5-1-1942

Cardiac arrhythmias

Frank John Peter
University of Nebraska Medical Center

THE CARDIAC ARRHYTHMIAS

Frank J. Peter

SENIOR THESIS

PRESENTED TO THE COLLEGE OF MEDICINE,
UNIVERSITY OF NEBRASKA,
OMAHA
1942

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INTRODUCTION

It is my opinion that in a paper of this type, in which no original work is done, little benefit is derived by anyone but the writer. In other words, the writer is benefited by the amount of knowledge he obtains while he is compiling his data. With this thought in mind I decided to choose a topic which would provide a maximum amount of instruction and which would at the same time prove interesting. The subject"Cardiac Arrhythmias" was finally chosen.because it seemed a very practical one, but primarily because it promised to be instructive along lines of which my knowledge had been none too complete.

Literature on this topic is very profuse--so profuse in fact that an all-inclusive study could not even be attempted. Furthermore, there are many related topics that must be considered; in the "History",for instance, one certainly could not omit mention of invention of the stethescope and of the electrocardiograph, nor could one omit mention of discovery of the important cardiac drugs, notable digitalis and quinidine. In discussing "Diagnosis", an explanation of the electrocardiograph, with interpretation of the various readings concerned in the "Arrhythmias", is an essential. Thus, even

a scanty knowledge of the subject involves several closely allied fields.

"Cardiac Arrythmias" seems an improper title, for many of the conditions herein discussed are not arrythmias in the literal sense of the word. However, the bradycardias, tachycardias and other conditions which are not arrhythmic, are universally included under this heading. Particularly are they included by the National Heart Association, whose classification is used in this paper.

An attempt has been made to present only the most pertinent facts. In the "History" only the pioneers in each phase of the subject are mentioned. In the body of the paper only the most logical or most widely accepted explanation of each "Arrhythmia" is offered. The result is a rather bulky paper which at the same time is only a very brief sketch of the Arrhythmias.

Imhotep first observed the pulse in 2980 B.C.
Hippocrates was next with observations on dropsy
Erasistratos dealt in anatomy of the human heart
Celsus' De Re Medicina, with venesection, was not far apart
Dioscorides, in 60 A.D. advocated diuretic squill
Galens study of the pulse temporarity filled the bill
da Vinci drew the heart long ere Mussolini
Then came fibrinous pericarditis a la Benivieni
da Carpis' mention of dilated heart was no collosus
Because Botallo soon described the ductus arteriosus
Conano discovered valves in veins in the spring of 1540
Aortic aneurism by Vesalius was diagnosed anti morti
While Servetus mentioned pulmonary circulation to his flocks
Fernel and Pare ascribed aortic aneurism to the great pox
Caesalpino his cohorts with "circulation" beguiled
And Schenck case records based on symptons compiled
d'Acquapendente created the anatomical theater in 1603
Albertini theorized on palpitation and syncope
Aselli in 1622 discovered the lacteal vessels
But then came William Harvey.

HISTORY

Nearly all historians who have concerned themselves with cardiology, have begun their dissertations with an account of William Harvey. Briefly, Harvey (43) proved the circulation of the blood and the manner in which it was accomplished, and he predicted the existence of the capillary circulation. Although he contributed nothing directly pertinent to this paper, his very scientific thesis on the anatomy and physiology of the heart, published in 1628, laid the foundation for the subsequent discoveries, which will be discussed herein.

Niels Stenson (33) in 1664, wrote the first treatise in which was included a theorization regarding the origin and integrity of cardiac action. Writing "On the Muscular Nature of the Heart", he related, "I saw in certain splendid preparations linear structures running on the surface of the heart, which however were not lymphatics, but dissection showed them to be truly nerves. A nervous plexus was demonstrated by Fallopius at autopsy, who believed that they were dispersed not only over the surface but that they penetrated into the interior."

In concluding the same article Stenson said, "If it be true, and the truth of an observation is dependent upon its correctness, that in the heart nothing is lacking which would make it a muscle, and not denying that muscle has been demonstrated in the heart, the heart thus

is not a substance sui generis, and so cannot generate certain substances as heat, innate warmth, the seat of the soul; nor can it produce certain humors as blood, certain spirits or vitality. But however this may be, I am examining solely the substance; thus, from the fibers proceeds all movements of the heart, occuring as a phenomenon of its own----, which subject, however, I wish to largely omit at present and defer a consideration of it until a later time."

Stephen Hales (9) in 1733 made the first outstanding contribution on the physiology of the cardiovascular system following the exposition of Harvey. His experiments proved that cardiac rate was accelerated concomitant with withdrawal of blood from the vascular system. Hales in "An Account of Some Hydraulic and Hydrostatical Experiments Made on the Blood and Blood-Vessels of Animals", in which he determined blood pressure in animals, particularly horses, by affixing brass pipe and glass tubes to the crural arteries and allowing the blood to flow vertically under its own pressure, with intermittent withdrawals of one quart of blood, made the observation that, "The horse's pulse beat 40 strokes in a minute, before he was disturbed or tied down; but when the glass tube was fixed to the artery, it beat 65 in a minute, and as the horse grew fainter, the pulse was more and more accelerated, so as

to beat an 100 times or more in a minute; whence we see that the pulse is weak and quick, when the heart is supplied with little blood; which is the case in the hectic fevers, etc."

De Sénac (43) in 1749, was the first physician to use quinine in palpitation. His early and extraordinary observation is probably without doubt the very beginning of the relatively modern quinidine therapy of cardiac arrhythmia.

Senac believed that remedies for palpitation were the "stomachics", the "cordials" and the "sedatives". The stomachic remedies have appeared to various physicians as a resource against palpitations, for it is often in the stomach that their cause resides; if they do not arise from this as an immediate cause, there is in many cases an occasional cause which sets the other in motion." The majority, he says, of those who are subject to palpitations are hypochondriacs, the functions of their stomachs are deranged, and this derangement troubles the action of the heart.

Senac, then, believed that palpitation was excited by the viscera, and the viscera in turn excited by faulty digestion; therefore, remedies which would facilitate digestion, could be regarded as remedies for palpitation. In this regard, he quoted, "Of all the stomachic remedies

the one whose effects have appeared to me the most constant and the most prompt in many cases, is quinine mixed with a little rhubarb. Long and rebellious palpitations have ceded to this febrifuge, seconded with a light purgative."

Sénac (30) entertained a curious idea about the remote cause of movements of the heart. He thought these motions were transmitted by an animal spirit situated in the brain and spinal marrow. He thought of this spirit as an extremely elastic fluid, which was put into action by the impression of the blood on the delicate tissue of the parietes of the heart and the columns of the ventricles.

John Babtist Morgagni (43) in a letter to a friend in 1761 described heart block in an "epileptic" patient, stating that, "You will suspect, whether the rarity of the pulse be, in fact, a very uncommon sympton, to remain after an epilepsy, in hypochondriac patients, when you shall have compared this observation of mine with that of the celebrated Gerbezius which describes the pulse of a strong hypochondriac man, 'who was now and then subject to slight epileptic paroxysms, as being so very slow, that before the subsequent pulsation followed that which went before, three pulsations would certainly have passed in another healthy person'." Morgagni, in this first description of heart block, gave no explanation for the decrease in pulse rate, however.

William Withering, (35) who through his extensive experimentation with, and use of the foxglove, incited a widespread us of the drug, deserves more than passing mention here. In 1775 his attention was drawn to digitalis by the discovery that it was important in the cure of dropsy. This remedy was a decoction of herbs which an old woman in his native town, Shropshire, had compounded. After analyzing the remedy, Withering found the important ingredient to be foxglove. At first he made the leaves of the plant into a decoction, then into an infusion, and still later he used the powdered form.

On the basis of Witherings description of the patients he treated, it has been assumed that some of them suffered from auricular fibrillation. Withering, however, recommended the use of digitalis in dropsy and anasara only, and was careful to state that it was valueless in the treatment of ovarian cysts and similar condition. He did not understand how the drug acted in dropsy nor did he differentiate its action on cardiac dropsy from its action on other forms of dropsy. Because he had frequently noticed retardation of pulse in patients treated with the drug, he knew that it exerted some action on the heart. He wrote, "That it has a power over the motion of the heart, to a degree yet unobserved in any other medicine, and that this power may be converted to

salutary ends."

In 1816, Laënnec, (41) through his invention of the stethescope, initiated the art of auscultation. Willius and Keys, (43) discussing William Einthoven in regard to his contributions to electrocardiography, make the statement, "William Einthoven was awarded the Nobel Prize in medicine for his contributions which so greatly advanced the study of modern cardiology and which are as important, perhaps, to the cardiologist as the use of digitalis is to the cardiac patient." If Einthoven deserves this tribute, and there is little doubt that he does, Laennec certainly deserves one equally as great, for Laënnec made available to all practitioners a simple, inexpensive, but very valuable piece of diagnostic equipment--and that almost a century before invention of the electrocardiograph.

With the stethescope Laënnec first "heard the language of pathology". He wrote extensively on cardiology and, as far as can be determined, was the first to expound logical correlation between cardiac action and rhythm. He stated that, "In every case the heart gives two distinct pulsations for one beat of the arterial pulse." He believed that the first sound was due to contraction of the ventricles, that the second sound, which followed immediately afterward, was due to contraction of the auricles, and that there then followed, "a very short,

yet well-marked interval of repose." Laennec, dividing a cardiac cycle, in regard to time, concluded that, "a fourth (or third) belongs to the systole of the auricles, a fourth (or somewhat less) to the state of quiescence, and two-fourths to the systole of the ventricles." His observations, expecially in regard to causation of the second sound, although not accurate, are non the less remarkable.

Laennec recognized the phenomenon of "dropped beat" but did not theororize as to its cause; also, he recognized regular and irregular arrhythmias, stating simply that "these irregularites occur most frequently in cases of dilation".

In 1825 Hope (43) experimented on donkeys in order to satisfy himself in regard to the cause of the heart sounds. He succeeded in examining the hearts of stunned asses, in which respiration had been artificially maintained after the pericardium was opened, and proved that the second heart sound was dependent on the abrupt closure of the aortic and pulmonic valves.

Parry (41) in 1825 published his "Collected Works", which included a description of "rapidity of pulse" and palpitation coincident with exophthalmic goiter. This recognition of tochycardia as being the probably result of hyper-thyroidism, in some cases, was accompanied by no

explantion, however.

Adams (30) in 1827, discussed a patient who had died as a result of "apoplexy". He had noticed a remarkable slowness of the pulse, which generally ranged at the rate of thirty per minute. When, at autopsy, he found that the heart had undergone some fatty myocardial degeneration, he decided that the "apoplexy" had been due to a condition existent in the heart, reasoning that the slowness of the heart had caused a relative venous stasis in the brain which was the direct cause of the "apoplexy". Adams, thus, was probably the first to describe heart block in relation to the syndrome which later was to bear the name of Adams and Stokes.

Nineteen years later, Stokes (39) published complete data on several of his patients who had evidenced slow pulse and had suffered from intermittent attacks of syncope, which he described as "pseudo-apoplexy". In Stokes' series of seven cases with permanently slow pulse he found at autopsy that: organic disease of the aorta on the valves, or both, existed in five; fatty degeneration, or "weakened myocardium" existed in three. Stokes concluded his paper with the statement that, "The preceeding observations are published with view of drawing the attention of the Profession to a combination of cerebral and cardiac phenomena, of which our knowledge is still imperfect."

In 1871, Traube (43) read his classic paper on "A Case of Pulsus Bigeminus" before the Berlin Medical Society. This report contained what is probably the first clear picture of pulsus alternans. Because of his extensive experiments on animals, Traube had long been acquainted with the condition designated by him as "pulsus bigeminus". He had been able to produce the phenomenon in animals by two different procedures; first, by greatly prolonged suspension of artificial respiration in animals with sectioned vagi, wherein he noted that, "Where the heart has been deprived of the influence of the inhibitory spinal nervous system, we see the pulsus bigeminus appear under circumstances similar to those in which the vagi are intact, that is, after the arterial tension is appreciably reduced and shortly before considerable reduction in the frequency of the pulse takes place, the low pulsus tardi appears, which portends the failure of the left ventricle."

Second, Traube discovered that the phenomenon would appear every time, shortly after poisoning in an animal with the vagi sectioned, when a substance was administered which stimulated the heart through the entire "inhibitory spinal nervous system". From these experiments he concluded that two conditions were necessary for the appearance of pulsus bigeminus; first, the heart must be released from the influence of the "inhibitory spinal

nervous system"; second,there must be some agent circulating in the blood, which increases the irritability of the cardiac component of the "inhibitory spinal nervous system", which is still functional.

The case presented by Traube to the Berlin Medical Society concerned a patient whom he had treated for cardiac decompensation. Traube had noticed that when the patient had become digitalized a condition, which he designated as "pulsus alternans, a variation of pulsus bigeminus", ensured. When digitalis therapy was discontinued, the phenomenon of "pulsus alternans" soon disappeared. Traube concluded that, "since digitalis belongs to those agents which stimulate the inhibitory nervous system of the heart", the pulsus alternans had been caused by this drug. This finding correlated with his experimental observations as described above.

Waller (25) in 1887 published a paper concerning his experiments on the leading off of the action currents of the heart by means of contact electrodes. The import of these early electrophysiological experiments on the heart is self evident. Waller was the first to demonstrate that the currents set up by the beating of the heart in animals could be recorded without opening the thorax. He was the first, also, to obtain an electrocardiogram of the action of the human heart.

Waller discovered that if a pair of electrodes were strapped to the front and back of the chest and connected with a Lippmann's capillary electrometer (a device incorporating a capillary column of mercury in which the mercury was caused to move by changes in potential between mercury and sulfuric acid electrodes) the mercury would be seen to move slightly but sharply at each beat of the heart. The movements of the column of mercury were photographed on a traveling plate simultaneously with those of an ordinary cardiographic lever. The result is shown in a reproduction of one of Wallers original recordings, figure 1.

Wallers chief problem then remained to determine whether the electrical variation was physiological and not due to a mechanical alteration of contact between the electrodes and the chest wall caused by the heart's impulse. He reasoned that physiological variation should precede the movement of the heart, while this could not be the case if the variation were due to altered contact. He therefore made more accurate time measurements, running the kymograph as rapidly as possible without rendering the initial points of the curves too indeterminate. The result of this experiment is shown in figure 2, which proves that the electrical phenomenon began slightly before the cardiographic lever began to rise. Waller con-

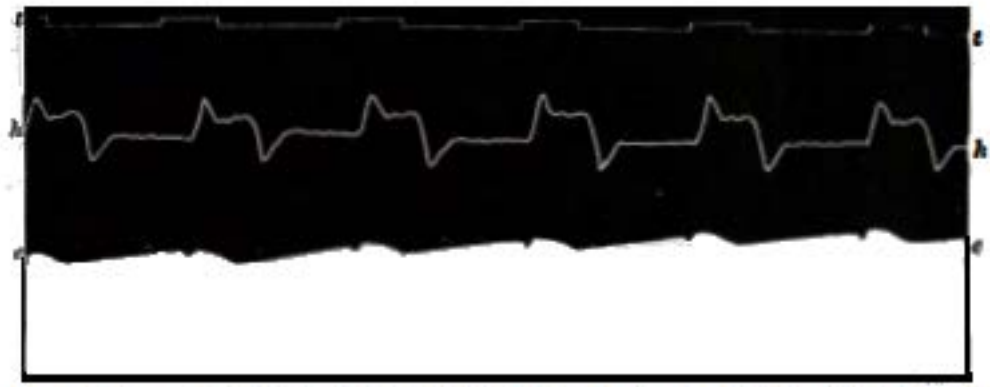

FIG. 1. Man. Heart led off to electrometer from front and back of chest (front to Hg; back to H_2SO_4).
e.e. electrometer. *h.h.* cardiograph. *t.t.* time in seconds.

Figure 1. From Cardiac Classics (43).

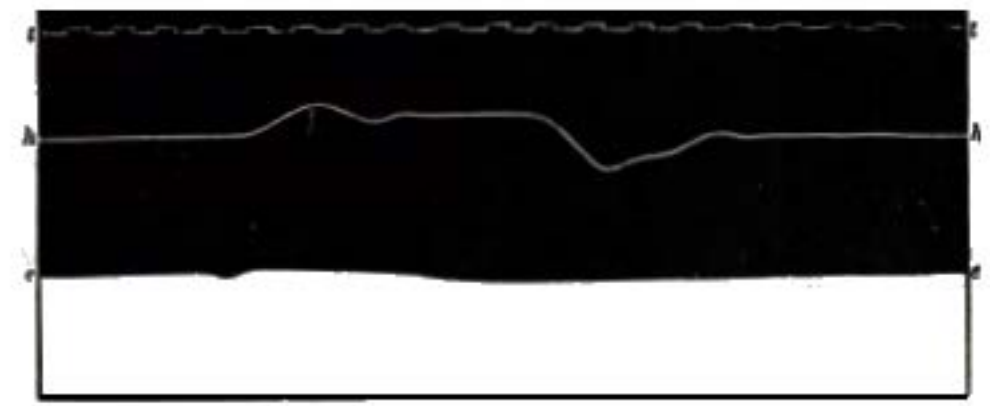

FIG. 2. Man. Heart led off to electrometer from front and back of chest (front to Hg; back to H_2SO_4).
e.e. electrometer. *h.h.* cardiograph. *t.t.* time in $\frac{1}{25}$th sec.

Figure 2. From Cardiac Classics (43).

cluded, after deducting .01 second for "lost time" of the cardiograph, that the electrical impulse preceded the mechanical impulse by no more than .015 second. He further concluded that in the human heart the duration of systole was 0.35 second.

Waller later proved beyond a doubt that the electrical variation of the human heart was demonstrable by taking leads off of other body surfaces. He used the two hands, one hand and one foot, and the mouth with any extremity, obtaining similar results in each instance.

MacWilliam (32) in 1887 published his paper on "Fibrillar Contraction of the Heart", in which he discussed the results of certain experiments on animals. Ludwig and Hoffa (41) in 1850 had shown that the application of faradic currents to the ventricles of the dog's heart caused an abolition of the normal beat, during which there was a considerable fall in arterial blood pressure. MacWilliam duplicated, then enlarged on the experiments of Ludwig and Hoffa. MacWilliam found that faradic stimulation of the ventricles caused a "rapid succession of incoordinated peristaltic contractions", which, he theorized, were due to a lack of harmony in the contraction and relaxation of the minute muscular fibers composing the ventricular walls. After stimulating isolated ventricles and finally isolated bits of ventricular tissue from the

very apex of the heart, MacWilliam concluded that: (1) Neither the nervous nor mechanical connection between auricles and ventricles is necessary for the effective contraction of the latter. (2) The state of arrhythmic fibrillar contraction is essentially due to certain changes occuring within the ventricles themselves. (3) The ventricles contain within themselves the entire mechanism necessary for the execution of regular coordinated beats. (4) As the complexity of the cardiac musculature increases, so increases the complexity of movement upon stimulation. (5) The duration of the movement varies with the excitability of the ventricular muscle.

MacWilliam also produced fibrillar contractions with certain drugs. He showed that the auricles, when stimulated by faradic current, would go into a rapid flutter, the movements of which, unlike those in the case of the ventricles, would be coordinated. Finally,MacWilliam showed the vagal stimulation had no effect on ventricular fibrillation, but that it inhibited on stopped the movements in "auricular flutter". From this he concluded that the vagus nerve had no connection with ventricular muscle.

Bristowe (41) in 1888, described paroxysmal tachycardia as a clinical entity for the first time, antedating by a year the report by Bouveret in which the term

"Paroxysmal Tachycardia" was first used. Bristowe's opening statement was very descriptive, to wit, "The subject to which I wish to direct attention is that of extremely rapid pulsation, occuring for the most part in intermittent paroxysms of variable duration, in hearts structurally and texturally sound, and in persons otherwise healthy."

Bristowe saw his first case of paroxysmal tachycardia in consultation with another physician. He was greatly impressed by the apparent spontaneity of the attach, in which the cardiac beats were counted at the rate of 246, but even more impressed by the absolute lack of distress in the patient. In the latter regard he stated, "Had I not known that the patient's heart was beating with extraordinary rapidity it would never have struck me, from watching her and conversing with her, that there was anything the matter with her."

