CARDIAC INFARCTION.

A STUDY OF 125 CASES.

THESIS FOR DEGREE OF M.D. OF GLASGOW UNIVERSITY

PRESENTED BY :-

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INTRODUCTION.

Coronary Occlusion is still regarded by many clinicians as an interesting but rare condition.

Five years study has convinced me that it is not uncommon. During that period I have seen 70 cases in hospital practice where no special provision is made for admission of such cases. It is a common experience of clinicians that once having seen the unusual condition they will frequently encounter it again.

The numbers in the present series are too large to be explained by such coincidence and it is possible that this disease is on the increase. The more probable explanation, however, is that special investigation has revealed its presence where it would hitherto have been overlooked.

A.W.Harrington is much impressed by its frequency in private practice.

In seventeen years (1911-1929) he saw thirty two cases whilst during the last three years he has seen twenty three.

I have had the opportunity of consulting his records and my conclusions are based on my personal experience of 70 cases augmented by the study of these additional 55 cases.

Much of the present work is corroborative and its

value lies in its scope rather than its originality. The study of coronary occlusion is comparatively young and the tempering of experience is still necessary.

I have endeavoured to summarize the main clinical features by the typical case and to describe the unusual features which may be encountered.

I have classified the cases in various groups according to their main outstanding clinical features and their ultimate prognosis.

I have found that previous classifications do not suffice and no doubt with further experience the present classification will also be incomplete.

The clinical part of the work has been under the constant supervision of A.W. Harrington. The electrocardiographic part has been entirely personal. I have taken the records and made the interpretation in every hospital case. I have been content to record my electrocardiographic findings and have refrained from theorizing on these results. Increasing experience has made the drawing of conclusions more difficult. Each case has presented its own peculiar abnormalities and made me realise more and more that the electrocardiogram is but the galvanometric record of change in relative electric potential; that the axial relationships are not always constant and that the instrument is not perfect.

For these reasons I have been hesitant to assign a special value to each minute variation in conformation or diminution in wave amplitude which may occur.

My results are in the main confirmatory but I feel they will serve as a necessary link between the apparently divergent views of others.

The sequential alterations of the electrocardiogram in coronary occlusions have been established. Daily electrocardiographyin certain of my cases has enabled me to record these frequent variations and I have described the sequence usually encountered.

I have met with most types of electrocardiographic abnormality during the course of this study and I have attempted to classify these according to their importance.

On three occasions, where single tracings were obtained, I found no abnormality of the electrocardiogram. I place less importance on this than I should have done two or three years ago. Changes may be slow in one case, rapid in another and without several tracings it is unwise to assume that they did not or will not occur.

Finally I have studied the question of relationship between electrocardiographic abnormality and the site of the cardiac infarction. My opportunities for such a study have been

exceptional.

The post mortem lesions were investigated in seventeen cases in which previous electrocardiographic tracings were obtained. This number exceeds that of any previous publications. Most of the pathological lesions secondary to coronary occlusion were encountered and the electrocardiographic changes were many and varied.

I feel that I have sufficient data to establish the fact that certain of the changes of the electrocardiogram are dependent on the site of the cardiac infarction.

Infarction of the heart from thrombotic and much more rarely from embolic occlusion of the coronary arteries has been known to a large number of pathological and clinical observers for many years. Hammer (1) as early as 1878 described a case which had been correctly diagnosed during life.

Lindsay Steven (2) in 1887 established the corelation of coronary disease with both acute infarct and chronic cardiac failure. He,however,considered embolism and multiple embolism to be the causative factor in most of the cases.

Notwithstanding a complete pathological survey of the subject by Marie (3) in 1896 the association of the clinical syndrome with pathological findings was slow. Status Anginosus was considered to be a grave complication of angina pectoris and nothing more.

Older text books paid but scant attention to the subject. In Douglas Powell's (4) article on Diseases of the Myocardium, in Allbutt's System (1909) the description of coronary thrombosis is confined almost entirely to the rapidly fatal seizure, and he states that the symptoms require "but brief notice".

It was not until the Herrick's (5) publication in 1912 that medical attention was focussed on the subject: One

type of angina was placed on a definite pathological basis and it was appreciated that the Coronary Thrombosis was not necessarily fatal. Previous workers in Europe had published similar conclusions but their work was unheeded. Following a second publication by Herrick (6) in 1918 an extensive American literature appeared. Prominent in this were articles by Levine (7 & 8) Libman (9), Wearn (10), Hamman (11), and Christian (12).

Although Clifford Albutt (13) in his Diseases of the Arteries and Angina Pectoris (1915) described the symptoms and recognised that dyspnoea and rapid fall in blood pressure are constant, that restlessness is often terrible, that the pain may be persistent for days and that a visceral perforation may be simulated, little attention was paid to the subject in this country until comparatively recently when papers have been published by A.G. Gibson (14), McNee (15), Coombs and Hadfield (16), Parkinson and Bedford (17), Cowan (18), Allan (19), and East Baine & Cary (20). The contribution of Parkinson and Bedford is an exhaustive survey of the subject.

In spite of extensive and ever growing study this important condition is still too frequently regarded as interesting but unusual.

As Allan (21) states "Disease of the Coronary Arteries is a common disease". In his study of 1000 consecutive post mortem examinations made in the Western Infirmary

over 37% presented lesions of these vessels recognisable by naked eye. About 10% died more or less directly from the effect of coronary lesions.

We are inclined to believe that coronary disease is on the increase. It is true that when one is keenly on the outlook for any condition one is more apt to recognise it and the increase may only be apparent, but the following figures carefully compiled by Dr. Steven Faulds (22) are suggestive:- In 1893/97 of 1158 Autopsies at the Glasgow Royal Infirmary coronary disease was noted in 19, while in 1923/27 of 2008 Autopsies it was noted in 86.

During the past 5 years we have been able to obtain records of 125 cases. 55 of these were seen in private practice by A.W.H, 70 were hospital cases.

Post mortem examinations were carried out in 21 of these cases and electrocardiographic examination in 66 cases.

ETIOLOGY:- There would appear to be no specific cause of coronary thrombosis. Many of the patients had little or no history of previous illness. Two main types were encountered.

1. Arterio-sclerotic, the florid hyperpietic who had previously led a strenuous life.

2. The prematurely grey asthenic.

SEX:- The condition affects males more than females. In the present series 97 were males and 28 females.

AGE:- Coronary thrombosis is a disease of the latter years of life. The youngest in our series, Case 61, was 33 years of age. Post Mortem report in this case is indefinite but is suggestive of syphilitic aortitis.

Age -	30/40	40/50	50/60	60/70	70/80	80 and over.
Private Cases -		2	26	13	8	6
Hospital Cases -	4	18	29	16	3	

The average age of Levine's(23) group of 145 cases was 57.8 but the condition occurred most frequently between the ages of 60 to 69. In the present series the greatest number of cases (55) is found in the 50/60 group.

FAMILY HISTORY:- If Cohn's (24) theory that coronary thrombosis is an accidental gross manifestation of physiological ageing of the heart is correct, it would appear that this accident is more apt to occur in certain families. Frequently we have found in the family history that near relatives have died of or been affected by similar conditions.

ANGINA PECTORIS:- A previous history of angina pectoris was obtained in 27 of 93 cases.

Cases 2, 3, 4, 5, 6, 10, 11, 15, 16, 18, 20, 22, 28, 30, 37, 50, 53, 70, 71, 74, 76, 80, 86, 88, 89, 90, 91. It is probable that many cases of angina are due to intermittent claudication with temporary ischaemia of the muscles, and finally block occurs with ischaemic necrosis, (Case No.91). The greater proportion of our cases, however, gave no history of previous angina and many were emphatic that they had no cardiac symptoms before the onset of their attack.

Syphilis was an infrequent factor. 7.4% of 1164 cases admitted to the wards had a positive Wassermann Reaction. Of 70 hospital cases the Wassermann Reaction was positive in 6. In these 6 cases, however, syphilitic involvement of the aorta was a definite factor in the production of coronary occlusion.

DIABETES;- Was not an important factor. Only 3 out of 125 cases had a previous diabetic history, 4, 65, 90.

PREVIOUS INFECTION;- There is a tendency at present to regard a focus of previous extra cardiac infection as the casual factor in the production of the actual thrombosis (Neild (25) and Peel (26).) In case 55 suppurative nasal polypus may have been the cause of embolic infarct. Preceding infection was rare and it has been our experience that inflammatory lesion elsewhere, usually phlebitic, has followed rather than preceded the cardiac infarction. (Carey F. Coombs(27).

The present series of cases may be classified under the following five groups:-

I. Sudden Death. This group is not included in our series of cases but we have met with several "brought in dead" on receiving days where the lesion has been found post mortem.

II. Sudden Onset with severe anginal pain and rapid death. Cases 5, 19, 21, 53, 61, 71, 76 and 87. (8).

III. Sudden Onset with symptoms comparatively easy to recognise and

(a) Death within days or weeks. Cases 1, 2, 3, 4, 6, 8, 9, 10, 15, 16, 18, 20, 22, 30, 35, 55, 60, 72, 73, 78, 88, 89, 91. (24).

(b) Progressive myocardial weakness and gradual circulatory failure. Cases 7, 11, 12, 13, 14, 17, 33, 38, 43, 45, 63, 67, 68, 77. (14)

(c) Recovery with persistent angina. Cases 23, 28, 35, 36, 37, 40, 41, 42, 44, 46, 48, 49, 50, 51, 54. (15).

(d) Apparent complete recovery. Cases 24, 25, 27, 29, 31, 32, 39, 40, 47, 52, 58, 59, 62, 65, 66, 69, 74, 75, 84. (19).

IV. Sudden Onset but symptoms referred to some other Viscus.

(a) Abdominal Type. Cases 9, 15, 16, 18, 27, 30, 31, 40, 54, 56, 57, 61, 62, 63, 82, 84, 88.

(b) Respiratory Type. Cases 8, 17, 24, 31, 44, 48, 50, 51, 58, 72, 79, 81.

V. No history of sudden catastrophe but myocardial degeneration and gradual cardiac failure.

Coronary Thrombosis, unlike Angina Pectoris, commonly occurs at night while the patient is at rest. In twenty one

of our cases, however, the onset of symptoms occurred when the patient was at or returning from work. Cases Nos. 1, 5, 10, 21, 23, 29, 32, 34, 35, 40, 44, 48, 49, 54, 57, 59, 67, 73, 74, 79, 85.

Pain is a commanding feature in most cases. It occurs suddenly and is often of great and even agonising severity. Patients who had previously anginal attacks stated that "This was something quite different and more severe". It is usually substernal in situation but may radiate upwards into the neck, across the chest into one or both arms, and downwards into the abdomen. It is gripping or vice like; a common description being that "the chest seems to be crushed in a vice". Rest does not bring relief; pain may persist for hours and even days.

Amyl Nitrite is ineffective. (One of our cases, No.23, however, obtained relief from pain when given Amyl Nitrite by the House Physician).

The patient is usually restless, constantly tossing and turning and angor animi may be marked.

Dyspnoea is frequent. In some cases the attack may be ushered in by intense dyspnoea. Cases Nos. 4, 34, 50, 65,92.

Gastric disturbance occurred in most of our cases. Vomiting may be persistent but gradually subsides if the patient survives. The tongue rapidly becomes furred and the breath offensive.

Physical examination:- The facies is that of shock. It is ashen grey and the lips are a little cyanosed. Sweating

may be profuse. The pulse is usually frequent, small and soft.

We have noted most types of irregularity, auricular fibrillation, Cases 12, 38, auricular flutter, Cases 5, 68; extra systole, Cases 2, 57; full heart block, Cases 29, 55; partial heart block, Cases 4, 81; pulsus alternans, Cases 30, 86. This is in marked contrast to ordinary angina in which during the attack the pulse is little affected. Clifford Albutt (13) states that in angina "Amid the agitation of patient and attendants the heart, assumed to be the protagonist in the conflict, often seems to be the one impassive actor!

Blood pressure generally falls rapidly, the fall being more marked in systolic, and in one of our cases pressure was as low as 65/45 m.m. Hg. Case No. 67. This fall is best appreciated where patient's pressure has been known before the attack, Case 91. The hyperpietic who previously had blood pressure of over 200 m.m. Hg. may still have an apparently normal systolic reading. The relatively small pulse pressure is of value in such cases. Case 36. The blood pressure does not fall immediately in all cases. Chart taken from one of our hospital cases shows a delay in fall for at least 24 hours after the attack. Case 27.

HEART SOUNDS:- The heart sounds are as a rule peculiarly soft and distant. Gallop rhythm was noted in several cases.

PERICARDITIS:- Pericarditis varying from evanescent fine localised friction to persistent coarse generalised friction was noted in 20 of our cases. Effusion was present in two of A.W.H's private cases.

TEMPERATURE :- Slight irregular pyrexia is often present in the first few days of illness. In a number of our cases temperature has remained normal or sub-normal during the whole course of illness. In Case 24, a good recovery, pyrexia continued for several weeks.

Dullness to percussion and moist rale at the bases of both lungs were frequently present. The association of chest pain, lung dullness and fever led to the diagnosis of pneumonia in two cases. (Cases Nos. 24, 31.) Acute oedema of the lungs is not infrequent. (Cases Nos. 1, 62, 77).

URINE:- Occasionally albuminuria was present. Glycosuria was found in 3 of our cases. Urobilin and bile were present in 2 of the hospital cases.

Thrombosis was found in 5 cases and in our experience this complication added to the gravity of the prognosis. The commonest site of thrombosis was the femoral vein. (Cases Nos 17,24,30,67, 83.) Jugular Vein thrombosis was present in one case (Case No.17).

EMBOLISM:- Infarction of lung is common. Characteristic signs and symptoms were present in 7 cases. Cases Nos. 17, 50, 74, 77, 79, 81, 83.

In one of our cases No.30, the terminal signs and

symptoms were suggestive of cerebral embolism.

Embolism of right femoral artery was found post mortem in Case No. 17.

Slight Jaundice was present in 2 cases.

LEUCOCYTOSIS:- Leucocytosis was present in all acute cases examined except Cases Nos. 1, 11, 38, 43.

The average leucocyte count was 12000 per cmm. The highest count recorded was 24,600 per cmm. (Case No.30).

SUDDEN ONSET BUT SYMPTOMS REFERRED TO SOME OTHER VISCUS.

Abdominal Type :- It is frequently forgotten that gastric discomfort may be the outstanding and for a time the only symptom of cardiac disease. Dr. A. Bruce McLean (28) states that about 10% of cases sent to him for radiological examination of the gastro-intestinal tract have either clinical or X-ray evidence of cardiac disease. Case 56, which showed electro-cardiographic tracing of the type associated with cardiac infarction was referred by A.B. McL. In coronary thrombosis sickness and vomiting is an almost constant feature and when this is associated with abdominal pain the possibility of error in diagnosis can be readily understood. We have met with three types of abdominal complex in our series.

1. Flatulent Dyspepsia :- In this type of which cases 30, 31 and 57 are good examples the patient had been troubled for some time with flatulent dyspepsia. Although in these cases there was definite clinical evidence of coronary thrombosis the patients persisted that their only discomfort was gastric.

2. Acute Abdomen :- Although pain is usually sub-sternal at the onset it tends to radiate both upwards to the shoulders and arms and downwards to the abdomen.

In nine cases acute abdominal pain heralded the onset. Cases No. 21, 22, 27, 40, 45, 57, 81, 84, 87.

The provisional diagnosis was incorrect in all these cases. Laparotomy for acute abdomen was performed in 5 of 70 hospital cases. Cases 8, 18, 27, 54, 62.

3. Gall Bladder Colic :- Anderson (52) has shown that the abdominal type of cardiac infarction may closely simulate gall bladder colic. An operation was performed on Case No. 54 after an attack of what was thought to be typical gall bladder colic. No abnormality of the gall bladder was detected.

The association of gall stones with cardiac disease, Case No. 15, is well recognised and this adds further to the difficulty of differential diagnosis. We are of opinion, however, that the difficulty is not so great when the possibility of coronary thrombosis is kept in mind.

Respiratory Type :- Hypostasis of the lung bases was present in most cases. In two cases, 24 and 31, the sudden pain in the chest, dyspnoea, dullness and rale at the bases of lungs led to the provisional diagnosis of pneumonia. In cases, 48, 50, 53, 58, pain in the left side of the chest and slight fever were incorrectly assumed to be due to pleurisy.

The development of friction rub following infarction of lung was wrongly interpreted. Case No. 53.

In cases 17 and 80 the main clinical features were almost entirely respiratory and led to a tentative diagnosis of tumour of lung.

A similar misinterpretation of signs was made in one of Alice J. Marshall's (29) cases.

PROGNOSIS:- There is no disease in which prognosis is more uncertain. Recovery is common and may take place notwithstanding the occurrence of complications. The continuance or absence of pyrexia is not helpful. Continued low blood pressure need not be of grave import. Pulsus alternans was detected in one case which made good recovery. Pericarditis and even pericardial effusion were present in cases which recovered. Abnormalities of cardiac rhythm, whilst adding to the gravity of prognosis did not prevent recovery. In Case No. 38 in which auricular fibrillation persisted recovery took place. Temporary heart block was present in Case No. 29 who made a good recovery. Persistent full heart block was present in Case No. 56 who died of pneumonia several months after coronary lesion. Auricular flutter and full heart block was present in Case No. 68 who lived for fully a year.

It has been our experience that the development of thrombosis elsewhere adds to the gravity of prognosis. We cannot express any definite conclusion on the value of high leucocytosis; in our limited experience it has not been helpful.

Prognosis must always be guarded. It cannot be too strongly emphasised that death may occur suddenly at any time even in a patient who is apparently doing well. Cases Nos. 3 and 16.

ELECTROCARDIOGRAPHY IN CORONARY OCCLUSION.

The introduction in 1903 by Einthoven of the string galvanometer which consisted of a very delicate thread of platinum or silvered glass suspended between the poles of a stationary electro magnet and deflected by the most minute heart curves was an epoch making event in the study of heart disease.

The electrocardiograph, based on the string galvanometer in the hands of Lewis, Wenckeback and others soon became of extreme value not only in experimental research but also as an aid to diagnosis.

By its means the study of disordered cardiac mechanism was placed upon a rational basis, the arrhythmias were now classified and their significance gauged.

One of the most difficult problems of medicine is the detection of early evidence of myocardial disease and within recent years most electrocardiographic research has been directed along this channel.

In 1910 Eppinger and Rothberger (30) showed that characteristic electrocardiograms were obtained in lesions of one or other main branch of the bundle of His.

Eppinger and Stoerk (31) diagnosed during life a lesion of the right branch of the bundle which was subsequently confirmed post mortem.

Lewis (32) has summarized the typical features of the branch bundle block electrocardiogram.

1. The chief deflections are greater than normal in amplitude.
2. The initial phases have an unusual duration.
3. The chief and final deflections are oppositely divided; the course is broadly diphasic.

Cowan and Bramwell (33) in a study of 24 cases of bundle-branch block came to the conclusion that its presence indicates a definite myocardial lesion but if unaccompanied by signs of cardiac insufficiency is not necessarily of grave prognostic significance.

From the gross changes of bundle branch lesion the next step was the study of lesser variations in the electrocardiogram.

Oppenheim and Rothschild (34) suggested that lesions of the arborisation of the bundle produced typical electrocardiographic changes. Arborisation block was evidenced by low voltage QRS with broadening and notching or splintering.

Lewis (35) did not accept this view that this type of electrocardiogram need be associated with a particular lesion of the left Purkinje network.

Wilson and Herdman (36) regarded a number of these tracings as indicative of incomplete heart block.

All were agreed that these changes were definite evidence of myocardial disease.

In 1919 Herrick (37) described Inversion of T wave in Leads I and II in cases of Coronary Occlusion. Similar changes had been found by Smith (38) after experimental ligation of coronary artery in dogs. Pardee (39) in 1920 was the first to record a change in the electrocardiogram occurring immediately after an occlusion. He states "curves of this sort are obtained in experimental work, also if the records are taken during the first few hours after tying off a coronary branch. The typical features of these records is that the ST interval does not start from the base line of the record but comes off from the QRS group at some point above the base line".

Wearn (40) described diminution of amplitude of the electrocardiographic waves in two patients suffering from coronary thrombosis. Cowan (41) found distinct changes in the electrocardiogram of a patient suffering from progressive myocardial degeneration examined over a period of years. Describing these changes he states "In 1922 it was abnormal, PR interval being long and the QRST interval excessive. In 1924 there was little change but the deflections generally were larger. In 1925 the electrocardiographic complex had altered. The deflections were still larger and the QRST had altered, the QRS interval measuring 0·18 to 0·20 seconds while SIII was succeeded by a comparatively large deflection with a thickened downstroke which merged into T".

Parkinson and Bedford (42) recorded changes in 28 cases. Repeated electrocardiograms were taken. They found that electrocardiograms obtained within a week of sudden occlusion showed RT interval either above or below the isoelectric level - the extent of RT deviation was usually about 1-3 mms., but occasionally reached 6 mms. The RT elevation was frequently flat topped or plateau like, but sometimes continued to rise to form a summit. These changes were best seen in Leads I and III and were constantly opposite in these leads - elevation of RT in I being accomplished by depression of RT in III and vice versa. RT deviation was occasionally present in one lead only.

T waves were frequently absent and when they returned they were in opposite direction of RT deviation. When RT deviation had disappeared the RT preceding the T wave usually exhibited a noticeable upward tendency. This last phenomenon has been described and designated the Coronary T wave by Pardee (43) and Cove Plane T by Oppenheim and Rothschild (44).

Parkinson and Bedford further demonstrated that tracings obtained a few weeks after acute onset conformed to one of two types. These they termed T I type or T III type according to the presence of T inversion in I or III.

Willius (45) in his study of coronary occlusion divided the abnormalities into ten types :-

(1) Inversion of T wave.
(2) Pardee T wave.
(3) Incomplete Block.
(4) Complete Block.
(5) Bundle Branch Block.
(6) Auricular Fibrillation.
(7) Inversion of P.wave in III.
(8) Splintering in 2 leads.
(9) Prolongation of QRS.
(10) Prolongation of QRST.

He found it unusual for electrocardiogram to return to normal lines in less than six months to one year.

Gilchrist and Ritchie (46) in a Critical Survey of 148 cases in which serial electrocardiograms had been taken, concluded that"even although control electrocardiograms and post mortem confirmation of the clinical diagnosis be lacking these sequential alterations of the RT segment and of T occurring in the course of a short period of time are strong presumptive evidence of myocardial infaretion".

PRE INFARCTION ELECTROCARDIOGRAM.

In 5 cases I obtained electrocardiograms before the onset of characteristic symptoms of infarction.

In Case No. 71 three days before terminal infarct - the tracing showed no abnormality.

In Case No. 39 tracing one month before infarct - the electrocardiogram was of the left axis deviation type. There was no abnormality of RT or T wave.
A tracing taken one month after infarct was similar except for slight diminution of amplitude of the waves.
In three of these cases there was abnormality of Lead III.

In Case No. 31 there was diminution of amplitude of QRS with slightly elevated RT.

In Cases Nos. 30 and 53 there was slight shouldering of RT with inversion of T wave.

I have found this type of abnormality in a large series of patients suffering from typical angina of effort.

In Cases Nos. 30 and 31 typical sequential alterations of Lead III were recorded during the subsequent attack.

In four of our five cases the electrocardiogram indicated

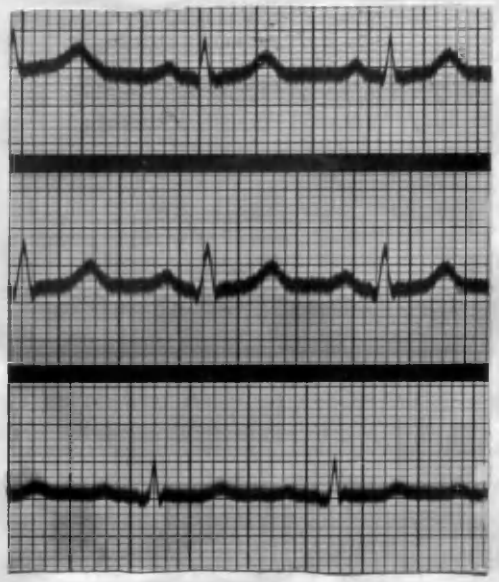
abnormality before the onset of symptoms.
In three of these the abnormality was in Lead III.
Whilst abnormality of this Lead is not uncommon in many apparently normal individuals - the presence of inverted T wave with preceding upward shouldering even if found in this one Lead should be regarded as possible indication of coronary involvement.

It is interesting to note that in Case No. 31 the electrocardiogram was suggestive of cardiac infaraction several days before the onset of typical symptoms.

Case No. 71.

3 days before terminal infarction.

20.5.31

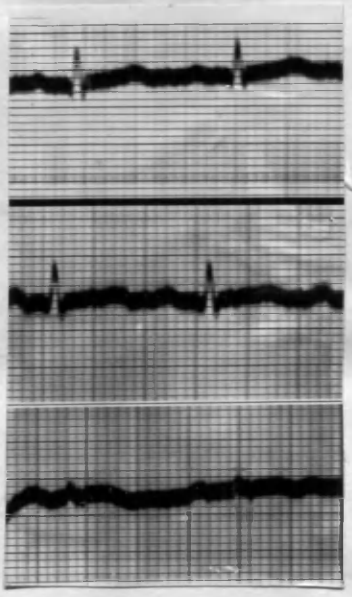


Apart from slight diminution of amplitude the electrocardiogram is normal.

Case. No. 31.

Before Onset

25.3.30.



T upright in all leads.

PR - ·16.

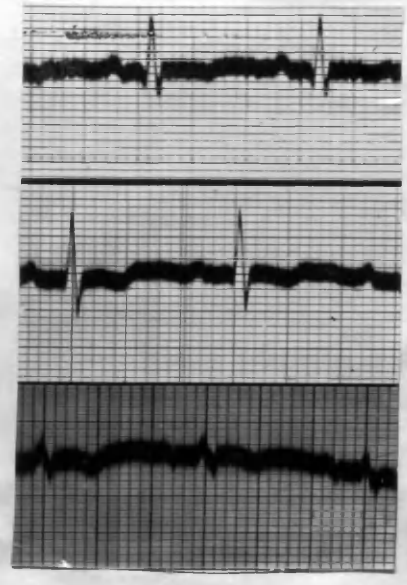
QRST = .36

QRS in III - small amplitude.

Case No. 31.

9.4.30.

2 Days after onset



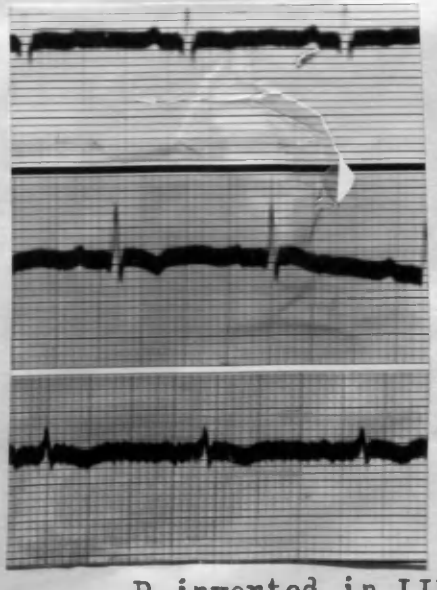
P inverted in III.

T inverted in III - diphasic in I & II.

QRST = ·28 - ·32

14.4.30

7 Days after onset

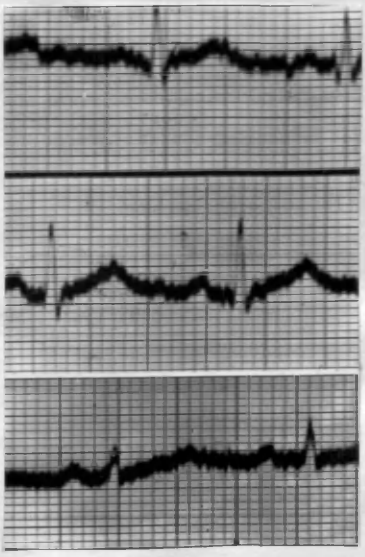


P inverted in III

T inverted in III and II.

Case No. 31.

4 months after onset.

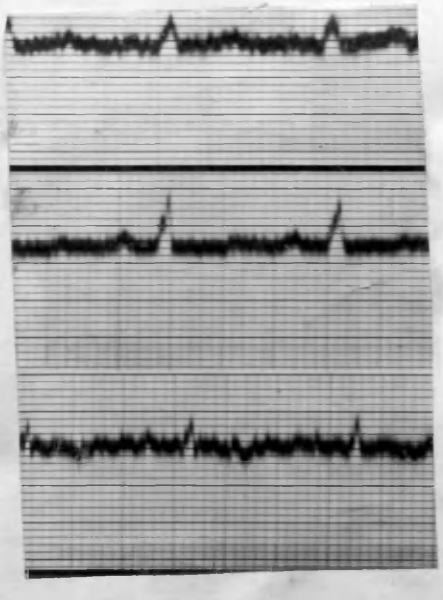


Lead III still of diminished amplitude - otherwise no abnormality.

Case No. 30.

Before Onset

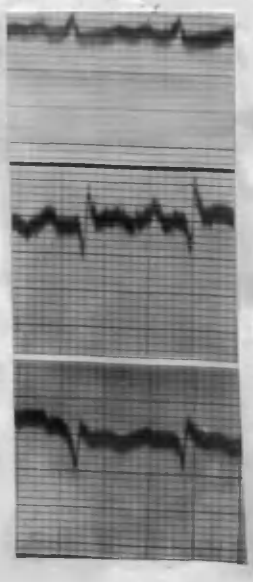
18.3.30



QRS splintering.
T wave inversion in III.

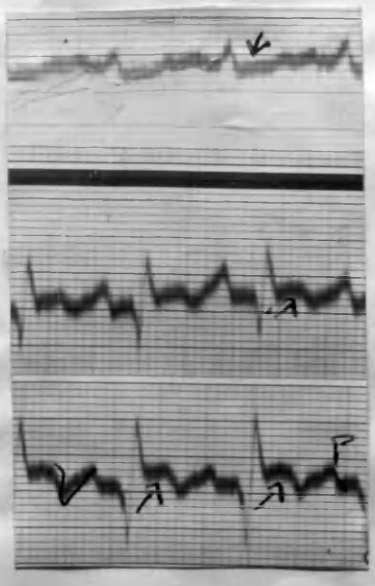
After Onset
1.5.30.

3.5.30



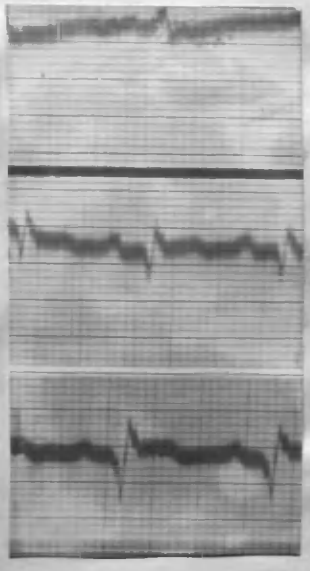
Increased amplitude
of Q wave in II and III
RT elevation in II & III.

4.5.30



Inverted T In III
RT elevation in
II and III.

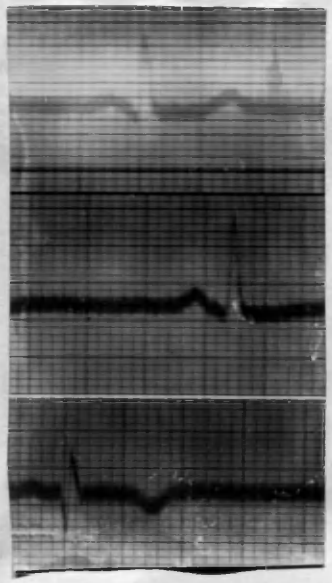
7.5.30



T wave no longer
inverted in III.

Case No. 53.

5 days before Terminal Infarct.

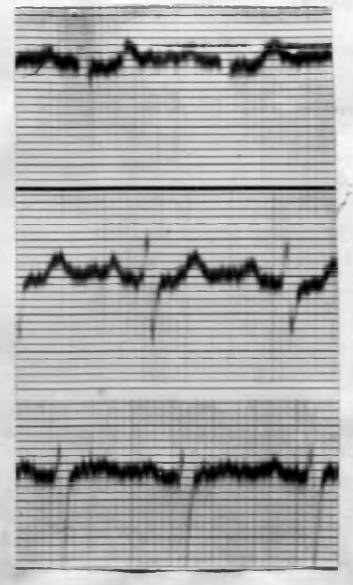


RT elevation with inverted T in I III.

Cowan Wave present.

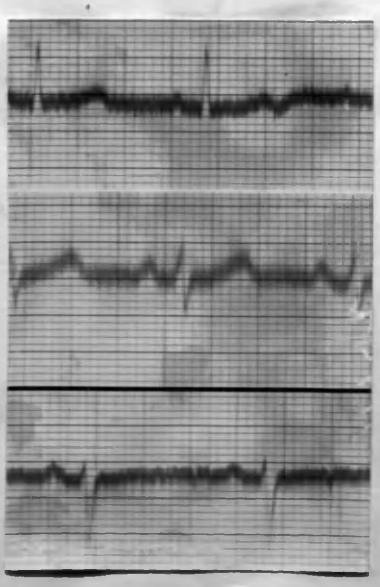
Case No. 39.

One month before infarct.



Left Ventricular Preponderance.

One month after infarct



Slight diminution in amplitude of QRS in all Leads. No T wave inversion.

ELECTROCARDIOGRAM DURING ACUTE PHASE.

Tracings were obtained in 36 of our cases during the acute stage of illness, (Cases No. 1, 2, 3, 4, 5, 6, 8, 9, 10, 13, 14, 16, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 42, 43, 54, 55, 66, 67, 68, 92, 93.)

The changes in the electrocardiogram varied.

(1) Axis Deviation.

(2) Sequential changes of Parkinson and Bedford.

(3) Increased amplitude of QIII (Cowan).

(4) Broadening and Splintering of QRS.

(5) Prolongation of QRST.

(6) Inversion of P III.

(7) Abnormality of Rhythm.

(8) Normal Electrocardiogram.

I. **AXIS DEVIATION.**

Left axis deviation was present in almost every case. This was in agreement with pathological findings. In every case examined post mortem there was enlargement, in some cases very marked, of the left ventricle. In case 24 the electrocardiogram was of the right axis deviation type until six months after attack when it became left axis deviation. X-ray examination of this case in the early stages showed no enlargement of the heart downwards and to the left.

2. 25 serial tracings were obtained. 15 of these were of the sequential type described by Parkinson and Bedford.
7 were of the TI type (Cases No. 1, 2, 3, 23, 24, 25, 34).
9 were of the TIII type (Cases No. 15, 16, 23, 27, 29, 30, 31, 32, 93).
Case No. 23 showed sequential alterations characteristic of both types.
In our cases there would appear to be a definite order of change - we shall describe those changes as seen in TI type

(1) Elevation of RT I.
(2) Elevation of RT I and II.

(3) Diphasic T I.
(4) Inversion of T I.
(5) Diphasic T II.
(6) Inversion of T I and T II
(7) Gradually increasing inversion of T I.

(8) Diminution of Inverted T II.
(9) T II absent, amplitude of T I diminished.
(10) Upright T II.
(11) T I absent
and occasionally
(12) Return to normal - Upright T I.

RT DEVIATION.

RT deviation was present in 22 of our cases. RT I was elevated in 11 of these and RT III in 11. (RT I Cases No. 1, 2, 3, 4, 5, 8, 23, 24, 26) (RT III Cases No. 13, 15, 16, 27, 28, 29, 30, 31, 33, 93, 93).

Oppenheim (47) has described elevation of RT six hours after obstruction of coronary artery. In two of our cases it was present within 12 hours. This elevation varied from a slight upward rounding RT (or more correctly ST) to a marked elevation of over 6 mm. (Case No. 29). This case made a good recovery and I have not found the extent of RT deviation to be of any prognostic value (Ernstene A.C. 48). The gradual inversion of T wave usually commenced within 2 to 13 days.

INVERSION OF T WAVE.

Gradual inversion of T wave commences between the third and thirteenth day. In Case No. 34 T I and T II inverted 36 hours after onset. In Case No. 6 RT remained elevated for 18 days after which the typical sequential alterations of T wave occurred. In Cases No. 28, 33 and 38 RT was still elevated 4 to 6 weeks after onset, and in Cases No. 41, to 50 five to six months after onset.

In none of our fatal cases did T II become inverted. In one of these, daily tracings were obtained for fully a month.

In 3 of these cases inversion of T wave was succeeded by a further elevation of RT. It may be that inversion of TII is evidence of attempt at recovery and that the return of RT indicates further spread of infarction.

RETURN TO NORMAL.

The return to upright T wave may occur within six months. Typical abnormality may persist, however, after fully a year.

In Case No. 23, however, TII became upright in 7 days and TIII in 8 days.

Case No. 24 TI, TII were still inverted after one month, but had both become upright in 6 months.

Case No. 25, TI and TIII were still inverted 10 weeks after.

Case No. 27, TII and TIII were still inverted 5 weeks after.

Case No. 32, TII and TIII were still inverted 6 weeks after.

Case No. 40, TII and TIII were still inverted 3 weeks after, TII upright 11 weeks after.

Case No. 46, TIII was inverted 3 weeks after.

Case No. 62, TIII was inverted 22 days after, upright 2 months after.

Case No. 29, TIII was still inverted 6 months after.

Case No. 47, TI inverted 5 months after.

Case No. 23, TII became upright for the second time after 5 months. TI was still inverted 15 months after.

I have described the typical changes but in some of our cases, as already indicated, sequential alterations may occur rapidly from day to day (Case No. 23).

Variations from bundle branch block - sharp inversion of T wave - almost normal electrocardiogram may take place within a period of days (Case No. 16).

To draw conclusions from a single electrocardiogram may lead to gross error.

Whilst the sequential alterations are the most dramatic electrocardiographic changes met with in coronary occlusion they are unfortunately from the diagnostic point of view, not always encountered and one has frequently to depend on other electrocardiographic evidences of myocardial disease to confirm the diagnosis of coronary involvement.

None of the changes which I now describe are characteristic of sudden coronary occlusion, but rather of chronic myocarditis secondary to progressive coronary obstruction. Not only do serial tracings in these cases show no sequential alterations but the abnormality is persistent or gradually progressive. Once only have I seen a broadened and splintered QRS return

to normal. From a study of over 3000 tracings in which I have but rarely found these electrocardiographic changes in other types of myocardial disease (and these I have detailed below) I have been convinced that they are positive evidence of interference with coronary circulation.

3. Cowan recorded gradual increase in amplitude of QIII in progressive myocardial fibrosis.

This abnormality occurred in 14 of our tracings (Cases No. 2, 13, 15, 16, 27, 28, 29, 30, 32, 33, 34, 36, 37, 41) and was associated with inversion of PIII in 4 of our cases (Cases No. 28, 33, 36, 37) where it occurred sequential alterations were not obtained even when repeated tracings were taken.

I have found this to be the most common change in the electrocardiograms of over 150 cases of angina pectoris and have also noted its presence in 5 cases of acute rheumatism and 11 cases of pericarditis.

