stimulation of certain vagus fibers increase the size and force of the contractions of the heart and increase its tonicity without modifying the cardiac rate.

Tonus allows the heart wall to resist stretching during diastole. The force which stretches the heart during diastole is the pressure of the blood flowing from the great veins. This flow will continue until an equilibrium is established between venous pressure and the tonicity of the heart.

A normal tone aids in maintaining the efficiency of the heart (1) by resisting over-filling, and (2) by promoting an initial tension which is favorable to an effective contraction. Changes in tone are essential in order that the ventricles may be capable of receiving varying quantities of blood, but an excessive tone may lead to a ventricular capacity which is too small and a diminished tone may admit too large a volume of blood and, therefore, lead to dilatation and an inefficient emptying of the ventricles.

The relation of tonus to dilatation and cardiac insufficiency in the individual case is obscure and is a subject which requires further careful investigation.

In order to intelligently interpret the myocardial activities several other features should be held prominently in mind. The law of *"All or None"* or of *"Maximal Contractions"* was discovered by Bowditch in 1871. He showed that if a stimulus was strong enough to induce a contraction, the cardiac muscle responded to that stimulus with all the contractile power of which it was capable at that particular moment; also that the size of the contraction was independent of the strength of the exciting stimulus; a small stimulus, if effective, produced a contraction just as large as a stronger stimu-

*Amer. Jour. Physiol., 1906, xv, 1.

†Arch. f. d. ges. Physiol., 1895, lx, 139.

lus. When the heart muscle was stimulated it responded with a maximal contraction or none at all.

That the cardiac muscle cells possess a *"Refractory Period"* was discovered by Marey in 1875. He was able to show that there was a period beginning just before systole and continuing a short time after it during which the heart will not respond to stimuli even if these are of great strength. The studies of Engelmann have demonstrated that during the refractory period the properties of excitability, conduction and contractility are all abolished. After systole excitability is gradually restored so that, whereas immediately after the refractory period the heart will respond only to stimuli of great strength, as diastole advances the minimal stimulus necessary to produce a contraction becomes progressively smaller. Engelmann showed in like manner that the conductivity and contractility gradually increased with the lengthening of the time between the end of the refractory period and the time when the stimulus was applied.

When we come to study the functions of the primitive cardiac tube in the lower vertebrates (as for example the frog, in which portions of the primitive tube still exist, as the sinus venosus, auricular canal and the aortic bulb), we find that all of its parts possess in a high degree the power of originating stimuli, but the posterior portion of the tube as represented by the sinus venosus is even more excitable than the other parts of the tube, hence normally the cardiac contractions start from the sinus.

The capability of parts of the frog's heart, other than the sinus, to originate stimuli resulting in contraction, is demonstrated by the Stannius experiment. When a ligature is so applied as to separate the sinus venosus from the auricle, the sinus will continue to contract rhythmically, but the rest of the heart ceases to move; after a time, however, the auricle and ventricle again begin to beat, but at a rate slower than that of the sinus and quite independent of the sinus rhythm. If now a second ligature be applied between the auricle and the ventricle, the auricle will continue to beat and after a short pause the ventricle will begin to contract rhythmically at a rate slower than that of the auricle and independent both of the sinus and of the auricle.

There are several phenomena presented in this experiment which particularly attract our attention.

1. The capacity of each chamber to initiate rhythmic contractions independent of the other parts of the heart.

2. The rate of the spontaneous rhythmic contractions is fastest for the sinus, intermediate for the auricle, slowest for the ventricle.

3. In the intact heart, before such ligatures are applied, the rate of the rhythmic contractions of the whole organ is determined by the rate of the sinus, i.e., by the portion which has the fastest rate, or, in other words, that which has the greatest excitability. In a similar manner, when the sinus is cut off, the auricle sets the pace for the ventricle.

As was pointed out in the preceding section on the histology of the mammalian heart certain special structures have been found in different portions of the heart which have many features in common. These are the *"Sinus Node,"* the *"Auriculo-ventricular Node"* and the *"Bundle of His."* A study of the functions of these structures leads us to believe that they are remnants of the primitive cardiac tube and retain the qualities of the original tube in a higher degree than other portions of the cardiac musculature.

Anatomical, morphological and developmental evidence indicate that the *"Sinus Node"* is the normal pacemaker of the mammalian heart. This evidence has been reinforced by considerable experimental work, a brief digest of which may be found in Thomas Lewis' valuable work, "The Mechanism of the Heart Beat," Chapter IV.

There is certain evidence that under pathological conditions the *"Auriculo-ventricular Node"* may become the pacemaker of the heart, giving rise to a form of heart activity that is known as *"Nodal Rhythm."*

In other pathological conditions when the conducting path from the auricle to the ventricle is severed (termed auriculo-ventricular block), the evidence is in favor of the assumption that the main trunk or one of the branches of the *"Bundle of His"* originates the stimuli which set the pace for the ideo-ventricular rhythm which is then established.

CHAPTER IV

Graphic Aids to Diagnosis

Two of the aids which have of late been extensively utilized to interpret myocardial function are the *polygram* and the *electrocardiogram*. The *polygram* is a graphic, synchronous record of two or more parts of the circulatory system, usually of the radial and jugular pulses. The main facts of clinical importance which have been obtained from polygraphic studies of the circulation have been derived from a comparison of the relations of the auricular and ventricular activities. The value of records of the apex beat, carotid, brachial and radial arteries, depends on the fact that they represent more or less accurately the activities of the left ventricle, while the movements of the jugular veins or pulsating liver give us a certain insight into the activities of the right auricle.

In clinical work we usually make use of synchronous records of the radial and jugular tracings (Figure 2). The principal waves of the jugular tracings are, the wave of auricular systole (a); the wave synchronous with, and probably due to carotid pulsation (c); the (v) wave, due to rising auricular pressure during the ventricular systole; the depression (x) is mainly due to the relaxation of the auricle after its systole; the depression (y) is due to emptying of the auricle after the opening of the tricuspid valve; the closure of the semi-lunar valve is frequently marked by a notch in the ascending limb of the (v) wave, and the two portions of the wave have been designated (v^s) and (v^d) (Rihl), to indicate their relations to systole and diastole. The opening of the tricuspid valve is indicated by the termination of the (v) wave.

A fourth wave, which sometimes appears in diastole and called the (h) wave (Gibson), is believed to mark the closure of the auriculo-ventricular valves.

The first step necessary in analyzing the jugular tracing is to locate the (c) wave. To accomplish this one measures accurately with dividers the distance of the foot point of any given radial wave from the line marking the beginning of the radial tracing. One then measures off the corresponding distance from the start-

ing place of the jugular tracing; at the distance equivalent to 0.1 second preceding this point will be found the (*c*) wave, since the pulse wave requires 0.1 second to travel from the carotid to the radial at the wrist. In the normal tracing the (*a*) wave will be found preceding the (*c*) wave by 0.2 second. The (*v*) wave follows the (*c*) wave, separated from it by the depression (*x*). The depression (*y*) follows the (*v*) wave.

In various arrhythmic conditions of the heart there will be found variations in the relations of these waves of the jugular pulse. These variations afford us the means of determining the time relations of the auricular and ventricular activities, and from these can be deduced certain abnormalities in the fundamental properties of the cardiac tissues.

Much time and annoyance in taking polygrams may be saved if one systematically pays attention to the following apparently insignificant details. One should always first examine his instrument to see if all parts, particularly the tambours, are in good working order; the clockwork (usually two sets) should be wound. If smoked paper is to be used, an ample supply should be prepared, so that one need not interrupt his work for this purpose during the record-taking period. For smoking the paper a large candle, a kerosene lamp, burning camphor or a gas burner fitted with a fantail may be employed.

Personally I prefer the gas burner when practical, as by this means the smoke is laid on the paper more evenly and less heavily than by the other methods. If the ink polygraph is used, the pens should be carefully cleaned and filled with the writing fluid.

The patient should be placed on a couch or bed with clothing loosened, to allow access to the parts which it is wished to investigate. He should be in a comfortable position, so that he may relax and lie quietly. In taking a record of the jugular pulse, the shoulders should be slightly raised, the head somewhat flexed forward and rotated to the right and supported by pillows so that the sternocleidomastoid muscles may be perfectly relaxed. When a radial tracing is taken, the position of the radial artery should be marked with a skin pencil; this will be found to facilitate greatly the correct adjustment of the spring of the instrument over the radial artery. The spring of the instrument should rest immedi-

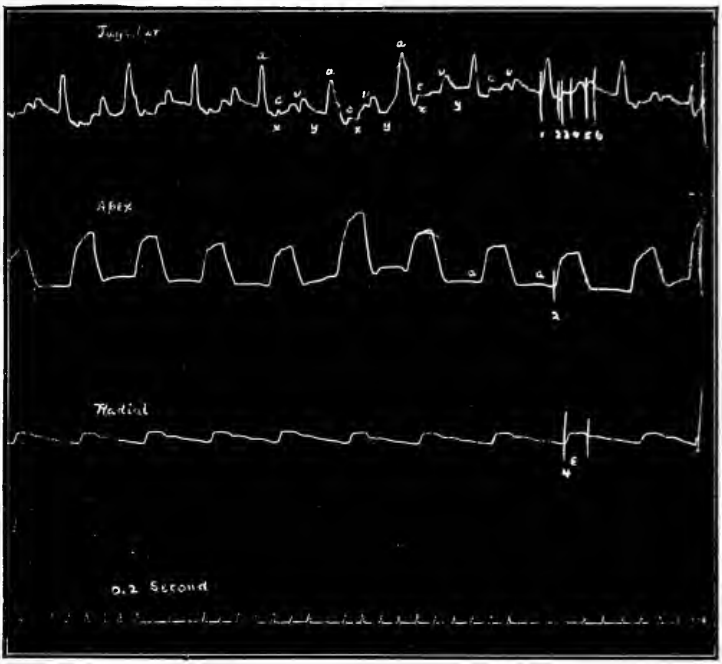

FIGURE 2. NORMAL POLYGRAM

1, beginning of auricular contraction; 2, beginning of apex beat; 3, beginning of carotid wave; 4, beginning of radial wave; 5, closure of semi-lunar valves; 6, opening of tricuspid valve; E = 3 – 5 = ventricular systolic period.

ately over the artery to avoid distortion of the form of the pulse tracing, which a lateral displacement may cause. If the pressure of the spring on the arterial wall is equivalent to the diastolic pressure of the patient, the movements of the spring will be maximal; if this gives a movement to the writing lever greater than is desired, the excursions may be reduced by slightly increasing or diminishing the pressure of the spring. (In polygraph work the time relations of the various tracings are usually of greater importance than the size of the various waves.) In adjusting the brachial cuff of the Uskoff or Erlanger apparatus, the pressure is usually raised to a point equivalent to the patient's diastolic pressure. (In using this instrument one sometimes meets with difficulty in securing a sharp "footpoint" for the waves of the brachial record.) The receiving apparatus for an apex tracing may be a small Mackenzie cup or one of the more elaborate forms of the so-called "cardiographs," which may be strapped to the chest wall or held by an assistant. The position of the apex thrust should be marked with a skin pencil and care should be taken to adjust the receiver to this point. (A receiver placed inside the apex beat will often record the systolic retraction of the tissues near the apex, giving the so-called "inverted cardiogram.") The jugular tracings are best taken with a Mackenzie cup about one and one-half inches in diameter and slightly flattened on one edge. The cup is placed over the jugular bulb, just above and about one-half inch to the right of the right sternoclavicular junction. The flattened edge of the cup should be parallel to the upper border of the clavicle and should be held gently in position by the operator's hand, which is steadied by resting lightly on the chest wall; the cup is provided with a pin-hole vent, which may be covered by the operator's finger, who can thus adjust the internal air pressure without removing the cup. Any considerable pressure of the cup over the veins must be avoided. The receiving cup may be shifted about to obtain the point of maximum venous pulsation; at times the best tracings are obtained at a point higher up on the neck.

In taking tracings of the movements of the liver, a larger cup with a flattened edge should be employed; the abdomen should be relaxed and the cup brought in contact with the under surface of the liver.

Records of the left auricle have been taken by introducing into the esophagus a stomach tube with a single lateral opening covered with a delicate rubber membrane. The tube must be introduced to a point which brings the rubber membrane opposite the pulsating auricle. In taking such records a preliminary course of training is necessary to accustom the patient to the use of the tube.

The changes in air pressure produced in the various receiving devices are transmitted to the tambours connected with the writing levers by rubber tubing; each of these tubes should be provided with a small lateral valve closed automatically by a spring which can be released at any time to regulate the internal air pressure.

When all the levers are seen to be correctly adjusted and moving freely, the time marker is started and finally the clockwork moving the paper. At times, when the respiratory movements distort the jugular tracing, it will be found of advantage to have the patient hold his breath while the polygram is being taken. From time to time the paper should be stopped so that synchronous points on the various curves may be indicated for the sake of subsequent measurement. The smoked paper records are fixed by passing them through a bath of varnish.

The *electrocardiograph* records the differences in electrical potential of the heart muscle during its activity. That every contraction of a muscle is accompanied by certain definite changes in electrical potential has been known since 1856, when Kölliker and Müller detected the action current of the frog's heart by applying electrodes to its surface. When a wave of contraction passes over a muscle, that portion of the muscle which is actively contracting is electrically negative to all other parts of the muscle. It is the object of electrocardiography to detect those differences of potential which occur during the contraction of the heart muscle and to record them graphically. This has been made possible clinically by the genius of Professor Einthoven, of Leyden, to whom we are indebted for the construction of an exceedingly delicate galvanometer known as *Einthoven's String Galvanometer*. This instrument consists of a powerful electromagnet activated by a storage battery of five to six amperes furnishing about ten volts. Between the poles of this electro-magnet is stretched a delicate filament of platinum or of silvered quartz; the diameter of one of these threads

is from two to four microns (about half the diameter of a red blood-corpuscle) and their resistance is about 5,000 ohms. When a current of electricity passes through this string, at right angles to the lines of force of the magnetic field of the electro-magnet, the string is deflected to one side or the other, according to the direction in which the current passes. An arc light passing through a system of condensing lenses and Zeiss objective and projection oculars, magnifies and focusses the shadow of the string on a photographic plate or film.

A Weston normal element, furnishing one volt in connection with a rheostat, is so arranged that the operator may send any desired fraction of this current through the string in either direction, and thus determine the sensitiveness of the instrument at any particular moment. The tension of the string may be increased or diminished at will by means of a micrometer screw, thus readily varying its sensitiveness. The string is usually adjusted so that the passage of one millivolt of current causes a deflection of the shadow of one centimeter. This has been the standard used in the records presented in this entire book.

When a patient is placed in the galvanometer circuit by attaching suitable electrodes to the surface of the body, it is found that certain differences of potential are present which cause a deflection of the string (the so-called "skin current," probably due to the glandular activities of the body). In order that the delicate differences of potential of cardiac activity may not be obscured by this "skin current," it is neutralized by introducing into the circuit in the opposite direction, by means of a rheostat, a sufficient portion of the current from a single dry battery cell to exactly counterbalance the "skin current."

For the purpose of enclosing the patient in the circuit, various forms of electrodes are employed, usually metal vessels containing solutions of common salt, in which the extremities are immersed. A more satisfactory electrode devised by Dr. H. B. Williams is used in our work at the Presbyterian Hospital. It consists of a large thin flexible sheet of German silver; this is moistened with hot salt solution and bandaged to the extremities with flannel strips.

A Jacquet timer, recording fifths of a second, is placed in front of the camera so that the time intervals are recorded simultaneously

with the movements of the string. The photographic apparatus which we have employed makes use of a 200-foot kodak film moved by an electric motor, with convenient devices for numbering, exposing, cutting off, and removing the film according to the length desired. In the Presbyterian Hospital wires are run from the laboratory to the several wards, so that records are easily taken of patients without removing them from their beds. Records thus taken at a distance are known as *telecardiograms*.

The patient under observation should be warm, relaxed and perfectly quiet, since tenseness of the muscles, muscular movements, shivering, talking, coughing, etc., cause irregular movements of the string. (These, however, are usually easily differentiated from the movements due to heart activity.)

The steps necessary to be taken in making an electrocardiogram are as follows:

1. Attachment of three electrodes to the patient (right arm, left arm and left leg).
2. The estimation of the resistance in each of the three leads.
3. The standardization of the galvanometer.
4. The compensation of the "skin current."
5. The recording of the movements of the string of the galvanometer under the influence of the heart action.
6. The development and printing of the films.

When no current is passing through the string of the galvanometer the electrocardiogram represents a base line which shows the string at rest. When the current passes through the string it is deflected and these deflections are shown in the electrocardiogram as waves varying in height and length. The principal features of the lettering of the waves of the normal electrocardiogram, as first suggested by Einthoven, are shown in Figures 3, 4, 5. The wave (*P*) corresponds to the systole of the auricles; the waves (*Q*, *R*, *S*, *T*) correspond to the systole of the ventricles; of these waves (*R*) and (*T*) are most constant and are best understood. (*Q*) and (*S*) may either one or both be absent in the electrocardiogram of the healthy subject. A satisfactory interpretation of all of the elements of the electrocardiogram is not yet possible, but the following summary indicates the significance of the main features as most generally ac-

cepted at the present time: (Some interpretations less generally accepted are placed in brackets.)

P = The auricular systole. (Conduction through the auricle or activity of the sinus region plus conduction through the auricle.)

P to *R* = The time occupied in the conduction of the impulse from the auricle to the ventricle, normally 0.12 to 0.17 second.

Q = The first evidence of ventricular activity; probably portions of the ventricular muscle at some distance from the base.

R = The activity of the basal portions of both ventricles. (Impulse conduction from base to apex.)

S = Activity of apical portion of both ventricles. (Impulse conduction from apex to base.)

S-T = A balance of the potential between base and apex. (An absence of conduction.)

T = The final activity of the ventricle; probably the basal portion near the roots of the great arteries. (Change of electrical potential accompanying contraction of the whole ventricle.)

T to the following *P* = The diastolic period when no current is being developed.

U = The relaxation of the ventricles. (Electrical variation of the arteries.)

In general it may be said that there are two views as to the activities which produce the differences in electrical potential: (1) that all the waves accompany the excitation process; (2) that a part of the waves are due to excitation or conduction and the remainder accompany the activity of contraction.

Sufficient evidence to make a decision between these views is not yet at hand.

The character of the electrocardiographic curve varies with the parts of the body from which it is derived. In routine work it is customary to take three records from each patient designated as follows:

NORMAL ELECTROCARDIOGRAM

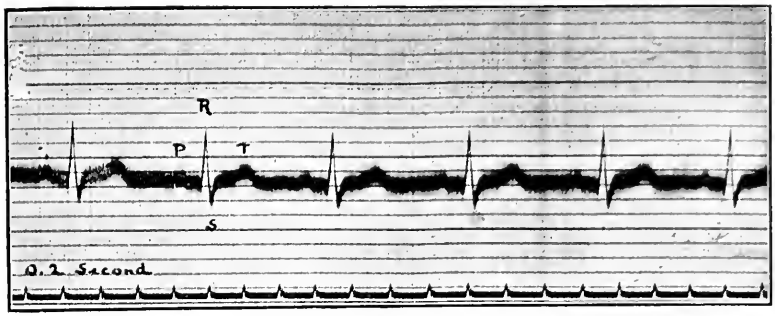

FIGURE 3
Lead I (right arm—left arm)

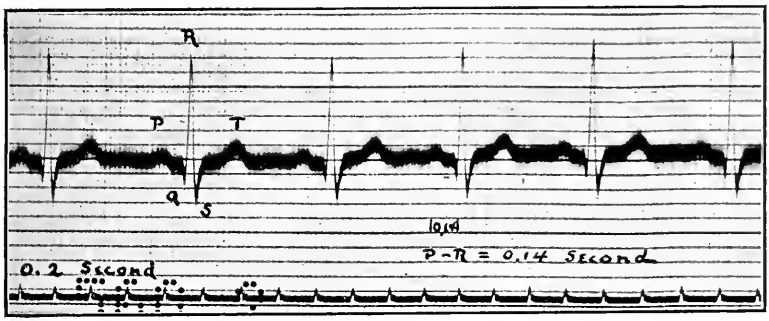

FIGURE 4
Lead II (right arm—left leg)

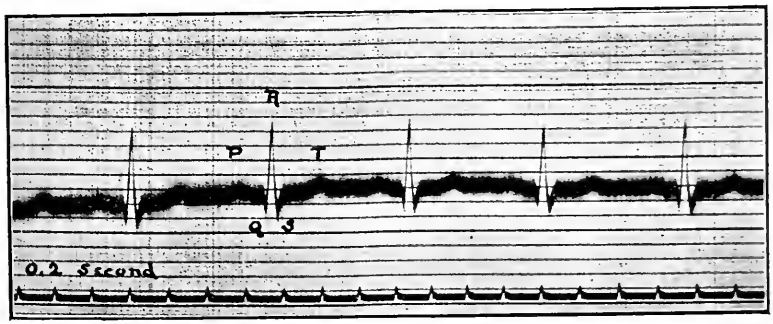

FIGURE 5
Lead III (left arm—left leg)

LEAD I

Current from right arm and left arm.

LEAD II

Current from right arm and left leg.

LEAD III

Current from left arm and left leg.

Electrocardiograms obtained from a normal individual by the three leads as described present different features. The wave (*P*) is positive in *all leads*. (*P*) to (*R*) interval varies slightly in the *three leads*. All the waves of *lead* II are greater than those of *leads* I and III. The wave (*R*) is positive in *all leads*. (*T*) is usually positive in *all leads,* but is occasionally negative in *lead* III. Even in normal individuals there is a considerable range of variation in the electrocardiogram which lies within the limits of the normal. Among these physiological variations may be mentioned a shortening of the diastolic period in increased frequency of the heart, and variation in the aptitude of the (*R*) wave synchronous with respiration; the increase in the size of the (*T*) wave with increased exertion; the changes in (*Q*) and (*S*) coincident with the changes of the position of the heart in the thoracic cavity.

In comparing the value of the polygram and the electrocardiogram as means of interpreting the changes in anatomical and functional conditions of the heart we should observe first of all that these two methods record different sets of phenomena. The polygram is a graphic time pressure curve. The electrocardiogram is a graphic portrayal of the variation of electrical potential during muscular activity. By both of these methods we can study the functions of stimulus production, irritability, conductivity and contractility, but each method has certain advantages and is successful in points where the other is inadequate. They do not portray the same phenomena and are therefore supplemental and corroborative rather than identical. For this reason we have adopted a scheme for taking combined records, i.e., we often record on the same film figures of the electrocardiographic and time pressure curves of the arterial and venous pulses. Conclusions drawn from both types of records are remarkably in accord, thus strengthening the evidence obtained.

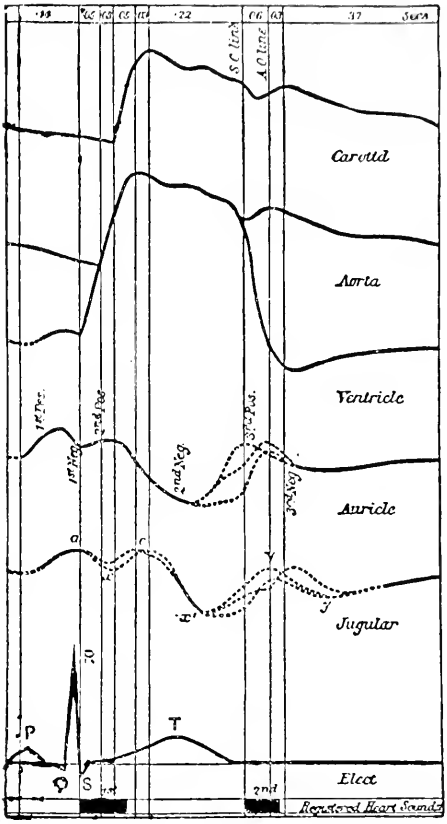

FIGURE 6

Diagram of the pressure changes in the cardiac chambers and their time relations to the aortic, carotid, jugular and electrocardiographic curves and heart sounds. (After Lewis.)

CHAPTER V

Classification of Disturbances of Myocardial Function

The ideal analysis of myocardial functions is based on an examination of the fundamental properties of the muscle cells. Such an examination would show that—

Stimulus production	is	normal,	increased	or	diminished.	
Excitability	"	"	"	"	"	"
Conductivity	"	"	"	"	"	"
Contractility	"	"	"	"	"	"
Tonicity	"	"	"	"	"	"

If there is a departure from the normal (i.e., a depression or an increase in one of these properties) the abnormality may involve the entire musculature or a limited portion of it. Such a classification should therefore indicate the location or site of the abnormality. That is to say, it should indicate the particular part involved, as, for example, the sinus node, the auricular tissue, the auriculoventricular node, the bundle of His or one of its branches, the ventricular muscle, etc., etc.

To complete such a classification the etiological condition should be investigated and we should assign the change in function to some underlying condition, anatomical, nutritional, reflex, etc.

While at present the state of our knowledge and our means of obtaining evidence in regard to all these factors are too incomplete to permit us to make a final analysis in every instance and to assign the existing abnormality to a definite change in one or more of the fundamental properties of cardiac muscle, with an exact site and an accurate causal factor, we are able to accomplish this to a limited extent, but the advances of the past decade, the result of careful clinical observations and well-conceived experiments on animals give us promise of a more extensive knowledge in the near future. It is important that the study of remedies should be based upon a similar analysis so that we may know, for example, whether a particular drug or hydrotherapeutic procedure will heighten or depress excitability in a particular portion of the heart and thus lead

us to its logical utilization in correcting an abnormal state of this property.

As an example of the present possibilities of the use of such a classification, we may cite a case in which we have evidence that all the fundamental properties are normal except that of conduction. We may also have evidence that the conduction is depressed and that the abnormality is localized in the cells of the bundle of His, if the patient has been taking large doses of digitalis (we know that digitalis depresses conductivity), we may find that the withdrawal of this drug allows the cells to recover their property of conduction to the normal degree.

CLINICAL CONSIDERATIONS

For the clinician one of the most common and often an early feature of change in myocardial function is an alteration in the rate and rhythm of the heart action. Since we are to consider these myocardial changes from the standpoint of the everyday practitioner it will be well for us to take our start from this point, and to subdivide our cases into classes, grouped on the basis of easily elicited physical signs, viz. varieties of rate and rhythm. By the employment of graphic methods and other means at our disposal we will endeavor to further analyze each group and to point out as far as we are able the fundamental properties which are disordered, the site of the abnormality and its cause. We will therefore consider our cases in the following groups:

A Regular.
B Irregular.

1. Bradycardia.
2. Tachycardia.
3. Sinus arrhythmias.
4. Extrasystoles.
5. Alternation.
6. Complete irregularity.

THE REGULAR HEART

By the term regular heart we understand one which conforms to the rule (*regula*) of the normal heart. As opposed to the term

regular, the term irregular heart includes all changes in rate, rhythm and character of cardiac contractions which do not conform to the rule of the normal heart.

What then are the rules of the normal heart beat?

1. The *rate* of the normal heart is not fixed at any definite figure; it is dependent on the needs of the body at any particular moment. The heart is a pump which has its greatest efficiency when it is maintaining the needs of the individual organism with the least expenditure of cardiac energy. Any very considerable variation in rate will either fail to meet the needs of the body, or will meet these needs with lack of economy in the expenditure of energy. Hence, the rate, whether too fast or too slow, which *per se* falls short in maintaining an adequate circulation or which does this with an undue expenditure of energy, is inefficient and, therefore, not according to the rule of the normal heart.

Within such limits the rate may vary and yet be normal, but such variations must be gradual. Change in rate in the normal heart is accomplished by a change in the length of the diastolic period, but to conform to the rule the difference in length of successive diastolic periods must be infinitesimal. A regular heart always has a rate within the normal limits, but a heart with a normal rate may be irregular since in some other features it does not conform to the rule of the normal heart.

2. The *rhythm;* the rule of the normal heart is that it must not only beat rhythmically, but also that the rhythm must have a special well-defined character; the individual cycles which compose the rhythm must all be of the same kind and size, and the diastolic portions of successive cycles must be of equal length.

A regular heart is always rhythmic, but a rhythmic heart is not necessarily regular.

The *pulsus alternans* is an example of heart activity which is perfectly rhythmic and yet, according to our definition, is irregular; it is composed of alternating large and small beats which succeed each other at equal intervals and is therefore absolutely rhythmic, yet since the kind of rhythm does not conform to the rule, since successive beats are unequal in size, it falls into the class of irregularities.

3. The *pacemaker* of the regular heart is the sinus node;

when any other portion of the heart either customarily or occasionally initiates the stimulus which results in a contraction, this heart must be included in the class of irregular hearts.

4. In the regular heart the wave of contraction must sweep over its chambers in *an orderly sequence* and the *stimulus must follow the path which we have learned to recognize as normal.* Any deviation in the path which the stimuli follows or any abnormality in the sequence of contraction of the chambers brings it into the group of irregular hearts.

5. In the regular heart not only must *the stimulus* sweep over the heart by the normal paths and in the normal direction, but it *must travel at a speed which is normal.* Any delay in transmitting stimuli places a heart among those classified as irregular.

6. Among other features to which the heart must conform to be considered regular are uniformity in the size and duration of successive systoles, and a condition of the muscle mass which is somewhat short of complete relaxation during diastole.

The departure from the normal in (1) *rate* and (2) *rhythm* are easily detected by the ordinary methods of inspection, palpation and auscultation. An abnormal (3) *pacemaker;* an unusual (4) *path* or *direction* taken by the *stimulus;* (5) a delay in the *speed* of the *passage* of the *stimulus* and the finer *variations* (6) in the *character of the contractions of the ventricles* are often best detected by the employment of graphic methods, but when one has once become familiar with the evidence obtained by such means, physical signs are quite sufficient in the majority of instances to afford us data upon which to base a correct interpretation of the abnormalities which are present.

THE IRREGULAR HEART

In the preceding section it was stated that the group of irregular hearts includes all those which show a departure from the normal in rate, rhythm and character of contractions. In the succeeding paragraphs an attempt was made to define the "rules" of normal cardiac activity. We will next consider the various types of irregular hearts which are distinguished by well-defined changes in rate and rhythm.

ABNORMAL CHANGES IN RATE

Under irregularities of the heart are included all those changes of rate which exist at the expense of the functional efficiency of the heart. The normal heart is a machine which provides the individual at any particular moment with a sufficient blood supply, and at the same time is working with an economical expenditure of energy; it is working at an optimum. The adaptation of the rate of the heart to the needs of the body is controlled very largely through the extracardial nerves. Anatomical and functional evidences show that for the most part the fibers of the right vagus and the right accelerator (sympathetic) nerves terminate in the tissues in the region of the sinus node while the left vagus and left accelerator are more particularly distributed to the auriculo-ventricular node and the tissues junctional between the auricle and ventricle. By reflex activity through these paths the rapidity of stimulus production is modified. There is considerable physiological and clinical evidence that both these nerves possess what is known as "tone," that their activity is continuously modifying the stimulus production of the cells of the heart; the vagus tends to hold this property in check, the accelerator tends to heighten its activity; it is through a correct balance of these forces that the heart activity is varied with the momentary demands of the organism.

Hypertonus of either of these nerves results in a heart rate abnormally rapid or abnormally slow.

Among the factors which modify the rate of the heart are individual differences in the age, size of body, build, work, temperature, nervous constitution, arterial pressure, etc., etc.

CHAPTER VI

Bradycardia. Heart Block

It is well to recall first of all that a slow pulse is not necessarily synonymous with a slow heart. The heart contractions may be of such unequal strength that only a portion of them are detected in the radial artery; some of the systoles may be so lacking in force that the resulting arterial wave may be insufficient to affect the pressure in the radial artery, or again they may even fail to open the aortic valves (*pulsus frustrans*). We should therefore always check up our finding of a slow pulse by counting the apex beat by auscultation. Hence a radial count alone is not sufficient to establish the existence of a bradycardia.

All really slow hearts are comprised in two classes:

1. True Bradycardia.
2. Heart Block.

1. In *true bradycardia* all the chambers of the heart contract at a slow rate and in the normal sequence and relationship. It might be fairly questioned whether such hearts should be included in the class of irregular hearts, since although slow, their activity is usually efficient in maintaining an adequate circulation, economical in the expenditure of energy, and in other respects conforming to the rules of the normal heart. However, some of these hearts are too slow to properly supply all parts of the organism with sufficient blood, and therefore this group of bradycardias may fairly be included among the irregular hearts.

A slow heart is not a very rare occurrence, a rate between 50 and 60 is common in tall persons, in those with increased arterial pressure, aortic stenosis, pregnancy, convalescence from acute fevers, in typhoid fever, meningitis, chronic nephritis, cerebral hemorrhage and tumors; it is often associated with jaundice and some digestive reflexes, such as vomiting, etc. When we attempt to classify these heterogeneous clinical manifestations it seems reasonable to divide them into two groups:

(*a*) *Toxic agents,* which probably have a direct depressing effect on the sinus node (typhoid fever, jaundice).

(*b*) *Heightened vagus tone,* either from direct irritation of the pneumogastric center (meningitis, cerebral hemorrhage), or a reflex activity (vomiting, pregnancy, increased arterial tension, etc.).

It is to be noted that a true bradycardia is due to a depression of activity of the sinus node, either through the chemical constitution of its blood supply, or through the nervous influences brought to it through the extracardial nerves, particularly the right vagus. The change which takes place in the node is a depression of the property of the formation of stimulus material or of its excitability, or both; at present we have no clinical method of determining which one of these properties is the one affected in any particular case.

At a later time the effects of vagus activity will be more fully discussed; in passing it will be sufficient to note that by the administration of atropin, vagus impulses may be cut off and thus a clinical estimate may be made of the influence which it has hitherto been exerting.

A true bradycardia is never encountered with a rate under 40. Probably every heart with a rate less than this belongs to the second group of slow hearts, viz.:

HEART BLOCK

2. Which is the result of interference in the conduction of stimuli from one part of the heart to another. Theoretically such an abnormal condition may occur in any part of the musculature of the heart; practically it is rarely recognized, except when it involves the bundle of His or one of its branches. Here the cells of the conducting system are grouped in a narrow band so that a very limited lesion or functional derangement of moderate extent is sufficient to produce marked clinical phenomena.

In accordance with the degree of functional disorder we may recognize:

(*a*) Total Heart Block, complete dissociation.
(*b*) Partial Heart Block, partial dissociation.
(*c*) Delayed Conduction, without dissociation.

(*a*) TOTAL HEART BLOCK; COMPLETE DISSOCIATION

It will be recalled, as was pointed out in the paragraphs on the anatomy and function of the heart tissue, that stimuli normally originate in the sinus node, thence spread over the auricle to the auriculo-ventricular node of Tawara, where connection is made with the bundle of His; through this the impulses pass to be distributed first by the two branches of the bundle, and later by its subdivisions and their connections with the Purkinje's fibers to all parts of the ventricular muscle. It will also be recalled that in the Stannius experiment on the frog's heart, when the second cut or ligature is applied so as to separate the auricle and the ventricle, the auricle continues to contract rhythmically in the normal manner and after a considerable pause the ventricle begins to contract at a slow rhythm entirely independent of the auricular contractions. This is precisely what happens in man when the property of conduction of the bundle of His is destroyed. The auricles continue to contract in a normal manner in response to the rhythmic stimuli arising in the sinus node; these impulses are unable, however, to pass the obstruction in the bundle of His and hence are unable to influence the activity of the ventricle. Since, however, the uninjured portions of the bundle still possess the fundamental properties of the production of stimulus material and excitability, stimuli will be set free at this point and the ventricle will respond by slow rhythmic contractions entirely independent of the contractions of the auricle and of the stimuli originating in the normal pacemaker. This condition is known as *complete dissociation* and the activity of the ventricle as the *ideo-ventricular rhythm.*

(*b*) PARTIAL HEART BLOCK; PARTIAL DISSOCIATION

If the bundle of His is not completely functionally severed but is merely injured so that the property of conduction is depressed (that is to say if the formation of the molecules upon which the conduction of impulses is dependent is abnormally slow) the ventricle may not respond to every impulse from the auricle. This condition is known as *partial dissociation.* If the ventricle ordinarily responds to the stimuli from the normal pacemaker, and only occasionally fails to contract in this manner, the condition is known as the

dropped beat. If the ventricle responds to every second or third auricular contraction it is called a *2 to 1* or a *3 to 1* rhythm. Or, if for every 5 beats of the auricle we have 3 contractions of the ventricle the condition is known as *partial dissociation* with a *3 to 5 rhythm.* At times the periods of ventricular response may be so long that an occasional stimulus may be initiated in the bundle and we then have a *partial dissociation with interspersed ideo-ventricular contractions* (*"escape of the ventricle"*).

(*c*) DELAYED CONDUCTION WITHOUT DISSOCIATION

The conducting tissues may be so affected that the rate of conduction may be much less than the normal so that the passage of the stimulus from the auricle to the ventricle consumes a period of time appreciably in excess of what is usual; if, however, the ventricle responds to each stimulus originating at the pacemaker there is no dissociation. This form of impaired function may easily pass over to a partial heart block, or even a complete block and a single case may exhibit grades of conduction changes comprising delayed conduction, partial and complete block on successive observations.

PATHOLOGY

Heart block of all degrees has been produced experimentally by various procedures which have had for their object the destruction or injury of the bundle of His. Ligature of the bundle in the perfused heart (Humblet) and the dog's heart in situ (Erlanger), crushing by means of an auriculo-ventricular clamp (Erlanger), section of the bundle in the perfused heart (Cohn and Trendelenburg) have uniformly produced some degree of block whenever subsequent histological examination demonstrated injury to the bundle. Heart block has been produced by stimulation of the vagus (Chauveau) and as a direct result of asphyxia (Lewis, Sherrington). Various degrees of temporary or permanent block have been produced by the injection of various cardiac poisons such as digitalis (Cushny, Tabora), adrenalin (Kahn), aconite (Cushny), muscarine and physostigmine (Rothberger and Winterberg).

Cases which have exhibited the evidence of heart block have almost invariably shown some histological alteration in the bundle

of His when such an examination has been made. As a general rule the degree of heart block corresponds with the extent of the histological change which is found in the bundle; a complete heart block usually corresponds to a complete destruction of the bundle, while delayed conduction is more apt to be associated with a moderate degree of infiltration,* in the exceptional case a complete block may occur with but very little apparent histological change† and a case of partial block may exhibit an extreme degree of bundle destruction. These exceptional cases merely emphasize the fact that histological observations cannot always be relied on to measure the degree of functional impairment.

Bachmann‡ has collected from the literature sixty-three cases of heart block reported since 1899. Complete clinical and histological data were obtained in twenty-four or these; in the others the information obtained was interesting, but incomplete in all details. In all cases where a complete transverse lesion was found, there had been a complete heart block. In a group of cases showing variations from partial to complete block, the extent of the histological examination did not show changes which could be regarded as proportional to the degree of functional disturbance. In five cases, including those showing both partial and complete block, no histological alterations in the bundle of His were found. Bachmann calls attention to the fact that other parts of the conducting system were not minutely studied, hence the evidence of absence of all histological change is not absolutely conclusive in these cases.

Probably the lesion of the bundle of His which has been most frequently found is the result of a syphilitic infection, either a gumma or an old syphilitic scar. Calcareous nodules, chronic inflammatory changes, fibrosis, calcareous degeneration and necrosis involving the bundle, have been found; more rarely an ulcer penetrating the septum; atheromatous changes in the central fibrous body; fibroid and epithelial tumors have been described. Acute inflammatory conditions may be present with leukocyte infiltration and degeneration of the cells of the bundle.

*Pardee: Arch. Int. Med., 1913, xi, 641.
†Krumbhaar: Arch. Int. Med., 1910, v, 583.
‡Bachmann: Jour. Exp. Med., 1912, xvi, 25.

ETIOLOGY

In addition to the cases of distinct syphilitic origin, others seem to bear a direct relationship to the more acute infections. More or less severe cases of heart block have followed diphtheria, typhoid fever, influenza, septic poisoning, puerperal fever and pneumonia; these diseases naturally supply the etiological factor in heart block as found in youth and young adults. In elderly people the lesion, when not syphilitic, is often merely a phase of one of the common general chronic inflammatory or degenerative processes, whose etiology is still for the most part shrouded in so much obscurity. The degenerative changes accompanying arteriosclerosis of the coronary arteries are sometimes associated with various degrees of heart block.

There is a group of cases, the majority of which show only mild grades of interference with conduction, which are undoubtedly of rheumatic origin. Mackenzie was the first to draw attention to this group. Many of them have had pericarditis or endocarditis particularly with involvement of the mitral valve. There seems to be little question that the acute and subacute rheumatic inflammatory processes have a tendency to implicate not only the pericardium and the endocardium, but the myocardium as well. Keith's examination of hearts which had been observed clinically by Mackenzie* showed that the inflammatory process had a tendency "to extend from the base of the valve into the central fibrous body, and to involve the bundle."

It has been pointed out that experimentally toxic doses of digitalis may produce block in the normal heart; the administration of such doses is, of course, impossible in many, but the effect of moderate doses of digitalis and other drugs of the same group on hearts with impaired conduction may often be observed in the clinic. Given in such cases digitalis usually lengthens the conduction time and may even induce a partial or a complete block. The question is still unsettled as to whether digitalis acts in these cases directly on the heart tissues or through the vagus nerve.

Heart block has been produced experimentally by stimulation of the vagus nerve. It is a question whether a clinical heart block

*Mackenzie: Diseases of the Heart, p. 179.

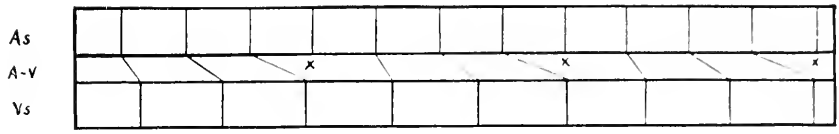

FIGURE 7

DELAYED CONDUCTION PRODUCING PARTIAL BLOCK

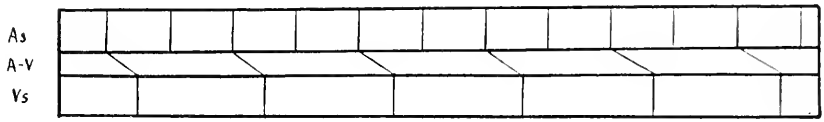

FIGURE 8

PARTIAL BLOCK

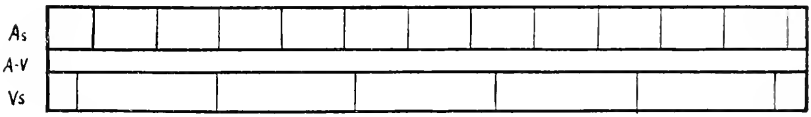

FIGURE 9

COMPLETE BLOCK

Diagrams showing the mechanism of various degrees of heart block. As = auricular contraction. A-V = conduction from auricle to ventricle (note the variation in the length of this period). Vs = ventricular contractions.

can be initiated by vagal changes, but in damaged hearts the conduction abnormalities may be accentuated by vagal reflexes. A very beautiful illustration of this influence is a case reported by Mackenzie*; the conduction time was usually slow and the reflex obtained by swallowing repeatedly produced a partial block when the patient was under the influence of digitalis. Conduction disturbances following vagus stimulation have been studied by Robinson and Draper,† who have published some very beautiful electrocardiograms showing changes in conduction of various degrees. They reached the conclusion that the left vagus has as a rule a greater influence on the property of conduction than the right vagus.

IDENTIFICATION

Clinical: When the pulse rate is under 60 one may suspect some degree of interference with the property of conduction; in such a case, however, one should always compare the rate of cardiac contractions as determined by auscultation with the pulse rate, since not infrequently one finds a large number of ventricular systoles which are so inefficient as to make no impression on the radial pulse (when at a later time we take up the discussion of extrasystoles and auricular fibrillation it will be pointed out that in these conditions many ventricular contractions may be detected on auscultation over the precordium which afford in the radial artery no evidence of their presence).

If the heart is perfectly rhythmic and has a rate in the neighborhood of 30 it is practically certain that a complete block is present. A faster ventricular rate does not, however, rule out the possibility of a complete block, 8 out of 34 cases of complete block which I have studied by graphic methods had a ventricular rate of over 45.

In a partial block the ventricle may contract rhythmically or at times may be quite arrhythmic, the rate, while usually a slow one, is as a rule faster than in complete block. A partial block is to be suspected when the ventricular rate suddenly changes to one half its former rate. A single dropped beat is usually due

*Ibid., p. 340, also Plate IV, Figs. 258 and 259.

†Jour. of Exper. Med., xv, No. 1, 1912.

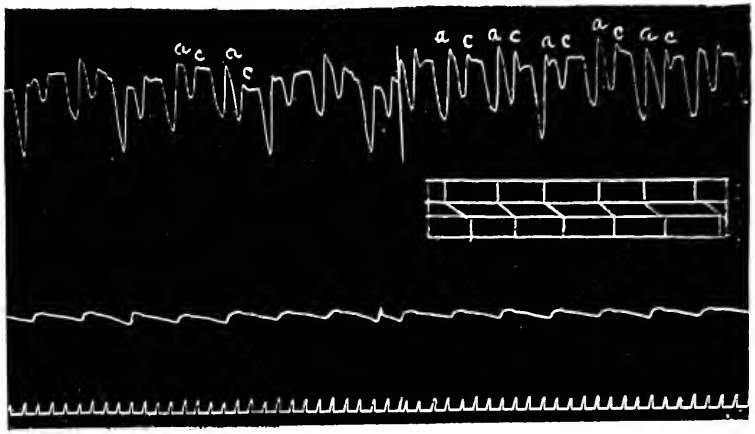

FIGURE 10

Delayed conduction. No dissociation. *a-c* interval = 0.3 second. Auricular rate = 73. Ventricular rate = 73.

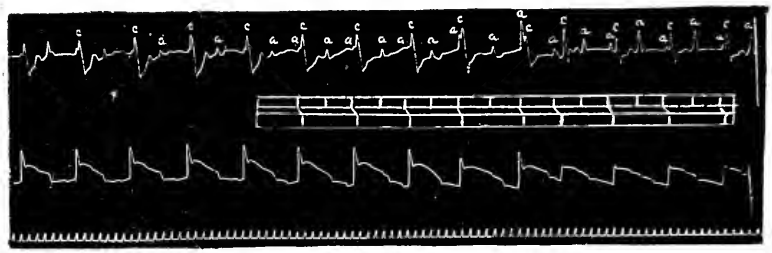

FIGURE 11

Partial block. For the most part this is a 2 to 1 block, but occasionally an extra auricular impulse passes the block. Auricular rate = 92. Ventricular rate = 42.

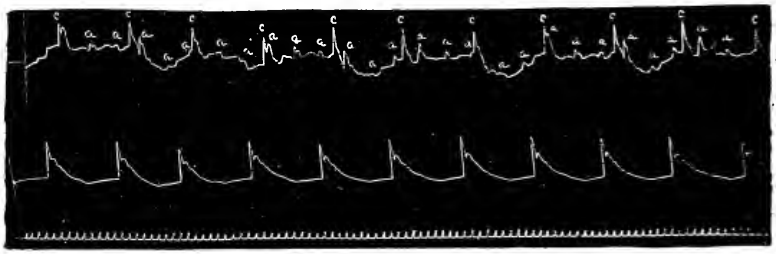

FIGURE 12

Complete block. Auricular rate = 98. Ideo-ventricular rate = 31.

to a partial block, but this can only surely be determined by the evidence of graphic records.

When the pulsations of the jugular vein are visible they are of considerable aid in making a diagnosis of the character of the irregularity. In complete block the jugular pulsations are usually rhythmic, but occur at much more frequent intervals than and quite independent of the ventricular impulses. Occasionally there may be seen a large jugular pulsation in place of one of the usual smaller ones, and if this phenomenon is closely observed it will be apparent that it occurs at a time when the ventricle contracts synchronously with the jugular pulsation, that is when the auricles and ventricles contract simultaneously. In incomplete block the jugular pulsations usually bear a definite numerical ratio to the ventricular contractions, for example, we may see in a 2 to 1 block, two jugular pulsations to one apex beat, or three jugular to two ventricular in a 3 to 2 block, etc.

In delayed conduction it is sometimes possible to detect a longer than the normal interval between the first venous wave and the carotid pulsation, or this prolonged interval may become evident when we note the time by which the first wave of the jugular pulse precedes the apex beat.

In a few cases of block a low muffled sound may be heard during ventricular diastole; this is the auscultatory evidence of the auricular contraction which is sometimes heard; when this occurs soon after the second sound or a distinct interval before the first sound it sometimes gives the impression of a reduplication of the second or of the first sound, as the case may be.

In certain cases of mitral stenosis associated with heart block there have been described (Mackenzie) short, harsh murmurs which occurred synchronously with the pulsations of the jugular vein and quite independent of the ventricular contractions. These were ascribed to the acceleration of the flow of blood from auricle to ventricle at the time of the independent auricular systole. When present this sign should be of assistance in recognizing the independent activities of the auricles and ventricles; personally it has been my fortune to see only one case of this kind.

The Adams-Stokes syndrome, attacks of unconsciousness associated with a slow pulse, should always suggest the possibility of

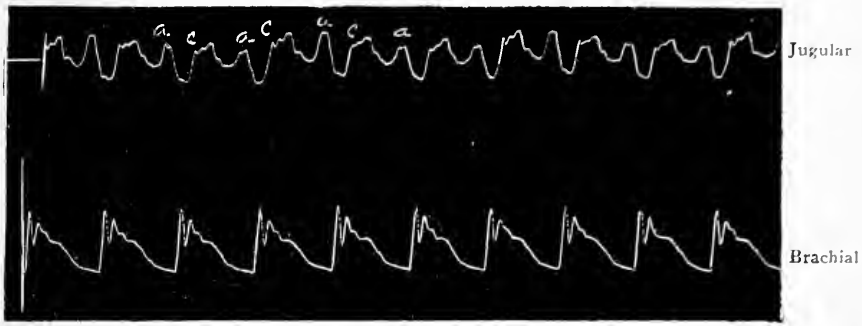

FIGURE 13

Delayed conduction *a-c* interval = 0.35 second.

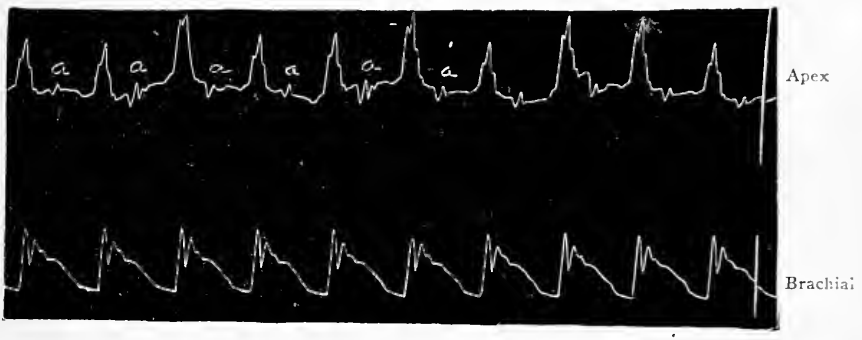

FIGURE 14

Delayed conduction. Apex and radial tracings. The auricular wave is seen in the apex curve and precedes the ventricular wave by 0.35 second.

heart block. It should be remembered, however, that heart block and the Adams-Stokes syndrome are not interchangeable terms.

A fluroscopic examination often will demonstrate the independent activities of the chambers.

Polygraphic tracings (Figures 10, 11 and 12) will usually give conclusive evidence of conduction defects when these are present. If this property is only moderately depressed, the ventricle (as shown by the apex or arterial tracing) will have a normal or slow rate and the intersystolic periods will be uniform in length (Figure 10), the jugular tracing, representing the activity of the right auricle, will show an *a* wave preceding each *c* wave at a uniform interval, but this *a-c* interval will exceed 0.2 second, the time occupied by the normal *a-c* interval.

If conduction is a degree more defective the arterial pulse may show a rhythmic activity indicated in the diagram (Figure 7), the diastolic periods gradually increase in length until the longest pause is reached at the time of a "dropped beat," then suddenly this period is shortened only to be again gradually lengthened until another beat is dropped. When we examine the auricular diagram we see that the auricular (*a*) waves recur at regular intervals; the *a-c* interval following the "dropped beat" may be normal, 0.2 second, in length(it usually exceeds this), but each successive *a-c* interval is longer, since the property of conduction is becoming more and more exhausted, and each ventricular response is thus delayed until one of the auricular impulses reaches the junctional tissues while they are still in the refractory state, hence no impulse is conveyed to the ventricle and a "dropped beat" results. At the time of the next auricular impulse the long preceding rest has considerably restored the functional condition of the auriculoventricular bundle, consequently the *a-c* interval is much shorter and the ventricular response is prompt. This rhythmic lengthening of the ventricular cycle superficially resembles the respiratory *sinus arrhythmia,* it can be differentiated from this condition by observing that in conduction defects (1) the rhythmic change in the length of the ventricular cycles is not synchronous with the phases of respiration; (2) the *a* waves of the jugular tracing are separated by equal intervals; (3) the *a-c* intervals exceed 0.2 second.

When the conduction is even more abnormal the arterial pulse

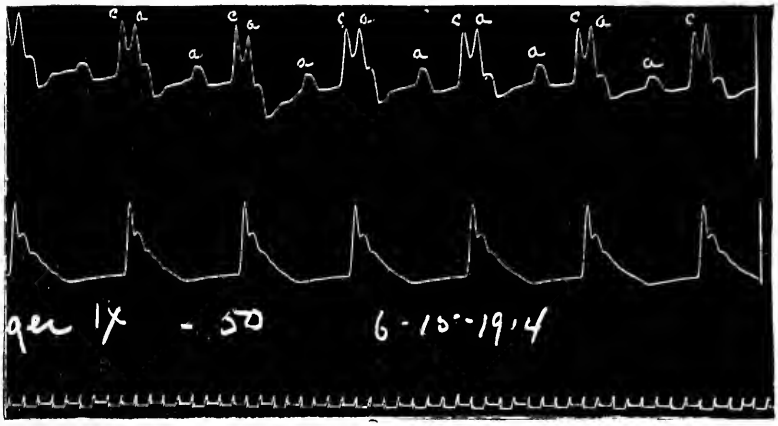

FIGURE 15
2 to 1 block. Auricular rate 70. Ventricular rate 35.

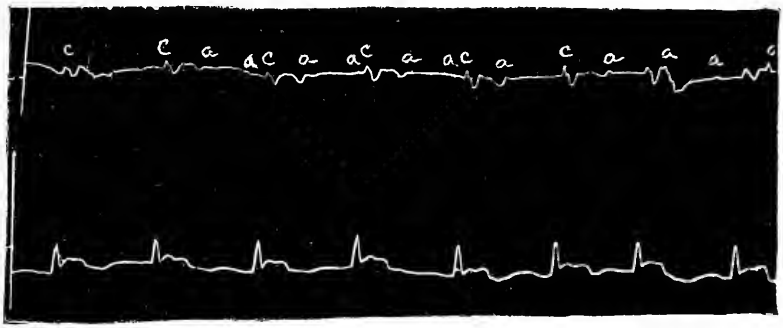

FIGURE 16
Complete block

will be slow (usually 40 to 50 per minute). In the jugular record rhythmically recurring *a* waves will be found (Figures 8 and 11), but the ventricle responds only to every other or every third impulse from the normal pacemaker. The *a-c* interval when present may be of normal duration; it is usually prolonged.

In complete block (Figures 9 and 12) the arterial pulse is slow, usually 30-35 per minute and perfectly rhythmic, in the jugular tracing are found the equally spaced *a* waves which bear no fixed relation to the ventricular waves. The *a* waves are equidistant from one another as are also the *c* waves but the two rhythms are entirely independent of one another.

The *Electrocardiogram* furnishes evidence of conduction defects which is even more clear than the polygram.

As has been pointed out, the *P* wave represents auricular activity, *Q R S T* ventricular activity. Normally the *P-R* interval, measured from the beginning of the *P* wave to the beginning of the *R* wave, is between 0.14 and 0.18 second; a *P-R* interval occupying more than 0.18 second indicates a delay in the passage of the stimulus from the auricle to the ventricle and is due to a defect in the property of conduction.

The simplest form of this irregularity is shown in Figures 17, 21 and 22, the pulse is beating slowly and rhythmically at a rate of 60. Each ventricular complex is of the normal type and is preceded by a *P* wave; the *P-R* interval is always of the same length, but is excessively long, measuring 0.37 second (Figure 17). Such a heart, on physical examination, might show little deviation from the normal, but the electrocardiogram makes very evident the underlying defect.

The records of cases of partial block resulting in the "dropped beat" is shown in Figures 18 and 23. The *P* waves are picked out from such a record with little difficulty. The ventricular complexes (*Q R S T*) are quite normal in form except when they are distorted by a superimposed *P* wave. The *P* waves of the first two cycles shown in the record (Figure 18) are easily recognized, if one measures the time between these *P* waves (approximately 0.6 second) and, beginning with one of these easily identified *P* waves, lays off on the remainder of the record intervals similar in length, one will find at each one of these points a wave either clearly de-

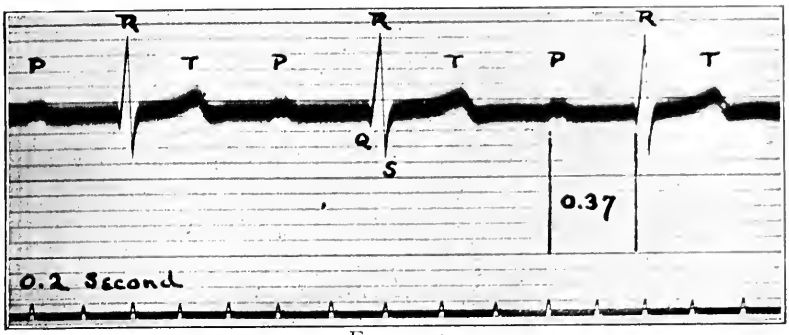

FIGURE 17

Delayed conduction. Every ventricular complex (QRST) is preceded by an auricular complex (P). The P-R interval is excessively long, 0.37 second. The notch in the P wave is a slight abnormality not infrequently seen in cases of mitral stenosis. Auricular rate = 60. Ventricular rate = 60.

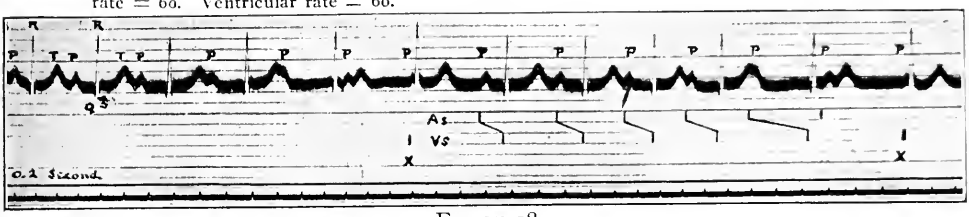

FIGURE 18

Partial block. Auricular rate = 102. Ventricular rate = 89. P-R = 0.18 to 0.43 second. *Note* gradual lengthening of the P-R interval resulting in the "escape of the ventricle" at x, also the rhythmic lengthening and shortening of the ventricular cycles. P recurs at equal intervals of time, but its relation to the ventricular waves varies, *note* how the P wave distorts Q.R.S. and T at various points.

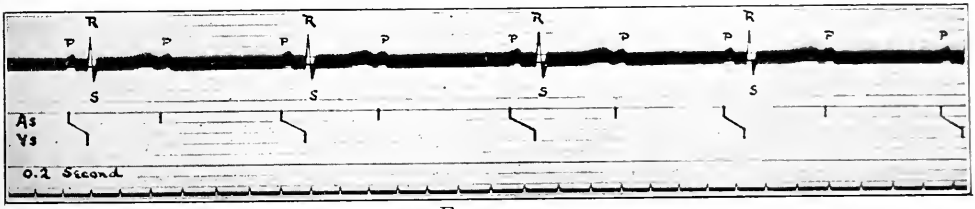

FIGURE 19

Partial (2 to 1) *block.* Auricular rate = 86. Ventricular rate = 43. Every other auricular impulse is blocked. P-R interval = 0.15 second. *Note* alternate short and long auricular cycles (sinus arrhythmia) and slow regular contractions of the ventricle.

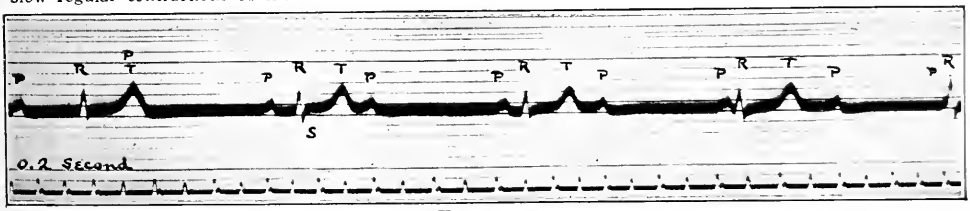

FIGURE 20

Complete block. Auricular rate 60. Ventricular rate 37. The auricular and ventricular activities are entirely independent of each other. *Note* P is slightly notched. The ventricular complex (RST) is normal in type except when distorted by a superimposed P wave.

fined or appearing as a notch changing the normal form of the ventricular complex; these are the *P* waves representing the auricular activity which recur rhythmically at equal time intervals. The *P-R* intervals vary in length from 0.18 second to 0.43 second; this gradually lengthening indicates a progressive exhaustion of conductivity, at last (at x) the period becomes so long that the ventricle contracts spontaneously (known as the "escape of the ventricle") without waiting for an impulse to reach it from the normal pacemaker; the auricular impulse (indicated by the *P* which merely notches the ascending limb of the *R* wave at x) reaches the ventricle while it is in the refractory period, hence there is no ventricular response to this impulse. After the rest thus afforded to the junctional tissues the ventricle responds promptly to the next auricular impulse and the *P-R* interval measures only 0.18 second only again to be gradually lengthened. The record shows an auricular rate of 102, a ventricular rate of 89. The mechanism which underlies the gradual lengthening and the sudden shortening of the ventricular cycles is quite evident.

Another type of partial block is shown in Figures 8 and 19. In this case the ventricle responds to every other auricular impulse. The *P-R* interval conforms to the normal length (0.15 second) but the exhaustion of the *A-V* bundle is shown in its inability to transmit the next succeeding impulse coming down from the sinus, so that ventricular responses and "dropped beats" alternate, showing a 2 to 1 block. The existence of a sinus arrhythmia evidenced by the alternating short and long periods between the auricular contractions, may be, in this case, an additional element in favoring a partial block. If the auricular responses were equidistant it is quite possible that the junctional tissues would have recovered sufficiently to convey the impulse to the ventricle, but the shortened auricular diastole which regularly follows a ventricular response does not allow enough time for the recovery of the functionally defective conduction.

A comparison of these two cases of partial block represented in Figures 18 and 19 is interesting. In the first case (Figure 18) the auricle contracts regularly, the ventricle irregularly. In the second case (Figure 19) the auricle contracts irregularity, the ventricle regularly and at a much slower rate than in the preceding case. The

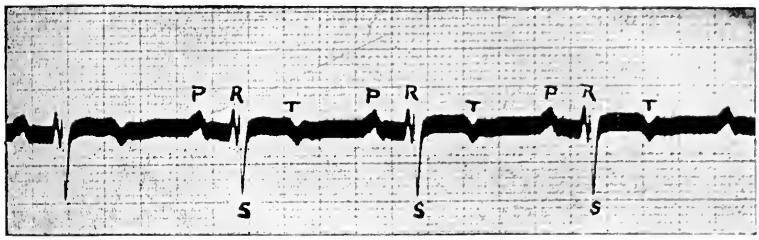

FIGURE 21

Delayed conduction. *P-R* interval = 0.2 second.

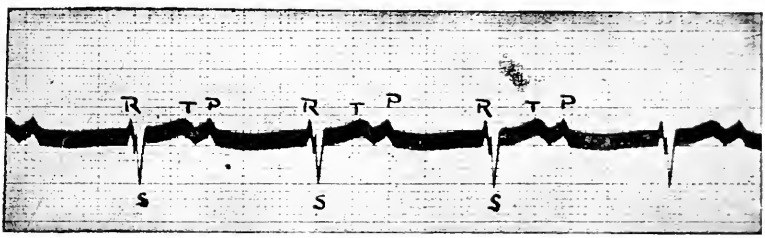

FIGURE 22

Delayed conduction. *P-R* interval = 0.6 second. From the same case as Figure 21 after a considerable amount of digitalis had been given.

irregularity in each case is a rhythmic one. In the first case a regular auricle associated with a defect in conduction produces a rhythmic irregularity of the ventricle. In the second case the rhythmic irregularity of the auricle associated with a defect in conduction produces a regular activity of the ventricle.

The electrocardiogram of a case of complete block is shown in Figure 20. Here it is to be noted that the ventricular complexes (*R S T*) are normal in form except when they are distorted by superimposed *P* waves which recur at regular intervals but with no fixed relation to successive ventricular complexes. The *P* waves are equidistant from one another but fall in any portion of the ventricular cycle (both systole and diastole). The ventricle is contracting at the rate of 37 and is perfectly regular, the auricle with a slight arrythmia contracts at a rate of 60. The activities of the upper and lower chambers are quite independent. This complete dissociation of auricles and ventricles is entirely characteristic of complete block. No impulses can pass from the auricle to the ventricle and each has its independent rhythm. The pace of the auricle is set by the normal pacemaker, the sinus node. The ideo-ventricular pacemaker is in this case located in the bundle of His above its bifurcation (if the impulses which initiate the contractions of the lower chamber were in some other portion of the ventricular musculature, as is sometimes the case, the ventricular complexes would be of an entirely different type).

Only those cases have been described which represent distinct types of block; it should be said, however, in passing that a single case may present various degrees of block at different times, delayed conduction, partial and complete block may present themselves in a single case at successive periods and more rarely one may follow a case passing through the stages of complete and partial block back to a condition of normal activity.

A number of the simpler and more common types of partial and complete auriculo-ventricular block have been discussed; this list, however, by no means exhausts the varieties of heart block which are seen in the clinic. Block associated with sinus irregularities, extrasystoles and auricular fibrillation are some of the types which will be taken up in subsequent chapters.

The differentiation of a block produced by an organic lesion

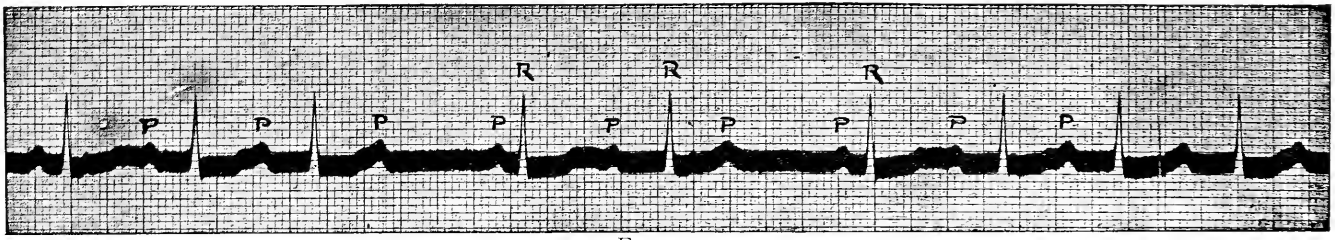

FIGURE 23

Partial block. Note variation in the length of the *P-R* interval. Occasionally the auricular impulse is blocked.

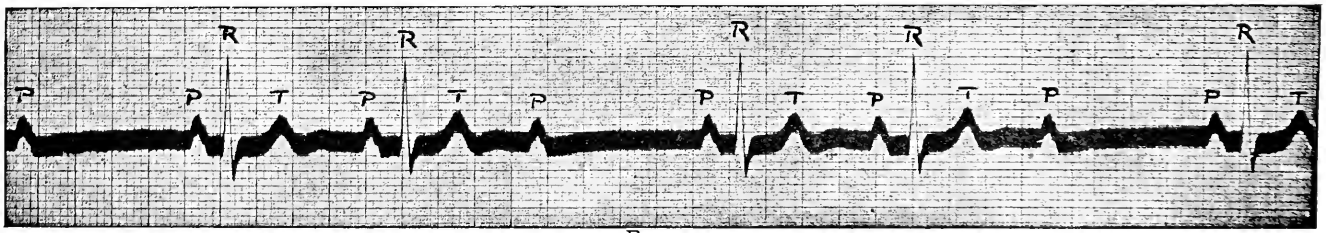

FIGURE 24

Partial block. The ventricle fails to respond to every third auricular impulse.

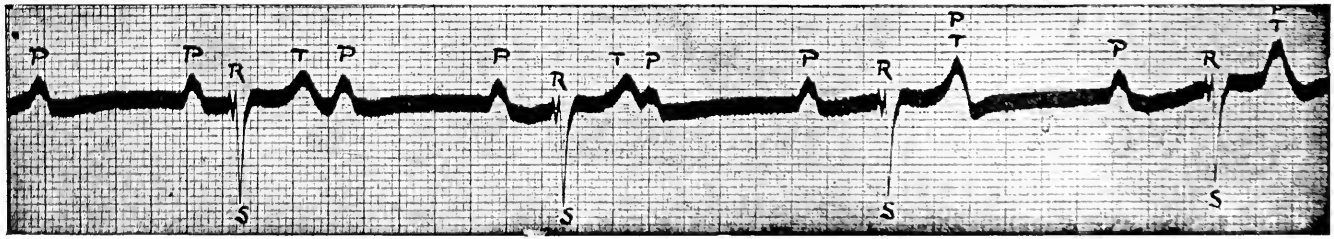

FIGURE 25

Complete block. Auricular rate 70. Ventricular rate 32. The auricular and ventricular rhythms are quite independent.

from that resulting from a hypertonic condition of the vagus may often be made by noting the effect of the administration of atropin. In cases of vagus block, the paralyzing of the terminal nerve endings with atropin abolishes the block.

CLINICAL FEATURES AND SIGNIFICANCE

The milder grades of interferences with the property of conduction are often associated only with those signs which we have described in pointing out the means for their clinical recognition. The patient may be entirely unconscious of any abnormality and his attention may first be called to the condition by the physician who discovers the arrhythmia. While a moderate degree of delayed conduction may be accompanied by no other symptoms, its recognition and true evaluation is important, for it means, as a rule, intrinsic myocardial defect. This may be either an organic or a chemical change in the muscle cells. It may be permanent or temporary, but it nearly always means some sort of damage to the slender bundle of muscle connecting auricles and ventricles. In taking this stand, I am quite aware that a considerable degree of impaired conduction may be produced both experimentally and clinically by stimulation of the left vagus nerve, and yet I believe that clinically these vagus effects are usually only in evidence when the tissues under their control are functionally damaged.

The recognition of this defect is also important, because it usually indicates extensive myocardial change. It is the most evident feature because the integrity of the *A-V* bundle is essential to the sequential co-ordination of auricles and ventricles, and yet it is rare to find a lesion of the bundle without other widespread damage in the walls of both the upper and lower chambers. Indeed, it is the rule, rather than the exception, that the injury to the *A-V* bundle is merely a part of the pathological process involving a large portion of the heart muscle.

The identification of this defect is of considerable practical value, since these hearts are particularly susceptible not only to vagus influences, but also to drugs of the digitalis group. Some degree of block may often be initiated in a normal heart by the administration of toxic doses of digitalis. In those showing an abnormal conduction, digitalis is frequently a potent influence in accentuat-

ing the defect. It does not follow from this that digitalis is always contraindicated in conduction abnormalities. Some of these patients seem to improve when the block is increased, but in such cases it should be given with caution and with a knowledge of the effect which may be expected.

A complete functional destruction of the bundle of His is not necessarily followed by cardiac insufficiency. I have had under my observation for the past three years a young woman referred to me by Professor Janeway, in whom a complete heart block was discovered during a routine examination. She has at no time given any evidence of cardiac insufficiency and during this period has completed a course of training as nurse in the Presbyterian Hospital, involving no small amount of physical exertion.

Cardiac insufficiency in cases of dissociation is more often due to the inability of a damaged ventricle to maintain its part, rather than the result of the cutting off of the normal auricular impulses.

During the course of acute rheumatic fever, diphtheria, pneumonia and other infectious diseases, one should be on the outlook for conduction disturbances. These are met with not only during the height of the active process, but also during convalescence. I have seen its development on several occasions, some days after the defervescence of an acute lobar pneumonia, in diphtheria long after the subsidence of the acute symptoms, and in rheumatism when the patient was beginning to move about. Under these conditions the block is frequently only a temporary affair and eventually disappears with the removal of the toxins and the restitution of the cells of the myocardium. The indiscriminate use of digitalis in the acute infections is to be deprecated. It may be of great service when properly used, but may also do distinct injury unless carefully adapted to the needs of the individual patient.

In complete block the rate of the ventricle is quite independent of reflex influences; excitement may increase the auricular rate in its accustomed manner, but the ventricular rate is undisturbed. The same has been noted in regard to the effects of the administration of alcohol and chloroform (Mackenzie).

We have discussed the cases of heart block in which the arrhythmia is the only symptom and in which there is no altera-

tion in the general blood distribution, also a group of cases in which the arrhythmia may or may not be the only evidence of a myocardial lesion, but in which the heart is unable to maintain an adequate circulation, with a result that the ordinary symptoms of cardiac insufficiency ensue. In the latter we conclude that there is always a defective ventricular muscle in addition to the abnormal condition of the *A-V* bundle.

There remains for our consideration a group of cases which is characterized by symptoms which are directly dependent upon the arrhythmia for their development. This is the group that has long been recognized as the Adams-Stokes syndrome. Its distinctive features are attacks of unconsciousness, with or without convulsive movements, associated with a sudden slowing of the usual pulse rate. The seizures are probably the result of a sudden cerebral anæmia attending the abrupt slowing of the left ventricle. The attacks may be very infrequent and occur at intervals of months or may follow one another with great rapidity, so that "twenty to thirty" may be counted in twenty-four hours. They differ greatly in their duration and in their severity. Sometimes the loss of consciousness is merely momentary or it may be prolonged for several minutes. The breathing may be at first stertorous and be followed later by apnœa, or the respiration may be normal throughout the attack. If the attack is momentary there is usually a pallor of the face; in a prolonged attack there is venous congestion with extreme cyanosis. The convulsion may consist of slight twitching of the face or of one arm or in the more severe paroxysms it may become general. The attack is usually ushered in with a sudden decrease in the ventricular rate and with pauses between the beats of varying duration. The change may consist in a fall of rate from the normal to the neighborhood of 30, or, in cases with a block of considerable duration and an established rate of 30, the rate may suddenly fall to 7 or 8 per minute. During the attack the veins of the neck may be prominent and are seen pulsating rhythmically at the rate of 60 or over per minute. After the attack the heart will usually be found to be beating rhythmically at about 30 per minute. This sudden change in an established ideo-ventricular rhythm, with its associated loss of consciousness, is additionally suggestive of serious damage to the ven-

tricular muscle, for a normal ventricle should maintain its ideo-ventricular rhythm unimpaired.

The exciting cause of this syndrome is often found in a little unusually physical exertion. In extreme cases, walking a short distance has been found sufficient to induce an attack, while in others a moderate amount of exertion is endured with impunity.

Since the ordinary path by which the ventricles are influenced is severed, we do not know the mechanism through which exercise may affect them. It may be by a direct effect on the myocardium of an altered blood supply, or possibly by reflex influences through those few fibers of the extracardial nerves which are known to terminate in the ventricular muscle.