From his observations, Bristowe concluded that paroxysmal tachycardia, as far as the heart is concerned, is a purely functional disorder, and that any actual cardiac disease found to be present must be considered as merely coincidental.

In 1893, His (17) presented his now famous paper on the function of the atrioventricular bundle, together with a description of events leading to discovery of that entity.

It was the current teaching of the time that the ganglia were the autonomic centers of the heart. Several men, notably Engelmann and Gaskell, believed that the heart muscle itself is able to originate rhythmic stimuli. None had been able to prove their point, however, and because of the interesting controversy, His decided to study the embryological development of the heart to ascertain whether or not it is able to beat before it has nerves and ganglia. He followed the development of the cardio-nervous system through several vertebrates and in each instance proved that the heart beat before it received nerves or ganglia. However, one point remained mysterious to His, namely, the conduction of the stimulus from one part of the heart to the other.

Gaskell (43) had shown that in the frog and turtle the conduction is made by way of the muscles. His reasoned therefrom, that the conduction mechanism is similar in the human and attempted to prove such a muscular connection by examining serial sections in the various embryonic stages. His' description of his results is classical --"After extensive investigation I was able to find a muscle bundle which connects the auricular and ventricular septal walls, and which apparently had not been observed before, because it is only visible in its entire distribution when the septal walls are cut exactly in the hori-

zontal direction. The bundle arises from the posterior wall of the ventricle near the auricular septum in the alrioventricular groove; it joins the upper edge of the ventricular septum and ramifies, coursing on the septum anteriorly until it branches near the aorta into a right and left branch, the latter terminating in the base of the aortic cusp of the mitral valve."

His could not state with certainty whether the bundle actually conducted the impulses from the auricle to the ventricle, as he did not perform any experiments dealing with severance of the bundle. He concluded, however, that, "Its presence, in all events, is contrary to the opinion of those, who, in the absence of such a muscular connection between the auricle and ventricle, attempt to prove the necessary presence of a nerve conduction."

Einthoven (25), in 1903, employed the galvanometer, the invention of Johannes Schweigger, to measure the electric current produced by the action of the heart. Waller's capillary electrometer (previously described) had been attended by much difficulty and many errors. In Waller's instrument, the mercury, because of its inertia produced curves which were not exact; thus, the recordings were not accurate measurements of the electrical changes accompanying the heart beats.

Einthoven's instrument is essentially composed of a

thin silver-coated quartz filament, which is stretched like a string in a strong magnetic field. When an electric current is conducted through this filament, the filament reveals a movement which can be observed and photographed by means of considerable magnification. Furthermore, it is possible to regulate the sensitivity of the galvanometer very accurately by tightening or loosening the string.

Besides eliminating the problem of inertia as occured in the capillary electrometer, the galvanometer presented many other advantages, namely; (1) Less bulk in the moving medium--a quartz filament could be made only one-tenth as thick as the mercury column. (2) Ease of regulation, by means of changing tension on the filament. (3) Elimination of false results which occured in the capillary device due to changes in potential in the mercury and sulphuric acid baths. (4) Elimination of friction, which existed in the older instrument in the form of dust particles in the tube. (5) Because of its physical principle, the galvanometer is more easily isolated, electrically. By perfecting Schweigger's invention, Einthoven introduced a practical method for electrocardiography which has remained fundamentally unchanged.

Auricular fibriallation was first described by Hering (30) in 1903 and designated by him as "pulsus irregularia

perpetuus". Hering recognized the brevity of the cardiac cycle, even in relation to the increased heart rate. He theorized that a cardiac arrhythmia can consist of two different types, resulting either from (A),an abnormal beharior of the stimulus, or (B) an abnormal behavior of the irritability of the heart or certain of its parts or (C), when both conditions are present. In regard to the condition at hand he stated, "The Pulsus irregularis analysed here, which is observed in valvular heart disease, coronary sclerosis, and myocardial diseases, is lasting and for that reason, I called it in the title Pulsus irregularis perpetuus; it is furthermore the same whether the patients heart beats faster or slower, as for example, after giving digitalis: it does not arise under the influence of respiration, it is not then a Pulsus irregularis respiratius". Because there was no "respiratory annhythmia", Hering reasoned that the abnormaility did not result from the indirect influence of the "extra-cardial cardiac nerves", since he believed, "there is no Pulsus irregularis produced by the extra-cardial nerves save that seen in respiratory disturbances".

With the above evidence at hand, he reasoned, further, that the condition takes its origin from a cause within the heart, probably of myogenic origin, whether produced by extra stimuli or by premature or delayed normal stimuli.

Herings description of auricular fibrillation, although neither concise nor complete, was the first attempt at a scientific description of the condition. In a few years, several men, notably Rothberger and Winterberg, Mackenzie, and Lewis were to make much more detailed investigations--these will be discussed later.

Ritchie (10) in 1906 gave the first clear account of extra-systole, which he termed the "commonest form of cardiac irregularity. He stated that an extra-systole, "is a ventricular contraction which, instead of occuring in response to a stimulus arriving from the auricle as normally, occurs independently in the form of a premature heart-beat separated by a somewhat prolonged pause from the succeeding beat, which is exaggerated".

Tawara (13) in 1906, after detailed micro-anatomy, showed that the Purkinje fibers represented the end branches of the auriculo-ventricular bundle. He also described the A-V node, to which his name is sometimes attached, as a localized swelling on the A-V bundle consisting of a close interlacement of fibers. It was Tawara who first made clear the origin, course, and endings of the whole auriculo-ventricular conduction system.

Keith and Flack (13) verified the truth of Tawara's discovery in 1907 and set out to trace the evolution of the auriculo-ventricular connecting system. They also

desired to ascertain if, in the region wherein the beat of the heart was believed to begin; namely, at the termination of the superior vena cava in the right auricle, there existed tissue of the nature of the node which Tawara discovered at the beginning of the A-V bundle. They found in all mammalian hearts, at the expected site, a collection of peculiar muscular tissue. They named this tissue the sinoauricular node, inferring from its position, and from its resemblance to the tissue at the beginning of the ventricular bundle, that this node was concerned in the inception of the heart beat.

There is considerable controversy among historians as to who presented the first clear description and logical explanation of auricular fibrillation. There is no doubt that Hering, as previously mentioned, was the first to discover the condition clinically. However, his observations and description left much to be desired. According to White (41) its clinical existence was suspected by Cushny in 1906, and was proved in 1909 by Rothberger and Winterberg, and by Lewis independently. Other historians, notable Major (30), and Willius and Keys (43), believe that the condition was first accurately described by Mackenzie in 1908.

The evidence at hand shows that Mackenzie (30) did the most complete original work on the condition, although

he was aided considerabley in his final conclusions by the electrocardiographic work of his pupil, Lewis (26). MacKenzie first observed the condition in a patient whom he had observed at intervals until her death in 1898. He made simultaneous recordings of both arterial and venour pulse on the patient, intermittently, from 1892 to 1898, As shown in figure two (figures 119 and 120), the venous pulse (liver) was of the auricular type until 1898. At that time the patient became very ill and her heart became rapid and irregular. As soon as she exhibited partial recovery Mackenzie again made polygraphic recordings with the result shown in the third tracing (figure 121) of figure two. He concluded, from this, that all evidences of auricular activity had disappeared, and, when at post-mortem examination he found the auricle distended and thin walled, he concluded, further, that the auricle had become distended, atrophied, and paralyzed.

Mackenzie then followed a series of similar cases over a number of years, but in each of these it was discovered at post-mortem that the auricles were hypertrophied. Mackenzie then reasoned that since the auricles were hypertrophied they must have been contracting during his period of observation,and that his earlier assumption, in regard to atrophy and paralysis, was wrong. Since the auricles had exhibited no pulsution of their own, he in-

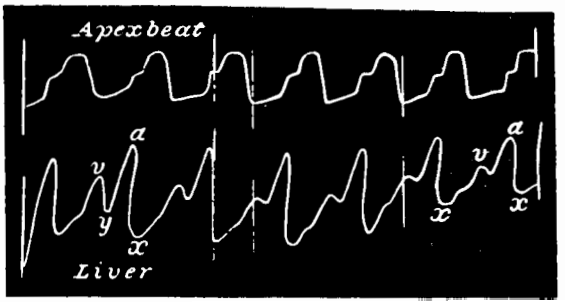

FIG. 119. The liver pulse shows a well-marked wave (*a*) due to the auricle (Case 48. 1892).

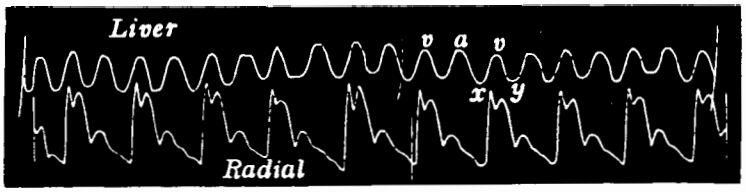

FIG. 120. There is still a well-marked wave in the auricle (Case 48. 1897).

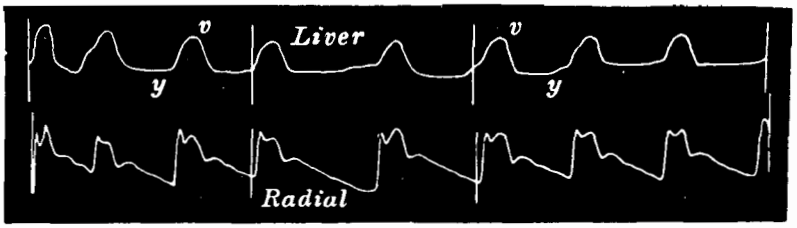

FIG. 121. Showing the irregular rhythm, characteristic of auricular fibrillation. W

Figure 3. From Cardiac Classics (43).

ferred that they had been contracting simultaneously with the ventricles. Because he could not conceive of any other possibility to explain the facts, he suggested that the stimulus for simultaneous contraction arose in the auriculo-ventricular node. He, therefore, called the condition "nodal rhythm" in his explanation which appeared in his book (43) in 1908. Mackenzie had realized, even before publication of his results, that his explanation of "nodal rhythm" was far from being established.

Cushny (30) in 1906 was the first to suggest that auricular fibrillation might be the factor of prime importance. He derived this conclusion from the fact that there was great resemblance of the radial tracings in a case of paroxysmal irregularity in the human subject to the tracings from a dog, in which experimental auricular fibrillation had been produced. When Cushny's observation was called to the attention of Mackenzie, Mackenzie agreed that auricular fibrillation might have something to do with the etiology of his "nodal rhythm", but failed to realize the real significance of the condition, believing that it was just a transient affair and could not be at the "bottom of the cases" that went on for years.

In 1909, Lewis (26) who had been studying the condition extensively by means of electrocardiography, finally published his results. Lewis had been able to detect in

the electrocardiogram of experimentally produced fibrillation, certain oscillations during ventricular diastole, which were induced by the fibrillating auricle. Lewis (43) also examined the electrocardiograms of typical cases of Mackenzies' "nodal rhythm", and found the auricular oscillations also present. Thus, auricular fibrillation was established as the real etiology in "pulsus irregularis perpetuus", or "nodal rhythm".

Lewis (41), in explaining the mechanism of auricular fibrillation, states, "The walls of the auricle stand in the distolic position; systole, either complete or partial, is never accomplished; the wall, as a whole is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement; rapid, minute, and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface."

Mackenzie (30) had recognized the occurence of "extra-systole" as early as 1902. However, it was not until 1913, after he had made a long series of observations that he published his opinion on the condition. He believed that it was due to some part of the heart's structure being temporarily more excitable than the normal starting place. Before the time of Mackenzie, the presence of extra-systole had been regarded as serious and, as a result, patients

had been subjected to unnecessary treatment and fear. From his series of cases, Mackenzie concluded that: (1) "From such facts as these, that healthy men and women may present this form of irregularity, it can be gathered that extra-systoles in themselves are signs of no significance so far as the efficiency of the heart if concerned." (2) "It may therefore be stated that when the extra-systole is the only abnormal sign, the prognosis is a favorable one, and where it is associated with other signs the prognosis is to be based upon these other signs."

Jolly and Ritchie (20) in 1911 were the first to distinguish "auricular flutter" from auricular fibrillation. In the three cases which they had observed prior to their publication, they found auricular rates of from 168 to 350 per minute while the ventricular rate was either normal or only slightly increased. In each case the auricles and ventricles beat rhythmically and there was no evidence of the arrhythmia found in auricular fibrillation. Jolly and Ritchie, after extensive electrocardiographic studies concluded that the relative slowness in the ventricular contractions was due to depression of the A-V node or to a lesion involving that area. They made no allusions in regard to origin of the auricular tachycardia, however.

This procession of events may be concluded with a brief description of the discovery of quinidine by Frey (12).

As previously mentioned, Sénac had discovered over one hundred years previously, that quinine acted beneficially in cases of obstinate palpitation. The discovery that quinine has a specific effect on auricular fibrillation was discovered in 1914 by Wenckebach (12) who made the observation incidental to treating cases of malaria in which auricular fibrillation was also present. Frey developed quinidine, an isomer of isomer of quinine in 1918. He subsequently showed that it has a more effective action in abolishing auricular fibrillation than has quinine itself.

Quinidine, which is currently used in auricular flutter, auricular fibrillation, extra-systoles and other forms of heterotopic rhythm, is more effective than digitalis, which does not arrest the fibrillation but protects the ventricle from the auricular impulses. In caseswhich react favorably to quinidine, the oscillations of the auricle are seen to become progressively slower and coarser, while the rhythm of the ventricle generally quickens; then the fibrillation is suddenly replaced by normal movements of a rapid rhythm and this passes into regular beating of auricle and ventricle at 70-80 beats per minute (14).

The Cardiac Conduction System; Its Relation to Cardiac Rhythm.

The beating heart owes its characteristics to five basic physiological properties, namely; (1) rhythmicity, (2) conductivity, (3) irritability, (4) contractility, and (5) tonus (23).

According to Katz (21) it can be shown that the heart has the peculiar property of maintaining its beat as soon as the embryo becomes viable, and that this beating continues until death terminates the existence of the individual. This automatic rhythmic property of the heart is not dependent upon its connections with the rest of the body since not only the heart of the cold-blooded animal, but that of the mammal and even that of man, has been made to beat after its removal from the body.

It has been demonstrated that this property of rhythmicity in the mammalian heart resides in the muscle of the heart and not in the nervous structures associated with it (21). The theory of neurogenic origin of the mammalian heart beat has been thoroughly disproved, and the myogenic origin is almost universally accepted. It has been shown clearly that the property of rhythmicity in the heart is peculiar to nodal tissue, and that under normal circumstances, the ordinary cardiac musculature

and probably even the Purkinje fibers do not share this property. Any seeming rhythmicity in cardiac tissue other than nodal has been ascribed by thoughtful students to the presence of small islets of nodal tissue in the beating strips, or to abnormal circumstances which give rise to the development of rhythmicity in fibers which normally do not display this property. (40)

Since under ordinary circumstances all parts of the heart, including nodal tissue, are discharged when the activating process reaches them, it follows that of several rhythmic foci, or active pacemakers, the fastest will soon dominate all the rest. (29) The fastest rhythmic focus in the nodal tissue of the heart is normally located in the sinus node, usually near its head. This region, therefore, will become the normal pacemaker of the heart, and will be responsibe for initiating the events of the heart beat (21). If for any reason this primary pacemaker is suppressed, either temporarily or permanently, or if its discharge is prevented from spreading through the heart, secondary or tertiary pacemakers will take up its function, but at a slower rate. The fastest of these remaining pacemakers will be the one to gain control.

There are various theories regarding the control of the primary pacemaker. Most authorities are now of the

opinion that impulses are received at the sino-auricular node, although they do not actually originate there (4). Katz (21) states, "The primary pacemaker is under the control of the nervous system, the rate of its activity being normally determined by the tonic discharges reaching it along the vagus and sympathetic nerves. The former is inhibitive in its action, the latter augmentative."

Irritability is that property of living tissue which permits it to respond when stimulated. Without irritability there would be no heart beat, since the heart muscle would not respond to the stimulation periodically produced by the pacemaker. The measure of irritability can be derived from the intensity and duration of the stimulus required to evoke a certain response, or from the magnitude of the response evoked by a given stimulus (36).

Figure four represents a diagram of the conduction system of the human heart. This conduction system is composed of a specialized "embryonal" muscular type of tissue (41). It is the pathway whereby the muscular units of the heart are coordinated in their action and it is com - posed of: (1) The Sino-Auricular Node, (2) The Auricular System, (3) The Auriculo-Ventricular Node, (4) The Main Bundle of His, (5) The Right and Left Bundle Branches, (6) The Purkinje Ramifications. This communication system carries an impulse or stimulus which excites the muscular

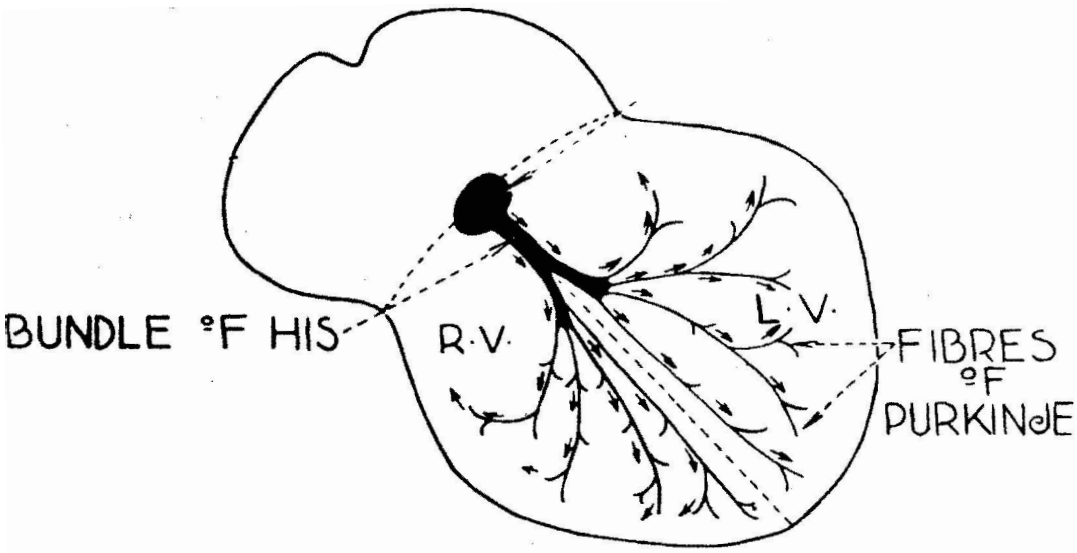

Figure 4. Diagram of the Conducting system
From Electrocardiography (29).
Explanation on page 32.

units to coordinate contraction. The exact nature of the stimulus is unknown, however, it may be said that it is a from of electrical energy which can be registered on a sufficiently sensitive galvanometer (25).

As has already been mentioned, the Sino-Auricular Node is known as the pacemaker, inasmuch as the stimulus to contraction is initiated in this area and spreads through the remainder of the conduction system to reach every muscular unit. The S-A Node is also sometimes known as the Node of Keith and Flack. It lies in front of the lateral part of the superior vena cava in a sulcus where the vein joins the right auricle. It is spindle shaped, measuring about 1 cm. in length, 3 mm. in breadth, and 1 mm. in thickness, and is covered by epicardium and epicardial fat. Small processes from it spread into the auricular musculature. It is supplied by a single artery. Histologically it is composed of interlacing fibres similar to muscle cells but without striation.

The conduction system of the auricles has been a controversial issue. Some observers have stated that it is similar to the ventricular structure, while others maintain complete absence of any definite system with direct muscular transmission (41).

The Auriculo-Ventricular Node or Node of Tawara is about 6 mm. x 3 mm. x 1 mm. It lies in the lower portion

of the atrial septum. It is not well defined and is difficult to dissect anatomically from the main bundle which leads from it.

The Bundle of His or atrio-ventricular bundle originates from the above described node and courses through the septum membranaceum. It is about 1 cm. in length and shaped as a narrow flat band. It can best be located as just under the posterior cusp of the aortic valve. It courses downward beneath the endocardium into several branches. One of the main stems passes to the right ventricular wall with the moderator band and there subdivides.

