

THE VELOCITY OF THE BLOOD FLOW IN
CARDIOVASCULAR DISEASE AS CLINICALLY
ESTIMATED BY MEANS OF SODIUM CYANIDE

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by

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INTRODUCTION AND REVIEW OF THE LITERATURE

The velocity of the Blood Flow as defined by Blumgart⁽¹⁾ is the time required for a certain length of fluid column to pass to a given point, or conversely the time a certain cross-section of fluid takes to flow a definite distance.

The mean velocity of a stream of fluid through a rigid tube is directly proportional to its cross-sectional area, and the difference in pressure from point to point. According to a physical law - that of Poiseuille - the product of the cross-sectional area multiplied by the pressure head, and divided by the coefficient of viscosity gives the velocity of flow of the fluid. Such a physical law cannot however be applied to an estimation of the velocity of the blood flow in the animal body, owing to the constantly changing conditions present in the vascular system. The state of the peripheral vascular bed is in a constant state of flux. As Krogh and others have shown, certain capillaries may temporarily be partially or completely closed.

Peripheral vaso-dilatation by lowering resistance tends to increase the velocity of the blood flow, as the latter varies inversely as the resistance. On the other hand, by increasing the cross-sectional area of the flowing stream, the same factor tends to decrease the velocity.

This and other factors, termed by Clifford Albutt 'Vasomotor Breezes', have tended to make the problem of the estimation of the velocity of the blood flow in man complicated, and investigators have been compelled to resort to direct methods in attempting an accurate estimation.

The blood velocity may be measured as the distance covered by a particle of blood in a fixed interval of time, or conversely as the time taken to complete a known distance. From an experimental point of view, it has been found simpler to estimate the time taken, rather than the distance traversed by the particle of blood.

Methods have been devised, by means of which the speed of the circulating blood can be measured over selected pathways. Methods applicable to man, with any claim to accuracy, depend on the rapid injection of a substance into a peripheral vein, and its detection at some more distant part of the body.

The time interval between such an injection, and the arrival of the fastest particle of blood, through the designated portion of the circulation, has been termed the circulation time for that particular pathway. The detection of the arrival of the substance chosen, at a particular point, depends upon some physiological property of this substance such as colour, taste, radio-activity, neuro-muscular stimulation or vaso-dilator effect.

The rate of the blood flow varies greatly in the different parts of the circulation. The rate is slowest in the capillaries, increasing along the great veins, as the blood approaches the heart. In the arterial system the speed is greatest in the large trunks,

and decreases gradually towards the periphery. In the lungs, there is a separate circulation, which is to a certain extent independent of the systemic circulation. The term 'Pulmonary Circulation time' refers to the time necessary for a given particle to appear in the left auricle, after its previous entrance into the Pulmonary Artery.

The circulation time measured represents the velocity flow of the fastest particle through the shortest path. That slight possible variations in the length of the larger vessels might alter the circulation time significantly, is opposed by the findings of certain observers. The contention that the circulation time can be appreciably altered by changes in the number of available capillary pathways through the lungs, has been met by Blumgart and Weiss. These observers emphasize the constancy of their findings in the same persons on different days, and conclude that such vasomotor effects, if present, are not of sufficient importance to alter the significance of the results.

The clinical significance of the use of the circulation time as a measure of the mean velocity of blood flow has been accepted.

In the selection of a method, clinically suitable for the estimation of the circulating time in normal and pathological states, a review of the literature is essential. Each of the different methods, which have been described for estimation of the velocity of the blood flow, offers certain advantages, and each possesses limitations, which must be considered.

In the present investigation the method first described by

Robb and Weiss, whereby the stimulant properties of Sodium Cyanide on the respiration are utilised as a signal reaction was chosen. The advantages and limitations of the method will be discussed later. An adequate flow of blood to the tissues implies inter alia an adequate speed of transportation of the blood to the sites of utilization. This aspect - the velocity of the blood flow - has received somewhat meagre attention in the past, due chiefly to the technical difficulties involved. From a survey of the available literature, it is evident that sufficiently accurate methods of estimating the velocity of the blood flow in man have only comparatively recently been devised. These methods, in fact, have been involved mainly during the last decade.

Harvey, whose discovery of the circulation of the blood was made in 1628, wrote: "The circuit of the blood is accomplished now more rapidly, now more slowly according to the temperament, age, etc. of the individual, to external and internal circumstances, to naturals and non-naturals, sleep, rest, food, exercise, affections of the mind and the like." It was not until 1733, however, that the physiologist Stephen Hales, attempted the first estimation of the velocity of the blood flow. He computed the rate of flow in the aorta of the horse. Hering in 1827 studied the circulation time in the horse by injecting a solution of Potassium Ferrocyanide at one point, and estimating its time of arrival in the blood at another point in the vascular circuit. He tested samples of withdrawn blood by the Prussian Blue reaction, and found that the substance required 26.2 seconds to pass from one jugular vein to the other jugular vein.

In 1850 Volkman constructed a pendulum device by which he attempted to gauge the circulation time in the horse.

In 1858 Vierordt designed an instrument by which he extended his observations to other animals - rabbits, cats, and dogs. He found that the velocity of the blood flow over analagous paths in different animals, bore an inverse relationship to their size, that is to say, the smaller the animal the greater the velocity. He also observed that the circulation times were related to the pulse rates. He inferred that the circulation time in man from one external jugular vein to the other external jugular would occupy 23.1 seconds if the heart rate were 72 beats per minute. Repeated attempts followed to measure the circulation time in the arteries and veins, by means of mechanical devices such as manometers. These attempts failed.

In 1894 G.N.Stewart attempted to gauge the velocity of the blood flow by a conductivity method. A hypertonic solution of sodium chloride was injected intravenously into animals, and its time of arrival in another vessel determined by means of electrodes and a sensitive galvanometer.

This method is not applicable to man. Stewart also utilized Methylene Blue injections - observing the arrival of the dye in the common carotid artery by transillumination. He studied the circulation time over many pathways in various animals, and also in different organs. In dogs, he found that the circulation time from the right ventricle to the aorta, varied between 1.7-8 seconds, according to the size of the dog used.

Bornstein, in 1912, attempted to measure the circulation time in man by a simple procedure. He caused patients to inhale a mixture of air and 5.7% carbon dioxide. As soon as the arterial blood carrying the increased amount of carbon dioxide arrived at the respiratory centre, there followed an increase in the depth of the respiration. The circulation time was taken as the time interval between the inspiration of the mixture and the first deep respiration. This method is of limited applicability, as difficulty in detecting the amplified respirations will occur when applied to patients suffering from dyspnoea - a condition in which the estimation of the circulation time is of value.

Koch, in 1922, described a method of measuring circulation time by means of a dye. This method consisted in the injection of 1 c.c. of a 1.6% solution of fluorescin into the cubital vein of one arm, and then collecting samples of blood every 5 seconds from the corresponding vein in the other arm. The moment of appearance of the dye determined the circulation time. The dye traversed the veins to the right ventricle, the pulmonary circulation, the left chambers of the heart, aorta, arteries of the arm, the peripheral capillaries of the arm, and finally the selected vein. Technical difficulties in this method were to detect faint traces of Fluorescin in the blood. Other drawbacks, e.g. the necessity for two venepunctures, and frequent sampling of the blood, interfere with the trustworthiness of the method. Despite these difficulties, the general behaviour of the blood circulation time was fairly accurately estimated by Koch in normal subjects and in pathological conditions. In 51

normal subjects between the ages of 15 and 79 years, he found that the average circulation time was 20.4 seconds.

Klein and Heinen, in 1929, described a similar method to the above - using Congo Red in place of Fluorescin. The same objections hold good with this method as with the method of Koch.

Hirschon and Mandel, in 1922, suggested the use of calcium chloride intravenously to determine the circulation time. 5 c.cs of a 10% solution of calcium chloride were rapidly injected intravenously. A feeling of warmth and a sensation of heat followed the course of the drug throughout the body. This sensation was taken to indicate the arrival of the drug at various neuro-muscular junctions. These observers used this method in an attempt to study the blood velocity through the pulmonary circulation in patients in whom an artificial pneumothorax had been therapeutically induced.

Kahler, in 1930, employed the same technique and drug to estimate the circulation time to the head, neck, hands, and feet. The calcium chloride method, although an improvement on the dye methods, has certain disadvantages viz: danger of venous thrombosis from the drug, risk of tissue slough at the site of injection if leakage of the drug occurs. There is also the possibility of the drug itself affecting the circulation time. A certain number of patients, moreover, failed to respond. On the whole this method is of limited applicability, as the reaction is of an entirely subjective character.

Milldolevi, in 1925, and Koch, in 1928, inserted an electrode through the skin next to the radial artery. A concentrated salt solution was then injected intravenously, and its time of arrival

registered by means of a galvanometer attached to the electrodes.

(2)
Blumgart and Yens, in 1927, described the radium emanation method. A solution of sodium chloride was made radio-active by exposure to radium emanations. Small non-toxic doses of the salt were injected intravenously, and the time of arrival at one or more parts of the circulation was registered by means of a special detector placed over the heart or a suitable artery, and sensitive enough to detect the earliest trace of radium.

Blumgart and Yens at that time summarized the problem as follows: "Theoretically the most desirable measurement of the velocity of the blood flow consists in establishing the separate velocities of each minute portion of the blood along the many separate paths: when one considers that the innumerable vessels in the body are constantly changing in size and elasticity, and that the blood is a suspension of corpuscles in a fluid medium, the impossibility of fulfilling the ideal requirements becomes obvious. The problem is further complicated; any mean velocity measurements, which depend on the insertion of a mechanical device into the blood stream defeats its end, and can, therefore, not be considered for clinical application. The most feasible method appears to be the injection of some substance at one point in the body, and the measurement of the time of its arrival at another point."

They also concluded that the substance chosen should fulfil the following requirements:

- (1) The substance must not be toxic in the amounts used.
- (2) The substance should not be present previously in the body, as estimation of additional amounts of substances already within the body is always subject to error.

- (3) The substance must not in any way disturb the very phenomena under investigation. Toxicity would introduce such an error. The introduction of hypertonic salt solution would also cause an error, as it would alter the blood volume, vary the speed of the blood flow, and thereby modify the very phenomena under investigation.
- (4) The substance should disappear from the body with a sufficient rapidity to allow of repeated measurements.
- (5) The substance must be readily detectable in minute amounts; were this impossible, varying dilutions of the substance would be all the more likely to produce correspondingly variable results.

They concluded that the use of the active deposit of radium yielded a method which fulfilled the foregoing criteria, and permitted clinical application in normal subjects and in pathological states.

Blumgart and Weiss,⁽³⁾ in 1927, estimated the velocity of the blood flow in normal individuals by means of the radium emanation method. The time which elapsed between the injection of the active deposit into an antecubital vein, and its time of arrival in the right chambers of the heart was measured, and termed the arm-heart time; for it was a measurement of the velocity of the venous blood of the arm to the heart. With the detector placed over the right auricle they found the average 'arm-heart time' to be 7 secs.

The time interval between the injection of the substance into an antecubital vein, and its arrival in a corresponding position in the other arm, was termed the arm-arm time. The average time for the radio-active substance to traverse this pathway was found to be 18 secs.

The time taken for the blood to pass through the heart was

estimated at one second. The rate of flow through the arteries was supposed to be twice that in the veins. Therefore taking 18 seconds as the average arm-arm time, and subtracting 7 seconds as arm-heart time, and $3\frac{1}{2}$ seconds for the arterial time, an average pulmonary circulation time of $6\frac{1}{2}$ seconds was obtained.

This method made possible a separate estimation of the rate of blood flow through the pulmonary and peripheral circulation, and for the first time made possible the determination of the circulation time from the site of injection to the right side of the heart.

Blumgart and Weiss concluded that their method was accurate, that considerable variations of the dose of radio-active substance did not influence the results obtained, that the substance injected was non-toxic in the amounts administered, and that no co-operation on the part of the patient was necessary, and that measurements could be repeated after as short an interval as three hours.

These observers obtained measurements of the circulation time also in pathological states. These will be referred to later.

The radium emanation method, while undoubtedly possessing all the properties claimed for it, is unsuitable for general clinical application owing to the bulkiness of the apparatus, expense, and the technical proficiency required.

⁽⁴⁾
Weiss, Robb, and Blumgart, in 1929, showed that following the intravenous injection of suitable doses of histamine in man, the following phenomena occurred:

- (1) An intense flush of the face and neck - this flush was caused by the arrival of the histamine in the small vessels of the face and neck.

- (2) A salty or metallic taste was experienced in the mouth just before or simultaneously with the appearance of the flush.
- (3) The cardiac rate was found to increase at the commencement of the flush, and return to normal with the fading.
- (4) A throbbing headache - presumably due to dilatation of the intracranial vessels, causing stretching of the meninges occurred in some cases; and persisted for varying periods up to a day.

The phenomenon of the flush was used as the signal reaction in measuring the circulation time. The results obtained by this method were found to correspond closely to those obtained by the radium emanation method.

Dyspnoea was found to occur in cases of emphysema, and in the presence of circulatory failure. In cases of severe anaemia, no flush was obtained, and it was concluded that the method was not applicable to cases of coronary thrombosis.

Bain, in 1934,⁽⁵⁾ confirmed the above observations. He used a solution of histamine acid phosphate in doses of .001 mg. per kilo of body weight, and obtained a good flush in just over 50% of his cases. The metallic taste in the mouth he found to be of little value in estimating the circulation time.

The disadvantages attendant on the use of histamine as a reagent were concluded to be -

- (1) Headache, which occurred in 25% of his cases. When this occurred, it prejudiced a second observation, as apprehension tends to cause an outpouring of adrenaline, which by constriction of the vessels might delay the onset of the flush.
- (2) Dyspnoea which occurred in a few cases.

The method was unreliable in cases of thyrotoxicosis, in patients with ruddy complexions, and in those with flushed faces due to nervousness or other causes.

Severe reactions were noted in 3 cases, and 1 death was recorded. The average 'arm-face time' was found to be 22 seconds (with a range 19-25 seconds), in subjects whose hearts, blood and thyroids were clinically normal.

The observations recorded by this observer in pathological states will be referred to later.

The histamine method has the following advantages over the radium emanation method.

- (1) Simplicity. No complicated apparatus or technique is required.
- (2) Cheapness.
- (3) The test can be repeated at intervals of 5-10 minutes if necessary.

It possesses the common advantage of being an objective reaction (if the metallic taste is ignored), not requiring the co-operation of the patient.

Bain while admitting the attendant disadvantages, concluded that the method was accurate, easily performed single handed, and furnished an answer quickly.

Neubauer, in a series of studies on the choleric action of the bile salts, introduced the use of sodium dehydrocholate. This substance was found to be practically the only bile salt, which could be injected intravenously without deleterious effect. A disagreeable side action was the intensely bitter taste experienced after injection of even small amounts of the salt.

Winternitz, Deutch and Brill utilised this property of the drug for determination of the circulation time. They determined the moment of the appearance of the bitter taste, following an intravenous injection of 5 c.cs of a 20% solution of sodium dehydrocholate. They found that the sensation was very transitory, lasting 10-20 seconds only, so that the test could be repeated with the needle in situ. No undue subjective sensations, except occasional nausea, were noted. The drug appeared to have little or no effect on the circulation itself, and no alteration in the heart rate was noted.

Tarr, Oppenheimer and Sagar,⁽⁶⁾ in 1933, published the results of their observations on the circulation time in various clinical conditions, determined by the use of sodium dehydrocholate. A positive response was invariably obtained. 5 c.cs of a 20% solution of the drug were rapidly injected into an arm vein, and the time interval between the injection and the appearance of the bitter taste, measured by means of a stop-watch. The subjects were as far as possible under basal conditions (the test was done fasting - before breakfast), and the patient's co-operation secured.

Occasionally some of their patients experienced nausea, and in a small percentage vomiting followed the injection.

About 1% of their cases experienced pain in right upper quadrant of the abdomen - presumably due to sudden gall bladder distension.

The observers checked their measurements by the use of calcium gluconate as a reagent, and found the value by the two methods in the same patient to be within the bounds of experimental error.

In normal subjects an average circulation time of 13 seconds, with extremes of 10-16 seconds, was obtained. This time agreed with the results of Winternitz and his co-workers.

The sharpness of these normal times was contrasted favourably with the wider fluctuations found with the radium emanation and histamine methods.

The results confirmed what previous investigators had shown: That in normal adults the circulation time bears little or no relationship to the subject's weight, height, pulse rate, or pulse pressure.

The obvious disadvantages of the method are that it is entirely subjective, and depends on the co-operation of the patient. It is of course unreliable in patients, whose sense of taste is impaired.

A fairly large-bore needle is necessary for the rapid injection, necessitating the use of novocaine as a local anaesthetic. The technique elevates the procedure to the status of a minor operation.

The reagent is relatively costly.

The results obtained by Tarr et alii, in pathological states will be subsequently discussed.

(7)
Kremer and Robertson, in 1935, used decholin for estimating the circulation time, and found that it possessed advantages over the histamine method. In subjects whose pulse rates were under 40 per minute, the end point was indeterminable. Under such circumstances the decholin apparently diffuses in the blood stream. The above investigators measured the circulation time in a small series of patients, with raised basal metabolic rates, and their conclusions will again be referred to.

(8)
Fishberg, Hitziz, and King, in 1933, measured the circulation time by injecting intravenously a solution of saccharin. 2.5 grains of soluble saccharin in 2 c.cs of distilled water, were rapidly injected into a suitable arm vein, and the time interval between the injection and a sweet sensation in the mouth measured, the pathway being designated arm-tongue.

The normal arm-tongue circulation time was found to be 9-15 $\frac{3}{4}$ seconds.

This method in common with the decholin method possesses the serious disadvantage of being entirely subjective.

(9)
Hitziz, in 1933, made a further study of the circulation time by the use of ether, η iii with the same quantity of normal saline were rapidly injected into an antecubital vein, and the patient instructed to announce the moment when he smelled the drug, the smell of which he was familiarised with. In addition, an observer smelled the subject's breath and announced its arrival. It was claimed by Hitziz that the end point by this method was sharp and unmistakable.

He found that the normal circulation time in the pathway designated arm-pulmonary capillaries varied between 3 $\frac{1}{2}$ -8 seconds.

This method is to a certain extent dependent on the patient's co-operation, and appears to be only partially objective. A further disadvantage is the occurrence of pain at the site of injection. This was reported to occur in 25% of the subjects tested

(10)
Robb and Weiss, in 1933, published results of their observations on the measurement of the circulation time in normal subjects by the

use of a solution of sodium cyanide in appropriate doses. This drug had been previously administered to man and animals and found to be a safe and effective stimulant of respiration. Its use for measurement of the circulation time in man and animals was first suggested in 1922.

Robb and Weiss measured the time elapsing between the intravenous injection of the cyanide into an antecubital vein, and the appearance of the signal reaction - in this case being an increase in the amplitude of the respiration.

They measured separately the pulmonary and peripheral venous circulation times, by using two or more different sites for injection:

- (1) A peripheral vein of the fore-arm or foot was chosen, depending on the peripheral circulation to be studied. The circulation time from the sites of injection they named arm-carotid time, and foot-carotid time respectively.
- (2) External jugular vein. By injection into this vein a measure of the crude pulmonary circulation time was obtained, but this included a short peripheral venous pathway.

By difference between the two, the venous velocity index was devised, thus providing a practical estimation of the velocity of venous blood flow, but not an exact measure of the circulation time from the point of injection to the right side of the head.

They estimated the arm-carotid circulation time in 35 normal subjects, and in 21 of the 35 subjects the crude pulmonary circulation time.

The following results were obtained in normal subjects

(1) arm-carotid - cyanide reaction time -

| | |
|--------------|----------------------|
| Normal range | 9-21 seconds |
| Average | <u>15.6</u> seconds. |

These figures are in harmony with the results obtained by the radium emanation method in normal subjects - the arm-arm circulation time being 17.5 seconds.

(2) Crude pulmonary circulation time -

| | |
|--------------|---------------------|
| Normal range | 7-14 seconds |
| Average | <u>10.6</u> seconds |

The index of venous velocity, which is devised by subtracting the crude pulmonary circulation time from the arm-carotid time, expresses the circulation time for approximately three-quarters of the venous pathway from the antecubital vein to the heart, and not the entire arm-heart circulation time. The venous circulation time according to observations by the radium emanation method is more variable, and of less significance than the pulmonary circulation time. Weiss and Robb concluded that this was true also according to the cyanide method. The individual measurements for the venous circulation time ranged from 1-7 seconds - the average venous velocity index for the 21 subjects being 4.5 seconds. These results are again in harmony with the radium emanation method, in which the average arm-heart circulation time was 6.6 secs.

It was concluded by the above observers that the histamine reaction time, because of the peripheral location of its site of reaction is constantly longer than the arm-arm circulation time of the radium emanation method. The pathways differ in character and length. This is reflected in the difference in the circulation

time obtained by the two methods.

The practical agreement throughout between the results obtained by the radium emanation and cyanide methods is held by Weiss and Robb to indicate that the reaction time to cyanide is a trustworthy measure of the circulation time.

From the results of their observations, the same authors concluded that the cyanide method of measuring the circulation time fulfilled the following requirements:

- (1) Cyanide is non-toxic in the doses used.
- (2) In repeated observations no serious effects followed the intravenous administration.
- (3) The change in respiration is eminently suitable for the rôle of a signal reaction.
- (4) The abrupt onset, and conspicuous increase in respiratory amplitude occur at any phase of the normal respiratory cycle, and can be registered either graphically, or by means of a stop-watch.
- (5) Cyanide itself does not influence the velocity of the blood flow during the first circulation of the blood after injection, until the respiratory response has occurred. This observation is supported by the absence of any change in the heart rate, or arterial pressure prior to the occurrence of the increase in respiratory amplitude.
- (6) A wide range of effective but safe dosage of sodium cyanide exists sufficiently removed from the dangerous dose to warrant its use in man.
- (7) The method is pre-eminently objective in character, and is independent of the subject's co-operation.

Again, according to Robb and Weiss, the substance chosen, in this case sodium cyanide, should, and does, possess the following properties:

- (1) Should not influence the velocity of the blood flow until the signal reaction has occurred.
- (2) The substance, and its effect on the body, should be rapidly inactivated so that measurements may be repeated after short intervals of time if necessary.
- (3) The signal reaction, which is of primary importance in the method, must be objective in nature, and readily discernible in normal and pathological cases. The method also should, if possible, be suitable for graphic registration.
- (4) The reaction time proper of the substance, after arrival in the reacting organ, should be a negligibly small fraction of the entire reaction time, which consists of the circulation time, plus the reaction time proper.

(11)
Wood, in 1936, using sodium cyanide in a study of the circulation time, experienced difficulty in gauging the adequate dose. In some cases alarming respiratory embarrassment followed. Owing to this difficulty decholin was substituted.

For the purposes of the present investigation the methods which appeared to fulfil the above-mentioned requirements most accurately, and to possess most advantages and fewest limitations, were the histamine and sodium cyanide methods. They appeared to be eminently suitable for clinical application despite Wood's results.

The disadvantages of the radium emanation method, considered by

most observers to be most accurate, as it is made the subject of comparison, outweigh the advantages it possesses. The same considerations apply to the various dye methods.

The entirely subjective nature of the other methods already discussed, render them unsuitable for general application. The histamine and sodium cyanide methods having been finally chosen, the histamine method was first proceeded with.

HISTAMINE METHOD.

The drug was used according to the methods described by Weiss, Robb and Blumgart, and Bain.

The dosage recommended by Bain - 0.001 mg. of the drug per kilo of body weight being employed.

Ampoules were made containing 0.128 mg. histamine acid phosphate in η x saline. η ss of this solution for every stone of body weight was the calculated dose, e.g. the dose for a 10 stone patient being η v of the solution.

The requisite dose was injected into an antecubital vein, and an attempt made to measure the time interval between the injection and the first appearance of the flush on the face or neck. This time interval was measured by means of a stop watch.

Difficulty was experienced in timing the onset of the flush, but the most serious drawback was the occurring of severe headache, following the injection (notwithstanding the fact that the precaution was taken of keeping the patient recumbent following the injection). In several cases the headache was of such severity that it prejudiced the use of the test on the other patients in the ward.

The occurrence of a metallic taste in the mouth was found to be too variable to be of value as a signal reaction. For these reasons the use of the histamine method was discarded, and the sodium cyanide method finally employed.

SODIUM CYANIDE.

Pharmacology. Mode and site of action. Before proceeding to a description of the technique used and results obtained, it is desirable to discuss some of the pharmacological actions of the drug.

Loevenhart and his co-workers, in 1918, tested the efficiency of respiratory stimulants on dogs. The most powerful stimulation resulted from sodium cyanide intravenously injected - the degree and duration of the stimulation depending on the rapidity of the intravenous injection.

A decided temporary stimulation of respiration was found to occur with 0.1 c.c. of 0.05% solution per kilo of body weight. This stimulation could be maintained by injecting 0.1 c.c. of 0.05% of the drug every 1½-3½ minutes. This fact was held to indicate rapid disintoxication of the drug - supposedly by conversion into thiocyanate. This dosage occasioned but little alteration on the circulation. The fatal dose was 2½-6½ c.cs of 0.05% of solution per kilo of body weight, i.e. about 20 times the effective respiratory stimulant dose - an ample margin of safety was therefore guaranteed.

Loevenhart concluded that the effects on the respiration were

central in origin, as they were not affected by section of the vagi or atropine.

The same observer asserted that -

- (1) these small effective doses increased both the rate and the depth of the respiration, cyanide being one of the strongest stimulants of the respiratory centre.
- (2) The respiratory centre is more sensitive to cyanides than the vasomotor centre, and this again is more sensitive than the vagal centre.
- (3) Larger doses slow the rate but greatly increase the depth of respiration, and toxic doses depress and paralyse the centre.

(12)

Sollman considers that the effect of cyanide on the circulation is analogous to that of asphyxia, and that the vasomotor centre is intensely stimulated with a corresponding rise of blood pressure. A marked dilatation of the renal vessels is stated to occur, this effect being apparently due to a paralysis of the arterial muscle. A dilatation of the coronary vessels also occurs. The above author concludes that although the action of cyanides on the central nervous system bears a close resemblance to the phenomena of asphyxia, the oxygen supply to the tissues is not lessened.

If applied directly to the frog's heart, cyanides cause a standstill. There is no evidence of this action in mammals.

Fate of cyanide in the body. Cyanides are very unstable. They change readily even by mere exposure to light and air, and undergo still greater decomposition in the body. Part of the radicle combines with sulphur-containing molecules to form

sulphocyanides. A portion is excreted unchanged by the lungs, while the fate of the remainder is unknown. Sulphocyanide is found in human parotid saliva, and constantly in human urine.

During the present investigation of the velocity of blood flow an attempt was made to study the fate and excretion of the sodium cyanide injected. A description of the method and results obtained will be furnished later.

(13)
Loevenhart, Schlonovsky and Seybold, in 1922, employed various drugs to estimate the circulation time. In the course of their investigations on animals, they injected sodium cyanide intravenously, and always obtained a stimulation of the respiration within a few seconds, when an appropriate dose was used. This suggested to these observers the applicability of sodium cyanide to determine the circulation time in animals, and they suggested its use for that purpose in man.

The advantages found working with animals were, that the drug could be used on the intact animal, that it could be used over and over again on the same animal, and that it made chemical or electrical examination of the blood unnecessary.

The rapidity of action of the cyanides has been known since the first observation on their toxicological action. In fact, their popular reputation, as the deadliest poison, is based on this rapidity of action, and not on the smallness of the dose.

When cyanide was used in proper dosage in animals, Loevenhart et alii found that the effect passed off, within less than 1 minute as a rule, and that the injection could be repeated any number of times. No deleterious effects followed upon the repeated injection

of the drug in animals. Using the marginal ear vein as the site of injection, they found the average circulation time to equal 3.97 seconds in animals. They also found that a slow injection delayed the reaction time, and that accurate observations could only be made when the injection of the cyanide occupied one second or less - the more rapid the injection, the more rapid the onset of the reaction.

Beyond a certain optimum the reaction time was independent of dosage. The optimum dose in dogs was found to be 0.75 c.c. of 0.02% solution sodium cyanide. Injections of the drug usually slowed the pulse rate in unanaesthetised dogs in the dosage here employed. In 25 of their cases the pulse rate was slowed in 20, and only in 2 cases was the rate increased by 3-5 beats. In dogs, under ether anaesthesia, the dosage had to be increased 50-300% in order to stimulate the respiration.

Loevenhart and his co-workers concluded that a determination of the reaction time to sodium cyanide in rabbits and dogs, gave a figure within 1 second of the complete circulation time. Sodium cyanide stimulated all the medullary centres, but the most marked effect was on the respiration.

Small doses caused an increase in the rate and amplitude of respiration, whereas toxic doses produced a fleeting stimulation, followed rapidly by shallow irregular breathing, and finally by paralysis of respiration and death.

According to Loevenhart, the respiratory stimulant dose of sodium cyanide in man, when injected rapidly into a vein, is 3-5

mgm., which is approximately 0.04-0.07 mg. per kilo of body weight, whereas the fatal dose of the injected drug in man and animals was estimated to be 20 times the respiratory stimulating dose.

The toxicity of sodium cyanide is stated by Sollman to be considerably lower than that of several of the glucosides and alkaloids employed in clinical medicine.

Cumulative effects do not readily occur from the administration of cyanide, because of its rapid inactivation in the body, which, according to Loevenhart, progresses at the rate of 1.5-2 mg. per minute in man. The drug is converted in part into relative innocuous sulphur compounds, and in part into less closely related nitrogenous compounds.

Solis Cohen and Gittens maintain that excretion occurs as hydrocyanic acid in the breath and as cyanide in the urine. The above data on the pharmacology of the cyanide group indicate that sodium cyanide can be safely administered to man in amounts sufficient to produce a distinct stimulation of the respiration, and that the dose can be repeated after a short interval of time.

Robb and Weiss made a comparison of the results obtained by the three methods of estimating the circulation time - Radium emanation, histamine, and cyanide.

The histamine method measures the circulation time from arm to blood vessels of the face and brain.

The radium emanation method measures the circulation time from an antecubital vein of one arm to brachial artery of the opposite arm, a pathway more clearly defined, and less subject to variation.

The histamine reaction time, because of the peripheral location of its site of reaction, is consistently longer than the arm-arm circulation time of the radium emanation method. The two pathways differ in length - this is reflected in the difference in the circulation time obtained with the two methods.

The practical agreement throughout between the circulation time estimated by the radium emanation method (17.6 seconds), and the cyanide method (15.6 seconds) is held by these observers to indicate that in normal subjects the reaction time to cyanide is a trustworthy measure of the circulation time.

The longer average histamine reaction time of 23 seconds contrasts strongly with their 15.6 seconds cyanide reaction time.

According to Robb and Weiss such divergence between the results obtained with methods employing pathways regarded as essentially the same, required explanation.

In order to verify the existence of a significant difference between the reaction times obtained with the two pharmacological methods, they estimated the reaction times of both sodium cyanide and histamine in eight subjects under identical conditions. Their observations showed that in the same individual the cyanide reaction time was without exception shorter than the reaction time of histamine. The difference on the average was 6.5 seconds, which was sufficient in their minds to indicate a difference in the vascular site of the reaction.

Histamine is known to exert its dilatatory action directly on the minute vessels of the skin and brain. The flush obtained is due mainly to its primary action on the small veins. The reaction

time of histamine therefore is the time required for the blood to flow to the sub-papillary vessels rather than to a more proximal part of the large arteries, as is the case with the radium emanation method. The prolonged reaction time of histamine had been previously attributed to a considerable slowing of the blood flow in the smaller vessels.

Hering estimated the capillary circulation time as 5 seconds and according to Koch's observations, the capillary circulation would correspond to 8 seconds in man. The increase in respiratory amplitude, following cyanide injection, has been assumed to be due to a direct action of the drug on the respiratory centre. This explanation of its site of action would require that the cyanide must be delayed in arrival at the respiratory centre, because of the similarity in blood supply and circulation time to the skin of the face and to the brain.

Thus, unless there is a considerable difference in the rapidity of the action of these two substances, after their arrival in the brain capillaries, their reaction times should be in close agreement. Since the large difference of 6.5 seconds between the two methods cannot be ascribed to a more rapid action of the cyanide after its arrival, it must be explained by a shorter pathway and circulation time of the blood to the site of the cyanide action. Such a postulation, according to Robb and Weiss, would exclude the respiratory centre as the site of cyanide stimulation of the respiration.

(14)

Harriet Owen and Robert Gessel, in 1931, helped to throw additional light on the mechanism of the action of cyanide on the

respiration. They noted the effects of the injection of sodium cyanide into the carotid arteries after occlusion of the vertebral and external carotid arteries, and after denervation of one carotid sinus.

Injection of sodium cyanide on the side on which the innervation of the carotid sinus was intact, invariably produced increased pulmonary ventilation. Injection on the denervated side produced relatively small or no increase in ventilation; followed by depression.

By painting the innervated sinus with sodium cyanide, increased ventilation was obtained. This response was demonstrated to be due to a local action. Intravenous injection of sodium cyanide after double vagotomy, and double sinus denervation was found to be relatively ineffective, or entirely ineffective in augmenting ventilation. Later depression of ventilation was not uncommon.

Similar results were obtained if the common carotid arteries and internal occipital arteries were occluded as a substitute for denervation.

In addition, it was found that the injection of cyanide into the fourth ventricle of the brain produced either immediate excitation or depression without initial excitement.

These observers confirmed the investigations of Heymans and Dantrebande, made in the same year. They concluded from their observations that sodium cyanide exerted its action locally, and that this action was predominantly exerted on the carotid sinus.

The carotid sinus is a dilatation present at the bifurcation

of the common carotid artery. Numerous sensory receptors lie in the deeper parts of the adventitia here.

The sinus is in contact with the respiratory centre, through afferent nerves, and it has been proved by the experiments of Heymans and his co-workers that the nerve endings in this region are sensitive to changes in the chemical condition of the blood.

Perfusion of the innervated carotid sinus, with precautions taken to prevent the fluid reaching the medullary centres, produces striking respiratory effects, which can be proved to be reflex in origin as they are abolished by section of the sinus nerves.

Heymans believes that the sensory area, which responds to changes in the chemical composition of the blood, is located in the carotid body, and not in the sinus itself. The carotid body, however, is intimately related anatomically to the sinus.

According to the evidence obtained by Owen and Gessel, the reaction time to cyanide is the time required for the blood to flow to the carotid sinus of the carotid artery. This provides a rational explanation for the shorter reaction time of the cyanide method, and also its correspondence with the results obtained with the radium emanation method.

Since the action of cyanide on the carotid sinus of man had not been demonstrated, Robb and Weiss resorted to indirect methods to throw additional light on the mechanism of its action.

They determined the cyanide reaction time and the actual circulation time simultaneously in 7 individuals. An adequate amount of sodium cyanide, dissolved in 5 c.cs of a 50% solution of

glucose was rapidly injected into the antecubital vein of the arm, and the circulation times of both the cyanide and the glucose obtained.

The usual method of registering the reaction time of the cyanide was used. The circulation time of glucose was determined by its appearance in the arterial blood, as shown in blood samples taken from the femoral artery at known intervals of time.

In each case satisfactory estimations of both circulation times were obtained. These results showed close agreement of the reaction time of cyanide and circulation time of blood to the femoral artery. This close agreement was accepted by Robb and Weiss as substantial, though indirect evidence - that, in man as well as in animals, sodium cyanide acts upon the carotid sinus. Thus the cyanide reaction time measures the velocity of blood flow between a chosen vascular area, and a large artery as does the radium emanation method. They also concluded that sodium cyanide produces an immediate increase of respiration upon arrival in the carotid sinus, and that the time required for the cyanide to act after its arrival at the sinus must be a relatively small part of the total reaction time.

These conclusions make sodium cyanide a particularly suitable substance for a study of the estimation of the circulatory rate in cardio-vascular disease.

Sodium Cyanide - Dosage.

From his experiments on animals, Loevenhart concluded that the greater the concentration of sodium cyanide used, and the smaller

the volume injected, the more abrupt and intense was the resulting stimulation of the respiration.

Robb and Weiss in their observations on the circulatory rate employed a 2% aqueous solution of sodium cyanide. This percentage according to them, permitted the rapid injection of an effective dose of cyanide in small volume, without a significant alteration in the blood volume or velocity of the blood flow. The same observers concluded that the quantity required to produce an optimal respiratory response, was roughly proportional to the subject's body weight. In their series of normal cases, the optimal dose ranged from 5-10 mg. sodium cyanide, corresponding to 0.25-0.5 c.cs of a 2% solution, or 0.07-0.19 mg. per kilo of body weight, the average optimal dose equally 7 mg. 0.35 c.cs of a 2% solution, or 0.11 mg. per kilo of body weight respectively.

The above system of dosage was used by Robb and Weiss for the measurement of the arm-carotid circulation time. For an estimation of the crude pulmonary circulation time, 2/3rds of the above dose was found to be sufficient.

Three times the optimal dose was administered by Robb and Weiss to one of their normal cases, and no untoward reactions followed, proving that the range of the safe but effective dose of sodium cyanide is wide. In the present study of the blood velocity rate, the arm-carotid circulation time was estimated.

Before proceeding to a study of the velocity of the blood flow in cardio-vascular disease, the circulatory rate was estimated in a small series of normal subjects.

The estimation of the velocity of the blood flow in normal subjects.

The subjects chosen, adult males, 14 in number, were subjectively normal, and in clinical examination the cardiovascular and endocrine systems were found to be healthy. The resting heart rates and blood pressure were all within the accepted normal range.

The ages of the subjects varied from 21-46 years, and their weights from 10-14 stones.

The dose of the drug varied from 0.3-0.4 c.c. of 2% solution, and was calculated roughly according to the subject's weight, on the basis of 0.11 mg. per kilo of body weight. In a few cases no signal reaction followed the injection of 0.2 c.c. of the drug, but the same cases responded later to larger doses.

Shortly after commencing the present study, it was found that if the solution of the drug used was 2 or more days old, the signal reactions obtained varied in intensity, and in some cases no response occurred, even with the larger doses. No adequate reason could be obtained from the supplying firm, so subsequent supplies of the drug were obtained from a different firm, whose medical director kindly supplied the following data re the preparation and keeping qualities of sodium cyanide solution: "This solution is prepared by taking a fresh clean piece of pure sodium cyanide, and dissolving in a quantity of sterile glass-distilled water, so that the solution will be above 2% of cyanide.

"The solution is then assayed chemically, and the volume

adjusted accordingly with sterile glass-distilled water, so as to contain 2% of cyanide, calculated as sodium cyanide, and filtered into suitable small sterile bottles. It has been found that if the solution is kept in well-filled bottles at 4°C. in the dark (that is chill room temperature), very little decomposition occurs. After six weeks' storage under such conditions, we have found the strength to be 1.9%.

"If, however, the solution is kept in a partially-filled bottle at room temperature, the rate of deterioration is very rapid - about 10% per week."

The drug supplied by this firm was found to give satisfactory results, and in view of the above report, the precaution was taken of keeping the solution under as cool and dark conditions as possible.

Technique of administration.

The estimation of the circulatory rate was carried out as far as possible under semi-basal conditions. Three to four hours had elapsed since the previous meal, and the subjects had rested for a short time prior to the measurement.

The subject was placed in a semi-recumbent position, with the arm selected for the injection at the level of the right auricle. The requisite dose was drawn into a graduated 1 c.c. Record syringe, with an attached No.15 hypodermic needle. The arm was suitably sterilised, and the drug rapidly injected into a convenient antecubital vein. The rapidity of the injection was such that it occupied on the average less than 0.5 second. When venous stasis was required for successful venepuncture, the

subject's arm was manually compressed, the hand immediately withdrawn on the needle entering the vein, and a time interval allowed for adequate restoration of the normal venous flow before injection.

The circulatory rate, that is the time interval between the completion of the injection of the drug and the onset of the signal reaction, was measured by means of a stop-watch. Graphic registration of the rate was, however, thought preferable, and a Mackenzie-Lewis polygraph was utilised for this purpose.

The respiratory excursions were registered by means of an aluminium cup, strapped to the chest wall, and attached by rubber tubing to a tambour actuating a pen on the polygraph. The aluminium cup was not found to register the respiratory excursions satisfactorily, so a Paul Ebert's pneumograph was substituted. This instrument gave satisfactory results, and possessed a further advantage in that it could be tied over the subject's clothing.

On completion of the intravenous injection, a signal was given and a suitable indication instantly made on the recording paper. The time interval between this mark and the commencement of the increase in respiratory amplitude was measured by the polygraph time marker, each oscillation of the marker equalling one-fifth second. The subjects were not informed as to the nature of the signal reaction as it was feared that this knowledge might prejudice the accurate registration of the increase of respiratory amplitude.

Normal subjects - Results.

Graphs are presented from the 14 normal subjects, demonstrating the measurement of the circulatory rate. The time of injection of the drug, and the first increase in respiratory amplitude are indicated - the distance between the two, representing the time interval between the injection of the cyanide into an antecubital vein, and the arrival of the drug at the carotid sinus.

In all cases the signal reaction was characterised by a sharp increase in respiratory amplitude. This increase was clear cut and its time of onset easily recorded by a stop-watch.

The circulation times obtained by the graphic and stop-watch methods were in close agreement - on the average not varying more than 1 second.

The increase in respiratory amplitude lasted 10-20 seconds, and was accompanied in most cases by an increase in the respiratory rate.

Simultaneous with, or immediately subsequent to the respiratory reaction, an increase in the heart rate occurred in the majority of the cases. This was found to be slight in most, but in one case the heart rate rose from 60-80 beats per minute, and this higher rate was maintained for 4 minutes.

No significant increase was found in the blood pressure readings taken immediately after the reaction - a rise of from 2-4 mms. Hg. being found on the average.

Local Effects. Slight and fleeting pain was complained of by 2 of the subjects following the venepuncture.

General Effects. No untoward effects were experienced as a rule, with the exception of one case in whom a mild condition of collapse followed 20 minutes after the injection of the drug. This was however concluded to be psychical in origin, as some difficulty had been experienced in venepuncture.

The sensations experienced by the subjects consequent on the respiratory stimulation, were on the whole not unpleasant. A sense of suffocation was described by a few cases.

A taste of "bitter almonds in the mouth" was experienced by 2 of the cases, following shortly upon the respiratory disturbance.

The following are the individual circulatory rates (arm to carotid), obtained in the 14 normal subjects.

| | | |
|-------|-----------------|---------|
| No. 1 | $13\frac{3}{5}$ | seconds |
| 2 | 17 | " |
| 3 | $15\frac{3}{5}$ | " |
| 4 | 18 | " |
| 5 | $15\frac{4}{5}$ | " |
| 6 | $16\frac{2}{5}$ | " |
| 7 | $11\frac{3}{5}$ | " |
| 8 | $15\frac{1}{5}$ | " |
| 9 | $12\frac{1}{5}$ | " |
| 10 | $12\frac{4}{5}$ | " |
| 11 | $11\frac{3}{5}$ | " |
| 12 | 12 | " |
| 13 | $15\frac{4}{5}$ | " |
| 14 | $13\frac{1}{5}$ | " |

In the above series of 14 normal subjects the average arm-carotid circulatory rate is 14.35 seconds, or $14\frac{2}{5}$ with a range of 11.6-18 seconds.

This result is in agreement with that obtained by Robb and Weiss in 35 normal subjects - namely an average arm-carotid circulatory rate of 15.6 seconds.

The range 11.6-18 seconds is however narrower than that of 9-21 seconds, which was obtained in their series.

Wood in a series of 10 normal subjects obtained an average arm-carotid circulatory rate of 15.75 seconds; with this result also, the figure 14.35 seconds is in harmony.

Average arm-carotid circulatory rate 14.35 seconds

Normal range 11.6-18 seconds

In the series of patients with pathological signs and symptoms of the cardiovascular system, the technique of administration, and method of registration, were similar to those employed in the measurement of the circulatory rate in normal subjects. The dosage employed was calculated on a similar basis.

Graphs demonstrating the registration of the velocity of the blood flow are presented, relative to each case.

Clinical notes, including a short abstract of the history and physical examination, with special reference to the cardiovascular system are appended.

Electrocardiograms, taken to elicit further evidence of cardiac abnormality, are, where possible, included.

In order to demonstrate any change in the heart rate, occurring in relation to the increase in the respiratory amplitude,

a record of the pulse rate was in some cases obtained, and recorded simultaneously on the same graph. This was done by means of a glycerine pelote adjusted to a brachial artery, and attached to a second tambour on the polygraph.

CARDIOVASCULAR DISEASE.

The velocity of the blood flow in 129 patients with pathological signs and symptoms of the cardiovascular system, has been measured and graphically recorded. 132 such graphs demonstrating this measurement are presented.

A simultaneous tracing from the brachial artery is recorded in a few of the graphs.

Any significant change occurring in the blood pressure, estimated immediately after the respiratory stimulation, is recorded.

The 129 patients studied in this series have been classified and divided into the following groups:

| | |
|--|---------------------|
| 1. Arterial hypertension - hyperpiesia | 36 cases |
| 2. Rheumatic heart disease | 20 " |
| 3. Cardiac failure | 20 " (23 graphs) |
| 4. Thyrotoxicosis | 18 cases |
| 5. Specific heart disease | 12 " |
| 6. Myocardial degeneration | 5 " |
| 7. Effort syndrome | 4 " |
| 8. Angina pectoris | 3 " |
| 9. Auricular fibrillation | 3 " |
| 10. Anaemia | 3 " |
| 11. Acute rheumatic endocarditis (pyrexia) | 2 " |
| 12. Hypo-thyroidism | 2 " |
| 13. Bradycardia (vagal) | 1 " |

The ages of the patients ranged from 13-76 years. Technical difficulties and lack of co-operation preclude the use of this method of estimating the circulatory rate in children. In case No.4 - a boy of 13 years - graphic registration was unsatisfactory, the bizarre respiratory excursions, due to sobbing, obscuring the onset of the signal reaction, which in this case was measured by means of a stop-watch. The graphic method was successfully employed in patients aged 15 years, whose co-operation had been gained.

GROUP IArterial Hypertension - Hyperpiesia

36 patients, aged 34-65 years, with established arterial Hypertension are included in this group. The term Hypertension is taken to denote an established systolic blood pressure reading of over 160 mms. Hg. and a diastolic pressure of over 90 mms. Hg.

No previous history of cardiac failure was obtained in any case, and on careful examination no evidence of the condition was detected.

The majority of the patients demonstrated subjective evidence of pathological involvement of the cardiovascular system.

26 of the cases complained of varying degrees of effort dyspnoea.

In 16 cases complaint was made of diffuse praecordial pain, present while at rest, but intensified by exertion. The pain was described as dull in character, and chiefly localised to the left sub-mammary region.

6 cases presented the syndrome of 'Angina of effort'. In them the pain was substernal, with typical radiation, and lasted for seconds or minutes, being relieved by rest or nitrites.

In 3 cases a history of hemiplegia was obtained, and in one case objective evidence of this condition remained.

In 1 case (Case No.94), a history suggestive of the 'Adams-Stokes' Syndrome was given, and Case No.47 suggested a previous

attack of paroxysmal auricular flutter.

27 cases complained of palpitation occurring on exertion or at rest.

General symptoms complained of by the majority were - giddiness, headaches, and exhaustion.

Clinical evidence, indicative of cardiovascular disease, e.g. left ventricular enlargement, peripheral vascular sclerosis etc., was present in the majority of the cases.

Electrocardiographic examination revealed varying degrees of myocardial degeneration, viz. slurring of the Q,R,S complex, and partial heart block.

Of the few cases in which the blood N.P.N was estimated, only one (No.126), showed evidence of nitrogen retention.

The circulatory rate in the 36 patients assigned to this group, ranged from 9.4 secs - 21.8 secs, producing an average of 12.82 secs. This average is 1.53 secs less than the average normal figure of 14.35 secs.

No consistent relationship between the blood pressure, ventricular rate, or weight of the patient, and the circulatory rate, was found to exist.

In case No.28, whose systolic blood pressure was 300 + mms. Hg, the rate was 10.8 secs, while No.127 weighing nearly 19 st., gave a measurement of 9.8 secs. Case No.2 whose circulatory rate measured 21.8 secs, showed no clinical evidence of cardiac failure, but an X-ray examination demonstrated "Chronic inflammatory changes in the lungs."

Other cases showing a slight prolongation of the velocity of blood flow in this group were:

| | | |
|-------|------------------|------------------------------------|
| No.15 | Circulatory rate | 16 secs. |
| 93 | " " | 17.2 secs. Angina of effort. |
| 94 | " " | 16.4 secs. 'Adams-Stokes Syndrome' |
| 102 | " " | 16.4 secs. |

(Some engorgement of the cervical veins was present, but no other clinical evidence of failure)

105 Circulatory rate 16.6 secs. Angina of effort.

The average figure of 12.82 secs, slightly less than the normal average figure obtained in this group, is in keeping with the findings of Blumgart, Weiss and Robb. These observers, using the histamine method, concluded that the circulatory rate in patients with essential arterial hypertension, but without circulatory failure, was normal.

Blumgart and Weiss, by means of the radium emanation method, demonstrated that the velocity of the blood flow in patients with arterial hypertension, but free from signs of circulatory failure, is either normal or slightly slower than normal.

Blumgart suggests that this slight slowing in the blood velocity in hypertension, in the absence of signs of circulatory failure, in some patients may be related to the back pressure effects of arterial hypertension, within the pulmonary vessels, and quotes Gerhardt's experimental evidence for this view. This worker observed that increased arterial pressure in the greater circulation produced an increase in the volume of the lesser circulation. Such an increase in the amount of blood in the lungs would lead to increased cross-sectional diameter of the stream of blood flowing

through the lungs. Slowing in the blood flow, with prolongation of the pulmonary circulation time, would then occur, since the velocity of flow is conversely proportional to the cross-sectional diameter of the stream.

Further support for this hypothesis is derived from the experiment of Wearn, Barr and German, who observed that slight compression of the abdominal aorta in animals caused considerable dilatation of the alveolar capillaries.

The average circulatory rate of 12.82 secs, in this series of 36 patients, with established hypertension, is however in contrast with the results obtained by Bain. This observer, using the histamine method, in 29 cases of hyperpiesia, found a prolongation of the circulatory rate in all but 2 cases. These 29 cases were without signs of circulatory failure. Where the patient suffered from dyspnoea on exertion, or effort angina, the rate was found to be further prolonged. In his series of cases he failed to obtain a normal rate in established hyperpiesia, and concluded that the test was of value in differentiating such cases from raised blood pressure of 'functional origin'.

GROUP II

Rheumatic Heart Disease

20 patients, aged 13-49 years, suffering from chronic heart disease, are included in this category. All the cases in the group could be designated 'Compensated valvular disease' i.e. on careful examination no clinical signs of failure were detected. A previous history of this condition was excluded.

All the patients gave a previous history of 'rheumatic infection' viz. rheumatic fever, chorea, growing pains, or tonsillitis.

In all but 2, symptoms referable to the cardiovascular system were complained of. Such symptoms were effort dyspnoea, palpitation, giddiness, faintness, and praecordial discomfort.

3 cases are examples of pure aortic disease, while in 8 the mitral valve appeared to be alone involved. The remaining 9 cases showed a combination of aortic and mitral lesions.

The circulatory rate in the 20 patients belonging to this group ranged from 8.2 secs to 19.2 secs, with an average rate of 12.4 secs, nearly 2 secs less than the average normal of 14.35 secs.

The anatomical site of the lesion did not appear to influence the circulatory rate.

Case No.75, whose circulatory rate was 19.2 secs, was a woman who had suffered for 5 years from effort dyspnoea, and on examination was slightly cyanotic.

In case No.82 the circulatory rate was 15.8 secs. The cardiac rhythm in this case was irregular and the electrocardiogram revealed the presence of auricular extrasystoles.

Case No.8, with a rate of 15.4 secs, showed electrocardiographic evidence of a mild degree of partial heart block.

Case No.23, whose circulatory rate was 10.4 secs, had an irregular cardiac rhythm, due to ventricular extrasystoles.

In Case No.124 circulatory rate 14.2 secs, a mild attack of rheumatic fever had occurred 10 days previously. At the time of the test the temperature was normal and the joint manifestations had subsided, but the heart rate was 108.

The average circulatory rate of 12.4 secs obtained in this group is in harmony with the results obtained by other methods.

Blumgart and Weiss, using the radium emanation method, concluded that in valvular disease, with compensation established, the circulation time was normal or slightly prolonged, according to the clinical condition of the patient.

Robb and Weiss, by means of the sodium cyanide method, estimated the circulatory rate in 10 patients with chronic valvular disease, without circulatory failure. Their measurements were found to be within the normal range, though there was a tendency towards stasis and retardation of blood flow.

The average arm-carotid rate in their 10 cases was 18.3 secs.

GROUP III

Cardiac Failure

20 patients, aged 20-76 years, are included in this group. All cases were suffering from rheumatic, hypertensive or arterio-sclerotic heart disease, with subjective and objective signs of failure. The presence of the following criteria of cardiac failure was determined in the clinical examination of the case:

- (1) Physical signs of pulmonary stasis.
- (2) Peripheral oedema.
- (3) Hepatic enlargement and tenderness.
- (4) Ascites.

The diagnosis of failure, however, was on occasion considered justifiable in the presence of pulmonary congestion alone.

13 of the patients were suffering from rheumatic heart disease, 3 from hypertension, and 2 from arterio-sclerotic heart disease. In 2 cases the blood Wassermann reaction was positive.

In 3 cases only was the cardiac rhythm regular. In the remaining 17 the arrhythmia was due to auricular fibrillation, auricular flutter, or the presence of extrasystoles.

The circulatory rate of the group ranged from 17.6 secs to 39.6 secs, the average rate being 23.8 secs or 9.5 secs longer than the average normal rate of 14.35 secs.

In cases Nos.11, 14, and 122, the circulatory rate measured 17.6, 20.4, and 20 secs. All three cases had received treatment by digitalis prior to the measurement. No.14 had received known

quantities of strophanthin, in the other two the dosage of the digitalis preparation administered was unknown. Signs of pulmonary congestion alone constituted the diagnosis of failure. Case No.14 had a palpably enlarged liver, but this was in all probability due to a local lesion, as the patient was suffering from chronic cholecystitis.

In Case No.88 three measurements of the circulatory rate were made at intervals of 6 weeks. The first measurement was made before the institution of treatment and was 23.2 secs.

When the 2nd estimation was made 6 weeks later, the patient has been digitalised, treated with diuretics, and a hydrothorax tapped. Physical signs of pulmonary stasis, and slight peripheral oedema were still present. The circulatory rate at this stage measured 23.6 secs. The patient meanwhile had lost 7 lbs in weight.

The 3rd and last estimation was carried out in another 6 weeks time. The patient was under a maintenance dose of digitalis, no peripheral oedema was present, and only minimal signs of pulmonary congestion. The circulatory rate finally measured 19.2 secs - 3.4 secs faster than the original rate.

In Case No.103 2 measurements of the rate were carried out. This case gave a previous history of failure, and was critically ill - gallop rhythm being present. The primary estimation was made as before prior to treatment when the patient was water-logged. The rate measured 39.6 secs.

4 weeks later a 2nd measurement showed a rate of 24.2 secs, i.e. 15.4 secs faster than the 1st. The patient at this time was 6 lbs lighter in weight, but still presented signs of pulmonary

stasis, and slight peripheral oedema. She was under a maintenance dose of digitalis at the time of this estimation.

These 2 cases (Nos. 88 and 103) demonstrate the relationship between the circulatory rate and the improvement in the patients' clinical condition.

5 cases (Nos. 44, 61, 70, 78 and 91), suffering from hypertensive or arterio-sclerotic heart disease, were examples of 'left ventricular failure'. In 3 the history was suggestive of 'cardiac asthma', while in Case No. 70 a probable attack of coronary thrombosis had occurred. In these 5 cases the only physical sign of failure was detectable in the pulmonary system, viz. basal crepitations. In No. 61 the liver was clinically enlarged - but this was in all probability a result of excessive indulgence in alcohol.

The circulatory rate of this small group ranged from 20 secs to 33.6 secs.

The signal reaction following the injection of cyanide, in this group of patients, with cardiac decompensation, differed in several respects from the normal reaction, and the reaction occurring in patients without failure. The respiratory response to cyanide in cases of circulatory failure was characterised by a more moderate increase in the respiratory amplitude. The onset of the signal reaction was in general more gradual than normal, and the duration of the respiratory stimulation longer.

Some difficulty was experienced in timing the onset of the signal reaction by the stop-watch method, and in this group the times obtained by the two methods were not always in agreement.

This prolongation of the respiratory stimulation by cyanide in cases of circulatory failure is in agreement with the postulation, by Blumgart and Weiss, of a 'stringing out' of the blood stream, in these cases, during its passage through the heart and lungs.

In circulatory failure the modification of the respiratory reaction to cyanide can most probably be accounted for by the fact that the concentration of the drug in the blood stream, arriving at the carotid sinus, is somewhat lowered owing to the diminished velocity of the blood flow through the pulmonary circuit. In all the cases in this group, however, the signal reaction was distinctive enough to allow of its graphic registration.

Blumgart and Weiss, using the radium emanation method, found that in cases of circulatory failure, owing to the phenomenon of 'stringing out' of the blood stream, an increased dose of the radio-active substance was necessary. This observation was supported by Weiss, Robb and Blumgart, using histamine as a reagent.

In this series of cases of circulatory failure, larger doses as a rule were found to be necessary to produce adequate respiratory stimulation, e.g. Case No.85, in which no respiratory response followed injections of 6 mg. or 7 mg. of sodium cyanide. It was not until 8 mg. of the drug were employed that the circulatory rate was successfully measured.

A similar experience was found with Case No.70. It was concluded that in the presence of failure, larger doses of sodium cyanide are necessary. This conclusion is in harmony with the

view expressed by the observers noted above, using the radium emanation and histamine methods, but is in direct contrast to the findings of Robb and Weiss. These observers, using sodium cyanide, expressed the view that smaller doses of the drug were adequate in cardiac failure. The average optimal dose employed by them, under these circumstances, was 0.26 c.cs of a 2% solution or 0.084 mg. per kilo of body weight - this being approximately 25% smaller than the average normal dose. They concluded that this smaller dosage indicated a lowering of the threshold for cyanide stimulation during circulatory failure.

In this series of cases of cardiac failure, no untoward results followed the injection of sodium cyanide, even when the patient appeared critically ill.

Case No.14 complained of severe upper abdominal pain following the estimation. This was concluded to be co-incidental, and due to the presence of a pathological gall-bladder.

The average figure of 23.8 secs obtained in this group, demonstrating a slowing of the circulatory rate in cardiac failure, is in agreement with the results obtained by all investigators using other methods. Koch, although his studies were limited to 5 c.c. samples of blood, was able to prove a definite slowing of the blood stream in congestive heart failure.

Blumgart and Weiss (radium emanation method), found prolongation of the circulatory time in cases of failure - in some cases up to 108% above the extreme upper limit of normal.

Weiss, Robb and Blumgart (histamine method) concluded that a

markedly prolonged circulation time was always associated with severe circulatory failure, but that the prolongation of the reaction time was not necessarily proportional to the severity of the clinical condition.

Robb and Weiss (sodium cyanide method), Tarr, Oppenheimer and Sagar (sodium dehydrocholate method), and Hitziz (ether method), among the other investigators, confirmed this prolongation of the circulatory rate in circulatory failure.

Of the cases are suggestive of so-called primary thyrotoxicosis, while in 3 an enlargement of the gland was present. In 2 cases arrhythmia, due to auricular fibrillation, and signs of circulatory failure were evident. 5 cases gave a previous history of rheumatic infection, and showed signs of rheumatic endocarditis in addition to the effects of the hyper-thyroid state. 13 of the patients complained of symptoms relative to the cardiovascular system; and in all cases, on clinical or electrocardiographic examination, some abnormality of the cardiovascular system was present.

The circulatory rate in this group ranged from 5.4 sec. to 15.6 sec., with an average rate of 9 sec. or 9.3 sec. less than the average normal time of 14.55 sec.

In Case No. 27 circulatory rate 11.6 sec., auricular fibrillation and crepitations at the lung bases indicating failure were present.

Case No. 30, showing more marked signs of failure, gave a rate of 14.6 sec. Signs of mitral stenosis due to previous rheumatic infection, were present in this patient.

In Cases No. 34, 39, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100 - a continuation of the study.



GROUP IV

Thyrotoxicosis

18 patients, aged 15-59 years, are included in this group. Biochemical estimations of the basal metabolic rate were not carried out, but all cases exhibited subjective or objective evidence of thyroid dysfunction. 15 of the cases are examples of so-called primary thyrotoxicosis, while in 3 an adenomatous condition of the gland was present. In 2 cases arrhythmia, due to auricular fibrillation, and signs of circulatory failure were evident. 5 cases gave a previous history of rheumatic infection, and showed signs of rheumatic endocarditis in addition to the effects of the hyper-thyroid state. 13 of the patients complained of symptoms relative to the cardiovascular system; and in all cases, on clinical or electrocardiographic examination, some abnormality of the cardiovascular system was present.

The circulatory rate in this group ranged from 4.4 secs to 14.6 secs., with an average rate of 9 secs or 5.3 secs less than the average normal time of 14.35 secs.

In Case No.27 circulatory rate 11.6 secs, auricular fibrillation and crepitations at the lung bases indicating failure were present.

Case No.30, showing more marked signs of failure, gave a rate of 14.6 secs. Signs of mitral stenosis due to previous rheumatic infection, were present in this patient.

In Cases No.34, 69, 84 and 96 - a combination of rheumatic

endocarditis and thyrotoxicosis - the circulatory rate measured 12.4, 6.8, 10.2, and 8 secs respectively.

In the 2 cases with signs of circulatory failure, the average circulatory rate is 13.1 secs compared to the rate of 23.8 secs, which is the average for the failure group.

In contradistinction to the failure group the signal reaction in this group was generally characterised by a sharp onset, high increase in respiratory amplitude, and relatively short duration of respiratory stimulation.

No untoward effects followed the injection of the cyanide in this group.

The average circulatory rate of 9 secs obtained in this group is again in agreement with other results.

(16)
Blumgart, Gargill and Gilligan were the first to estimate the blood velocity in patients suffering from thyrotoxicosis.

In thyrotoxic patients with no clinical evidence of cardiovascular disease, these observers found that the velocity of the blood flow was strikingly increased, the circulation times in some cases being the most rapid observed in any condition.

In 4 patients with clinical evidence of cardiovascular disease, they found that the velocity of the blood flow, though increased, was slower than the average velocity in the uncomplicated thyrotoxic patients. Estimating the basal metabolic rates of their cases, they concluded that the increased velocity of the blood flow paralleled the rise in the basal metabolic rate.

Tarr, Oppenheimer and Sagar, using the decholin method in

thyrotoxicosis without cardiac failure, obtained an average circulatory rate of 9 secs in contrast to their normal average rate of 13 secs.

In cases with cardiac failure the average circulatory rate was 13 secs.

The results obtained in the present series are in close agreement with the above results, and as the above observers remark, the circulatory time period in patients with congestive heart failure and Graves' disease, appears to be an arithmetical average of what would be expected in either condition alone.

Kremer and Robertson also using decholin as an agent, concluded that in thyrotoxicosis a raised basal metabolic rate resulted in a shortened circulation time. They observed, however, that it was impossible to predict the degree of rise of basal metabolic rate from the circulation time.

GROUP V

Specific Heart Disease

12 patients, aged 35-61 years, are included in this group. In 9 of the cases the blood Wassermann reaction was positive, while in 2 of the remaining cases a previous history of luetic infection was admitted.

In all 12 cases dyspnoea on effort was complained of, and in 9 cases praecordial pain or constriction related to exertion was recorded. Other symptoms complained of were palpitation and giddiness.

No previous history of cardiac failure was obtained in any case, and this condition was excluded by clinical examination.

All cases exhibited signs of involvement of the cardiovascular system, clinically or on electrocardiographic examination.

In 6 cases typical signs of aortic incompetence were present, while in the other 6 aortitis, or myocardial degeneration, was concluded to be present.

The circulatory rate in this group ranged from 8.6 secs to 25.6 secs, with an average rate 14 secs, approximately equal to the normal average rate of 14.35 secs.

In Case No.101 - a female patient - the rate was 25.6 secs. In this case the heart was markedly enlarged and 'gallop rhythm' was present. No signs indicative of failure were noted on

clinical or radiological examination.

Case No. 113, with a rate of 8.6 secs, had only experienced symptoms for 1 month prior to the estimation, and expressed himself as being perfectly well until this time. As all the cases complained of a relatively severe effort dyspnoea, and as dyspnoea is usually a symptom of late onset in aortic incompetence and aortitis, the obtaining of an average rate of 14 secs in this group would appear to support the view that dyspnoea and cardiac pain, in the above circumstances, may be due to a reflex nervous mechanism, and not to congestion of the pulmonary blood system.

Blumgart suggests that the above symptoms, occurring in patients with aortitis, may be due in part to a transitory functional disproportion between the right and left chambers of the heart. The above normal rate of 14 secs is in harmony with the findings of Blumgart and Weiss, who on examining 7 cases of specific heart disease, obtained normal circulatory rates in the absence of circulatory failure.

Case No. 114, with a circulatory rate of 11 secs, showed evidence of myocardial degeneration on electrocardiographic examination. No adequate cause for this was found.