It should always be remembered that the terms *heart block* and *Adams-Stokes syndrome* are not synonymous. The former designates a dissociated activity of the auricles and ventricles, and may continue indefinitely without the attacks of cerebral anæmia and unconsciousness, which are characteristic of the Adams-Stokes complex.

COURSE AND PROGNOSIS

Every case of heart block, even those of the mild degree showing only a prolonged auriculo-ventricular interval or occasional dropped beats, demand close observation over a long period of time. This is not because the defect in the *A-V* bundle is in itself a serious matter, but because it is usually indicative of more obscure and more extensive lesions of the other parts of the myocardium, and because it often affords the earliest evidence of a functional impairment which is progressive. It is rarely the only evidence of heart damage; usually one finds, in addition, a valvular defect, signs of a pericarditis, dilatation, hypertrophy, or other direct evidences of more extensive myocardial damage.

A mild degree of the depression of conduction in itself rarely induces cardiac insufficiency, but aside from the concomitant lesions which may be present and which have been referred to above, such hearts have a "margin of safety" under the normal. In my experience a very large proportion of hearts which show this abnormality during the course, or subsequent to the acute manifestations of influenza, pneumonia or typhoid fever, entirely recover

their normal function. When the defect follows diphtheria, rheumatism or a syphilitic infection, it is more apt to be permanent.

The prognosis in these mild types depends primarily on the functional condition of the heart, aside from the arrhythmia due to the injury to the bundle. If the lesion of the bundle is stationary, and if it is the only evidence of cardiac damage, it may be regarded with little apprehension. Patients do not die of mild degrees of heart block, nor is the reserve force of the heart greatly reduced thereby.

Unfortunately, in a considerable number of instances, a persistent heart block shows itself to be part of a progressive lesion. Symptoms of ventricular damage gradually develop or the evidences of bundle defect gradually or abruptly indicate a transition to a more severe type of abnormal function.

It is at the time of the sudden change from a mild degree of block to the more severe grades that the attacks of unconsciousness and convulsions, which are characteristic of the Adams-Stokes syndrome, are wont first to appear. The cerebral anæmia seems to be induced by the sudden transition from the faster to the slow rate. When the block becomes complete and the lower chamber takes on its slow rhythmic ideo-ventricular rhythm, there is less liability to these seizures. Later there may be other abrupt falls in rate (manifestly due to defects of the ventricular myocardium) and once more the attacks of unconsciousness appear with renewed severity and frequency.

A severe grade of heart block is a serious condition. It is usually associated with widespread myocardial damage and consequent cardiac insufficiency. The latter is most commonly the cause of the gradual incapacitating of the patient and ultimately of his death. That these associated conditions are usually the factors of moment is evidenced by the fact that one sees cases of complete block who for years follow their accustomed activities unconscious of any abnormal circulatory condition and are incommoded only when other evidences of myocardial change appear.

The development of fits, which, however, occurs in only a small percentage of those afflicted with heart block, is a grave sign. The first attack may be fatal and the only prodrome recognized may have been a slow or irregular heart action. More commonly the

patient has a number of attacks often with very little apparent harm. The attacks once established are prone to recur at shorter intervals and with increasing severity. One cannot predict when a seizure is likely to prove fatal. Even the majority of those who develop the Adams-Stokes syndrome, however, do not die in one of the attacks. They are far more apt to succumb to a gradually increasing cardiac insufficiency terminating in heart failure without cerebral symptoms.

Prognosis, on the whole, should rest on a study of the extent and progress of the myocardial defect and an estimate of the ventricular efficiency. Few patients survive the inception of attacks of cerebral anæmia more than two or three years. The end may come at any time. Exceptionally the duration of life is longer. Several of Edes' cases lived seven or eight years after the onset of the Adams-Stokes syndrome. A case reported by Osler lived thirty years after the discovery of brachycardia and seven years after the first syncopal attack. Gerhardt has reported three cases in which syncopal attacks and heart block completely disappeared.

CHAPTER VII

The Extrasystole

In the routine examination of the pulse our attention is frequently attracted by a form of irregularity which has the following characters: the rhythm is for longer or shorter periods that of a normal pulse, but at intervals this rhythm is interrupted by a pause during which one may get the impression that one pulse beat has failed in its normal sequence; it appears as if one pulse beat had been omitted and the impression is often described as "a dropped beat" or as "an intermittent pulse." When we come to verify our impressions by more careful observation we may find that, during this pause in which we at first thought a beat had been missed, we are able to detect on delicate palpation, a small pulse wave which had at first escaped our attention; this wave is usually much smaller than the waves of the normal rhythm; it occurs at a time which is a little too early for the occurrence of a beat of the normal rhythm and is followed by a pause which is somewhat greater than the interval between the beats of the normal rhythm; this pause is usually followed by a pulse wave which is a little larger and more forcible than the waves of the normal. This irregularity is known as an *extrasystole*. It is evidently the result of a ventricular contraction which has occurred too early and which is less forcible than the normal rhythmic contractions of the heart; it is therefore also known as *premature contraction*. On auscultating such a heart we will detect a rhythmic series of normal sounds interrupted at intervals by a group of sounds which are weaker and occur earlier than those of the normal cycles; this first and second sounds of the weak group are followed by a silence which is considerably longer than the normal diastolic period.

In some of the hearts of this group the extrasystolic contraction will be represented by a single sound only, and no corresponding wave even of an abortive character can be detected in the peripheral arteries. These signs indicate that the premature beat was wanting in force sufficient to open the aortic valve. The question of the

opening of the aortic valve depends on three factors: (*a*) the energy of the premature ventricular contraction; (*b*) the volume of the blood in the ventricle at the moment; and (*c*) the blood pressure in the aorta. These factors depend in turn upon the time of the occurrence of the extrasystole. If this comes early in diastole the contractile power of the ventricle will have recovered to only a moderate degree; the volume of blood in the ventricle will then be small and the aortic pressure will be near its highest point; hence it is hardly probable that the aortic valves will be opened and such a premature contraction will be accompanied by the first heart sound only; the second sound, due to the closure of the aortic valve, will be absent and there will be no corresponding pulse wave. If, however, the extrasystole comes later in the diastolic period, contractility will have more completely recovered; the volume of blood which has passed into the ventricle will be greater and the aortic pressure to be overcome much less; hence the aortic valve will be opened; the second heart sound will be heard and the small extrasystolic wave may be felt at the wrist.

PATHOLOGY AND ETIOLOGY

In the sections on the physiology of the heart it was pointed out that all portions of the musculature of the heart have the property of excitability, that is that any muscle cell can respond to stimuli at any time except during the "refractory period" which lasts for a short time after the cell has been stimulated. Also that normally stimuli are rhythmically originated at the "sinus node" and sweep over the tissues of the heart in an orderly manner, exciting to activity its chambers in a definite sequence.

If electrical stimuli of the proper strength be applied by means of suitable electrodes to the wall of the heart of the experimental animal (frog, turtle rabbit, dog, etc.), it will respond by a contraction, no matter what portion of the musculature is excited; the activity thus produced will spread downward in the direction taken by physiological stimuli and also from the point of stimulation upward toward the sinus node, i.e., in a direction the reserve of that of physiological stimuli, and the chambers of the heart will contract in the order in which the stimuli reach them. Contractions thus excited from an abnormal focus are known as extrasystoles, and,

according to their point of origin, are known as auricular, ventricular, etc.

If, in this manner, the heart is systematically studied by applying stimuli in the various phases of the cardiac cycle while the heart is beating rhythmically, it will be found that for a period beginning just before and extending a short time after systole, the heart is not excitable even by very powerful stimuli, i.e., the heart is in the "refractory phase" because the molecules upon which the fundamental properties of cardiac muscle depend have been decomposed into their constituent ions. Now the extrasystole which has been experimentally produced throws the heart muscle into the "refractory phase" so that the next physiological stimulus of the rhythmic series arising at the sinus node will reach the muscle cells lower down when they are inexcitable, hence it will be ineffective in producing a systole. The next systole will not occur until it is brought into being by the next spontaneous stimulus which is formed at the sinus node and which occurs exactly at the moment at which it would have occurred had there been no extrasystole. This lengthened diastolic period which follows the extrasystole is known as the *"compensatory pause."* When the time consumed between the last normal heart beat preceding the extrasystole and the normal beat following the compensatory pause is exactly equal to the time occupied by two beats of the normal rhythm, the long diastolic pause following the extrasystole is known as a *"complete compensatory pause;"* when the interval between the last spontaneous systole and the post-compensatory systole is less than the interval between two systoles of the normal rhythm, the compensatory pause is called *"incomplete."*

A study of the compensatory pause in the mammalian heart reveals the following facts: (*a*) When the sinus node is stimulated the extrasystole is not followed by a compensatory pause. (*b*) When the auricle is stimulated the compensatory pause is usually incomplete. (*c*) When the ventricle is stimulated the compensatory pause is complete. These facts may be explained on the following grounds: As soon as the stimulus material at the node is destroyed by its direct stimulation, the construction of the material is immediately recommenced and reaches the explosive point at an interval just equal to the period of the normal rhythm. When the auricle is stimulated early in the diastolic period (see Figure 26) the stim-

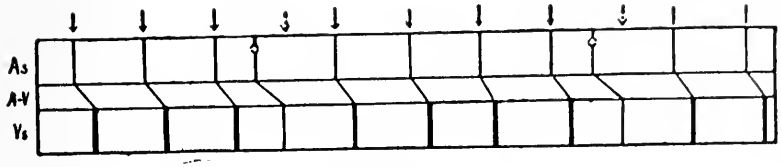

FIGURE 26

Extrasystole arising from the auricle near the sinus.

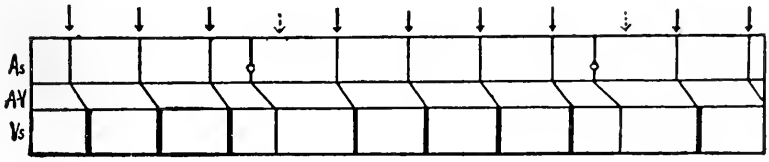

FIGURE 27

Extrasystole arising from a point low down in the auricle.

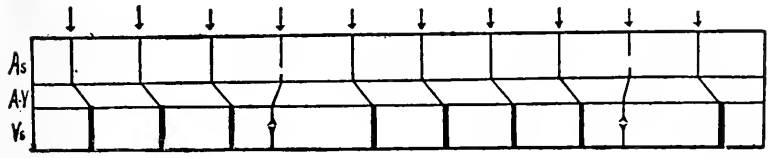

FIGURE 28

Extrasystole arising from a point in the ventricle.

Diagrams to illustrate the mechanism of the extrasystole starting from various parts of the heart muscle. The arrows indicate the points of origin and the directions taken by the stimuli. Dotted arrows indicate the time at which the normal stimulus at the sinus node should reach maturity if its formation was not interrupted by the extrasystole. The thickness of the lines representing ventricular systole indicate the relative effect of the normal beat and the extrasystole in maintaining an adequate circulation. *As* = auricular systole. *A-V* = auriculo-ventricular bundle. *Vs* = ventricular systole.

ulus is conveyed not only to the ventricle but also upward to the node and will destroy the spontaneously forming stimulus material at the node before it has reached the explosive point, hence the interval between the last physiological stimulus and the post-extrasystolic stimulus will be somewhat less than two cycles of the normal rhythm. When the auricular stimulation occurs somewhat later in diastole the retrograde stimulus may reach the node coincident with the explosion of the rhythmically formed stimulus material, hence in this instance the post-extrasystolic pause will be fully compensatory. When the ventricle is stimulated (see Figure 28) the retrograde stimulus reaches the sinus node during its refractory period just after its physiological stimulus and the post-extrasystolic stimulus will exactly equal the period between two beats of the normal rhythm and the post-extrasystolic pause will be fully compensatory. This explanation indicates how extrasystoles arising from different parts of the auricles may have compensatory pauses either complete or incomplete. It may be stated, as a general rule, that the nearer to the sinus node is the point of stimulation initiating an extrasystole, and the earlier it occurs in diastole, the shorter will be the post-extrasystolic pause; and, conversely, the farther from the sinus node is the point of origin of the extrasystole and the later it occurs the more nearly will the post-extrasystolic pause be compensatory.

Electrocardiographic studies have further shown that the stimuli originating extrasystoles may pass over the musculature of the heart by the normal paths (nomodrome extrasystole), or, since the stimuli may originate from some point far removed from the normal path or may be shunted from this path by abnormal conditions of the muscles which form an obstruction to their passage, they may take an unusual course through the cardiac tissue (allodrome extrasystoles). A discussion of these abnormal paths and their variegated but characteristic electrocardiographic records will be left for a later paragraph.

Extrasystoles have been produced experimentally in many ways other than the employment of electrical stimuli. Mechanical irritation, heat, the application of irritating salts, obstruction of the great veins (Stassen), clamping of the aorta (Hering), ligation of a branch of the coronary artery (Lewis), the injection of digitalis

and atropin (Cushny), adrenalin (Kahn), muscarine and physostigmine (Rothberger and Winterberg). Under proper conditions extrasystoles have been produced in the isolated perfused heart and in the mammalian heart in situ after all nervous connections have been severed, hence it is probable that their cause is an increased excitability of the muscle cells usually quite independent of nervous influences, though Kraus and Nicolai have produced them by vagus irritation.

The conditions of the experimental production of extrasystoles have been set forth at some length since it is upon inferences from these data that our conception of the pathological conditions underlying the extrasystole, as met with in man, is based. Very little indeed is known of the histological changes associated with the production of extrasystoles and there still remains here a field for careful and exhaustive research. Clinically extrasystoles are found far more frequently in those with slow hearts and often they may be made to disappear by moderate exercise which quickens the heart rate. The experimental evidence seems to indicate clearly that the extrasystole occurs because some cardiac muscle cells become more excitable than those of the sinus node and it is therefore on this ground, easy to understand why an increase in excitability should be more apparent during a slow rate, since in the faster rates the excitability of the node is greater than in the slow rates; under such conditions the abnormal irritability of some portion of the auricle or ventricle must be considerable to make itself evident.

It also seems fair to assume from the experimental evidence that nutritional disturbance may play an important part in increasing the excitability of heart muscle; an atheroma with a narrowing of the coronary artery or one of its branches may be the pathological counterpart of the ligation of the branches of the coronary which has been shown by Lewis to regularly produce extrasystoles.

Numerous toxic agents are known to be associated with the production of extrasystoles; they are quite common in many febrile conditions, notably in acute rheumatic fevers. One of the very common phenomena produced by the administration of large doses of digitalis (at least to patients having damaged hearts) is the appearance of ventricular extrasystoles; on the withdrawal of this drug they disappear. Nicotine is another of the cardiac poisons

which is clinically prominent as a cause of extrasystoles. The "tobacco heart" is one in which premature beats have become so frequent as to make themselves uncomfortably evident. Excessive tea drinkers are subject to this form of irregularity. Premature beats are found in persons of all ages; they are rare in the first decade of life and are most common after the age of 50. They are considerably more common among men than among women.

Extrasystoles are probably very much more common than is generally supposed; it has been estimated that a majority of persons reaching middle age have had extrasystoles at some period. They are frequently met with in those who afford other signs of impairment of the heart, such as valvular disease, myocardial degeneration and the cardiac complications of nephritis, but premature contractions are also not uncommonly found in those whose hearts have no discoverable abnormality other than this irregularity.

Premature contractions are exceedingly common in individuals of the neurotic type; they may sometimes be induced by irritation of the skin and in persons subject to this irregularity, merely plunging the hands into cold water is sufficient to develop it. They are often associated with digestive disturbances, particularly when accompanied by flatulency. As has been mentioned exercise will frequently cause the temporary disappearance of extrasystoles, but if carried to the point of fatigue the irregularity is prone to become more evident than before. In those predisposed to them, suspension of respiration for a few seconds will sometimes induce these premature contractions. When present in the upright position they will often disappear as soon as the subject lies down, even though this change in position is accompanied by a slight diminution in the rate of the heart. Extrasystoles are quite common during convalescence from infectious diseases.

IDENTIFICATION

Clinically, the starting point for establishing the presence of the extrasystole is to determine whether the patient has a fundamentally normal cardiac rhythm, which is broken on occasions more or less frequently. When the interruptions occur at infrequent intervals, as is the case in the majority of these patients, the detection of the fundamental rhythm is comparatively easy. If one palpates the

radial artery there are long periods during which the pulse is perfectly regular, then occasionally this regular rhythm is broken by a pause which is too long to fit the fundamental rhythm, or one may detect a very small pulse wave followed by a pause longer than that ordinarily separating the waves of the normal rhythm. When one listens to the heart sounds they will be heard for long periods as a normal rhythmic series until this series is broken by the occurrence of one or two indistinct heart sounds which follow the last normal sounds too early and which are in turn followed by a pause longer than that occupied by the interval between the heart sounds of the periods of normal rhythm. The small premature waves detected in the radial and the indistinct premature first (or first and second) sounds heard over the precordium, each followed by a more or less complete compensatory pause, are our usual common evidences of the presence of extrasystoles. Whether one hears at the time of the premature beat a first and second heart sound or only a first heart sound depends, as has been pointed out in a preceding paragraph, on whether the extrasystolic contraction has, or has not opened the aortic and pulmonary valves.

If murmurs are present during the periods of normal rhythm, they are much less distinct in the premature cycle and may be absent. The mitral systolic is the murmur which can most easily be detected in the extrasystolic cycle; the presystolic is more rarely heard; while aortic murmurs are absent or shortened in consonance with the action of the valve which may fail to open, or open only for a brief period. I have recently seen a case presenting extrasystoles in which no heart sounds could be heard, both first and second sounds being replaced by loud harsh murmurs. At the time of the extrasystole one could hear four murmurs following each other at equally spaced intervals. The first and second of these murmurs were louder and a little longer than the third and fourth; the fourth murmur was followed by a considerable pause which was succeeded by a repetition of the two murmurs which constituted the auscultatory evidence of the ordinary rhythmic activity of the heart.

Another type of rhythm which is easily recognized as due to extrasystoles is the so-called "bigeminus." Here the radial pulse shows a rhythmic series composed of a large wave, a short pause, a small wave and a long pause. This sequence is repeated again

and again. The repeated recurrence of two pulse waves followed by a pause has given rise to the very expressive term "coupled rhythm." It consists of a wave of the fundamental rhythm followed by a premature beat and its compensatory pause. This rhythm is one of the common manifestations of toxic doses of digitalis. When an extrasystole occurs every third beat it gives rise to a rhythm that was formerly described as the "pulsus trigeminus."

When extrasystoles occur quite frequently and at very irregular intervals it is sometimes more difficult to assure oneself, by the ordinary physical signs, that the irregularity is due to premature contractions, but careful observation will usually discover a fundamental rhythm, interrupted by beats which occur too early, are followed by a pause and each time they appear give the impression of "coupling."

Inspection of the jugular pulse is frequently an aid in making the diagnosis of an extrasystole. The two venous waves which one ordinarily sees during the fundamental rhythm are often replaced at the time of the premature contraction by a single venous wave larger than the others. This wave is due to the inability of the vein to discharge its contents into the auricle at this moment, since the pressure in the auricle is abnormally high, the ventricle being in systole and the auriculoventricular valves being closed. This is, of course, more in evidence when the origin of the extrasystole is in the ventricular wall and the auricle and ventricle contract simultaneously.

Whether an extrasystole is auricular or ventricular in origin can only be definitely decided by graphic records and yet the trained observer who has sharpened his powers of differentiation by correlating his physical signs with the evidence of the graphic records, can often, by noting the length of the compensatory pause and the character of the heart sounds of the premature beat, quite correctly assign a particular extrasystole to its proper category.

A graphic record of the radial or of the apex beat is often sufficient evidence to establish the presence of the extrasystole. Such a record (Figures 30, 31 and 32) shows a series of similar waves recurring at equal intervals. This rhythm is more or less frequently interrupted by a small wave which occurs too early to fit into the

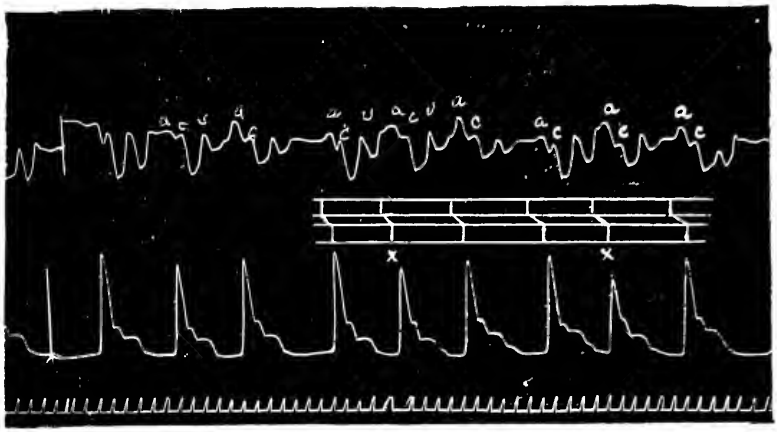

FIGURE 29
Auricular extrasystole at x. The compensatory pause is incomplete.

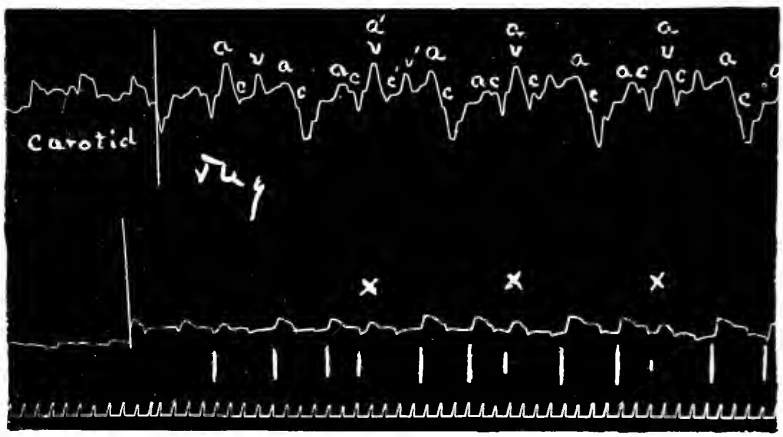

FIGURE 30
Auricular extrasystole at x. *a'* of extrasystole superimposed on preceding *v* wave. The compensatory pause is incomplete.

fundamental rhythm. It is followed by a pause longer than that between two beats of the fundamental rhythm, which in turn is followed by a wave which is usually a little larger than the average wave of the rhythmic series and which is the first of a new series of rhythmic waves. In the case of an extrasystole which originates in the ventricle the post-extrasystolic pause is fully compensatory (see Figures 32 and 33). When the extrasystole has its origin higher up in the cardiac tissues, the pause is "incomplete" (Figures 29, 30 and 31). The reason for this has been explained in a preceding paragraph (page 60).

THE POLYGRAM

Auricular Extrasystoles. The jugular tracing throws additional light on the mechanism (Figures 29 and 30). Figure 29 shows a rhythmic series of waves *a c v*, which is several times (at *x*) interrupted by a similar group which occur too early; it is clear that the auricle contracts too soon and is followed by a sequential contraction of the ventricle.

Another case of auricular extrasystole is shown in Figure 30; here the premature contraction of the auricle occurs earlier in the cycle than was the case in Figure 29, so that the auricular premature wave *a'* is superimposed on the *v* wave of the preceding group; the simultaneous contraction of the ventricle and the auricle causes an unusual temporary stasis in the jugular vein, hence this large wave (*v' a'*), The extrasystole is followed by a compensatory pause which is "incomplete."

The *Nodal Extrasystole* is illustrated (.r Figure 31). In this instance our conception is that the premature contraction starts at a point in the tissues junctional between auricles and ventricles; from this point the stimulus sweeps upward to the auricle and downward to the ventricle so that these chambers contract practically simultaneously, hence the waves *a'* and *c'* of the jugular coincide. The retrograde stimulation of the auricle has destroyed the usual stimulus material accumulating at the normal pacemaker; the building up of stimulus material is, however, at once recommenced and this reaches maturity in the normal time which is shown by the fact that the time elapsing between the wave *a'* of the extrasystole and the succeeding *a* wave is exactly the interval of the normal rhythmic series.

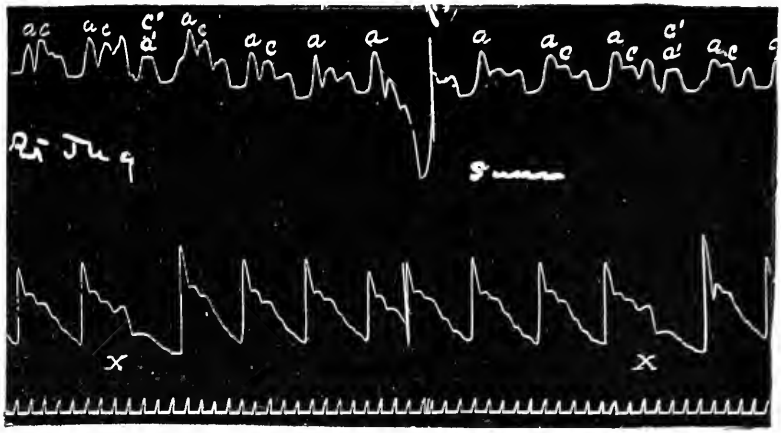

FIGURE 31

Nodal extrasystole at x. In the jugular tracing the *a* and *c* waves of the extrasystole occur simultaneously. The compensatory pause is incomplete.

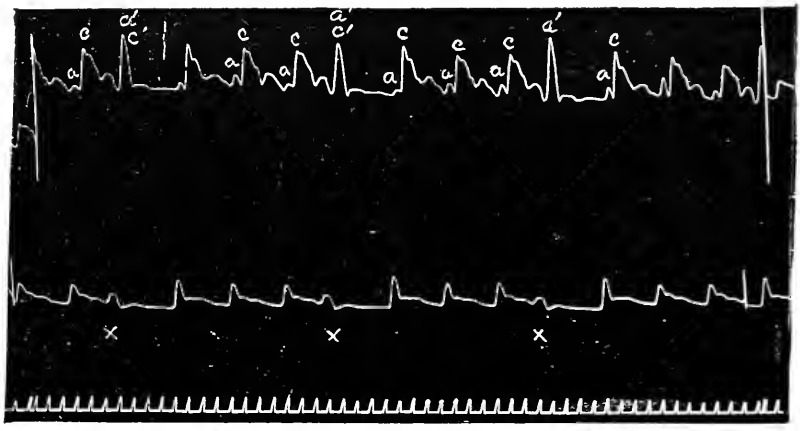

FIGURE 32

Ventricular extrasystole at x. In the extrasystolic cycle the auricle and ventricle contract simultaneously (*a' c'*). The compensatory pause is complete.

Ventricular Extrasystoles are shown in Figure 32. The auricle, as represented by the *a* waves of the jugular record, contracts rhythmically, but occasionally (*x*) the ventricle contracts prematurely so that at these times the auricle and ventricle contract simultaneously and their activities are represented by a large wave (*a' c'*) in the jugular tracing. The absence of the *v'* wave in the extrasystolic cycle which is quite evident in the records is due to the empty condition of the ventricle at the time of the premature contraction. It is to be noted that the post-extrasystolic pause is fully compensatory. Figure 33, with its diagrammatic analysis, shows a ventricular extrasystole which occurs every third beat giving rise to the so-called "pulsus trigeminus."

Mixed types of extrasystoles are not infrequently seen in a single case. A tracing of such a patient is shown in Figure 34. Here one may make out the following sequence: normal beat, auricular extrasystole, ventricular extrasystole. The analysis of the polygraph in these cases is sometimes quite difficult. The analysis of the tracing shown in Figure 34 was subsequently verified by electrocardiographic records in which the analysis is much less difficult.

THE ELECTROCARDIOGRAMS

As a rule the identification of the kind and point of origin of the extrasystole is most accurately made by means of the electrocardiographic record. The most distinctive features of extrasystoles are that (1) they occur too early, and (2) they are followed by a pause greater than the normal intersystolic pause.

To fix clearly the phenomena which the electrocardiogram discloses, upon which we base conclusions as to the point of origin of the extrasystole, let us recall just what the movements of the string of the galvanometer represent. At any given moment the deflection of the string indicates the algebraic sum of the differences of electrical potential of the heart as a whole. When the stimulus arises at the sinus node (the normal pacemaker) and passes over the heart in a sequential, orderly manner, a series of deflections occur which we have learned to recognize (see Chapter IV) as the normal differences of electrical potential for successive instants of the cardiac cycle. If now the stimulus arises from some point of the cardiac musculature other than the "*sinus node*" it is quite evident that the

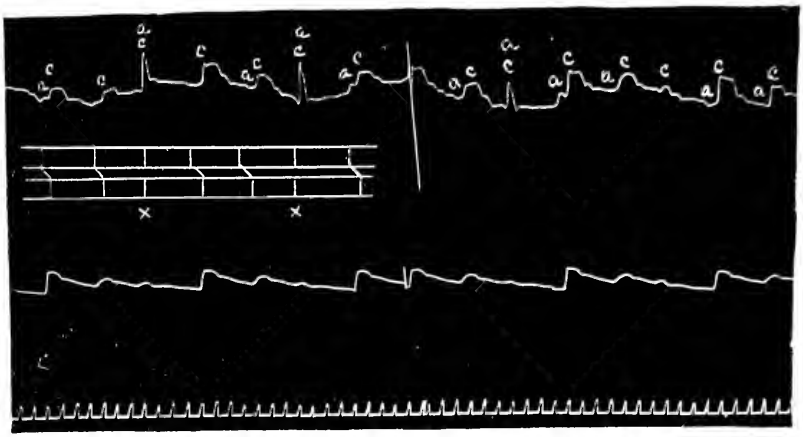

FIGURE 33
"Pulsus trigeminus" due to an extrasystole, which occurs every third beat.

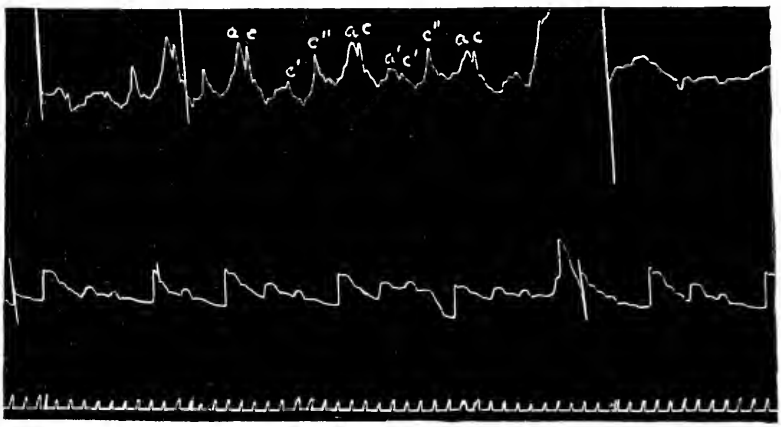

FIGURE 34
Ventricular and auricular extrasystoles in a single record. *a c* = normal cycle, *a' c'* = auricular extrasystole. *c''* = ventricular extrasystole.

impulse passing by abnormal paths and reaching portions of the cardiac tissues at intervals quite at variance with the normal will produce differences of electrical potential at successive moments of the cardiac cycle quite different from the normal. How great are the variations in electrical potential which result from the extrasystolic contractions may best be appreciated by a study of the curves which are here reproduced.

Auricular Extrasystoles. When the focus from which the extrasystole arises is at or near the sinus node the electrocardiographic complexes are usually of the normal form. Such a record is shown in Figure 35. It is composed of a series of complexes, each of which is practically of the normal type. Each cycle is opened by a *P* wave, which at its proper interval is followed by a normal ventricular complex, *Q R S T*. In the center of the record the fundamental rhythm is broken by a cycle (*x*) which, although normal in other respects, occurs prematurely and is followed by a pause which is not quite long enough to be completely compensatory. This premature contraction must have arisen at or near the sinus node, since the various parts of the cardiac musculature have been stimulated by paths and in a sequence which is the normal one.

The curve reproduced in Figure 36 shows an extrasystole which has arisen high up in the auricle near the sinus. Here the extrasystole has occurred so early that its *P* wave is superimposed on the *T* wave of the preceding cycle producing a wave which is equal to *P* + *T*. The pause following the extrasystole is incomplete.

It has been shown by Lewis* that if the auricle of an animal is made to contract by applying artificial stimuli to various portions of the auricular tissue, the resulting electrocardiographic records will be greatly modified. When the point of stimulation is at or near the sinus node the *P* wave is upward in direction and of a form which we have come to regard as normal; as the point of stimulation is made more and more remote from the sinus the *P* complexes become irregular in form and may be directed downward or show a diphasic variation. We are therefore led to infer that in the human electrocardiogram an upward single *P* wave represents an auricular contraction originating at or near the sinus node; a downward directed *P* wave indicates an origin in the lower part of the

*Heart, 1910, ii, 27.

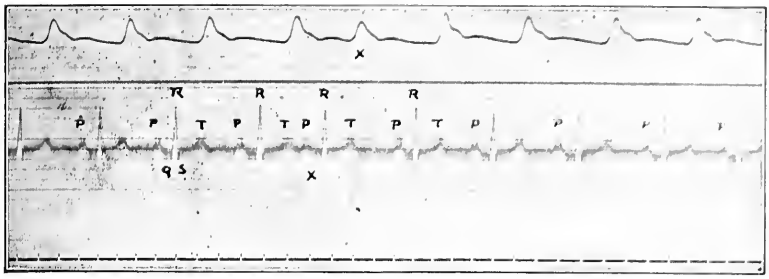

FIGURE 35

Auricular extrasystole at x. Compensatory pause incomplete. *P* = auricular contraction. *R-T* = ventricular contraction. Brachial tracing above.

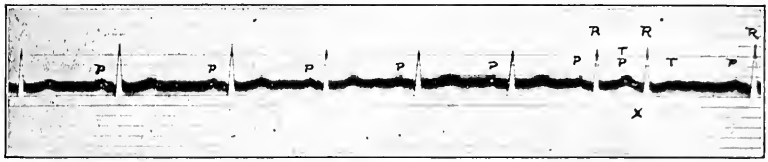

FIGURE 36

Auricular extrasystole at x. The auricular wave *P* of this extrasystole is superimposed on the *T* wave of the preceding ventricular complex.

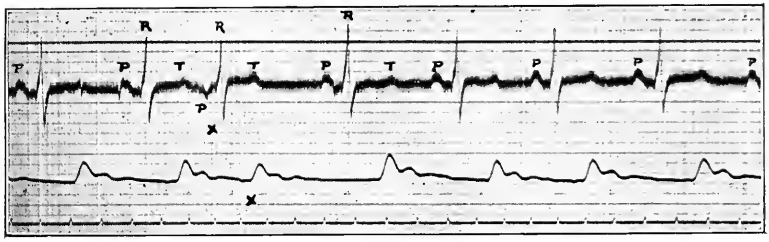

FIGURE 37

Extrasystole at x arising from a point low down in the auricle. *P* is directed downward in the extrasystole. *P* following extrasystole is diphasic. Below is brachial tracing.

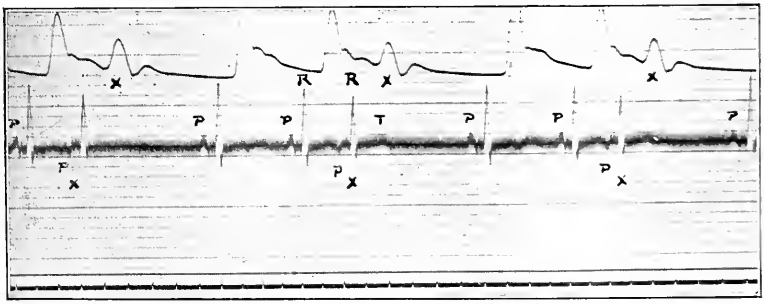

FIGURE 38

"Pulsus trigeminus" caused by an auricular extrasystole, which occurs every third beat at x. *P* is reversed in extrasystole, indicating a point of origin low down in auricle. Radial tracing above.

auricle; a notched or diphasic *P* wave indicates an intermediate point of auricular origin.

An extrasystole which arose in the lower part of the auricular tissue is shown in Figure 37. The complexes of the ordinary rhythm are normal in form except that the *P* waves are rather too broad and have summits which are slightly flattened; the extrasystolic cycle (*x*) is initiated by a *P* wave which is directed downward but is followed by a ventricular complex which is normal in form, indicating that the ventricular response to the premature auricular activity was the result of an impulse which passed down through the *A-V* bundle and over the ventricular musculature by the normal paths in a perfectly orderly manner. It may be noted in passing that the auricular complex which immediately follows the extrasystole has a form somewhat different from the *P* waves of the succeeding normal cycles; this is not an unusual occurrence and suggests that the auricle has not as yet entirely recovered its normal function.

Figure 38 displays a rhythm which was formerly known as the "pulsus trigeminus." It consists of a series of two normal beats followed by an auricular extrasystole. The impression produced on the palpating finger by a pulse of this type is indicated by the radial curve taken simultaneously with the electrocardiogram. All the auricular (*P*) complexes of this record show an unusual diphasic form, suggesting that even those impulses which originated at the sinus node have taken an abnormal path through the auricular tissue. The *P* waves of the extrasystole (*x*) are clearly reversed, indicating an origin low down in the auricle.

The ventricular extrasystole presents in the electrocardiogram (Figure 41), a complex far removed from that of the normal ventricular contraction. The abnormal point of origin and the consequent abnormal path which the impulse follows usually produces a much greater difference of electric potential than does the impulse which descends from the auricle and follows the normal path through the *A-V* bundle and its branches. The auricle contracts at regular intervals, so that often when an extrasystole occurs the ventricular and auricular contractions are simultaneous. The little wave representing auricular activity will then occur during the time of ventricular activity and is usually relatively so small that it is

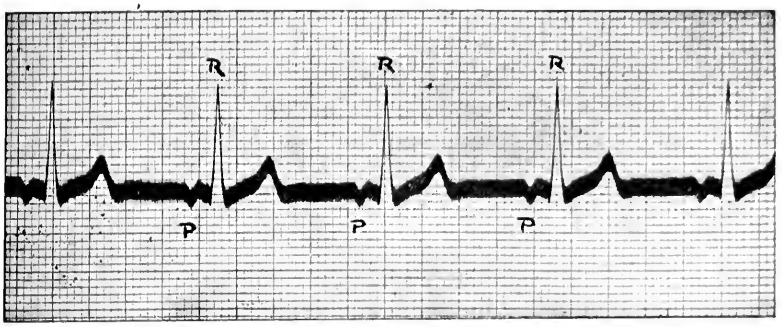

FIGURE 39

Lead II. Every *P* wave is of an abnormal form indicating an abnormal point of origin in the auricle or an abnormal path through the auricular wall.

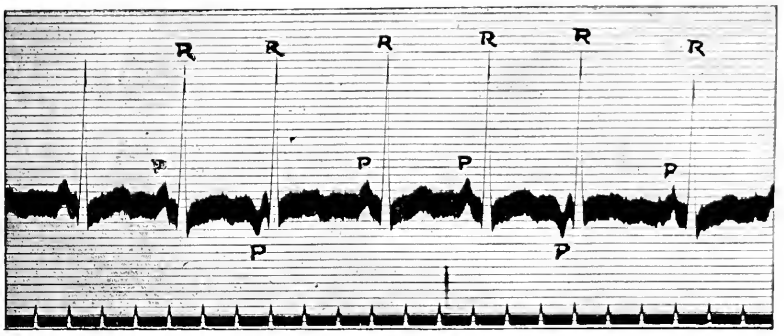

FIGURE 40

Auricular extrasystoles from a point low down in the auricle. Note the short *P-R* interval of the extrasystoles and the incomplete compensatory pauses.

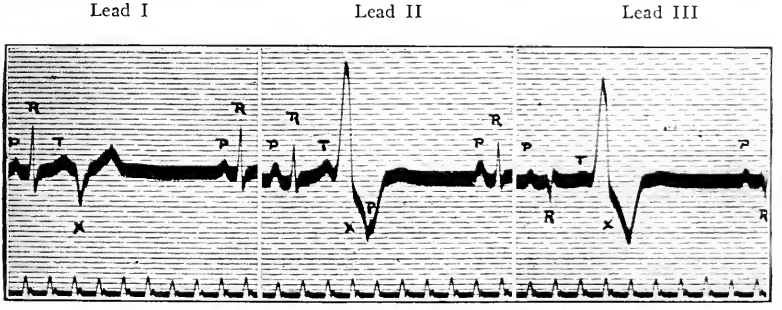

FIGURE 41

Ventricular extrasystole at x arising from a point at the base of the left ventricle. Showing similarity in the complexes obtained by leads II and III. Compare *P R T* = normal complex and extrasystolic complex x.

submerged in the large waves of the ventricular complex. Figure 41 shows an electrocardiogram taken from a patient by the customary three leads. The first and last complexes of each lead are the normal for this individual, between these are seen the extrasystoles. It is to be noted that the form of the extrasystolic waves are very similar in leads II and III, but that these differ very materially from the extrasystole pictured in lead I. The similarity of form of the extrasystolic complexes of leads II and III is usual. The complex of lead I may be similar in form to that of lead II, but it is usually quite different. The submerged auricular wave which occurs during the extrasystole can be seen (only in lead II) as a small notch (*P*) in the final dip of the extrasystolic complex.

Systematic studies of the electrical complexes obtained by stimulating various portions of the right and left ventricles both when the branches of the bundle of His are intact and when one of the branches has been cut, have shown that a comparison of the records* taken by lead I and lead II will indicate the point from which the extrasystole has its origin.

The prominent types are shown in Figures 42, 43, 44 and 45. The direction of the principal deflection in leads I and II with the points of origin of the extrasystoles may be tabulated as follows:

TYPE	DIRECTION OF PRINCIPLE DEFLECTION		POINT OF ORIGIN OF STIMULUS.
	LEAD I.	LEAD II.	
1	up	up	Right ventricle near base
2	up	down	" " " apex
3	down	up	Left ventricle near base
4	down	down	" " " apex

A type of curve which is not infrequently met with is shown in Figure 46. Two ventricular extrasystoles appear in this record. Each is preceded by a *P* wave which occurs at its regular rhythmic

*Rothberger and Winterberg: Archiv. für die ges. Physiologie, 1913, cliv, p. 571.

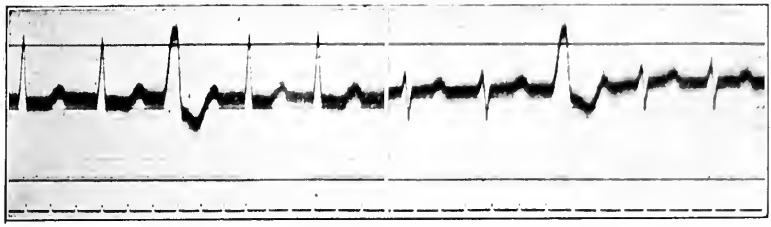

FIGURE 42

Type. 1. Ventricular extrasystole arising from a point in the right ventricle near the base.

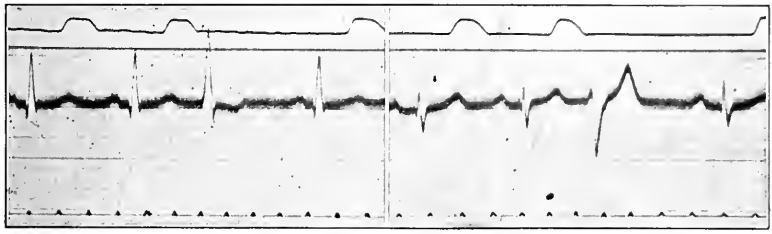

FIGURE 43

Type 2. Ventricular extrasystole arising from a point in the right ventricle near the apex. Above brachial tracing the extrasystole produces no arterial wave.

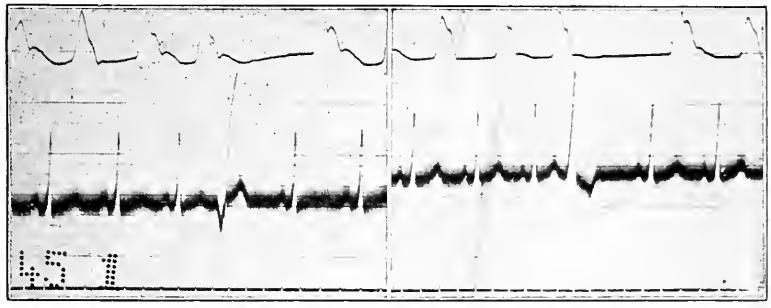

FIGURE 44

Type 3. Ventricular extrasystole arising from a point in the wall of the left ventricle near the base. Radial tracing above.

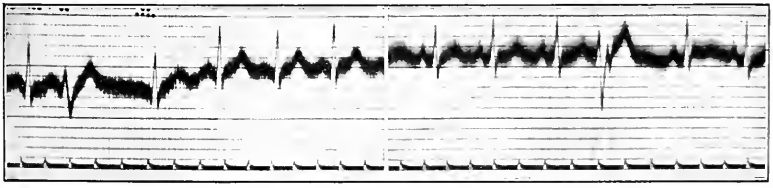

FIGURE 45

Type 4. Ventricular extrasystole arising from a point in the wall of the left ventricle near the apex.

interval. At first sight one might regard this as an impulse which had its origin in the auricle and which was shunted off by an abnormal path through the ventricular wall. One notices, however, that the length of the *P-R* interval of the normal complexes is unusually long (over 0.2 second), while the interval between *P* and the onset of the extrasystolic complex is very brief (0.1 second). It is therefore evident that insufficient time has elapsed between *P* and the onset of the extrasystole to permit of the passage of the stimulus from the auricle to the ventricle, and we must conclude that the ventricle has contracted in response to a stimulus initiated independently in its own wall.

A contrast to this case is shown in Figure 47. Here the ventricular extrasystole (at *x*) occurs relatively early and the auricular contraction *P* is seen as a step on the descending limb of the large extrasystolic wave. The arterial tracing which accompanies this as well as many of the preceding electrocardiograms shows the relatively small wave which is produced in the arterial tree by the extrasystole. This evident lack of efficiency of the premature contraction in maintaining an adequate circulation is due to two factors (1) the abnormal sequence of the stimulation of the muscle fibers of the ventricle results in a contraction which is relatively incoördinated, and the propelling power of the ventricles is less than under the normal conditions; (2) on account of the prematurity of its contraction the ventricle is less well filled with blood, hence a smaller volume is expelled into the aorta.

The nodal extrasystole. The majority of extrasystoles which one sees in the clinic have their origin in some portion of the ventricular wall. Auricular premature contractions are far less frequent. A still more rare form of extrasystole is shown in Figure 48. In this curve the extrasystolic complex is only slightly changed from the ventricular complex of the fundamental rhythm, the following pause is fully compensatory and the presence of *P* in its normal rhythmic position following the principal wave of the extrasystole shows that the rhythm of the auricle has not been disturbed. Since the ventricular portion of the extrasystolic complex has a form not unlike the ventricular complexes of the sequential rhythm and yet clearly is not the result of auricular activity, we conclude that its point of origin is at some point high up in the auriculo-ventricular bundle

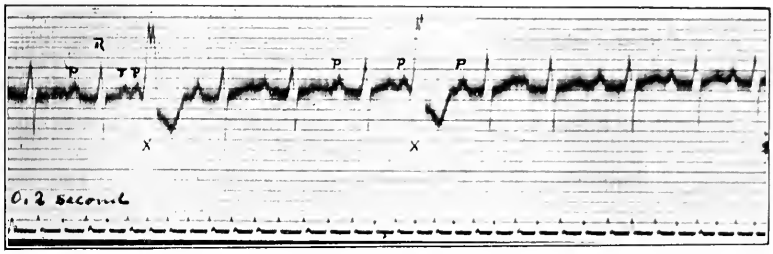

FIGURE 46

Ventricular extrasystole at x. The auricle contracts rhythmically, as shown by *P* waves. *P-R* interval = 0.3 second. The extrasystole does not originate in the auricle.

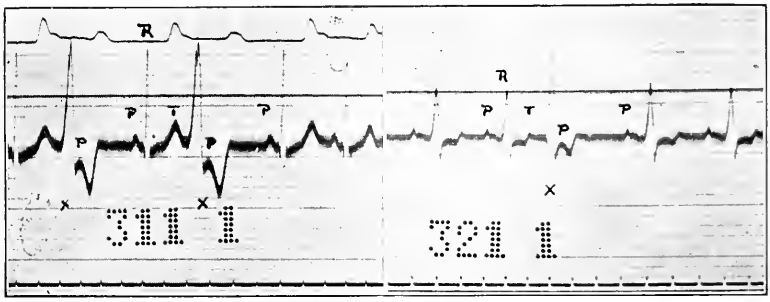

FIGURE 47

Ventricular extrasystole at x, showing submerged *P* waves. Brachial tracing above. Extrasystolic pause is fully compensatory.

FIGURE 48

Nodal extrasystole at x. At time of extrasystole auricle and ventricle contract simultaneously. Origin of ventricular impulse is high up in the *A-V* bundle.

and that its subsequent course through the ventricular wall follows the normal channels. This is known as the *nodal extrasystole*.

The interpolated extrasystole is another rare form of premature contraction. An extrasystole always ventricular in origin occurs between two beats of the normal rhythm without otherwise disturbing the orderly course of either the auricular or the ventricular rhythm (Figures 49, 50 and 51).

In Figures 52 and 53 are shown two types of "pulsus bigeminus," each due to an alternation of normal cardiac contractions and extrasystoles; the extrasystoles of Figure 52 arise in the wall of the right ventricle near the apex; the premature contractions of Figure 53 arise in a point in the left ventricular tissues near its base.

Extrasystoles of different points of origin frequently are met with in the same patients on separate occasions and sometimes in close succession. Figure 54 shows auricular extrasystoles at A and ventricular extrasystoles at X. The auricular extrasystoles have an incomplete, the ventricular a complete compensatory pause. Figure 55 shows an alternation of ventricular extrasystoles (x) and normal ventricular complexes. At the center of the record (Y) the sequence is further disturbed by the occurrence of a ventricular extrasystole from an entirely new point of origin.

THE CLINICAL SIGNIFICANCE

of the extrasystole is one of considerable importance. Most of us have followed the career of patients who have had occasional extrasystoles for a number of years and often we can secure a history of the existence of this form of irregularity for many years, antedating our own observations, yet we rarely see a case of cardiac insufficiency which can reasonably be attributed to this irregularity *per se*. The patient is often quite conscious of what they often describe as a "thumping" in the precordial region, "fluttering of the heart," or "palpitation." On examination a large number of these sensations can be shown to be due to the presence of extrasystoles. These sensations are often the occasion of considerable alarm to the patient particularly when they are first discovered and the physician who assures them that this irregularity in itself is of very little significance and rarely is the forerunner of more serious

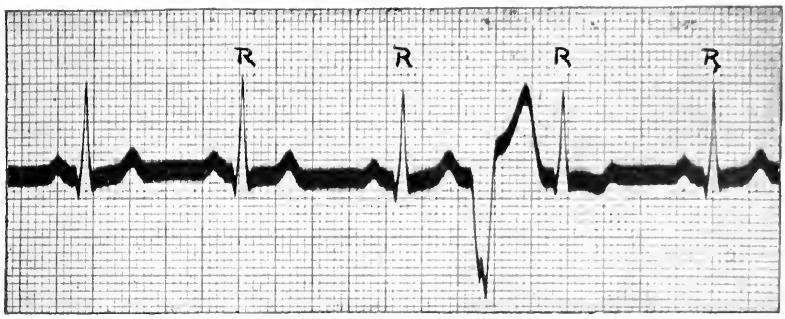

FIGURE 49
Interpolated extrasystole.

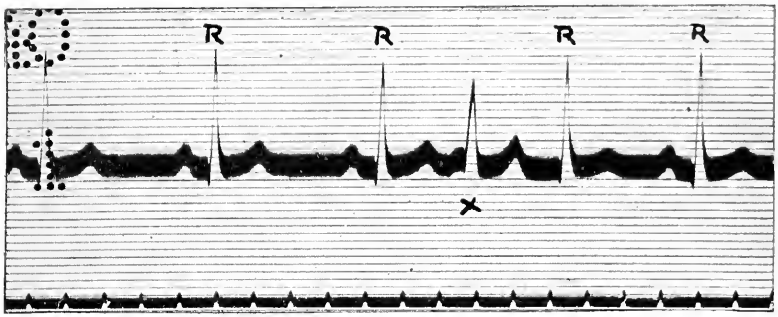

FIGURE 50
Patient G. Lead I. Interpolated extrasystole at *X*. Ventricular extrasystole type 1.

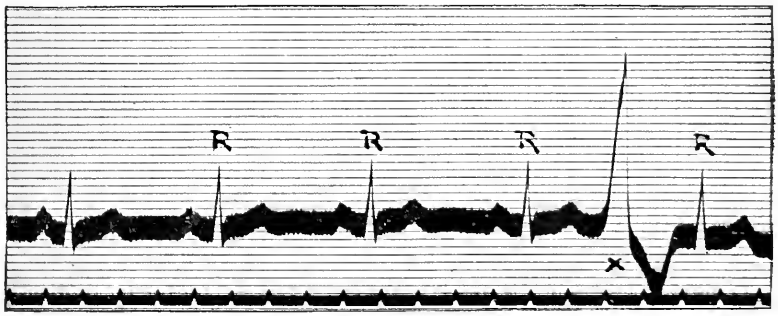

FIGURE 51
Patient G. (same as Figure 50.) Lead II. Interpolated ventricular extrasystole type 1.

trouble does the patient a great service in removing his grounds for anxiety.

When, however, we see cases which show extrasystoles at very frequent intervals and particularly when the extrasystoles arise from more than one focus our prognosis should be much more guarded, such irregularities are evidences of more serious myocardial defects. The rapid and persistent increase in the number and a multiplication of the foci of origin of extrasystoles point to advancing myocardial changes and are often associated with symptoms indicating cardiac insufficiency. Curiously enough some of the patients in whom I have discovered extrasystoles occurring constantly and in great numbers were quite unconscious of cardiac irregularities.

A more prolonged study of the different types of extrasystoles, their points of origin and their frequency may eventually lead us to modify our prognosis in accordance with such findings, but as yet our facts do not warrant more positive statements. Our prognosis ultimately rests on the extent of myocardial damage, and the extrasystole is merely one of the symptoms which suggest that the defective muscle is little or much affected.

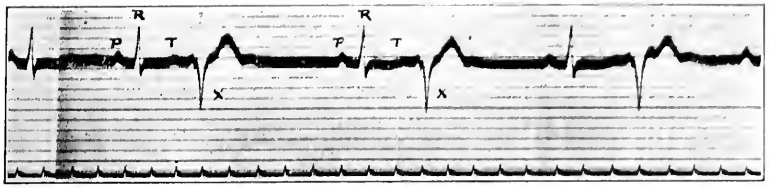

FIGURE 52

"Bigeminus." The extrasystoles (x) arise from the wall of the right ventricle near the apex (Type 2).

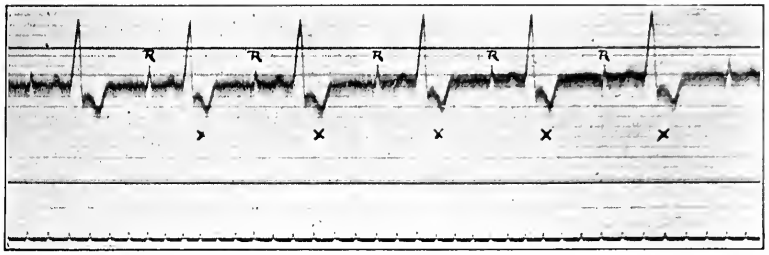

FIGURE 53

"Bigeminus." The extrasystoles (x) arise from a point in the wall of the left ventricle near the base (Type 3).

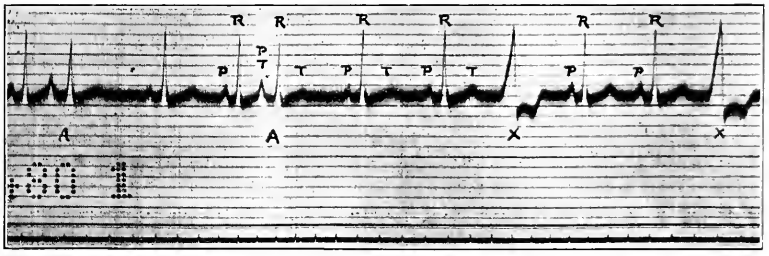

FIGURE 54

Extrasystoles from different points of origin. A = auricular extrasystole with incomplete compensatory pause. X = ventricular extrasystoles with complete compensatory pause.

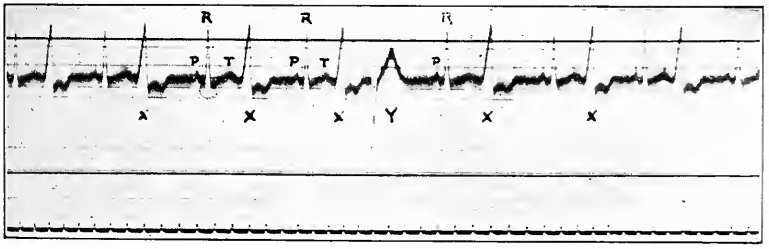

FIGURE 55

Two types of ventricular extrasystoles. X arising from the right ventricle near the base. Y arising from the left ventricle near the apex (Types 1 and 4).

CHAPTER VIII

Tachycardia

A heart rate of abnormal rapidity is one of the most frequent phenomenon observed by the physician. For purposes of the present discussion one may classify all such cases in two groups:

I. ACCELERATED HEARTS.

II. PAROXYSMAL TACHYCARDIA.

The main clinical feature which distinguishes these groups is the manner in which the transition from the normal to the abnormal rate is accomplished. In the case of the *accelerated heart* the transition from the slow to the rapid and from the rapid to the slow rate is gradual; in a very brief period the heart cycle may become so shortened that the rate per minute is increased 50 per cent., and yet, as observed by palpation or auscultation, the length of any two successive cycles is so nearly identical that neither the finger nor the ear is able to detect the minute differences which go to make up the change.

In the *paroxysmal tachycardia* the onset and the offset of the change in rate is abrupt and the observer and even the patient is usually able to detect the sudden transition without difficulty.

THE ACCELERATED HEART

ETIOLOGY AND PATHOLOGY

It has already been pointed out that the rate of the normal heart is not fixed, but varies with the needs of the body at any particular moment. This rate adjustment is brought about through the regulatory mechanism of the extra cardial nerves. In the conditions now to be considered the underlying factors are many and complicated, but we may recognize three important elements which individually or in association may produce an abnormal acceleration of the heart:

(A) *The outside demands on the heart may be excessive.*

A full discussion of the demands on the heart which originate outside of the cardio-regulatory nervous mechanism and the cardiac

tissues themselves, the nature of such demands and their modus operandi, important and interesting as they are, would lead us outside of the limits which we have set in these chapters devoted to the subject of myocardial function. However, this outside call for increased cardiac activity must never be lost sight of in analyzing the response of the cardiac tissues to these demands. A simple illustration of the response of the heart to increased demand is seen in the effect of work. As a general rule it may be stated that the response to physical exertion of an individual with a good myocardium is shown in an increased blood pressure. One with a defective myocardium shows an abnormal acceleration of the heart rate. With a normal heart muscle under efficient regulation and a normal vasomotor tone, moderate exercise causes an increase of cardiac rate, but with rest the rate should return to its usual level in the space of a very few minutes. That the demands of exercise produce an intrinsic physiological effect on the myocardium is evidenced by the fact that the normal electrocardiogram constantly shows under such stress definite though small changes; in addition to the shortening of the diastolic period (*T-P*) there is an increase in the size of waves *P* and *T* and a deepening of *S*.

(B) *The extracardial nerves may be at fault in their regulatory capacity.*

It is quite evident in certain accelerated hearts that the fine nervous adjustments are unbalanced. The activity of the vagi are depressed or there is an excessive activity of the accelerators, such a lack of balance mainly affects the heart through its pacemaker, the sinus node. This is probably the mechanism of the rapid changes of rate in emotional conditions, the so-called "labile pulse" of neurasthenics and the more persistent rate increase in certain organic lesions of the central nervous system and of the peripheral nerves supplying the heart.

(C) *The heart muscle may be defective* and responds to normal outside demands with abnormal acceleration. The direct application of heat to the myocardium is known to increase the cardiac activity. Bacterial and chemical toxins set free in many of the infectious diseases are recognized as efficient agents in causing functional or organic changes of the myocardium, which are the basis of a response in rate out of proportion to the stress.

While we can sometimes designate one of these particular factors, excessive outside demands, defective nerve regulation or myocardial damage, as the cause of the increased heart rate, the problem is usually more complicated. No doubt frequently two or all of these elements play a part. In the present state of our knowledge we are often at a loss in deciding which link in the chain is at fault, and, if more than one, their relative importance.

Fever is nearly always accompanied by an acceleration of heart rate, and so uniform is this phenomenon that the well-known Liebermeister's rule of an increase of 8 pulse beats for each degree of temperature above the normal is found approximately accurate, albeit, with many exceptions. Whether this is brought about by the increased temperature of the blood passing through the heart, or by the chemical action of associated toxins on the regulatory nervous mechanism, or on the cells of the cardiac muscle, is undecided.

The increased heart rate of *shock* is undoubtedly due to local or general vaso-motor disturbance with its reflex demands on the heart to maintain an adequate blood pressure. A similar explanation seems probable for *Graves' disease,* and the excessive administration of thyroid extract in which the evidence points to the damaging effect of toxins on the vaso-motor apparatus, rather than the heart muscle. The "labile pulse," wide pulse pressure, flushing, local sweating and tremors characteristic of this disease suggest that the toxins chiefly attack the sympathetic nervous system, possibly incidentally producing a hypertonus of the accelerator nerves, and probably act on the heart muscle only in an indirect manner. *Pregnancy* probably has only a reflex effect on cardiac activity.

Exhausting diseases (tuberculosis, etc.) and *convalescence from wasting diseases* (typhoid, etc.), nearly always show some degree of increase pulse rate. Each one of these conditions, febrile or afebrile, with toxic and nutritional disturbances may affect the outside demands on the heart, the functional balance of the extracardial nerves, or the cardiac muscle, and in each instance the effort should be made to determine and apportion the relative responsibility of each of these factors in the acceleration of the heart. The *severe anemias,* high grades of chlorosis, marked secondary anemias (as in malignant disease), and the primary pernicious forms are invariably associated with an increase in heart rate. In the extreme

grades of anemia the cardiac muscle shows an advanced degree of degeneration with fatty infiltration and hemorrhages,* so that we have little hesitancy in ascribing the altered heart activity to the direct toxic or nutritional effect on the myocardium.

In *valvular disease* the mechanical defect must be considered. The volume output is unusual and the normal bodily calls for blood are met by an increased heart rate. In the majority of these cases, however, the disease which was the agent in distorting the valves has also injured the myocardium and this, in association with the change in cardiac tone resulting from dilatation and hypertrophy, are important influences in modifying heart rate. Changes in the myocardium are produced by *acute rheumatic fever* and *other infectious diseases* with a resulting acceleration of heart rate. These changes may be chemical with no demonstrable histological abnormality, or there may be fatty degeneration and fibrous replacement, so that we meet with many degrees of functional impairment.

MECHANISM

The main link in the mechanism through which the increased rate of the "accelerated heart" is produced is the "sinus node," the normal pacemaker of the heart. Here the fundamental properties of "stimulus formation" or "excitation" or both, become heightened. This change may be intrinsic, that is to say, the chemical processes of the muscle cells of the node are so changed that they form and explode stimulus material more rapidly, or the change may be brought about by the modifying impulses showered on the node by the extracardial nerves. The sinus node is particularly influenced by impulses brought to it by the right vagus and the right accelerator.†

The distinguishing feature of the "accelerated heart" is that the sinus node retains its function as the pacemaker of the heart. This is shown by the graphic records which indicate that the impulse formation arises at the normal point and spreads through the auricle, the bundle of His and the ventricle in a normal orderly fashion. There are several facts, however, which indicate that, in these "accelerated hearts" other portions of the musculature may have their properties of "stimulus formation," "excitability," and perhaps also "conduc-

*Lazarus: "Pernicious Anemia," Nothnagel's Practice, Phila., 1906, p. 283.
†Robinson and Draper: Jour. Exp. Med., 1911, xiv, p. 227.

tion" heightened. It is known that the fibers of the left vagus and of the left sympathetic are in the main distributed to portions of the heart below the sinus node,† and experimental evidence indicates that cutting the left vagus and stimulating the left sympathetic have a considerable effect in increasing the heart rate. Again in certain "accelerated hearts" it may be seen that systole, which in the normal heart has a very constant length, is shortened. This is only conceivable on the ground that one or more of the fundamental properties of cardiac muscle mentioned above are quantitatively changed.

The principal change from the normal in the cardiac cycle of the accelerated heart is a shortening of the diastolic period. From this it follows that the rest period of the heart is curtailed and the time allowed for the recovery of the property of "contractility" is considerably less than in the heart working at the normal rate, hence the contractile power is less. Furthermore there is less opportunity for the heart to receive its normal quota of blood, hence the volume output is smaller. It follows as a result of these two factors that the pulse is smaller in volume and of diminished force.

IDENTIFICATION

Little need be said of the clinical recognition of the "accelerated heart;" the pulse may be counted either by palpation at the wrist or perhaps more accurately by auscultation at the apex. If one is present during the change from a slow to a faster rate this is best detected by counting the pulse in 10 second intervals, omitting every other 10 seconds. Neither the finger nor the ear can detect the small differences in the lengths of the successive diastolic periods, but the variations in length of the cycles separated by considerable periods is easily made out. The volume output of the heart is usually somewhat diminished with the acceleration of the rate and the consequent diminution in the peripheral arterial wave may be quite evident.

The *polygram* of the accelerated heart conforms to the normal except that the diastolic period is shortened. This is at times so marked that the *a* wave may be superimposed on the preceding *v* wave. The jugular tracings (Figures 56 and 57) show a normal sequence of waves, *a*, *c*, *v*. Figure 56 is a record of a girl, 15 years of age, suffering from rheumatic myocarditis and adherent pericardium.

†Cohn and Lewis: Jour. Exp. Med., 1913, xviii, p. 739.

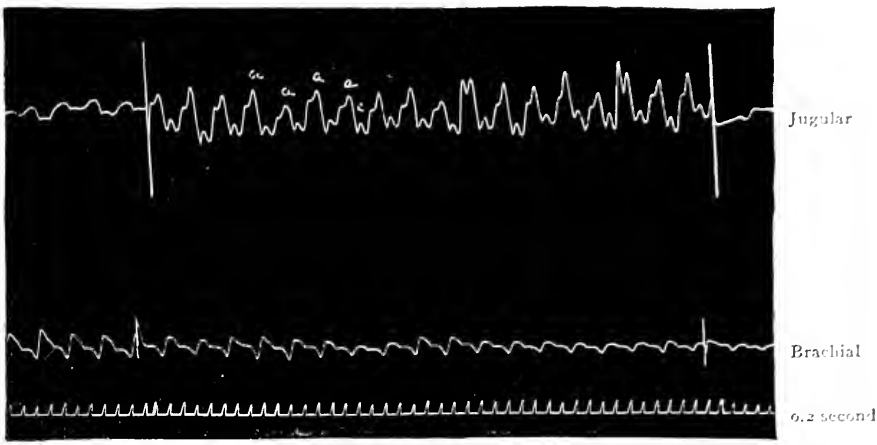

FIGURE 56
Accelerated heart. Rate 145. Patient suffering from rheumatic myocarditis.

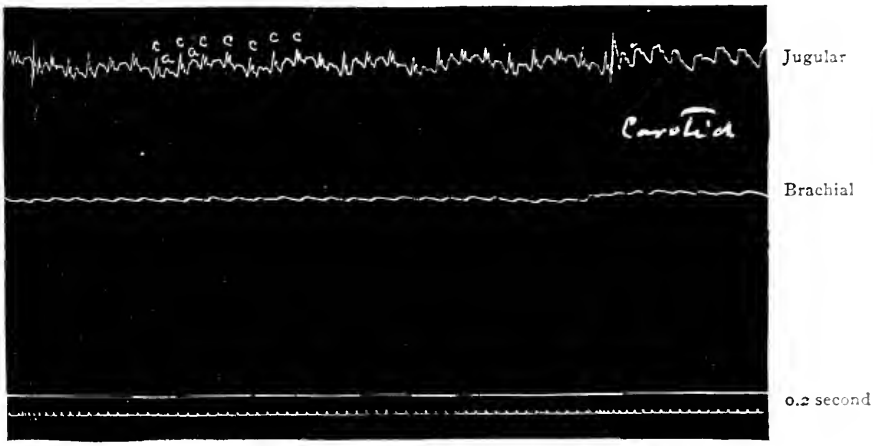

FIGURE 57
Accelerated heart. Rate 138. Case of Graves' disease.

The rate at the time the record was taken was 145 and the rapidity was in part due to excitement, as her pulse at rest was commonly 120. The slightest physical exertion at this time would send her pulse to 160, suggesting a marked instability of the sinus node.

In Figure 57 is shown a tracing of a case of Graves' disease; the rate is 138. It is evident from the jugular tracing that the normal pacemaker is in control and that the rapid rate depends upon the shortening of the diastolic period.

Electrocardiograms of accelerated hearts are presented in Figures 58 and 59. Figure 58, from a case of Graves' disease, shows a short diastolic period, but the sequence of waves is normal. Figure 59 was obtained from a case of cerebral hemorrhage, a few hours before death. The diagnosis was confirmed by autopsy and it seems clearly a case in which the nervous regulatory mechanism is at fault. The *P* and *T* waves in this record overlap. Careful measurement suggests that the earlier of the two peaks represents the auricular contraction which occurs before the preceding ventricular systole is completed. This curve simulates quite closely the records obtained experimentally during the stimulation of the right sympathetic ganglion by Rothberger and Winterberg.*

THE CLINICAL SIGNIFICANCE AND PROGNOSIS

of the accelerated heart depend on the underlying condition and to determine this, the responsibility of excessive outside demands, lack of balance between the elements of the nerve regulatory mechanism and defects of the myocardium, must be correctly apportioned. In general one may say that excessive outside demands and unbalanced nerve control acting on a heart with its myocardium intact, are usually more readily corrected, and hence of less serious import to the patient than when the heart acceleration depends upon an intrinsic defect of the myocardium. But even a normal myocardium may be worn out by the excessive activity induced by extracardial conditions, and a defective myocardium properly handled may recover full functional efficiency. The tests by which we may gauge the integrity of the heart muscle and its reserve force will be discussed in a later chapter.

*Archiv. f. d. ges. Physiol., 1910, cxxxv, p. 557, Fig. 18, d.

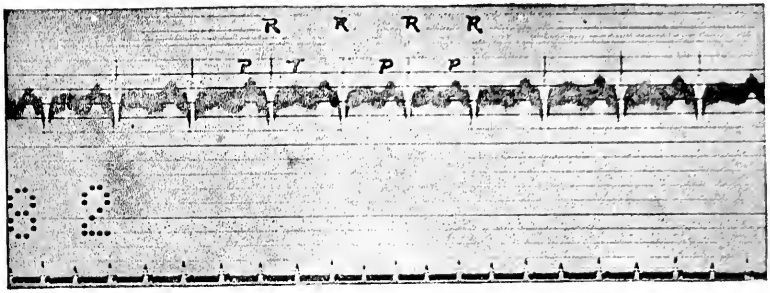

FIGURE 58

Accelerated heart. Rate 148. Case of Graves' disease.

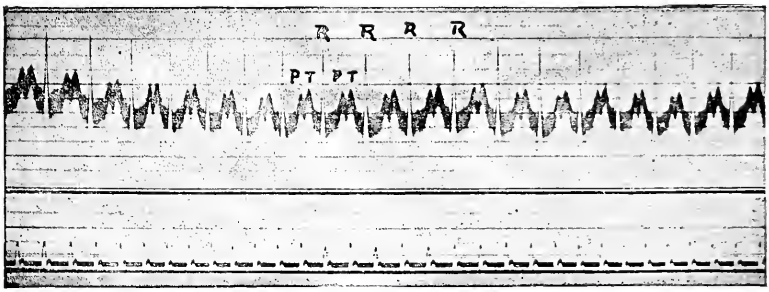

FIGURE 59

Accelerated heart. Rate 184. Terminal tachycardia of cerebral hemorrhage.

CHAPTER IX

Paroxysmal Tachycardia

Acceleration of the heart rate, which has been discussed in the preceding chapter, is exceedingly common and is important as a symptom associated with many conditions. Paroxysmal tachycardia, which we are now to examine, is relatively rare, and is associated with phenomena so distinct and definite that the syndrome deserves consideration as a clinical entity.

This group is particularly characterized by the suddenness of the change in the rate of the heart. The acceleration in rate occurs as a paroxysm whose onset is abrupt and whose termination is equally sudden. The change in rate, both of the onset and offset of the attack, occurs in a period of time less than that occupied by one normal cardiac cycle. The duration of the paroxysms are extremely variable. They may last for only a few beats or may continue for minutes, hours or days. The longest attack which has come under my notice was continued for 28 days. This variability is the rule not only comparing different cases, but also in the successive attacks of a single individual. The relative time consumed by the paroxysms and the intervals of slow rate is very variable, but in nearly all instances the slow periods exceed the paroxysmal periods by a considerable margin.

MECHANISM

An analysis of the paroxysms shows that it is composed of a series of contractions having their origin in some part of the cardiac musculature other than the sinus node; in other words, a rapid succession of extrasystoles; in some point of the heart wall excitability is raised to such a point that for a period stimuli are set free at an abnormally rapid rate, and, in accordance with the law that the most excitable portion of the heart sets the rate for the less excitable portions, this excessively irritable point usurps the function of the pacemaker, and for the time the normal pacemaker, the sinus node, is buried in the flood of stimuli arising from this new point of origin. Usually all of the contractions of a given paroxysm arise from a single point and spread over the heart

muscle by the same path. This is shown by the similarity of the waves obtained in graphic records. For the most part, the contractions are rhythmic, hence their rate is to a degree a measure of the rate of stimulus formation and the excitability of the irritable point.

During the period of slowing, the sinus node regains its ascendency and sets the pace. If one studies carefully the periods of slow rate, one will almost invariably discover isolated extrasystoles occurring more or less frequently. These are usually of the same type as those which go to make up the beats of the paroxysm, and are often of material assistance in determining the particular point in the heart in which the extrasystoles of the paroxysms have their origin. It is conceivable that any portion of the heart muscle may be capable, under suitable conditions, of assuming the rôle of pacemaker for a limited period of time. We are certainly able to define paroxysms which have their origin in the wall of the auricle, in the region of the auriculo-ventricular node and in the right and left ventricles. Most of the paroxysms have an auricular origin. Ventricular paroxysmal tachycardias are comparatively rare.

When the point of origin is in the auricle, the ventricle usually responds promptly and in the usual manner to each auricular impulse. At times, however, the electrocardiographic records suggest that the stimulus has taken a path through the ventricle wall, somewhat removed from the normal, or again the exciting effects of the frequent stimuli may be seen in a depression of the bundle contractility, as evidenced by an abnormally long period between the auricular and ventricular contractions.