The Left Bundle Branch is located beneath the posterior aortic cusp and passes downward subendocardially toward the apex. The subdivisions ramify over the lateral wall of the left ventricle.

The Purkinje System ramifies over the entire walls of both ventricles subendocardially as a network of the terminal twigs of the sub-bundle branches. They penetrate into the muscle substance and are distributed to each muscular unit. Histologically they are fine interconnecting fibres (4).

THE ELECTROCARDIOGRAPH;
ITS SIGNIFICANCE IN REGARD TO ARRHYTHMIAS

The basic principles of the electrocardiograph have been described in the "history"; therefore more detailed description of the instrument is not necessary here.

In its early years the electrocardiograph was used chiefly as an aid in the explantion of cardiac arrhythmia, tachycardia, and bradycardia, having proved to be more satisfactory than the mechancial graphic methods previously employed because of the greater ease of technique and interpretation and because of the more complete information afforded. As time went on, however, it was learned that more important data about the heart than the explanation of abnormalities of rate and rhythm are shown by the electrocardiogram, from a study of the shape, direction, amplitude, and time relations of the individual waves or deflections (41).

Katz (21) states, "There is nothing in the electrocardiogram that gives any information, except inferentially, concerning the property of contractility and the property of tonus." The electrocardiogram can give no information of how vigorously the heart is beating, nor whether the heart has good or poor tone (24). The electrocardiogram in reality depicts only where the im-

pulse originates and the pattern of its spread through the cardiac musculature, therefore giving information regarding the properties of rhythmicity, conductivity, and irritability only (21).

It must be emphasized that the electrocardiograph does not take the place of such other diagnostic methods as history taking, percussion, auscultation and roentgenology, but it does obviate in large part the need of taking mechanical graphic records of arterial and venous pulses and of the apex impulse (42). Finally, it is important to remember that the electrocardiogram may be perfectly normal even in the presence of serious heart disease, for example, in those conditions in which rhythmicity, conductivity, and irritability are not affected.

The first and fundamental step in studying electrocardiograms is to become familiar with the so-called leads. An "electrocardiographic lead" is the connection of any two parts of the body by electrodes and wires with the recording galvanometer. Although two electrodes may be attached to any parts of the body (if they are not too far from the heart and too close together) to lead the heart current to the galvanometer, it has been found by experience that the most suitable lead points for routine clinical use are the forearms, the left leg, and the precordium at the cardiac apex (45).

At the present time, four leads are ordinarily used for each patient studied, although a few cardiologists have recently incorporated the use of a fifth lead, another chest lead (38). Whereas the use of four or five leads is now considered standard in modern electrocardiography, it was customary to use three routine leads up until 1932 or 1933 (37). These three leads are now called the "classical" leads, and since they are adequate to demonstrate most arrhythmias, illustrations, for the most part, will be confined to them.

Lead I consists of the connection of right lower arm to one end of the galvanometer string and of the left lower arm to the other end so that the preponderant spread of the action current (which has been called the wave of relative negativity) in the direction of the lead, that is from right arm to left, is represented normally in the electrocardiogram by an upright deflection of the string shadow, while its reverse direction is represented by an inverted deflection. Lead 2 consists of a similar arrangement substituting left leg for left arm. Either leg may be used with little or no difference of electrical potential during the cardiac cycle; the left leg is, however, the customary lower lead point. In Lead 3 the left leg lead continues to be the lower contact, while the left arm is substituted for the right arm. Thus these three lead

points, when connected, form a triangle, which is essentially equilateral. Electrically and geometrically, Lead 2 is equal to the sum of Leads I and 3, since the difference of electrical potential between right arm and left leg is about the same whether the lead points are connected directly or in a roundabout way. Therefore the wave P_2 should equal P_1 plus P_3, Q-R-S_2 should equal Q-R-S_1 plus Q-R-S_3, and T_2 should equal T_1 plus T_3 (these letters refer to auricular and ventricular deflections in the electrocardiogram soon to be discussed, while the appended numbers refer to the particular leads--1, 2,and 3). Similarly Lead 2 minus Lead 1 equals Lead 3, and Lead 2 minus Lead 3 equals Lead 1. Lead 4, also called the chest lead, consists of connection of the precordium at the cardiac apex to the galvanometer by an electrode to which the right arm wire is attached while the other or so-called indifferent electrode is placed on the left leg (or back of the thorax at the angle of the left scapula (42).

The relationship of the electrocardiogram to the anatomical elements of the heart is shown in figure 5. The P wave represents the passage of the impulse from its origin in the S-A node through the auricles, stimulating them to contraction. The P-Q (or P-R when Q is absent) represents the delay at the A.V. node and the main bundle of His. The Q R S portion of the electrocardiogram is

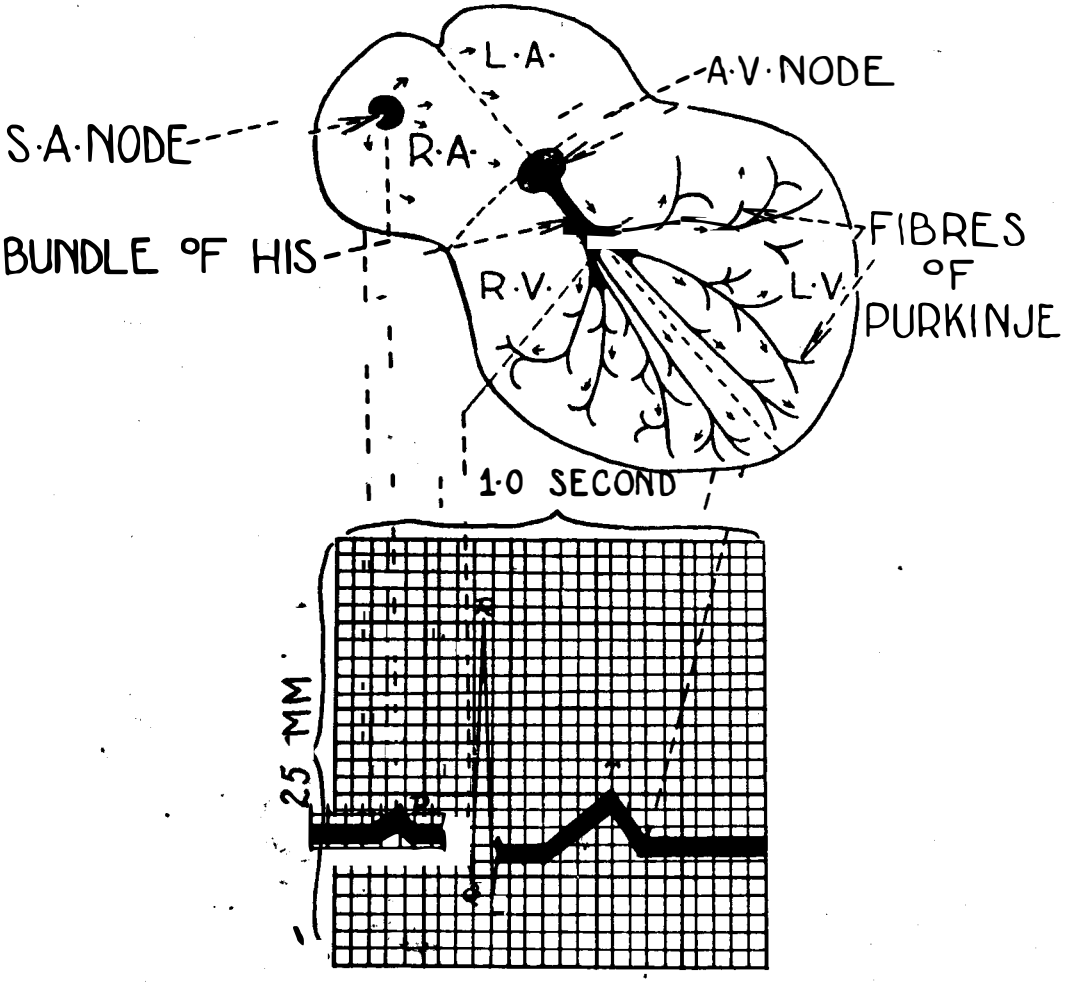

Figure 5. Relationship of EKG to Anatomical Elements Of the Heart.
From Electrocardiography (28).
Explanation on Page 40.

related to the spread of the stimulus through the bundle branches, the Purkinje fibers, and the terminal network, stimulating the ventricles to contraction (29). The Q R S complex is also called the "initial ventricular deflection" since its consists of a triad of waves occurring at the very beginning of ventricular contractions (31). The T wave represents the recession of stimulus in the ventricles. Since the T wave occurs during ventricular systole (the ventricles are contracting even though the stimulus has recessed), it is combined with the Q R S waves and cllaed the "ventricular complex" (31).

Figure six shows a more complete analysis of the cardiac cycle as portrayed by the electrocardiogram. Besides breaking the graph up into its components, this diagram also shows the presence of a U wave. The exact import of the U wave is unknown, although it is now generally considered to be a manifestation of electrical stress within the heart resulting from changes in position of the heart during ventricular relaxation (34).

Figure seven, taken from Katz (21), is a series of diagrams showing the temporal correlation of electrical and mechanical events (above) with the electrocardiogram (below) in a human heart with a cycle of 0.76 second. The intervals between diagrams are 0.02 second except between 1 and 2, 6 and 7, 25 and 26, 26 and 27, 30 and 31,

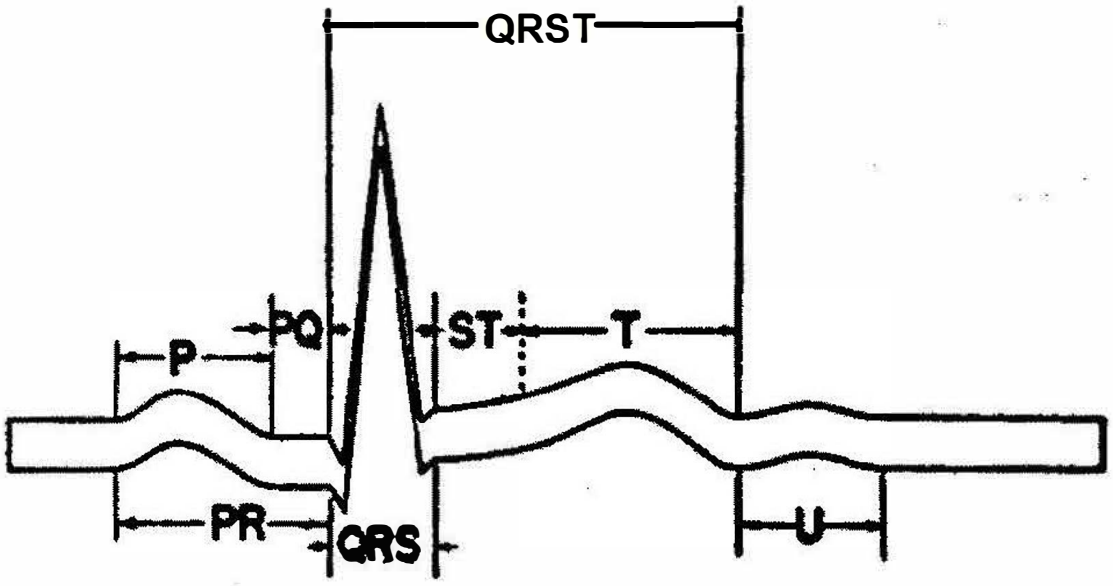

Figure 6. Elements of a Normal EKG Wave.
From Electrocardiography (21).
See page (41) for explanation.

and 31 and 32--this was done in the original for purposes of spacing. The blacked out portion of the electrocardiogram shows how much of the record had been written up to the time represented by each diagram. As the excitation wave spreads, the area passed through is depolarized. In the diagrams the depolarized state in the heart is shown by cross-hatching and that of the polarized or repolarized stage by the absence of cross-hatching. The walls of the aorta and veins are stippled. The special muscular tissue --the conduction system, except for the Purkinje fibers, are shown in black.

Attention should be paid to the changes in size and shape of the cardiac chambers, aorta, superior vena cava, coronary sinus and pulmonary veins, as well as to the changes in thickness of the walls of the auricles and ventricles and the opening and closing of the aortic and A-V valves in the successive diagrams. Note that the series starts at 1 with the heart in diastole, the A-V valves open, the seminlunar valves closed and the impulse discharged in the sinus node still within the node. At 2 the impulse has begun to spread through the auricles. At 4 the auricles have begun to contract and cause the A-V valves to close partially. At 7 stimulation of the auricles is completed. At 8 the repolarized state has begun to reappear in the auricles. At 9 the impulse has

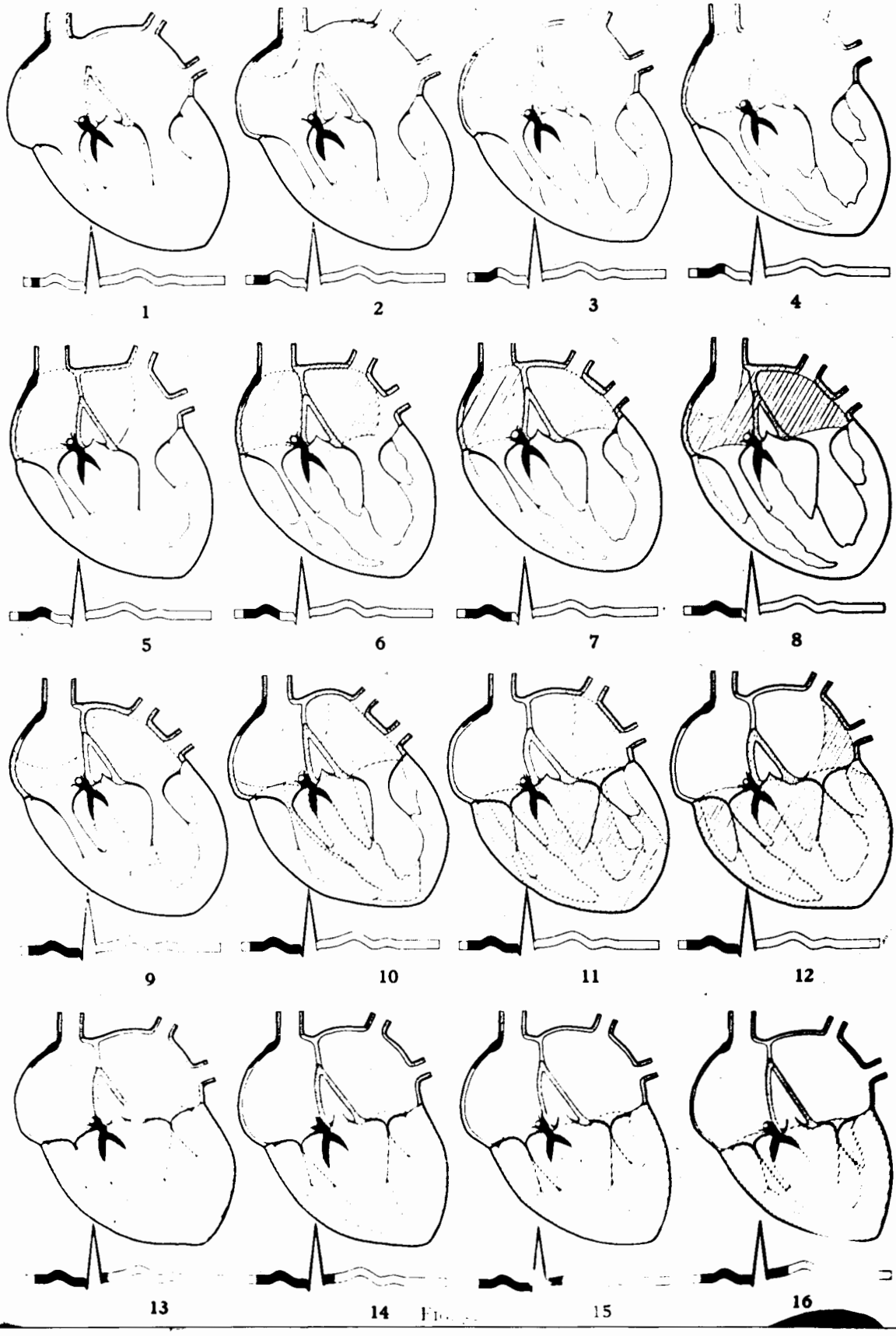
1
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Figure 7. (Other side of page).
Temporal Correlation of Electrical and
Mechanical Events in the Human Heart,
with the EKG.
From Electrocardiography (21).
See Page 42 for Explanation.

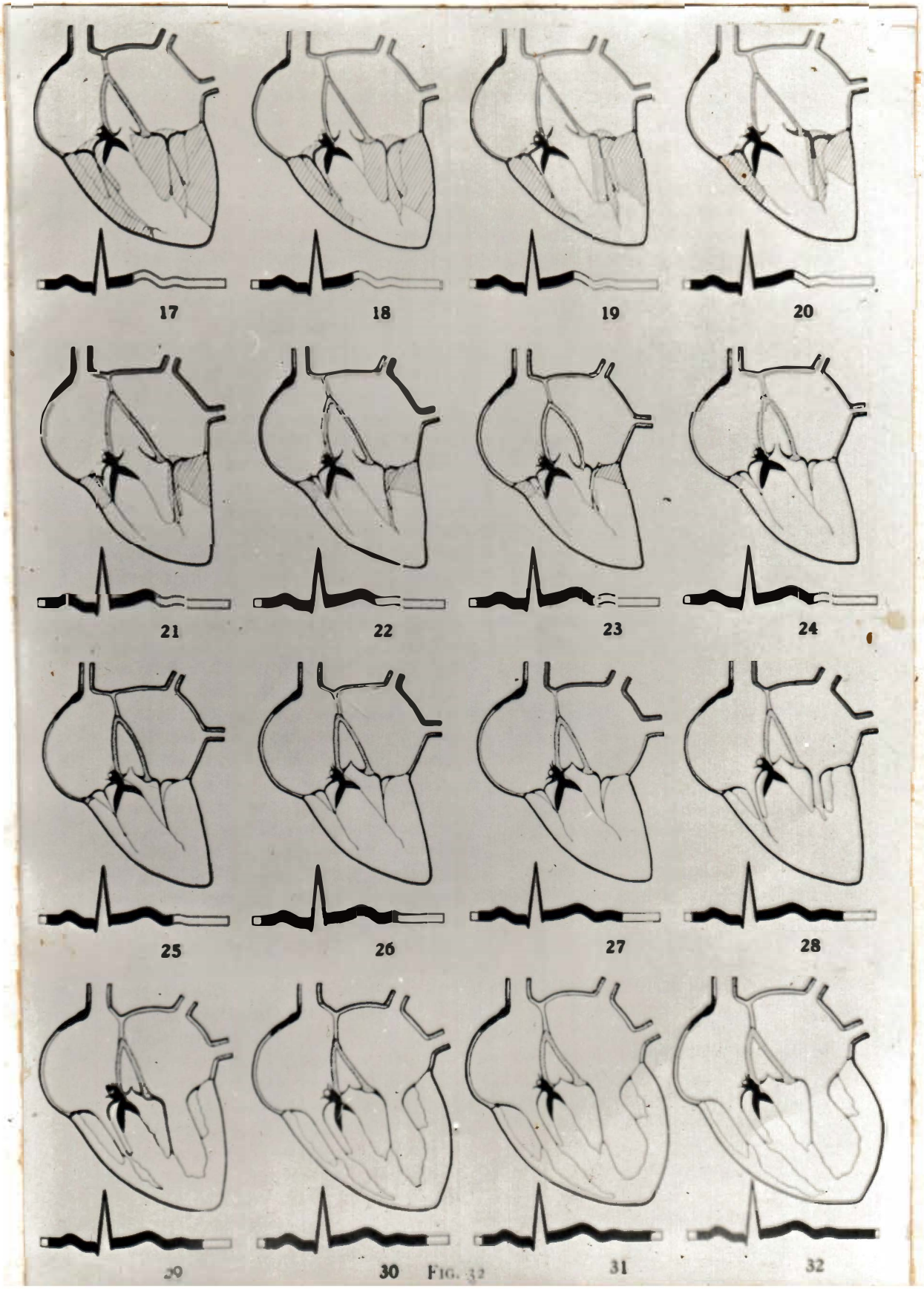

FIG. 32

begaun to spread in the ventricles. At 11 the A-V valves are closed as ventricular contraction begains. At 13 the ejection of the ventricles begins with the semilunar valves open, and stimulation of the ventricles is completed. At 14 repolarization in the auricles is completed. At 15 repolarization in the ventricles has begun. At 26 repolarization in the ventricles is completed, relaxation of the ventricles has begun, and the semilunar valves have closed. At 28 the A-V valves have again opened and filling of the ventricles has begun. At 32 the heart cycle is completed.