4. BROADENED AND SPLINTERED QRS.

Abnormality of QRS was a frequent occurrence. Splintering and broadening was the most common. In two cases (Cases No. 10 and 35) the change in QRS was suggestive of Right Bundle Branch Block Type. As with other features

of electrocardiogram the coronary occlusion QRS variation may be transient. In Case No. 15 QRS changed from day to day.

In three of my cases the main abnormality was in the QRS complex. QRS was broadened and splintered in all these cases. In two there was broadening of the RS interval and slurring of the S wave. A similar type of abnormality was met with as a transient feature in the sequential alterations of QRST complex in Case No. 15. In these other cases, however, this abnormality still persisted months after.

In the third case of this group there was prolongation and notching of QRS complex of the type associated with Aborisation Block by Oppenheim and Rothschild.

5. PROLONGATION OF QRST.

There is still much difference of opinion with regard to the significance of prolongation of QRST. Pardee (49) suggests that "For heart rates of 70 or over the maximum normal duration of systole be set at .40 seconds measured from the beginning of the QRS group to the end of the T wave. In some of our cases QRST was at first definitely prolonged beyond .46 (Case No. 23) and diminished with improvement to the patient.

6. INVERSION OF P III.

In five of our cases in the present Series PIII has been inverted. In two of these the inversion was associated with persistent elevation of RTIII. The amplitude of QIII was increased in all these cases. This inversion may be transitory and I have seen PIII vary; upright, diphasic and inverted in a single record (Case No. 37). In 11 cases of acute pericarditis the electrocardiogram has shows this abnormality.

Willius (50) has recorded this change and is inclined to regard it as evidence of coronary sclerosis.

7. ABNORMALITY OF RHYTHM.

I have seen most types of abnormality of rhythm. In some of the cases this abnormality was the striking feature of the electrocardiogram.

Case No. 70 in which there was a history of repeated attacks of persistent anginoid pain showed paroxysmal ventricular tachycardia.

In Case No. 56 full heart block persisted from the onset of attack.

The most frequent type of abnormal rhythm was that due to ventricular extra systole and both during the acute stage and in the stage of recovery. Parkinson and Bedford (42)

were impressed by this frequency which appears to be more than accidental.

8. NORMAL ELECTROCARDIOGRAM.

In two of my cases which recovered, No. 54 and 66, and in one which died, No. 71, the electrocardiogram showed no marked abnormality. Similar findings have been recorded by Wearn (51) Anderson (52) and Campbell (53).

In the absence of serial tracings I cannot draw any definite conclusion from these. Theoretically it would be possible that a small infarction involving a "quiet" area of the heart need not cause electrocardiographic alteration. (Craib 54).

Recent work by Wolferth and Clackwood (55) would suggest that the introduction of a fourth lead may minimise the possibility of error.

SMALL AMPLITUDE QRS.

The amplitude of QRS varies considerably in normal individuals. The upper limit of normal has been fixed at 16 mms. and the lower at 7 mms. The possibilities of error in assessing wave amplitudes are familiar to anyone acquainted with the technique of electrocardiography.

During marked circulatory failure it is common to find the amplitude much diminished and with circulatory improvement a gradual increase of amplitude takes place. Wearn (40) has recorded diminished amplitude of QRS in Coronary occlusion.

I have found this diminution of amplitude most frequently in those tracings where sequential alterations occurred and diminution was most marked in that lead which first showed elevation of the RT interval i.e. in Lead I in the TI type of electrocardiogram and in Lead III in the TIII type.

In one of my cases two years after what appears to have been a coronary occlusion and a few days before sudden death the amplitude of QRS was much diminished in all leads - (maximum 3 mms.).

ELECTROCARDIOGRAPHY IN THE RECOVERY STAGE.

In those cases where tracings could only be obtained some time after the onset of illness the electrocardiographic changes resembled one or other of the types encountered where serial tracings were recorded.

1. Left axis was only abnormality in Cases No. 52 and 82.

2. Cases No. 44, 45 and 47 had inversion of TI. Case No. 51 showed inversion of TII and TIII and in Cases No. 46 48 and 49 TIII was inverted.
   RT III in Case No. 50 was elevated 5 months after the attack.

3. QIII was increased in amplitude in Case No. 50.

4. QRS was broadened and splintered in Case No. 85.

5. PIII was inverted in Cases No. 50 and 83.

6. Auricular Fibrillation was present in Cases No. 38 and 43 In the latter case fibrillation was first recorded two years after the onset. Recurring ventricular extra systoles were found in Case No. 69 and paroxysmal ventricular tachycardia in Case No. 70.

ABNORMAL QRS IN ACUTE PERICARDITIS.

For some time after Pardee's description of RT elevation in coronary occlusion this electrocardiographic phenomenon was thought to be peculiar to the condition. Since then it has been noted during acute rheumatism, rheumatic carditis and in pericarditis with effusion, uraemia and lobar pneumonia. A possible explanation of this distortion in these diseases would be (a) localised inflammatory myocarditis, or (b) localised anoxaemia of cardiac muscle.

In eleven cases of pericarditis inversion of P wave was present.

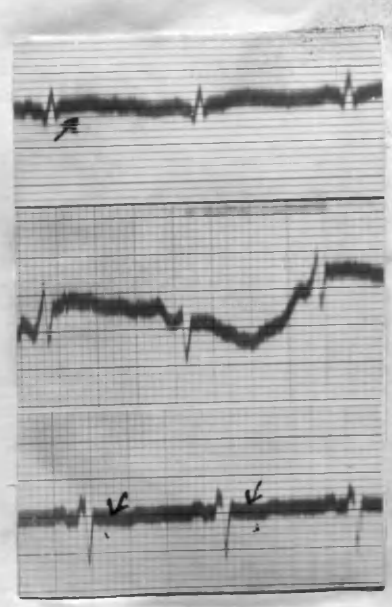
In three cases I have found abnormalities of electrocardiogram similar to those encountered in coronary occlusion. In one case there was small amplitude QRS and elevation of RT in I. Three months later P wave was inverted in III. In the second case RTI elevation was later succeeded by RTI depression and still later by inversion by TII and TIII. In the third case there was inversion of T wave in all leads.
Three months later T was upright in all leads.

SEQUENTIAL ELECTROCARDIOGRAMS.

T I Type.

Case No. 23.

9.3.29.



PR = •12 secs.

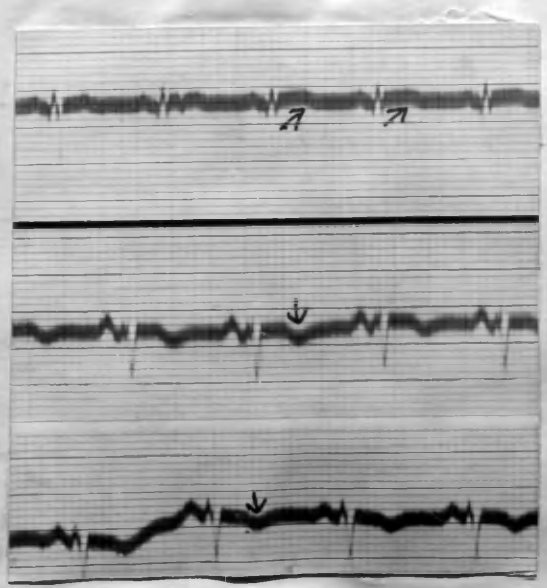
P is notched in III

Left ventricular preponderance

High RT interval in I

R notched in III.

10.3.29.



PR = •14 secs.

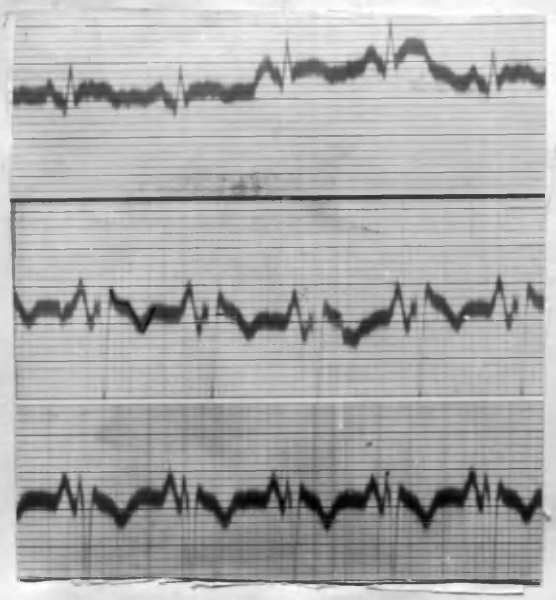
T inverted in II and III.

QRST = .36 secs.

R notched in II

Case No. 23

11. 3. 29



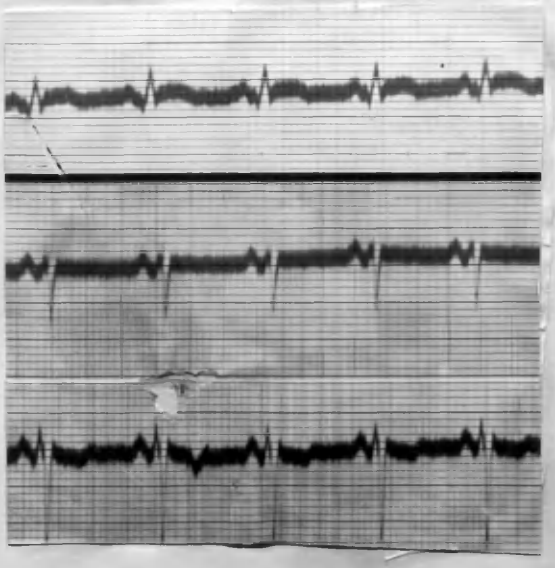
RT elevated in I

PR = .14

QRST = .36

T inverted in II and III

12. 3. 29.

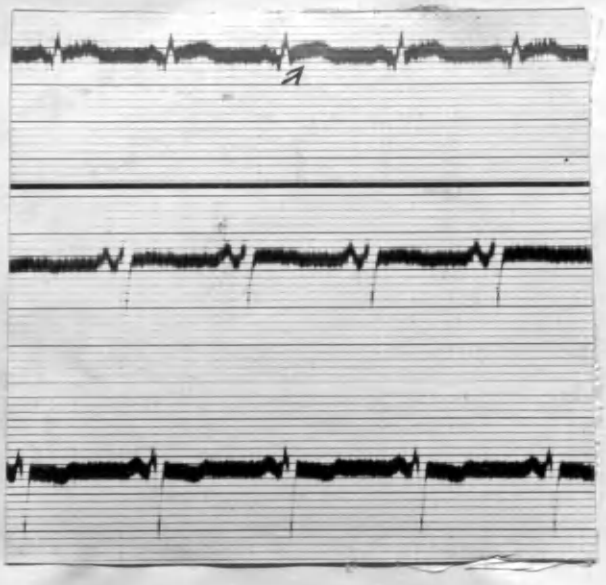


T wave absent in II

T wave inverted in III but less marked than on 11/3/29,

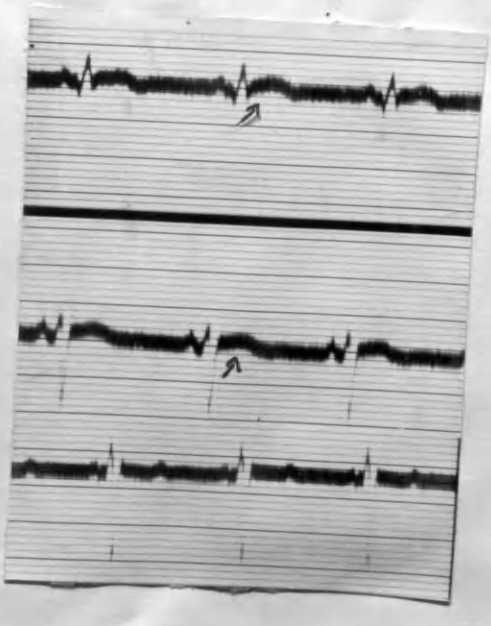
RT in I plateau type.

13. 3. 29



Gradual diminution of T wave in III
RT plateauing in I.

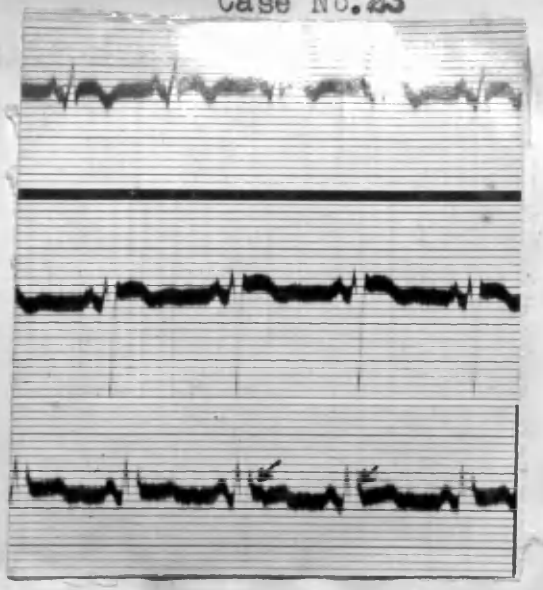
14. 3. 29.



Elevated RT in I and II
T wave upright in III

Case No. 23

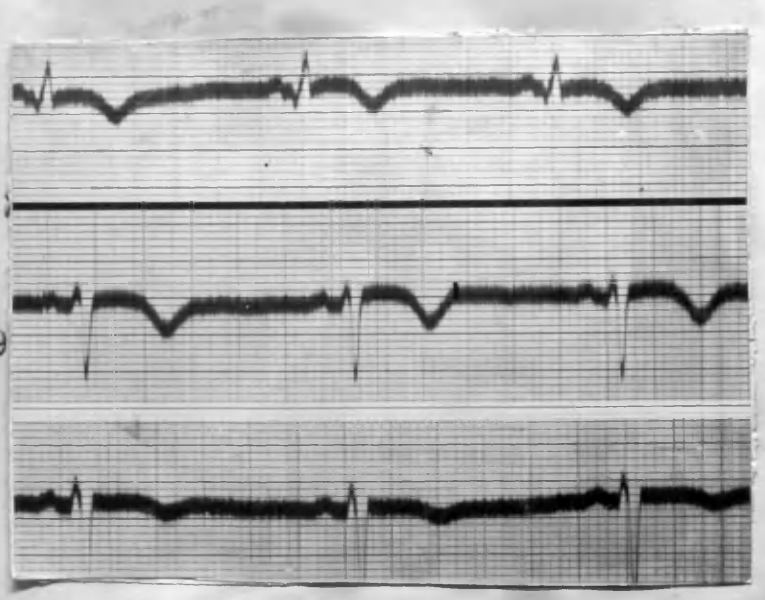
18. 3. 29.



T Wave inverted in I

Elevation of RT in II.

Cowan wave in III.



.29

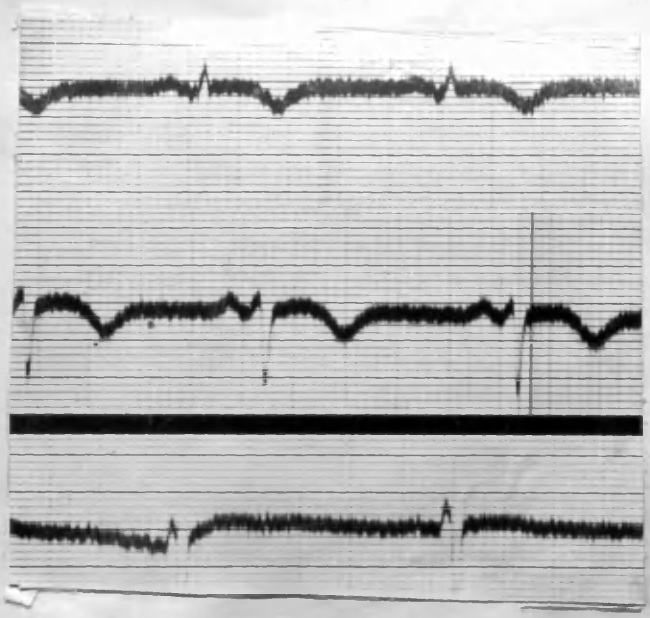
Inversion of T in I, II & III leads.

PR = .14 secs.

QRST = .48 secs.

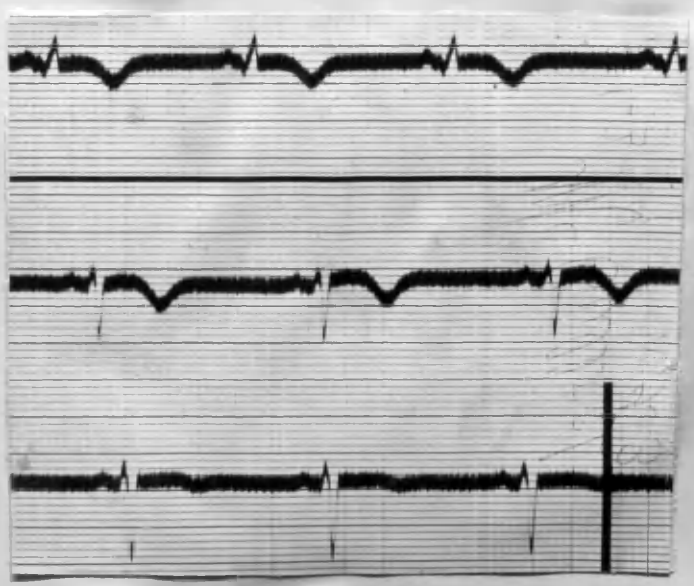
Case No. 23

25.3.29



T wave absent in III.

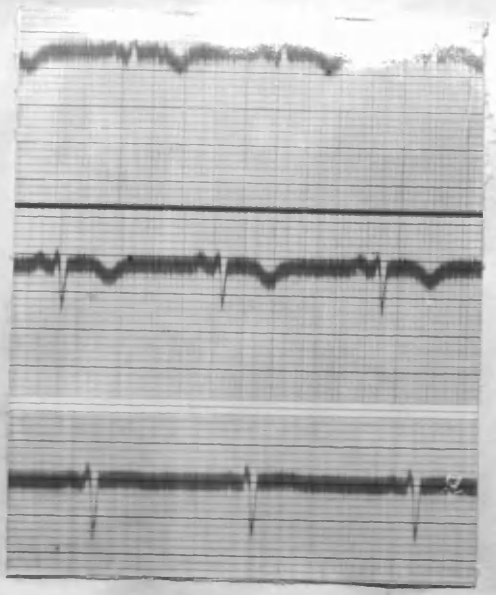
27.3.29.



QRST = ·52 - ·48

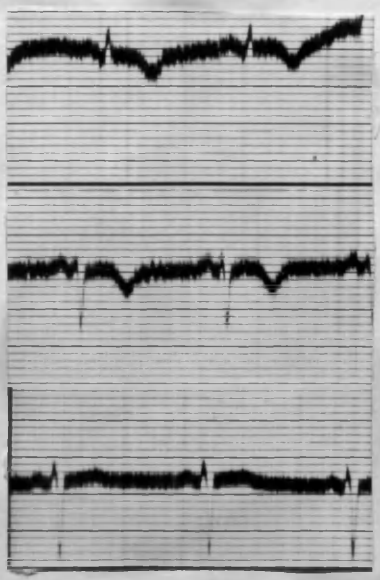
Case No. 23.

30.3.29



QRST = •48 +

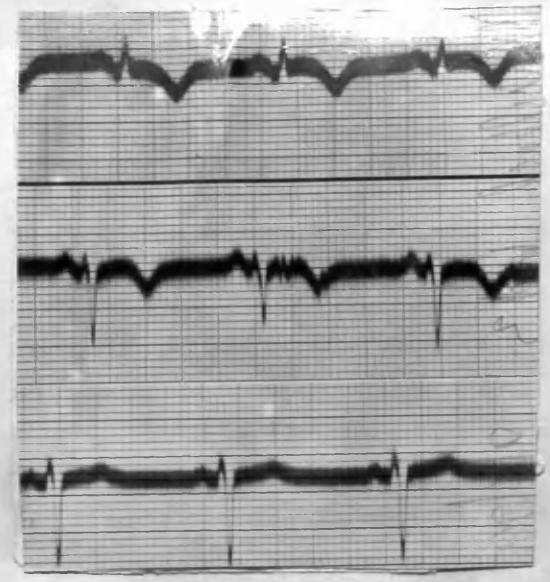
4.4.29.



QRST - •48

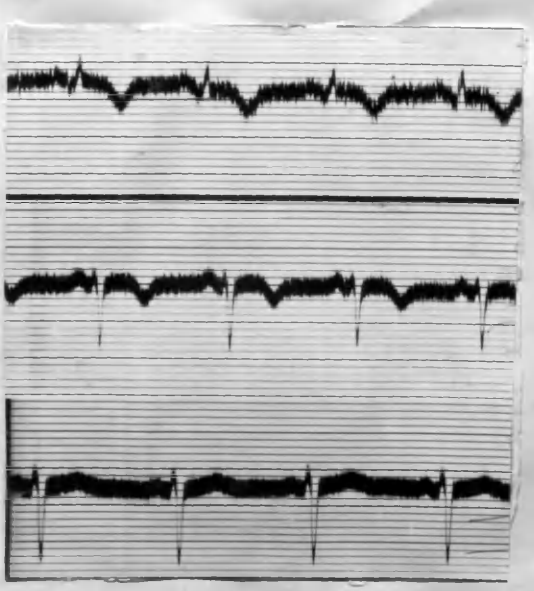
Case No. 23.

9.4. 29.



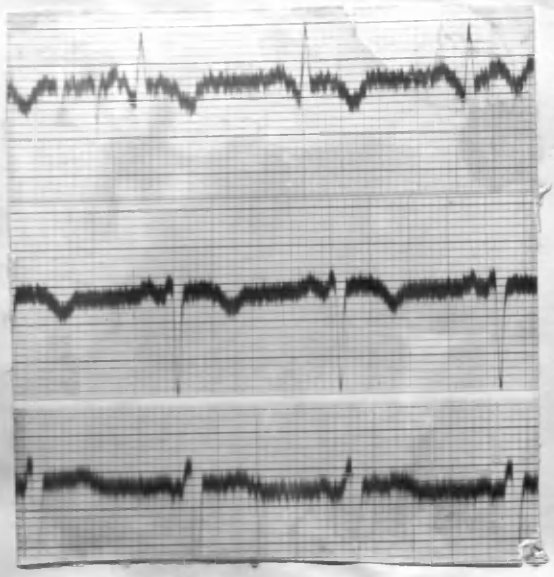
QRST = •44

12.4.29.



Case No. 23.

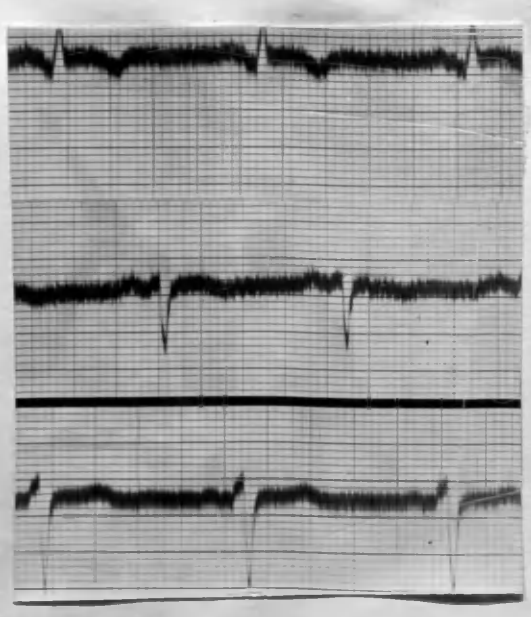
13.5.29



QRST = .40 - .36

R wave thickening in II & III.

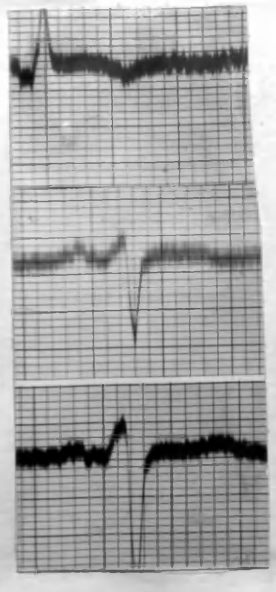
5.8.29



Inverted in I
QRST = .34 secs.
Splintering of R wave in II & III.

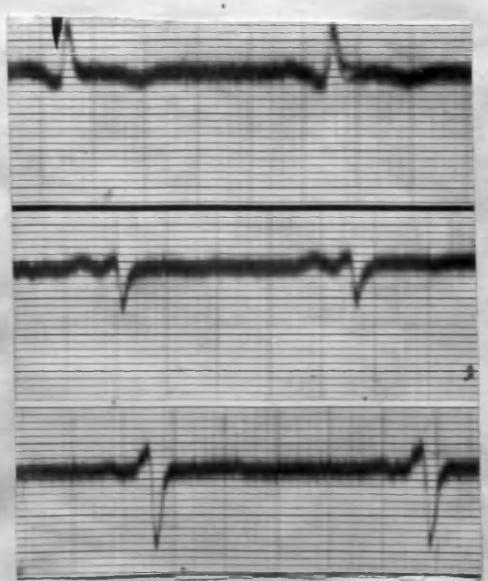
Case No. 23.

14.11.29.



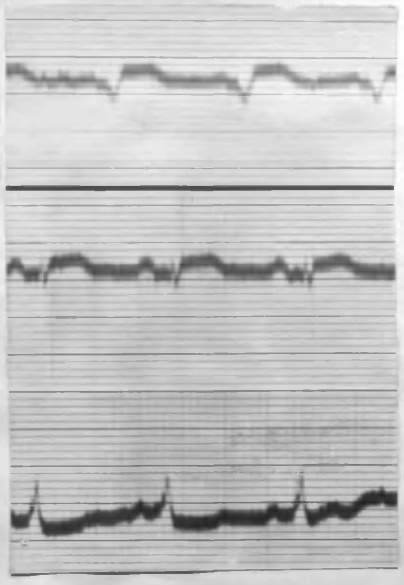
Left Ventricular Preponderance
RT slightly elevated in I
QRST = .40.
T now upright in II and III.

5. 6. 30.

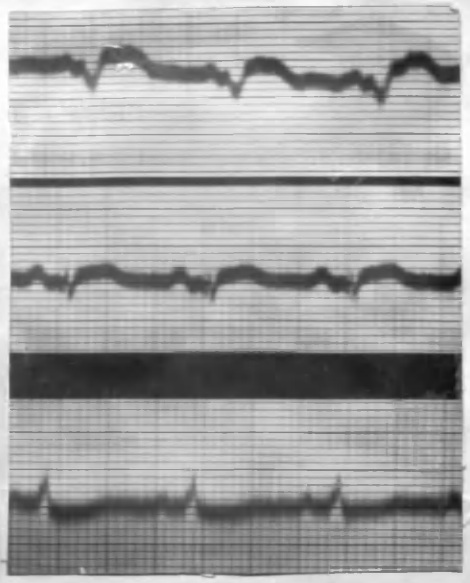


Case No. 24.

14.4.30

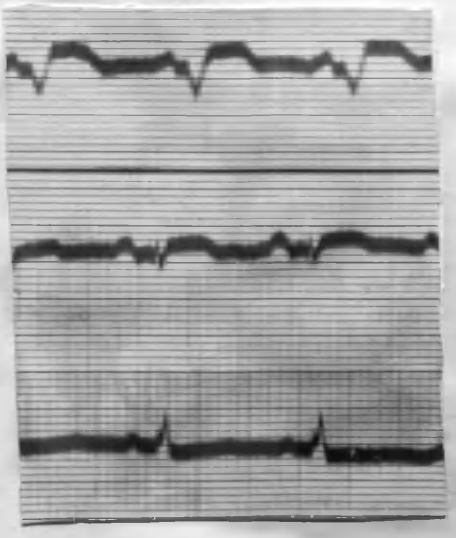


15.4.30.

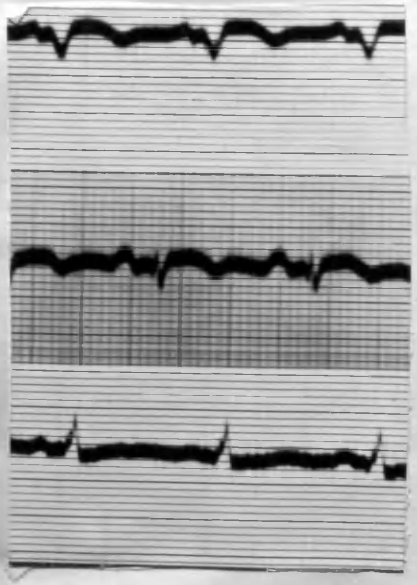


PR - .16
Right Ventricular Preponderance.
RT elevation in I & II
depression in III.
QRS notched in all leads.

16.4.30



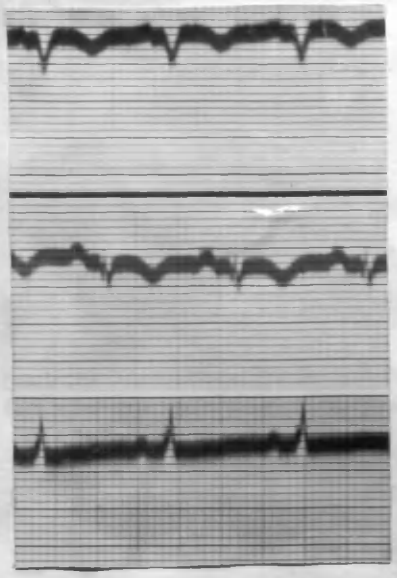
18.4.30.

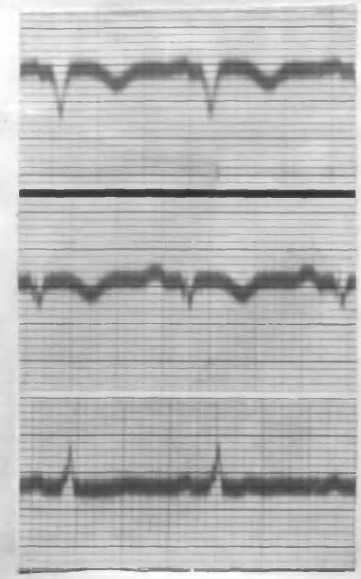


Note - inverted T in I and II.

Case No. 24.

29.4.30



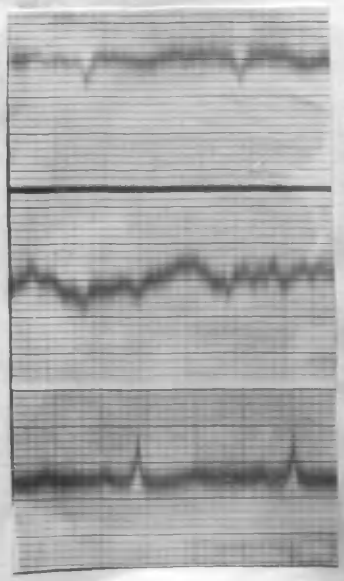


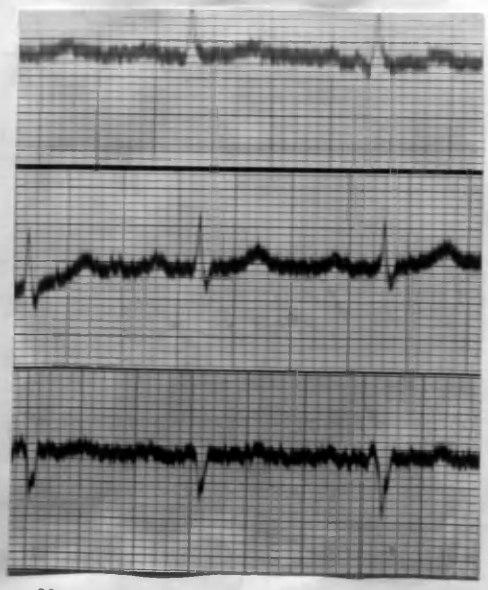
4. 5. 30.

Tracings show little change.

6 months after onset.

14.5.30



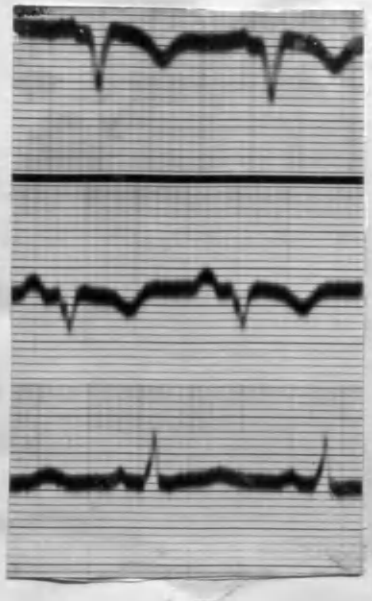


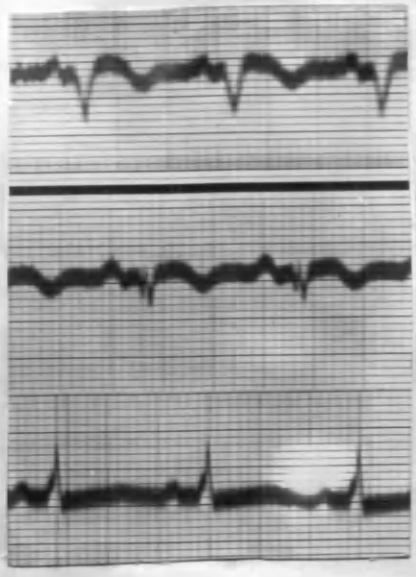
7.10.30

Now left ventricular preponderance
T wave upright in all leads.

Case No. 24.

19.4.30

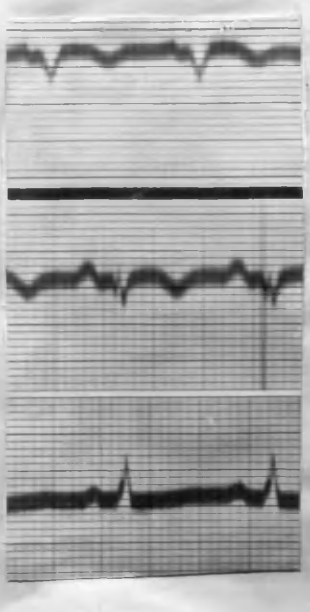


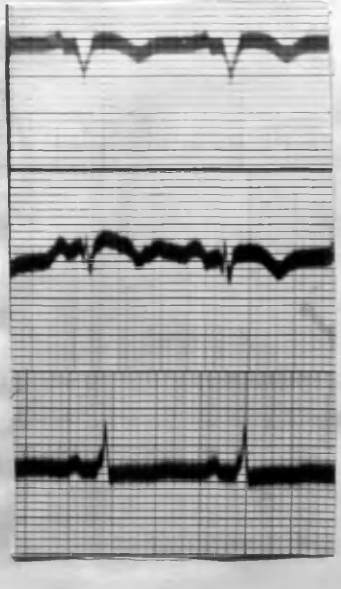


22.4.30

T inverted in I and II
upright in III.

23.4.30



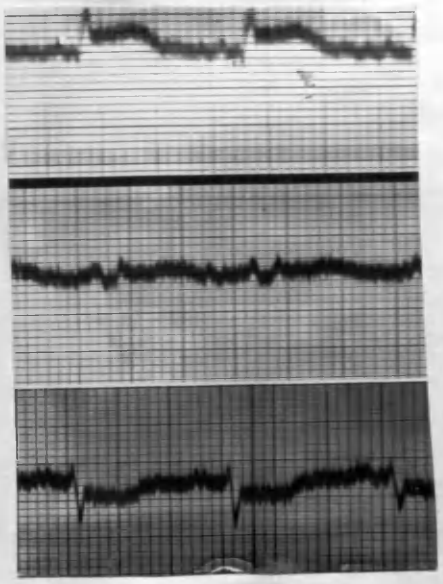


18.5.30.

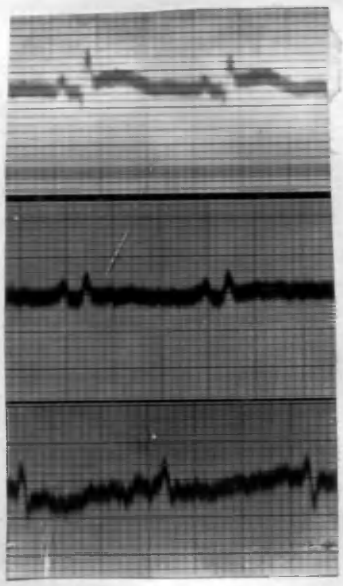
Gradual diminution of amplitude of T in III.

Case No. 25.

30.5.31



6.6.31.

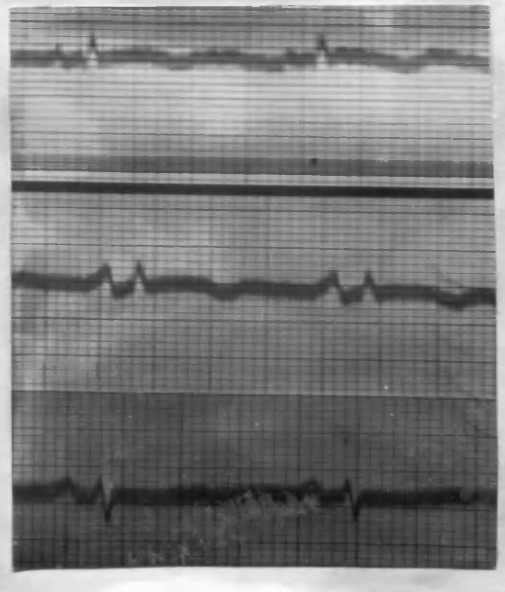


Left ventricular preponderance
RT elevation I
depression in III

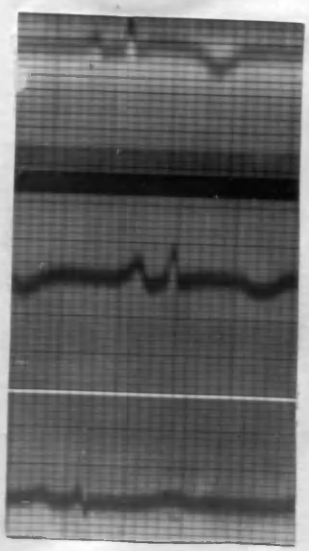
Lead 1
12.6.31.

Elevated RT
Commencing inversion of T

15.6.31.



29.6.31

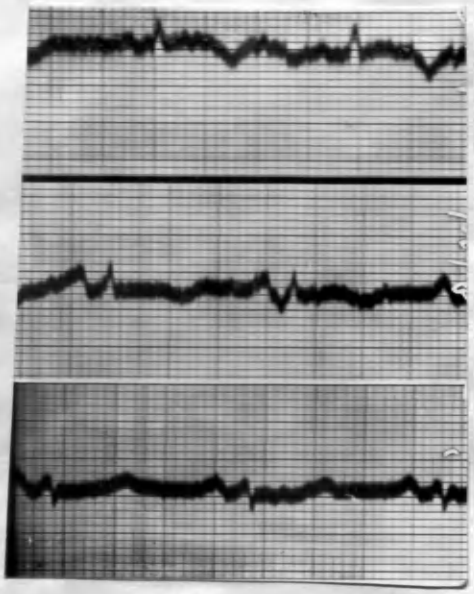


T now inverted in I & II.