It has been shown by Erlanger* that stimuli may pass over the conducting system of the heart in a direction opposite to the normal. We have evidence that this occurs in paroxysms of ventricular origin, and that the auricular contraction is a response to stimuli reaching it from the ventricle.

EXPERIMENTAL PRODUCTION

In a previous chapter it has been pointed out that single extrasystoles may be produced experimentally by applying mechanical

*Arch. Int. Med., 1913, xi, p. 362.

or electrical stimuli to various portions of the cardiac musculature. If a properly spaced series of such stimuli are applied to the wall of the heart, a tachycardia will instantly result, composed of a succession of extrasystoles. During such an artificial paroxysm, the activity of the normal pacemaker is submerged by the stimuli set free from the new focus. When the artificial stimuli are withdrawn the tachycardia terminates abruptly. The normal pacemaker immediately regains its ascendency and the normal rhythm is resumed. Such paroxysms may be induced by stimulation of either the auricle or the ventricle. When the ventricle is thus excited, the stimuli are transmitted upward to the auricle, a direction the reverse of the normal, and the contractions follow instead of precede the ventricular contractions. These retrograde stimuli pass the bundle of His with less velocity than those which pass over the heart in the normal direction, hence a part of them may be blocked and the auricle may fail to respond to each ventricular contraction. Tachycardias have been experimentally produced by the administration of aconitin (Cushny), muscarine (Rothberger and Winterberg), by an abrupt increase of the blood pressure (Hering), and by ligature of the coronary arteries (Lewis); a production of attacks of tachycardia by ligation of the coronaries particularly elicits our interest, since it more nearly approximates conditions which we may encounter clinically. Lewis* found that obstruction of the blood flow in the right coronary was usually, and that of the descending branch of the left coronary was invariably, followed by isolated ventricular extrasystoles, as the nutrition of that portion of the ventricular wall supplied by these vessels became progressively impaired, extrasystoles appeared at shorter and shorter intervals, until finally there was established a rapid series of rhythmically recurring extrasystoles, constituting a true paroxysmal tachycardia. Under these conditions the stimuli became retrograde and the auricular followed the ventricular contraction. The extrasystoles were rhythmical and graphic records showed that in a given case all the extrasystoles had a single point of origin. In dogs rates between 300 and 420 per minute were obtained. The phenomenon occurred both when the vagi were intact and when they were sectioned, showing that the disturbance

*Heart, 1909-10, i, p. 98.

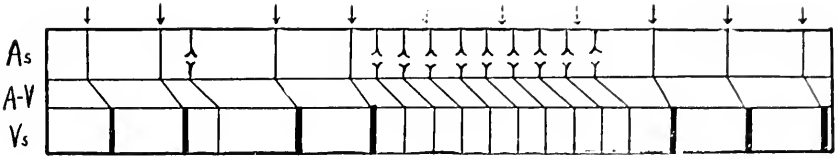

FIGURE 60

Diagram showing mechanism of auricular tachycardia. One isolated extrasystole is indicated and a short paroxysm composed of a rhythmic series of similar extrasystoles. The temporary pacemaker is located in the wall of the auricle. The conduction time (*A-V* period) is lengthened during the paroxysm.

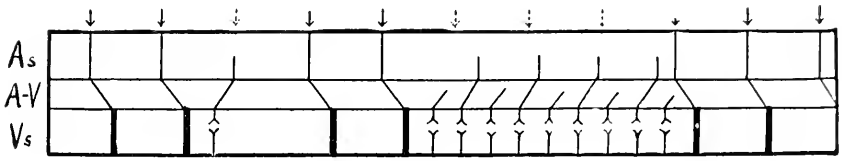

FIGURE 61

Diagram showing the mechanism of ventricular tachycardia. One isolated ventricular extrasystole and a short paroxysm composed of a rhythmic series of similar extrasystoles. The temporary pacemaker is located in the ventricular wall. During the paroxysm the auricle contracts in response to "retrograde stimuli" passing upward from the ventricle, every other impulse from the ventricle is blocked.

The arrows indicate the points of origin and the direction taken by the stimuli. Dotted arrows indicate the time at which the normal stimulus at the sinus node should reach maturity if its formation were not interrupted by the extrasystole. The thickness of the lines representing ventricular systole indicate the relative effect of the normal beat and the beats of the paroxysm in maintaining an adequate circulation. As = auricular systole. A-V = time of transition from auricle to ventricle or the reverse. Vs = ventricular systole.

had its origin in the wall of the heart and could not be ascribed to altered central innervation. When the ligature was removed and the circulation became re-established, the paroxysm abruptly ceased and the sinus node resumed its function of pacemaker.

The diagrams, Figures 60 and 61, indicate the mechanism of the paroxysmal attacks. Figure 60 represents a focus of abnormal irritability situated in the wall of the auricle. The impulses are set free so rapidly that the stimulus material forming at the sinus node is destroyed before reaching maturity. As soon, however, as the abnormal irritability of the auricular wall is lost, the accumulation of stimulus material at the sinus node continues for the normal period and thus the node resumes its rôle of pacemaker. Figure 61 represents an abnormal focus in the ventricular wall, which, for a short period, becomes the pacemaker of the whole heart. Here the ventricular impulses become retrograde, that is, they passed upward over the *A-V* bundle and stimulated the auricle from below. These impulses are frequently blocked, as is indicated in the diagram, in which the auricle responds only to every other ventricular impulse.

It might be supposed from this review of the mechanism of these disturbances that paroxysmal tachycardias would be frequent sequelæ of single extrasystoles. This is not the case. Isolated extrasystoles are extremely common. Probably most individuals reaching the age of 50 have had extrasystoles at one time or another, but attacks of true tachycardia are comparatively rare. On the other hand, it may be said that probably every true paroxysm is preceded by isolated extrasystoles.

PATHOLOGY

Little is known of the histological changes which may form the anatomical basis of paroxysmal tachycardia. In my own series only two cases have had a fatal termination, and in neither of these was a post mortem permitted. In the literature several autopsies have been reported and these have all shown more or less excessive myocardial change—sclerosis, fibrosis, atrophy, and arterial degeneration, particularly of the coronaries. One does not feel that we have as yet evidence of any definite pathological lesion which

is characteristic. Experimental evidence suggests that the cause may be found in the intracellular chemical change induced by variations in the blood supply in the heart, which may or may not show degeneration of the myocardium.

ETIOLOGY

In no one of my series of 33 cases of paroxysmal tachycardia have I been able to obtain a history of a similar condition in an ancestor or in any immediate relative. My youngest case was a girl who had her first attack when 9 years of age; the oldest a man of 69, whose paroxysms had annoyed him for 2 years. One patient, a man of 44, has suffered from attacks over a period of 20 years. The distribution by decades of the time of onset in my series is as follows:

Decade	Under 10	10-20	20-30	30-40	40-50	50-60	60-70
Number of Cases	1	4	8	6	6	5	3

Among the 33 cases which I have observed, 23 were males and 10 females. The following tabulation indicates that the syndrome occurs about twice as often in men as in women.

	Hoffmann*	Lewis†	Hart	Total
Men	6	18	23	47
Women	4	11	10	25
				72

An analysis of my cases presents the following factors, which may have a bearing direct or indirect on the condition of the myocardium. Alcohol was used to excess by 4; tobacco by 2 of the men. Severe gastrointestinal disturbance had preceded the attacks for several years in 3 of the women; nearly all had a history of one or more of the infectious diseases of childhood; in one case the onset of tachycardia followed 6 months after a severe infection of the middle ear; in another yellow fever antedated the attacks by 2 years. There had been frank attacks of acute articular rheumatism, followed by endocarditis with valvular defects, in 4 cases; a syphilitic infection was demonstrable in 4 cases, 3 of which

*Die Electrocardiographie, Wiesbaden, 1914.
†Clinical Disorders of the Heart Beat, London, 1913.

showed evidence of myocardial damage other than the attacks of tachycardia. Several of the series had taken considerable doses of digitalis; in one a physician whose arrhythmia had been wrongly diagnosed as complete irregularity and auricular fibrillation had taken very large doses, and it seems to me that this was undoubtedly an important agent in increasing the irritability of the heart muscles. The attacks in a young patient of my series, a boy of ten, immediately followed a race in which he participated, at which time the physician who saw him found evidence of acute dilatation. A case of mild Graves' disease, in which the pulse averaged 100, has shown on several occasions paroxysms lasting only a few minutes in which the rate was between 160 and 170. Valvular defects were present in 12 of my patients; the mitral valve was involved in 10, of which 4 were cases of well-marked stenosis; two patients had aortic insufficiency and one had defects of both the aortic and mitral valves; 15 cases showed various degrees of cardiac enlargement. In many cases the irritability of the heart muscle seems to require a very small exciting factor to induce an attack. The patient will usually ascribe the onset to flatulence, some emotional disturbance or unusual physical exertion; any one of these is probably an efficient cause to call forth an attack in a myocardium suitably damaged.

SYMPTOMS

The symptoms associated with paroxysmal tachycardia are of great variety, and show great differences from individual to individual. This is doubtless in a large measure due to the extent of damage present in the myocardium and the ability of the heart to meet the tax thus exacted. The patient is practically always conscious of the abrupt onset and termination of the attacks. They usually describe the attacks as beginning with one or two "thumps" or "throbs" in the precordial region, followed by a sensation of fluttering in the chest, which is terminated by another "thump" or "flop," and the attack is over. The amount of anxiety is always greater in the early attacks; as the patient becomes more or less accustomed to the paroxysm he is less alarmed, and a momentary pause in his activities may be the only evidence to show that he knows the attack is on. This absence of alarm I have noticed

particularly in young adults who have had attacks for a number of years, but whose hearts show no anatomical abnormality and functional disturbance characterized only by the attacks of extrasystoles at more or less infrequent intervals.

One of my patients, whose attacks have continued for several days, was quite unconcerned even when his heart was beating at 170. He rarely voluntarily assumed a recumbent position on account of the attacks and it was difficult to convince him that rest at these times was important.

In those who have an associated valvular lesion, and in those with evidence of marked arterial changes, a greater discomfort and attendant anxiety are closely associated with the symptoms referable to the cardiac insufficiency which is induced by, or the precordial pain which accompanies, the attack.

At the onset patients often complain of palpitation in the chest and a swelling and pulsation of the vessels of the neck. Often they have eructations of gas, nausea and vomiting. There may be a "gone," sinking feeling, and, if the attack is prolonged, sweating, coldness, great lassitude and an intolerable feeling of weakness. They may have a sensation of palpitation or of bounding in the chest, shortness of breath or a sensation of suffocation. In one case under my observation attacks were invariably accompanied by a watery diarrhœa; in another by frequent micturition.

In the prolonged attacks, increase of the cardiac dulness to the left can sometimes be made out and the symptoms of circulatory embarrassment terminate the picture. The veins are not properly emptied, but are engorged, and there is pronounced cyanosis. The liver may be increased in size and become tender to palpation. There may be edema of the extremities; there may be cough with profuse thin, or blood-streaked, expectoration, with the physical signs of pulmonary congestion.

The paroxysms are often attended with headache and dizziness, more rarely with momentary or prolonged periods of unconsciousness, which may be explained on the basis of cerebral anemia. Pain is sometimes prominent. A feeling of oppression and of constriction of the chest accompanies the attacks in nearly all patients to a greater or less degree. The pain is usually precordial, and is sometimes sharp, suggesting a real angina, and may radiate into

the arms and back; sometimes one can detect areas of hyperesthesia over the chest and arms, following the distribution of one or more of the upper thoracic and lower cervical nerves. A few patients complain of numbness and tingling of the extremities.

A progressive cardiac insufficiency may terminate in general anasarca, pulmonary edema, collapse and occasionally death. As a rule, however, the signs of cardiac insufficiency are very moderate, and even when present to an extreme degree clear up with great rapidity, following the abrupt ending of the rapid heart action. The absence of alarm, the facial change of expression from one of anxiety to complete calm; the abrupt change from dyspnea to quiet breathing; the sudden cessation of pain; the subsidence of engorged veins of the neck coincident with the termination of the paroxysm present some of the most remarkable and agreeable clinical phenomena with which we are familiar.

The signs of pulmonary congestion and edema of the extremities may require a period of days for their subsidence, the rapidity depending to a considerable degree on the functional efficiency of the heart when it has resumed its normal rate.

As illustrating the character of severe attacks terminating fatally, one case which I had the opportunity to observe closely for a period of months, may be described.

A man, 55 years of age, who had a leutic infection 20 years earlier, had a heart moderately enlarged to the left and a faint systolic murmur at the apex. Between the attacks his pulse was about 70 with many extrasystoles. At all times there was evidence of a moderate degree of cardiac insufficiency. A description of the attacks, obtained from the patient, was as follows:

"The exact cause of these attacks of syncope and tachycardia, which come as often as twenty times in one day and have been absent as long as 26 days, cannot be determined. Many times he has been wakened from his sleep by dizziness to become unconscious and have a typical attack. Again, a slight exertion, as walking, going up stairs or straining to pass water, may be followed by an attack, but these same exertions, or even more severe ones at another time, may have no harmful effect. The attack comes on suddenly with dizziness, grayness before the eyes and a buzzing in the head like an organ. There are no premonitory symptoms. Un-

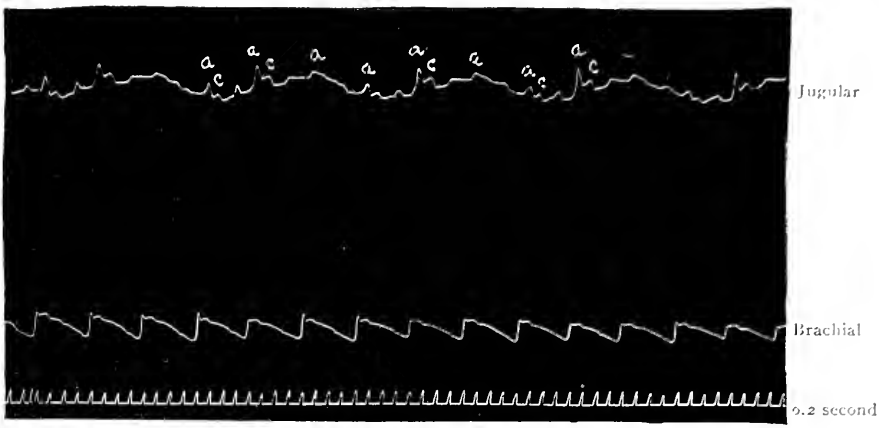

FIGURE 62

Normal. Rate 82. Respiratory rate 24. For paroxysm of auricular tachycardia in the same individual see Figure 63.

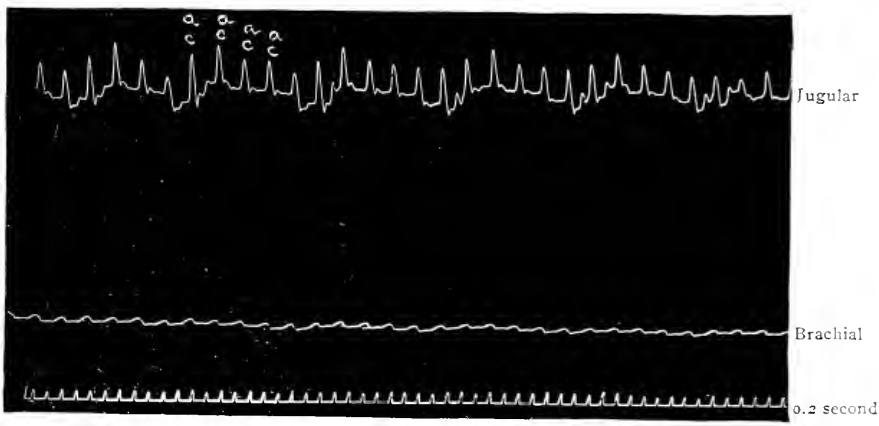

FIGURE 63

Auricular tachycardia. Rate 182. Respiratory rate 30. For record of the same case between attacks see Figure 62. Compare in the two records the volumes of the arterial and venous pulses. The jugular records are quite different in form, the large wave of the paroxysm = $a + c$.

consciousness follows rapidly, and when he comes to his heart is beating very rapidly, 200 to 250 to the minute. There is a choking sensation, as if a ball were in the throat, and he is shaking all over. There is never any pain over the heart or down the arm. At times he has been struck down as if by electricity without warning, again he has simply had dizziness and grayness, without losing consciousness. The tachycardia lasts a varying length of time, sometimes for only ten minutes, at other times all day. During its continuance he has great gastric disturbance, with frequent vomiting. He cannot forecast the end of the attacks until it is at an end. Then, at times, a violent regular beating of the heart is succeeded by two or three irregular beats, as if something shook the heart, and this is immediately followed by two or three tremendous throbs of the heart with each of which there is a feeling as if fresh air were forced into his throat and head and the attack stops suddenly as it began."

His paroxysms of tachycardia continued for 5 years, becoming gradually more frequent, and he finally died during an attack.

IDENTIFICATION

The conditions other than paroxysmal tachycardia which afford a heart rate of over 160 are extremely rare. During the paroxysm the pulse is exceedingly small, often irregular in force and frequently cannot be detected at the wrist. Under these conditions our examination should at once be directed to the precordial region. The apex beat may be imperceptible to the touch or, when palpable, may give the impression of complete irregularity. The heart sounds may be indistinct and have a fetal character; often they are sharp and distinct; as a rule, they are perfectly rhythmic, but so rapid that the rate can be only approximately estimated; this is best accomplished by counting short (5 seconds) periods. If one is fortunate enough to be making observations at the beginning or at the termination of the attack, the change in rate is readily detected. The transitions are usually accompanied by one or two large forcible beats, with loud sounds and unusually large pulse waves. The change in rate is quite abrupt. In the absence of such an observation the patient will frequently establish the diagnosis by his description of the sudden onset and termination of the attacks. Valvu-

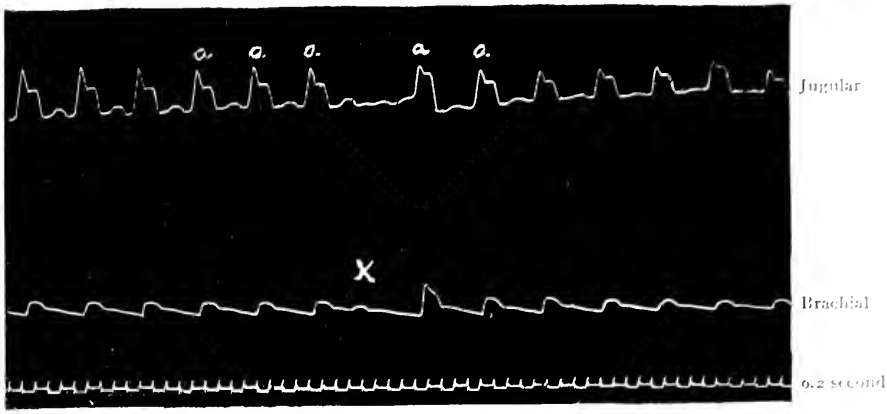

FIGURE 64

Rate 72. Between attacks same individual as Figure 65. At X is shown an extrasystole with an incomplete compensatory pause. Note the *a* wave is large and the *a-c* interval is of normal length.

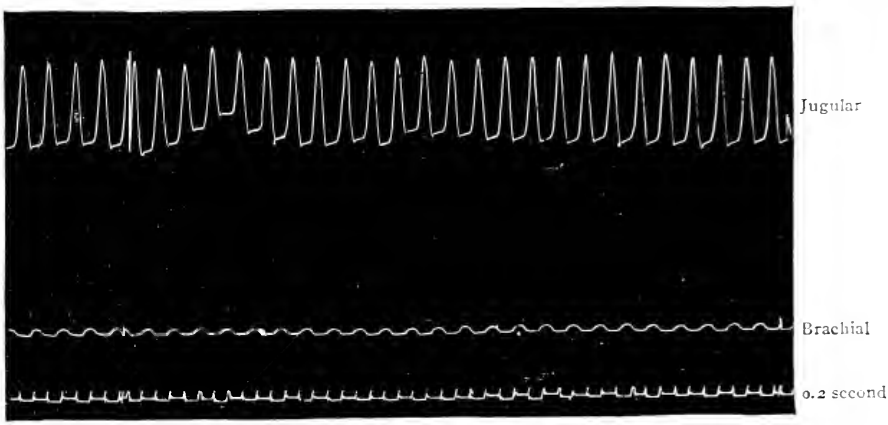

FIGURE 65

Auricular tachycardia rate 174. For slow rate see Figure 64. The large venous waves are the result of the simultaneous contraction of the auricle and the ventricle. Each one of these waves belongs to two cycles. They are composed of a *c* wave of a cycle just completed and an *a* wave of a cycle just beginning. The *a-c* interval is abnormally long.

lar murmurs, if present during the slow rate, sometimes cannot be detected during the paroxysm. In some cases a heart without murmurs during the slow period will develop a loud systolic murmur during the paroxysms.

During the slow periods extrasystoles followed by pauses, more or less fully compensatory, can usually be detected; sometimes they are very frequent, more often only occasional. Single extrasystoles are quite common between paroxysms which are of short duration and which follow one another at brief intervals.

During the paroxysms the veins of the neck are prominent, distended, hard and pulsate with great rapidity. Often two pulsations of the jugular may be seen to correspond to each systole of the heart.

In most instances the attacks are not affected by the position assumed by the patient and continue whether he sits up or lies down without change in rate.

When seen only between the attacks the diagnosis rests largely on the history, but the patient's description of the attacks is usually so clear that there is little difficulty in classifying the abnormal activity.

The cases which present the most obscure diagnostic problems are those with very frequent short paroxysms separated by equally short periods of slow rate broken by frequently occurring extrasystoles. These are often wrongly classified as complete irregularity due to auricular fibrillation. They may usually be assigned to their correct category by means of a careful and prolonged study of the ordinary physical signs. Their status may be absolutely settled by graphic records.

The *polygram* brings out clearly some features of the paroxysms which are observed with great difficulty by the ordinary means of eliciting physical signs.

In Figures 62 and 63 are shown brachial and jugular tracings taken from a woman 35 years of age. Figure 62 shows the usual condition of her pulse; the rate is 82; the arterial pulse is of good size and well sustained; the jugular pulse shows a normal sequence of waves *a*, *c* and *v*; the *a-c* interval is normal (less than 0.2 second). Figure 63 is a record taken during her second paroxysm, which lasted 2 days without interruption. At the time the tracing was

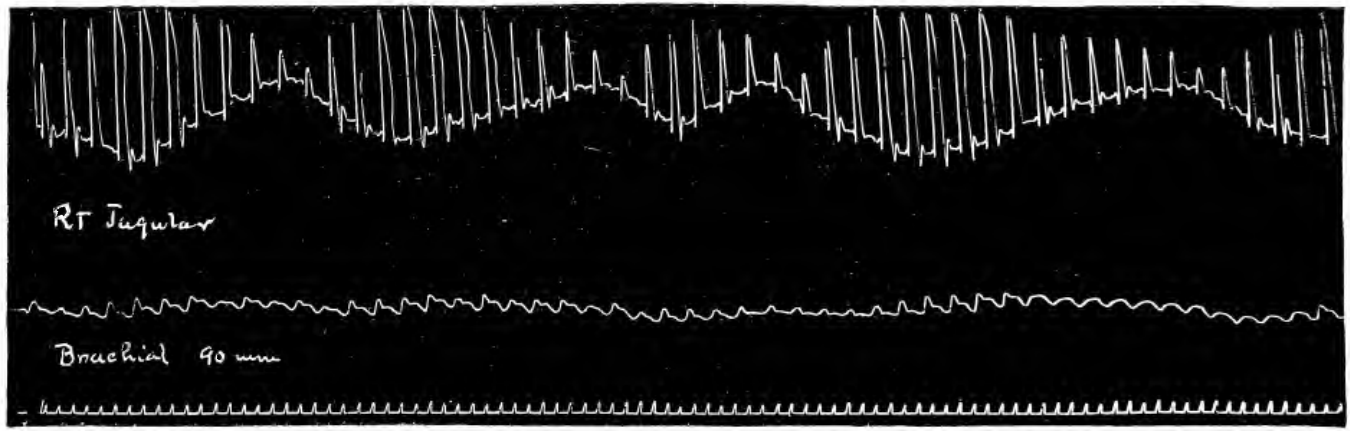

FIGURE 66

Auricular tachycardia taken during a paroxysm. Ventricular rate 158. Note the very large venous waves due to venous stasis and the respiratory curve upon which the jugular pulsations are superimposed.

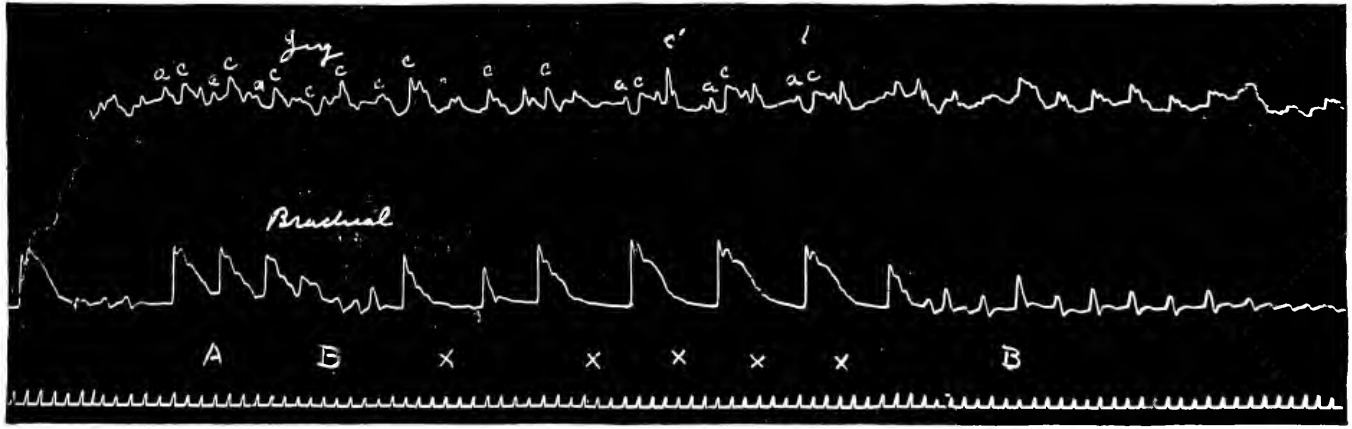

FIGURE 67

Ventricular paroxysmal tachycardia. Beginning and termination of attacks. At A normal rate of 90 per minute. At X ventricular extrasystoles, which show in the jugular but not in the brachial. At B paroxysms of tachycardia, rate 170 per minute.

secured the attack has been under way for 24 hours. The heart at this time was beating rhythmically at a rate of 182 per minute. The small volume of the brachial pulse is in great contrast to that of the slow periods. The venous curve shows, in place of the well-defined waves of the slow heart rate, one large wave and one small notch to each cycle. The interpretation is that the auricle and the ventricle are contracting simultaneously, so that the veins are unable to empty into the right auricle in the normal manner. The large jugular waves, much greater than the jugular waves of the normal period, are due to a summation of the *a* and *c* waves. It will also be seen that during the paroxysm the *a-c* interval is considerably prolonged (over 0.3 second), indicating that there is a delay in the conduction of the stimulus from the auricle to the ventricle. This is not an uncommon feature in tachycardias, the excessive functional demand on the slender *A-V* bundle leading to its partial exhaustion.

In these two figures the respiratory curve is brought out in the venous tracing. That in this case the dyspnea was not very marked is evidenced by the facts that the breathing was 24 during the slow rate and only 30 during the attack, and that the excursion is not very much greater during the paroxysm.

Tracings from another case of auricular tachycardia are shown in Figures 64 and 65. As in the preceding case, the contrasts between the cardiac rates (72 and 174) and the arterial pulse volumes of the two periods are shown in the brachial tracings. During the paroxysm (Figure 65) only one large wave appears in the jugular to each cardiac cycle.

The slow period (Figure 64) is interrupted at one point (*X*) by an extrasystole with an incomplete compensatory pause, hence we may conclude that it probably had its origin in the auricular wall. It is a series of such extrasystoles which constitute the paroxysm.

Figure 66 was taken from a man of 36 during a prolonged paroxysm. The ventricular rate is 158 and is perfectly rhythmic. The jugular tracing shows the great venous congestion and the very large waves which are due to simultaneous contractions of auricle and ventricle; conduction is delayed. The exact point in the auricle which has become the temporary pacemaker for the whole heart cannot be definitely settled from the polygraphic record. The

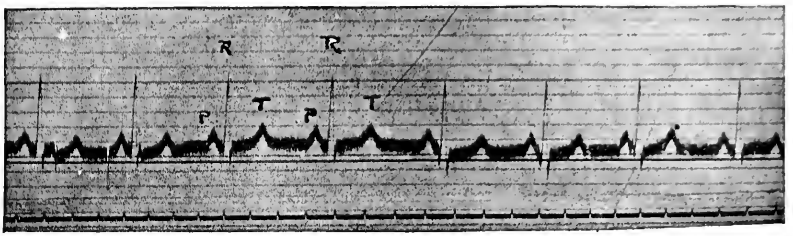

FIGURE 68

Normal. Rate 80 Same case as Figure 69. Lead II.

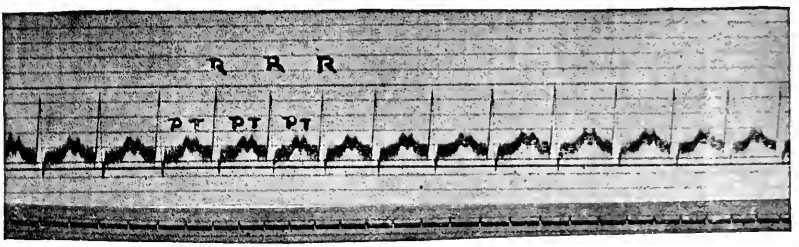

FIGURE 69

Taken during a paroxysm, rate 167. Lead II. Same patient as Figure 68. Note inversion of P, which notches the summit of T. Auricular tachycardia. The pacemaker of the heart is in the lower part of the auricle.

respiratory curve upon which the large jugular waves are superimposed show that, in spite of the prolonged attack, the breathing is not greatly accelerated; at this time it was 22 to the minute, but quite irregular.

A rare tracing from a case of ventricular tachycardia is reproduced in Figure 67. The brachial shows at A the usual rate for this patient between attacks (92 per minute). At X isolated extrasystoles, each with a complete compensatory pause, occur; the premature beats are so weak that they make practically no impression on the brachial pressure. At *B* are shown two short paroxysms of tachycardia, indicating the manner of the abrupt onset and termination of the attacks. The *a-c* interval of the "normal" rhythm of this patient was always longer than that of a normal heart, measuring nearly 0.3 second.

During the paroxysm the auricle contracted in response to the "retrograde stimulus" from the ventricle; this cannot be conclusively made out in the polygram, but is substantiated by electrocardiographic records (see Figure 80). The irregularity of this pulse is so extreme that it might easily have been mistaken for a case of "complete irregularity" and auricular fibrillation, had no graphic records been secured.

The *electrocardiogram* gives us information in regard to paroxysmal tachycardia which we can obtain by no other method. Through this agency we have discovered the real mechanism of the attacks. The knowledge acquired in this way tends to emphasize the importance of the muscle cell changes and to minimize the rôle played by the extra cardial nerves in inducing this change in cardiac activity. These graphic records convince us that a new point in the heart wall has become the temporary pacemaker of the heart. The proof is most clearly demonstrated, if we study the records of such a case as is shown in Figure 80, where the evidence is complete in a single curve. This is from a case of ventricular paroxysmal tachycardia, a condition of extreme rarity, hence it will be better to first direct our attention to the more common forms, namely, tachycardias of auricular origin.

Such a case is illustrated in Figure 69, which was taken during a paroxysm in which the heart rate was 167. Figure 68 was secured from the same patient a few hours after the cessation of the

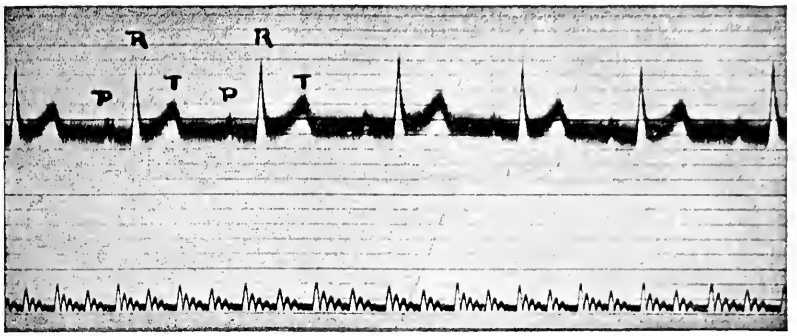

FIGURE 70

Same patient as Figure 71. Rate 76 between attacks. Taken by lead II. *P* is slightly notched, otherwise the curve is normal.

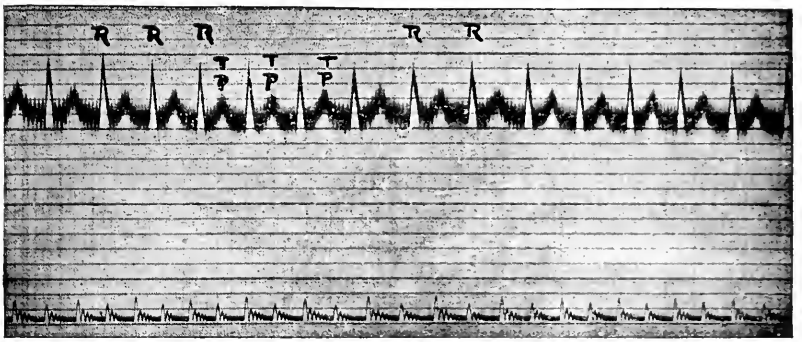

FIGURE 71

Auricular paroxysm, rate 174, lead II. From same individual as Figure 70. The small *P* wave is submerged in the large *T* wave.

attack. This record shows a perfectly normal curve for a heart with a rate of 80. Both records were taken by lead II (right arm and left foot). If we should superimpose the ventricular portion (beginning of *R* to the end of *T*) of the cycles shown in Figure 68 on one of the cycles of Figure 69, we would find that they correspond in every particular, except that the summit of the *T* wave shows constantly a deep notch. If we compare the records further, we note that in Figure 69 there is no wave which corresponds to the well-marked *P* wave of Figure 68. Careful measurement shows that the notch in the *T* wave (Figure 69) occurs at exactly the time at which a *P* wave of the normal rhythm should precede the *R* wave, hence we conclude that the positive *P* wave of the normal rhythm is replaced by a negative wave notching the *T* wave of the paroxysm.

In studying the auricular extrasystole, it was shown that when the premature beat started from a point in the auricle at some distance from the sinus node, the *P* wave of the electrocardiogram was distorted in form, or even completely reversed in direction, hence in the records under consideration we are led to conclude that the paroxysm shown in Figure 69 is composed of a series of extrasystoles having their origin at a point in the auricular wall considerably removed from the site of the normal pacemaker.

Figure 70, taken by lead II, shows a normal electrocardiogram, except for a slight notching of the *P* wave. Figure 71 was taken from the same patient during an attack which lasted for one hour, during which the heart rate was 174. Here the ventricular portions of the two records are almost identical, except that the waves of the paroxysm are a trifle smaller than those of the slow rate. During the paroxysm no *P* wave can be definitely located; in this case it was probably so small that it caused no distortion of the relatively large *T* wave.

Electrocardiograms of another case of auricular paroxysmal tachycardia are shown in Figures 72 and 73; both records were taken by lead III (left arm and left leg).

When Figure 72 was taken the heart rate was 75 per minute. This record shows several abnormal features; the *P* wave is slightly notched and *R* is directed downward (the latter feature is quite usual in hypertrophy of the left ventricle), *T* is also directed down-

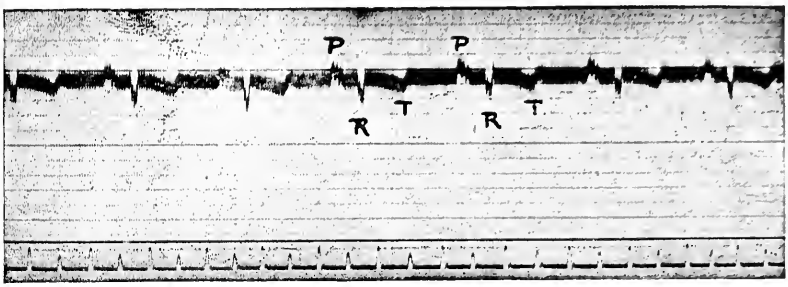

FIGURE 72

Slow period, same case as Figure 73. Rate 75. Lead III. *P* is notched, *R* and *T* have a downward direction.

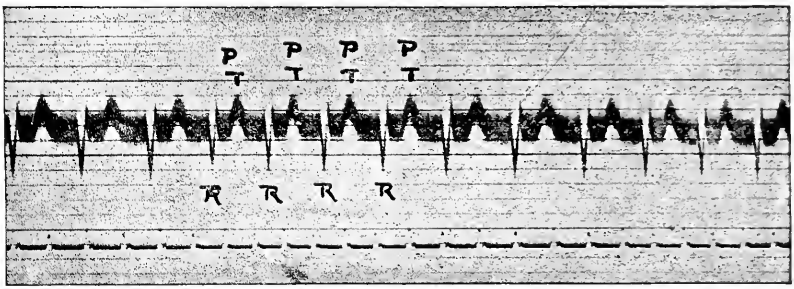

FIGURE 73

Auricular tachycardia, rate 168. Lead III. Same patient as Figure 72. Ventricular complex increased in size. *T* has become a positive wave. *P* is superimposed on *T*.

ward. During the paroxysm (Figure 73) the rate is 168. *R* is still directed downward and is increased in amplitude, suggesting a dilatation of the left ventricle. The slow wave between the *R* waves is an algebraic sum of the waves *P* and *T* of the new rhythm.

The next case, illustrated in Figures 74 and 75, shows some interesting features, during the slow period (rate 76 per minute) the *P* wave is unusually broad. *R* is slightly notched and the rhythm is broken by an extrasystole, which is plainly of ventricular origin. The paroxysm (rate 188) is composed of *R* waves followed by a depression, which in all probability are reversed *P* waves, having their point of origin in the lower part of the auricle, possibly near the *A-V* node. The *P-R* interval is prolonged, measuring over 0.2 second, exhibiting the delay in conduction which is not an uncommon feature of these cases. In this instance the complexes of the paroxysm probably represent extrasystoles of auricular origin and do not conform to the type of the isolated extrasystole which interrupts the slow rhythm (Figure 74).

Figures 76, 77 and 78 depict the mode of transition from the slow to the rapid and from the rapid to the slow rate in different cases.

The onset of a paroxysm can be seen in Figure 76 and the dislocation of the pacemaker from the sinus node to a point low down in the auricle is indicated by the change in forms of the *P* wave from a positive to a negative deflection. In the first cycle of the paroxysm the reversed *P* wave falls at the apex of the *T* wave, but subsequently notches the earlier portions of this part of the ventricular complex.

The offset of a paroxysm is shown in Figure 77. The bizarre complexes which intervene between the paroxysm and the slow rhythm probably represent extrasystoles of unusual types, and are doubtless the kind of cardiac activity which give the patient the subjective sensation of "throbs" or "thumps" at the time of the transition.

Another transition from a heart beat of 160 to one of 70 is shown in Figure 78. The curve is somewhat distorted by the movements of the patient produced by the sensations experienced at the time of the termination of his attack.

The most convincing evidence of the nature of the mechanism

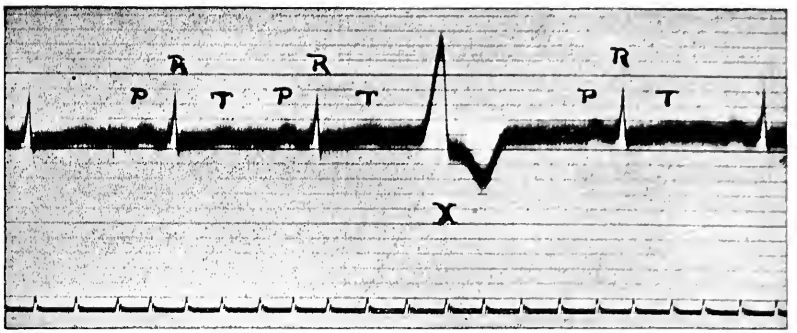

FIGURE 74

Rate of 76 interrupted by a ventricular extrasystole. Same individual as Figure 75. Lead II.

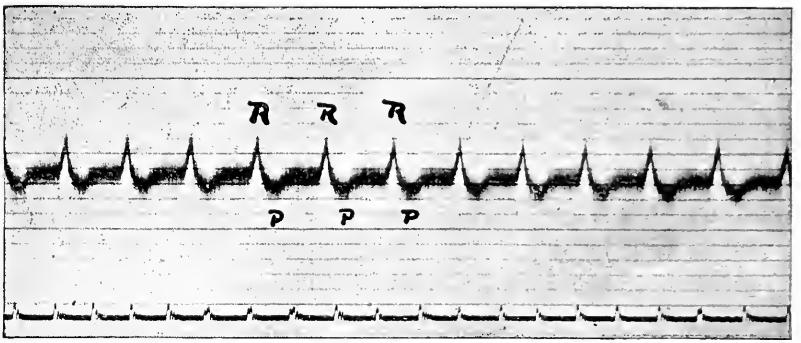

FIGURE 75

Paroxysm of tachycardia, rate 188. Lead II. From same case as Figure 74. Note reversal of *P* wave and prolonged *P-R* interval. Abnormal pacemaker probably situated near the *A-V* node.

in paroxysmal tachycardia is brought to view when we are fortunate enough to secure in a single record periods of slow rates interrupted by single extrasystoles, continued into periods of tachycardia. Such records are shown in Figures 79 and 80.

A short paroxysm of tachycardia (rate 168), changing to a slow rate (80) broken by extrasystoles, is shown in Figure 79. The patient, from whom this curve was taken, was a physician, 65 years of age, in whom the diagnosis of "complete irregularity," due to auricular fibrillation, had been repeatedly made. The correct diagnosis was hardly possible until electrocardiographic records were secured. The slow rate is interrupted by auricular extrasystoles (X_1) and another type of extrasystole (X_2) which has its origin in the ventricular wall. The auricular premature beats have their origin high up in the auricle, since the P wave of the extrasystole is a positive wave, as is shown by the waves which are clearly the sum of T and P. The paroxysm is composed of both kinds of extrasystoles, but the auricular type predominates, which is also the case in the period of slower cardiac activity

The electrocardiogram of a case of ventricular tachycardia* is shown in Figure 80. Tachycardias of this type are extremely unusual. The bizarre forms of the complexes of his slow rate (80) are seen in the short diastolic (T-P) interval, the broad P wave, the long P-R interval and the unusual form of the R waves. These features alone suggest serious myocardial damage. From time to time there appear isolated ventricular extrasystoles (X). The paroxysm (rate 200) is composed of complexes similar in form to those of the isolated extrasystoles. Between the large waves of the paroxysmal period are seen small waves *(P)* which occur with every other cycle. These undoubtedly represent auricular contractions due to retrograde stimuli arising in the ventricle. It appears that every other impulse from the ventricle is blocked. This record conforms in many particulars to the curves obtained experimentally after tying one of the coronary arteries, hence a tentative diagnosis may be made of partial coronary obstruction. The patient is still alive (3 years after the record was taken), hence the diagnosis has not been verified.

*A complete record of this case will be found in Heart, 1912, iv, p. 128.

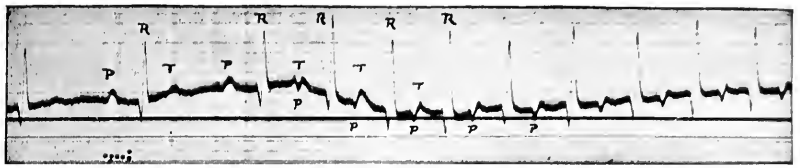

FIGURE 76

Transition from slow rate to paroxysm. Note dislocation of pacemaker to a point low down in the auricle as evidenced by the abrupt change in the direction of the *P* wave. The ventricular complex is unchanged except as its contour is broken by the reversed *P* wave. Compare Figure 69.

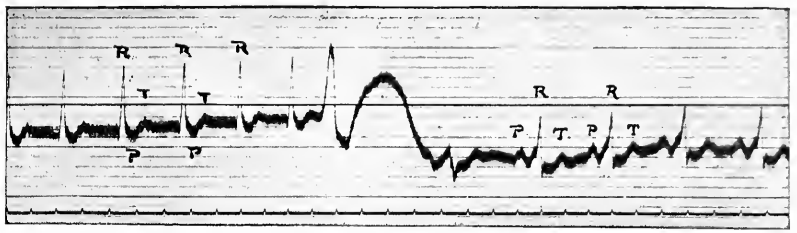

FIGURE 77

Abrupt termination of a paroxysm. Transition distorted by extrasystoles of ventricular origin. Pacemaker of paroxysm located near the *A-V* node. Compare Figure 75.

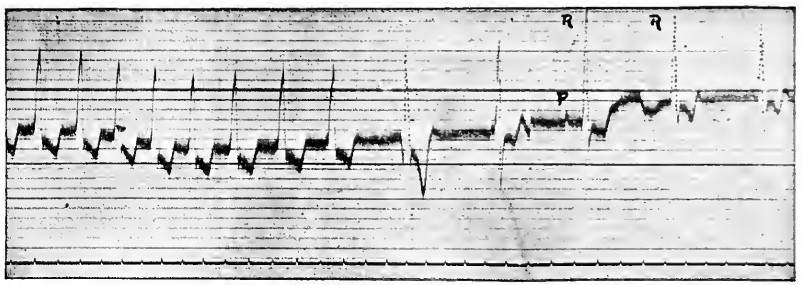

FIGURE 78

Auricular tachycardia, rate 160. Transition to rate of 70. Origin low down in the auricle.

CLINICAL SIGNIFICANCE AND PROGNOSIS

There is little doubt that every subject of paroxysmal tachycardia has a defect of the myocardium that must be seriously considered. The prognosis is most difficult. Some patients over a period of many years have attacks which incommode them but little and the attacks become less severe, less alarming, and in some instances disappear altogether. Some have only a few attacks before the fatal termination.

I have never seen a case that was fatal until a number of attacks had occurred, nor have I found such a case reported in the literature.

The condition of the heart in the intervals between attacks is important as an aid in determining the seriousness in the individual case. If at these times the heart shows no abnormality other than occasional extrasystoles, one can be reasonably sure that there is no imminent danger. If, however, marked valvular defects are present, if there is evidence of an old inflammation of the pericardium, if there is a general arteriosclerosis, if extrasystoles constantly occur at very frequent intervals, and if the heart is embarrassed in maintaining an adequate circulation in the periods of slow rate, the paroxysms will rightly be viewed with much apprehension. The paroxysm is very exhausting to the heart. If the myocardial damage is made evident by the attacks only, the heart will probably successfully carry this stress; if, however, other evidences of myocardial damage exist, the strain of the paroxysm is a far more serious matter. The patients who do particularly well are young subjects with no evidence of cardiac abnormality between attacks. Middle-aged and elderly individuals sooner or later invariably develop other evidences of myocardial insufficiency and, while they may have many and frequent attacks of tachycardia without serious manifestations, the ultimate outlook is less favorable.

The frequency and duration of the individual attacks do not seem to be very important factors in determining the prognosis. Much more important is the severity of the attacks as estimated by the degree of circulatory embarrassment, cardiac dilatation, the congestion of lungs and liver and edema of the extremities. At-

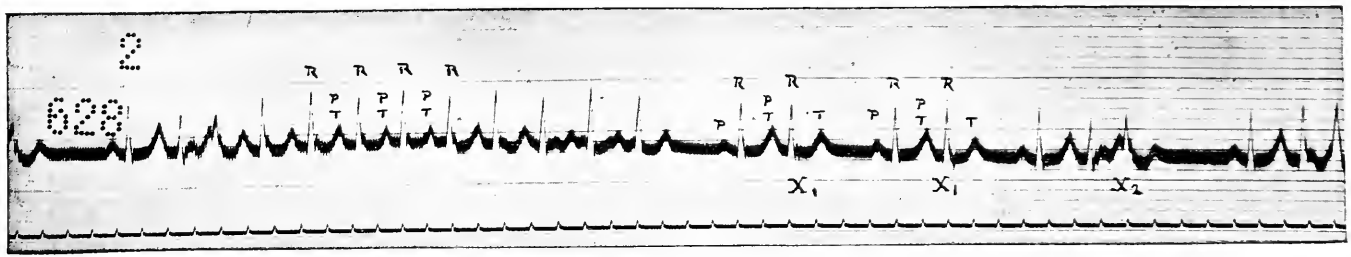

FIGURE 79

Paroxysmal tachycardia, rate 168. Transition to slow rate which is interrupted by extrasystoles of two types (X_1, X_2). The attack is composed of extrasystoles of both types. X_1 are evidently auricular premature beats, X_2 probably have a ventricular origin.

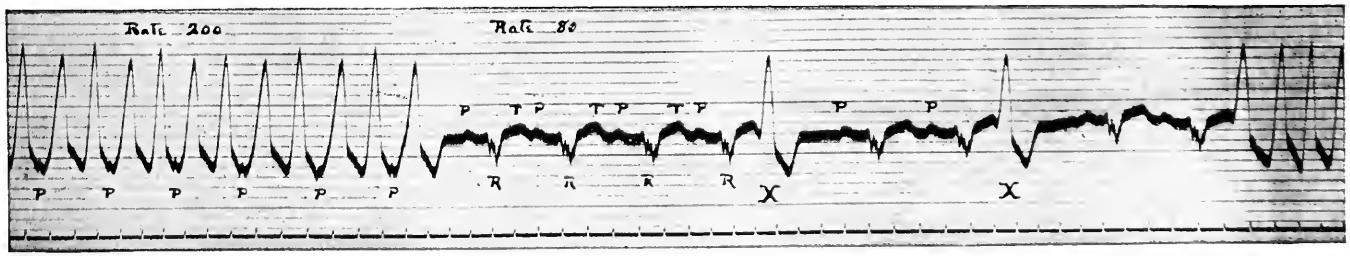

FIGURE 80

Ventricular paroxysmal tachycardia, rate 200. The complexes of the slow rate (80) are very abnormal in form. Isolated ventricular extrasystoles at X. Paroxysm made up of complexes of same type as the single ventricular extrasystoles. During tachycardia the auricle responds to every other retrograde stimulus from the ventricle (P). Time intervals = 0.2 second.

tacks associated with unconsciousness should be viewed with gravity.

With a history of a moderate number of attacks over a number of years in a young adult, with intervening periods of normal heart action, one may usually give a good prognosis. When the patient is more advanced in years and has paroxysms of increasing frequency and severity, and intermediate periods characterized by signs of cardiac insufficiency, the outcome of any particular attack is doubtful, the prognosis for the future is not good.

There is another rare form of paroxysmal tachycardia in which the attack consists in a short period of auricular fibrillation. This will be discussed in the chapter on auricular fibrillation.

CHAPTER X

Auricular Flutter

Closely allied to "auricular paroxysmal tachycardia," discussed in the last chapter, is an abnormal functional activity of the heart usually designated as *auricular flutter.** The terms "auricular tachycardia" (Robinson), "auricular tachyrhythmia" (Hoffmann), and "auricular tachysystole" (Rihl) have also been applied to this condition.

The chief distinguishing feature of this group is the rapid, rhythmic, coördinated systoles of the auricle, the contractions usually occurring at a rate between 250 and 300 per minute. The auricular rate is so rapid that the ventricle is unable to respond to each impulse so that the ventricular rate is always slower than the auricular. The abnormal activity may occur in short paroxysms lasting only a few minutes or may be continued for days or weeks. It seems quite probable that this peculiar activity differs essentially from that of auricular paroxysmal tachycardia only in respect to the rate of the auricular contractions; in paroxysmal auricular tachycardia the auricular rate usually does not exceed 250 per minute and the ventricles respond to each auricular stimulus; in auricular flutter the auricular rate is much faster and the ventricles are unable to respond to each auricular stimulus.

EXPERIMENTAL PRODUCTION

As early as 1887 MacWilliam* described the phenomena which result from the application of a weak faradic current to the exposed auricular wall as follows: "It sets the auricles into a rapid flutter . . . the movements are regular: they seem to consist in a series of contractions originating in the stimulated area and thence spread over the rest of the tissue. The movement does not show any distinct sign of incoördination: it looks like a rapid series of contraction waves passing over the auricular wall." Under these conditions the ventricular rate is accelerated but is usually one-half or less than one-half of the auricular rate. In a heart beating 140 to

*Jolly and Ritchie, Heart, 1910-11, ii, 77.

*Journ. of Physiology, 1887, viii, 296.

180 per minute such faradization may induce an auricular rate of 500 to 600 per minute while the ventricular rate may be 200 to 300 per minute. If faradization of the auricles is stopped the "auricular flutter" may continue for a considerable time and then the auricle may resume its physiological rate.

In the frog's heart "auricular flutter" lasting as long as two minutes, starting suddenly and terminating abruptly, may be induced by a single induction shock applied to the sinus† or some portion of the auricle.‡

While the auricles are in "flutter" vagus stimulation may change the flutter into a condition of "fibrillation" and slow the ventricle; it does not, however, slow the coördinated contractions of the auricle. It is possible, as suggested by Ritchie, that excessive stimulation of the accelerator nerves may be a factor in producing flutter in an otherwise healthy heart.

MECHANISM

Experimental and electrocardiographic evidence indicates that auricular flutter is characterized by a rapid rhythmic series of auricular contractions having their origin in some point of the auricular musculature other than the sinus node. Nearly all paroxysms of auricular flutter are preceded and followed by extrasystoles which interrupt the physiological rhythm more or less frequently; the extrasystoles are auricular in origin and probably arise in the wall of the upper chamber at a point which becomes the pacemaker for the paroxysm. That the irritability of this point is very great may be concluded from the great rapidity of the auricular systoles. The mechanism is the same as that of auricular paroxysmal tachycardia but in flutter the auricular rate is so great that the capacity of the bundle of His to convey stimuli is exceeded and the ventricle responds only to every second or third auricular impulse. In most cases the ventricular response is perfectly rhythmic and there is one ventricular contraction to two or three auricular contractions. Less commonly the ventricular contractions are arrhythmic and respond at one time to each second auricular impulse, at another time to each third or fourth impulse from the upper chamber.

†Lovén: Mitteilungen vom physiol. Laboratorium in Stockholm, 1886, iv, 16.
‡Engelmann: Arch. f. d. ges. Physiol., 1897, lxv, 109.

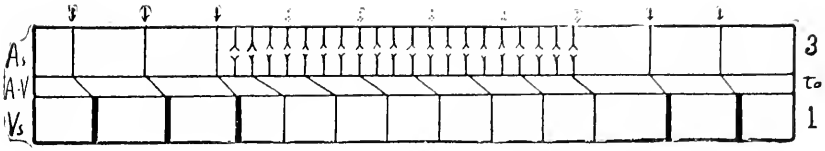

FIGURE 81

Paroxysm of auricular flutter. During the attack the auricles contract rhythmically. The ventricles contract rhythmically at a slower rate, the ventricle responds to every third auricular impulse.

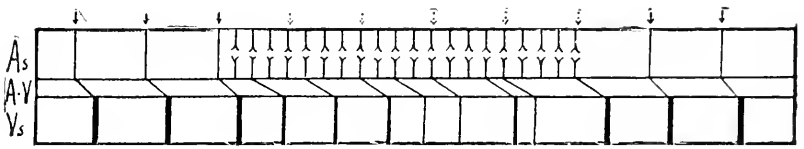

FIGURE 82

Paroxysm of auricular flutter with irregular ventricular response. Such an extreme grade of ventricular irregularity may simulate the activity of auricular fibrillation, or a lower grade of irregularity may be mistaken for extrasystoles. The ventricle responds to the first, second, third or fourth auricular impulse.

Diagrams to illustrate the mechanism of auricular flutter of different types. The arrows indicate the points of origin and direction taken by the stimuli. Dotted arrows indicate the time at which the normal stimuli at the sinus node should reach maturity if its formation were not interrupted by the abnormal impulse starting from a lower point in the auricle and traveling upward. The thickness of the lines representing ventricular systole indicate the relative effect of the several contractions in maintaining an adequate circulation. The obliquity of the *A-V* line indicates the varying length of the conduction time. *As* = auricular systole. *A-V* = conduction from the auricle to the ventricle. *Vs* = ventricular systole.

The activity may be regarded as an auricular tachycardia with a functional depression of the property of conduction in the *A-V* bundle. We conclude that a real depression of conduction exists because we know that in "paroxysmal tachycardia" the ventricle may respond to the auricular impulses at a rate above 230 per minute, yet in "auricular flutter" the rate of the lower chamber of the heart is usually not above 120; rarely it attains a rate of 160 per minute. Ritchie* has reported a patient with a ventricular rate at times under 40; in this case there was probably an organic lesion of the bundle of His.

Figure 81 shows in diagrammatic form the mechanism of a paroxysm in which the ventricle responds rhythmically to every third auricular impulse; during the attack the ventricular rate is accelerated but is only one-third the rate of the auricle. Each ventricular systole of the paroxysm is less forcible, since the property of contractility has not had the same time to recover as is permitted during the physiological rate. The exhaustion of the capacity of conduction in the *A-V* bundle, due to the abnormal shower of auricular impulses, is indicated by the obliquity of the line representing the period of the passage of the stimulus from the auricle to the ventricle.

In Figure 82 are plotted the auricular and ventricular activities of a paroxysm of flutter in which the ventricular response is very irregular; the lower chamber follows the first, second, third or fourth auricular impulse in a seemingly haphazard fashion. The conduction period is variable and prolonged. The ventricular contractions have a force proportional to the preceding diastolic period. The difficulty of differentiating such a mechanism from that of "auricular fibrillation" is apparent. If the ventricular response had been rhythmic up to the time of the final beats of the paroxysm, it is easy to see how the pulse and heart sounds might suggest the occurrence of an extrasystole only.

ETIOLOGY AND PATHOLOGY

The reported cases of auricular flutter indicate that it occurs considerably more often among men than women. It may occur at any age; the earliest subject which has been put on record was 5

*"Auricular Flutter," London, 1914, 36.

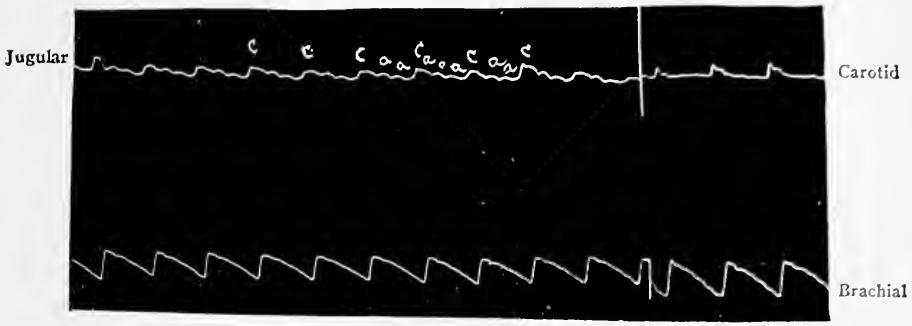

FIGURE 83

Auricular flutter with regular ventricular response. Auricular rate 276. Ventricular rate 92.

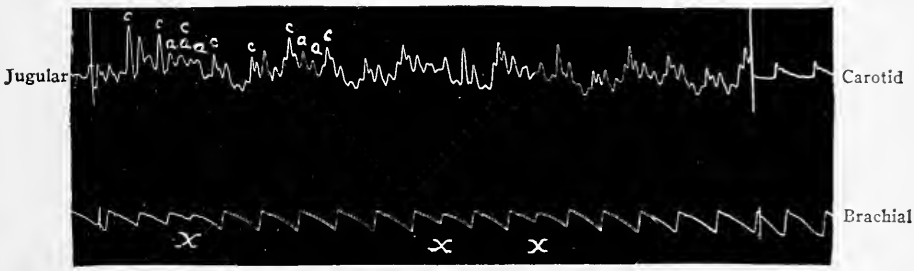

FIGURE 84

Auricular flutter with irregular ventricular response. Auricular rate 280. Ventricle usually responds to fourth auricular impulse, at X it responds to second auricular impulse.

years old. All the cases which I have observed, with one exception (14 years), have been over 50 years of age. Ritchie in his analysis of 49 cases found that 70 per cent. occurred after the fortieth year.

Auricular flutter rarely occurs without some other evidence of damage to the cardiac tissues; about a third of the cases show a defect of the mitral valve. Dilatation of the auricles is a common antecedent condition. Pericarditis has been present in several cases. General arteriosclerosis in which the coronaries have participated has been found in a number of instances.

The acute infections, such as diphtheria and rheumatic fever, have been the evident causative agent in about 20 per cent. of the cases thus far reported. Evidence of a syphilitic infection is obtained in at least 10 per cent.

It has been suggested that an abnormal balance of external nervous control may be an element in the production of auricular flutter, but no anatomical lesion which would indicate a removal of vagus influence or a hypertonic activity of the accelerators has thus far been demonstrated.

Such evidence as is at hand leads us to believe that this abnormal activity has its origin in a lesion in the auricular wall which constitutes a focus of increased irritability.

In the few post-mortems which have been reported, in those who have been the subjects of auricular flutter, histological examinations have failed to demonstrate a particular focus in the auricular wall to which one could ascribe the functional change, but general inflammatory and degenerative changes of the myocardium are not wanting. Dilatation of the auricles with fibrous, fatty or lymphocytic infiltration of the walls is the most common finding. Atheroma of the coronaries and calcareous deposits in the arterial wall suggesting an interference with the nutrition of the heart musculature have been found in several instances. These lesions frequently involve a large part of the heart muscle and may include the sinus node and *A-V* bundle. Ritchie (Case III) found changes in the sinus node consisting of lymphocytic infiltration. Hemorrhage and granular degeneration of the nodes are reported by Hume.* I have obtained autopsies in three cases, men of 51, 54 and 55 years, re-

*Heart, 1913-14, v, 25.

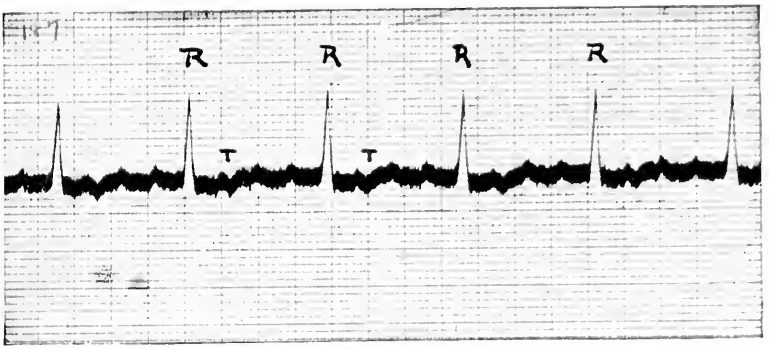

FIGURE 85
Patient H. S. Lead I. Auricular flutter. Time lines = 1/25 second.

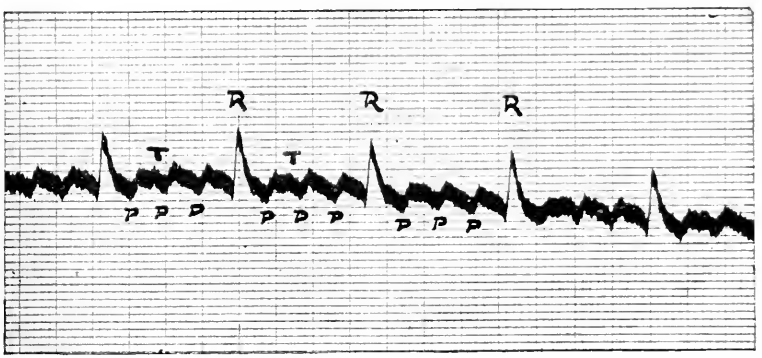

FIGURE 86
Patient H. S. Lead II. Auricular flutter. Time lines = 1 25 second.

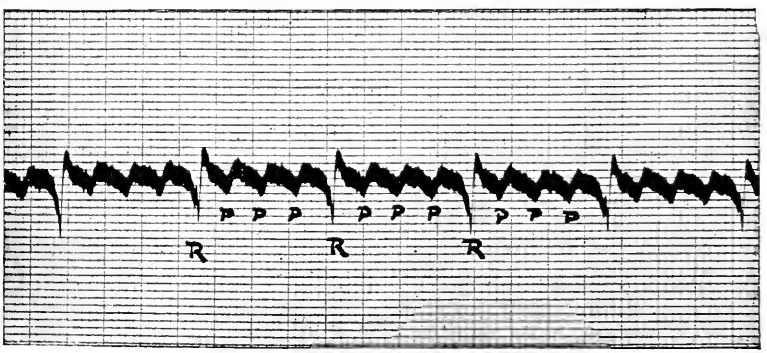

FIGURE 87
Patient H. S. Lead III. Auricular flutter. Time lines = 1 /25 second.
Figures 85, 86, and 87 taken from the same subject. Auricular rate 336 per minute. Regular ventricular response to every fourth auricular impulse, ventricular rate 84.

spectively. Each showed sclerosis of the coronaries and extensive fibrous myocarditis; in each very little normal heart muscle could be found. Each had an old infarct of the left ventricular wall.

IDENTIFICATION

A careful history and physical examination may lead us to suspect "auricular flutter," but one can only be sure of the correctness of the diagnosis when fortified by the evidence of graphic records. The pulsation of the veins of the neck gives us certain information in regard to the activity of the right auricle, a very rapid rhythmic pulsation of the jugular vein, showing a continuous series of waves at absolutely equal time intervals and two or three times as rapid as the ventricular rate, as determined by auscultation, suggests an auricular flutter, but it is quite evident that by mere inspection it is most difficult to count and correctly determine the spacing of the small venous waves. In cases of established auricular flutter I have repeatedly tried to elicit auscultatory evidence of the rapid auricular activity with complete failure.

The ventricular contractions may be perfectly rhythmic and so accelerated that one may suspect a true "paroxysmal tachycardia" (see Chapter IX). As a rule, in "auricular flutter" the ventricular activity is less rhythmic and not as fast as is the case in "paroxysmal tachycardia." The irregular ventricular activity of "flutter" is most often mistaken for the far more common disturbance known as "auricular fibrillation" (see Chapter XI). In most cases of "auricular flutter" the arrhythmia is not as great as in "auricular fibrillation" and in the latter the ventricular form of the venous pulse may aid in distinguishing the two conditions; however, without the assistance of graphic records the separation of these groups is practically impossible.

When the ventricular rate is only 40 or less and perfectly rhythmic, one at once suspects a condition of heart block. If in such a case the jugulars are pulsating rhythmically at a rate of 200 or more per minute, one can be reasonably sure that a condition of "auricular flutter" coexists.

There are certain types of irregular ventricular response when the auricle is in flutter which simulate forms of extrasystolic activity. For example, if for considerable periods there is a ventricu-

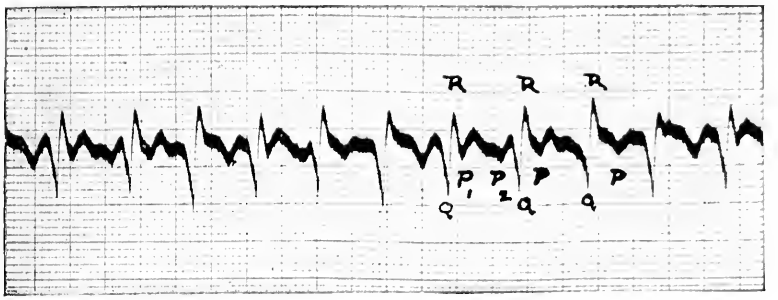

FIGURE 88

Auricular flutter. Lead I. Auricular rate 332. Ventricular rate 166. As:Vs::2:1. Time = 1/25 second.

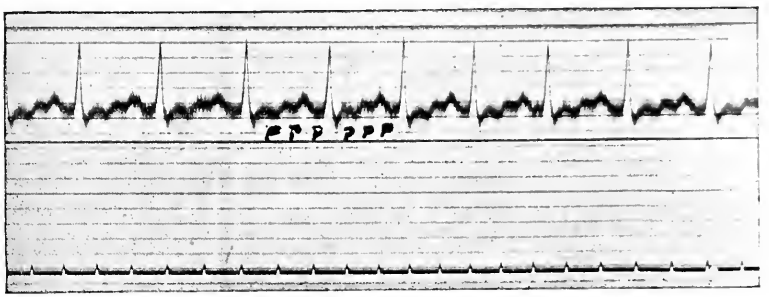

FIGURE 89

Auricular flutter. Lead II. As:Vs::4:1. As = 492. Vs = 123. Time = 0.2 second.

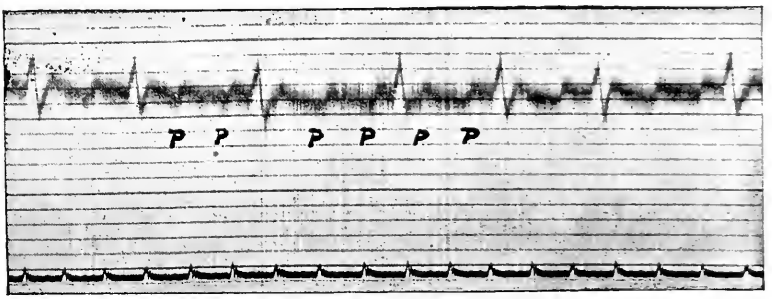

FIGURE 90

Auricular flutter with irregular ventricular response. Lead III. As = 280. Time = 0.2 second.

lar response to every third auricular impulse and this established rhythm is broken by a ventricular response to the second auricular impulse, which is in turn followed by a ventricular contraction after four auricular systoles, the early beat and the succeeding pause may give one the impression of an extrasystole with a compensatory pause (see Figure 82).

The *polygram* is often of material aid in making a diagnosis of auricular flutter and the jugular tracing may demonstrate the rapid rhythmic activity of the right auricle. The analysis of the jugular curve is, however, often obscure, since the record of the waves of auricular activity is distorted by the *c* and *v* waves characteristic of the normal venous tracing. We should bear in mind that the *a*, *c*, *v* and *h* waves of the normal jugular pulse do not follow one another at exactly equal intervals of time, and when we can detect in the jugular record such a rhythmic series of waves two, three or four times as rapid as the ventricular rate, we have strong grounds for suspecting a condition of auricular tachycardia.

Figure 83 was secured from a case of "auricular flutter" in which there were regularly three auricular contractions to one ventricular. The ventricular rate was 92, the auricular rate 276 per minute. One of the *a* waves of each cycle is simultaneous with the *c* wave. The ventricle contracts in perfect rhythm.

A type of irregular ventricular response is shown in Figure 84. The jugular record is composed of a rhythmic series of *a* waves at a rate of 280 per minute, which can be picked out by careful measurement; the pure auricular record is distorted by *c* and *v* waves of each cycle and the whole is superimposed on the respiratory curve. The ventricle is contracting at a rate of 102 per minute; the ventricle usually responds to the fourth auricular impulse, but occasionally (at *X*) it responds to the second auricular impulse. This type of irregular ventricular response would strongly suggest occasional auricular extrasystoles were it not for the evidence obtained from the jugular tracing. The analysis of both of these polygrams was verified by electrocardiographic records taken at the same time.

The *electrocardiogram* must be our final court of appeal in substantiating a diagnosis of "auricular flutter." Even here the evidence is sometimes obscure, and it is wise to have records taken by the three standard leads in order to be certain of our interpretation.

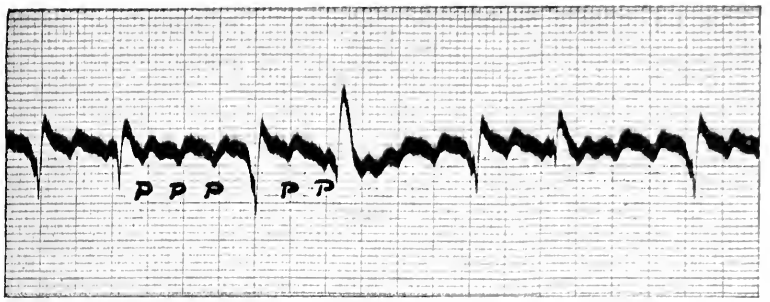

FIGURE 91

Auricular flutter. Lead III. Irregular ventricular response. One ventricular extrasystole. Time = 1/25 second.

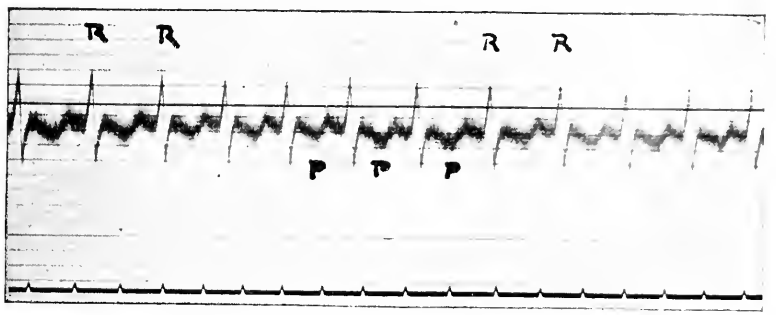

FIGURE 92

Auricular flutter. Patient H. Auricular rate 388. Regular ventricular response rate 194. Time = 0.2 second.

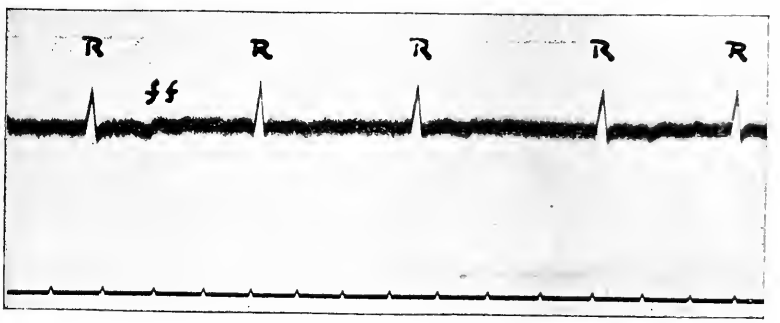

FIGURE 93

Auricular fibrillation. Taken from same patient (H.) as Figure 92, but 15 days later. Time = 0.2 second.

Figures 85 (lead I), 86 (lead II) and 87 (lead III) were taken from the same patient at intervals of about one minute and indicate the differences in the records secured by different derivations. Usually the analysis is most easily made from leads II and III, but this is not always the case. In these records the ventricle is beating rhythmically at a rate of 84 per minute; the auricle is contracting at a rate of 336 per minute. One of the auricular (P) waves of each cycle is submerged in the R defection of the ventricular cycle. The T wave of the ventricular complex is evident only in leads I and II as a slight distortion of the rhythmically recurring P waves.

In Figures 88, 89, 90 and 91 are shown four records from four distinct cases of auricular flutter indicating the variations which such a group of cases may present.