Case No. 115, circulatory rate 13.6 secs, gave a history suggestive of recent coronary thrombosis, although this assumption was not supported by the electrocardiogram.

Case No. 116, circulatory rate 17.2 secs, was a case of myocardial degeneration secondary to chronic bronchitis and emphysema.

No. 117, whose circulatory rate was 17.6 secs, was a female patient, who had undergone the operation of thyroidectomy.

GROUP III

Cardiac Failure

20 patients, aged 20-76 years, are included in this group. All cases were suffering from rheumatic, hypertensive or arterio-sclerotic heart disease, with subjective and objective signs of failure. The presence of the following criteria of cardiac failure was determined in the clinical examination of the case:

- (1) Physical signs of pulmonary stasis.
- (2) Peripheral oedema.
- (3) Hepatic enlargement and tenderness.
- (4) Ascites.

The diagnosis of failure, however, was on occasion considered justifiable in the presence of pulmonary congestion alone.

13 of the patients were suffering from rheumatic heart disease, 3 from hypertension, and 2 from arterio-sclerotic heart disease. In 2 cases the blood Wassermann reaction was positive.

In 3 cases only was the cardiac rhythm regular. In the remaining 17 the arrhythmia was due to auricular fibrillation, auricular flutter, or the presence of extrasystoles.

The circulatory rate of the group ranged from 17.6 secs to 39.6 secs, the average rate being 23.8 secs or 9.5 secs longer than the average normal rate of 14.35 secs.

In cases Nos.11, 14, and 122, the circulatory rate measured 17.6, 20.4, and 20 secs. All three cases had received treatment by digitalis prior to the measurement. No.14 had received known

quantities of strophanthin, in the other two the dosage of the digitalis preparation administered was unknown. Signs of pulmonary congestion alone constituted the diagnosis of failure. Case No.14 had a palpably enlarged liver, but this was in all probability due to a local lesion, as the patient was suffering from chronic cholecystitis.

In Case No.88 three measurements of the circulatory rate were made at intervals of 6 weeks. The first measurement was made before the institution of treatment and was 23.2 secs.

When the 2nd estimation was made 6 weeks later, the patient has been digitalised, treated with diuretics, and a hydrothorax tapped. Physical signs of pulmonary stasis, and slight peripheral oedema were still present. The circulatory rate at this stage measured 23.6 secs. The patient meanwhile had lost 7 lbs in weight.

The 3rd and last estimation was carried out in another 6 weeks time. The patient was under a maintenance dose of digitalis, no peripheral oedema was present, and only minimal signs of pulmonary congestion. The circulatory rate finally measured 19.2 secs - 3.4 secs faster than the original rate.

In Case No.103 2 measurements of the rate were carried out. This case gave a previous history of failure, and was critically ill - gallop rhythm being present. The primary estimation was made as before prior to treatment when the patient was water-logged. The rate measured 39.6 secs.

4 weeks later a 2nd measurement showed a rate of 24.2 secs, i.e. 15.4 secs faster than the 1st. The patient at this time was 6 lbs lighter in weight, but still presented signs of pulmonary

stasis, and slight peripheral oedema. She was under a maintenance dose of digitalis at the time of this estimation.

These 2 cases (Nos.88 and 103) demonstrate the relationship between the circulatory rate and the improvement in the patients' clinical condition.

5 cases (Nos. 44, 61, 70, 78 and 91), suffering from hypertensive or arterio-sclerotic heart disease, were examples of 'left ventricular failure'. In 3 the history was suggestive of 'cardiac asthma', while in Case No.70 a probable attack of coronary thrombosis had occurred. In these 5 cases the only physical sign of failure was detectable in the pulmonary system, viz. basal crepitations. In No.61 the liver was clinically enlarged - but this was in all probability a result of excessive indulgence in alcohol.

The circulatory rate of this small group ranged from 20 secs to 33.6 secs.

The signal reaction following the injection of cyanide, in this group of patients, with cardiac decompensation, differed in several respects from the normal reaction, and the reaction occurring in patients without failure. The respiratory response to cyanide in cases of circulatory failure was characterised by a more moderate increase in the respiratory amplitude. The onset of the signal reaction was in general more gradual than normal, and the duration of the respiratory stimulation longer.

Some difficulty was experienced in timing the onset of the signal reaction by the stop-watch method, and in this group the times obtained by the two methods were not always in agreement.

This prolongation of the respiratory stimulation by cyanide in cases of circulatory failure is in agreement with the postulation, by Blumgart and Weiss, of a 'stringing out' of the blood stream, in these cases, during its passage through the heart and lungs.

In circulatory failure the modification of the respiratory reaction to cyanide can most probably be accounted for by the fact that the concentration of the drug in the blood stream, arriving at the carotid sinus, is somewhat lowered owing to the diminished velocity of the blood flow through the pulmonary circuit. In all the cases in this group, however, the signal reaction was distinctive enough to allow of its graphic registration.

Blumgart and Weiss, using the radium emanation method, found that in cases of circulatory failure, owing to the phenomenon of 'stringing out' of the blood stream, an increased dose of the radio-active substance was necessary. This observation was supported by Weiss, Robb and Blumgart, using histamine as a reagent.

In this series of cases of circulatory failure, larger doses as a rule were found to be necessary to produce adequate respiratory stimulation, e.g. Case No.85, in which no respiratory response followed injections of 6 mg. or 7 mg. of sodium cyanide. It was not until 8 mg. of the drug were employed that the circulatory rate was successfully measured.

A similar experience was found with Case No.70. It was concluded that in the presence of failure, larger doses of sodium cyanide are necessary. This conclusion is in harmony with the

view expressed by the observers noted above, using the radium emanation and histamine methods, but is in direct contrast to the findings of Robb and Weiss. These observers, using sodium cyanide, expressed the view that smaller doses of the drug were adequate in cardiac failure. The average optimal dose employed by them, under these circumstances, was 0.26 c.cs of a 2% solution or 0.084 mg. per kilo of body weight - this being approximately 25% smaller than the average normal dose. They concluded that this smaller dosage indicated a lowering of the threshold for cyanide stimulation during circulatory failure.

In this series of cases of cardiac failure, no untoward results followed the injection of sodium cyanide, even when the patient appeared critically ill.

Case No.14 complained of severe upper abdominal pain following the estimation. This was concluded to be co-incidental, and due to the presence of a pathological gall-bladder.

The average figure of 23.8 secs obtained in this group, demonstrating a slowing of the circulatory rate in cardiac failure, is in agreement with the results obtained by all investigators using other methods. Koch, although his studies were limited to 5 c.c. samples of blood, was able to prove a definite slowing of the blood stream in congestive heart failure.

Blumgart and Weiss (radium emanation method), found prolongation of the circulatory time in cases of failure - in some cases up to 108% above the extreme upper limit of normal.

Weiss, Robb and Blumgart (histamine method) concluded that a

markedly prolonged circulation time was always associated with severe circulatory failure, but that the prolongation of the reaction time was not necessarily proportional to the severity of the clinical condition.

Robb and Weiss (sodium cyanide method), Tarr, Oppenheimer and Sagar (sodium dehydrocholate method), and Hitziz (ether method), among the other investigators, confirmed this prolongation of the circulatory rate in circulatory failure.



GROUP IVThyrotoxicosis

18 patients, aged 15-59 years, are included in this group. Biochemical estimations of the basal metabolic rate were not carried out, but all cases exhibited subjective or objective evidence of thyroid dysfunction. 15 of the cases are examples of so-called primary thyrotoxicosis, while in 3 an adenomatous condition of the gland was present. In 2 cases arrhythmia, due to auricular fibrillation, and signs of circulatory failure were evident. 5 cases gave a previous history of rheumatic infection, and showed signs of rheumatic endocarditis in addition to the effects of the hyper-thyroid state. 13 of the patients complained of symptoms relative to the cardiovascular system; and in all cases, on clinical or electrocardiographic examination, some abnormality of the cardiovascular system was present.

The circulatory rate in this group ranged from 4.4 secs to 14.6 secs., with an average rate of 9 secs or 5.3 secs less than the average normal time of 14.35 secs.

In Case No.27 circulatory rate 11.6 secs, auricular fibrillation and crepitations at the lung bases indicating failure were present.

Case No.30, showing more marked signs of failure, gave a rate of 14.6 secs. Signs of mitral stenosis due to previous rheumatic infection, were present in this patient.

In Cases No.34, 69, 84 and 96 - a combination of rheumatic

endocarditis and thyrotoxicosis - the circulatory rate measured 12.4, 6.8, 10.2, and 8 secs respectively.

In the 2 cases with signs of circulatory failure, the average circulatory rate is 13.1 secs compared to the rate of 23.8 secs, which is the average for the failure group.

In contradistinction to the failure group the signal reaction in this group was generally characterised by a sharp onset, high increase in respiratory amplitude, and relatively short duration of respiratory stimulation.

No untoward effects followed the injection of the cyanide in this group.

The average circulatory rate of 9 secs obtained in this group is again in agreement with other results.

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Blumgart, Gargill and Gilligan were the first to estimate the blood velocity in patients suffering from thyrotoxicosis.

In thyrotoxic patients with no clinical evidence of cardiovascular disease, these observers found that the velocity of the blood flow was strikingly increased, the circulation times in some cases being the most rapid observed in any condition.

In 4 patients with clinical evidence of cardiovascular disease, they found that the velocity of the blood flow, though increased, was slower than the average velocity in the uncomplicated thyrotoxic patients. Estimating the basal metabolic rates of their cases, they concluded that the increased velocity of the blood flow paralleled the rise in the basal metabolic rate.

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thyrotoxicosis without cardiac failure, obtained an average circulatory rate of 9 secs in contrast to their normal average rate of 13 secs.

In cases with cardiac failure the average circulatory rate was 13 secs.

The results obtained in the present series are in close agreement with the above results, and as the above observers remark, the circulatory time period in patients with congestive heart failure and Graves' disease, appears to be an arithmetical average of what would be expected in either condition alone.

Kremer and Robertson also using decholin as an agent, concluded that in thyrotoxicosis a raised basal metabolic rate resulted in a shortened circulation time. They observed, however, that it was impossible to predict the degree of rise of basal metabolic rate from the circulation time.

All cases exhibited signs of involvement of the cardiovascular system, clinically or on electrocardiographic examination.

In 5 cases typical signs of aortic incompetence were present, while in the other 5 aortic, or myocardial degeneration, was considered to be present.

The circulatory rate in this group ranged from 9.8 secs to 25.8 secs, with an average rate of 14.37 secs, approximately equal to the normal average rate of 14.37 secs.

In Case No. 131 - a female patient - the rate was 25.8 secs. In this case the heart was markedly enlarged and "gallop rhythm" was present. No signs indicative of failure were noted in

GROUP V

Specific Heart Disease

12 patients, aged 35-61 years, are included in this group. In 9 of the cases the blood Wassermann reaction was positive, while in 2 of the remaining cases a previous history of luetic infection was admitted.

In all 12 cases dyspnoea on effort was complained of, and in 9 cases praecordial pain or constriction related to exertion was recorded. Other symptoms complained of were palpitation and giddiness.

No previous history of cardiac failure was obtained in any case, and this condition was excluded by clinical examination.

All cases exhibited signs of involvement of the cardiovascular system, clinically or on electrocardiographic examination.

In 6 cases typical signs of aortic incompetence were present, while in the other 6 aortitis, or myocardial degeneration, was concluded to be present.

The circulatory rate in this group ranged from 8.6 secs to 25.6 secs, with an average rate 14 secs, approximately equal to the normal average rate of 14.35 secs.

In Case No.101 - a female patient - the rate was 25.6 secs. In this case the heart was markedly enlarged and 'gallop rhythm' was present. No signs indicative of failure were noted on

clinical or radiological examination.

Case No. 113, with a rate of 8.6 secs, had only experienced symptoms for 1 month prior to the estimation, and expressed himself as being perfectly well until this time. As all the cases complained of a relatively severe effort dyspnoea, and as dyspnoea is usually a symptom of late onset in aortic incompetence and aortitis, the obtaining of an average rate of 14 secs in this group would appear to support the view that dyspnoea and cardiac pain, in the above circumstances, may be due to a reflex nervous mechanism, and not to congestion of the pulmonary blood system.

Blumgart suggests that the above symptoms, occurring in patients with aortitis, may be due in part to a transitory functional disproportion between the right and left chambers of the heart. The above normal rate of 14 secs is in harmony with the findings of Blumgart and Weiss, who on examining 7 cases of specific heart disease, obtained normal circulatory rates in the absence of circulatory failure.

No adequate cause for this was found.

Case No. 83, circulatory rate 15.6 secs, gave a history suggestive of recent coronary thrombosis, although this assumption was not supported by the electrocardiogram.

Case No. 104, circulatory rate 17.0 secs, was a case of myocardial degeneration secondary to chronic bronchitis and emphysema.

No. 109, whose circulatory rate was 17.0 secs, was a female patient, who had undergone the operation of thyroidectomy a year

GROUP VI

Myocardial Degeneration

The 5 members of this group, whose ages ranged from 23 to 63 years, were classified as suffering from myocardial degeneration of varied or unknown aetiology.

Praecordial pain or discomfort was the principal symptom in all, while 4 cases complained of dyspnoea on exertion. Arterial hypertension was absent in all cases, and cardiac failure was excluded by the history and physical examination.

The circulatory rate ranged from 10.2 secs to 17.6 secs. with an average rate of 13.9 secs - approximately equal to the average normal rate of 14.35 secs.

Case No.26, with a circulatory rate of 11 secs, showed evidence of myocardial degeneration on electrocardiographic examination. No adequate cause for this was found.

Case No.83, circulatory rate 13.6 secs, gave a history suggestive of recent coronary thrombosis, although this assumption was not supported by the electrocardiogram.

Case No.104, circulatory rate 17.2 secs, was a case of myocardial degeneration secondary to chronic bronchitis and emphysema.

No.109, whose circulatory rate was 17.6 secs, was a female patient, who had undergone the operation of thyroidectomy a year

previously. The early history of growing pains and the cardiac signs, suggested a rheumatic endocarditis.

Case No.110, circulatory rate of 10.2 secs, showed clinical enlargement of the left ventricle, and electrocardiographic evidence of myocardial degeneration.

The average circulatory rate of 13.9 secs in this group agrees with the approximately normal rate found by Blumgart and Weiss (radium emanation method), in similar pathological cardiac conditions.

GROUP VII

'Effort Syndrome'

4 patients, all males, aged 24-44 years, constitute this group.

In all 4 cases praecordial pain on exertion was complained of, and in 3 effort dyspnoea was an additional cause of disability. General symptoms complained of were giddiness, palpitation, and exhaustion. In 2 of the cases shock was blamed for the onset of the symptoms. All cases had refrained from work owing to their disability.

The circulatory rate in this group ranged from 6.2 secs to 10.4 secs - an average rate of 8.2 secs, which is 6 secs less than the normal average rate of 14.35 secs, and is slightly less than the average rate of 9 secs obtained in the thyrotoxic group.

In cases No.55, 57 and 76, with circulatory rates of 7.4, 6.2 and 10.4 secs respectively, the blood pressure readings were of the high normal range. This and the moderate tachycardia, present in all cases, were the sole abnormalities detected on clinical examination. The electrocardiograms showed slight departures from the normal in all cases.

The 4 cases exhibited tremor in the outstretched hands and brisk or exaggerated deep reflexes.

In Cases No.55 and 57, nystagmus was observed, Case No.57 being a case of 'Miner's nystagmus'. In this patient the thyroid

gland was palpably enlarged. Case No.106 admitted a previous luetic infection, but this had been adequately treated, and the blood Wassermann reaction was negative.

The diagnosis of 'Effort syndrome' in these cases was arrived at by a process of exclusion. Some difficulty was experienced in differentiating the syndrome from thyrotoxicosis, the 2 conditions showing a similar increase in the pulse pressure, and signs of nervous imbalance being common to both. In Case No.57, although the thyroid gland was palpably enlarged, the patient weighed over 12 stones and no loss of weight was admitted.

In this group the signal reaction was characterised by a sharp onset and deep increase in respiratory amplitude. Case No.55, on experiencing the respiratory stimulation, 'collapsed' and complained of severe praecordial pain, which was stated to last for half an hour afterwards.

The rapid average circulatory rate of 8.2 secs obtained in this group is in keeping with the quick rates found by Bain in this syndrome, and measured by means of the histamine method, and observations of Tarr, Oppenheimer and Sagar who, in 4 patients with neuro-circulatory asthenia whose basal metabolic rates were normal, found quickened circulatory rates.

In Case No.55, following the increase in respiratory amplitude severe substernal pain, lasting 10 secs, was experienced. The blood pressure (systolic) taken immediately was found to have risen 10 mm. Hg.

GROUP VIII

Angina Pectoris

3 patients, males, aged 44-52 years, constitute this group. The members of this group were not considered to be suffering from arterial hypertension, there was no history of antecedent luetic infection, and the blood Wassermann reaction was in all cases negative. Effort dyspnoea was complained of in the 3 cases, but pain was the presenting symptom. This pain had a typical radiation in all the cases and was induced by exertion and relieved by rest.

The circulatory rate in this group ranged from 11.6 secs to 13.4 secs - an average rate of 12.4 secs or nearly 2 secs quicker than the average normal 14.35 secs. Case No.38 was a severe case of angina pectoris, the patient's walking capacity being limited to 100 yards. The circulatory rate was 12.2 secs in this case.

In Case No.111, circulatory rate 11.6 secs, the pain had recently occurred at rest, and had been accompanied by dyspnoea. There was a suggestion of gallop rhythm present. In this case, and in No.72, both patients admitted to being heavy cigarette smokers.

In Case No.38, following the increase in respiratory amplitude severe substernal pain, lasting 10 secs, was experienced. The blood pressure (systolic) taken immediately was found to have risen 10 mms. Hg.

The average circulatory rate of 12.4 secs in this group of 3 cases of angina pectoris, without signs of cardiac failure, is in contrast with the prolongation of the histamine flush time obtained by Bain in cases of effort angina. This observer concluded that effort angina should be regarded as a symptom of failure - meaning that the heart has failed to keep pace with the needs of the circulation.

In Case No. 5, circulatory rate 17.6 secs, the atrricular fibrillation was the result of 'stale' myocardial degeneration. This case was complicated by the presence of chronic pulmonary tuberculosis and emphysema. The temperature was normal for some few weeks prior to the estimation. The ventricular rate was 79, and no digitalis had been administered.

Case No. 89, circulatory rate 15.8 secs, gave a history of previous failure. She had been heavily dosed with a digitalis preparation and had just recovered from an attack of nausea and vomiting due to this. The electrocardiographic appearance supported this assumption.

In Case No. 120, circulatory rate 15.8 secs with a history of 5 attacks of rheumatic fever, there was no clear previous history of failure, but the patient had been given large doses of digitalis for 7 weeks prior to the estimation.

GROUP IX

Auricular Fibrillation

The 3 patients, aged 35-64 years, included in this group, were suffering from auricular fibrillation without signs of circulatory failure detectable on clinical examination. The circulatory rate ranged from 15.8 secs to 17.6 secs - an average rate of 16.4 secs, or 2 secs longer than the average normal rate of 14.35 secs.

In Case No.6, circulatory rate 17.6 secs, the auricular fibrillation was the result of 'senile' myocardial degeneration. This case was complicated by the presence of chronic pulmonary tuberculosis and emphysema. The temperature was normal for some few weeks prior to the estimation. The ventricular rate was 78, and no digitalis had been administered.

Case No.89, circulatory rate 15.8 secs, gave a history of previous failure. She had been heavily dosed with a digitalis preparation and had just recovered from an attack of nausea and vomiting due to this. The electrocardiographic appearance supported this assumption.

In Case No.120, circulatory rate 15.8 secs with a history of 5 attacks of rheumatic fever, there was no clear previous history of failure, but the patient had been given large doses of digitalis for 7 weeks prior to the estimation.

Cases No.89 and 120 are both examples of over-digitalisation. It may be assumed that the velocity of the blood flow in these 2 cases was slower previous to the administration of the drug.

Blumgart and Weiss (radium emanation method), found that a definite increase in the velocity of the blood flow occurred subsequent to the administration of digitalis. This was demonstrated in 6 out of 8 cases of congestive failure, to whom digitalis had been administered. Blumgart and Weiss also observed that the circulatory rate was unaffected in normal cases to whom the drug had been given, and they concluded that digitalis has no stimulating effect on the circulation, but tends to increase the speed of the blood flow only when the latter is already slowed.

GROUP X

Anaemia

3 patients, aged 39, 50 and 57 years respectively, are included in this group. All complained of general symptoms of anaemia viz. dyspnoea on exertion, palpitation, faintness etc. All cases were free from signs of congestive failure. The circulatory rate in the group ranged from 9.8 secs to 10.8 secs - an average rate of 10.4 secs, or approximately 4 secs quicker than the average normal rate of 14.35 secs.

In Case No.29, circulatory rate 10.6 secs, the haemoglobin percentage was 58. The cause of the anaemia was not ascertained in this case, but there was a history of malaria. In addition the patient showed signs of arterio-sclerosis.

Case No.62 was suffering from an advanced degree of anaemia, secondary to abdominal carcinoma, and was markedly emaciated, weighing 6 st. 6 lbs. The circulatory rate measured 9.8 secs.

In Case No.63, circulatory rate 10.8 secs, suffering from dysphagia, in addition to anaemia, the diagnosis made was microcytic anaemia - Plumer-Vincent syndrome.

The average circulatory rate of 10.4 secs in this group agrees with the results obtained by other methods. Blumgart, Gargill and Gilligan, using the radio-active method in cases of anaemia free from congestive failure, noted that the velocity of the blood flow tended to increase in proportion to the degree of anaemia.

The pulse rate was observed by them to be more closely related to changes in the velocity, than to variations in the degree of anaemia.

Tarr, Oppenheimer and Sagar (decholin method) in a study of 18 cases of anaemia, free from signs of congestive failure, observed a slight quickening of the circulatory rate - the average rate being 1 sec shorter than the average normal.

Bain concluded that in cases of anaemia the histamine flush time was quickened, but as anaemic patients failed to flush satisfactorily his results are indefinite.

GROUP XI

Acute Rheumatic Endocarditis - Pyrexia

The 2 patients studied under this heading were males, aged 17 and 27 years.

The circulatory rates were 7.3 secs and 7 secs respectively - an average rate of 7.2 secs, or approximately half the normal average rate of 14.35 secs.

Case No.49, circulatory rate 7.4 secs, gave a history of previous acute rheumatism, and a recent similar attack a week prior to examination. Signs of endocarditis were evident, and at the time of estimation of the circulatory rate the heart rate was 92, and temperature 100°.

Case No.92, circulatory rate 7 secs, gave a history of rheumatic fever 4 weeks previously, and at the time of examination was still complaining of joint symptoms. The temperature was 99.8° and heart rate 100. The presence of developed mitral stenosis was noted.

The above average circulatory rate of 7.2 secs is the quickest average rate in the groups studied in this series. The increase in the velocity of the blood flow is largely due to the increase in basal metabolism occasioned by the pyrexia.

In a case of bacterial endocarditis with pyrexia, Tarr, Oppenheimer and Sagar observed a much quickened circulatory rate.

Blumgart and Weiss, in 6 patients convalescent from rheumatic

fever but without evidence of valvular damage, found a slightly quickened circulatory rate. Their cases were apyrexial at the time of examination. This increase in speed was attributed to cardiac hyperactivity following upon the infection.

2 patients (males) aged 44 and 47 years, are classified in this group. The circulatory rates in the 2 cases were 22 beats and 27.6 beats. An average rate of 24.8 beats or 30% above the average normal of 14.55 beats.

Case No. 7, circulatory rate 22 beats, weighed 14 st. 5 lbs, and complained of effort dyspnea and increasing lethargy. A gynaecological operation at the age of 24 years had resulted in an artificial menopause. The heart rate was 68 and temperature sub-normal at the time of examination.

This patient had been receiving treatment with thyroid extract 6 months prior to the estimation of the rate.

Case No. 37, circulatory rate 27.6 beats, had received no treatment for the condition. Arterial hypotension was present. This case was a more typical example of the hypo-thyroid state. Physiological estimations of the basal metabolic rates were not made.

The average circulatory rate of 24.8 beats in this group, demonstrating a marked prolongation of the velocity of the blood flow, is comparable with the slowing observed by Hillgart, McGill and Gillies, who found that the degree of slowing in the blood velocity in myxedema corresponded closely with the degree to which the basal metabolic rate was lowered. They also observed that

GROUP XII

Hypo-Thyroidism

2 patients (females) aged 44 and 47 years, are classified in this group. The circulatory rates in the 2 cases were 22 secs and 27.6 secs. An average rate 24.8 secs or 10 secs slower than the average normal of 14.35 secs.

Case No.7, circulatory rate 22 secs, weighed 14 st. 5 lbs, and complained of effort dyspnoea and increasing lethargy. A gynaecological operation at the age of 24 years had resulted in an artificial menopause. The heart rate was 60 and temperature sub-normal at the time of examination.

This patient had been receiving treatment with thyroid extract 6 months prior to the estimation of the rate.

Case No.37, circulatory rate 27.6 secs, had received no treatment for the condition. Arterial hypertension was present. This case was a more typical example of the hypo-thyroid state. Biochemical estimations of the basal metabolic rates were not made.

The average circulatory rate of 24.8 secs in this group, demonstrating a marked prolongation of the velocity of the blood flow, is comparable with the slowing observed by Blumgart, Gargill and Gilligan, who found that the degree of slowing in the blood velocity in myxoedema corresponded closely with the degree to which the basal metabolic rate was lowered. They also observed that

following thyroid therapy the rise in metabolic rate, and increase in velocity of blood flow, took place simultaneously and closely paralleled each other.

GROUP XIII

Only 1 patient, who was unclassified, was assigned to this group.

Case No.71. The circulatory rate measured 15 secs. This was a case of sinus bradycardia, due to excessive vagal action. He gave a history of several 'collapses' and had been diagnosed as suffering from the Adams-Stokes syndrome.

gradually increased, the slopes of the two curves being almost identical. The alkaline change was also checked by pH determinations. The loss of cyanide is probably due to volatilization of HCN produced by ionization, leaving residual CN^- in solution. The loss of chemical activity does not parallel the loss of physiological activity which falls off at a great rate, many solutions being physiologically inactive in seven days, and in some cases even shorter time.

Estimation.

The cyanide was estimated in urine etc., by the usual methods of toxicological analysis and in the solutions for the stability tests by ordinary volumetric methods.

Factors affecting excretion.

It was first considered necessary to examine the quantity of cyanide present at the end of various time intervals. This was done in three cases, the results of which indicated that thirty

THE EXCRETION OF SODIUM CYANIDE WHEN
ADMINISTERED INTRAVENOUSLY IN SMALL DOSES

Stability.

Solutions were kept in a cool, dark room and analysed at various time intervals. At no time was any solution found to contain ammonia, carbonate, or formate, but the concentration of cyanide steadily decreased whilst the alkalinity of the solution steadily increased, the slopes of the two graphs being almost identical. The alkaline change was also checked by pH determinations. The loss of cyanide is probably due to volatilisation of HCN produced by ionisation, leaving residual NaOH in solution. The loss of chemical activity does not parallel the loss of physiological activity which falls off at a great rate, many solutions being physiologically inactive in seven days, and in some cases even shorter time.

Estimation.

The cyanide was estimated in urine etc., by the usual methods of toxicological analysis and in the solutions for the stability tests by ordinary volumetric methods.

Factors affecting excretion.

It was first considered necessary to examine the quantity of cyanide present at the end of various time intervals. This was done in three cases, the results of which indicated that thirty

minutes was an ample time interval. From 15% to 18% was excreted in this time, whilst in the following period of thirty minutes, from 1.3% to 3.0% was excreted and in subsequent periods from 0.0% to 0.9%.

The condition of the bladder at the start of the experiment appears to affect the percentage excretion. In six cases the average percentage cyanide excreted with an empty bladder was 17.6%, the lowest result being 15.0%. For the same cases with the bladder not emptied before the experiment the average was 13.2%, the highest result being 14.0%.

The quantity of cyanide voided in the urine was remarkably constant and was capable of duplication when the same case was examined with a day or so between each injection.

Varying dosage of the cyanide from 4 to 8 mgms did not cause a corresponding variation in the percentage excreted in the urine which was roughly constant at 18% with two cases, and 24% with a third case.

One case with a dose of 8 mgms noticed a cyanide flavour in the mouth after the injection, the saliva was kept and on examination was found to contain 1.1 mgms or 14.0% of the injected cyanide.

Normal and pathological heart cases each excrete the same percentage cyanide in the same time. This percentage is 17.65% with a deviation from 12.3% to 23.7% in twenty-three cases with empty bladders in half an hour. The concentration per unit volume per unit time was not constant and an increased percentage output would be expected after the use of diuretics.

Diuretics used on five cases were water, diuretin, ammonium chloride, and salyrgan; the results obtained varying from 24.3% to 78%, a marked increase over the 17.3% obtained on the same cases without diuretics.

The distribution between urine and breath was examined in one case, but due to the inconvenience of the apparatus it could not be applied to other cases, although the excretion was checked by the use of test papers, it was shown that cyanide was mainly excreted in the early stage of the reaction. In the one case examined 75% was found in the breath in half an hour, and 12.5% in the urine in the same time.

In all cases the urine was examined before the injection for cyanide and in no case could it be found in detectable quantity.

The urine was examined in many cases before and after the experiment for albumin, blood, reducing substances, and a microscopical examination was made of any deposit. In no case was any abnormal constituent found but which was originally present, in some cases a deposit of phosphates was produced that could be accounted for by change of concentration as measured by specific gravity.

DISCUSSION

In the present study of the velocity of the blood flow in cardiovascular disease, the pathway selected for measurement of the circulatory rate was the arm-carotid route. No attempt was made to divide this portion of the circulatory system into its component parts.

On reviewing the average circulatory rates in the 13 classified groups in this study, the 2 conditions of clinical importance, in which a slowing of the velocity of the blood flow was found to occur, were congestive heart failure and hypo-thyroidism.

The term congestive heart failure denotes passive engorgement, occurring as a result of cardiac disease, whether appearing in the pulmonary vascular bed, the systemic circuit, or in both of these areas.

The group under review included cases of so-called left ventricular failure, i.e. pulmonary engorgement consequent upon a lowering of the reserve power of the left ventricle.

This isolated left-sided failure is encountered only when the functional capacity of the right ventricle is adequate to master the increased resistance in the pulmonary circulation, which results from inefficiency of the left side of the heart. The dynamics of the circulation are then so altered that the pressure in the systemic veins is unchanged despite the engorgement of the lesser circulation.

In the cardiac failure group in all cases in which signs of pulmonary stasis were detected, the circulatory rate was prolonged, independent of signs of right-sided failure.

The diminution in the velocity of the blood flow in congestive heart failure has been interpreted as indicative that the cardiac output is likewise diminished. Harrison,⁽¹⁷⁾ in his study of the dynamics of heart failure has demonstrated that ample evidence exists to prove that the fundamental disturbance in failure occurs, first, behind the left ventricle, and consists in a rise in the capillary and venous pressures.

Statistical analysis of the data of Blumgart and Weiss indicates that slowing in the blood flow through the lungs, precedes this rise in venous pressure. Blumgart maintains that a stage of engorgement is present prior to the increase of pressure in the veins, and that it is only after the veins have become filled to the limit of their capacity that additional amounts of blood cause a rise in pressure.

A correlation of the results of the circulatory rate measurements in failure, with the degree of disability of the patients, failed to yield a strict relationship. In general, however, the prolongation of the rate was found to parallel the severity of the condition.

The speed of the blood flow in the 2 cases of hypo-thyroidism approximated that found in the cardiac failure group, although the 2 patients were free from evidence of circulatory insufficiency.

In the case receiving thyroid therapy, the circulatory rate

was 5.6 secs quicker than in the untreated case. The increase in blood velocity occurring when the metabolic rate is raised, and necessitating an increased amount of work by the heart, has been held by some observers to offer a rational explanation for the development of clinical manifestations of myocardial insufficiency following thyroid therapy. The striking increase in the velocity of the blood flow in the thyrotoxic group emphasizes the strain under which the heart labours in this condition.

The increased velocity of blood flow in thyrotoxicism probably occurs to meet the changes of the elevated basal metabolic rate, and not as a result of a toxic effect on the heart.

The 2 cases in this group, complicated by signs of cardiac failure, showed circulatory rates approximately normal. In these 2 patients the signs of the overactive gland were detectable.

Thyroid gland dysfunction, and other forms of heart disease, frequently co-exist in the same individual, and the evidence of the thyroid activity may be very obscure.

The finding of a normal circulatory rate in the presence of circulatory failure, should occasion search for other factors tending to increase the blood velocity, e.g. hyperthyroidism, pyrexia, and severe anaemia.

In these 'masked thyrocardiacs' the diagnosis of the thyroid dysfunction is extremely important from the point of view of successful treatment.

Markedly increased circulatory rates were found in the 4 cases classified under the 'effort-syndrome' group. This syndrome,

which is synonymous with the terms neuro-circulatory asthenia, and 'irritable heart', resembles closely the condition of thyrotoxicosis and differential diagnosis is often difficult or impossible.

Some observers have found the exophthalmos and enlargement of the thyroid gland present in 50% of their cases of neuro-circulatory asthenia. Other writers regard many cases diagnosed as 'effort syndrome' subjects as cases of atypical Graves' disease.

The criteria of diagnosis appear to be the absence of a marked loss of weight, and a normal heart rate during sleep and rest.

The increased velocity of the blood flow noted in the 3 cases of anaemia, is a mechanism whereby an adequate supply of oxygen is maintained to the tissues. The greater the diminution in the haemoglobin percentage, the greater the extent to which the blood supply must be increased to supply the adequate amount of oxygen.

SUMMARY AND CONCLUSIONS

The term - velocity of blood flow - has been defined, and a historical resumé of the problem, associated with its measurement, enumerated.

The various methods devised for the estimation of the circulatory rate have been critically reviewed, and their attendant advantages and limitations discussed. The methods, whereby the drugs histamine and sodium cyanide are used as chemical agents, were chosen to measure the circulation time over selected pathways of the circulation.

The histamine method was discarded after a trial owing to difficulty experienced in timing the reaction signal accurately, and the occurrence of unpleasant sequelae. The sodium cyanide was finally chosen.

Sodium cyanide, when injected intravenously in suitable doses produces a temporary but pronounced stimulatory effect on the respiration, characterised by an increase in the respiratory amplitude, and usually in the rate of the respiration.

Simultaneous to, or immediately following this phenomenon, an increase in the heart rate occurs. The effect on the blood pressure is insignificant. The action of the drug is exerted on the carotid sinus.

The respiratory disturbance lasts from 10-20 seconds - a slightly longer time occurring in cases of circulatory failure.

This action of sodium cyanide upon the respiration is eminently suitable as a signal reaction, and is capable of graphic registration.

Mention has been made of the method of preparation of the solution of the drug, and the rate of its decomposition noted. In a number of normal and pathological cases it has been demonstrated that sodium cyanide is rapidly excreted via the lungs and kidney, chiefly by means of the former route. The rate of renal excretion is affected by the presence of preformed urine, and the action of diuretics. No evidence of renal damage has been found to follow the injection of the drug.

In 146 observations no local or serious general untoward results have followed the intravenous injection of the drug in doses of 0.25 to 0.45 c.c. of a 2% solution - representing 5-9 mg. sodium cyanide. The adequate dose has been found to be roughly proportional to the patient's weight.

In cases of circulatory failure slightly larger doses have been found necessary.

The method is entirely objective, not unpleasant, and is entirely independent of the subject's co-operation.

The technique of administration and means of graphic registration are simple, and require a minimum of assistance.

A quick result is furnished and the method can be used at the bedside or in the consulting room. The portion of the circulation designated arm-carotid pathway has been used in all cases.

146 graphs representing the measurement of the circulatory rate

along this pathway are presented. These graphs represent 14 normal subjects and 129 pathological cases.

The average normal circulatory rate equals 14.35 secs. The patients suffering from various aspects of cardiovascular disease have been classified into 13 groups. The results are here summarized:

- (1) Essential hyperpiesia - established hypertension.
Average circulatory rate 12.82 secs.
(Range 9.4 - 21.8 secs.)
- (2) Rheumatic Heart Disease.
Average circulatory rate 12.4 secs.
(Range 8.2 - 19.2 secs.)
- (3) Cardiac Failure.
Average circulatory rate 23.8 secs.
(Range 17.6 - 39.6 secs.)
- (4) Thyrotoxicosis.
Average circulatory rate 9 secs.
(Range 4.4 - 14.6 secs.)
- (5) Specific Heart Disease.
Average circulatory rate 14 secs.
(Range 8.6 - 25.6 secs.)
- (6) Myocardial degeneration.
Average circulatory rate 13 secs.
(Range 10.2 - 17.6 secs.)
- (7) Effort Syndrome.
Average circulatory rate 8 secs.
(Range 6.2 - 10.4 secs.)
- (8) Angina Pectoris.
Average circulatory rate 12.4 secs.
(Range 11.6 - 13.4 secs.)

- (9) Auricular Fibrillation.
Average circulatory rate 16.4 secs.
(Range 15.8 - 17.6 secs.)
- (10) Anaemia.
Average circulatory rate 10.4 secs.
(Range 9.8 - 10.8 secs.)
- (11) Acute Rheumatic Endocarditis - Pyrexia.
Average circulatory rate 7.2 secs.
(Range 7 - 7.4 secs.)
- (12) Hypo-Thyroidism.
Average circulatory rate 24.8 secs.
- (13) Bradycardia. 15 secs.

The velocity of blood flow is an important characteristic of the circulation. In various pathological states of the circulation considerable deviations from the normal are found to be present.

In cases of cardiac failure, regardless of the aetiology, and in cases of hypo-thyroidism, considerable prolongation of the circulatory rate takes place, while in cases of thyrotoxicosis, neuro-circulatory asthenia, and anaemia, quickening of the rate has been shown to occur. The prolongation of the rate in circulatory failure roughly parallels the clinical condition of the patient, improvement in the latter state being reflected in a shortening of the time.

The velocity of the blood flow is therefore a reflection of the condition of the myocardium, and its estimation a useful additional clinical test to evaluate the state of the cardiac reserve. In cases of failure this estimation is of some

prognostic significance.

In patients exhibiting signs of cardio-vascular disease, with adequate cardiac compensation, the velocity of the blood stream is within the normal range. The estimation of the velocity is of value in the differential diagnosis of dyspnoea - a symptom covering many causes. The distinction between dyspnoea resulting from early left ventricular failure and that of bronchogenic origin being a case in point.

The finding of a normal circulatory rate in a case of circulatory failure should indicate a complication such as thyrotoxicosis, pyrexia, or anaemia.

In the present investigation the constant agreement with the results obtained by means of other methods, notably the radium emanation, permit the conclusion that the results obtained by the sodium cyanide method are a trustworthy measurement of the circulatory rate.

The above investigation was carried out at the Liverpool and District Hospital for Diseases of the Heart, and I am indebted to Dr. Harris for permission to examine the cases recorded. For the estimation of the excretion of sodium cyanide, I am indebted to Mr. C.V. James, M.Sc., for his help.

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Case 2.

Sex: Male. Married.

Age: 64 years.

Occupation: Insurance agent.

History: 12 months. Breathlessness on exertion, giddiness, palpitation, attacks of faintness, and exhaustion.

2 weeks ago, while at rest, suddenly felt faint and lost consciousness for a few seconds. Has experienced two similar attacks during the last 10 days.

Previous Illnesses: 'Inflammation of kidneys' 6 years ago.

Family History: Nil relevant.

Habits: Alcohol and tobacco moderate.

Examination: Pale man. Weight 9 stones.

Circulatory System: Heart beat, 80; regular.

Blood-pressure, 180/90

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Mitral area soft systolic bruit.

Aortic 2nd sound accentuated.

Lungs: Breath sounds - harsh vesicular.

No adventitious sounds.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin, trace present.

Nervous System: N.A.D. Optic discs and retinal fields normal in appearance.

Case 2 (continued)

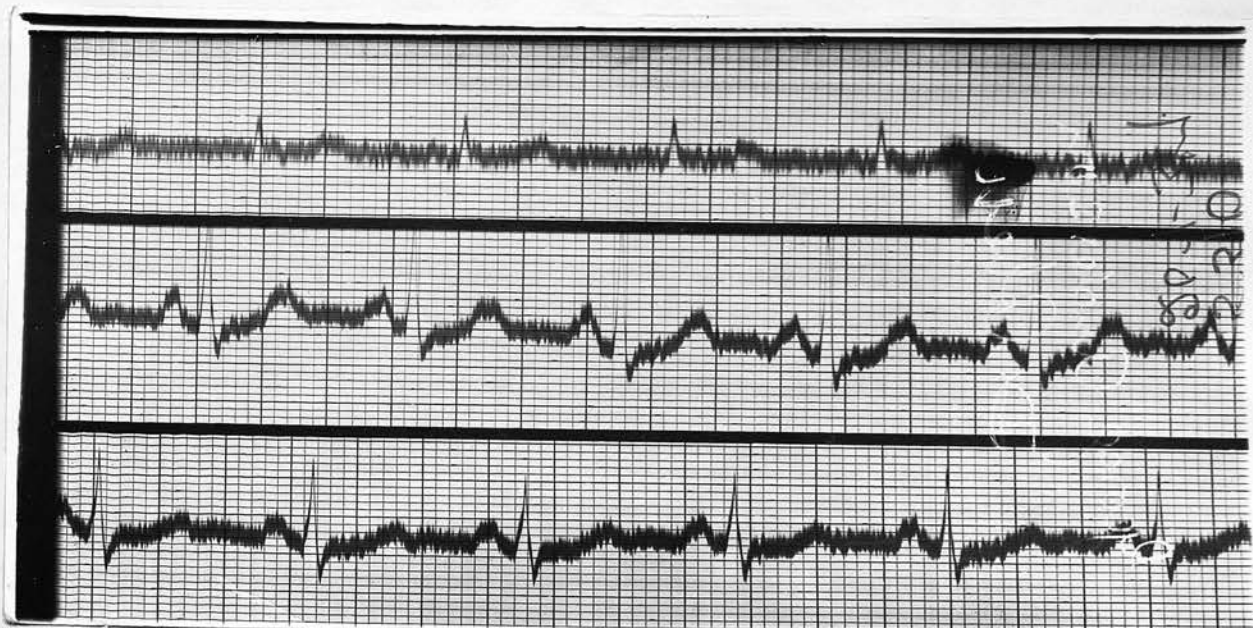
W.R.: Negative.

Electrocardiogram: Nil abnormal.

X-ray Examination: "No appreciable cardiac enlargement.
Chronic inflammatory changes present in lungs."

Diagnosis: Hyperpiesia.

Circulatory Rate: $21\frac{4}{5}$ secs.



Case 9

Sex: Female. Married.

Age: 46 years.

Occupation: Housewife.

History: 6 months. Dyspnoea and praecordial pain on exertion. Upper thoracic constriction, giddiness, exhaustion and somnolence.

3 months. Palpitation on exertion, and at rest.

2 months. Amenorrhoea.

Previous Illnesses: Haemoptysis 6 months ago. X-ray examination at that time showed evidence of chronic bronchitis.

Tonsillitis frequent attacks.

Influenza.

Family History: Childless. No history of miscarriages.

Examination: Obese woman. Weight 14 st. 6 lbs.

Circulatory System: Heart rate, 74.

Blood-pressure, 214/122

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Occasional extrasystole present.

Mitral area systolic bruit.

Aortic 2nd sound accentuated.

Lungs: Rhonchi scattered over both lungs.

Abdomen: N.A.D.

Oedema: Nil.

Case 9 (continued)

Urine: Albumin trace.

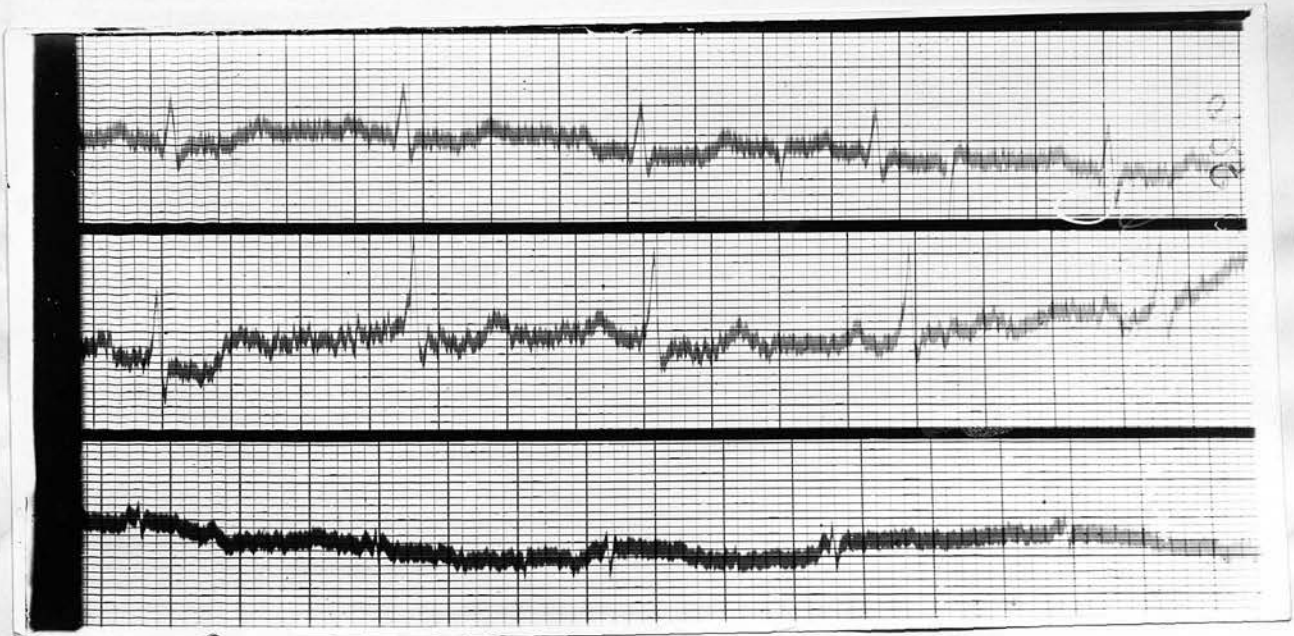
Nervous system: N.A.D.

W.R.: Negative.

Electrocardiogram: Right ventricular extrasystoles.
Left ventricular preponderance.
Lead III flat.

Diagnosis: Hyperpiesia.
Myocardial degeneration.

Circulatory Rate: $9\frac{3}{5}$ secs.



Case 12.

Sex: Female. Widow.

Age: 53 years.

Occupation: Nil.

History: 3 years. Palpitation, tinnitus and insomnia.
Dyspnoea on exertion and lack of energy.

Previous illnesses: 'Rheumatism' aet. 37 years.
Tonsillitis.
Influenza.

Family History: Menopause aet. 49 years. No children.

Examination: Weight 11 st. 9 lbs.

Circulatory System: Heart rate, 70; regular.
Peripheral vessels sclerosed.
Blood-pressure, 244/120.
Apex beat: 6th interspace, outside
mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R.: Negative.

Case 12 (continued)Electrocardiogram: NOT AVAILABLEDiagnosis: Hyperpiesia.
Myocardial degeneration.Circulatory Rate: 11 secs.

Case 13.

Sex: Female. Married.

Age: 55 years.

Occupation: Housewife.

History: 1 year. Palpitation and weakness. Giddiness, insomnia, and nervousness. Occipital headaches and flushings.

Previous Illnesses: Influenza.

Family History: 7 children; 4 miscarriages.
Menopause aet. 47 years.

Examination: Weight 9 st. 12 lbs.

Circulatory System: Heart beat, 80; irregular.
Blood-pressure, 220/104.
Apex beat, not palpable.
Heart sounds: Gallop rhythm present.
Mitral area, systolic bruit.
Aortic area, harsh systolic bruit
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Nervous System: N.A.D.

Case 13 (continued)

Electrocardiogram: P₂ split.
QRS dropped beats.
T₃ inverted.
Left ventricular preponderance.

Diagnosis: Partial heart block.
Myocardial degeneration.
Hyperpiesia.

Circulatory Rate: $14\frac{2}{5}$ secs.



GROUP 1

36 cases

Essential hyperpiesia - established hypertension
Average circulatory rate 12.82 secs.
(Range 9.4 - 21.8 secs.)

Case 15.

Sex: Male. Married.

Age: 48 years.

Occupation: Labourer.

History: 1 month. Attacks of giddiness on changing his position, especially marked on getting out of bed in the morning.

Palpitation, frontal headaches, and insomnia.

Previous Illnesses: Pneumonia 1 year ago - aet. 47 years.
Off work 1 month.

Habits: Alcohol and tobacco moderate.

Examination: Weight 11 st. 7 lbs.
Arcus senilis present.

Circulatory System: Heart rate, 74; regular.
Blood-pressure, 178/100.
Peripheral vessels sclerosed.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

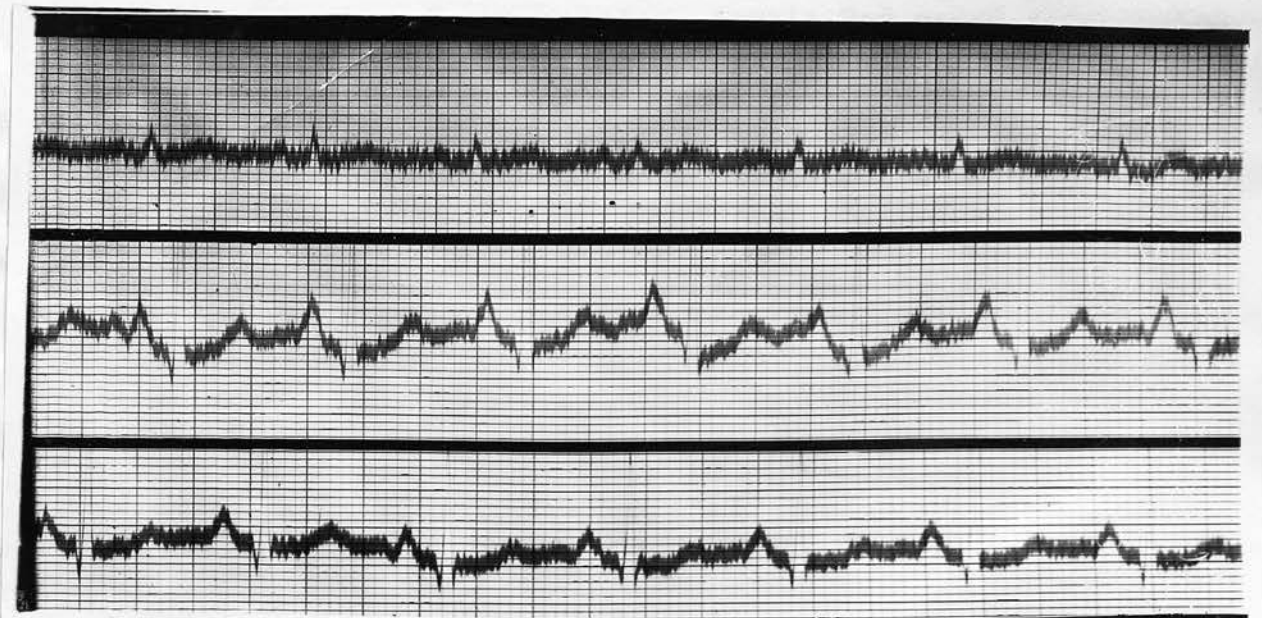
Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R: Negative.

Case 15 (continued)Electrocardiogram: Lead I Low Voltage.R₁ slightly slurred.Q₂ and Q₃ present.Diagnosis: Hyperpiesia.Circulatory Rate: 16 secs.

Case 16.

Sex: Female. Married.

Age: 62 years.

Occupation: Housewife.

History: 5 years. Frontal headaches and giddiness.
4 years ago was informed her blood-pressure was high
and has led an inactive life since.

1 month ago had an attack of influenza for which she
was confined to bed for 5 days. Since then has felt
'shaky and easily exhausted'.

Previous Illnesses: Prolapsus uteri - operation.

Family History: 2 children; history of 3 miscarriages.

Examination: Weight 11 st. 4 lbs.

Circulatory System: Heart rate, 78; regular.
Blood-pressure, 186/106.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

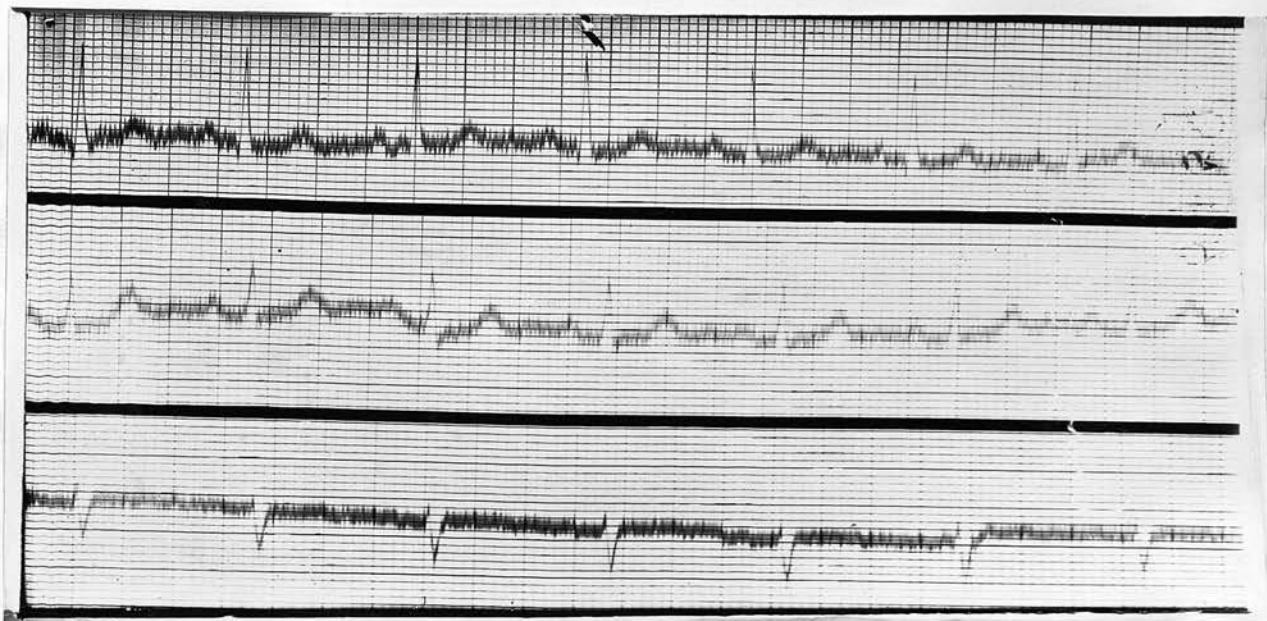
W.R:

Case 16 (continued)

Electrocardiogram: LEFT VENTRICULAR PREPONDERANCE
Q.R.S. SLURRED
T₃ FLAT

Diagnosis: Hyperpiesia.

Circulatory Rate: $14\frac{2}{5}$ secs.



Case 17.Sex: Female. Married.Age: 59 years.Occupation: Housewife.History: 1½ years. Dyspnoea and praecordial pain on exertion.
Palpitation and giddiness.
Rheumatic pains limbs and back.4 weeks ago had an influenzal attack which necessitated
10 days' stay in bed. Her symptoms have been worse since.Previous Illnesses: Growing pains, childhood.
Influenza.
Diphtheria and typhoid fever in childhood.Family History: 22 children. History of 2 miscarriages.
Menopause aet. 46 years.Examination: Obese florid woman. Weight 13 st. 2 lbs.Circulatory System: Heart beat, 80; regular.
Blood-pressure, 224/128.
Apex beat: 5th interspace, just outside
mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 17 (continued)Nervous System: N.A.D.W.R.: NEGATIVEElectrocardiogram: NOT AVAILABLEDiagnosis: Hyperpiesia.Circulatory Rate: $14\frac{2}{5}$ secs.

Case 19.

Sex: Male. Married.

Age: 55 years.

Occupation: Dock labourer.

History: 1 year. Pain in chest, brought on by exertion, and eased by rest. Severity of the pain is such that he is forced to stand still. Pain is lower retro-sternal in position, lasts a few seconds, and radiates towards the left nipple. Can now walk only 300 yards in comfort.

Pain is brought on more readily if walking is done shortly after a meal.

Has been treated for 'Indigestion'.

Previous Illnesses: Nil.

Family History: Nil relevant.

Habits: Alcohol and tobacco very moderate.

Examination: Pale man, weight 12 stone.

Circulatory System: Heart beat, 75; regular.

Blood-pressure, 184/92.

Apex beat: 5th interspace at
mid-clavicular line.

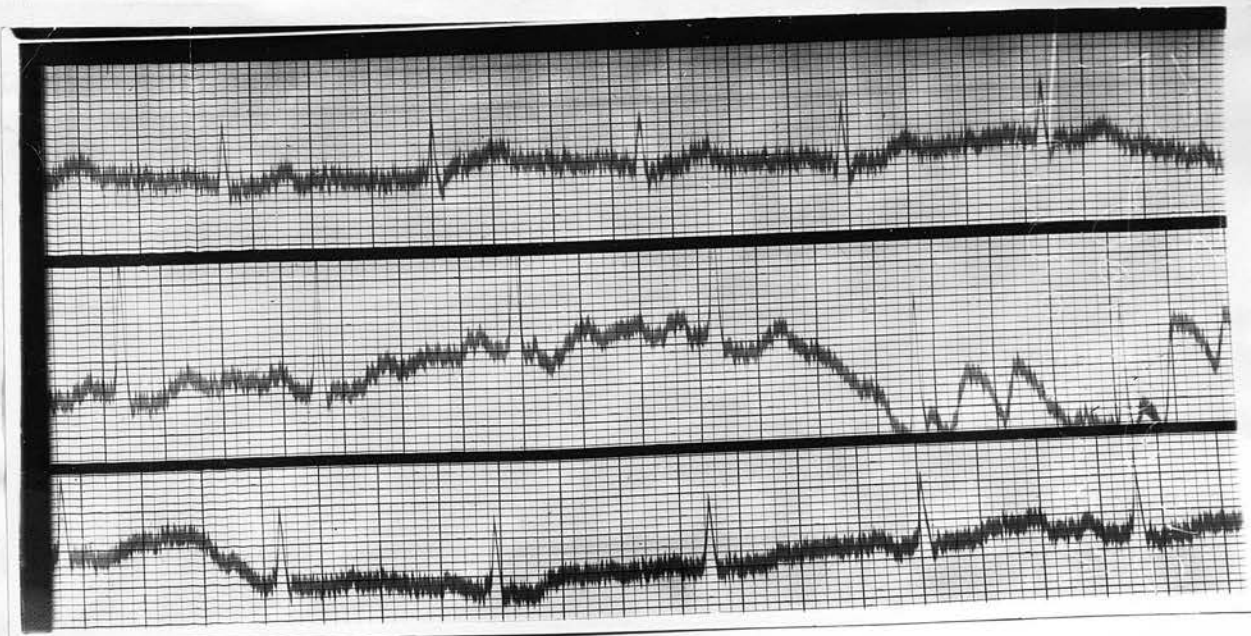
Heart sounds: aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 19 (continued)Nervous System: N.A.D.W.R.:Electrocardiogram: T₃ flat.Diagnosis: Hyperpiesia.
Angina of effort.Circulatory Rate: $10\frac{1}{5}$ secs.

Case 24.

Sex: Female. Married.

Age: 55 years.

Occupation: Housewife.

History: 3 years. Frontal headaches and 'sensation of pressure' in head. Giddiness and tinnitus.

Irritability, insomnia, anorexia, and abdominal discomfort after meals.

1 year. Above symptoms - increased in intensity.

Previous Illnesses: Influenza.
Nephrectomy (right) aet.42. Calculus.

Family History: 5 children. No history of miscarriages.

Examination: Pale woman, weight 8 stones.

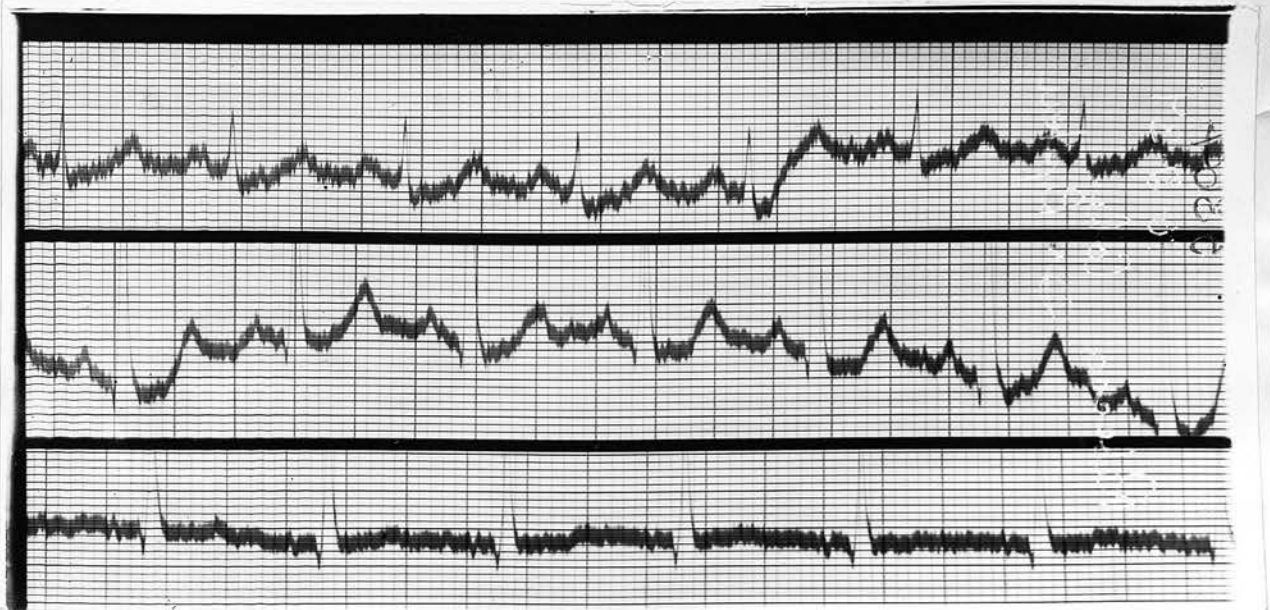
Circulatory System: Heart rate, 78; regular.
Peripheral vessels sclerosed.
Blood-pressure, 208/102.
Apex beat: 5th interspace, just outside mid-clavicular line.
Heaving in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D. Prolapsus uteri; pessary worn.

Oedema: Nil.

Urine: Albumin trace.

Case 24 (continued)Nervous System: N.A.D.W.R: Negative.Electrocardiogram: Q₂ and Q₃ present.
S slurred.Diagnosis: Hyperpiesia.Circulatory Rate: 10 secs.

Case 28.Sex: Female. Married.Age: 59 years.Occupation: Housewife.

History: 1 year. Dyspnoea on exertion, such as climbing one flight of stairs or going up a small hill. More recently dyspnoea after meals. Palpitation on exertion. 6 months. Left sub-mammary pain on exertion. Pain radiates to both shoulders and down the arms. Giddiness.

All the above symptoms have increased during the past 2 months, following an attack of influenza.

Loss of weight (1 stone in last 6 months)

Previous Illnesses: Influenza.
Growing pains, childhood.

Family History: 4 children, no miscarriages.
Menopause aet. 56 years.

Examination: Plethoric woman, weight 12 st. 7 lbs.

Circulatory System: Heart beat, 92; regular.
Blood Pressure, 300 + /130.
Apex beat: 6th interspace outside mid-clavicular line.
Heaving in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Case 28 (continued)

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

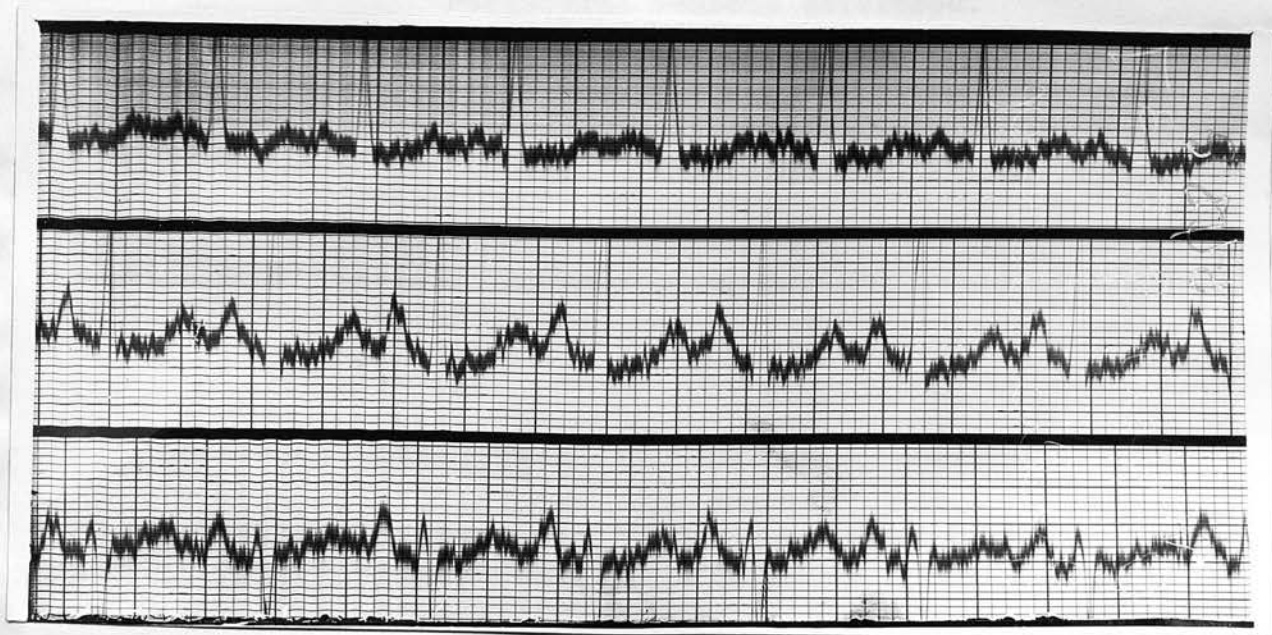
W.R: Negative.