T more sharply inverted
in I and II
upright in III.

16.7.31

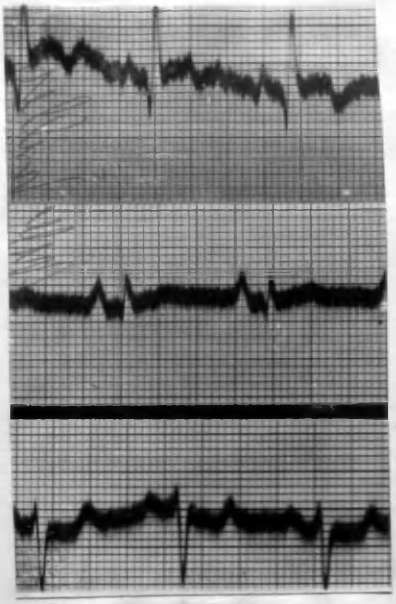
10 weeks after onset.



T wave still inverted in I.
Diphasic in II.
Small amplitude of QRS.

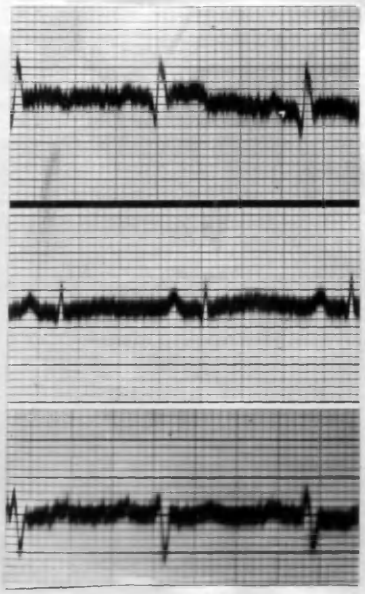
Case No. 26.

22.2.28

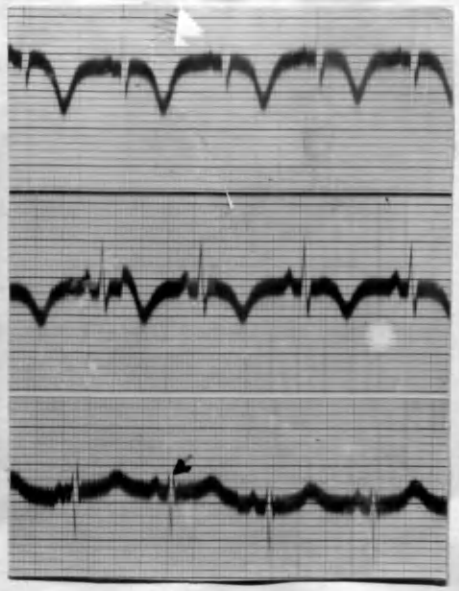


Left Ventricular Preponderance
PR - .20.
RT elevation in I; depression in III.
T wave upright in III.

2.3.28
10 days later



RT elevation in I still present.



15.10.28. - 36 hours after onset.

PR = ·12

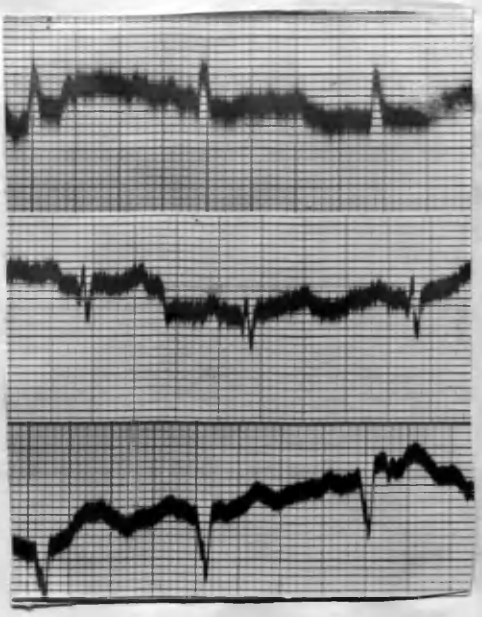
QRST = ·46

Left Ventricular Preponderance

T inverted and exaggerated in I, II.

Case No. 35.

Three days after attack.



2.10.27.

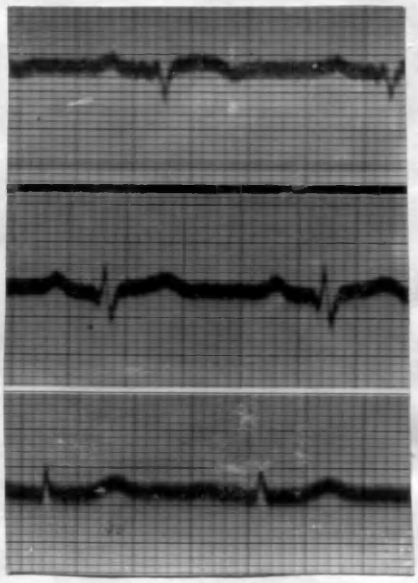
Left ventricular preponderance.

PR = •16.

QRS notched in II and III.

T is inverted in I.

8. 5. 31.



PR = .22

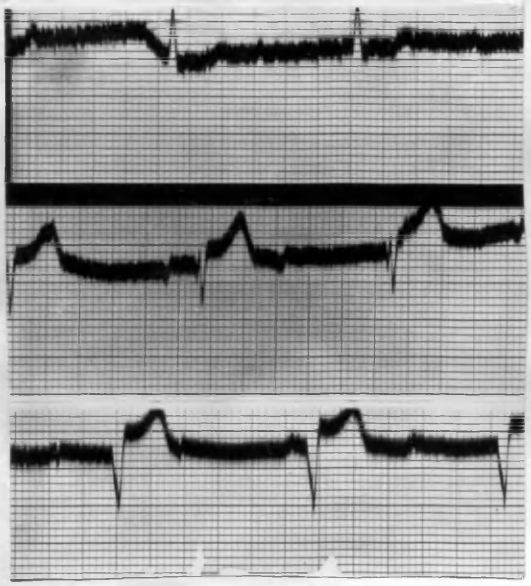
Right Ventricular Preponderance.
QRST = .36
Plateauing of RT in I
Small amplitude QRS.

SEQUENTIAL ELECTROCARDIOGRAMS.

T III Type.

24 hours after onset.

17.6.28.



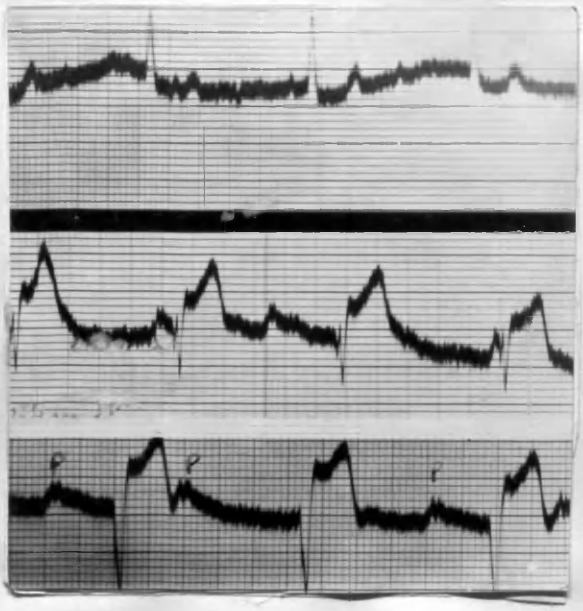
Full Heart Block.

Elevated RT in II and III

RT depressi on in I.

Case No. 29.

18.6.28.



Left Cardiac Preponderance.

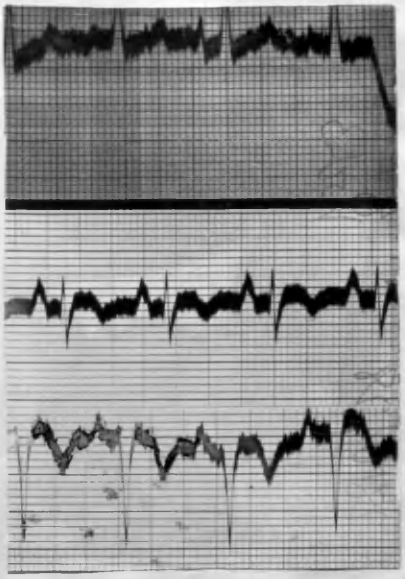
High RT in II and III

Auricular Rate 90 p.m.

Ventricular 52 p.m.

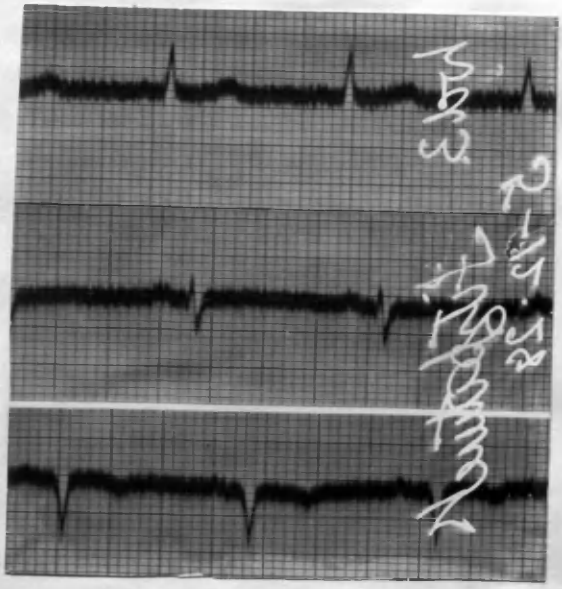
Case No. 29.

22.6.28



PR = ·20
T inverted in II Sharply inverted in III
RT still slightly elevated in III.

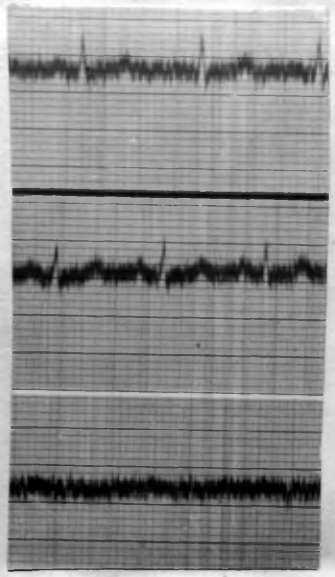
5.12.28



PR = .16
T inverted in III.

Case No. 29.

29. 3. 29.



Small normal deflection in I and II.

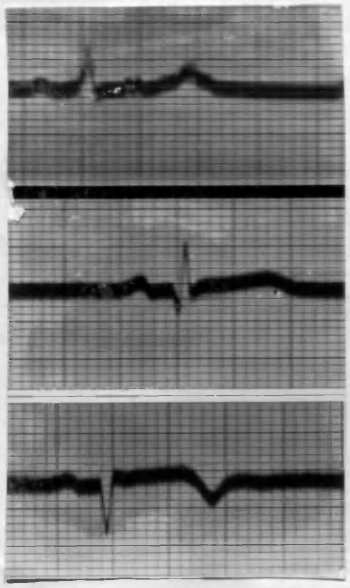
Note - T wave upright in II.

QRST absent in III (Faulty connection?)

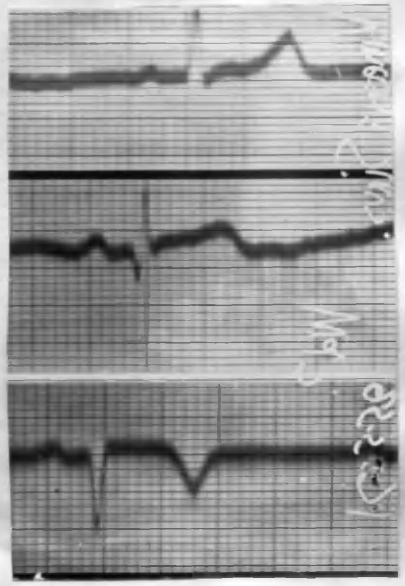
Case No. 27.

24.5.31

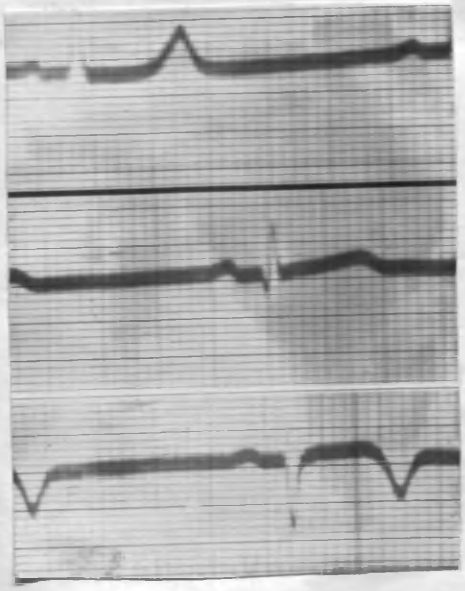
2 days after onset.



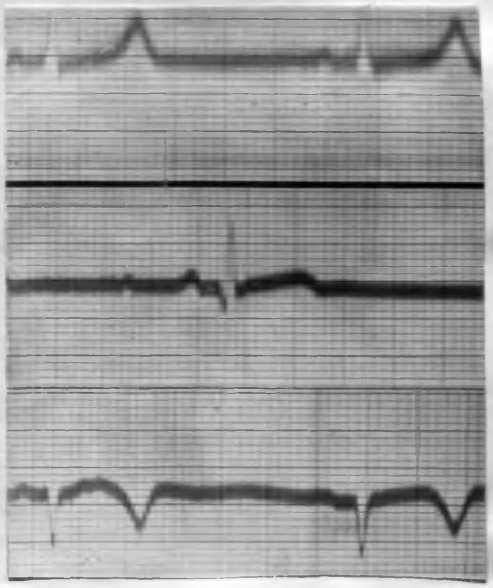
25.3.31.



27.5.31.



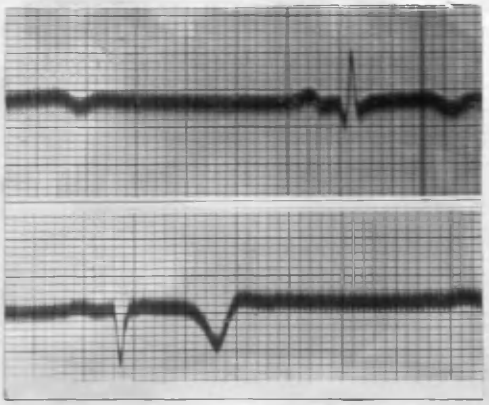
28.5.31

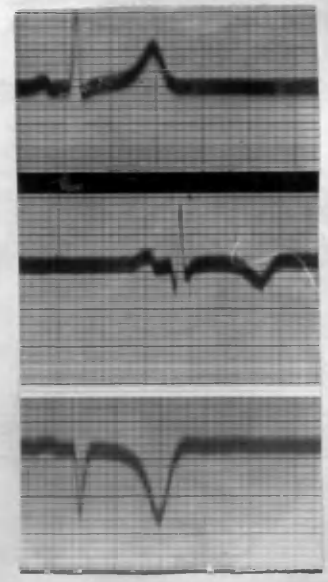


Note gradual inversion of T Wave in III.

Case No. 27.

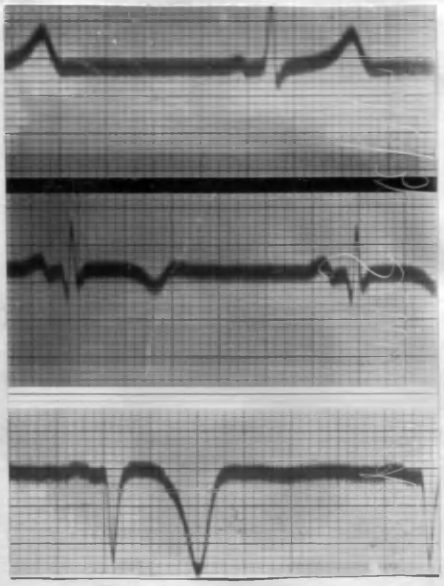
30.5.31

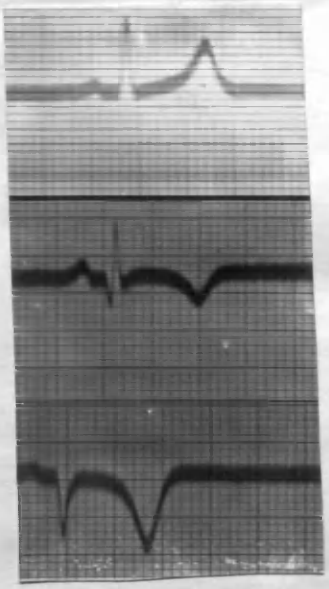




3.6.31.

5.6.31.



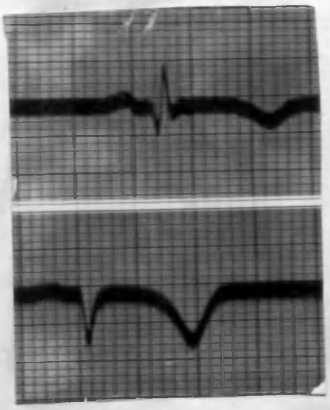


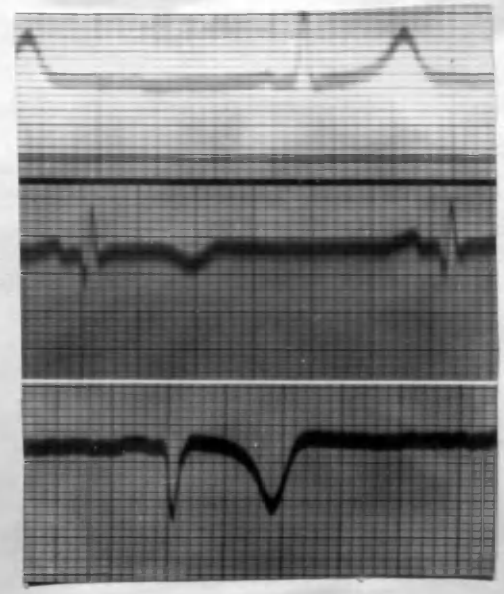
6.6.31.

Note. T wave in II becomes gradually inverted.
T wave in III of marked amplitude.

Case No. 27.

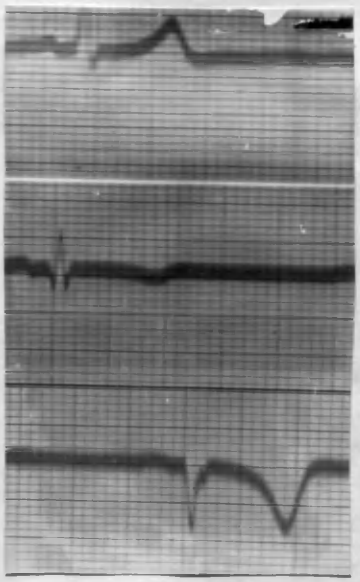
12.6. 31.

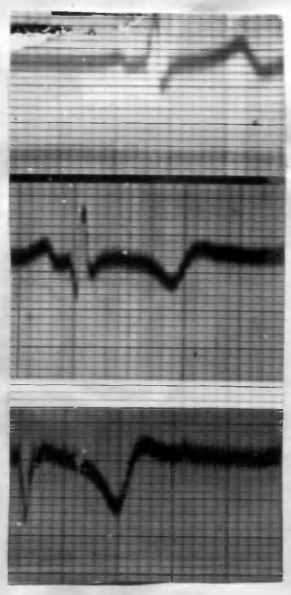




15.6.3

25.6.31.





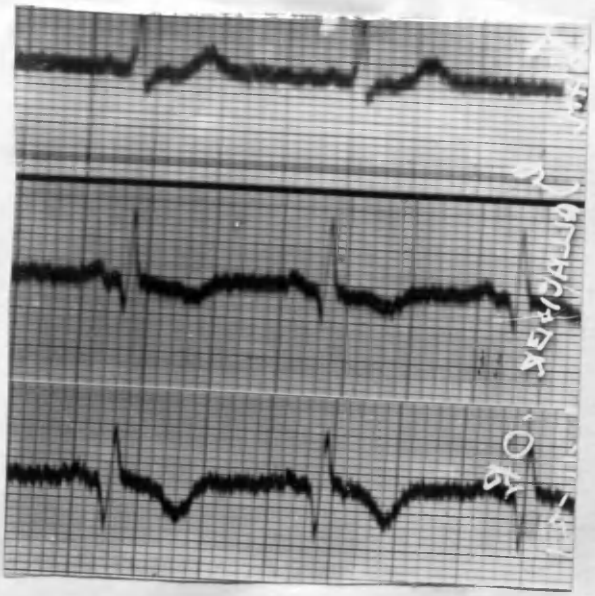
30.6.3

Little alteration during the next month.

Case No.40.

3 weeks after onset.

4.4.31.



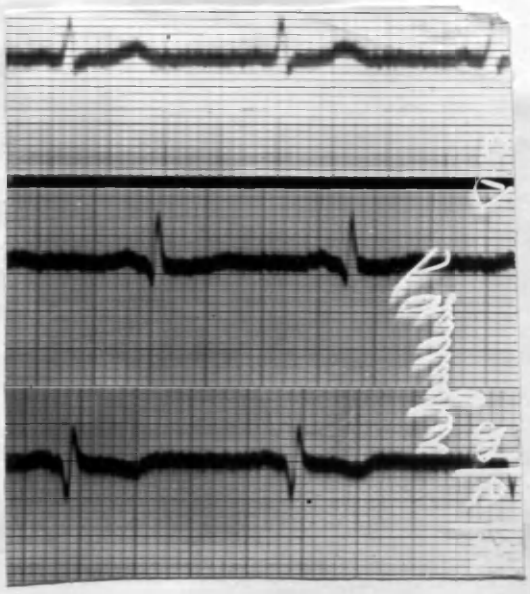
Left ventricular preponderance.

T inverted in II; sharply inverted in III.

Lead III QRS complex - Q wave is greatly increased.

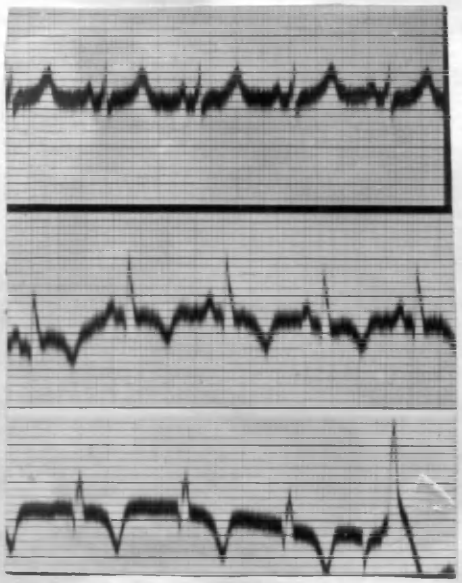
11 weeks after onset.

29.5.31.



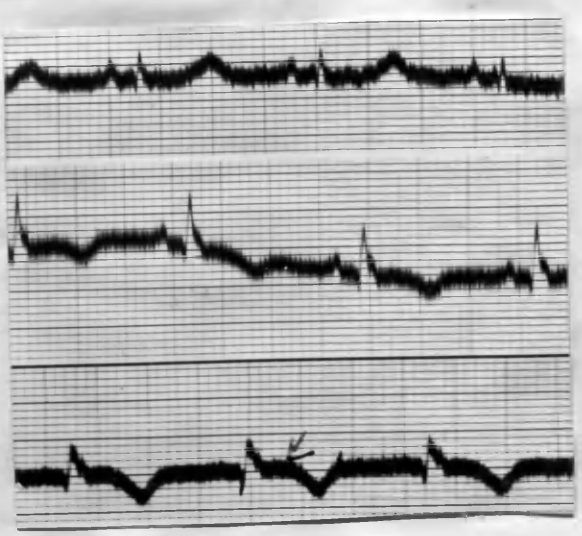
T wave in II no longer inverted and inversion in III is much less.

11 days after attack.



QRST - .40 secs.
T is sharply inverted in II and III.
R is splintered in I and III.

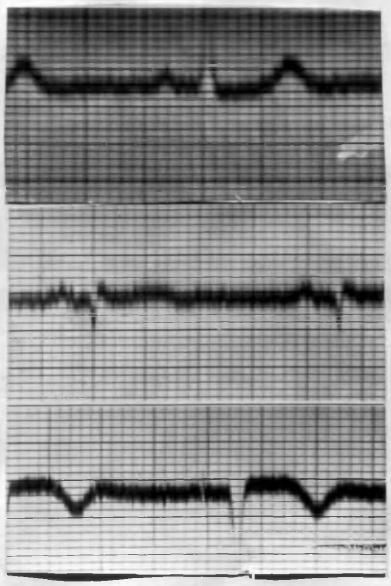
One month later.



T is less sharply inverted.
Note. "Shouldering" of RT in Lead III
QRST = .36

Case No. 64.

Seven Days After.

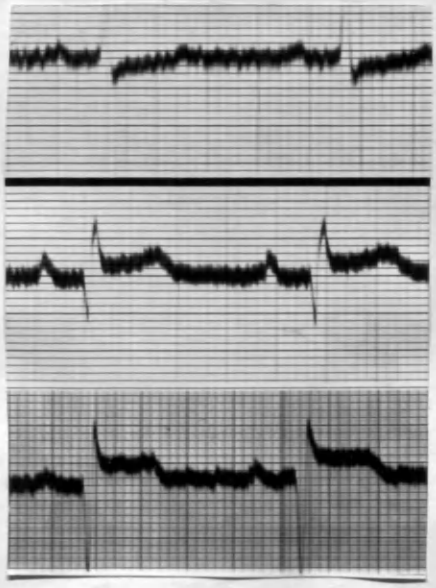


Elevation of RT in II and III.

T inverted in III.

Case No.92.

1 day after Onset.



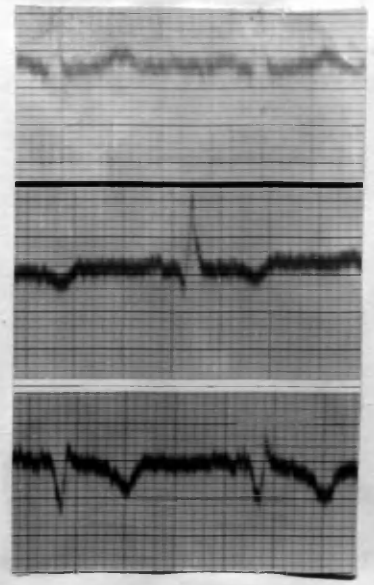
RT elevation in II and III.
depression in I.

Q in III is +

Case No. 51.

Six weeks after onset.

9.9.30.



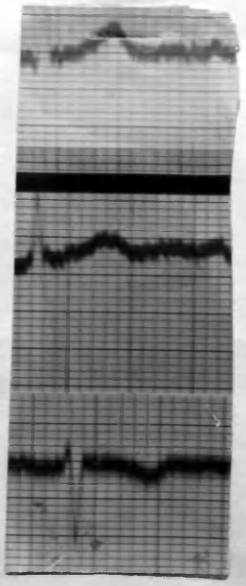
Left ventricular preponderance.

T inverted in II - sharply inverted in III

Note variation in amplitude of terminal wave in QRS complex in III.

Case No. 46.

3 weeks after sudden onset of pain in left side of chest.



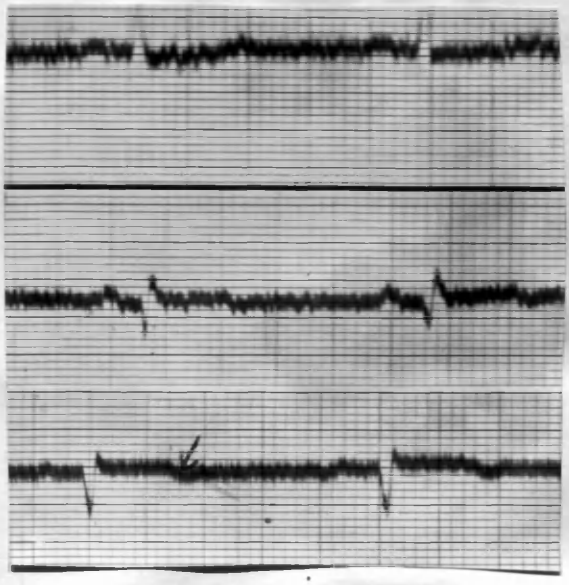
Left ventricular preponderance.

T inverted in III.

QRST = .32.

Case No. 92.

2 days after Onset.



Diminution of wave amplitude.

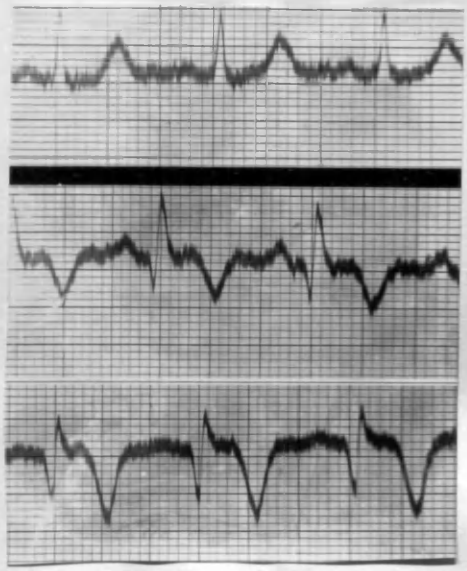
RT is still inverted in II and III.

T is becoming inverted in III.

Q is still + in III.

Case No. 93.

1 month after Onset.



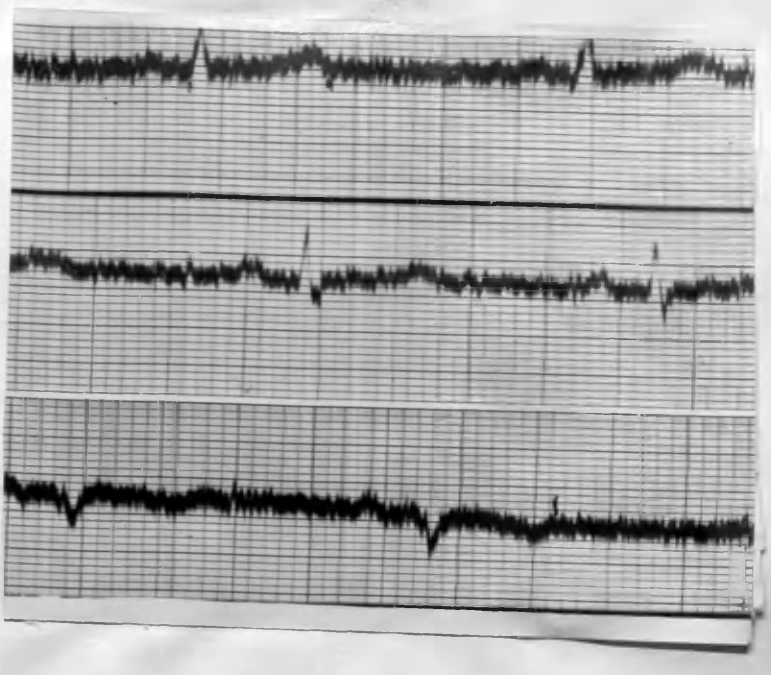
Tracing taken 30 days after severe attack and 12 hours after second attack lasting a few minutes.

Note. Elevation of RT in II and III.
Inversion of T in II and III.
Q wave in III is of markedly increased amplitude.

Case No.62.

22 days after onset of acute abdominal pain - Laparotomy.

27.11.29.



QRS = ·06

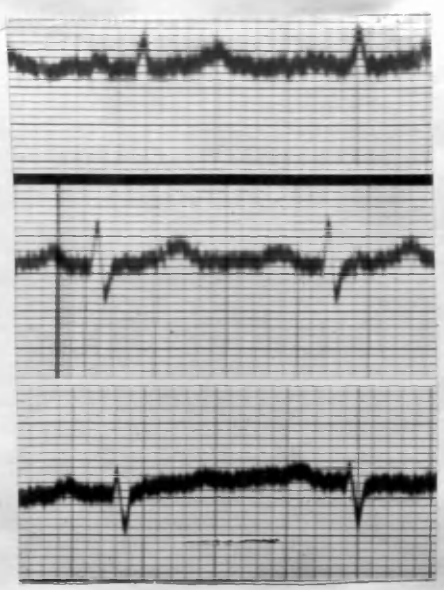
QRST - ·36 - ·40

T inverted in III.

Splintering in Lead III.

Case No. 62.

4.1.30.



Left Ventricular Preponderance.

QRST = .36

Note change in QRS in III.

R wave now present.

T wave upright.

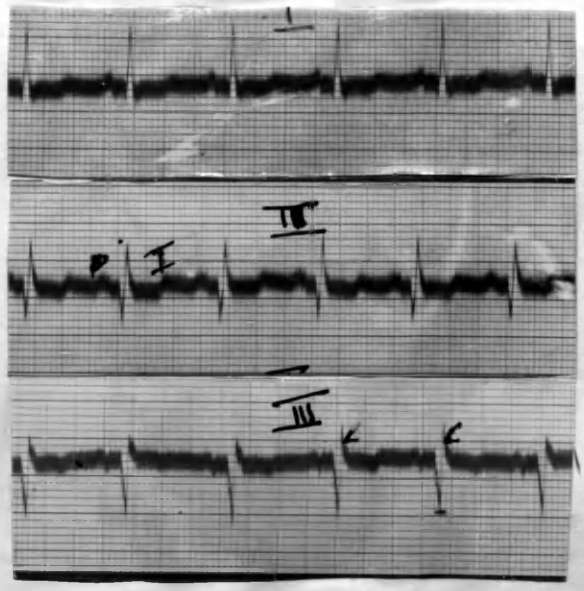
ELECTROCARDIOGRAMS showing -

(1) Increased amplitude of Q III with inversion of P III.

(2) Broadened and splintered QRS.

Case No. 37.

22.10.28.



Left Ventricular preponderance.

PR = .12

P inverted in III

QRS in II is abnormal.

T is displasic (?) in all leads.

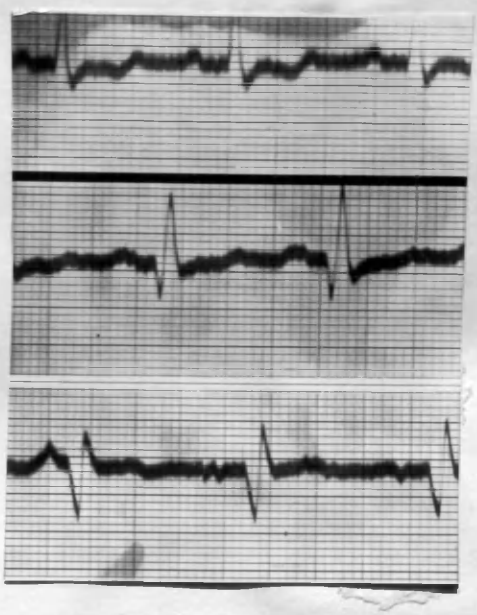
The appearance in leads II & III is almost as if a normal QRS had been rotated.

The terminal wave of Cowan in QRS III is prominent.

Case No. 37.

3 years later.

13.2.32.



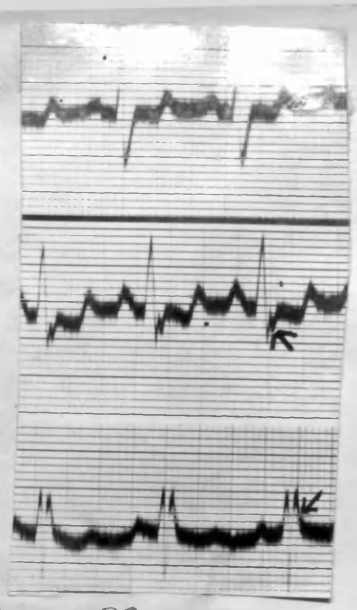
T is inverted in III.

Q III is +

Note. Variation of P in III - upright - diphasic inverted.

Case No. 36.

12 days after attack.



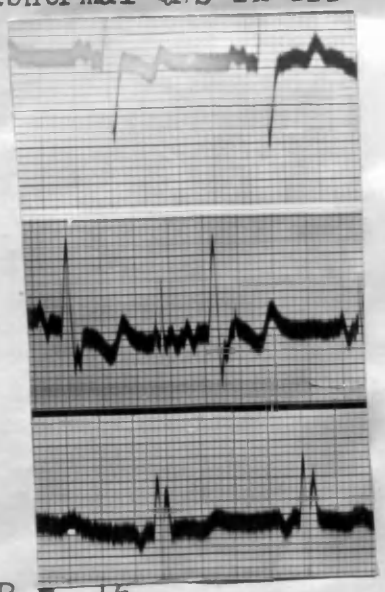
7.5.28.

PR = ·20 secs.
QRS = .12 secs.

T diphasic in II and III

QRST = ·40

Note abnormal QRS in III



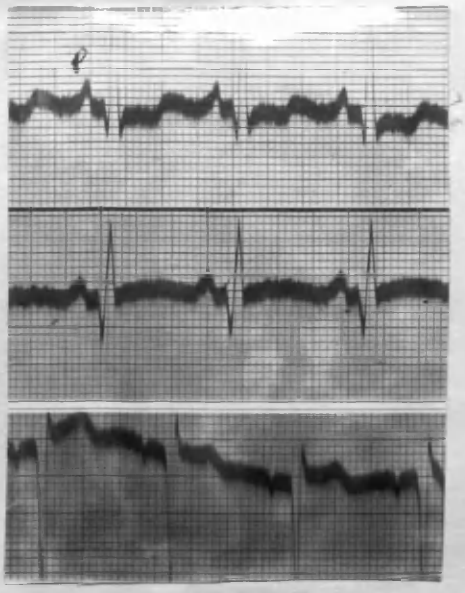
25.6.28.

PR = .16
P bifurcated in I
P inverted in III.

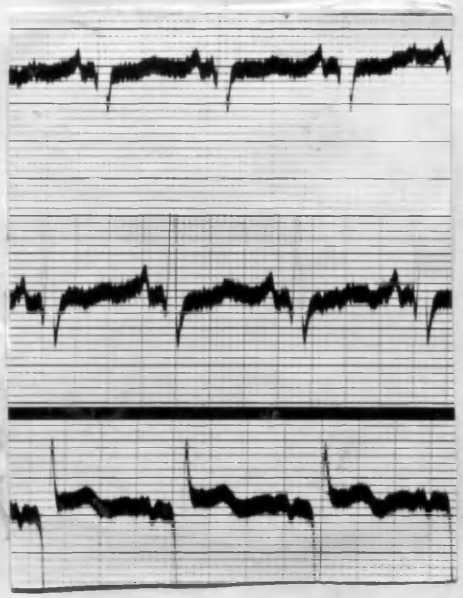
Case No.28.

1 month after attack.

3. 8. 28.