In Figure 88 is reproduced a curve taken from a patient by lead I. In this instance the ventricular rate is 166; the auricular rate is 332 per minute. The ventricular and auricular complexes are in part superimposed so that the analysis at first glance seems obscure; by the aid of records taken by leads II and III (not reproduced) we could clearly establish a rhythmic rapid activity of the auricle at double the rate of the ventricle. The question arises in this case as to which of the auricular stimuli excites the activity of the lower chamber. We cannot answer this question positively but we have strong evidence for presuming that the *earlier* of the auricular stimuli (P_1) is the one to which the succeeding ventricular contraction is the response. If the response was to the stimulus delivered at P_2 the conduction time (P_2-Q) would be abnormally short. While it is not inconceivable that in certain cases the property of conduction may be heightened, all our experience goes to show that in those cases of auricular flutter in which we have positive evidence, conduction is normal or depressed (usually the latter). It is never demonstrably shortened, hence we are led to believe that in every case the conduction time is longer than the normal and therefore in the instance shown in Figure 88 it is probable that the ventricle responds to P_1 rather than to P_2.

A case in which the lower chamber response follows four auricular contractions is depicted in Figure 89. The ventricular rate is 123; the auricular rate is 492. Both chambers contract rhythmically, but the auricle four times as often as the ventricle.

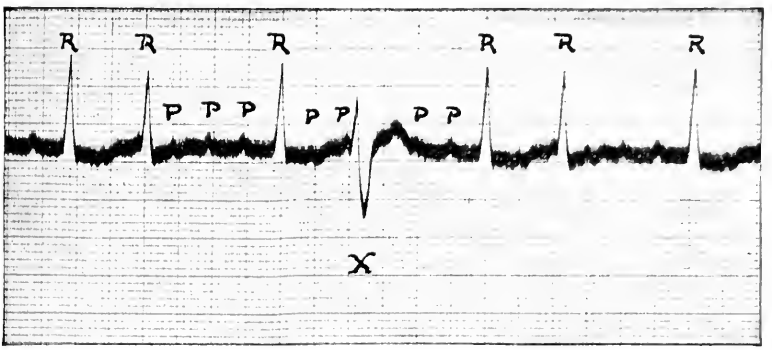

FIGURE 94

Patient K. Auricular flutter with irregular ventricular response and ventricular extrasystole. Time 1/25 second.

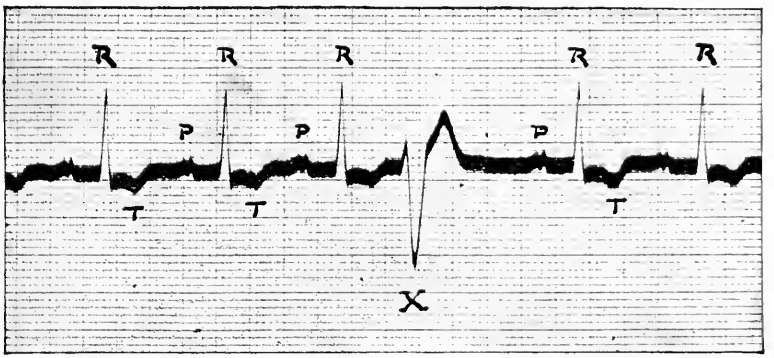

FIGURE 95

Patient K. Sequential rhythm taken from same subject as Figure 94. Taken 11 days later. Note same type of ventricular extrasystole. Time 1.25 second.

Irregular ventricular responses are shown in Figures 90 and 91. In Figure 90 the auricle is beating rhythmically 280 times per minute; the ventricle responds to every second or third auricular stimulus.

In Figure 91 the response is to the second, third or fourth auricular stimulus. This record is further complicated by an unusual complex indicating one ventricular beat having its origin in a point in the ventricular wall quite different from the other ventricular contractions, which are of supraventricular origin.

A record from a case of "flutter" with a very rapid rhythmic response is represented in Figure 92. The auricular rate is 388; every other auricular complex is submerged in a ventricular complex which occurs 194 times per minute. The ratio of the rate of the upper to the lower chambers is as 2 is to 1. A record from the same case (Figure 93) taken 15 days later, after the patient had taken digitalis, shows complete irregularity and a rate of 46 per minute. There are at this time no coördinated contractions of the auricle, but its activity is one of "fibrillation."

CLINICAL COURSE AND SIGNIFICANCE

Auricular flutter is occasionally the only evidence obtainable of a defective myocardium, though quite commonly extrasystoles precede and follow the paroxysms. In such patients careful examination fails to reveal any organic change in the valves, endocardium or pericardium, and the only evidences of functional disturbance are those elicited during the paroxysm. During the attack, which comes on abruptly and terminates suddenly, the patient may be very uncomfortable. He is conscious of an unusual commotion in the chest; the accelerated and irregular activity of the ventricle may be the cause of considerable apprehension; this may be accompanied by some dyspnœa, precordial distress and prostration if the paroxysm is prolonged. Some attacks may extend over days or even weeks, the earlier alarm and dyspnœa may subside, and the patient may resume his usual occupation aware only of the continuing "palpitation."

In most cases there are other evidences of myocardial damage and the "auricular flutter" throws an additional load on a heart

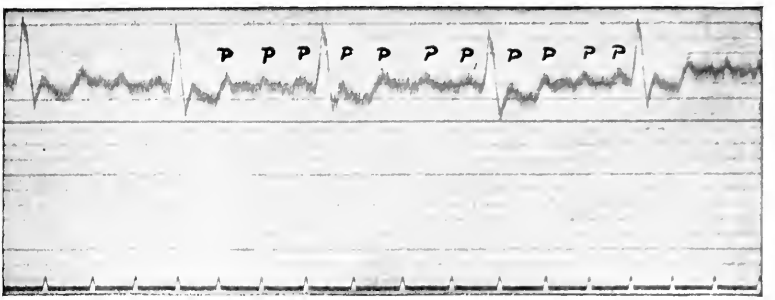

FIGURE 96

Patient M. K. March 11, 1912. Auricular flutter. Time 0.2 second.

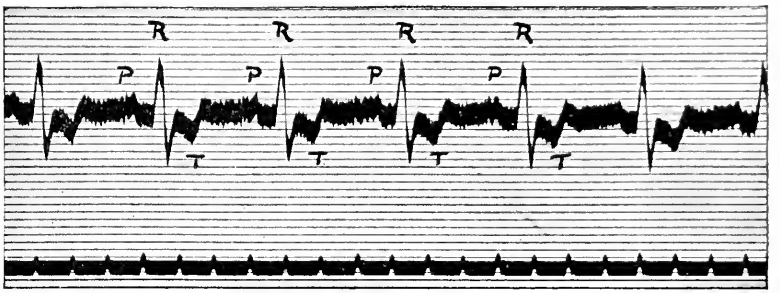

FIGURE 97

Patient M. K. December 12, 1914. Sequential rhythm. Time 0.2 second.

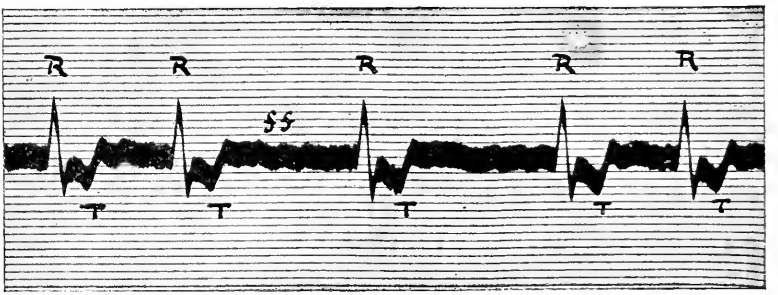

FIGURE 98

Patient M. K. December 28, 1914. Auricular fibrillation. Figures 96, 97 and 98 are taken from the same subject, the first of these was taken during an attack of pneumonia 2 1/2 years before the subsequent records.

already overtaxed. In such patients the general signs of cardiac insufficiency may have been present before the onset of the auricular acceleration, or the unusual stress occasioned by the new rhythm may be too much for a heart barely able to preserve an adequate blood stream; its narrow margin of safety is quickly exhausted, and signs and symptoms of cardiac insufficiency rapidly appear. The extent and severity of the symptoms depend to a very large degree on the condition of the heart before the attack; the auricular flutter may last for days or weeks, yet ultimately the heart may recover a normal rhythm and perform its work with reasonable efficiency; or in a short time there may develop dyspnœa, congestion of the liver and lungs, edema of the extremities, Cheyne-Stokes respiration, giddiness, unconsciousness and collapse.

A patient may have many attacks of auricular flutter or it may appear only as a terminal event. Once established, the attacks are prone to recur and each one is apt to persist for a longer time. Occasionally one sees attacks of flutter alternating with periods of normal rhythm; more often "auricular flutter" passes into "auricular fibrillation," which may persist indefinitely or may, in turn, give way to a physiological rhythm. With a return to a normal rhythm the symptoms usually improve.

The tendency to resume a normal rhythm is seen in Figures 94 and 95, taken from the same patient at intervals of eleven days. Figure 94 shows auricular flutter at 300 per minute, with an irregular ventricular response interrupted at *X* by a ventricular extrasystole. In Figure 95 is seen the sequential rhythm of eleven days later interrupted by an extrasystole of the same type as that which occurred during the period of "flutter."

Figure 96 was taken from a patient during her first paroxysm of flutter, which had its onset during an attack of lobar pneumonia in March, 1912. In December, 1914, she returned to the hospital with broken cardiac compensation. Her record taken at that time (Figure 97) shows a sequential rhythm. A few days later she began to fibrillate (Figure 98) and has continued this condition up to the present time (6 months later).

The clinical significance of auricular flutter lies in the fact that it indicates a considerable degree of damage present in the auricular wall. That the damage may be temporarily repaired is indicated

by the recovery of normal rhythm, but the tendency to repeated and more severe attacks suggests that usually the repair is incomplete.

The welfare of the patient depends to a large degree on the condition of the ventricle. With a normal ventricular muscle the patient will withstand many attacks of "auricular tachycardia" with comparative immunity. With a damaged ventricle the outlook is much less propitious. Unfortunately the myocardial damage is rarely limited to the auricle. In "auricular flutter" a slow, regular response of the ventricle is favorable; a rapid, irregular ventricular response makes the outlook more serious. The change to a condition of auricular fibrillation and a slowing of the ventricle under digitalis are to be regarded as a favorable sequence of events. The return to a normal rhythm is to be welcomed but by no means assures complete recovery.

CHAPTER XI

Auricular Fibrillation

The group which we are now to consider is characterized by a "complete irregularity" of the pulse. Waves of varying sizes, large and small, separated by intervals long or short, follow each other in a confused succession. The pulse is absolutely devoid of rhythm. The closest study will not enable us to predict whether the next event in the series is to be a forcible, or a weak impulse, a pause short or long. We may have a series of small waves separated by unequal intervals occasionally mingled with large waves, or large and small waves and longer or shorter pauses may be jumbled together in the utmost confusion.

In the older literature this pulse has been described and labeled with many different names, *"pulsus arhythmicus," "deficiens," "inaequalis," "intermittens," "irregularis,"* it has been called the "mitral pulse." The pulse rate may be fast or slow, it may exceed 200, or may be under 40.

The heart shows the same degree of irregularity, indeed by auscultation the absolute irregularity is made more apparent than by palpating the radial. The heart activity is, I think, best described by the now discarded term *"delerium cordis."* The apex impulses are unevenly spaced, and forcible and weak thrusts are mixed in an erratic series.

The heart sounds show the same absence of rhythm both in time and force. They vary greatly in intensity. Each contraction may be represented by a first and second sound, or at more or less frequent intervals the first sound alone is audible suggesting that the feeble contraction has failed to open the aortic valves.

The credit of separating this type of disordered myocardial function from others belongs in a very large degree to Mackenzie,* who pointed out the constant association of the "ventricular form of venous pulse" and "complete irregularity" of the ventricle. At this time he ascribed these phenomena to a paralysis of the auricles. Later he amplified his views in a paper based on the study of 500

*Study of the Pulse, London, 1902.

cases.† In a series of subsequent papers he modified his theory of the underlying defective mechanism, and since he was led to believe that the auricle and ventricle contracted simultaneously, he assumed that a point near the *A-V* junction was the source of the impulse which simultaneously excited both the upper and the lower chambers. He further assumed that this abnormal pacemaker was located in the node of Tawara‡ and therefore introduced the term "nodal rhythm"§ to designate this group.

The real explanation of the activity of the completely irregular heart was made clear by the electrocardiographic studies of Rothberger and Winterberg¶ and of Thomas Lewis.‖ To these investigators, working independently, is jointly due the distinction of conclusively demonstrating that in these cases there is no gross auricular contraction, but that the auricular wall is in a continuous state of fibrillation. To those who are interested in the facts upon which their conclusions are based a study of the original papers is commended.

EXPERIMENTAL PRODUCTION AND MECHANISM

When the auricle of an exposed heart is faradized the coördinated contractions of the chambers as a whole cease, there is no auricular systole, and the muscle wall assumes the relaxed condition of diastole, the muscle mass is, however, not at rest, it manifests a continuous activity which consists of fine irregular waves with here and there a sharper twitching movement. The movement has been likened to the appearance of the squirming of a bunch of worms. This is what is known as *fibrillation of the auricle*. It is similar in appearance to the fine fibrillary movements which are not infrequently seen in the tongue. When such a condition is produced experimentally it is accompanied by a complete irregularity of the ventricle, a venous pulse of the ventricular form and an electro-

†Amer. Jour. Med. Sci., 1907, cxxxiv, 12.

‡Quart. Jour. of Med., 1907-8, i, 39.

§With our present clearer insight into the mechanism of the completely irregular pulse, the term "nodal rhythm" should not be used in this connection. It should be reserved to designate a rare but definite cardiac activity in which the pacemaker is located in the *A-V* junctional tissues.

¶Wien. klin. Wochenschr., 1909, xxii, 839.

‖Heart, 1909-10, i, 306.

cardiogram which shows a continuous series of small irregular deflections, which are met with in no other condition, and are undoubtedly due to the abnormal auricular activity.

Lewis studied 3 horses, each having complete irregularity, the electrocardiograms were similar to those obtained from men with complete irregularity. On quickly opening the chest the auricles could be seen in a condition of fibrillation, and the ventricles were contracting in the characteristic irregular manner. Cushney and Edmunds,* who were familiar with the experimental production of auricular fibrillation, in discussing a case of complete ventricular irregularity in man were the first to suggest that this phenomenon might be associated with a fibrillating auricle.

Auricular fibrillation has been produced experimentally by increasing the intra-auricular pressure (Lewis), by stimulating the vagus and accelerator nerves (Morat and Petzetakis) by the application of heat to the myocardium (Lagendorff) and by toxic doses of drugs, digitalis (Francois-Frank), nicotine (Pezzi and Clerc), pilocarpine (Busquet).

It has been suggested by Lewis† that fibrillation is due to a highly irritable condition of many points in the auricular musculature, each of which "is independently and spontaneously elaborating heterogenetic impulses," that is to say he views the condition as closely allied to the auricular extrasystole, but instead of there being one irritable point there are many, and the multiple impulses thus set free neutralize or reinforce one another in an utterly haphazard fashion.

Recently Garrey‡ has taken exception to this view, and substitutes the theory that the condition is due to many small blocks between the fibers of the auricular musculature, so that the fibers receive their stimuli not by the usual paths, but in such a manner that the stimuli pass slowly from cell to cell by a circuitous path, thus causing contraction waves in a shifting series of ring-like undulations.

There is little question that the ventricular contractions are the result of irregular impulses received from the auricle for the electrocardiographic ventricular complexes are usually of the form which

*Amer. Jour. Med. Sci., 1907, cxxxiii, 67.

†Mechanism of the Heart Beat, London, 1911, p. 192.

‡Am. Jour. Physiol., 1914, xxxiii, 397.

are secured from stimuli arising in a point above the level of the junctional tissues. There are several factors which may have an influence in determining the instant at which the ventricle shall respond: (a) it is conceivable that a stimulus is effective only when of a certain magnitude, and that such a size is only attained when a considerable number of minute auricular impulses bombard the junctional tissues at the same instant, (b) this constant shower of auricular stimuli may have a modifying effect on the excitability or the conductivity of the bundle, (c) the underlying disease which has caused the damage to the auricular musculature may not have left the *A-V* bundle unscathed.

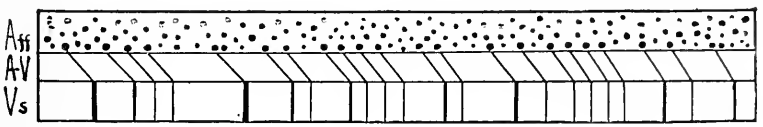

Figure 99

Diagram to illustrate our conception of the mechanism of auricular fibrillation. Aff dots of various sizes indicate stimuli of various magnitudes arising in many parts of the auricular wall neutralizing or reinforcing or blocking one another. When effective the impulse is transmitted through the junctional tissues (*A-V*) and the ventricle responds (*Vs*) at irregular intervals with a varying degree of force, roughly proportional to the length of the preceding diastolic period. It is to be noted that the auricle is in a state of continuous activity, but there is no coördinated contraction: also that the stimuli which call forth a ventricular contraction arises in a point above the ventricular tissues.

The peculiar susceptibility of cases of auricular fibrillation to stimulation† of the left vagus and the drugs of the digitalis group suggests a distinct change in the functional activity of the junctional tissues.

The force of the successive ventricular contractions depend mainly on the period of rest which precedes any particular contraction, thus permitting a more complete filling of the ventricle and a recovery of its contractile power, but as has been pointed out by Einthoven and Kortweg‡ this relationship is not always maintained, hence other factors must be at work which are not completely understood.

The accompanying diagram (Figure 99) portrays in a graphic manner the conception of the mechanism, the upper reaches of the junctional tissues are being continuously bombarded by a shower of

†Draper and Robinson: Jour. Exp. Medicine, 1911, xiv, 217.
‡Heart, 1915, vi, 107.

small impulses from the auricular segments; at irregular intervals these stimuli become effective, and a ventricular activity, irregular in time and force, is the result.

PATHOLOGY

Every heart showing fibrillation of the auricles which has been exhaustively examined has given evidence of gross or histological damage of its tissues, but as yet no lesion has been described to which we may definitely attribute the abnormal functional activity. Grossly one finds valvular defects, most frequently mitral stenosis, hypertrophy and dilatation, pericarditis and coronary sclerosis. Mackenzie early in his studies pointed out the frequency with which dilatation and hypertrophy of the auricles is met with in these cases. Histological examination almost invariably reveals evidence of acute or chronic inflammatory changes of the myocardium, leucocytic infiltration, or fibrosis and atrophy of the muscle cells. In several instances the blood supply of parts of the auricular wall has been found deficient. In some cases structural changes have been described in the sinus node or in the *A-V* node, in others these tissues have revealed nothing abnormal to the most thorough search.*

The recognized fact that fibrillation of the auricles may be only temporary suggest that in a certain number of cases, the cause may be a toxin, or a temporary nutritional disturbance.

Since it has been shown experimentally that over distension of the auricle may lead to fibrillation, it seems reasonable to suppose that an auricular wall damaged by disease, under the stress of a defective valve, or the demands of a general arteriosclerosis, may readily fall into fibrillation. This would explain the onset in a large number of the cases that are seen in the clinic.

ETIOLOGY

Auricular fibrillation is met with in all decades of life, but is extremely rare in those under ten years of age. In a personal experience with over 300 cases I have seen only one case under the age of ten. A curve of the age incidence (Figure 100) indicates that 89 per cent. of the cases occur between the ages of 21 and 60. The first

*Cohn: Heart, 1912-13, iv, 221.

sharp elevation occurs during the years when rheumatism is prevalent, the second elevation occurs in the decade when the rheumatic period overlaps the time at which arteriosclerotic changes become a prominent feature.

It is seen more frequently in men than in women, the proportion being about 2 to 1.

The association with mitral disease is very noticeable, 60 per cent. of the cases showed a mitral stenosis, with or without mitral incompetence, in 5 per cent. there was evidence of mitral insufficiency

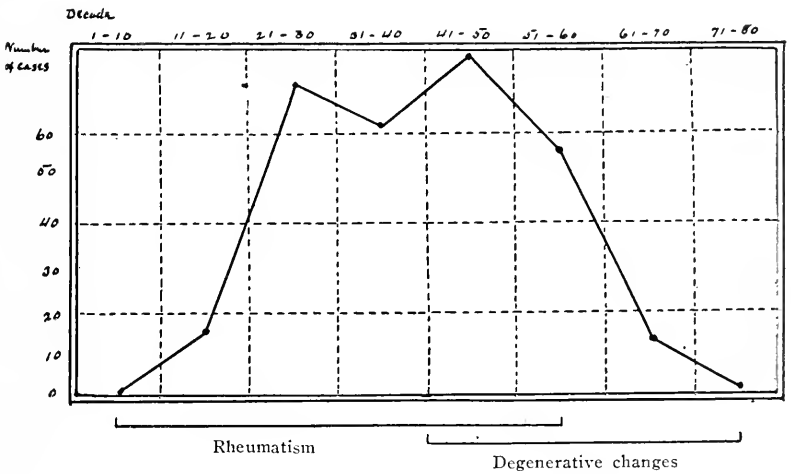

FIGURE 100

Age incidence of 300 cases of auricular fibrillation arranged by decades.

without stenosis. In a few cases there was an aortic lesion as well as a mitral defect. In only 7 instances have we seen fibrillation in hearts with defects of the aortic valves only.

Distinct evidence of one or more attacks of *acute rheumatic fever* was obtained in over one-half of the cases studied. The well-known frequency with which rheumatism affects the mitral valve is further evidence that this disease is the underlying cause of the myocardial defect. It is not, however, common to see auricular fibrillation during the first attack of rheumatic fever, the damaged heart does not usually go into fibrillation until some years after the evidence of a valvular defect has been established. This phase is further empha-

sized when we compare the age incidence of rheumatism and of fibrillation; according to Church* 57 per cent. of the mitral attacks of acute rheumatism occur under the age of 20. Our chart of the age incidence of fibrillation shows that in only 16 cases† did the onset occur before the twentieth year, and only 149 cases‡ showed fibrillation during the first four decades. This suggests that fibrillation points to a rather advanced degree of tissue damage. The stretching of the injured auricular muscle under the stress caused by a defective valve may well be an important factor in producing this type of abnormal function.

We have seen fibrillation develop during the course of a lobar pneumonia on six occasions.§

In several cases with mitral disease the original lesion could be attributed to an attack of scarlet fever.

Attacks of influenza seem to be the only etiological factor that could be obtained in some of the patients. The most careful scrutiny of a case now under observation has given no explanation of a well-marked mitral stenosis and auricular fibrillation other than repeated attacks of grippe.

We have seen fibrillation of the auricles in four cases of Graves' disease, two of these gave an old history of attacks of acute rheumatic fever and each presented evidence of a mitral stenosis.

Aside from the rheumatic cases, and those in which the myocardial damage may be attributed to one of the acute infections, there is a considerable group which includes those suffering from degenerative changes, such as general arteriosclerosis, chronic nephritis, emphysema, etc. This group is composed of patients who, for the most part, have passed their fortieth year, and in it males largely predominate.

Syphilis and alcohol appear to be prominent etiological factors in this group. Many of these have no discoverable evidence of valvular disease, a soft blowing systolic murmur in the mitral area is,

*System of Medicine: Allbutt and Rolleston; Vol. ii, Pt. 1, 603.

†8 per cent. of those having a rheumatic etiology.

‡77 per cent. of those of rheumatic origin.

§In 126 cases of pneumonia carefully studied by Dr. A. E. Cohn, fibrillation occurred in 12 cases (personal communication).

however, not an uncommon feature. Several had dilatation of the arch of the aorta, and insufficiency of the aortic valves.

Identification

Clinical. Palpation of the radial pulse in most instances is sufficient to permit us to assign this group of cases to their correct category. The complete irregularity is at once evident, the pulse beats are irregular both in time and force. As a rule the stronger beats follow the longer pauses, but the disorderly sequence of large and small waves separated by intervals long or short, allows us to make a very strong conjecture as to the kind of abnormal cardiac activity with which we have to deal. There are other cases in which the complete irregularity is not so easily detected by palpation. When the pulse volume is diminutive, when the pulse pressure is relatively small, the recognition of the unequal size of the successive waves may be quite difficult. In cases with a rate of over 160 the pulse waves may show very insignificant differences in size, and the time intervals may show variations only to be detected by the most careful measurements secured by instrumental means. There are certain patients with pulse rates between 70 and 80, and with the waves placed at such even intervals that an uncorroborated examination of the radial pulse is quite insufficient to classify it as one of complete irregularity.

Before going farther we should emphasize the point that while practically every case of "auricular fibrillation" is characterized by a pulse of "complete irregularity," there are other conditions which may also afford a "completely irregular" pulse which can only be differentiated by exact instrumental methods, while in nineteen out of twenty instances a "complete irregularity" of the pulse correctly indicates a condition of "auricular fibrillation," the twentieth may be the result of an entirely different heart activity. The types which may notably give rise to this confusion are certain "sinus arrhythmias," and cases with very frequent extrasystoles, particularly when they arise from several points in the heart wall (Figure 120), or are interpersed with short runs of paroxysmal tachycardia (Figures 79 and 80).

If the veins of the neck are prominent inspection will show that the jugular pulsations are synchronous with the apex beat, no pre-

systolic wave can be seen, the venous pulse is of the ventricular form.

Auscultation of the apex will corroborate the complete irregularity discovered in the pulse, and at times will make clear an irregularity which was not discovered when palpating the wrist. The sounds vary greatly in intensity and follow one another at unequal intervals. Often the heart beats are far more numerous than the arterial pulsations, some contractions are too feeble to perceptibly increase the lumen of the radial artery, and others fail even to open the aortic valve. These latter are represented by a first heart sound only, the second sound is wanting.

The question as to whether the aortic valves are or are not opened by any particular ventricular contraction depends on the relative pressures in the aorta and the left ventricle. If the diastolic period is very short, the contractile power of the ventricle will have had little time in which to accumulate, at the same time the pressure in the aorta will be relatively high. Under such conditions the valves will not be opened. On the other hand given a longer pause the next succeeding ventricular contraction will have recovered its contractility to a greater degree, the aortic pressure will be relatively low, and the aortic valves will be opened.

In some cases where the degree of irregularity is moderate, it may be difficult to distinguish this from a condition of extrasystole. It is helpful in such cases while listening to the heart beats to concentrate the attention on the beats which occasionally occur too early, and which simulate the premature beat of the extrasystole, the diastolic period following these early contractions of complete irregularity usually do not have the same length, nor as a rule do they have as long a duration as that which would constitute the "compensatory pause" of an extrasystole, and thus may be differentiated.

If valvular defects are present murmurs are usually detected, but at times their character is quite different from those heard before the inception of the complete irregularity. If the heart is beating very rapidly the murmurs are less intense, and the sharp snap of the first sound, so characteristic of mitral stenosis, may become muffled. The intensity of the murmurs vary

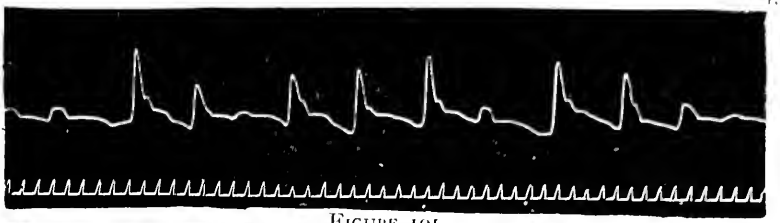

FIGURE 101

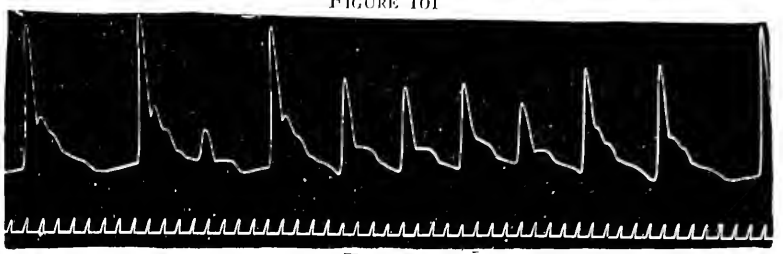

FIGURE 102

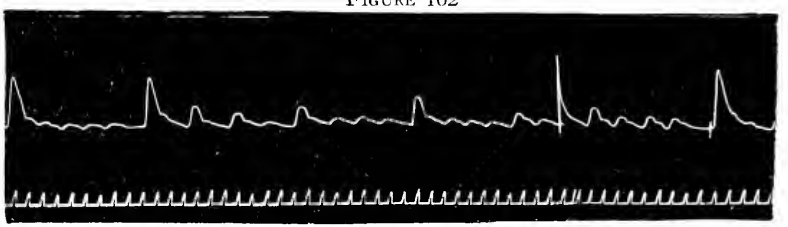

FIGURE 103

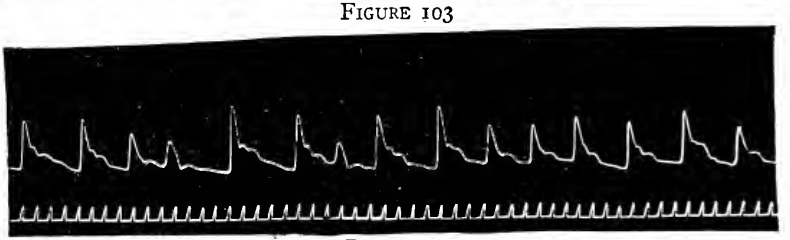

FIGURE 104

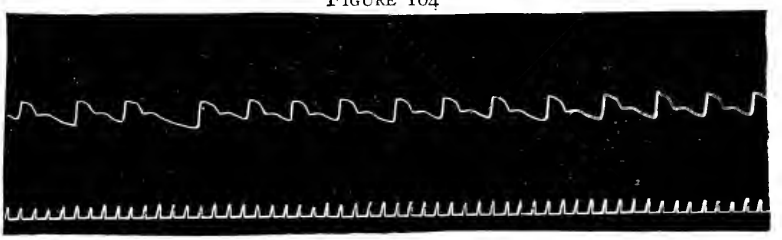

FIGURE 105

Arterial tracings from cases of auricular fibrillation showing the great variety of pulses which are seen. All of these records show complete irregularity.

from cycle to cycle. The more forcible beats are accompanied by relatively loud and harsh murmurs, the more frequent feeble contractions by murmurs of less conspicuous intensity.

There is frequently an increase in the intensity and duration of the mitral diastolic murmur. It may also assume a rougher quality. This may be explained on the basis of the increased volume of blood under considerable pressure in the right auricle, which is possibly distended and is never emptied by an effective contraction.

For the most part, the time relations of the murmurs remain as before the change to complete irregularity, systolic and diastolic murmurs continue to occupy their established position in cardiac cycle. Mackenzie long ago pointed out one exception to this general rule, namely, the disappearance of an established presystolic murmur in cases of mitral stenosis which changed from a physiological rhythm to one of complete irregularity. His explanation of this phenomenon assumed that the presystolic murmur of mitral stenosis was due to auricular systole, and that the failure of the auricular contraction accounted for the disappearance of this murmur. While this phenomenon is frequently seen it is not invariable as has been evidenced both by the ordinary methods of eliciting physical signs* and by occasional graphic records of the heart sounds which may be found in the literature. Authorities are not at all agreed that this presystolic murmur is due to auricular systole, and the persistence of the murmur in certain cases of undoubted auricular fibrillation lends support to the view that other explanations of the mechanism of the production of this murmur may not be disregarded.

The character of the complete irregularity of the arterial waves are clearly brought out by taking a tracing of one of the peripheral arteries (radial, brachial or carotid), and by means of a simple graphic record of this kind (see Figures 101, 102, 103, 104 and 105) one can hardly miss a correct diagnosis.

The great variation in the size and time intervals of the arterial pulse waves is shown in Figures 101, 102 and 103. In Figures 101 and 102 the waves are for the most part of large volume,

*Hart, Med. Rec., New York, 1911, lxxx, 2.

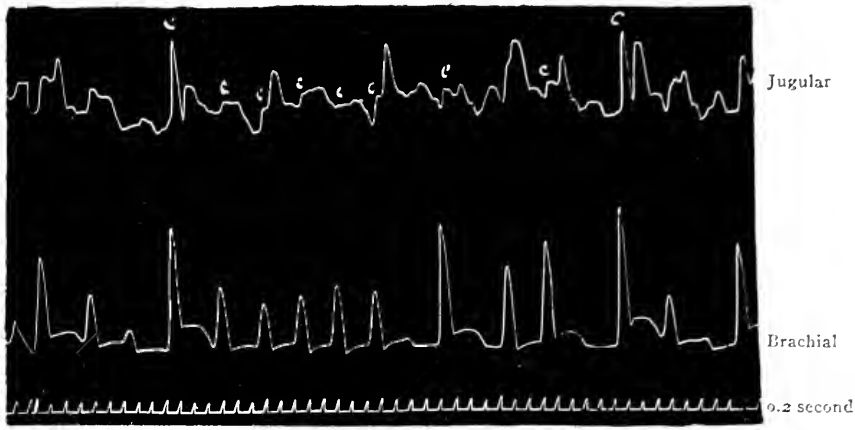

FIGURE 106

Auricular fibrillation. Great variation in the size of the arterial waves and the intervals between them. Jugular completely irregular, *a* absent, *c* and *v* waves fused. Depression *x* absent.

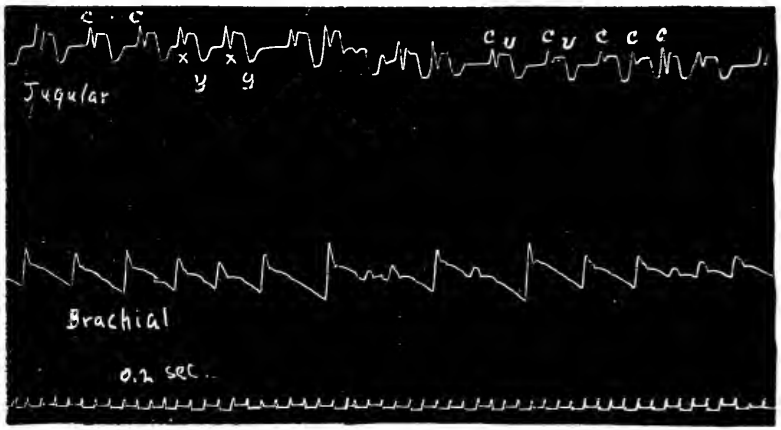

FIGURE 107

Auricular fibrillation. At certain places the form of the arterial record might suggest an extrasystole. In the jugular record note the "ventricular form" of the venous pulse, the *a* wave is absent.

but show considerable differences in force and time intervals. Figure 103 shows a brachial pulse rate of 138. Many of the smaller waves could not be detected by palpation of the radial artery, hence it is easily seen how a count of the radial alone would have been very misleading in determining the rate of the heart beat. Figures 104 and 105 are both taken from cases of auricular fibrillation. They indicate an unusual degree of rhythmicity, but careful measurements will show that the uniformity is apparent rather than real. In both of these records there are waves which a casual observer might mistake for extrasystoles, but close attention reveals not only a lack of uniformity in what one might take to be waves of the physiological rhythm, but further a period of very inconstant length both before and after the small waves which thus differentiate them from the intervals which one finds on either side of the extrasystole as commonly observed.

Polygrams make the diagnosis still more clear (see Figures 106, 107, 108, 109 and 110). In these records the arterial curves show in considerable variety the features that have already been dwelt upon. In addition to this, the jugular reveals features which are characteristic of this form of irregularity. It is itself completely irregular. The time intervals between the waves indicate a complete absence of rhythmicity. The size of the waves show great variations, which are quite independent of the phase of respiration in which they occur. Furthermore, the venous pulse is of the "ventricular form," that is to say all the positive waves occur during the ventricular systolic period, the *a* wave, the representative in the normal jugular of auricular systole is absent, *c* and *v* waves may be made out, the *c* wave usually varying in size proportional to the synchronous ventricular contraction. The *v* wave is usually a large broad elevation which frequently begins earlier than the *v* wave of the physiological rhythm and may be fused with the preceding *c* wave (Figures 106 and 109). The explanation of this phenomenon is found in the distended condition of the auricle which is probably never effectually emptied, hence the ventricular pressure is more readily transmitted by way of this blood column to the large veins. This feature may be further emphasized by a relatively insufficient tricuspid valve. The *v* wave is promptly terminated by the opening of the tri-

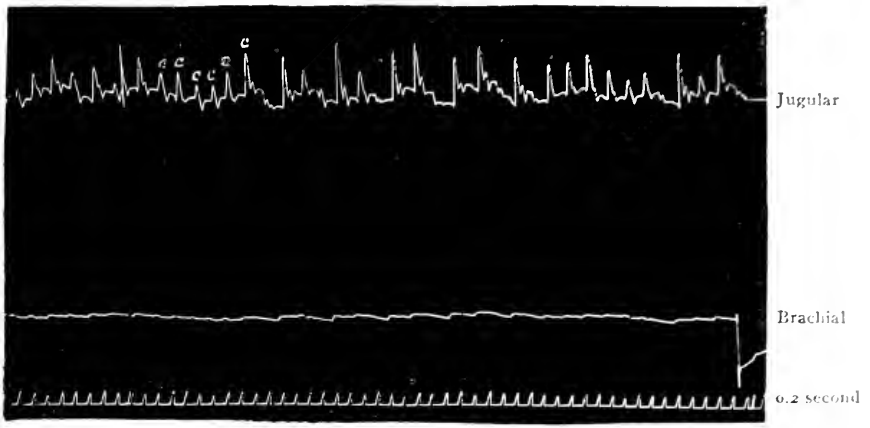

FIGURE 108

Auricular fibrillation with a very rapid ventricular response. From a case of general arteriosclerosis with high blood pressure.

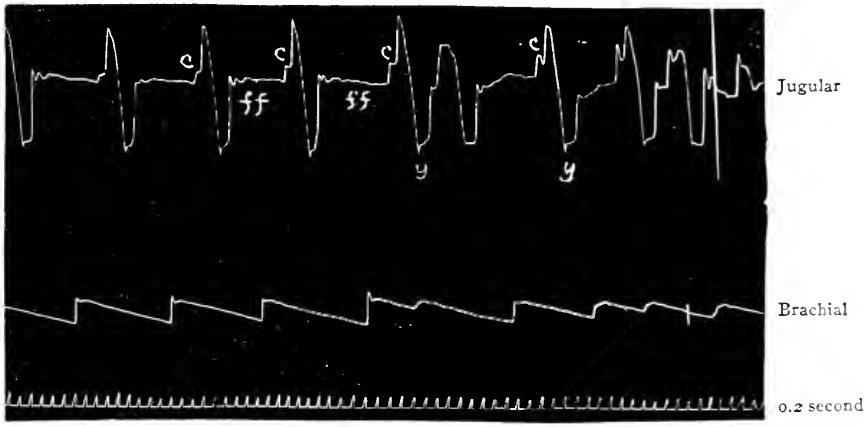

FIGURE 109

Auricular fibrillation with slow ventricular response. Note absence of *a* wave, absence of depression *x*, unusual depth of *y*, small oscillations during diastole (*ff*).

cuspid valve, and the discharge of blood under abnormal pressure in the auricle into the ventricle. An examination of the several polygraphic records indicates the variety in the form and time of the beginning of the *v* waves. As a general rule the cases of auricular fibrillation of long standing with overdistended auricles and veins show a tendency for the *v* wave to become fused with the *c* wave, and the depression *x* may entirely disappear. The unusually deep depression *y* seen in Figure 109 indicates an unusual fall of pressure when on the opening of the tricuspid valves the contents of the distended auricle are poured into a relaxed and dilated ventricle.

Figure 108 illustrates the kind of curves often secured from the cases of auricular fibrillation with a very rapid ventricular response in old arteriosclerotic cases, the pressure changes in the brachial and radial are often quite small, and it is difficult to obtain good arterial tracings.

A slow ventricular rate is recorded in Figure 109. The brachial waves are well marked and extremely irregular at a rate of 60 per minute. In the jugular tracing there are seen during diastole some very small waves marked *ff*. These minute fluctuations in venous pressure are not infrequently obtained in auricular fibrillation when the ventricular response is deliberate, they have been ascribed to the undulating auricular activity, but it is quite probable that they are due to vibrations induced in the vein by the pressure of the cup used as a receiver.

Synchronous records of the apex beat and carotid are reproduced in Figure 110. At *x* are seen weak ventricular contractions, which are barely forceful enough to overcome aortic pressure, and presumably would be too weak to be detected in the radial.

The *electrocardiogram* usually merely corroborates the evidence of auricular fibrillation obtained by the simpler methods of examination, but in certain obscure cases the electrical records are essential to a correct diagnosis. We have seen cases that have been carefully studied by skilful clinicians in which the irregularity was mistakenly interpreted as due to auricular fibrillation, and under vigorous treatment the patients grew worse rather than better. The galvanometric records disclosed the fact that

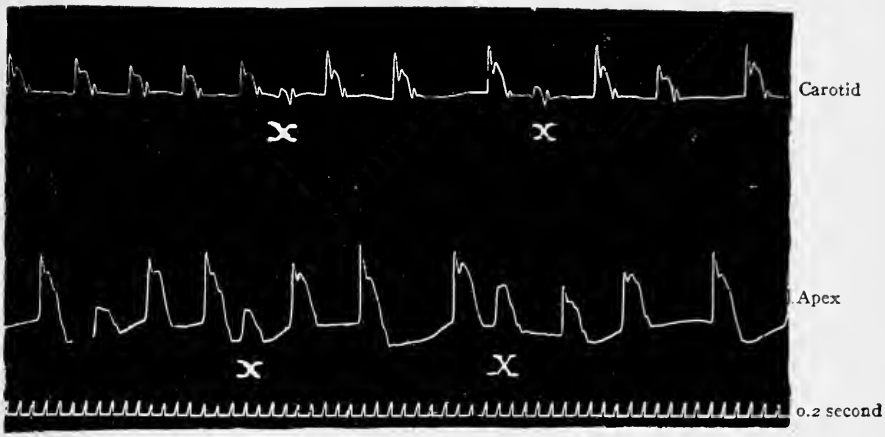

FIGURE 110

Auricular fibrillation. Apex and carotid tracings. At *x* the weak ventricular contractions are barely able to open the aortic valves.

these "complete irregularities" were not caused by "auricular fibrillation," and a proper revision of the treatment resulted in immediate benefit to the patient.

The distinctive features of the electrocardiagram of auricular fibrillation are:

1. Complete irregularity in the time intervals between the ventricular complexes.

2. An absence of the *P* wave.

3. A series of small waves which are continuous throughout the whole cardiac cycle.

If one examines a number of electrocardiograms from cases of auricular fibrillation such as are shown in Figures 111, 112, 117, etc., the first thing that arrests our attention is the unequal spacing of the ventricular complexes. Some show this in a greater degree than others, but an absolute rhythmicity is almost* never seen. The parallelism between the irregularity of the ventricle and the arterial pulse is shown in Figure 111 in which simultaneous curves of the galvanometer and the pressures from a cuff placed on the brachial are recorded.

If we study the ventricular complexes carefully we are led to the conclusion that they have a normal form save for some distortion produced by the small waves of auricular activity which are present during the whole cardiac cycle, both systole and diastole. The ventricular complex may or may not be introduced by a *Q* deflection, both the ascending and descending limbs of the *R* wave are sharp and abrupt, *S* may or may not appear. *T* is usually present, the curve which is the result of the contraction of the lower chamber has the same characters and is of the same duration as is found in the same heart when the rhythm is of the physiological type. The records shown in Figures 121 and 122 were taken from the same patient, Figure 121, in June, 1910, when the auricles were contracting efficiently. Figure 122 was taken 10 months later, when the auricles were in fibrillation. A comparison of the ventricular portions of the two records show a marked similarity, the *R* and *T* deflections are of the same order, and of the same duration. We must therefore conclude

*An exception to this general statement is seen in cases of auricular fibrillation complicated by an *A-V* block. See Chapter XV.

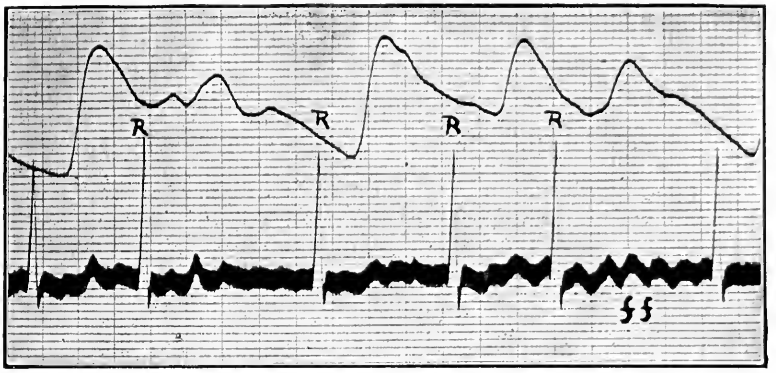

FIGURE 111

Auricular fibrillation. Note the complete irregularity in both time and force of the brachial waves. The ventricular complexes of the electrocardiogram show the same irregularity, the *P* wave is absent, the small oscillations (ff) vary greatly in size and duration, but are present throughout the whole cardiac cycle.

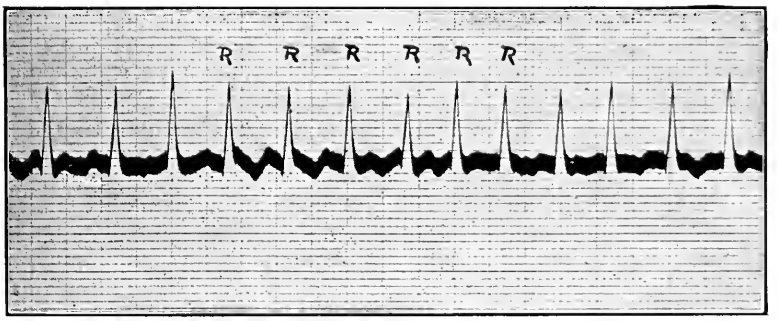

FIGURE 112

Auricular fibrillation with rapid ventricular response. *R* varies in height according as it coincides with the summit of one of the small waves or a depression between two small waves.

that even when the auricles are in a state of fibrillation the ventricles receive their stimuli from a point above the bundle of His, and that the stimulus travels over the ventricle by a path that is normal in all respects. The ventricle responds to a supraventricular impulse.

The second feature of importance in establishing the nature of the cardiac activity from a study of these records is the absence of the *P* wave, the normal representative of gross auricular contraction. In certain records (Figures 117, 123 and 127) one might at first glance question whether in some of the cycles a wave preceding the *R* deflections was not in reality a *P*, but an examination of a number of cycles will soon convince one that the inconstancy in the size, contour and time relation to the beginning of ventricular activity, make it necessary to find another interpretation.

In the electrocardiogram of the normal heart it may be recalled that in the period from *T* to *P*, the diastolic interval, there is a line without any suggestion of deflection. In the records now under discussion (Figures 111, 112, 113, etc.) this period is evidently occupied by a continuous series of uneven oscillations. These are the representatives of the rapid, irregular fibrillary activity of the auricles. Further examination will convince one that these small deflections are not limited to the diastolic period, but are continued during systole as well, so that the whole curve is modified by an unending series of these small fluctuations. It is for this reason that in many of the records the ventricular complexes seem to be of abnormal form. They are merely distorted by the superimposition of these representatives of unceasing auricular activity. The *T* wave being relatively small is frequently very much changed in appearance. The *R* deflection when large is only slightly modified, but will fluctuate in height (Figures 111 and 112) according as it is coincident with a summit of, or a depression between the small waves. The oscillations are usually arrhythmic, and vary in frequency. When the rate can be estimated it will be found to be between 400 and 600 per minute: they are usually most conspicuous in records taken by the second and third leads.

The great variety in the rate, size and rhythm of the waves

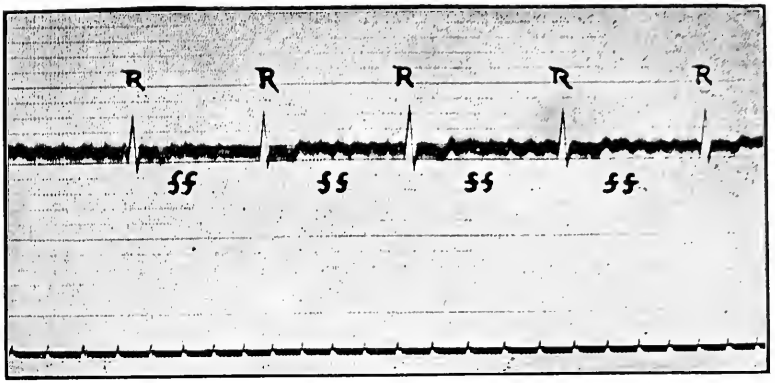

FIGURE 113

Auricular fibrillation with ventricular response which is almost rhythmic. The auricular oscillations are small, but distinct, constant and quite uniform in size.

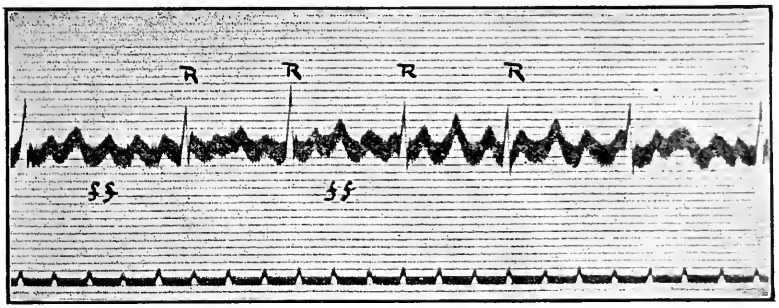

FIGURE 114

Very large waves of auricular activity which distort the ventricular complex to a marked degree.

of fibrillary activity as shown in the electrocardiogram indicates great differences in the algebraic sum of the electrical potentials developed from moment to moment in the same auricle, and in different auricles. The oscillations may be small and fairly rhythmic throughout the whole record, as seen in Figure 113, or large and very arrhythmic (Figure 114). Some curves show a great variation from cycle to cycle (Figures 111 and 112), others small fluctuations which are inconspicuous but quite constant (Figures 115 and 125). One may at times be confused by small rapid oscillations due to tremor of the skeletal muscles, when during the recording period the extremities are held tense and unrelaxed, these fluctuations are very fine and much more rapid than the waves of auricular fibrillation. They may be seen in Figures 116, 117 and 118, taken from the same patient on successive days before, during and after a paroxysm of auricular fibrillation. These muscular tremors are met with in patients under excitement who make a voluntary effort to hold themselves quiet. If the rapidity of the oscillations are noted they should not be confounded with the waves of auricular fibrillation which have a much slower period of vibration.

In cases such as are shown in Figures 113, 114 and 115, the arrythmia of the ventricles is only moderate, and it is plain that palpation of the radial or auscultation of the heart might fail to reveal the "complete irregularity." The graphic records, however, make clear the nature of the abnormal activity. The ventricle is seen to be definitely arrhythmic, *P* waves are absent, and the continuous series of oscillations, large or small, are seen during the whole cycle. In Figure 115 a simultaneous jugular curve has been recorded. In this may be noted the absence of the *a* waves, the *c* and *v* waves participating in the ventricular arrhythmia, and an absence of the depression *x* due to the early onset of the *v* wave, the fine oscillations *ff* are also apparent, but as has been pointed out in an earlier paragraph these are probably not characteristic of the fibrillating auricle, but are due to a venous thrill induced by the pressure of the cup used as a receiver.

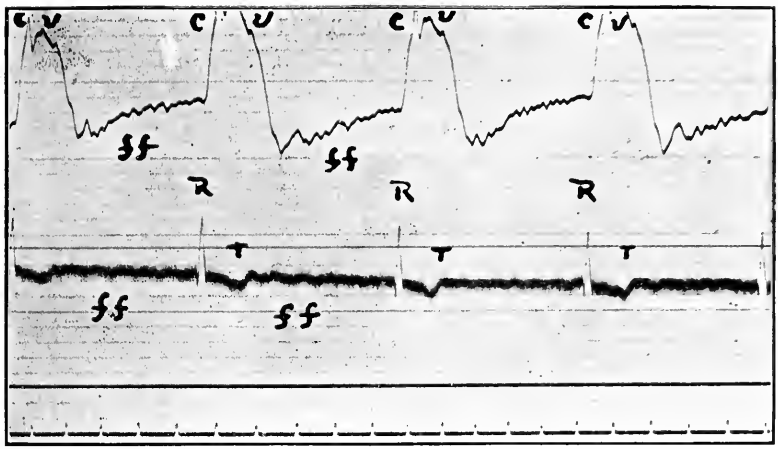

FIGURE 115

From a case of auricular fibrillation with a slow ventricular response. The form of the ventricular complex indicates that the stimulus which called it forth originated in the supraventricular tissues. *P* is absent. *R* and *T* are but slightly distorted by the superimposed oscillations of auricular activity (*ff*). Upper curve obtained from the right jugular vein, *a* is absent, *c* and *v* have coalesced.

CLINICAL FEATURES

Practically all patients with auricular fibrillation suffer from some degree of cardiac insufficiency. The functional disability of the heart may show a very wide range in different patients, and in the same patient at different times it may vary from a condition of almost complete heart failure to one in which there is ordinarily no evidence of an inability on the part of the heart to maintain an adequate circulation, and in which symptoms of insufficiency develop only under unusual stress. The degree of circulatory embarrassment depends in a very large measure on the integrity of the ventricular muscle. The valvular defect which so often has preceded the development of fibrillation has thrown upon the ventricles abnormal work, and perhaps injury which may, however, have been completely met by a compensatory hypertrophy. More often the underlying disease which has damaged both valves and auricular walls has also attacked the ventricles, leaving them an easy prey to the stress which the inception of the new rhythm imposes. If therefore the ventricular myocardium is extensively damaged one may expect the rapid development of the classical symptoms of cardiac insufficiency, lowered arterial pressure, increased venous pressure, slow capillary flow, œdema, cyanosis, dyspnœa, congestion of the lungs, liver, kidneys, etc., etc. On the other hand with a reasonably healthy ventricular muscle the strain occasioned by the disorder in the upper chambers may be supported with comparatively little evidence of abnormal blood distribution.

Fibrillation once established usually persists to the end of life, hence the term *pulsus irregularis perpetuus,* which was formerly used to describe the arterial features. However, one sees cases from time to time in which the normal pacemaker reasserts its control, and a physiological rhythm is regained. This has occurred in 6 per cent. of the cases which have come under my observation. Such a case is shown in Figures 116, 117 and 118, taken on successive days, April 19, 20 and 21, 1911. The records of the 19th and 21st show a sequential rhythm, on the 20th (Figure 117) the auricles were in fibrillation.

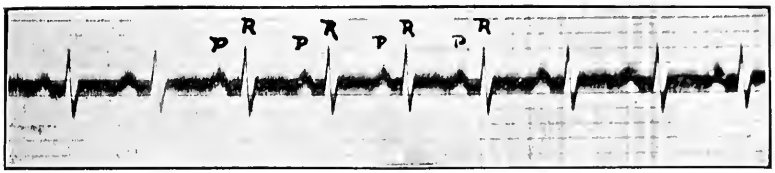

FIGURE 116

The two following figures were obtained from the same patient on successive days. April 19, 1911. The rhythm is sequential. The heart is rhythmic, the auricle has its normal activity, as indicated by the regular appearance of the *P* deflection.

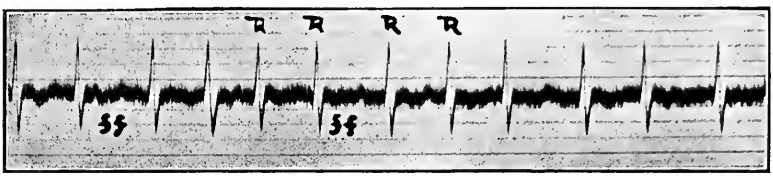

FIGURE 117

Same patient as Figures 116 and 118. Obtained April 20, 1911. At this time the auricle was temporarily fibrillating. Note the similarity in the form of the ventricular compleves in these three records. In this curve *P* is absent, there is complete irregularity, the small deflections (*ff*) due to the fibrillating auricle are present.

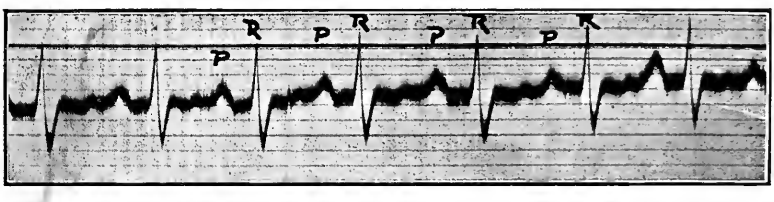

FIGURE 118

Obtained from the same patient as Figures 116 and 117, on April 21, 1911. The auricle has resumed coördinated contractions and the rhythm is again sequential. In all three of the above records note the fine rapid oscillations which are due to a tremor of the skeletal muscles.

Under "paroxysmal tachycardia" reference was made to an unusual form of paroxysm consisting of a short run of auricular fibrillation, with a rapid irregular ventricular response interrupting an ordinary sequential rhythm. Such a paroxysm is shown in Figure 119. The earlier portion of the record shows a number of cycles during which the sinus node is evidently the pacemaker. The rhythm is broken at *A* by an auricular extrasystole; this is followed by a normal cycle, then a bizarre curve which it is difficult to classify, but which may possibly represent an abnormal ventricular contraction and two ectopic auricular contractions; these, in turn, are succeeded by a short period of auricular fibrillation with a rapid irregular ventricular response. The paroxysm of fibrillation lasted for about a minute and the sequential rhythm, broken by occasional auricular extrasystoles, reappeared. Patients showing this condition are very few in number, and in my experience it is very unusual to secure the graphic evidence of the transition from the sequential rhythm to the paroxysm of auricular fibrillation. This curve was obtained from a patient seen in consultation by Professor Longcope and was recorded by my assistant, Dr. Strong.

These paroxysms may appear over a period of many years. A case reported by Cushny and Edmunds had attacks for twenty years. Robinson* has reported careful studies in a case which had probably suffered from attacks for twelve years.

The transitory character of the paroxysms has been somewhat difficult to explain. A number of patients in whom I have observed this phenomenon were suffering from lobar pneumonia; here it is quite likely that the toxins of the disease had a direct action on the muscle cell. It has been observed in hearts which showed no other clinical evidence of a pathological lesion. Mackenzie† has suggested that digitalis may be a possible factor in causing the auricular fibrillation. Robinson has advanced the hypothesis that in certain cases the paroxysm may be due to an alteration in the blood supply to the auricular musculature. He also reports a case‡ which was directly attributed to poisoning with hydrogen sulphid.

*Arch. Int. Med., 1914, xiii, 208.

†Heart, 1910-11, ii, 295.

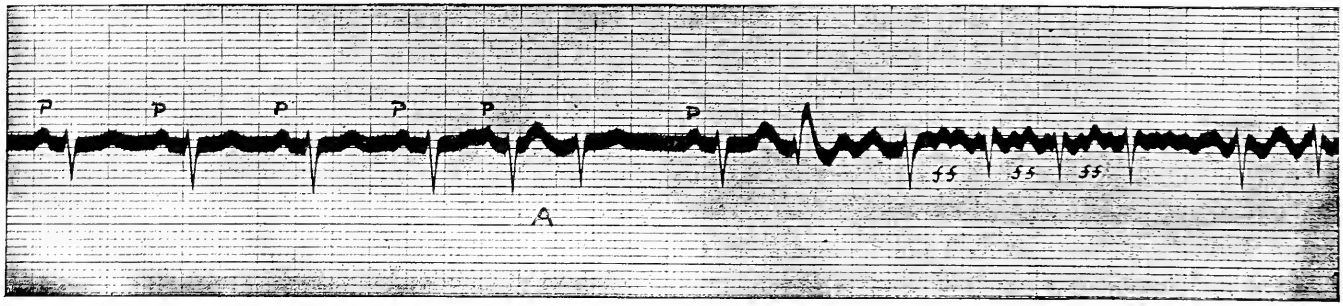

FIGURE 119

Transition from a sequential rhythm to a paroxysm of auricular fibrillation. Auricular extrasystole at *A*. The record was taken by lead III.

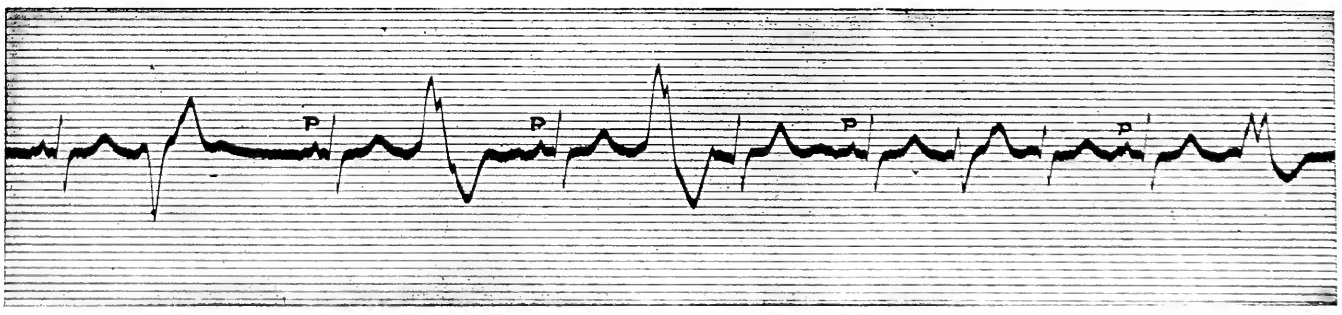

FIGURE 120

Extreme irregularity caused by extrasystoles arising from several points of origin in the heart muscle. The difficulty of distinguishing such an irregularity from auricular fibrillation, without the aid of graphic records is apparent.

The patient is not necessarily conscious of the irregular heart action. At times they apply for examination on account of the sensation of "fluttering" or "thumping" in the precordial region or complain of "palpitation." More frequently dyspnœa or œdema are the symptoms which bring them to the physician, and the erratic and tumultuous heart activity has passed unnoticed. Very few of them complain of anginal pains, but tenderness in the precordial region is a symptom frequently elicited during the examination with the finger, or the bell of the stethoscope. In cases which have been watched over a long period, one will often see the following series of events: (1) Several attacks of rheumatic fever with arthritis and endocarditis, most frequently involving the mitral valve. (2) Later extrasystoles, often auricular in origin. (3) The heart becomes gradually dilated under physical stress, and the auricles begin to fibrillate, and usually continue this activity up to the time of death, which may be postponed for many years.

Such a sequence of events is shown in Figures 121, 122 and 123. The patient, a man of 50, had a sharp attack of rheumatic fever, followed by endocarditis, when he was 20 years of age. At that time he was seen by Dr. E. G. Janeway, who told him he had a mitral stenosis. I first saw him in 1909, when he became conscious of an irregularity of the heart. At that time the rhythm was sequential, but he had occasional extrasystoles, which were verified by polygraphic tracings. On June 7, 1910, the curve shown in Figure 121 was obtained. It shows an extrasystole of auricular origin, and a split *P* wave. During an attack of pneumonia, in February, 1911, his auricles began to fibrillate. Figure 122 was obtained on April 28, 1911, and Figure 123 secured April 9, 1915. This has continued up to the present time (July, 1916). He is now in excellent health, and is able to conduct a business involving large responsibility, but slight physical exertion.

The rate of the heart beat in auricular fibrillation is extremely variable. One sees cases in which the rate does not exceed 40 a minute, and others which exceed 200. Under appropriate treatment it is not unusual to see a rate of 140 reduced to 100 in a very short time.

‡Jour. Amer. Med. Assn., 1916, lxvi, 1611.

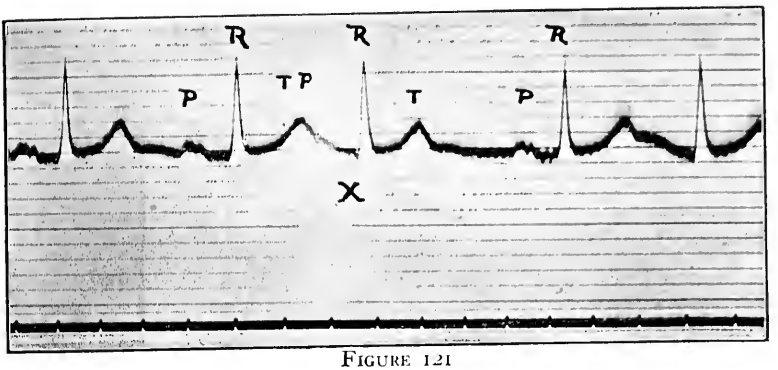

FIGURE 121

Patient B. S., June 7, 1910. The auricle is contracting as a whole. *P* is broad and split. Extrasystole of auricular origin at *x*. Note fine tremor of skeletal muscles.

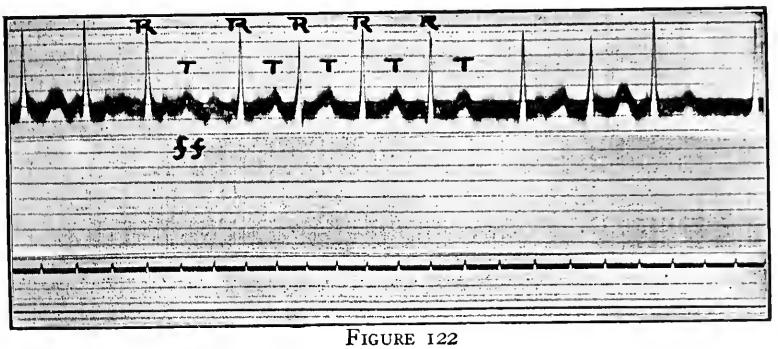

FIGURE 122

Patient B. S., April 28, 1911. The auricles are now fibrillating, the rhythm is rapid and very irregular.

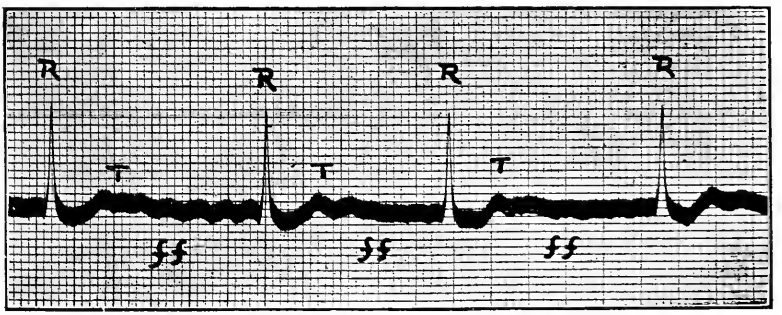

FIGURE 123

Patient B. S., April 9, 1915. Figures 121, 122 and 123 are all from the same subject. The auricles are still fibrillating, but the ventricular response is much less rapid and more evenly spaced.

The palpation of the radial pulse is a very insufficient criterion of the condition of the circulation. While the palpation of the pulse alone may be sufficient to establish a diagnosis, and while by this method one immediately detects the complete irregularity in force and frequency which characterize this group, the count of the radial pulse may be misleading. Frequently the number of the impulses which can be counted at the wrist is far below the actual number of cardiac contractions. Only those waves which are of considerable volume and force can be felt at the wrist (see Figure 125), and many small ventricular contractions expend their force before reaching the radial, and some even fail to open the aortic valves. These small contractions are ineffectual in maintaining an adequate circulation, yet are exhausting to the heart muscle, for we know that, in accordance with the law discovered by Bowditch, every contraction of heart muscle is maximal, that is to say, if it contracts at all, it exhausts all of the energy stored as contractile material in its muscle fibers at any particular moment; hence it is evident that however small a contraction may be, it must be taken into consideration in estimating the gravity of the condition of any particular heart.

The inadequacy of the observations on the radial pulse alone is well illustrated in Figure 124. Here the lower margin of the shaded area indicates the radial count. If one were guided by this alone, one would have said that on admission the cardiac rate was under 70 and never above 100. The upper boundary of the shaded area is the count taken by auscultation at the apex, and represents much more accurately the true condition, the admission rate being 127; the gradual reduction to the neighborhood of 60 makes the real improvement apparent.

The term *"pulsus deficiens"* has for a long time been used in describing pulse phenomena (Traube, Hering, Wenckebach, etc.), but each author has used it with a different meaning; some have considered it synonymous with *"pulsus intermittens,"* others have applied it to an absence of ventricular contraction, which breaks the ordinary rhythm; it has been used in describing extrasystoles and pulse alternans.

By *"pulse deficit"* we mean the difference between the number of cardiac contractions and the number of impulses which can be palpated in the radial artery. The best way of determining the pulse

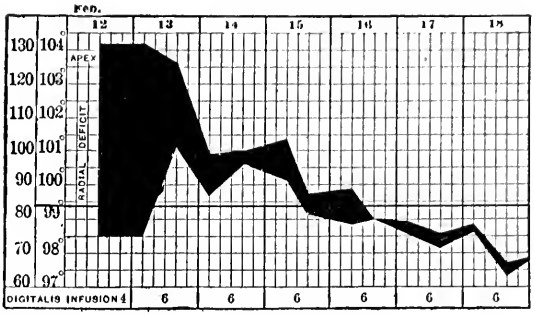

FIGURE 124

The shaded area represents the pulse deficit; the upper edge is the apex rate; the lower edge is the radial rate. Figures in digitalis column indicate dosage of the infusion in drams per twenty-four hours. Patient in bed during period represented in the figure.

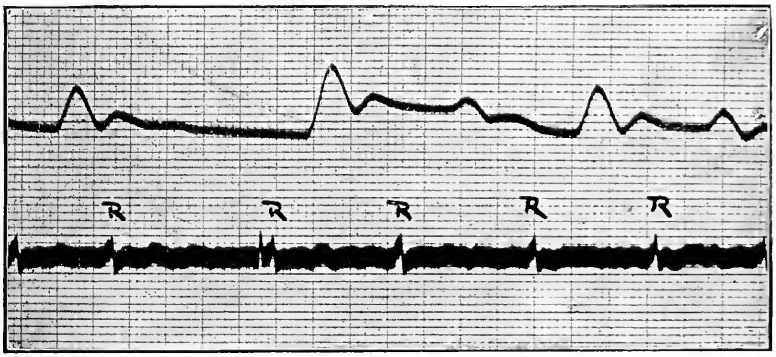

FIGURE 125

Auricular fibrillation. Simultaneous records of the cardiogram and the brachial pulse. Showing the mechanism of the "pulse deficit." Every other heart beat has little or no effect in raising the pressure in the brachial artery.

deficit is to have the apex counted by auscultation by one observer, while another is simultaneously counting the radial (these observations must cover a period of not less than a full minute, on account of the extreme irregularity of the pulse in many of these cases a count of one-quarter or one-half minute only is much less accurate). When one is obliged to make these observations unaided, the apex and the radial counts may be made in successive minutes. This of course does not give an absolutely accurate deficit, but it is extraordinary how closely the counts of successive minutes will coincide even when the heart and radial show the most extreme degrees of irregularity. After a little practice one may be able simultaneously to auscultate the apex and palpate the radial, thus determining the number of beats which fail to reach the wrist in the period of a minute.

In Figures 124 and 126 the upper margin of the shaded area represents the apex count, the lower margin the radial count, and the width of the shaded area represents the deficit at any particular point in the curve.

The relative deficit. In many cases of auricular fibrillation, particularly where improvement has occurred and the heart has become less irregular, slow, and fairly compensated, it will be found that the count at the apex and the radial are identical. Even in these cases the individual waves show a considerable variation in force and size. This is brought more clearly to view if the cuff of a blood-pressure apparatus is placed on the arm and the radial is counted while varying degrees of pressure are applied through the cuff. This difference in the pressure values of successive waves we have termed the "relative deficit," as contrasted with the absolute deficit, when without brachial pressure some waves fail to reach the radial.

The following observation will serve to illustrate this point: The patient was a gentleman who had fibrillating auricles for something over two years, with at times an apex rate of 160 and an absolute deficit of over 50. Later his heart was fairly compensated, and he was able to supervise large business interests which required a daily attendance at his office of six to eight hours. When we last saw him

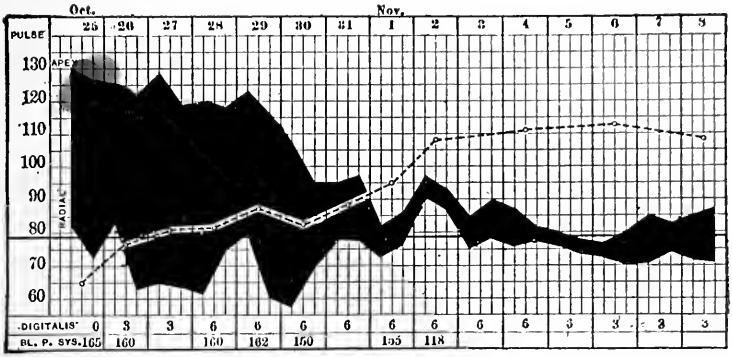

FIGURE 126

The shaded area represents the pulse deficit; the upper edge is the apex rate, the lower edge the radial rate. The broken line indicates the "average systolic blood-pressure" (compare these values with the figures at the bottom of the chart, which show the systolic blood pressure determined by the usual method). Figures in digitalis column indicate drams of the infusion per twenty-four hours.

his apex rate was 64, radial rate 64, deficit 0. On applying the brachial cuff the following counts were obtained:

Brachial pressure	Radial count per minute
140 mm. Hg.	0
130 mm.	50
120 mm.	58
110 mm.	62
100 mm.	64

While he had no absolute deficit, his relative deficit was quite evident when the pressure values of the waves of one minute were thus studied. This relative deficit may be detected in all cases of auricular fibrillation; it is rarely seen in other cardiac arrhythmias. We have found that such observations on the relative deficit are of real diagnostic value in corroborating a condition of auricular fibrillation which palpation and auscultation have led us to suspect.

The usual way of estimating blood pressure is entirely fallacious in auricular fibrillation. The accustomed method of obliterating the brachial artery by cuff-pressure and then by gradually lowering the pressure to determine the systolic blood-pressure by the height of the mercury column at which the pulse wave below the cuff is detected by palpation or auscultation is obviously of little value when practically each pulse wave has a different pressure value.

If such a pulse is observed for a period of a minute it will be found that only a small fraction of the total number of cardiac contractions have a pressure value, approximating the systolic blood-pressure as determined by this method. Figure 126 illustrates the inaccuracy of this method in these cases. Systolic blood-pressure taken by the usual method would signify that the successive blood-pressures of this patient were 165, 160, 160, 162, 156, 155, 148, etc., and that as her condition improved the blood-pressure was lowered. As a matter of fact, only a few beats could be detected below the cuff when exerting a pressure at these levels, and while doubtless her systolic blood-pressure was momentarily at these levels, they in no way indicate the efficient pressure of the blood column. As we shall show later, her systolic blood-pressure really increased with the improvement in her condition.

A much more valuable estimate of the force of the blood-stream

can be obtained by estimating the blood-pressure by another method; *the average systolic blood-pressure.*

To obtain what for convenience we have termed "the average systolic blood-pressure," the apex and radial are counted for one minute, then a blood-pressure cuff is applied to the arm, and the pressure raised until the radial pulse is completely obliterated; the pressure is then lowered 10 mm., and held at this point for one minute, while the radial pulse is counted; the pressure is again lowered 10 mm., and a second radial count is made; this count is repeated at intervals of 10 mm. lowered pressure until the cuff-pressure is insufficient to cut off any of the radial waves (between each estimation the pressure on the arm should be lowered to zero). From the figures thus obtained the average systolic blood-pressure is calculated by multiplying the number of radial beats by the pressures under which they came through, adding together these products and dividing their sum by the number of apex-beats per minute, the resulting figure is what we have called the "average systolic blood-pressure." The following two observations made on a patient will indicate the method of computation:

B.S., April 29, 1910. Apex, 131; radial, 101; deficit, 30.