Electrocardiogram: P₂ large.

Left ventricular preponderance.

Diagnosis: Hyperpiesia.

Circulatory System: $10\frac{4}{5}$ secs.



Case 33.

Sex: Male. Married.

Age: 57 years.

Occupation: Joiner.

History: 3 years. Generalised headaches and giddiness.
Headaches come on in 'spasms' and last a day or so.
Eyes recently tested and glasses prescribed.
Palpitation while in bed.
20 years. Abdominal discomfort after meals.
X-ray examination 5 years ago, no organic lesion found.
Unable to work 1 year.

Previous Illnesses: Influenza.

Family History: 4 children.

Habits: Alcohol and tobacco moderate.

Examination: Obese, plethoric man, weight 12 st. 2 lbs.

Circulatory System: Heart rate, 72; regular.
Peripheral vessels sclerosed.
Blood-pressure, 260/140.
Apex beat, not palpable.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin nil; sugar present.

Case 33 (continued)

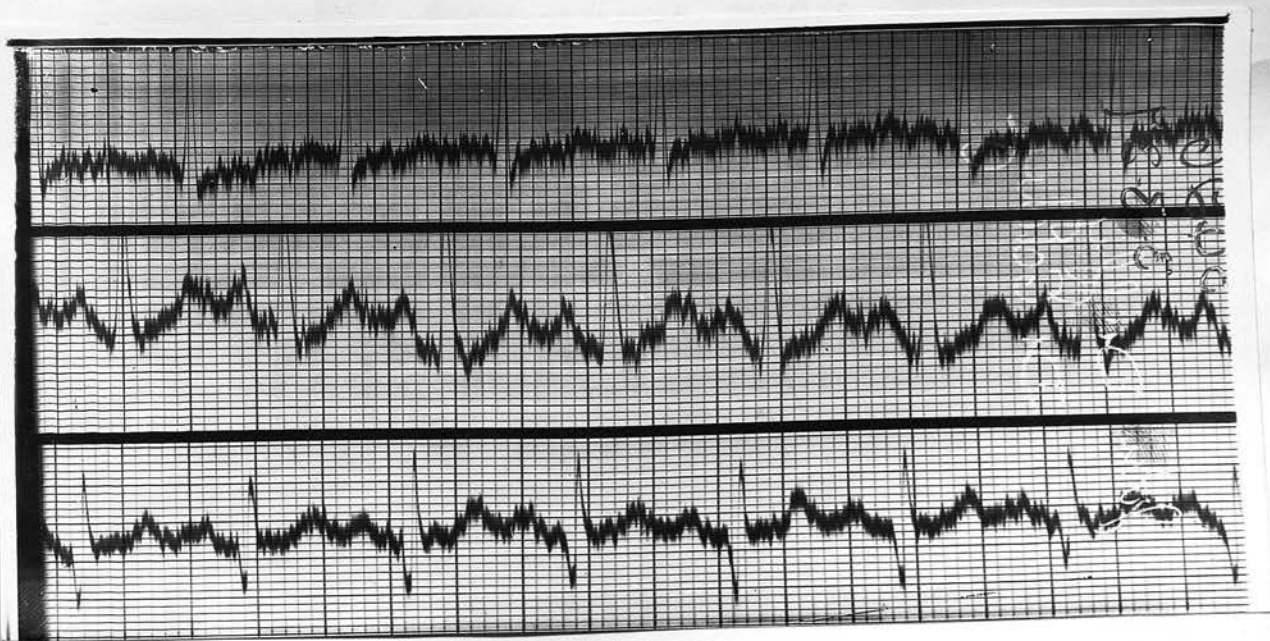
Nervous System: N.A.D. Retinal arteries sclerosed.

W.R: Negative.

Electrocardiogram: Q.R.S. SLURRED.

Diagnosis: Hyperpiesia.

Circulatory Rate: $12\frac{2}{5}$ secs.



Case 35.

Sex: Male. Married.

Age: 46 years.

Occupation: Clerk.

History: 5 years. Occasional severe frontal headaches.
2 years ago, when being examined for Life Insurance purposes, was told his blood-pressure was 'over 180'. Was then treated by rest and a low protein diet.
Now complains of dyspnoea on exertion, headaches, and lack of energy.

15 years. Symptoms suggestive of peptic ulcer.

Previous Illnesses: Nil.

Family History: Nil relevant.

Habits: Alcohol nil; tobacco heavy.

Examination: Weight 11 st. 6 lbs.

Circulatory System: Heart rate, 80; regular.

Blood pressure, 210/115.

Apex beat: 5th interspace, just outside mid-clavicular line.

Forcible in character.

Heart sounds: Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Case 35 (continued)

Urine: N.A.D.

Nervous System: N.A.D.

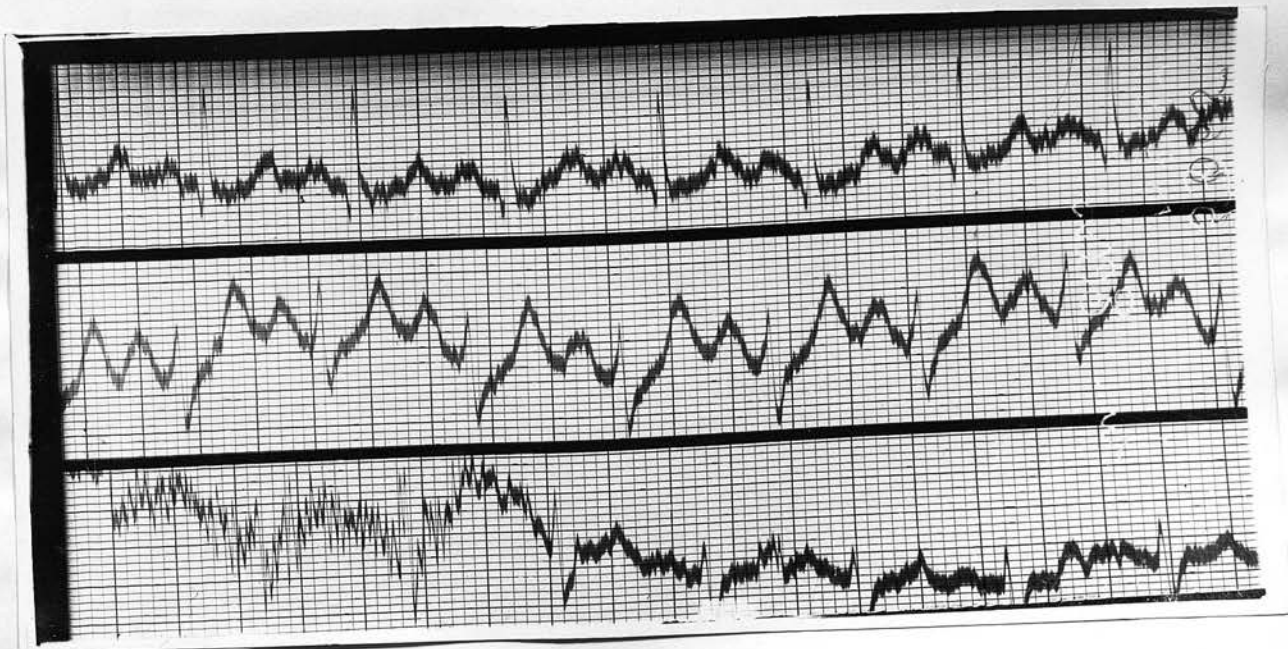
W.R: Negative.

Blood: Non-protein nitrogen, 47 mgm. per 100 ccs.

Electrocardiogram: P₂ large and split.

Diagnosis: Hyperpiesia.

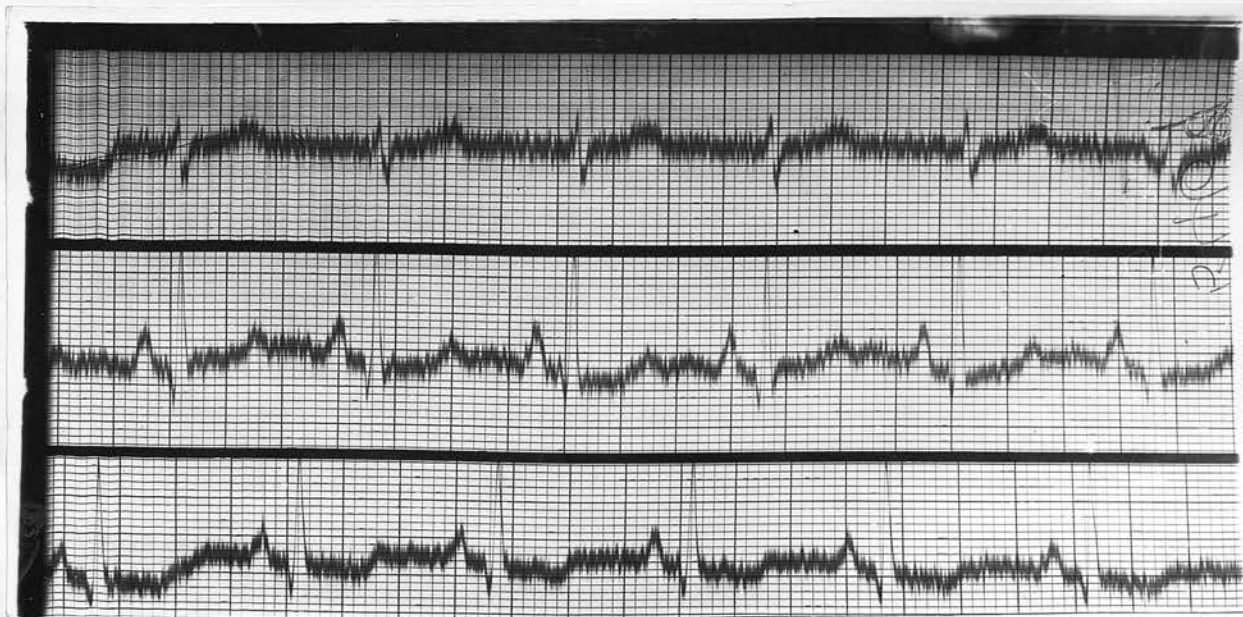
Circulatory Rate: $14\frac{2}{5}$ secs.



Case 47 (continued)Oedema: NilUrine: N.A.D.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: Q₂ and Q₃ present.S₁ slurred.T₃ inverted.Diagnosis: Hyperpiesia.

Myocardial degeneration.

? Paroxysmal auricular flutter.

Circulatory Rate: $12\frac{2}{5}$ secs.

Case 52.

Sex: Female. Married.

Age: 59 years.

Occupation: Housewife.

History: 1 year. Nausea and morning vomiting.

3 months. Dyspnoea and praecordial pain on exertion.
Palpitation, giddiness, and insomnia. Epistaxis.

2 months. Feeling of 'Light-headed'.

Previous Illnesses: Variola and Typhus (!), 1919.

Family History: 4 children; no history of miscarriages.

Habits: Alcohol - immoderate.

Examination: Obese. Weight, 14 st.3 lbs.

Circulatory System: Heart Rate, 72; regular.

Blood-pressure, 210/110.

Apex beat, not palpable.

Heart sounds: Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: Tenderness right hypochondrium. ? Cholecystitis.

Oedema: Nil.

Urine: Albumin trace.

Case 52 (continued)Nervous System: N.A.D.Electrocardiogram: Left ventricular preponderance.S₃ split.T₁ flat.Diagnosis: Hyperpiesia.
Myocardial degeneration.Circulatory Rate: $11\frac{1}{5}$ secs.

Case 58.

Sex: Female. Married.

Age: 51 years.

Occupation: Housewife.

History: 4 years. Giddiness, palpitation, insomnia, and lack of energy.

2 years. Praecordial pain and slight dyspnoea on exertion

Previous Illnesses: Rheumatic fever and growing pains, childhood.

Family History: 3 children; menopause aet. 49 years.

Examination: Florid. Weight 12 st. 12 lbs.

Circulatory System: Heart rate, 78; regular.

Peripheral vessels sclerosed.

Blood pressure, 214/104.

Apex beat: 6th interspace outside
mid-clavicular line.

Heart sounds: Mitral area, systolic bruit.

Aortic area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

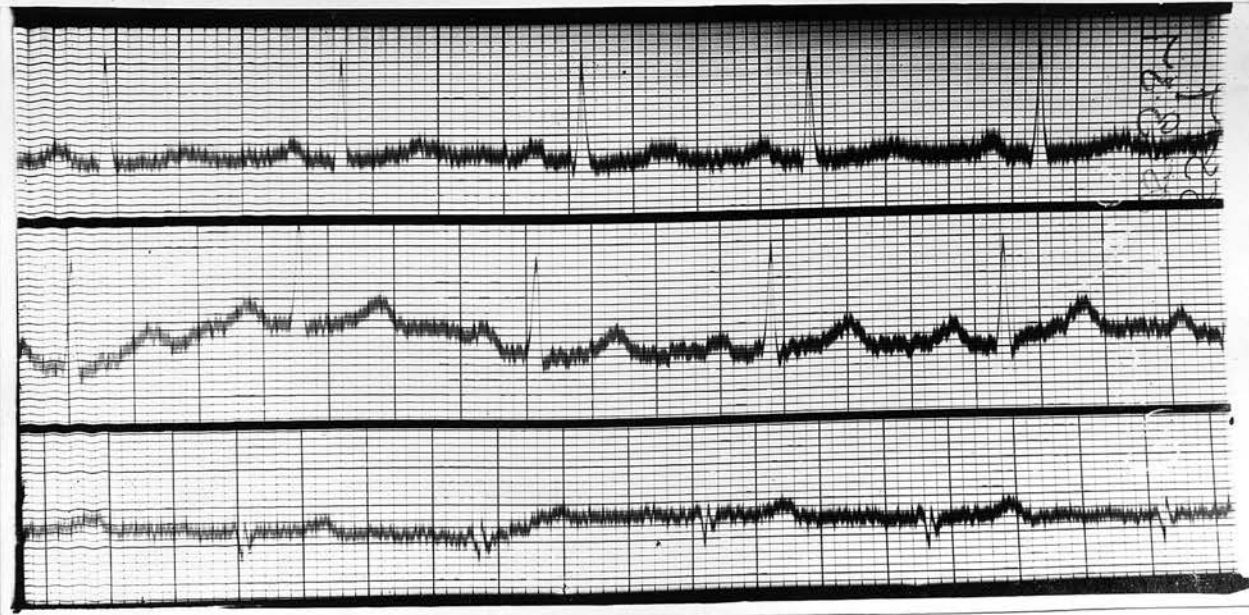
W.R: Negative

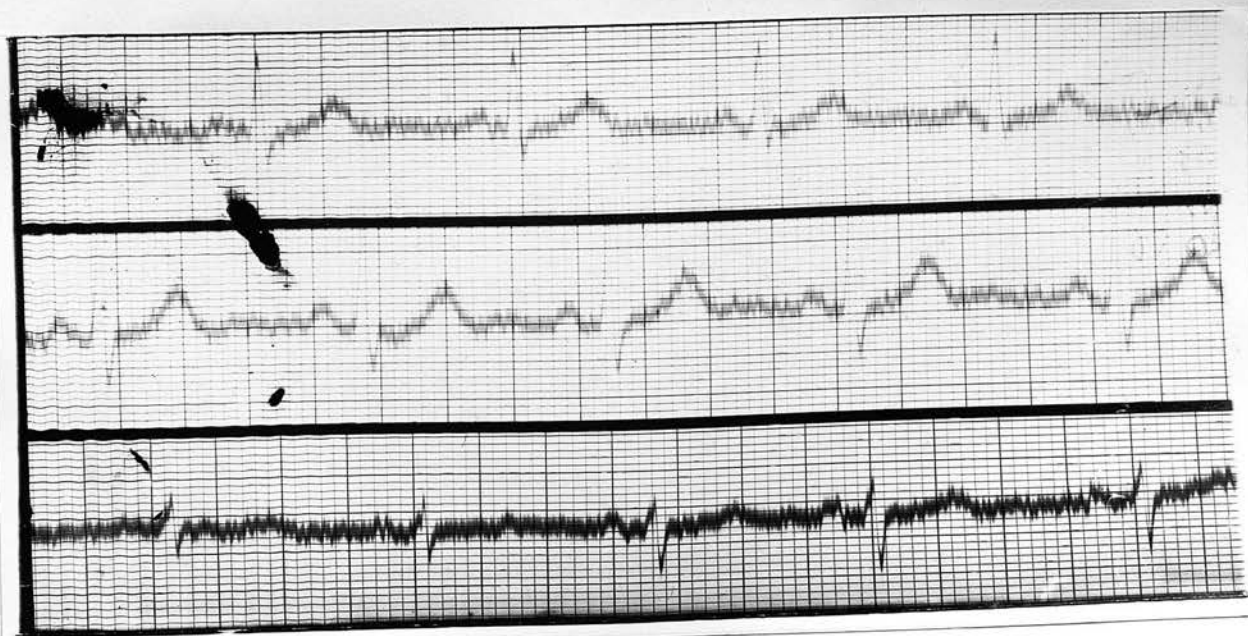
Case 58 (continued)

Electrocardiogram: P - R interval, 0.2 secs.
Left ventricular preponderance.
QRS III slightly slurred.

Diagnosis: Hyperpiesia.

Circulatory Rate: 13 secs.



Case 64 (continued)W.R.: Negative.Electrocardiogram: LEFT VENTRICULAR PREPONDERANCE.
Q.R.S. SLURRED.Diagnosis: Hyperpiesia.
Disseminated sclerosis.Circulatory Rate: $13\frac{2}{5}$ secs.

Case 65.

Sex: Female. Married.

Age: 45 years.

Occupation: Housewife.

History: 4 years. Occipital headaches, palpitation, exhaustion, and giddiness. Praecordial pain on exertion. All symptoms increasing in intensity.

1 year ago had a 'stroke', resulting in a left-sided hemiplegia. Necessitated a period of 10 weeks in bed.

Previous illnesses: Pneumonia aet. 33 years.

Family History: 7 children. 2 miscarriages.
Father died 'stroke'.

Examination: Weight, 10 stones.

Circulatory System: Heart rate, 72; regular.

Blood-pressure, 254/142.

Peripheral vessels sclerosed.

Apex beat: 6th interspace outside
mid-clavicular line.

Heaving in character.

Heart sounds: Mitral area, systolic bruit.

Aortic area, harsh systolic bruit

Aortic 2nd sound markedly
accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Case 65 (continued)

Nervous system: Signs, left hemiplegia.
Retinal arteries sclerosed. Optic discs normal.

W.R: Negative.

Blood: Non-protein nitrogen, 36 mgm. per 100 ccs.

Electrocardiogram: P - R interval greater than 0.2 secs.
Left ventricular preponderance.

Diagnosis: Hypertension. Myocardial degeneration.

Circulatory Rate: $11\frac{1}{5}$ secs.



Case 68.

Sex: Male. Married.

Age: 47 years.

Occupation: Window cleaner.

History: 4 years cough (winter) with slight amount of sputum.
3 years. Increasing dyspnoea on exertion.
2 years. Frontal headaches. Weight increasing.
1 year. Frequency of micturition.

Previous illnesses: Influenza; gonococcal infection aet. 22 years.

Family History: 3 children.

Habits: Alcohol and tobacco moderate.

Examination: Weight 12 st. 12 lbs.

Circulatory System: Heart rate, 78; regular.
Blood-pressure, 168/110.
Apex beat: 5th interspace at mid-clavicular line.
Forcible in character.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 68 (continued)Nervous System: N.A.D.W.R.: NEGATIVEElectrocardiogram: LEAD III LOW VOLTAGEDiagnosis: Hyperpiesia.Circulatory Rate: $11\frac{4}{5}$ secs.

Case 74.

Sex: Female. Married.

Age: 63 years.

Occupation: Housewife.

History: 6 years. Frontal headaches, giddiness, and palpitation.
2 years. Dyspnoea and left sub-mammary 'ache' on exertion.

Treated with thyroid extract recently (dose unknown).

Previous Illnesses: Influenza.
Tonsillitis.

Family History: Nil relevant.

Examination: Weight 11 st. 12 lbs.

Circulatory System: Heart rate, 80; regular.
Blood-pressure, 212/106.
Apex beat: 6th interspace outside
mid-clavicular line.
Heaving in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

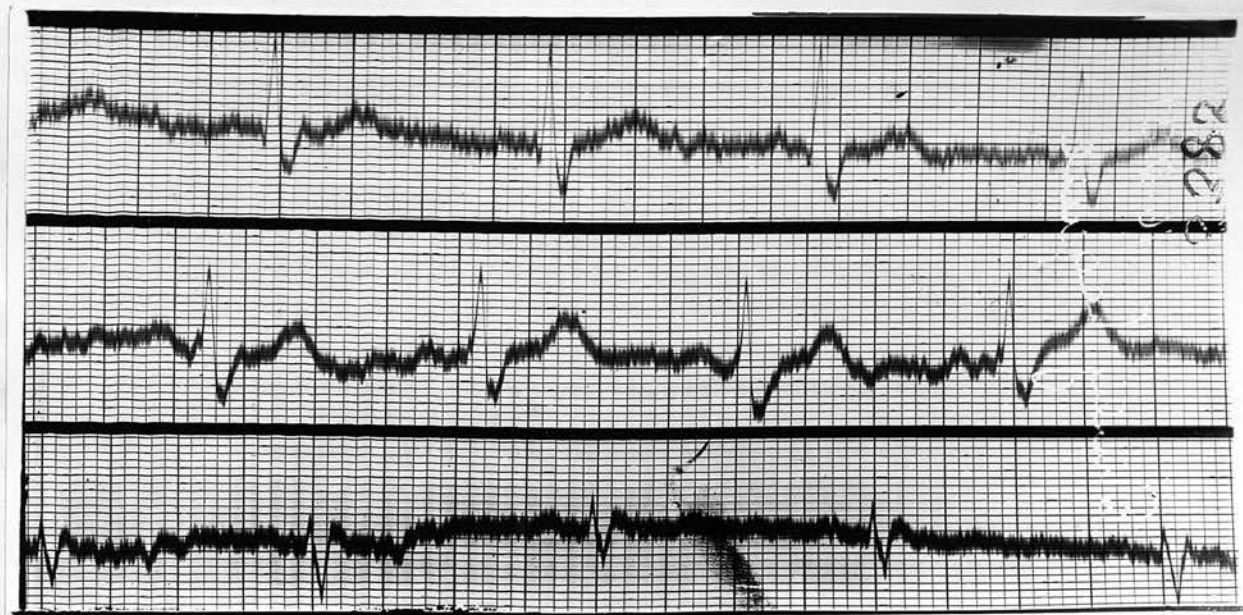
Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

Case 74 (continued)W.R.: Negative.Blood: Blood urea 40 mgm. per 100 ccs.Electrocardiogram: Left ventricular preponderance.T₃ inverted.Diagnosis: Hyperpiesia.

Myocardial degeneration.

Circulatory Rate: $10\frac{2}{5}$ secs.

Case 81.Sex: Female. Single.Age: 50 years.Occupation: Shop assistant.

History: Well until 3 months ago when she experienced sudden pain in her head, followed by unconsciousness 3 - 4 hours. On regaining consciousness found she had lost power in her left leg and left arm. Power returned in a few hours' time. In bed 10 days. Since then giddiness, 'muddled feeling in her head', palpitation and dyspnoea on exertion and insomnia.

Previous Illnesses: Measles
 Whooping cough }
 Scarlet Fever } Childhood.
 Diphtheria } →

Family history: Mother died 'stroke'.Examination. Weight 11 st. 2 lbs. Plethoric.

Circulatory System: Heart rate, 78; regular.
 Peripheral vessels sclerosed.
 Blood-pressure 228/120.
 Apex beat: 5th interspace just outside mid-clavicular line.
 Heaving in character.
 Heart sounds: Mitral area, systolic bruit.
 Aortic area, systolic bruit.
 Aortic 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.

Case 81 (continued)

Oedema: Nil.

Urine: Albumin, trace.

Nervous system: N.A.D.

W.R.: Negative.

Blood: Non-protein nitrogen 33 mgm. per 100 ccs.

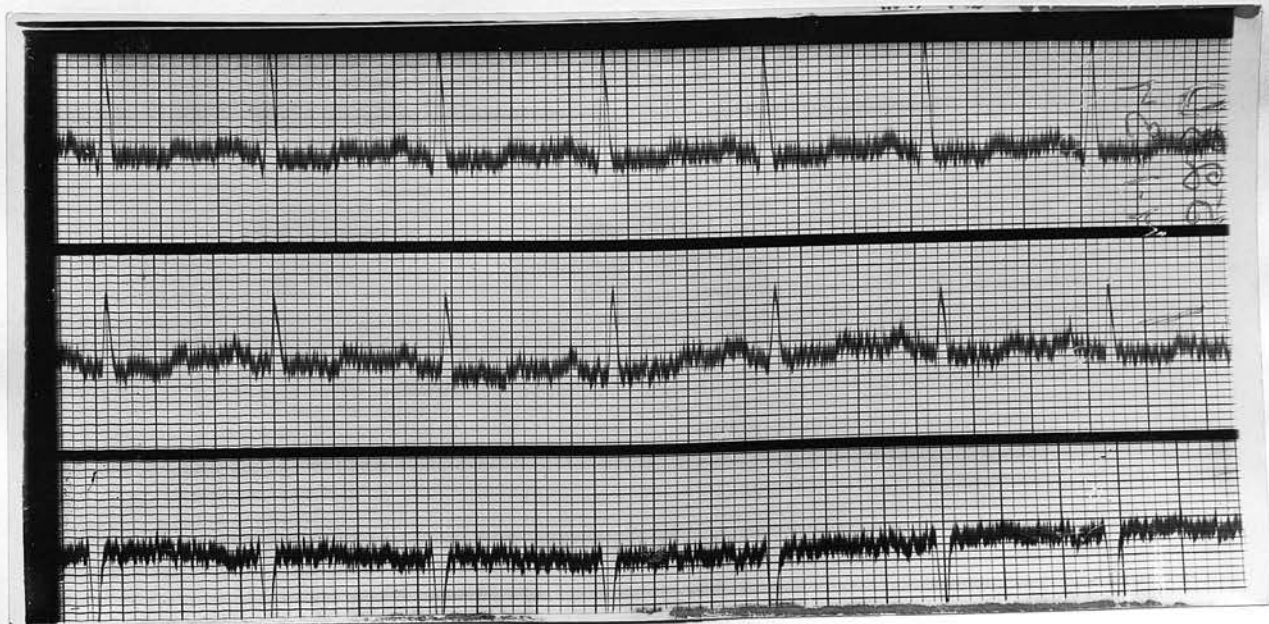
Electrocardiogram: P₂ split.

T₂ inverted; T₁ biphasic.

Left ventricular preponderance.

Diagnosis: Hyperpiesia. Myocardial degeneration.

Circulatory Rate: $9\frac{2}{5}$ secs.



Case 87.

Sex: Male. Married.

Age: 34 years.

Occupation: Insurance agent.

History: 3 weeks. Cough, with small quantity of sputum.
Dyspnoea on exertion.
Palpitation at night, which causes insomnia.

Previous Illnesses: Influenza,

Family History: 2 children.

Habits: Alcohol moderate; tobacco: heavy cigarette smoker.

Examination: Weight, 10 st. 6 lbs.

Circulatory System: Heart rate, 78; regular.

Blood-pressure, 166/100.

Apex beat: 5th interspace at mid-clavicular
line.

Forcible in character.

Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Fine tremor, hand.
Deep reflexes brisk.

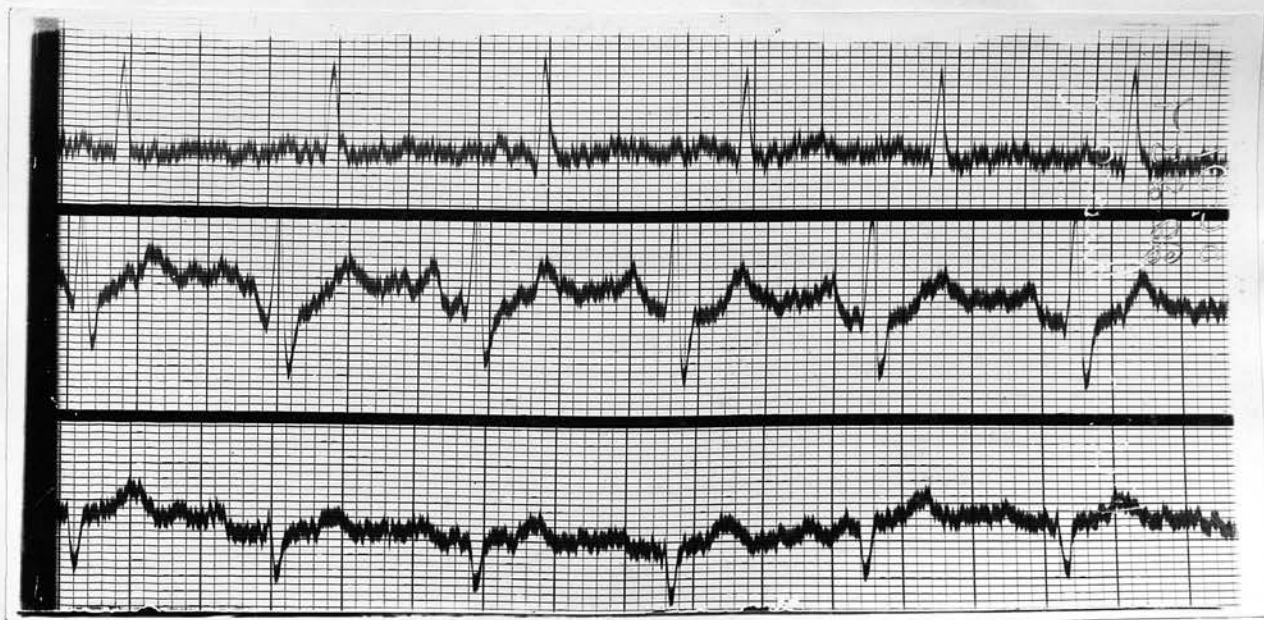
W.R.: NEGATIVE

Case 87 (continued)

Electrocardiogram: Left ventricular preponderance.
S slurred.

Diagnosis: Hyperpiesia.

Circulatory Rate: $11\frac{4}{5}$ secs.



Case 90 (continued)

Nervous System: Slight tremor, hands.

W.R: Negative.

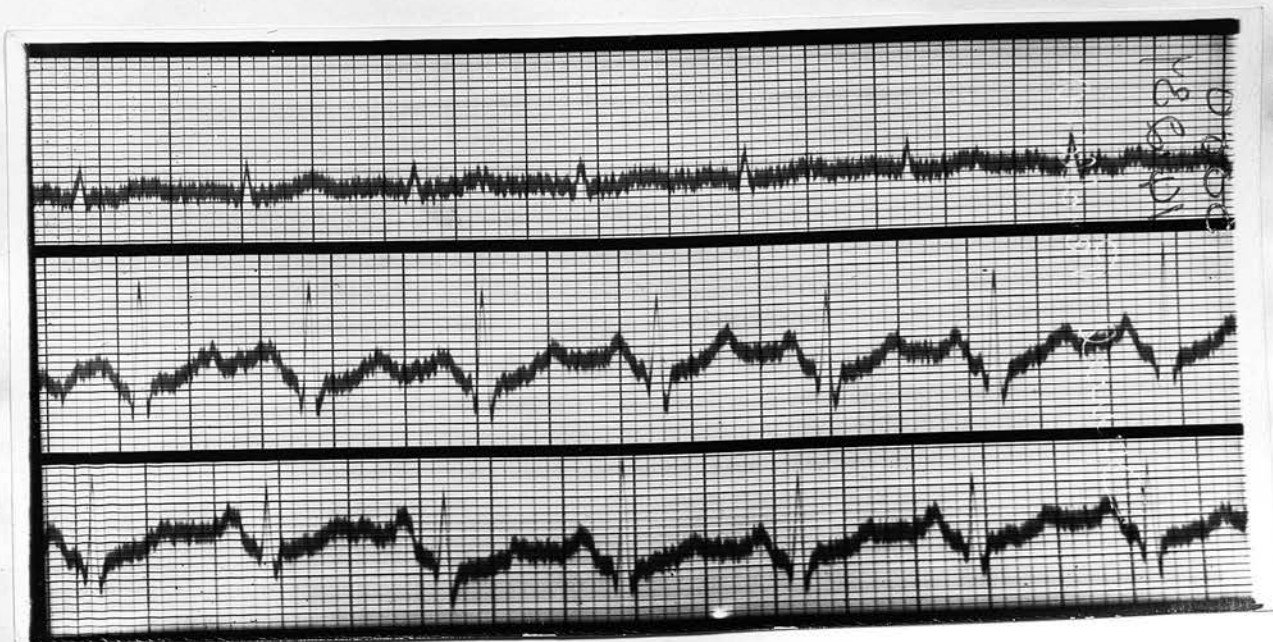
Electrocardiogram: S₂ and S₃ slurred.

Q₂ and Q₃ present.

X-ray Examination: "Heart normal in size. Aortic shadow normal."

Diagnosis: Hyperpiesia.

Circulatory Rate: 14 secs.



Case 93.

Sex: Male. Married.

Age: 64 years.

Occupation: Foreman labourer.

History: 2½ years sub-sternal pain induced by exertion, relieved by rest and nitrites. Pain radiates to left shoulder and down left arm as far as the elbow.

2 years. Dyspnoea and palpitation on exertion.

Previous Illnesses: Nil.

Habits: Alcohol and tobacco moderate.

Examination: Pale. Weight, 11 st.

Circulatory System: Heart rate, 70; regular.

Peripheral arteries sclerosed.

Blood pressure, 180/90.

Apex beat: 6th intercostal space, just outside mid-clavicular line.

Heaving in character.

Heart sounds: Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

W.R: Negative.

Case 93 (continued)

Electrocardiogram: Left ventricular preponderance.
P - R interval greater than 0.2 secs.

Diagnosis: Hyperpiesia.
Angina of effort.

Circulatory Rate: $17\frac{1}{5}$ secs.

Case 94.

Sex: Male. Married.

Age: 59 years.

Occupation: Motor driver.

History: 18 months ago, while driving a car, felt 'a click in his brain', and suddenly lost consciousness for 3 seconds. Same night had a similar attack, lasting only 1 second. 3 days later had 5 attacks within 6 hours. Kept in bed following 3 weeks when he returned to work. Kept well until 9 months ago when a further attack occurred, when hurrying in the street. Several attacks since. Said to get pale during the attack. No limb movements have been noticed. Attack is followed by a headache.

15 months. Dyspnoea and lower sub-sternal pain, radiating to left shoulder, on exertion.

Off work 9 months.

Previous Illnesses: Nil.

Family History: Nil relevant.

Habits: Alcohol moderate. Tobacco - heavy smoker.

Examination: Florid. Weight, 13 stones.

Circulatory System: Heart rate, 48; regular.

Blood-pressure, 178/80.

Apex beat, impalpable.

Heart sounds: Mitral area, systolic bruit.

Aortic area, systolic bruit.

Aortic 2nd sound accentuated.

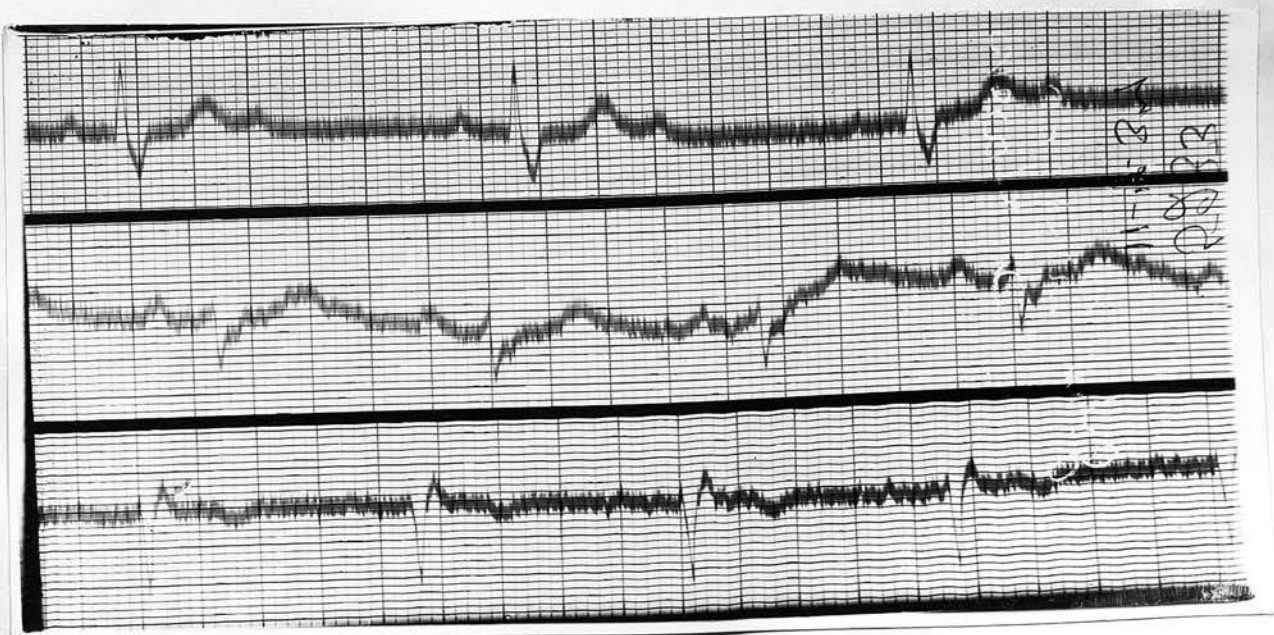
Lungs: N.A.D.

Case 94 (continued)Abdomen: N.A.D.Oedema: NilUrine: N.A.D.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: P - R interval 0.2 secs.QRS complex: Left ventricular preponderance.
Dropped beats in Lead I.T₃ inverted.Diagnosis: Heart block.

'Adams-Stokes Syndrome'.

Myocardial degeneration.

Hyperpiesia.

Circulatory Rate: $16\frac{2}{5}$ secs.

Case 99.

Sex: Male. Married.

Age: 61 years.

Occupation: Labourer.

History: 2 years. Dyspnoea and palpitation on exertion.
Symptoms getting progressively worse. Unable to
work 18 months. Loss of weight (1½ stone) last 2 years.

Previous Illnesses: Pneumonia aet. 25 years.
V.D. denied.

Habits: Alcohol and Tobacco moderate.

Examination: Pale. Weight 10 st. 5 lbs.
Arcus senilis present.

Circulatory System: Heart rate, 78; regular.
Peripheral vessels sclerosed.
Blood-pressure, 240/110.
Apex beat: 5th interspace outside
mid-clavicular line.
Heart sounds: Distant. Gallop rhythm present
Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

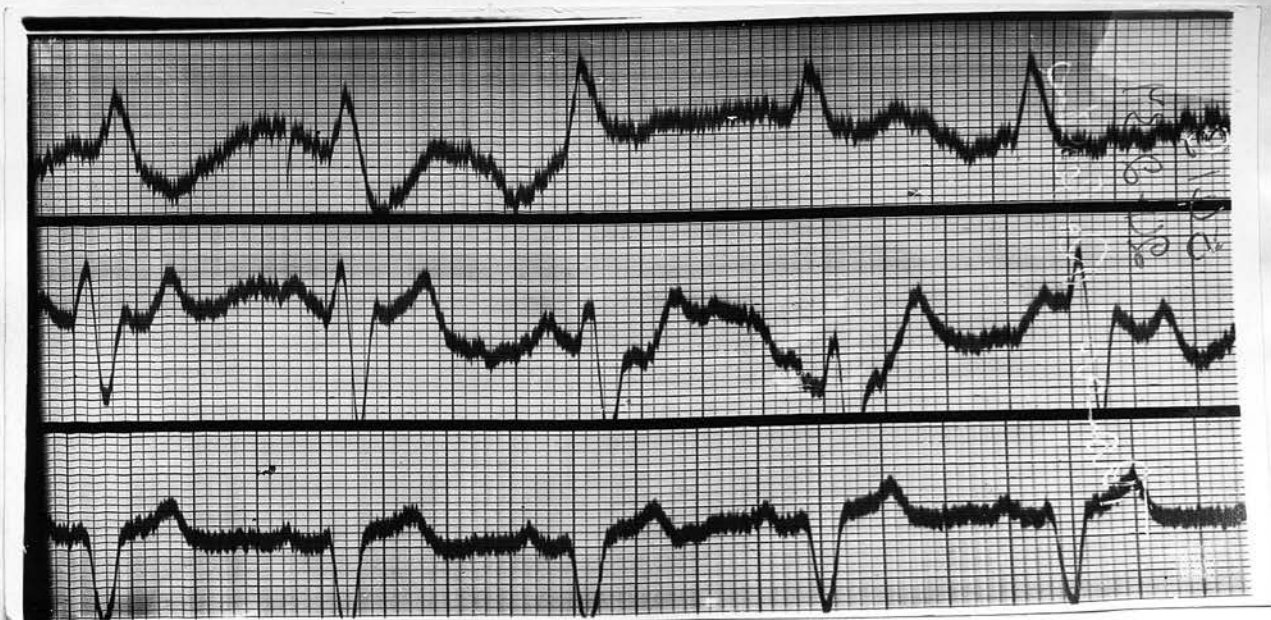
Case 99 (continued)

W.R.: Negative.

Electrocardiogram: Left ventricular preponderance.
QRS widened.
T inverted.
Right bundle branch block.

Diagnosis: Hyperpiesia.
Myocardial degeneration.

Circulatory Rate: $14\frac{4}{5}$ secs.



Case 100.

Sex: Male. Married.

Age: 60 years.

Occupation: Railway porter.

History: 4 years. Attacks of palpitation, becoming more frequent. Dull frontal headaches. Giddiness. Off work 2 weeks.

Previous Illnesses: Nil.

Habits: Tobacco moderate, alcohol nil.

Examination: Weight, 10 st. 11 lbs.

Circulatory System: Heart rate, 82; regular.

Blood-pressure, 220/126.

Apex beat: 5th interspace outside
mid-clavicular line.

Forcible in character.

Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R.: Negative.

Case 100 (continued)

Electrocardiogram: P - R interval, 0.26 secs.

Q₃ present. Lead III Low Voltage.

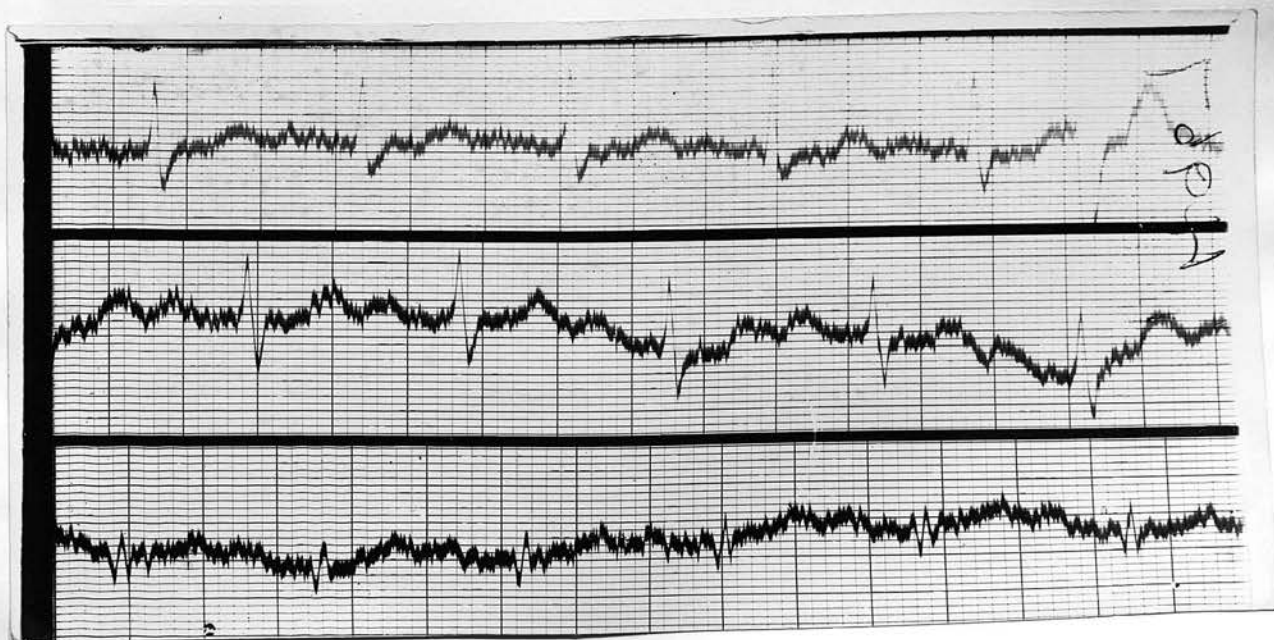
Right ventricular extrasystoles.

Diagnosis: Hyperpiesia.

Myocardial degeneration.

Partial Heart Block.

Circulatory Rate: $12\frac{2}{5}$ secs.



Case 102.

Sex: Female. Single.

Age: 59 years.

Occupation: Nil.

History: 5 years Dyspnoea, palpitation on exertion.
Giddiness and faintness. Slight praecordial pain
on exertion.

Previous illnesses: Nil. Menopause aet. 52 years.

Examination: Weight, 9 st. 1 lbs.

Circulatory System: Heart rate, 80; regular.
Cervical veins engorged.
Blood pressure, 212/114.
Apex beat: 7th interspace at
mid-axillary line.
Heaving in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

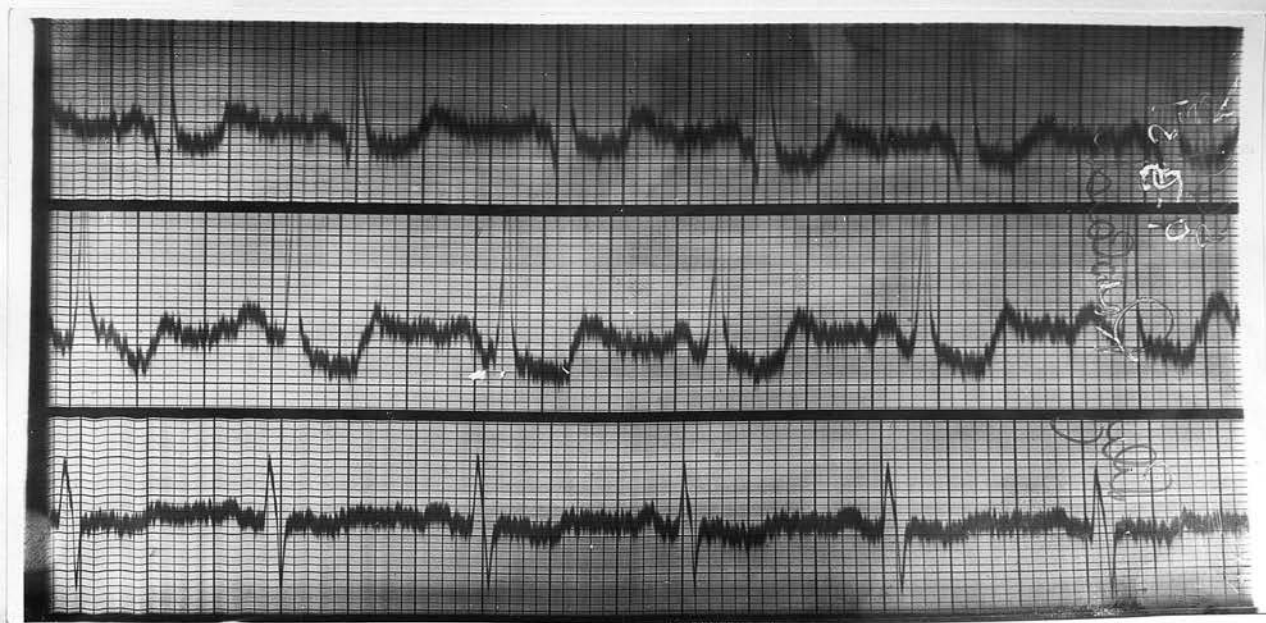
W.R: Negative.

Case 102 (continued)

Electrocardiogram: Q.R.S. SLURRED.
R-T. DEPRESSION.
LEFT VENTRICULAR PREPONDERANCE

Diagnosis: Hyperpiesia.

Circulatory Rate: $16\frac{4}{5}$ secs.



Case 105.

Sex: Female. Married.

Age: 65 years.

Occupation: Housewife.

History: 2 years. Sub-sternal pain, induced by exertion, and eased by rest. Pain is severe in character, radiates to the left shoulder, and down the arm to the elbow.

6 months. Pain more easily induced. At present can only walk about 10 yards without distress. The exertion of turning in bed is sufficient to induce an attack.

4 weeks. Slight cough, dyspnoea on exertion, palpitation, and a sense of constriction in the chest.

Previous Illnesses: Tonsillitis.
Influenza.

Family History: 3 children. 2 miscarriages.

Examination: Weight, 10 st. 4 lbs.

Circulatory System: Heart rate, 78; regular.
Peripheral vessels sclerosed.
Blood pressure, 254/150.
Apex beat, not palpable.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: A few scattered rhonchi posteriorly both lungs.

Abdomen: N.A.D.

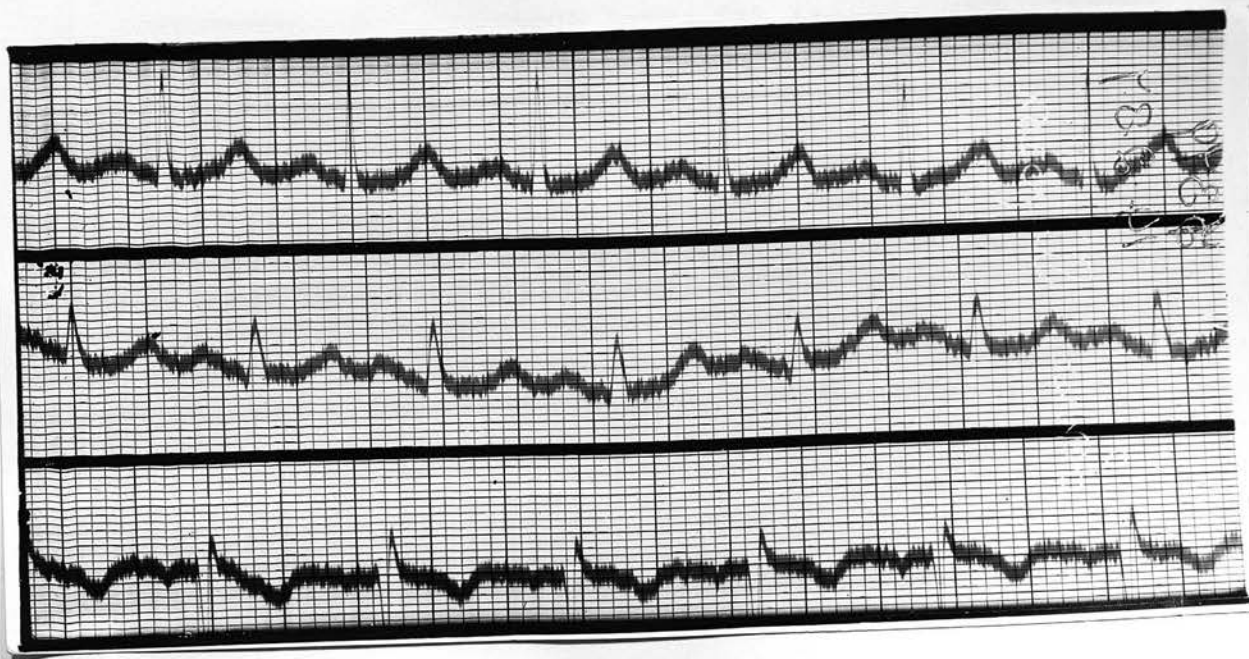
Oedema: Nil.

Urine: Albumin trace.

Case 105 (continued)Nervous System: N.A.D.W.R: Negative.Electrocardiogram: Left ventricular preponderance.T₃ inverted.Diagnosis: Hyperpiesia.

Angina of effort.

Myocardial degeneration.

Circulatory Rate: $16\frac{3}{5}$ secs.

Case 107.

Sex: Male. Married.

Age: 58 years.

Occupation: Labourer.

History: 12 months. Dyspnoea and praecordial pain on exertion and eased by rest. Site of pain is indicated over a wide area extending from nipple to nipple.

No radiation.

Frontal headaches.

Unable to work, 12 months.

Previous Illnesses: Luetic infection, 1906: treated for several years.

Malaria, dysentery, and typhoid fever, aet. 30-40 years.

Habits: Alcohol immoderate; tobacco moderate.

Examination: Weight, 11 st. 6 lbs.

Circulatory System: Heart rate, 82; regular.

Blood-pressure, 240/96.

Apex beat: 5th interspace outside mid-clavicular line.

Heaving in character.

Heart sounds: Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

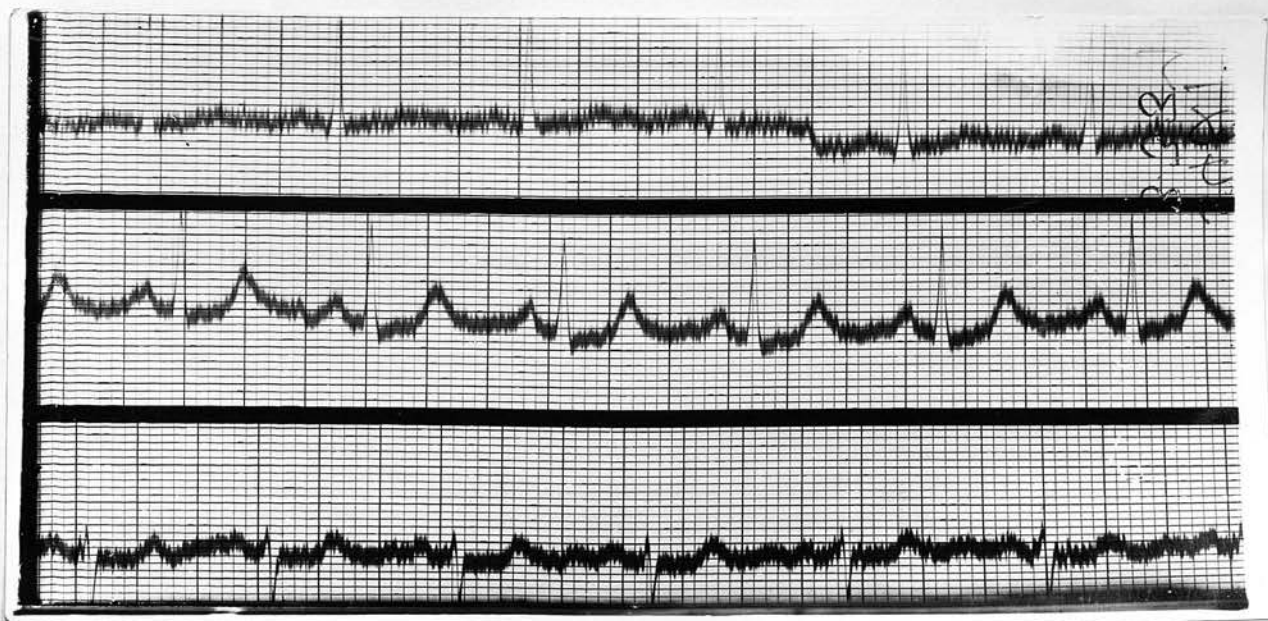
Case 107 (continued)

W.R.: Positive. Cerebro-spinal fluid Wassermann negative.

Electrocardiogram: Left ventricular preponderance.

Diagnosis: Hyperpiesia.

Circulatory Rate: $13\frac{1}{5}$ secs.



Case 116.

Sex: Male. Single.

Age: 36 years.

Occupation: Rent collector.

History: 3 months. Dull aching left sub-mammary pain which does not radiate, has no relation to exertion, and occurs occasionally while at rest in bed.

Dyspnoea on exertion. Palpitation and giddiness.

Kapt at work, which entails a lot of walking.

Previous Illnesses: Influenza.
V.D. denied.

Habits: Alcohol nil; tobacco moderate.

Examination: Weight, 13 st. 7 lbs.

Circulatory System: Heart rate, 74; regular.
Blood-pressure, 172/110.
Apex beat: 5th interspace at mid-clavicular line.
Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Vasomotor flushing present.
Thyroid gland palpable.

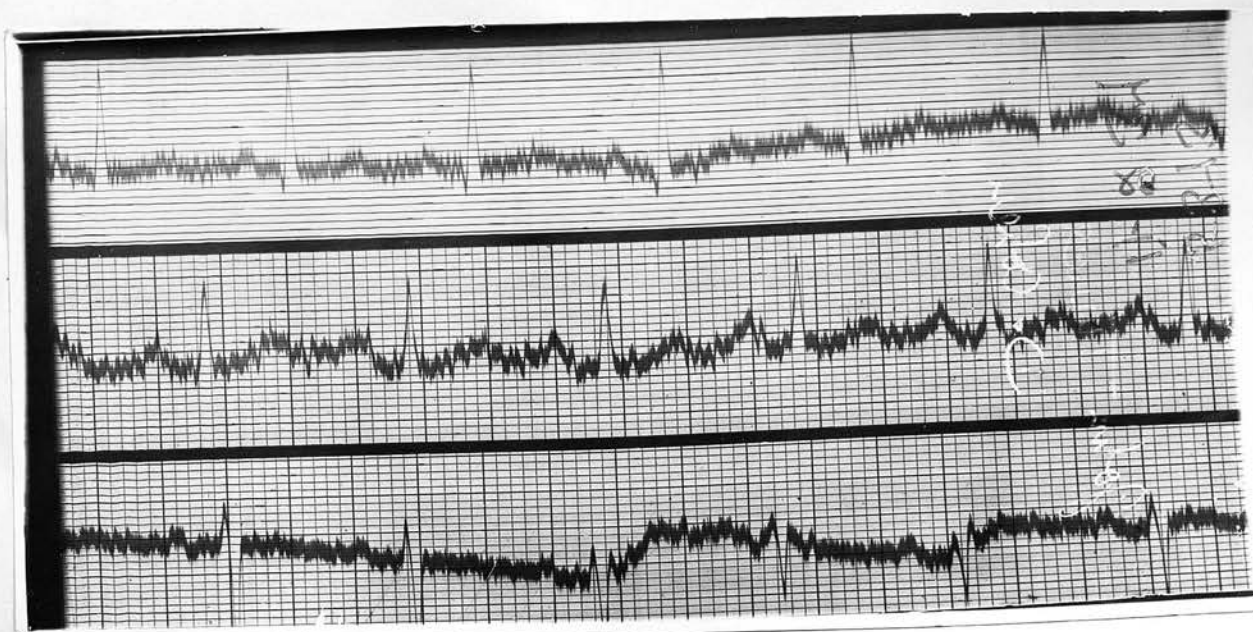
W.R: Negative.

Electrocardiogram: Left ventricular preponderance.
QRS II slightly slurred.

Case 116 (continued)

Diagnosis: Hyperpiesia.
 Myocardial degeneration.

Circulatory Rate: $10\frac{1}{5}$ secs.



Case 117.

Sex: Female. Married.

Age: 61 years.

Occupation: Cleaner.

History: 18 months. Giddiness in attacks.
Dyspnoea on exertion.

6 months. Praecordial pain on exertion, relieved by rest. Pain is sub-sternal and left sub-mammary, and radiates to the left shoulder and down left arm to the finger-tips. Relieved by Nitrites. Pain is followed by weakness in the left arm.
Palpitation.

3 months. Cough. Mucoid sputum.

Previous illnesses: Influenza.

Family History: No children. No history of miscarriages.

Examination: Weight, 9 st. 11 lbs.

Circulatory System: Heart rate, 80; regular.

Blood-pressure, 208/110.

Praecordium tender.

Apex beat: 6th interspace outside
mid-clavicular line.

Forcible.

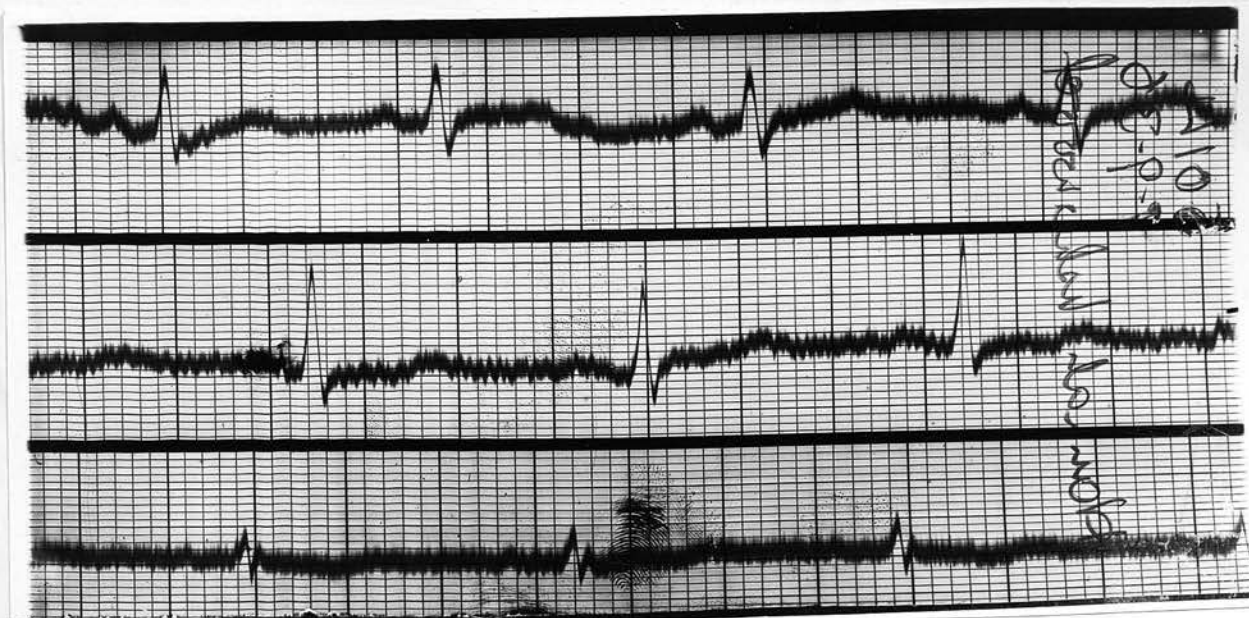
Heart sounds: Mitral area, systolic bruit.

Aortic area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: Scattered rhonchi.

Abdomen: N.A.D.

Case 117 (continued)Oedema: Nil.Urine: N.A.D.Nervous System: Hands, coarse tremor.W.R: Negative.Electrocardiogram: R₁, R₂ slurred. R₃ split.
T₃ inverted.X-ray Examination: "Chronic inflammatory changes in lungs.
? slight diffuse dilatation aortic arch."Diagnosis: Angina of effort.
Myocardial degeneration.
Hyperpiesia.
Chronic bronchitis.Circulatory Rate: 10 seconds.

Case 118.

Sex: Male. Married.

Age: 48 years.

Occupation: Fitter.

History: 2 years. Dyspnoea on exertion, attacks of giddiness and palpitation. Frontal headaches.

6 months. Praecordial pain, vaguely localised in left sub-mammary region, on exertion. No radiation.

Weight gradually increasing.

Previous Illnesses: Influenza.
Gastric ulcer, aet. 44 years.

Habits: Alcohol and tobacco moderate.

Examination: Weight 15 stones.