Left Ventricular preponderance
PR = .16 secs.
RT is depressed in I elevated in III.
P is inverted in III.
Q is + + in III



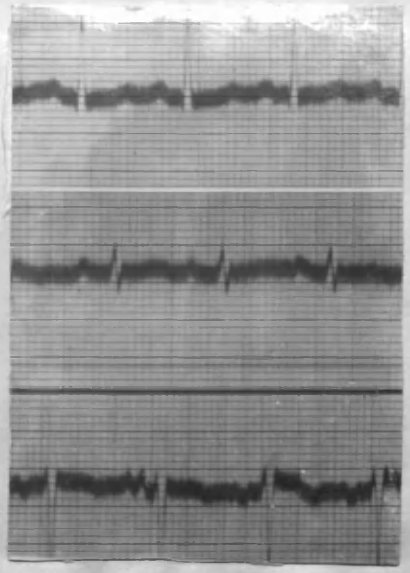
10.8.28.

P is still inverted in III.
RT is no longer depressed in I but remains elevated in III.

Case No. 33

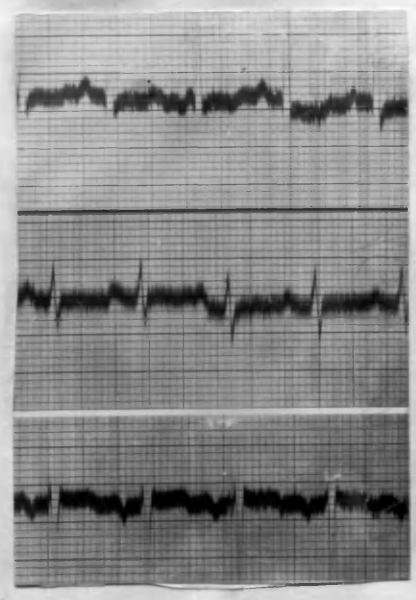
7 days after onset of severe praecordial pain.

18.4.29.



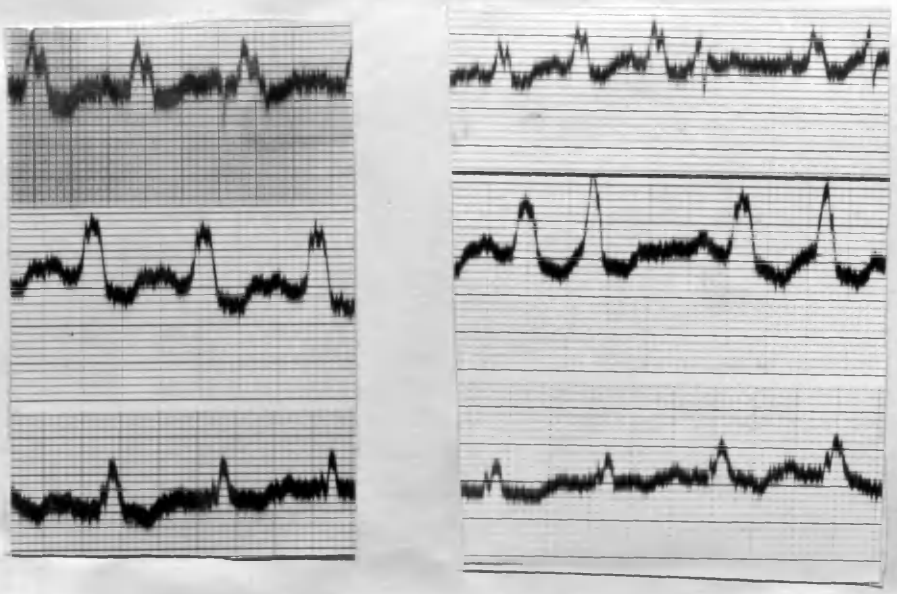
Left ventricular preponderance.
Poor T wave.
RT is slightly elevated in III.
P is inverted in III.

25.5.29.



There is little change in the electrocardiogram.

3. 2. 28.



1.3.28.

Arborisation Block (QRS = ·16)

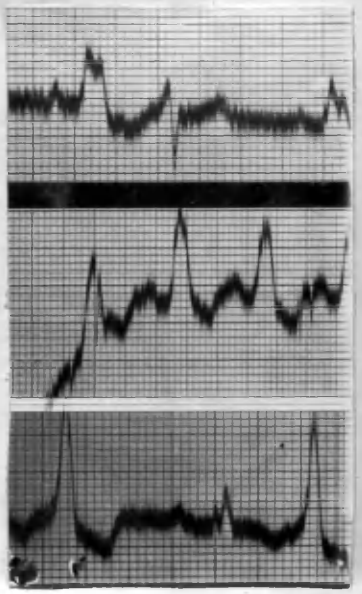
QRS broadened and splintered.

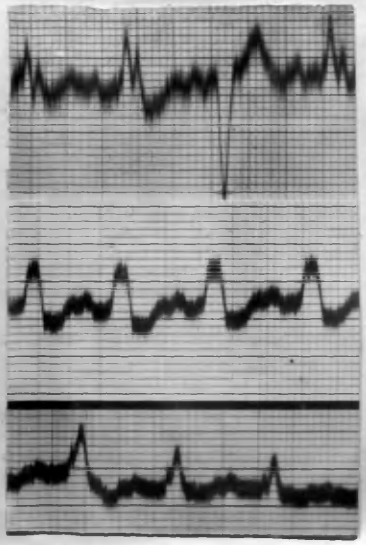
RT depression in I and II.

T inverted in all leads.

Recurring extra systoles Lead II (1.3.28)

5.3.28





9.3.28.

Broadening and splintering of QRS

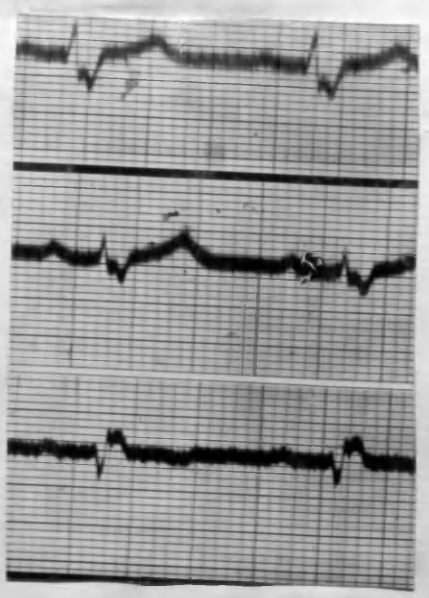
Extra systoles - Lead III (5.3.28)

Lead I (9.3.28)

Case No.41.

Three weeks after attack.

24.9.30.

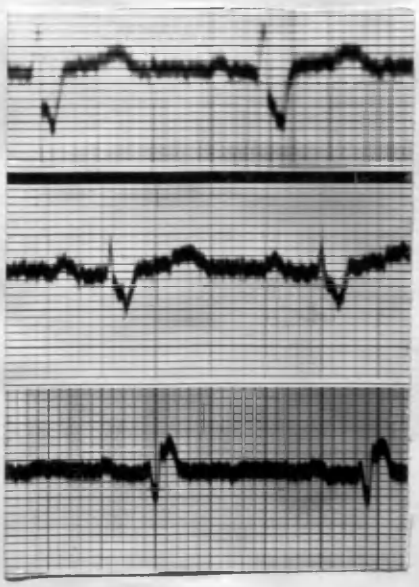


PR = ·20

Note - Abnormal QRS = ·12.

RT elevation ? in III
depression in I and II.

Roberts
25.4.31.

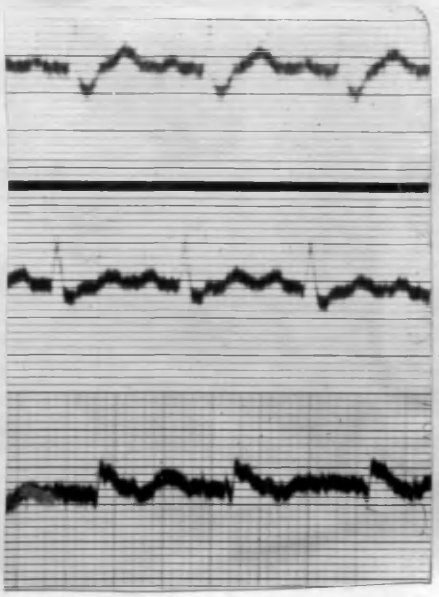


Increased amplitude of waves in all leads.
Otherwise there is little change.

Case No.42.

Progressive Cardiac weakness 1 month.
Pericarditis - 2 days before (173.30).

19.3.30.



Note abnormal QRS

RT elevation in III

depression in I and II

T wave inverted in III.

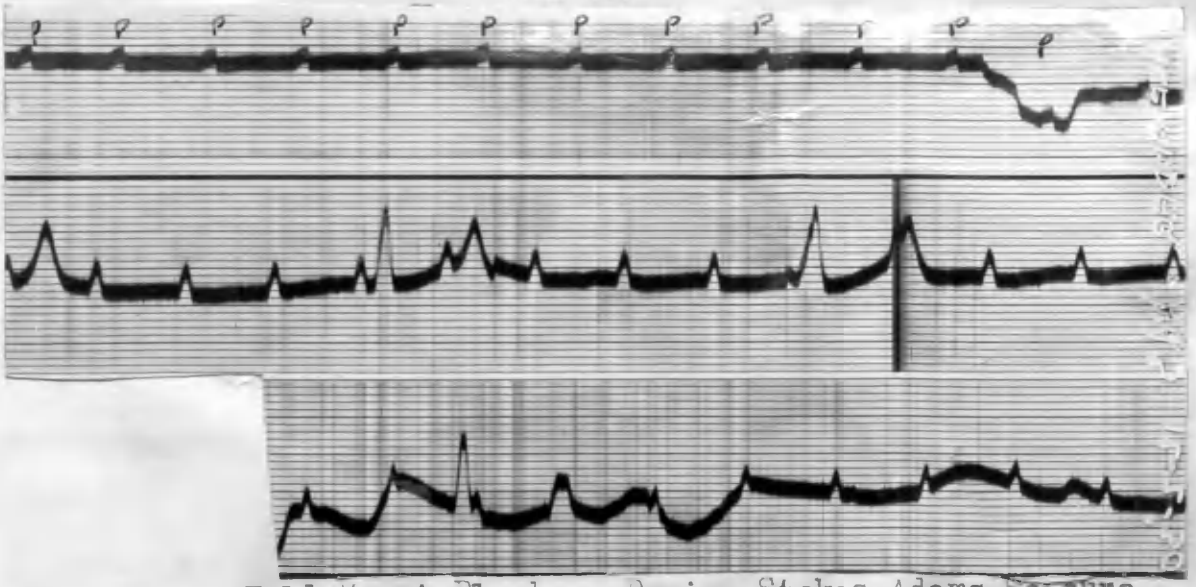
There was no change in subsequent tracings taken over a period of one year.

ELECTROCARDIOGRAMS showing

Abnormality of Rhythm.

Case no. 55.

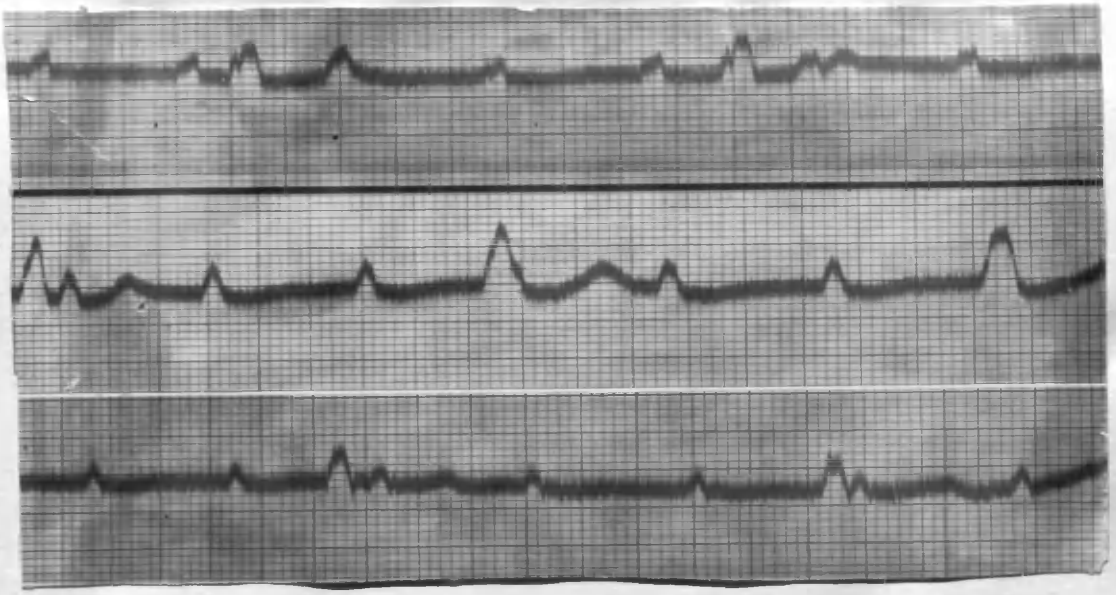
12.2.30



Full Heart Block. - During Stokes Adams Seizure

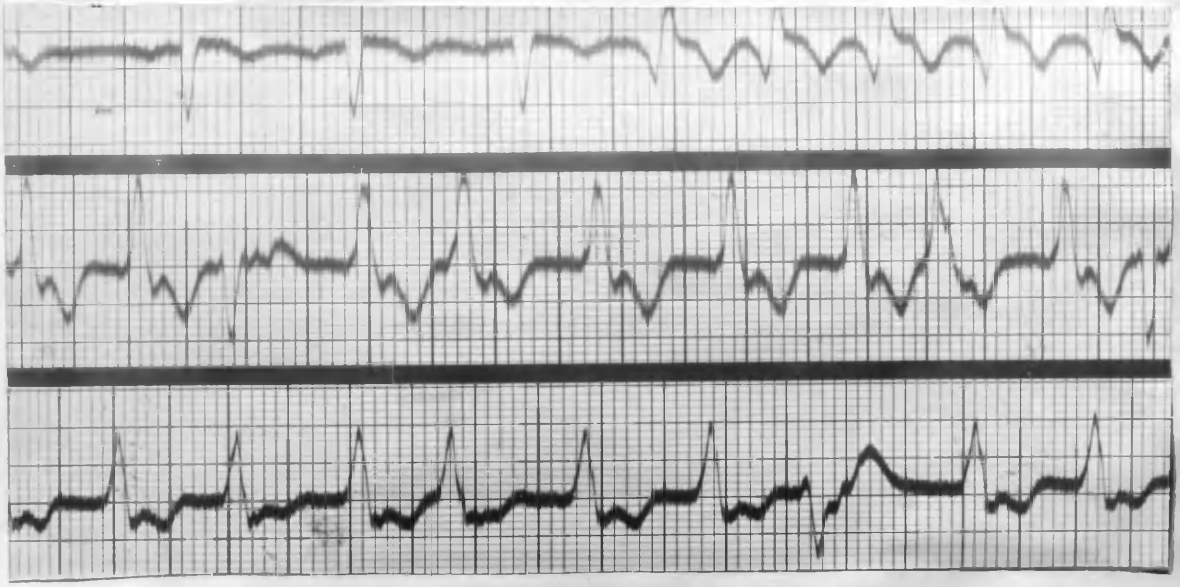
Note absence of ventricular complex in I.

4.10.30.

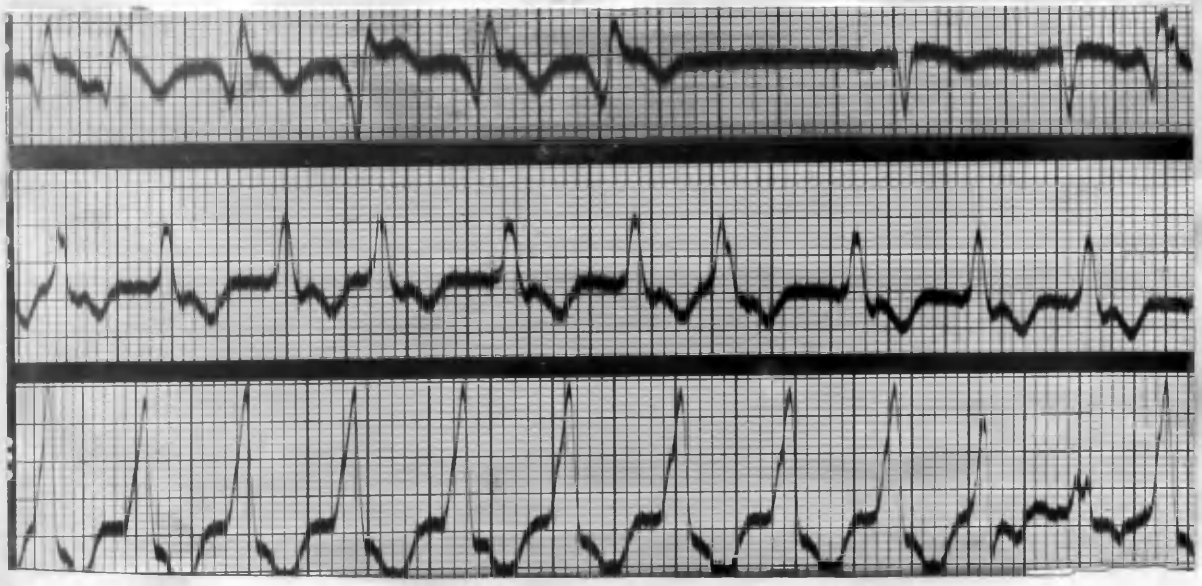


Splintering and Broadening of QRS

QRST - ·50 secs.



Frequent attacks of praecordial pain, dyspnoea and palpitation.
Tracings taken during attack.



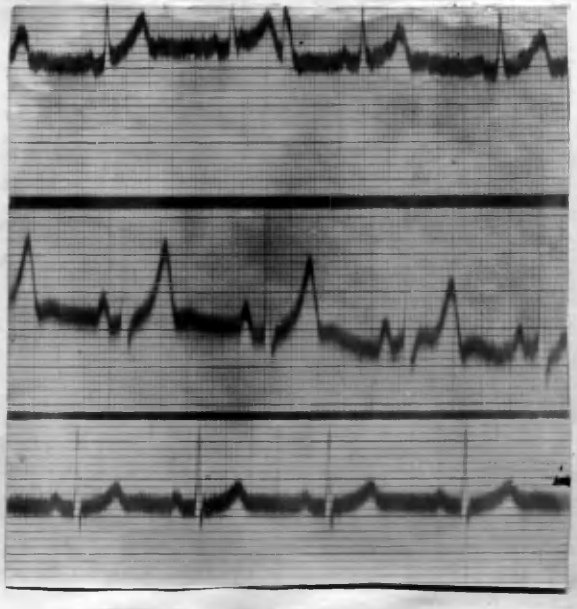
Frequent ventricular extra systoles
Paroxysmal ventricular tachycardia.
Note inversion of PQRST in I.

NORMAL ELECTROCARDIOGRAMS.

Case No. 54.

6 days after onset.

4.10.28.



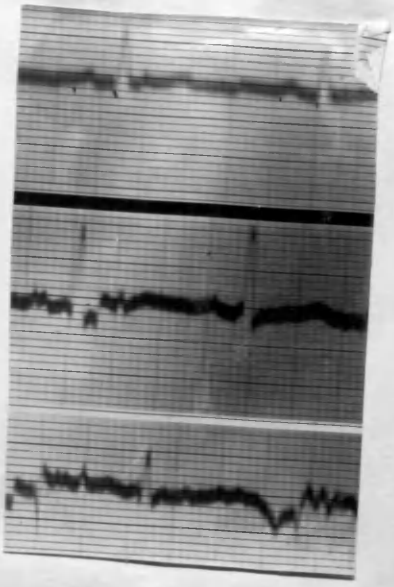
PR = ·16

QRST - ·40

T IS + in II

No RT deviation

Case No. 66.



PR = 18.

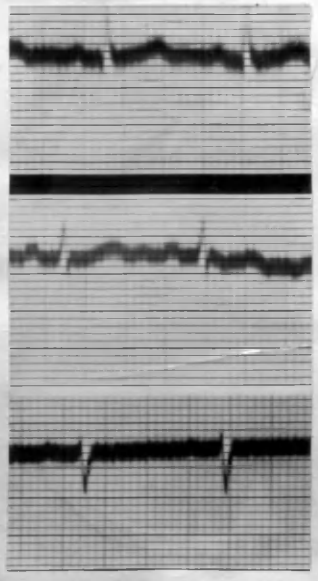
T wave upright in I and II.

absent in III.

No RT deviation.

ELECTROCARDIOGRAMS AFTER RECOVERY.

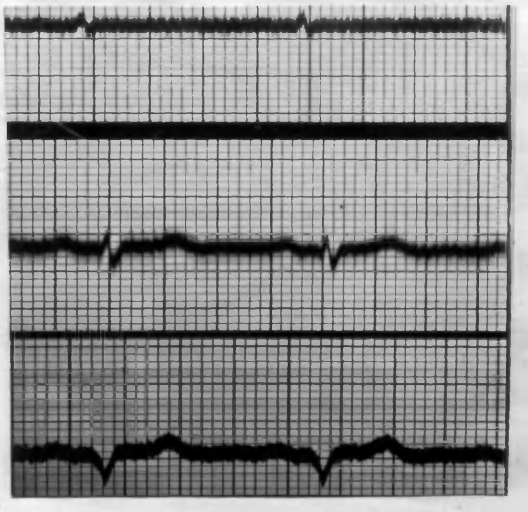
One year after first attack.
Three months after second attack.



Left ventricular preponderance.

T wave absent in III.

15 months after attack.



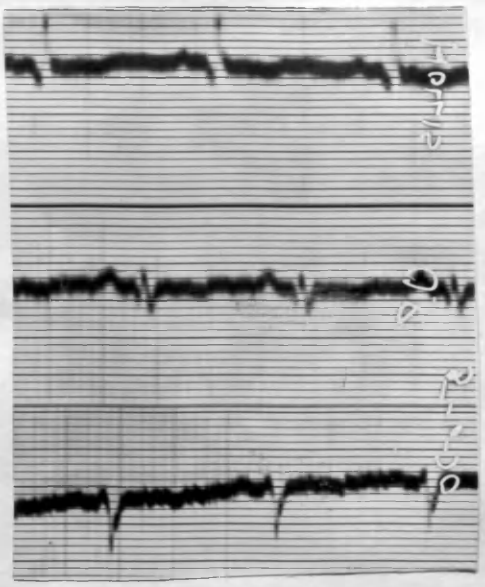
Small amplitude QRS.

Left axis deviation.

Case No.44.

2 months after attack.

5.6.30



Left Ventricular Preponderance.

PR = ·16

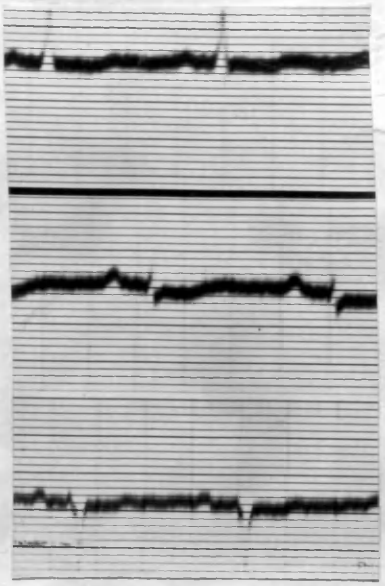
T inverted in I

QRS notched in all leads.

Case No. 45.

6 months after first of many anginal attacks.
7 days before terminal attack.

17.6.30.



Left ventricular preponderance.

RT depression in II.

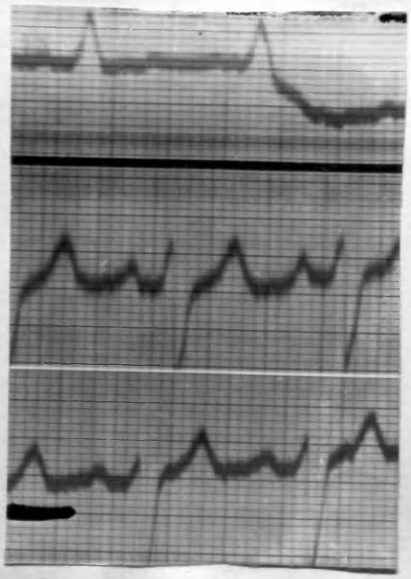
T inverted in I.

Poor T amplitude in all leads.

Case No. 47.

Five months after severe attack of Status Anginosus.

27.6.31.



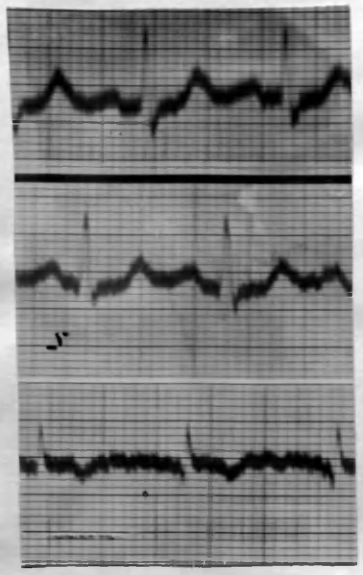
Left ventricular preponderance.

T inverted in I.

Case No. 48.

Three months after attack.

27.12.30.

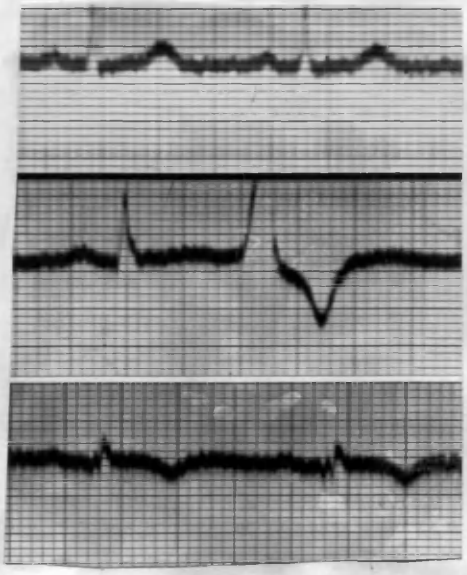


RT elevation in III.

T wave inverted in III.

Case. No.49

After three months.



Small amplitude QRS in III

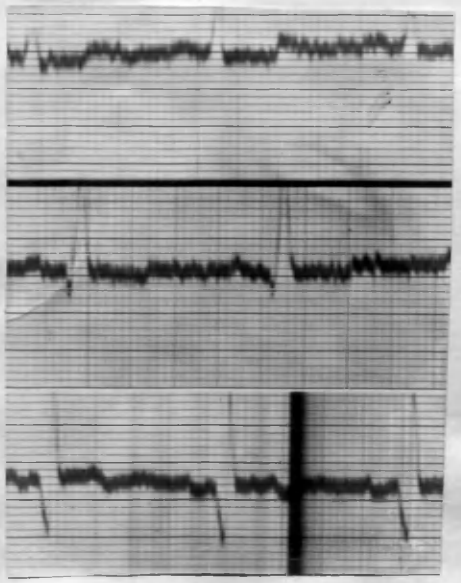
Elevated RT and inverted T in III.

Extra Systole in Lead II.

Case No. 50.

2.9.31.

Five months after attack.



RT depression in I.

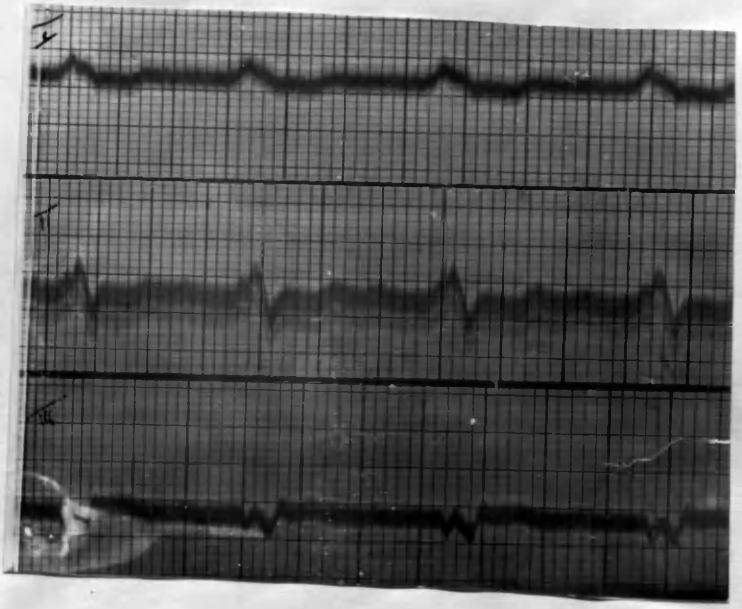
elevation in III.

Q+ in III

T wave inverted in II.

Case No. 85.

Three months after attack.



Left Ventricular Preponderance.

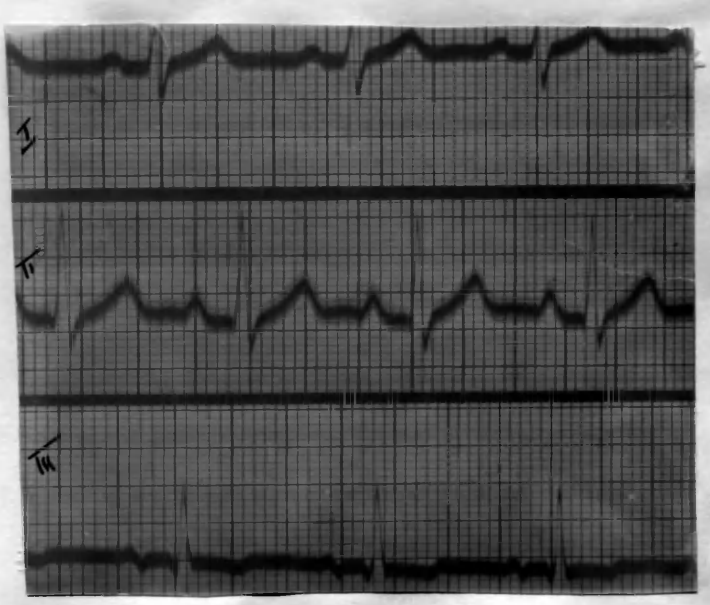
QRS = .14. QRS splintering in II and III.

RT slightly elevated in III.

Right Bundle Branch Block ?

Case No. 83.

Five months after.



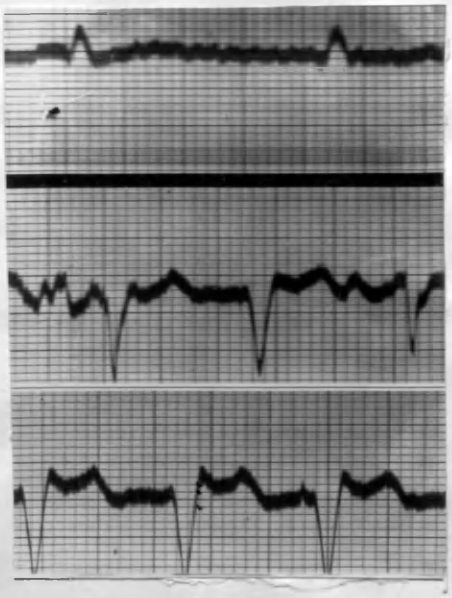
Inversion of P in III.

Inversion of T in III.

Case No. 38.

4 weeks after onset.

4.5.31.



Auricular fibrillation.
Left Ventricular preponderance.

QRS - .12

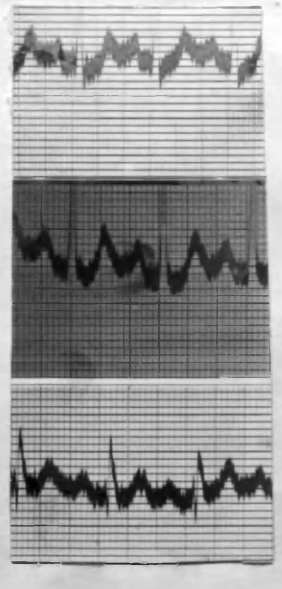
QRST = .34 secs.

RT elevated in II and III.

Case No.43.

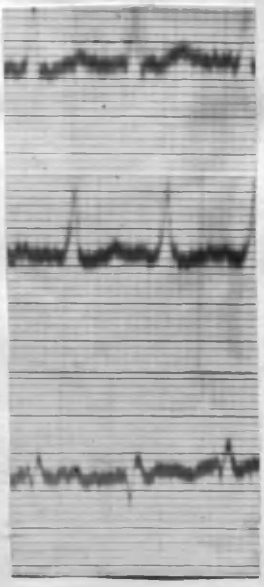
2 months after attack.

9.5.27.

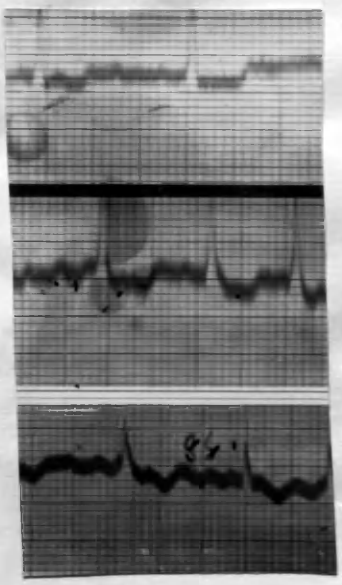


RT slightly depressed in I and II.
Abnormal QRS complex in III.
Q wave amplitude variation.

1.2.30



Auricular Fibrillation
Small amplitude QRS in III
Q wave is + in III and varies in amplitude.



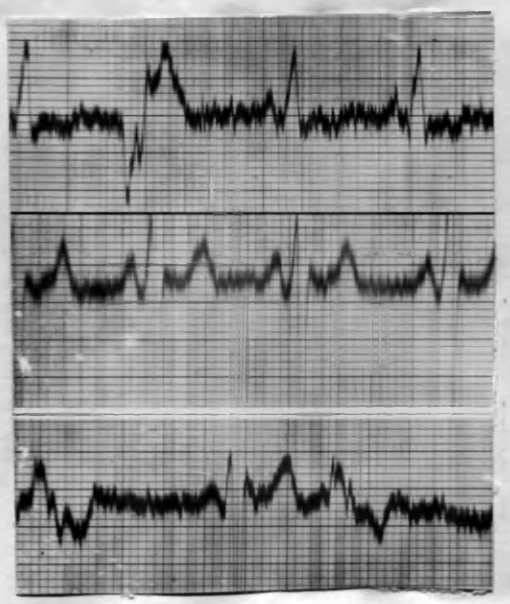
6.6.31.

A.F.
Note change in QRS in III
It is now of normal type.
T inverted all Leads.

Case No. 69.

After attack.

3.11.28.

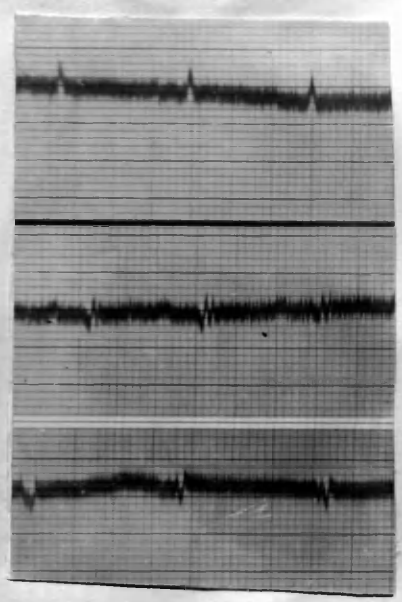


Left Ventricular Preponderance.
QRS is increased.
Recurring R Ventricular Extra Systoles.
QRST = .36 secs.

Case No. 63.

Two years after attack of "gall stones". Seven days before sudden death.

8.4.29.

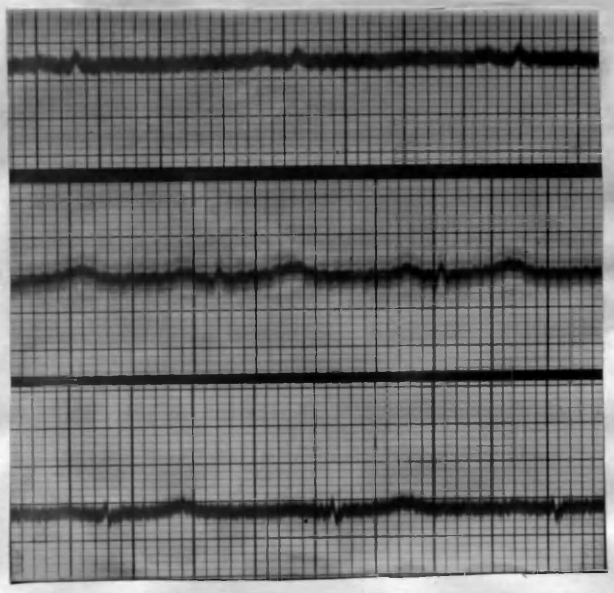


Small QRS amplitude in all leads.

T wave absent.

Case No. 90.

Five months after attack.



Small amplitude QRST.

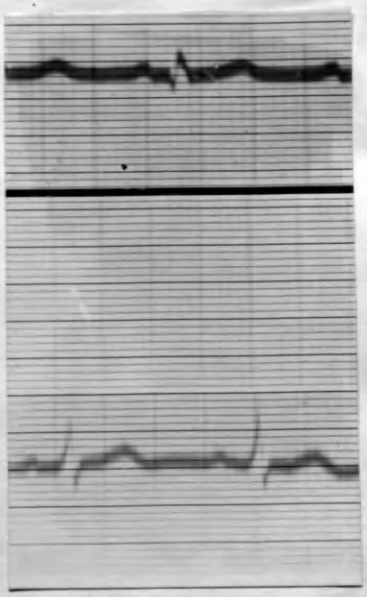
No axis deviation.

T upright in all leads.

[illegible] [illegible] [illegible] IN PENICILLIFERS.

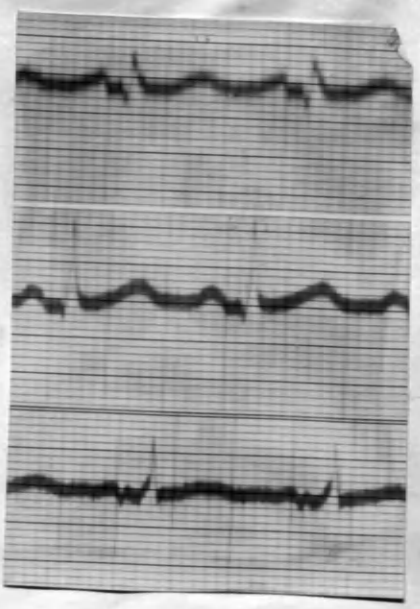
Taken from patient
suffering from
Pericarditis.
5/30.

12.7.30



Small deflection QRS in I.

18.8.30.

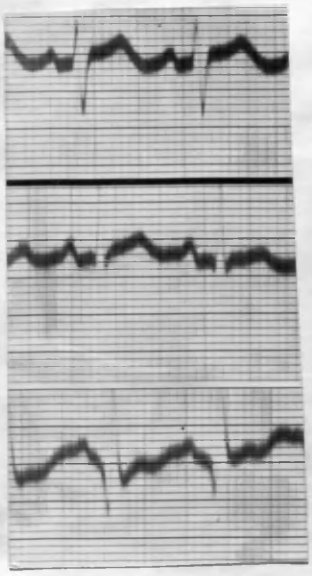


P wave notched and inverted in III.