Brachial pressure.	Radial count.	
100 mm. Hg.	0	
90 mm.	13	$13 \times 90 = 1170$
80 mm.	$47 - 13 = 34$	$\times 80 = 2720$
70 mm.	$75 - 47 = 28$	$\times 70 = 1960$
60 mm.	$82 - 75 = 7$	$\times 60 = 420$
50 mm.	$101 - 82 = 19$	$\times 50 = 950$
		Apex = 131) 7220

Average systolic blood-pressure 55 plus

B.S., May 11, 1910. Apex, 79; radial, 72; deficit, 7.

Brachial pressure.	Radial count.	
120 mm. Hg.	0	
110 mm.	44	$44 \times 110 = 4840$
100 mm.	$64 - 44 = 20$	$\times 100 = 2000$
90 mm.	$72 - 64 = 8$	$\times 90 = 720$
		Apex = 79) 7560

Average systolic blood-pressure 95 plus

The estimation of blood-pressure by this method gives us a simple and approximate measure of this factor of the heart's work. The diastolic pressure may be roughly determined by taking a graphic record with the Erlanger or Uskoff instruments and noting the pressure at which the average excursion of the pulse waves is maximal.

Examined by this method, the two groups, rheumatic and arteriosclerotic, show very different features.

In the rheumatic-mitral stenosis group, one not infrequently sees an average systolic blood-pressure under 70 mm. of mercury; these are usually cases with rapid rates (over 140) and a marked degree of cardiac insufficiency. With improvement the blood-pressure rises and may reach 120 mm., rarely 140.

In the arteriosclerotic group with fair compensation the average systolic blood-pressure is usually 160 mm. or over, and only falls below this when insufficiency becomes evident; in this group when the pressure, estimated in the above manner, falls below 140 mm. myocardial failure is very threatening.

The irregular activity of the ventricle is unquestionably the result of the peculiar activity of the auricles; while the upper chambers exhibit no gross contractions their walls show an incessant activity composed of irregular incoördinated contractions of the muscle fibers; impulses from the auricles are showered upon the junctional tissues in an entirely haphazard fashion, and the ventricles respond in utter confusion devoid of rhythm, and with extreme variation in the force of the succession beats.

In certain cases the junctional tissues between auricles and ventricles is somewhat damaged so that conduction between the chambers is depressed and fewer auricular impulses can reach the ventricles (Figure 113); under these conditions the ventricular rate is not excessive, and the contractions show more uniformity in force. Here one is impressed with the small amount of disability which results from the fibrillating auricle; it is little more than a venous reservoir, and takes little part in moving the blood, and if the irregular impulses which are initiated in its wall do not disturb the ventricle too greatly, the circulation is maintained with a reasonable degree of efficiency. The fibrillary activity may be continued for years, and in itself is not at all incompatible with life or a proper distribution of the blood to the various organs.

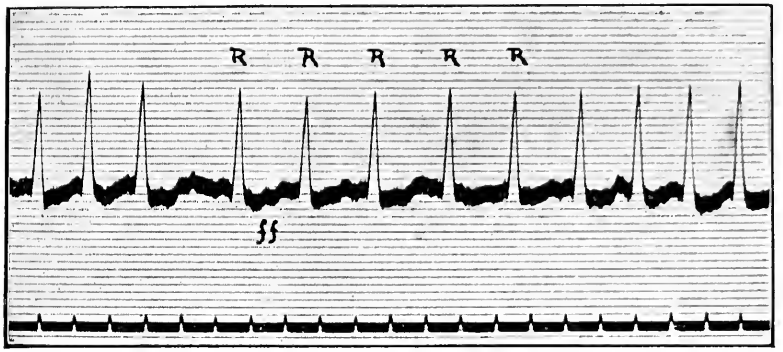

FIGURE 127

Auricular fibrillation. Patient C. F. Heart very rapid and irregular (see Figure 128).

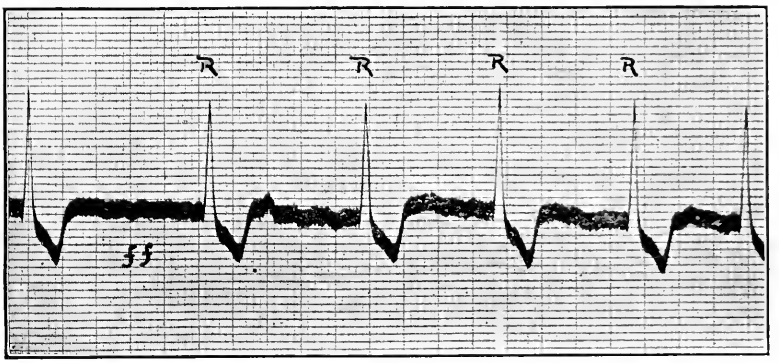

FIGURE 128

Auricular fibrillation, patient C. F., taken one week after Figure 127. The auricles are still fibrillating, but the heart has become slow under appropriate treatment. This is a favorable response to therapeutic measures, hence in this case the prognosis is good.

PROGNOSIS

This depends largely on two factors; first the integrity of the ventricular muscle, and second the facility with which the shower of impulses coming from the auricle can be blocked. With a weak, dilated ventricle showing irritability as evidenced by frequent ventricular extrasystoles arising from points in the ventricular tissue other than the *A-V* bundle, the outlook is bad, and yet under skillful treatment and favorable conditions, such a heart may occasionally recover a considerable degree of efficiency, and life may be prolonged several years. Mere dilatation of the ventricle if otherwise healthy need cause us much less concern if careful treatment is instituted soon after the beginning of the complete irregularity; but the longer the period between the onset of fibrillation and the employment of correct therapeutic measures, the more difficult is it to restore the damaged ventricle. In the majority of instances digitalis will effectually block the erratic auricular impulses, and give the ventricle the lengthened diastolic period so necessary for its recuperation. In a few cases digitalis either cannot be taken in sufficient amounts, or fails to obstruct the stimuli from the upper chamber. In these the immediate prospect is exceedingly alarming.

The degree of the response of the heart to treatment (see Figures 127 and 128), and the amount of reserve force which can be secured for it, are our best indications of what the future holds in store. Many of these hearts may be brought to a fair degree of efficiency, but the reserve force is never very great, and neglect of treatment, or over-exertion will almost invariably entail an attack of cardiac insufficiency. Each insult of this kind is met with greater difficulty and with each attack the outlook becomes more grave. Auricular fibrillation indicates a very serious myocardial defect; heart failure may be postponed for many years, but the majority succumb within ten years of its onset.

The signs which point to a favorable prognosis are:—

1. The resumption of a physiological rhythm.
2. The maintenance of a rate under 70.
3. The absence of a pulse deficit.
4. The absence of extrasystoles.
5. An average systolic blood-pressure of over 110 in the rheumatic group, and of over 160 in the arteriosclerotic group.

The symptoms which make the outlook grave are:—

1. A ventricular rate remaining for more than a few days above 130.
2. A persistent pulse deficit of 20 or over.
3. The occurrence of frequent ventricular extrasystoles.
4. A falling average systolic blood-pressure.
5. A ventricular rate which shows wide fluctuations under slight physical or emotional stress.

The gravity of a given case is often indicated by the *amount of treatment* requisite to secure a slow ventricular rate without a pulse deficit; some cases require very little treatment, and in these the immediate prognosis is good; others yield only to the most active therapeutic measures applied over a very long period. In such the danger is about proportional to the therapeutic measures found necessary.

VENTRICULAR FIBRILLATION

Ventricular fibrillation has been recognized as a terminal event in experiments on animals. When an extreme degree of ventricular irritability is produced by faradization of the ventricle (Levy) or by cutting off its blood supply* the coördinated contractions cease. These are succeeded by ineffectual twitching of the muscle wall and finally by diastolic relaxation with fine undulatory movements of the surface, and in a few seconds the animal is dead. The condition has also been experimentally produced by introducing a bubble of air into the coronary artery.† In these observations the animals occasionally recovered.

In man, ventricular fibrillation, unless of only momentary duration, is incompatible with life. Hoffmann‡ has reported, with electrocardiographic records, a condition which he interpreted as a period of ventricular fibrillation occurring at the end of a paroxysm of tachycardia; the patient ultimately recovered. As far as I know, this is the only instance in which it has been suggested that such an outcome is possible, and it is not altogether clear that Hoff-

*Lewis: Mechanism of the Heart Beat, London, 1911, p. 160.
†Morat and Petzetakis: Compt. rend. Soc. de Biol., 1914. lxxvii, 222, 377.
‡Hoffmann: Heart, 1911-12, iii, 213.

mann's records may not be interpreted as a series of ventricular extrasystoles arising from several points of origin. Ventricular fibrillation, as a terminal event, has been studied by Robinson* and by Halsey.†

It seeems not at all improbable, as McWilliams‡ suggested, that in a certain number of cardiac cases sudden death may be due to the abrupt onset of ventricular fibrillation. Levy§ has shown that the administration of low percentages of chloroform, by inhalation to animals, produces a high degree of ventricular irritability. Under this condition small reflex sensory stimulation or the struggling of the animal is sufficient to induce ventricular fibrillation and sudden death. If adrenalin was given intravenously to an animal under light of chloroform anæsthesia, ventricular fibrillation at once ensued. Levy believes that the cause of chloroform death in man is the onset of ventricular fibrillation and has collected from the reports of sudden death under chloroform a number of cases which strongly support his view.

The period of danger is at the time the patient is getting very little chloroform, at the beginning of its administration or when it is given intermittently. To avoid this danger, all nervous excitement for the patient must be excluded. No manipulations must be attempted until he is well under the anæsthesia. The chloroform must be given continuously and in considerable amounts. Adrenalin should not be used in conjunction with chloroform.

*Jour. Exp. Med., 1912, xvi, 291.
†Heart: 1915, vi, 67.
‡Brit. Med. Jour., 1889, i, 6.
§Heart, 1912-13, iv, 319.

CHAPTER XII

Auricular Flutter, Tachycardia and Fibrillation

These forms of arrhythmia have been discussed in separate chapters, but it may not be amiss to say a few words about their close association and to examine the slight modifications in mechanism which may lead to the transition of one into another.

In 1887, McWilliams,* in investigating the activities of the dog's heart under different experimental conditions, called attention to the variation in the nature of the response of the auricles to faradic currents of greater and less intensities. He found that (1) when he stimulated the auricular tissues with a weak faradic current the auricles contracted at a very rapid rate in perfect rhythm. If, however, the strength of the stimulating current was increased, (2) the auricles ceased to contract as a whole, the walls relaxed and assumed the diastolic position, but the muscle movements did not stop; the whole surface of the auricles took on an undulatory, incoördinated activity with irregular twitching, but with no actual systole of the upper chamber. Finally, (3) when stimulating current was withdrawn, the auricles resumed their accustomed deliberate coördinated contractions. In the first instance, the ventricles responded rhythmically, in response to each auricular contraction or more often to every other auricular impulse. Under the conditions of the second stage of the experiment, the ventricles responded in a haphazard fashion with complete irregularity. Finally, when the auricles recovered their accustomed activity, the ventricles responded in a normal manner.

Since these earlier observations, these different activities have been elaborately studied and are now recognized as "auricular flutter," "paroxysmal auricular tachycardia" and "auricular fibrillation." McWilliams' observations have been verified by a number of other investigators. Hirschfelder† was able to produce at will "auricular tachycardia" or "auricular fibrillation," according to the strength of the faradizing current. The same phenomena

*Jour. Physiol., 1887, viii, 296.

†Bull. Johns Hopkins Hosp., 1908, xix, 322.

were observed by Robinson,* who found, further, that if the right vagus was stimulated during a period of experimental tachycardia there was a transition to auricular fibrillation, while stimulation of the left vagus did not inhibit the auricular tachycardia, but blocked a portion of these impulses so that the ventricle failed to respond to every auricular contraction. He believes that in the experi-

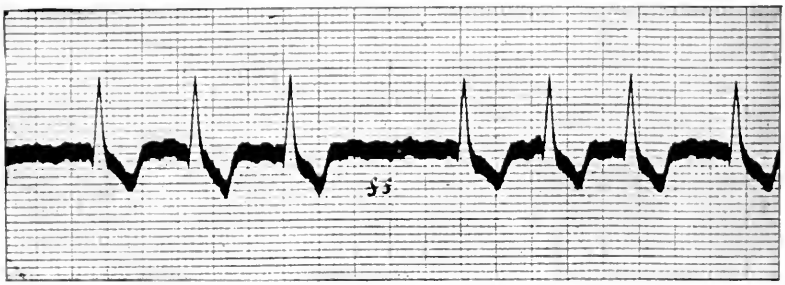

Figure 129

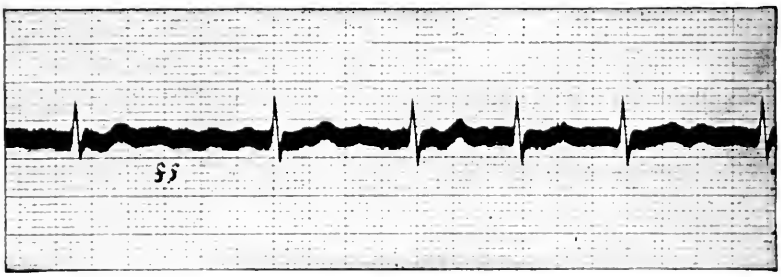

Figure 130

mental animal faradization of the auricles produces a mixed effect which is a combination of "flutter" and "fibrillation."

If one examines a number of electrocardiograms taken from a series of patients with complete irregularity, one is at once impressed with the great variety in the size and the rhythmicity of the waves representing the auricular activity In one they are so small as to be almost imperceptible and show no tendency to rhythmicity;

*Jour. Exp. Med., 1913, xviii, 704.

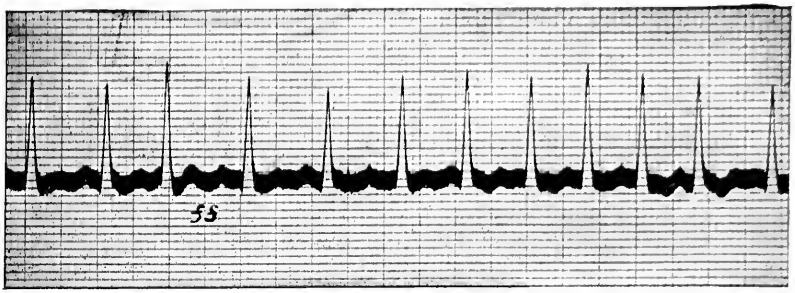

FIGURE 131

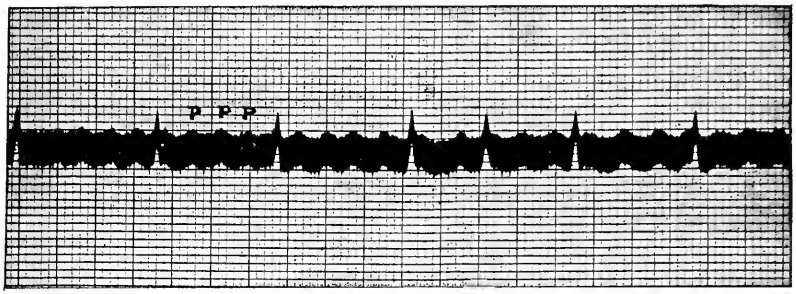

FIGURE 132

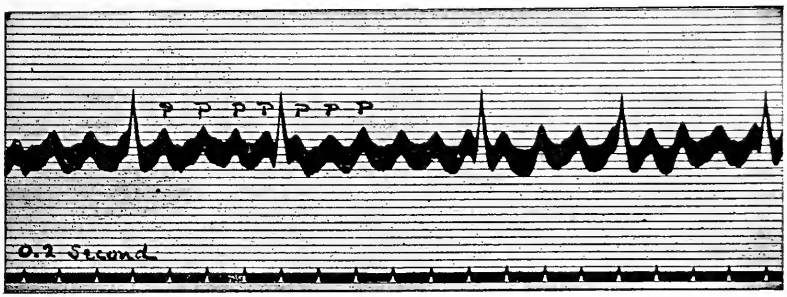

FIGURE 133

Figures 129-133.—Records from five different cases of complete irregularity. Arranged to show gradations in the size and rhythmicity of the undulations representing auricular activity and the ventricular response.

in another these waves may be very large and recur at quite definite intervals.

A series of records of this kind is shown in Figures 129, 130, 131, 132 and 133. They have been arranged to show the gradations that are seen in a collection of such curves. They vary from small irregular deflections to those of considerable size and degree of rhythmicity. One may say that, as a rule, as the auricular waves increase in size, the tendency is for them to become rhythmic, and also for the ventricle to respond to the auricular impulses at more regular intervals.

When one examines the first record (Figure 129) of this series, it conforms quite definitely to our conception of the classical picture of auricular fibrillation with the minute, very irregular fibrillary manifestations and the complete irregularity in ventricular response.

In the last record of the series (Figure 133) the undulatory waves are very much larger and quite rhythmic. The ventricular response is irregular, but not more so than might be seen in a case of "auricular flutter" with a block of varying degree. An inspection of the intervening records show gradations which suggest that, under various pathological conditions which are not understood, and at present cannot be differentiated, there may be all grades of auricular activity, such as the incoördinated contraction of individual muscle fibers, contractions in which a group of fibers are coördinated and contractions resulting from a coördinated activity of the entire musculature.

The close relationship of "auricular flutter" and "auricular fibrillation" may be further inferred from the ease with which, in the individual patient, the activity may change from one form to the other. Instances of this transition have been illustrated in the chapter on "auricular flutter" (Figures 92, 93, 96, 97 and 98).

It has been suggested that in "auricular flutter" the auricular contractions arise not from the normal pacemaker, the sinus node, but from some point of abnormal irritability in the auricular wall; in other words, that they are true auricular extrasystoles. If such is the case, it would bring our ideas of the mechanisms of "auricular flutter" and "auricular tachycardia" into a very close relationship. Such a conception is borne out by a study of the records

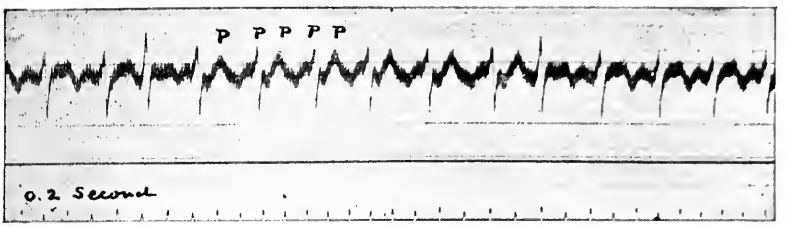

FIGURE 134

Record of T. K. showing transition from complete irregularity (auricular fibrillation) to rhythmic auricular flutter with a 2 to 1 ventricular response (*V's* = 120, *A's* = 240) and the return to complete irregularity.

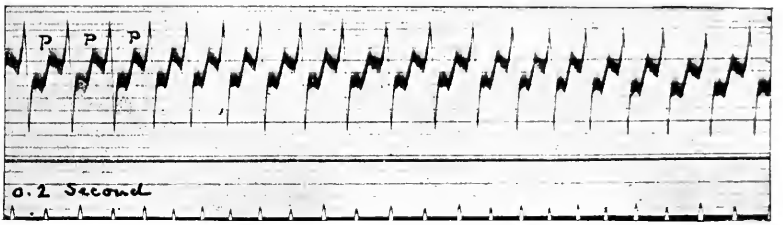

FIGURE 135

Record of T. K. taken a few minutes after Figure 134, showing rhythmic tachycardia (*V's* = 240). The ventricle responds to each auricular impulse.

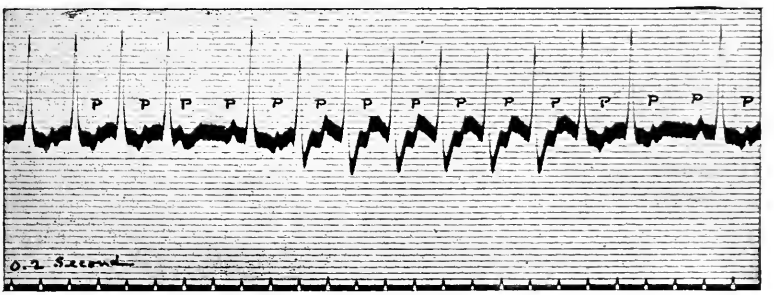

FIGURE 136

Transition from auricular flutter with irregular response to rhythmic tachycardia (*V's* = 180.)

(Figures 134, 135 and 137) from a patient who was under observation for a period of less than two hours, during which these curves and a number of others were secured. The beginning and the end of the electrocardiogram (Figure 134) can be interpreted as auricular fibrillation. In the center of this record there is a rhythmic period during which there appears an auricular flutter, with a ventricular response, to every other auricular impulse. A few minutes later this passed into a period of rhythmic auricular tachycardia (Figure 135), during which the ventricular rate is just double the rate of the rhythmic period of Figure 134. It seems evident that the auricle is still in flutter and that now the ventricle is responding to each auricular contraction.

Figure 137 is a polygraph secured from the same patient a few minutes later and shows the transition from a period of flutter, with irregular ventricular response, to one of rhythmic tachycardia. The electrocardiogram of another patient showing a transition from an auricular flutter, with an irregular ventricular response, to a typical paroxysm of auricular tachycardia, terminating again in auricular flutter, is presented in Figure 136. This patient was under observation for many weeks and her heart recovered a normal rhythm. During none of this time was it possible to obtain any conclusive evidence of auricular fibrillation.

Such observations suggest that there is a very close relationship between these types of abnormal auricular activity.

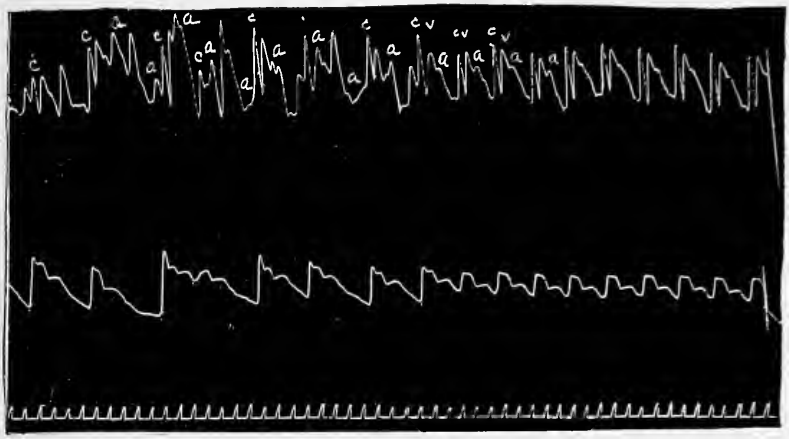

FIGURE 137

Polygraph of T. K. taken immediately after Figures 134 and 135, showing transition from auricular flutter with irregular response to rhythmic tachycardia.

CHAPTER XIII

Alternation

The palpation of the arterial pulse often presents to the clinician a series of waves which alternately vary in size. The waves recur with perfect rhythmicity for considerable periods. There is a large wave, then a small wave, another large wave followed by a small wave, and so on for hours or days. This condition is known as alternation of the pulse. The term alternation is not confined to this pulse phenomenon, but is also used to designate a similar alternating activity of the heart and of the jugular veins.

The term alternation requires further definition than the mere statement that large and small pulse waves follow one another. Alternation should be reserved for that activity of the heart in which forcible and weak contractions succeed one another rhythmically, and in which the interval between the large and small beats is equal to or greater than the interval between the small and the large beats. It is to be distinguished from the pulsus bigeminus, in which every other cardiac contraction is an extrasystole, and also from the pseudo-alternans detected in both veins and arteries, which is directly dependent on respiratory movements.

EXPERIMENTAL PRODUCTION AND MECHANISM

Alternation has been studied extensively in both cold- and warm-blooded animals. In the course of experimental studies of various kinds, it occurs spontaneously and has also been produced by utilizing various poisonous substances. It has been seen in the isolated and perfused heart and in the heart exposed by removing a portion of the chest wall. In the suspended heart, it has been provoked by withdrawing the supply of Ringer's or Locke's solution, by the substitution of carbon dioxid for the oxygen, by the introduction of hæmolytic serum, etc., etc. This activity has been induced in the heart in situ by the introduction of digitalis and aconitine (Cushny) and antiarin (Straub) and glyoxylic acid (Hering, Rihl, Kahn and others). Alternation may appear during the ap-

plication of electrical stimulation to the myocardium and while the extracardial nerves are being manipulated.

The difference in the successive waves may be in their contour, rather than their size. Their dissimilarity is practically always demonstrable by a time pressure curve, but the electrocardiogram furnishes evidence of alternation only on very infrequent occasions. When this is present, it consists in an alternating height of the *R* wave, more rarely in a change in the elevation of the *T* wave.

The mechanism of alternation is still a matter of considerable speculation. The majority of investigators ascribe this abnormal function to a defect in the *contractility* of the heart. It has been supposed that under pathological conditions different muscular fibers have different refractory periods, so that at one time all the fibers are active, at another only a portion take part in the contraction (Wenckebach, Mines). A view that has received considerable support is that the cardiac fibers differ in their *excitability* and, hence, respond to stimuli at uneven intervals (Gaskell, Kronecker). The abnormal activity has also been thought to be dependent on a defect in *conductivity* (Engelmann, Muskens). It is clear that as yet the evidence is not conclusive in regard to the fundamental properties of cardiac tissues which are at fault in the production of this irregularity. On clinical grounds, it seems to me that in alternation we are dealing with a phenomenon which may be produced by a defect of contractility at one time and at another time by a defect of conductivity. I believe that further study will permit us to segregate our cases of alternans into groups, each of which may be shown to be due to a different defect.

A presentation of the various theories of alternation and a critical review have been published by Gravier* in a recent thesis.

PATHOLOGY

There are reported in the literature about twenty autopsies in subjects in whom alternation of the heart has been a prominent feature and in whom this arrhythmia has been verified by graphic methods. Some of these reports are not at all complete; others give in considerable detail the histological findings obtained from

*L'Alternance du cœur, Paris, 1914.

a very exhaustive study of the cardiac muscle. Practically all of the hearts thus examined have presented anatomical lesions of greater or less extent. There is usually an hypertrophy, particularly of the left ventricle, with some degree of degeneration of the muscle fibers due to a perivascular sclerosis. These lesions are, as a rule, quite extensive and are not limited to any special part of the cardiac tissues. In some, the lesions have involved portions of the conduction system, but this finding is not at all constant. We may conclude that, as yet, we have no evidence which permits us to assign this functional disturbance to a special anatomical lesion.

The ease with which alternation may be provoked in the normal cardiac tissue of experimental animals by the administration of toxic substances, such as aconitine, digitalis, glyoxylic acid, strophanthin, veratrin, etc., has suggested that this functional abnormality may be the result of a cell intoxication. Digitalis must always be thought of as a possible factor, but one frequently sees alternation in patients who have never had this drug. Hering has described alternation in a man poisoned with strychnine. As alternation is not an uncommon accompaniment of advanced nephritis, it has been suggested that it is a manifestation of uremic poisoning.

The remarkable properties of glyoxylic acid in producing alternation experimentally, and the fact that this acid may be a product of abnormal metabolism, gives us a hint of another possible source of an auto-intoxicant.

ETIOLOGY

Our ideas of the frequency of the occurrence of the various arrhythmias of the heart is changing very greatly. The routine painstaking examination of large numbers of patients has demonstrated that irregularities which were once believed to be very exceptional are in reality not uncommon. This, for example, has been our experience with "auricular flutter." At one time this was regarded as a very unique finding; today it is not unusual to have more than one such case always under observation. A similar transition has occurred in our notions of the frequency of alternation. Not long ago it was thought to be rather a rare symptom; to-day, according to the statistics of some observers, it ranks

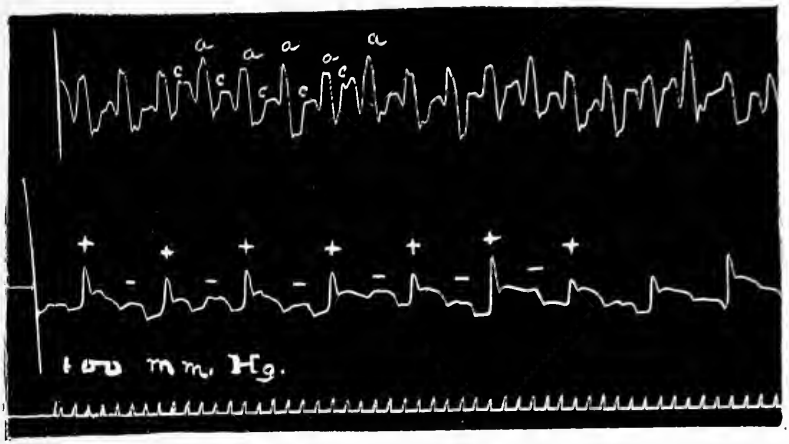

FIGURE 138

Alternation in brachial and jugular. Note variation in amplitude of *a* waves. Taken from same case as Figure 139, only a few minutes separate the two records.

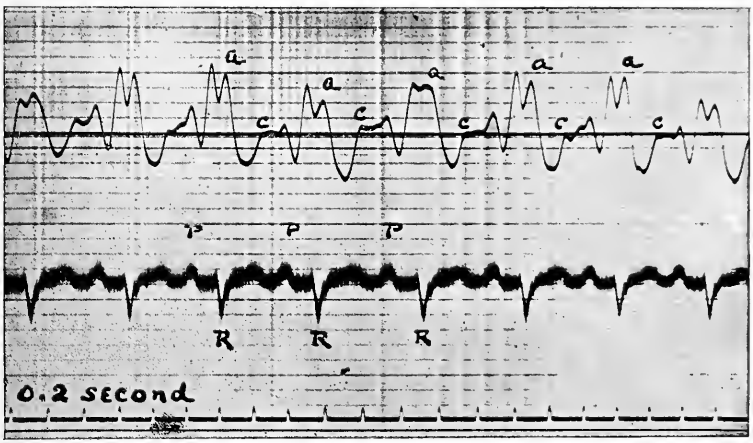

FIGURE 139

Jugular and electrocardiogram taken simultaneously. By palpation the radial was alternating at this time. There is no evidence of alternation in the electrocardiogram. Same case as Figure 138.

in frequency only second to the extrasystole. In a recent study of three hundred cardiac cases, White,* during a period of eight months, discovered no less than seventy-one cases of alternation. He found this irregularity in thirty-three per cent. of all the cases showing any degree of cardiac decompensation in which he secured graphic records.

Alternation is more frequent in the old than in the young, but has been observed in all ages from fifteen to seventy-five years. It is a common manifestation in myocarditis with hypertension. In my own experience it has most often been seen in cases of chronic nephritis or general arteriosclerosis, which have recently been admitted to the hospital wards.

Alternation is rather uncommon in the course of the acute infections, but it has been noted in pneumonia, diphtheria, typhoid fever and rheumatism. It is seen in cases of chronic valvular disease and pericarditis which are of rheumatic origin.

Alternation has been observed in acute dilatation (Mackenzie) in a case showing myocardial infarcts (Gallavardin) and in strychnin poisoning (Hering).

White obtained a positive Wassermann reaction in fifteen per cent. of his cases, and a history of overindulgence in alcohol, tobacco and tea was notable. In an analysis of forty-five cases of alternation, Windle† observed that the associated conditions presented the following order of frequency: (1) Arterial and myocardial disease; (2) chronic heart disease due to rheumatism; (3) pneumonia, and (4) acute rheumatic carditis.

In a considerable number of patients showing alternans, this is by no means the only evidence of myocardial defect. In association are found many other types of cardiac arrhythmia. A latent alternation often becomes evident when the heart rate is accelerated, hence alternation is a very common accompaniment of very rapid hearts, particularly those showing paroxysmal tachycardia (Figures 140, 150 and 151). Many also present signs of imperfect conduction. Alternation is quite common in cases of "auricular flutter" (see Figure 146) and this is not surprising when we recall that in the latter condition conduction defects and tachy-

*Amer. Jour. Med. Sc., 1915, cl, 82.
†Quart. Jour. Med., 1912, vi, 453.

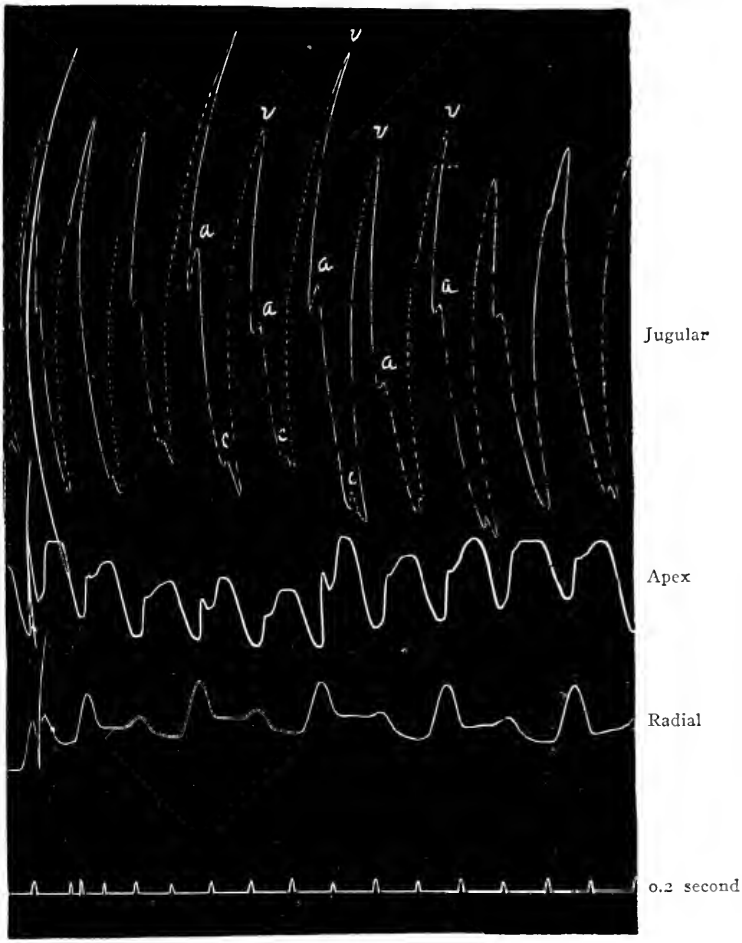

FIGURE 140

Alternation of apex, radial and jugular. Tachycardia rate 200.

cardia are very often in evidence. The association of extrasystoles is a frequent finding and an alternation which may have been quite unnoticed may become very pronounced in the cycles immediately following an extrasystole. This is so common that it has been given a special name, the "post-extrasystolic alternans."

It seems to me that the evidence is very suggestive that in certain cases of alternans the exciting cause of this irregularity is really a lack of proper nutrition of the myocardial cells. Under conditions of stress, the tissues have too little oxygen or there is an accumulation of CO_2 or possibly other toxins which may be the intermediary products of abnormal metabolism.

IDENTIFICATION

The more pronounced types of alternation may be detected by palpation of one of the peripheral arteries. This is naturally more easily accomplished when the large and small arterial waves show a considerable difference in the systolic blood pressures. This difference usually does not exceed ten millimeters of mercury, but occasionally may be as much as twenty millimeters (Rihl). With the smaller variations in pressure this important type of irregularity often passes unobserved, but if one is constantly on the outlook for it in cases in which it may be suspected, such as nephritics, general arteriosclerotics, etc., it will be discovered with considerable frequency. There are several simple means which may aid in the detection. Palpation of the pulse should always be made with the tips of several fingers, not with one finger alone, and while one is making the observation the pressure exerted on the arterial wall should be changed, as this often accentuates the differences in the size of the waves. A latent alternans may frequently be made more evident by partly occluding the artery above the palpating fingers. This may be accomplished by exerting pressure on the brachial artery by means of the cuff of a sphygmonanometer or by digital pressure of the axillary artery (Gallavardin and Gravier). The pressure thus applied should be varied in amounts, as it is difficult to predict in advance what degree of pressure will best accentuate the pulse differences. A little exertion to increase the rate of the heart may be of service in rendering the alternation more evident.

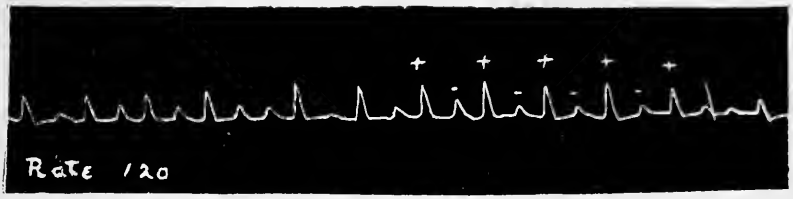

FIGURE 141

Radial tracing showing alternation and probably one extrasystole.

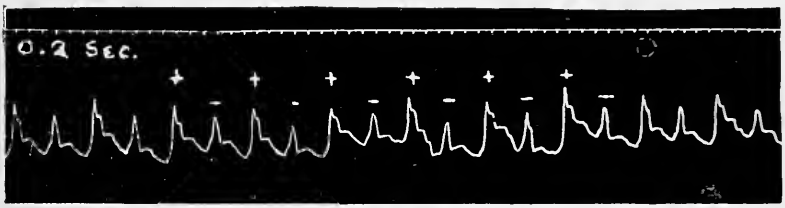

FIGURE 142

Radial tracing showing alternation. While this record was being taken a pressure of 85 mm.Hg. was applied to the brachial artery through a sphygmomanometer cuff in order to bring out the differences in amplitude of the successive waves.

In palpation the attention must be directed not only to the variation in amplitude of the successive waves, but also to their spacing; if the impression is obtained that the interval between the small and succeeding large wave is greater than the interval between the large wave and succeeding small wave, the irregularity is more probably an extrasystolic "bigeminus" than an alternation. This is the irregularity which has most often been confused with alternation.

Palpation of the precordial region may give evidence similar to that secured from palpation of the peripheral arteries, but this is usually an uncertain aid. Auscultation of the heart rarely reveals a difference in the intensity of the sounds of the strong and weak beats. When murmurs are present, they occasionally vary in intensity, but a change in heart sounds normal and pathological are far more characteristic of the extrasystolic irregularities and auricular fibrillation than they are of alternation.

In rare cases of alternation, inspection of the venous pulse shows a great variation in amplitude of every other cycle, thus giving us a clue to the type of the irregularity (see Figure 140).

Unless instrumental means are employed as a routine, many cases of alternation will escape recognition. Painstaking observations with the sphygmograph are our only sure means of detecting pulsus alternans in a very large number of instances. These records, taken under the various conditions outlined in discussing palpation of the pulse, present to us the minute variations in the amplitude and spacing of the succession waves, which will often escape even the highly cultivated tactile sense of the expert.

Typical sphygmograms are presented in Figures 141 and 142. In these records the difference in the amplitude of the alternate waves and their rhythmic spacing is well illustrated. Figure 141 is a radial tracing from a case in which the alternation was quite evident from palpation of the pulse. Figure 142, also taken from the radial pulse, was only brought out distinctly when a brachial cuff, with pressure 85 mm. Hg., was applied. Some cases of alternans have been published with graphic records in which the successive waves differ in duration rather than amplitude.*

Graphic records of the apex beat are, as a rule, rather unsat-

*Gravier, loc. cit., p. 44.

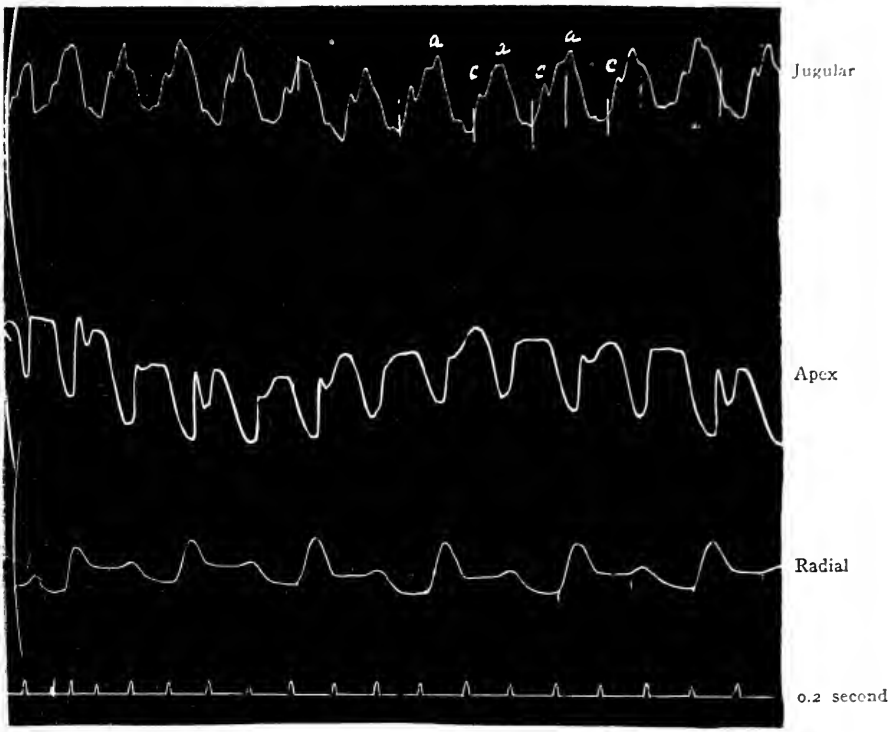

FIGURE 143
Polygram showing alternation in radial and apex.

isfactory in detecting this condition. It is, however, fairly well shown in Figures 140 and 143. In these records the alternation in the radial is quite evident. The alternation in the apex activity is indicated by the notching of every other wave of the cardiogram. Hering has pointed out that tracing, taken in the precordial region near the base of the heart, sometimes shows an alternation when it is absent in records taken near the apex.

The respiratory movements of the chest are prone to affect the contour of the waves of the cardiogram, but in these records the evidence is quite conclusive that the variation in the waves is quite independent of the breathing. It may be noted in this connection that in cases with a slow pulse and rapid respiration, when the pulse rate is approximately double that of the respiration, the peripheral arteries may show a difference in the size of the successive beats which simulates alternation. The pulse wave coincident with inspiration is smaller than that corresponding to expiration. This pseudo-alternans is easily differentiated by having the patient hold his breath when the amplitude of the pulse waves at once becomes uniform.

Alternation may or may not appear in the venous tracings and may assume several forms. There may be alternation of the *c* waves, of the *v* waves or of the *a* waves.

In Figure 144 is shown a well-marked alternation in the arterial record, but no evidence of this irregularity in the jugular. Respiratory movements are much more likely to distort the phlebogram, hence in cases of doubt it is always a wise precaution to secure the record during a period of suspended breathing.

Alternation in the *c* wave is quite evident in Figure 145. Here the large *c* wave corresponds to the large wave of the brachial, which is quite what one would expect. Occasionally one sees cases in which the small *c* wave corresponds to the large peripheral arterial wave. The explanation of this phenomenon is difficult.

Alternation of the auricle is seen in experimental work and may be diagnosed in the clinic by an alternating amplitude in the *a* wave of the jugular tracing. Such a record is exhibited in Figure 138.

On the whole, evidence of venous alternation is rather rare and its significance has not been carefully studied.

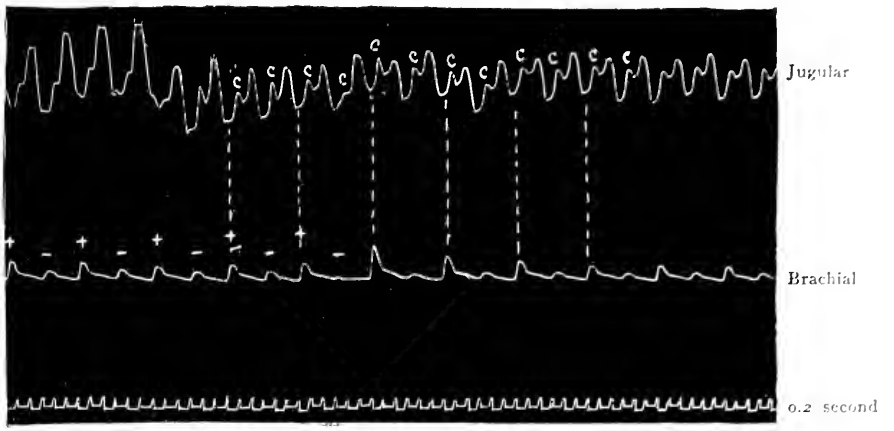

FIGURE 144
Alternation of brachial, no alternation in jugular.

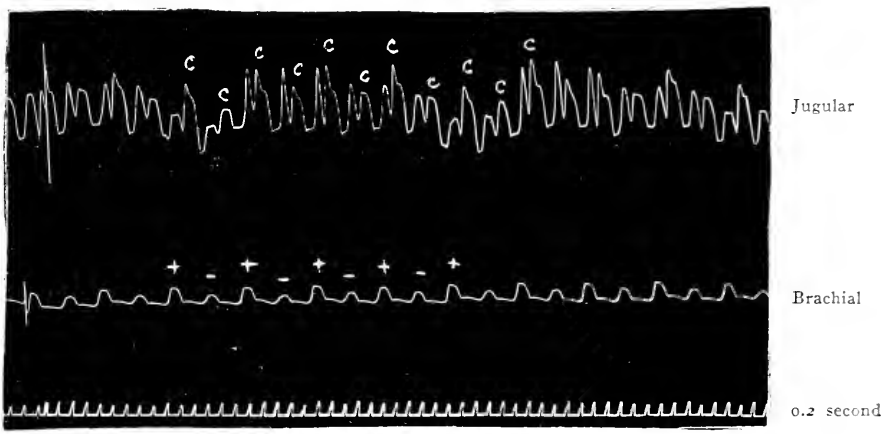

FIGURE 145
Alternation of brachial and of *c* wave of tracing taken from above the clavicle.

A very beautiful exhibition of alternation involving radial apex and jugular curves, is shown in Figure 140. This record was secured from a man suffering from advanced myocardial disease several months before his death. This curve was taken during an attack of paroxysmal tachycardia, during which the rate was 200 per minute. The phlebogram is difficult to analyze, since the *a, c* and *v* waves are fused together. At other times there was also evidence of a prolonged *a-c* interval, so this element also complicates the analysis. It would seem that there is in this case alternation of the *v* wave, due to alternating ventricular activity, which is very prominent on account of an insufficient tricuspid valve. There also would appear to be an alternation of the auricle, as evidenced by the differences in amplitude of the *a* waves, but this is less certain.

The electrocardiogram rarely shows evidence of alternation. The differences in amplitude of the waves of the peripheral arteries may be quite evident, and yet the successive cycles of the heart, as portrayed by the galvanometric curves, may present great uniformity (see Figures 139, 146, 148 and 149). In Figure 146 are presented simultaneous curves of a radial pulse and the electrocardiogram. The latter record shows that we are dealing with a case of auricular flutter with an irregular ventricular response. The radial waves distinctly alternate in amplitude, but it would seem that their rhythmic spacing is due to a delay in the transmission of the smaller waves to the periphery, rather than to a true alternating cardiac activity.

Figure 147 was secured from an old gentleman with cardiac decompensation and a pulse which, on palpation, closely simulated an alternans. The combined record shows that the irregularity is really not due to alternation, but to ventricular (*Vx*) and auricular (*Ax*) extrasystoles.

A true alternans, which is complicated by a single premature beat, is presented in Figure 148. The abnormal form of the usual ventricular complex suggests a lesion of one branch of the *A-V* bundle.

Rarely one obtains electrocardiographic evidence of alternation of the heart. This may consist in varying amplitudes of the *R* or of the *T* waves. Two such curves are shown in Figures 150

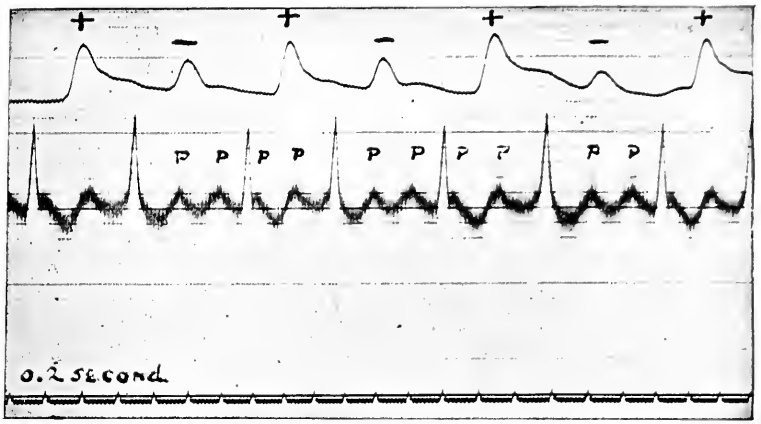

FIGURE 146

Radial showing alternation. The electrocardiogram shows auricular flutter with irregular ventricular response.

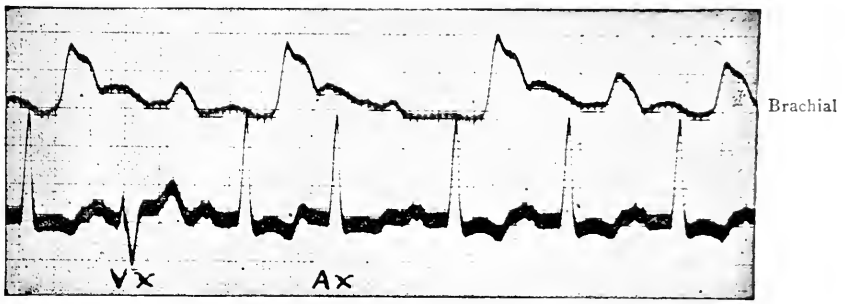

FIGURE 147

Pseudo-alternation of brachial due to ventricular (*Vx*) and auricular (*Ax*) extrasystoles.

and 151. Figure 150 was from a woman suffering from paroxysmal tachycardia; between the attacks neither radial tracings nor the galvanometer showed evidence of alternation. Soon after the onset of the paroxysm, the alternation could be detected both in the peripheral arteries and in the electrocardiogram. The electrocardiographic record shows a well-marked variation in amplitudes of the *R* waves. Figure 151 was secured from a man who at various times gave electrocardiographic evidence of defective conduction, extrasystoles, etc. He had frequent short paroxysms of ventricular tachycardia, never exceeding three minutes in duration. In some of these attacks the alternation in the ventricular complexes appeared, as shown in the cut. He left the hospital with a fairly regular, but evidently much diseased, heart. Unfortunately, he removed to another city, and we have been unable to secure information in regard to the outcome.

CLINICAL FEATURES

I have never seen a patient who was conscious of the alternation of his pulse. It is the other commonly associated symptoms which bring him to the physician. There is almost always some dyspnœa; this may be of mild degree or of the most extreme type. Many of them have Cheyne-Stokes breathing, some have nocturnal dyspnœa, which occurs in paroxysms and wakes them from their sleep. Patients with alternans are frequently the subjects of anginal pain. It is quite clear that the alternation is merely one of the manifestations of a failing heart, but one that is of considerable significance.

Alternation appears in varying degrees. The variation in the pressure values of the successive waves is never very great (5-10 mm. Hg.). The larger differences are easily detected by the finger; the smaller can be made out only by the aid of instruments of precision. It may be continuous for hours or days, or may be detected only in short runs of a few beats. It is associated with many other cardiac irregularities. It often appears in a few beats following an extrasystole and then is lost until introduced by another extrasystole. It has a tendency to become more evident when the heart rate is quickened and to disappear as the heart becomes slower. One often sees alternation of the pulse in patients suffer-

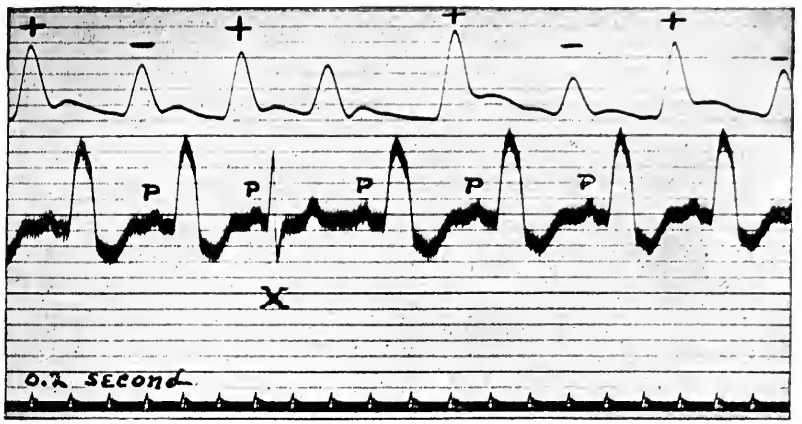

FIGURE 148

Alternation of radial. The electrocardiogram suggests that there is a lesion of one branch of the *A-V* bundle. Complex (*x*) appears to be of a more normal form, but this type of beat was very unusual in this patient.

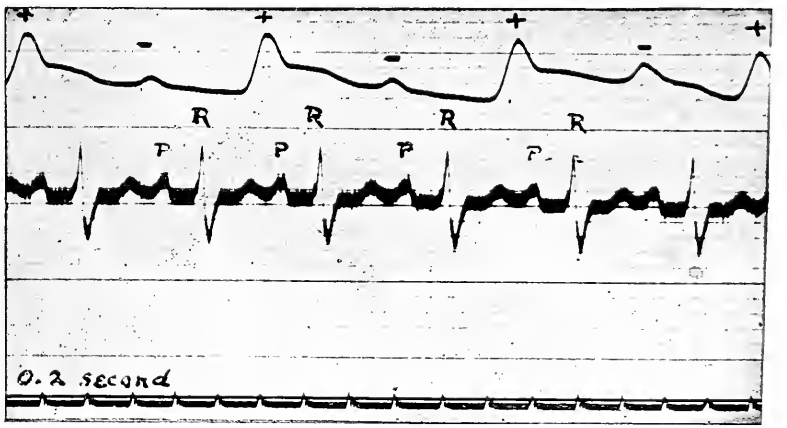

FIGURE 149

Radial shows alternation. No evidence of alternation in the electrocardiogram.

ing from advanced nephritis when they are first admitted to the hospital, which disappears after twenty-four hours' rest in bed.

PROGNOSIS

Most observers are in agreement that alternation is a sign of very grave import. This certainly holds for the well-marked alternation, which is usually detected by palpation and which continues for long periods. While in some cases this may be practically the only sign of the failure of the heart to carry its load, other signs are usually present, such as angina, dyspnœa, periodic breathing, œdema, etc. All these emphasize the seriousness of the changes in the myocardium. But even without these concomitant symptoms, a well-marked continuous alternans should arrest the examiner's attention and lead him to offer an extremely guarded prognosis.

The lesser grades of alternation, those which are not persistent, those which are made evident only when the heart rate is very greatly accelerated, and those which occur during the acute infections, but which are not prolonged for any considerable time, probably should be regarded as much less significant of grave myocardial defect. As yet these cases have not been studied in sufficient numbers or followed up carefully enough to permit us to draw more definite conclusions in regard to the prognosis.

As I have already suggested, further study may show us that in one case alternation is due to or associated with a defective conduction, while in another abnormal irritability may be the prominent associated change. It may be that on the basis of such a classification this sign may have a new value in prognosis.

In a study of the largest number of cases by a single observer, White* found alternation in 33 per cent. of all the heart cases showing any degree of cardiac decompensation, in which he secured graphic records. When his report was made, sufficient time had not elapsed to permit him to draw accurate conclusions as to the ultimate outcome of his cases.

Lewis regards alternans as an evidence of a disproportion between the ability of the heart muscle and the work it is called upon to perform. Hence, this irregularity may become manifest

*Am. Jour. Med. Sc., 1915, cl, 82.

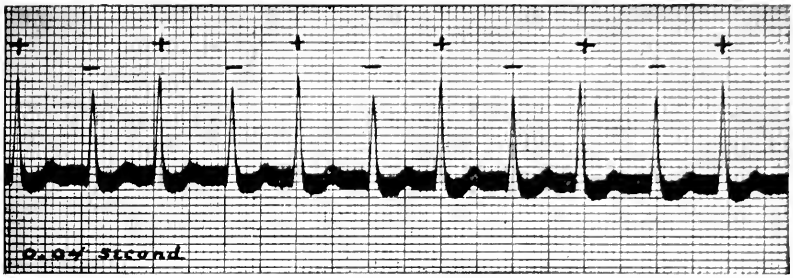

FIGURE 150

Tachycardia rate 156. The electrocardiogram shows alternation in the amplitude of the *R* deflections. Note that the short *R* is a trifle nearer the succeeding than the preceding tall *R*.

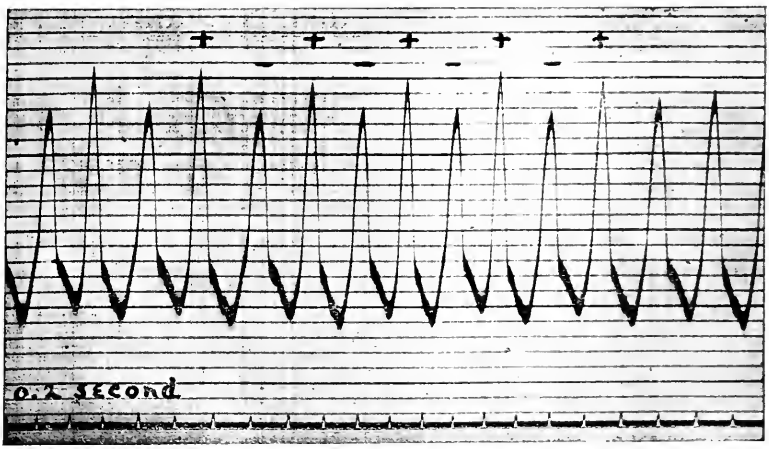

FIGURE 151

From a case of paroxysmal ventricular tachycardia. Rate 205. The electrocardiogram shows alternation.

when the heart muscle is reasonably healthy, if the demands upon it are excessive, and also when the diseased or poisoned muscle is endeavoring to perform work of which it is barely capable. In cases of paroxysmal tachycardia, in which on the strength of other evidence we feel reasonably sure that the ventricular muscle is not seriously damaged, an alternation of the heart is of much less significance than when it appears in a heart with a slow rate.

If we consider only these hearts with a slow rate, our personal experience would agree with that of Vaquez,* who finds three degrees of alternation which may be arranged in the order of their prognostic value, as follows: (1) Prolonged continuous alternation; (2) post-extrasystolic alternation; (3) transitory alternation.

A fatal termination may be expected in continuous alternation within a few months. I know of no case of this kind in which death has been delayed more than two years.

*XVII, Internat. Congress of Medicine, London, 1913, vi, 164.

CHAPTER XIV

The Influence Exerted by the Extracardial Nerves

In discussing the theories of the nature of the heart beat, it was pointed out that the cells of the myocardium intrinsically possess the fundamental properties through which the activities of the heart are initiated and maintained; these are modified and adapted to the momentary needs of the body through the agency of the extracardial nerves. It is conceivable that a departure from the normal efficiency of this adjustment may arise in two quite distinct ways: (1) The muscle cells may be so changed that one or more of their fundamental properties may be more than normally sensitive to nerve influences; (2) the nerve mechanism may be defective, and modifying influences abnormally large or small may thus be brought to bear on the cells of the myocardium.

It is, therefore, important that we should examine the manner in which the myocardial activities are modified by the extracardial nerves and the clinical manifestations which are the result.

ANATOMY AND PHYSIOLOGY

The extracardial nerves which modify the activity of the heart are the cardiac branches of the two vagi and branches of the cervical sympathetics. The vagus arises from nuclei lying in the medulla in the lower part of the floor of the fourth ventricle, passes out of the medulla in a groove between the restiform and olivary body, escapes from the skull through the jugular foramen and passes down the neck in the sheath of the carotid artery. About the level of the lower border of the thyroid cartilage the nerve is joined by branches of the sympathetic; at the level of the first rib the cardiac branches are given off; on the right side they follow the sheath of the innominate artery and on the left pass in front of the aorta, thus reaching the cardiac plexus.

The sympathetic fibers come from the spinal cord by way of the four or five upper thoracic spinal roots and pass through the first thoracic ganglion either to the inferior cervical ganglion or

directly to join the main vagus trunk. The fibers derived from the sympathetic are known as the accelerator nerves of the heart.

The cardiac plexus is composed of nerve fibers which can be traced over the posterior surface of the auricles and over the auriculo-ventricular groove to the ventricles. The ultimate distribution of the vago-sympathetic fibers is not entirely clear, but the recent work of Keith and Flack, Oppenheim, Dogiel and Cohn strongly suggest that some of them terminate in the muscle cells of the sino-auricular node, while others end in the node of Tawara or follow the bundle of His and its branches into the muscle cells of the ventricles. These histological studies suggest that the specialized muscle cells of the nodes and *A-V* bundle play an important rôle in receiving the modifying impulses conveyed to the heart by the vago-sympathetic nerves.

Stimulation of the vagi causes (1) a slowing of auricles and ventricles, (2) a depression of conductivity, and (3) probably a diminution in force of the contractions of the left ventricle. Stimulation of the sympathetics causes an acceleration of the heart rate.

Studies on the activities of the extracardial nerves have revealed marked functional differences in the right and left vagus and in the two sympathetics. These differences are qualitative, as well as quantitative. Thus, in his experimental work on dogs, Cohn* found that stimulation of the right vagus usually caused the arrest of all the chambers of the heart, but appeared to have very slight direct influence either on conduction or the activities of the ventricle. On the other hand, stimulation of the left vagus had only a moderate slowing effect on the auricles, but a very definite depressing influence on the rate of conduction between auricles and ventricles. His conclusions as to the differences in the distribution of the fibers of the right and left vagi are shown in Figure 152.

Rothberger and Winterberg† have shown that a corresponding difference exists in the distribution of the right and left sympathetic fibers, stimulation of the right stellate ganglion caused an increased auricular rate without conduction changes, while stimulation of the left stellate ganglion shortened or abolished the conduction time, calling forth the suggestion that the irritability of the *A-V* node

*Jour. Exp. Med., 1912, xvi, 732.

†Arch. f. d. ges. Physiol., 1911, cxli, 217; 1910, cxxxv, 506, 559.

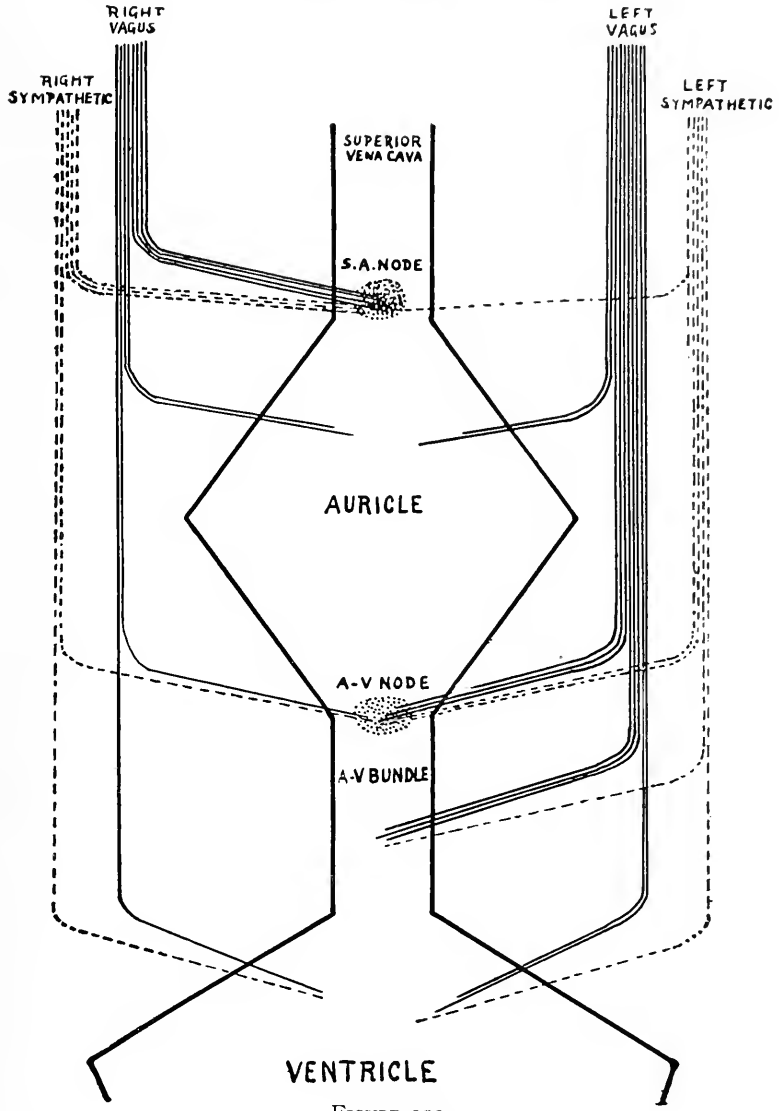

FIGURE 152

Diagram after Cohn (modified) indicating the distribution of the fibers of the right and left vagi and the right and left sympathetics.

It is to be noted that the right vagus and the right sympathetic are in the main distributed to the sino-auricular node and the auricle. The left vagus and the left sympathetic have a preponderating influence over the auriculo-ventricular node and bundle. The data upon which this diagram is based were obtained principally from the conclusions drawn from their experimental work by Cohn and by Rothberger and Winterberg.

had been thus increased so that it had assumed the rôle of pacemaker for the heart. The inferences as to the distribution of the fibers of the sympathetic, drawn from the observations of Rothberger and Winterberg, have been incorporated in the diagram (Figure 152).

Antedating the experimental work above outlined, Robinson and Draper‡ had shown that in man right and left vagus pressure produced distinct qualitative differences. Their method was to make digital pressure over the carotid sheath sufficient to obliterate the carotid pulse. They concluded from their electrocardiographic studies that the right vagus had a more evident influence on the rate and force of ventricular contractions, and that the left vagus had a pronounced effect in modifying conduction. Clinically, vagus pressure should be employed only on one side at a time; alarming standstill of the heart may result from bilateral pressure.

The contrast in the effect of the activities of the right and left vagi are shown in Figures 153 and 154. Both of these records were obtained from a case of paroxysmal tachycardia. The arrows indicate the time at which vagus pressure was made. Figure 153 was taken during pressure on the right vagus; Figure 154, during pressure on the left vagus. The tachycardia was caused by a very rapid auricular activity to which the ventricle made a corresponding rapid response. Pressure on the right vagus (Figure 153) immediately changed the rate of the pacemaker from 172 to 56 per minute. In Figure 154 is shown the effect of left vagus pressure: the ventricular rate is immediately changed from 172 to 86, but it is evident that the mechanism of the altered function is quite different from that shown in Figure 153. The auricular rate is unchanged, but the ventricle responds only to every other auricular impulse. Alternate auricular impulses are blocked, hence the ventricular rate is halved. Here it is evident that stimulation of the right vagus exerted its influence mainly on the auricle, stimulation of the left vagus had no effect on the auricle, but modified the *A-V* junctional tissues in such a way that a partial block was induced. Numerous attacks of tachycardia, while this patient was under observation, permitted us to repeat these observations on a number of occasions.

‡Jour. Exp. Med., 1911, xiv, 217; 1912, xv, 14.

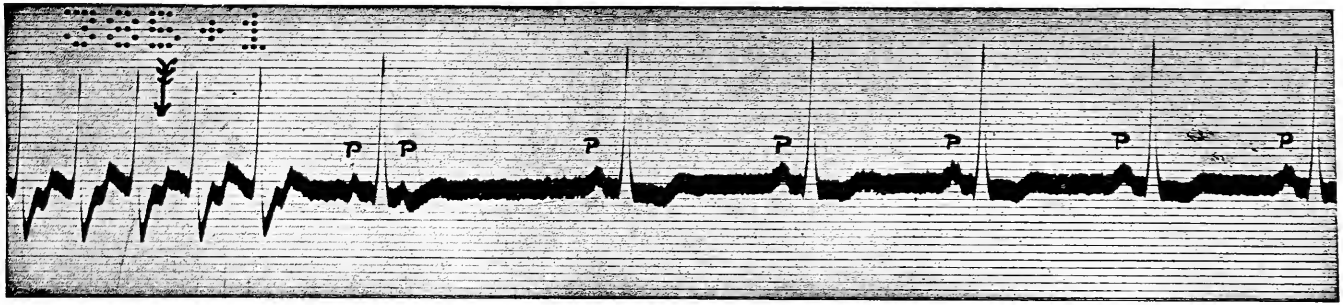

FIGURE 153

Patient E. B. "Paroxysmal tachycardia." The arrow indicates the time at which pressure was made on the *right vagus*. The slowing of the heart is effected by a diminution in the rate of the pacemaker, the sinus node.

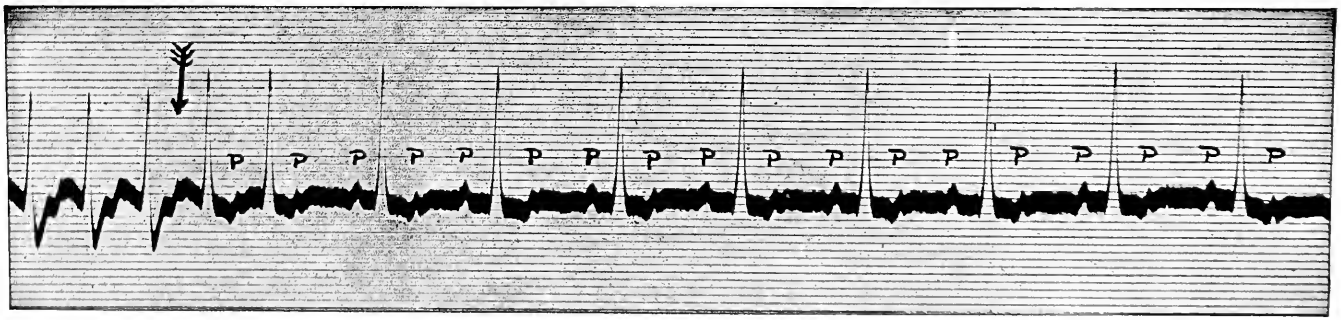

FIGURE 154

Patient E. B. "Paroxysmal tachycardia." The arrow indicates the time at which pressure was made on the *left vagus*. The auricle is not slowed, but every other auricular impulse is blocked so that the ventricular rate is halved.

Ashner* first observed that pressure on the eyeball caused a slowing of the pulse. He traced the course of these nerve impulses through the trigeminus to its nucleus and thence by fibers to the vagi. This oculocardiac reflex has been studied by many observers; a digest of their work may be found in Levin's paper.† In general, when the nerve tracts are intact, right and left ocular pressures correspond rather closely in their effects to right and left vagus pressures (Figures 155 and 156).

CLINICAL TYPES

The "accelerated heart" and its relation to vagus and sympathetic activities have been discussed in a preceding chapter and need not detain us at the present.

The slow regular heart, with a rate of 60 or somewhat less, is a type often seen. It has been referred to in an earlier chapter (VI) and is usually due to excessive vagus influence constantly at work. It has little significance, except as an indication of a high degree of vagus tone.

The slow heart, with or without sinus arrhythmia, significant of heightened vagus activity, is not infrequently met with in association with other symptoms suggestive of excessive vagus influence. These symptoms are: paleness of the face, tendency to myopia, low blood pressure, moist skin, asthma, gastric hypersecretion, hyperchlorhydria, rapid gastric motility, spasmodic constipation, etc. Patients presenting this symptom complex have been grouped under the term "hypervagotonic." Over against the group comprised in this syndrome one sees many patients presenting an opposing series of symptoms: tachycardia, flushing of the skin, gastric hyposecretion, etc. It is believed that these patients are the subjects of increased sympathetic activity, hence they have been classified as "hypersympathicotonics." These groups may further be distinguished by their reaction to drugs. The "hypervagotonic" group reacts to the administration of atropine by increased pulse rate, relief of asthmatic breathing, diminished gastric secretion, motility, etc., to pilocarpin with sweating, salivation, etc. The symptoms of the "hypersympathicotonics" are aggravated by the administration

*Wien. klin. Wochnschr., 1908, xliv, 1529.

†Arch. Int. Med., 1915, xv, 738.

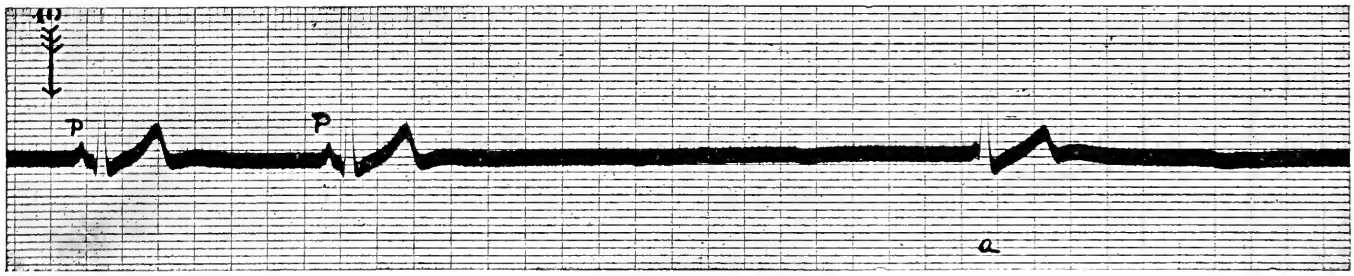

FIGURE 155

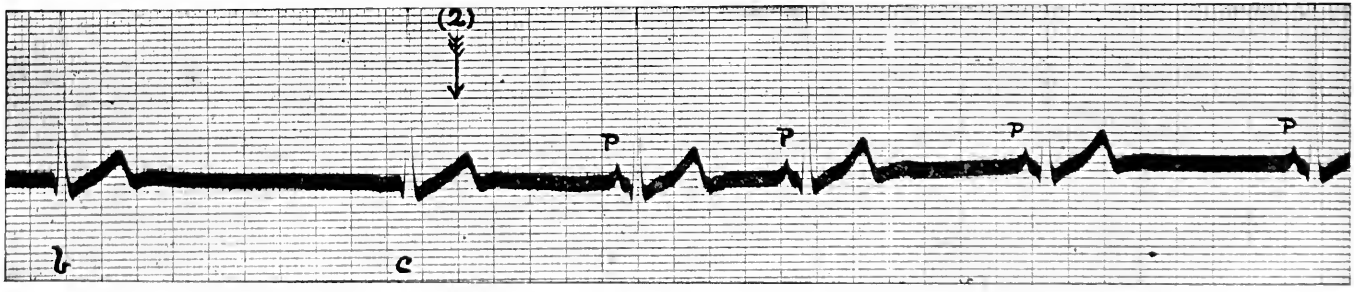

FIGURE 156

Figure 156 is a continuation of Figure 155. Arrow (1) indicates the beginning and arrow (2) the termination of right ocular pressure in a young man of the "hypervagotonic type." Note complete cardiac standstill with "ventricular escape" (*a*), (*b*) and (*c*). On the release of pressure the sinus node reasserts its function of pacemaker.

of atropine; they do not react to pilocarpin, but respond to epinephrin with tachycardia, hypertension and glycosuria. As a rule, the "hypervagotonics" show a marked response to vagus and ocular pressure; in the "hypersympathicotonics" these reflexes are diminished or absent. Such studies assist us in formulating our impressions as to the relative importance of the extracardial reflexes in modifying the cardiac activity in the individual case. With such abnormal tone either of the vagus or of the sympathetic mechanism, one may feel less suspicion that an altered myocardium is responsible for the changed heart activity.