Circulatory System: Heart rate, 78; regular.
Blood-pressure, 230/120.
Apex beat not palpable.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

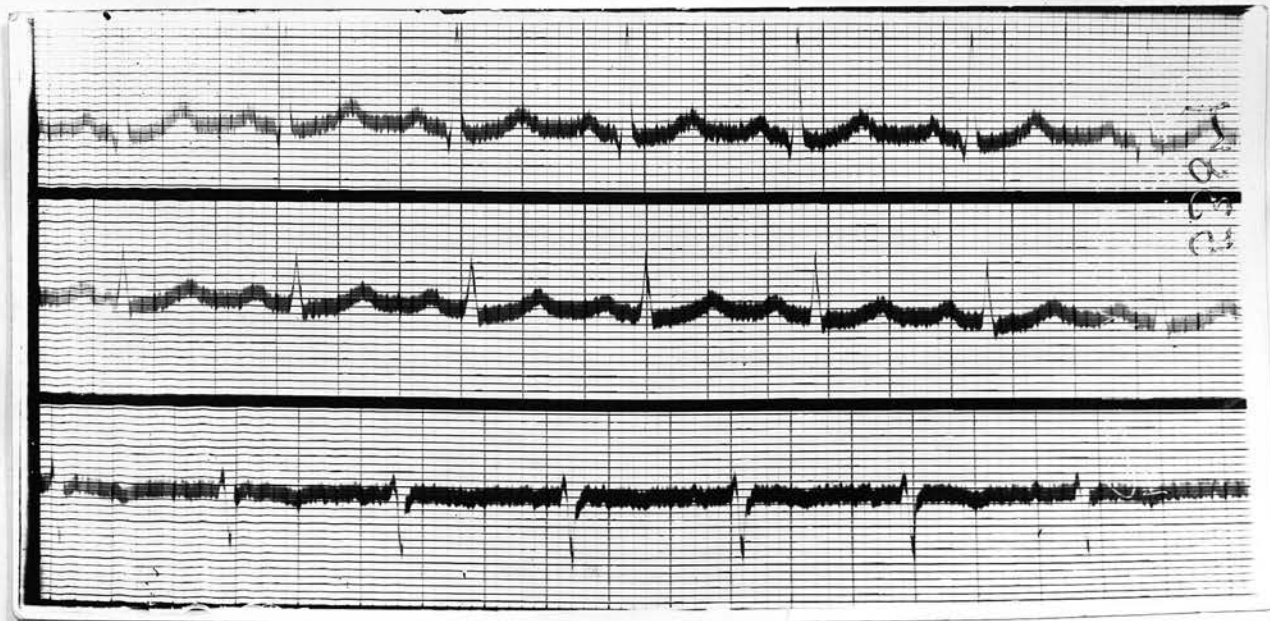
W.R: Negative.

Case 118 (continued)

Electrocardiogram: Left ventricular preponderance.
 T_3 inverted.

Diagnosis: Hyperpiesia.

Circulatory Rate: $9\frac{2}{5}$ secs.



Case 123.

Sex: Female. Married.

Age: 47 years.

Occupation: Housewife.

History: 3 years. Dull left sub-mammary pain on occasions.
Palpitation and throbbing in throat.
Lack of energy and depression.
Slight dyspnoea on exertion.

Previous Illnesses: Influenza aet. 42 years.

Examination: Weight, 13 st. 4 lbs.

Circulatory System: Heart rate, 76; regular.

Blood pressure, 178/100.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R.: Negative.

Case 123 (continued)

Electrocardiogram: NOT AVAILABLE

Diagnosis: Hyperpiesia.

Circulatory Rate: $12\frac{1}{5}$ secs.

Case 126.

Sex: Male. Single.

Age: 65 years.

Occupation: Cab driver.

History: 2 months. Frontal headaches. Giddiness.

6 weeks ago suddenly lost power in left leg and left arm. Speech unaffected. Loss of power lasted a few minutes, and he was able to walk shortly afterwards.

1 month. Palpitation and insomnia.

Off work 6 weeks.

Previous illnesses: Cholecystectomy performed 18 months ago.

Habits: Alcohol immoderate, tobacco moderate.

Examination: Weight 13 stones.

Circulatory System: Heart rate, 74; regular.

Blood-pressure, 250/130.

Peripheral vessels sclerosed.

Apex beat: 5th interspace outside mid-clavicular line.

Forcible in character.

Heart sounds: Muffled.

Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin.

Case 126 (continued)

Nervous System: N.A.D.

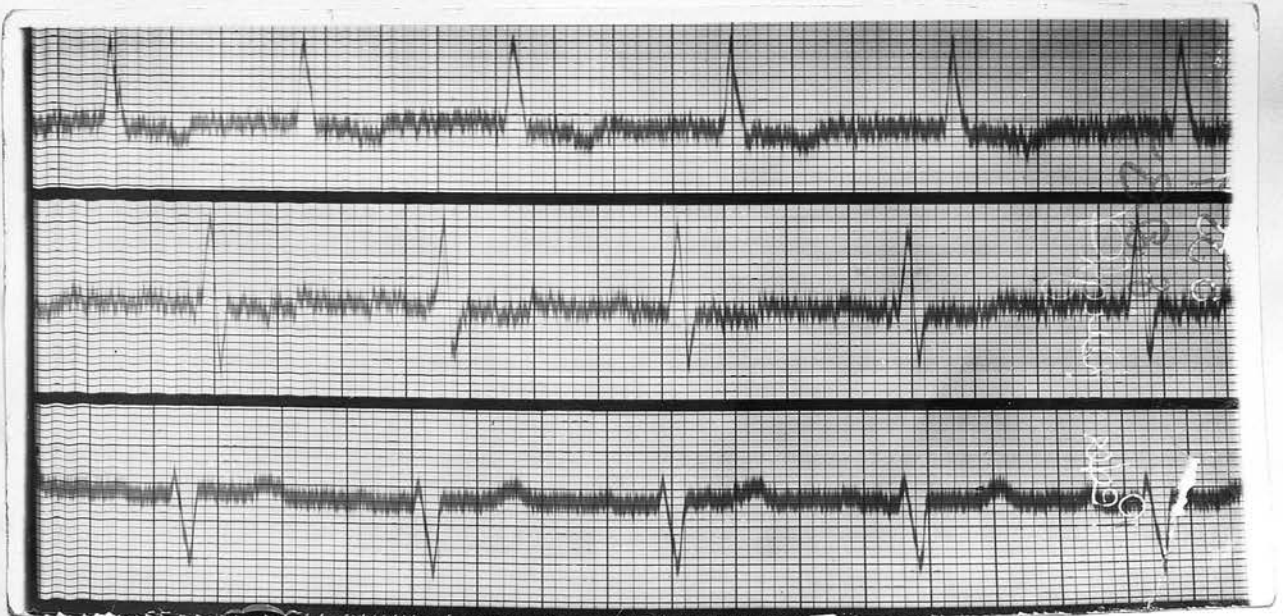
W.R: Negative.

Blood: Non-protein nitrogen 60 mgm. per 100 ccs.

Electrocardiogram: Left ventricular preponderance.
T₁ inverted.

Diagnosis: Hyperpiesia.
Myocardial degeneration.

Circulatory Rate: $14\frac{2}{5}$ secs.



Case 127.

Sex: Male. Married.

Age: 46 years.

Occupation: Grocer.

History: 4 years. Dyspnoea on exertion, occasional left sub-mammary pain on extreme exertion. Palpitation, giddiness.

2 years. Frontal headaches, drowsiness.

Weight gradually increasing.

Previous illnesses: Influenza.

Family History: 6 children.

Habits: Alcohol nil; tobacco moderate.

Examination: Obese. Weight 18 st. 11 lbs.

Circulatory System: Heart rate, 84; regular.

Blood-pressure, 196/104.

Apex beat: Impalpable.

Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Nervous System: N.A.D.

Urine: N.A.D.

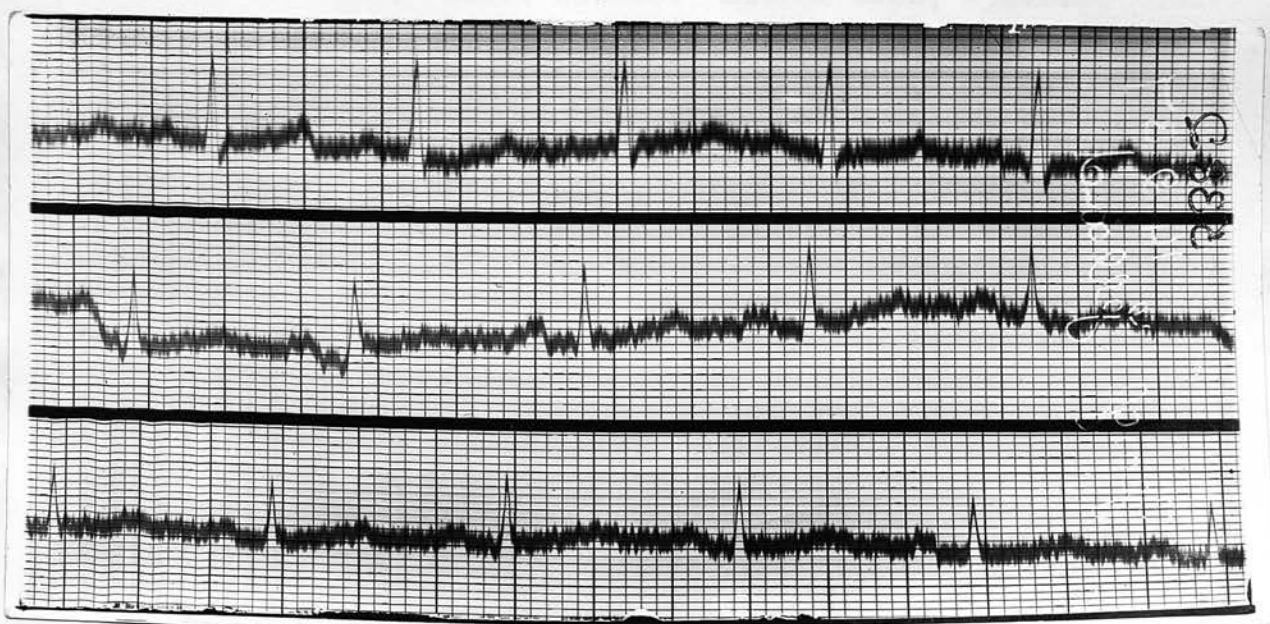
Case 127 (continued)

W.R.: Negative.

Electrocardiogram: P₂ slurred.

Diagnosis: Hyperpiesia.
Obesity.

Circulatory Rate: $9\frac{4}{5}$ secs.



Case 129.

Sex: Female. Married.

Age: 48 years.

Occupation: Housewife.

History: 3 months. Throbbing sensation in crown of head.
Occasional giddiness and palpitation.
Left sub-mammary pain on exertion.

Previous Illnesses: Chronic rheumatism arms and legs.

Family History: No children. No miscarriages.
Menopause aet. 47 years.

Examination: Weight 7 st. 8 lbs.
Anaemic.

Circulatory System: Heart rate, 77; regular.
Blood-pressure, 222/104.
Apex beat: 5th interspace at
mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

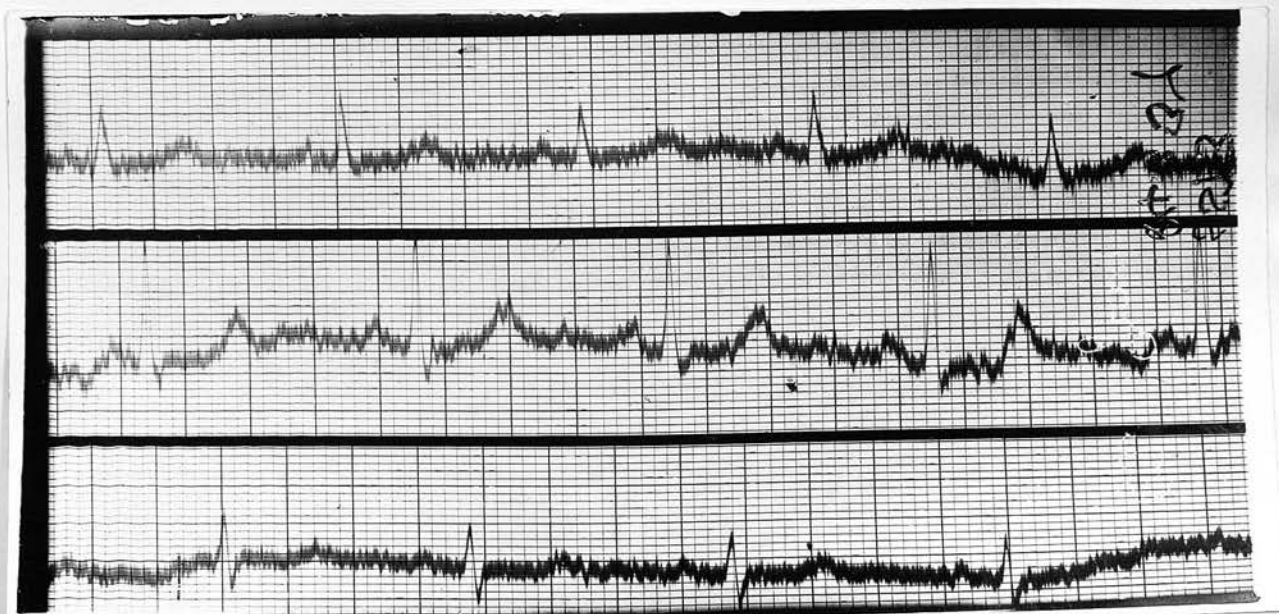
Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

Case 129 (continued)W.R.: Negative.Electrocardiogram: S₂ and S₃ slurred.Diagnosis: Hyperpiesia.Circulatory Rate: $10\frac{1}{5}$ secs.

GROUP 2

20 cases

Rheumatic Heart Disease.

Average circulatory rate 12.4 secs.

(Range 8.2 - 19.2 secs.)

Case 1

Sex: Female. Married.

Age: 49 years.

Occupation: Housewife.

History: 6 years. 'Thumping' sensation in the throat accompanied by a smothering feeling. Pain in the chest on exertion and relieved by rest. The pain is sub-sternal in location and radiates over a wide area. Dyspnoea and palpitation on the slightest exertion. Attacks of giddiness and faintness.

10 years. 'Flatulence' and abdominal discomfort after meals.

Previous illnesses: Tonsillitis, numerous attacks.
Influenza.

Family History: Nil relevant.
3 children; no history of miscarriages.

Examination: Obese woman. Weight 15 stones.

Circulatory System: Heart rate, 90; regular.
Blood pressure, 238/45.
Apex beat: 7th interspace, mid-axillary line.
Forcible.
Supra-sternal pulsation +
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic and diastolic bruits.

Lungs: N.A.D.

Abdomen: Tenderness over gall-bladder area.

Case 1 (continued)

Oedema: Nil

Urine: N.A.D.

Nervous System: N.A.D.

W.R.: Negative.

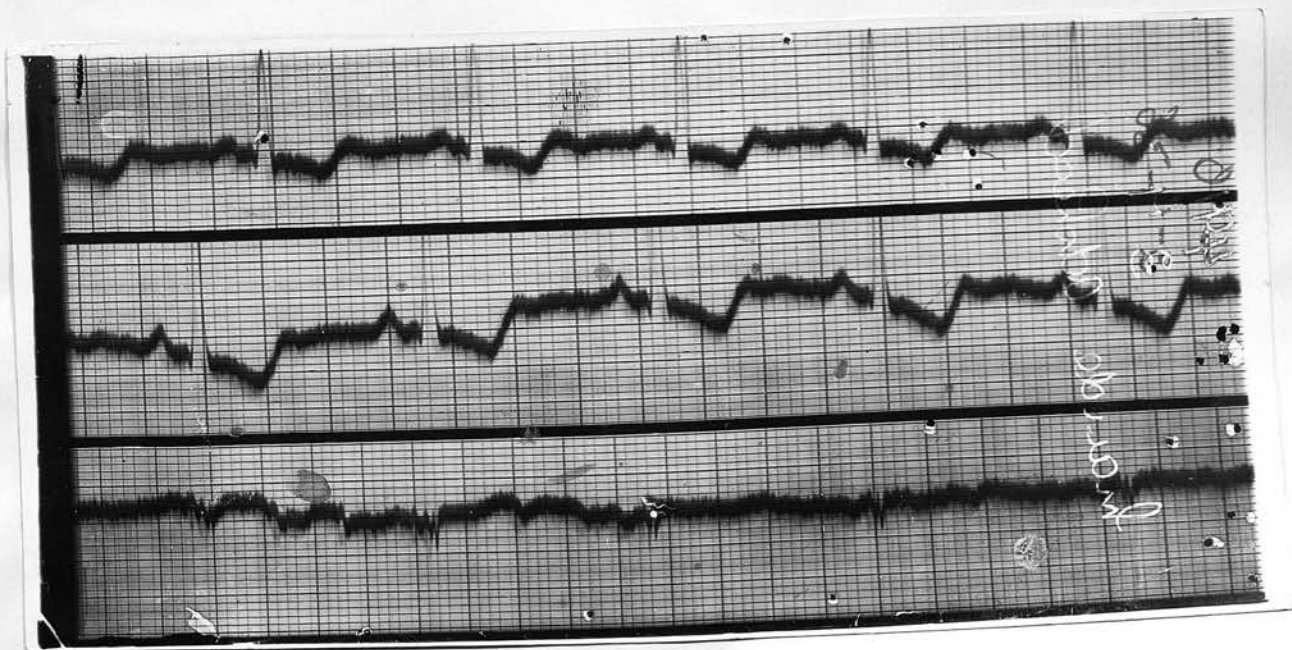
Electrocardiogram: Left ventricular preponderance.

T₁ and T₂ inverted. Lead III Low Voltage.

X-ray Examination: "General cardiac enlargement, chiefly left ventricular."

Diagnosis: Aortic incompetence. Rheumatic.
Chronic Cholecystitis.

Circulatory Rate: $10\frac{1}{5}$ secs.



Case 4.

Sex: Male. School-boy.

Age: 13 years.

History: 5 years. 'Growing pains' in legs on and off.

3 years. Nervousness.

Weight has increased satisfactorily.

Previous Illnesses: Tonsillitis aet.7 and aet.9.

Whooping cough aet.6.

Examination: Well-nourished boy.

Circulatory System: Heart rate, 78; irregular. Sinus arrhythmia.

Blood-pressure, 122/78.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Mitral area, systolic and
diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R: Negative

Case 4 (continued)

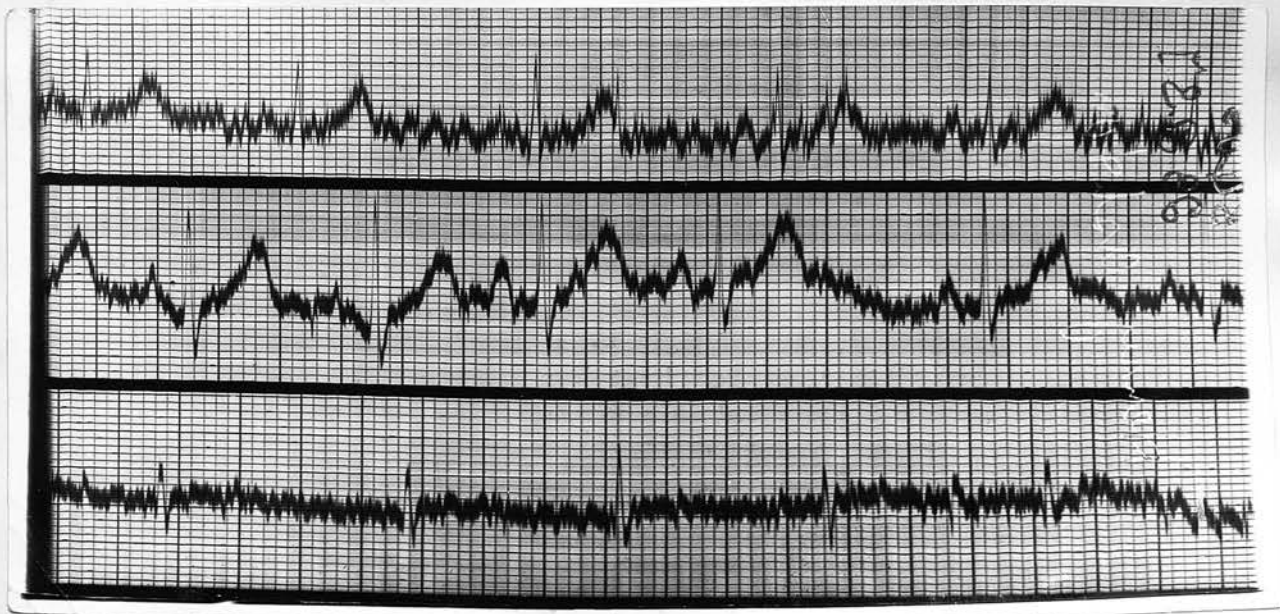
Electrocardiogram: Sinus Arrhythmia.

T₃ flat.

Diagnosis: Mitral stenosis. Rheumatic.

Circulatory Rate: $11\frac{1}{5}$ secs.

(measured by stop-watch method. Graphic record unsatisfactory as patient commenced crying on injection of the drug)



Case 5Sex: Female. Married.Age: 31 years.Occupation: Housewife.History: 1½ years. Intermittent left sub-mammary pain, not related to exertion.

Dyspnoea and palpitation on exertion.

Giddiness and exhaustion.

Previous illnesses: Growing pains in childhood.

Tonsillitis frequently.

Influenza.

Family History: No children; no history of miscarriages.Examination: Obese woman. Weight 13 stones.Circulatory System: Heart rate, 76; regular.

Blood-pressure, 136/76.

Apex beat: 5th interspace outside
mid-clavicular line.

Heart sounds: Mitral area, rough systolic bruit.

Aortic 2nd sound ringing.

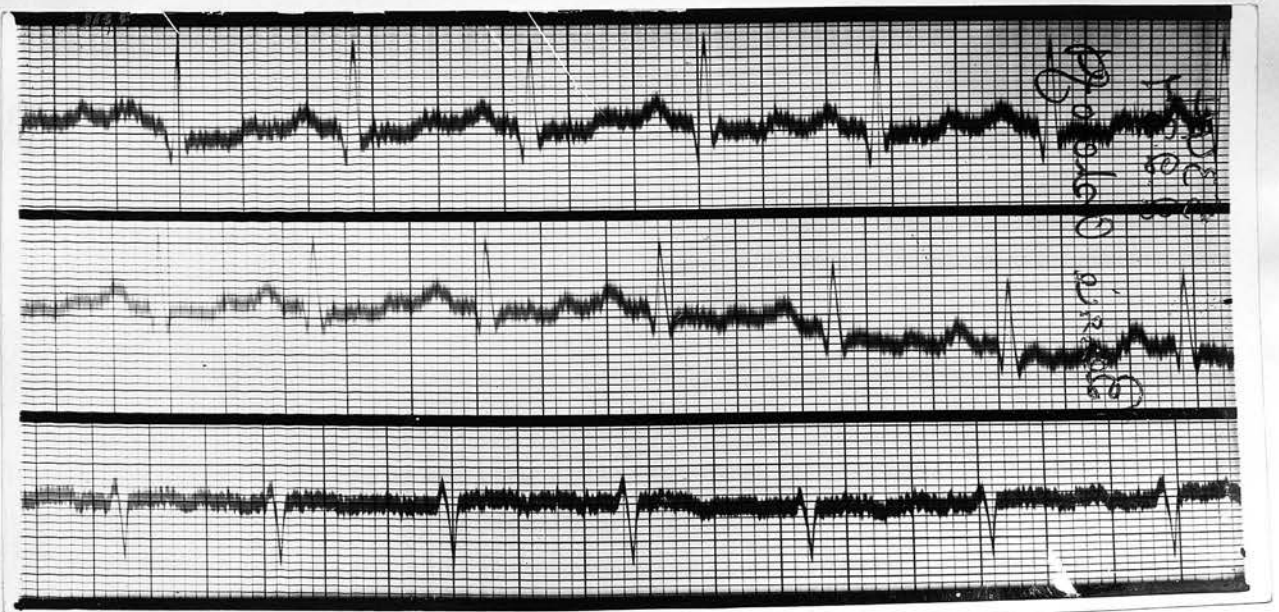
Diastolic bruit left border
sternum 2nd interspace.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.Nervous system: N.A.D.

Case 5 (continued)W.R.: Negative.

Electrocardiogram: P - R interval greater than 0.2 secs.
Left ventricular preponderance.
T₃ inverted.

Diagnosis: Aortic incompetence.
Mitral incompetence.
Myocardial degeneration.
Rheumatic.

Circulatory Rate: $12\frac{3}{5}$ secs.



Case 8.

Sex: Female. Single.

Age: 27 years.

Occupation: Clerk.

History: 5 months ago had a sudden attack of dyspnoea which lasted 5 minutes. 2 further attacks have occurred since, the last time being 3 weeks ago.

3 weeks. Dry cough, faintness, exhaustion, and palpitation.

Confined to bed 3 weeks during which time has had night sweats.

Previous Illnesses: Rheumatic fever aet. 21 and 23 years.

Chorea aet. 10 years.

Tonsillitis, several attacks;
tonsillectomy aet. 17 years.

Measles, aet. 18 years.

Family History: N.A.D.

Habits: Moderate smoker.

Examination: Weight 8 st. 7 lbs.

Circulatory System: Heart rate, 80; regular.

Temperature sub-normal.

Blood-pressure, 118/60.

Apex beat: 5th interspace outside
mid-clavicular line.

Pre-systolic thrill at apex.

Heart sounds: Mitral area, presystolic and
systolic bruits.

Aortic area, systolic bruit.

Diastolic bruit left border
of sternum 3rd interspace.

Case 8 (continued)

Lungs: Occasional moist sound at both bases.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous system: N.A.D.

W.R.: Negative.

Electrocardiogram: P - R interval 0.2 secs.

P large and widened.

Q₂ and Q₃ present.

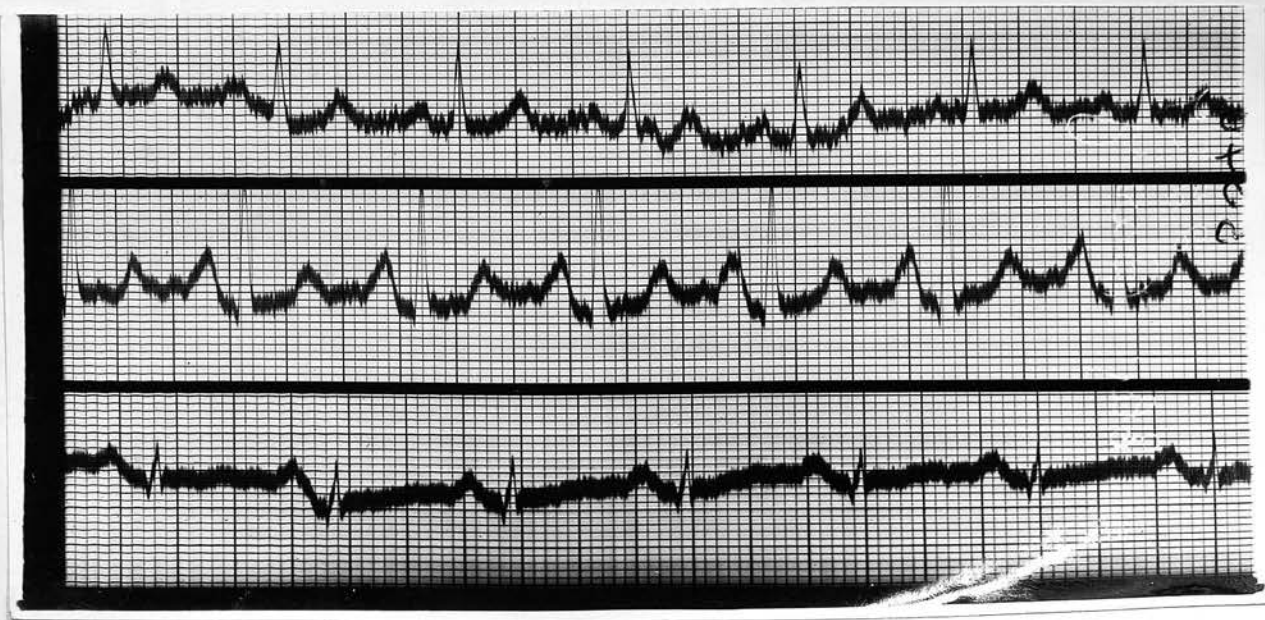
Normal rhythm.

Diagnosis: Mitral stenosis.

Aortic incompetence.

Rheumatic.

Circulatory Rate: $15\frac{2}{5}$ secs.



Case 18

Sex: Female. Single.

Age: 21 years.

Occupation: Machinist.

History: 12 months ago had an attack of 'Gastric influenza' for which she was in bed for a fortnight. This was followed by palpitation, dyspnoea on exertion, and attacks of giddiness and faintness.

14 days ago collapsed while at work and has been in bed since.

Also complains of cough with expectoration and loss of weight.

Previous Illnesses: Growing pains in child-hood.

Examination: Weight, 8 stones.
Slight cyanosis.

Circulatory System: Heart rate, 82; regular.
Temperature sub-normal.
Blood-pressure, 124/60.
Apex beat: 5th interspace outside
mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area, presystolic bruit.
Aortic area, diastolic bruit.
Pulmonary 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 18 (continued)

Nervous system: N.A.D.

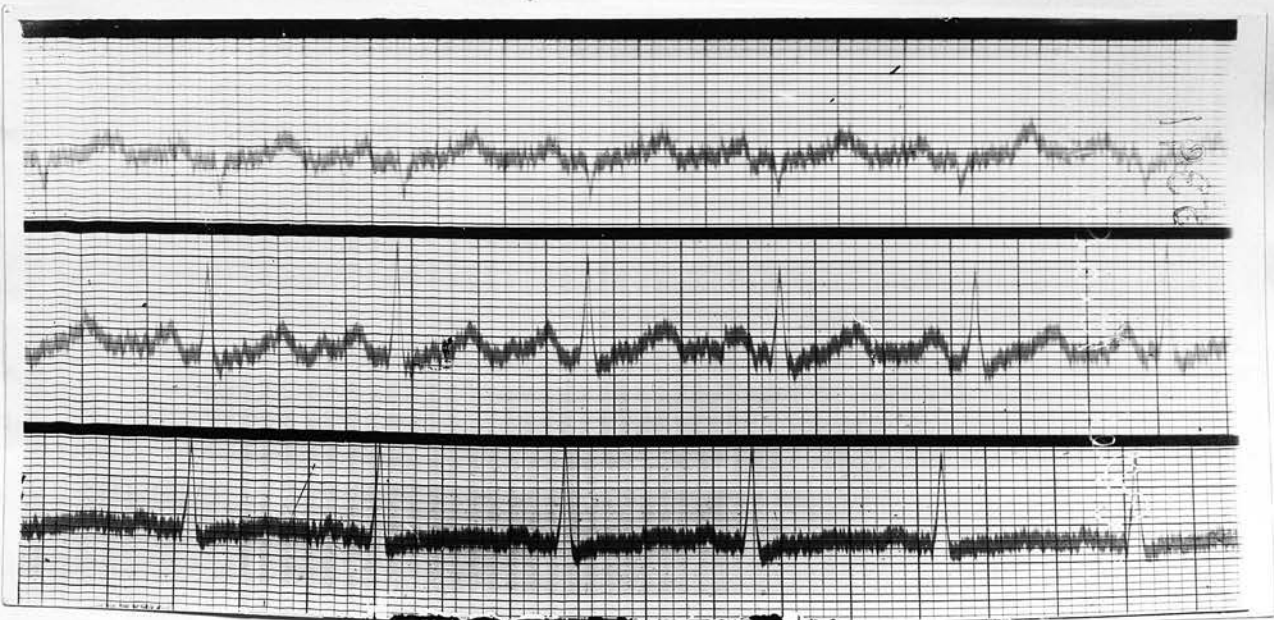
W.R.: Negative.

Electrocardiogram: Right ventricular preponderance.
T₃ flat.

X-ray examination: "Slight Left Auricular enlargement.
No evidence pulmonary pathology."

Diagnosis: Mitral stenosis.
Aortic incompetence.
Rheumatic.

Circulatory Rate: $12\frac{3}{5}$ secs.



Case 21.Sex: Male. Married.Age: 44 years.Occupation: Laundryman.History: 6 months left sub-mammary pain, which does not radiate. Pain is induced by exertion and eased by rest.

3 months ago 'collapsed' at work owing to the severity of the pain and spent 3 weeks in bed.

2 months. Dyspnoea on exertion and more recently has complained of a dry cough.

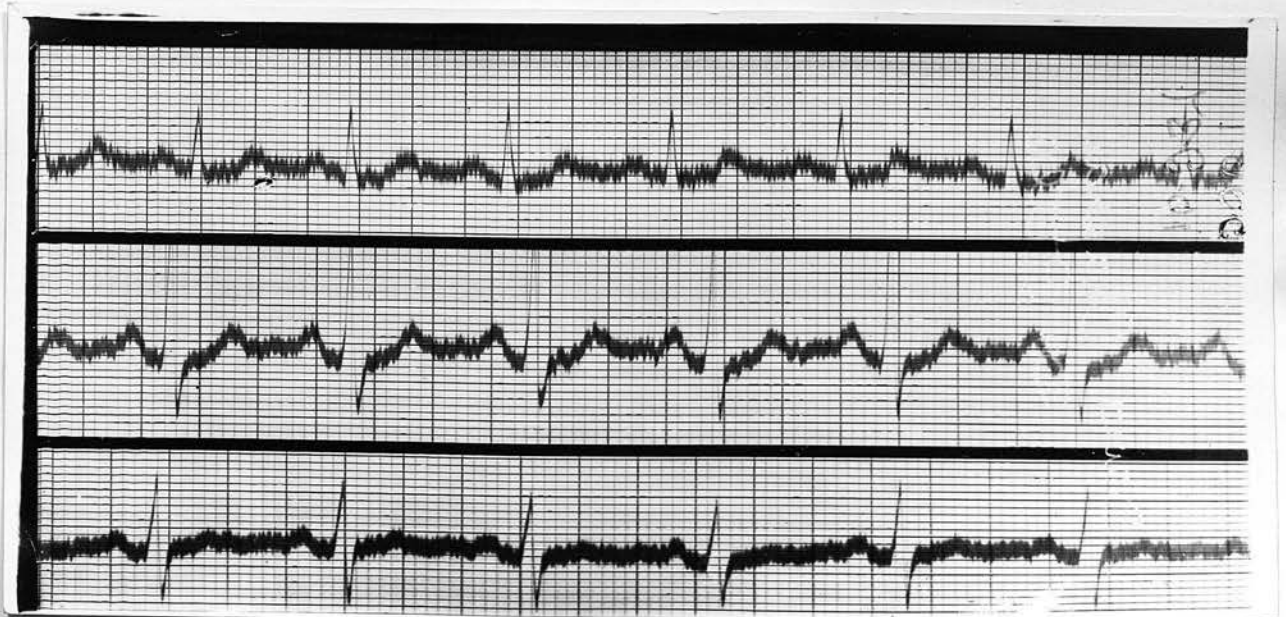
Previous Illnesses: Rheumatic fever aet. 12 years.
Scarlet fever, childhood.
Influenza.Family History: Nil relevant.Habits: Alcohol nil; tobacco moderate.Examination: Weight 8 st. 2 lbs.
Fingers clubbed.Circulatory System: Heart rate, 84; regular.
Blood-pressure, 134/70.
Carotid pulsation marked.
Apex beat: 6th interspace outside mid-clavicular line.
Heaving in character.
Systolic retraction 4th - 6th interspaces.
Systolic thrill base.
Heart sounds: Mitral area, presystolic and systolic bruits.
Aortic area, systolic and diastolic bruits.

Case 21 (continued)Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: S₂ slurred.Diagnosis: Aortic stenosis and incompetence.

Mitral stenosis.

Rheumatic.

Adherent pericardium.

Circulatory Rate: $13\frac{4}{5}$ secs.

Case 23.Sex: Female. Single.Age 17 years.Occupation: Packer.History: 6 months. Syspnoea on exertion, palpitation and nervousness.

4 months. Attacks of frontal headache and vomiting lasting 2 days and occurring every fortnight.

Remained at work until 4 days ago when one of the above attacks occurred.

Previous illnesses: Tonsillitis, several attacks.Family History: Nil relevant.Examination: Pale. Weight 9 st.7 lbs.

Temperature: 97.4°

Circulatory System: Heart rate, 86; irregular.

Blood-pressure, 128/76.

Apex beat: 5th interspace just
outside mid-clavicular line.

Forcible in character.

Heart sounds: Numerous extrasystoles.

Mitral area, harsh systolic
bruit conducted
posteriorly.

Aortic area, soft systolic bruit.

Pulmonary 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 23 (continued)

W.R.: Negative.

Nervous System: Deep reflexes brisk.

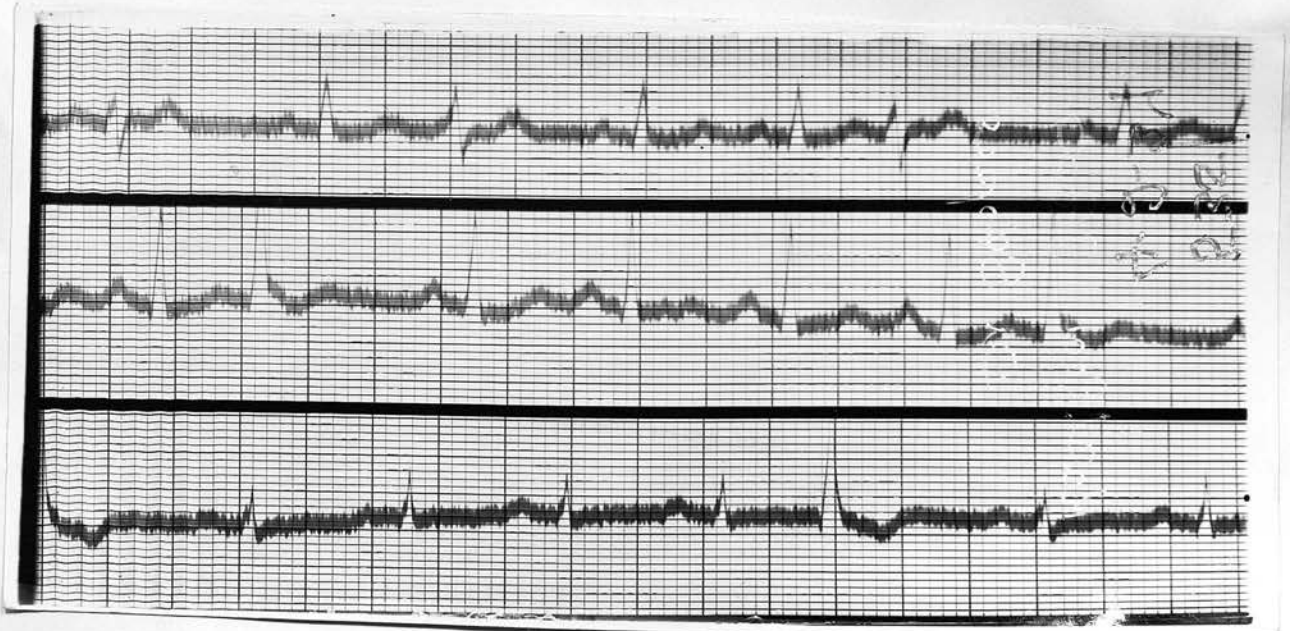
Thyroid gland diffusely enlarged.

Electrocardiogram: Numerous right ventricular extrasystoles.

Diagnosis: Mitral incompetence.

Rheumatic.

Circulatory Rate: $10\frac{2}{5}$ secs.



Case 36.Sex: Female. Single.Age: 26 years.Occupation: Domestic servant.History: 3 years. Palpitation on exertion and after meals.
Exhaustion, giddiness, and syncope.
Catamenia regular.Previous Illnesses: Rheumatic fever, aet.15 years.
Growing pains, childhood.
Tonsillitis, several attacks.
Diphtheria aet.10 years.
Pyelitis aet 23 years.Family History: Nil relevant.Examination: Weight 11 stones.Circulatory System: Heart rate, 80; regular.
Blood-pressure, 128/82.
Apex Beat: 5th interspace outside
mid-clavicular line.
Heart sounds: Mitral area, harsh systolic
bruit.
Aortic area, systolic and
diastolic bruits.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 36 (continued)

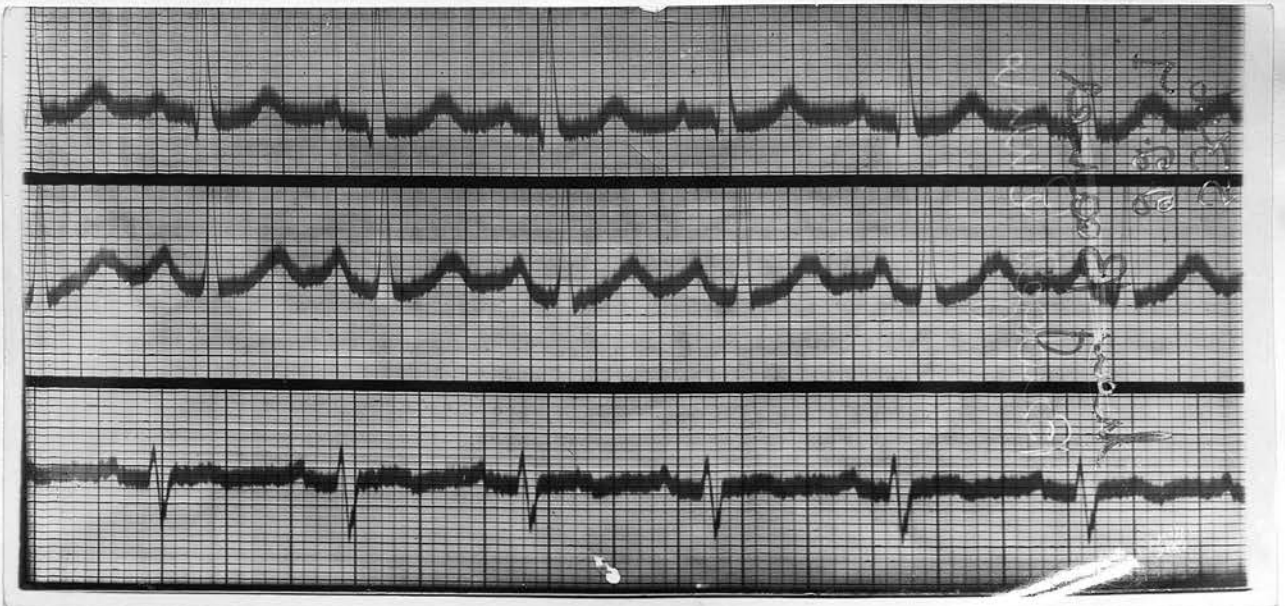
W.R.: Negative.

Nervous System: N.A.D.

Electrocardiogram: Left ventricular preponderance.

Diagnosis: Aortic incompetence.
Mitral incompetence.
Rheumatic.

Circulatory Rate: $11\frac{4}{5}$ secs.



Case 46

Sex: Female. Single.

Age: 31 years.

Occupation: Nurse.

History: 3 years. Dyspnoea on exertion.

More recently palpitation and exhaustion.

18 months ago had an attack of loss of consciousness. Lasted a few seconds. Said to have gone pale during the attack. Since then has had 3 similar attacks, the last one being 1 week ago.

No tongue-biting or incontinence.

('Have been diagnosed Stokes-Adams Syndrome'.)

Previous Illnesses: Rheumatic fever aet. 26 years.

Tonsillitis, numerous attacks.

Family History: Nil relevant.

Catamenia regular.

Examination: Weight 9 st. 6 lbs.

Circulatory System: Heart rate, 76; regular.

Blood-pressure, 128/80.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Mitral area, systolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 46 (continued)

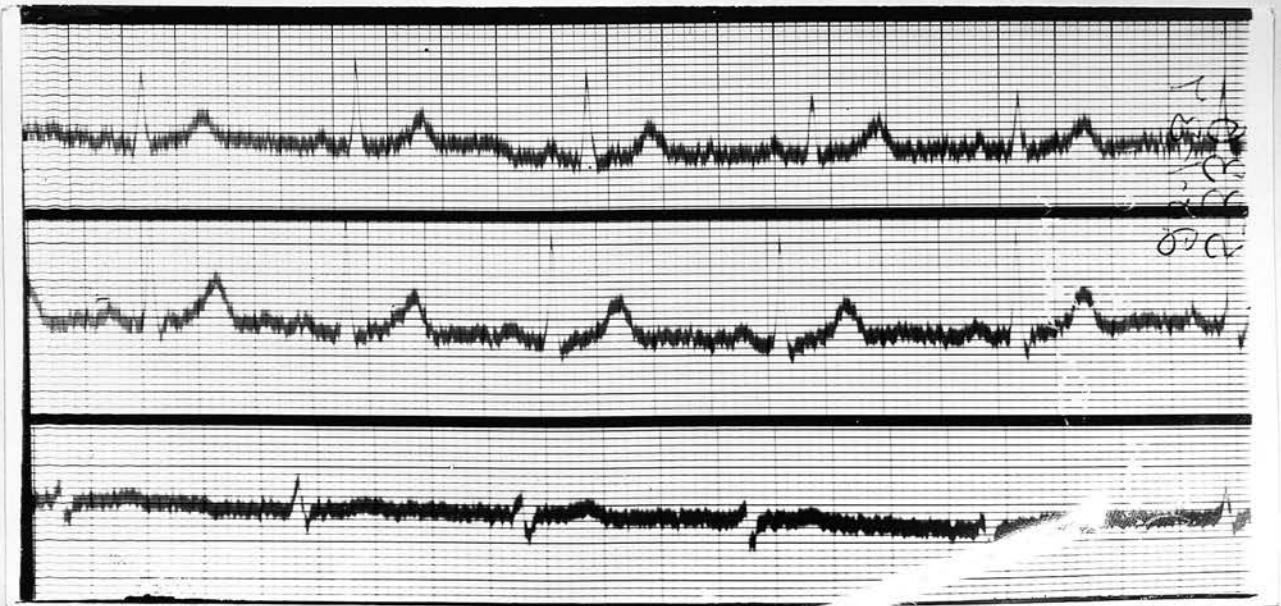
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: QRS III Slurred.

Diagnosis: Mitral incompetence.
Rheumatic.

Circulatory Rate: $13\frac{2}{5}$ secs.



Case 51.Sex: Female. Single.Age: 34 years.Occupation: Domestic servant.

History: 1 year ago had an attack of rheumatic fever, said to be severe, for which she was in bed for 8 weeks.
 Now complains palpitation and dyspnoea on exertion.
 Giddiness, unable to work 1 year.

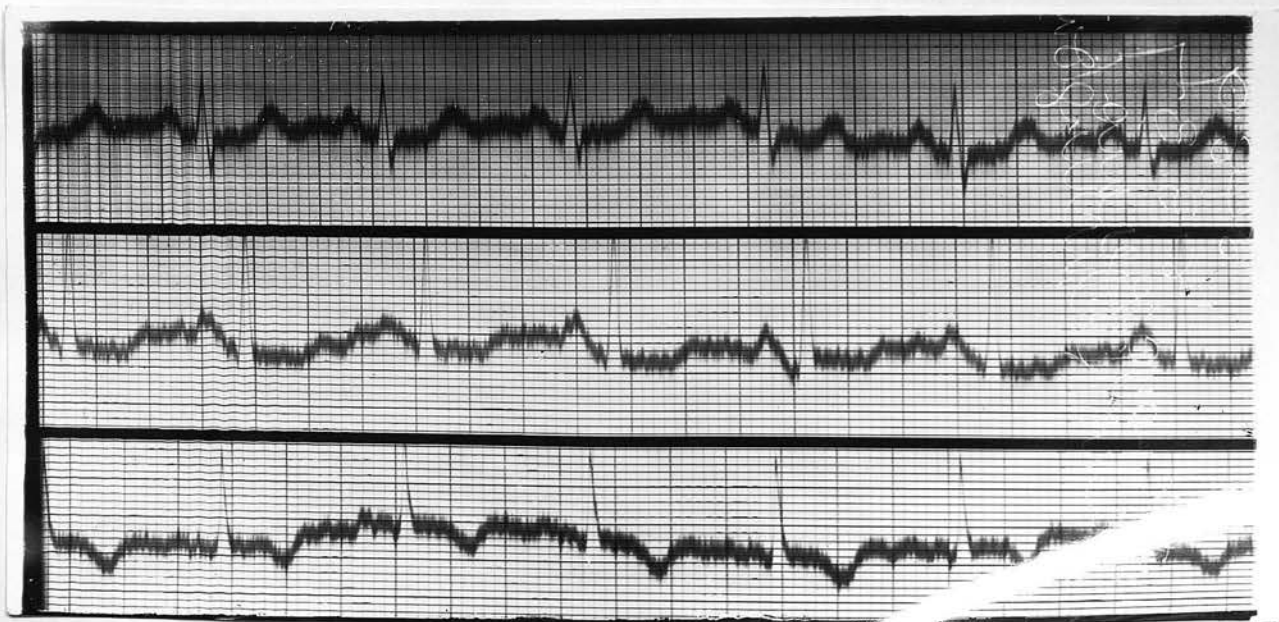
Previous Illnesses: Rheumatic fever aet. 17 years.
 Bronchitis.

Family History: Nil relevant.

Examination: Obese woman. Weight 13 st. 2 lbs.
 Malar flush.

Circulatory System: Heart rate, 75; regular.
 Blood-pressure, 140/90.
 Apex beat: 5th intercostal space outside mid-clavicular line.
 Presystolic thrill at apex.
 Heart sounds: Mitral area, presystolic and systolic bruits.
 2nd sound at apex reduplicated.
 Aortic area, systolic bruit.
 2nd Pulmonary sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 51 (continued)Nervous System: N.A.D.W.R: Negative.Electrocardiogram: P₂ notched.
T₃ inverted.Diagnosis: Mitral stenosis.
Rheumatic.Circulatory Rate: 14 secs.

Case 59.Sex: Male. Single.Age: 22 years.Occupation: Hairdresser.History: 14 days ago, attack of palpitation lasting 6 hours and accompanied by 'heaviness and pain in the chest'. Since then has complained of weakness. Off work since.Previous Illnesses: Rheumatic fever, aet. 4 years.
aet. 12 years.
Tonsillitis, several attacks.Family History: Nil relevant.Habits: Heavy cigarette smoker.Examination: Weight 9 stones.Circulatory System: Heart rate, 90; regular.

Temperature sub-normal.

Blood-pressure 102/48.

Apex beat: 6th interspace outside mid-clavicular line.

Forcible in character.

Systolic thrill base.

Presystolic thrill apex.

Heart sounds: Mitral area, presystolic and systolic bruits.

Aortic area, harsh systolic and diastolic bruits.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 59 (continued)

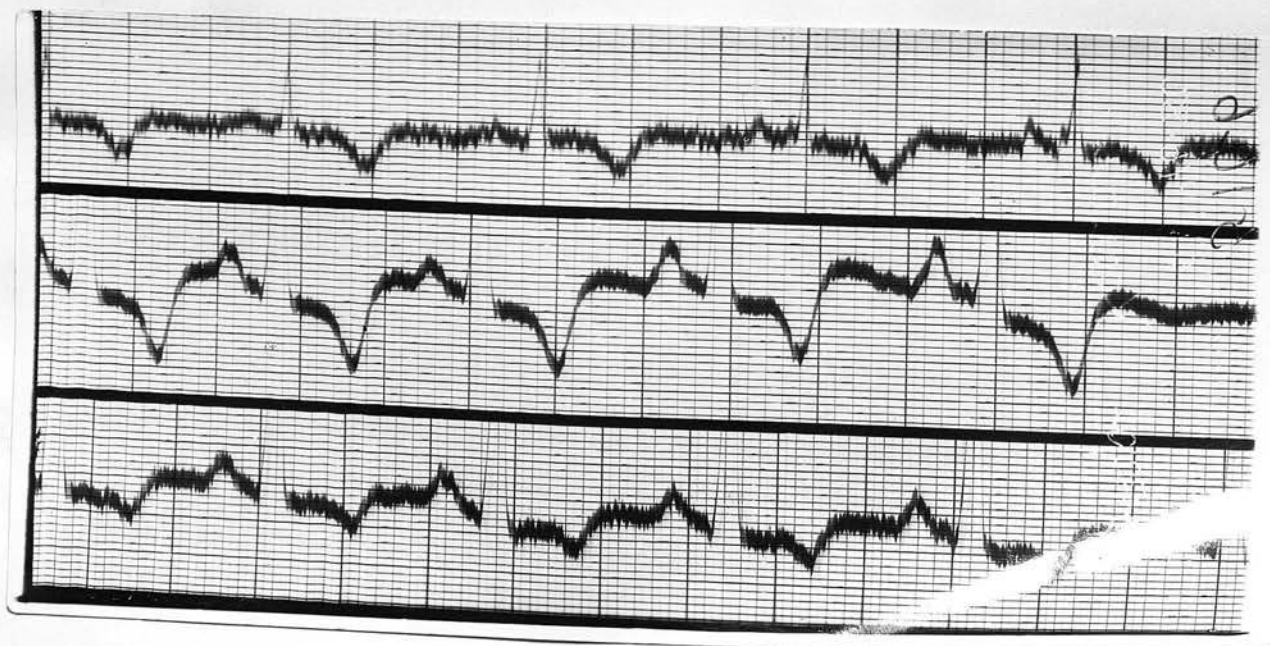
Nervous System: N.A.D.

W.R.: Negative.

Electrocardiogram: Sinus Arrhythmia.
T inverted all leads.
T₂ sharp and large.

Diagnosis: Aortic stenosis and incompetence.
Mitral stenosis.
Rheumatic.

Circulatory Rate: $10\frac{4}{5}$ secs.



Case 60.Sex: Female. Married.Age: 30 years.Occupation: Housewife.

History: 2 years. Occasional attacks of 'difficulty in breathing' which occur at night, shortly after falling to sleep. 3 such attacks during last 3 months. Attack lasts 10 to 15 minutes and is accompanied by slight praecordial pain, palpitation, and faintness.

1 year. Dyspnoea and palpitation on exertion. Giddiness.

Previous Illnesses: 'Acute rheumatism' on and off last 10 years.
 Growing pains, childhood.
 Tonsillitis every winter.
 Influenza.
 Kidney abscess.

Family History: Nil relevant. No children or miscarriages.
 Menses regular.

Examination: Pale. Weight 9 st. 4 lbs.

Circulatory System: Heart rate, 82; regular.
 Blood-pressure, 142/78.
 Apex beat: 6th interspace at mid-clavicular line.
 Heart sounds: Mitral area, systolic bruit.
 Aortic area, systolic bruit.
 Diastolic bruit, left sternal border 3rd interspace.

Lungs: N.A.D.

Case 60 (continued)

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

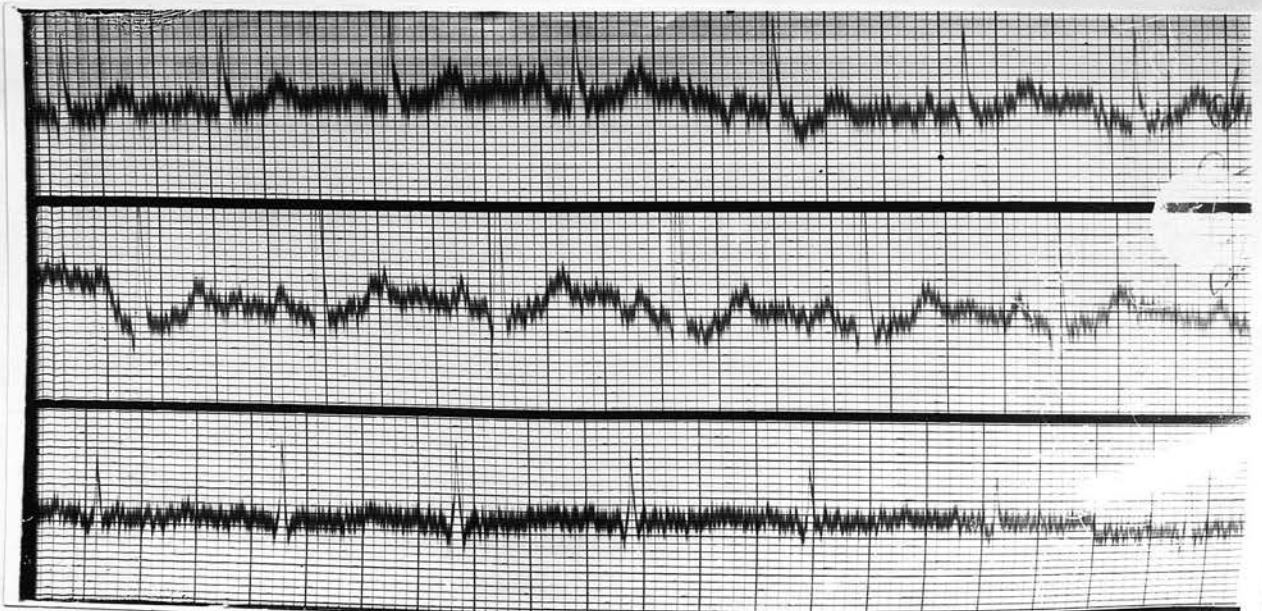
W.R: Negative

Electrocardiogram: Q₂ present.

T₃ flat.

Diagnosis: Aortic incompetence.
Rheumatic.

Circulatory Rate: $11\frac{4}{5}$ secs.



Case 67.

Sex: Female. Single.

Age: 23 years.

Occupation: Typist.

History: 3 years ago, after influenza, was told her heart was affected. An electrocardiographic examination at that time showed prolongation P - R interval and inversion of T waves. ? digitalis effect. Kept well until 1 month ago, when she again developed influenza. In bed 15 days. On getting up complained tiredness and palpitation.

Previous Illnesses: Growing pains, childhood.
Influenza.

Examination: Weight, 8 st. 1½ lbs.

Circulatory System: Heart rate: 74; regular.

Blood pressure, 140/80.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Mitral area, loud systolic
bruit.

Pulmonary 2nd sound accentuated

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

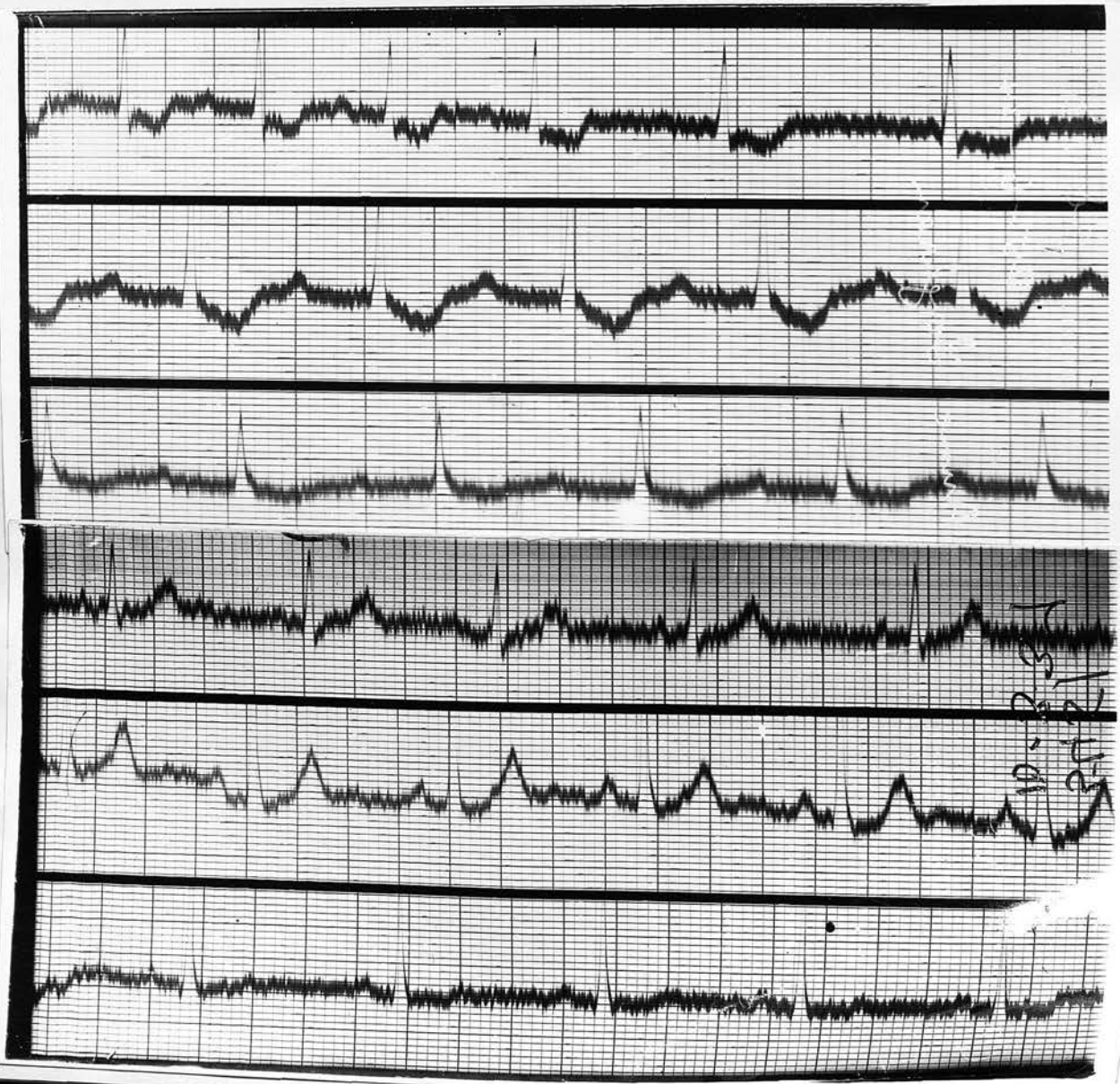
Case 67 (continued)

W.R.: Negative.

Electrocardiogram: (1) P-R. INTERVAL PROLONGED
T WAVES INVERTED
(2) NIL. ABNORMAL.

Diagnosis: Mitral incompetence.
Rheumatic.

Circulatory Rate: $10\frac{2}{5}$ seconds.



Case 75.

Sex: Female. Single.

Age: 48 years.

Occupation: Nil.

History: 5 years, Dyspnoea on exertion; palpitation, giddiness, and exhaustion.

Occasional praecordial ache.

Previous Illnesses: 'Growing pains', childhood.
Influenza.

Family History: Nil relevant.

Examination: Weight, 9 stones. Slightly cyanotic.

Circulatory System: Heart rate, 76; regular.

Blood pressure, 124/70.

Apex beat: 5th interspace just
outside mid-clavicular line.

Heart sounds: Mitral area, presystolic bruit.
Pulmonary 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

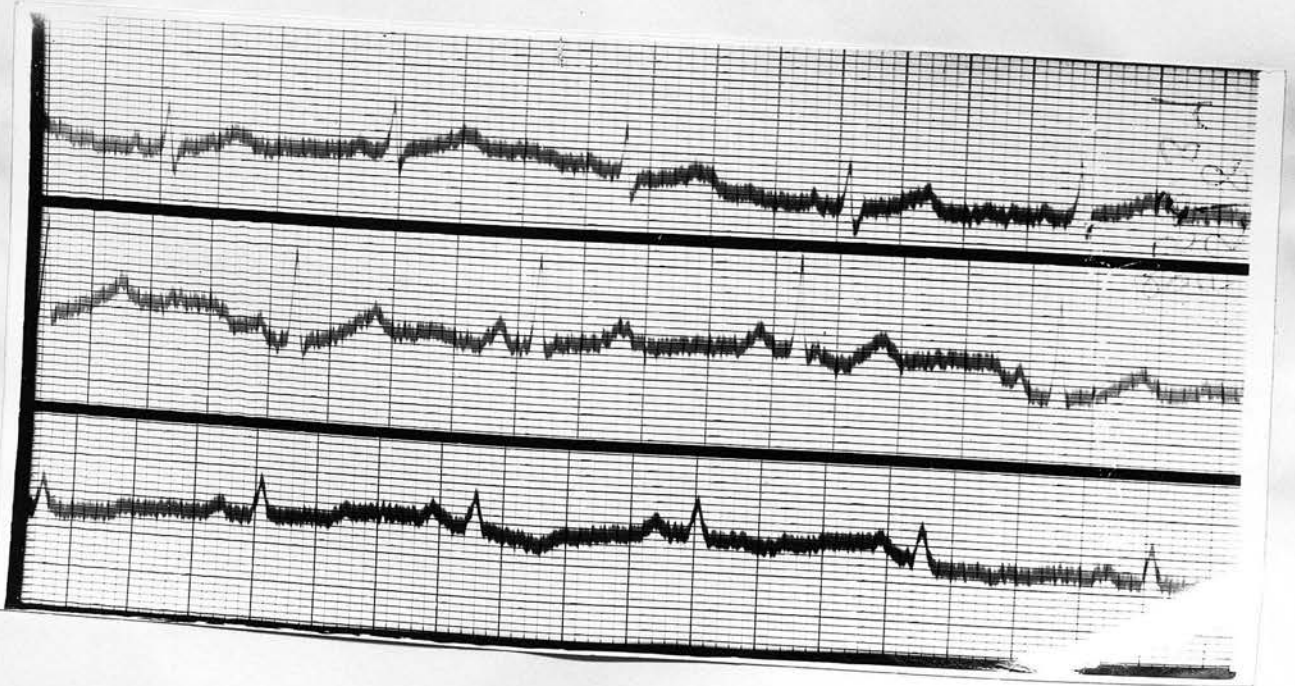
Case 75 (continued)

W.R.: Negative.

Electrocardiogram: R₃ slightly slurred.

Diagnosis: Mitral stenosis.
Rheumatic.

Circulatory Rate: $19\frac{1}{5}$ secs.



Case 82.

Sex: Female. Single.

Age: 23 years.

Occupation: Shop assistant.

History: 15 months. Dyspnoea and palpitation on exertion. Giddiness and exhaustion. Feeling of constriction in chest. Unable to work 15 months and in bed 3 months. Now complains dyspnoea on exertion (slight).

Previous Illnesses: Rheumatism in legs aet. 21 years.
Tonsillitis, frequent attacks;
tonsillectomy aet. 15 years.
Influenza.
Scarlet Fever aet. 17 years.

Family History: Nil relevant.

Examination: Weight 7 st. 2 lbs.