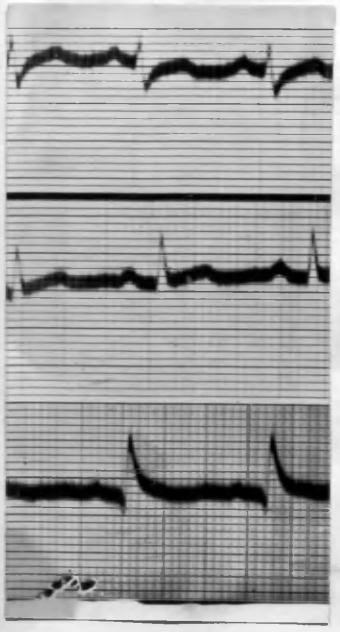
Tracing from boy suffering from acute pericarditis.
Gross generalised pericardial friction.
Consolidation of base of left lung.

13. 8. 31.

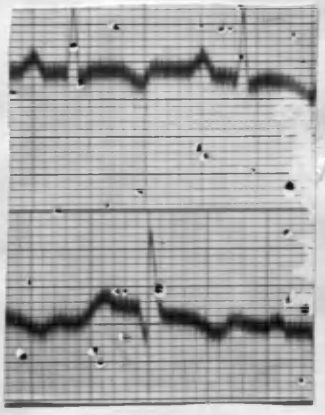
Note elevation
of RT in Lead I.



Leads II & III only.



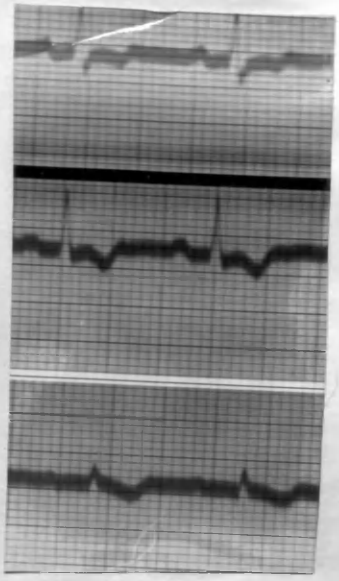
5 days later.
RT in Lead I is now
depressed.
QRS in 000 has changed.



14 days later.
Pericardial Effusion
T inverted in II & III.

Taken from patient suffering from acute rheumatism complicated by pericarditis.

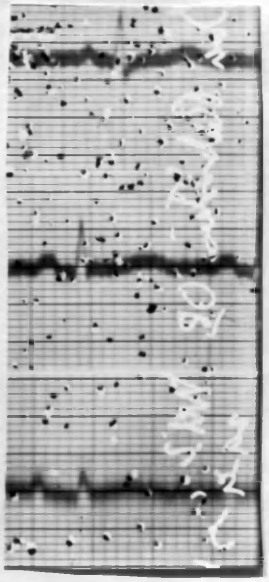
9.6.31.



T wave diphasic in I
inverted in II and III.
Small amplitude QRS in III.

Three months later

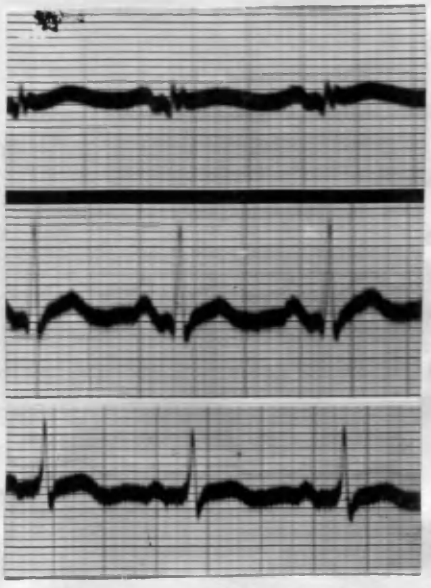
29.9.31.



T is now upright in all Leads.

Pericarditis in May 1930.

5.6.30.



Small deflection and splintered QRS in I

Slight plateauing of RT in I.

CORRELATION OF ELECTROCARDIOGRAPHIC ABNORMALITIES WITH PATHOLOGICAL FINDINGS.

A STUDY OF 17 CASES.

Once it was recognised that certain of the electrocardiograms obtained in coronary occlusion could be separated into two types designated the TI or TIII according to whether inversion of T wave occurred in Lead I or Lead III it was naturally asked if these changes were characteristic of particular pathological lesions and further, provided relationship between electrocardiographic abnormality and post mortem findings could be established would the prognostic value of the electrocardiogram be enhanced.

Within recent years attemptshave been made to correlate electrocardiographic abnormality with the site of the infarction. Any conclusions drawn from an individual study must be of limited significance.

Typical TI or TIII changes are not constantly present in coronary occlusion and indeed, are absent in the majority of cases.

It cannot be accepted that the electrical upset in the fatal case and in the case which recovers are alike. The effect of sudden occlusion must be two fold. There is first the local effect of production of infarction and the throwing out of function of part of the cardiac muscle and second the general effect of disorganisation of rhythmic cardiac contraction. This second factor or heart shock

will be personal and variable and on its presence and degree will depend the development of at least some of the arrhythmias whilst it cannot be disregarded as a possible cause of distortion of the electrocardiogram.

Parkinson and Bedford (56) were of opinion that TI or TIII type of inversion may be determined by the size and site of the infarct and its relation to the apex rather than by particular artery involved.

Barnes (57) suggested that the TI type follows infarction of the anterior surface of the left ventricle and apex and that TIII type is related to infarction of the posterior surface with or without apical involvement.

In a study of six cases I (58) found that the electrocardiograms were all abnormal but the type of abnormality was not constant. It varied from the more or less simple left cardiac preponderance to the more definite bundle branch lesions.

Ritchie and Gilchrist (59) in their survey of reported post mortem cases (44 in number) stated "that until further evidence has been acquired the form of the electrocardiographic distortion cannot be regarded as a definite localising sign of the infarct."

Cowan (60) found no characteristic electrocardiographic changes in 4 out of 12 cases of gross myocardial damage

damage examined post mortem.

I have now had the opportunity of examining post mortem 21 cases and in 17 of these I had obtained electrocardiograms.

I intend at a later date in conjunction with A. W. Harrington to make a more complete study of these cases. At present I shall be content to record my conclusions with regard to correlation briefly and with reservations. The accumulated experiences of many workers over a period of years will be necessary before the possibilities or limitations of attempted correlation can be realised.

This is intended as an addition to the previous findings of others and as a stimulus for future work. To expect finality in such a study is to seek disillusionment.

There is no mathematical accuracy in the production of infarct. Anatomical and pathological variations are frequent.

Gross (61) has shown that the anatomical distribution of the coronary arteries is variable. The right coronary artery is usually the smaller of the two and rises from the anterior aortic sinus. It passes to the right in the auriculo ventricular groove and supplies blood to the right auricle, most of the right ventricle and posterior parts of the left ventricle and of the interventricular

septum. The left coronary artery arises from the left aortic sinus and passes to the left in the auriculo ventricular groove. It divides almost at once into two main branches, the larger of which descends in the inter-ventricular sulcus while the smaller continues along the auriculo ventricular groove. These supply the anterior two thirds of the ventricular septum, most of the left ventricle and a small portion of the anterior surface of the right ventricle. The main horizontal branches are vertical; branches of the two arteries anastomose with each other at their extremities and from these rings of vessels many nutrient branches which anastomose are given off to the muscle.

Campbell (53) has done excellent work on the subject by X-raying hearts injected with barium.

He found that on the anterior surface there is surprisingly little variation in the vessels but on the posterior surface the size and distribution of the posterior artery vary greatly and that the arterial supply to any part may vary in different cases.

He confirmed Gross's findings that

(1) Anastomoses exist between the right and left coronary arteries both in capilliary and precapilliary distribution.

(2) Anastomoses exist between the branches of each coronary artery.

(3) Anastomotic distribution is irregular but abundant.

He did not investigate Gross's fourth statement that anastomoses exist between the coronary arteries and vessels from the adjacent and attached organs. He found that the neuro muscular tissue is specially safeguarded by many alternative channels of supply. In two of his cases stenosis of the specific artery to the node and bundle did not cause alteration of electrocardiogram.

He agreed with Gross that gradual obliteration brings further increase in the anastomotic supply.

Even this extensive ansatomoses is usually insufficient to compensate for occlusion of a large vessel.

Cowan (62) however, has seen at least five cases in which blocking of one of the main arteries was well compensated by enlargement of the other and only a few patches of fibrosis were found on microscopic examination.

The types of lesion produced will vary with rate of occlusion of artery. Gradual stenosis may allow of anastomotic compensation but generally leads to ischaemic fibrosis.

If the occlusion is sudden necrosis results - the extent of infarct depending on the size of the vessel involved.

The area of infarct is less than that supplied by the occluded artery - this is only partially explained

by surrounding anastomoses; Thebesian vessels play some part.

Muir (63) has demonstrated that direct blood supply from the ventricle is also a definite factor.

The electrocardiographic alterations produced by cardiac infarction will vary according to

(1) The arterial distribution.

(2) The anastomotic compensation.

(3) The presence or absence of previous fibrosis.

In none of my cases was the infarct the sole evidence of myocardial involvement. The left ventricle was enlarged and there was varying degree of myocardial fibrosis. In some of these this fibrosis was more striking than the infarct itself and without records before its occurrence one cannot assign all the electrocardiographic abnormalities to the development of infarct.

The artery occluded was not always the same.

Of 21 Autopsies :-

(1) The descending branch of the left coronary was occluded in 9.

(2) The orifice of the left coronary in 1.

(3) The lateral descending branch of the left coronary in 1.

(4) The orifice of the left coronary and the right coronary $\frac{1}{2}$" from its orifice in 1.

(5) The descending branch of the left coronary and the transverse branch of the right coronary in 3.

(6) The descending branch of the left coronary and right coronary sclerosis in 1.

(7) The right coronary orifice in 1.

(8) The transverse branch of the right coronary in 1.

(9) The artery to the bundle of His in 1.

(10) There was no macroscopic evidence of complete occlusion in 2.

In 39 of Levine's series of 46 Autopsies the vessel involved was the descending branch of the left coronary.

This preponderance of the involvement of the descending left coronary has been noted by various workers. Muir (63) attemps to explain this. He says "It seems possible that by the impinging of the heart on the chest wall at this point an artery already diseased suffers what may be called slight traumatism and thus the lesion is increased; it is certainly the case that disease in this situation is often more marked than elsewhere."

The Site and Extent of Infarct was not always the same in Obstruction of a Particular Vessel.

	ARTERY OCCLUDED.	SITE & EXTENT OF INFARCT.
I.	Descending Branch of the Left Coronary Artery.	Apex and anterior wall of left ventricle in 5 cases. Apex of both left and right ventricles in 1 case. Apex and left side of septum in 3 cases.
II.	Orifice left Coronary Artery.	Apex and anterior wall of the left ventricle.
III.	Lateral Descending Branch of Left Coronary.	Apex and anterior wall left ventricle.

	ARTERY OCCLUDED.	SITE & EXTENT OF INFARCT.
IV.	Descending Branch of Left Coronary Artery and Transverse Branch of Right Coronary.	Anterior part of left ventricle at apex. Left side of septum and posterior wall of left and right ventricles.
V.	Left Coronary Orifice and Right Coronary ~~Orifice~~ Artery ½" from its origin.	Aneurysm of apex and infarction of posterior wall of left ventricle.
VI.	Transverse Branch of Right Coronary.	Posterior wall of left and right ventricles at the base.
II.	Sclerosis of Left and Right Coronaries	Fibrosis of apex and posterior walls of both ventricles.
III.	Artery of the Bundle of His.	Infarct in the region of the bundle.

Notwithstanding the many possible fallacies, the majority of my cases have shown a definite relationship between the type of electrocardiographic abnormality and the site of the lesion.

In 6 cases (1, 2, 3, 4, 5, and 8) there was elevation of RT in Lead I of the electrocardiograms. In all of these there was occlusion of descending branch of left coronary artery with infarction involving the anterior part of the apex of the heart.

In 4 cases (10, 13, 15 and 16) there was elevation of RT in Lead III of the electrocardiograms. In all these cases there was occlusion of the transverse branch of the right coronary artery with infarction involving the posterior part of the base of the heart.

In one case (68) the electrocardiogram showed full heart block. In this case there was occlusion of one of the vessels to the bundle with small recent infarction in the region of the bundle. There was, in addition, much fibrosis of the bundle. As full heart block was present in this case for a year before death the recent infarct was not the causal factor. The only new abnormality that developed during the patient's terminal attack was electrocardiographic inversion of T wave in Lead III.

The 5 cases in which no relationship was established (6, 7, 9, 11 and 12) were not satisfactory.

In case 6 Lead I, and in case 9 Lead III only were established, whilst the tracings in cases, 7, 11 and 12 were not taken during the acute phase of illness. My experience has been that Elevation of RT in Lead I with subsequent inversion of T wave in Lead I has been associated with occlusion of the left coronary artery and infarction involving the anterior part of the apex of the heart.

Elevation of RT in Lead III with subsequent inversion of T wave in Lead III has been associated with occlusion of the transverse branch of the right coronary artery and infarction involving the posterior and basal part of the heart.

Interesting clinical pathological problems arise.

There is a preponderating involvement of the descending left coronary artery post mortem and yet the TI and T III types of electrocardiographic abnormalities are almost equal in the recovery cases.

Can one assume that the prognosis is better in infarction involving the posterior and basal part of the heart and would careful pathological investigation reveal much more frequent evidence of previous infarction of this region?

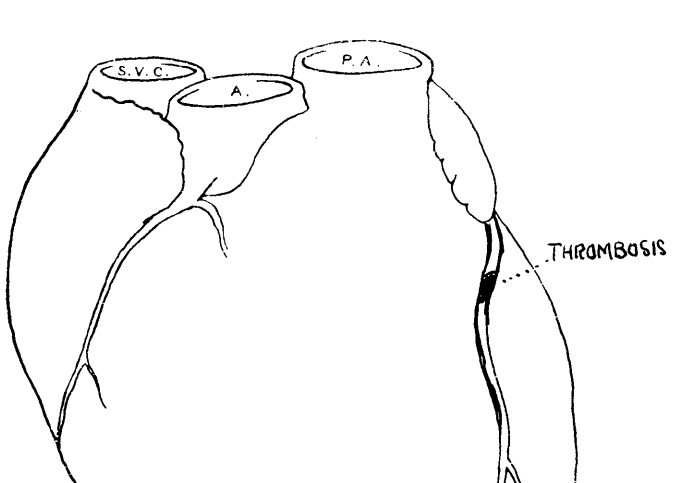
Case No. 1.

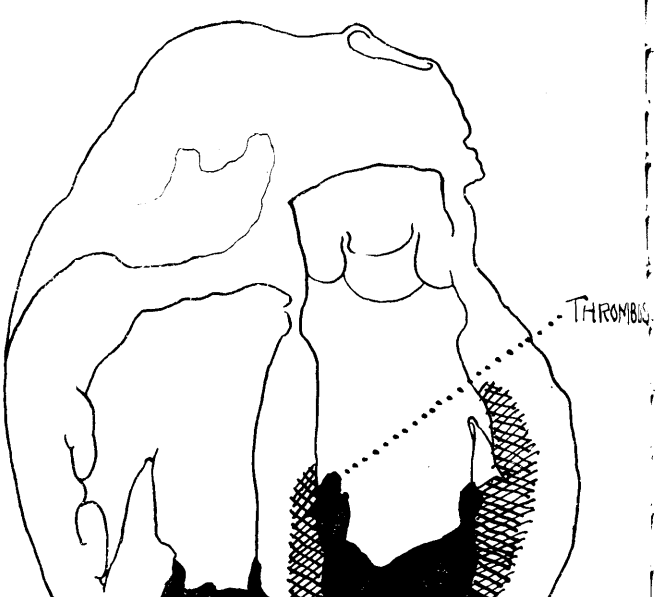
Post Mortem Summary.

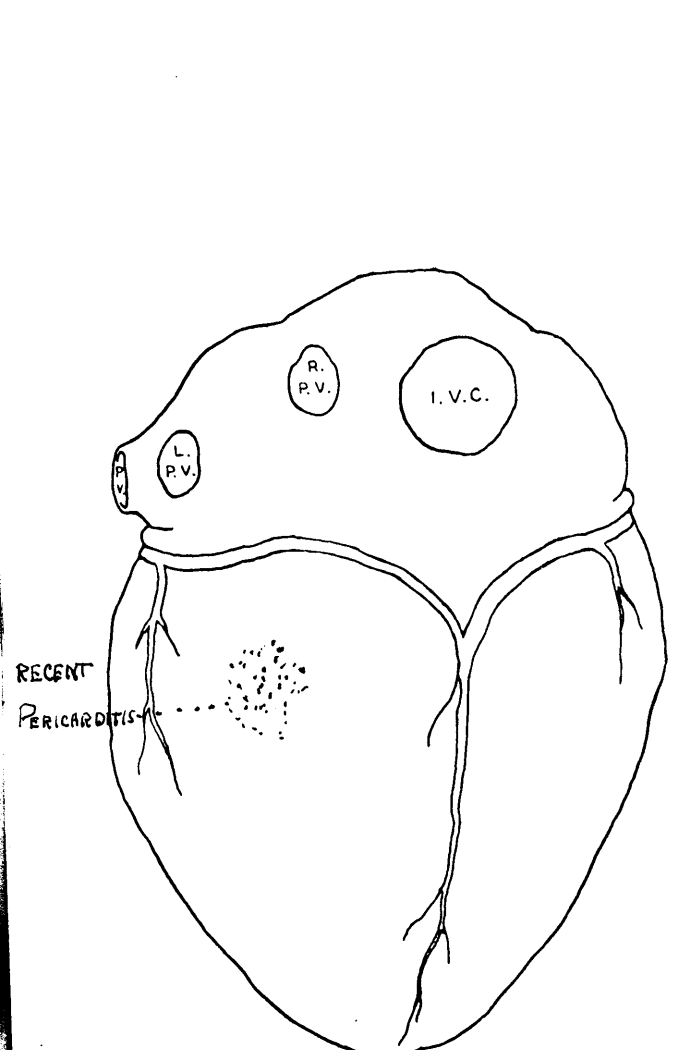
The heart was greatly enlarged - the left ventricle showed considerable hypertrophy.

There was almost globular bulging of lower two-thirds of the ventricle. The surface showed purplish staining; there was marked softening.

On opening the heart an infarction was found involving the lower one third of the left ventricle wall; left side of lower part of septum and right ventricle at apex. The coronaries were atheromatous and the left descending branch was completely blocked by thrombus of about one inch from its origin.


S.V.C.
A.
P.A.
THROMBOSIS


THROMBOSIS
INFARCTION

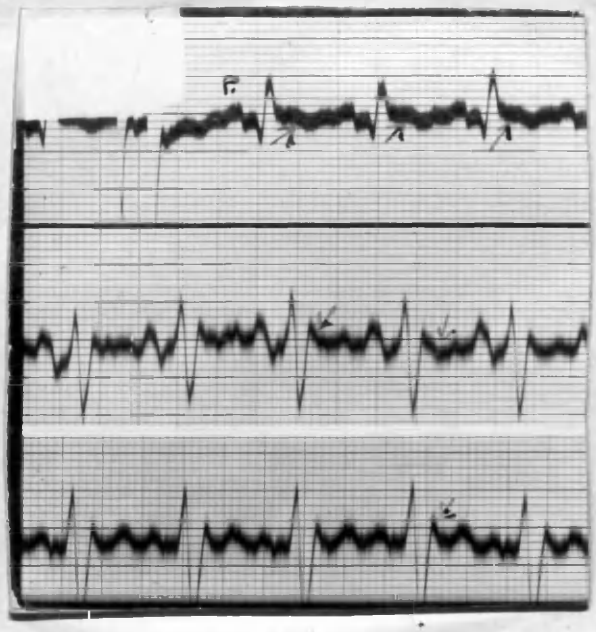

R. P.V.
I.V.C.
L. P.V.
P. V.
RECENT
PERICARDITIS


INFARCTION

CASE NO. 1.

ELECTROCARDIOGRAPHIC RECORDS.

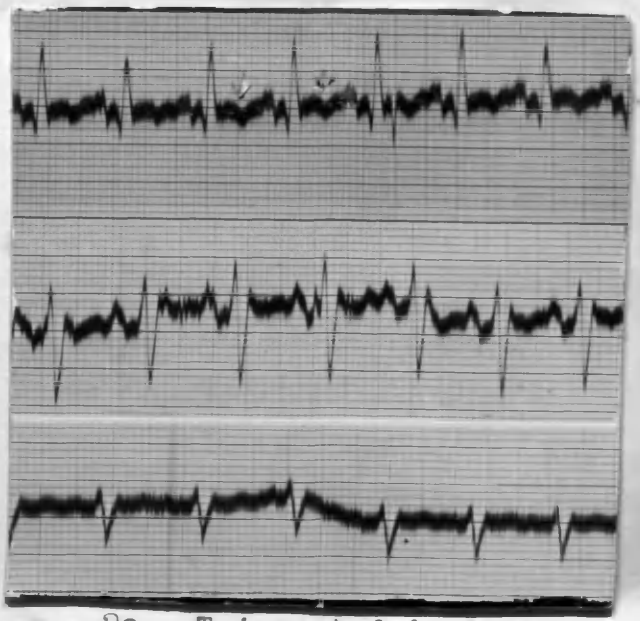
14.1.27



P notched in I, II, III.
PR = .16 QRST = .40 - .36
QRS = .16 RT of "Pardee" type in I
RT depressed in II & III

Left Cardiac Preponderance.

17.1.27



PR = .20 T inverted in I
QRST = .36
Q + in I
P notched in I & II
Left cardiac preponderance.

Case No. 2.

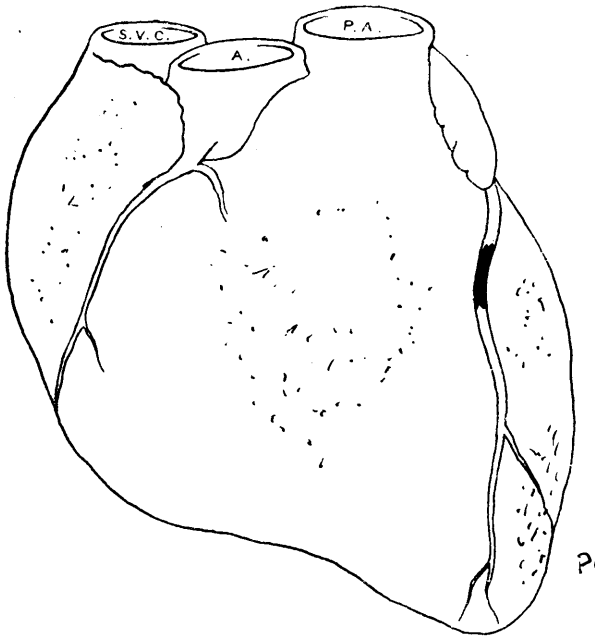
Post Mortem Summary.

The heart was moderately enlarged weighing 16 ozs. There was slight excess of purulent fluid in the pericardial sac and a patchy recent pericarditis involving the apex of the left ventricle and the upper part of the right ventricle posteriorly to a slight extent. The lower third of the left ventricle was of a peculiar salmony tinge, soft in consistency and bulged outwards to a slight degree. On opening the heart an infarction of the lower third of the left ventricular wall corresponding to this area described was found. There was thrombus adherent to it on its inner aspect. The whole ventricular wall presented a peculiar mottled appearance.

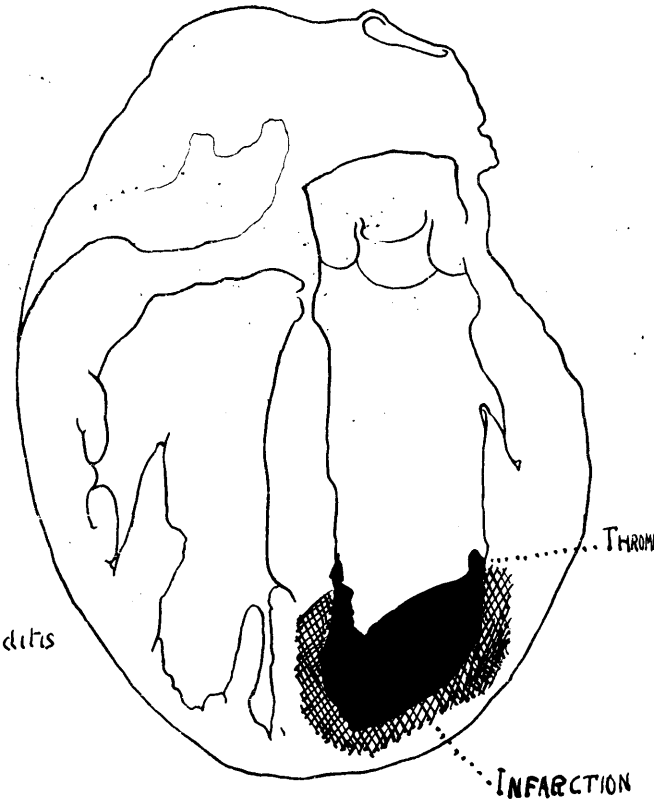
The coronaries were atheromatous and a large calcareous plaque with thrombus was found a short distance from the orifice of the left main descending coronary branch. The valves were apparently healthy.

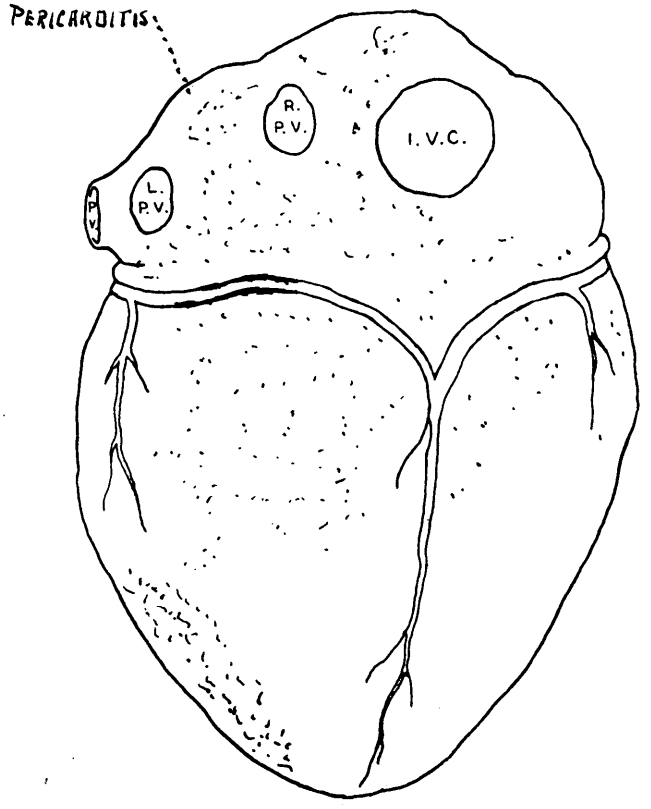
The left lateral transverse branch was sclerotic. Other points worthy of note were the presence of advanced chronic interstitial nephritis, and fibrosis of the pancreas. There was hypostatic congestion of the lungs, chronic cholecystitis and gastro-intestinal catarrh.

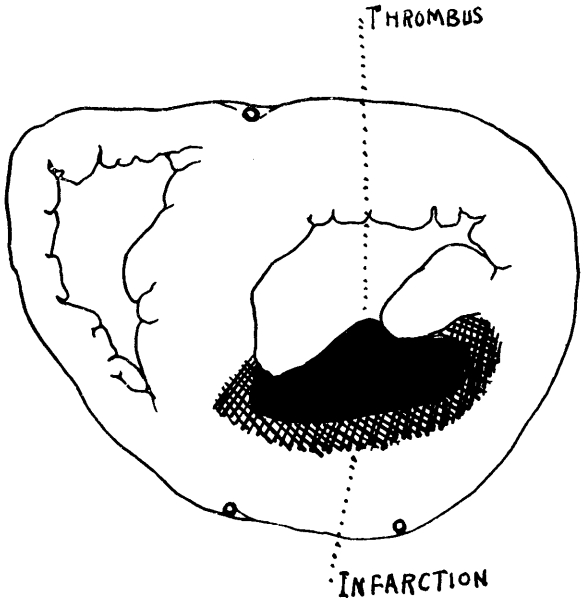
Recent Pericarditis



old Pericarditis

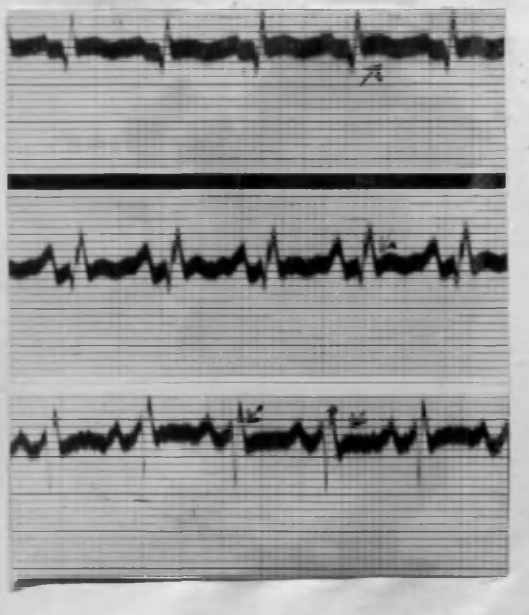




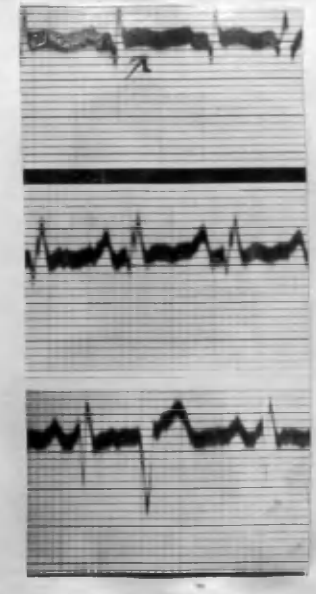


Case No. 2.

5. 8. 29.



6.8.29.



RT interval above the base line

Depression in III.

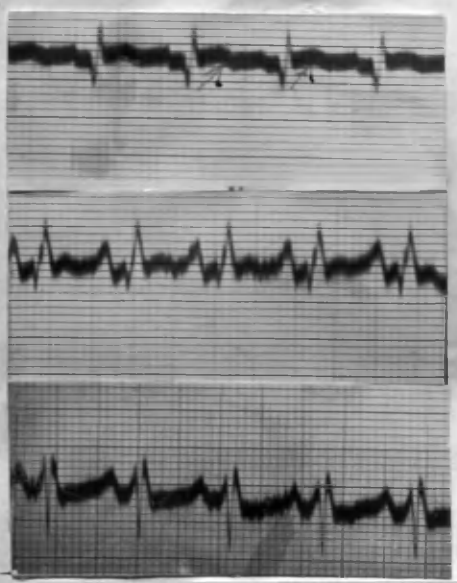
PR = ·20

Splintering of QRS in II.

Cowan Wave in III

Left Ventricular Extra Systole.

7. 8. 29.



Case No. 3.

Andrew McAlpine.

External. The body is that of a well built but poorly nourished man. There is a notable pallor of the body surface.

Thorax. Heart. There is a slight excess of blood-stained serous fluid in the pericardial sac. The heart is arrested in diastole. There are some patches of old pericarditis over both auricular and ventricular surfaces. A large recent area of pericarditis has produced a fairly firm adhesion between visceral and parietal pericardial layers over the anterior aspect of the lower third of the left ventricle. All the valves appear normal. There is hypertrophy of both ventricles with dilatation - the right side being most notably involved. Both auricles are dilated and there is marked subendocardial fibrosis on the left side. The myocardium shows old and recent infarctions. The left ventricle near its base shows extensive areas of fibrosis - the anterior wall and anterior half of the inter-ventricular septum, with the greater part of the lateral wall show a patchy but almost confluent fibrosis involving the inner two thirds of the musculature. Approaching the apex roughly in the middle third of the ventricle - the fibrous area extends posteriorly to involve two-thirds of the inter-ventricular septum, while on the lateral ventricular wall the scarring is not so obvious. The apical third of the

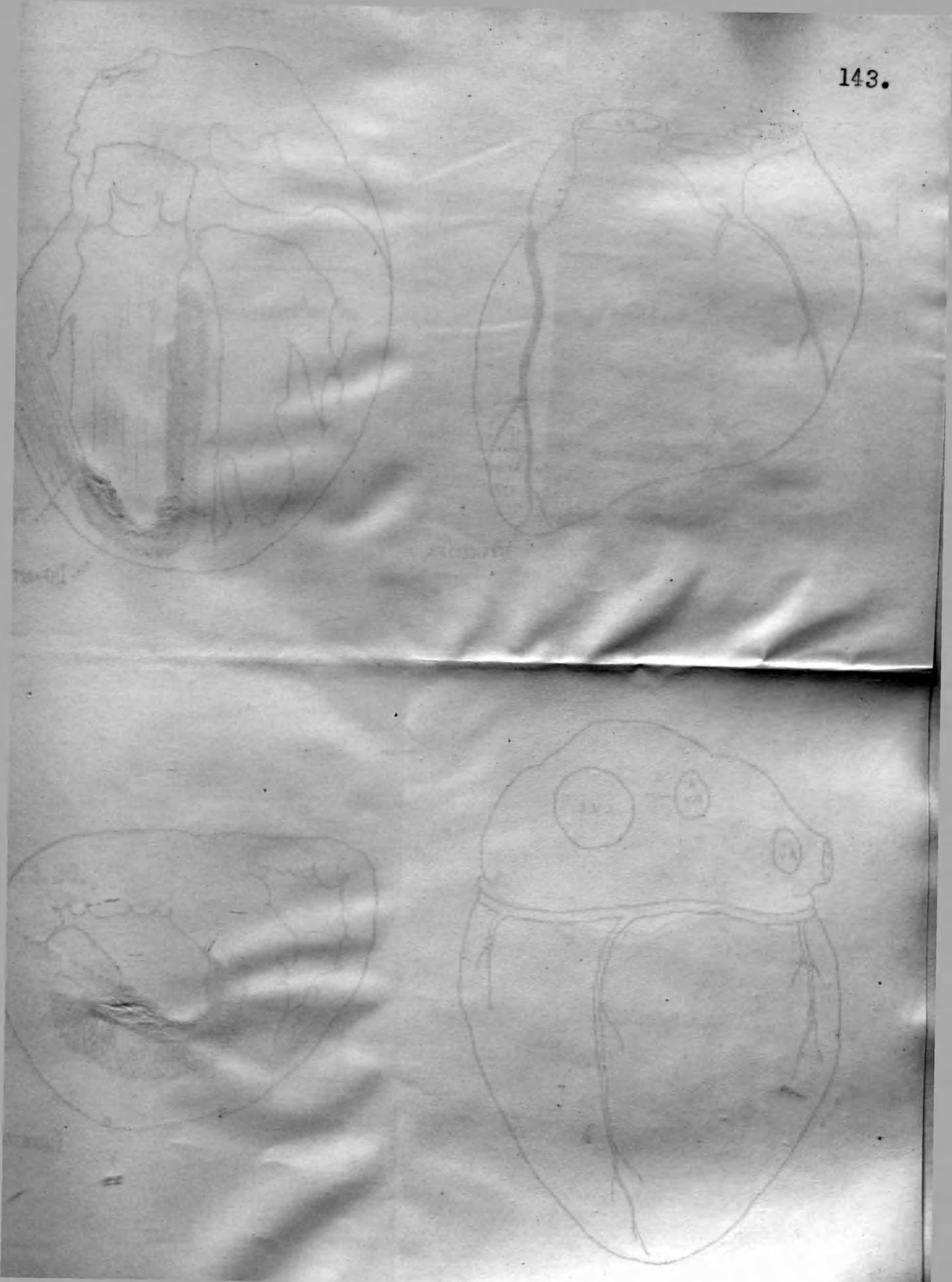
left ventricle (covered with adherent pericardium) shows continuity of the old-standing fibrosis with a recent infarction which involves practically the whole muscular wall. Recently organised thrombus is adherent to the endocardium over the apex and adjoining area of the ventricle. There is a generalised sub-endocardial fibrosis at the left ventricle most marked on the anterior and septal walls.

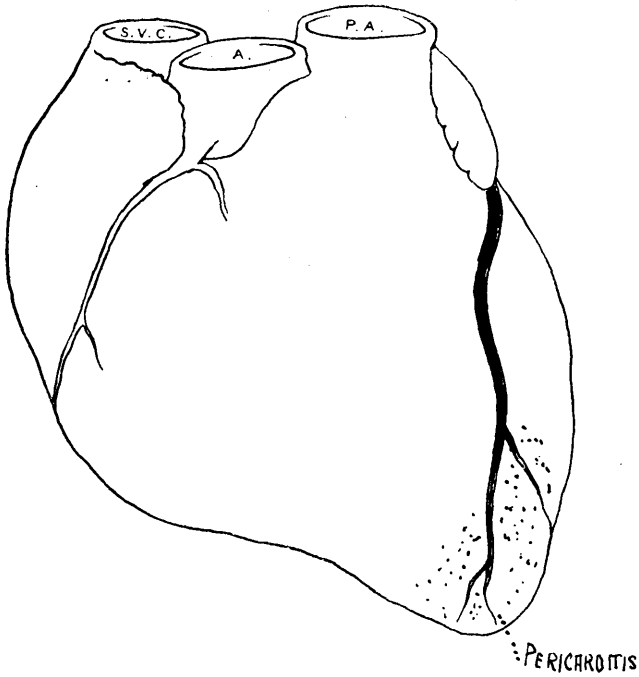
The coronary orifices appear normal although a few small atheromatous patches are evident in the neighbouring aortic wall. The left descending coronary artery shows a considerable atheromatous patch covered with recent thrombus which completely flocks the lumen about ¼" from its origin. Traced distally the lumen of the vessel shows an old standing complete blockage about 1" below the recent obstruction, beyond this the lumen of the artery cannot be appreciated.

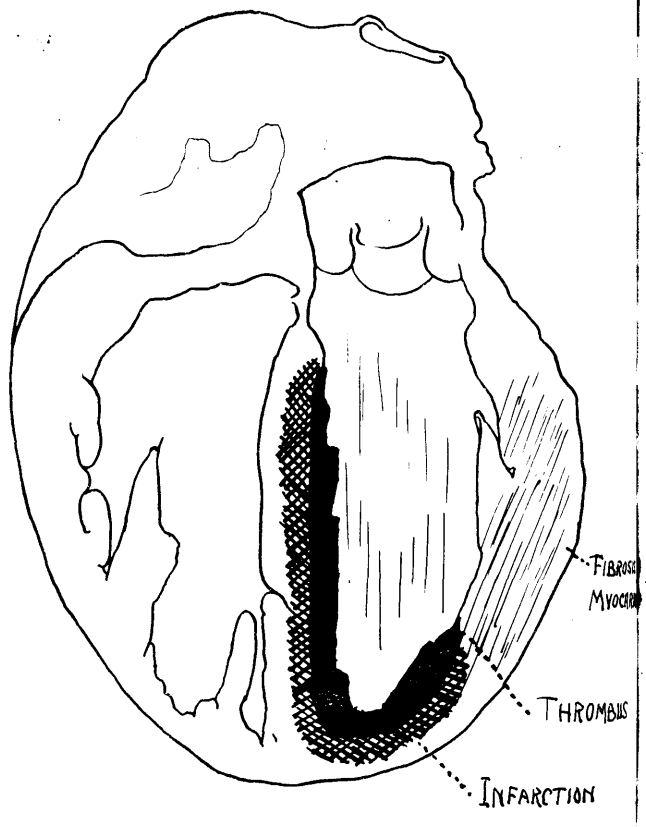
The right coronary vessel is dilated, sclerotic and atheromatous. The artery bifurcates about 1" from the posterior-inter-ventricular septum and the transverse branch shows a marked degree of narrowing. The descending branch shows nothing of note.