Figures 155 and 156 are parts of a continuous curve and afford a record of the effect of right ocular pressure secured in a young man of 22 of the hypervagotonic type. His usual heart rate was under 60, with a moderate degree of sinus arrhythmia. He was the subject of attacks of asthma, gastric hypersecretion and hyperacidity and constipation. At the point in the electrocardiogram marked by the arrow, (1) pressure was made on the right eyeball, which was released at (2). After the beginning of the pressure there was one normal heart beat; from this point until the pressure was released, the auricle was in complete arrest, as indicated by the absence of the *P* wave. After a complete cardiac standstill of 3.4 seconds, there is a "ventricular escape," (a) followed by two similar phenomena (b) and (c) with intervening periods of standstill each somewhat over two seconds.

The "ventricular escape" is of such a form that we feel assured that it originated from a point high up in the *A-V* bundle, probably in the region of the *A-V* node. It is another illustration of the ability of the cells of the myocardium other than the sinus node to initiate contractions.

On the removal of the ocular pressure, the sinus node resumes its pacemaking function and auricular contractions (*P*) reappear.

The most common irregularity of the heart which is due to vagus influences is the "*respiratory sinus arrhythmia*" of children and of young adults (Figures 157, 158, 159 and 160). This is frequently discovered when palpating the radial. It consists in a rhythmic lengthening and shortening of the heart cycles coincident with the respiratory movements of the chest; the longest cycles usually appear in expiration; during inspiration the cycles are per-

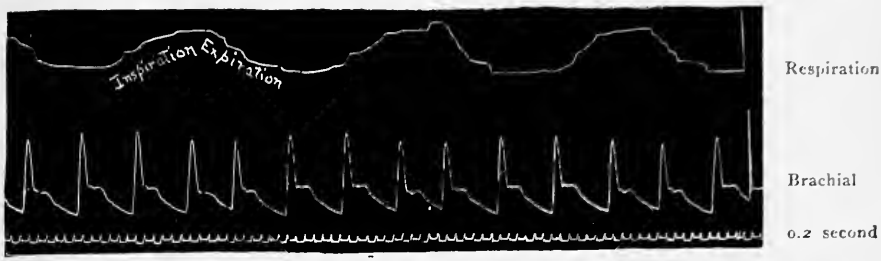

FIGURE 157

Respiratory sinus arrhythmia. Upper curve respiratory movements. Lower curve brachial pulse. Time 0.2 second.

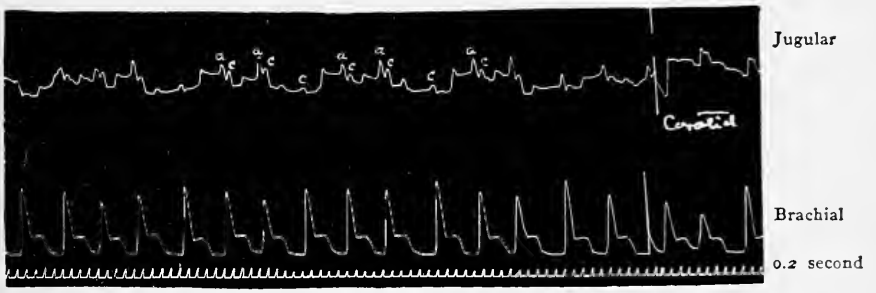

FIGURE 158

Respiratory sinus arrhythmia. Upper curve jugular pulse. Lower curve brachial pulse. Time 0.2 second. Rhythmic variation in length of cycles varies with respiration. The auricle participates in the irregularity, the *a-c* interval is normal.

ceptibly shortened. Forced respiration may produce an arrhythmia hitherto unnoticed or may exaggerate an irregularity which has been present during natural breathing. The most pronounced modification can be secured by having the patient take a full inspiration and hold the breath for 15 to 30 seconds; while holding the inspired air the pulse becomes slow and suddenly quickens when respiration is resumed.

Figure 161 is the electrocardiogram of a boy of 12 who ordinarily showed a mild degree of sinus arrhythmia. To verify the nature of the irregularity, under instructions he drew a deep inspiration at (1) and held his breath; at (3) expiration was allowed. The slowing of the auricles and of the whole heart is quite evident. There was a distinct change in the form of the *P* wave while the breath was held (2) and this did not recover its normal contour until natural breathing was resumed (4).

This is the only common forme of cardiac irregularity met with in young children and, hence, has been named by Mackenzie the "youthful arrhythmia." It is also not infrequently seen in young adults, particularly in those with a rather unstable nervous organism. The irregularity is due to changes, induced by varying degrees of intrathoracic pressure, in the vagus influences conveyed to the sinus node, thus modifying the rate of impulse formation of the pacemaker of the heart.

This irregularity is sometimes in evidence in patients showing abnormal types of respiration. Figure 162 was obtained from a man of 50 with an advanced grade of nephritis exhibiting Cheyne-Stokes respiration. The change in pulse rate during the transition from deep breathing to a period of apnœa is illustrated in the tracing. During the active respiratory movements, the heart cycles occupied 1.4 seconds; during apnœa, the rate increased so that each cycle consumed approximately 0.6 second. It is probable that in this case the change in vagus tone is central in origin, depending on the accumulation of carbon dioxide in the blood; the heart has endeavored thus to compensate for the disordered respiratory function.

The graphic records of a case in which the arrhythmia was due to peripheral stimulation of the vagus, are presented in Figure 163. This was secured from an extremely neurotic young man of

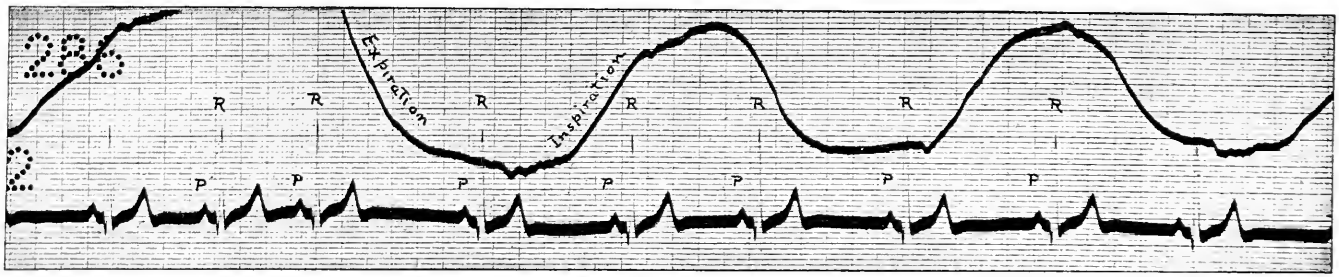

FIGURE 159

Respiratory sinus arrhythmia. Respiratory curve and electrocardiogram. The *P-R* interval is normal. Time intervals 0.04 second.

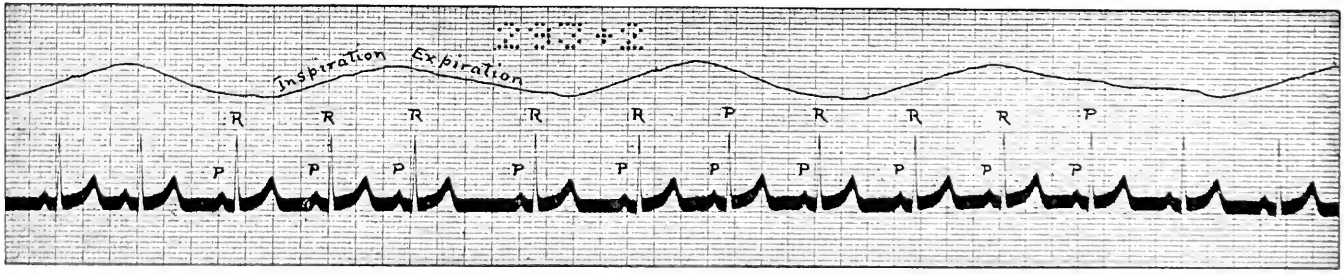

FIGURE 160

Respiratory sinus arrhythmia. Respiratory curve and electrocardiogram. Time intervals 0.04 second. *P-R* interval normal, does not vary. *P* is somewhat smaller when cycles are lengthened.

22, who was seen in consultation because it was thought that he was suffering from auricular fibrillation on account of the complete irregularity of the pulse. The upper curve of the record was obtained by placing a receiving cup over the upper part of the neck. The movements are due to the efforts of the patient to swallow and expel air from the stomach. The change in the cardiac cycles are clearly synchronous with these irregular movements of the œsophagus. Electrocardiograms showed conclusively that the complete irregularity of the heart was not due to auricular fibrillation, but was caused by the varying rate of stimulus production in the normal pacemaker, induced by the spasmodic muscular movements during "cribbing."

The *identification* of this form of irregular pulse is usually a simple matter. The rhythmic change in the length of the cycles is synchronous with respiration, and may be exaggerated by forced respiratory movements. The pulse waves usually show very slight variations in size; the only noticeable departure from the normal is the rhythmic variation of the intervals between the beats. The cardiac irregularity is of the same nature, first and second sounds follow each other at normal equal intervals, but the time interval between the second and first sounds shows a rhythmic change in length. The venous pulse (Figure 158) shows a normal succession of *a*, *c* and *v* waves, but the intervals between these groups show the same grade of irregularity as the arterial pulse. The electrocardiogram (Figures 159, 160 and 161) presents a series of normal auricular and ventricular complexes. The ventricular curves are of normal duration, and the irregularity shows a departure from the usual physiological rhythm only in a variation in the length of the diastolic period.

From the evidence presented in regard to the relative influences of the right and left vagus nerves on the sinus node and on the junctional tissues, it may be inferred that the right vagus is the most important factor in producing this irregularity. Exceptionally one meets with a case of "sinus arrhythmia" in which the polygram shows an *a-c* interval of varying length, the electrocardiogram a similarly changing *P-R* interval. This indicates that the passage of stimuli from the auricle to the ventricle has been delayed and in these one is led to the conclusion that the tone of

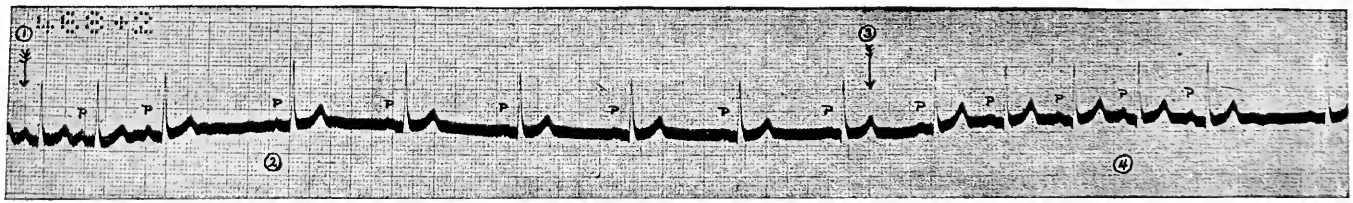

FIGURE 161

Sinus arrhythmia in a boy of 12. At (1) he took a deep inspiration and held his breath; after 8 seconds at (3), expiration occurred and normal breathing was resumed. Note change in form of *P* complex, which only regains its normal shape at (4).

the left vagus also is being rhythmically modified, thus affecting the rate of the ventricular response.

The *clinical significance* of "respiratory sinus irregularities" is very slight. The important element is the recognition of their true character, thus distinguishing them from the forms of abnormal cardiac activity of a more serious nature.

Patients showing this arrhythmia do not develop cardiac insufficiency which can be attributed to the irregularity. It is frequently met with in neurasthenics in whom no other evidence of cardiac abnormality can be found at the time, or subsequently. In the majority of children it will spontaneously disappear before puberty. It has been studied in robust school boys, soldiers in training and other healthy individuals. It is not indicative of myocardial disease and requires no treatment. No drugs or other therapeutic measures are needed, nor should those who present this symptom limit their customary activities.

There is another considerable group of *sinus arrhythmias which bear no relation to the respiratory movements.* These disorders of the heart mechanism have been classified in subgroups by Lewis as follows: (1) sudden cessation of the whole heart beat; (2) phasic variations of pulse rate, in which a retardation and subsequent gradual acceleration of the whole heart occurs; (3) an irregularity of the whole heart in which shorter and longer pauses are mingled indiscriminately.

(1) Sudden cessation of the whole heart beat. This is a form of irregularity which is seen with extreme rarity. It has been known as "sino-auricular block," since, on rather theoretical grounds, it was supposed to be due to an interference with the passage of the impulse from the sinus node to the auricular tissues. The phenomenon consists in the dropping out of a single beat; that is to say, there is a pause during which there is no evidence of activity of any part of the heart. The length of this pause is commonly a trifle less than two beats of the usual rhythm. Since at present we have no means of detecting the activity of the sinus node other than the effects which it has on the auricle, there seems to be no direct way of establishing the fact that the node continues its normal activity but that its stimulus is interrupted on the way to the auricle. From the close similarity which

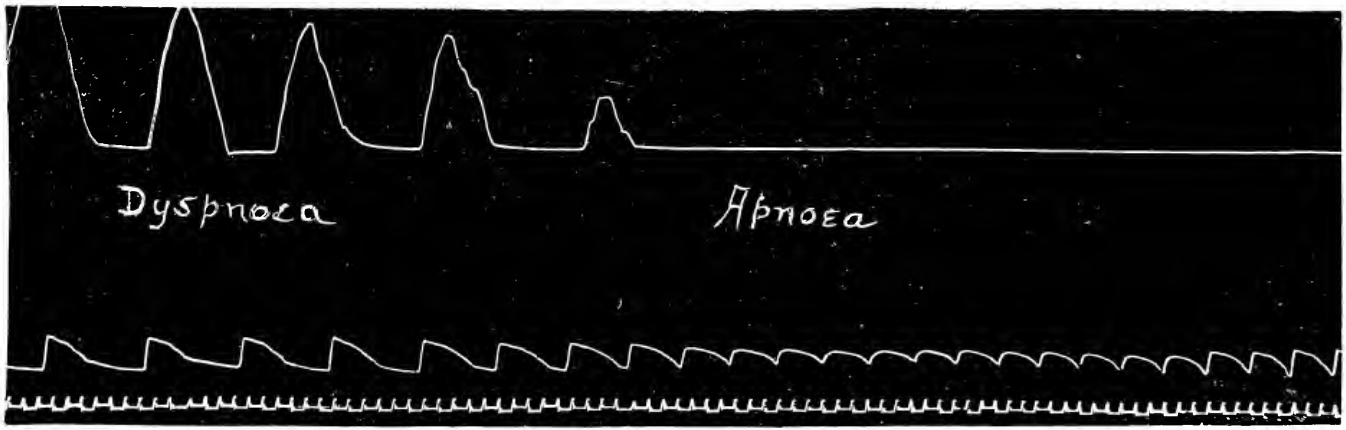

FIGURE 162

From a case of nephritis with Cheyne-Stokes respiration. Upper curve respiratory movements. Lower curve brachial pulse. Time 0.2 second. Rate of heart during apnœa 100, at height of dyspnœa about 43 per minute.

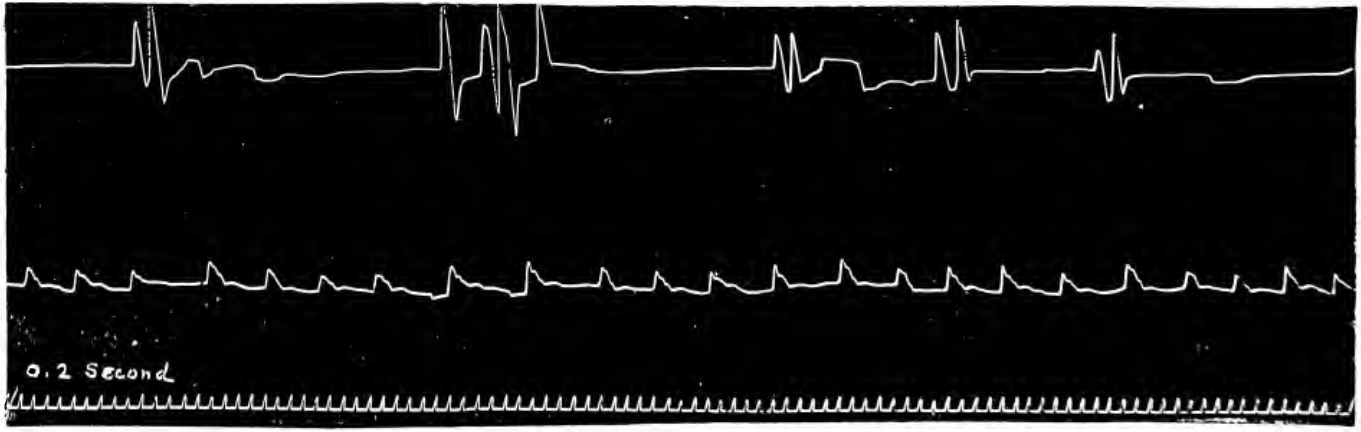

FIGURE 163

Sinus arrhythmia from a "cribber." The upper curve represents the movements of the neck when the patient was swallowing air. The lower curve, the brachial pulse, simulates the complete irregularity of auricular fibrillation.

this phenomenon seems to bear to the irregularities (*phasic variations of pulse rate*) discussed in the next paragraph, since these two types of irregularity are seen in the same patient, since in certain cases (Eyster and Evans)* it can be induced by pressure on the right vagus nerve and abolished by atropine, it seems to me there is little doubt that it is a vagus effect and the term "sino-auricular block" should be dropped as inconsistent with the evidence which we possess at the present time.

This irregularity can be surely recognized only by instruments of precision. The polygraph shows an absence of ventricular activity in both arterial and venous tracings. The jugular record shows no evidence of an *a* wave during the pause.

The electrocardiogram merely shows a pause nearly or quite equal to two cycles of the usual cardiac rhythm without evidence of either auricular or ventricular activity.

This irregularity is usually associated with other evidences of abnormal cardiac function; most of the reported cases have shown some degree of auriculo-ventricular block.

Figure 165 is the record (lead I) of a man of 55 with evidences of myocardial degeneration. At other times he had showed extreme grades of cardiac arrhythmia with many ventricular extrasystoles, paroxysmal tachycardia, etc., etc. At the time the record was taken he had recovered to a considerable degree and the heart was for him fairly regular and for weeks showed only the abnormalities here presented. It is to be noted that the *P* wave is slightly diphasic, that the *R* wave is reversed, that the whole ventricular complex has a very abnormal form and the *P*-*R* interval is excessively long. One "dropped beat" is evident in the electrocardiogram. The interval including the "dropped beat" (R_3 to R_4) is a trifle less than the length of two cycles of his usual rhythm (R_1 to R_2).

The record (Figure 164) of a young girl suffering from rheumatic pericarditis shows a prolonged standstill of the heart. At other times the change in the length of the cardiac cycles was less abrupt, so that one would have included her arrhythmia under group 2, "phasic variations of the pulse rate"; indicating, as we have already suggested, that no sharp line may fairly be drawn between these

*Arch. Int. Med., 1915, xvi, 832.

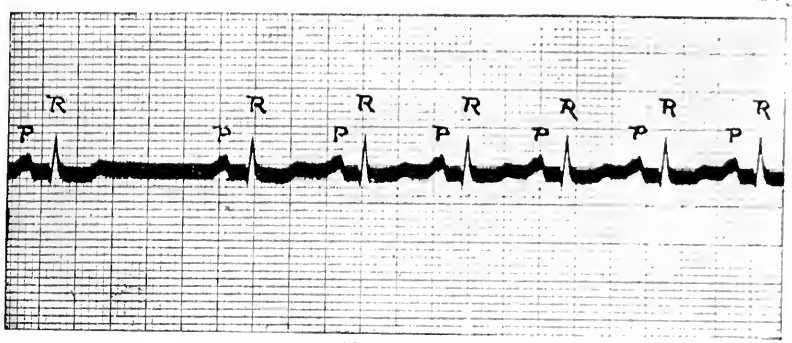

FIGURE 164

Sinus arrhythmia not respiratory. "Dropped beat." From a young woman with rheumatic pericarditis. *P-R* interval=0.2 second.

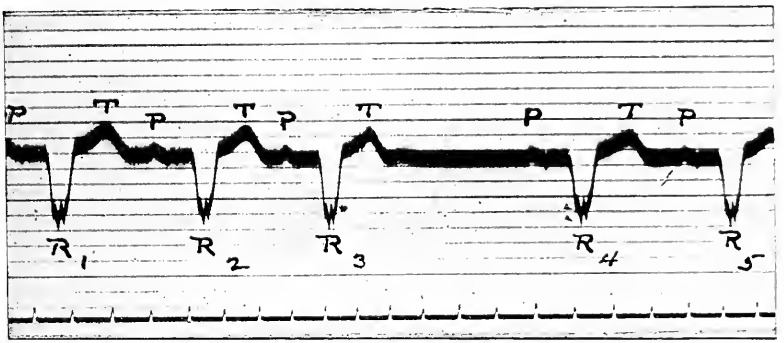

FIGURE 165

From a man 55 years old with myocardial degeneration. "Dropped beat." So-called "sino-auricular block." All the complexes are very atypical (see text). *P-R* intervals 0.22 second. Time 0.2 second.

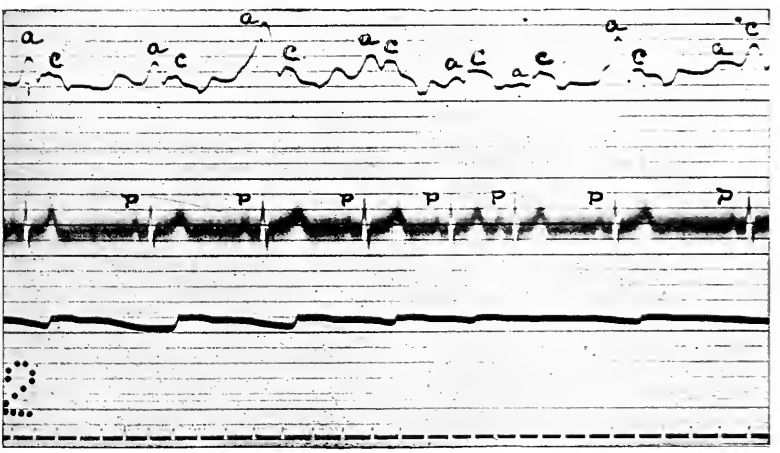

FIGURE 166

Records from above downward jugular tracing, electrocardiogram, brachial tracing, time 0.2 second. From a young woman with hyperthyroidism. The arterial pulse is completely irregular, but both the jugular and electrocardiographic records indicate an auricle with irregular but otherwise normal contractions.

groups. The somewhat prolonged *P-R* interval suggests a hypertonus of the left as well as of the right vagus.

2. Phasic variations of pulse rate (Figures 167, 168 and 169). Here there are alternating periods of rapid slowing and equally rapid acceleration of the heart. In some cases this change is quite rhythmic, the heart beating 10 or 15 times between the individual cycles of the maximum length, in other cases the time elapsing between similar phases is very variable. The variation in the length of the individual cycles is always quite independent of respiratory movements.

Very little is known of the mechanism of this form of irregularity, but the evidence seems to point to vagus influences acting on a sinus node which is functionally damaged. I believe the mechanism responsible for its production is quite similar to that of the single dropped beat described above. The identification may be suspected when a rhythmic series of quickening and slowing heart beats is detected by palpation or auscultation. The type of irregularity is securely established by graphic records.

Figure 167 is the record of a man with general arteriosclerosis, with a rhythmic change which occupied about 18 heart cycles. Figure 168 is from a girl of eleven with acute endocarditis; when the acute process subsided the arrhythmia disappeared. The electrocardiogram of a boy of fourteen with acute rheumatic endocarditis and arthritis is reproduced in Figure 170. The varying length of the *P-R* interval in this record is suggestive either of excessive activity of the left vagus or of an *A-V* node peculiarly susceptible valescence, but reappeared coincident with a recurrence of the arthritis.

3. Irregularity of the whole heart in which shorter and longer pauses are mingled indiscriminately. This arrhythmia closely simulates the pulse features of complete irregularity of auricular fibrillation of the slow type (Figures 163 and 166). But the mechanism of its production is quite different. The sino-auricular node acts as the pacemaker of the heart, but the rhythmic character of the formation of stimulus-material is disturbed, probably due to the reception of impulses varying in intensity which reach the sinus node through the right vagus. The auricle responds irregu-

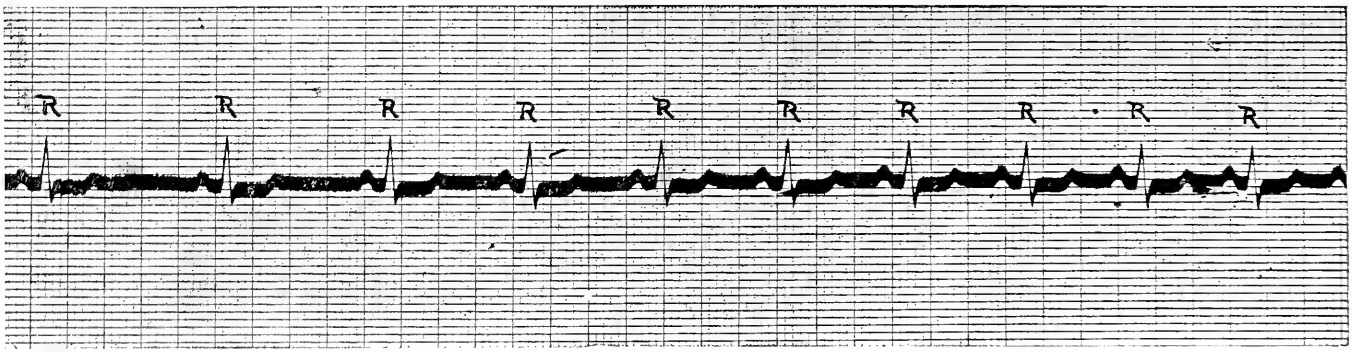

FIGURE 167

Sinus arrhythmia. Phasic variations independent of respiration. From a man with general arteriosclerosis.

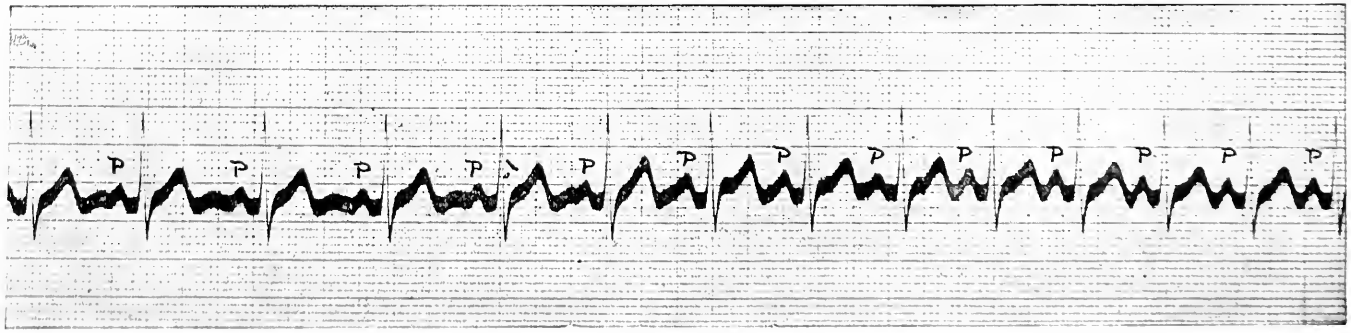

FIGURE 168

Sinus arrhythmia with phasic variations independent of respiration. From a girl of 11 with acute endocarditis.

larly and the ventricle follows the auricular contractions with a corresponding arrhythmia, but with no other abnormal features. In short, the whole heart assumes the arrhythmia of the pacemaker, but the chambers respond sequentially and otherwise normally.

Recognition of this type of irregularity by the ordinary physical signs is difficult. If the pulsation of the veins of the neck are prominent, one may be able to detect the presystolic *a* wave, indicative of coördinated, but arrhythmic, auricular activity. Barring this resort must be had to graphic records. Both polygrams and electrocardiograms show nothing abnormal except the unequal diastolic periods. The *a*, *c* and *v* waves of the jugular record show the normal relationship (Figure 166 and 169). The auricular and ventricular complexes of the electrocardiogram have a normal sequence and the usual form (Figures 166 and 171). The polygram is to be distinguished from that of auricular fibrillation by the presence of the auricular as opposed to the ventricular form of venous pulse; the electrocardiogram shows a *P* wave of normal type and an absence of the small diastolic oscillations so characteristic of auricular fibrillation. The condition is to be distinguished from the auricular extrasystole by the absence of intervals which represent pauses of a compensatory character and by the normal contour of the *P* wave in the galvanometric records.

The polygraphs (Figures 166 and 169) show the complete irregularity of the arterial pulse, an auricle which is active and equally arrhythmic, a considerable variation in the length of the diastolic period and the normal sequential relation of auricle and ventricle.

Figure 166 was from a young woman of 20 with symptoms which suggested hyperthyroidism, but with no evidence of myocardial disease other than the arrhythmia. The electrocardiogram (Figure 171) was obtained from a woman of 25 during an attack of acute rheumatic endocarditis.

CLINICAL FEATURES AND SIGNIFICANCE

Sinus irregularities which bear no relation to respiration are seen more frequently in the young, but are not limited to this period of life. They may be discovered accidentally in those who show no other evidence of disease. They are occasionally seen in the period of convalescence following the acute infectious diseases. They are

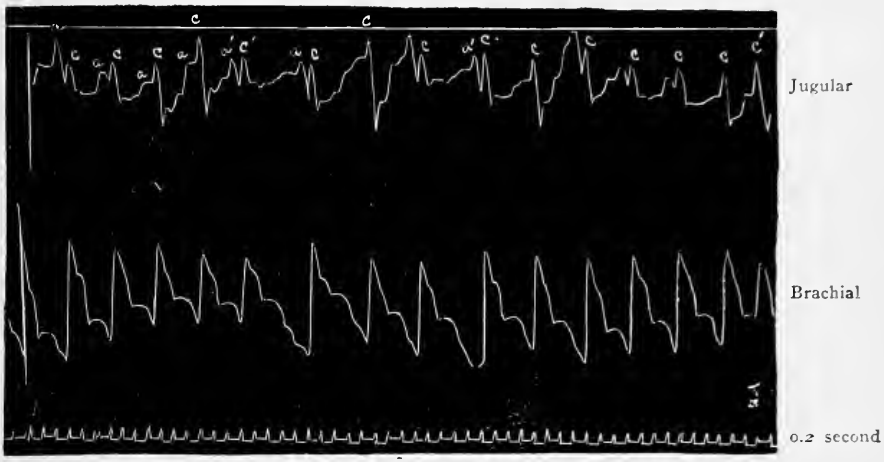

FIGURE 169

Brachial and jugular tracings from a young woman with a completely irregular pulse. Sinus arrhythmia independent of respiration.

sometimes associated with the administration of large doses of digitalis.

These irregularities are usually seen in association with a slow pulse rate, sometimes they are abolished by exercise or a febrile condition in which the pulse is accelerated. Most of them show a very prompt response to vagus or ocular pressure (Figures 155 and 156). Frequently they disappear under the administration of atropine.

At the Presbyterian Hospital, in the routine examinations during the past year, we have studied and recorded graphically seventeen cases of sinus arrhythmia in which the irregularities were quite independent of respiratory movements. Eight of these cases were associated with other forms of arrhythmia suggesting a myocardial defect, such as extrasystoles, partial auriculo-ventricular block, etc. The nine remaining cases belonged exclusively to the type of irregularity now under discussion. Among these there was one adult and one seventeen; the rest were all under fourteen years of age. Six were males and three females. Of these nine cases, five had physical signs of definite endo- or pericardial lesions; one was an overgrown athletic boy with a moderate degree of cardiac hypertrophy, one had chorea; the remaining two showed no definite signs of disease, although one was slightly dyspnœic and the other was of a high-strung, nervous temperament. In two of these cases the irregularity disappeared, in the others it still persisted at the time of the last examination.

Clinically, it is important that we should recognize the nature of these arrhythmias and distinguish them from the types of more serious moment with which they may be confounded.

Such an activity of the heart is in itself, I believe, not a serious matter, but my present impression is that it indicates a definite myocardial change which may be very transitory, but which may well bear careful observation.

The exciting causes are undoubtedly the nervous impulses conveyed to the heart through the vagi, but these are probably peculiarly effective, since they are acting on a heart which is damaged and, hence, unusually susceptible to outside influences.

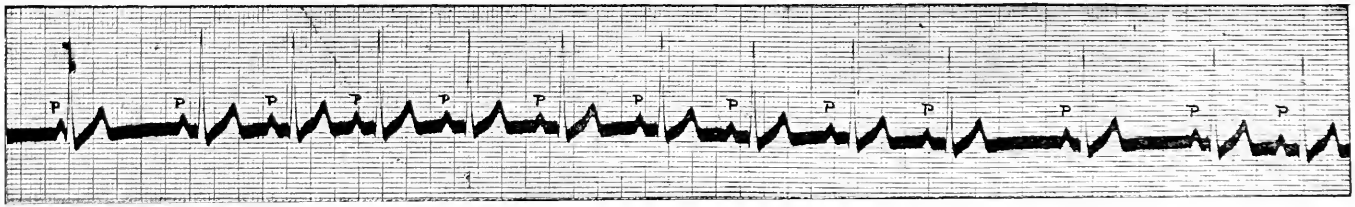

FIGURE 170

Sinus arrhythmia. Phasic variations independent of respiration. Note variations in the length of the *P-R* interval suggesting functional changes in the *A.V.* node or bundle.

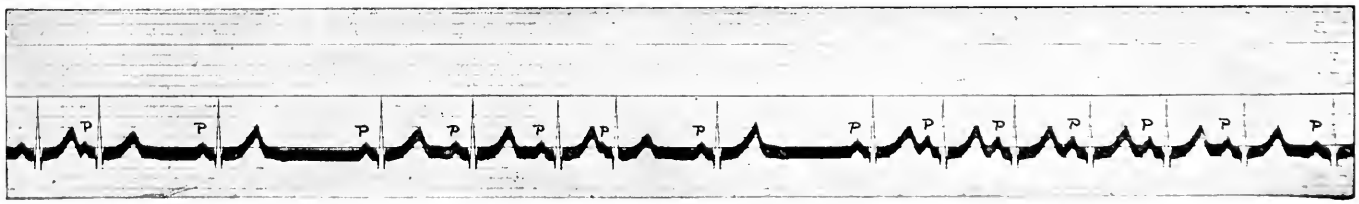

FIGURE 171

Sinus arrhythmia. Complete irregularity. From a woman during an attack of acute endocarditis.

CHAPTER XV

Mixed Arrhythmias

In the preceding pages the arrhythmias have been discussed as individual types. This is the usual form in which they are seen in the clinic and it is, perhaps, simpler to study them primarily from this standpoint.

It has been pointed out from time to time how close is the association of some of the types; for example, that a single heart may pass through the phases of extrasystole, auricular flutter, fibrillation and paroxysmal tachycardia and repeat any one of these abnormal types of functional activity.

There are other instances by no means rare in which the arrhythmia depends upon the alteration of more than one of the fundamental functions of the cardiac muscle. Changes in the rate of stimulus formation at the sino-auricular node are not infrequently associated with a defect in the capacity of conduction in the bundle of His. Increased irritability of various portions of the musculature, as evidenced by extrasystoles or fibrillation, is often found associated with a depression of the property of conduction. One might spend considerable time in enumerating the various combinations which are seen, but it will suffice us to discuss several of the more common and distinctive types of the mixed arrhythmias, bearing in mind that these by no means exhaust the assortment that are encountered.

Perhaps the most common type of the mixed irregularities are those in which *a definite sinus arrhythmia is associated with a defect in the conductivity of the A-V bundle.* The electrocardiogram of a well-marked case of this group is shown in Figure 172. The lower line of numerals represent the measurements in fractions of a second of the *P-R* intervals, the upper the *P-P* intervals. The *P-R* time was usually excessive in this case and in the record shows a variation in length from 0.18 to 0.43 second. The *P-P* intervals, which indicate the rate of stimulus formation at the sinus, also presents a variation in length with a tendency to a rhythmic, gradual increase followed by a diminution in the length of these

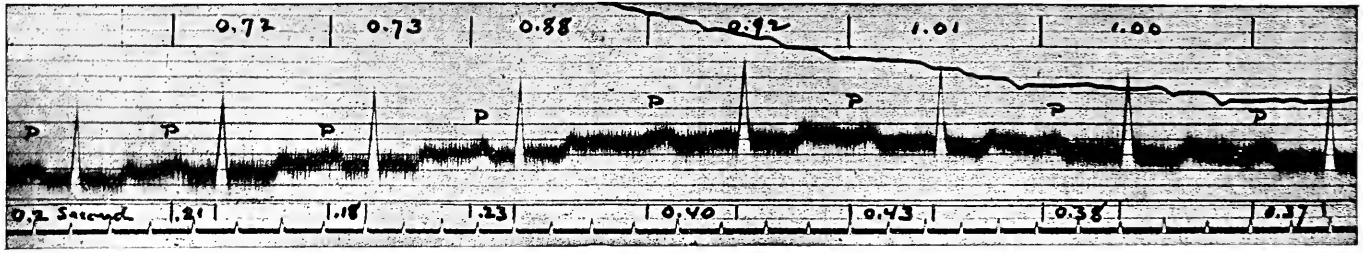

FIGURE 172

Delayed conduction and sinus arrhythmia. The upper row of figures indicate the length in seconds of the auricular (*P-P*) intervals. The lower row of figures indicate the length of the conduction (*P-R*) intervals.

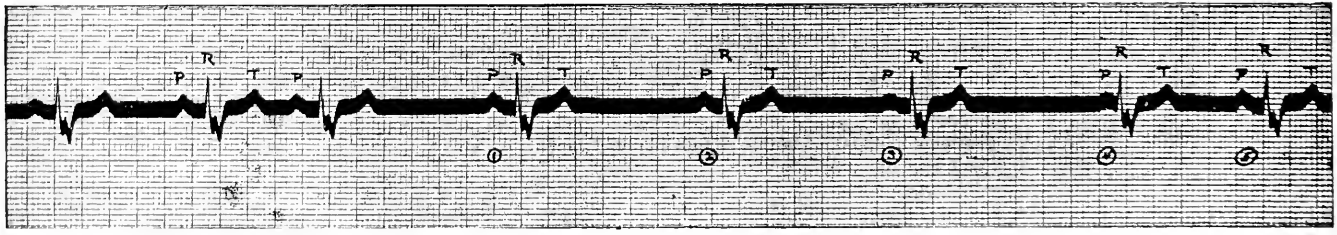

FIGURE 173

Sinus arrhythmia and *P-R* intervals of varying length. Gradual change in the form of the *P* wave.

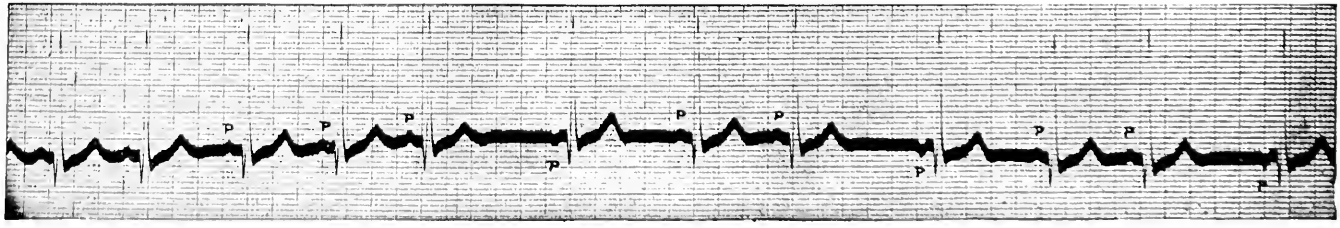

FIGURE 174

Sinus arrhythmia and change in the form of *P*, suggesting a new point of origin for the auricular stimuli.

intervals. The extreme differences are about one-third of a second. The ventricular arrhythmia is the resultant of these two factors and the cycles occupy from 0.69 to 1.01 seconds. The arrhythmia was accentuated by forced respiratory movements. Just before this curve was secured the patient took a deep inspiration and held the breath; the slow expiratory movement is indicated by the portion of the respiratory curve in the latter half of the record. It is quite probable that the arrhythmia is in a large measure due to vagus influences, the sinus activity varying with the tone of the right vagus, the conductivity of the bundle changing with the tone of the left vagus (see Chapter XIV on the Influence of the Extracardial Nerves).

In Figures 173 and 174 are exhibited the electrocardiograms of two cases in which *sinus arrhythmias are associated with changes in the contour of the P waves*, indicating a shifting of the pacemaker from the sinus node to some other point in the auricular wall. In Figure 173 a small, but definite, change in the *P* deflection (1, 2, 3, 4) is accompanied by a shortening in the *P-R* interval; at 5 the *P* wave recovers the form characteristic of the normal pacemaker and the *P-R* interval measures 0.2 second.

Figure 174 presents less variation in the *P-R* interval, but a more marked dislocation of the pacemaker is suggested by the complete inversion of the auricular complex.

A functional abnormality of this character indicates a mild degree of auricular myocardial defect, not in itself sufficient to embarrass the patient, but which serves as a signal to the physician that unless stationary it may be the forerunner of more serious damage, with corresponding mischief to the circulation.

A mixed arrhythmia, which is extremely common, is *the association of ventricular extrasystoles with auricular fibrillation.* It is met with not infrequently in subjects with auricular fibrillation to whom digitalis has been administered in considerable doses. It is also seen in those who have never received digitalis. Most often the extrasystoles occur at infrequent intervals and are all of one type. The record shown in Figure 175 was obtained from a patient who was not taking digitalis. It presents rather an extreme degree of the irregularity. The extrasystoles are of three distinct types, indicating as many points of abnormal ventricular irritability. By

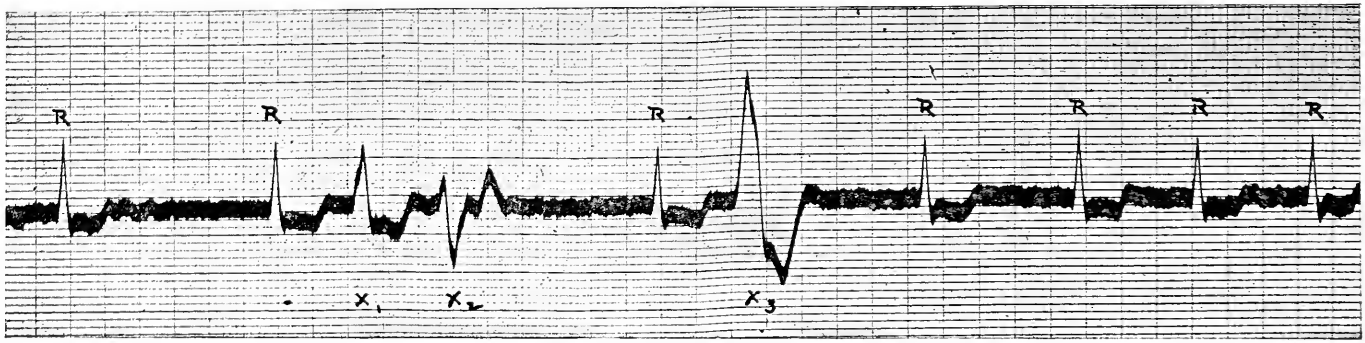

FIGURE 175
Auricular fibillation, complicated by ventricular extrasystoles. x_1, x_2 and x_3, three distinct types of extrasystoles.

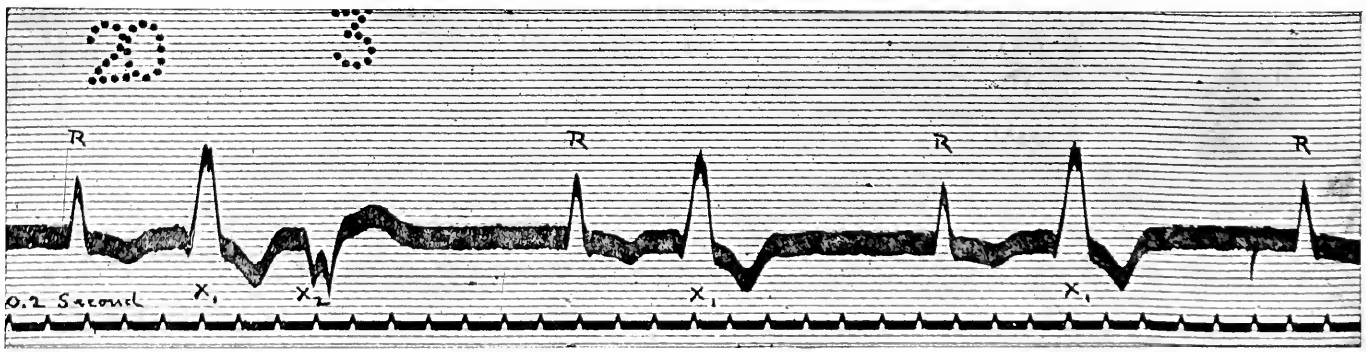

FIGURE 176
Auricular fibrillation, complete *A-V* block and ventricular extrasystoles of two types. For polygram of this case see Figure 177.

means of the ordinary methods of physical examination, one may find considerable difficulty in correctly interpreting this type of irregularity. One who has studied a great many hearts of this kind learns to distinguish by auscultation certain contractions which differ from the majority by their "flopping" character, and these he may suspect to be extrasystoles. They can only be surely analyzed with the aid of electrocardiographic curves.

The appearance of a large number of ventricular extrasystoles in a case of auricular fibrillation indicates that, in addition to an irritable auricle, we have an abnormally irritable ventricle. The muscle damage is very extensive. When the extrasystoles arise from many foci, we must conclude that the lesions are even more widely distributed. Nearly all of these patients have a severe grade of cardiac insufficiency which is extremely difficult to control.

In discussing the prognosis of auricular fibrillation, it was pointed out that the future of the patient depended to a very large degree on the integrity of the ventricular muscle. Here we have clear evidence that the ventricular wall is extensively diseased. The gravity of the prognosis increases with the multiplication of the number and types of ventricular extrasystoles.

AURICULAR FIBRILLATION AND HEART BLOCK

In 1909 James Mackenzie* described a group of four cases presenting a slow rhythmic pulse which suggested a complete heart block, but in which polygraph curves failed to reveal any auricular activity. He suggested that there was also in these cases a bradycardia of the auricle and that the auricles and ventricles were contracting simultaneously in response to a stimulus arising in the region of the *A-V* node. Accordingly, he designated this type as "nodal bradycardia." This acute observation was soon followed by an exhaustive study of one of the cases of this group by Lewis and Mack,† using Einthoven's galvanometer. They were able to show that the auricle was in a condition of fibrillation and that the ventricles had assumed the slow ideo-ventricular rhythm ordinarily seen in complete heart block. The post-mortem examination of this

*Heart, 1909-10, i, 23.

†Quart. Jour. Med., 1909-10, iii, 273.

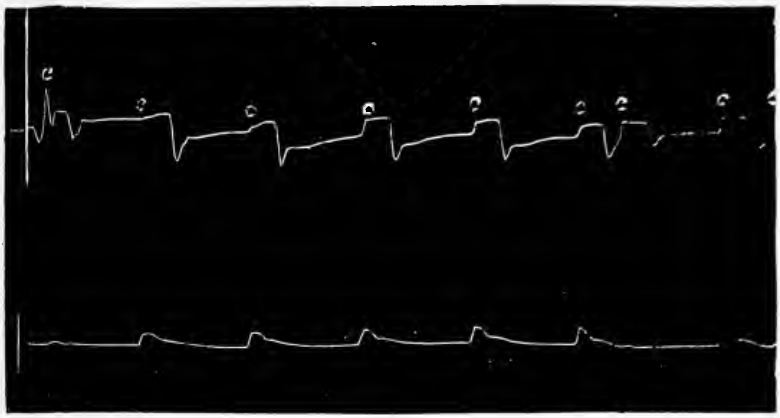

FIGURE 177

Auricular fibrillation, complete *A-V* block and ventricular extrasystoles. Rate 35. For electrocardiogram of this case see Figure 176.

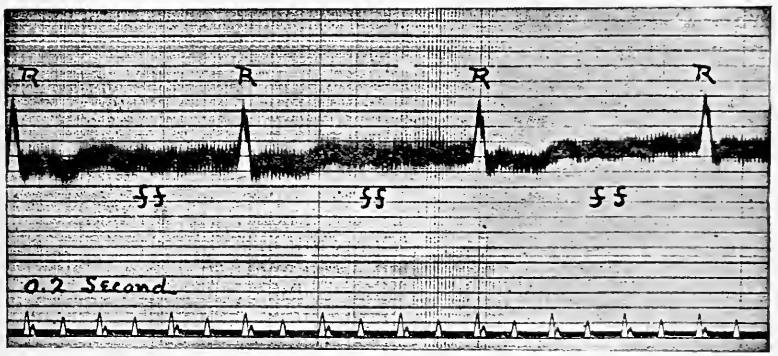

FIGURE 178

Auricular fibrillation and complete *A-V* block. Note rhythmicity of ventricular complexes

heart‡ revealed a lesion completely severing the bundle of His, and organic damage to the auricular wall sufficient to explain the existence of auricular fibrillation.

It has been my fortune to observe two cases presenting this symptom complex. One of these died while under observation, but permission for an autopsy could not be obtained.

The mechanism of this rather unusual functional activity seems reasonably clear. There are no coördinated contractions of the auricles; they are in a state of fibrillation and, as is usual in this condition, irregular impulses are being constantly showered on the functional tissues. Since, however, there is a complete functional severance of the *A-V* bundle, all these impulses are blocked and none of them reach the ventricle. Thus cut off from stimuli derived from the upper portions of the heart, the ventricle initiates its own stimuli and a typical ideo-ventricular rhythm becomes established.

Among the etiological factors found have been syphilis, rheumatism and general arteriosclerosis. As in other conditions of block, the association of mitral disease is more common than other valvular lesions.

The effect of digitalis and drugs of the same order in producing a complete block in cases of auricular fibrillation will be considered in another place.

Of the cases reported in the literature only one (Mackenzie, case 4) was under fifty years of age, the others were from fifty-one to sixty-eight years old. It is seen less frequently in women than in men.

Complete block will, of course, be inferred when the ventricles are found to be beating rhythmically at a rate in the neighborhood of thirty per minute. When a rhythmic pulsation of the jugulars two or three times the rate of the ventricles is absent, an associated auricular fibrillation may be suspected.

In the polygraph the arterial records show a slow rhythmic rate of about thirty per minute and a corresponding rhythmic jugular of the ventricular form. All *a* waves are absent and there is no evidence of gross auricular activity (Figure 177).

The electrocardiogram (Figure 178) presents a slow rhythmic

‡Cohn and Lewis: Heart, 1912-13, iv, 15.

contraction of the ventricle characteristic of complete block, an absence of all P waves and the small undulations (ff) pathognomonic of auricular fibrillation.

The galvanometric record of another case is shown in Figure 176. Here the activity is further complicated by ventricular contractions (x_1, x_2) originating in abnormal points in the ventricular wall, indicating an excessive irritability. The record is suggestive of the effect of digitalis when administered in large doses to cases of auricular fibrillation, but, in this instance, neither digitalis nor any other drug of this group was being given. The abnormal activity was entirely due to the myocardial defects. In this case the administration of atropine produced a considerable increase in the ventricular contractions of the normal type (R).

Further details have been reported in a paper on "Functional Heart Block."* A polygram secured from this same patient is reproduced (Figure 177). The arterial pulse is slow, 35 per minute, and rhythmic, except toward the end of the curve, where two extrasystoles appear. The jugular tracing shows an absence of all a waves and is of the ventricular form.

Most of these cases present signs of a considerable degree of cardiac insufficiency, and it is evident that the defective myocardium is unable to maintain an adequate circulation. In neither of the two cases which I have had under observation have there been attacks of unconsciousness or convudsions, but several of the reported cases have exhibited phenomena which allow them to be grouped under the Adams-Stokes syndrome. The convulsions are due, as in other cases of heart block, to cerebral anæmia following an abrupt lowering of the heart rate.

In these cases the myocardial damage is so extensive that it makes the prognosis exceedingly grave. The termination may occur at any time in a convulsive seizure or, as in the two cases which I have been able to follow, the course may be a progressively weakening heart with ultimate failure.

Probably three or four years would be the longest period of life which could be expected after the discovery of such serious myocardial defects.

*Hart: Amer. Jour. Med. Sc., 1915, cxlix, 62.

HEART BLOCK AND EXTRASYSTOLES

An unusual phenomenon occasionally met with in cases of complete block is presented in Figure 179. This was taken from a case, seen in consultation with Dr. Frank Grauer, of Adams-Stokes disease, who died in a convulsion two years after these observations were made. His usual ventricular contractions were perfectly rhythmic and at a rate of thirty per minute, the auricular rate was 86. At rare intervals there appeared a single premature ventricular beat, sometimes two of these presented in succession. That the origin of these extra contractions must have been in the region of the *A-V* node, or, at any rate, high up in the *A-V* bundle, may be inferred from the form of the ventricular complexes of the electrocardiogram (Figure 180), taken on the same day. There are two possible explanations of this activity: (1) The block, which is ordinarily complete, occasionally becomes partial and the ventricle responds with a delay in the conduction period to an auricular impulse; the complete character of the block for most of the time makes this explanation seem improbable. (2) The *A-V* node or bundle is excessively irritable at times and the usual ideo-ventricular rhythm is interrupted by what may be called a nodal or bundle extrasystole. On theoretical grounds, one might assume that such a quickening of the ventricular activity would be an event favoring an improved distribution of the blood. My observations on this patient were not sufficiently prolonged to determine whether such was the case.

The graphic records of another case of heart block are presented in Figures 181 and 182. The polygraph shows a perfectly rhythmic activity of the auricles at a rate of 78 and of the ventricles at a rate of 37, but with a complete dissociation of the upper and lower chambers of the heart. This curve would suggest the ordinary type of complete heart block. When, however, we come to examine the electrocardiographic record (Figure 181) it presents some unusually interesting features. Here, again, there is evidence of complete dissociation of auricles and ventricles. The *P* wave is, however, quite abnormal in form. Instead of a single positive wave, it is diphasic and it is, therefore, probable that the auricular pacemaker is a point in the muscle at a considerable dis-

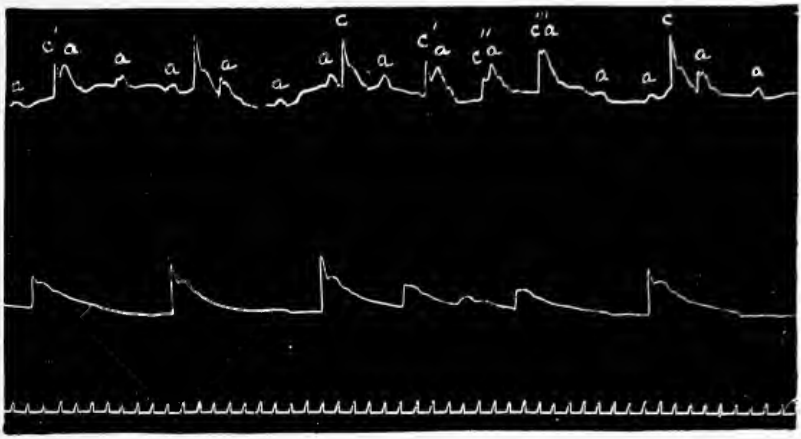

FIGURE 179

Complete *A-V* block. As rate = 86. Vs rate = 30. Complicated by extrasystoles *c'*, *c''*, *c'''*. For electrocardiogram of this case see **Figure 180.**

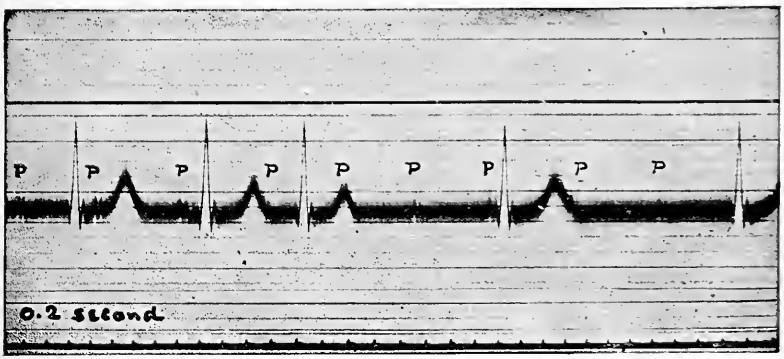

FIGURE 180

Complete *A-V* block and nodal extrasystoles. *As* rate = 86. *Vs* rate = 30. For polygram of this case see **Figure 179.**

tance* from the sino-auricular node. Still more interesting is a study of the ventricular complexes. These are similar in form, but are readily distinguished from the curve of a normal ventricular contraction. The records taken by leads I and II (not here reproduced) showed ventricular complexes of an abnormal type. In lead I they were directed upward, in lead II and lead III (Figure 181) they were directed downward. These complexes conform to the type obtained experimentally by Eppinger and Rothberger,† through a destruction of the left branch of the bundle of His. We, therefore, conclude that in the case under discussion there existed a lesion dividing the main stem of the *A-V* bundle, causing a dissociation of auricular and ventricular activities, and, further, that there was a destructive lesion in the branch of the *A-V* bundle which is distributed to the left ventricle. The ideo-ventricular rhythm, therefore, probably arose, not as is usual from a point in the main stem of the bundle, but in the right ventricular wall.

It is interesting to observe how widely is distributed the property of rhythmicity in the heart muscle. This heart, with a ventricular pacemaker very remote from the normal site, maintained an almost perfect rhythmic activity over long periods of time and, on many occasions, when he was under observation.

*Lewis: Heart, 1910, ii, 27.
†Ztschr. f. klin. Med., 1910, lxx, 1.

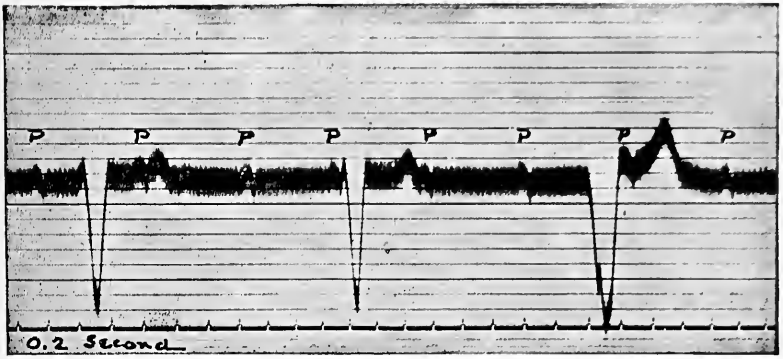

FIGURE 181

Complete *A-V* block with destruction of the left limb of the *A-V* bundle. Note diphasic *P* complex. For polygram of this case see Figure 182.

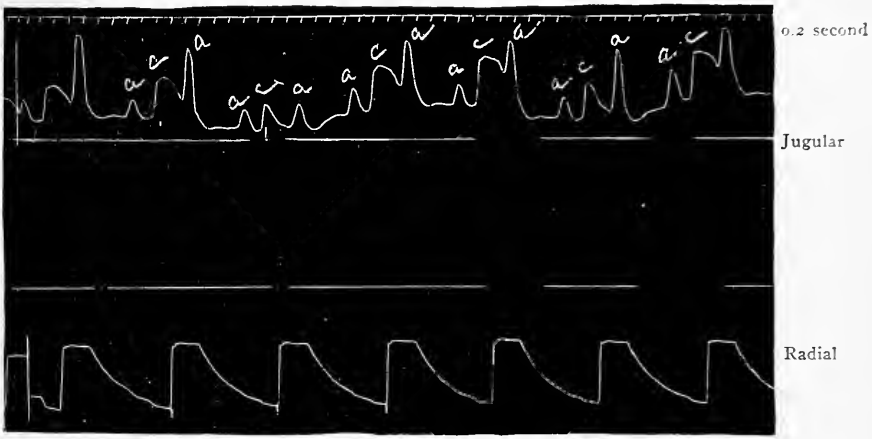

FIGURE 182

Complete *A-V* block. *As* rate=78. *Vs* rate=37. For electrocardiogram of this case see Figure 181.

CHAPTER XVI

Position of the Heart and Changes in the Disposition of the Muscle Mass

A discussion of changes in position of the heart in the chest cavity would lead us outside of the limits which we have set to the subject matter of these pages. Since, however, we have laid considerable stress on the use of the electrocardiogram as a means of studying myocardial function, and have utilized not a small portion of our space in describing the various characters of these records and the means for their analysis, it becomes necessary to examine certain deviations from the usual type which are due to changes in the position of the heart and which, therefore, must be distinguished from those which are due to intrinsic myocardial alternations.

The amplitude and direction of the records obtained by the three leads usually employed in clinical work depend on the algebraic sum of the currents developed in the heart at any given moment, but any one of the leads will record only those currents which pass in the same plane as the line connecting the points from which the particular lead is secured. Einthoven* has shown that if the strength and direction of the current developed by the heart is represented by a line of given length and direction the relative size of the deflections obtained by the three leads will be proportional to the projection of this line on the sides of an equilateral triangle whose sides represent the connections of the points from which the current is derived from the body. This conception is illustrated in the diagram (Figure 183) reproduced by permission from a paper by Pardee.† In this diagram the line xy represents the strength and direction of the original current arising in the heart. The amount of current recorded in each of the leads is measured by the projection of this line on the sides of the triangle formed by connecting the points from which the current is led off. The magnitude of the deflections of leads I, II and III will be proportional to the lines x_1y_1, x_2y_2 and x_3y_3. In the normal heart the

*Lancet, 1912, i, 853.
†Jour. Amer. Med. Assn., 1914, lxii, 1311.

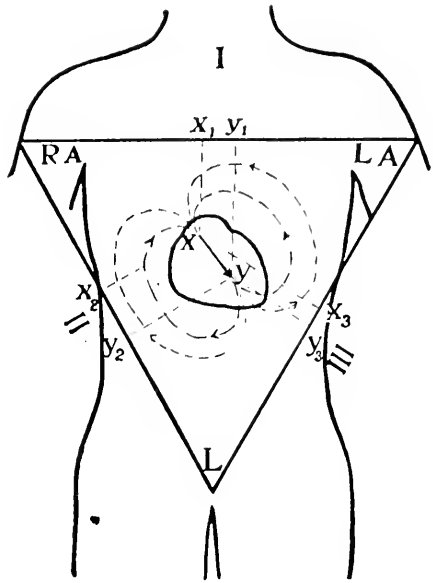

FIGURE 183

After Pardee. Diagram illustrating the relative size of the waves of the electrocardiogram in the different leads obtained from a current which is represented in force by the length and in direction by the position of *XY*.

RA right arm; LA left arm; L left leg. Roman numerals designate the leads represented by the sides of the triangle.

x_1 y_1, x_2 y_2 and x_3 y_3 indicate the relative amplitudes of the reflections secured from the three leads.

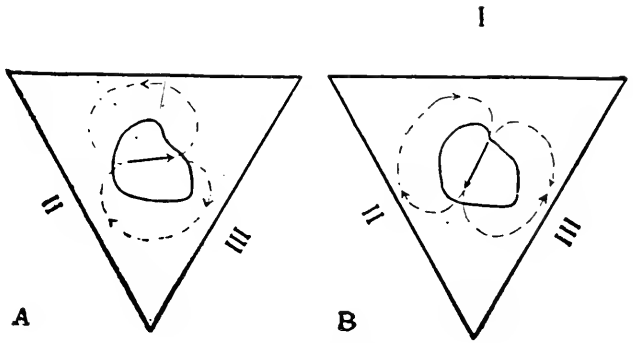

FIGURE 184

After Pardee. Diagram illustrating the variations in direction of the current in the different leads caused by differences in direction of the current developed in the heart.

waves of lead II are larger than those obtained by either lead I or lead II. Einthoven, Fahr and De Waart* and Williams† have demonstrated mathematically that reckoned from a fixed point in the cardiac cycle lead II minus lead I equals lead III, and if two of these values are known the third can be correctly calculated.

The direction of the waves of a given lead depends on the direction of the flow of the current in the heart. This is illustrated by the diagram (Figure 184), in which the effect on the direction of the current in the three leads, due to a difference of direction of currents in the heart, is shown by comparison.

The most common variations which one sees are differences in the relative amplitude of the deflections due to changes in the axis of the heart caused by respiration or an alternation in the position of the body. During inspiration the waves *R* and *T* are smaller in lead I and larger in lead III. When the body is turned from the left to the right side there is a deepening of the *S* wave in lead II. Grau‡ has found that the *S* wave of lead II was made greater by pathological conditions (left pleural effusions, etc.) which displaced the heart to the right.

The records of a case showing an extreme change in the cardiac axis is shown in Figures 185, 186, 187, 188, 189 and 190. The patient had complete transposition of the viscera and the heart was on the right side of the chest. The records were obtained by the following leads: I = right arm — left arm, II = right arm — left foot, III = left arm — left foot, IV = left arm — right arm, V = left arm — right foot, VI = right arm — right foot. It is plain that by leads I, II and III (those usually employed) the waves depart from the normal in direction and in amplitude in the different leads. By reversing the electrodes (leads IV, V and VI) the electrocardiogram of a normal heart is secured.

A one-sided hypertrophy of the heart produces a change in the electrical axis and, hence, a corresponding change in the galvanometric records taken by the three customary leads. This observation was first reported by Einthoven,‖ who called attention to the fact that in many cases of *left hypertrophy* the *R* wave in

*Arch. f. d. ges. Physiol., 1913, cl, 275.
†Amer. Jour. Physiol., 1914, xxxv, 292.
‡Zeitschr. f. klin. Med., 1909, lxix, 281.
‖Arch. Internat. de Physiol., 1906, iv, 132.

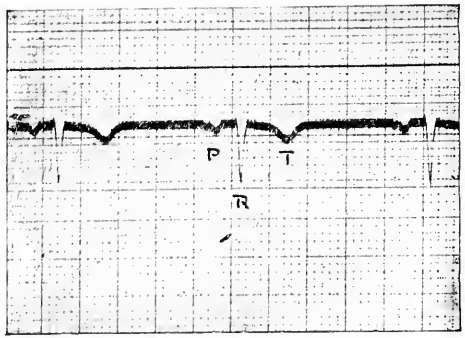

FIGURE 185
Lead I (right arm—left arm).

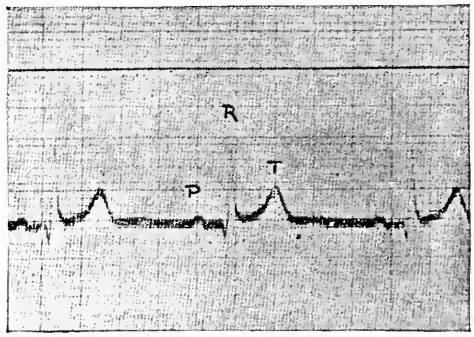

FIGURE 186
Lead II (right arm—left foot).

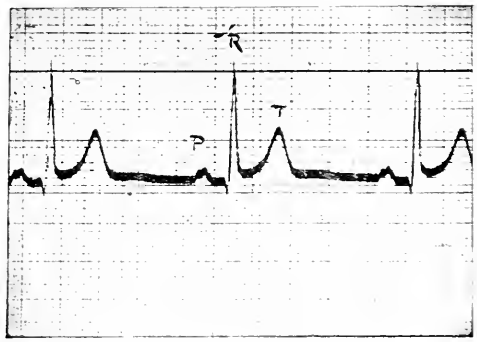

FIGURE 187
Lead III (left arm—left foot).

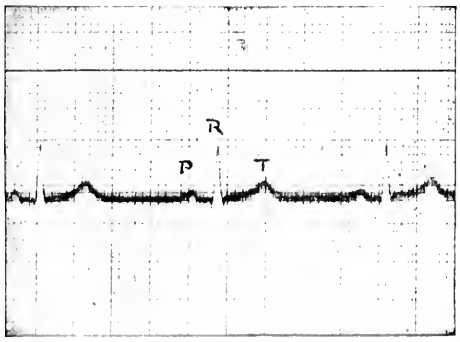

FIGURE 188
Lead IV (left arm—right arm).

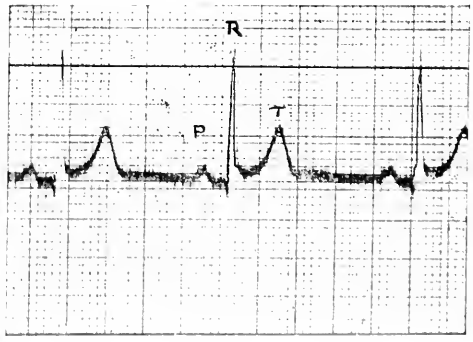

FIGURE 189
Lead V (left arm—right foot).
A case of dextrocardia with transposition of the viscera.

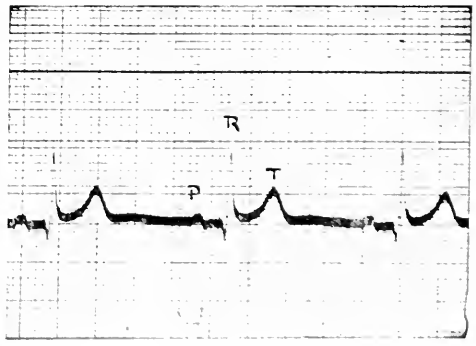

FIGURE 190
Lead VI (right arm—right foot).

lead I was increased in size and in lead III was diminished or even directed downward, while in *right hypertrophy* R was small or negative in lead I and increased in amplitude in lead III. The constancy of this phenomenon has been questioned by Hering and others and a number of apparent exceptions have been discussed in a recent paper by Bridgman.† It is probable that some of the discrepancies in the correlation of the heart condition and the changes in the electrocardiograms have been due to insufficient evidence as to the relative masses of the right and left chambers of the heart. In a very careful study of this question, Lewis‡ has pointed out that the usual methods of examination are quite insufficient to determine the relative degrees of hypertrophy of the right and left ventricles. As a rule, he found that his cases of mitral and pulmonary stenosis, with clinical evidence of right-sided hypertrophy, showed the characteristic electrocardiograms of right hypertrophy, as indicated by Einthoven, while cases of hypertension and aortic insufficiency, with signs of left hypertrophy, showed the graphic evidence ascribed to left hypertrophy. There were, however, a number of discrepancies and these he explains on the ground that the clinical evidence of right or left hypertrophy was not conclusive. In a small number of cases, which he was able to study by separating the chambers by dissection and taking their weights he was able to show that the electrocardiographic records corresponded to the degree of preponderance of the right or the left heart.

Fraser* produced experimental right and left hypertrophy in rabbits by the injection of adrenalin, spartein and bacterial toxins, and was able to show a correlation between the changes in the electrocardiograms and careful post-mortem examinations.

It seems to me that the evidence is reasonably conclusive that *right ventricular predominance* is characterized by a diminution in the size of R and a deepening of S in lead I, an increase in R and a lessening of S in lead III, while *left ventricular predominance* is shown by an increase in the amplitude of R and a decrease in S in lead I, and a shortening of R and a deepening of S in lead III.

†Arch. Int. Med., 1915, xv, 487.
‡Heart, 1914, v, 367.
*Jour. Exp. Med., 1915, xxii, 292.

LEAD I

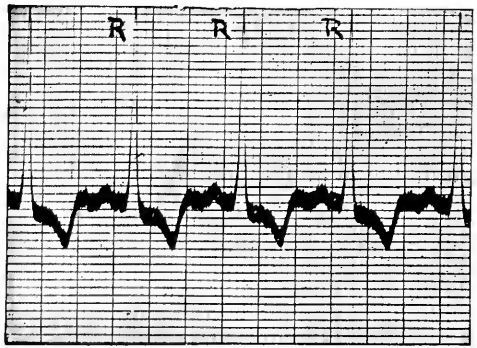

FIGURE 101

LEAD II

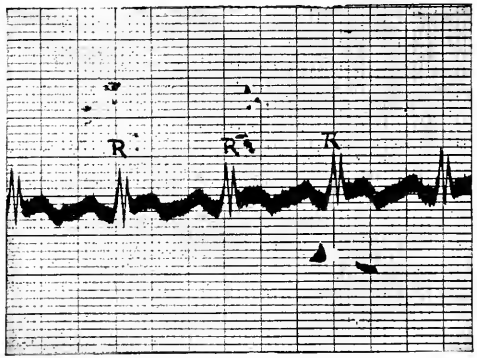

FIGURE 102

LEAD III

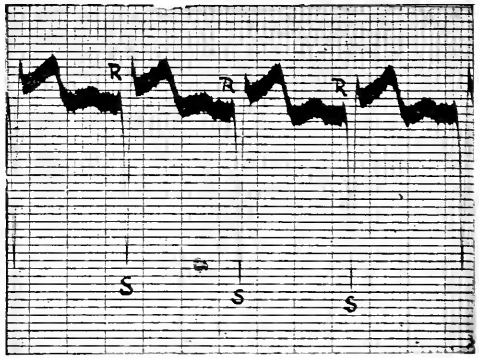

FIGURE 103

Leads I, II and III from a case of long standing hypertension with hypertrophy of the left ventricle.

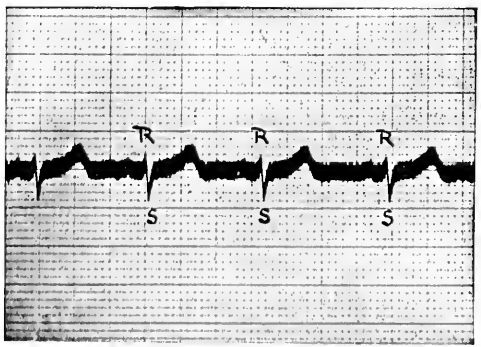

FIGURE 104

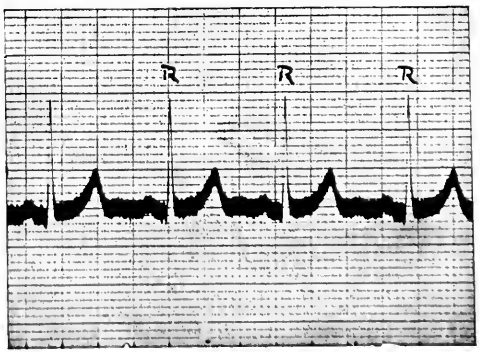

FIGURE 105

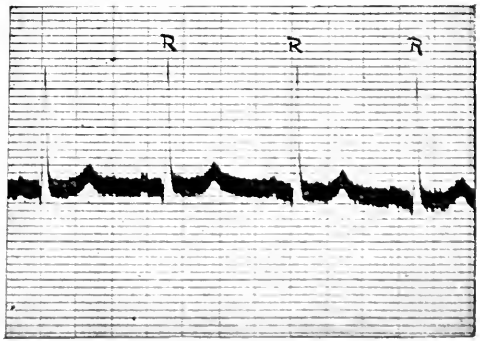

FIGURE 106

Leads I, II and III from a case of mitral stenosis with hypertrophy of the right ventricle.

A comparison of the records obtained in right and left ventricular predominance are shown on pages 239 and 241. Figures 191, 192 and 193 were secured from a case of long-standing hypertension (225-260 mm. Hg.), with physical signs and radiographic evidences of marked left ventricular hypertrophy. Figures 194, 195 and 196 are from a patient with mitral stenosis and clinical and radiographic evidences of right-sided hypertrophy.