This remarkable series of diagrams, for reasons that are self-evident, exemplify the saying that, "A picture is worth a thousand words."

The Cardiac Arrhythmias; Etiology, Diagnosis (Including Symptoms and Signs), and Treatment.

The value of complete and concise history-taking as an aid to diagnosis in cardiac arrhythmias is as important as in any other phase of medicine. In the "past history", special reference should be made to diseases associated with heart conditions, such as rheumatic fever, syphilis, hypertension, exophtholmic goiter, scarlet fever, nephritis, anemia and chronic pulmonary disease (27). The patients social life, habits and professional status warrants consideration--particularly in regard to mental or physical overwork. Family history may furnish important clues to diagnosis. In eliciting a history of the "present illness", particular attention should be paid to any pain which might be interpreted as being of cardiac origin; also, attention should be paid to complaints of palpitation or of respiratory abnormalities.

With these facts in mind, and with a picture of normal cardiac activity at hand, for purposes of comparison, we may proceed with a discussion of the arrhythmias.

Sinus Mechanisms

Sinus Tachycardia

Etiology: Sinus Tachycardia is the term given to an increased contraction rate of 100 or more per minute (3). The impulses all arise in the normal site, i.e., the sinus node, and follow a normal pathway as previously described. The rhythm remains regular. Simple sinus tachycardia (figure 8) has no prognostic significance.

Its most usual causes are:

(1) Normal.

- (a) After exercise
- (b) In Infants

(2) Abnormal

- (a) Hyperthyroidism
- (b) Fever
- (c) Fright

Diagnosis:

(1) Regular rate of over 100.

(2) Gradual increase as distinguished from the sudden onset of paroxysmal tachycardia.

(3) Slight changes in rate by holding of breath.

(4) Increase in rate with exercise.

(5) May be slowed by vagal pressure.

(6) Rate may be altered by use of drugs.

As may be seen, the E. K. G. is simply a fast normal.

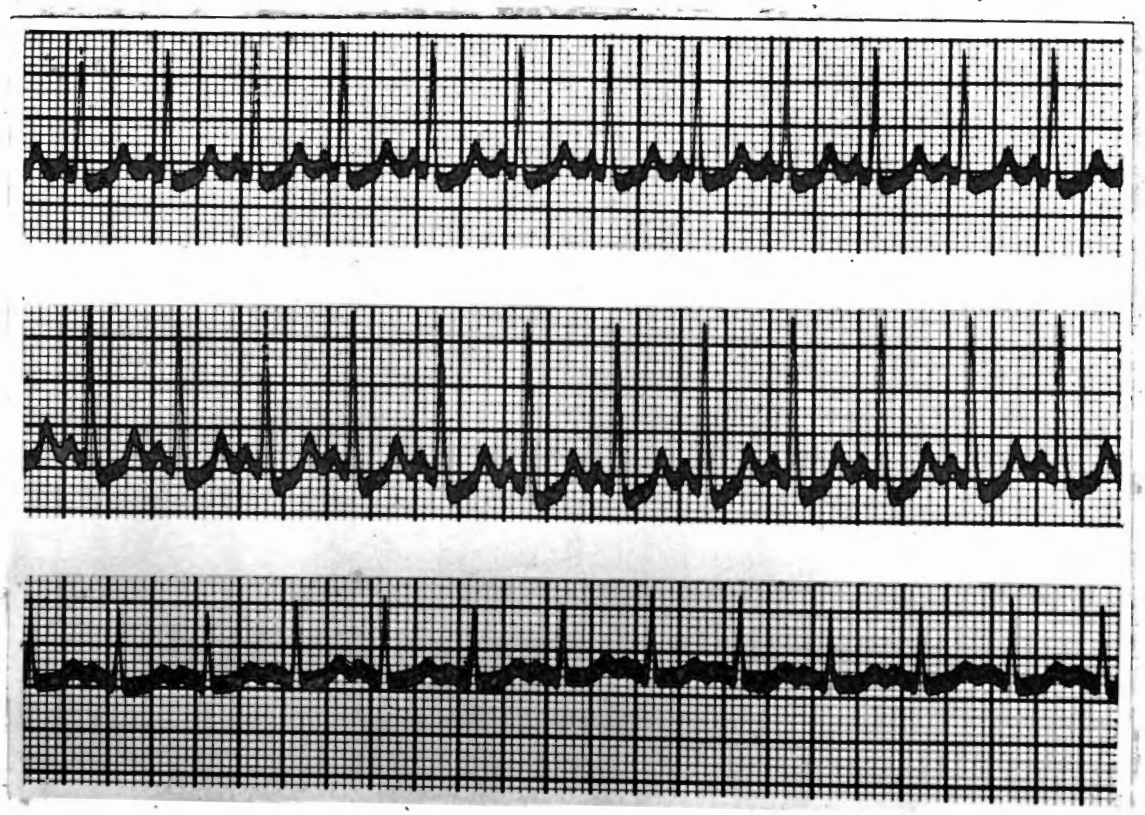

Figure 8. EKG. (diagram) Showing Sinus Tachycardia.

Summary: Normal sinus mechanism with rapid rate, without conduction deformities.

From Electrocardiography (29).

Explanation on page 48.

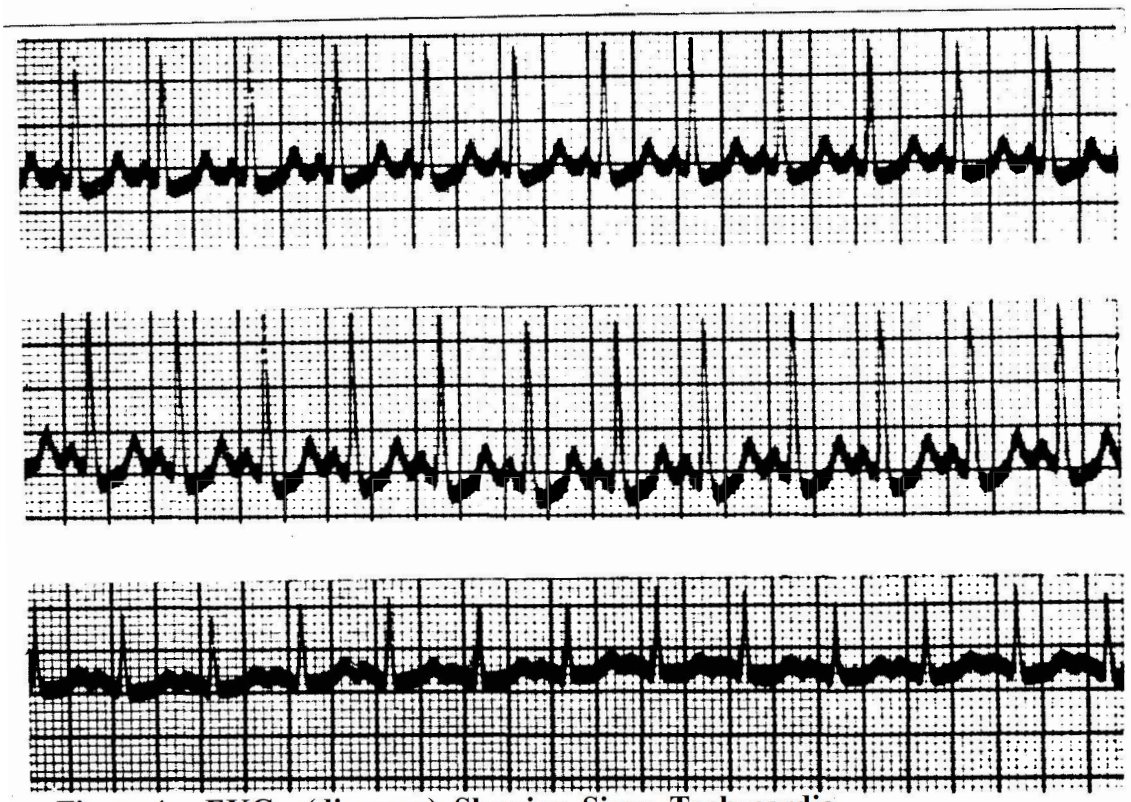

Figure 4. EKG. (diagram) Showing Sinus Tachycardia.

Summary: Normal sinus mechanism with rapid rate, without conduction deformities.

From Electrocardiography

(29). Explanation on page 48.

Treatment: Avoidance of causative factors wuch as over-exercise or fright. Treatment of underlying cause in cases of hyperthyroidism or fever.

Sinus Bradycardia

Etiology: This bradycardia (slow beat) sometimes called normal bradycardia means simply that the impulse of stimulation is produced very slowly by the sino-auricular node (36). Except that stimuli are sent out at infrequent intervals, they otherwise proceed normally.

Arbitrarily, a bradycardia is present when the rate is less than 50-60.

Bradycardia occurs in::

1. Jaundice.
2. After prolonged fevers.
3. Athletes.
4. Cerebral hemorrhage.
5. Brain tumor.
6. Hydrocephalus.
7. Meningitis.
8. Transient during fainting and after parturition.
9. After large doses of pilocarpine, aconitine and morphine.

Diagnosis: Clinically, it is important that we distinguish this from heart block. Test is as follows: Pace

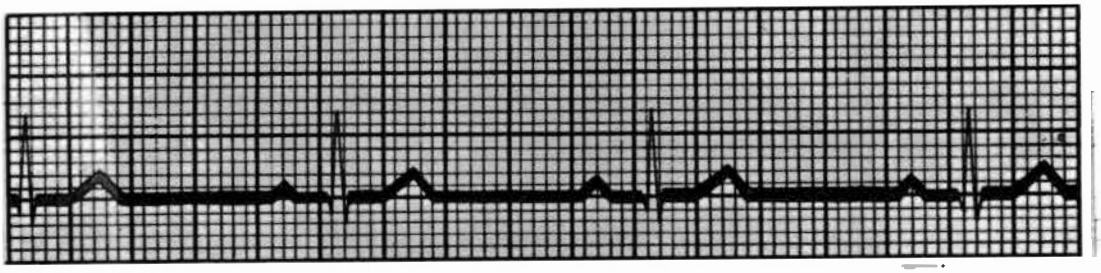

Figure 9. EKG (diag.) Showing Sinus Bradycardia.

Summary: Shows normal pathway but slow emission of stimuli by pacemaker (S-A node).

From Electrocardiography (29).

Explanation on page 50.

heart for a few cycles, then use Vagal or Ocular pressure. As the heart stops and a cycle drops out if the next cycle is picked up on its regular paced time, HEART BLOCK and not BRADYCARDIA is probably present. In other words, there is a 2:1 block. In bradycardia, after Vagal pressure the next cycle will not be picked up on its regular paced time.

In this arrhythmia the electrocardiogram (figure 9) is simply a slow normal.

Treatment: That of the underlying cause. Belladonna and atropine may be useful when extreme slowness gives rise to syncope.

Sinus Arrhythmia

Etiology: This is the most common form of arrhythmia and has no clinical significance. It is physiological. This phenomenon is vagal in origin and the irregularity in the cardiac rhythm is due to irregular impulse formation in the sino-auricular node(15).

Most common causes are:

1. Deep breathing in normal adults.
2. Common in childhood with quiet breathing.
3. If it occurs in adults with quiet breathing it implies myocardial damage.

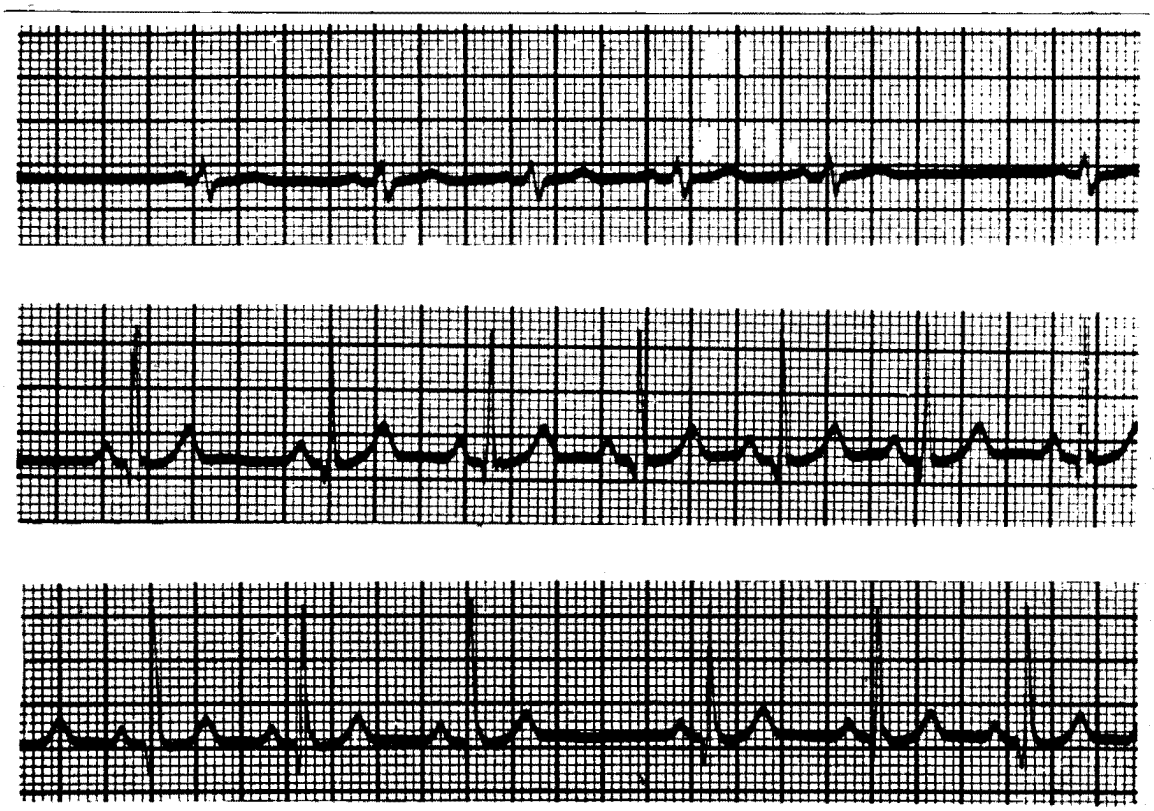

Figure 10. EKG (diag.) Showing Sinus Arrythmia.
Summary: QRS complexes diphasic in Lead I and upright in Leads II and III. T waves are upright and of normal contour. P-R interval 0.16 second. Waves within normal limits except for irregularity of S-A emission.
Ecplanation on page 54.

4. May occur as result of digitalis administration.

5. May occur during convalescence.

Diagnosis: Easily made clinically by use of stethescrope because of the "regularity in the irregularity". The waves of the electrocardiogram do not deviate from the normal except in temporal relations i.e., from one wave to another (figure 10). It is sometimes important to differentiate this condition from auricular fibrillation. This is done by increasing the heart rate, in which case the latter is intensified, while sinus arrhythmia becomes abolished.

Treatment: None, unless myocardial damage indicated, then treat as per any organic lesion.

Sinus Premature Systole

Etiology: The sinus node may emit an impulse to contraction out of sequence and for no known reason. This mechanism in rare and of no practical interest clinically(5).

Diagnosis: The EKG shows a premature systole with a P wave and ventricular deflections in all leads which are indistinguishable from those characteristic of the basic sinus rhythm. The cycle preceding and the cycle following the premature beat have a combined length less than two normal cycles. If the premature systole arises in

a region of the sinoauricular node different from the site at which the basic rhythm arises, its P wave will be somewhat abnormal. It is then indistinguishable from premature systoles arising elsewhere in the auricle.

Treatment: None, unless findings indicate underlying heart disease.

AURICULAR MECHANISMS

Auricular Premature Systole

Etiology: These are premature contractions arising in an ectopic auricular focus (36). Because this ectopic focus sends out an impulse which evokes a ventricular response premature to the normal rhythm, the auricle and ventricle are in a refractory state which precludes the possibility of response to sino-auricular stimulation in its regular place; therefore, following the premature contraction, there is a so-called "compensatory pause".

This is a very common type of arrhythmia, occurring in both normal and diseased hearts. The condition usually results from increased irritability of the heart muscle in which it arises (29). Such increased irritability may be due to various influences, such as:

(1) Nervous (vague or sympathetic).

(2) Toxic (pneumonia, influenza.

(3) Chemical (digitalis, nicotine, calcium, barium, chloroform).

(4) Changes in the heart muscle itself.

Excess coffee, tea or tobacco, fatigue, and constipation may produce premature contractions. Psychic disturbances sometimes may be the underlying cause. Premature systoles are common in organic heart disease, especially the rheumatic, hypertensive and coronary sclerotic types.

Diagnosis: Sometimes the extra systole may be felt by the patient who feels as if his heart had "jumped" or "turned over." Clinically the coupling may be readily recognized with the stethoscope.and sounds somewhat as follows:

Lub dub--Lub dub--Lub dub ub dub-----Lub dub--lub dub

(Premature Contraction) (Compensatory Pause)

Differentiation as to source of the extra systole cannot definitely be made with the stethoscope but circumstantial evidence may be obtained. Those of auricular origin are not followed by a full compensatory pause. Those of ventricular origin are more noticeable to the patient than the auricular type. Premature beats of auricular origin may sometimes be detected at the radial pulse.

In the EKG when the stimulus arises from within the auricle the P wave only is deformed. It may be low, in-

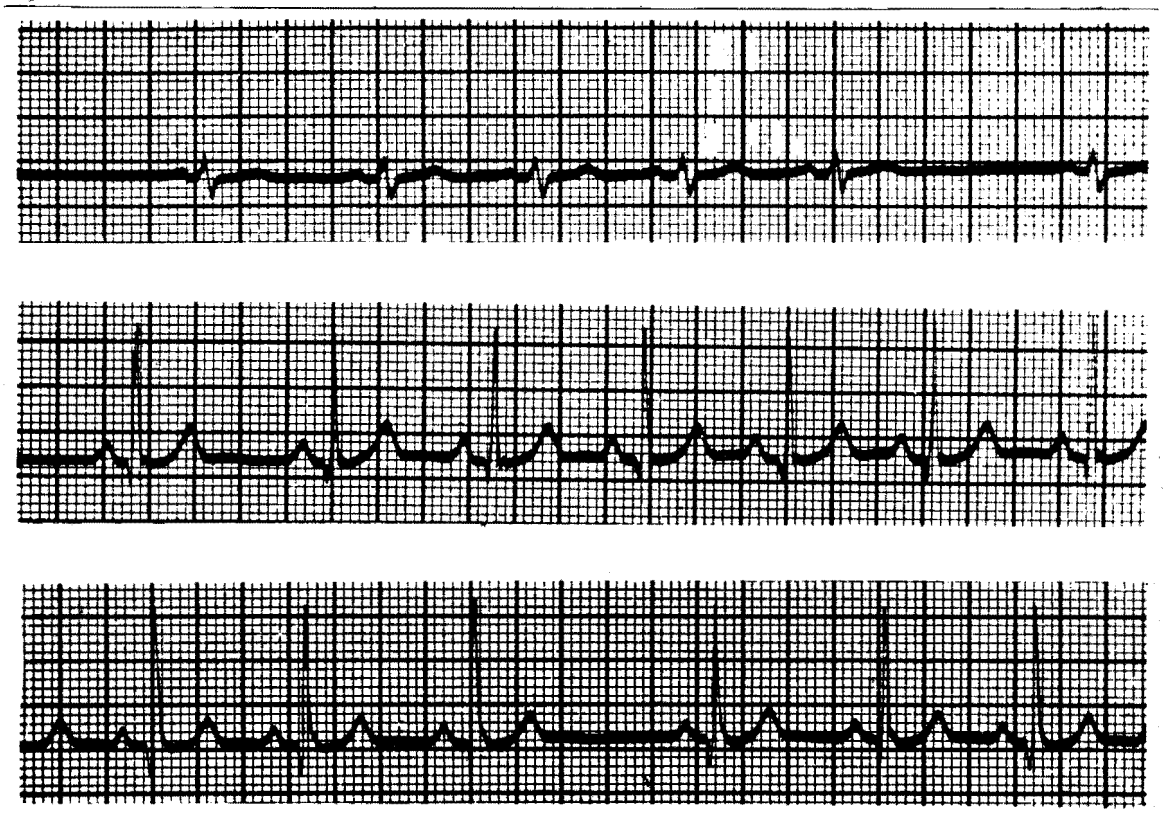

Figure 10. EKG (diag.) Showing Sinus Arrythmia.
Summary: QRS complexes diphasic in Lead I and upright in Leads II and III. T waves are upright and of normal contour. P-R interval 0.16 second. Waves within normal limits except for irregularity of S-A emission.
Ecplanation on page 54.

verted or diphasic under these circumstances. The P-R interval is shortened or lengthened, but the ventricular complex, the QRST, usually remains unchanged, as in (figure 11). The ventricular pathway, however, may be abnormal, producing a deformity of the QRS (21).