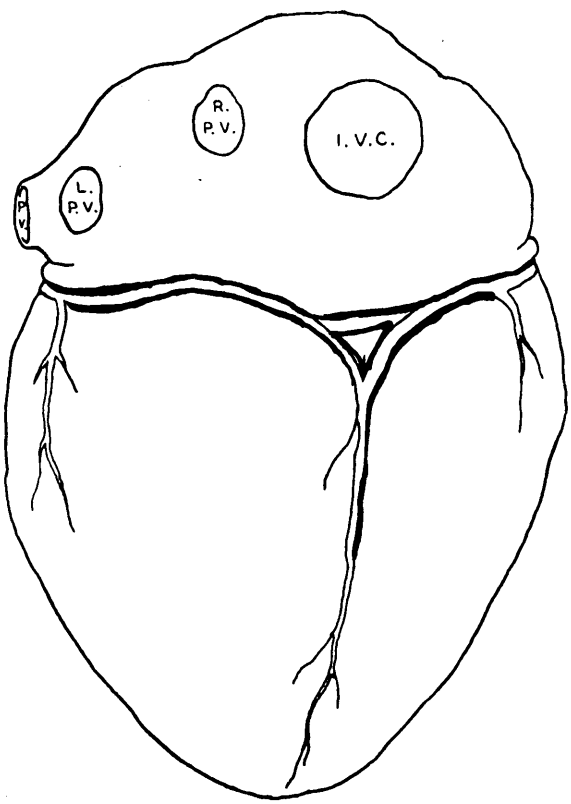
The aorta is notably sclerotic and slightly atheromatous.

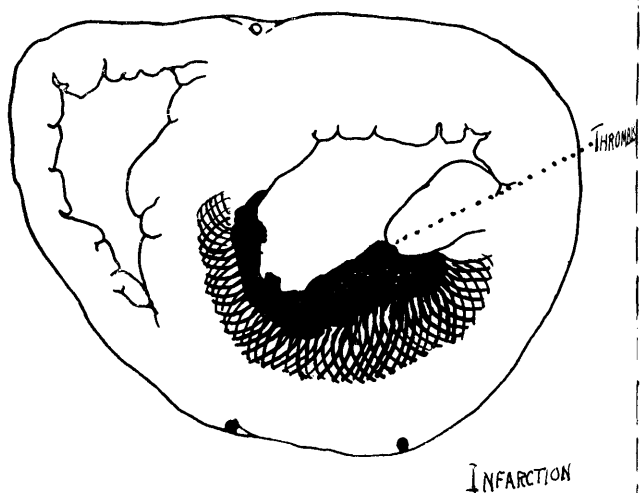
Lungs. Both lungs show chronic bronchitis, emphysema and apical scars. There is intense oedema of both upper lobes.




S.V.C.
A.
P.A.
PERICARDITIS

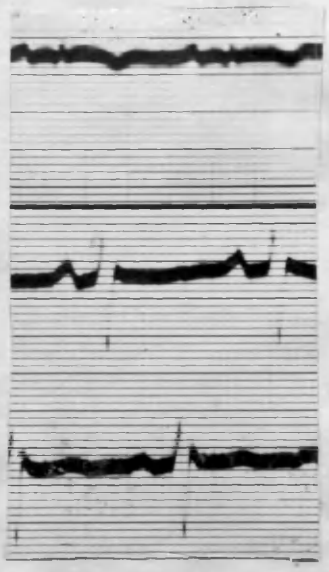

FIBROSED MYOCARDIUM
THROMBUS
INFARCTION

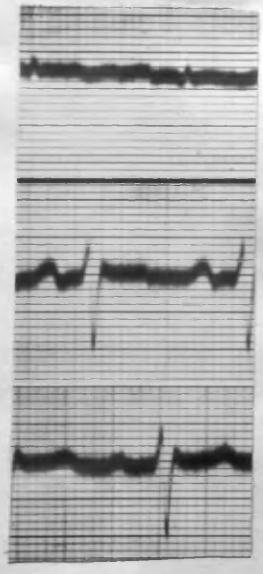

R. P.V.
I.V.C.
L. P.V.
P.V.


THROMBUS
INFARCTION

Case No. 3.

6.3.30.

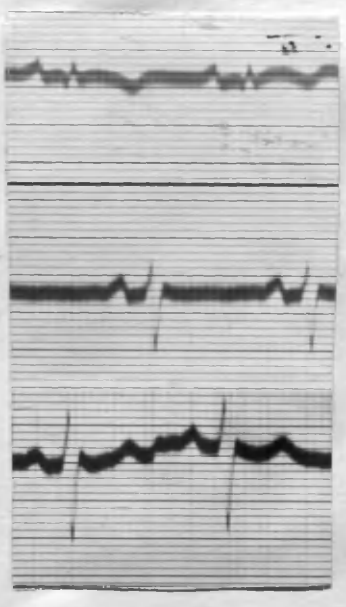


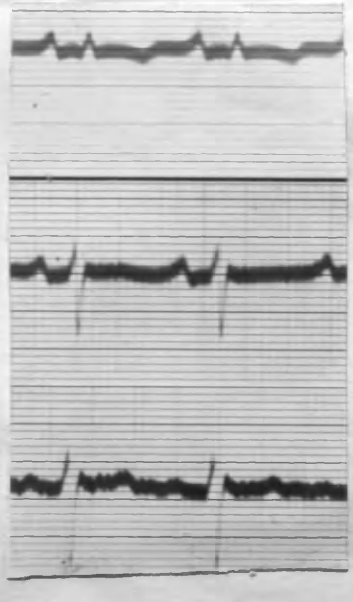


7.3.30.

Small amplitude QRS in I.

9.3.30.



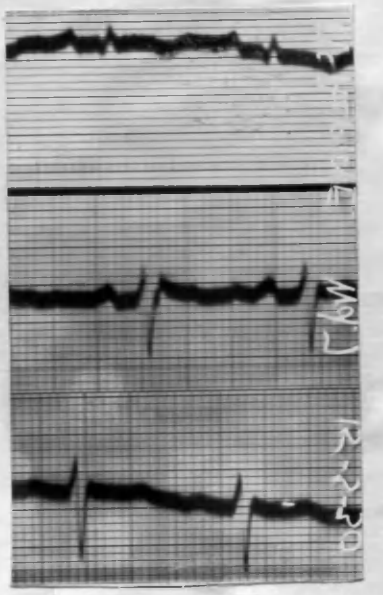


10.3.30

T in I definitely inverted.

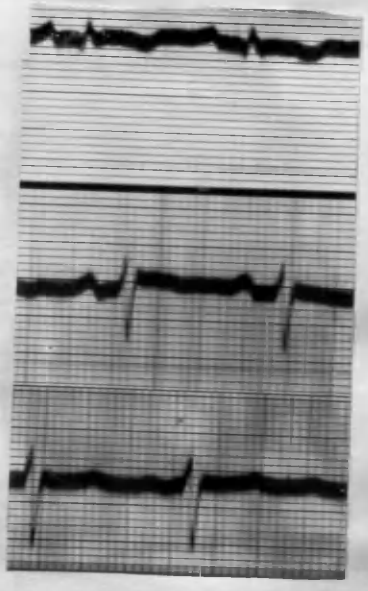
Case No. 3.

12. 3. 30



Left Ventricular preponderance
Small amplitude QRS in I
RT slight elevation in I & II.
T wave absent in II.

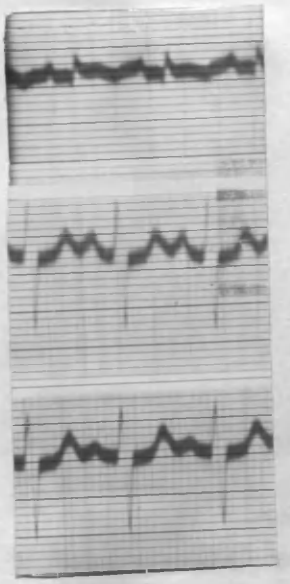
15. 3. 30.



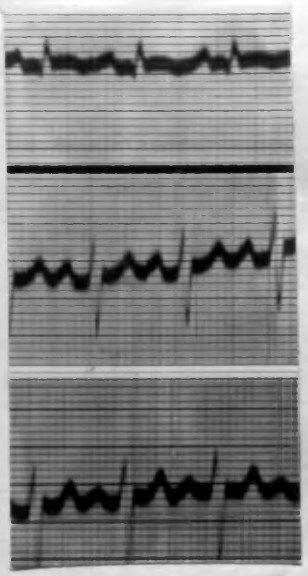
T in I more definitely inverted.

Case. No. 3

19. 2. 30



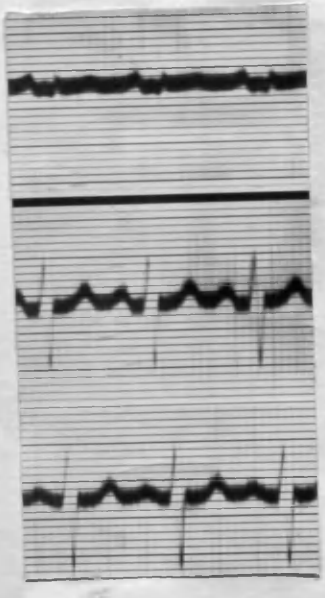
20. 2. 30



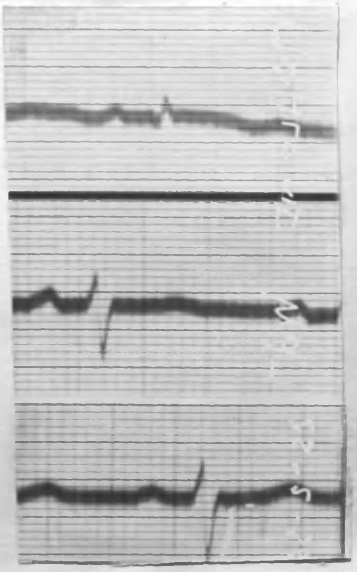
Elevation of RT in I

T wave present in II.

21. 2. 30



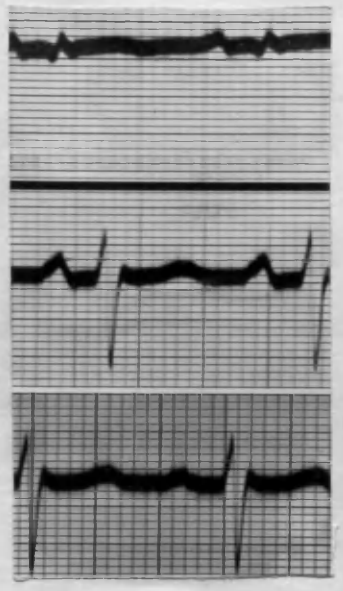
25. 2. 30.



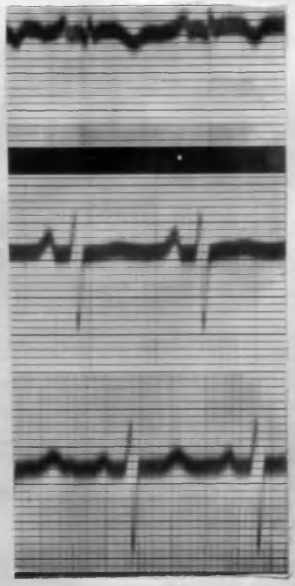
Small amplitude QRS in 1.

Case No. 3.

27. 2. 30



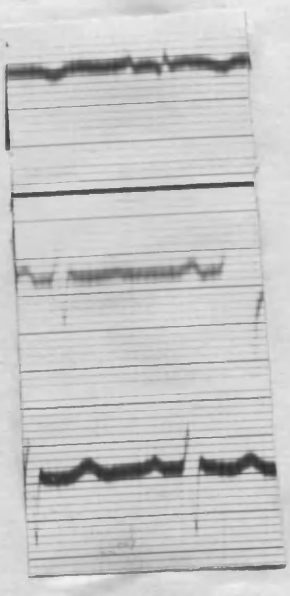
2. 3. 30



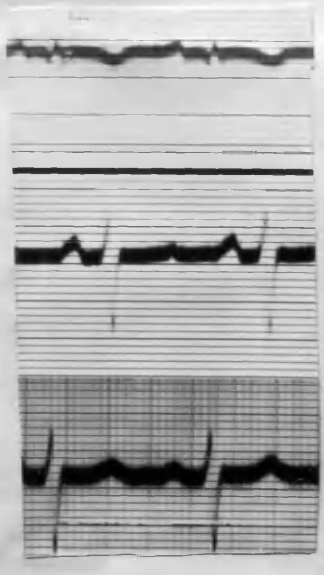
Inverted T in I

Flattening on T in II

4. 3. 30



5. 3. 30

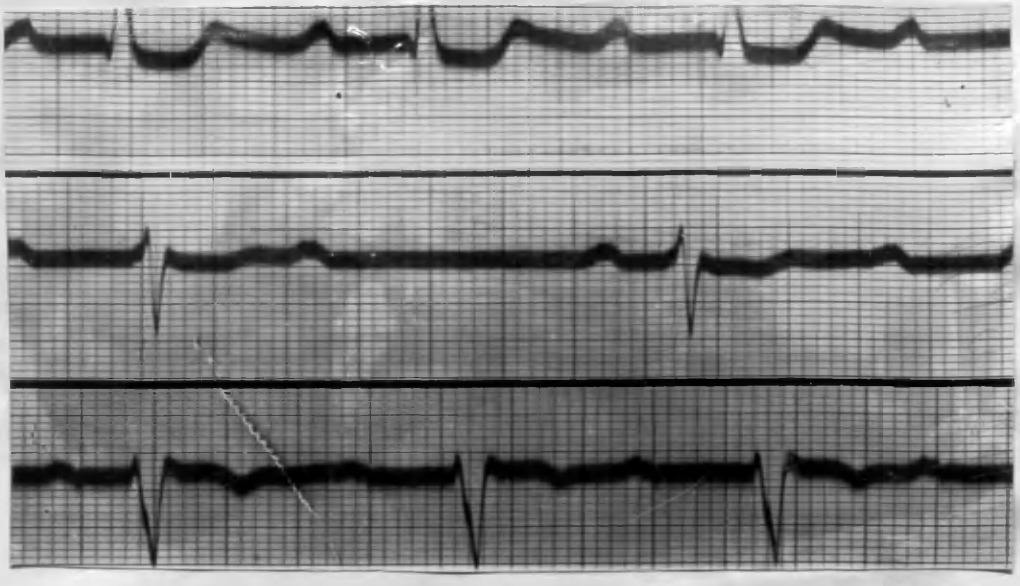


T wave again present in II

POST MORTEM SUMMARY.

Excerpt from British Medical Journal of 11th April 1931.

A post-mortem examination was made by Dr. Alice J. Marshall. The heart was soft and there was marked dilatation of the right auricle and right ventricle. There was an infarction of the muscle of the left ventricle involving the whole of the apical region and extending outwards for at least two inches on the anterior wall. In the left lateral wall of the left ventricle there was also a small recent infarction which lay midway between apex and base, in the line of the left lateral descending branch of the posterior coronary artery. Old thrombus was adherent to the inner surface of the left ventricle over the infarcted area. There were no gross changes in the valves, but the aorta showed advanced syphilitic aortitis. The orifices of the coronary arteries were of normal calibre, but the vessels were atheromatous. The right coronary artery was patent throughout its course, though its walls were much thickened. There was marked narrowing of the main descending branch of the left coronary artery, three-quarters of an inch from its orifice, while the lateral descending branch was completely obstructed by old thrombus about one inch from its orifice.



Left ventricular preponderance.

RT elevation in III

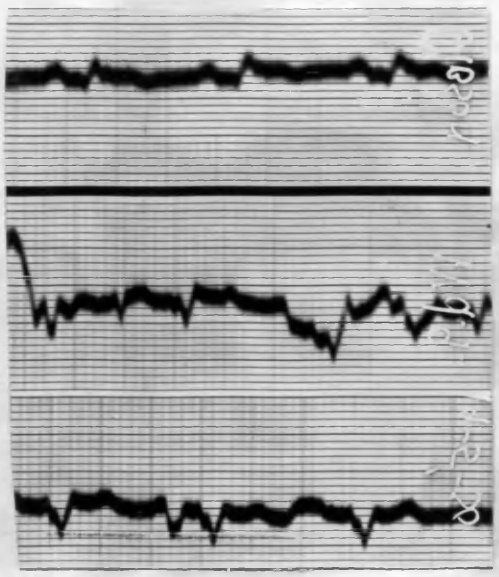
depression in I

Potential Heart Block PR = .32

In Lead II Partial Heart Block.

Case No. 4.

19.5.30.



Small amplitude QRS.

Left ventricular preponderance.

RT elevation in I.

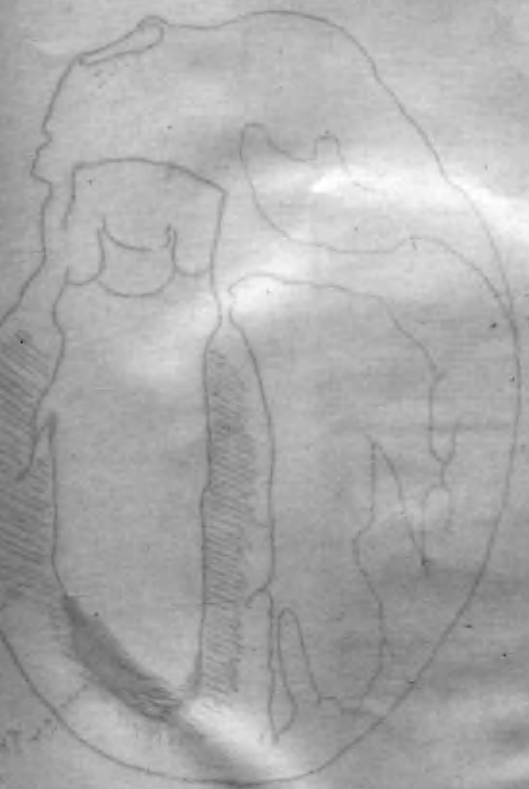
Case No. 5.

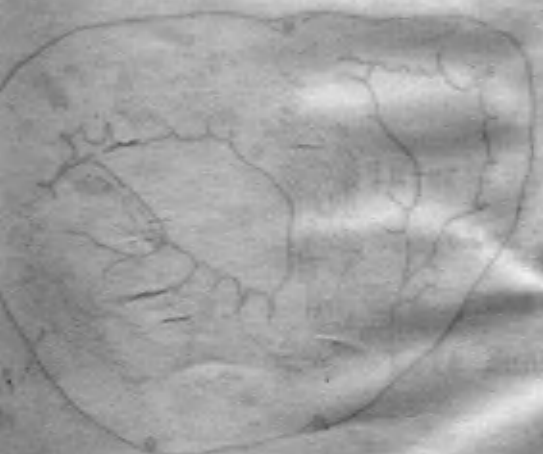
Post Mortem Summary.

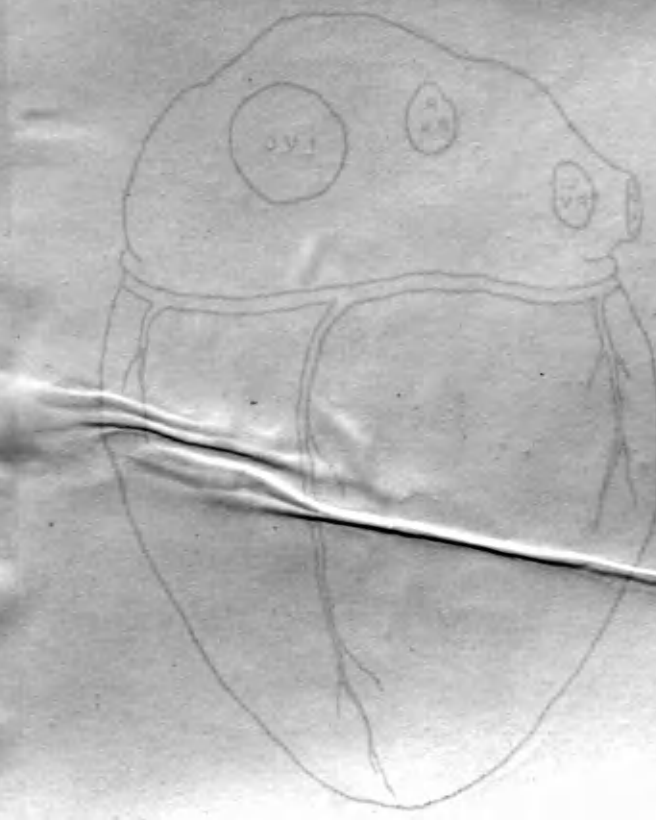
The heart was enlarged. There was thinning of apex with a patch of recent pericarditis the size of a sixpence.

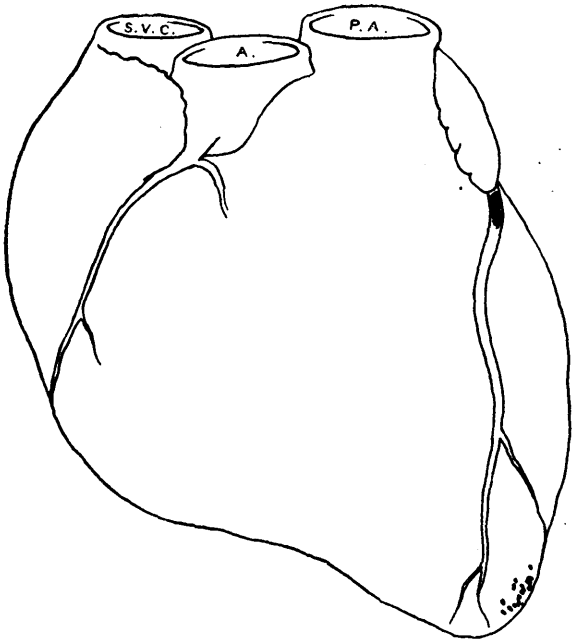
Descending branch of left coronary blocked by thrombus organised to wall of vessel ½" from origin. Transverse branch healthy with patent right coronary 1" beyond its origin, was narrowed and atheromatous; completely occluded for 1". Aorta healthy.

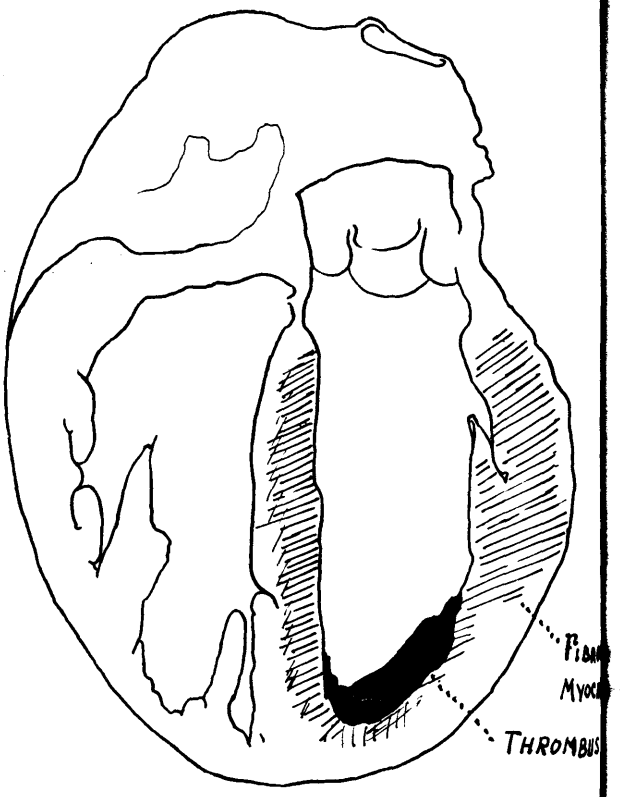
The myocardium at apex of left ventricle showed cloudy swelling and necrosis. There was generalised myocardial fibrosis, affecting chiefly the left ventricle.

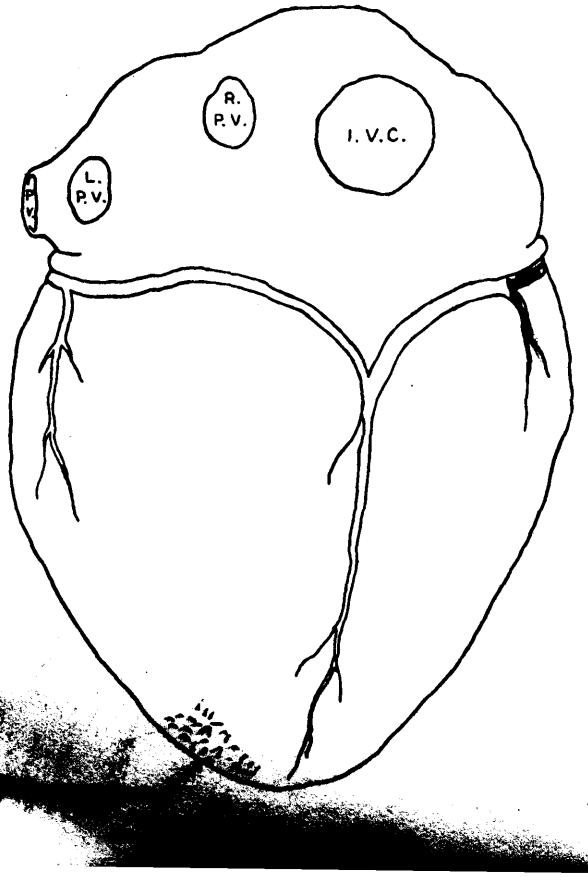


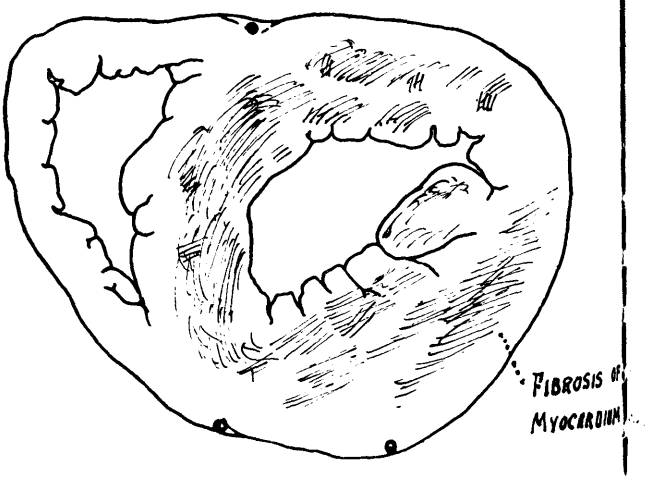




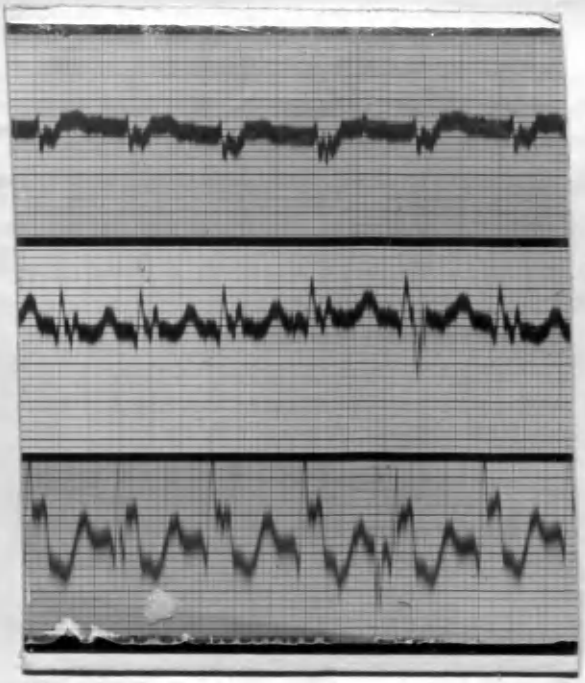

S.V.C.
A.
P.A.


THROMBUS


R.
P.V.
I.V.C.
L.
P.V.


FIBROSIS OF
MYOCARDIUM

Case No. 5



Pulse Rate 116 p.m.

PR = .24 secs.
QRS = .16 secs.

Inversion of QRS in I

Elevated RT interval in I

2/1 Auricular Flutter ?

Left Bundle Branch Block.

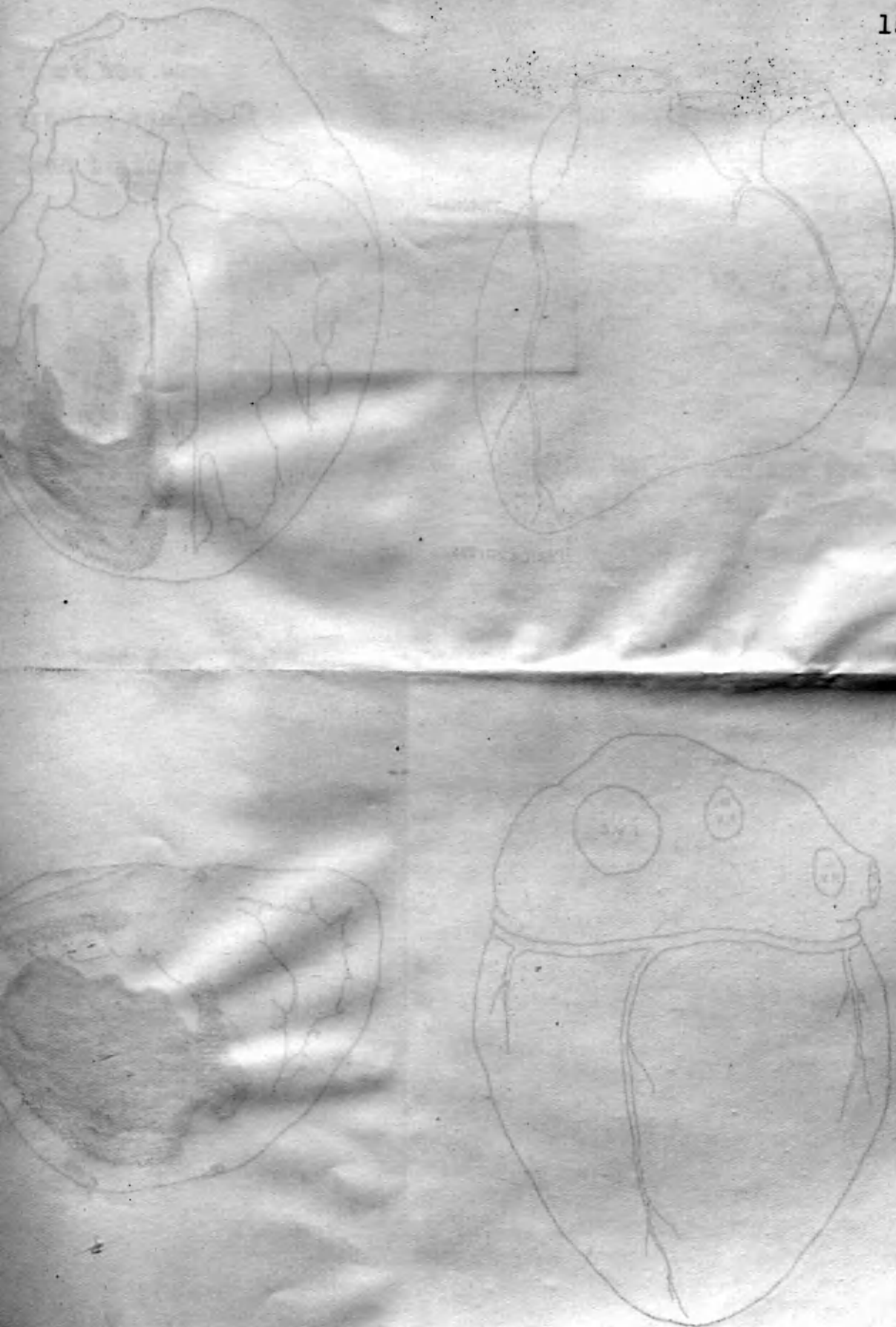
Case No. 6.

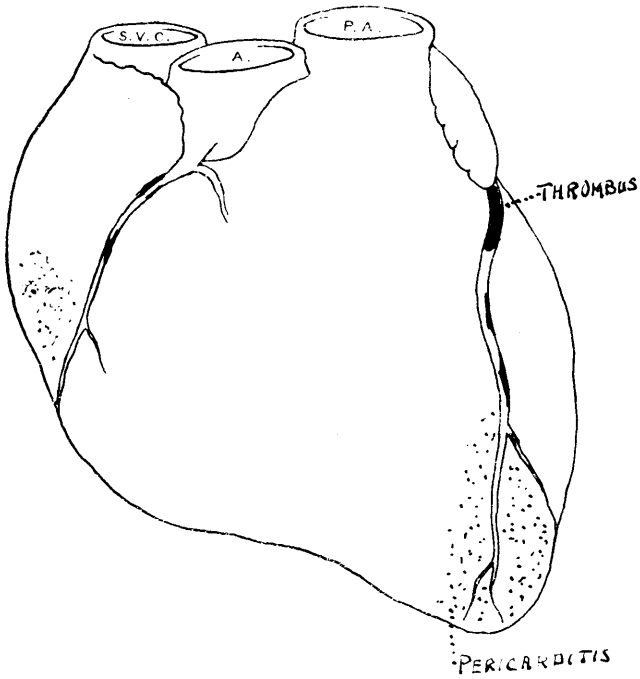
Post Mortem Summary.

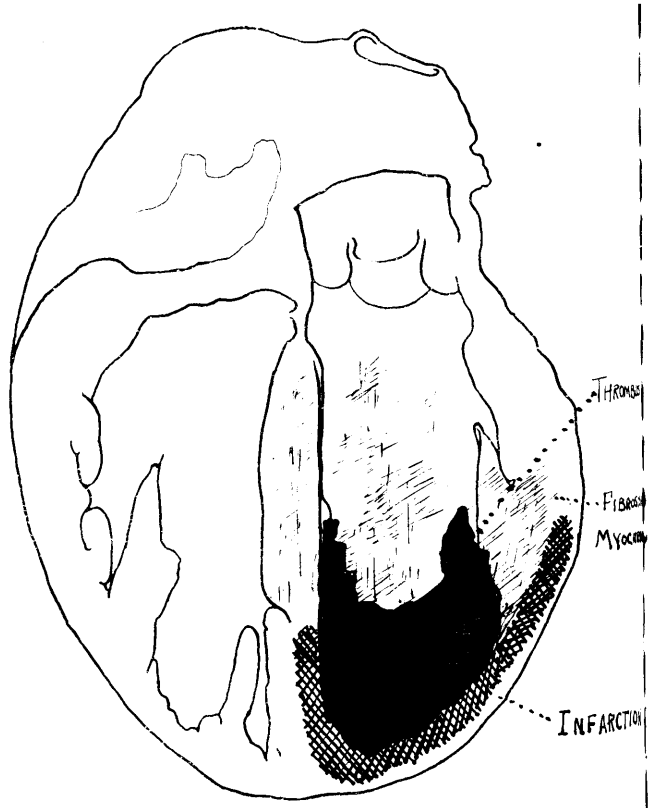
Heart was enlarged.

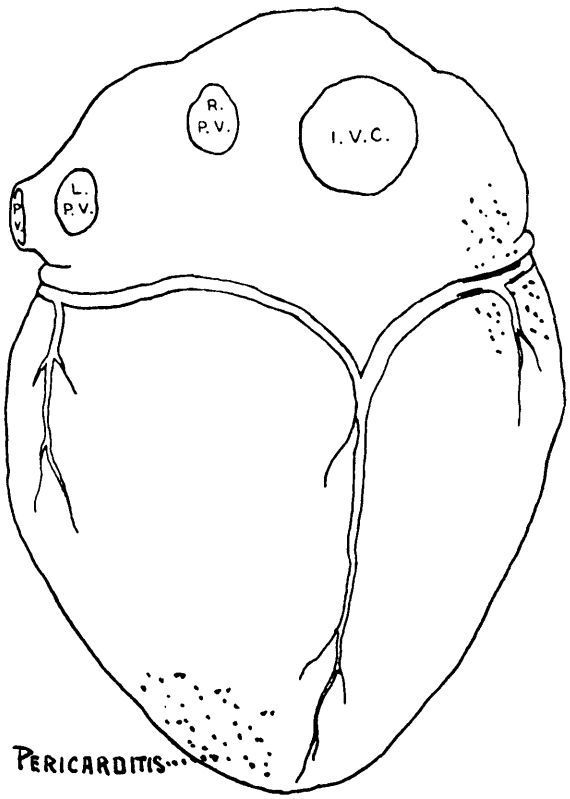
On anterior surface of left ventricle is old pericarditis. Left ventricle is half full of ante mortem clot. Heart well thinned and divided into layers. Myocardium thin, pale, and degenerate. Aorta atheromatous.

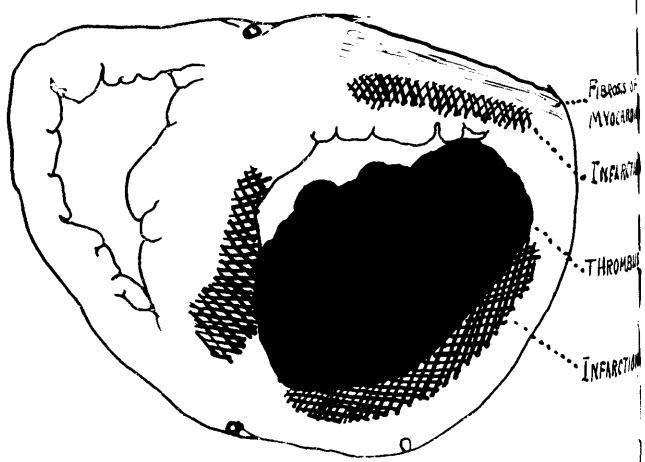
There was marked atheroma of aorta. The pericardiac sac showed much recent pericarditis. There was old pericarditis on anterior surface of left ventricle. Descending branch of left coronary artery was blocked by thrombus about 1" from its origin. The left ventricle was full of ante-mortem clot. There was infarction of the apex of left ventricle, the infarction involving to a slight degree the left side of septum. The heart wall in this region was thinned and divided into layers.