Circulatory System: Heart rate, 78; irregular. Extrasystoles.
Blood-pressure, 138/60.
Apex Beat: 5th interspace at
mid-clavicular line.
Diffuse.
Heart sounds: Mitral area, systolic and
diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

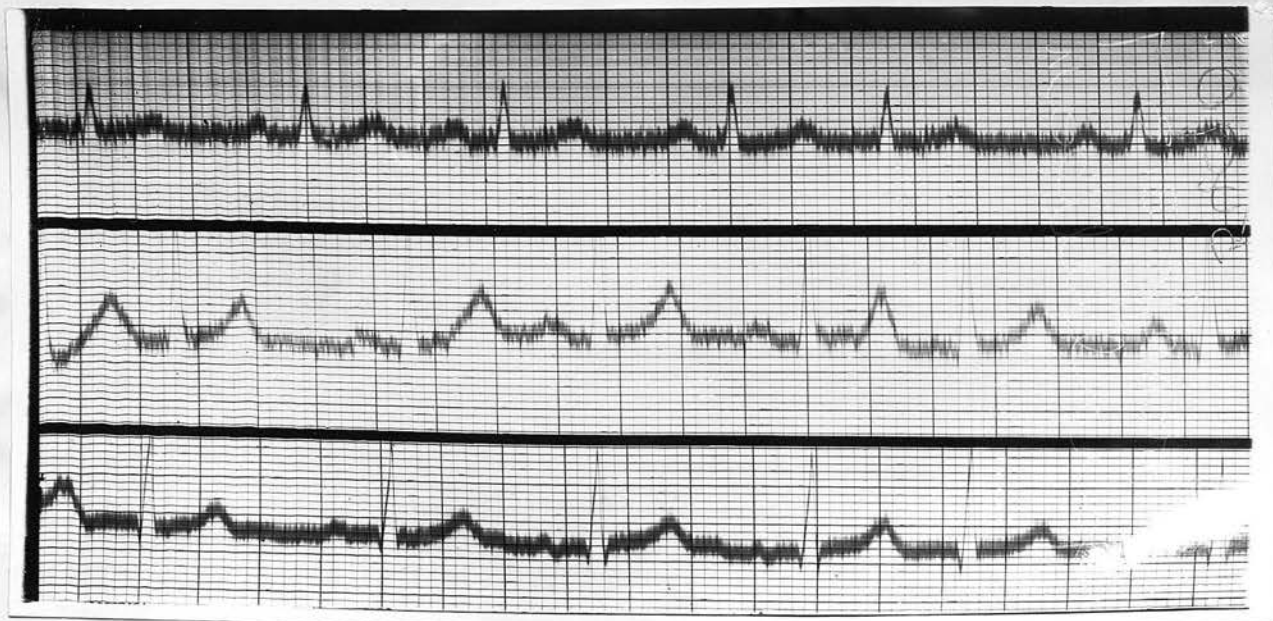
Case 82 (continued)

W.R.: Negative.

Electrocardiogram: R₁ slurred.

Diagnosis: Mitral stenosis.
Rheumatic.

Circulatory Rate: $15\frac{4}{5}$ secs.



Case 86.

Sex: Male. Single.

Age: 17 years.

Occupation: Nil.

History: 2 years. Dyspnoea on exertion and exhaustion.
Following an attack of pleurisy and 'congestion of
the lungs'. Has been resting on and off since.

Previous Illnesses: Growing pains, childhood.
Tonsillitis.
Scarlet fever, aet. 6 years.

Examination: Pale. Weight 8 stones.

Circulatory System: Heart rate, 80; regular.
Blood-pressure, 126/84.
Apex beat: 6th interspace outside
mid-clavicular line.
Forcible in character.
Presystolic thrill at apex.
Heart sounds: Mitral area, presystolic bruit.
Aortic area, systolic and
diastolic bruits.
Pulmonary 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 86 (continued)

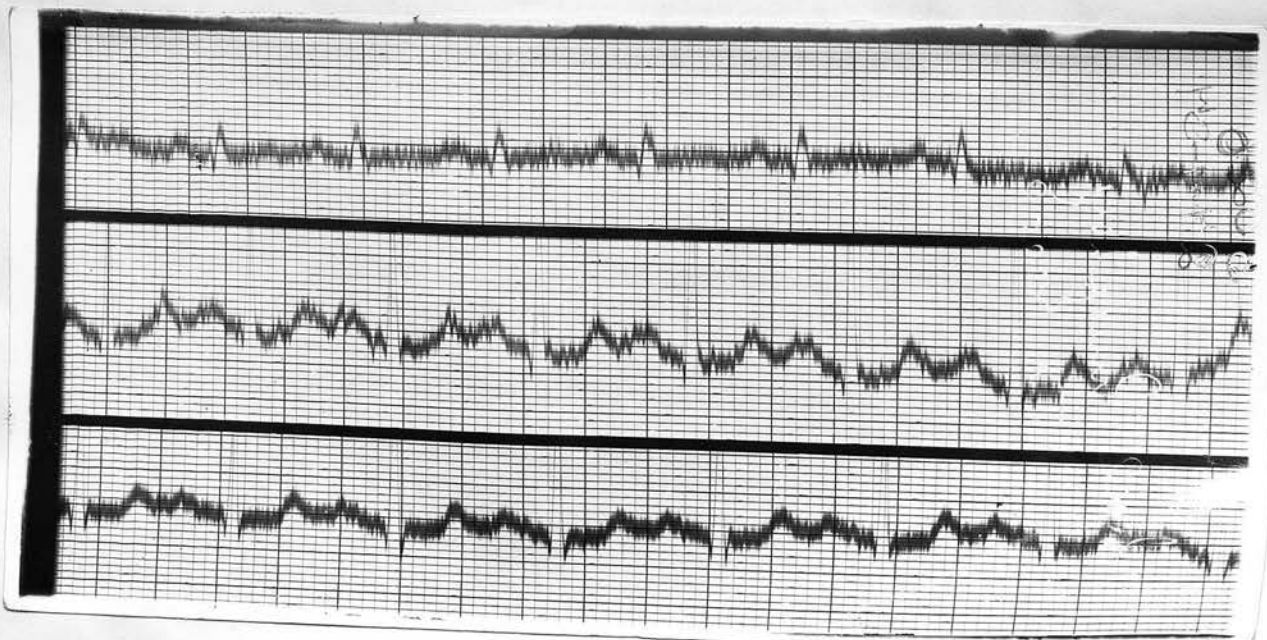
Nervous system: N.A.D.

W.R.: Negative.

Electrocardiogram: R₁ notched.

Diagnosis: Mitral stenosis.
Aortic incompetence.
Rheumatic.

Circulatory Rate: $13\frac{2}{5}$ secs.



Case 97.

Sex: Female. Married.

Age: 26 years.

Occupation: Housewife.

History: 4 months ago, while in bed at night, experienced a sudden attack of palpitation which lasted a few moments, and has not recurred. Since then has complained of slackness and weakness in the legs.

Able to continue her household duties.

Catamenia regular.

Previous Illnesses: Growing pains, childhood.
Measles.

Family History: 1 child.

Examination: Flushed appearance. Weight 9 st. 2 lbs.

Circulatory System: Heart rate, 82; regular.

Blood-pressure, 112/70.

Apex beat: 5th interspace at mid-clavicular line.

Diffuse in character.

Heart sounds: Mitral area, systolic and diastolic bruits.

Aortic area, systolic bruit.

Pulmonary 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil

Urine: N.A.D.

Case 97 (continued)

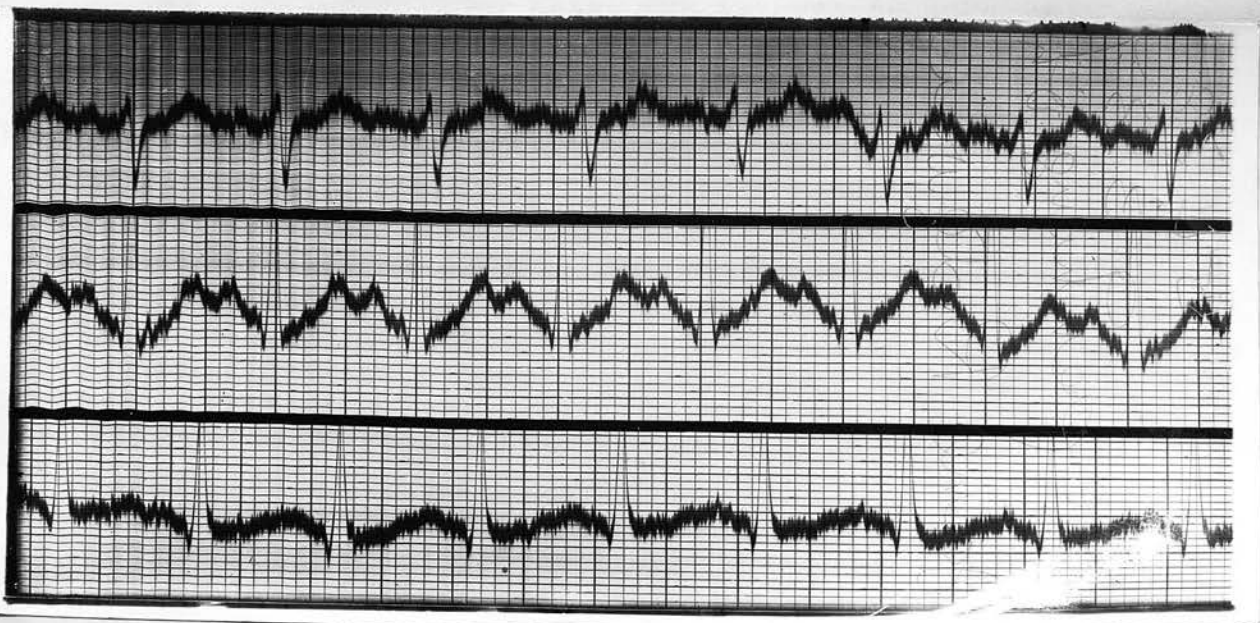
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: Right ventricular preponderance.
QRS slurred.

Diagnosis: Mitral stenosis.
Rheumatic.

Circulatory Rate: $8\frac{1}{5}$ secs.



Case 124.

Sex: Male. Single.

Age: 17 years.

Occupation: Nil.

History: 10 days ago, sudden attack pain and swelling in knee joints, preceded by a sore throat and accompanied by a temperature of 102°. Temperature, joint pains, and swelling subsided in 5 days' time, under the influence of salicylates and at present no pain or discomfort is complained of.

Previous Illnesses: Rheumatic fever, aet. 10 years.
Tonsillitis.

Habits: Alcohol and tobacco nil.

Examination: Weight 7 st. 6 lbs. Temperature normal.

Circulatory System: Heart rate, 106; regular.

Blood pressure, 104/58.

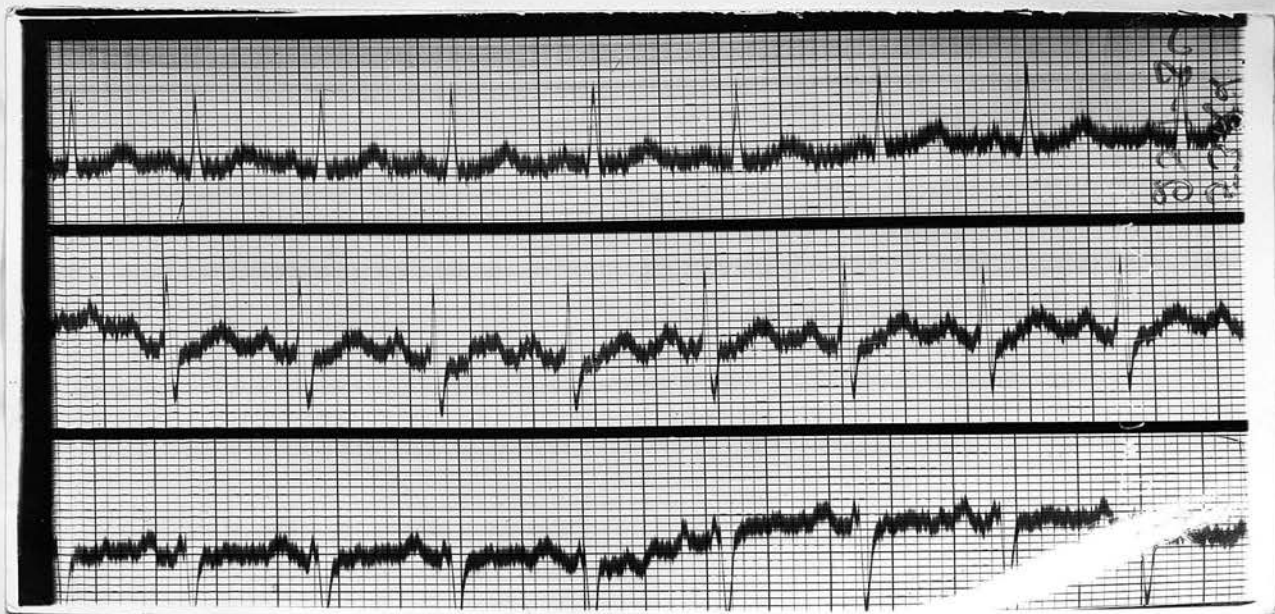
Apex beat: 5th interspace outside
mid-clavicular line.

Heart sounds: Mitral area, harsh systolic bruit.
Aortic area, systolic and
diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Case 124 (continued)Urine: N.A.D.Nervous System: N.A.D.W.R.: Negative.Electrocardiogram: Left ventricular preponderance.
T₃ flat.Diagnosis: Mitral stenosis.
Aortic incompetence.
Rheumatic.Circulatory Rate: $14\frac{1}{5}$ secs.

Case 125.

Sex: Female. Single.

Age: 23 years.

Occupation: Nurse.

History: Recently under treatment for a septic finger. Medically examined and was dismissed her employment, said to have 'Heart Trouble'. Has been in bed 14 days and now complains dyspnoea on exertion. Giddiness and faintness in the mornings.

Previous Illnesses: Tonsillitis.
Nephritis
? Chorea, childhood.
Appendicitis.
Whooping cough.
Measles.

Examination: Weight, 9 st. 2 lbs. Anaemic.

Circulatory System: Heart rate, 84; regular.
Temperature sub-normal.
Blood-pressure, 130/78
Apex beat: 5th interspace inside
mid-clavicular line.
Heart sounds: Mitral area, harsh systolic bruit.
Aortic area, diastolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 125 (contd.)

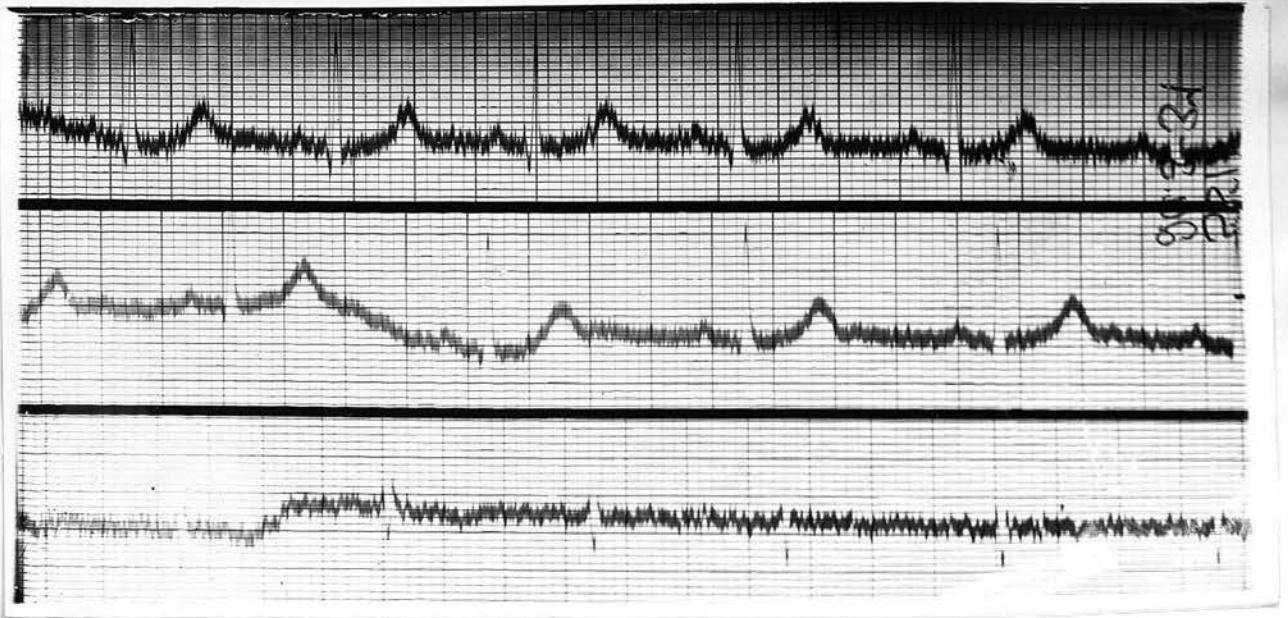
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: LEFT VENTRICULAR PREPONDERANCE

Diagnosis: Aortic incompetence.
 Mitral incompetence.
 Rheumatic.
 Anaemia.

Circulatory Rate: $8\frac{2}{5}$ secs.



Case 128.

Sex: Male. Single.

Age: 17 years.

Occupation: Clerk.

History: 14 days. Palpitation, commenced while in bed.
Giddiness.

Previous Illnesses: Growing pains, childhood.
Tonsillitis.
Scarlet fever.

Examination: Weight, 10 st. 1 lb.
Temperature, 97.8°.
Pale.

Circulatory System: Heart rate, 87; regular.
Blood-pressure, 146/84.
Apex beat: 5th interspace at
mid-clavicular line.
Slightly forcible.
Heart sounds: Aortic area, diastolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous system: N.A.D.

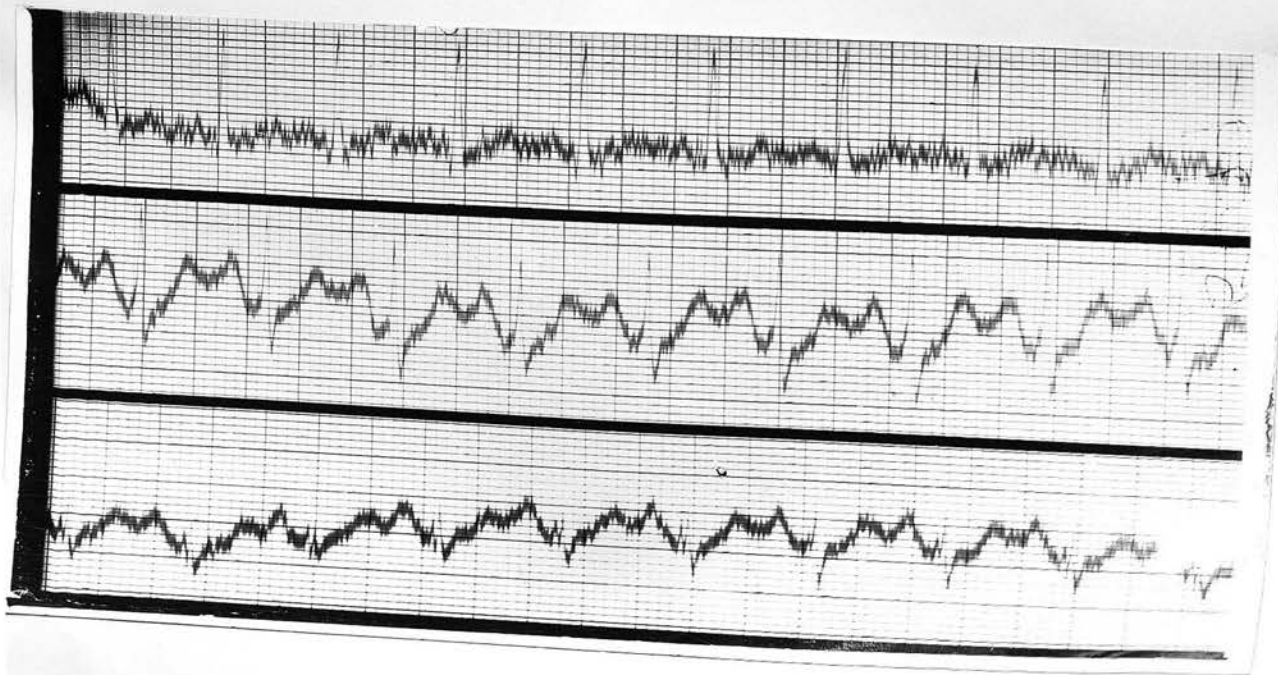
Case 128 (continued)

W.R.: Negative.

Electrocardiogram: Left ventricular preponderance.

Diagnosis: Aortic incompetence.
Rheumatic.

Circulatory Rate: $8\frac{3}{5}$ secs.



Case No.

Sex: Female. Single.

Age: 20 years.

Occupation: Nil.

History: 3 years. Increasing weakness & exertion
dyspnoea, orthopnoea, cough with sputum.

1 year. Spontaneous haemoptysis on several
occasions.

GROUP 3

20 cases

Family History: Nil.

Cardiac Failure.

Average circulatory rate 23.8 secs.

(Range 17.6 - 39.6 secs.)

Family History: Nil relevant.

Build: Weight 11 st. Stature 5 ft. 6 in.

Respiratory System: Heart rate, 96; regular.

Blood pressure, 126/80.

Sp. grav. 5th intercostal space
mid-clavicular line.

Heart sounds: Murmur area, Presystolic murmur
Diastolic 3rd and 4th intercostal spaces.

Roentgen: No pulmonary vascular congestion.

Abdomen: N.A.B.

Urine: Nil.

Case 10.

Sex: Female. Single.

Age: 20 years.

Occupation: Nil.

History: 3 years. Increasing dyspnoea on exertion.
Palpitation, exhaustion, cough with sputum.

1 year. Sputum has been blood-stained on several occasions.

6 months. Experienced left Respiratory pain frequently.

Previous Illnesses: Growing Pains, childhood.

Tonsillitis, several attacks.

Measles, Whooping Cough and Pneumonia, aet. 11. years, followed by persistent cough for 3 years.

'Tuberculous abscess at bottom of spine', aet. 7 years.

Family History: Nil relevant.

Examination: Weight 11 st. Cyanosis of cheeks and ears.

Circulatory System: Heart rate, 86; regular.

Blood pressure, 126/84.

Apex beat: 5th interspace inside
mid-clavicular line.

Heart sounds: Mitral area, Presystolic bruit.
Pulmonary 2nd sound accentuated.

Lungs: Harsh vesicular breath sounds.
Crepitations bases.

Abdomen: N.A.D.

Oedema: Nil.

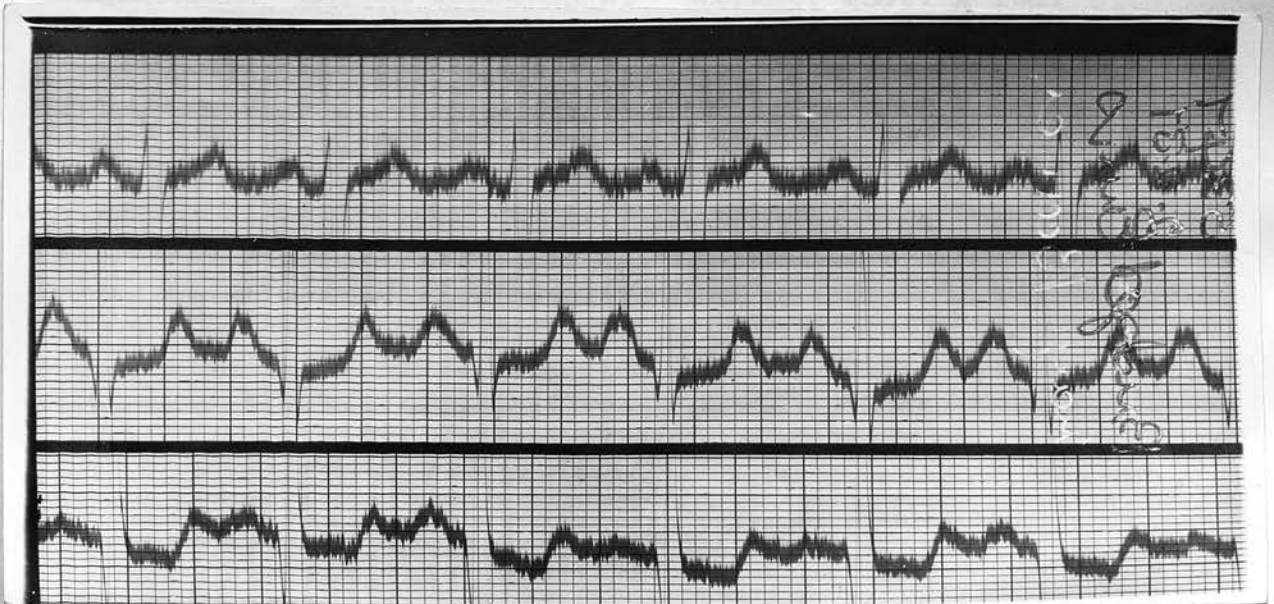
Case 10 (continued)Urine: Albumin trace.Nervous System: N.A.D.W.R: Negative.

Electrocardiogram: P₂ large and split.
 RT Depression in II and III
 Right ventricular preponderance.

X-ray Examination: 'Heart shadow, left auricular enlargement.
 Signs of Pulmonary stasis'.

Diagnosis: Congestive failure.
 Mitral stenosis.
 Rheumatic.

Circulatory Rate: $20\frac{4}{5}$ secs.



Case 11.Sex: Male. Married.Age: 47 years.Occupation: Grinder.

History: 3 months ago, had an attack of 'influenza', followed by 'bronchitis'. Since recovering from the influenza attack, has experienced increasing shortness of breath on exertion, and cough. The dyspnoea is now so marked, that he is unable to walk more than 50 yards without distress. For the last six weeks, has been taking 'digitalis leaf', on his own initiative and in a haphazard fashion. Had a previous attack of dyspnoea 3 months ago, which necessitated his confinement to bed for a month.

Previous Illnesses: Rheumatic fever, four attacks;
 aet. 12 years. 23 years.
 " 18 years. 39 years.
 'Rheumatism in right shoulder joint.'
 Influenza.

Examination: Sallow appearance. Weight, lost. 2 lbs.
 Lips, cyanotic.

Circulatory System: Heart rate, 80; irregular.
 Blood pressure, systolic 136
 Apex beat: 5th interspace outside
 mid-clavicular line.
 Heart sounds: Auricular Fibrillation.
 Mitral Area, systolic and
 diastolic bruits.

Lungs: Scattered crepitations basesAbdomen: N.A.D.Oedema: Nil.

Case 11 (continued)

Urine: N.A.D.

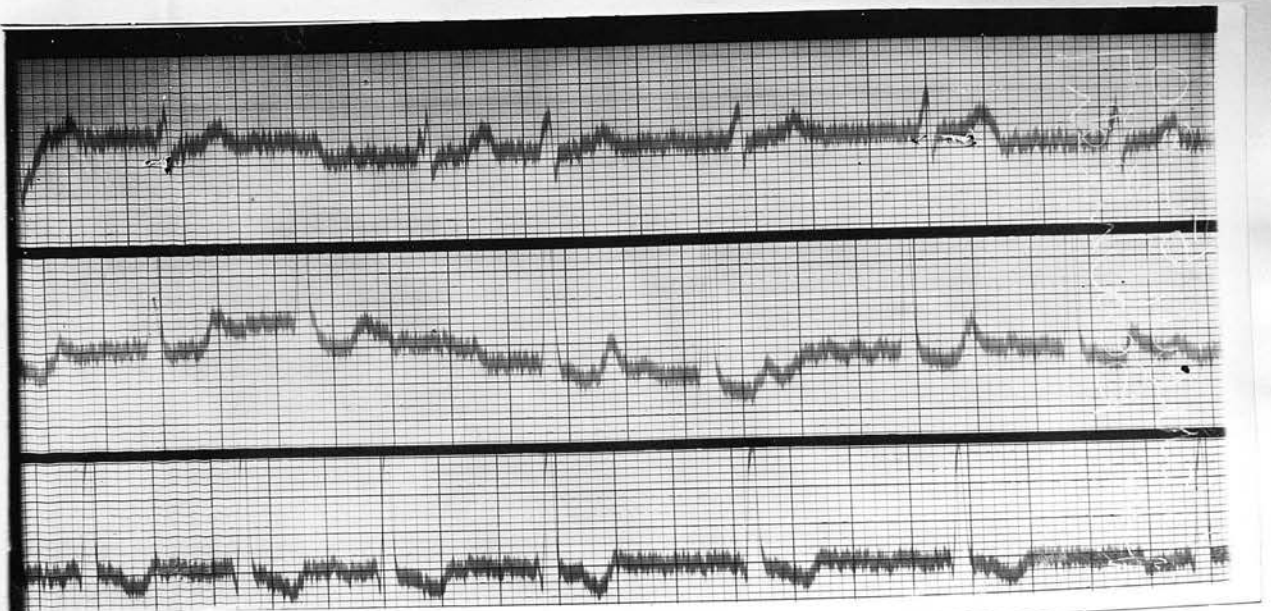
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent. QRS slurred
T₃ inverted. Auricular Fibrillation.

Diagnosis: Auricular Fibrillation.
Mitral Stenosis.
Rheumatic.

Circulatory Rate: $17\frac{2}{5}$ secs.



Case 14.Sex: Female. Married.Age: 48 years.Occupation: Housewife.

History: 10 years. Dyspnoea on exertion. Palpitation and exhaustion. Has been in bed for months at a time during the last 10 years. Been confined to bed for the last 6 weeks.

Now complains dyspnoea, palpitation, epigastric pain and vomiting, cough with slight sputum.

Previous Illnesses: Frequent attacks of 'Rheumatism' since childhood. No direct history of Rheumatic Fever.

Tonsillitis.

Family History: Childless. No history of miscarriages.

Examination: Weight, 10 st. 12 lbs.

Orthopnoeic.

Circulatory System: Heart rate, 80; irregular.

Blood pressure, systolic 140.

Apex beat: 6th interspace outside mid-clavicular line.
Diffuse in character.

Heart sounds: Auricular Fibrillation.

Mitral area, systolic and diastolic bruits present.

Pulmonary 2nd sound accentuated.

Lungs: Percussion note impaired.

Crepitations at bases.

Abdomen: Liver enlarged.

Gall Bladder enlarged and tender.

(History suggestive of Cholecystitis last 6 years).

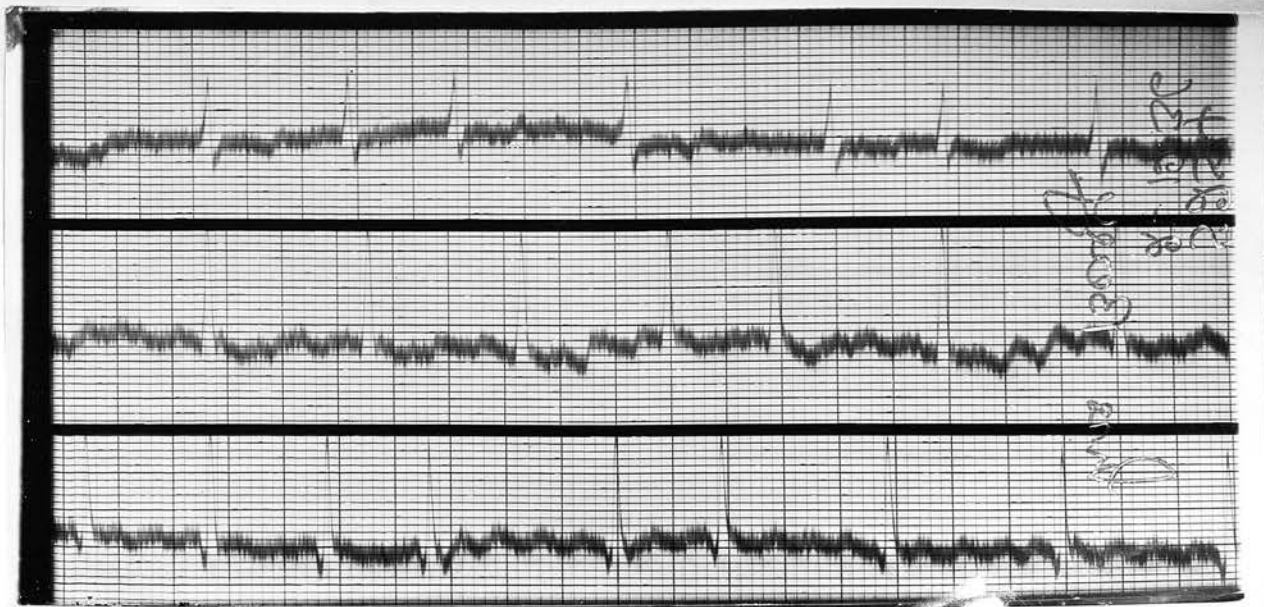
Case 14 (continued)Oedema: Nil.Urine: N.A.D.Nervous System: N.A.D.W.R: Negative.

Electrocardiogram: P absent.
Q₂ Q₃ present.
R - T depression.
Auricular fibrillation.

Diagnosis: Auricular fibrillation.
Mitral stenosis.
Rheumatic.
Chronic cholecystitis.

Circulatory Rate: $20\frac{2}{5}$ secs.

Was being treated by daily intra-muscular injections of strophanthin 0.5 mg.



Case 31.

Sex: Male. Married.

Age: 61 years.

Occupation: Dock Labourer.

History: 3 years. Dyspnoea on exertion, and cough. Continued at work until 1 month ago, when following an attack of influenza, he experienced increasing dyspnoea, swelling of the feet and 'stomach', palpitation, and cough with frothy sputum.

Been confined to bed, 14 days.

Previous Illnesses: Influenza.
Bronchitis.

Habits: Alcohol moderate; Tobacco, heavy smoker.

Examination: Cyanosis, face and ears. Orthopnoeic.
Weight 10 st. 4 lbs.

Circulatory System: Heart rate, 92; irregular.
Blood pressure, 132/80
Apex beat: Not palpable.
Heart sounds: Auricular fibrillation.
Mitral area, systolic bruit.

Lungs: Slight emphysema.
Percussion note impaired both bases.
Crepitations both bases.

Abdomen: Liver enlarged. 1 hand's breadth below costal margin.
Tender. Ascites present.

Oedema: Slight, ankles and up to knees.
Sacral oedema.

Case 31 (continued)

Urine: Albumin present.

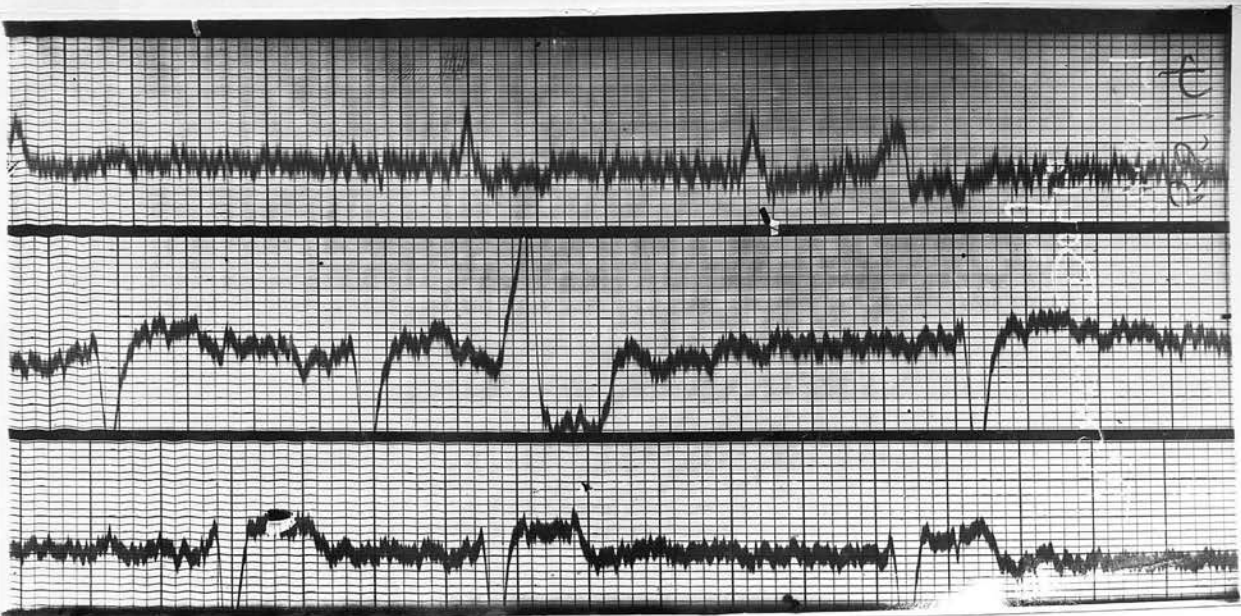
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P numerous waves.
Left ventricular preponderance.
RT depression.
? Auricular flutter.

Diagnosis: Auricular flutter.
Myocardial Degeneration.

Circulatory Rate: $25\frac{2}{5}$ secs.



Case 42.

Sex: Female. Married.

Age: 50 years.

Occupation: Housewife.

History: 9 years. Dyspnoea on exertion. Palpitation and exhaustion.

1 year. Cough with sputum - frothy.

Treated on and off for 8 years for 'Heart Disease'.

Previous Illnesses: Growing pains, childhood.
Tonsillitis, many attacks.

Family History: No children.
No history Miscarriages.
Menopause, æt. 47 years.

Examination: Weight 10 st. 6 lbs.
Cyanosed.
Cough troublesome.

Circulatory System: Heart rate, 94; irregular.
Blood pressure, 134/90.
Apex beat: 6th interspace outside
mid-clavicular line.
Heart sounds: Mitral area, systolic and
diastolic bruits.
Aortic area, systolic bruit.
Pulmonary 2nd sound accentuated.
Auricular fibrillation.

Lungs: Sputum frothy.
Crepitations both bases.

Case 42 (continued)

Abdomen: Liver, palpable and tender (3 finger breadths below costal margin).

Oedema: Ankles.

Urine: Albumin present.

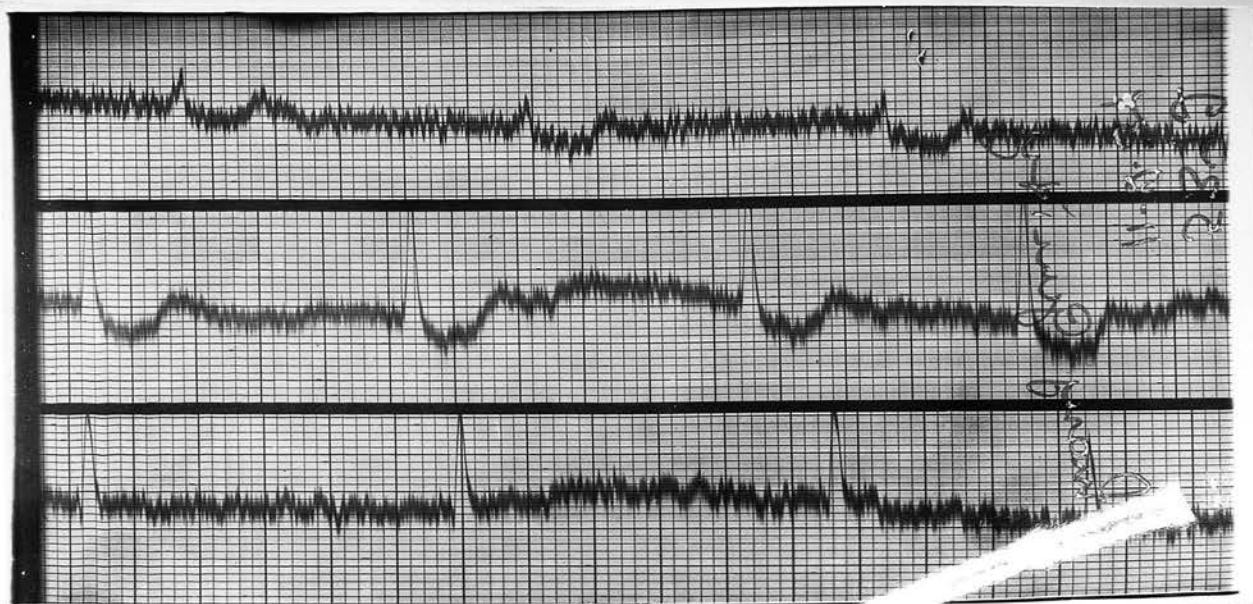
Nervous System: N.A.D.

W.R: Positive.

Electrocardiogram: Irregular rhythm, P absent.
R₁ R₃ splintered, Q₃ present.
RT depression, T₃ flat.

Diagnosis: Congestive failure.
Auricular Fibrillation.
Mitral Stenosis.
Rheumatic.

Circulatory Rate: $23\frac{3}{5}$ secs.



Case 44.Sex: Male. Widower.Age: 58 years.Occupation: Painter and Decorater.History: 5 years. Dyspnoea on exertion, and a sensation of constriction in the chest.

More recently attacks of giddiness and palpitation.

Kept at work up till 6 months ago.

20 years. Suffered from 'Acute gout' - sudden attacks of pain and swelling in both feet.

Previous Illnesses: Influenza, several attacks.

Gout.

Gonorrhoea, aet. 33 years.

Family History: Father and grandfather died 'cerebral haemorrhage'.Habits: Alcohol, beer 3 pints per diem.

Tobacco moderate.

Examination: Weight 10 st.

Slight Cyanosis.

Circulatory System: Heart Rate, 90; irregular.

Blood pressure, 270/150.

Apex beat: outside mid-clavicular line.
5th interspace.

Diffuse in character.

Heart sounds: Auricular Fibrillation.

Mitral Area, systolic bruit.

Aortic area, systolic bruit.
2nd sound
accentuated.Lungs: Basal Crepitations.Abdomen: N.A.D.

Case 44 (continued)Oedema: Nil.Urine: Albumen present.Nervous System: N.A.D.

Retinal arteries sclerosed.

Electrocardiogram: P absent.

QRS widened and slurred; irregular.

Left ventricular extrasystole in lead III.

T₃ inverted.T₃ biphasic.

Auricular fibrillation.

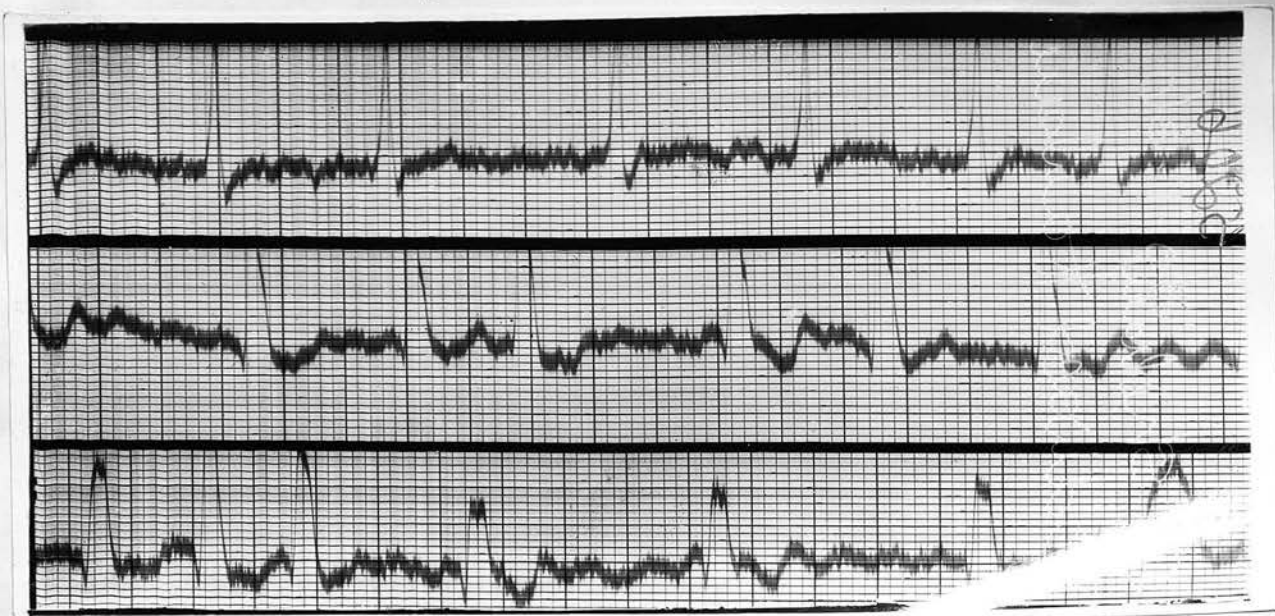
W.R.: Negative.Blood: Uric acid, 8 mgm per 100 ccs.Diagnosis: Auricular fibrillation.

Bundle Branch Block.

Myocardial degeneration.

Hyperpiesia.

Gout. ? Plumbism.

Circulatory Rate: $22\frac{1}{5}$ secs.

Case 56Sex: Female. Married.Age: 46 years.Occupation: Housewife.

History: 3 months ago, noticed feet swollen at night time. Followed by swelling of the stomach. Cough and increasing dyspnoea. Spent one month in bed, and was relieved, but on getting up, all the signs and symptoms returned; and she now complains of cough, frothy sputum - recently blood-stained, dyspnoea on exertion and also at rest, palpitation and insomnia (due to dyspnoea).

Previous Illnesses: Tonsillitis, many attacks.

16 years ago, following childbirth, was in bed '3 weeks' with 'Heart Trouble'.

Family History: 9 children.

No miscarriages.

Menopause, aet. 44 years.

Examination: Icteric Tinge.

Orthopnoeic.

Weight 10 st. 7 lbs.

Circulatory System: Heart rate, 130; irregular.

Cervical veins, congested.

Blood pressure: systolic, 126.

Apex beat: 5th interspace outside mid-clavicular line.

Diffuse.

Heart sounds: Gallop Rhythm present.

Auricular Fibrillation.

Mitral Area, systolic and diastolic bruits.

Aortic area, systolic bruit.

Lungs: Creptitations, left base.

Right, hydro-thorax.

Case 56 (continued)

Abdomen: Ascites present.

Liver enlarged; 1 hand's breadth below costal margin.
Tender.

Oedema: Feet and legs to knees.

Sacral Oedema.

Urine: Albumin present.

Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P. absent.

QRS slurred.

Auricular fibrillation.

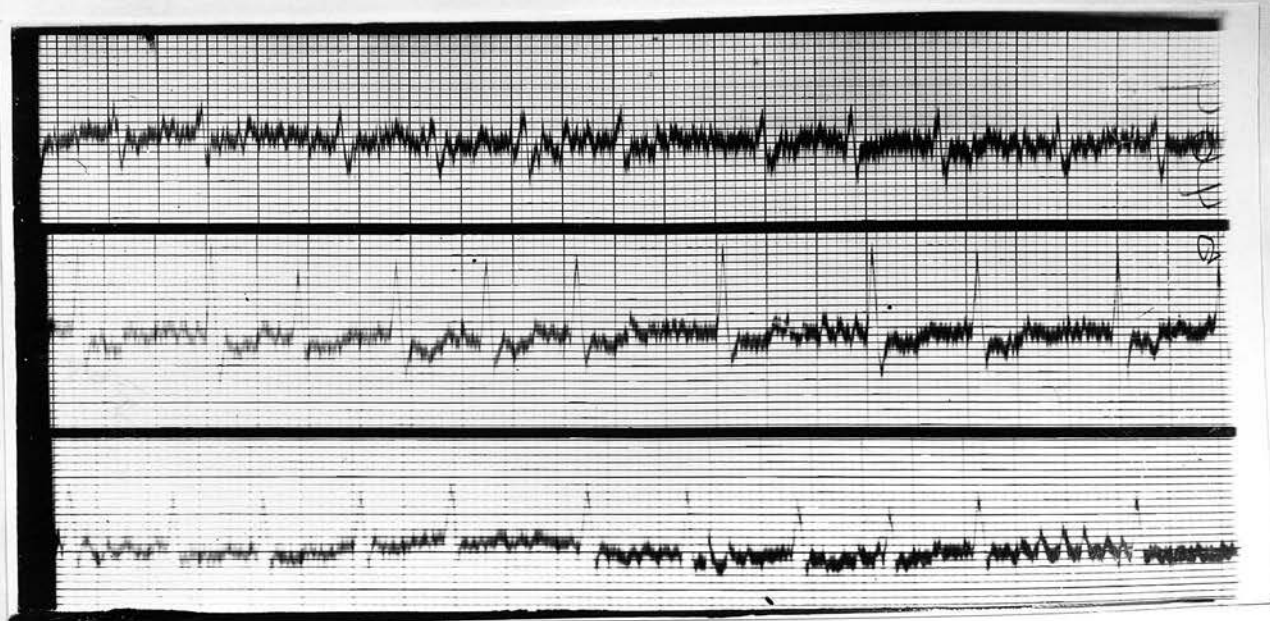
Diagnosis: Congestive failure.

Auricular Fibrillation.

Mitral Stenosis.

Rheumatic.

Circulatory Rate: $33\frac{1}{5}$ secs.



Case 61.

Sex: Male. Married.

Age: 60 years.

Occupation: Labourer.

History: 2 years. Dyspnoea on exertion. Cough with scanty sputum.

4 months. Attacks of nocturnal dyspnoea (cardiac asthma?).

Previous Illnesses: Influenza.

Family History: Nil relevant.

Habits: Alcohol, immoderate.

Tobacco, moderate.

Examination: Dyspnoeic.

Weight 8 st.

Circulatory System: Heart rate, 84; irregular.

Blood pressure, 220/110

Apex beat: 5th interspace outside
mid-clavicular line.

Heart Sounds: Gallop rhythm.

Mitral area, systolic bruit.

Aortic area, systolic bruit.

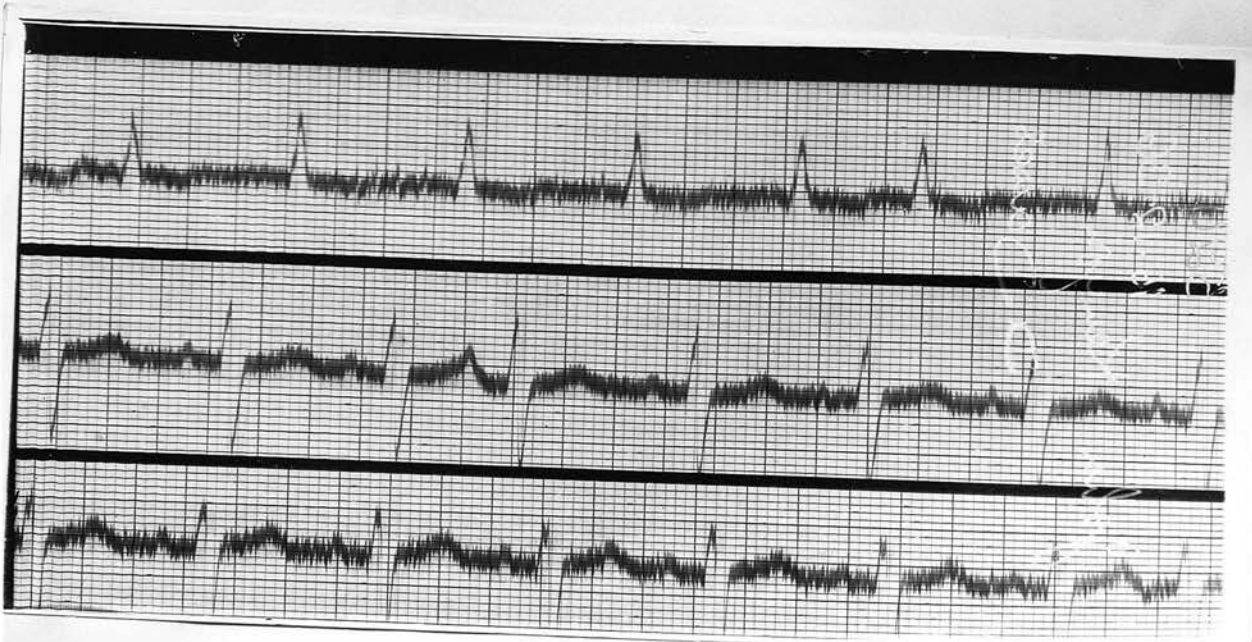
Aortic 2nd sound accentuated.

Lungs: Crepitations bases.

Abdomen: Liver enlarged, 2 finger breadths below costal margin.

Oedema: Nil.

Urine: Albumin.

Case 61 (continued)Nervous System: N.A.D.W.R.: Negative.Electrocardiogram: Left ventricular preponderance.
QRS slurred.
Auricular extrasystoles.Diagnosis: Cardiac Asthma.
Myocardial Degeneration.
Hyperpiesia.Circulatory Rate: 20 secs.

Case 70.

Sex: Male. Married.

Age: 57 years.

Occupation: Garage Worker.

History: 4 months, dyspnoea on exertion - getting worse, 6 weeks ago, while walking to work, experienced a sudden gripping sensation in his chest, and collapsed, vomited, and was sent home to bed, where he has been since. Complains now palpitation, faintness and loss of appetite.

Previous Illnesses: Rheumatic Fever, childhood.
Influenza.

Family History: Nil relevant.

Habits: Alcohol and Tobacco, moderate.

Examination: Pale.
Slightly dyspnoeic.
Weight 12 st. 7 lbs.

Circulatory System: Heart rate, 76; regular.
Peripheral vessels sclerosed.
Blood pressure, 98/74.
Apex beat: Not palpable.
Heart sounds; distant.
Mitral area, systolic bruit.
Aortic area, systolic bruit.

Lungs: Crepitations both bases.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Case 70 (continued)

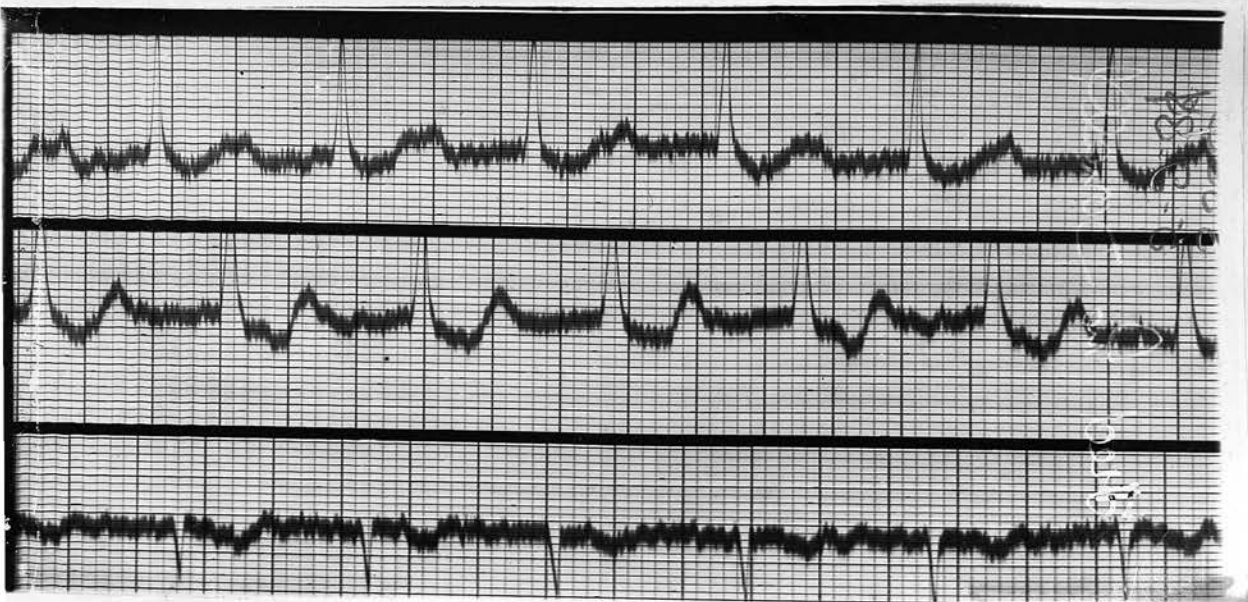
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: Left ventricular preponderance.
R-T depression.

Diagnosis: Coronary thrombosis.
Myocardial degeneration.

Circulatory Rate: $20\frac{3}{5}$ secs.
(Blood-pressure taken immediately afterwards, systolic 124.)



Case 78.Sex: Male. Married.Age: 53 years.Occupation: Clerk.History: 6 weeks ago developed a dry cough which was followed by dyspnoea and praecordial pain on exertion.

3 weeks. Nocturnal attacks of dyspnoea - ? cardiac asthma.

Palpitation and giddiness.

Previous illnesses: Malaria 1903.
Scarlet fever, childhood.
V.D. denied.Family History: Father died aet.56 'heart failure'.Habits: Alcohol and tobacco - moderate.Examination: Weight 13 st. Dyspnoeic.Circulatory System: Heart rate, 76; irregular.
Blood-pressure 208/116.
Peripheral vessels sclerosed.
Apex beat: 6th interspace outside mid-clavicular line.
Heaving in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.
Extra systoles present.Lungs: Rhonchi both lungs. Crepitations bases.Abdomen: N.A.D.Oedema: Nil.

Case 78 (continued)

Urine: Albumin present.

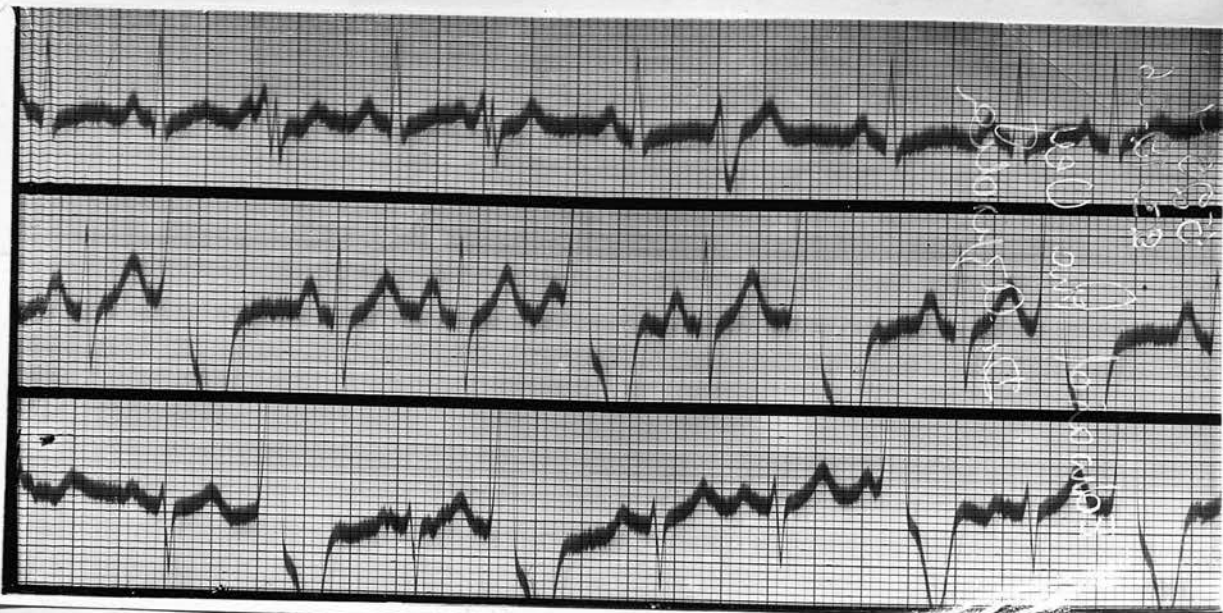
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: Left ventricular preponderance.
Right and left ventricular extrasystoles.

Diagnosis: Hyperpiesia.
Myocardial degeneration.
Cardiac asthma.

Circulatory Rate: 26 secs.



Case 80.Sex: Female. Married.Age: 51 years.Occupation: Housewife.

History: 3 years. Dyspnoea and palpitation on exertion.
 18 months ago treated in bed for 2 months for
 'heart failure'. Kept fairly well until 3 weeks
 ago, when following an attack of influenza,
 dyspnoea increased, and a cough with sputum developed.
 Managed to do her housework with difficulty.

Previous illnesses: Scarlet fever, childhood.
 Tonsillitis many times.

Family History: 2 children. 2 miscarriages after 1st pregnancy.
 Menopause, aet. 44 years.

Examination: Weight 6 st. 10 lbs.
 Dyspnoea.

Circulatory System: Heart rate, 126; irregular. Pale.
 Jugular congestion present.
 Blood-pressure, 118/50.
 Apex beat: 6th interspace outside
 mid-clavicular line.
 Diffuse.
 Heart sounds: auricular fibrillation.
 Mitral area, systolic and
 diastolic bruits.
 Aortic area, systolic and
 diastolic bruits.

Lungs: Crepitations bases.

Abdomen: Liver enlarged 2 finger breadths below costal margin.
 Tender.

Case 80 (continued)Oedema: Nil.Urine: Albumin trace.Nervous System: N.A.D.W.R.: Negative.Blood Count: Red blood cells 3,000,000 per cm.Electrocardiogram: P absent.

QRS slurred.

Auricular fibrillation.

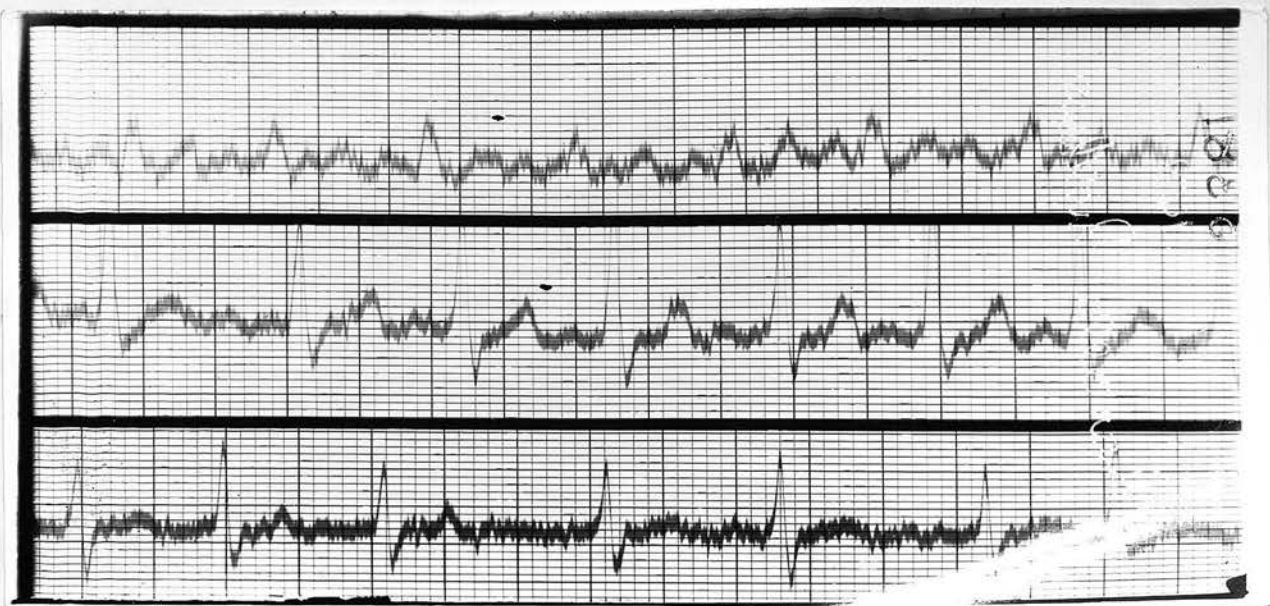
Diagnosis: Congestive failure.

Auricular fibrillation.

Aortic incompetence.

Mitral stenosis.

Rheumatic.

Circulatory Rate: 21 secs.

Case 85.

Sex: Male. Married.

Age: 65 years.

Occupation: Dock labourer.

History: 7 years. Dyspnoea on exertion, palpitation and cough. 6 years ago in bed 6 weeks with 'heart trouble', after which he resumed work and kept more or less well until 3 years ago, when he was compelled to go to bed for a month.

Unable to work 18 months.

In bed on and off 6 months.

3 weeks ago had influenza. This was followed by swelling of feet and bad cough.

Previous illnesses: Rheumatic fever, 4 attacks. The last being 25 years ago.

Tonsillitis, quinsy - many times.

Influenza.

Habits: Alcohol and tobacco moderate.

Examination: Pale man. Weight 11 st. 4 lbs.

Dyspnoeic.

Circulatory System: Heart rate, 60; irregular.

Blood-pressure 178/92.

Apex beat: 6th interspace outside mid-clavicular line.

Heart sounds; auricular fibrillation.

Mitral area, systolic and diastolic bruits.

Aortic area, diastolic bruit.

Case 85 (continued)

Lungs: Percussion note impaired bases.
Crepitation bases.

Abdomen: Liver enlarged 3 finger breadths below costal margin.
Tender.
Ascites present.

Oedema: Feet and legs. Sacrum.

Urine: Albumin present.