Extrasystoles sometimes occur in rhythmic sequence. They may follow every regular beat, with a continual coupling which is known as pulsus bigeminus. When every third beat is an extrasystole, it is a pulsus trigeminus; and if every fourth beat is an extrasystole it is a pulsus quadrigeminus. The irritation produced by digitalis is the most frequent etiologic factor for this type of phenomenon, in which case a state of digitalis poisoning is indicated (11).

Treatment: If underlying heart disease is shown to be present the condition must be treated accordingly. If there is no heart lesion, sedation may be used if the symptoms are disturbing to the patient, otherwise no treatment is necessary.

Auricular Tachycardia (Paroxysmal)

Etiology:This is by far the most common and least important type of paroxysmal tachycardia, occurring about six times more often than the ventricular type. It is a sudden rapid heart action which originates in an ectopic

focus in the auricle, and is not primarily associated with organic heart disease (40).

Diagnosis: In this arrhythmia the rates vary from 180-250, are usually about 200. The rate remains fixed for any one attack. Separate attacks are apt to have rates in the vicinity of one another; for example, 184, 180, 186, etc. It is often started by dreams, gas, sudden emotion, fright, sudden changes of position, anesthetics, etc. In the E K G the diagnosis of supraventricular tachycardia is made when the heart rate is over 180 beats per minute and regular, and the Q R S complex has a duration of less than 0.10 second (21). See figure 12.

In long runs of supraventricular paroxysmal tachycardia it is not always easy to determine the point of origin of the ectopic beats. If a P wave is found with an approximately normal P-R duration, the origin can be said to be auricular. When no P wave is seen or an inverted P wave is present with a shorter than normal P-R interval, or when such a P wave can be shown to follow the Q R S complex, then the origin is said to be in the A-V node. But the decision is not readily made in most cases; hence, whenever doubt exists, the attempt to locate the focus is not pursued further and the record is grouped in the more inclusive term supraventricular paroxysmal tachycardia. When a run of rapid heart action occus with Q R S complexes

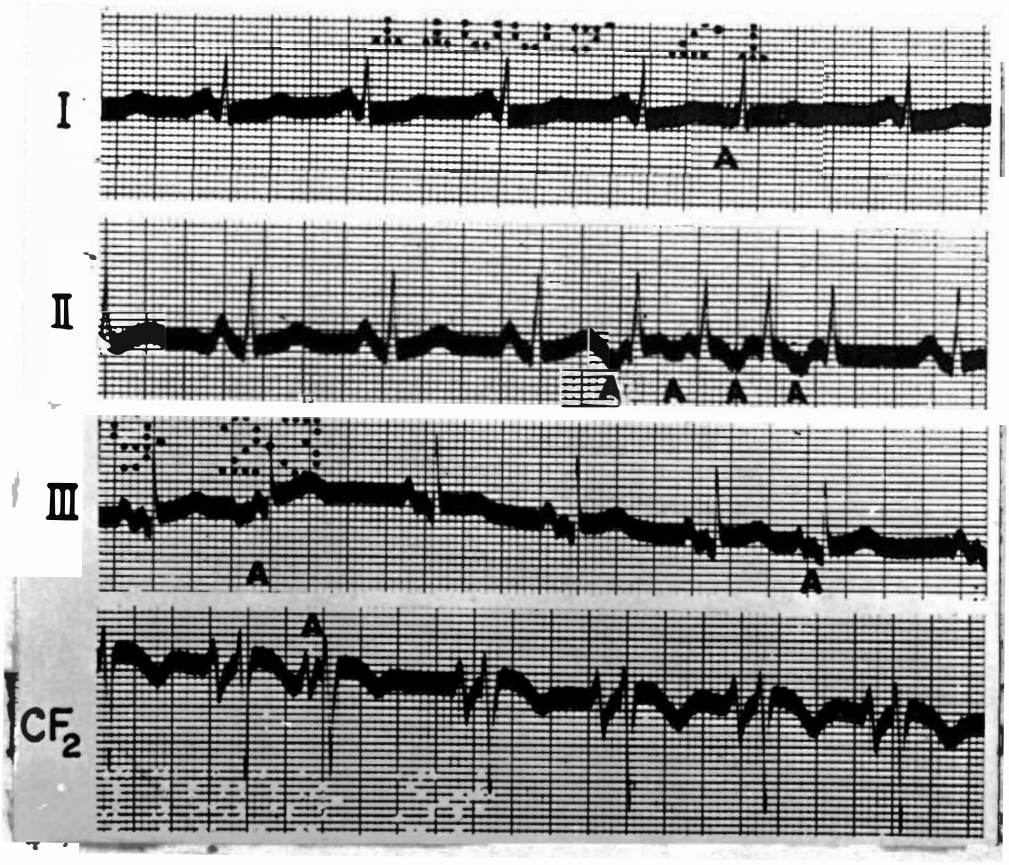

Figure 12. EKG Showing Auricular Tachycardia.

Summary: Single premature systoles (a) occur in leads I, III and CF_2 (chest lead). In Lead II there is a quartet of premature systoles (a). The P-P invervals in this short paroxysm of tachycardia are uniform.

From Electrocardiography (21).

Mechanism explained on page 59.

of normal duration, two other possibilities should be considered, namely, auricular flutter with regular 1 to 1 or 2 to 1 ventricular response, and auricular fibrillation. In the event of the latter, closer inspection of the record will reveal the irregularity in the timing of the ventricular complexes(28).

Treatment: Have patient hold deep breaths or exert vagal pressure. Pressure on the eyeball will slow the heart by reflex mechanism--this method is best if the patient is on the operating table. If the symptoms occur often and are distressing to the patient, quinidine sulfate may be given.

Auricular Flutter

Etiology: Auricular flutter is a very rapid regular auricular beat which replaces the normal auricular beat. The rate of the auricle varies between 200 and 380, but following quinidine administration, it may fall to as low as 120. Auricular flutter is found in both paroxysmal and chronic persistent forms; the latter state may continue unchanged for years.

The ventricles almost never beat as rapidly as the auricles, that is, a 1:1 ratio is extremely rare. Commonly the ratio of ventricular to auricular beats is 1:2; less often 1:4 or even 1:5 and 1:6. Not infrequently the ventric-

ular response is irregular. In exceptional circumstances there may be complete A-V heart block with an idioventricular rhythm.(20).

Auricular flutter is the transient physiologic mechanism which usually precedes auricular fibrillation and its etiologic background is the same. It occurs most commonly in long standing cases of mitral stenosis or hypertension. It may be seen in thyrotoxicosis and in arterior-sclerotic heart disease with coronary occlusion (1).

The mechanism of auricular flutter and fibrillation has been a matter of some controversy. The theory of circus movement proposed by Sir Thomas Lewis is, and has been, the generally accepted explantion to date (26). Lewis believes that the impulse in auricular flutter arises in the right auricle near the S-A node, in the region of the entrance of the great veins. He traces the pathway in the wall of the right auricle in a circular fashion about the superior and inferior venae cavae; this he has called circus movement. In auricular flutter the pathway remains constantly the same, and the flutter waves in the electrocardiogram are similar. Each one follows closely upon its fellow, giving the curve a regular saw-tooth appearance.

Diagnosis: Auricular flutter should be suspected

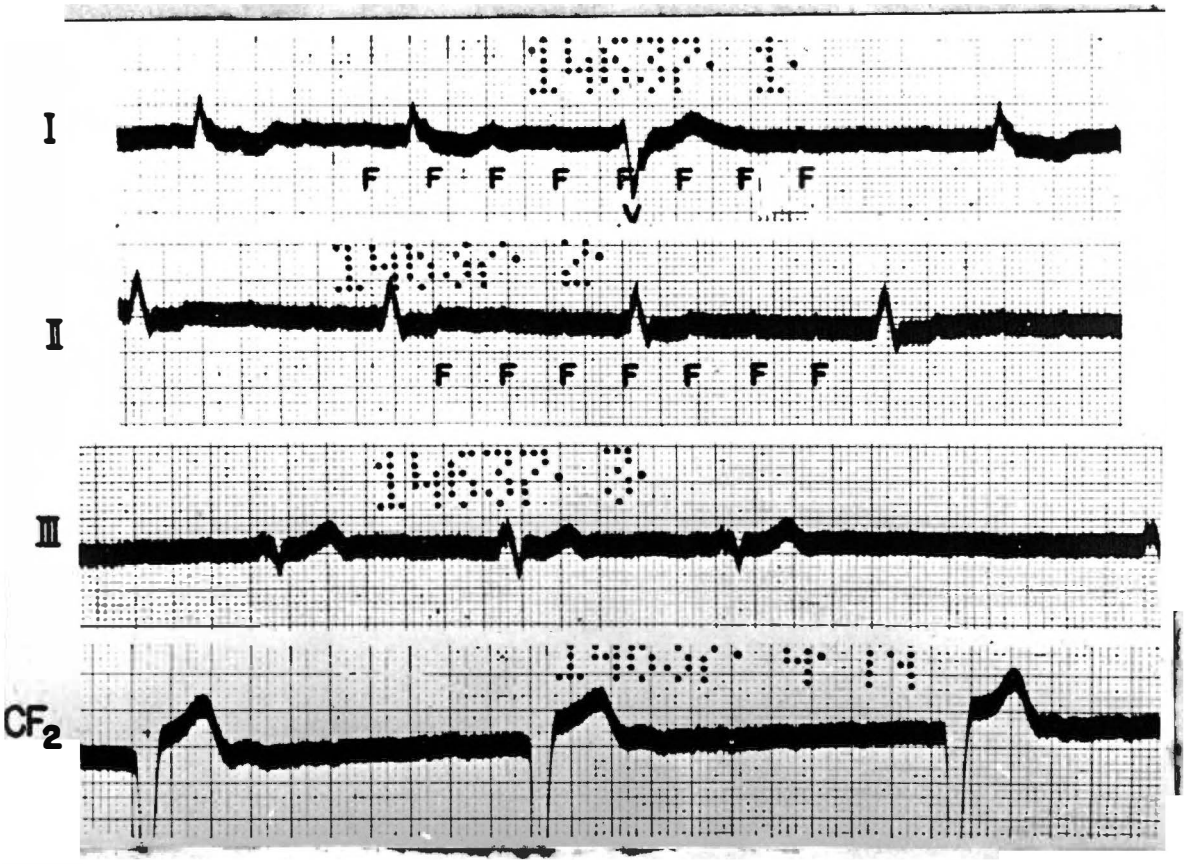

Figure 13. EKG Showing Auricular Flutter.

Summary: Auricular flutter with irregular conduction varying from 3:1 to 7:1. Is attended by ventricular premature systoles (V). The flutter waves are seen best in leads I and II as tiny upright waves, regular in appearance and spacing--rate 188. The bradycardia of the ventricles suggests some depression of the A-V junctional tissue.

From Electrocardiography (21).

Mechanism explained on page 64.

when the ventricular rate is about 150 per minute and the rhythm regular. Under these conditions the rate is unaffected by change in position, by rest, or by moderate exercise. There is generally marked, temporary slowing of the ventricles during carotid sinus pressure. When this occurs, the rhythm may become decidely irregular. The electrocardiogram will confirm the diagnosis(24).

An E K G is usually required to establish the diagnosis, especially to distinguish the condition from auricular fibrillation and from occurence of frequent premature systoles. The key to the electrocardiographic diagnosis of pure auricular flutter is the occurence of auricular activity in the form of so-called F waves, which occur at a rate of from 250 to 380 per minute. These are absolutely regular in spacing, size and contour. At times they cause a continuous undulation of the galvanometer string which is disturbed only by the Q R S T complexes. On occasion they appear more like P waves except that their frequency is too high and they are abnormal in contour. See figure 13.

Treatment: Both digitalis and quinidine have been advocated for reasons inherent in each drug. The vogue now seems to be to use quinidine preceeded by digitalis. In this way, the risk of too great an increase in the ventricular rate, while the auricular rate is slowing, is eliminated (42).

Auricular Fibrillation

Etiology: In this arrhythmia the site of impulse initiation has left the sinus node and is replaced by multiple foci in the auricular musculature. Each single focus sends out stimuli at a rapid rate as in the preceding mechanism, auricular flutter. Thus, auricular fibrillation may be said to be composite auricular flutter. These foci cause localized contraction waves of the auricular muscle which interfere with one another (7).

The etiology has also been considered as due to a circus movement within the auricle which does not follow a constant path. In this case the rate of the circus movement is considered as being between 350 and 500 per minute. The ventricles respond irregularly, the rate depending in large measure upon the facility of auriculoventricular conduction (26).

Auricular fibrillation is most frequently associated with organic heart disease, especially when this is due to arteriosclerosis, hypertension or rheumatic fever. In paroxysmal form, auricular fibrillation may occur at any time during the course of these conditions, but when established permanently, it is usually a manifestation of an advanced form of the underlying disease. Also, auricular fibrillation, expecially in paroxysmal form, is seen occasionally in persons without evidence of organic heart

disease (41).

Diagnosis: Complete irregularity of the heart beat is the cardinal sign of auricular fibrillation. No dominant rhythm can be identified. When the ventriculr rate is over 150 or below 70, it may be difficult to detect the gross irregularity. In the former the irregularity can be brought out by carotid sinus pressure or digitalis, both of which produce AV block; in the latter, by exercise which increases the ventricular rate. Because of the rapid, weak heart action, many beats are not propagated to the peripheral pulse; hence, the apex rate is faster than the pulse rate (pulse deficit). Both apex and pulse rates should be counted simultaneously. Besides the marked arrhythmia, the other signs are those of the underlying heart disease, if any exists (40).

The electrocardiogram is always diagnostic in showing the gross irregularity in spacing of the ventricular complexes (except when there is complete heart block) and the presence of rapid persistent undulations, instead of P waves. These undulations are irregular in spacing, amplitue and contour (29). See figure 14.

Treatment: In the vast majority of cases, auricular fibrillation is readily controlled by digitalis. Although the auricular effect of this drug is to accelerate the rate of fibrillation in the auricles, its primary action

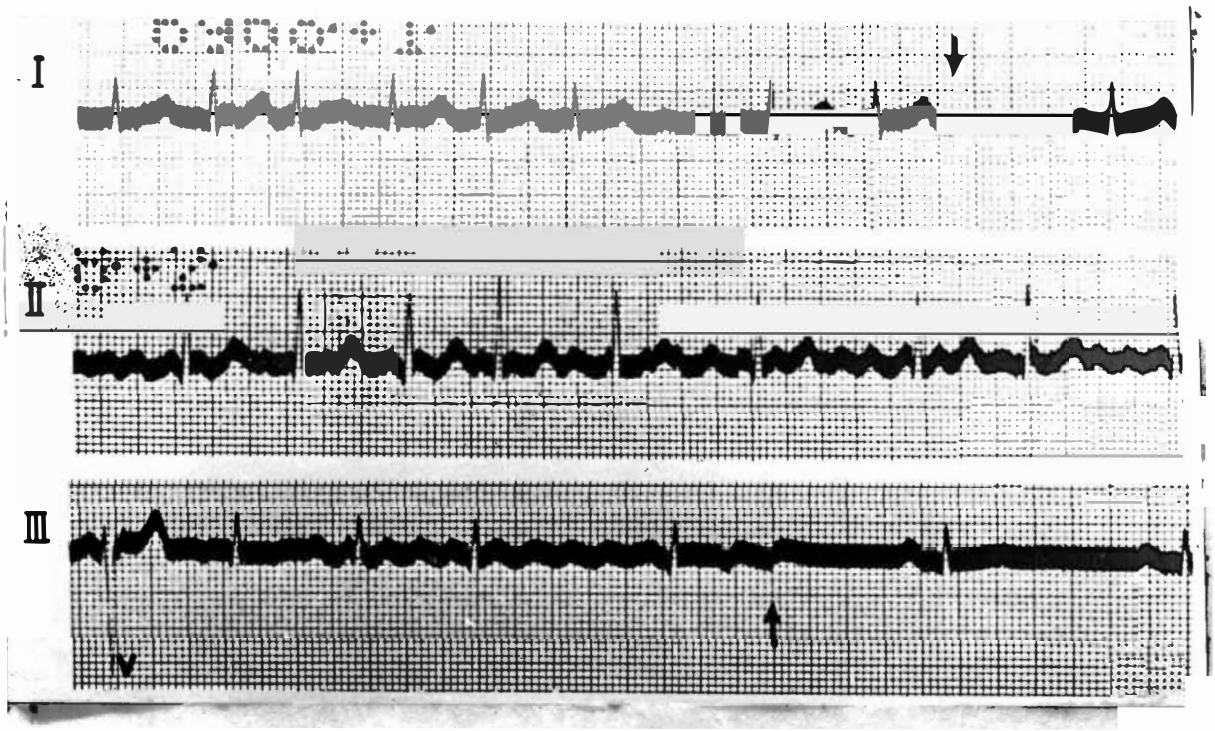

Figure 14. EKG Showing Auricular Fibrillation (Paroxysmal).

Summary: Arrows in I and III show termination of period of fibrillation. During the fibrillation no P waves are seen, instead only undulatory waves, irregular in timing, contour and amplitude--rate about 340. This, with gross irregularity in spacing of ventricular complexes; identifies the condition.

From Electrocardiography (21).

Mechanism explained on page 66.

is to increase the grade of AV block, thereby reducing the number of impulses passing to the ventricles. Quinidine may be employed when the following conditions have been satisfied: Fibrillation is of short duration, advanced rheumatic heart disease is not present, no embolic phenomena have occurred, no marked cardiac enlargement is present and there is no congestive heart failure. In about 50 per cent of patients the drug allows a resumption of sinus rhythm (21).

Wandering Pacemaker

Etiology: Wandering pacemaker, as the name implies, is a continual change of the location of the pacemaker governing the entire heart. The shift may be from one part of the sinus node to another, or from the sinus node to the A-V node and back again. This latter phenomenon, like the former, is in reality a variant of sinus arrhythmia and carries the same significance. It is consequently a normal variant. Unlike sinus arrhythmis where the change in rate is due to alterations within a single pacemaker, the change in rate in wandering pacemaker is due to suppression of one pacemaker while another takes over, which is turn is suppressed, while a third takes over, and so on until the process is repeated (15).