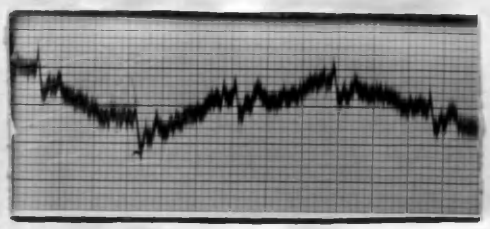

S.V.C.
A.
P.A.
THROMBUS
PERICARDITIS


THROMBUS
FIBROSIS OF MYOCARDIUM
INFARCTION


R. P.V.
I.V.C.
L. P.V.
P. V.
PERICARDITIS

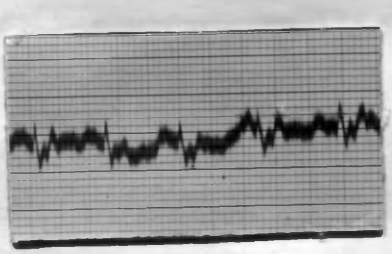

FIBROSIS OF MYOCARDIUM
INFARCTION
THROMBUS
INFARCTION

There was some difficulty with the apparatus at this time, and no tracing was taken until ten days after the delirious attack. Lead I alone could be taken.

Lead I  17. 7. 29.

QRS is splintered and prolonged .12 secs.

T wave absent.

Lead I  25. 7. 29

PR = .16 secs.

QRS = .12 secs.

There is no definite T wave but rather a tendency to downward plateauing.

P wave is notched.

Post Mortem Summary.

Heart greatly enlarged. Hypertrophy and dilatation ofboth ventricles. Several patches of pericarditis.

(a) Anterior wall of right ventricle at base.

(b) Apex.

(c) Posterior wall of left ventricle at base.

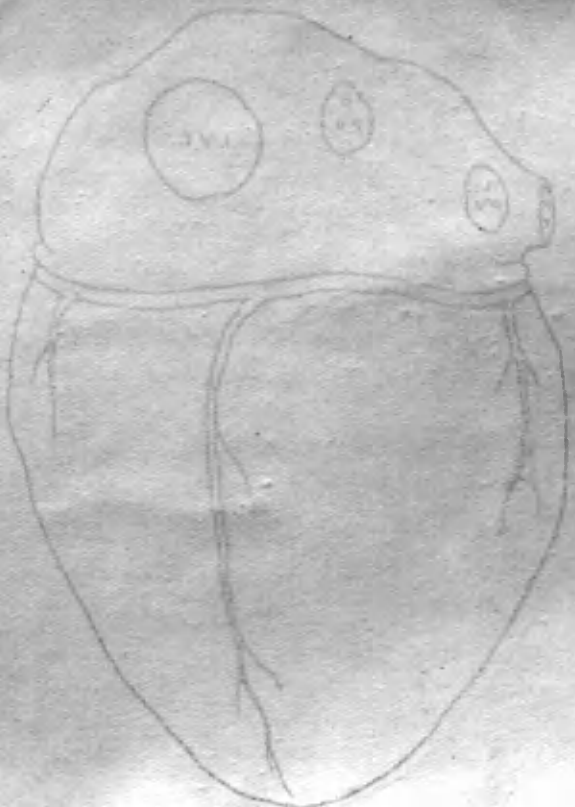
Myocardium soft - cloudy swelling and fibrosis.

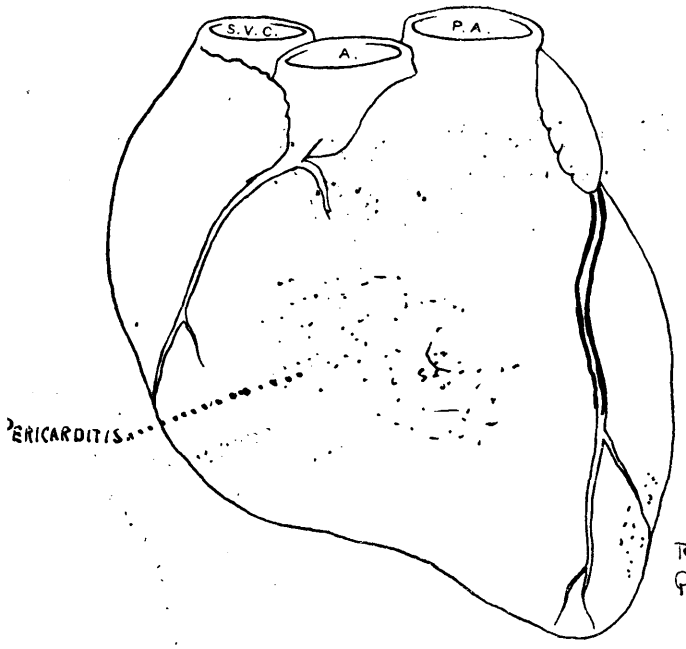
Endocardium - thickened white patches.

Papillary muscles of left ventricle fibrosed.

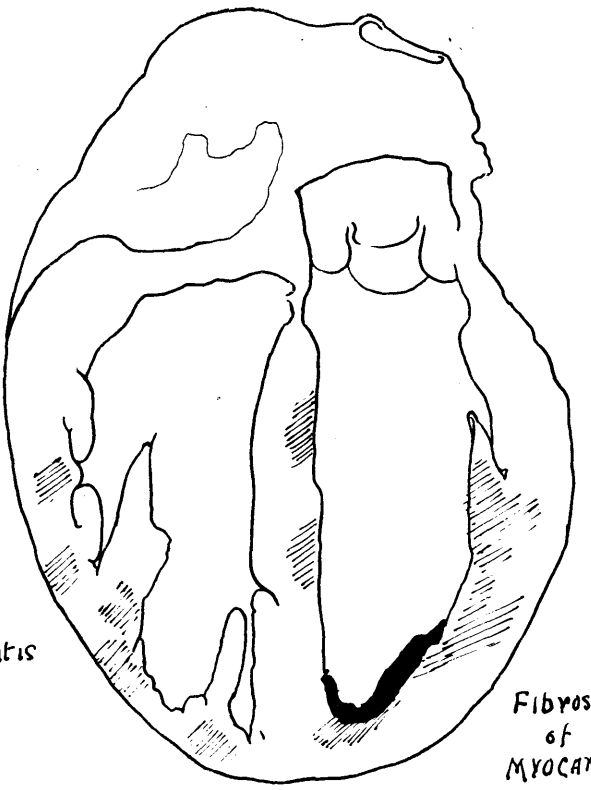
Right coronary - almost occluded 1½" from orifice by calcareous material, and degeneration involves the whole descending part.

Left coronary - Transverse Branch greatly narrowed, descending branch 1" from origin shows extreme sclerosis.

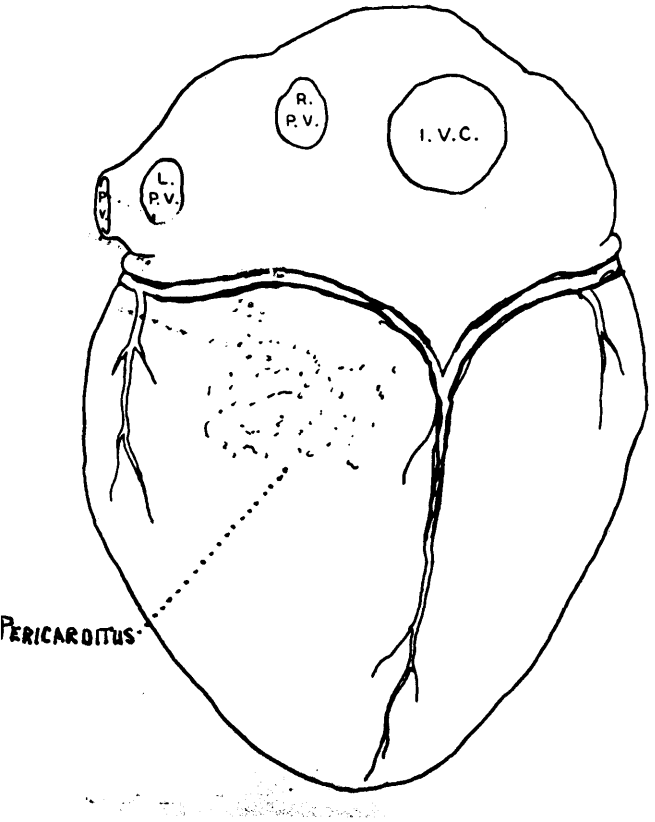


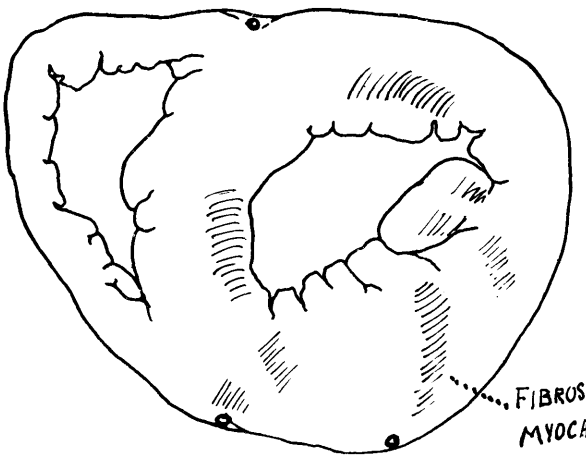


Recent Pericarditis



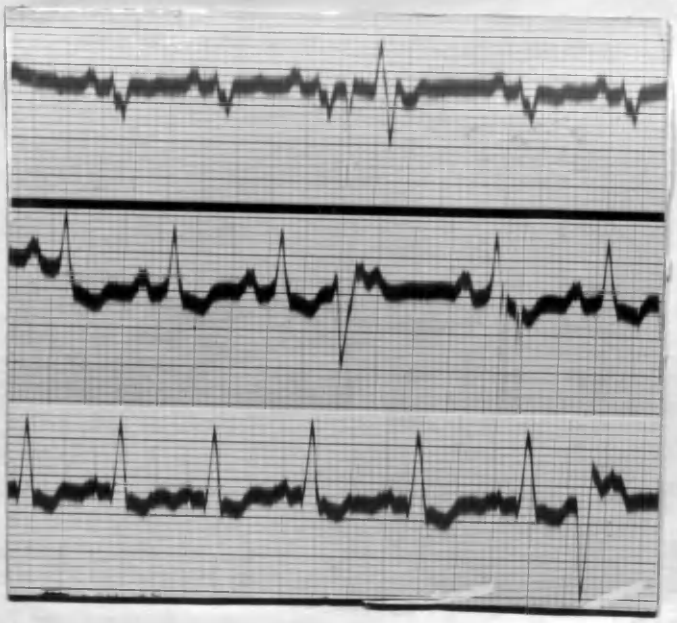
Fibrosis of Myocardium





Case No.7.

21. 8. 28.



P notched in I, II, III

PR - .20 secs.

RT = .32 secs.

QRS - .12 secs.

QRS splintered in I

T inverted in II and III.

Left Bundle Branch Block ?

Irregular left Ventricle Extra Systoles.

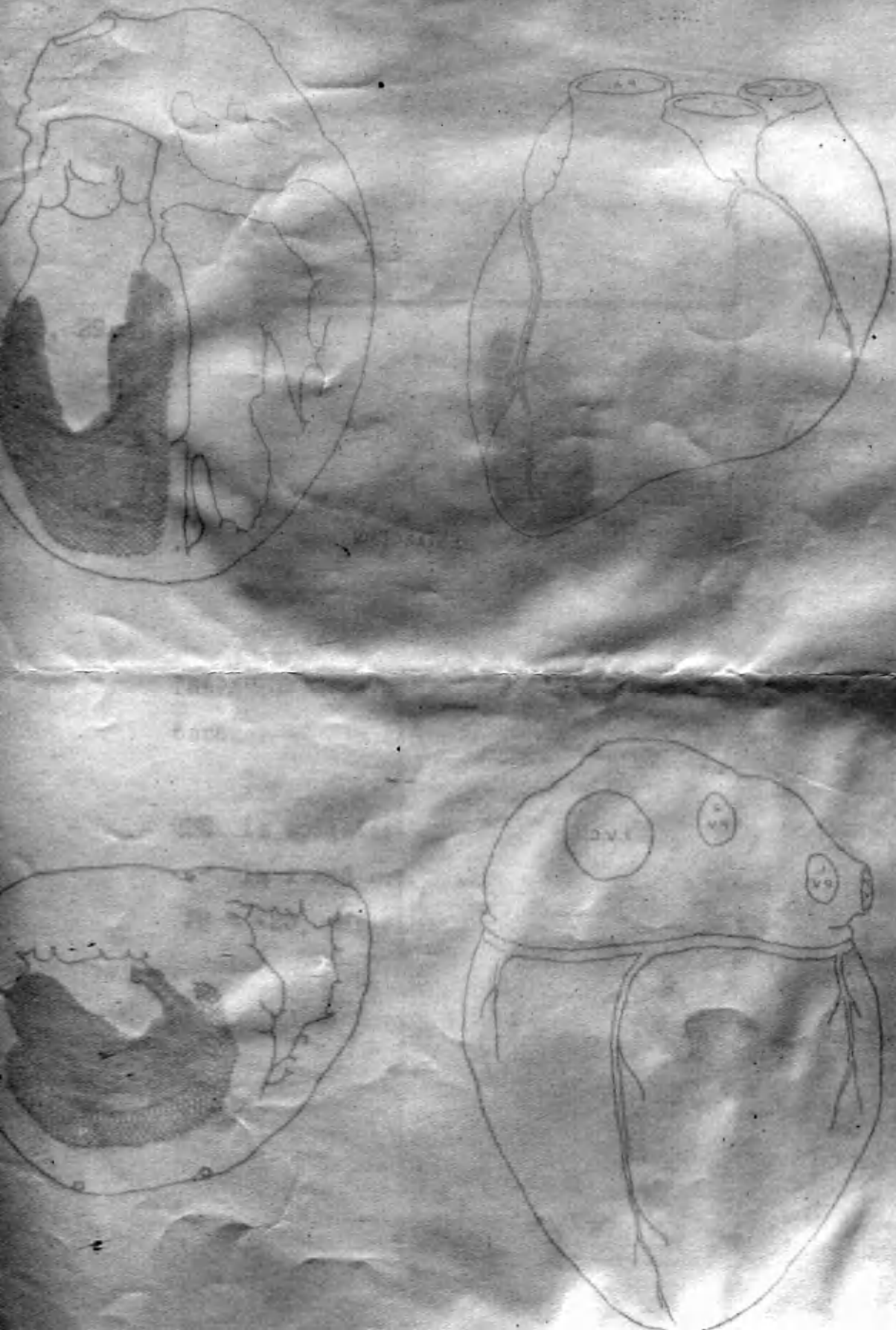
8. VI. 29.

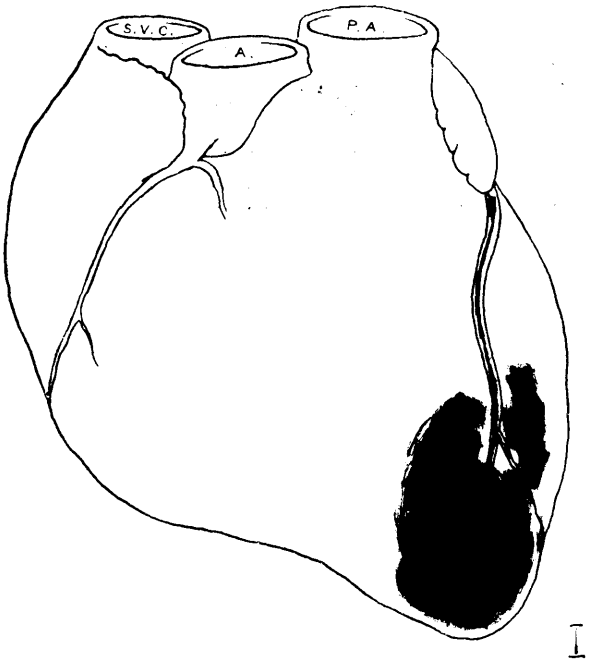
The body is that of a well developed man. There is a 3" operation scar in the epigastric region.

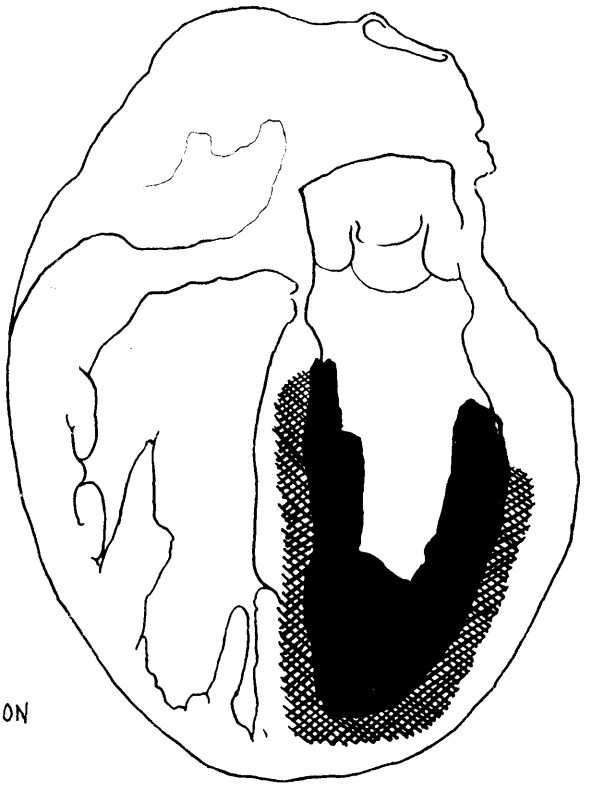
The heart is enlarged showing considerable hypertrophy of the left ventricle except on its anterior aspect near the apex where the wall seems thin on palpation. Over this area the pericardium is firmly adherent to the wall. A large calcareous plate is palpable in the course of the descending branch of the left coronary artery, about 1½" from its orifice.

The aorta appears remarkably healthy to the naked eye, except for a small atheromatous patch about ½" below the origin of the subclavian artery; here the aorta is thinned as if from a scar.

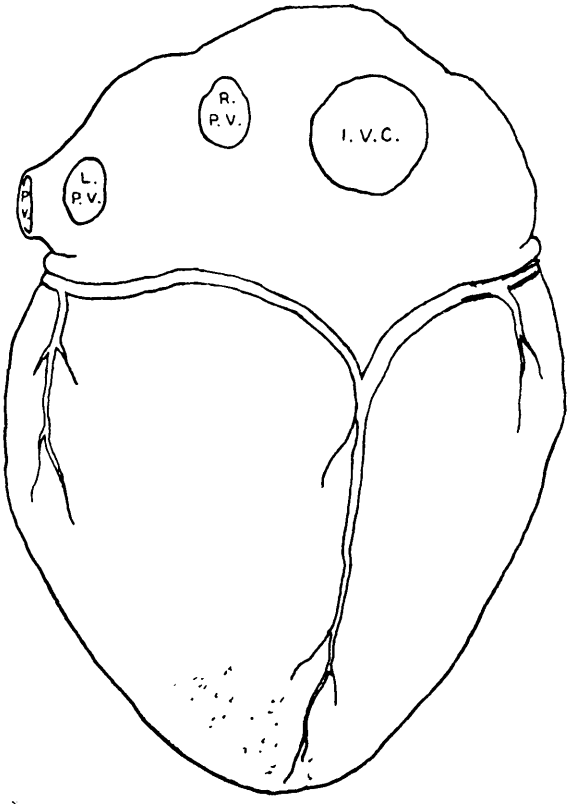
The lungs show oedema and some chronic bronchitis. There is scarring at both apices.

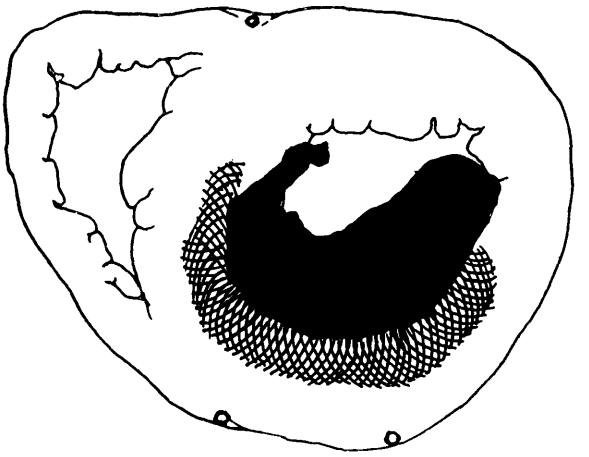






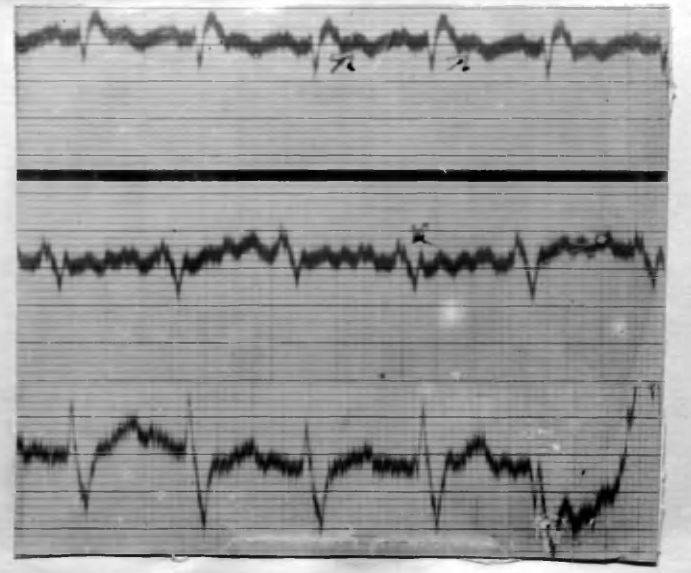
INFARCTION





ELECTROCARDIOGRAPHIC RECORD.

7. 6. 29.



QRS is prolonged .20 secs.

The apparently elevated RT in I must be taken, because of its time relationship, as part of QRS.

The undulations in II are not established in I or III QRS is splintered.

In I and occasionally in II a bifurcated P, giving PR = .20, can be established.

Case No. 9.

Post Mortem Summary.

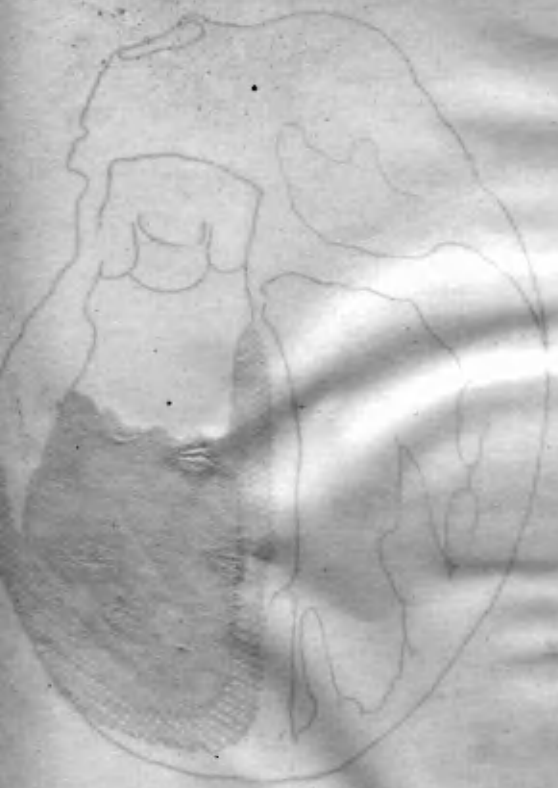
Stoutly built elderly man.

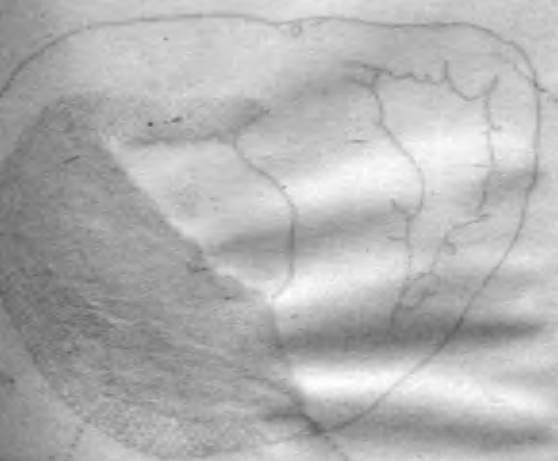
Heart.

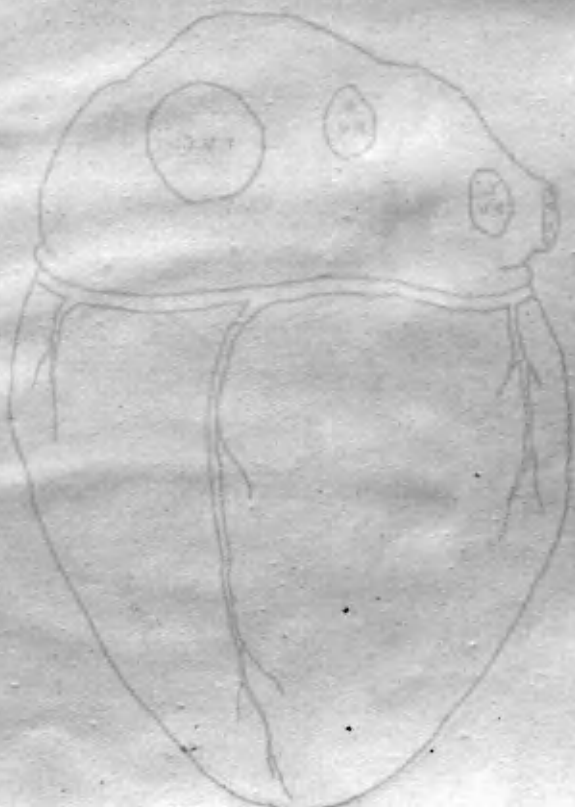
Several ozs. of fluid blood in pericardial sac.

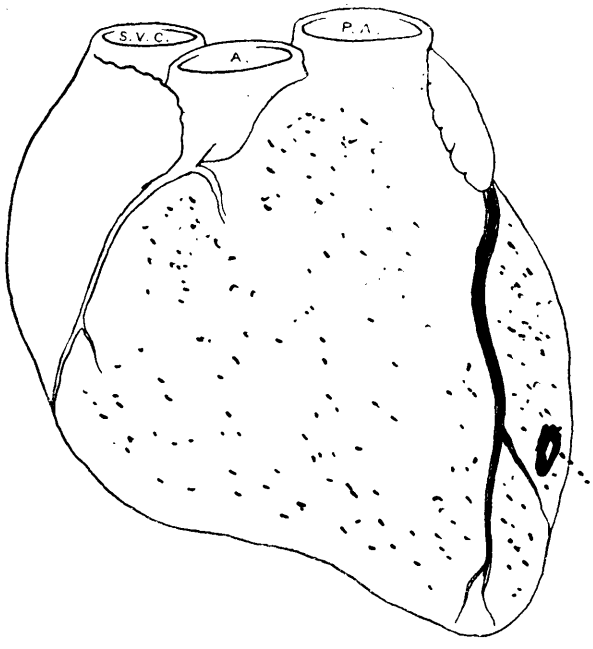
Recent pericarditis - Visceral and pariestal adherent. Whole apex is site of recent infarction and there is fairly large rupture on anterior aspect, about 2½" from apex. Right ventricle is dilated. Aortic shows patchy atheroma - atheromatous ulceration and arterio sclerosis.

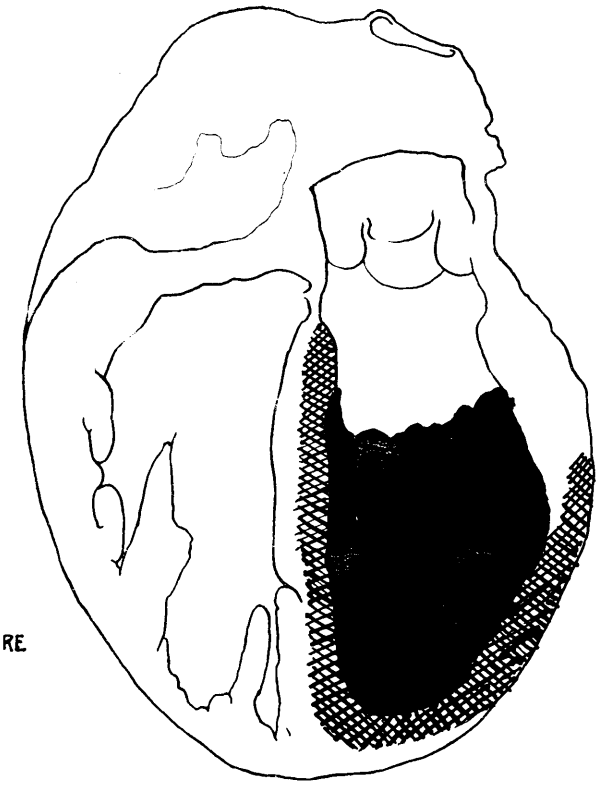
Further examination showed the descending branch of the left coronary completely blocked by thrombus. Infarction involved the whole of the apex of the left ventricle and left side of the septum.

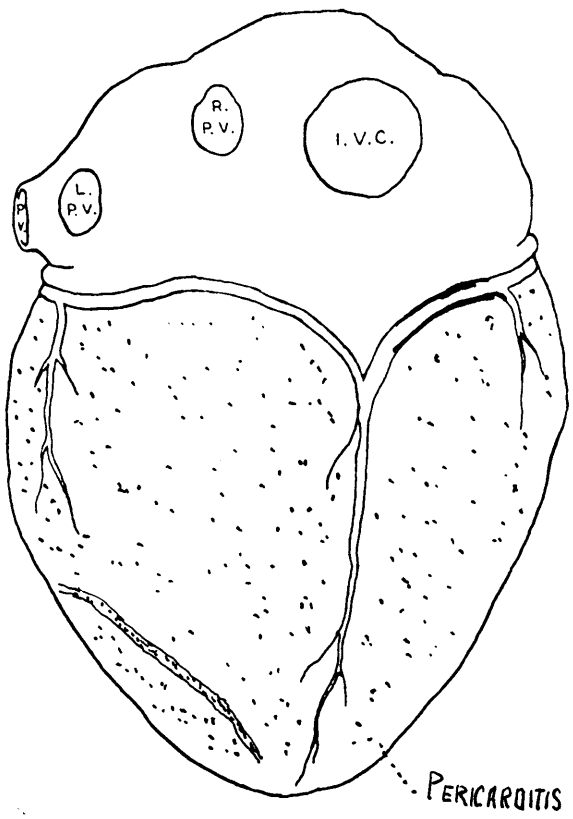


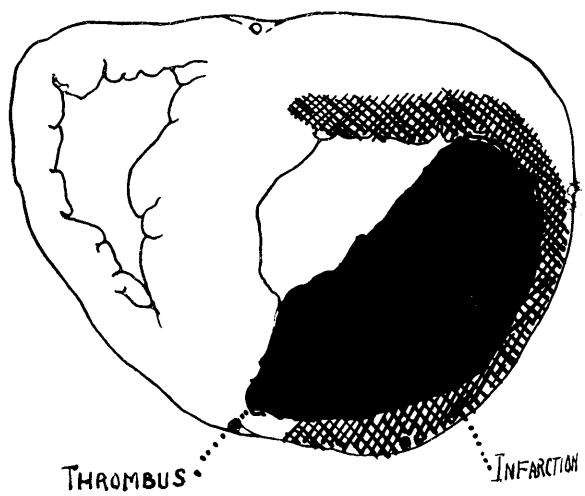





S.V.C.
A.
P.A.
SITE
OF
RUPTURE

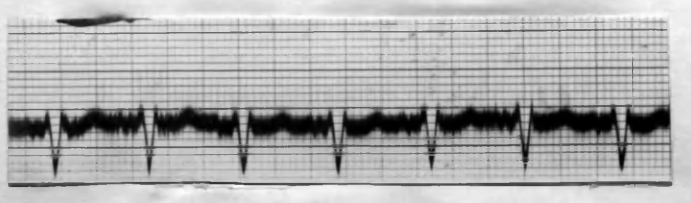



R.
P.V.
I.V.C.
L.
P.V.
PERICARDITIS


THROMBUS
INFARCTION

ELECTROCARDIOGRAPHIC RECORD.

24. 4. 29.



III only.

S is +.

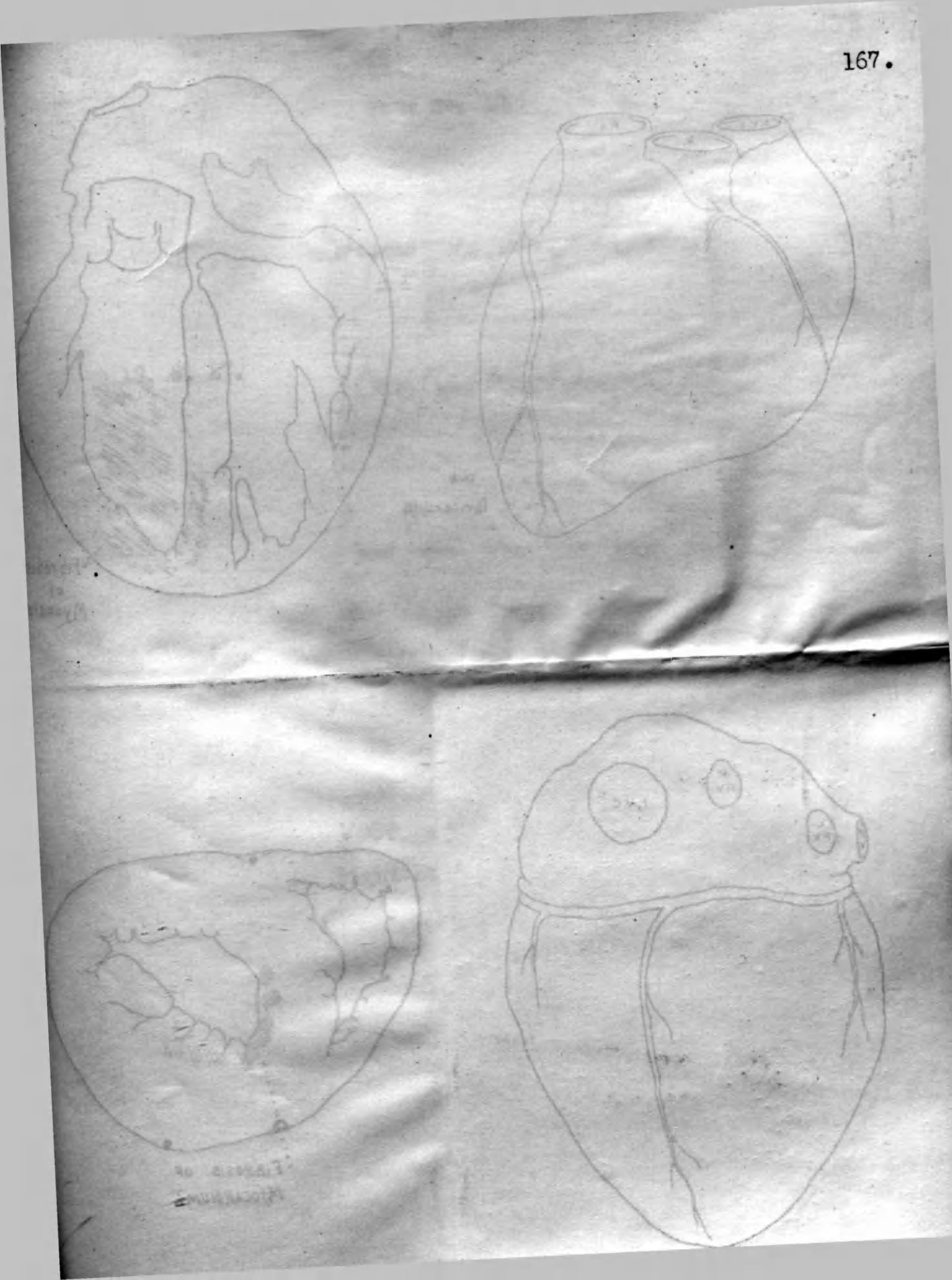
QRST = .32 secs.

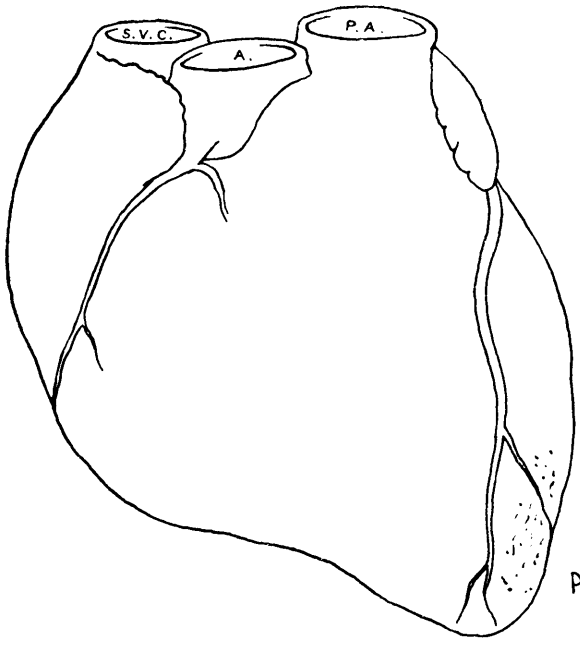
P is notched and apparently inverted.

PR = .12 secs.

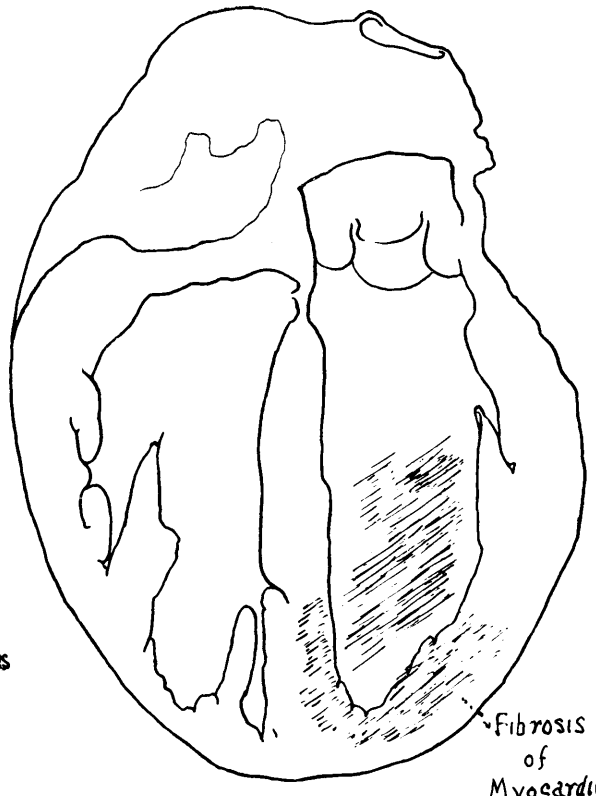
Post Mortem Summary.

Heart was much enlarged—three to four times normal size. There was aneurysm of heart wall at apex, this being due to thinning and fibrosis of myocardium in this region. The left coronary orifice was completely obliterated. The right coronary orifice was blocked ½" from its origin. There was a small recent infarction of the posterior aspect of left ventricle.