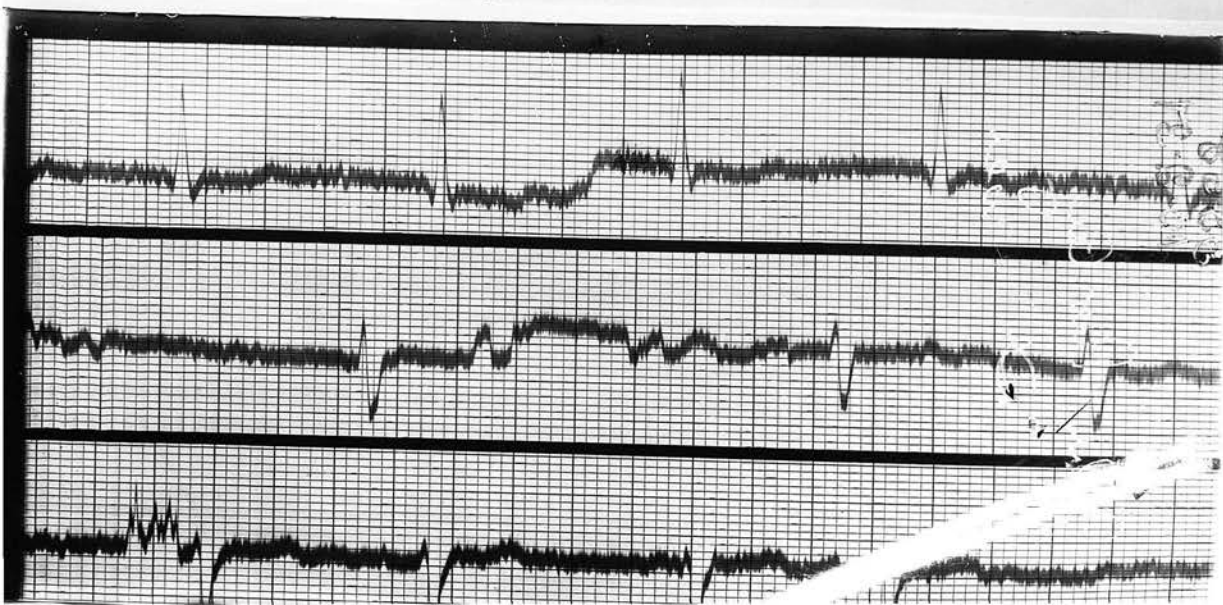
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent.
T flattened.
QRS slurred.
Left ventricular preponderance.

Diagnosis: Congestive failure.
Auricular fibrillation.
Aortic incompetence.
Mitral stenosis.
Rheumatic.

Circulatory Rate: $23\frac{1}{5}$ secs.



Case 88.

Sex: Female. Married.

Age: 36 years.

Occupation: Housewife.

History: 15 years. Intermittent rheumatic pains shoulders and limbs.

2 years. Dyspnoea on exertion. More recently at rest.

2 months. Choking sensation in chest.

Unable to do any housework for last 2 months, and confined to bed for the past month.

1 month. Cough with blood-stained sputum.

Palpitation and fluttering sensation in the chest, exhaustion and anorexia.

Previous illnesses: Rheumatic fever. 3 attacks. The last at aet. 21 years.
Chorea, childhood.
Pneumonia, aet. 7 years.

Family History: 2 children. 2 miscarriages.

Examination: Orthopneic. Weight 6 st. 9 lbs.
Cyanosed.
Icteric tinge face and conjunctivae.
Jugular veins congested.

Circulatory System: Heart rate, 110; irregular.
Apex beat: 5th interspace outside mid-clavicular line.
Diffuse in character.

Case 88 (continued)Circulatory System:

(Contd.)

Heart sounds: auricular fibrillation.

Mitral area; systolic and
diastolic bruits.

Pulmonary area; systolic bruit.

Lungs: Right hydrothorax.
Crepitations, left base.Abdomen: Liver enlarged one hand's breadth below costal margin.
Tender.
Ascites nil.Oedema: Feet, legs, and sacrum.Urine: Albumin.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: P waves absent.
S slurred.
QRS widened.
T₂ T₃ flat.Diagnosis: Congestive failure.
Auricular fibrillation.
Mitral stenosis.
Rheumatic.Circulatory Rate: $23\frac{1}{5}$ secs.

Case 88 (continued)

6 weeks later.

Patient had been digitalised, hydrothorax tapped, and treated with diuretics - ammonium chloride and salyrgan.

Examination: Weight 6 st. 2 lbs. (loss of 7 lbs.)

Circulatory System: Heart rate, 80; irregular.

Lungs: Basal crepitations.
Pleural friction right lung.

Abdomen: Liver not palpable.

Oedema: Slight sacral oedema.

Circulatory Rate: $23\frac{2}{5}$ secs.

6 weeks later.

Patient still under digitalis (Tr Digitalis x t.d.s).

Circulatory System: Heart rate, 70; irregular.

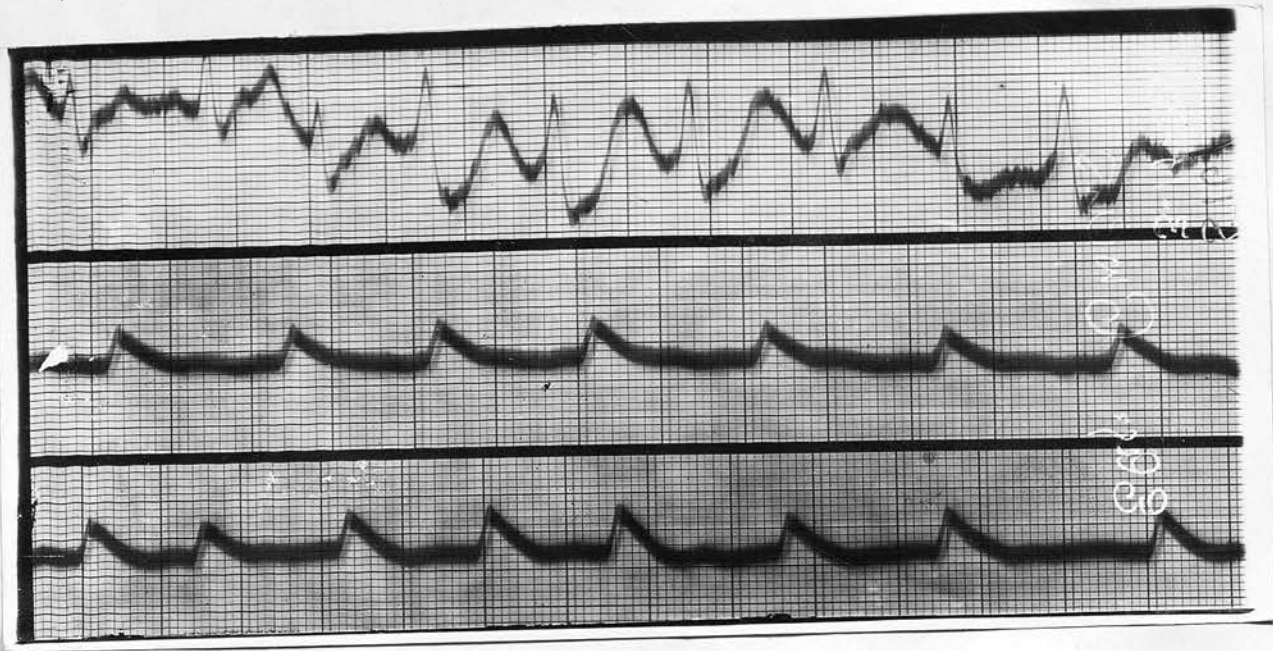
Lungs: Occasional basal crepitations.

Oedema: Nil.

Abdomen: N.A.D.

Circulatory Rate: $19\frac{1}{5}$ secs.

Case 88 (continued)



Case 91.

Sex: Male. Married.

Age: 61 years.

Occupation: Plumber.

History: 3 years. Praecordial distress - no actual pain on exertion. Dyspnoea on exertion.

Occipital headaches. Exhaustion. Giddiness.

Pains in calves of legs on walking - eased by rest.

Cough on occasions - during night time.

Previous illnesses: Pneumonia, aet. 57 years.

Haemoptysis, aet. 60.

X-ray reported normal.

Family History: Nil relevant.

Habits: Alcohol immoderate. Tobacco moderate.

Examination: Obese plethoric man. Weight 13 st.

Circulatory System: Heart Rate, 68; regular.

Peripheral vessels sclerosed.

Blood-pressure, 230/130.

Apex beat: impalpable.

Heart Sounds: Aortic area; systolic bruit.

Aortic 2nd sound accentuated.

Lungs: Crepitations both bases.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Case 91 (continued)

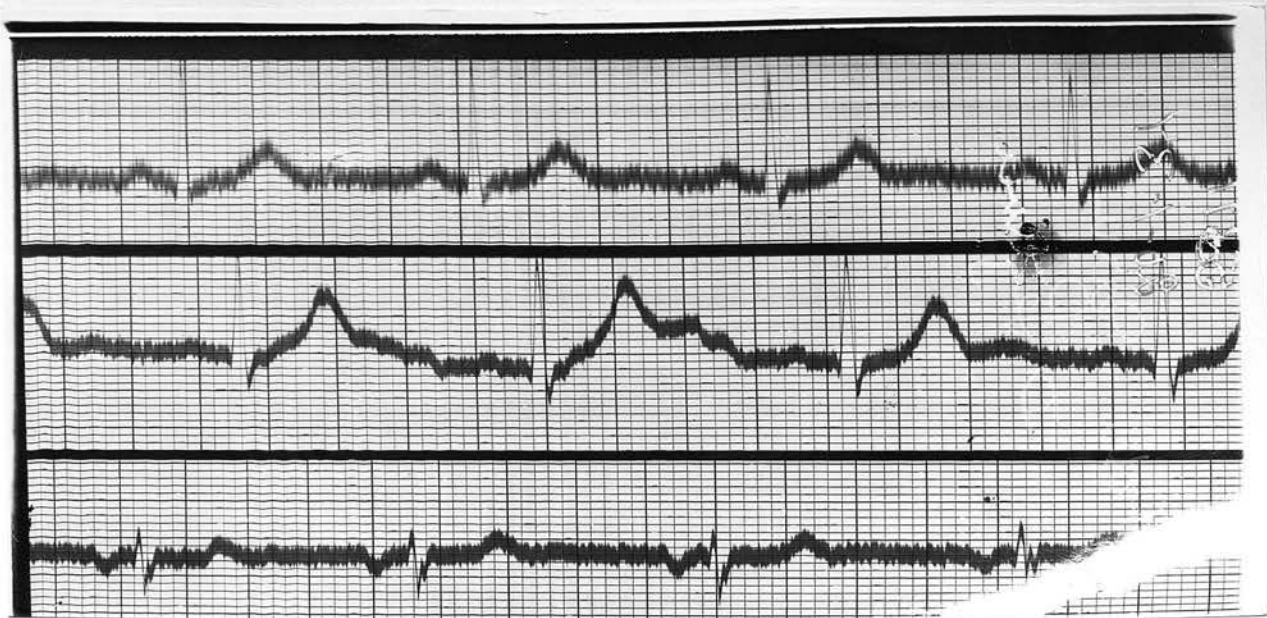
Nervous System: N.A.D.

W.R: Positive.

Electrocardiogram: P₃ inverted.
Left ventricular hypertrophy.
S₃ notched.

Diagnosis: 'Cardiac Asthma'.
Myocardial degeneration.
Intermittent claudication.
Hyperpiesia. ? Plumbism.

Circulatory Rate: $33\frac{3}{5}$ secs.



Case 95.

Sex: Female. Married.

Age: 35 years.

Occupation: Housewife.

History: 1 week. Palpitation and fluttering in chest. Commenced suddenly while in bed, has continued since and caused insomnia.

1 week. Dyspnoea on exertion, discomfort in region of left breast. Giddiness and slight cough.

Previous illnesses: Rheumatic fever, aet.7.
Tonsillitis.
Scarlet fever, aet.5.
Diphtheria.
Influenza.

Family History: 1 child.

Examination: Weight, 11 st. 6 lbs.

Circulatory System: Heart rate, 110; irregular.
Blood-pressure, ? 160/90.
Apex beat: 6th interspace outside mid-clavicular line.
Diffuse.
Heart sounds: auricular fibrillation.
Mitral area; systolic and dyastolic bruits.
Aortic area; systolic and dyastolic bruits.

Lungs: Basal crepitations.

Abdomen: N.A.D.

Case 95 (continued)

Oedema: Nil.

Urine: N.A.D.

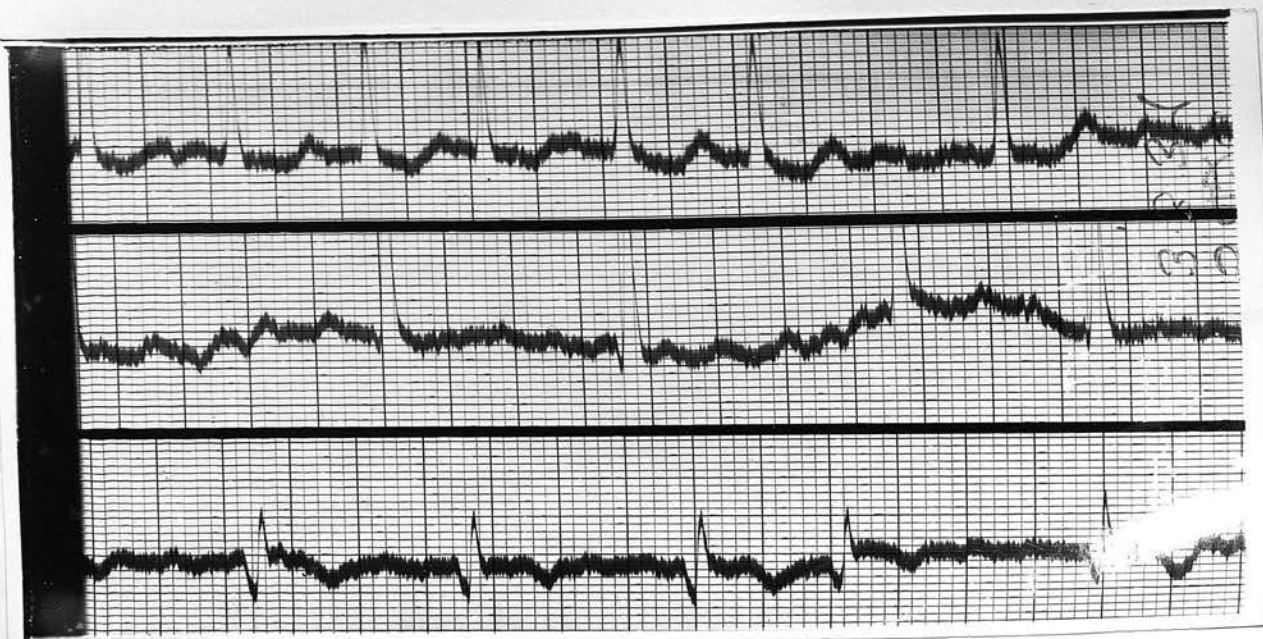
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent.
QRS slurred.
T₃ inverted.
Auricular fibrillation.

Diagnosis: Auricular fibrillation.
Mitral stenosis.
Aortic incompetence.
Rheumatic.

Circulatory System: $23\frac{2}{5}$ secs.



Case 98.Sex: Female. Single.Age: 25 years.Occupation: Book-binder.History: 3 years. Dyspnoea and palpitation on exertion.
Faintness.

2 months. Cough with slight sputum.

Unable to work last 2 years.

Previous illnesses: Chorea, aet. 10 years.
Growing pains, childhood.
Tonsillitis.
Scarlet fever, aet. 4 years.Examination: Orthopnoeic.
Cyanosis face and ears.
Weight, 7 st. 12 lbs.Circulatory System: Heart Rate, 110; irregular.
Blood-pressure ? 116/80.
Apex beat: 6th interspace outside
mid-clavicular line.
Diffuse in character.
Heart sounds: auricular fibrillation.
Mitral area; systolic and
diastolic bruits.
Aortic area; ? diastolic bruit.Lungs: Crepitations bases.Abdomen: Never enlarged to 2 finger breadths below costal margin.
Tender.Oedema: Nil.

Case 98 (continued)

Urine: Albumin trace.

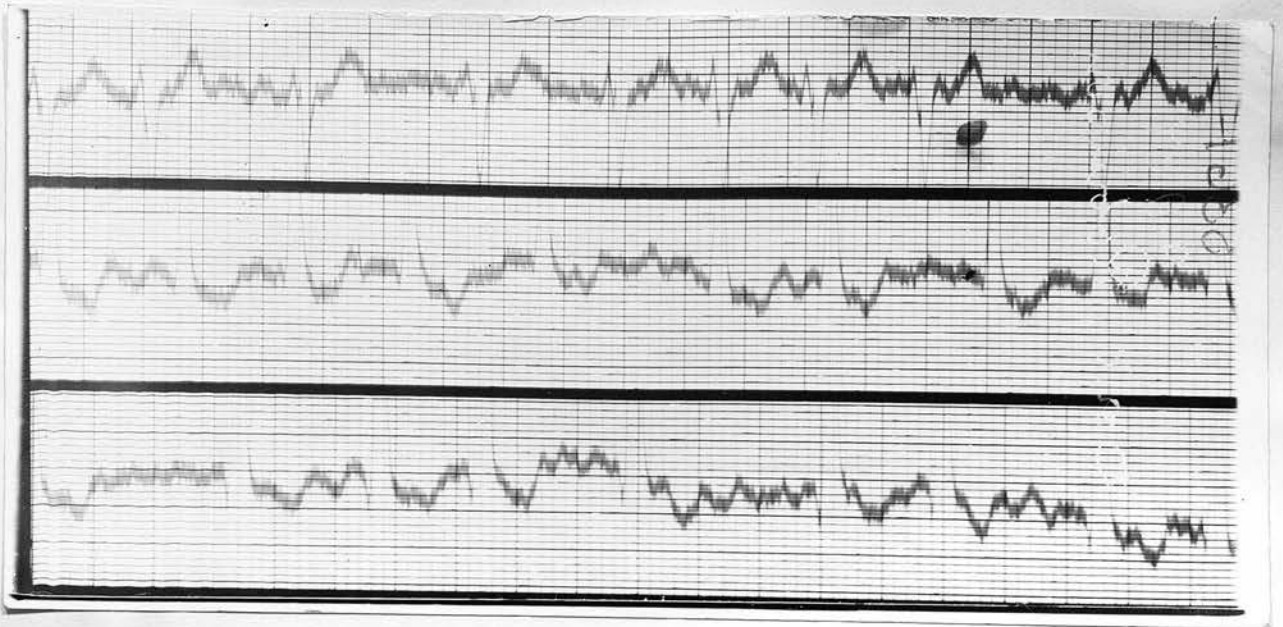
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent.
Right ventricular preponderance.
Auricular fibrillation.

Diagnosis: Congestive failure.
Auricular fibrillation.
Mitral stenosis.
Rheumatic.

Circulatory Rate: $21\frac{3}{5}$ secs.



CASE 103.

Sex: Female. Married.

Age: 49 years.

Occupation: Housewife.

History: 7 years ago first experienced dyspnoea - gradually getting worse.

2 years ago had 'dropsy' and was confined to bed for several months suffering from 'cardiac failure'. Kept well until 10 months ago, when she caught a bad cold. This was followed by increasing dyspnoea and swelling of the feet. Dyspnoea has more recently occurred during the night, and is accompanied by a sensation of suffocation. In addition has suffered from palpitation, exhaustion, and cough with frothy sputum.

Unable to do any house-work for past 10 months.

In bed for past month.

Previous illnesses: Growing pains, childhood.
Tonsillitis.
Scarlet fever.

Family History: 3 children. 2 miscarriages.

Examination: Orthopnoeic. Pale. Weight 8 st. 12 lbs.

Circulatory System: Heart Rate, 100; irregular.
Blood-pressure, systolic 150.
Apex beat: 6th interspace outside
mid-clacivular line.
Heaving in character.
Systolic thrill, base of heart.

Case 103 (continued)Circulatory System:

(Contd)

Heart sounds: auricular fibrillation.

Gallop rhythm.

Mitral area: systolic and
dyastolic bruits.Aortic area: rough systolic
bruit and
diastolic bruit.Lungs: Percussion note impaired at bases.

Basal crepitations.

Abdomen: Liver enlarged 3 finger breadths below costal margin.

Tender. Ascites nil.

Oedema: Ankles, legs and sacrum.Urine: Albumin.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: P absent. Irregular.

Ventricular extra-systole in lead iii.

Left ventricular preponderance.

T₁ T₂ inverted.Diagnosis: Congestive failure.

Auricular fibrillation.

Aortic stenosis and incompetence.

Mitral stenosis.

Rheumatic.

Circulatory Rate: $39\frac{3}{5}$ secs.

Case 103 (continued)

4 weeks later.

Examination: Weight 8 st. 6 lbs. (loss of 6 lbs).

Circulatory System: Heart rate, 58.

Lungs: Crepitations, bases.

Abdomen: Liver edge just palpable.

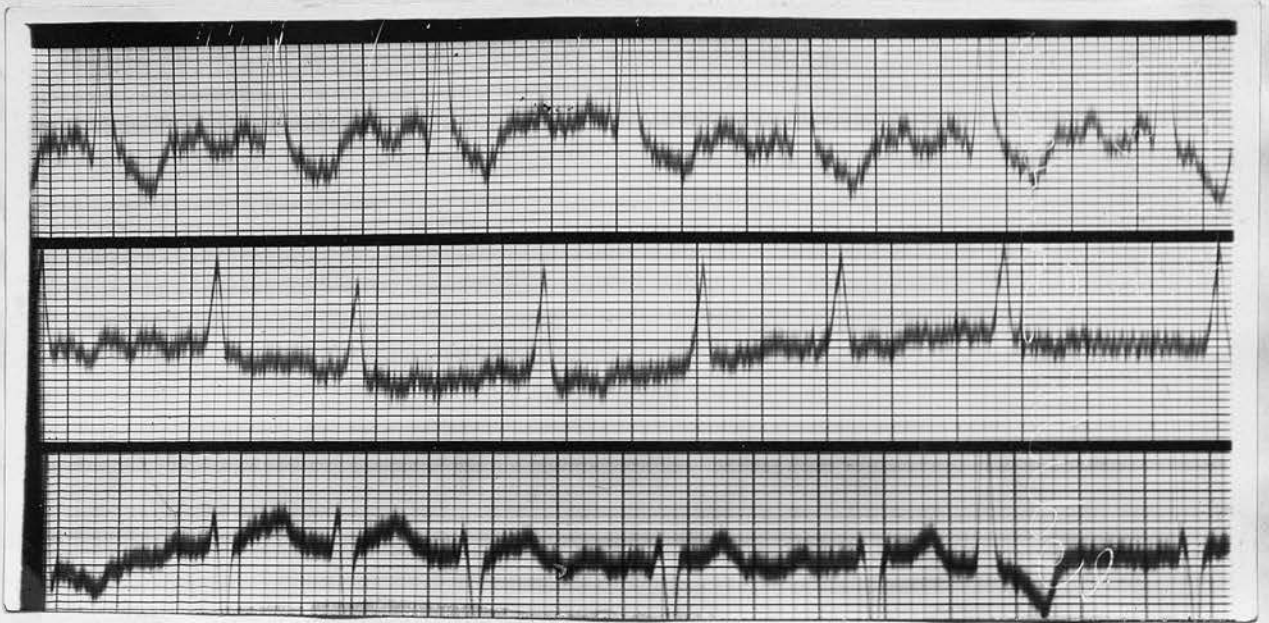
Oedema: Slight over sacrum.

Patient has been treated with salyrgan injections and digitalis (now on Tr. Digitalis η x B.D.).

Circulatory Rate: $24\frac{1}{5}$ secs.

Patient died suddenly 6 weeks after 2nd examination.

Case 103 (continued)



Case 112.

Sex: Female. Married.

Age: 76 years.

Occupation: Housewife.

History: 10 years. Giddiness, dyspnoea on exertion, palpitation and exhaustion.

6 weeks. 'Bad breathing'. Insomnia due to dyspnoea. Cough with copious sputum.

Previous illnesses: Nil.

Family History: 15 children. Menopause, aet. 50 years.

Examination: Weight 12 st. Cyanotic and dyspnoeic.

Circulatory System: Heart rate, 87; irregular.

Jugular veins congested.

Blood-pressure, 272/120.

Apex beat: 7th interspace at anterior axillary line.

Heart sounds: Auricular fibrillation.

Mitral area; systolic bruit.

Base; systolic bruit.

Lungs: Basal crepitations.

Abdomen: Liver enlarged 1 hand's breadth below costal margin.
Tender.

Oedema: Slight, ankles.

Urine: Albumin trace.

Nervous System: N.A.D.

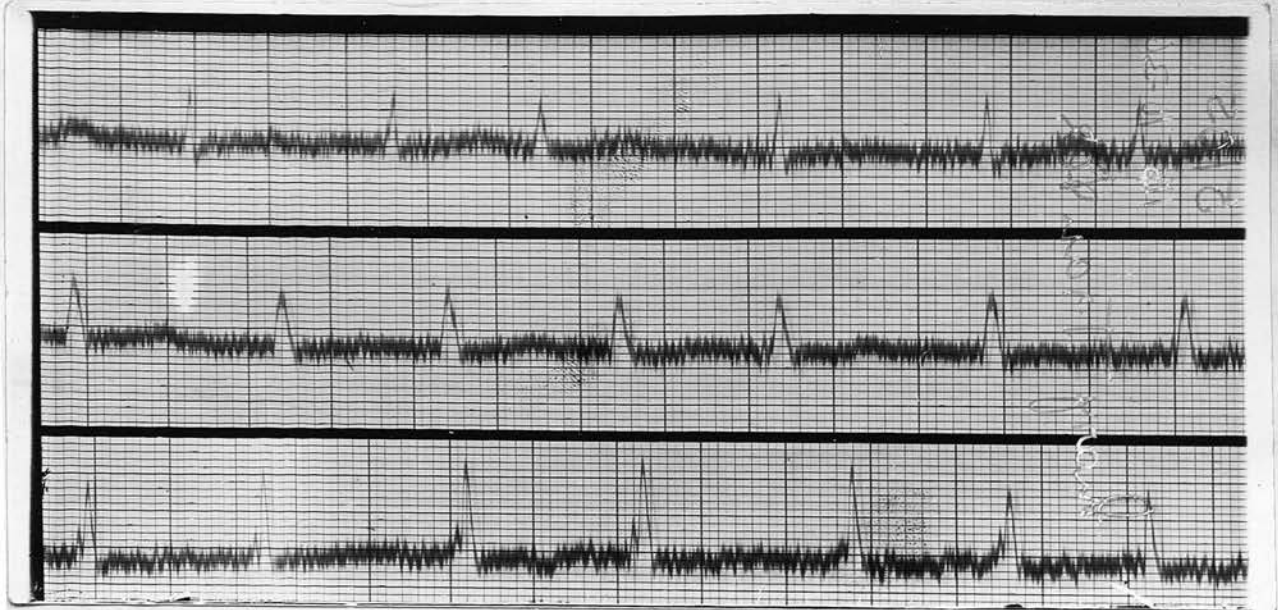
W.R: Negative.

Case 112 (continued)

Electrocardiogram: P absent.
T flat all leads.
R₃ split.
R₂ slurred.
Auricular fibrillation.

Diagnosis: Congestive failure.
Auricular fibrillation.
Hyperpiesia.
Myocardial degeneration.

Circulatory Rate: $21\frac{4}{5}$ secs.



Case 115.

Sex: Male. Married.

Age: 42 years.

Occupation: Riveter.

History: 4 years. Dyspnoea and palpitation on exertion.
Cough with small amount of sputum.
Above symptoms followed Pneumonia 4 years ago.
Off work 4 years.

Previous illnesses: Chorea and scarlet fever, childhood.

Habits: Alcohol and Tobacco moderate.

Examination: Weight 9 st.

Circulatory System: Heart rate, 92; irregular.

Blood-pressure, 130/82.

Apex beat: 6th interspace outside
mid-clavicular line.

Diffuse in character.

Heart sounds: auricular fibrillation.

Mitral area: systolic and
diastolic bruits.

Lungs: Basal crepitations.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

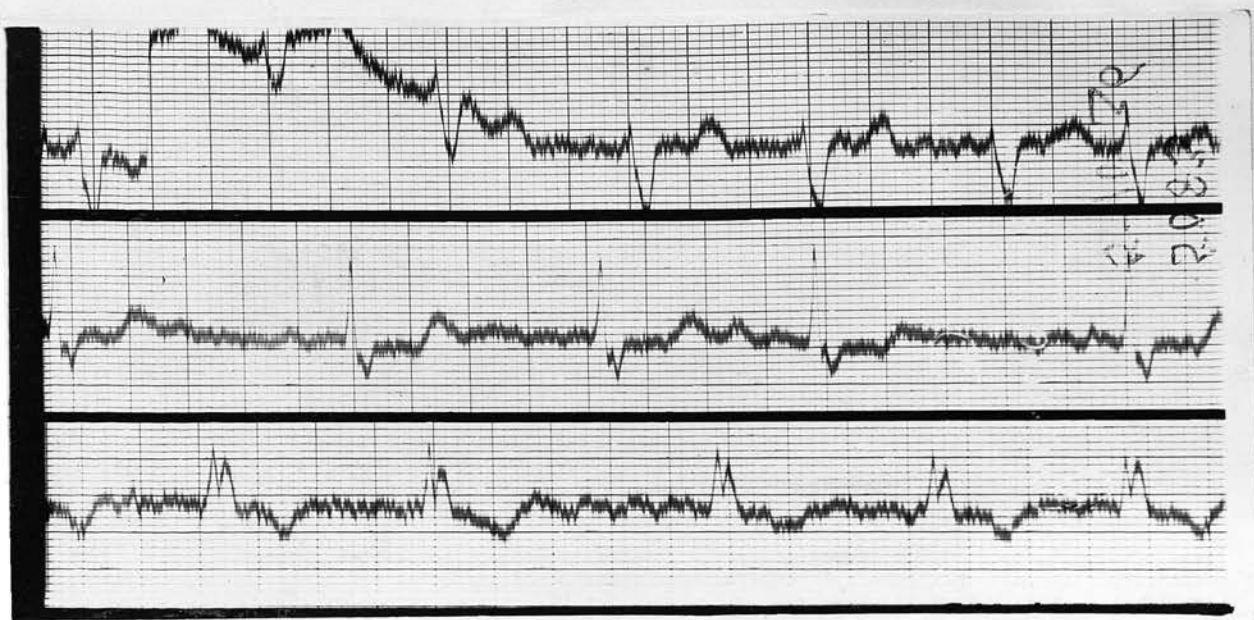
W.R: Negative.

Case 115 (continued)

Electrocardiogram: P absent. Irregular.
 T₃ inverted.
 Right ventricular preponderance.
 QRS complex. Notched.
 Auricular fibrillation.
 Right bundle branch block.

Diagnosis: Auricular fibrillation.
 Mitral stenosis.
 Bundle branch block.
 Rheumatic.

Circulatory Rate: $23\frac{4}{5}$ secs.



Case 122

Sex: Female. Married.

Age: 49 years.

Occupation: Housewife.

History: Well until 3 months ago, when she developed a cough. This was followed by dyspnoea, swelling of feet. Treated in bed and given digitalis. She has been up now for 14 days, and complains dyspnoea and palpitation on exertion.

Previous Illnesses: Growing pains in childhood.
Influenza.

Family History: 9 children.
No history miscarriages.

Examination: Slightly cyanosed.
Weight 7 st. 6 lbs.

Circulatory System: Heart rate, 100; irregular.
Blood pressure 136/?70.
Apex beat, 6th interspace outside
mid-clavicular line.
Heart sounds: auricular fibrillation.
Mitral area, systolic and diastolic bruits.

Lungs: Crepitation bases.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 122 (continued)

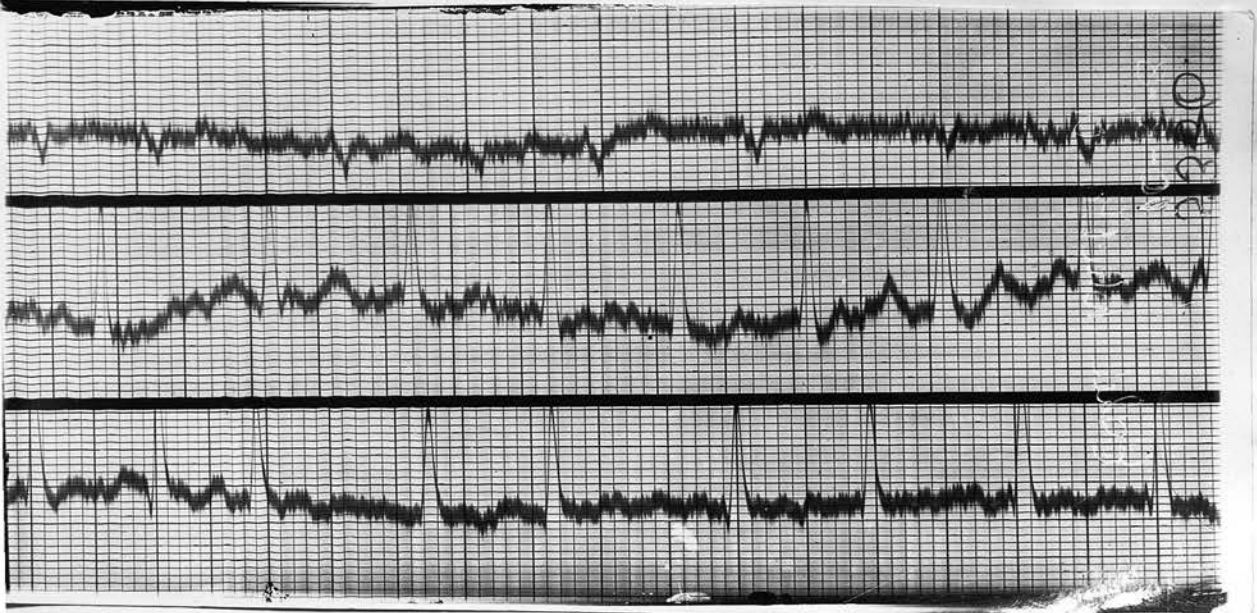
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent.
S₃ slurred.
Auricular fibrillation.

Diagnosis: Congestive failure.
Auricular fibrillation.
Mitral stenosis.
Rheumatic.

Circulatory Rate: 20 secs.



Case 3.

Sex: Female. Single.

Age: 38 years.

Occupation: Factory worker.

History: Always been liable to 'fainting attacks'.

2 years. Symptoms, palpitation, and vague paroxysmal pain on exertion. Exhaustion and occasional vertigo, insomnia, and an increasing degree of perspiration.

GROUP 4

18 cases

Previous illnesses: Rheumatism. Influenza.

Thyrotoxicosis.

Average circulatory rate 9 secs.

(Range 4.4 - 14.6 secs.)

Examination: Pale skin. Heart rate 100. Blood pressure 120/70. Regular.

Circulatory System: ----- Very regular.

Blood pressure, 120/70.

Max beats per interspace mitral aortic valve line.

Tricuspid in character.

Heart sounds: Murmur area, systolic murmur.

Aortic 2nd sound accentuated.

Temp: N.I.D.

Pulse: N.I.D.

Weight: 111.

Case: N.I.D.

Examination: 21.4 sec. patient in hands.

Very regular rhythm.

Tricuspid sound palpable.

2nd, 3rd sound palpable.

Case 3.

Sex: Female. Single.

Age: 38 years.

Occupation: Factory worker.

History: Always been liable to 'Fainting Attacks'.

2 years. Dyspnoea, palpitation, and vague praecordial pain on exertion. Exhaustion and occasional giddiness, insomnia, and an increasing degree of nervousness are also complained of. Feet have recently been noticed to swell at night time.

Previous illnesses: Tonsillitis. Influenza.

Family History: Father died aet.65. "Blood Pressure"
Mother died aet.43. "Heart Failure"

Examination: Pale woman. Weight 6 st. 10 lbs. Catamenia regular.

Circulatory System: Heart Rate, 84; regular.
Blood-pressure, 165/70.
Apex beat: 5th interspace outside mid-axillary line.
Forcible in character.
Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Fine tremor present in hands.
Deep reflexes brisk.
Thyroid gland palpable.
Eyes, palpebral fissures widened.

Case 3 (continued)

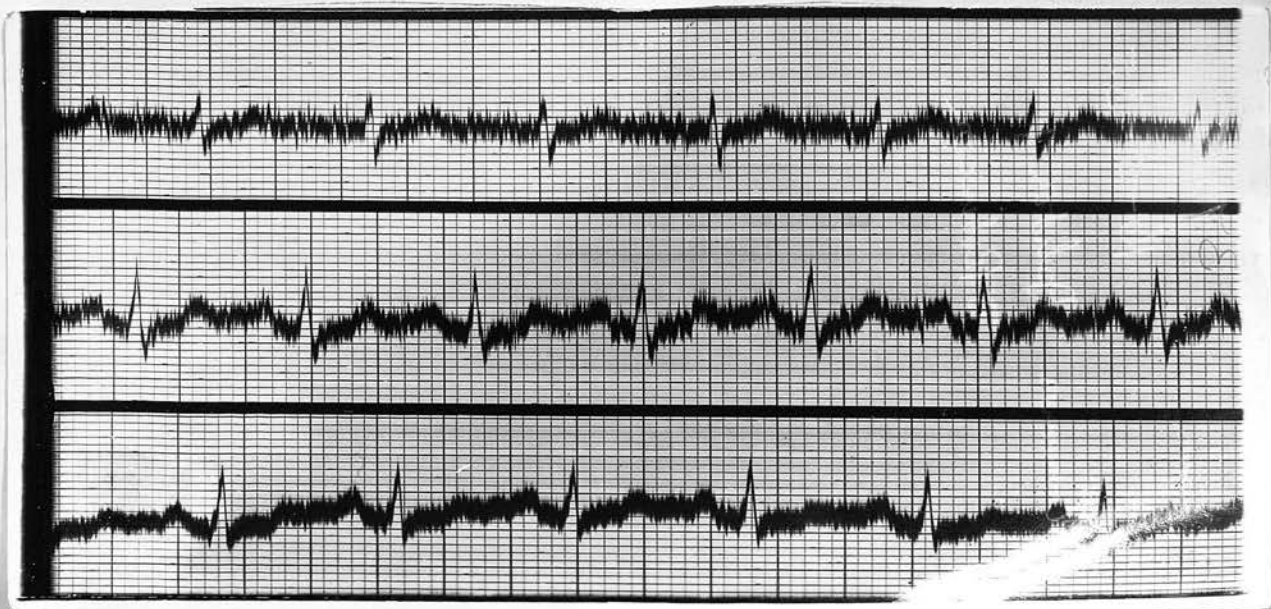
W.R.: Negative.

Blood count: Red blood cells 4,500,000
Haemoglobin 84%.

Electrocardiogram: R₁ and R₃ slurred.

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $9\frac{3}{5}$ secs.



Case 22.

Sex: Female. Married.

Age: 42 years.

Occupation: Housewife.

History: 4 years. On and off dyspnoea on exertion. Attacks of palpitation and giddiness. Exhaustion and 'aching over the heart'.

2 years. Nervousness and 'flushings'. Loss of weight. Recently attacks of diarrhoea.

Previous illnesses: Operation on uterus aet.35. ? Myomectomy.

Family History: Nil relevant.
3 children. 1 miscarriage.

Examination: Flushed nervous woman. Weight 8 st.

Circulatory System: Heart rate, 90; regular.
Blood-pressure, 120/80.
Apex beat: 5th interspace at mid-clavicular line.
Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound slightly accentuated.

Lungs; N.A.D.

Abdomen: N.A.D.

Oedema: Albumin Nil. Sugar trace.

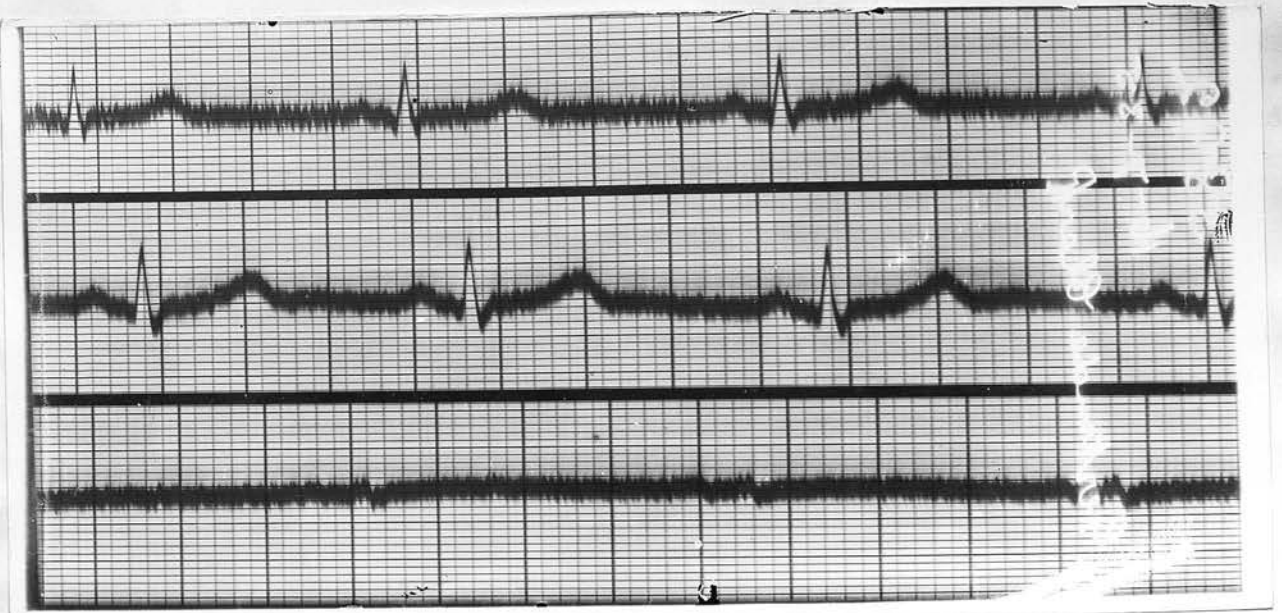
Nervous System: Tremor in hands. Palms moist.
Deep reflexes brisk.
Thyroid gland palpable - no irregularity noted.
Eyes, palpebral fissure widened.

Case 22 (continued)

Electrocardiogram: P₃ absent, S₁ S₂ slurred.
QRS₃ low voltage. T₃ flat.

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $4\frac{2}{5}$ secs.



Case 25.

Sex: Female. Married.

Age: 52 years.

Occupation: Housewife.

History: 5 years. Palpitation, giddiness and exhaustion.
Nervousness. Occasional left sub-mammary pain,
not related to exertion.

3 years. Abdominal discomfort after meals.

Previous illnesses: Influenza.
Diphtheria in childhood.

Family History: Nil relevant.

Examination: Weight 8 st. 4 lbs.
Nervous woman.

Circulatory System: Heart rate, 90; regular.
Blood-pressure, 180/90.
Apex beat: 5th interspace just outside
mid-clavicular line.
Forcible in character.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Tremor hands. Deep reflexes brisk.
Eyes prominent.
Thyroid gland not visibly enlarged.
Palpable - hard.

Case 25 (continued)

W.R: Negative.

Electrocardiogram: Left ventricular preponderance.
R₂ notched.

X-ray Examination: "General cardiac enlargement".

Diagnosis: Hyperpiesia.
Thyrotoxicosis.
Adenoma of thyroid.

Circulatory Rate: $7\frac{3}{5}$ secs.



Case 27.Sex: Female. Married.Age: 54 years.Occupation: Housewife.History: 3 years. Dyspnoea on exertion. Palpitation and nervousness.

2 years. Swelling in neck. Giddiness and Faintness.

Family History: 2 children. No miscarriages.
Menopause aet. 48 years.Previous Illnesses: Nil.Examination: Weight 9 st.Circulatory System: Heart rate, 120; irregular.
Blood-pressure, systolic 134.
Apex beat: 5th interspace just outside mid-clavicular line.
Diffuse in character.
Heart sounds: Systolic bruit over praecordium.Lungs: Crepitation both bases.Abdomen: Nil.Oedema: Nil.Urine: Albumin trace.Nervous System: Thyroid gland enlarged and irregular.
Eyes prominent.
Slight tremor hands.W.R.: Negative.

Case 27 (continued)Electrocardiogram: Not available.

Diagnosis: Congestive failure.
 Auricular fibrillation.
 Thyrotoxicosis - thyroid adenoma.
 (Patient awaiting thyroidectomy)

Circulatory Rate: $11\frac{3}{5}$ secs.

Case 30.

Sex: Female. Married.

Age: 42 years.

Occupation: Housewife.

History: 2 years. Dyspnoea on exertion, and more recently at rest. Palpitation and occasional cough. Symptoms getting worse.

Previous Illnesses: Chorea, aet. 12 years.

Family History: 4 children. No history of miscarriages.

Examination: Nervous woman. Weight 10 st.

Circulatory System: Heart rate, 120; irregular.
Blood-pressure, systolic 140.
Apex beat: 6th interspace outside mid-clavicular line.
Diffuse in character.
Heart sounds: Auricular fibrillation.
Mitral area, systolic and diastolic bruits.

Lungs: Crepitations both bases.

Abdomen: Liver edge palpable. Ascites nil.

Oedema: Ankles slight.

Urine: Albumin trace.

Nervous System: Thyroid gland diffusely enlarged.
Tremor hands.

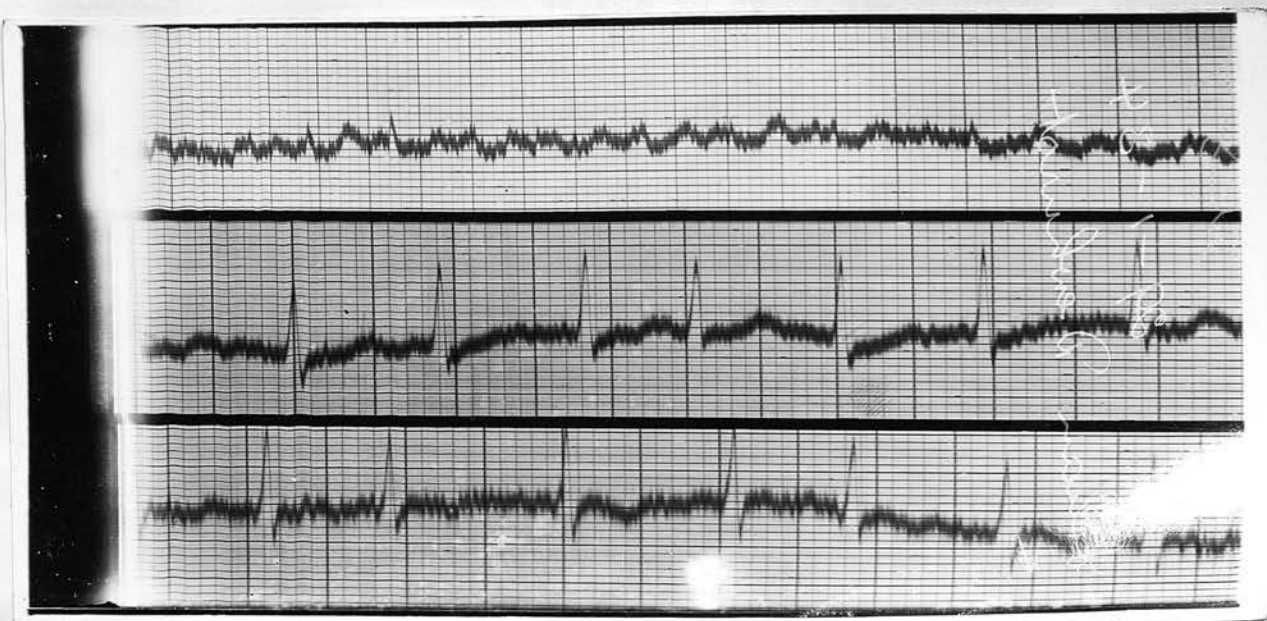
W.R: Negative.

Case 30 (continued).

Electrocardiogram: P absent.
T₃ inverted.
Auricular fibrillation.

Diagnosis: Congestive failure.
Auricular fibrillation.
Mitral stenosis - rheumatic.
Thyrotoxicosis.

Circulatory Rate: $14\frac{3}{5}$ secs.



Case 32.Sex: Female. Single.Age: 35 years.Occupation: Nil.

History: Perfectly well until 1 year ago, when she had pneumonia, for which she was in hospital for 3 weeks. While convalescent she had a 'relapse', and since then has complained of tightness and a crushing feeling in the chest, and inability to take deep breaths. Complains also of palpitation while lying on the left side. This causes insomnia. Dyspnoea, and a pricking pain under left breast on exertion. Sweats easily especially in hands, and is very nervous. Appetite good, but loss of weight (? quantity).

Previous illnesses: Pneumonia as above.
Scarlet fever. Childhood.

Family history: Nil relevant.Examination: Flushed. Weight 10 st. 2 lbs.

Circulatory System: Heart rate, 110; regular.
Blood-pressure, 154/80.
Apex beat: 5th interspace inside mid-clavicular line.
Heart sounds: Soft systolic bruit, mitral area.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 32 (continued)

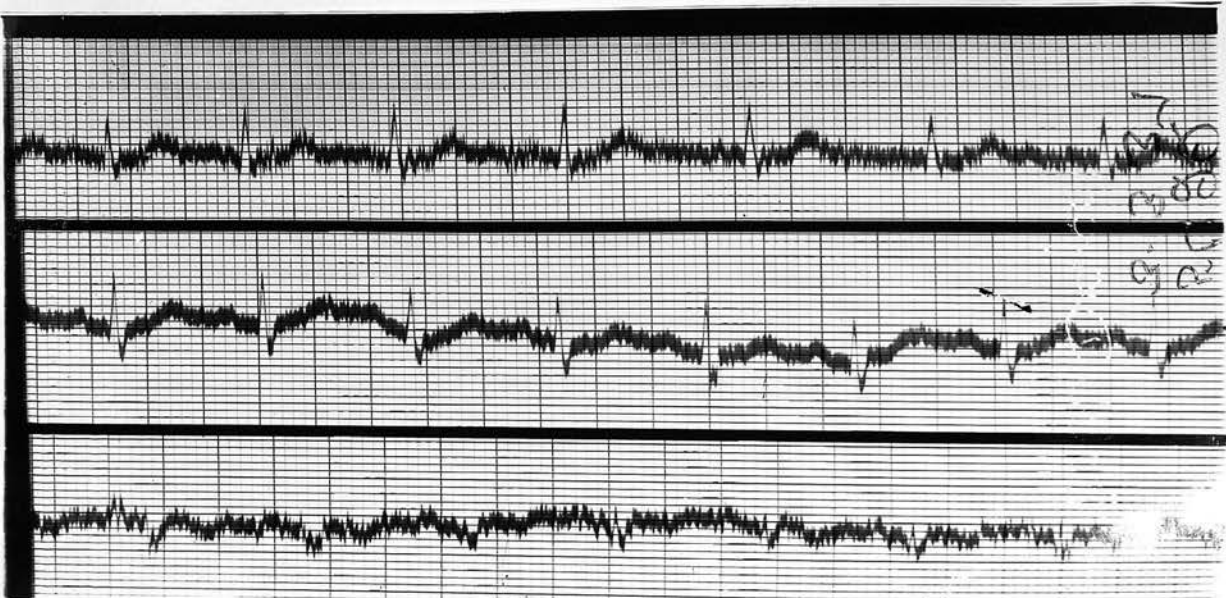
Nervous System: Palms dripping with sweat.
Fine tremor hands.
Deep reflexes brisk.
Thyroid gland palpable.

W.R: Negative.

Electrocardiogram:

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $7\frac{3}{5}$ secs.



Case 34.

Sex: Female. Single.

Age: 35 years.

Occupation: Telephone operator.

History: 3 years dyspnoea on exertion. Attacks of palpitation, exhaustion, syncope, and irritability. Sweats easily and flushes frequently. Slight loss of weight and 'beating in the neck'.

6 months. Praecordial ache on exertion.
Ankles swollen at night time.

Previous illnesses: Tonsillitis - 3 attacks.
Scarlet fever. Childhood.

Family History: Nil relevant.

Examination: Anxious-looking woman. Weight 8 st.

Circulatory System: Heart rate, 104; regular.
Blood-pressure, 142/76.
Apex beat: 5th interspace outside mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area, Systolic bruit.
Aortic area, Diastolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

W.R: Negative.

Case 34 (continued)

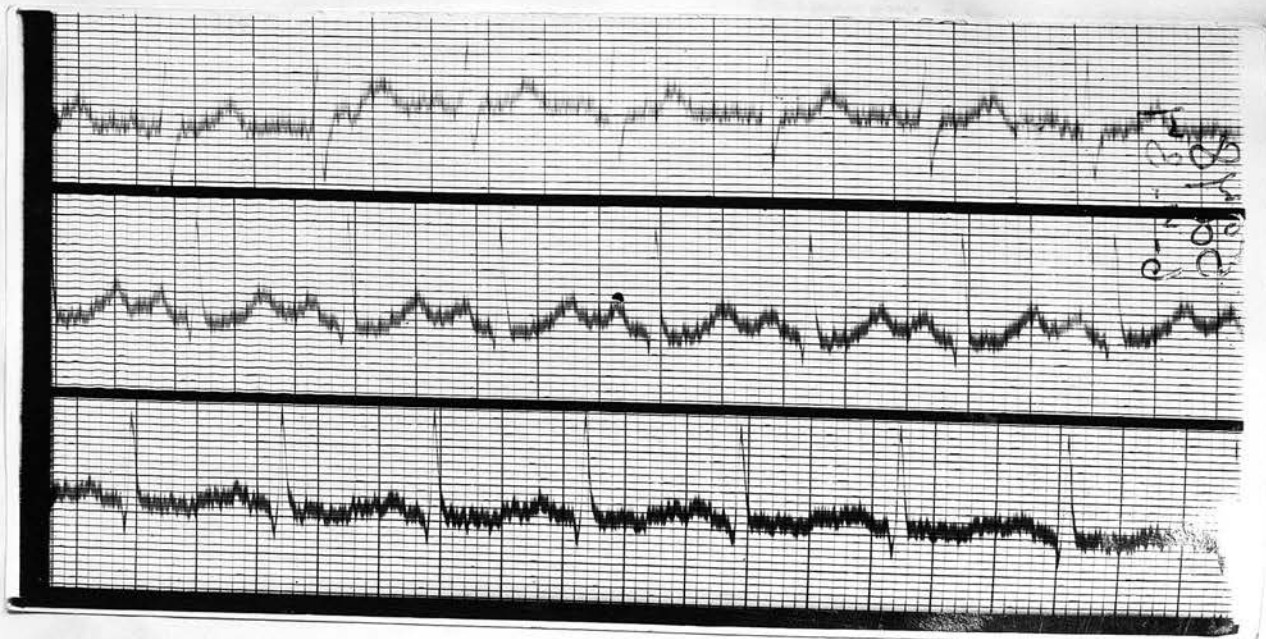
Nervous System: Slight proptosis. Palpebral fissures widened, lid lag present. Tremor hands. Palms moist. Deep reflexes brisk.

Thyroid gland, palpable - diffusely enlarged.

Electrocardiogram: Q₂ and Q₃ present.
S slurred. T₃ inverted.

Diagnosis: Aortic incompetence. Rheumatic. Thyrotoxicosis.

Circulatory Rate: $12\frac{2}{5}$ secs.



Case 39.

Sex: Female. Married.

Age: 59 years.

Occupation: Housewife.

History: 6 years. Dyspnoea and palpitation on exertion. Slight precordial pain on exertion, radiating in right shoulder. Insomnia. Frontal headaches, and giddiness. Epigastric discomfort after meals.

Previous illnesses: Scarlet fever, childhood.

Family History: 3 children. No history of miscarriages.

Examination: Nervous woman. Heart.

Circulatory System: Heart Rate, 86; regular.

Blood-pressure, 130/82.

Apex beat: 6th interspace outside mid-clavicular line.

Heart sounds: Mitral area, systolic bruit.
Pulmonary area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Thyroid gland, palpable. Nodular.

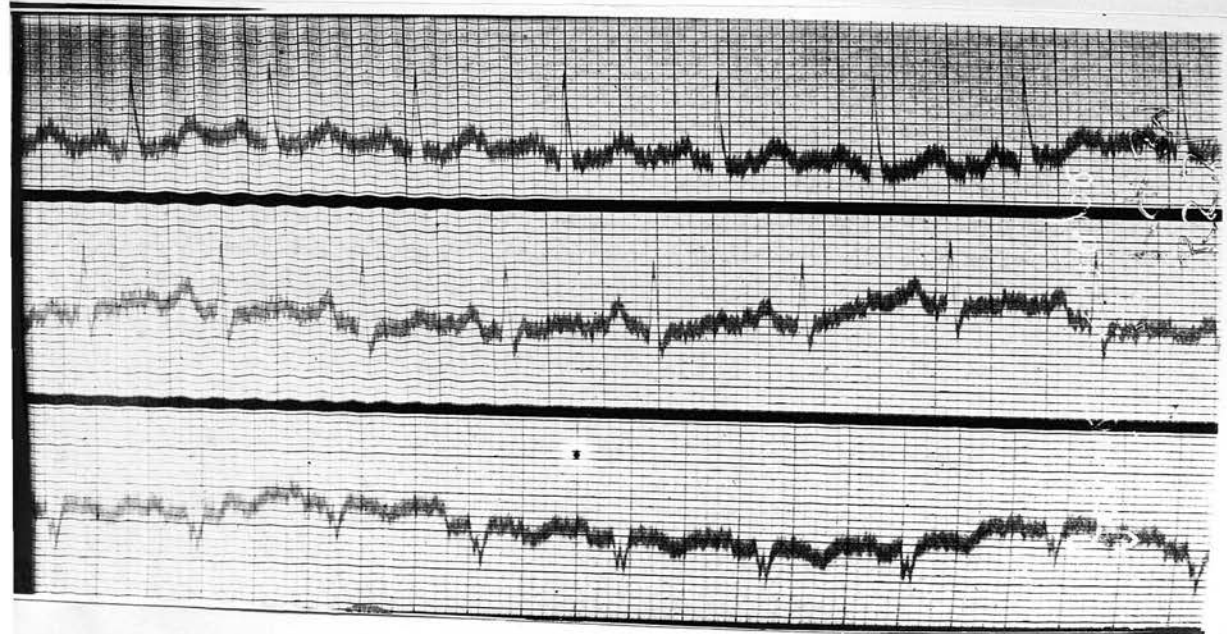
W.R: Negative.

Case 39 (continued)

Electrocardiogram: Q.R.S. SLURRED.
T₃ INVERTED
LEFT VENTRICULAR PREPONDERANCE

Diagnosis: Myocardial degeneration.
Thyroid adenoma.

Circulatory Rate: $9\frac{4}{5}$ secs.



Case 40.Sex: Female. Married.Age: 25 years.Occupation: Housewife.History: 3 years. Attacks of palpitation, worse at night, and accompanied by praecordial pain. Dyspnoea on exertion. Exhaustion and faintness, insomnia.

1 year ago was a hospital in-patient when she was told she was suffering from 'Graves' disease' and treated with rest in bed and medicine.

6 months. All symptoms have got worse.

Previous illnesses: Scarlet fever, aet.19 years, followed by 'Neurasthenia'.

Pneumonia aet.22 years.

Family History: 1 child. 2 miscarriages before birth of child. Is now 5 months pregnant.Examination: Flushed nervous patient.Circulatory System: Heart rate, 92; regular.

Blood-pressure, 136/72.

Apex beat: 6th interspace at anterior axillary line.

Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 40 (continued)

Nervous System: Tremor hands. Palms moist.
Pupils dilated. React to light and accommodation.
Deep reflexes brisk.
Thyroid gland diffusely enlarged.

W.R: Negative.

Electrocardiogram:

NOT AVAILABLE

Diagnosis: Thyrotoxicosis. Myocardial degeneration.

Circulatory Rate: $9\frac{2}{5}$ secs.

Case 45.Sex: Female. Married.Age: 27 years.Occupation: Housewife.History: 2 years. Frontal headaches, palpitation, and vomiting. 1 or 2 such attacks per month.

Nervousness, depression, irritability, flushings, and loss of weight. Giddiness, lack of energy, insomnia and nightmares.

Previous illnesses: Tonsillitis, many attacks.
Tonsillectomy aet. 25 years.
Measles in childhood.Family History: 1 child. Catamenia regular.Examination: Weight 7 st. 2 lbs.Circulatory System: Heart rate, 90; regular.
Blood-pressure 124/60.
Apex beat: 5th interspace just outside mid-clavicular line.
Forcible in character.
Heart sounds: mitral area, systolic bruit.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: Albumin trace.

Case 45 (continued)

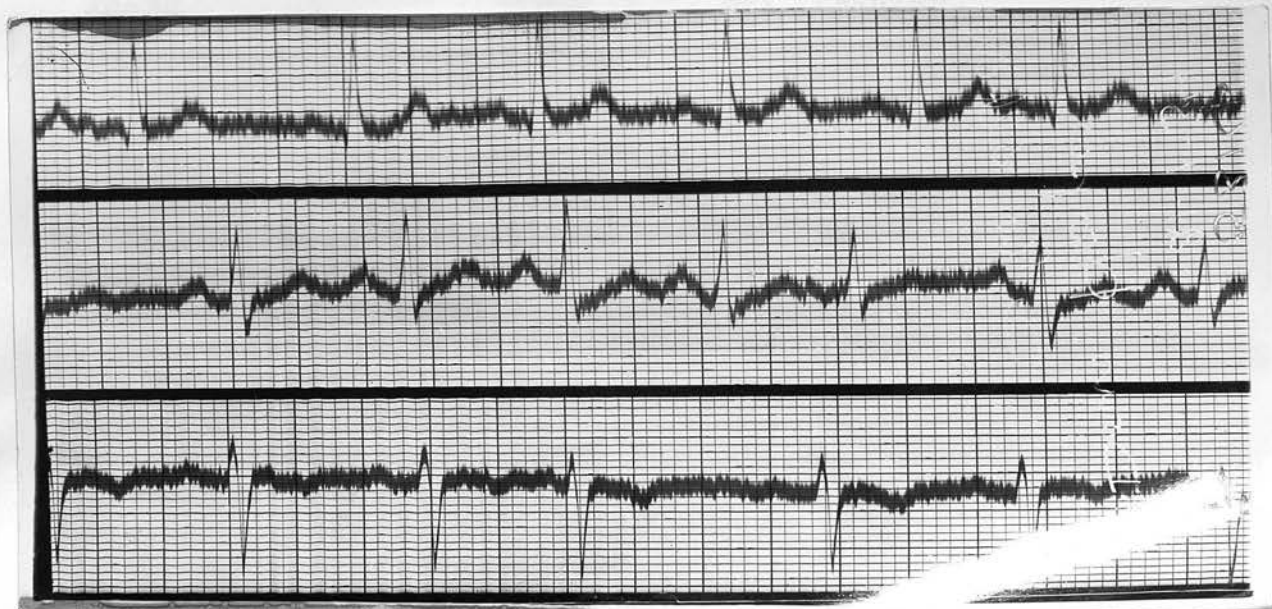
Nervous System: Fine tremor hands. Palms moist.
Deep reflexes brisk.
Thyroid gland palpable.
Pupils dilated. React to light and accommodation

W.R: Negative.

Electrocardiogram: Sinus irregularity.
T₃ inverted.
Left ventricular preponderance.

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $8\frac{1}{5}$ secs.



Case 53.Sex: Female. Married.Age: 36 years.Occupation: Housewife.History: 12 months. Frontal headaches and nausea.
3 months. Attacks of giddiness, and on one occasion she fainted in the street.

6 months. Loss of weight - 1 stone.

For the same period, nervousness, insomnia, and sweats easily. Palpitation and slight dyspnoea on exertion.

All above symptoms followed a shock - motor-car accident 12 months ago.

Previous illnesses: Nil.Family History: 2 children. 1 miscarriage 12 months ago.Examination: Flushed appearance. Weight 9 st. 3 lbs.Circulatory System: Heart rate, 84; regular.

Blood-pressure 170/90.

Apex beat: 5th interspace inside
mid-clavicular line.

Heart sounds: closed in all areas.

Aortic 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 53 (continued)

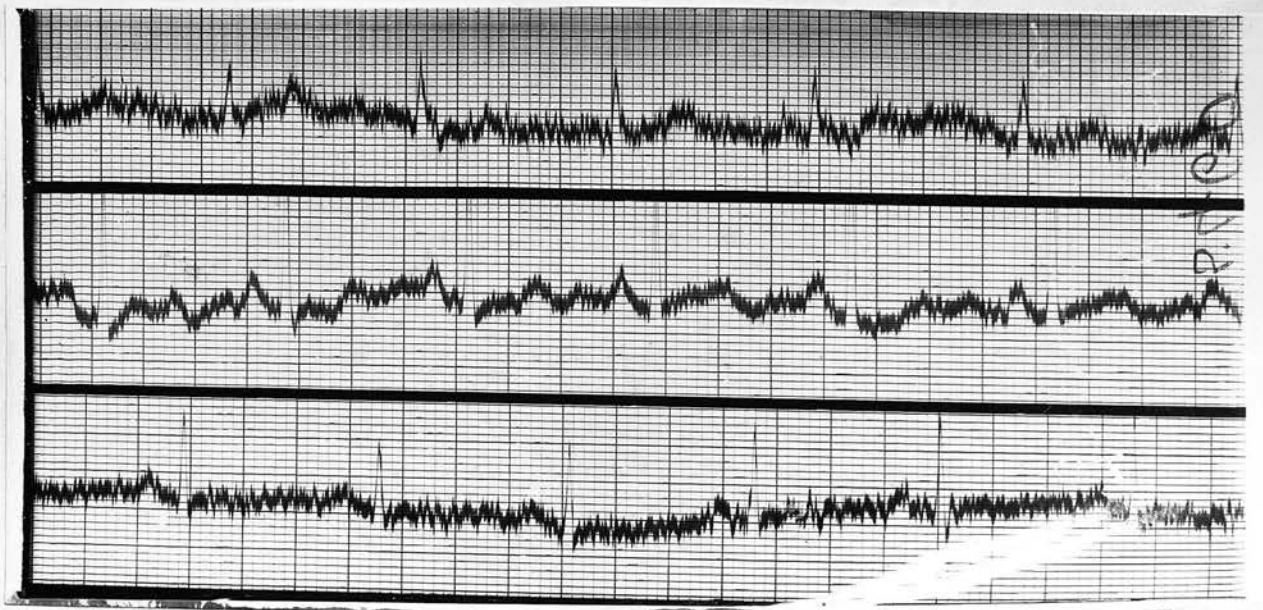
Nervous System: Slight fine tremor, hands. Palms moist.
Deep reflexes brisk.
Thyroid gland not visibly or palpably enlarged.