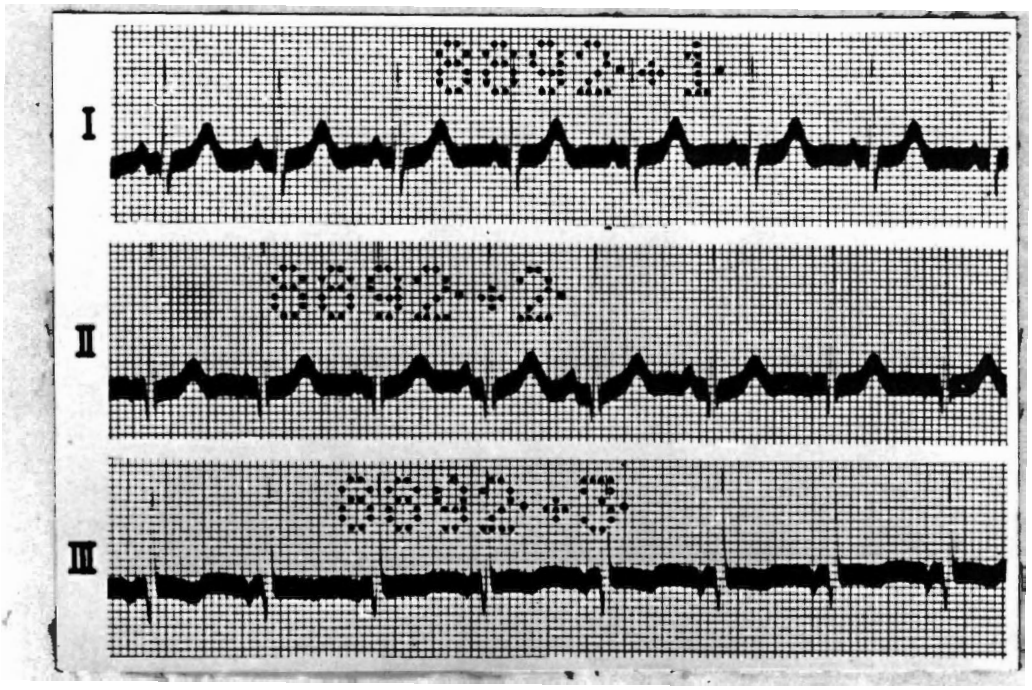

Figure 15. EKG Showing Wandering Pacemaker (Between Sinus and A-V Nodes).

Summary: As the P waves become shorter in duration and smaller in Lead I and more peaked and norrower in lead III, the P-R interval shortens to about 0.10 second, evidence of A-V nodal control. Is slowing of rate as A-V node takes control.

From Electrocardiography (21).

Mechanism explained on page 70.

Diagnosis: Can be definitely made only my means of E K G. The electrocardiographic diagnosis of wandering pacemaker to the A-V node is made when in an arrhythmic record the P wave in a single lead undergoes change in size, shape or even direction simultaneously with changes in the P-R interval, some of which become less than 0.12 second. Since the slower pacemakers are nearer to the ventricles, the P-R interval shortens as the rate slows. As a rule, the smaller and inverted P waves occur in the slower beats and the taller P waves in the more rapid beats. The transition in the P wave in not abrupt but occupies several beats to go from one contour to the other extreme, indicating a progressive shift through intermediary pacemakers (40). See figure 15.

Treatment: Since this is often a physiological phenomenon, no treatment is necessary unless there is an accompanying organic lesion.

AURICULOVENTRICULAR(A-V) NODAL MECHANISMS

A-V Nodal Premature Systole

Etiology: Extrasystoles which originate in the A-V node represent an irritable focus in that region. Their clinical significance is the same as premature contractions originating from within the auricles. The stimulus passes into the auricles in a retrograde fashion and into the

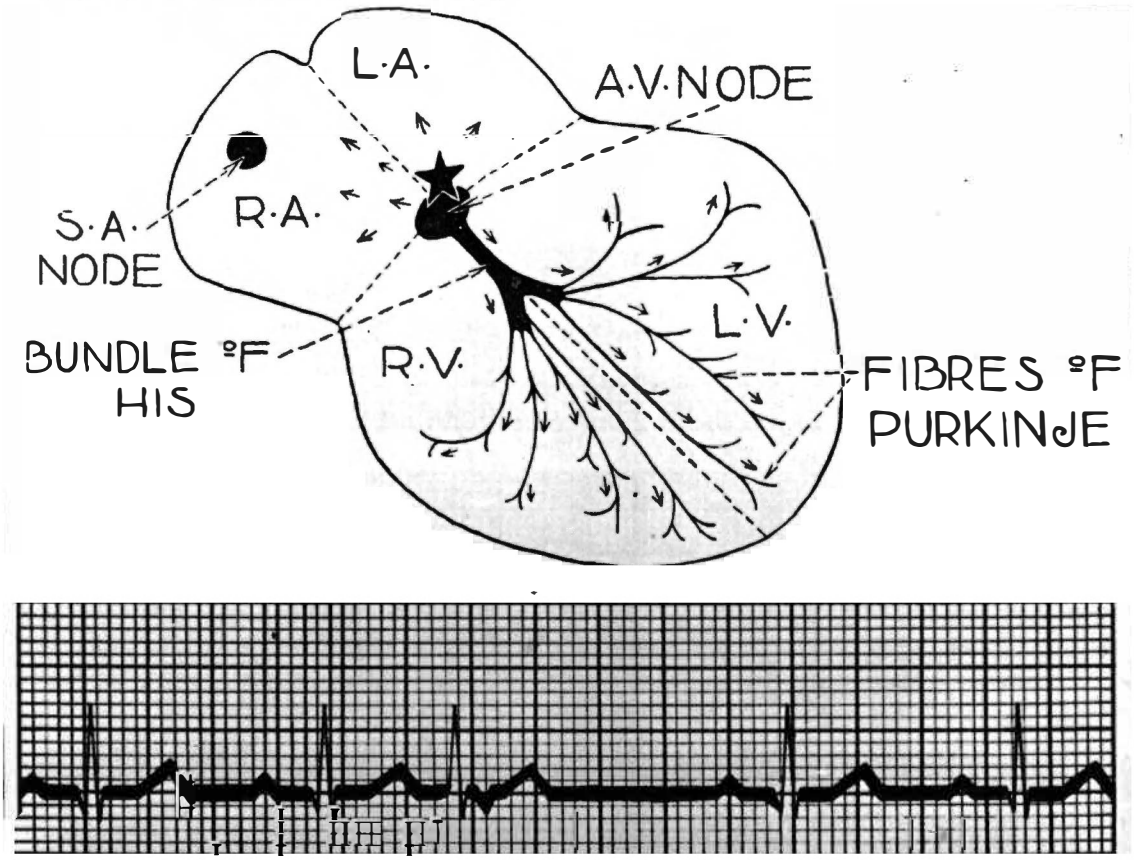

Figure 16. EKG (diag.) Showing Premature Systole of Nodal Origin.

Summary: Extrasystole arises from A-V node--designated by a star. Stimulus passes into auricles in retrograde fashion--into ventricles as ordinarily. No P wave is found before the QRS complex for this reason. Here, the P wave is inverted--(see third cycle above), and follows the QRS complex.

From Electrocardiography (29).

Mechanism explained on page 72.

Ventricles through the main bundle and the branches in the usual fashion. The auricular contractions and ventricular contractions thus occus simultaneously (5). Certain cardiologists believe that frequent and persistent extrasystoles presage the occurrence of a paroxysmal tachycardia. There is no doubt that a paroxysmal tachycardia of nodal origin usually is preceded by the occurrence of extrasystoles of A-V nodal origin. However, few patients with nodal extrasystoles develop the paroxysmal rhythm(15).

Diagnosis: The diagnosis is made electrocardiographically by the short or negative PR interval, usually with an abnormal P. The absence of a P wave may mean that the P is buried in the QRS or may indicate auricular standstill. Auricular fibrillation with complete heart block may be ruled out by using chest leads (38). See figure 16.

Treatment: That of underlying heart condition.

A-V NODAL RHYTHM

Etiology: Nodal rhythm is an infrequent disturbance in which the impulse arises in some part of the A-V node because of loss of the normal sinus control. This condition may on rare occasions appear in excessive digitalization or quinidine, acute infections, or organic damage in the sinus region, commonly the result of coronary artery disease (15). The impulse originates in the A-V

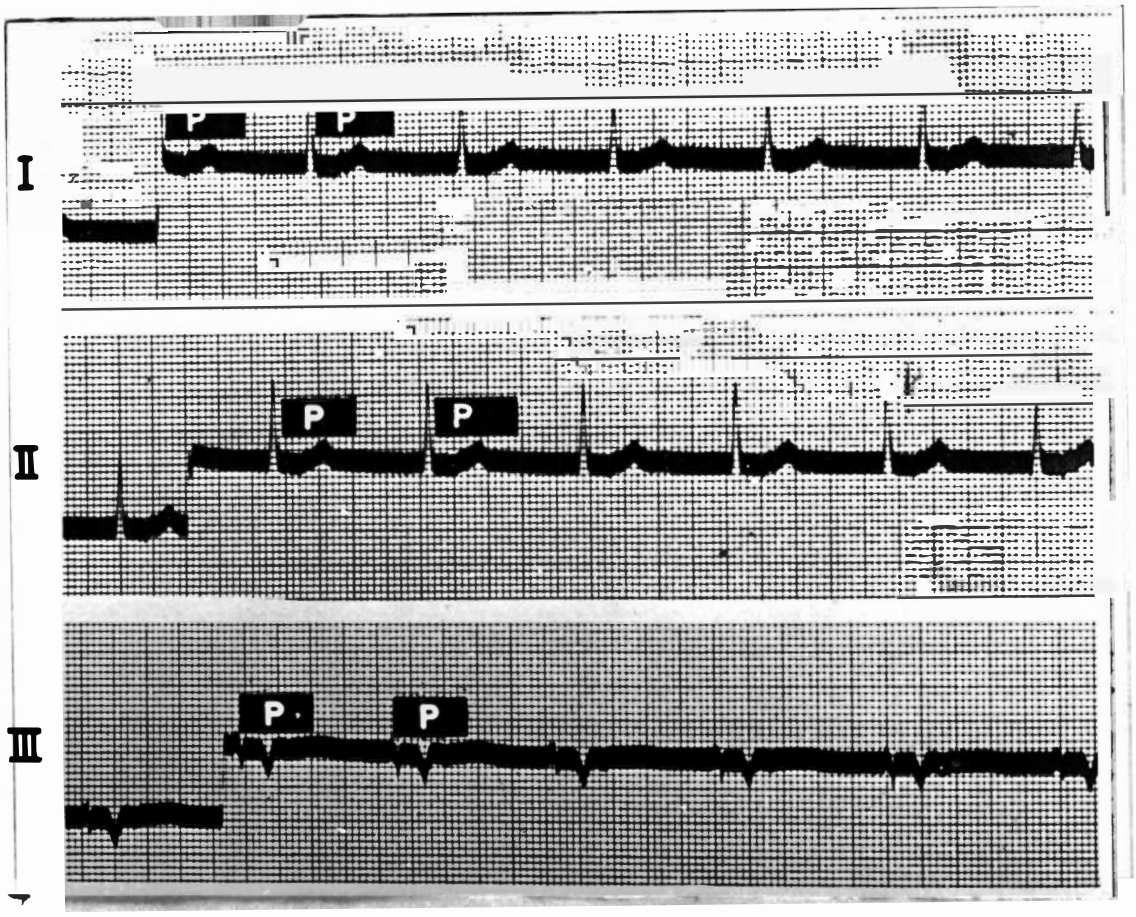

Figure 17. EKG Showing A-V Nodal Rhythm.

Summary: Rate regular--70. The P wave follows the QRS by 0.18 second and appears as notch on S-T segment in leads I and II and as a peaked inverted wave in Lead III. The auricular complexes (P) are retrograde in nature.

From Electrocardiography (21).

Mechanism explained on page 74.

node and is transmitted to the ventricles and in retrograde fashion to the auricles. The auricular contractions, represented by abnormal P waves, may be lost in the QRST complex, as in the nodal extrasystole. Occasionally, however, they may be detected. The ventricular complex usually has a normal countour (29).

Diagnosis: Diagnosis is made by E K G findings as mentioned above. Auricular fibrillation with complete heart block may be ruled out by using chest lead. See figure 17.

Treatment: the symptomatology of paroxysmal tachycardia of nodal origin is nowise different from that of auricular origin. The prognosis is also equally benign. In cases of persistent nodal rhythm atropine will abolish the type associated with vagal depression; ephedrine may also be used. If organic reasons are present treat accordingly. If digitalis intoxication is the cause regulation of this drug should be made (11).

A-V Nodal Tachycardia

Etiology: This condition is very rare. It has the relatively unimportant clinical significance of auricular paroxysmal tachycardia and a mechanism somewhat similar to that of ventricular paroxysmal tachycardia with with regular retrograde auricular response, although in

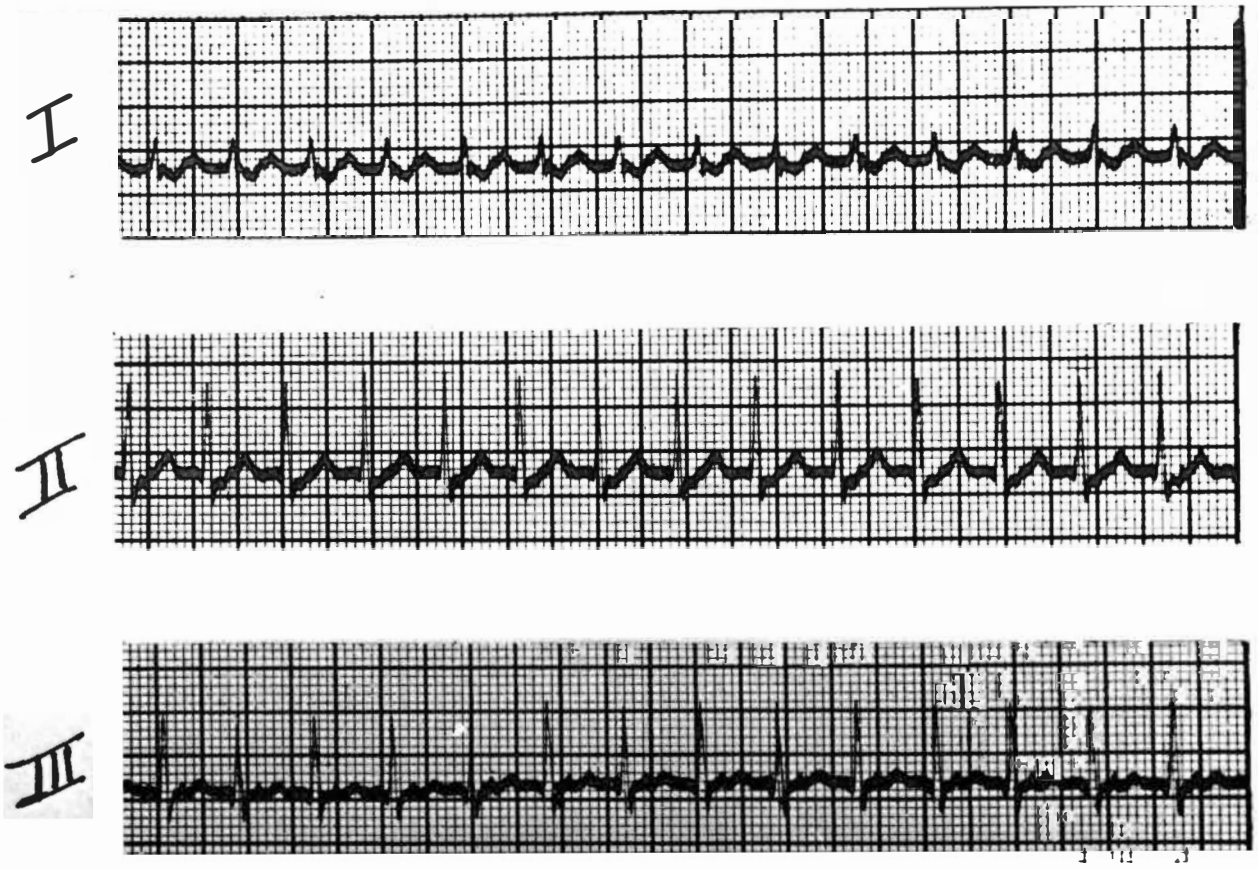

Figure 18. EKG (diag.) Showing A-V Nodal Tachycardia.

Summary: Atypical auricular complexes in R-T segments of all leads. QRS complexes normal. T waves upright in all leads.

From Electrocardiography (29).

Mechanism explained on page 76.

rare instances the auricular contraction may precede the ventricular even when the impulse starts in the auriculoventricular node (41).

When the site of the pacemaker is suddenly shifted from the sinus node to an ectopic focus, which in this case is the A-V node, the rate of stimulus formation is usually rapid. (28).

Diagnosis: Usually the person affected is conscious of the distrubance of rhythm, but in rare cases a paroxysm, long or short, may pass unnoticed except by the observer; this happens in insensitive persons or in individuals who are too ill to appreciate this complication. The general complaint is of a regular rapid palpitation or of a disagreeable sensation of fluttering in the chest in the region of the heart (29).

E K G's are important to determine the site of origin of the tachycardia. As shown in Figure 18, P waves are not visible; they have been lost in the QRST complex because of the rapid contraction rate of this mechanism. Because of this rapid rate the ventricular complex is also slightly changed.

Treatment: None unless there is an associated organic heart lesion.

A-V Nodal Escape

Etiology: Nodal escape is a rather uncommon mechanism. The sinus node is so slow in emitting stimuli to contraction that the ventricle sometimes beats of its own accord. Under these circumstances it is said to "escape" from the influence of the sinus node and the auricles.(15). It occurs during retardation of the sinus rhythm in the slow cycles of sinus arrhythmia, following sinus standstill during the pause after a premature systle,and in the intermittency of S-A block, and partial A-V block when the A-V node may take over control of the heart for one or several beats(27).

Diagnosis: This condition is of no significance, and is asymptomatic. It is diagnosed in the electrocardiogram when after a pause longer than normal there appears a ventricular complex which is normal in configuration, but differs slightly from the ventricular complexes of the sinus beats, in showing varying degrees of aberrant conduction. This complex either has no P wave in front of it to the sinus P waves, but so shortly before it hat the P-R interval is less than 0.12 second. When the sinus rhythm is regular, projection of the spacing of the P waves will identify the one occurring around the time of the nodal escape(21).

Treatment: This condition, alone, exemplifies physiological heart action, therefore no treatment.

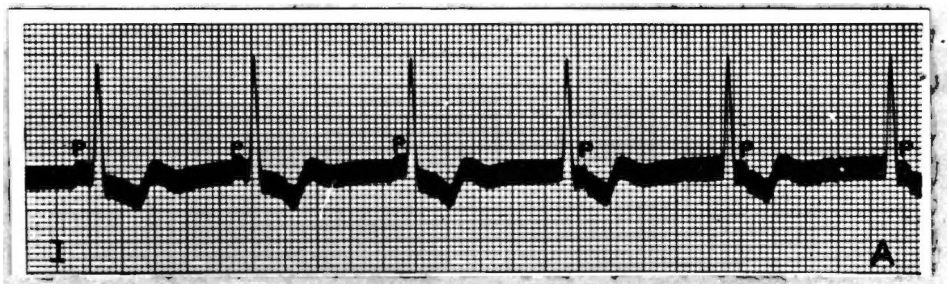

Figure 20. EKG Showing Idioventricular Rhythm.

Summary: The P waves show no relation to the QRS deflections. Long R-R interval indicating independent ventricular focus.

From Diseases of the Heart (16).

Mechanism explained on page 79.

Idioventricular Rhythm

Etiology: If the ventricular rhythm is controlled entirely by the A-V node, the condition is spoken of as idioventricular rhythm. The pacemaker for this rhythm may be in the His-bundle or below it. The rate in either is usually between 30 and 40 beats per minute but may be faster. Idioventricular rhythm is seen most commonly in complete auriculoventricular heart block, less often in certain of the parasytolic rhythms(16).

Diagnosis: If the pacemaker is below the bundle of His the QRS is markedly aberrant ("ventricular rhythm"). Theauricles may show a regular sinus, auriculoventricular nodal, or circus rhythm. In either case the auricular and ventricular complexes are unrelated. The positive diagnosis is made only by electrocardiogram. See Figure 20.

Treatment: That of the underlying heart condition.

Ventricular Escape

Etiology: The mechanism is the same as that in the last named condition (Idioventricular Rhythm), except that the A-V node initiates only occasional beats.