Lewis has called attention to the fact that infants under three months of age show an electrocardiogram of the right ventricular hypertrophy type which gradually changes to the normal type.

LEAD I

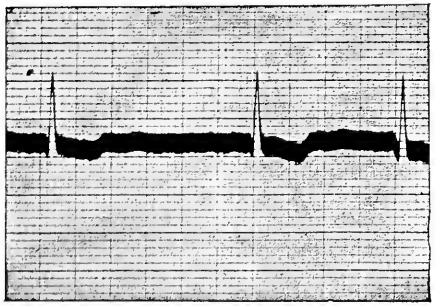

FIGURE 197

LEAD II

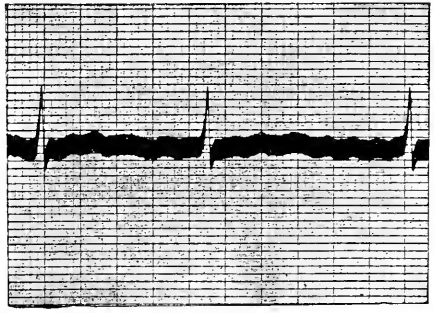

FIGURE 198

LEAD III

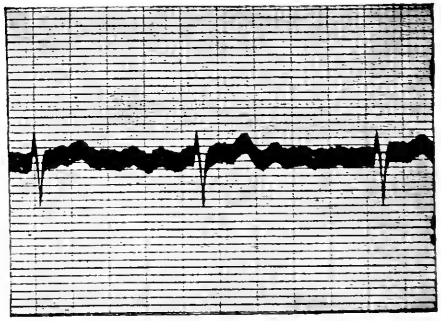

FIGURE 199

Leads I, II and III from a patient with mitral insufficiency, left hypertrophy and auricular fibrillation.

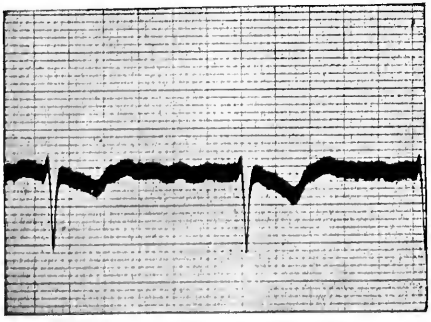

FIGURE 200

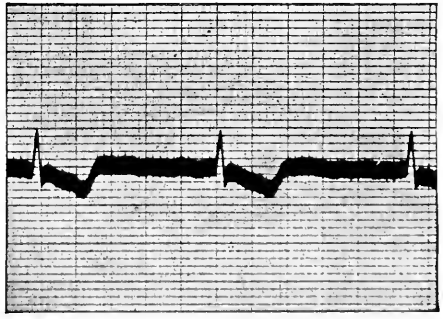

FIGURE 201

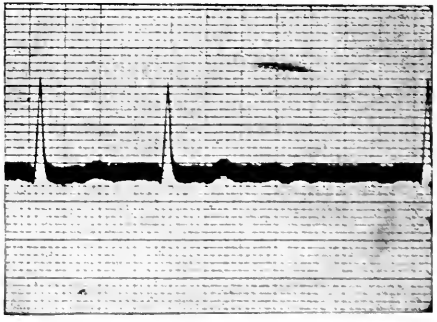

FIGURE 202

Leads I, II and III from a patient with mitral stenosis, right sided hypertrophy and auricular fibrillation.

CHAPTER XVII

Treatment

GENERAL PRINCIPLES

The object of the treatment of myocardial disease is the restoration of function. This may be accomplished by (1) the removal of an abnormal structural condition of the heart muscle, or, failing this, (2) maintaining a proper relation between the amount of work performed by the heart and its functional capacity.

Functional abnormalities are usually the result of organic structural changes; there is little question that we can correct these to some extent and possibly in certain instances restore the muscle cell to the normal. In other instances the functional derangement is dependent on chemical rather than histological changes in the muscle cells or nerve endings, these too are often capable of complete correction.

An adequate circulation may be secured by improving the condition of the heart itself, or by reducing the demands on the heart to a point where a defective myocardium may still be able to perform the necessary work.

It is obvious that etiological studies are of vital importance in determining the means suitable to be employed in the removal of the particular lesion. For example, the acute infections, rheumatism and syphilis, each present their individual problems and a corresponding solution.

Taken in hand in the early stages, myocardial changes due to such toxins may often be repaired and frequently complete restoration of function may be attained. However, as we know only too well our early efforts to avert fixed structural changes are all too frequently unavailing. Diphtheria has left behind degenerative changes, the active inflammatory reaction of rheumatism has subsided, but the bands of fibrous tissue separating or replacing muscle cells remain. An active syphilitic process may have been controlled, but the gumma may be replaced by scar tissue interrupting the continuity of functionally active myocardial tissue or may have produced changes in the walls of the blood vessels materially affect-

ing the nutrition of otherwise healthy contractile areas. Advancing years have brought with them blood vessels atheromatous and studded with calcareous deposits and the elasticity of their walls is all but gone. The outside demands of excessive physical exertion of the contracted kidney or of a general arteriosclerosis have left in their train dilated cardiac chambers with the myocardium over-stretched or thickened. The recognition of such structural changes demands that we meet the problem by a different method and by new agencies. Though we frankly admit that we cannot dissolve calcareous deposits or replace connective tissue with new muscle cells, we have by no means exhausted our opportunities of service to our patient. There are at our disposal a number of measures which may help to improve the myocardium and an even greater number through which we may influence the demands on the heart and thus limit the stress to the capabilities of the organ damaged beyond possibilities of restoration to the normal.

INDIVIDUALIZATION

Success in the treatment of myocardial disease is primarily dependent upon individualization. It is in this sphere that the newer methods of investigating the myocardium, to which such a considerable portion of this book has been devoted, are of greatest value. Up to the present time these studies have been in a large measure directed to discovering the nature of the abnormal function and in designating in each instance the fundamental property of the muscle cell which is at fault. Thus we are able to say with considerable certainty that in one case a defect in conductivity, in another abnormal irritability and in a third a change in contractility, is primarily the basis of the functional change. The effect of drugs and other remedial measures on these fundamental functions of cardiac tissue have been less completely investigated, but such studies as have been made have already furnished information of great value in determining the mode of their activities and in pointing the way to their employment in abnormal functional states. It is needless to suggest that further research along these lines is most desirable and should be a fruitful source of information. It should serve to extend our knowledge in a very useful field and to correct many of the notions now in vogue in

regard to the treatment and management of those suffering from diseases of the heart.

REST

The most important single measure at our command in the treatment of diseases of the heart is a curtailing of the work that the heart is called upon to perform. This does not mean that every case showing a defect of myocardial function needs rest, and it is one of the important duties of the physician to determine in each instance whether the demands on the myocardium are too great and if so to what degree they should be modified.

When the body is at rest, the heart liberates only a small part of the force of which it is capable; the remainder is called the "reserve force." This factor of safety is very considerable in the normal heart and it has been estimated* that the reserve force of the heart renders it able to perform thirteen times the amount of work which it accomplishes when the body is at rest. A moderate amount of physical exertion calls upon the heart for the expenditure of four times as much work as is required in a state of bodily inactivity, but the normal heart will readily perform this task year after year if its work is alternated with proper intervals of rest.

The insufficient heart is one in which the "reserve force" is below the normal. The depletion of the reserve may have been due to excessive demands on a normal heart or normal demands which cannot be met by a damaged heart. It is evident that the term "insufficient heart" is a relative one. The exhaustion of reserve strength may be very slight or of a degree that is barely compatible with life. When the heart is insufficient and the reserve force is depleted to a marked degree, rest is always indicated.

The first object attained by bodily rest is a diminished demand on the heart for work, i.e., the drain on the reserve force is curtailed. Important as is this factor, there are other associated benefits which should not be lost sight of in reckoning the value of this mode of treatment. Physical inactivity usually entails a slowing of the heart. This is brought about by reflex nervous influences affecting the pacemaker. In a normal heart beating at a rate of 70, the cardiac cycle occupies 0.862 second. In such a

*Lewy: Ztschr. f. klin. Med., 1896-97, xxxi, 320.

heart Edgren has estimated that duration of systole is 0.379 second, diastole 0.483 second and the time occupied in diastole in a twenty-four hour period is, therefore, over thirteen hours. In the accelerated heart the shortening of the cycle is almost entirely at the expense of the diastolic period, the length of systole remaining practically unchanged. In a heart beating 140 per minute, the total time occupied by diastole in twenty-four hours is reduced to less than four hours. The diastolic period is the time in which the myocardium obtains its rest and the above figures sufficiently indicate the great variations in this period of recuperation with different heart rates. During diastole the molecules are built up, upon which depend the fundamental properties of the muscle cell, and, other things being equal, the longer this period of recovery the more mature will be these molecules and the more effective their dissociation during systole. Thus the contractile power of the muscle cell is proportional to the period of rest preceding its utilization. The above is illustrative of the advantage to the heart of conditions permitting the fundamental properties of the muscle cell to reach their optimum during each cycle, and for this physical rest is one of our most useful agents.

Another important benefit to the muscle cell attained by prolonging its period of rest is dependent on the fact that the myocardium receives its nutrient supply of blood during diastole; it, therefore, follows that a lengthening of an abnormally short diastolic period affords a better opportunity for the muscle cells to receive their full quota of nutrient material, upon which must depend a normal functional activity and the storing of an adequate reserve.

The development of cardiac hypertrophy, so desirable in many cases, is dependent on an adequate blood supply, which can best be secured by prolonging the diastolic period.

The question may now be asked in what patients, the subjects of abnormal myocardial function, is rest* indicated and to what degree must it be employed? To such a question no categorical answer can be given, but a few suggestions will be offered which may help the physician in formulating the policy suited to the needs of the individual case.

*We would define "rest," as used in the present discussion, as any curtailment of the physical activity to which the individual is accustomed.

The object to be attained is to prevent myocardial damage or, if this is in process, to limit its extent; to maintain a normal distribution of the blood throughout the body, and to accomplish this without permanently reducing the reserve force, or, if the reserve force is already curtailed, to afford an opportunity for its restitution.

Absolute rest in bed is advisable in all cases where an active infective process is localized in the heart substance and in the active stages of all infectious diseases, the toxins of which are prone to attack the cardiac tissues. It is also indicated in the early stages for a heart which has become insufficient from any cause whatever, the insufficiency being suggested by a greater or less degree of dyspnœa and other evidences of improper blood distribution. Frequently, when the reserve force of a heart is only moderately depleted, a period of confinement to bed, while sometimes not absolutely necessary, will allow the patient to shorten materially the time required to recover his balance. In cases of acute or chronic severe cardiac insufficiency, with complete exhaustion of reserve force, absolute rest in bed is imperative.

Many mild infections and febrile conditions, such as bronchitis, indigestion, diarrhœa, etc., not ordinarily considered of serious import, attain a new significance when they occur in a subject with a weak cardiac muscle; such patients should be confined to bed until the complication has cleared up.

Few patients with extreme myocardial insufficiency can be kept flat in bed and some are far more comfortable and will improve quite as rapidly if they are allowed to spend all their time in a chair.

The length of time that a patient should remain in bed is often rather a difficult matter to decide. I think the more common error is to allow the patient up too soon, but in a certain number the confinement to bed may be overdone and there is a more rapid improvement if the heart is given the extra work which the change in position entails. The patient should be confined to bed until it is quite evident that the extreme insufficiency has disappeared and until he has accumulated enough reserve force to change his position and make certain simple movements without inducing symptoms of exhaustion of the reserve force. Changes to an upright position and the transition to walking and other exercises should

be very gradual and should depend on a study of the reaction of each individual to the work thus newly imposed. All advances should be made only under the explicit written directions of the physician.

Many cases of mild chronic myocardial insufficiency do not need to go to bed, limitation of their usual physical activities, change in occupation and definite periods of enforced inactivity are sufficient to allow the heart to recover a reasonable amount of reserve force. In arranging a program for each patient, the ingenuity of the physician will often be greatly taxed. Our prescription must be one which the patient can follow. If merely correct in theory, but impossible of accomplishment, it will be as little creditable to the sagacity of the physician as it is of benefit to the patient. We can do better than to advise a poor man, who has to climb three flights to his two-room tenement, to install an elevator. A temporary interruption or a change of occupation may be imperative, but the psychological, as well as the physical, effect on the patient must be carefully considered before this is advised.

Our discussion has hitherto contemplated conditions in which myocardial defects are associated with cardiac insufficiency. There are a vast number of patients with disorders of myocardial function, some undoubtedly due to real organic changes, who show no evidence of cardiac insufficiency. In the majority of these rest is not indicated, indeed they are better off following their usual mode of life, provided this does not involve excessive physical exertion, rather than modifying this and thus introducing a continuous state of introspection which may ultimately unfit them for both the work and the pleasures of life. There are a certain number, however, whose activities should be curtailed, at least for a temporary period, with the object of warding off a subsequent condition of exhaustion of the reserve force or of correcting a functional derangement which is a source of apprehension to the patient or his friends. Such individuals are greatly helped if we can assure them that the course of treatment is temporary and for the purpose of bringing about a complete restoration of the normal cardiac functions. It is the duty of the physician to sift carefully these cases, using all the facilities which modern methods have placed at his disposal, and, backed by the evidence thus ob-

tained, assist his patient by advising a mode of life suited to the individual conditions.

Fear and apprehension are very frequent accompaniments of myocardial disorders. The most potent influence that can be employed in correcting such psychological states is the kindly but firm assurance, based on real knowledge, given by the physician to the patient. Mental rest is, to some patients, as important as physical rest. Worry and excitement, acting reflexly through the extracardial nerves, accelerate the heart or exert their influence unevenly, rendering an irritable heart less able to preserve its rhythmic activity and thus reducing its efficiency. The method to be employed to combat such conditions again calls for the most painstaking efforts of the medical adviser in offering advice suited to the individual. Withdrawal from business or other usual occupations will, in many cases, augment rather than relieve nervous tension. Change of surroundings and mild, suitable diversions may serve our purpose in one case and utterly fail in another. Hence, the study of the psychology of each individual becomes an important part of the physician's duty.

In other portions of this book the significance of special disorders and the amount of rest suited to each is discussed.

EXERCISE

The indications for and method of transition from a state of complete bodily rest to movements requiring moderate muscular exertion have been touched upon in connection with the discussion of rest. It remains for us to consider the advantages which may be secured by exercise, the class of cases upon which regulated activities have a favorable effect and the methods which should be utilized in applying this form of treatment.

The most evident effects of moderate exercise on a normal heart are an immediate increase in rate and the blood pressure. If the exercise has not overtaxed the heart, its rate and the blood pressure will return to normal a few minutes after the cessation of the exercise. In general, the insufficient heart reacts to exercise in the same manner as the normal heart, *provided that it too is not overtaxed.* In order that a heart may not be overtaxed, the demands put upon it must fall below its maximal working capacity, which consists of the sum of the force necessary to carry on the cir-

culation while the body is at rest and its reserve force. The difference in the working capacity of the normal and the insufficient heart is a difference in the amount of reserve force.

There is no advantage to an insufficient heart in the increase of rate produced by exercise, but if the acceleration is not excessive and the period of its duration is brief, it usually has no detrimental effect. The heightened blood pressure in the first part of the aorta increases the amount of blood passing through the coronaries, and this furnishes more nutritive material to the myocardium. As a result of this, the individual muscle fibers become larger* and actually increase in number.† Hence, there is a thickening of the musculature which we recognize as hypertrophy of the heart. Exercise increases the work of the heart. The heart reacts to this increased work with hypertrophy. Hypertrophy means, at least for a time, an increased reserve force. Aside from the direct effect on the myocardium, exercise facilitates the return flow of the blood from the extremities and reduces the work of the heart ordinarily expended in this direction, while at the same time there is an increased flow of blood to the right side of the heart.

A discussion of the benefits to the patients from exercise other than those directly affecting the circulatory apparatus are beyond the scope of the present papers. But such items as the relief of congestion of the various organs of the body, the improvement in appetite and digestion, the betterment of the excretory activities of bowel, kidneys and skin, the betterment of respiratory conditions, etc., etc., should not be forgotten in estimating the value of this form of therapeutics.

In considering the classes of patients who may be benefited by a course of graded exercises, it may be well first of all to enumerate those in which any measures of this kind are absolutely contraindicated:

1. Acute infectious diseases in which a myocardial involvement has commenced or may be apprehended.
2. Acute dilatation after overexertion.
3. A heart which is not compensated during complete bodily rest.
4. Angina pectoris.

*Goldenburg: Virchows Arch., 1886, ciii, 88.
†Zielenko: Ibid., 1875, lxii, 29.

5. Cardiac asthma.
6. Chronic nephritis.
7. General arteriosclerosis with high blood pressure.

Among those to whom we may be confident that properly regulated exercises will be of benefit, are:

1. Young people with disproportionally small hearts. We see this condition not infrequently in young persons who have grown rapidly, but whose cardiac development has apparently failed to keep pace with the rest of the body. It occurs not only in those of sedentary habits, but also in boys who are exercising vigorously. These may show signs of cardiac insufficiency of a mild grade, with no discoverable abnormality other than a myocardium that is relatively small. These patients should not be deprived of their exercise, but for their irregular and often too violent activities a course of regular and carefully graded training should be substituted.

2. Those of sedentary habits whose cardiac tissues are sufficient in quantity, but deficient in quality. Here exercise acts as a direct stimulant to cardiac metabolism and the results of its employment are most gratifying.

3. Young persons with sinus arrhythmia. The instability of the pacemaker and its sensitiveness to reflex nervous influences, are often favorably affected by properly controlled exercise.

4. Obese patients in whom the cardiac reserve is not sufficient to support the extra burden of overweight. In these a strict dietary should play an important part. In the very aged, or when arteriosclerosis is present, exercise should be used with great caution.

Lying midway between the group in which exercise is distinctly contraindicated and the group in which we may invariably expect favorable results from its employment, are many cases in which the results of exercise cannot be as confidently predicted. We should always individualize, but in the cases under consideration a minute study of each patient's condition and his reaction to exercise is particularly required.

For the most part, these are patients who are convalescing from an acute myocardial process or chronic cases whose hearts have become decompensated; all of them show a greater or less impairment of reserve force and a period of rest must precede any measures directed toward increasing the work of the heart. This is

especially the case in those in whom the reserve force has been depleted by physical exertion which has exceeded the maximal working capacity of the heart. After a period of rest, during which the heart will have had an opportunity of adding something to its reserve force, massage or the mildest forms of resistance movements may be tried for brief periods; if the reaction of the patient to these mild procedures is favorable, they may be continued and gradually increased in the manner indicated below. If, however, the cardiac response is bad, rest must be continued and all forms of activity deferred to a subsequent and more propitious occasion.

The chief advantages of the introduction of a course of graded exercises are that both passive and active movements of this kind can be more accurately measured than in the case of voluntary indiscriminate activities, which the patient will undertake when not under close supervision. Progress is more uniform and convalescence is less apt to be interrupted by frequent temporary setbacks due to thoughtless excessive demands on the reserve force, and is therefore shortened.

In order that the patient may receive benefit from this form of treatment, it must be conducted under the closest supervision of the physician. The physician must either carry out the treatment in person or must be present during each of the earlier treatments and at intervals during the later stages, in order that he may observe the patient's reaction and advise as to the rapidity with which advances shall be made. When the effect of treatment is established, the physician may by degrees avail himself of the assistance of a trained attendant always working under his direction. On a number of occasions I have seen considerable harm as the result of the well-meaning, but injudicious, efforts of an attendant ignorant of the limits which should be imposed.

The physician must prescribe the kind of exercise, the length of treatment and the intervals of rest in each instance and vary these in accordance with the changes in the reserve force. The physician is guided in measuring the amount of exercise desirable by the reaction of the patient. This does not mean the subjective sensations of the patient, as these are often misleading. The best guides are a study of the pulse rate and blood pressure. One cannot lay down actual figures, but the increase in pulse rate during

the exercise should never be excessive. A good reaction, as indicated by the pulse rate, is one in which the rate increases during exercise (not over 20 beats per minute) and returns to the pre-exercise rate or below this within 3 minutes after the cessation of the exercise. Systolic blood pressure should show a maximum increase on the completion of the exercise. If the pressure rise is prolonged after the pulse rate begins to decline, it indicates that the work to which the myocardium has been subjected is excessive.

The most important principle to be observed in planning a course of graded exercises is to regulate the demands on the heart so that they shall never exceed its reserve force. If the treatment is commenced very early, that is, before the patient is allowed out of bed, or even when he is beginning to sit up, at a time when the reserve force is still small, a start should be made with gentle massage of the extremities, always working from the periphery toward the trunk. Short periods of massage (10 minutes) should alternate with ten-minute periods of rest, and the whole treatment should not consume more than half an hour. As the reserve force increases, the massage may include the whole body. The periods of rest may be shortened and the duration of treatment lengthened, but it should never exceed one hour. Following the course of massage, passive exercises may be introduced. Here the patient relaxes completely and his extremities are gently moved by the attendant. Next are added simple voluntary movements made by the patient at first slowly without resistance, later more rapidly and against resistance furnished by the attendant. This may, in turn, be followed by exercises involving self-resistance, in which certain muscles are contracted by a voluntary effort against resistance introduced by opposing sets of muscles (e.g., the patient imagines he is lifting a weight with one hand, etc., etc.).

All these exercises should be employed for short intervals only, alternating with periods of rest, and a period of complete relaxation in the horizontal position for half an hour or more should follow each treatment.

Along with these exercises the patient should be taught the proper method of breathing, a very important element in facilitating blood flow.

Patients with a fair amount of reserve force should be allowed

to walk short-measured distances on the level, later the distances may be increased and climbing may be tried. The changes should be very gradual and always controlled by a study of the effect which the prescribed exercise produces. A patient with a permanently damaged heart must be taught that there are definite limits to the stress with which his heart can definitely cope, and he must learn to live within these limits or pay the penalty. What the limits may be can only be determined by a careful study of the individual. The degree to which some patients with severe grades of myocardial insufficiency can improve is extraordinary. They may reach a point where they can do a surprising amount of heavy manual labor with impunity.

In selecting the form of exercise suitable for a patient whose heart has attained a fair degree of strength, it is often helpful to ascertain the kind of exercise in which he is proficient. On the links a trained golfer will make much less demand on his reserve than the beginner, even riding may be permissible for a skilled horseman.

It has been our purpose to outline the principles involved in applying systematic graded exercises, the details of the various methods as advocated by Herz and Zander, Oertel's Terrain cure, Schott's modification of the Swedish movements as used at Nauheim, Barringer's dumbbell exercises, etc., etc., are best studied in the original papers or in special treatises devoted to these subjects.

BLOOD-LETTING

Under ordinary conditions, the hydrostatic pressure in the vascular system tends to make the blood in the veins rise to the same heights as in the arteries. The force of the heart, assisted by the suction action of respiration and the muscular pressure on the veins, returns the blood to the heart at the same rate at which it is sent out. A prominent feature of many cases of cardiac insufficiency is an abnormal distribution of the blood. A weak myocardium, with loss of muscular tone, predisposes the cardiac chambers to dilatation; when the right ventricle is thus affected and tricuspid insufficiency ensues, the back pressure leads to an overfilling of the veins and right heart. It is under these conditions that bleeding is often of great value. The volume of the

blood brought to the heart is reduced and the embarrassment of the right heart is at least temporarily relieved.

Venesection has an effect that is essentially mechanical. It decreases the amount of blood brought to the right ventricle, hence this contracts more completely and more efficiently and forces more blood into the left heart, which, in turn, sends more blood into the aorta. This increases arterial pressure and, incidentally, the blood flow in the coronaries, thus improving the nutrition of the myocardium.

The indications for venesection are an overdistended right heart and venous engorgement in a patient whose condition demands prompt interference, where there is no time to wait for the action of drugs or other remedial measures or where these have failed. The procedure will often afford a respite during which other measures may have an opportunity to act.

Bleeding is best accomplished by opening a superficial vein at the bend of the elbow after applying to the upper arm a bandage sufficiently snug to obstruct the venous return. By puncturing the vein with a good-sized aspirating needle, to which is attached a rubber tube leading to a flask, much of the disorder incident to the cruder methods may be avoided. The blood flow may be facilitated by gentle mouth suction by the operator on another tube leading from the flask. The blood flow is also increased by getting the patient to close and open the hand, thus forcing the blood through the veins by muscle contraction.

The best results are usually secured by the removal of 300 to 500 cubic centimeters of blood.

DIET

There are few general principles which can be laid down as a guide in regulating the diet of those suffering from myocardial disease. The relation of protein intoxication to the degenerative changes of various organs, is a subject pregnant with interesting possibilities, but as yet our knowledge is but a step advanced beyond the sphere of pure speculation. If our therapeutics are to be used with a conviction which is based on a reasonable logic, the application of these hypotheses to the treatment of our patients must await the discovery of further facts which may link together the fragments which at present can only be called suggestive.

The beneficial effects of the administration of large quantities of sugar is based on evidence secured by perfusing the isolated heart with fluids containing sugars of varying amounts* and is supported by a considerable amount of rather unconvincing clinical evidence.† My own experience with this procedure has been limited and thus far inconclusive.

Regulation of the diet is important, but should be directed toward the correction of conditions frequently very remote from the heart and which influence the myocardium in ways indirect, but often efficacious.

Among such conditions the patient's *weight* should receive consideration as a factor of first moment. Excessive weight is a very common complication of chronic heart disease, because, as a rule, restriction in the amount of food has not paralleled the reduction in bodily activity. The requirements of the body at rest are less than twenty-five calories per kilogram of body weight. Those whose activities call for great physical work often utilize 50 calories per kilo. When a patient's mode of life is suddenly changed from one of severe physical exertion to one of comparative inactivity, the tendency is for him to continue taking the quantity of food which habit has established. As a result, the body requirements are more than supplied and the surplus is stored as fat, which soon is an additional burden to be carried by a heart whose reserve already shows too narrow a margin. It follows that when a physician curtails the physical activities of a patient with an insufficient heart, the diet should be restricted as a prophylactic measure. When an excessive diet has already produced a condition of obesity, the overweight must be gotten rid of by a carefully instituted reduction cure. The details of a régime suited to the needs of the various types which we meet need not detain us, but we should bear in mind that dependence must be placed almost entirely on the restricted caloric value of the ingested food. Exercises can be employed to only a very limited extent and drugs are worse than valueless. The weight should be reduced very gradually. I believe the best results are obtained when the loss is about one pound a week.

*Locke and Rosenheim: Zentralbl. f. Physiol., 1905, No. 20, Dec. 30.
†A. Goulston: Cane Sugar and Heart Disease, London, 1914.

The antitheses to these obese patients are not infrequently seen. They are below average weight and are poorly nourished. Most often they are young persons who are growing rapidly. Sometimes they have had repeated attacks of rheumatic fever. The whole musculature is below par and the myocardium has suffered along with the other muscles of the body. To these judiciously forced feeding is of great value.

Patients suffering from chronic nephritis with high blood pressure and a heart laboring beyond its capacity are best treated dietetically with foods, the end products of which make the least demand on the eliminative capacity of the kidney. The kind of food best suited to the individual case can only be determined by a careful study of the functional activities of the kidney in each case.

Those with infectious diseases running a febrile course with intercurrent myocardial involvement should be fed with small quantities of nutritious food at frequent intervals. The management of the diet does not differ from that of the uncomplicated disease, except that, as a rule, it is well to keep the administration of fluids within reasonable bounds.

In general, patients with well-marked cardiac insufficiency do better when given small meals at frequent intervals; five or six feedings in the twenty-four hours are usually better than three meals. The majority of the diet should consist of solid food.

Quite as important as the proper direction of the kind and quantity of the food is the *regulation of the water balance*. The relation of œdema, ascites, pleural effusion, etc., to myocardial insufficiency is intricate and a full discussion would lead us far afield. The volume of the circulating blood undoubtedly has a considerable influence, not only on the amount of work which the heart is called upon to perform, but also must be reckoned with as a direct factor in producing dilatation and hypertrophy. The most notable example of this condition is met with in the so-called "beer heart." Here the myocardial changes may be the result of several elements, such as the alcohol and carbohydrate content of the beverage, the severe physical exertion to which most of these patients have been subjected and the increased volume of the blood following the ingestion of enormous quantities of fluid. Practically all competent observers are agreed that the latter is the most important factor

in inducing the change in the heart muscle. The aspiration of the pleura, or the removal of ascitic fluid, will often relieve the strain on a laboring heart and furnish the starting point for the recovery of its reserve force. The disappearance of œdema is frequently the forerunner, rather than the result, of improved heart action. With this end in view, the stimulation of kidney activity and the reduction of the sodium chloride intake may be of considerable indirect benefit to the heart.

As a general rule, those suffering from myocardial insufficiency should not take over one and a half liters of fluid a day. Many, particularly those having œdema, are greatly benefited by a more restricted fluid intake. We are not as yet in a position to designate the exact types which will respond favorably to an extreme limitation of fluid ingestion, but not infrequently the employment of this method yields brilliant results. The diet suggested by Karell*

*Karrell Diet. For the first five to seven days, milk 200 cc. at 8 and 12 A.M., 4 and 8 P.M. No other food or fluid. Eighth day: milk as above and at 10 A.M. one soft-boiled egg; at 6 P.M., two pieces of dry toast. Ninth day: milk as above and at 10 A.M. and 6 P.M. one soft-boiled egg and two pieces of dry toast. Tenth, eleventh and twelfth days: milk as above, and at 12 M. chopped meat, rice boiled in milk and vegetables; 6 P.M., one soft-boiled egg. (No salt is used throughout the course. Salt-free toast and butter used. Small amount of cracked ice allowed with diet. All meat can often be advantageously omitted.) From Dr. Herbert S. Carter's Diet Lists, Saunders, Phila., 1914.

is one of the most satisfactory means of securing the desired fluid restriction. During such a course the patient should be confined to bed. Not infrequently a patient who has shown a prompt improvement on a Karell régime may be benefited by later using such a diet one day each week.

BEVERAGES

The regular use of alcohol in those suffering from functional or organic disease of the myocardium, is to be deprecated on theoretical grounds. There is little doubt that its habitual use affects favorably neither the heart muscle nor its controlling nervous mechanism. And yet I think there are many patients accustomed to the use of malted and spirituous liquors by years of habit in whom the entire withdrawal of all alcoholic beverages is a distinct detriment. To young adults, all alcoholic drinks should be forbidden: elderly people accustomed to its use often do better if allowed

moderate quantities. Light wines, good whiskey or brandy are to be preferred to malted drinks representing an equal amount of alcohol. These should be taken at meal-time and in amounts definitely prescribed. Tea and coffee should be regulated much in the same way as alcohol, they are not to be indiscriminately forbidden and in certain instances the effect of the contained caffeine is of distinct value.

COLD APPLICATIONS

The application of cold to the precordium, by means of an ice-bag or a Leiter's coil, often seems to be of advantage in rapid, overacting hearts and in tachycardias of sinus origin. The rational explanation of such a procedure present difficulties. We know that the formation of stimulus-material is slowed by a direct application of cold to the sinus region, but it is hardly conceivable that a precordial icebag will produce a degree of cold sufficient to penetrate to such a depth.

The precordial poultice and hot fomentation are frequently comforting to the patient. It is doubtful whether they have any more profound therapeutic effect.

BATHS

In a certain number of patients suffering from myocardial disease, a course of baths seems to be of considerable value. Waters containing carbon dioxid and simple brine have been employed with satisfactory results. The reaction of the individual patient must be studied. The first bath should be a weak one, just below body temperature and should last not over six minutes. If the patient is comfortable, if there is no increase of dyspnœa, and if the pulse and heart action are improved, the baths may gradually be made stronger, the temperature lowered and the time lengthened. The method is almost wholly empirical and we have little knowledge of the physiological processes through which the beneficial results are obtained. Nervous reflexes, following cutaneous stimulation and altered vaso-motor tone, are probably elements of importance, but much careful observation and scientific study is necessary before we can offer a logical explanation of the modus operandi and a more accurate outline of the indications for and against these measures.

SPA TREATMENT

The value of spa treatment, I believe, has been greatly overestimated. The benefits derived from a visit to a well-organized resort are due mainly to the ordered régime to which the patient is subjected under the watchful care of a skilful physician, the relaxation from the cares of business and home, the change of scene and opportunities for properly regulated pleasures. The specific value of special waters and complicated apparatus are of very minor importance.

The miraculous cures which are often claimed in the name of the spa may be equalled in the experience of any skilful practitioner.

Most patients are much more readily controlled in a place removed from accustomed ties and responsibility, and where it is the fashion to follow the minute directions of the medical adviser.

The conscientious physician, in considering the probable value to be derived by his patient from spa treatment, must take into account the long journey, its attendant fatigue and expense. He must see to it that his patient is placed under expert medical care and is not allowed to drift into the net of the unbalanced faddist or the unscrupulous charlatan, who unfortunately are not less in evidence at the most famous cures than elsewhere.

CHAPTER XVIII

Treatment

DRUGS

In the present discussion of drugs in the treatment of abnormalities of myocardial function, we will limit ourselves to a consideration of a very small number and will examine only their direct action on the myocardial tissues or their indirect action through the vagus and accelerator nerves.

It is often most important in treating myocardial disorders to correct or modify the condition of organs other than the heart. Thus a change in kidney function or an alteration in the size of the peripheral arteries may be a far more efficient means of improving cardiac function than anything which we can do to the heart muscle directly. It is not, however, our purpose to discuss drug activity from this standpoint. We will, therefore, confine our attention to those drugs which have a direct action on the myocardium and will omit a consideration of the effect of the drugs of our selected list upon other organs of the body. These aspects are presented in the proportions which they deserve in the standard works on pharmacology and works devoted to the broader discussion of diseases of the heart. An excellent résumé of the experimental work has been compiled and discussed by Winterberg* and should be consulted by those particularly interested. The graphic methods of recording circulatory changes have greatly improved the detail and accuracy of our clinical observations. By these means we may discover minute alterations which have been hitherto impossible of detection. It is probable that the next few years will add much to our knowledge of the usefulness and uselessness of drugs.

ADRENALIN

The extract of the suprarenal gland finds its chief effect on the wall of the peripheral blood vessels. There is considerable evi-

*Handb. d. Path., Diagnostik, u. Therap. d. Herz. u. Gefässkrankungen, Leipzig, 1914, iii, H. 2.

dence that it also has an influence in increasing the activities of all of the fundamental properties of the myocardial cells, notably heightening the irritability and the contractility. It may be that this is due to a direct action on the muscle cell or, indirectly, through its effect on the sympathetic nerve for which this drug seems to possess a selective activity.*

In large doses and under special conditions (see "ventricular fibrillation") adrenalin may produce extreme myocardial irritability and many types of arrhythmia.†

On account of its influence in increasing the contractility and the tone of the heart muscle, it has been recommended in acute dilatation and in conditions of surgical shock. Until our knowledge is more complete, I believe that we should employ adrenalin with great caution in conditions of acute heart failure, recalling that in association with anæsthesias of light degree‡ it has a tendency to produce great irritability, which may result in ventricular fibrillation and a fatal outcome.

There is considerable experimental evidence which indicates that, while adrenalin acts as a constrictor on most of the peripheral arteries, it produces a widening§ of the lumen of the coronaries.

ALCOHOL

The effect of alcohol on myocardial activity is probably purely reflex and the result of functional changes in the central nervous system and modifications of vasomotor control. The slowing of the heart, following the administration of alcohol in febrile affections, is probably due to its influence in diminishing cerebral excitement. Alcohol has no effect on muscle when brought to it in the blood stream, but when applied directly to the muscle it weakens its contractions (Cushny).

AMMONIA

probably has no direct effect on the myocardium. Its influence in reducing heart rate is by reflex nervous inhibition. Its effect is quite rapid and disappears in a few minutes.

*Rothberger and Winterberg: Pflüger's Arch., 1911, cxlii, 461.
†Kahn: Arch. f. d. ges. Physiol., 1909, cxxix, 379.
‡Nobel and Rothberger: Ztschr. f. d. ges. Exp. Med., 1914, iii, 151.
§Janeway and Park: Jour. Exp. Med., 1912, xvi, 532, 541.

ATROPINE

modifies cardiac activity through its effect on the vagus nerve control. It probably has no direct influence on the myocardial cells, although given in toxic quantities to experimental animals it is said to depress the property of contractility (Ringer). The first effect of atropine is to stimulate the vagus through its center in the medulla and thus cause a slowing of the heart. This effect is very slight and quickly passes off. It is succeeded by an increase in heart rate due to a paralysis of the inhibitory terminations of the vagus nerves. That the influence is due to its action on the nerve endings is indicated by the fact that after its administration stimulation of the vagi has no effect on cardiac activity, whereas if the action were central stimulation of the vagi should still slow the heart. The accelerators are not affected by atropine.

Children show very little cardiac response to atropine. The reaction to the drug gradually increases with maturity and reaches its maximum in those about thirty years of age. In advanced years the reaction is less marked than in middle life. In man the slowing of the heart occurs about five minutes after the subcutaneous administration. This disappears in a minute or two. The maximum increase in heart rate appears twenty to thirty minutes after its exhibition. From this time the rate gradually lessens and returns in an hour or two to the rate which preceded its administration.

Atropine is useful in correcting a heart action which is too greatly slowed by an overacting vagus. Thus, in a heart block which is due to a hypertonic vagus, the block may be promptly broken by its administration. The slowing effect of morphine may be neutralized by atropine. When digitalis has been given to excess, a part of its influence may be promptly opposed by a hypodermic of atropine. Its administration is a useful diagnostic means of distinguishing the vagal influences in heart block and cases of suspected hypervagotonicity from those in which the defect is intrinsically in the muscle cell. If the disordered myocardial function is due to an excessive activity of the vagus, the atropine will speedily remove this influence.

In an adult with a heart rate under 60, 1.3 milligrams (1/50 grain) may be given subcutaneously and may be repeated when the effect has disappeared. Unfortunately, the drug is not well

suited for prolonged therapeutic effects, for if the dosage is continued at a level to maintain the vagus paralysis other symptoms, such as dryness of the pharynx, dilatation of the pupil, etc., make the patient very uncomfortable.

CAFFEINE

and the closely related substances, *theobromine* and *theophyllin*, exert an influence on both the extracardial nerves and on the muscle cells of the heart. In experimental animals caffeine may slow the heart by central stimulation of the vagus and depression of the accelerators (Fredericq). In man such an effect is very inconstant. Small doses in animals usually accelerate the heart by increasing the irritability of the sinus node (Cushny and Naten*). This effect is independent of any action on the regulatory mechanism of the heart, since it is seen when the accelerators are cut and when the vagus is paralyzed by atropine. In dogs large doses produce auriculo-ventricular dissociation (depression of conduction). In animals in which an artificial heart block has been produced by a destruction of the bundle of His, caffeine augments the irritability of the ventricle so that the ideo-ventricular rate is increased and many extrasystoles appear.† The evidence as to the effect of caffeine on contractility is somewhat conflicting, but inclines toward the view that in moderate doses it improves the working efficiency of the heart.

In a few patients to whom I have administered *theocin* as a diuretic, there have appeared immediately thereafter large numbers of extrasystoles and in one case a paroxysm of tachycardia, which, as far as could be determined, was unique for this individual. These observations have led me to be cautious in the use of caffeine and closely related drugs in myocardial conditions showing a high degree of irritability. It suggests that the excessive use of tea and coffee in susceptible individuals may be the cause of extrasystoles.

CAMPHOR

is one of our drugs which has long enjoyed a reputation as a direct stimulant to cardiac muscle. We have at present no conclusive evidence that it favorably affects the heart muscle. Its use

*Arch. Internat. de Pharmodyn et de Thérapie, 1901, ix, 169.
†Egmond: Pflüger's Arch., 1913, cliii, 39.

has been advocated in auricular fibrillation, but recent reports make it appear that it is of doubtful value in this condition.

CHLOROFORM

acts on the heart indirectly by stimulating the vagus center, thus slowing the heart, and also has a direct influence on the muscle cells of the heart. Chloroform depresses conductivity to such an extent that at times an auriculo-ventricular block is established. It may greatly increase the irritability of the ventricular wall so that many extrasystoles appear which may pass into a ventricular tachycardia and thence to ventricular fibrillation which is uniformly fatal. The observations of Levy* seem to indicate that the most detrimental effects to the myocardium are wont to occur during light anæsthesia, so that arrhythmias are most common when the patient is in the early stages of anæsthesia or is beginning to recover from its effects. The dangers of the use of adrenalin, in conjunction with chloroform, have already been noted (see "ventricular fibrillation" and "adrenalin").

DIGITALIS

Digitalis obtained from the purple foxglove is the most important member of a group of drugs which have a similar action on the heart. In this series are included *strophanthus, squills, hellebore, convallaria, apocynum* and several others less well known. Of this group of drugs digitalis has received by far the greater study and, since all the members of this series affect the myocardium in a similar manner,† our discussion will be in the main limited to digitalis and the alkaloids which are derived from it. The glucosides obtained from digitalis include digitoxin, digitophyllin, digitalin, digitalein and digitonin. All of these glucosides are present in the infusion; digitonin, being insoluble in alcohol, is absent from the tincture.

Pharmacological studies indicate that digitalis acts in a twofold manner on the functional activities of the heart. It has a direct effect on the muscle cells and an indirect influence through the vagus nerves. The influence on the fundamental properties of the muscle cells has been analyzed to some degree. *Tone:* this

*Heart: 1913, iv, 319.
†Cushny: Jour. Exp. Med., 1897, ii, 233.

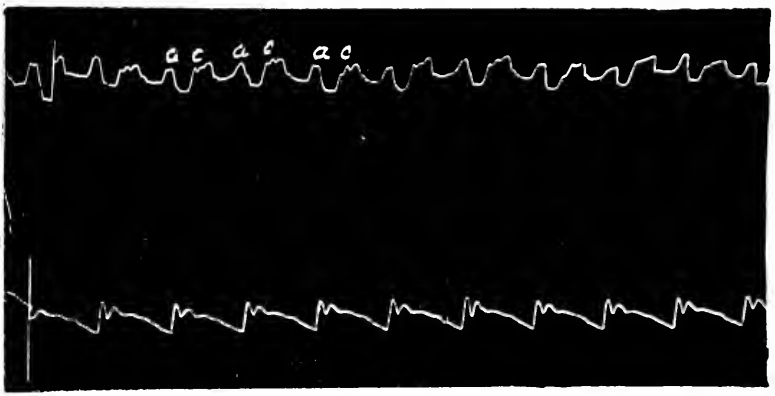

FIGURE 203
Digitalis effect. Delayed conduction. *a-c* intervals = 0.4 second.

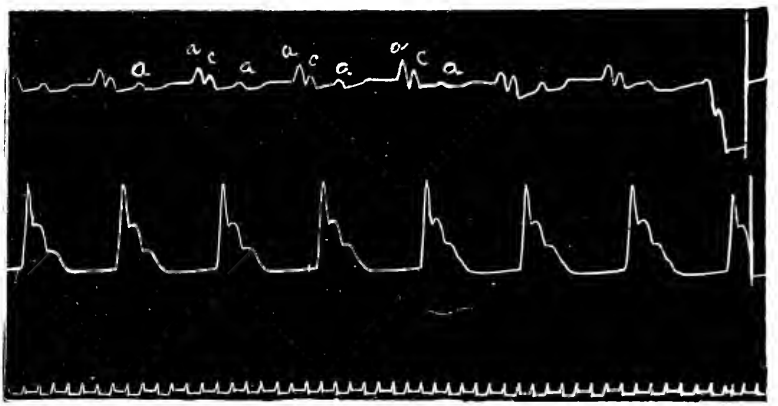

FIGURE 204
Digitalis effect. 2 to 1 block. Auricular rate 80. Ventricular rate 40.

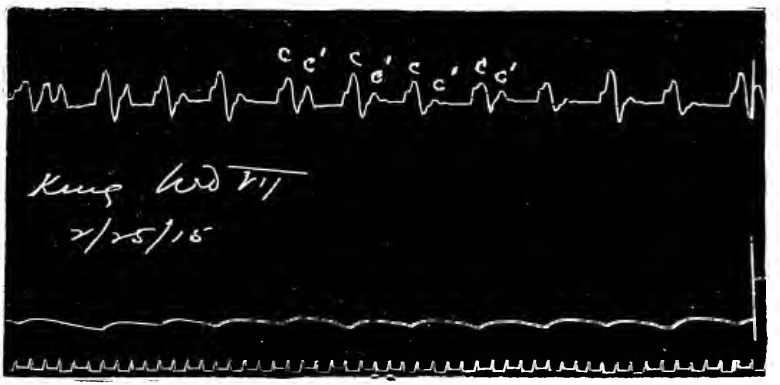

FIGURE 205
Digitalis effect. Coupled rhythm in a case of auricular fibrillation.

property is distinctly modified by digitalis, its direct action on the muscle is to increase its tone and to render relaxation less complete, the heart becomes smaller in systole and does not dilate so fully in diastole. Hand in hand with this direct action on the muscle, digitalis exerts a stimulating effect on the vagi, through this influence the heart is slowed and the tendency to diastolic relaxation is increased. Hence, the effect of digitalis on cardiac tone is the resultant of these two opposed factors and the tonicity will be increased or diminished according as the direct muscle effect or the indirect vagus effect is predominant. In a like manner the efficiency of the heart to empty itself will depend on the relative degree to which each of these factors comes into play. *Conductivity* is depressed so that one often sees in the experimental animal a complete auriculo-ventricular block. The depression of conductivity is due in part to vagus influences, since the block can at times be removed by cutting the vagi. However, there is some evidence which suggests that the dissociation is also due to a direct action of digitalis on the cells of the myocardium. The *irritability* of the heart muscle is increased by digitalis. Thus, in the isolated heart which has ceased to beat, the addition of digitalis to the perfusion fluid may induce rhythmic contractions. All these modifications of the fundamental properties of the heart muscle which have been observed in the experimental animal may be seen in man. We have as yet no accurate means of measuring the tonicity of the heart in the human subject, but most clinicians believe that it is increased by the administration of drugs of the digitalis group. A depression of conduction, even to the degree of complete auriculo-ventricular dissociation and increased irritability (as evidenced by the production of extrasystoles), are common sequelæ of the exhibition of digitalis in the clinic.

While the experimental study of digitalis has afforded us much valuable information in regard to the nature of its activity, it should be remembered that such investigations must of necessity be carried out on hearts with a myocardium approaching the normal or the damage which has been artificially produced is an acute change which is probably quite different from the myocardial changes which are commonly seen in the clinic and checked up at the post-mortem examination.

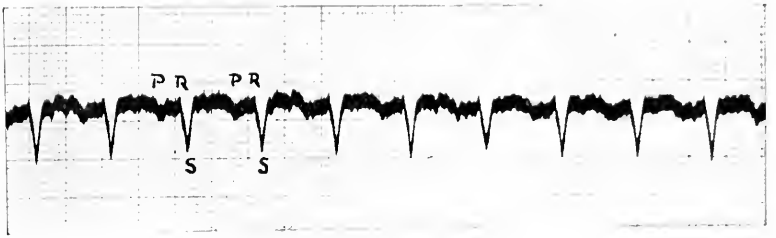

FIGURE 206
February 24, 1916. Before digitalis.

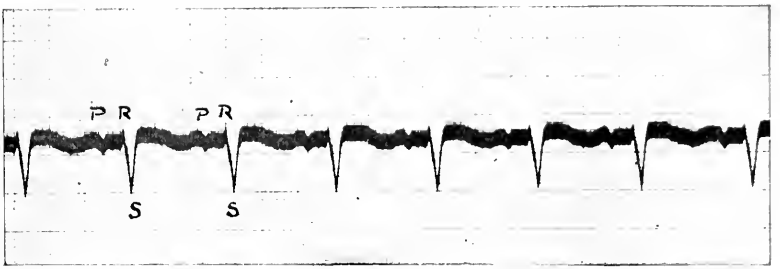

FIGURE 207
March 9, 1916. Sinus slowing.

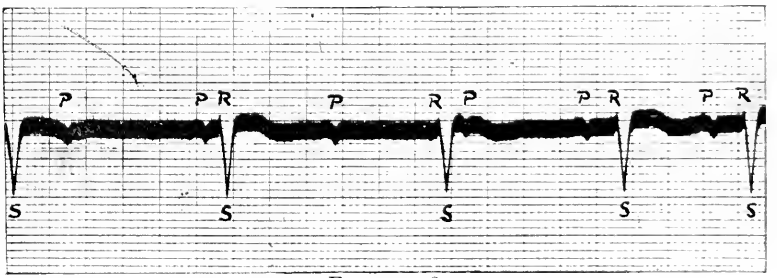

FIGURE 208
March 13, 1916. Complete block.

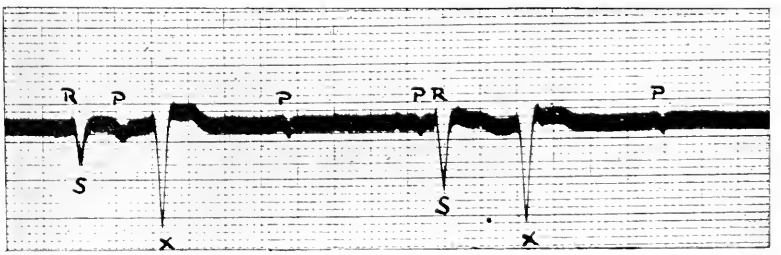

FIGURE 209

March 15, 1916. Complete block and coupled rhythm. Every other ventricular beat is an extrasystole.

All these records are from the same patient and show the progressive effect of considerable doses of digitalis in a myocardium susceptible to its influence. All of these records were taken by lead III.

It is, therefore, most fortunate that the polygram and electrocardiogram have come to our aid in the study of the effects of this and other drugs in man under the conditions which are seen in health and disease and which it is impossible to reproduce in the experimental animal.

The alterations in the rhythm of the heart under the influence of digitalis are: (1) a slowing (due to a depression of stimulus formation at the sinus node induced by vagal influences); (2) a slowing and development of arrhythmia, with modification of the property of conduction (due to change in the *A-V* junctional tissues through a direct effect on the myocardium and an indirect effect through the vagus), and (3) an increased irritability causing new forms of irregularity to appear (extrasystoles, fibrillation, etc.). Some of these changes may be detected by the ordinary methods of physical examination. The change in rate of sinus origin is usually only of moderate degree; when due to alteration in conduction, the slowing may be much more marked, so that, with complete block, the ventricular rate may be reduced to 30 a minute. These conduction changes may sometimes be verified by observing the rate of the jugular pulsations in the neck and comparing them with the apex impulse (see "conduction defects," Chapter VI). The increase in irritability is identified by watching for the development of complete irregularity (see "auricular fibrillation," Chapter XI) or of extrasystoles (see Chapter VII), notably the so-called "coupled rhythm," in which two beats are followed by a pause; the second of these beats is an extrasystole and the pause is the ordinary "compensatory pause" which is associated with extrasystoles. At first this digitalis effect may be observed as occasionally occurring extrasystoles, later every other heart contraction is an extrasystole with a compensatory pause and this is manifested as the "coupled rhythm."

The *polygram* helps us in detecting these digitalis effects and is a much more accurate indicator of the changes in heart rhythm than inspection, palpation and percussion.

Records from three cases under the influence of digitalis may be seen in Figures 203, 204 and 205. In Figure 1 the *a-c* interval is greatly prolonged and measures 0.4 second. In the case whose curve is shown in Figure 204 the brachial pulse rate is

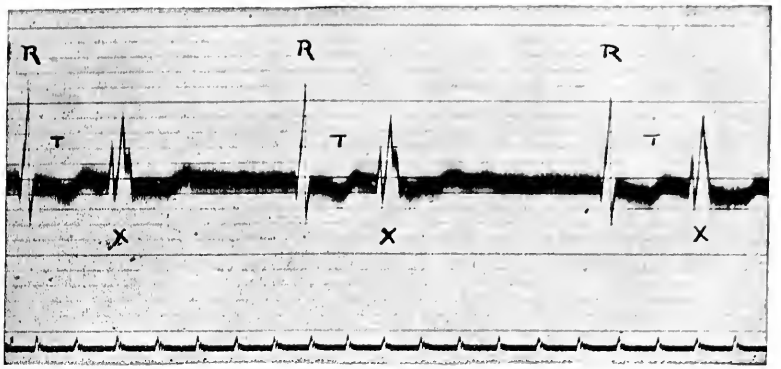

FIGURE 210

Coupled rhythm. Digitalis effect in a case of auricular fibrillation.

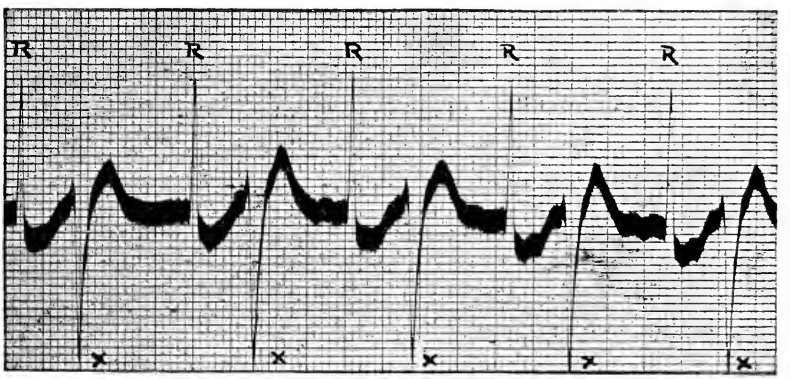

FIGURE 211

Coupled rhythm. Digitalis effect. Every other ventricular beat is an extrasystole. Case of auricular fibrillation.

perfectly rhythmic and only 40 a minute; the auricular rate is 80 and the slow ventricular rate is due to a 2 to 1 block. The patient whose tracing is presented in Figure 205 was a case of auricular fibrillation which showed the toxic effects of digitalis by the development of the coupled rhythm; the brachial shows a rate of 67 per minute, but examination of the jugular curve shows that the ventricle was beating at a rate of 134 per minute, but only every other one of these beats makes an impression on the brachial curve. The second beat of the couple is a ventricular extrasystole and is followed by a compensatory pause. A rhythm of this character and of such a rate indicates a high grade of irritability of the ventricular muscle and is a warning that digitalis must be immediately discontinued.

The *electrocardiograms* bring out these changes even more clearly. Figures 206, 207, 208 and 209 are all taken from the same patient at intervals of a few days during the administration of digitalis. Figure 206 presents the rapid rate (145) with a normal *P-R* interval before digitalis was commenced. (All these figures record lead III, in which in this case the *S* wave was abnormally deep, suggesting left ventricular preponderance; see Chapter XVI.) In Figure 207 is seen a slowing of the whole heart (sinus effect) and prolonged *P-R* interval (*A-V* conduction delay). Figure 208 shows still greater sinus slowing and a partial block. Figure 209 portrays an even less rapid sinus activity, complete block and the "coupled rhythm," in which the second beat of each pair is a ventricular extrasystole, indicating a high degree of myocardial irritability.

Records of two cases of auricular fibrillation, with different grades of ventricular irritability, are presented in Figures 210 and 211. It is quite evident that the irritability of the heart recorded in Figure 211 is very extreme, much more than that shown in either Figures 209 or 210. There is another very interesting change in the electrocardiographic records under the administration of digitalis which has been recently described by Cohn, Fraser and Jamieson.* This consists in a change in the contour of the *T* wave. A *T* wave which is positive in direction before the use of digitalis becomes smaller, diphasic or even directed downward when the

*Jour. Exp. Med., 1915, xxi, 593.

LEAD I

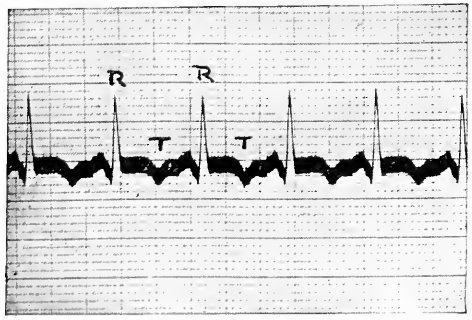

FIGURE 212

LEAD II

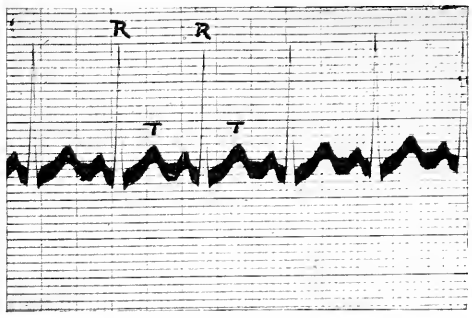

FIGURE 213
April 22, 1915. Before digitalis.

LEAD III

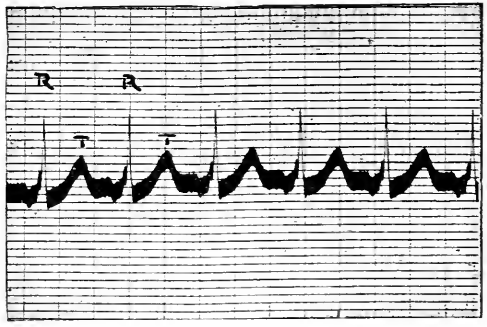

FIGURE 214

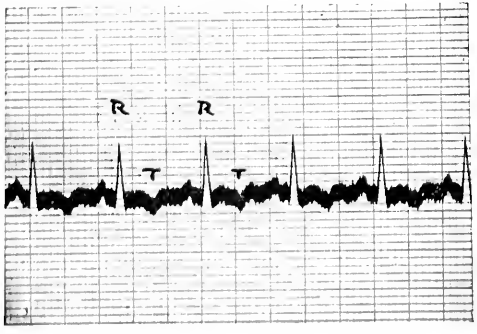

FIGURE 215

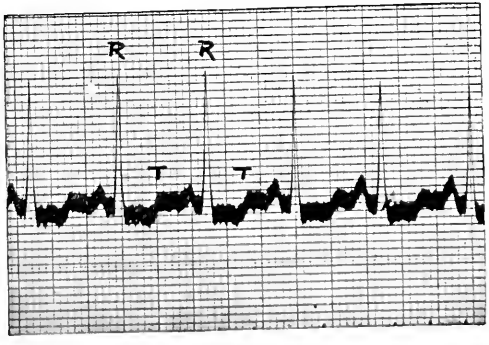

FIGURE 216
May 4, 1915, after digitalis, showing the change in the contour of the *T* wave.

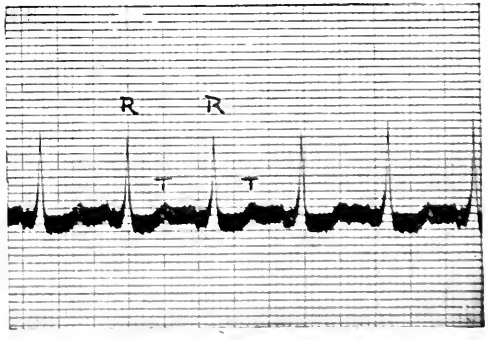

FIGURE 217

heart becomes digitalized. This is well shown by comparing Figures 212, 213 and 214, the three leads taken before and Figures 215, 216 and 217 secured after the administration of digitalis. This sign is a valuable guide in determining whether the heart muscle is being affected by the digitalis. The discoverers found it as the earliest evidence of digitalization in 34 out of 36 cases. They also call attention to the fact that influences other than digitalis may, on rare occasions, produce a similar modification of the size and direction of the *T* wave.

There is considerable clinical evidence that digitalis acts both on the muscle cells of the heart directly and indirectly through the vagi. The changes which occur in the *T* wave suggest this.* If the heart is brought thoroughly under the influence of digitalis, and the vagal influences are removed by administering atropine which paralyzes the terminals of the vagus in the myocardium, certain digitalis effects will still persist.† Digitalis has a greater influence on the damaged than on the normal heart muscle. This is particularly true of hearts which have been injured as the result of rheumatic fever and those which show conduction defects. Digitalis is particularly indicated in the dilated heart with a reduced muscle tone and in cases of tachycardia, where the rapid ventricular rate is due to impulses arising in abnormal foci in the auricles which are showered upon the *A-V* junctional tissues (auricular fibrillation and auricular flutter); here it is given with the purpose of blocking, partially or completely, the stimuli from the upper chambers.

Digitalis is usually contraindicated in those showing frequent extrasystoles, unless it can be shown by careful observation that it does not increase the myocardial irritability.

It is rarely of value in tachycardias of sinus origin such as are seen in certain forms of auricular paroxysmal tachycardia, Graves' disease and in the acute infections. In hearts showing a partial block the abnormality is usually increased by digitalis administration. It is not contraindicated by hypertension.

When a digitalis action is desired, it should be given until some physiological effect becomes apparent, but in pushing the dosage

*Cohn: Jour. Amer. Med. Assn., 1915, lxv, 1527.

†Cushny, Morris and Silberberg: Heart, 1912-13, iv, 33; Talley: Amer. Jour. Med. Sc., 1912, cxliv, 514.

the possible dangers should never be forgotten. Evidences of heart block and excessive irritability must be watched for and their development are the signs which should at once put us on guard.

The most important elements in selecting particular preparations of digitalis for administration are: (1) a high degree of physiological activity; (2) uniformity of physiological activity, and (3) familiarity on the part of the physician with the physiological activity. One and two can only be secured by obtaining the drug from reliable manufacturers who carefully select their digitalis leaves, employ a uniform method of extracting the active principles and standardize their product by physiological tests. The third element is secured by confining one's attention to the study of a small number of preparations and using these to the exclusion of all others. In my own practice a potent infusion, a standardized tincture, tablets of digipuratum, ampoules of digipuratum and crystalline strophanthine are the preparations which I have found satisfactory and which meet my needs. No rules for the amount of the drug which should be given can be laid down. Each case, in this respect, is a law unto itself, and the amount can be gauged only by studying the physiological effect in the individual. The maximal doses which may be used are: of an active infusion (freshly prepared) 30 cc. (1 ounce) in 24 hours; of a good tincture 4 cc. (1 fluiddram) in 24 hours; digipuratum by mouth 0.4 gram (6 grains) in 24 hours, intramuscularly 0.1 gram (1½ grains) three times a day; crystalline strophanthine intravenously ½ milligram dissolved in 8,000 parts of normal saline, not more often than once in 24 hours.

Unless there is special urgency, digitalis should be given by mouth, as all of the active preparations are very irritating to the tissues when administered subcutaneously. Hatcher and Eggleston* have shown that the nausea and vomiting which are produced by digitalis is due to their action on the central nervous system, rather than a local irritant effect on the stomach, hence the intramuscular or intravenous administration causes these symptoms as readily as when the drug is given by mouth. The toxic dose of strophanthin given intravenously is not far removed from the therapeutic dose, hence it should never be given to a patient who

*Jour. Pharm. and Exp. Therap., 1912, iv, 97.

has been recently taking any drugs of the digitalis series, and is indicated only in extreme emergencies.

The experimental work of Voegtlin and Macht,* who found that digitonin relaxes, while other alkaloids of digitalis constrict, the coronary arteries, suggests that in coronary spasm the infusion which contains digitonin should be a more useful preparation than the alcoholic extracts which contain no digitonin.

Digitalis is a drug of great power, our most potent agent in correcting certain defects of myocardial function, but its promiscuous use is not without danger and may be the direct cause of myocardial damage. In some cases it is absolutely contraindicated, in others it should be used boldly and with confidence. In those in which its use is of doubtful value, it should be administered with extreme caution.

OPIUM

and its alkaloids are not contraindicated by myocardial lesions *per se*, although the associated derangement of other organs (e.g., the kidneys) may make their use inadvisable. Their effect on the heart is probably entirely due to a stimulation of the vagus center in the medulla. In animals large doses of morphine slow the heart rate. This effect may be prevented or abolished by cutting the vagi or by the administration of atropine. It may produce‖ a sino-auricular or an auriculo-ventricular block and other forms of arrhythmia, notably sinus irregularities. It is believed that these changes are entirely due to vagus influences† (some hold that the accelerators are also depressed) and that morphine has no direct action on the muscle of the heart. In man therapeutic doses of morphine produce bradycardia in susceptible individuals.

Morphine is of considerable value in some cardiac emergencies, such as the paroxysmal tachycardias and other rapid hearts associated with conditions of great excitement and restlessness.

According to Macht,‡ the various alkaloids of opium differ in their effect on the coronary arteries. *Thebain, heroin* and *codeine* have little influence on controlling the lumen of these blood ves-

*Jour. Pharm. and Exp. Therap., 1913-14, v, 76.
‖Eyster and Meek: Heart, 1912, iv, 59.
†Cohn: Jour. Exp. Med., 1913, xviii, 715.
‡Macht: Jour. Amer. Med. Assn., 1915, lxiv, 1489.

sels; *papaverin* causes a marked relaxation and, therefore, should be useful in anginal pains due to coronary spasm.

THE NITRITES

in man cause a considerable acceleration of the pulse through reflex influences acting on the vagus center. This activity may be so marked that apparently the vagus is completely inhibited so that the condition simulates full doses of atropine. It seems pretty well established, however, that these influences are exerted on the medullary center and not, as is the case with atropine, on the terminations of the vagus nerve. Experiments on animals and clinical observations have failed in affording evidence that the nitrites have any direct action on the heart muscle. Hence, when it is indicated by reason of its other activities, it is not contraindicated on account of any action detrimental to the myocardium.

EXTRACTS OF THE POSTERIOR LOBE AND THE PARS INTERMEDIA OF THE PITUITARY GLAND have a considerable effect on the peripheral arteries and, aside from this, probably a direct action on the heart muscle. According to Wiggers,* they slow the heart, decrease its amplitude and increase its muscle tone.

STRYCHNINE

has no direct action on the cells of the myocardium. Its effect on the heart is an indirect one only and, therefore, it cannot fairly be classed as a drug useful in correcting intrinsic heart defects.

*Amer. Jour. Med. Sc., 1911, cxli, 502.

CHAPTER XIX

Treatment

INDICATIONS AFFORDED BY THE DIFFERENT TYPES OF RHYTHM

It is well to emphasize at the outset that the individual arrhythmias do not constitute distinct entities. They are merely symptoms of abnormal myocardial activity. At times an arrhythmia is the only means through which we may detect functional disorders of the heart, at others it is merely one of a large group of signs which demonstrate the defective character of the method in which the heart is performing its work. Their special value is found in the fact that they often reveal the particular fundamental properties of the muscle cells which are at fault, and thus suggest a new point to be attacked by therapeutic measures. The treatment of symptoms is often of very great value not only because a symptom in itself may at times actually endanger life, but also because its removal may be the starting point which leads to the correction of the more fundamental defect. The relief of pain during an acute infection may afford the rest which the body needs to reorganize its forces to combat successfully the toxins which are threatening its life. A bradycardia or a tachycardia may, through its intensity or frequent repetition, jeopardize life quite aside from the intrinsic influence which the underlying lesion may have on the circulation.

For these reasons I think we are fully justified in considering the arrhythmias in separate groups, both for the sake of such clues as may thus be afforded for their correction, as well as for the light which such a classification may throw on the conditions more fundamentally at fault.

It is needless to say that there is no drug or therapeutic measure which is a specific in correcting all irregularities of the heart. One form of treatment may be most beneficial in one type of arrhythmia and absolutely contraindicated in another type. This becomes perfectly apparent when we consider that to produce the different irregularities, modifications of different fundamental properties of the myocardial cells are necessary. A measure which may satis-

factorily control one form of arrhythmia may increase or even bring into being another type of irregularity.

HEART BLOCK

is the type of abnormality which indicates that there is an interference with the conduction of stimuli from one portion of the myocardium to another. Fundamentally, the object for treatment is to restore to the normal the functional capacity for conduction.

The methods to be selected in the treatment of heart block depend upon the etiology, the degree of the impairment of the property of conduction and the functional efficiency of the heart.

One most often sees a condition of *delayed conduction,* or of *partial block,* in the course of one of the acute infections, notably in cases of rheumatic fever. Usually this functional disturbance is transitory and passes off with the elimination of the toxins of the acute process. The treatment is entirely directed toward the general disease, antitoxin in diphtheria, salicylates in rheumatism, etc., free elimination in all. No treatment is especially indicated by the myocardial defect, which doubtless is frequently a chemical alteration rather than any histological change. If the conduction abnormality persists after the acute stages of the disease and into the period of convalescence, the patient should limit his activities for a considerable time, in the hope that the heart, not unduly taxed, may recover its normal function. If, after a reasonable period, the defect appears to have become permanent, the patient may gradually resume his usual activities, but under the careful observation of his physician, in order that an increased degree of block or any cardiac insufficiency may be detected at once and be corrected by appropriate measures.

The change from partial to complete block is, of course, always abrupt, but there are certain cases which show what we may designate as a *transition period;* that is to say, a very moderate block may become a partial block of marked degree and then return to a block of less severe type, or a partial block may become complete and in the course of a few hours the rhythm may revert to the partial type. As has been pointed out, it is during this period that the patient is most likely to develop syncopal attacks and hence is in a condition pregnant with considerable danger.

During this period, rest in bed should be insisted upon; this not only minimizes the possible dangers from the attacks of unconsciousness, but also reduces the demands on the heart to the lowest degree, thus possibly preventing a more advanced type of block and avoiding the attacks of syncope. It is in this period that atropine may often be used to advantage. Of this more will be said in a later paragraph. Digitalis is contraindicated.

Those in whom *complete dissociation* is thoroughly established usually can resume a moderate degree of activity, unless this is found to reduce the ventricular rate or to induce attacks of syncope. Those who are subject to attacks of unconsciousness must limit their activities, but are not, as a rule, confined to bed. They should be warned of the dangers which the fits entail and should never go about unattended. Some of these syncopal attacks seem to have a definite exciting cause, such as the stress of over-exertion or a reflex induced by gastrointestinal disturbances. Such causes should be thoroughly searched for, and the necessary measures for their avoidance instituted. There are reports in the literature which seem to indicate that some attacks of unconsciousness can be warded off or aborted by the use of atropine. We have no other means of relieving the syncopal attacks and I see no reason why atropine should not be tried.

With few exceptions, complete block, when once established, continues until the patient dies. From our knowledge of the pathology, we can rarely expect to restore the tissues to a normal capacity for conduction; however, the possibility of a syphilitic lesion and its removal should never be overlooked. In the presence of the history of infection and a positive Wassermann reaction, a vigorous course of antisyphilitic treatment is always indicated and even those in whom the evidence of lues is less convincing are entitled to the benefit of the doubt. The administration of mercury and potassium iodide should always precede the exhibition of salvarsan, which should be introduced cautiously and in small doses, which may gradually be increased. Success in securing a return of complete conductivity has been reported in some cases. The failure of antisyphilitic measures to restore the normal function does not prove that the scar which remains may not have been of syphilitic origin.

Nearly all grades of block are associated with a greater or less

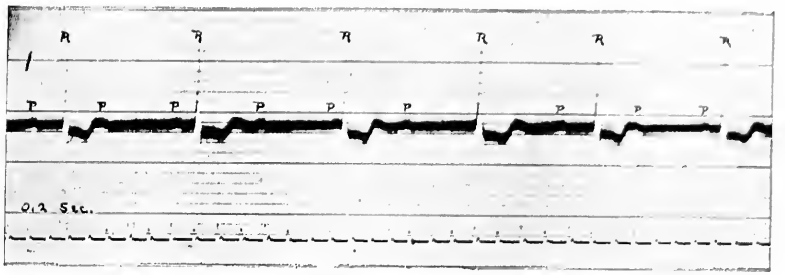

FIGURE 218
November 20, 10.51 A.M. Complete block. $As = 85$; $Vs = 46$.

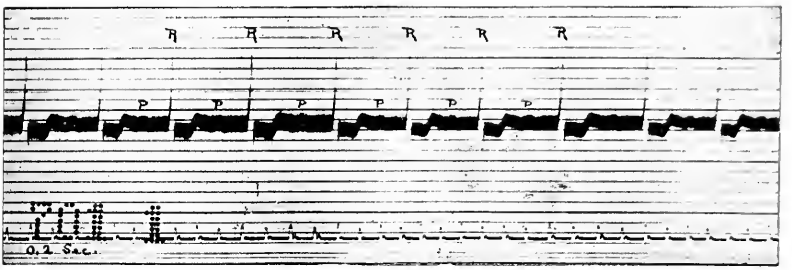

FIGURE 219
November 20, 10.56 A.M. Delayed conduction: twenty-five minutes after atropine *P-R* interval = 0.4 second. $As = 85$; $Vs = 85$.

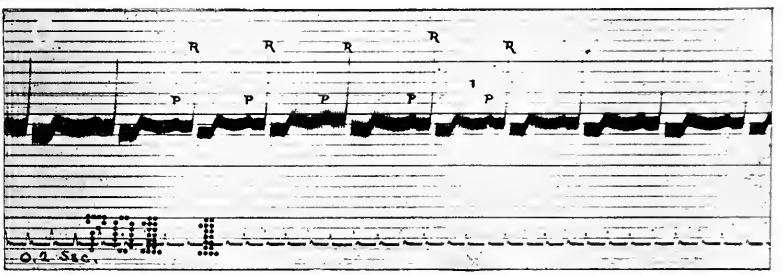

FIGURE 220
November 20, 11.01 A.M. Conduction slow. *P-R* interval = 0.22 second, $As = 85$; $Vs = 85$.

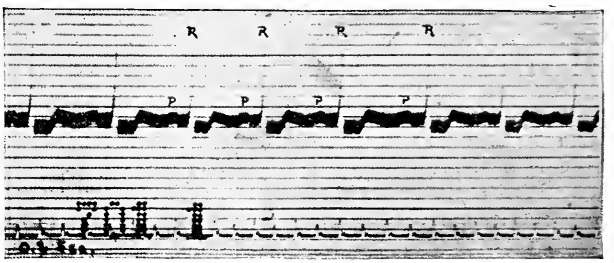

FIGURE 221
November 20, 11.06 A.M. *P-R* interval = 0.2 second; $As = 80$; $Vs = 80$. Complete block broken by the administration of atropine.

degree of cardiac insufficiency. This does not necessarily parallel the severity of the dissociation. This factor requires quite as much attention as the block, and for each patient appropriate measures must be employed to improve this phase of his difficulty.

As has been pointed out in another place, the activity of the vagus is sometimes an element in producing block. Although I believe it must be unusual for a vagus block to occur without other myocardial defect, it should always be taken into consideration as a possible factor. The administration of atropine may be used to remove vagus influences and thus determine their relative importance. For this purpose it may be employed subcutaneously in doses of 1.3 mg. (1/50 grain). Its effect should be apparent within thirty minutes. The effect of atropine usually disappears in a few hours. In some cases it is desirable to continue its influence and for this purpose 0.7 mg. (1/100 grain) may be given by mouth or subcutaneously at six-hour intervals for several days. The prolonged administration is apt to cause the patient considerable discomfort; his mouth becomes dry, and he cannot use his eyes on account of the loss of accommodation, hence its use must be discontinued.

Atropine is useful to counteract the effect of digitalis, when this has been given to a point where its toxic effect has become evident through the development of heart block. Here it probably influences only the vagus part of the digitalis action.