W.R:

Electrocardiogram: R₁ NOTCHED.

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $8\frac{3}{5}$ secs.



Case 66.Sex: Female. Married.Age: 48 years.Occupation: Housewife.History: 2 years. Frontal headaches - about once a month, lasting a few days.

18 months. Dyspnoea on exertion.

6 months. Left sub-mammary pain, lasting a few seconds, induced by exertion, and eased by rest. Pain radiates to the left shoulder and to the back.

1 month. Palpitation on exertion, giddiness, and insomnia. Recently noticed swelling of feet at night time.

Previous illnesses: Tonsillitis - several times.
Rheumatic pains - knees.Family History: 8 children. No miscarriages.
Mother died 'heart trouble' aet. 50 years.Examination: Weight 9 st. Flushed.Circulatory System: Heart rate, 90; regular.
Blood-pressure 148/92.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: closed.
Aortic 2nd sound accentuated.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.

Case 66 (continued)

Urine: N.A.D.

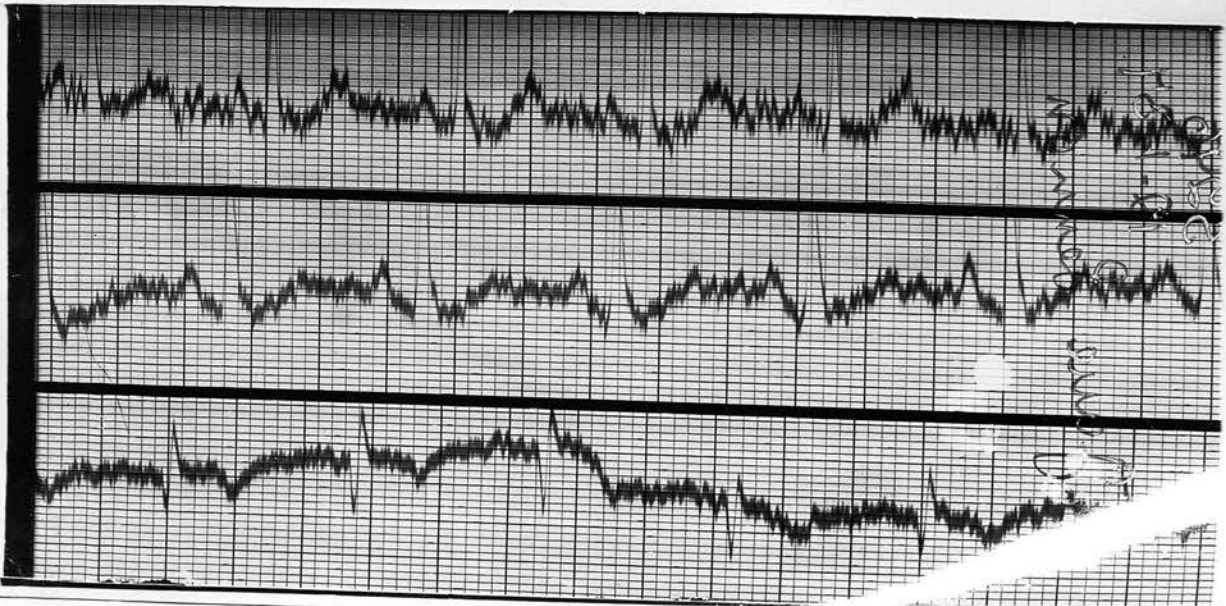
Nervous System: Tremor hands. Slight proptosis.
Thyroid gland palpable.
Deep reflexes brisk.

W.R: Negative.

Electrocardiogram: S slurred. $Q_3 +$
 T_3 inverted.

Diagnosis: Myocardial degeneration.
Thyrototoxicosis.

Circulatory Rate: $11\frac{1}{5}$ secs.



Case 69.Sex: Male. Single.Age: 21 years.Occupation: Nil.History: 18 months. Attacks of vertigo (twice a week).
Commence with frontal headache.

Dyspnoea on slight exertion.

Occasional left sub-mammary pain - no relation to
exertion.

Exhaustion. Insomnia. Slight loss of weight.

Symptoms followed shock - caused through falling
through a grid.Previous illnesses: Chorea. Aet. 14 years.
Tonsillitis. Tonsillectomy aet. 13 years.
Diphtheria, aet. 7 years.Examination: Nervous. Weight 8 st.Circulatory System: Heart rate, 94; regular.
Temperature sub-normal.
Blood-pressure 140/80.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: Mitral area, harsh systolic
bruit.
Pulmonary 2nd sound accentuated.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.

Case 69 (continued)

Urine: N.A.D.

Nervous System: Tremor hands.
Deep reflexes brisk.
Thyroid gland palpable.

W.R: Negative.

Electrocardiogram: Left ventricular preponderance.
R₃ notched.

Diagnosis: Mitral incompetence.
Rheumatic.
Thyrotoxicosis.

Circulatory Rate: $6\frac{4}{5}$ secs.



CASE 79.

Sex: Female. Married.

Age: 37 years.

Occupation: Housewife.

History: When aet. 21 years was medically treated for 'exophthalmic goitre'. This was said to have developed after a 'breast abscess'. She was much improved by treatment, and remained well until 3 years ago, when she collapsed - lost use of her legs, suffered from exhaustion and loss of weight. Kept in bed for a few months, and improved. Kept well until 4 months ago when she developed broncho-pneumonia. On recovery noticed dyspnoea, palpitation, exhaustion, and a sense of suffocation, which symptoms are still present. Loss of weight 2 st. during last 4 months.

Previous illnesses: Tonsillitis in childhood and adolescence.
Influenza.

Family History: 2 children. No history of miscarriages.

Examination: Weight 8½ st.

Circulatory System: Heart rate, 90; regular.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: aortic area, systolic bruit.

Blood-pressure 114/74.

Lungs: N.A.D.

Oedema: Nil.

Abdomen: Nil.

Case 79 (continued)Urine: Albumin.Nervous System: Bilateral proptosis. Skin moist.
Fine tremor hands.
Thyroid gland enlarged diffusely.W.R.: Negative.Electrocardiogram: P₂ large and split.
S slurred.
T inverted all leads.
Left ventricular preponderance.Diagnosis: Myocardial degeneration.
Thyrotoxicosis.Circulatory Rate: $7\frac{3}{5}$ secs.

Case 84.Sex: Female. Single.Age: 46 years.Occupation: Glove cutter.History: 4 months, dyspnoea on exertion, attacks of palpitation, nervousness, flushings, tinnitus, and loss of weight (about 6 lbs), exhaustion, insomnia, and sweating of hands.

All the above symptoms commenced suddenly.

Recently noticed swelling of her feet at night time.

Unable to work since commencement of illness.

Previous Illnesses: Growing pains, childhood.
Haematemesis.
Gastric ulcer aet. 23 years.Family History: Nil relevant.Examination: Weight 10 st.Circulatory System: Heart rate, 96; regular.
Cervical veins congested.
Blood pressure 144/80.
Carotid pulsation marked.
Apex beat: 5th interspace outside
mid-clavicular line.
Forcible.
Heart sounds: Mitral area, loud systolic
bruit.
aortic area, systolic bruit.
aortic 2nd sound accentuated.Lungs: N.A.D.

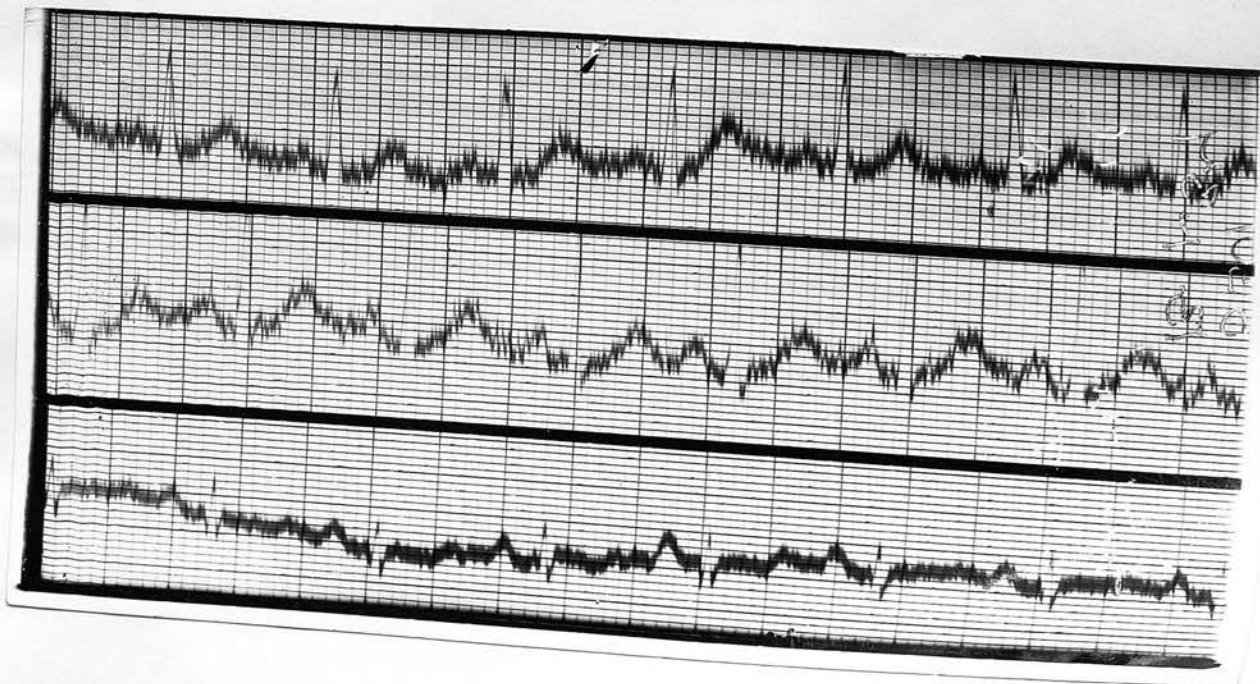
Case 84 (continued)Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Nervous System: Tremor hands. Palms moist.
Deep reflexes brisk.
Bilateral exophthalmos and lid lag.
Thyroid gland, palpable. Small nodule palpable,
left lateral lobe.

W.R: Negative.Electrocardiogram: T₃ inverted.Diagnosis: Thyrotoxicosis.

Adenoma of thyroid.

Old rheumatic endocarditis.

Circulatory Rate: $10\frac{1}{5}$ secs.

Case 96.Sex: Female. Single.Age: 28 years.Occupation: Domestic servant.History: 18 months. Dyspnoea and occasional praecordial pain on exertion. Palpitation worse at night time. Loss of weight, nervousness, and 'flushings'. Feet have been swollen at night recently.Previous illnesses: Tonsillitis.
Influenza.Family History: Nil relevant.Examination: Weight 7 st. 6 lbs.Circulatory System: Heart rate, 86; regular.
Blood-pressure 176/100.
Apex beat: 5th interspace outside
mid-clavicular line.
Forcible.
Heart sounds: Mitral area, presystolic and
systolic bruits.
Aortic area, systolic and
diastolic bruits.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

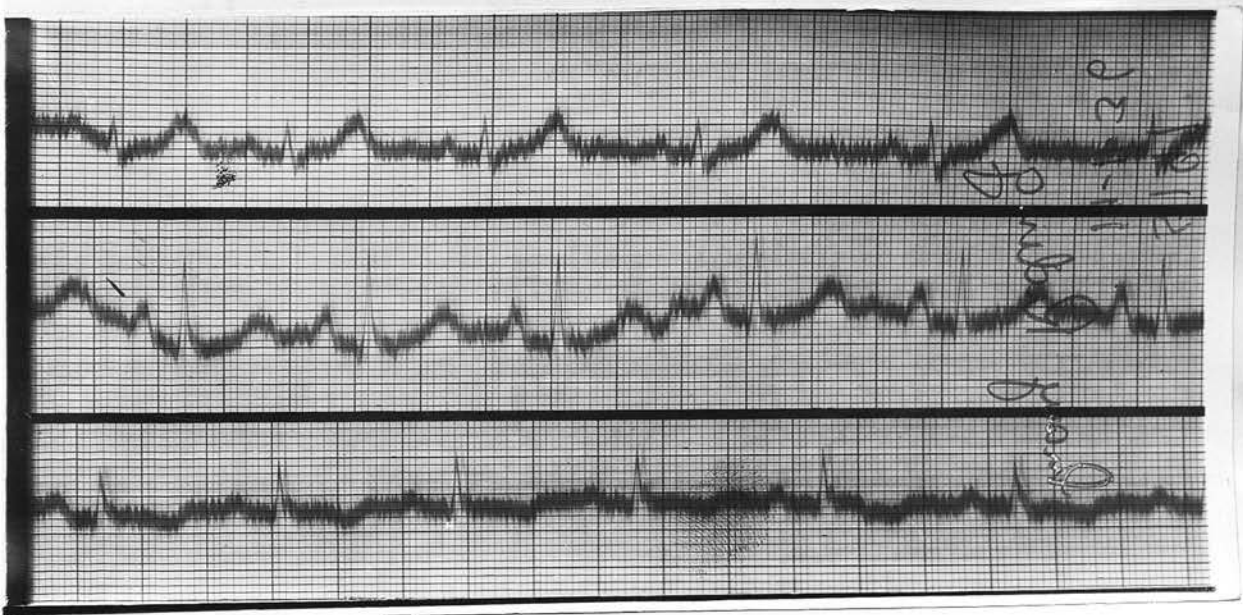
Case 96 (continued)

Nervous System: Tremor hands. Palms moist.
 Deep reflexes brisk.
 Moderate exophthalmos.
 Bilateral lid lag.
 Thyroid gland diffusely enlarged.

Electrocardiogram: R₃ split.
 S₂ S₃ slurred.
 P₂ notched.
 T₃ inverted.

Diagnosis: Mitral stenosis.
 Aortic incompetence.
 Rheumatic.
 Thyrotoxicosis.

Circulatory Rate: 8 secs.



Case 119.Sex: Female. Single.Age: 15 years.Occupation: Machinist.History: 3 Months. Loss of weight.

3 weeks. Palpitation and 'missing heart beats'.

Attacks of faintness. Dry cough.

Catamenia irregular.

Previous illnesses: Tonsillitis. Tonsillectomy aet. 9 years.
Measles, whooping cough - childhood.Examination: Weight 8 st. 5 lbs.Circulatory System: Heart rate, 88; regular.

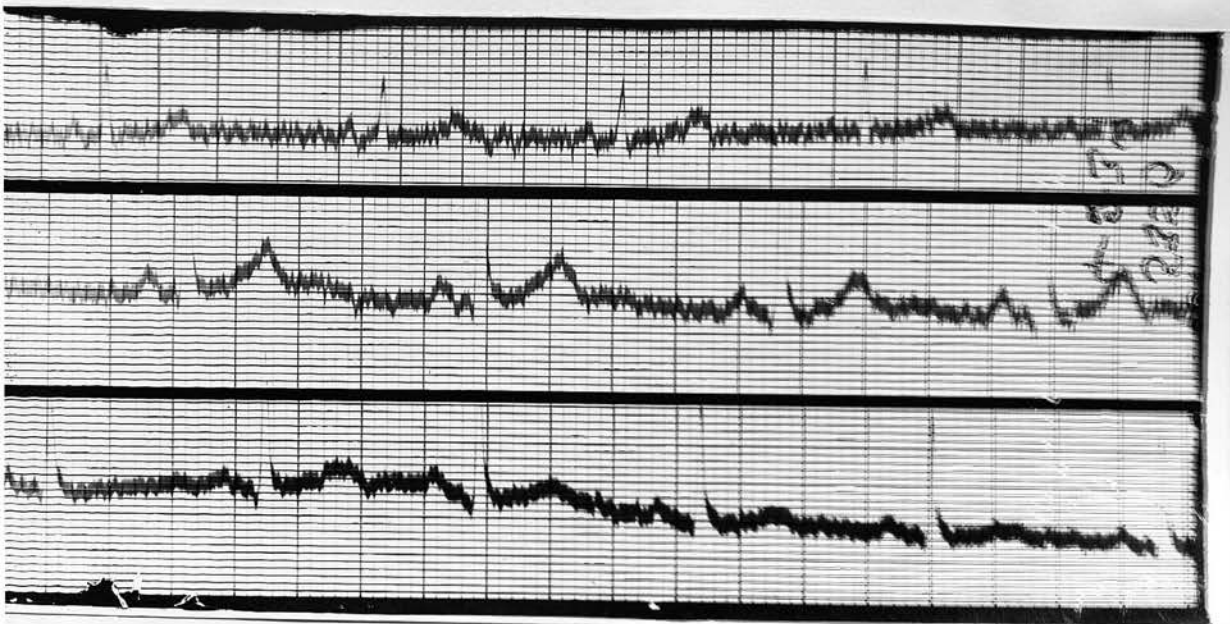
Temperature 97°.

Blood-pressure 118/60.

Apex beat: 5th interspace inside
mid-clavicular line.Heart sounds: mitral area, soft systolic
bruit.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: Albumin trace.Nervous System: Slight tremor hands. Palms moist.

Deep reflexes brisk.

Thyroid gland diffusely enlarged.

Case 119 (continued)W.R.: Negative.Electrocardiogram: Nil abnormal.X-ray examination: "Lungs - no evidence of Pul.Tub."Diagnosis: Thyrotoxicosis.Circulatory Rate: 9 secs.

Case 108.Sex: Male. Single.Age: 24 years.Occupation: Printer.History: 9 weeks. General 'shakiness' which commenced in the legs and spread to the arms. Nervousness, palpitation, and 'sweating in the hands'.
Loss of weight and exhaustion.

Unable to work 7 weeks and confined to bed 3 weeks.

Previous illnesses: 5 years ago 'nervous breakdown'.
In bed 3 weeks.
Tonsillitis.
Influenza.Family History: Father died 'arterio-sclerosis'.
Mother died 'stroke'.Habits: Alcohol and tobacco - nil.Examination: Weight 7 st. 7 lbs.Circulatory System: Heart rate, 100; regular.
Carotid pulsation marked.
Peripheral vessels sclerosed.
Blood-pressure 172/78.
Apex beat: 5th interspace at
mid-clavicular line.
Diffuse.
Epigastric pulsation marked.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic bruit.
Aortic 2nd sound accentuated.Lungs: N.A.D.Abdomen: N.A.D.

Case 108 (continued)

Oedema: Nil.

Urine: Albumin.

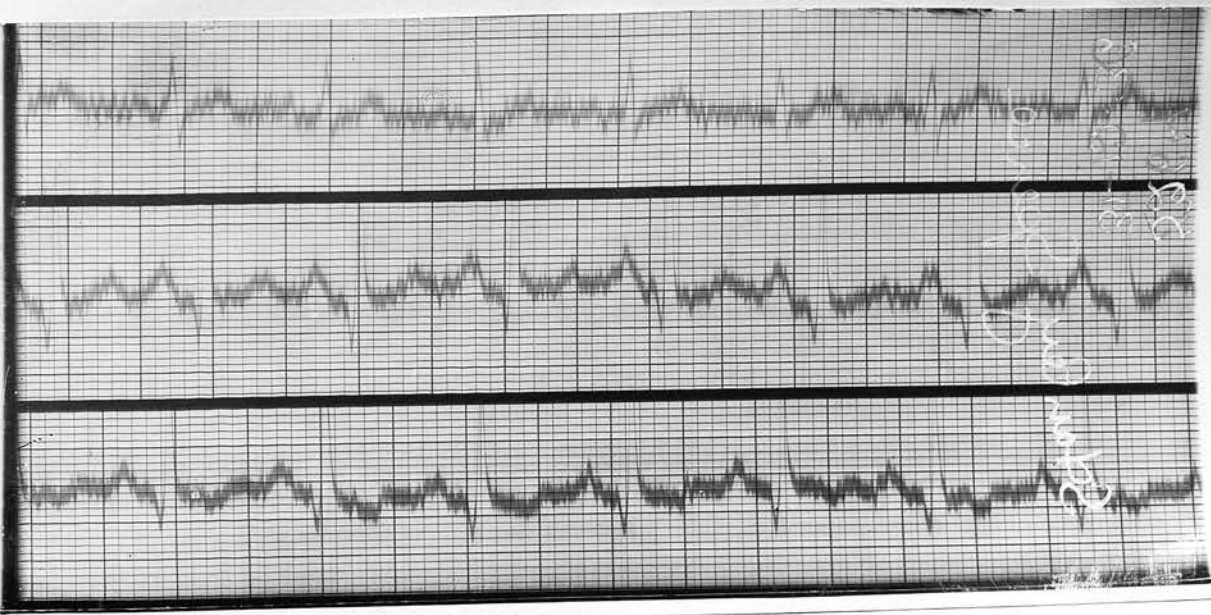
Nervous System: Marked tremor all limbs.
Palms moist.
Slight bilateral exophthalmos.
Palpebral fissures widened.
Bilateral lid lag.
Thyroid gland palpable.

W.R.: Negative.

Electrocardiogram: P₂ split. Q₂, Q₃ +.
S₂ S₃ slurred.
T₃ inverted.

Diagnosis: Thyrotoxicosis.

Circulatory Rate: $5\frac{4}{5}$ secs.



Case 12.

Sex: Male. Married.

Age: 61 years.

Occupation: Marine Engineer.

History: 3 weeks. Periodical pain on exertion, accompanied by a sense of constriction. Pain does not radiate. Episodes not precipitated on exertion. History of GROUP 5 conditions.

Previous Illnesses: 12 cases of Group 5 conditions, 1910-1920 years. Hypertension.

Specific Heart Disease.

Average circulatory rate 14 secs.
(Range 8.6 - 25.6 secs.)

Activity: Alcohol, tobacco

Examination: Weight 160 lbs.

Circulatory System: Heart rate 70-80. Systolic 120. Diastolic 80. Pulse 70-80.

Diagnosis: H.A.D.

Prognosis: H.A.D.

Comments: H.A.D.

Case 20.

Sex: Male. Married.

Age: 61 years.

Occupation: Marine Fireman.

History: 3 months. Praecordial pain on exertion, accompanied by a sense of constriction. Pain does not radiate. Dyspnoea and palpitation on exertion. Giddiness and faint sensations.

Previous Illnesses: Luetic infection, aet. 20 years.
Tonsillitis.

Family History: Nil relevant.

Habits: Alcohol, moderate; tobacco, heavy smoker.

Examination: Weight 8 stones.

Circulatory System: Heart rate, 76; regular.

Blood-pressure, 176/64.

Apex beat: 6th interspace at
mid-clavicular line.

Slightly forcible in character.

Heart sounds: Aortic area, systolic bruit.

Aortic 2nd sound accentuated and
ringing.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Case 20 (continued)Urine: N.A.D.Nervous System: N.A.D.W.R.: Positive.Electrocardiogram: P₂ large.T₃ inverted.

QRS slurred.

X-ray Examination: "Slight cardiac enlargement. Atheroma (calcified plaques) or aortic arch, but no appreciable aortic dilatation."Diagnosis: Angina of effort.
Myocardial degeneration.
Hyperpiesia.
Old specific infection.Circulatory Rate: $16\frac{1}{5}$ secs.

Case 41.

Sex: Male. Married.

Age: 51 years.

Occupation: Traveller.

History: 3 years. Dyspnoea on exertion. Occasional attacks of sub-sternal pain, while walking, and after meals. Pain eased by rest. Sense of constriction in the chest and giddiness.

Previous Illnesses: Luetic infection 20 years ago; treated.

Habits: Alcohol and tobacco nil.

Family History: Nil relevant.

Examination: Weight 9 stones.

Circulatory System: Heart rate, 72; regular.

Blood-pressure, 125/70.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Urine: N.A.D.

Nervous System: N.A.D.

Case 43.

Sex: Male. Widower.

Age: 52 years.

Occupation: Slaughter-house worker.

History: 1 month. Left praecordial pain on exertion eased by rest. Giddiness, faintness, and frontal headaches.

Previous Illnesses: Influenza.
Typhoid Fever.
Appendicectomy.
Gonorrhoea aet. 37 years.

Examination: Pale man. Weight 11 stones.

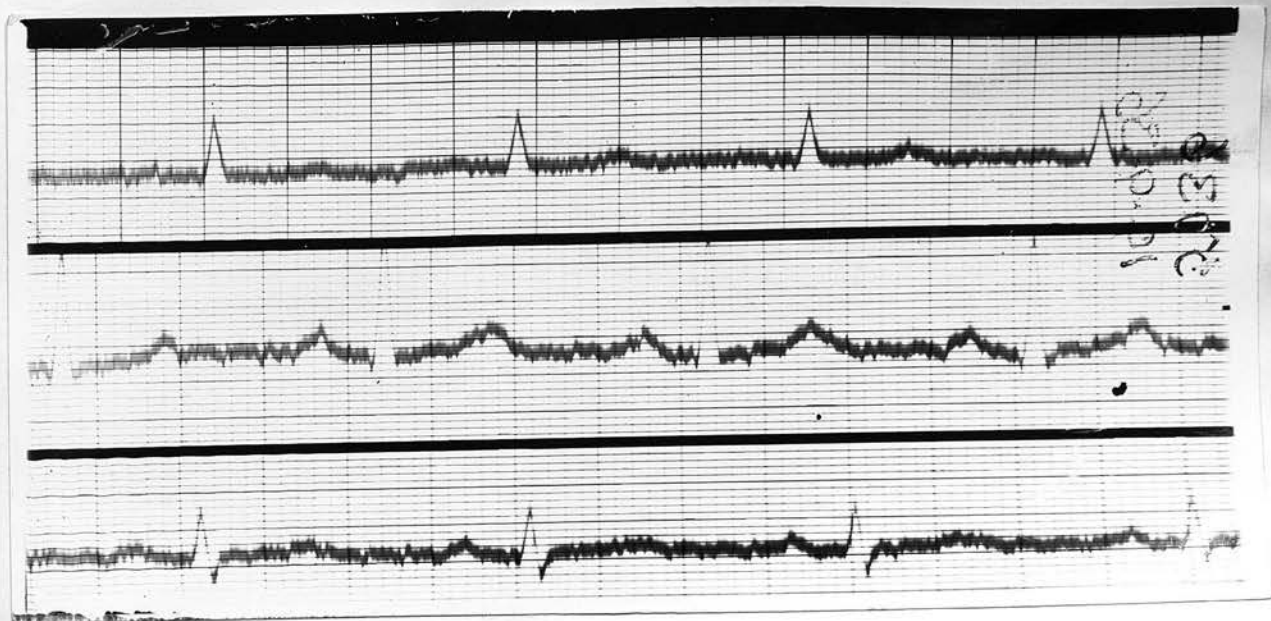
Circulatory System: Heart rate, 80; regular.
Pulse: 'Water Hammer' in character.
Capillary pulsation present.
Blood-pressure: 200/50.
Apex beat: 6th interspace at
anterior axillary line.
Forcible in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic and
diastolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin present.

Case 43 (continued)Nervous System: N.A.D.W.R.: Positive.Electrocardiogram: P₃ flat.X-ray Examination: "Cardiac enlargement, affecting mainly the left ventricle. Slight dilatation aortic arch. Old Pleuro-diaphragmatic adhesion right lung base.Diagnosis: Aortic incompetence.
Aortitis.
Specific infection.Circulatory Rate: $13\frac{4}{5}$ secs.

Case 48.Sex: Male. Married.Age: 37 years.Occupation: Tailor.

History: 2 months. Frontal headaches and dizziness. Increasing dyspnoea on exertion. Experiences a 'tightness in the chest' on walking too quickly or running, but no actual pain. Palpitation on exertion and occasional faintness. 3 weeks ago, had a profuse epistaxis which relieved his headache for a few days. Continued at his work.

Previous Illnesses: Nil. V.D. denied.Family History: 1 child.Habits: Alcohol nil. Heavy cigarette smoker.Examination: Pale man. Weight 8 st. 12 lbs.Circulatory System: Heart rate, 80; regular.

Pulse: Water Hammer.

Capillary pulsation.

Blood-pressure, 138/62.

Apex Beat: 6th interspace, outside mid-clavicular line.

Heaving in character.

Heart sounds: Mitral area, systolic and diastolic bruits.

Aortic area, systolic and diastolic bruits.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.

Case 48 (continued)

Urine: N.A.D.

Nervous System: Pupils equal and react light and accommodation.
Deep reflexes present.

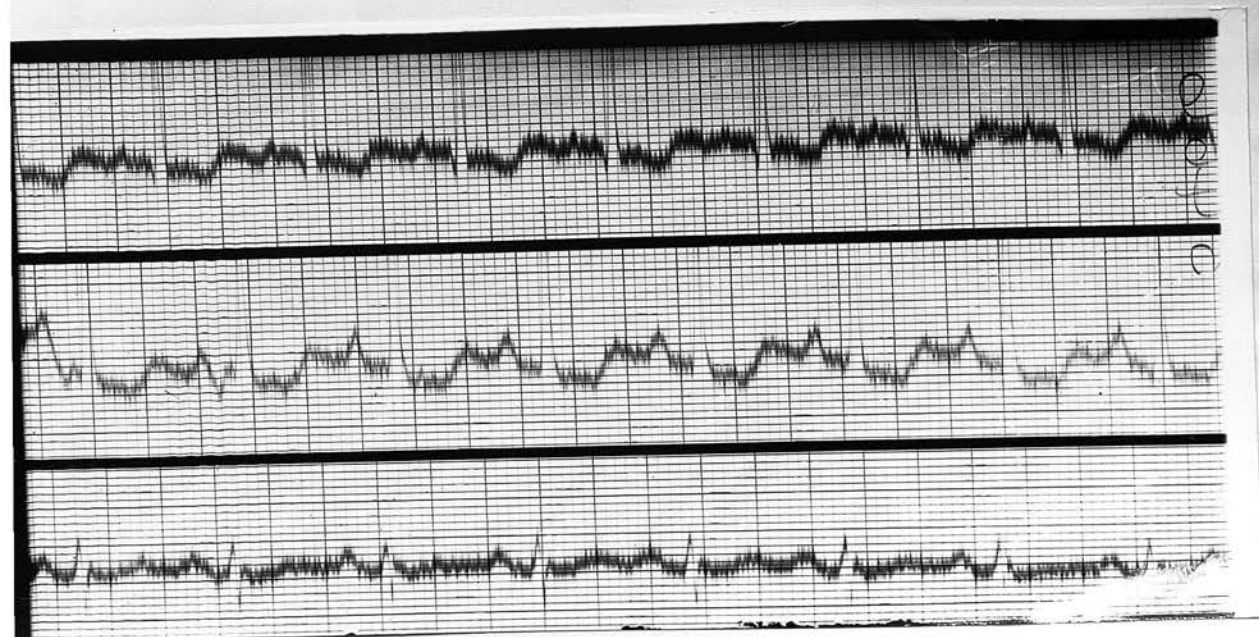
W.R.: Negative.

Electrocardiogram: T₁ inverted.

Left ventricular preponderance.

Diagnosis: Aortic incompetence. Aetiology ? specific.

Circulatory Rate: $12\frac{4}{5}$ secs.



Case 50.

Sex: Male. Married.

Age: 55 years.

Occupation: Shop assistant.

History: 2 years. Praecordial pain on exertion. Giddiness.
Dyspnoea on slight exertion. Easily tired.

Previous Illnesses: Luetic infection 1921. Energetically treated.

Family History: Nil relevant.

Habits: Alcohol and tobacco moderate.

Examination: Weight 12 stones.

Circulatory System: Heart rate, 76; regular.

Blood-pressure, 180/84.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Aortic 2nd sound accentuated and
ringing.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 50 (continued)

Nervous System: Pupils equal and react.
Deep reflexes present.

W.R: Positive.

Electrocardiogram: Left ventricular preponderance.

Diagnosis: Aortitis. Specific.
Myocardial degeneration.

Circulatory Rate: $11\frac{3}{5}$ secs.



Case 54.Sex: Male. Single.Age: 39 years.Occupation: Jobber.History: 2 months. Cough, slight, with scanty sputum, which
14 days ago was streaked with blood.

1 month. Dyspnoea on exertion. Palpitation.

1 year. Epigastric pain immediately after meals, relieved
by alkalies.Previous Illnesses: Tonsillitis.
Hydrocele operation.
V.D. denied.Family History: Nil relevant.Habits: Alcohol nil; tobacco moderate.Examination: Pale. Weight 10 st. 5 lbs.Circulatory System: Heart rate, 73; regular.

Blood-pressure, 108/60.

Apex beat: 6th interspace outside
mid-clavicular line.

Forcible in character.

Heart sounds: Mitral area, systolic and
diastolic bruits.Aortic area, systolic and
diastolic bruits.Lungs: N.A.D.Abdomen: N.A.D.Edema: Nil.Urine: N.A.D.

Case 54 (continued)

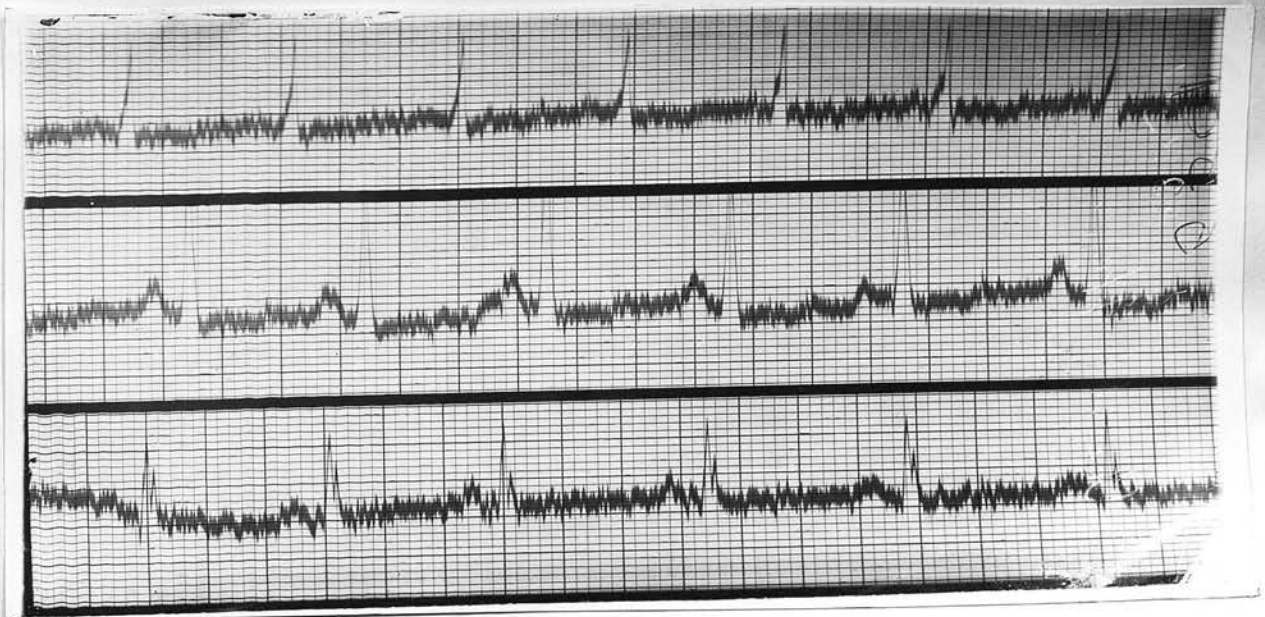
Nervous system: Pupils equal and react.
Deep reflexes present.

W.R: Positive.

Electrocardiogram: R₁ and R₃ notched.

Diagnosis: Aortic incompetence - specific.

Circulatory Rate: $14\frac{1}{5}$ secs.



Case 73

Sex: Male. Married.

Age: 55 years.

Occupation: Boiler Maker.

History: 12 months. Dyspnoea and palpitation on exertion.
9 months. Praecordial pain on exertion, lasting a few minutes, and relieved by rest. Pain difficult to localise, but chiefly sub-mammary.
Giddiness and faintness.
Unable to work 12 months.

Previous Illnesses: Luetic infection aet. 35 years.
Inadequately treated.

Family History: Father and mother both died 'heart trouble' about age 60 years.

Habits: Alcohol and tobacco moderate.

Examination: Weight 13 stone.

Circulatory System: Heart rate, 80; regular.
Blood pressure 144/96.
Apex beat, impalpable.
Heart sounds: closed in all areas.

Lungs: Slight degree emphysema.

Abdomen: Nil.

Oedema: Nil.

Urine: Albumin trace.

Nervous System: N.A.D.

Case 73 (continued)

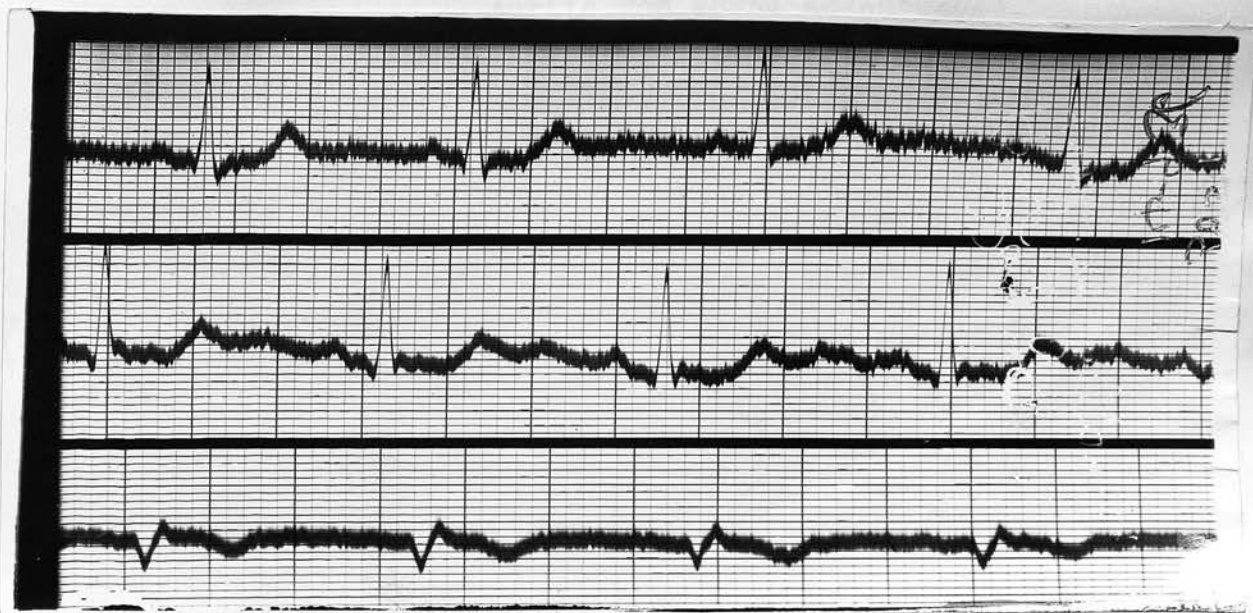
W.R: Negative.

Electrocardiogram: Q present.
Lead III low voltage.
T₃ inverted.

X-ray Examination: "No appreciable cardiac enlargement.
Aortic shadow shows normal appearances."

Diagnosis: Myocardial degeneration ? specific infection.

Circulatory Rate: $13\frac{4}{5}$ secs.



Case 77

Sex: Male. Married.

Age: 54 years.

Occupation: Window Cleaner.

History: 11 months. Palpitation, exhaustion and giddiness.
Slight dyspnoea and praecordial pain on exertion.

Previous Illnesses: Nil.

Family History: Nil relevant.

Habits: Alcohol nil.
Tobacco moderate.

Examination: Weight 10 stone.

Circulatory System: Heart rate, 68; regular.
Blood pressure 170/90.
Apex beat, 5th interspace inside
mid-clavicular line.
Heart sounds: Aortic area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

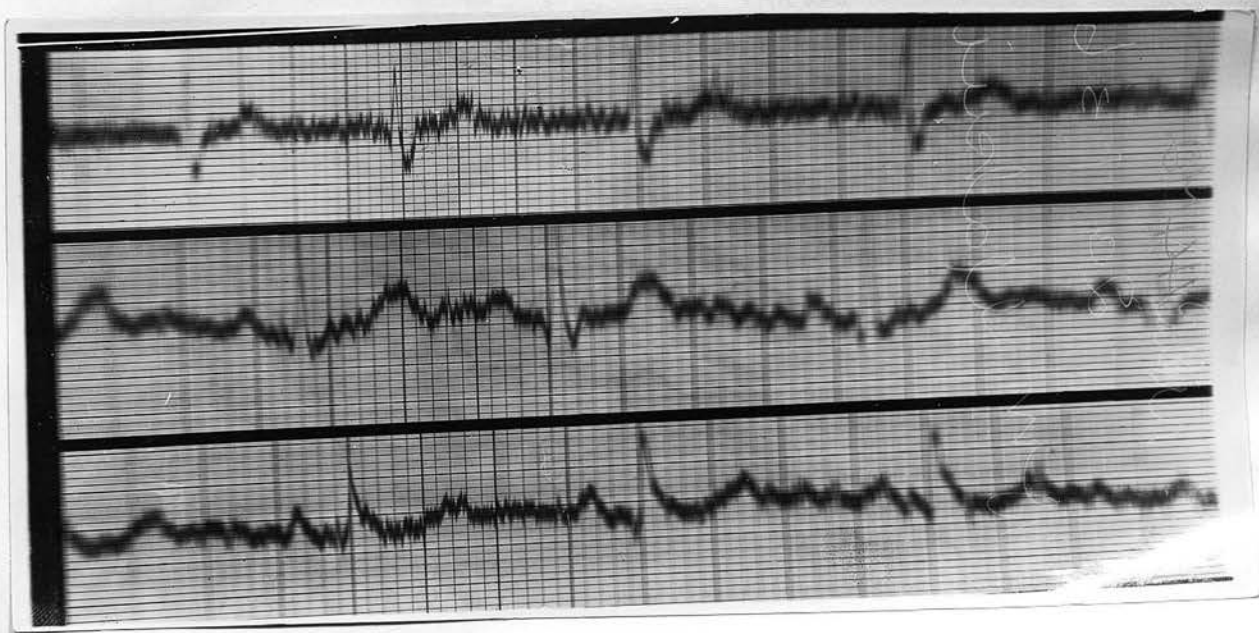
Urine: N.A.D.

Nervous System: Deep reflexes present.
Pupils equal and react.

W.R: Positive.

Case 77 (continued)Electrocardiogram: S₁ S₂ S₃ slurredDiagnosis: Myocardial degeneration.

Aortitis - specific infection.

Circulatory Rate: 12 $\frac{2}{5}$ secs.

Case 101

Sex: Female: Married.

Age: 52 years.

Occupation: Housewife.

History: 6 months. Dyspnoea on exertion, increasing, and now accompanied by palpitation and faintness. Cough at commencement, lasting a few weeks. Increasing difficulty doing her work. Insomnia and anorexia.

Previous Illnesses: Neuritis left arm 4 years ago.
Menopause aet. 40 years.

Family History: 3 children.
4 miscarriages.

Examination: Pale.
Weight 10 stone.

Circulatory System: Heart rate, 94; regular.
Pulse, 'water-hammer'.
Capillary pulsation present.
Blood pressure 190/60.
Apex beat, 6th interspace at anterior axillary line.
Heavy in character.
Heart sounds: Gallop rhythm present.
Mitral area, systolic and diastolic bruits.
Aortic area, systolic and diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Case 101 (continued)

Nervous System: Deep reflexes present.
Pupils equal and react.

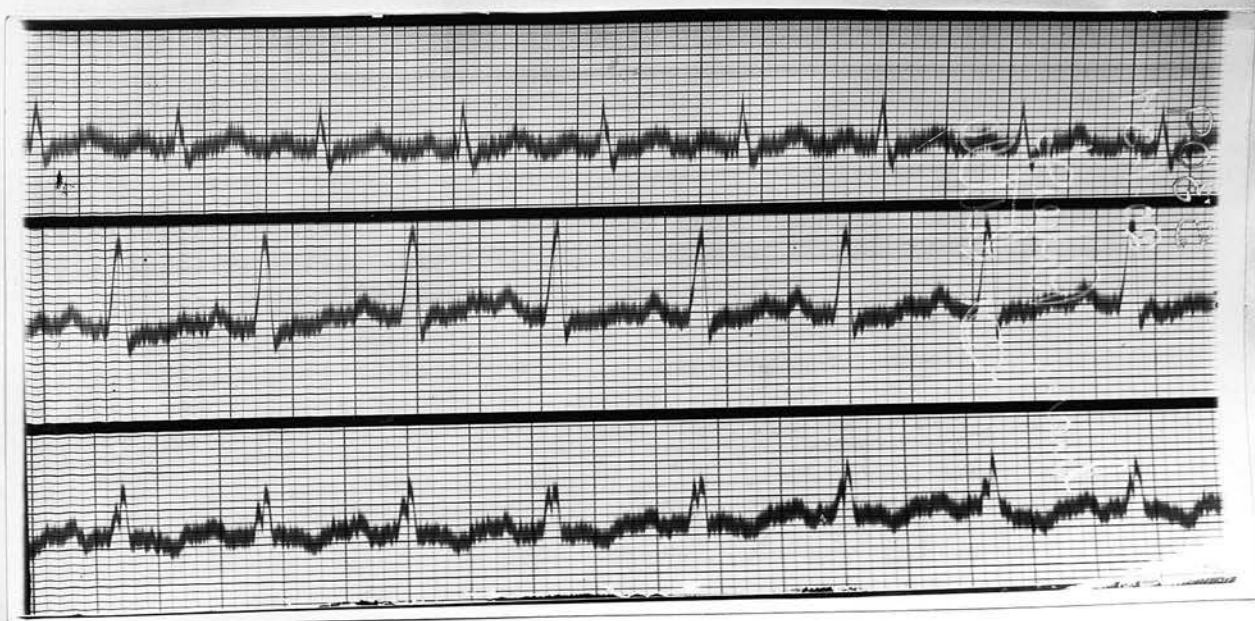
W.R: Positive.

Electrocardiogram: R₂ slurred.
R₃ split.
T₃ inverted.

X-ray Examination: "Marked general cardiac enlargement, mainly left sided. Slight diffuse dilatation of aorta."

Diagnosis: Aortitis, Aortic incompetence, specific.

Circulatory Rate: $25\frac{3}{5}$ secs.



Case 113

Sex: Male. Married.

Age: 48 years.

Occupation: Marine Engineer.

History: 1 month ago, had influenza. This was followed by attacks of giddiness, generally in the morning, and lasting a few minutes. Attacks have continued since. Dyspnoea on exertion, 1 month.

Previous Illnesses: Influenza.
V.D. denied.

Habits: Alcohol and tobacco moderate.

Examination: Weight 10 stone.

Circulatory System: Heart rate, 80; regular.
Blood pressure 218/88.
Pulse, 'water-hammer'.
Apex beat, 6th interspace at anterior axillary line.
Heavy in character.
Heart sounds: Mitral area, presystolic bruit.
Aortic area, systolic and diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R: Positive.

Case 113 (continued)

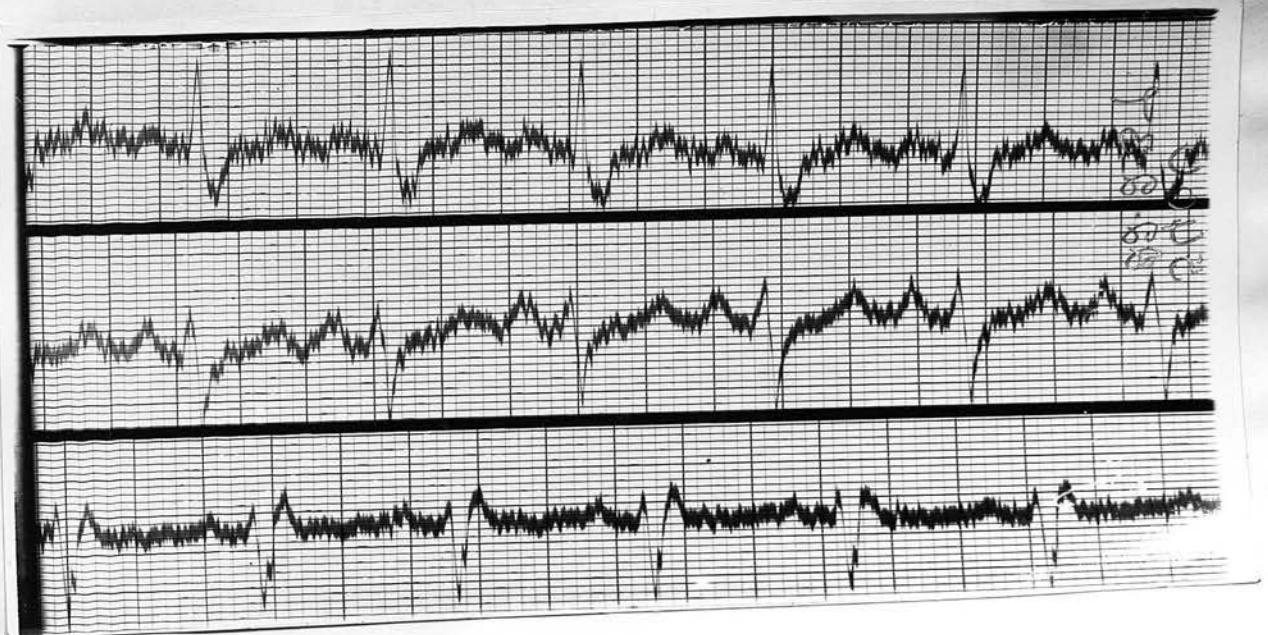
Electrocardiogram: Left ventricular preponderance.

S₁ slurred.

R₃ notched.

Diagnosis: Aortic regurgitation, specific.

Circulatory Rate: $8\frac{3}{5}$ secs.



Case 114.

Sex: Male. Married.

Age: 40 years.

Occupation: Tram Driver.

History: 4 years. Dyspnoea on exertion.

3 days ago, while at work, experienced sudden gripping, retrosternal pain, which did not radiate, and was accompanied by dyspnoea. Pain lasted ½ hour, and he was compelled to stop work.

4 years winter cough, and occasional giddiness.

Previous Illnesses: Luetic infection, aet. 18 years.

Tonsillitis.

Influenza.

'Goitre' aet. 34 years.

Family History: Father died 'heart failure'.

2 children.

Wife has had 4 miscarriages.

Habits: Alcohol, tobacco moderate.

Examination: Weight 12 stone.

Circulatory System: Heart Rate, 80; regular.

Blood pressure 124/100.

Apex beat, not palpable.

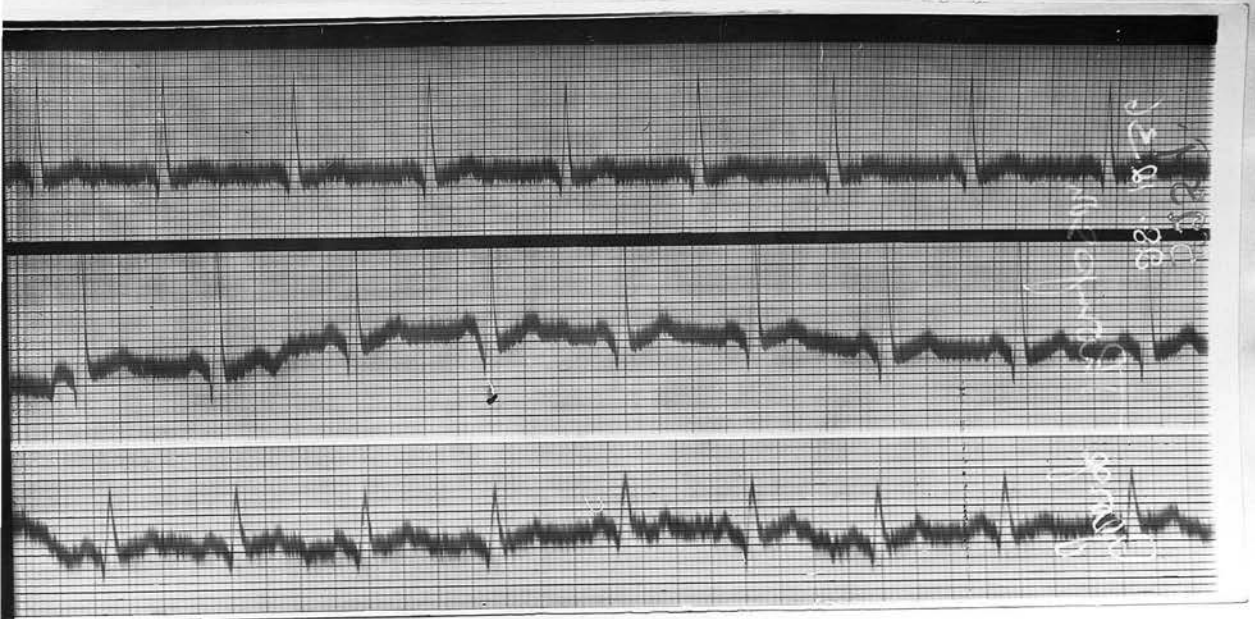
Heart sound: Aortic area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: Emphysema.

Breath sounds, harsh vesicular.

Abdomen: N.A.D.

Case 114 (continued)Oedema: Nil.Urine: N.A.D.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: Q₁ present.Q₂ + +Q₃ +X-ray Examination: "No appreciable cardiac enlargement.
Slight diffuse dilatation of aortic arch."Diagnosis: Aortitis specific.Circulatory Rate: $12\frac{2}{5}$ secs.

Case 121

Sex: Male. Single.

Age: 35 years.

Occupation: Salesman.

History: 6 months ago, discharged from hospital, after Broncho-pneumonia. Since discharge, complained of dyspnoea, and slight sub-sternal pain on exertion. Pain does not radiate.

Palpitation and lack of energy.

Unable to work since pneumonia.

Previous Illnesses: Tonsillitis.
Influenza.
Broncho-pneumonia.
V.D. denied.

Habits: Alcohol nil.
Tobacco moderate.

Examination: Weight 8 st. 2 lbs.

Circulatory System: Heart rate, 78; regular.
Pulse, suggestive 'water-hammer'.
Blood pressure 154/92.
Apex bent, 5th interspace outside mid-clavicular line.
Forcible in character.
Heart sounds: Mitral area, systolic bruit.
Aortic area, systolic and diastolic bruits.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 121 (continued)

Nervous System: N.A.D.

W.R: Positive.

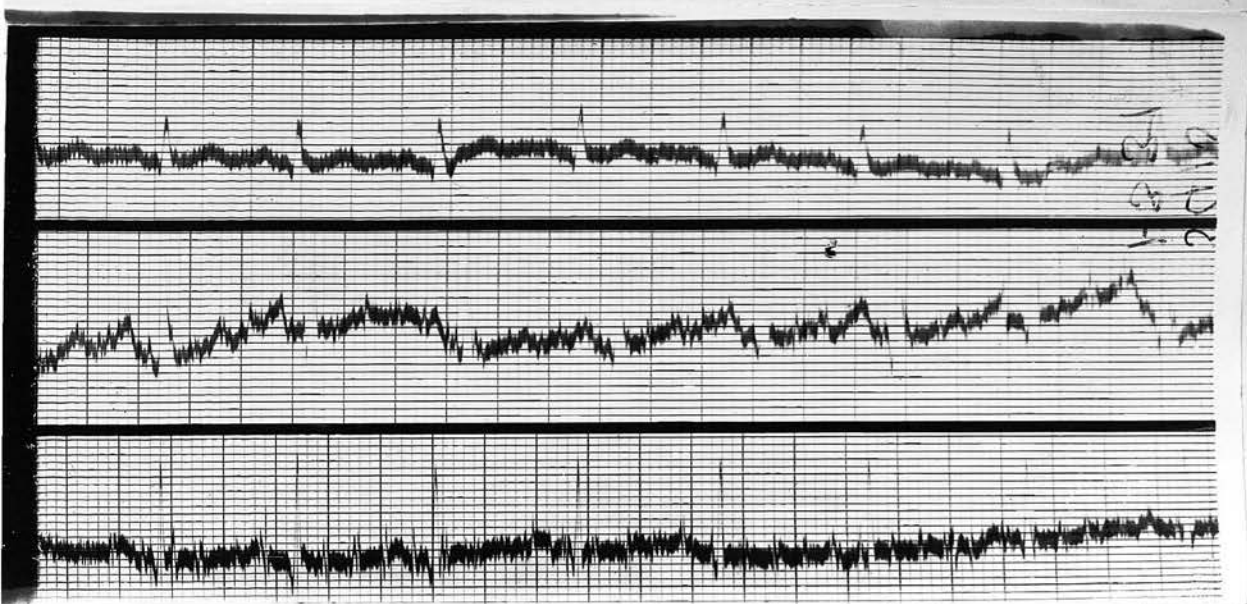
Electrocardiogram: R₁ slurred.

Q present.

X-ray Examination: "Left ventricular enlargement. Slight diffuse enlargement of Aortic arch."

Diagnosis: Aortic incompetence, aortitis, specific.

Circulatory Rate: $11\frac{4}{5}$ secs.



Case No.

Sex: Male, Single

Age: 49 years

Occupation: Journalist

History: 1 year. Pre-auricular pain on exertion. The pain is described as not being severe, but rather a twinge. It occurs 1/2 hour, minutes to the onset, and is relieved by rest.

GROUP 6

5 cases

Myocardial degeneration.

Average circulatory rate 13 secs.
(Range 10.2 - 17.6 secs.)

Previous Illnesses:

- Malaria
- Appendicitis
- _____
- _____

Family History: Nil relevant.

Habits: Alcohol nil; Tobacco 3 cigs. per week.

Examination: Weight, 15 st.

Circulatory System: Heart rate, 74; regular.
 Blood pressure, 100/70.
 Apex beat: 5th intercostal space, mid-clavicular line.
 Carotid arteries: closed in all areas.

Exam: N.A.B.

Arterial N.A.B.

Urine: N.A.

Case 26.

Sex: Male. Single.

Age: 29 years.

Occupation: Journalist.

History: 1 year. Praecordial pain on exertion. This pain is described as not being severe, but rather a twinge. It occurs over a wide area, radiates to the throat, and is eased by rest.

Palpitation on exertion.

3 years. Abdominal discomfort after meals - eased by alkalies.

Previous Illnesses: Influenza.
Malaria.
Appendicectomy.
V.D. denied.

Family History: Nil relevant.

Habits: Alcohol nil; tobacco 3 ozs. per week.

Examination: Weight, 12 st.

Circulatory System: Heart rate, 74; regular.
Blood-pressure, 120/70.
Apex beat: 5th intercostal inside
mid-clavicular line.
Heart sounds: closed in all areas.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Case 26 (continued)

Urine: N.A.D.

Nervous System: N.A.D.

W.R.: Negative.

Electrocardiogram: Q₃ present.

QRS complex slightly slurred Lead I.

S - T depression.

Diagnosis: Myocardial degeneration.

Circulatory Rate: 11 secs.



Case 83.

Sex: Male. Married.

Age: 63 years.

Occupation: Engineer.

History: 14 days ago, while at rest, experienced a sudden attack of lower sub-sternal pain, which did not radiate. Pain was very severe for 10 minutes, and gradually lessened. At the same time, felt giddy and faint. Praecordial soreness was present 3 - 4 days afterwards.

8 years ago, a similar - but milder - attack had occurred, and following this slight dyspnoea on exertion was noticed.

Previous Illnesses: Right nephrectomy aet. 37 years. Calculus.
Neuritis right shoulder 8 years ago.
V.D. denied.

Family History: Nil relevant.

Habits: Alcohol nil. Tobacco moderate.

Examination: Weight 10 stones.

Circulatory System: Heart rate, 80; regular.
Blood-pressure, 124/74
Apex beat: Impalpable.
Heart sounds: Mitral area soft systolic bruit.

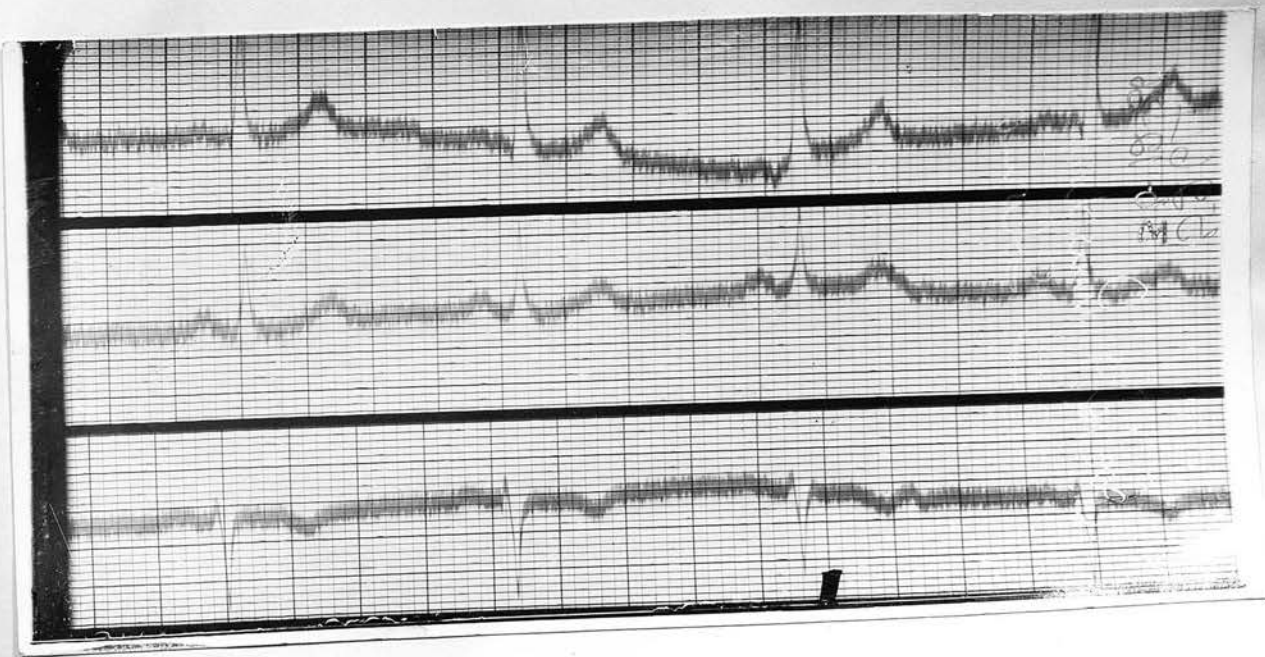
Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

Case 83 (continued)W.R.: Negative.Electrocardiogram: S slurred. T₃ inverted.
Left ventricular preponderance.Diagnosis: Coronary thrombosis.
Myocardial degeneration.Circulatory Rate: $13\frac{3}{5}$ secs.

Case 104.

Sex: Male. Married.

Age: 52 years.

Occupation: Motor Driver.

History: 3 years. Dyspnoea and occasional praecordial pain on exertion. Palpitation and exhaustion.

Cough - sputum scanty.

6 years. Winter cough, slight.

Previous Illnesses: Pneumonia aet. 49 years.
Symptoms followed shortly afterwards.

Habits: Alcohol and tobacco moderate.

Examination: Weight 8 st. 7 lbs.

Circulatory System: Heart rate, 100; regular.
Blood-pressure, 130/70.
Apex beat: Impalpable.
Heart sounds: closed all areas.

Lungs: Emphysema.

Breath sounds: harsh vesicular occasional rhonchi.

Sputum: Frothy; negative T.B.

Abdomen: N.A.D.

Oedema: Nil.

Urine: Albumin trace.

Nervous System: N.A.D.

W.R: Negative:

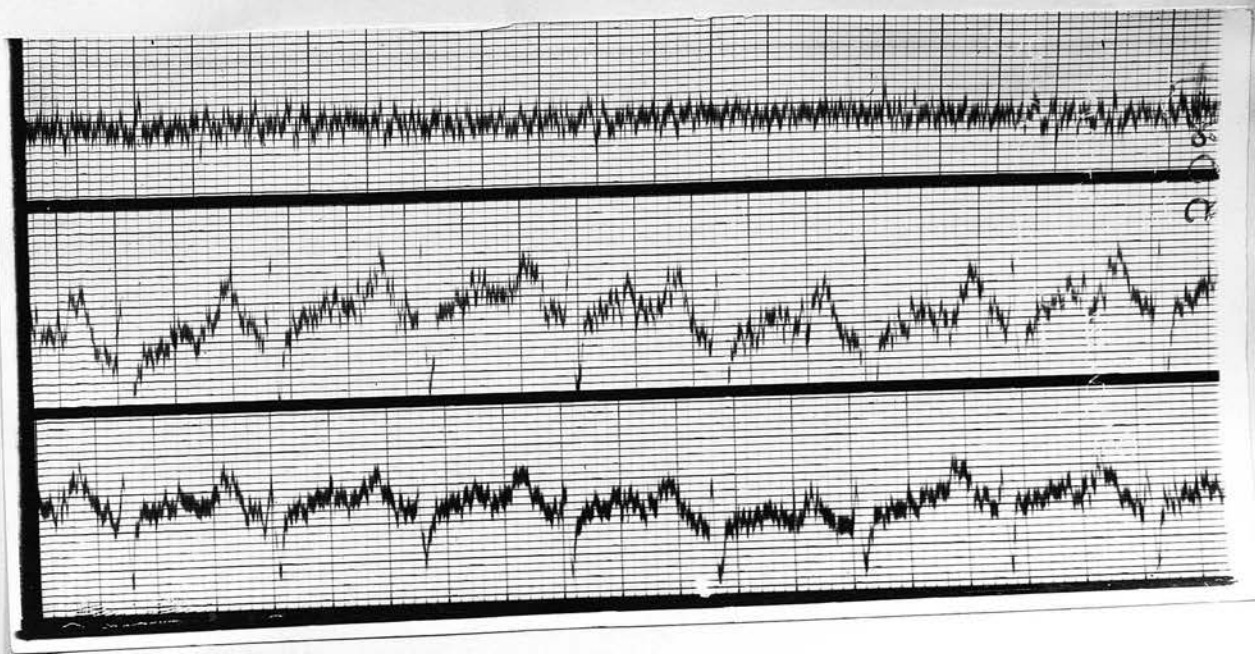
Case 104 (continued)

Electrocardiogram: QRS flat, Lead I.
Slightly slurred.