From a standpoint of anatomis diagnosis, no conclusions have been drawn. It holds no serious prog-

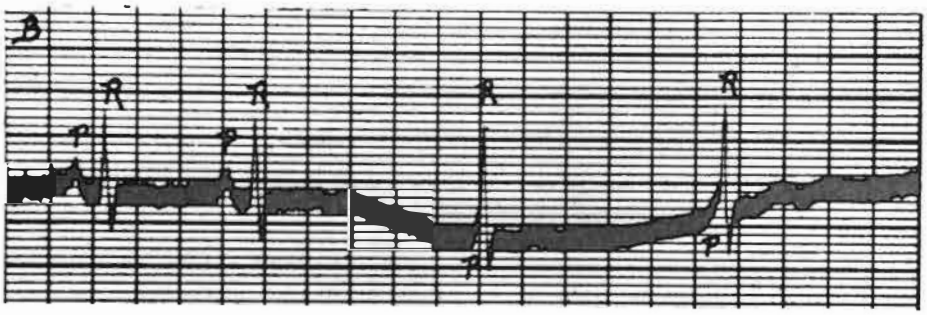

Figure 21: EKG Showing Ventricular Escape.

Summary: Sinus Arrythmia indicated by difference in R-R intervals. Independent beating of ventricles in last 2 segments indicated by no definite P wave before QRS complex.

From Heart Disease (41).

Mechanism explained on page 79.

nostic significance as it is usually due to hypertonicity of the vagus nerve depressing the sinus node. However, lowered vascular supply to the sinus node or inflammatory damage to the pacemaker may produce this mechanism (27).

Diagnosis: Is made by the E K G which shows ventricular complexes not associated with the P wave of the auricular complexes. See Figure 21.

Treatment: That of the accompanying of underlying lesion.

Ventricular Premature Systole

Etiology: In this mechanism the site of impulse formation is displaced from the sinus node into the ventricular muscle. There is a growing tendency among students of heart disease to regard extrasystoles with more care than in previous years. For example, a patient who has sustained an acute coronary thrombosis and develops frequent extrasystoles of ventricular origin sometimes develops a ventricular tachycardia from the same irritable focus. That patient with a hypertensive vascular disease develops ventricular extrasystoles coincidentally with mild congestive failure. Ventricular extrasystoles sometimes bring the patient with coronary sclerosis to the physician before angina

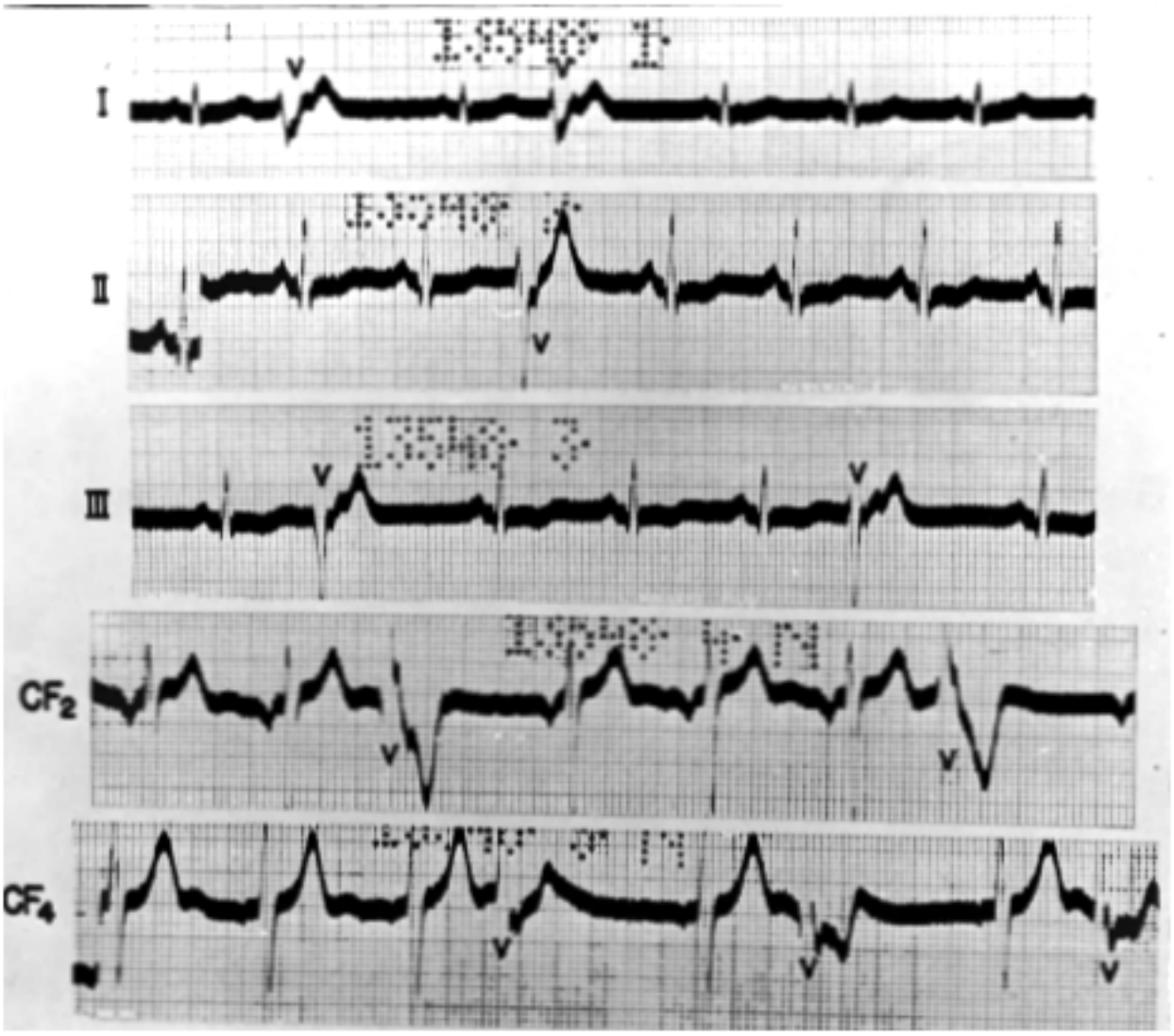

Figure 22. EKG Showing Ventricular Premature Systole.

Summary: Premature systoles (V) resembling each other in each lead is presumptive evidence of single ectopic focus. QRS inverted in limb leads and inverted in chest leads. Sinus P wave occurs at irregular intervals and causes notching of the S-T-T of the premature systoles.

From Electrocardiography (21).

Mechanism explained on page 83.

is a manifest symptom (5).

Diagnosis: Made by the E K G. The cardinal diagnostic sign of ventricular premature systoles is the presence of a premature bizarre QRST complex which is not preceded by a premature P wave and whose QRS complex is prolonged beyond the duration of the dominant QRS complexes (21). See Figure 22.

Treatment: Persons with this condition should have occasional check-ups by means of the E K G. The condition, in itself, is of no consequence, but cardiac conditions, as enumerated above must be watched for (27 .

Ventricular Tachycardia

Etiology: Ventricular tachycardia is essentially a regular succession of extrasystoles arising from an ectopic focus in the ventricular muscle. The impulses to contraction arise from this ectopic focus, and travel a retrograde pathway through the ventricular muscle. The regular auricular complexes are lost in the bizarre QRST complexes. The rate is usually rapid and the rhythm regular. The transposition to this focus is usually sudden and its reposition is also as sudden, hence it is a paroxysmal phenomenon (5

The known causative factors producting this mechanism are few. It is most frequently demonstrated as

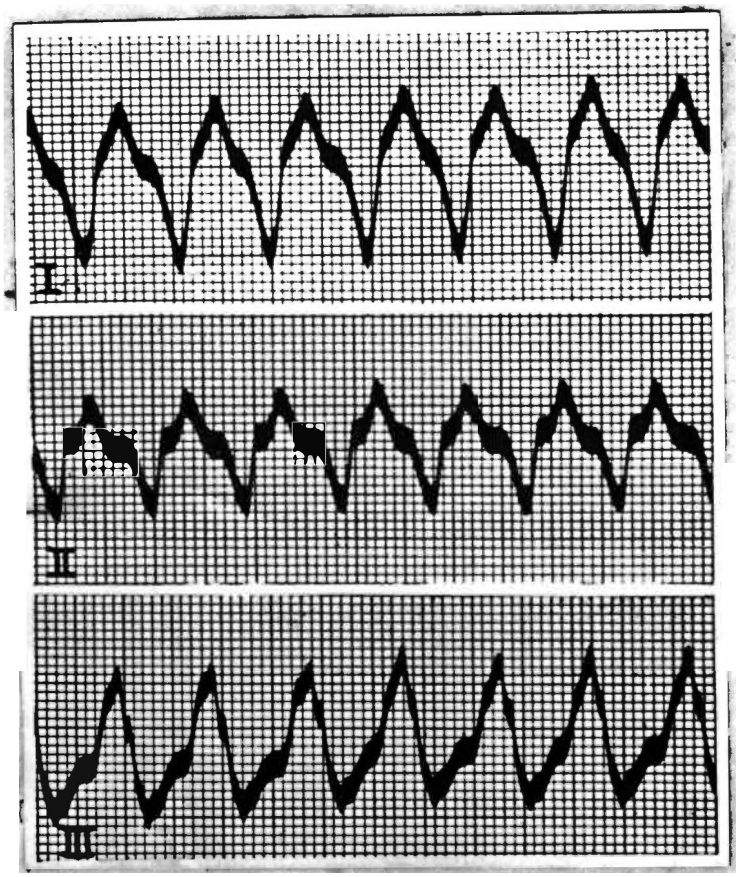

Figure 23. EKG Showing Ventricular Tachycardia.

Summary: Large bizarre ventricular complexes with loss of auricular complexes.

From Diseases of the Heart.

Mechanism explained on page 85.

a complication of an acute coronary thrombosis. It may also be produced by drugs which will sufficiently intoxicate the ventricular muscle, such as digitalis, squills or quinidine(11 .

Diagnosis: Primarily by E K G. The ventricular complexes are bizarre, are not quite evenly spaced, and may differ from one another in countour; pauses are seen during which sinus beats occur. The regular auricular complexes are lost in the bizarre QRST complexes (29). See Figure 23. The prognosis in this form of paroxysmal tachycardia is serious. It is more difficult to stop than the supraventricular type. Death is apt to occur from cardiac exhaustion after protracted attacks even in apparently normal individuals. Ventricular paroxysmal tachycardia is rarely unassociated with heart disease and the rapid rate adds a heavy burden to any already diseased myocardium. There is also the hazard that a with coronary disease and paroxysmal ventricular tachycardia may develop a fatal ventricular fibrillation.(36).

Treatment: Quinidine sulfate by mouth in full therapeutic doses.

Ventricular Fibrillation

Etiology: The mechanism of ventricular fibrillation is not definitely established. According to Katz (21)

four alternative theories have been advanced and depending on circumstances each may actually operate. These are:

"1. Extremely rapid regular beating of a single ectopic pacemaker--a parasystole;

2. Multiple rapidly beating ectopic pacemakers in competition with each other;

3. Continuous multiple reentries from several points, originating as off-shoots of a single initial impulse, and

4. A single, though somewhat irregular, self-perpetuating "mother circus ring" originating as an off-shoot of a single initial impulse."

In any event this condition is a terminal event in many disease entities during the last few moments of life. It may be produced by intoxic tion with many drugs such as caffein, digitalis, squills, and quinidine, and experimentally in animals by faradization of the ventricle. When ventricular fibrillation is initiated the circulation is so seriously impaired that death soon ensues (29).

Diagnosis: Implied when patient has feeble heart sound, thready or absent pulse and shows cyanosis. The E K G shows a series of rapidly occurring QRST complexes of bizarre type, each one different from its fellows(16). See Figure 24.

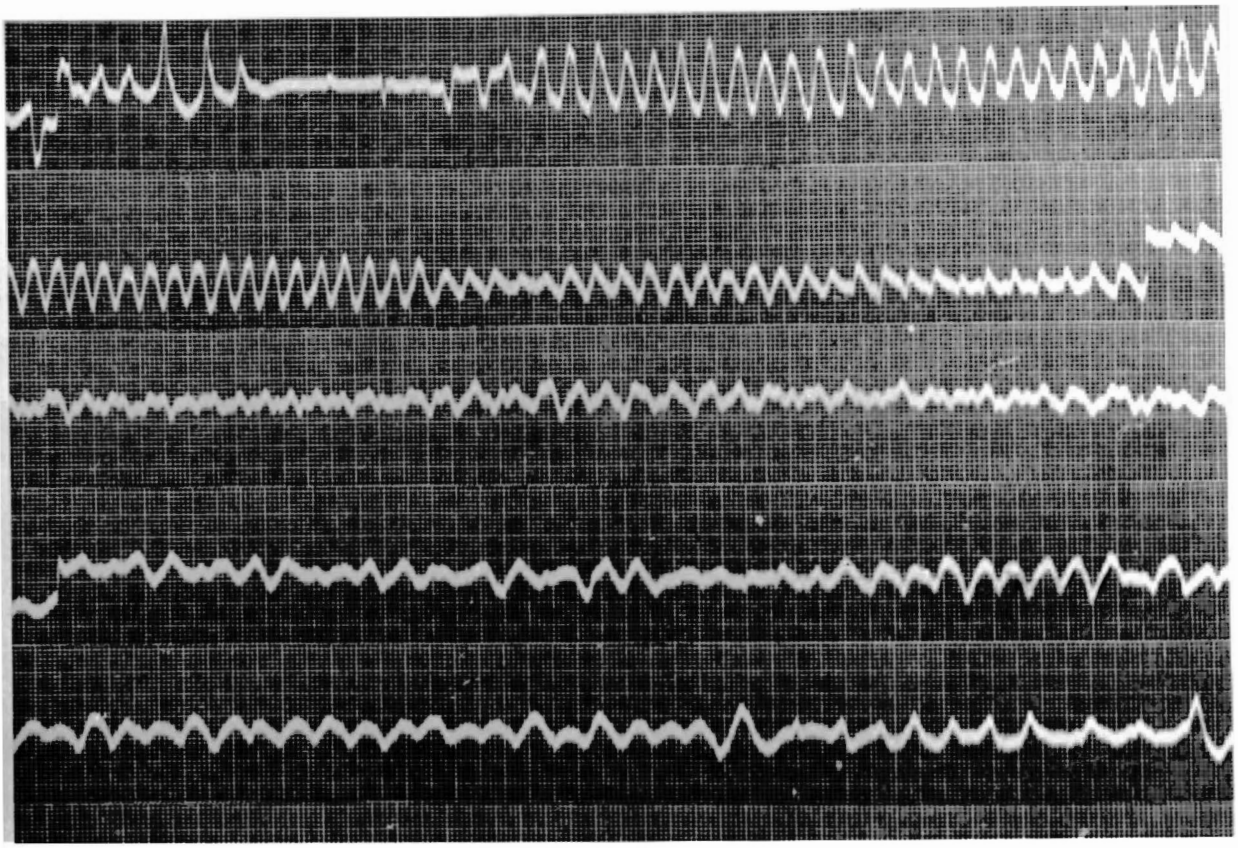

Figure 24. EKG Showing Ventricular Flutter and Fibrillation.

Summary: Continuous record of lead III. Shows paroxysmal ventricular flutter and fibrillation.

From Electrocardiography (21).

Mechanism explained on page 86.

Treatment: If the condition is due to depression by drug, this drug should be removed immediately. If the condition is due to organic disease the treatment must be according.

MECHANISMS OF DISTURBED IMPULSE CONDUCTION

Sino Auricular Block

Etiology: Sino-auricular block is a relatively rare condition in which neither the auricles nor the ventricles are activated by the normal pacemaker because the impulse is prevented from leaving the sinus node. S-A Block may lead to an occasional dropping out of one beat or to a definite regular or irregular intermittence of such beat omission. Thus once every fifth, fourth, third or second beat the heart will not be stimulated. More often, the dropped beats occur irregularly. On rare occasions two or three beats may be dropped out in succession (21).

The condition is due to increased vagal tone, most commonly the result of digitalis intoxication, less often appearing during acute infections or following quinidine.

Diagnosis: The patient may be aware of an occasional "skip beat", otherwise there are no symptoms. In the E K G all the deflections are dropped out for one or more beats, the pauses representing multiples of the normal

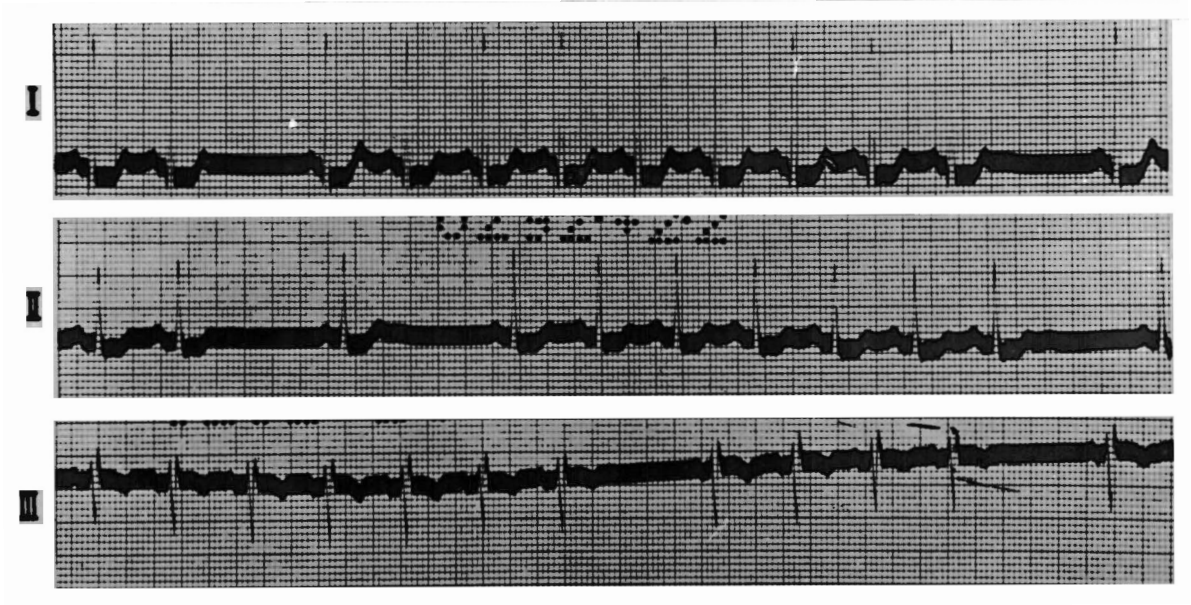

Figure 25. EKG. Showing S-A Block.

Summary: Record shows occasional dropping out of auricular and ventricular complexes, twice in leads I and III and three times in lead II. Mechanism diagnosed by finding that the P-P intervals bounding the pauses are almost exactly twice that of the usual P-P intervals.

From Electrocardiography (21).

Mechanism explained on page 90.

P-P interval. See Figure 25.

S-A block must be distinguished from other forms of intermittency, especially from intermittent A-V block, early premature systoles, and blocked auricular premature systoles. Examination of the jugular pulse in S-A block will show complete absence of all pulsation during the pause, whereas in A-V block the regularly spaced auricular waves can be made out, and in premature systoles a premature wave or group of waves will usually be detectable. S-A block must also be distinguished from marked sinus slowing where the change in rate is rather abrupt and from sinus standstill. As a matter of fact, S-A block is almost always an electrocardiographic diagnosis (17).

Treatment: If the condition is due to digitalis poisoning, this drug should be curtailed. If the condition gives rise to symptoms because of long pauses, atropine and ephedrine may be used.