The graphic evidences of the effect of the administration of atropine are shown in Figures 218, 219, 220 and 221. These records were obtained from a man who entered the hospital with signs of marked cardiac insufficiency, mitral incompetence and a dilated left ventricle. His heart was rapid and rhythmic, but showed a prolonged conduction interval (P-$R = 0.2$ second). He was given digitalis and after four days of its administration the heart suddenly became very slow (rate 46). An electrocardiogram taken at this time (Figure 218) presented the evidences of a complete heart block. He was given 1.3 mg. atropine subcutaneously, and the effect is shown in the succeeding records. Figure 219, taken twenty-five minutes after the atropine was given, indicates that there is no longer a block, although the conduction time is very long (P-$R = 0.4$ second) and the ventricular rate has increased to

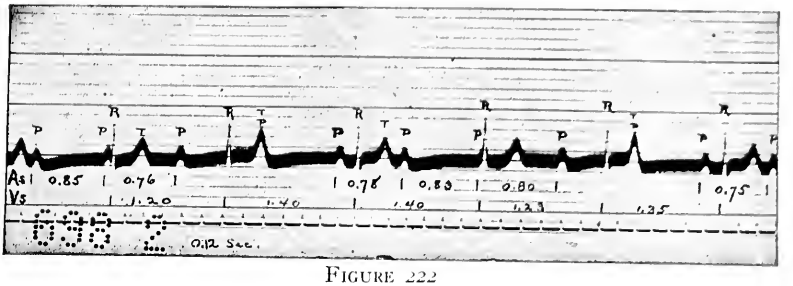

FIGURE 222
November 18, 3.40 P.M. Complete block. Before atropine. *As*=75; *Vs*=48.

FIGURE 223
November 18, 4.07 P.M. Complete block twenty-five minutes after atropine. *As* = 90; *Vs* = 52.

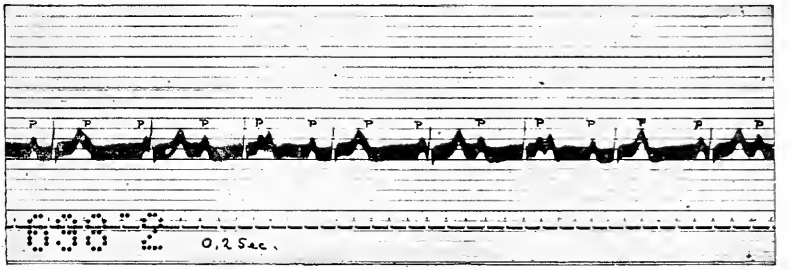

FIGURE 224
November 18, 4.10 P.M. Complete block. *As* = 100; *Vs* = 60.

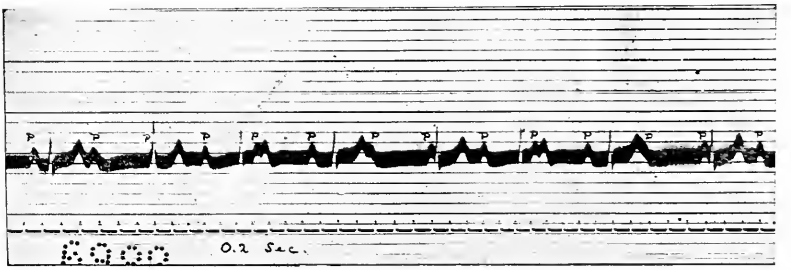

FIGURE 225
November 18, 4.12P.M. Complete block *As* = 100; *Vs* = 60.
A case of block not removed by atropine administration.

85 per minute. Five minutes later (Figure 220) the heart rate had not changed, but the *P-R* interval was reduced to 0.22 second. Thirty-five minutes after the atropine (Figure 221) the ventricular rate was 80, the *P-R* interval 0.2 second. This was the maximum atropine effect, which gradually wore away until, at the end of eight hours, complete block again appeared.

The results of the administration of atropine to another patient with a complete heart block (cause unknown) is presented in Figures 222, 223, 224 and 225. It will be seen that atropine did not abolish the block which remained complete throughout the period of observation. It did, however, cut off the vagus influences, with the result that both the auricular and ventricular rates were increased.*

The question of the administration of digitalis to patients showing defects of the property of conduction must be studied in each individual. To many showing signs of cardiac insufficiency and dilatation, its use is beneficial. In cases of established complete block, it is not contraindicated unless it produces excessive ventricular irritability and extrasystoles. In cases of delayed conduction, it may be given with caution. To those with partial block, it is usually harmful. Every patient with conduction defects to whom digitalis is given, should be under the close observation of his physician until its effects are fully established.

EXTRASYSTOLES

Our present knowledge indicates that an extrasystole represents an increased irritability of some portion of the myocardium. Whether this is due to a chemical alteration of the muscle cell, histological changes in the cell or to extracardial nervous influences which increase the excitability of the cell, is not altogether clear. From the evidence at our disposal, we may make the assumption that in different patients exhibiting extrasystoles the cell modifications are not necessarily of the same nature and that in one it may be a true histological, in another a chemical change and in a third a nerve influence. The object of therapeutic measures is to reduce the myocardial irritability and the assumption that the change in this property may be the result of such diverse influ-

*Hart: Amer. Jour. Med. Sc., 1915, cxlix, 16.

ences afford us several points of attack which may be considered in each case according to its individual merits.

There are certain substances which have an undoubted influence in the production and maintenance of extrasystoles. They probably act as direct toxins to the myocardial tissues. These are tea, coffee, tobacco and certain drugs, such as digitalis, theocin, adrenalin, chloroform, etc. The reason for the susceptibility of some hearts to these substances is entirely unknown. Some individuals can apparently use excessive quantities of tobacco with entire impunity, while in others a small amount of nicotine will produce a high degree of myocardial irritability. This I have repeatedly seen in susceptible individuals who have been closely observed to determine this point. In the same way certain patients show a similar tendency with digitalis in small doses, while others will take very large amounts without evidence of increased myocardial irritability.

The first attempt to reduce the excessive cardiac irritability should consist in the withdrawal of all such toxic substances. In many instances the discontinuance of the use of tea, coffee or tobacco will result in the disappearance of extrasystoles, in others the cure is not so simple, but it is not fair to condemn this method until it has been given a prolonged trial.

Another apparent source of myocardial irritability is an abnormal condition of the gastrointestinal tract. Whether such digestive disturbances act by producing substances which are direct toxins to the heart muscle or by purely reflex nervous influences is not known, but it seems very clear that in a certain number of instances the extrasystoles will disappear with a correction of the gastrointestinal disorder.

It is also wise to search for other possible sources of endogenous toxins or irritants which may cause an abnormal reflex and to remove them when possible.

A distinction should be made between the extrasystoles which are associated with other evidences of myocardial damage and those which occur as the sole evidence of disturbance of heart function. In the former case our treatment may be mainly directed to a correction of the more fundamental cardiac defect and often the extrasystoles will become less evident with the improvement of the other features. These are the patients in whom we are justi-

fied in trying the effect of digitalis, but it should be used with care, only under close observation and should be discontinued if it increases the irritability out of proportion to its otherwise beneficial effect. In those in whom extrasystoles are the only sign of abnormal cardiac activity, digitalis is contraindicated; they are often made worse by its administration. These patients should be told that they have no serious heart lesion, they should not curtail their ordinary physical activities and, if they are leading a sedentary life, they should be introduced to regular exercise in the fresh air and take a course of carefully graduated physical exercises, which in a very large number of instances will abolish the extrasystoles altogether. I know of no drug which will directly improve the cardiac condition, although the use of one of the bromides or valerian may be of service in tiding over a period of anxiety and apprehension.

While the patient's mind should be put at rest and he should be reassured, I think it is a matter of some importance that we should get rid of the extrasystoles if we can by the simple hygienic measures which we have outlined above. I have the impression that by allowing extrasystoles to go unchecked the abnormal focus in the myocardium acquires, as it were, a habit of assuming the rôle of pacemaker for the heart, and this, if uncontrolled, tends to become fixed and may lead to the development of abnormalities of more serious moment.

THE ACCELERATED HEART

In seeking methods of slowing the heart that is beating at too rapid a rate, we should always endeavor to single out the cog in the mechanism which is defective. When the demands of physical exertion exceed the capacity of the heart muscle, it must be curtailed; if it has led to actual heart strain and dilatation, the patient must be put to bed and digitalis may be found of benefit. If the excessive stress arises from the abnormal functional activity of other organs, these must be corrected as far as possible, and during the process the heart strength must be conserved by rest. When the increased rate is due to excessive accelerator influences, the cause may often be found in the deranged function of some organ remote from the heart. By rectifying this disorder, the

abnormal reflexes will be excluded and the tachycardia will disappear. In these conditions sedatives, such as the bromides and valerianates, are often of considerable service.

It is probable that by far the greater number of simple tachycardias are due to poisons which destroy the balance of the extracardial nerves or more often actually increase the irritability of the sinus node, such, for example, are tea, coffee, tobacco, alcohol, thyroid extract and the toxins of bacterial infection. The indication is clear that the absorption of these substances must be curtailed and their elimination facilitated. Beyond this, rest and the application of cold to the precordium are the means which afford the best results. Drugs such as digitalis, strychnine, aconite, etc., will be found of distressingly little value.

PAROXYSMAL TACHYCARDIA

Since our conception is that these attacks are due to the excessive irritability of some point in myocardial tissue, which, therefore, assumes the rôle of a pathological pacemaker, our efforts are directed to reducing this irritability to the normal. With this end in view, we study the condition of the heart between the paroxysms and correct, as far as may be, such abnormal functional activities, adjusting the patient's mode of life to the amount of stress which his heart is able to support. We endeavor to remove elements which may act as exciting causes to the paroxysm, such as sudden physical exertion, emotional excitement and gastrointestinal disturbances. We put the patient in the best possible physical condition in the hope that the attacks may be less frequent or that he may be better able to cope with the paroxysms when they arise. When the attack comes on, the patient should at once go to bed; this they usually do without advice, but exceptionally one sees an individual who will interrupt his activities only for a moment at the beginning of the seizure and will then go on with whatever he may be doing with seemingly little discomfort.

The paroxysms are prone to terminate spontaneously, and are so variable in length that it is exceedingly difficult to estimate the value of measures employed to arrest them. One fact is very certain, we have no one single means which invariably stops the attacks. In certain individuals the attacks can be stopped by various muscu-

lar movements or by assuming some special posture. The patients themselves will often discover the "trick" by which this end is attained or the physician may find one suited to the patient's needs by testing the methods which have proved successful in others. The following are a few of the means which may be tried: holding the breath after deep inspiration; strong deglutition movements (as has been suggested by Vaquez, this may be accomplished by having the patient swallow several large cachets containing some inert drug or other substance); assuming various bodily attitudes, such as lying on a couch with the head projecting beyond the edge and thrown back as far as possible, or flexing the head forward and bringing it down between the legs with the body curled up; slapping the chest; a long drink of very cold water; vomiting induced by tickling the pharynx or the administration of an emetic, such as mustard, zinc sulphate or the syrup of ipecac; cold applications to the chest wall. It is probable that all these methods act by stimulating the vagus either by mechanical pressure or by reflex influences. One can frequently stop an attack temporarily, sometimes permanently, by direct vagus pressure. This is accomplished by making digital pressure over the carotid sheath just below the angle of the jaw. Right vagus pressure is usually much more efficacious than left vagus pressure (see Figures 152 and 153); the nerves should be manipulated one at a time, compression should never be applied to both vagi at the same moment, as there is a possibility of causing serious arrest of the heart. Ocular pressure may be tried, but it is, as a rule, less effective than vagus pressure.

Digitalis is of little or no value in the short paroxysms, but in attacks lasting several days it may be pushed to the physiological limit. In the tachycardias of auricular origin we may hope to induce an *A-V* block; in those rare paroxysms, which arise from a focus in the wall of the ventricle, digitalis is contraindicated.

The intravenous use of crystalline strophanthine has been found successful in arresting the paroxysms on a number of occasions. A single dose of one-half milligram, dissolved in 8,000 parts of normal saline, may be given. This dose may be repeated after an interval of twenty-four hours.

No one of the above measures is always successful. Some paroxysms resist all the attempts made to arrest them and ulti-

mately stop spontaneously. Under these circumstances the patient must be made as comfortable as possible. He is allowed to assume the posture in which he is most at ease. Quiet and rest are attained by the administration of bromides, valerian, or, in conditions of great distress and restlessness, a hypodermic of morphine. There is a long list of drugs which have been reported to be efficient in stopping the attacks, among these are aconite, amyl nitrite, strychnine, veratrine, hypophysis extract, etc., etc. These probably are without any real effect and owe their reputation to the coincidence of their administration with the time of the spontaneous termination of the paroxysm.

SINUS ARRHYTHMIAS

We have seen that cardiac irregularities arising in the sinus node are due to a condition of the nodal tissue which makes it peculiarly susceptible to vagus influences. When the *rhythmic changes are synchronous with the respiratory movements,* we may regard them as entirely physiological, and as such they require no treatment. They do not indicate an abnormal condition of the myocardium, hence there is no reason for subjecting the individual presenting this irregularity to unusual restrictions or for limiting his accustomed activities.

The *irregularities of sinus origin, which are independent of the respiratory movements,* have a somewhat different significance. Here, I believe, that the cause of the instability of the nodal tissue is usually a true myocardial defect of which the functional change of the sinus node is often the earliest evidence. It is certainly wise that these patients should be closely watched, with the purpose of detecting at the earliest possible moment further signs of myocardial damage, should these present. In themselves, these irregularities are of little consequence; they do not demand treatment. These patients never show cardiac insufficiency without presenting signs of abnormalities of the heart other than this arrhythmia. When the irregularity is first discovered, the patient's activities should be moderately restricted, in order that the heart may not be subjected to any considerable stress pending the determination of the extent of the myocardial damage. If at the end of a reasonable period it appears that the irregularity is the only evi-

dence of disturbed function, the restrictions may be gradually relaxed.

AURICULAR FLUTTER

Auricular flutter indicates an exceedingly irritable condition of the auricle, which is contracting rhythmically at a very rapid rate. It is usually associated with a defect in the *A-V* junctional tissues, so that a part of the auricular impulses are blocked and the ventricle responds only to every other stimulus originating in the upper chamber. Hence the object of our therapeutic measures is *first* to increase the degree of block and thus reduce the number of the auricular stimuli which are able to reach the ventricle, and, *second,* to diminish the irritability of the auricular tissues so that the sinus may regain its ascendency and resume its rôle of cardiac pacemaker.

During the attack the patient should be in bed and as nearly horizontal as possible. Frequently the patient breathes more easily, if the head is somewhat elevated, and during urgent dyspnœa it may be necessary to allow him to sit up in a chair. He must be kept as quiet as possible and all friends and sources of emotional excitement must be excluded. Sleep and rest may be secured by the administration of bromides and valerian or even morphine, if the restlessness is excessive. Food should be given frequently in small amounts and in an easily digested form. As a rule, large amounts of fluid should be avoided.

As in the case of "paroxysmal tachycardia," which seems to bear a very close relationship to auricular flutter, stimulation of the vagus will sometimes slow the ventricles and even arrest the paroxysm. A number of methods for attaining this result have been detailed on page 286, and need not be repeated here. The effect of vagus stimulation is, however, usually transitory and is unreliable as a lasting therapeutic measure. On theoretical grounds the stimulation of the left vagus should have a greater effect in slowing the ventricles, while the right vagus should better control the auricular tachycardia. That this is not always the case is shown in Figures 226 and 227. These are portions of records secured from a man of forty-nine during an attack of auricular flutter lasting several days. His auricles were contracting at a rate of 300 per minute; his ventricular rate was 150 per minute. At the point in Figure 226 indicated by the arrow, digital pressure was made

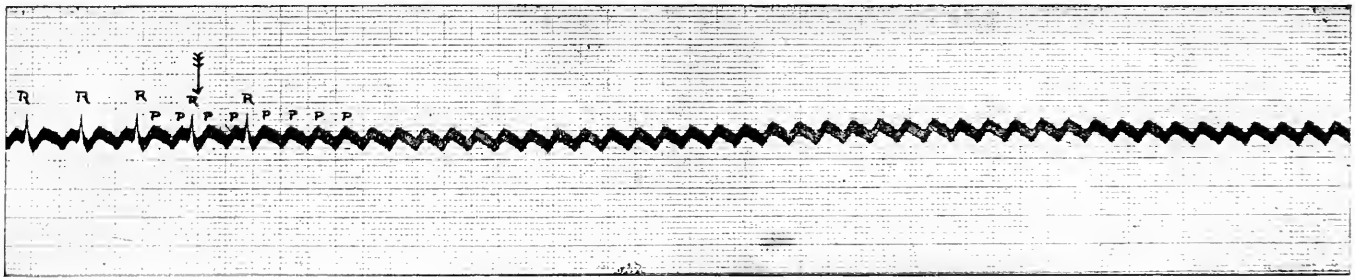

FIGURE 226

Case of auricular flutter. Auricular rate 300. Ventricular rate 150. The arrow indicates the time at which *right vagus pressure* was made. The ventricle did not contract while vagus pressure was continued.

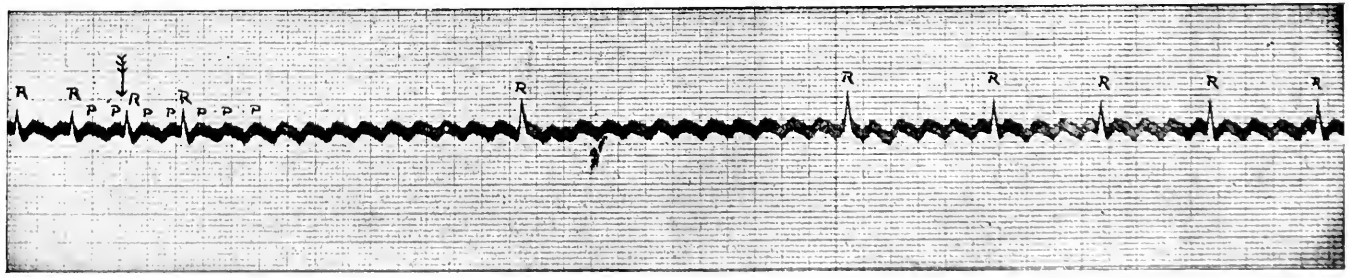

FIGURE 227

Same case as Figure 226, taken eight minutes later. *Left vagus pressure* was made at the time indicated by the arrow and continued for 10 seconds, the ventricle was at once slowed, but shortly after "escaped" while vagus pressure was still being made.

on the right vagus nerve and continued for ten seconds. During this time the ventricles did not contract, the auricular activity was not affected. One second after pressure was discontinued the ventricles resumed their contractions at a rate of 25 per minute. This rate gradually increased and at the end of five minutes the ventricular rate had returned to 150. The effect of left vagus pressure is shown in Figure 227. Here again it is evident that the auricular tachycardia is unchanged. There was a ventricular escape at the end of 2.4 seconds and the intervals rapidly shortened and a ventricular rate of 150 per minute was attained in nine seconds, while vagus pressure was still being made.

The drugs which are of most value in auricular flutter are digitalis and others belonging to this group. The object to be sought is an increase in the degree of auriculo-ventricular block, so that a portion of the impulses originating in the irritable auricle may be obstructed and the ventricular rate reduced. In order to secure a prompt effect digitalis must be administered in large doses. It may be given subcutaneously if the patient is in great distress, or if immediate relief is demanded strophanthin may be given intravenously, observing the pracautions that have already been suggested in the use of this powerful alkaloid. Exhibited by mouth in full doses, digitalis may require several days to reduce the ventricular rate to the normal, but a considerable slowing is usually seen in forty-eight hours. When the ventricular rate reaches about 70 per minute, although the auricles are usually still in a condition of flutter, the digitalis may be stopped, in the hope that a physiological rhythm may be recovered. If the ventricle shows a tendency to increase its rate, digitalis should be resumed. It is quite probable that soon after this the auricles will begin to fibrillate, but often the physiological rhythm will reappear with no intervening period of fibrillation. The question has been debated as to whether the fibrillation is due to the administration of the digitalis. This seems quite probable, hence it would seem wise to employ this drug only to a point where it controls the ventricular rate within reasonably normal limits, and from time to time to discontinue it, carefully observing the functional condition of the heart.

A typical favorable reaction to digitalis is shown in Figures 228,

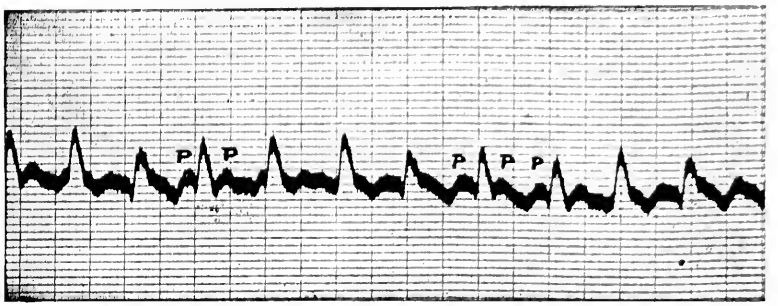

FIGURE 228 April 6. Before treatment. Auricular flutter with irregular ventricular contractions 150 per minute.

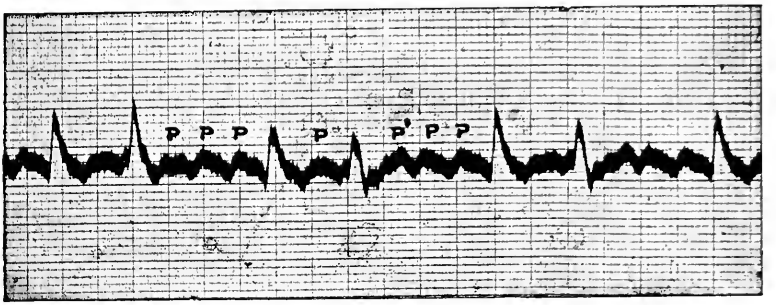

FIGURE 229 April 9. Ventricle irregular rate 100.

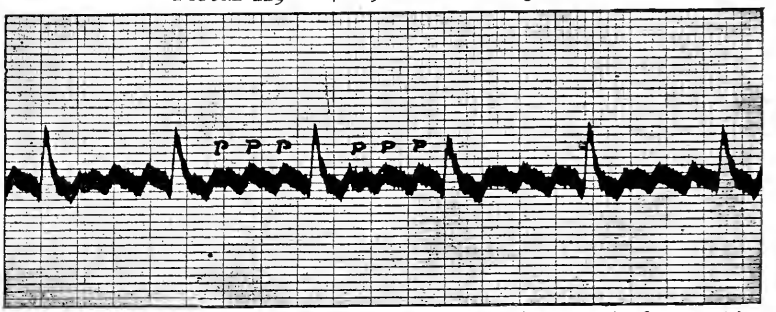

FIGURE 230 April 15. Ventricle regular, rate 75. Auricles still in flutter. After this digitalis was stopped.

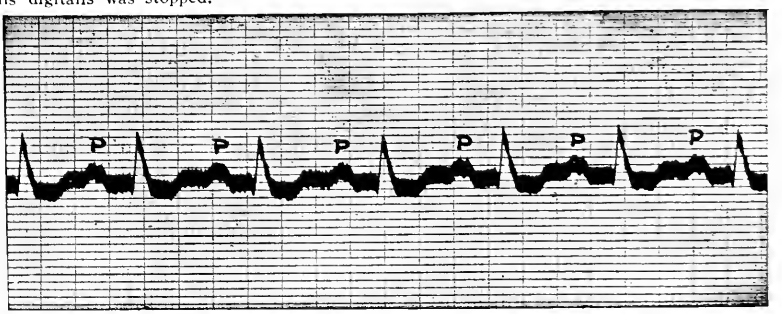

FIGURE 231 April 22. Regular sequential rhythm. Auricular flutter has stopped.

The above four records show the effects of well-regulated digitalis administration in a case of auricular flutter.

220, 230 and 231. On April 6, before treatment (Figure 228), the ventricular rate was 150 per minute; under large doses of digitalis the rate was reduced to 100 (Figure 229). The quantity of digitalis was then reduced, and on April 15 the activity was that presented in Figure 230. The auricles were still in flutter, with rhythmic contractions at the rate of about 300 per minute; the ventricles were also perfectly rhythmic, but responded only to every fourth auricular impulse. At this point digitalis was discontinued and a few days later the heart returned to a sequential rhythm (Figure 231). At no period was auricular fibrillation observed in this patient.

About one-half of the cases of auricular flutter recover a physiological rhythm, many of them pass into auricular fibrillation and, in a considerable number, this is continued for the remainder of life.

The use of potassium iodide, in association with digitalis, has given favorable results in some cases.* If there is evidence of a syphilitic infection, a course of mercury and iodide is indicated.

AURICULAR FIBRILLATION

It is most generally held that this condition is caused by a highly irritable condition of the auricular wall. The disturbances of the circulation are almost entirely due to the secondary effects on the ventricles, which are induced by the abnormal stimuli showered upon the junctional tissues by the frenzied activity of the auricles. The purpose of treatment is, therefore, to reduce the irritability of the auricles, in the hope of securing coördinated contractions of the auricular muscle. Failing in this, we should attempt to obstruct a portion of the impulses set free in the upper chamber and thus relieve the ventricles of the stimuli, which lead to such rapid and ineffectual contractions.

Our means to reduce the excitability of the auricular muscle are limited and most often unavailing, yet I believe that in every case of auricular fibrillation discovered near the time of the inception of the new rhythm, an attempt should be made to bring the auricular activity back to the normal. It has been pointed out that a sudden increase in intra-auricular pressure is probably an important factor in inducing the onset of fibrillation in a heart previously

*Ritchie: Auricular Flutter, 1914, p. 132.

damaged by disease. Anything which we can do to prevent venous congestion and overfilling of the auricles of a heart in which we suspect myocardial defects, should be employed as a prophylactic against fibrillation. If fibrillation has commenced, rest in bed is at once indicated, and in cases with evident venous stasis and overdistension of the auricles, a prompt phlebotomy may sometimes relieve the increased pressure and permit the auricle to resume a physiological activity. The active elimination of the toxins of the acute infections or of other poisons may be accompanied by the reappearance of normal auricular contractions. If the ventricular rate becomes very rapid, digitalis may be employed in full doses until the ventricles are slowed to about 90 a minute, but it should then be discontinued, for digitalis, undoubtedly, has the effect of increasing the irritability of the muscle cells of the heart, and in these early cases it is wise to remove this influence in the hope that the auricles may recover their normal coördinated contractions.

As has already been stated, our efforts to reduce auricular excitability and to secure a return to a normal rhythm are usually unsuccessful, auricular fibrillation becomes established and will probably continue to the end of life. It is in these cases, however, that the treatment of abnormal myocardial function obtains its most brilliant successes.

The attention of the physician must be centered on the ventricular activity, if the rate of the lower chamber is not over 75 per minute, and the heart is well compensated little need be done for the patient other than to see to it that his mode of life conforms to the limited amount of force which such a heart has in reserve. Physical and emotional strain must be avoided, the excessive use of tobacco, alcohol, tea and coffee are forbidden, gastrointestinal disturbances must be corrected and exposure to infections shunned.

With a more rapid and irregular ventricular rate, the indications for treatment are quite different. The patient should be put to bed at once and kept as quiet as possible. Food should be given at frequent intervals in easily digested forms, the amount of fluids taken should usually be restricted. Rest may often be secured by one of the simple hypnotics, veronal, trional, etc. The most important object to be attained is to block a portion of the hap-

hazard auricular impulses, so that these may cease to vex the overacting ventricles. This can nearly always be secured by the administration of digitalis or strophanthus. In a considerable number of these hearts the myocardial damage is not limited to the auricular wall, but has also involved the *A-V* bundle. This makes them peculiarly susceptible to digitalis influences, and with this drug it is usually easy to produce a considerable degree of auriculoventricular block. While rest in bed is essential to the successful treatment of these cases, it is quite easy to demonstrate that this alone is not sufficient, in the majority of instances, to reduce the ventricular rate to the desired point, outside demands in excess of the functional capacity of the heart are only partly responsible for the increased rate. This is due in a very large degree to the abnormal auricular activity and until this influence is checked the ventricles cannot be satisfactorily controlled. Hence, the administration of digitalis is practically always a necessity.

How much digitalis must we give? This question can be answered only by studying the individual patient. We must give it in sufficient amounts to secure its physiological effects, and this can be determined only by observing the reaction of each heart to the drug during its administration.

The method which I have found most satisfactory is to begin by giving by mouth a good infusion or the tincture. I use 30 c.c. (1 ounce) of the infusion or 4 c.c. (60 minims) of the tincture in each twenty-four hours until a definite physiological effect is observed. This is usually seen in from three to five days. The beneficial effects of rest and the administration of digitalis are shown in the graphic records of the brachial pulse, taken at intervals of two or three days from a single patient (Figures 232, 233, 234, 235, 236 and 237). I think the best method for watching the effect of the drug is to make frequent estimations of the apex rate, the radial rate and the pulse deficit, as described in the chapter on auricular fibrillation (page 164). When the rate of the heart, determined by auscultation over the apex, falls below 90 and the pulse deficit is less than 10, the dosage may be gradually diminished, but should be continued in sufficient amount to effect a still further slowing of the heart and a diminution in the pulse deficit. As a rule, I find that these hearts are most efficient if

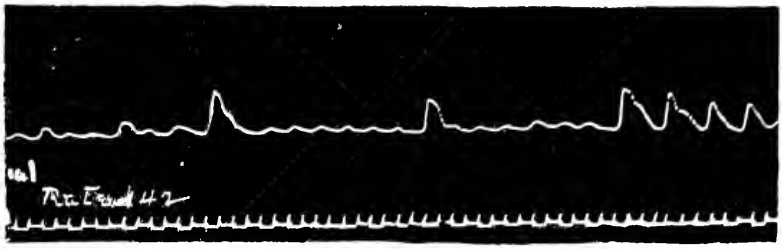

FIGURE 232 January 24. Rate 142.

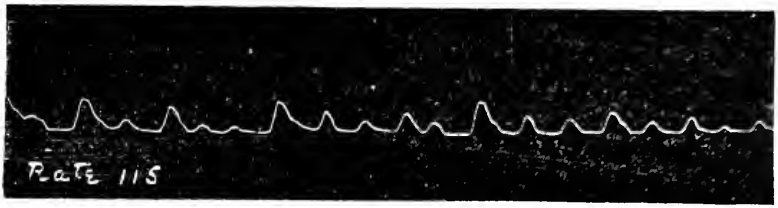

FIGURE 233 January 25. Rate 115.

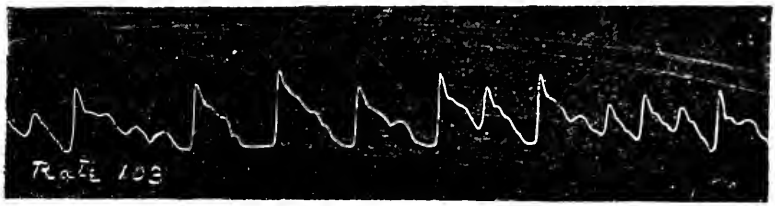

FIGURE 234 January 26. Rate 103.

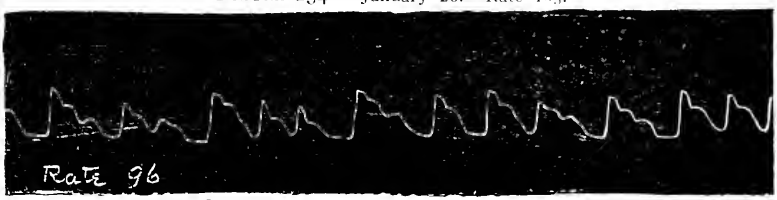

FIGURE 235 January 29. Rate 96.

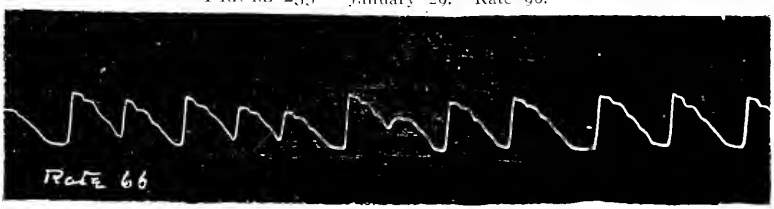

FIGURE 236 February 3. Rate 66.

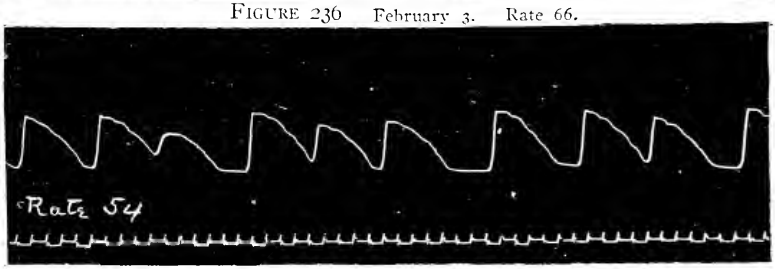

FIGURE 237 February 5. Rate 54.

A series of records taken from a case of auricular fibrillation showing the progressive effect of digitalis administration.

the rate is kept between 60 and 70 per minute with no deficit. When patients are observed in this way, it is very rare to see the disagreeable toxic effects formerly so common in digitalis therapy; nausea and vomiting are very infrequent, and it is almost never necessary to discontinue the drug on account of these symptoms. Excessively slow rates and the "coupled rhythm" are so unusual that they are curiosities. Notwithstanding the infrequency of the development of the signs of digitalis intoxication in patients to whom the drug is administered by this method, they occasionally arise and, should the heart rate fall below 50 or the "coupled rhythm" appear, digitalis must be stopped at once. When the rate has increased to 70, digitalis should be resumed in a smaller dose. The patient should be kept in bed for a week after the rate has reached 70. He may then be allowed to slowly resume his activities. Any notable acceleration of the heart or increase in the pulse deficit is a warning that he is exceeding the stress which his myocardium may safely support and his exertions must be correspondingly reduced. It is usually necessary to continue small doses of digitalis for an indefinite period. I have patients who have not missed their daily dose of digitalis for five years.

If the physical exertion is increased very cautiously and the heart rate is not allowed to exceed 70 a minute, hypertrophy will gradually develop and in some individuals the myocardium will recover an extraordinary capacity for work.

While a majority of those suffering from auricular fibrillation respond to a course of treatment as outlined above in a satisfactory manner, there are some who are exceedingly difficult to handle. These are usually cases of long-standing fibrillation which have been untreated, or those who have been treated intermittently with intervening periods of cardiac decompensation. They usually present evidences of extensive myocardial damage extending into the ventricular tissues and often show frequent ventricular extrasystoles. The polygrams of such a case are shown in Figures 238, 239 and 240. The effect of digitalis in these cases is to cause a great increase in the number of ventricular extrasystoles without materially slowing the ventricular rate. Figure 239 shows the record obtained on the second day of the administration of our usual initial dosage of digitalis. On account of the coupled rhythm,

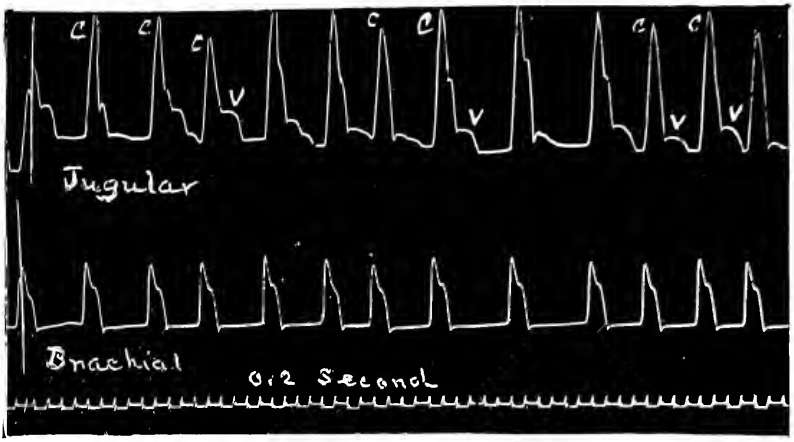

FIGURE 238

Patient "J. M." Auricular fibrillation. Record on admission to the hospital October 2.

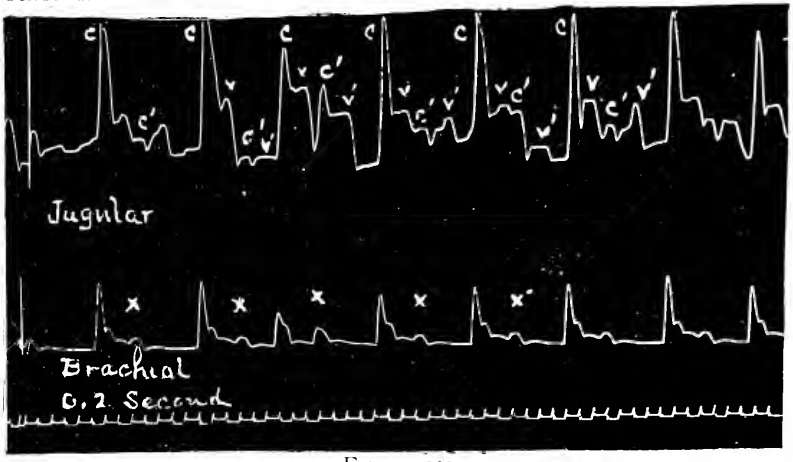

FIGURE 239

Patient "J. M." October 14. Result of the administration of 8 cc. tincture of digitalis in two days. "Coupled rhythm."

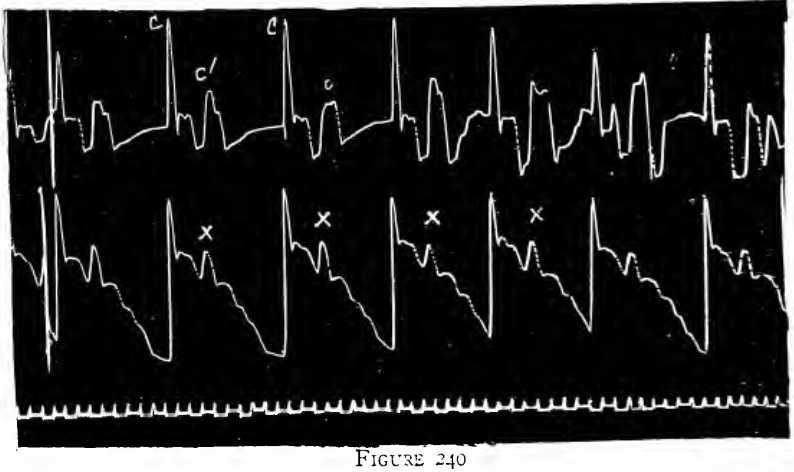

FIGURE 240

Patient "J. M." November 22. Result of the administration of 6 cc. of the tincture of digitalis in three days. Every other ventricular contraction is an extrasystole. A case of auricular fibrillation which reacted unfavorably to digitalis.

the drug was at once discontinued, the pulse remained rapid, but the extrasystoles disappeared, and five weeks later an attempt was again made to give digitalis, but in a smaller dose. After four days the coupled rhythm reappeared (Figure 240). Through rest, bromides, aspiration of chest fluid and diuresis this heart was finally improved so that it tolerated small doses of digitalis, and the patient was eventually able to leave the hospital and resume his occupation of watchman.

Each time that the ventricles are allowed to become rapid and the heart to become insufficient, it will be found more difficult to control the rate and secure a fair degree of compensation, and when to this is added a myocardial irritability, which is excessively increased by digitalis, it will require all the ingenuity of the physician to devise means to obtain an improvement in the cardiac function.

Occasionally one sees a patient whose condition is so grave that some immediate means must be employed to reduce the excessive heart rate. In these one sometimes can obtain almost miraculous results by the intravenous administration of strophanthin; this should never be given if the patient has been recently taking digitalis or strophanthus in any form.

When there is a history of a syphilitic infection and a positive Wassermann reaction, these patients should always be given a course of antisyphilitic treatment.

Figure 241 is the record of a case of auricular fibrillation three days after digitalis was commenced; two days later the pulse suddenly became very slow and on listening at the apex the coupled rhythm could be detected. The electrocardiogram taken at this time (Figure 242) indicated that the slow pulse rate was due to a complete heart block, associated with ventricular extrasystoles, which were too weak to show in the peripheral arteries.

In Figures 243, 244, 245 and 246 are presented a series of records taken from a patient during a course of digitalis treatment. On April 12 (Figure 243) the rate was 138; three days later (Figure 244) this was reduced to 100 beats per minute. On April 18 (Figure 245) the rate fell to 57 and the increased irritability of the ventricle was evidenced by the occasional appearance of extrasystoles. At this time digitalis was discontinued and

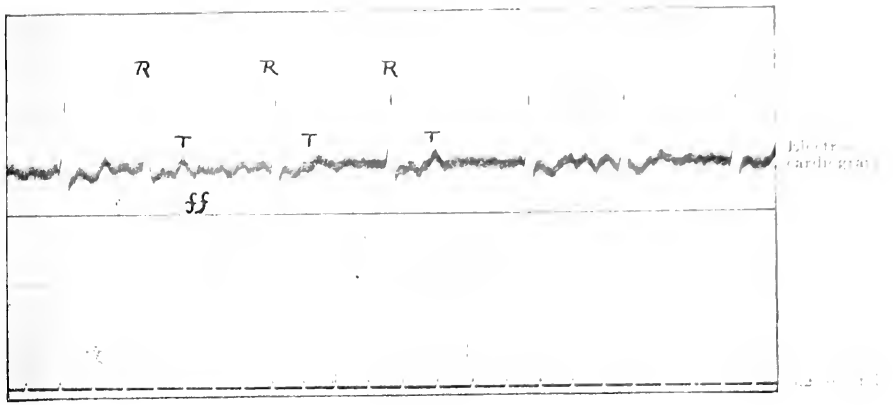

FIGURE 241

Patient "G." Auricular fibrillation. Rate 104. Digitalis had been taken for two days.

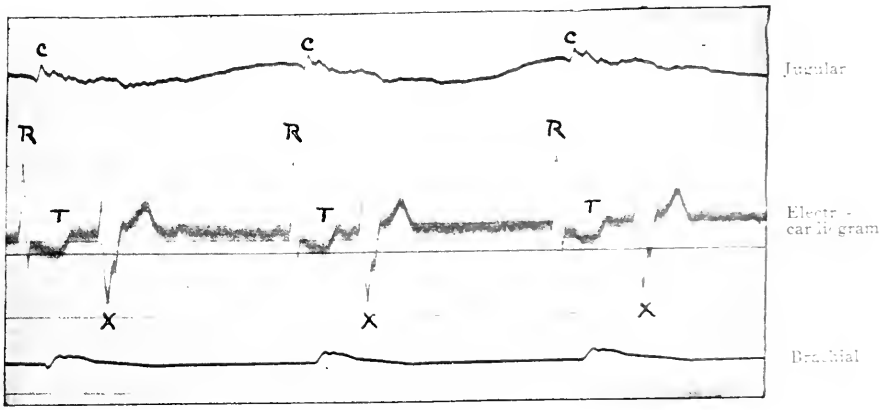

FIGURE 242

Patient "G." Fourth day of digitalis therapy. Note "coupled rhythm," every other ventricular contraction is an extrasystole which does not affect the jugular or the brachial pressures.

on May 4 (Figure 246) extrasystoles had completely disappeared and the heart rate was 94 per minute. This series of records also shows the influence of digitalis in modifying the form of the *T* wave.

The effect of digitalis on blood-pressure, in auricular fibrillation, has long been a matter of contention. Thirty years ago it was thought that the administration of digitalis elevated blood-pressure, but this view was controverted by many subsequent observers, such as Christeller, Fränkel, Heike, Hansen, Gross, Potain, and others. Their opinions have been summarized by Janeway,* who says: "All of the above observers fail to find any relation between the arterial tension and the circulatory improvement from digitalis."

Our present evidence justifies us in asserting that in the cases of cardiac insufficiency, where digitalis is of most value, it raises blood-pressure by slowing and increasing the force of ventricular activity.

The failure of former observers to recognize this fact was dependent on two elements: (1) That it was not then known that the benefits of digitalis administration are mainly evident in cases of auricular fibrillation, and (2) that they had no satisfactory method of estimating the blood-pressure in these cases, in which the successive contractions of the heart vary so greatly in force and time. Mackenzie says, in his "Monograph on Digitalis": "In our observation, even when the drug was pushed and caused nausea and heart irregularities, we could detect no appreciable effect upon the blood-pressure (except in one case)."

Since we have come to recognize that digitalis finds its chief usefulness in cases of auricular fibrillation, and have applied our method of estimating the "average systolic blood-pressure" to the study of this group, it has become clear to us that hand in hand with the improvement in the patient's condition the average systolic blood-pressure is elevated.

This is best made evident by the presentation of several charts, which have been selected from a considerable number, all of which show the same features.

Figure 126 shows the effect of rest and digitalis on a case under observation in the Presbyterian Hospital for two weeks. The dimi-

*The Clinical Study of Blood-pressure, New York, 1904, p. 210.

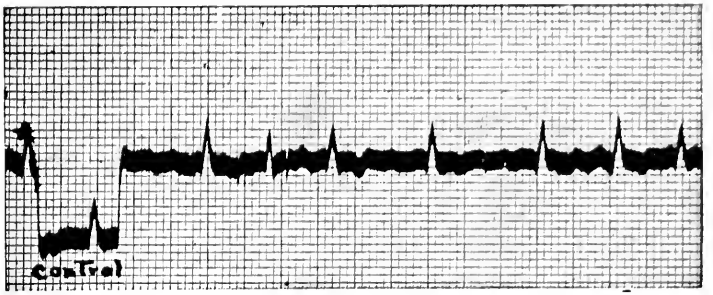

FIGURE 243 April 15. Rate 138.
(The "control curve" was made artificially to standardize the galvanometer string by introducing one millivolt of current into the circuit. All of the records were standardized in this way.)

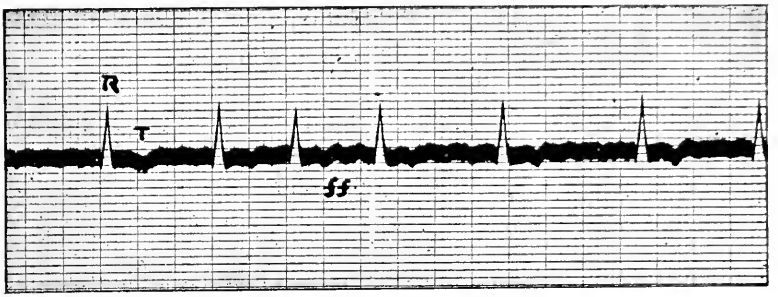

FIGURE 244 April 15. Rate 100.

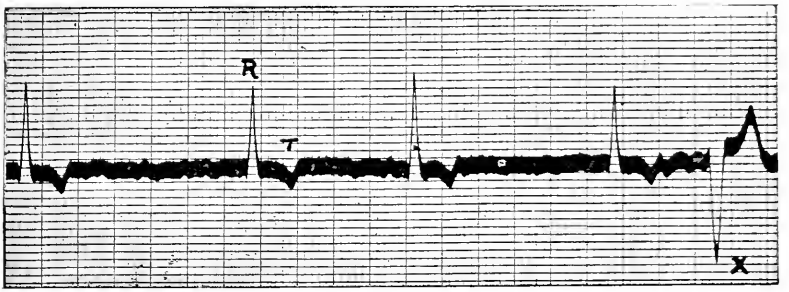

FIGURE 245 April 18. Rate 57. Ventricular slowing, appearance of extrasystoles and change in form of *T* wave.

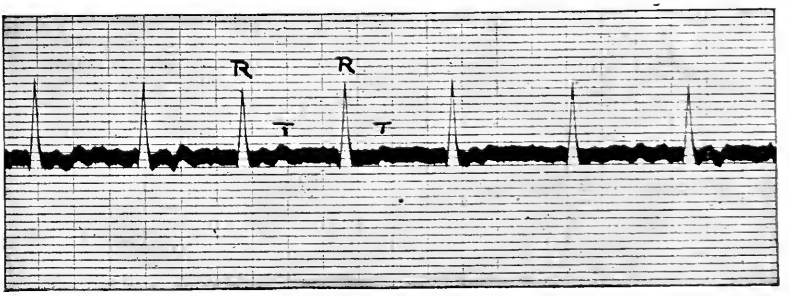

FIGURE 246 May 4. Rate 94. Digitalis stopped on April 19.
Effect of digitalis in a case of auricular fibrillation.

nution in the deficit and the gradual increase in the average systolic blood-pressure is quite clear. During this period all of the patient's symptoms improved, and she was able to leave the hospital to return to her home.

This chart also shows how misleading would have been the systolic blood-pressure estimations made by the ordinary method (see figures at the bottom of the chart), for at these brachial pressures only four or five waves per minute reached the radial during the three days following her admission. The diagram indicates that the full digitalis effect is not obtained for four or five days; this we have found to be quite usual.

Figure 247 is the chart of a man who, during the whole period of observation, insisted upon following his ordinary occupation. He was not confined to bed at any time, although we should have considered this the wisest course when he was first seen. He, therefore, illustrates the effect of digitalis independent of the influence of any considerable amount of rest in the horizontal position. The rise in blood-pressure coincident with a slowing of the pulse and diminishing deficit and the fall of pressure corresponding to an increase in the pulse rate and deficit stand out clearly. The observations cover a period of nineteen months, and the changes in the deficit and the blood-pressure could be closely correlated with his other symptoms. Whenever there was any considerable deficit or the blood-pressure fell this was associated with more or less dyspnœa, a lack of vigor and feelings of lassitude.

Figure 248 is that of a woman who was in the Presbyterian Hospital during the whole period of observation as represented by the diagram. The initial deficit on admission, which showed a marked and sudden diminution with the administration of a freshly made infusion of digitalis (an ounce was given on October 17), the first setback induced by getting out of bed, her subsequent improvement under rest and digitalis, the second setback brought on by an attack of hemorrhoids and the variations in blood-pressure, associated with the various stages of her progress, are all indicated in a graphic manner.

ALTERNATION

is a positive indication for limiting the work which the heart is called upon to perform. Although we may not as yet agree to

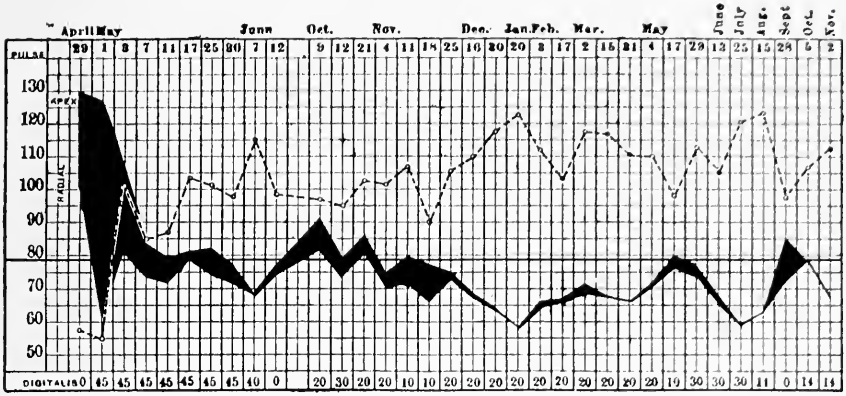

FIGURE 247

The shaded area represents the pulse deficit; the upper edge is the apex rate; the lower edge the radial rate. The broken line is the "average systolic blood pressure." The figures in the digitalis column indicate minims of the tincture per day. Patient not confined to bed.

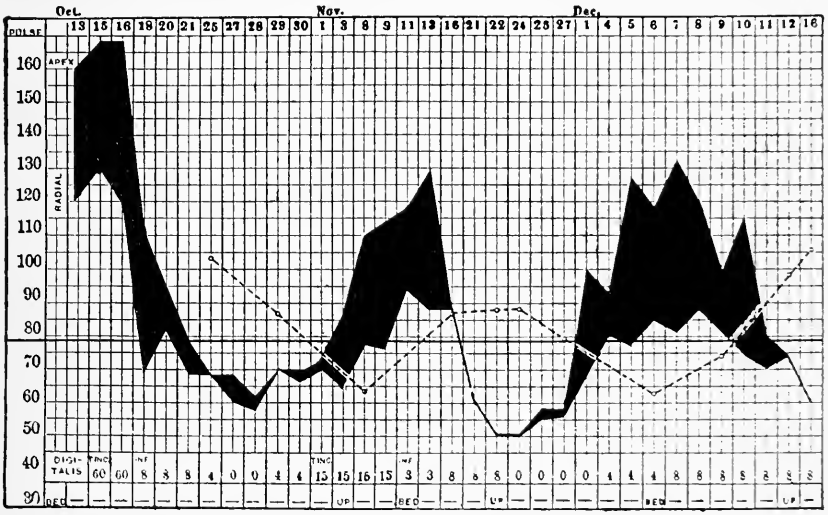

FIGURE 248

The shaded area represents the pulse deficit; the upper edge is the apex rate; the lower edge is the radial rate. The broken line indicates the range of the "average systolic blood pressure." Digitalis figures indicate minims of the tincture and drams of the infusion. October 13, admitted to hospital. November 3, up in chair one-half hour; November 9, up in chair two hours. December 4, up in chair four hours; at this time she had a crop of external hemorrhoids which caused much distress.

the exact mechanism underlying this form of irregularity, it is pretty generally conceded that it indicates a normal myocardium that is being overtaxed or more usually a very much damaged heart muscle for which even a moderate stress is too great. The soundness of this view is shown by the facts that rest in bed frequently is associated with at least the temporary disappearance of the alternation and that an alternation which is often very much in evidence when the heart is beating at an excessive rate may no longer be capable of detection when it is measurably slowed. Alternation is often a persistent matter, and it may be impossible and unwise to keep a patient in bed until it disappears. But when first discovered the patient should have the benefit of an absolute rest for at least a few days. This should be followed by gradually increasing exercise, the amount to be determined by the observations of the physician as to its effect on the activity of the heart.

It has been pointed out that while alternation is regarded by the majority of observers as an indication of defective contractility, it has not been conclusively proved that this fundamental property is alone at fault. It is certain that many cases are at least associated with a defect of one of the other properties, such as conductivity or irritability. It is also known that we can rarely produce a change in one of these fundamental properties without modifying the others. Hence, it is possible that by inducing a change in the irritability or the conductivity we may indirectly affect the property of contractility.

The above may explain the divergent views in regard to the use of digitalis in alternation. It is advocated by some workers and discountenanced by others. My own observations lead me to believe that it is of distinct value in some cases of alternation, and I have never seen harm done which I could directly attribute to the digitalis. In a case of alternation associated with auricular flutter, which I had the opportunity to follow for many months, digitalis was of undoubted value. In those cases in which digitalis slows the ventricular rate, it will often abolish an alternation. Digitalis is usually contraindicated in the psuedo-alternans due to extrasystoles.

My custom is to use digitalis in moderate doses, watching the

effect closely and stopping it after a reasonable period, if the heart does not respond favorably.

In view of the fact that alternation is experimentally readily produced by certain poisons, it seems logical that we should correct as far as possible disorders of metabolism or other sources of toxins, or at least secure their elimination as rapidly as may be. The correction of kidney and bowel functions not only assist in removing poisons, but also tend to diminish the work which the heart is called upon to perform.

According to Lewis, patients with alternation are not favorable subjects for general anæsthesia. If an anæsthesia must be used, ether, rather than chloroform, should be employed.

BIBLIOGRAPHY

BOOKS AND MONOGRAPHS

CUSHNY: *Pharmacology.* Third edition. Philadelphia. 1903.

CYON: *Les Nerfs du Cœur.* Paris. 1905.

CYRIAX: *Kellgren's Manual of Treatment.* London. 1903.

GRAVIER: *L'Alternance du Cœur.* Paris. 1914.

HIRSCHFELDER: *Diseases of the Heart and Aorta.* Second edition. Philadelphia. 1913.

HOFFMANN: *Die Electrocardiographie.* Wiesbaden. 1914.

HOFMANN: *Remedial Gymnastics for Heart Affections.* New York. 1911.

JAGIC: (Editor) *Handbuch der Pathologie, Diagnostik und Therapie Herz und Gefässerkrankungen.* Leipzig. 1914.

KAHN: *Das Electrocardiogram.* Wiesbaden. 1914.

KRAUS UND NICOLAI: *Das Electrocardiogram des gesunden und kranken Menschen.* Leipzig. 1910.

KREHL: *Die Erkrankungen des Herzmuskels und die nervösen Herzkrankheiten.* Second edition. Wien. 1913.

LE CLERQ: *Maladies du cœur et de l'aorta.* Paris. 1914.

LEWIS: *Mechanism of the Heart Beat.* London. 1911.

Lectures on the Heart. New York. 1915.

MACKENZIE: *The Study of the Pulse.* London. 1902.

Diseases of the Heart. Third edition. London. 1914.

MEYER: *Die Digitalis Therapie.* Jena. 1912.

MEYER UND GOTTLIEB: *Die experimentelle Pharmakologie.* Wien. 1914.

NICOLAI: *Die Mechanik des Kreislaufs. Nagel's Handbuch der Physiologie des Menschen.* Braunschweig. 1909.

RITCHIE: *Auricular Flutter.* New York. 1914.

TAWARA: *Das reizleitende System des Säugethier-Herzens.* Jena. 1906.

TIGERSTEDT: *Human Physiology.* Third edition. Translated by Murlin. New York. 1906.

WENCKEBACH: *Die Arrhythmia als Ausdruck bestimmter Functionsstörungen des Herzens.* Leipzig. 1903.

WIGGERS: *Circulation in Health and Disease.* Philadelphia. 1915.

HEART BLOCK

BACHMANN: *Jour. Exp. Med.*, 1912, xvi, 25.
BARRINGER: *Arch. Int. Med.*, 1909, iv, 186.
COHN: *Heart*, 1912-13, iv, 7; 1914, v, 5.
CHRISTIAN: *Arch. Int. Med.*, 1915, xvi, 341.
EDES: *Trans. Assn. Amer. Phys.*, 1901, xvi, 521.
EPPINGER UND ROTHBERGER: *Ztschr. f. klin. Med.*, 1910, lxx, 1.
ERLANGER: *Johns Hopkins Hosp. Bull.*, 1905, xvi, 234.
Jour. Exp. Med., 1905, vii, 676; 1906, viii, 58.
Amer. Jour. Physiol., 1905-6, xv, 153.
Amer. Jour. Med. Sc., 1908, cxxxv, 797.
EYSTER AND MEEK: *Heart*, 1914, v, 119.
FAHR: *Virch. Arch. f. path. Anat.*, 1907, clxxxviii, 562.
GERHARDT: *Deut. Arch. f. klin. Med.*, 1908, xcii, 485.
HART: *Amer. Jour. Med. Sc.*, 1915, cxlix, 62.
HERRICK: *Amer. Jour. Med. Sc.*, 1910, cxxxix, 246.
HEWLETT: *Jour. Amer. Med. Assn.*, 1907, xlviii, 47.
JAMES: *Amer. Jour. Med. Sc.*, 1908, cxxxvi, 469.
KRUMBHAAR: *Arch. Int. Med.*, 1910, v, 583; 1914, xiii, 390.
Univ. Penn. Med. Bull., 1908.
LEA: *Lancet*, 1915, i, 1289.
LEWIS AND OPPENHEIMER: *Quart. Jour. Med.*, 1911, iv, 145.
NEUHOF: *Amer. Jour. Med. Sc.*, 1913, cxlv, 513.
Jour. Amer. Med. Assn., 1914, lxiii, 577.
A. OPPENHEIMER AND B. S. OPPENHEIMER: *Proc. N. Y. Path. Soc.*, 1913, xiii, 123.
OPPENHEIMER AND WILLIAMS: *Proc. Soc. Exp. Biol. and Med.*, 1913, x, 86.
PARDEE: *Arch. Int. Med.*, 1913, xi, 641.
PRICE AND MACKENZIE: *Heart*, 1911-12, iii, 233.
THAYER: *Arch. Int. Med.*, 1916, xvii, 13.
WILSON: *Jour. Amer. Med. Assn.*, 1915, lxv, 955.

EXTRASYSTOLES

DANIELOPOLU: *Arch. d. mal. du cœur*, 1914, vii, 174.
DRESBACH AND MUNFORD: *Heart*, 1913-14, v, 197.
ERLANGER: *Amer. Jour. Physiol.*, 1906, xvi, 160.
FERRALIS AND PEZZI: *Arch. d. mal. du cœur*, 1916, ix, 1.

FLEMMING: *Quart. Jour. Med.*, 1911-12, v, 318.
GALLAVARDIN AND GRAVIER: *Lyon Med.*, 1914, cxxii, 830.
LASLETT: *Heart*, 1909-10, i, 83.
LEVI: *Heart*, 1913-14, v, 299.
LEWIS: *Heart*, 1910, ii, 27.
Quart. Jour. Med., 1911-12, v, 1.
Heart, 1913-14, v, 335.
LEWIS AND SILBERBERG: *Quart. Jour. Med.*, 1912, v, 333.
LEWIS AND WHITE: *Heart*, 1913-14, v, 335.
MACKENZIE: *Quart. Jour. Med.*, 1907-8, i, 131; 481.
ROTHBERGER AND WINTERBERG: *Arch. f. d. ges. Physiol.*, 1912, cxlvi, 385; 1913, cliv, 571.
Zentralbl. f. Physiol., 1906, xxiv, 1.
VINNIS: *Heart*, 1912-13, iv, 123.
WILSON: *Arch. Int. Med.*, 1915, xvi, 989.

TACHYCARDIA

COHN: *Jour. Exp. Med.*, 1912, xv, 49.
COHN AND FRASER: *Heart*, 1913, v, 93.
GALLAVARDIN: *Arch. d. mal. du cœur*, 1916, ix, 45.
HART: *Heart*, 1912-13, iv, 128.
HUME: *Heart*, 1911-12, iii, 89.
LEA: *Proc. Royal Soc. Med.* (Lond.), 1913, vi, 14.
LEWIS AND SCHLEITER: *Heart*, 1911-12, iii, 173.
LEWIS AND SILBERBERG: *Quart. Jour. Med.*, 1911-12, v, 5.
LIAN: *Arch. d. mal. du cœur*, 1915, viii, 193.
PARKINSON AND MATHIAS: *Heart*, 1914-15, vi, 27.
RIHL: *Deut. med. Wchnschr.*, 1907, xxxiii, 632.
ROBINSON: *Arch. Int. Med.*, 1915, xvi, 967.
ROTHBERGER AND WINTERBERG: *Zentralbl. f. Physiol.*, 1907, xxv, 1.
Arch. f. d. ges. Physiol., 1911, cxlii, 461.

AURICULAR FLUTTER

FULTON: *Arch. Int. Med.*, 1913, xii, 475.
HAY: *Lancet*, 1913, ii, 986.
HERTZ AND GOODHART: *Quart. Jour. Med.*, 1908-9, ii, 213.
HEWLETT AND WILSON: *Arch. Int. Med.*, 1915, xv, 786.
HIRSCHFELDER: *Bull. Johns Hopkins Hosp.*, 1908, xix, 322.

HUME: *Quart. Jour. Med.*, 1913, vi, 235.
Heart, 1913-14, v, 25.
JOLLY AND RITCHIE: *Heart*, 1910-11, ii, 177.
LEVINE AND FROTHINGHAM: *Arch. Int. Med.*, 1915, xvi, 818.
LEWIS: *Heart*, 1912, iv, 171.
MCWILLIAMS: *Jour. Physiol.*, 1887, viii, 290.
MATHEWSON: *Edinb. Med. Jour.*, 1913, xi, 500.
MORISON: *Lancet*, 1909, i, 39; 77.
NEUHOF: *Med. Record*, 1915, lxxxviii, 995.
PARKINSON AND MATHIAS: *Heart*, 1915, vi, 27.
RIHL: *Ztschr. f. exp. Path. u. Therap.*, 1911, ix, 277.
RITCHIE: *Edinb. Med. Jour.*, 1912, ix, 485.
Proc. Royal Soc. Edinb., 1905, xxv, 1085.
ROBINSON: *Jour. Exp. Med.*, 1913, xviii, 704.
WHITE: *Arch. Int. Med.*, 1915, xvi, 517.

AURICULAR FIBRILLATION

AGASSIZ: *Heart*, 1912, iii, 353.
BUSQUET: *Presse med.*, 1914, xxii, 41.
COHN: *Heart*, 1913, iv, 221.
COHN AND LEWIS: *Heart*, 1913, iv, 15.
COWAN: *Glasgow Med. Jour.*, 1914, lxxxi, 128.
CUSHNY AND EDMUNDS: *Amer. Jour. Med. Sc.*, 1907, cxxxiii, 66.
DRAPER: *Heart*, 1911-12, iii, 13.
EHRENREICH: *N. Y. Med. Jour.*, 1914, xcix, 269.
EINTHOVEN AND KORTEWEG: *Heart*, 1915, vi, 107.
FALCONER AND DEAN: *Heart*, 1912, iv, 87.
FREDERICQ: *Scalpel*, 1914-15, lxvii, 49.
GARREY: *Amer. Jour. Physiol.*, 1914, xxxiii, 397.
HART: *Med. Record*, 1911, lxxx, 2.
HART AND JAMES: *Amer. Jour. Med. Sc.*, 1914, cxlvii, 63.
HERING: *Münch. med. Wchnschr.*, 1912, lix, 750; 818.
HEWLETT: *Heart*, 1910, ii, 107.
Arch. Int. Med., 1915, xv, 786.
KILGORE: *Arch. Int. Med.*, 1915, xvi, 939.
LEA: *Quart. Jour. Med.*, 1911-12, v, 388.
LEWIS: *Heart*, 1909-10, i, 306; 1912-13, iv, 273.
Jour. Exp. Med., 1912, xvi, 395.

LEWIS AND MACK: *Quart. Jour. Med.*, 1910, iii, 273.
LEWIS AND SCHLEITER: *Heart*, 1912, iii, 173.
MACKENZIE: *Brit. Med. Jour.*, 1911, ii, 869; 969.
Quart. Jour. Med., 1907-8, i, 38.
Amer. Jour. Med. Sc., 1907, cxxxiv, 12.
MORAT AND PETZETAKIS: *Compt. rend. Soc. de biol.*, 1914, lxxvii, 222; 237.
PARDEE: *Jour. Amer. Med. Assn.*, 1915, lxiv, 2057.
Med. Record, 1915, lxxxvii, 710.
RIHL: *Ztschr. f. exp. Path. u. Therap.*, 1910, viii, 446.
ROBINSON: *Jour. Exp. Med.*, 1913, xviii, 704.
Arch. Int. Med., 1914, xiii, 298.
ROTHBERGER AND WINTERBERG: *Arch. f. d. ges. Physiol.*, 1910, cxxxi, 387.
Wien. klin. Wchnschr., 1909, xxii, 839.
WENCKEBACH: *Arch. f. Anat. u. Physiol.*, 1907, 1-24.
WIGGERS: *Arch. Int. Med.*, 1915, xv, 77.
WINTERBERG: *Arch. f. d. ges. Physiol.*, 1909, cxxviii, 471.

VENTRICULAR FIBRILLATION

GUNN: *Heart*, 1913-14, v, 1.
HALSEY: *Heart*, 1915, vi, 67.
HOFFMANN: *Heart*, 1912, iii, 213.
LEVY: *Heart*, 1912-13, iv, 319; 1913-14, v, 299.
Jour. Physiol., 1914, xlix, 54.
LEVY AND LEWIS: *Heart*, 1912, iii, 99.
MCWILLIAM: *Brit. Med. Jour.*, 1889, i, 6.
MORAT AND PETZETAKIS: *Compt. rend. Soc. de Biol.*, 1914, lxxvii, 222; 237.
NOBEL AND ROTHBERGER: *Ztschr. f. d. ges. exp. Med.*, 1914, iii, 151.
ROBINSON: *Jour. Exp. Med.*, 1912, xvi, 291.

SINO-AURICULAR BLOCK

ERLANGER: *Amer. Jour. Med. Sc.*, 1908, cxxxv, 797.
EYSTER AND EVANS: *Arch. Int. Med.*, 1915, xvi, 832.
GIBSON: *Practitioner*, 1907, lxxviii, 589.
HEWLETT: *Jour. Amer. Med. Assn.*, 1907, xlviii, 47.
JOACHIM: *Deut. Arch. f. klin. Med.*, 1905, lxxxv, 373.
LASLETT: *Quart. Jour. Med.*, 1908-9, ii, 347.

LEVINE: *Arch. Int. Med.*, 1916, xvii, 153.
MACKENZIE: *Brit. Med. Jour.*, 1902, ii, 1911.
RIHL: *Deut. Arch. f. klin. Med.*, 1908, xciv, 286.
SCHOTT: *Münch. med. Wchnschr.*, 1912, lix, 292.
WENCKEBACH: *Arch. f. Anat. u. Physiol.*, 1906, 297-354.

HEART BLOCK AND AURICULAR FIBRILLATION

COHN: *Heart*, 1911-12, iii, 23.
COHN AND LEWIS: *Heart*, 1912-13, iv, 15.
DRAPER: *Heart*, 1911-12, iii, 13.
ERLANGER AND HIRSCHFELDER: *Amer. Jour. Physiol.*, 1905-6, xv, 153.
FALCONER AND DEAN: *Heart*, 1912-13, iv, 87.
HART: *Amer. Jour. Med. Sc.*, 1915, cxlix, 62.
HUME: *Heart*, 1914, v, 149.
LEA: *Quart. Jour. Med.*, 1912, v, 388.
LEWIS: *Heart*, 1909-10, i, 351; 1911-12, iv, 15.
LEWIS AND MACK: *Quart. Jour. Med.*, 1909-10, iii, 273.
MACKENZIE: *Heart*, 1909-10, i, 23.
PRICE AND MACKENZIE: *Heart*, 1912, iii, 233.
SOUQUES AND ROUTIER: *Arch. d. mal. du cœur*, 1913, vi, 305.
WHITE: *Bost. Med. and Surg. Jour.*, 1915, clxxiii, 431.

NERVE CONTROL OF THE HEART

ASCHNER: *Wien. klin. Wchnschr.*, 1908, xliv, 1529.
COHN: *Jour. Exp. Med.*, 1912, xvi, 732; 1913, xviii, 715.
COHN AND FRASER: *Heart*, 1914, v, 93.
DOGIEL: *Arch. f. d. ges. Physiol.*, 1911, cxlii, 109.
ERLANGER AND HIRSCHFELDER: *Amer. Jour. Physiol.*, 1905-6, xv, 153.
FABRE AND PETZETAKIS: *Compt. rend. Soc. de Biol.*, 1914, lxxvi, 343.
FERRALIS AND PEZZI: *Arch. d. mal. du cœur*, 1916, ix, 1.
GANTER AND ZAHN: *Arch. f. d. ges. Physiol.*, 1913, cliv, 492.
HART: *Amer. Jour. Med. Sc.*, 1915, cxlix, 66.
KEITH AND FLACK: *Jour. Anat. and Physiol.*, 1907, xli, 172.
LEVINE: *Arch. Int. Med.*, 1915, xv, 758.
LOEPER AND MOUGEOT: *Compt. rend. Soc. de Biol.*, 1914, lxxvi, 104.
MEEK AND EYSTER: *Heart*, 1914, v, 227.

MINES: *Jour. Physiol.*, 1914, xlvii, 419.
MUSKENS: *Amer. Jour. Physiol.*, 1898, i, 486.
OPPENHEIMER AND OPPENHEIMER: *Jour. Exp. Med.*, 1912, xvi, 613.
PETZETAKIS: *Bull. et. mem. soc. med. d. hôp. de Paris*, 1914, xxxvii, 739.
RITCHIE: *Quart. Jour. Med.*, 1912-13, vi, 62.
ROBINSON: *Arch. Int. Med.*, 1915, xvi, 967.
ROBINSON AND DRAPER: *Jour. Exp. Med.*, 1911, xiv, 217; 1912, xv, 14.
ROTHBERGER AND WINTERBERG: *Arch. f. d. ges. Physiol.*, 1910, cxxxv, 500; 559; 1911, cxli, 217; 343.
WILSON: *Arch. Int. Med.*, 1915, xvi, 1008.

ALTERNATION

ESMEIN: *Arch. d. mal. du cœur*, 1913, vi, 385.
GALLAVARDIN AND GRAVIER: *Arch. d. mal. du cœur*, 1914, vii, 497. *Lyon Med.*, 1911, Dec. 19.
GALLI: *Arch. d. mal. du cœur*, 1916, ix, 40.
HERING: *Ztschr. f. exp. Path. u. Therap.*, 1912, x, 14.
HERRICK: *Jour. Amer. Med. Assn.*, 1915, lxiv, 739.
JOACHIM: *Münch. med. Wchnschr.*, 1911, lviii, 1951.
KAHN: *Arch. f. d. ges. Physiol.*, 1911, cxl, 471.
LEWIS: *Quart. Jour. Med.*, 1910-11, iv, 141.
MUSKENS: *Jour. Physiol.*, 1907, xxxvi, 104.
PEZZI AND DOUZELAT: *Arch. d. mal. du cœur*, 1914, vii, 81.
RIHL: *Ztschr. f. exp. Path. u. Therap.*, 1906, iii, 275.
VAQUEZ: *XVII, Internat. Congr. Med.*, London, 1913, vi, 157.
WHITE: *Amer. Jour. Med. Sc.*, 1915, cl, 82.
WINDLE: *Quart. Jour. Med.*, 1910, iv, 435; 1912, vi, 453.

DIGITALIS

BAILEY: *Jour. Pharmacol. and Exp. Therap.*, 1909, i, 349.
CHRISTIAN: *Arch. Int. Med.*, 1915, xvi, 341.
COHN: *Jour. Amer. Med. Assn.*, 1915, lxv, 1527.
COHN, FRASER AND JAMIESON: *Jour. Exp. Med.*, 1915, xxi, 593.
CUSHNY: *Jour. Exp. Med.*, 1897, ii, 233.
CUSHNY, MARRIS AND SILBERBERG: *Heart*, 1912, iv, 1.
DANIELOPOLU: *Compt. rend. soc. de biol.*, 1913, lxxiv, 969.
EGGLESTON: *Arch. Int. Med.*, 1915, xvi, 1.

GOTTLIEB: *Med. Klinik*, 1913, ix, 2061.
HART: *L. I. Med. Jour.*, 1913, vii, 217.
HATCHER AND BAILEY: *Jour. Amer. Med. Assn.*, 1907, xlviii, 1177.
HATCHER AND EGGLESTON: *Jour. Pharmacol. and Exp. Therap.*, 1912, iv, 97.
HEWLETT AND BARRINGER: *Arch. Int. Med.*, 1910, v, 93.
JAMES AND HART: *Amer. Jour. Med. Sc.*, 1914, cxlvii, 63.
JANEWAY: *Amer. Jour. Med. Sc.*, 1908, cxxxv, 781.
LEHNERT AND LOEB: *Therap. Monatsh.*, 1914, xxviii, 164.
MACKENZIE: *Heart*, 1911, ii, 273.
MARTINET: *Presse med.*, 1914, xxiii, 361; 433.
REINIKE: *Ztschr. f. klin. Med.*, 1913-14, lxxix, 441.
ROTH: *Bull. Hyg. Lab., U. S. P. H. and M. H. S.*, No. 102.
ROTHBERGER AND WINTERBERG: *Arch. f. d. ges. Physiol.*, 1913, cl, 217.
SCHLEITER: *Amer. Jour. Med. Sc.*, 1914, cxlviii, 343.
VOEGTLIN AND MACHT: *Jour. Pharmacol. and Exp. Therap.*, 1913-14, v, 76.