X-ray Examination: "No appreciable cardiac enlargement. Chronic inflammatory changes in lungs. No evidence bronchiectasis."

Diagnosis: Myocardial degeneration.
Chronic bronchitis and emphysema.

Circulatory Rate: $17\frac{1}{5}$ secs.



Case 109.Sex: Female. Married.Age: 23 years.Occupation: Housewife.History: 2 years ago, palpitation, 'fluttering in chest', praecordial pain, nervousness and loss of weight. Told at that time that her heart was affected.

1 year ago thyroidectomy was performed. Followed by improvement including gain of 1 stone in weight.

Now complains dyspnoea and occasional praecordial pain on exertion.

Faintness and exhaustion.

Previous illnesses: Growing pains, infancy.
Influenza.Family History: 1 child aet. 4 years. 1 miscarriage.
3 months pregnant.Examination: Weight 10 st. 2 lbs.
Thyroidectomy scar. Nil palpable.Circulatory System: Heart rate, 86; regular.
Blood-pressure, 130/80.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: Mitral area systolic bruit.
Pulmonary 2nd sound slightly
accentuated.Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Case 109 (continued)Nervous System: N.A.D.W.R.: Negative.Electrocardiogram: Q₁ present. T₃ inverted.

Left ventricular preponderance.

Diagnosis: Myocardial degeneration following thyrotoxicosis.Circulatory Rate: $17\frac{3}{5}$ secs.

Case 110.

Sex: Male. Married.

Age: 42 years.

Occupation: Store-keeper.

History: 18 years. Praecordial ache more or less constantly present and no relation to exertion.

Discharged from the Army in 1919. V.D.H.

4 years. Dyspnoea on exertion. Palpitation, giddiness, and faintness.

Symptoms have recently become intensified, following an attack of influenza, since when he has also suffered from insomnia and irritability.

Previous illnesses: Tonsillitis. Influenza.

Habits: Alcohol nil; tobacco moderate.

Examination: Pale. Nervous. Weight 9 st. 6 lbs.

Circulatory System: Heart rate, 86; regular.

Blood-pressure, 156/90.

Apex beat: 5th interspace just outside mid-clavicular line.

Heart sounds: Aortic 2nd sound slightly accentuated.

Lungs: N.A.D.

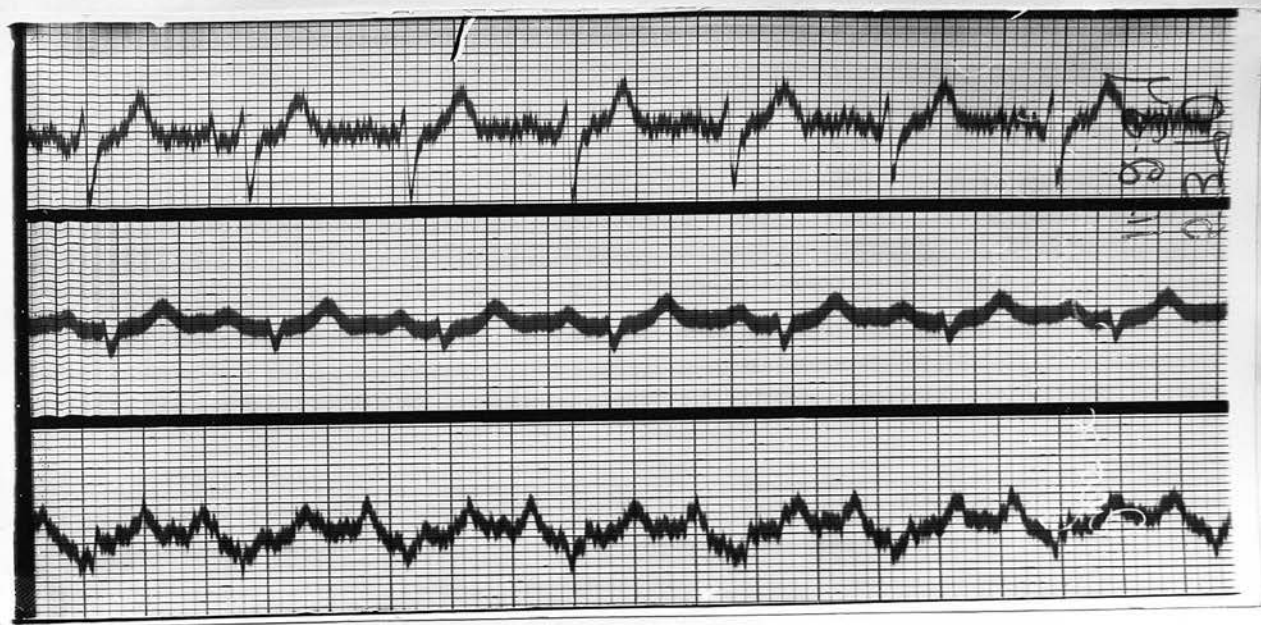
Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 110 (continued)Nervous System: N.A.D.W.R: Negative.Electrocardiogram: S₁ slurred.

Lead II Lead III Low Voltage.

Diagnosis: Myocardial degeneration.Circulatory Rate: $10\frac{1}{5}$ secs.

Date 25.

Sex: Male, Married.

Age: 28 years.

Occupation: Boot repairer.

History: 3 years ago, while working, had an accident, falling down
 in the solar plexus by a hammer. He was "knocked out"
 for 2 hours and spent 4 days in bed. Shortly after
 this accident, he was seized with at work with severe
 pain in the chest. The pain radiated down both arms and
 lasted a few minutes. Then told he was suffering from
 "Grippe". He was treated at different hospitals, at one of which
 he had "Myocarditis".

GROUP 7
4 cases

Effort Syndrome.

Severe attacks of pain, which has been very
 distant. Average circulatory rate 8 secs.
 (Range 6.2 - 10.4 secs.)
 Also complains of palpitation and giddiness.
 Unable to work 3 weeks. Loss of weight, 1 stone and
 3 months. -----

Previous Illnesses: Tuberculosis.

Habits: Alcohol nil; tobacco moderate.

Presenting: Pale, thin, emaciated man. Weight 8 st. 1 lb.
 Gait very slowly and with a marked stoop.

Circulatory System: Heart rate, 96; regular.
 Blood-pressure, 120/80.
 Apex beat: 5th intercostal space
 mid-clavicular line.
 Heart sound: Muted all areas.
 Aortic 2nd sound accentuated.

Exam: N.A.P.

Adm: N.A.P.

Case 55.Sex: Male. Married.Age: 24 years.Occupation: Boot repairer.

History: 3 years ago, while cycling, had an accident, being struck in the solar plexus by a handlebar. He was 'knocked out' for 2 hours and spent 14 days in bed. Shortly after this accident, he was seized while at work with sudden pain in the chest. The pain radiated down both arms and lasted a few seconds. Then told he was suffering from 'Nerves'. Attended different Hospitals, at one was told he had 'Disseminated Sclerosis'.

2 years. Numerous attacks of pain, which has been persistent for the last 3 months and now is only able to walk a short distance on account of praecordial pain and dyspnoea. Also complains of palpitation and giddiness. Unable to work 3 months. Loss of weight, 1 stone last 3 months.

Previous Illnesses: Tonsillitis.Habits: Alcohol nil; tobacco moderate.

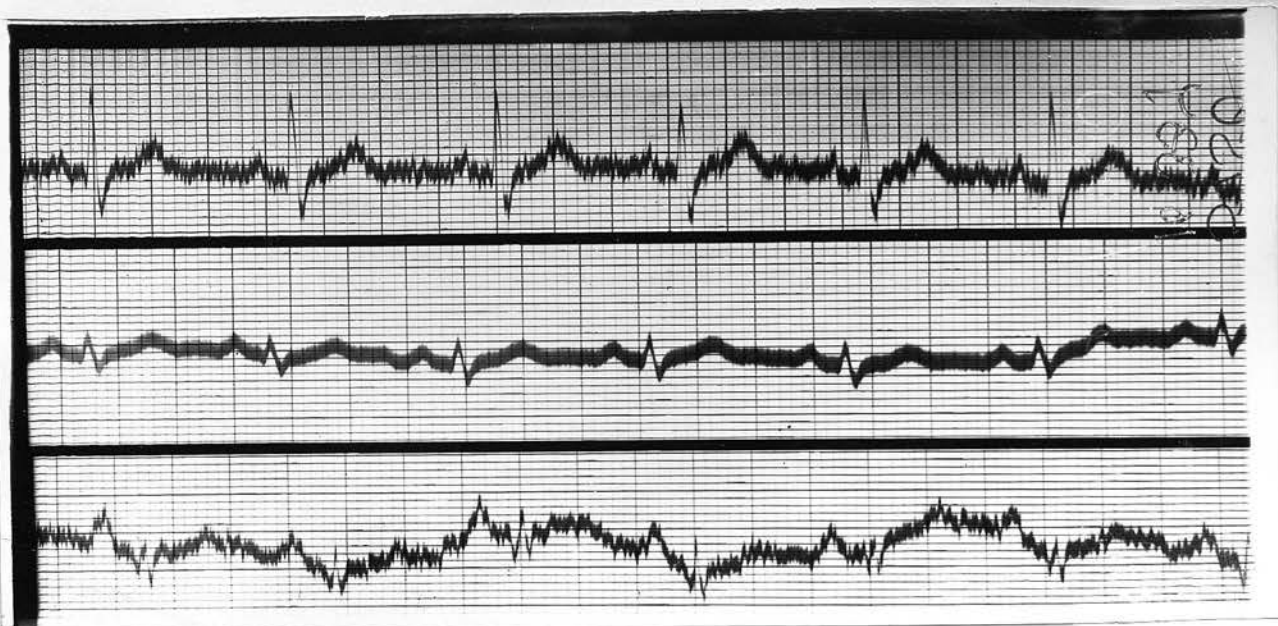
Examination: Pale, thin, anxious man. Weight 8 st. 1 lb.
Walks very slowly and with a marked stoop.

Circulatory System: Heart rate, 84; regular.
Blood-pressure, 150/90.
Apex beat: 5th interspace inside
mid-clavicular line.
Heart sounds: Closed all areas.
Aortic 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.

Case 55 (continued)Oedema: Nil.Urine: N.A.D.

Nervous System: Tremor hands; palms wet.
Bilateral nystagmus; pupils equal and react.
Deep reflexes exaggerated.
Plantar reflex, no response.
Abdominal reflexes, present.

W.R: Negative.Electrocardiogram: Lead II Low Voltage.Diagnosis: 'Effort Syndrome'.Circulatory Rate: $7\frac{2}{5}$ secs.

Case 57.

Sex: Male. Married.

Age: 41 years.

Occupation: Miner.

History: 2 years. Attacks of praecordial pain, radiating down both arms to finger tips and up to the neck, lasting a few seconds, brought on by exertion and relieved by lying down. First attack took place while down a mine.

Dyspnoea and palpitation on exertion.

Nervousness, exhaustion, and fainting attacks.

Previous Illnesses: Influenza several times.

Family History: Nil relevant.

Habits: Alcohol nil; tobacco moderate.

Examination: Weight 12 st. 6 lbs.

Circulatory System: Heart beat, 106; regular.

Blood-pressure, 165/94.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: Closed all areas.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: Flushes easily; tremor hands.

Deep reflexes brisk.

Bilateral horizontal nystagmus.

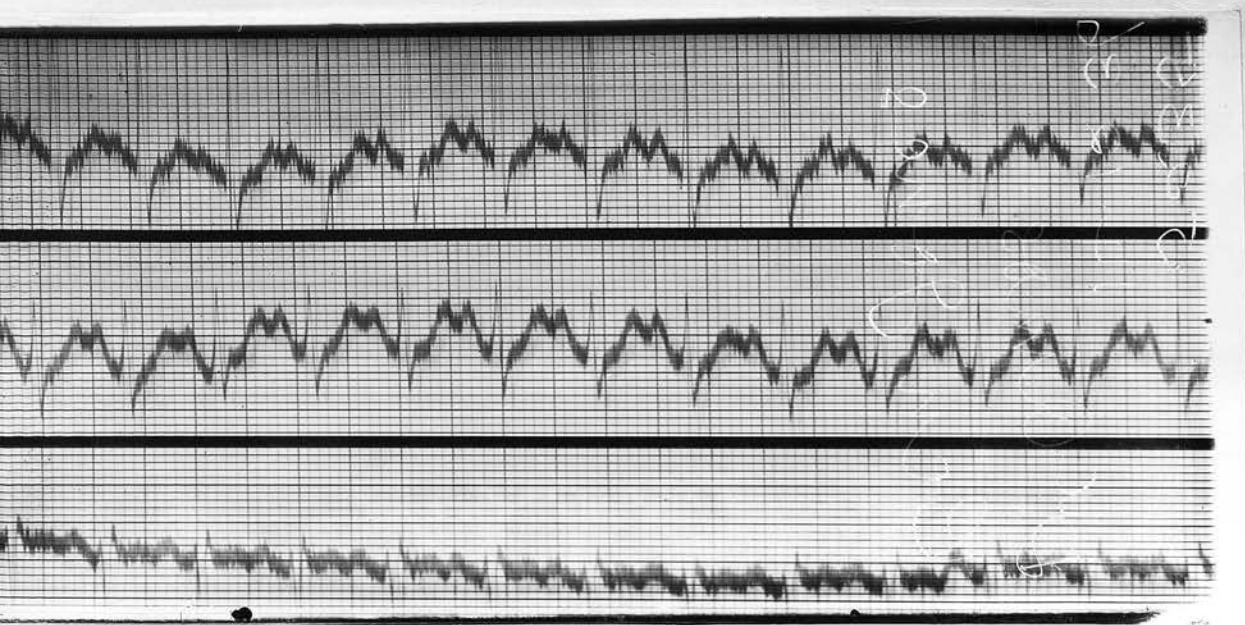
Thyroid gland palpable.

Case 57 (continued)

Electrocardiogram: Tachycardia.
Left ventricular preponderance.

Diagnosis: Myocardial degeneration.
Miner's nystagmus.
'Effort Syndrome'. ? Thyrotoxicosis.

Circulatory Rate: $6\frac{1}{5}$ secs.



Case 76.Sex: Male. Single.Age: 34 years.Occupation: Crane driver.

History: 3 months. Stabbing praecordial pain, localised in left sub-mammary region, and worse on doing heavy work. Giddiness, exhaustion, and nervousness. All symptoms following a severe shock.

Previous Illnesses: Pneumonia aet. 16 years.
Influenza aet. 25 years. V.D. denied.

Family History: Nil relevant.Habits: Alcohol nil; tobacco moderate.Examination: Weight 10 st.

Circulatory System: Heart rate, 94; regular.
Blood-pressure, 144/100.
Apex beat: 5th interspace inside mid-clavicular line.
Diffuse in character.
Heart sounds: Closed all areas.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Nervous System: Coarse tremor, hands.
Deep reflexes exaggerated.

W.R: Negative.

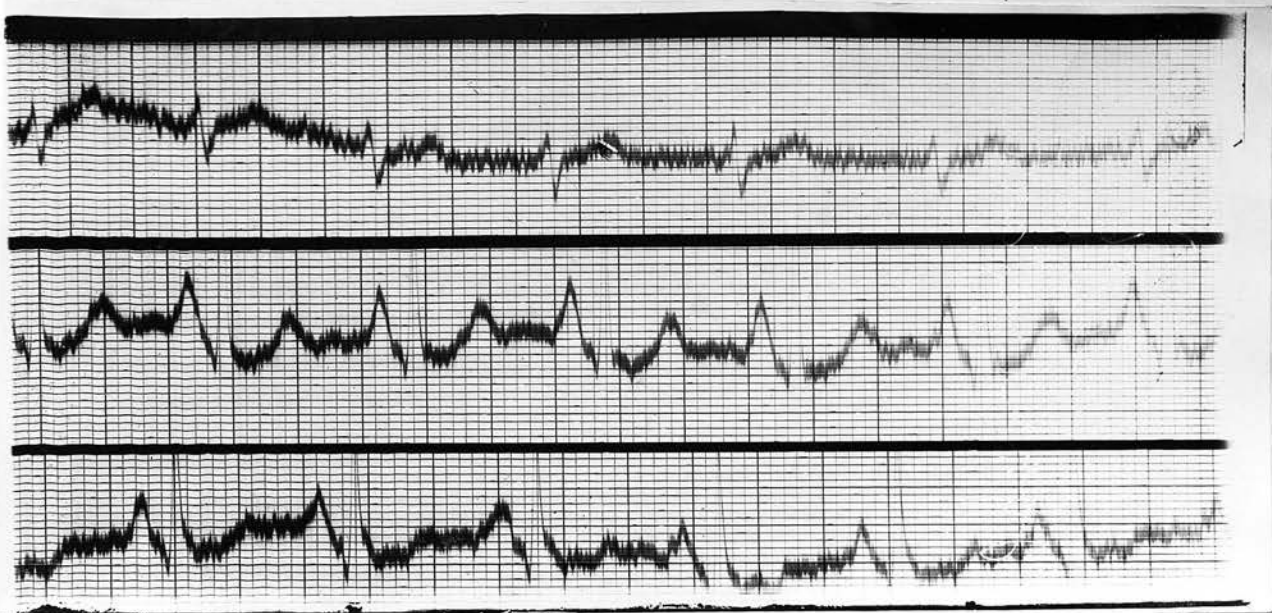
Case 76 (continued)

Electrocardiogram: Q₂, Q₃, present. S₁ slightly slurred.
P₂ and P₃ large.

X-ray Examination: "No appreciable cardiac enlargement.
Aortic shadow shows normal appearances."

Diagnosis: 'Effort syndrome'.

Circulatory Rate: $10\frac{2}{5}$ secs.



Case 106.Sex: Male. Married.Age: 44 years.Occupation: Nil.History: Discharged from Army, 1919, suffering from D.A.H., and is in receipt of a pension.

1 year. The following symptoms have been getting worse: Dyspnoea, palpitation, and praecordial pain on exertion. Exhaustion. States he has collapsed in the street on several occasions recently. Noticed eyes were more prominent recently.

Previous Illnesses: Luetic infection, 1919; treated adequately; given clean bill of health prior to marriage.Habits: Alcohol nil; tobacco moderate.Examination: Nervous man. Weight, 10 st.

Circulatory System: Heart rate, 96; regular.
 Blood-pressure, 130/68.
 Apex beat: 5 interspace at
 mid-clavicular line.
 Heart sounds: closed all areas.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.

Nervous System: Slight bilateral exophthalmos.
 Deep reflexes, brisk.
 Tremor hands.
 Thyroid gland not palpable.

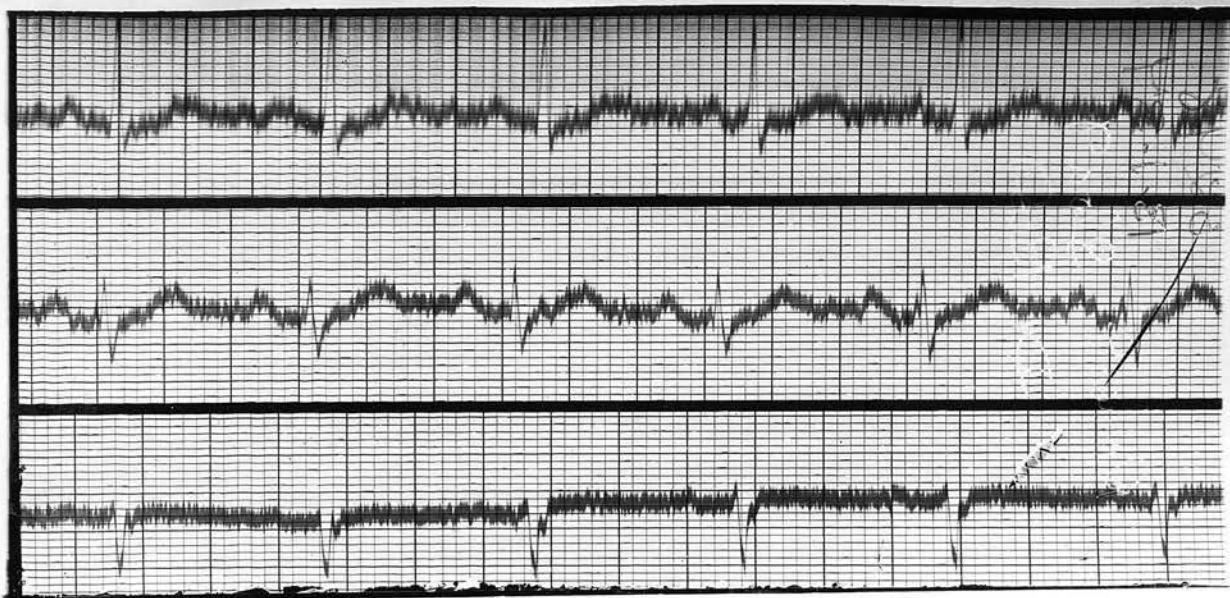
Case 106 (continued)

Electrocardiogram: Left ventricular preponderance.
T₃ flat; S₃ notched.

X-ray Examination: "No appreciable cardiac enlargement.
Aortic shadow shows normal appearance."

Diagnosis: 'Effort Syndrome'.

Circulatory Rate: 9 secs.



Case 14

Sex: Male - Married.

Age: 51 years.

Occupation: Dock Labourer.

History: 18 years. Increasing dyspnea on exertion.

A severe retrosternal pain, commencing lower end of sternum, radiating to neck, and down the left arm to the wrist. Always induced by exertion, and

GROUP 8

3 cases

Angina Pectoris.

Average circulatory rate 12.4 secs.

(Range 11.6 - 13.4 secs.)

Previous Illnesses:

Family History: All normal.

Habits: Alcohol and tobacco moderate.

Weight: 11 stone.

Regulatory System: Heart rate, 70, regular.

Blood pressure 120/80.

Apex beat, 5th intercostal space mid-clavicular line.

Heart sounds: Normal 1st and 2nd aortic spaces.

Unguis: N.A.O.

Respiratory: N.A.O.

Genitals: Nil.

Excretory: N.A.O.

Neurological: N.A.O.

Case 38Sex: Male. Married.Age: 51 years.Occupation: Dock Labourer.History: 1½ years. Increasing dyspnoea on exertion.

4 months retrosternal pain, commencing lower end of sternum, radiating to neck, and down the left arm to the wrist. Pain always induced by exertion, and ceased at once by rest. Worse in cold weather. Pain getting more frequent, and more easily provoked. Is now only able to walk 100 yards without distress. Unable to work 4 months.

Previous Illnesses: Nil. V.D. denied.Family History: Nil relevant.Habits: Alcohol and tobacco moderate.Examination: Weight 11 stone.Circulatory System: Heart rate, 78; regular.

Blood pressure 120/90.

Apex beat, 5th interspace at
mid-clavicular line.

Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.Abdomen: N.A.D.Oedema: Nil.Urine: N.A.D.Nervous System: N.A.D.

Case 38 (continued)

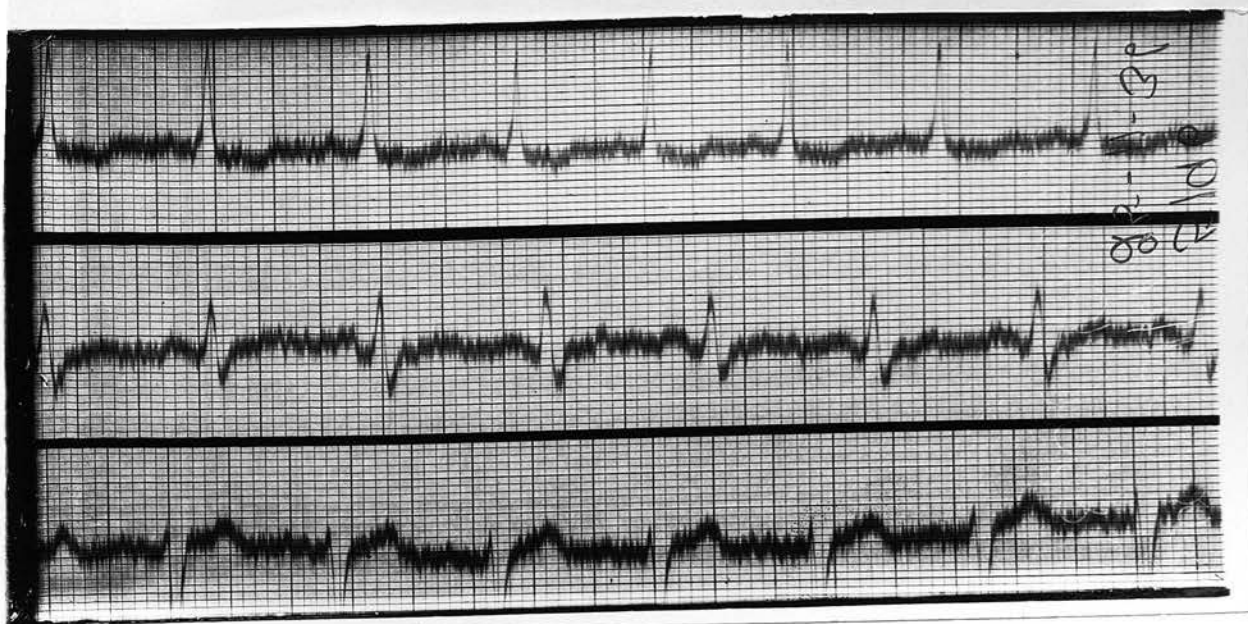
W.R: Negative.

Electrocardiogram: Left ventricular preponderance.

Diagnosis: Angina of effort.
Myocardial degeneration.

Circulatory Rate: $12\frac{1}{5}$ secs.

(Following increase in respiratory amplitude, severe sub-sternal pain, lasting 10 secs, was complained of. Blood pressure taken immediately was found to have risen to 142/80, and Heart rate to 82).



Case 72

Sex: Male. Married.

Age: 44 years.

Occupation: Shop Assistant.

History: 6 weeks. Praecordial pain on exertion. Pain radiates to neck, and is relieved by rest. Slight dyspnoea on exertion. Giddiness.

Previous Illnesses: Pneumonia and pleurisy aet. 24 years.
Loss of right arm in accident in childhood.

Family History: Nil relevant.

Habits: Tobacco, heavy smoker. Alcohol nil.

Examination: Obese one-armed man.
Weight 12 stone.

Circulatory System: Heart rate, 74; regular.
Blood pressure 168/96.
Apex beat, 5th interspace just outside mid-clavicular line.
Slightly forcible.
Heart sounds: Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

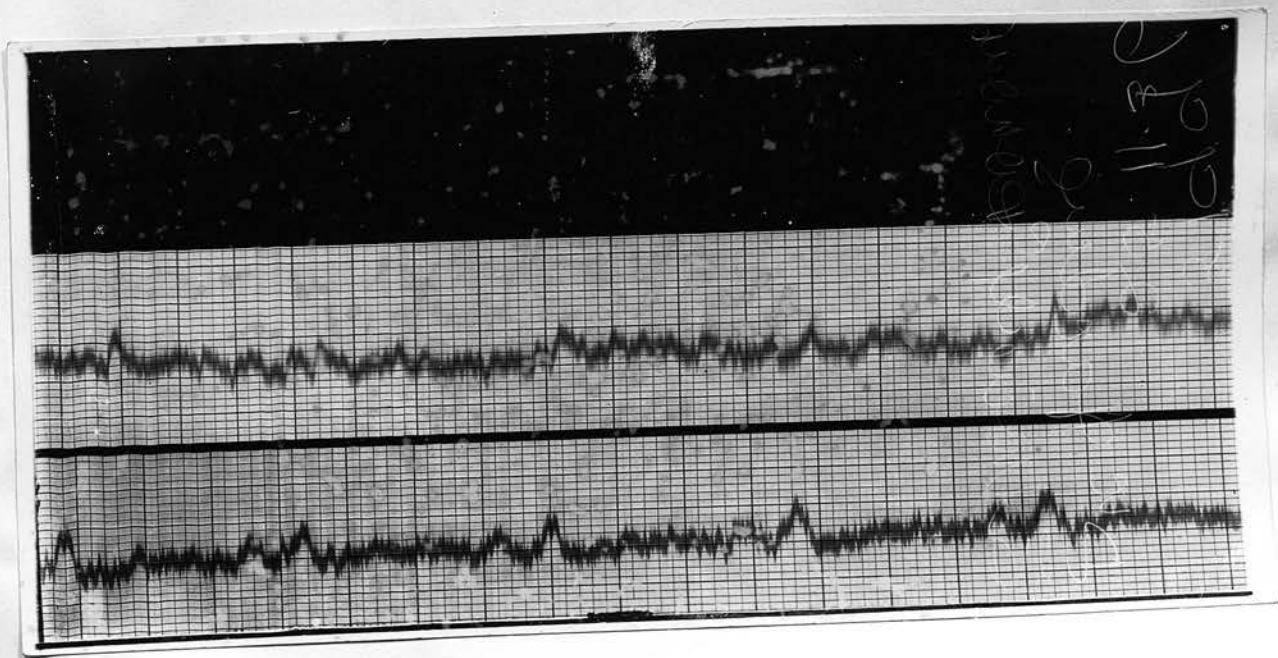
Case 72 (continued)

W.R.: Negative.

Electrocardiogram: Low voltage in the 2 leads.
QRS complex ?slurred.

Diagnosis: Angina of effort.

Circulatory Rate: $13\frac{2}{5}$ secs.



Case 111

Sex: Male. Married.

Age: 52 years.

Occupation: Estate Agent.

History: 1 year. Dyspnoea on exertion.

2 months, upper chest pain, radiates down left arm as far as the finger tips. Pain worse on exertion, and eased by rest, such as standing still. On several occasions, recently, pain has occurred while in bed, and has then been accompanied by dyspnoea.

1 month, severe temporal headaches.

Kept at work.

Previous Illnesses: Influenza.
Dysentery.
V.D. denied.

Habits: Alcohol moderate. Heavy cigarette smoker.

Examination: Weight 10 stone.

Circulatory System: Heart rate, 88; regular.
Blood pressure 150/100.
Apex beat, 5th interspace at
mid-clavicular line.
Heart sounds: suggestion of gallop rhythm.
Mitral area, systolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 111 (continued)

Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P₂ large.

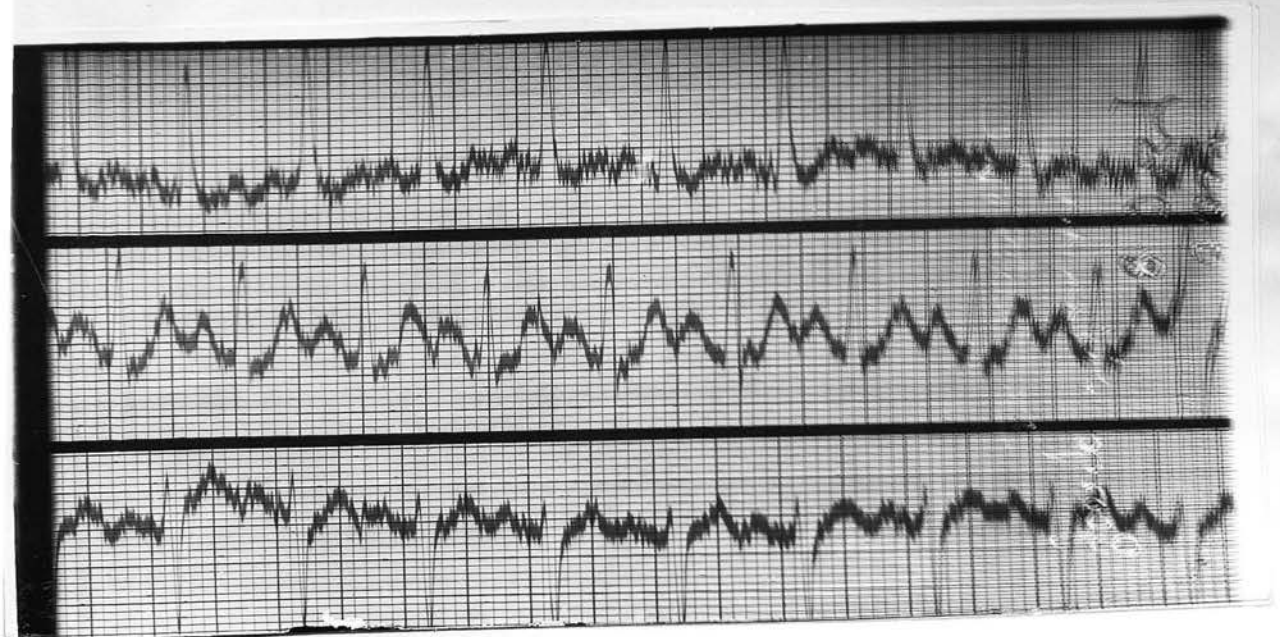
T₂ split.

Left ventricular preponderance.

Diagnosis: Angina of effort.

Myocardial degeneration.

Circulatory Rate: $11\frac{3}{5}$ secs.



GROUP 9

3 cases

Auricular Fibrillation.

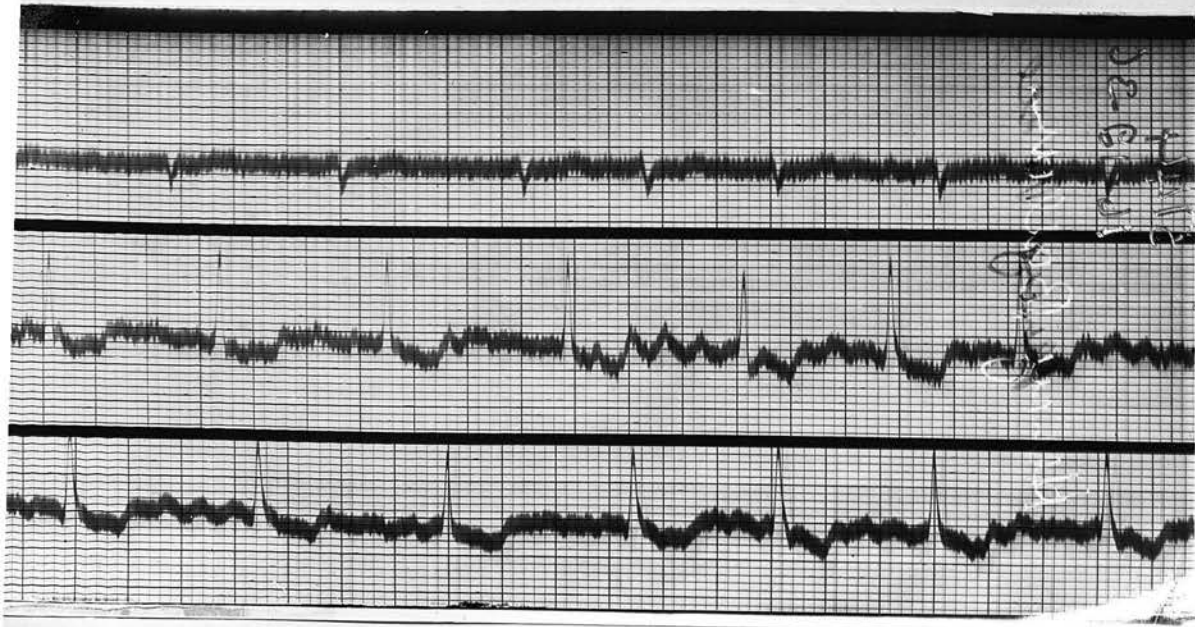
Average circulatory rate 16.4 secs.

(Range 15.8 - 17.6 secs.)

Case 6.Sex: Male. Married.Age: 64 years.Occupation: Insurance Agent.History: Following an attack of pneumonia 3 years ago, has been in indifferent health, loss of weight, cough and increasing dyspnoea. Ceased work 12 months ago, and has been confined to bed for the last month. Has recently suffered from attacks of nocturnal dyspnoea, lasting $\frac{1}{4}$ hour, and followed by the expectoration of blood-stained mucus.Previous Illnesses: Pneumonia 3 years ago.Habits: Alcohol and tobacco nil.Examination: Somewhat wasted looking man.
Weight 8 st, 9 lbs.
Slight degree of finger clubbing present.
Temperature normal.Circulatory System: Heart rate, 78; irregular.
Blood pressure 130/86.
Apex beat, 5th interspace inside
Mid-clavicular line.
Heart sounds: Auricular fibrillation
present.
Mitral area, systolic bruit.Lungs: Emphysematous. Percussion note impaired both
lungs posteriorly.
Breath sounds: Broncho-vesicular both upper lobes.
No adventitious sounds.
Sputum negative T.B. (3 examinations).Abdomen: N.A.D.

Case 6 (continued)Oedema: Nil.Urine: N.A.D.Nervous System: N.A.D.W.R: Negative.Electrocardiogram: Auricular fibrillation.

P absent.

S₂ S₃ slurred.T₃ inverted.X-ray Examination: "No appreciable cardiac enlargement.
Chronic bi-lateral apical T.B. No
evidence of bronchiectasis."Diagnosis: Auricular fibrillation.
Myocardial degeneration.
Chronic pulmonary tuberculosis.Circulatory Rate: $17\frac{3}{5}$ secs.

Case 89.

Sex: Female. Single.

Age: 36 years.

Occupation: Shop Assistant.

History: 15 years ago was in hospital with 'heart trouble'.
3 years ago, while at work 'Collapsed', an attack of coughing and difficulty in breathing. Was then in bed for 7 months, and has been in bed on and off since. 6 weeks ago similar attack.

Previous to this last attack, feet had been swollen during the day.

Just recovered from nausea and vomiting due to overdosage of digitalis.

Previous Illnesses: Influenza.
Tonsillitis.

Examination: Weight 6 st. 3½ lbs.

Circulatory System: Heart rate, 64; irregular
Blood pressure 108/72.
Apex beat 5th interspace outside
mid-clavicular line.
Diastolic thrill at apex.
Heart sounds: Mitral area, diastolic bruit.
Aortic area, systolic and diastolic bruit.

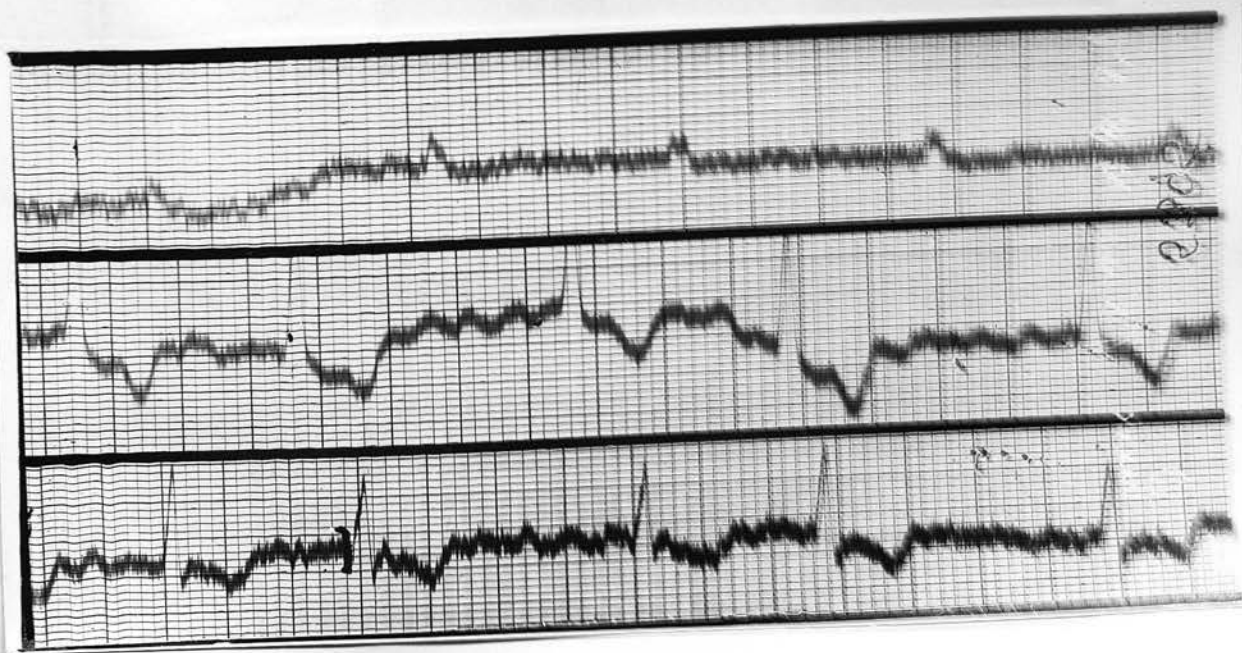
Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

Case 89 (continued)W.R: Negative.Electrocardiogram: P absent.
Auricular fibrillation.
T₂ T₃ inverted, ? Digitalis effect.Diagnosis: Auricular fibrillation.
Aortic incompetence.
Mitral stenosis.
Rheumatic.
Digitalised.Circulatory Rate: $15\frac{4}{5}$ secs.

Case 120

Sex: Female. Single.

Age: 35 years.

Occupation: Shop Assistant.

History: 8 weeks ago, developed influenzal symptoms viz. coughing, sore throat, and muscular pains. Stayed in bed a few days, and on getting up, found she was dyspnoeic on slight exertion, and had troublesome palpitation.

Ordered back to bed and has been there since.

Given digitalis by her doctor.

Previous Illnesses: Rheumatic fever,
aet. 10, 14, 18, 23, and 27 years.
Bronchitis and 'fluid on the lung' aet 28
years.

Examination: Weight 8 st. 10 lbs.

Circulatory System: Heart rate, 60; irregular.
Blood pressure 152/72.
Apex beat, 5th interspace at
mid-clavicular line.
Heart sounds: Auricular fibrillation.
Mitral area, systolic and diastolic bruits.
Aortic area, systolic bruit.
Diastolic bruit, left of sternum, 3rd
interspace.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Case 120 (continued)

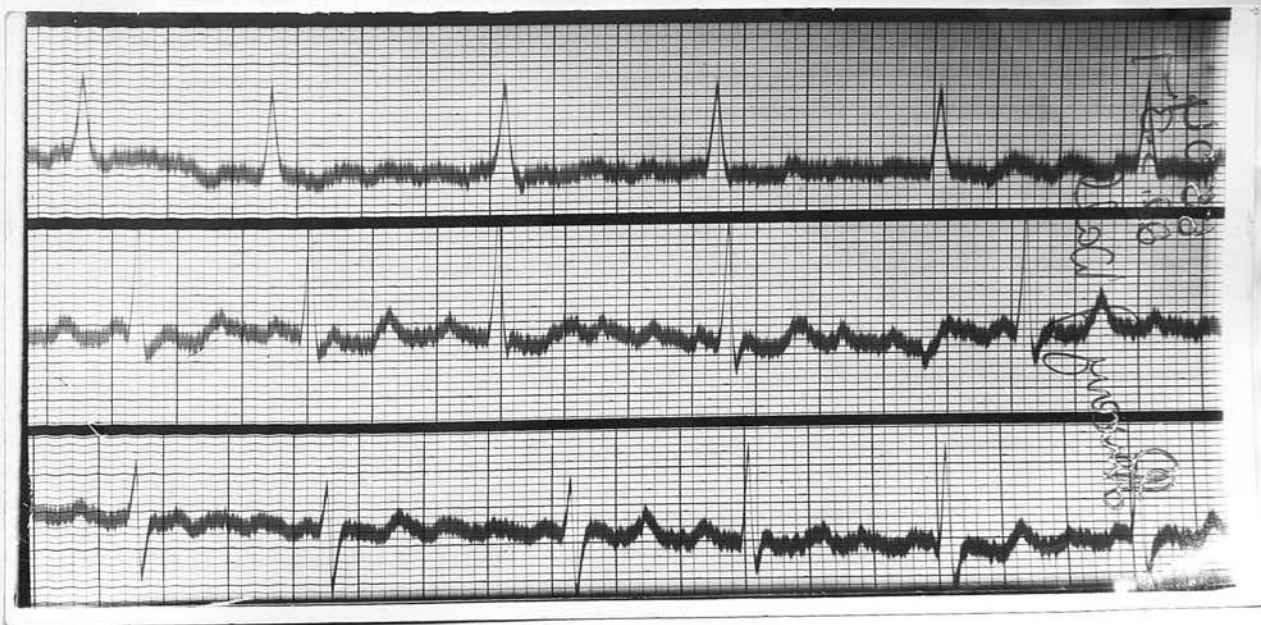
Nervous System: N.A.D.

W.R: Negative.

Electrocardiogram: P absent.
QRS Sl. slurred in leads i and ii.
Left ventricular preponderance.
Auricular fibrillation.

Diagnosis: Auricular fibrillation.
Mitral stenosis.
Aortic incompetence.
Rheumatic.
Digitalised.

Circulatory Rate: $15\frac{4}{5}$ secs.



Sex: Male. Married.
Age: 30 years.
Occupation: Truck Driver.

Present Illness: 10 weeks. Progressive and insidious onset of weakness, tiredness, loss of energy.

Previous Illness: Appendicitomy 1924.
Tubercular meningitis, operations 1932, and 1935.
Syphilis, treated with Salvarsan.

GROUP 10

3 cases

Anaemia.

Average circulatory rate 10.4 secs.
(Range 9.8 - 10.8 secs.)

Height: 5.4 ft.
Weight: 145 lb.
Temp: 98.6 F.
Pulse: 80.
Blood Pressure: 110/70.
Respiratory System: O.K.
Spleen: Enlarged.

Case 29

Sex: Male. Married.

Age: 50 years.

Occupation: Tram Driver.

History: 10 months. Dyspnoea and praecordial pain on exertion.
Giddiness, faintness, and lack of energy.

Previous Illness: Appendicectomy 1929.
Duodenal ulcer, operations 1930, and 1932.
Malaria during war service.

Habits: Alcohol nil. Tobacco moderate.

Family History: Nil relevant.

Examination: Weight 10 st. 4 lbs.

Circulatory System: Heart rate, 80; regular.
Peripheral vessels sclerosed.
Blood Pressure, 134/76.
Apex beat: 5th interspace at
mid-clavicular line.
Heart sounds: Mitral area, systolic bruit.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

W.R: Negative.

Case 29 (continued)

Blood Count: Red blood cells 4,200,000
Haemoglobin 58%
White blood cells 9,100

Electrocardiogram: T₁ flat.

Diagnosis: Myocardial Degeneration.
Anaemia.
Arterio-Sclerosis.

Circulatory Rate: 10 $\frac{3}{5}$ secs.



Case 62

Sex: Female. Married.

Age: 57 years.

Occupation: Housewife.

History: 7 years on and off. Left sub-mammary pain, brought on by wind. Occurs in bed. No relation to exertion.

12 months. Dyspnoea on exertion.

Abdominal distension and nausea after meals.

Previous Illnesses: Nil.

Family History: 1 child. Menopause aet. 49 years.

Examination: Anaemic emaciated woman.

Weight 6 st. 6 lbs.

Circulatory System: Heart rate, 86; regular.

Blood pressure 168/90.

Apex beat: 5th interspace at
mid-clavicular line.

Heart sounds: closed all areas.

Lungs: N.A.D.

Abdomen: Hard mass size of orange in right hypochondrium.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

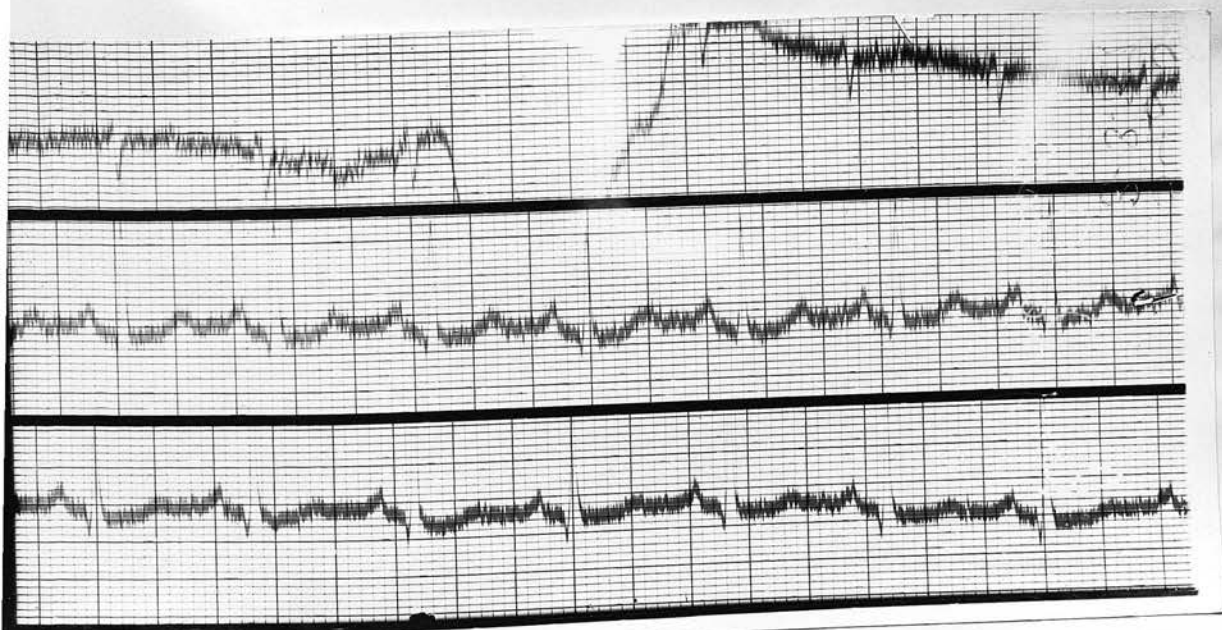
W.R: Negative.

Case 62 (continued)

Electrocardiogram: Q₃ present
Right ventricular preponderance.

Diagnosis: ? Carcinoma of Colon.
Anaemia.

Circulatory Rate: $9\frac{4}{5}$ secs.



Case 63

Sex: Female. Single.

Age: 39 years.

Occupation: Shop Assistant.

History: 2 years. Dyspnoea and palpitation on exertion.
Giddiness and exhaustion. No loss of weight.

8 weeks dysphagia (examined recently with oesophagoscope;
diagnosis, Plumer-Vincent Syndrome).

6 months Amenorrhoea.

Previous Illnesses: Nil.

Examination: Pale woman.

Weight 9 stone.

Finger nails brittle and spoon shaped.

Circulatory System: Heart rate, 82; regular.

Blood pressure 160/94.

Apex beat: not palpable.

Heart sounds: Mitral area, systolic bruit.

Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

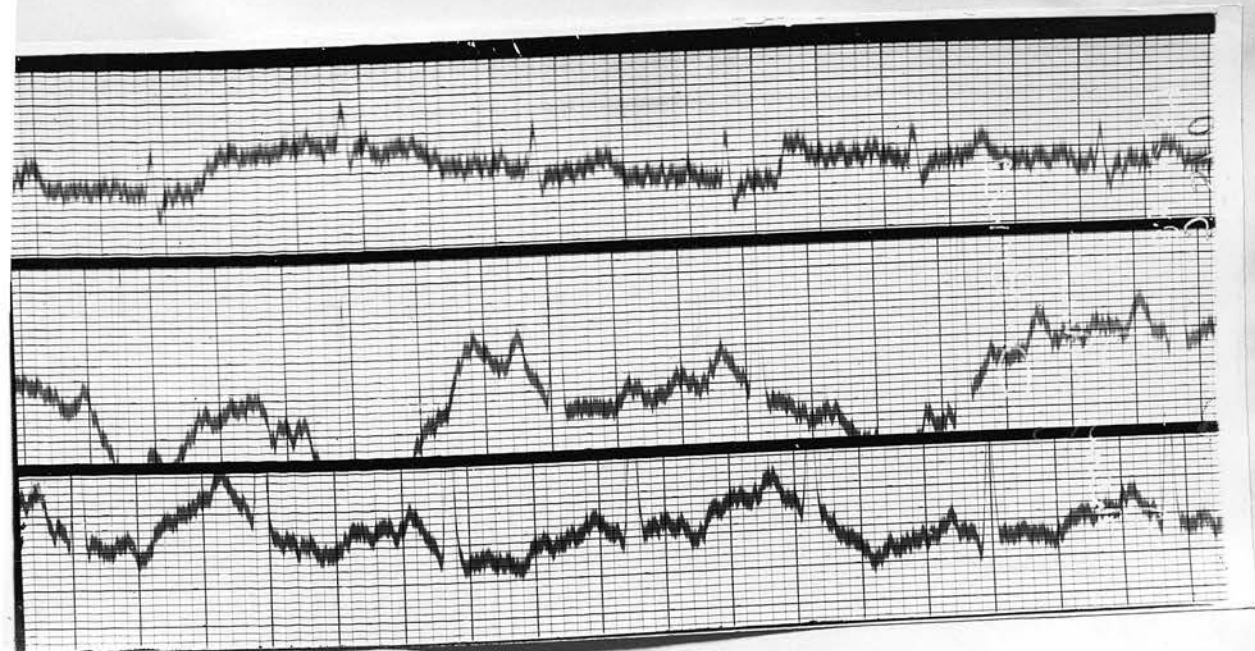
Urine: N.A.D.

Nervous System: N.A.D.

W.R: Negative.

Case 63 (continued)Electrocardiogram: P₂ large and split.T₃ inverted.Diagnosis: Microcytic Anaemia.

Plumer-Vincent Syndrome.

Circulatory Rate: $10\frac{4}{5}$ secs.

Case 49

Sex: Male, Ethnic:

Age: 17 years.

Occupation: Factory worker.

History: 12 months ago, had an attack of acute rheumatism. He was kept in bed then for 4 weeks. 6 months ago, had a similar attack, and stayed in bed 2 weeks. He then resumed his work, but a week ago, had another attack, and has been in bed since.

GROUP 11

2 cases

Previous Illnesses: Acute Rheumatism - Pyrexia.

Acute Rheumatic Endocarditis - Pyrexia.

Average circulatory rate 7.2 secs.

(Range 7 - 7.4 secs.)

Examination:

Temperature 101.2
Pulse enlarged and rapid

Circulatory System: -----

Blood pressure 115/75
Aortic valve, the first sound is
soft and muffled.
Heart sounds: Normal area, systolic murmur,
tricuspid area, systolic murmur.

Lungs: F.A.D.

Abdomen: F.A.D.

Genitals: F.A.D.

Urine: F.A.D.

Nervous System: F.A.D.

Case 49

Sex: Male. Single.

Age: 17 years.

Occupation: Factory worker.

History: 12 months ago, had an attack of 'acute rheumatism'. He was kept in bed then for 4 weeks. 6 weeks ago, had a similar attack, and stayed in bed 3 weeks. He then resumed his work, but a week ago, collapsed, and has been in bed since.

Complains now of dyspnoea on exertion, and giddiness.

Previous Illnesses: Tonsillitis many times.

Family History: Nil relevant.

Examination: Weight 9 stone.

Temperature 100°.

Tonsils enlarged and septic looking.

Circulatory System: Heart rate, 92; Sinus arrhythmia.

Blood pressure 118/76.

Apex beat, 5th interspace at
mid-clavicular line.

Heart sounds: Mitral area, systolic bruit.

Aortic area, systolic bruit.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

Case 49 (continued)W.R.: Negative.Electrocardiogram: Left ventricular preponderance.
T₂ large.Diagnosis: Acute rheumatic endocarditis.
Mitral incompetence.
Pyrexia.Circulatory Rate: $7\frac{2}{5}$ secs.

Case 92

Sex: Male. Married.

Age: 27 years.

Occupation: Seaman.

History: 4 weeks ago, pains and swelling knee joints and shoulders. Subsided in a week, to recur a few days ago. Still has slight joint pains.

Dyspnoea, palpitation and giddiness on exertion.

Previous Illnesses: Rheumatic fever aet. 15 years.
aet. 22 years.

Tonsillitis.

Malaria.

Influenza.

Habits: Alcohol and tobacco, moderate.

Examination: Weight 9 st. 2 lbs.
Temperature 99.8°.

Circulatory System: Heart rate, 100; regular.
Blood pressure 140/90.
Apex beat, 5th interspace inside
mid-clavicular line.
Heart sounds: Mitral area, diastolic bruit.
Pulmonary 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil.

Urine: N.A.D.

Nervous System: N.A.D.

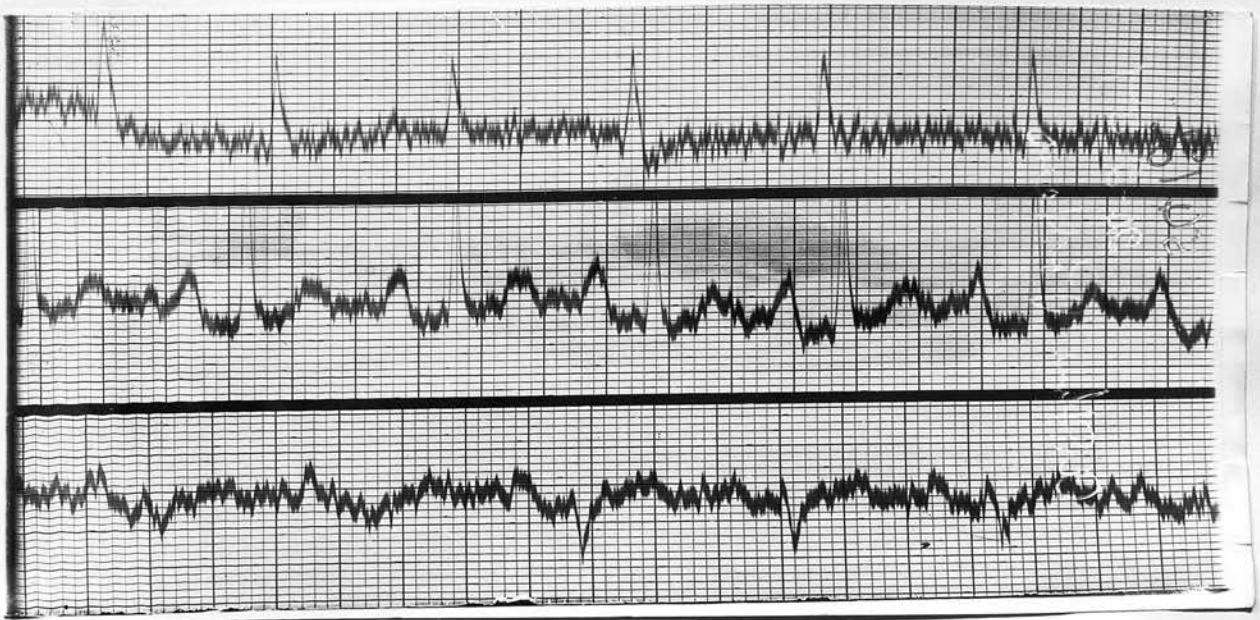
Case 92 (continued)

W.R.: Negative.

Electrocardiogram: P large. Flat topped and split.
S slurred.

Diagnosis: Mitral stenosis.
Acute rheumatism.
Pyrexia.

Circulatory Rate: 7 secs.



GROUP 12

2 cases

Hypo-Thyroidism.

Average circulatory rate 24.8 secs.

Family History: 3 children.

Occupation: Clerk.

Marital Status: Married.

Age: 35.

Circulatory System: Normal.

Image: N.A.B.

Address: N.A.B.

Education: N.A.B.

Case 37

Sex: Female. Married.

Age: 57 years.

Occupation: Housewife.

History: 3 years ago had operation for prolapsus uteri. Since then increasing dyspnoea on exertion. Continuous dull left sub-mammary pain, worse on exertion. Palpitation, giddiness, constipation, drowsiness, and increasing weight. Joint pains and increasing intolerance to cold weather.

Previous Illnesses: Nil.

Family History: 7 children; no history of miscarriages.
Menopause aet.43.

Examination: Very obese. Weight 15 stones.
Malar flush. Face puffy.
Hair and skin dry. Eyebrows thin.
Supra-clavicular padding.

Circulatory System: Heart rate, 60; regular.
Blood-pressure, 212/110.
Apex beat, not palpable.
Heart sounds: Distant.
Aortic 2nd sound accentuated.

Lungs: N.A.D.

Abdomen: N.A.D.

Oedema: Nil. Limbs 'puffy'.

Urine: N.A.D.

Case 37 (continued)

Nervous System: Deep reflexes sluggish.

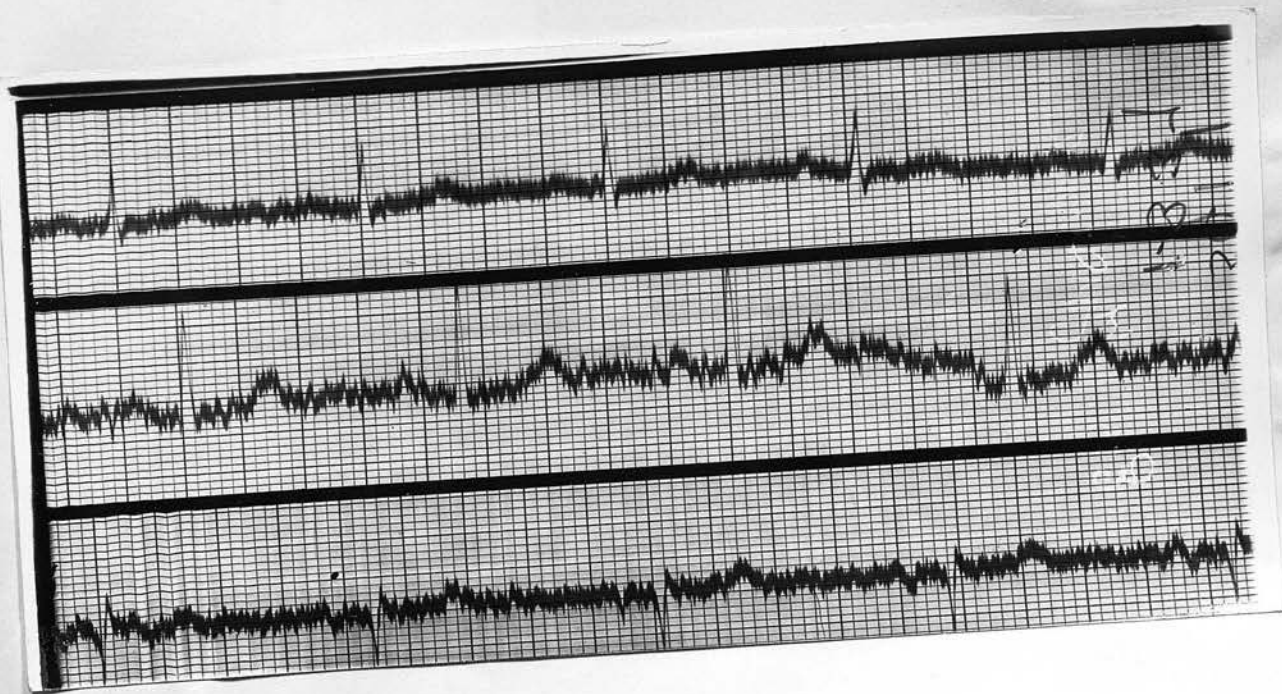
Knee Joints: Crepitus on movement.

W.R.: Negative.

Electrocardiogram: P - R interval 0.2 secs.
Left ventricular preponderance.

Diagnosis: Hyperpiesia.
Hypo-thyroidism.

Circulatory Rate: $27\frac{3}{5}$ secs.



Case 11

Sex: Male. Single.

Age: 30 years.

Occupation: Engineer.

History: 10 years. Has been subject to fainting. Has had about 6 attacks the last one being 3 weeks ago. Attack consisted of a momentary loss of consciousness. Said to go pale during attack. No muscular twitch, no loss of speech or control during an attack. He awakes in 1-2 minutes, and is able to resume his normal activities.

GROUP 13

1 case

Previous Illnesses:

Family History: Bradycardia. 15 secs.

Habit: Alcohol nil.

Tobacco: very moderate.

Examination: Fair.

Weight: 150 lbs.

Circulatory System: Heart rate, 40; regular. Blood pressure 110/70. Aortic back, 5th intercostal space. S2 normal. Heart sounds clear all over.

Urine: N.S.

Abdomen: N.S.

Reflexes: Nil.

Diagnosis: N.S.

Case 71 (continued)Nervous System: N.A.D.W.R: Negative.Electrocardiogram: P₁ flat.

Sinus bradycardia.

Diagnosis: Bradycardia, vagal.Circulatory Rate: 15 secs.