Intra-Auricular Block

Etiology: This condition is a probable sign of general myocardial involvement, since it is unusual for auricular damage to develop alone. Unless caused by digitalis, it is a sign of organic heart disease. As the name implies, the slowing of conduction in the

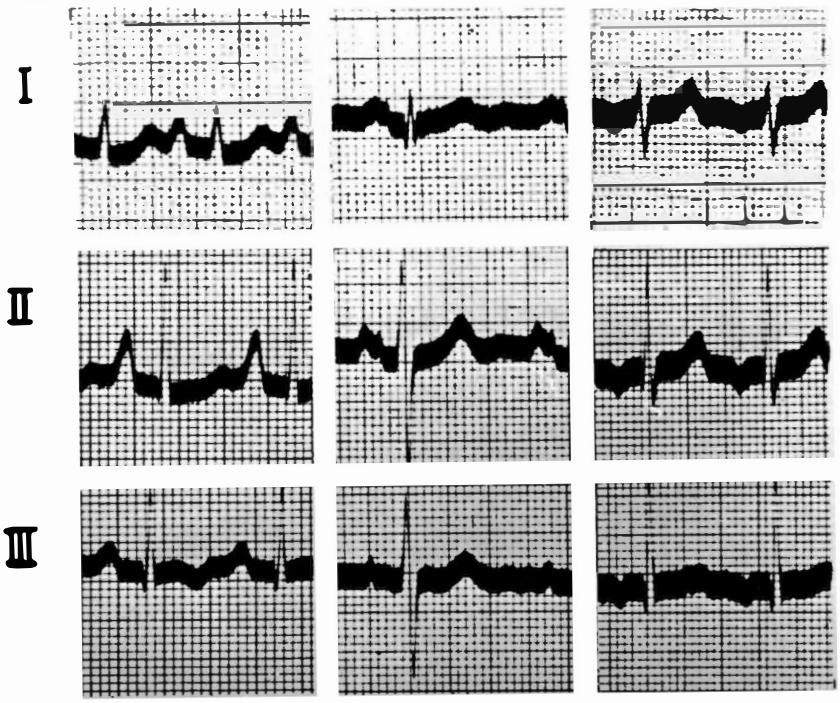

Figure 26, EKG Showing Intra Auricular Block.

Summary: Diagnosed by broad tall, notched or otherwise abnormal P waves appearing in the dominant rhythm and having P-R intervals longer than 0.12 second--as above.

From Electrocardiography (21).

Mechanism explained on page 92.

auricles is due to the organic lesion.

Diagnosis: May be identified by the E K G only. It should be diagnosed whenever the P wave is broad, and especially when at the same time it is both notched and tall, and when the P wave is definitely outside the lower limit of normal duration (0.12 second). A broad P wave should be considered the sign of intra-auricular block just as a prolonged QRS complex, its ventriular homologue,is considered the evidence of intraventricular block (27). See figure 26.

Treatment: That of the underlying organic lesion.

Auriculo-Ventricular Block

Etiology: This is a very common form of block and may be temporary, intermittent or permanent. It occurs in three chief forms:

1. Prolonged A-V conduction in which the A-V transmission time is prolonged but every impulse reaches the ventricles. This is commonly called first degree A-V block.

2. Partial A-V block (seonnd degree A-V block) in which some of the auricular impulses fail to reach the ventricles. The degree ofblock can vary from one preventing conduction of very few auricular impulses to one stopping almost all impulses (almost complete A-V block).

Partial A-V block can give rise to regular, periodic, or irregularly spaced blocked impulses. The commonest form of regular partial A-V block is the 2:1 A-V block, in which every other auricular impulse is blocked.

3. Complete A-V block in which none of the auricular impulses reach the ventricles. The heart is thus controlled by two pacemakers, the auricles by the sinus node and the ventricles by an ideaventricular pacemaker, apparently located just below the block in that ectopic focus having the fastest inherent rate of discharge. This type of block may be called third-degree A-V block 44).

Lesser grades and temporary forms of A-V block are caused most often by digitalis intoximation (rarely by quinidine), by acute infections, especially rheumatic fever, diphtheria, influenza, scarlet fever and other streptococcal infections. It also occurs in uremis and in anoxemis, expecially when there is coronary disease. Rarely, it may be caused by reflexes operating over the vagi, and it can be induced by carotid sinus pressure. Severe temporary forms of block may be caused by any of these conditions, but are more common in acute coronary closure.

The severe chronic forms occur especially in older persons and are caused more often by a degenerative pro-

cess such as follows coronary disease, by chronic inflammation, especially in rheumatic heart disease, or by syphilitic involvement of the mouths of the coronary arteries (8).

Diagnosis: Symptoms are uncommon. Palpitation is sometimes present in sensitive patients. When intermittence is present, the symptoms resemble those seen in premature systoles. In slow ventricular rates, the patient may be unpleaseantly aware of the vigorous, slow, forceful beats. Giddiness, faintness to complete unconsciousness, with muscular twitching or convulsions occur in the severer forms of heart block, this is known as the Adams-Stokes syndrome (39).

E K G is always necessary to confirm the diagnosis. This is made on finding a P-R interval longer than 0.21 second, or one of more P waves which are not conducted to the ventricle. However, prolongation of the P-R interval or blocked out P waves of other etiologies must be excluded; such causes operate as the result of interference. This in auricular premature systoles, paroxysmal tachycardia, auricular flutter (where F waves may be mistaken for P waves) auricular beats following ventricular and nodal premature systoles, and in the auricular beat occurring at the time of nodal escape, this mechanism appears and leads to either pro-

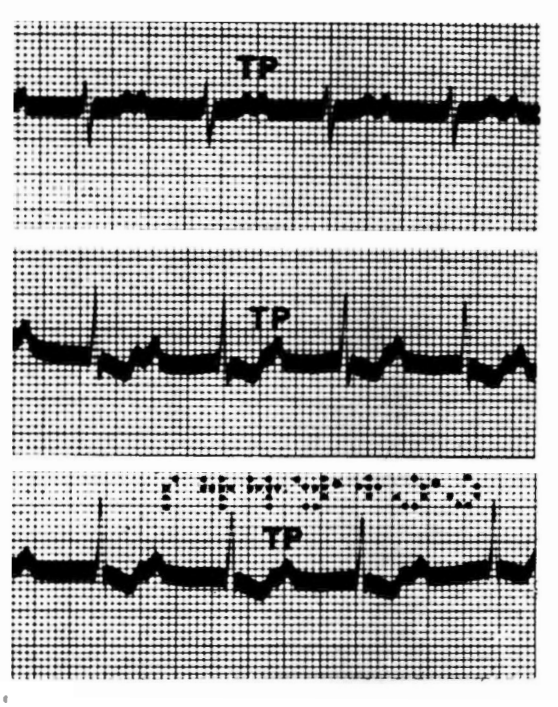

Figure 27. EKG Showing Prolonged A-V Conduction Time (1st Degree Block).

Summary: All sinus P waves are followed by QRST complexes but the conduction time is long.

From Electrocardiography (21).

Mechanism explained on page 94.

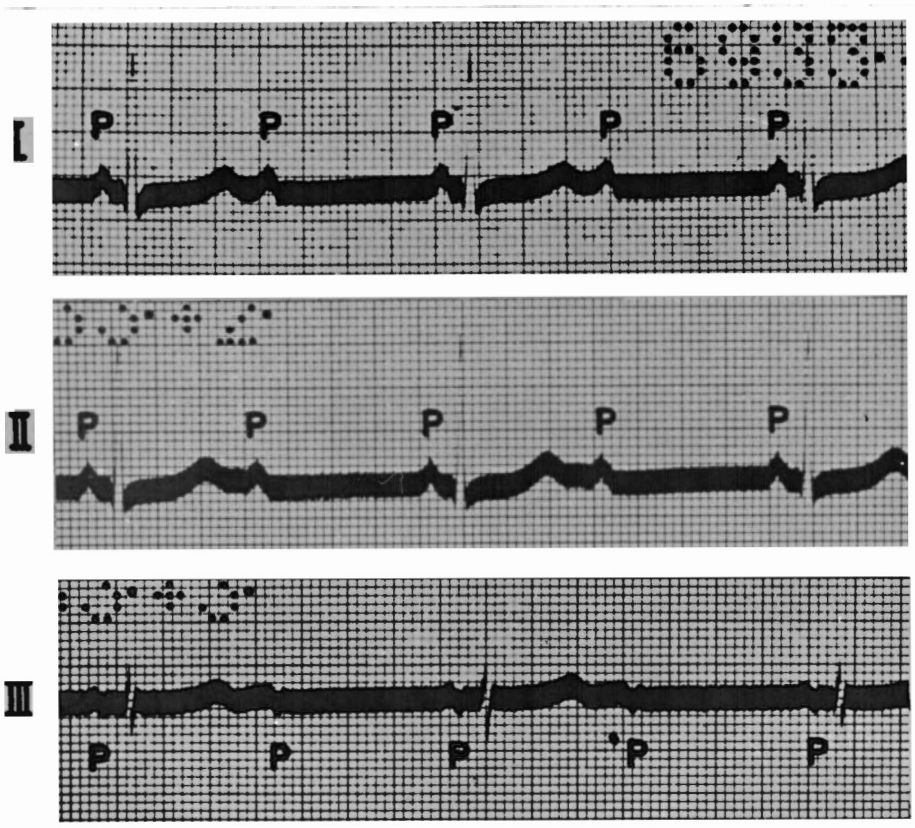

Figure 28. EKG Showing Partial A-V Block (2nd Degree).

Summary: Isolated P waves not followed by QRST complexes. In this illustration there is a 2:1 block since there are two P waves to each QRST complex.

From Electrocardiography (21).

Mechanism explained on page 95.

longed P-R intervals or blocked out P waves, or both. In all of these instances, the blocked P wave or prolonged P-R interval is due to the fact that the auricular impulse reaches the A-V juntional tissue during its normal relative or absolute refractory phase(21).

In first degree A-V block (Prolonged A-V Conduction) is present when all the sinus P waves are followed by QRST complexes but the P-R interval is prolonged beyond 0.21 second (Figure 27).

In second degree A-V block (Partial) the sinus P wave is not followed by a QRSTcomplex, and when this blocking cannot be explained on the basis of the interference which follows a premature nodal or ventricular systole or a nodal escape. The dropped beat may be an occasional one or it may occur frequently. The intermittence is defined by the ratio of P waves to QRST complexes; i.e., 7:6, 6:5, 5:4, 4:3, 3:2, 2:1, etc. See Figure 28.

In third degree A-V block (Complete) none of the auricular impulses reach the ventricles. The heart is thus controlled by two pacemakers, the auricles by the sinus node and the ventricles by an idioventricular pacemaker, apparently located just below the block in that ectopic focus having the fastest inherent rate of discharge. See Figure 29.

Treatment: If the condition is caused by digitalis or quinidine intoxication these drugs should be curtailed. If there is an associated organic condition, treatment should vary accordingly(29).

Intra-Ventricular Block

Etiology: Katz states "In view of the arbitrariness and practical unimportance of attempts to identify intra-ventricular block as due to left and right bundle involvements, we have made it a practice to classify intra-ventricular block in a manner which avoids localization. A localizing classification must be deferred until the time when there is a relative agreement as to the findings. If it is desirable to decide whether the block is chiefly in the right or left ventricle, the asynchrony in the activity of the right and left ventricle should be determined. This is an unnecessary procedure for ordinary purposes and is not always satisfactory."

Katz and his associates have therefore designated the intra-ventricular blocks by new terminology. The type which has been known as the left bundle-branch block is called the "common" type", that which has been known as the right bundle-branch block is called the "uncommon type", all others are known as indeterminate types(21).

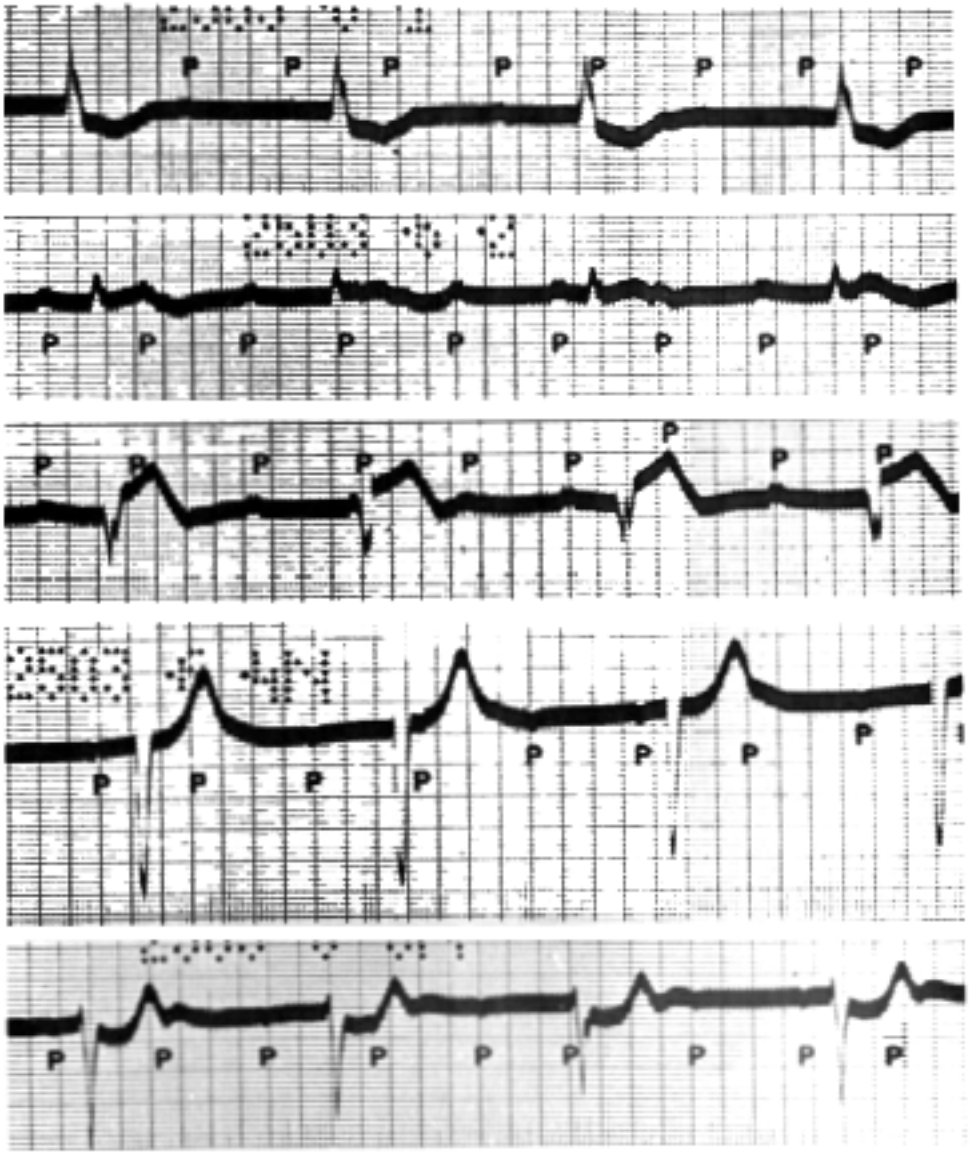

Figure 29. EKG Showing Complete A-V Block (3rd Degree)

Summary: Ventricular rate 34- regular. P waves show no relation to QRST complexes--i.e., they are irregularly spaced.

From Electrocardiography (21).

Mechanism explained on page 94.

Transient intraventricular block occurs in acute heart failure, recent myocardial infarction, acute coronary insufficiency, digitalis or quinidine excess, and acute infection. Its presence in diphtheria is a sign of ill omen(2).

The chronic form occurs most often in coronary sclerosis, less often in syphilitic and rheumatic heart disease. Clinically there are no c rtain signs of its presence, but gallop rhythm (presystolic or protodiastolic) is not uncommon, and a split apex beat may be present. (18).

Diagnosis: By EKG

In the common type, the QRS complex is upright in lead I and inverted in lead III and the amplitude of the QRS complex in these leads is more than 5 millimeters. In mostinstances the S-T segment and T wave are deviated in a direction opposite to the QRS complex of the lead. Lead II may resemble lead I or lead III. The chest leads usually resemble lead III, although on occasion they do not, especially (21). See Figure 30.

In the uncommon type, the QRS complex is inverted in lead I and upright in lead III, while the amplitude of the QRS complex in these leads is more than 5 millimeters. The S-T segment and T wave contour are variable, but in general the direction of their deviation is opposite to that of the major QRS phase. The QRS com-

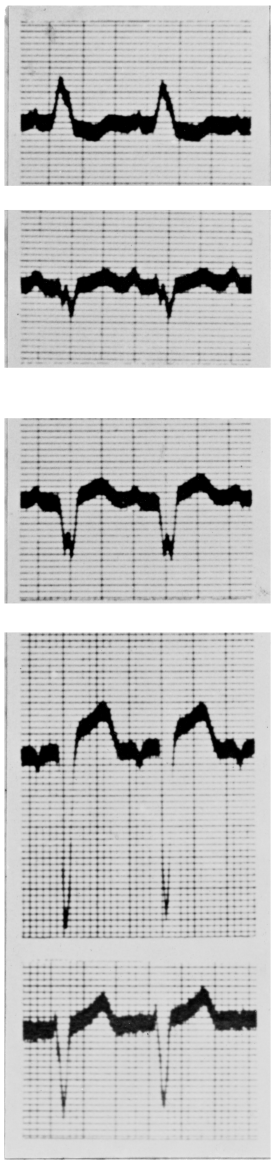

Figure 30. EKG Showing Uncommon Variety of Intra-ventricular (Left BranchhBundle) Block.

Summary: QRS upright in Lead I and inverted in Lead III. QRS segments notched. In CF_{21}, the QRS in down and prolonged, the ST is abnormally elevated and the P inverted.

From Electrocardiography (21).

Mechanism explained on page 100.

plexes in the chest leads are bizarre, notched, and may be polyphasic and M or W shaped; the T in CF_2 often may be inverted. See Figure 31.

All other forms of intra-ventricular blocks are classified under the general heading of "indeterminate types". These are too numerous, and their minor deviations, as shown by the EKG, are not of significant importance to mention here (21).

Treatment: That of the underlying organic disease.

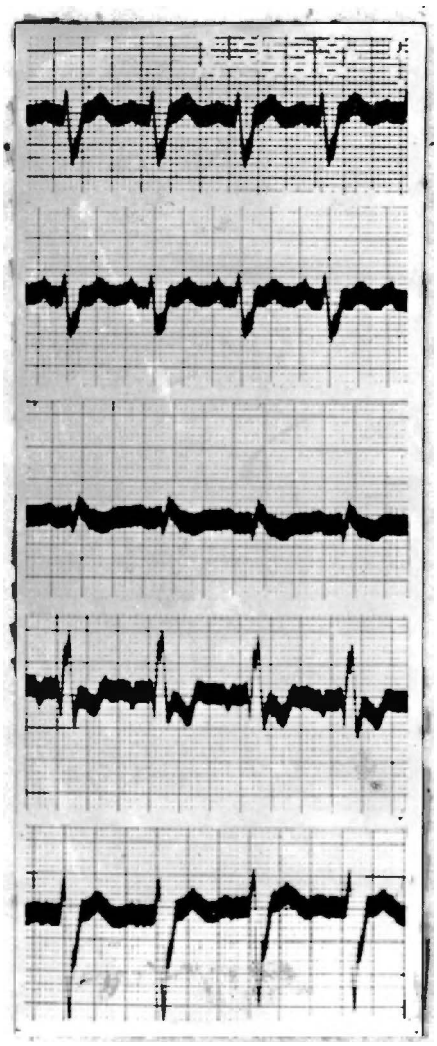

Figure 31. EKG Showing Uncommon Type (Right Branch Bundle) of Intraventricular Block.

Summary: Prolonged QRS throughout. QRS has amplitude of over 5 mm. in leads I and III; it is inverted in lead I and upright in lead III. In CF_2 the QRS is small, prolonged, slurred and W shaped.

From Electrocardiography (21).

Mechanism explained on page 100.

CONCLUSIONS

I. Cardiac rhythm depends on the site of origin of the impulse to contraction and upon the pathway it travels.

2. In the normal physiologic mechanism all stimuli arise from the sinus node and travel the normal pathway through the A-V node, main bundle of His, into the right and left branches and into the arborizations of the Purkinje system and the ventricular musculature.

3. Stimuli to contraction may occasionally arise in an ectopic focus----in this case the arrhythmia is often considered within physiological limits, but is considered pathological when it is evidence of toxicity, from drugs or otherwise, or when it is caused by an underlying organic lesion.

4. The heart is under the "external" influence of the vagus nerve and of sympathetic fibers. Vagus stimulation acts as a "brake" upon the heart, while sympathetic stimulation accelerates the heart rate. Various drugs may be used to enhance the action of either of the above systems. Most widely cardiac "stimulants" are ephedrine, atropine, and caffeine, while the "inhibitants" most used are quinidine and digitalis.

5. While many of the arrhythmias may be diagnosed by ordinary procedures, the EKG is an invaluable aid in the final analysis----in fact is often the only means of positive identification.

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