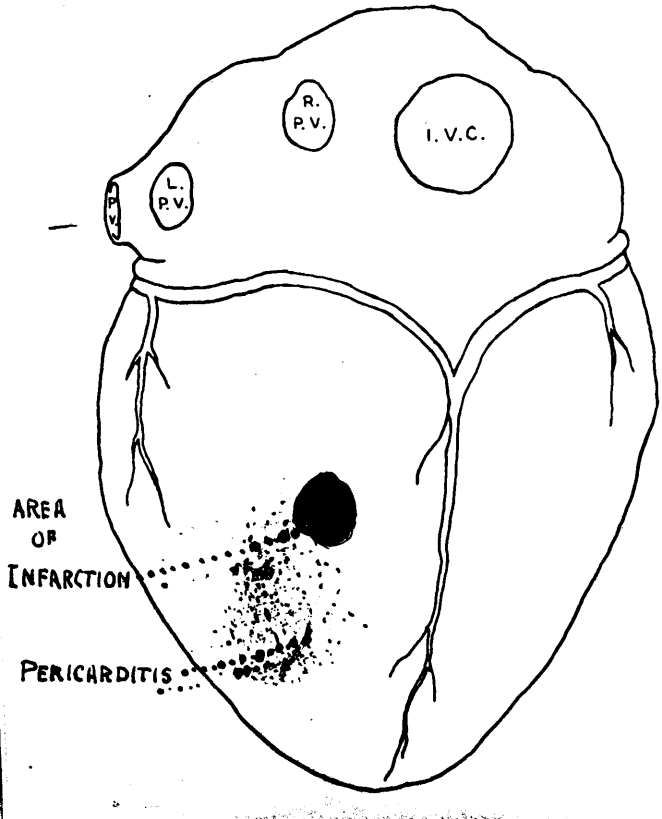




old
Pericarditis

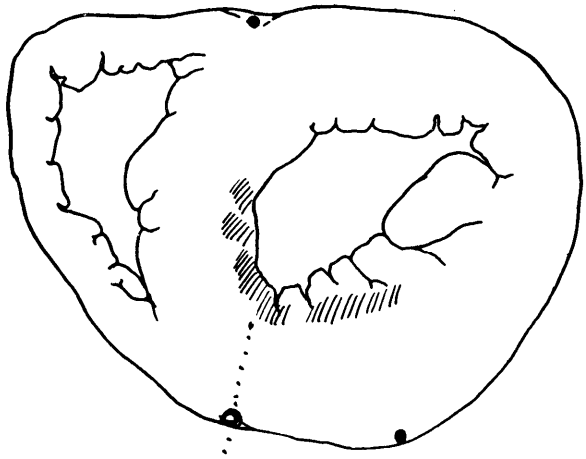


Fibrosis
of
Myocardium



AREA
OF
INFARCTION

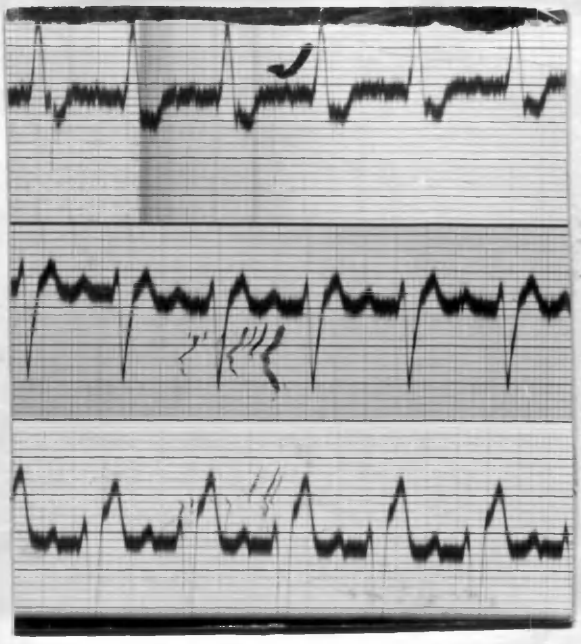
PERICARDITIS



FIBROSIS OF
MYOCARDIUM

Case No. 10.

13. 6. 27.



Pulse Rate 92 per min.

PR = .26 secs.

QRS = .16 secs.

QRST = .36 secs.

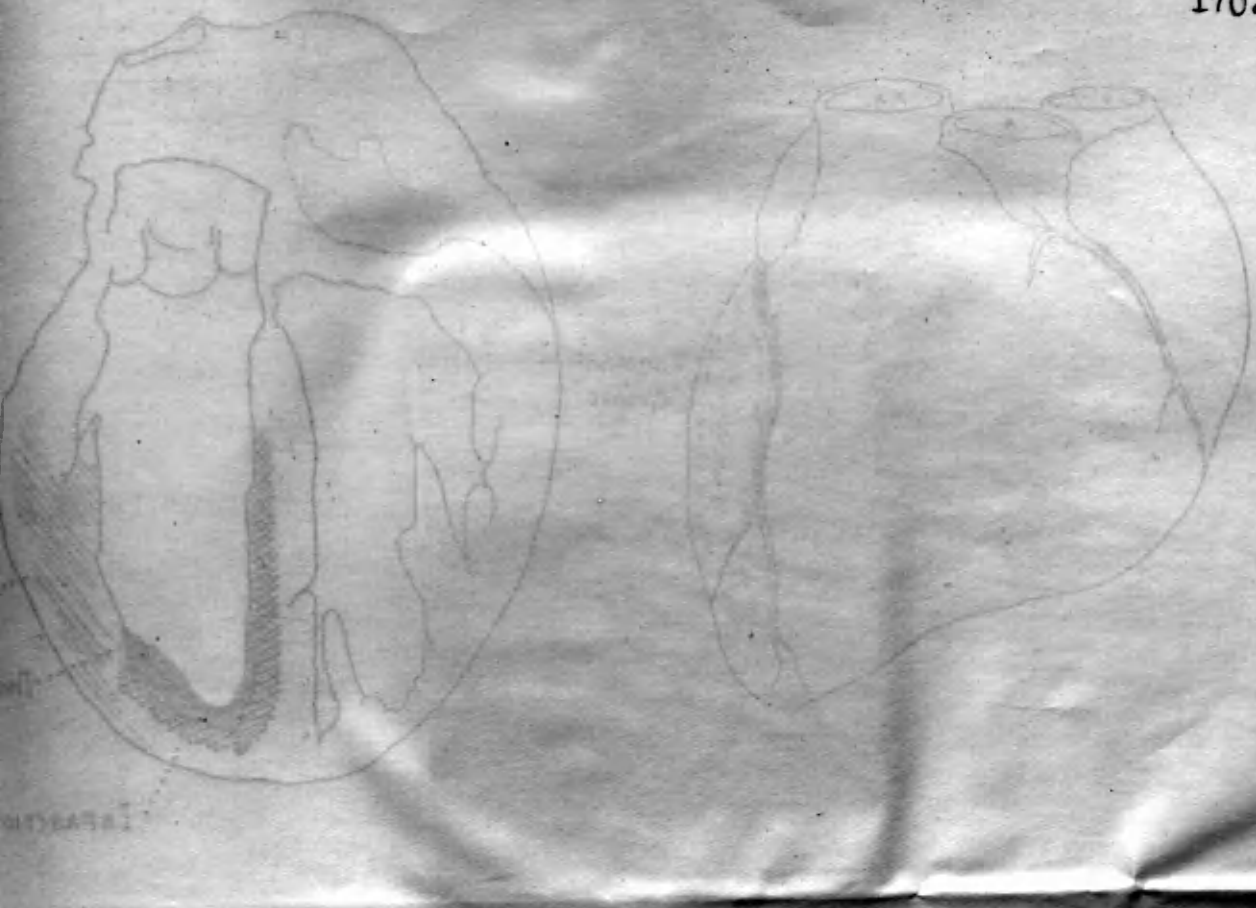
Right Bundle Branch Block.

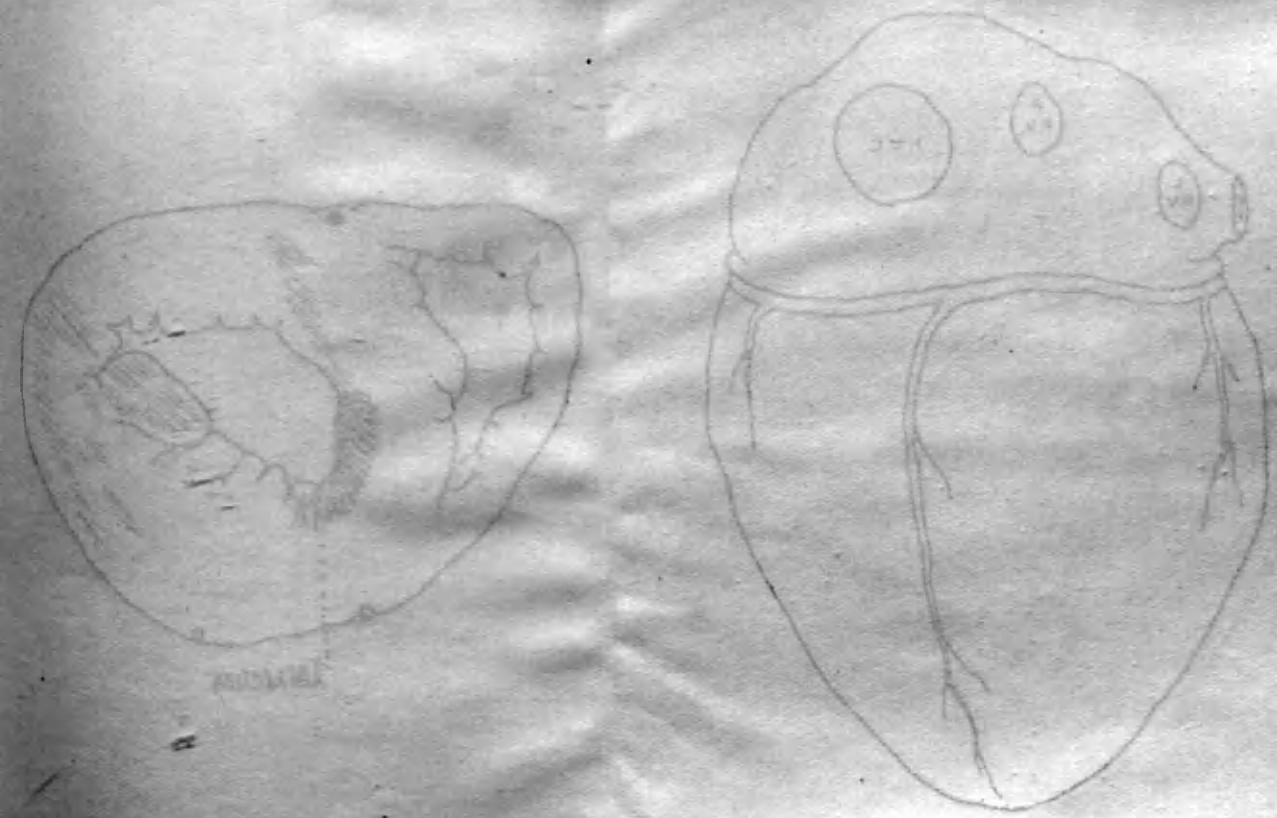
Post Mortem Summary.

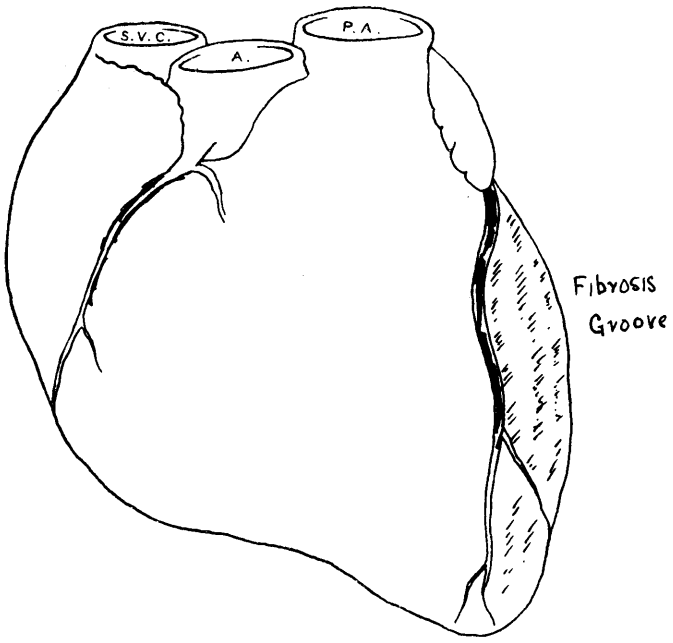
Heart was greatly enlarged, 28 ozs.

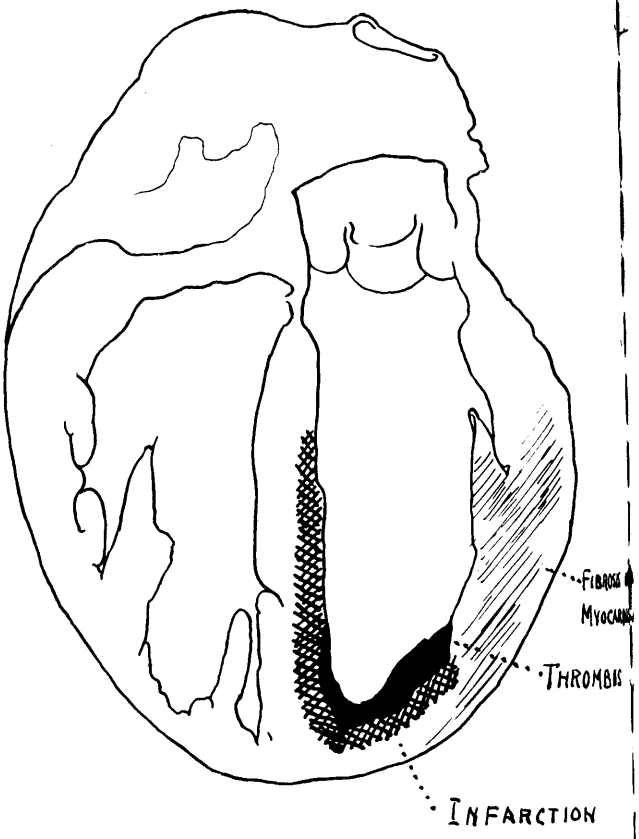
Coronaries hard and nodular. Soft area from commencement of descending branch of left coronary. This area was thinned. Descending left coronary was completely blocked by thrombus.

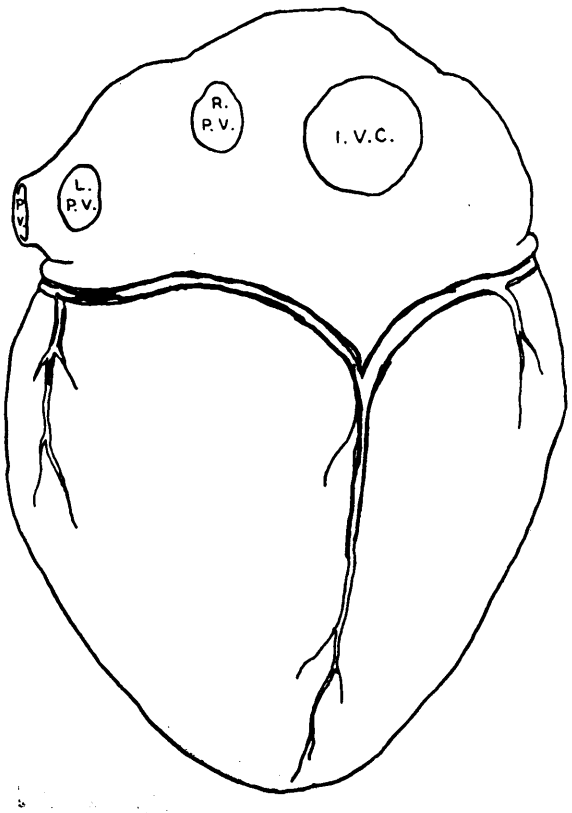
Further examination showed softening to be due to fibrosis and thinning of the myocardium in this region. There was in addition widespread fibrosis of left ventricle. At the apex of left ventricle and half way up left side of septum was recent infarction.

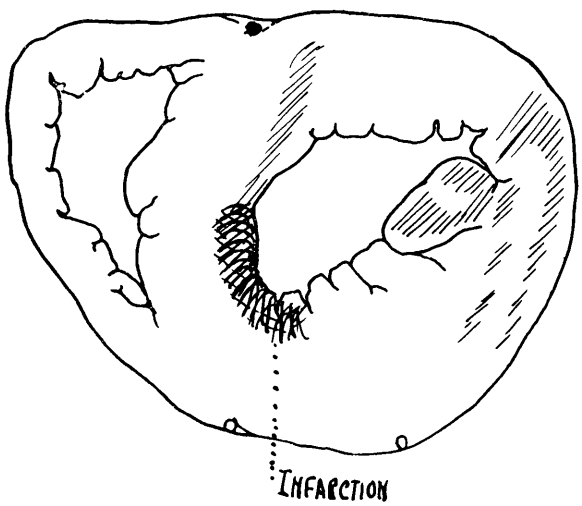





S.V.C.
A.
P.A.
Fibrosis
Groove

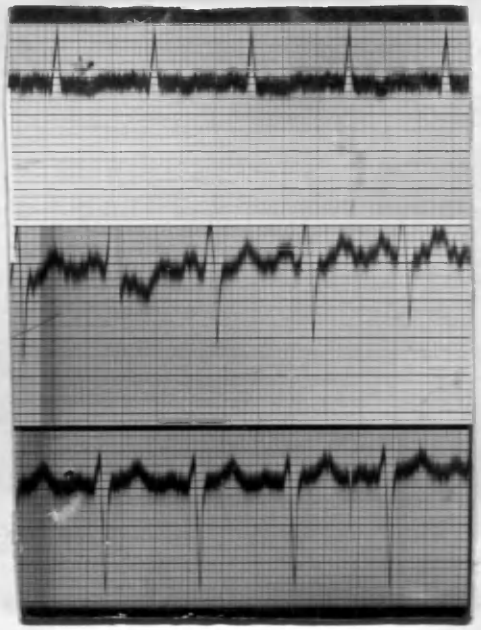

Fibrosis
Myocardium
Thrombus
Infarction


R.
P.V.
I.V.C.
L.
P.V.


Infarction

Case No. 11

ELECTROCARDIOGRAPHIC RECORD.



Pulse Rate - 109 per min.

Left Ventricular Preponderance.

PR = .14 secs.

QRS = .10 secs.

QRST = .34 secs.

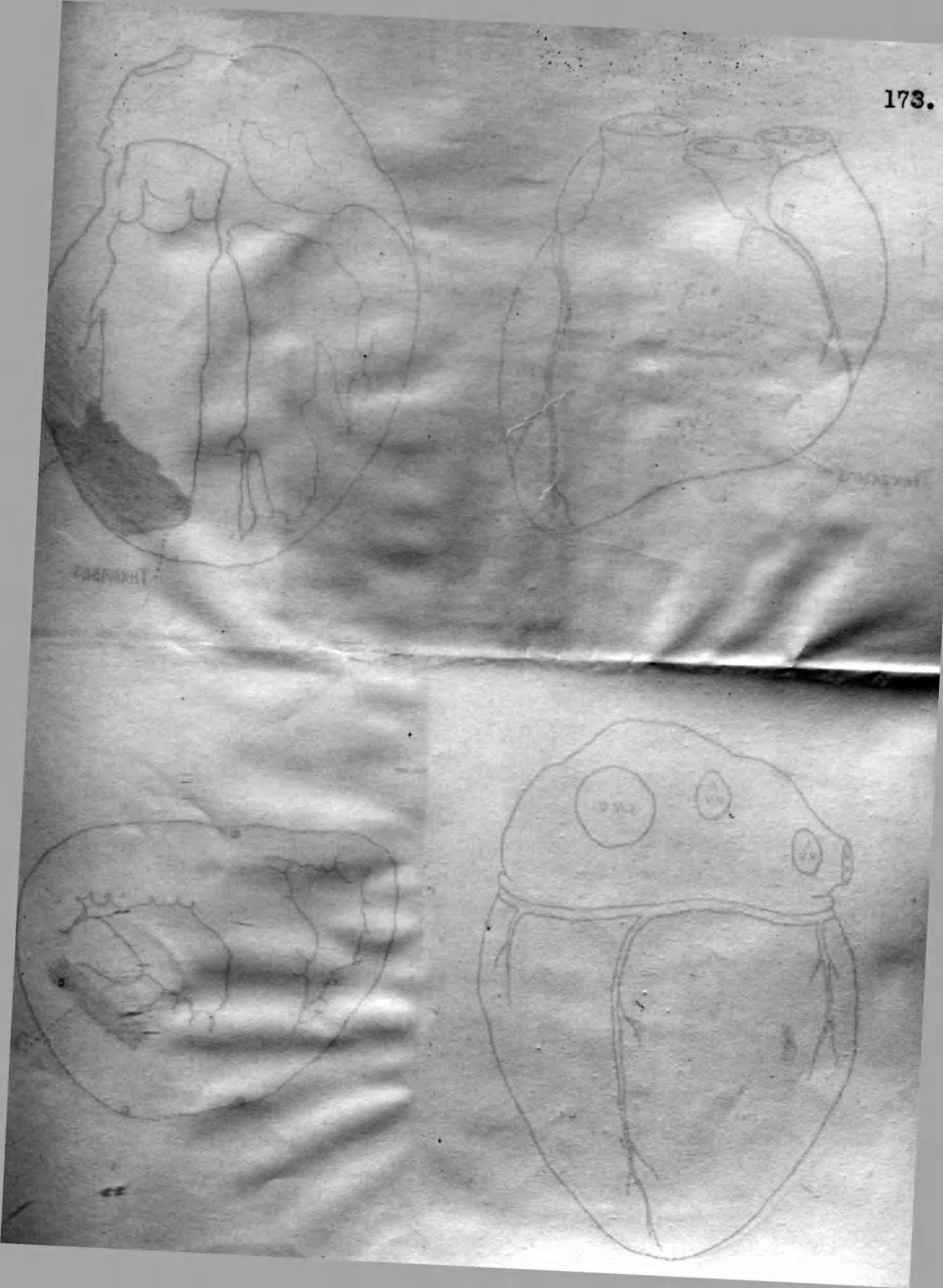
T inverted in I.

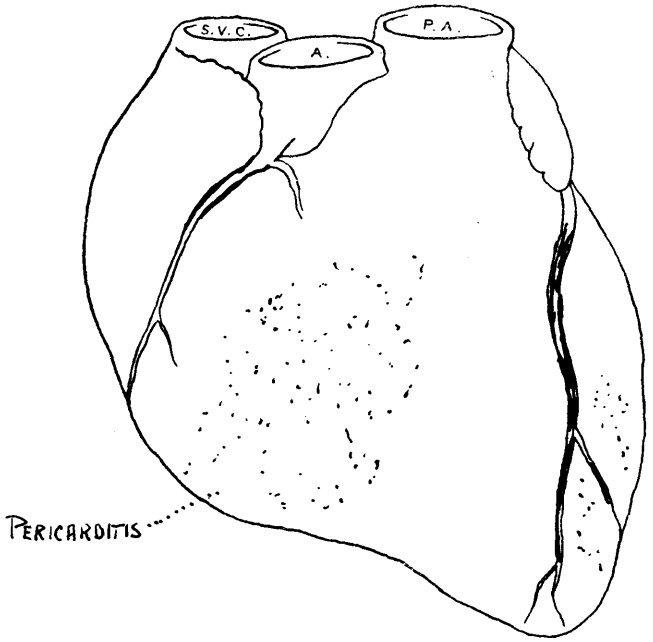
Case No. 12.

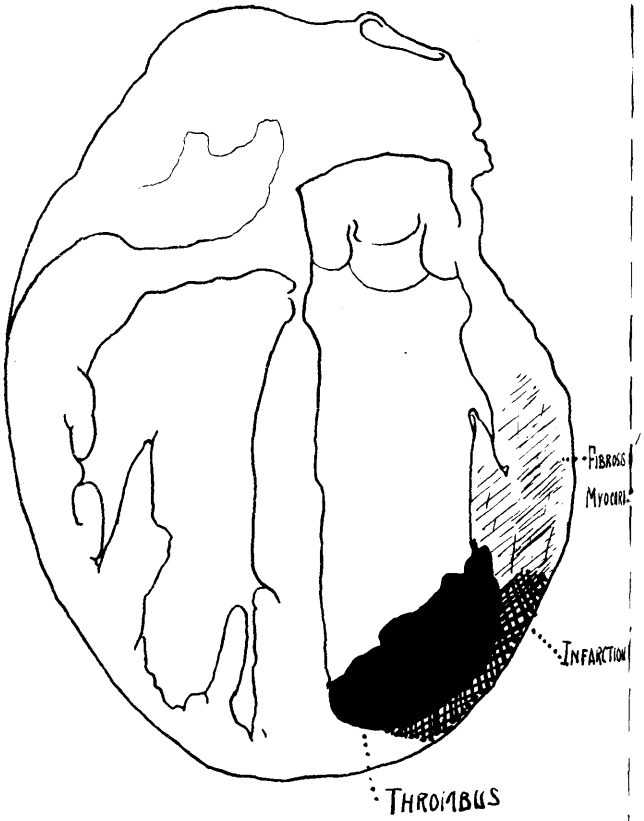
Post Mortem Summary.

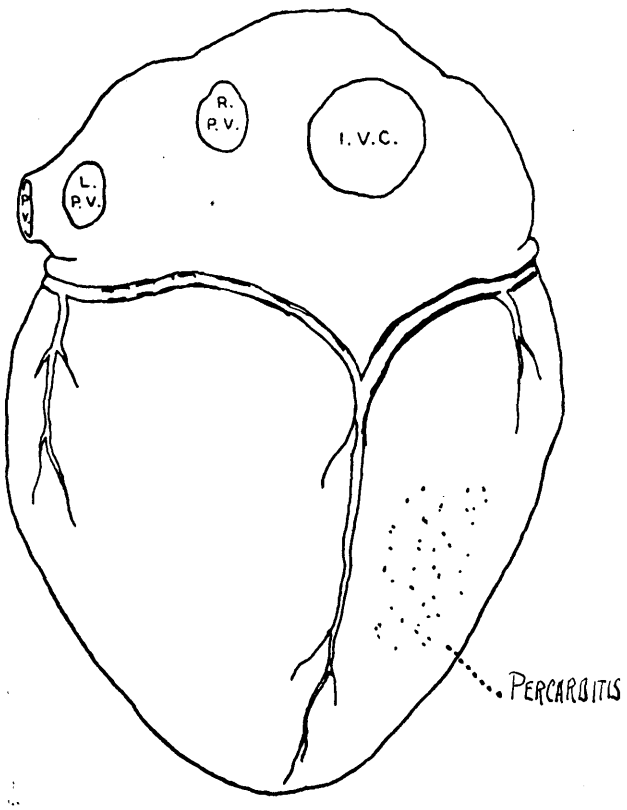
THORAX. Heart : The pericardial sac was very large and the heart greatly hypertrophied. This was principally due to hypertrophy of the left ventricle with some dilatation. The right ventricle was moderately dilated with some hypertrophy also. There was no valvular disease sufficient to cause deformity but there was some thickening of the aortic cusps. About half an inch above the left posterior cusp there was an aneurism about the size of a hazelnut. It was shallow and had a wide orifice which looked as if it had arisen as a rupture. It was an old aneurism and mostly filled with thrombus. Its situation kept it clear of any interference with the conducting apparatus. There was extreme atheroma with calcification causing much narrowing of both coronary arteries. There was the remains of an old infarction situated in the ventricular wall underneath the left papillary muscle and involving it to a considerable extent. There were globular thrombi in the ventricle over this area. There were also thrombi in both auricular appendages.

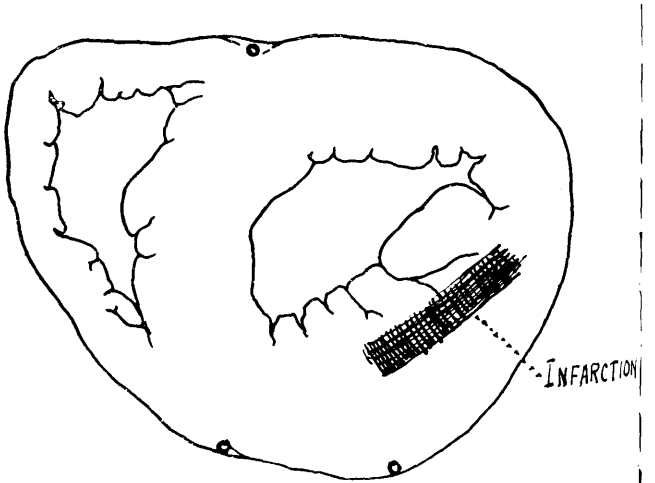
Lungs: These showed considerable anthracosis and emphysema. There was a small old infarction at the left base and a large recent one occupying nearly the whole of the middle lobe of the right lung. Associated with this was pleurisy with a considerable amount of effusion.

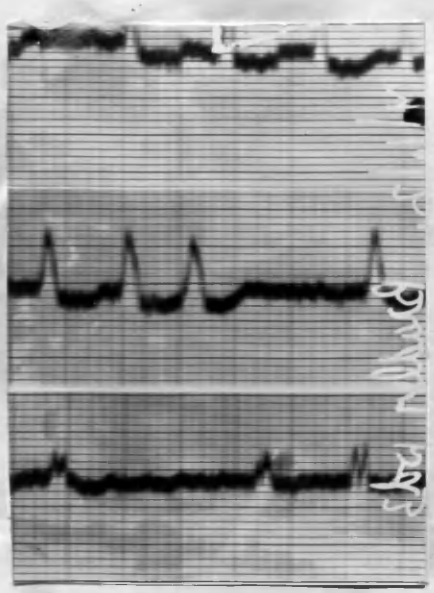



S.V.C.
A.
P.A.
PERICARDITIS


FIBROUS
MYOCARD.
INFARCTION
THROMBUS


R.
P.V.
I.V.C.
L.
P.V.
PERCARDITIS


INFARCTION



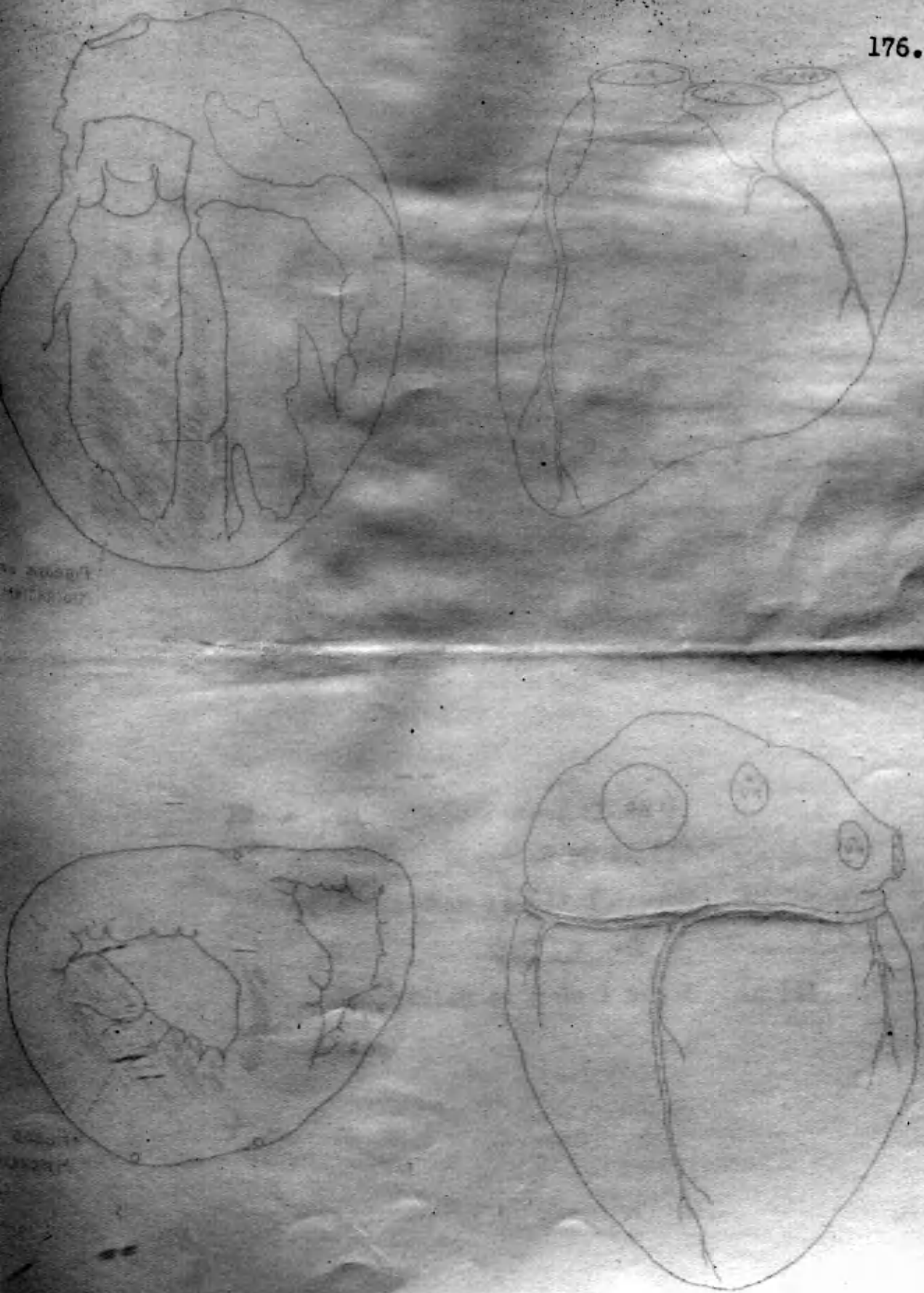
Auricular Fibrillation.

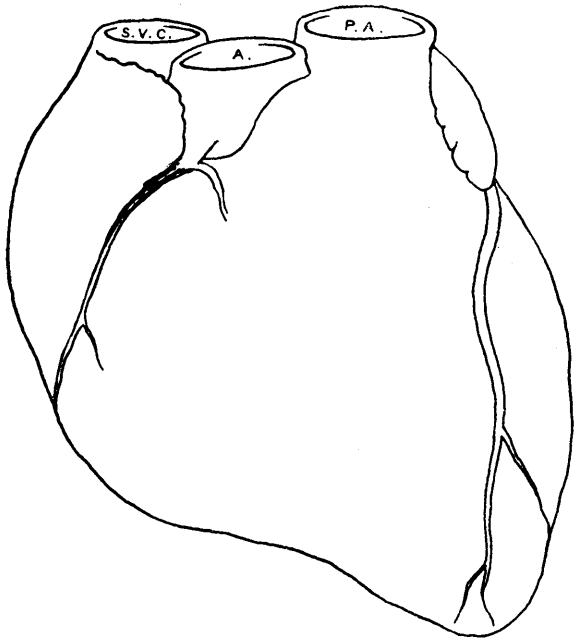
RT depressed in I & II.

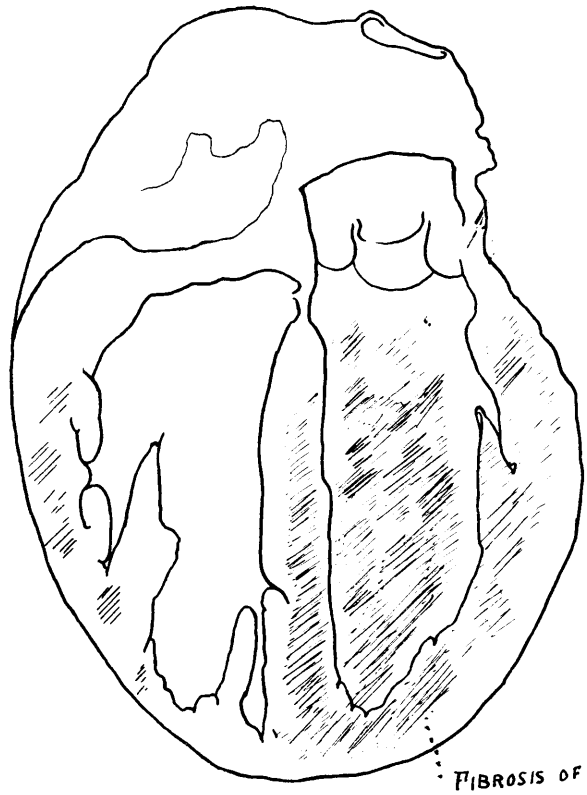
QRS splintered in III.

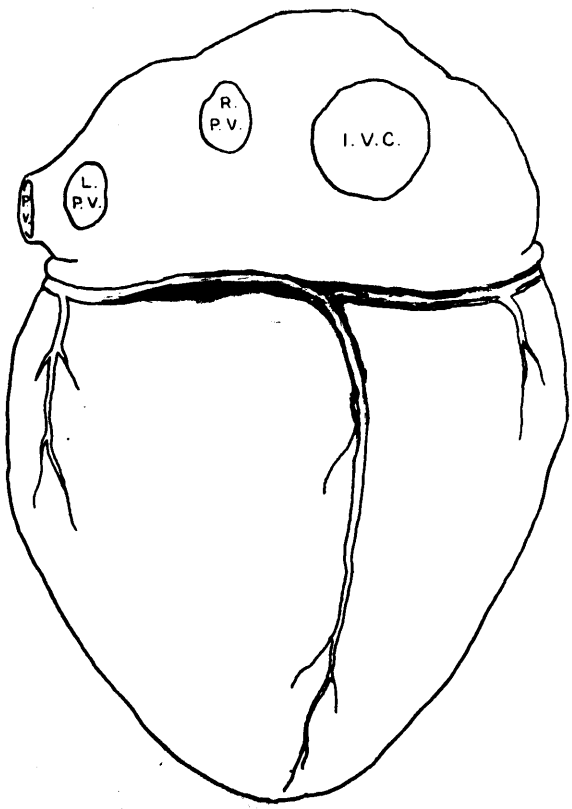
Case No. 13.

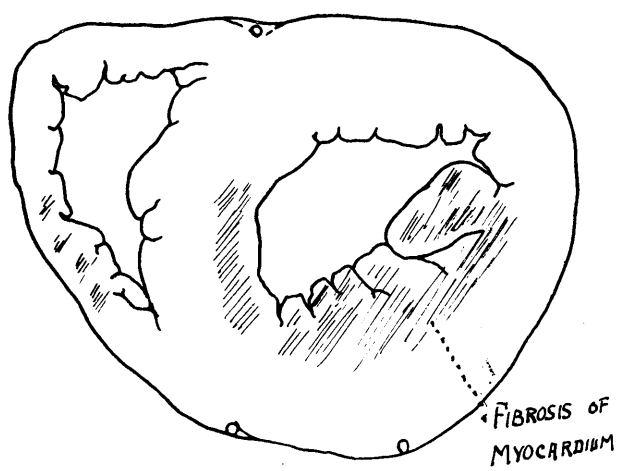
HEART : The organ is considerably enlarged and very soft in consistence. There is dilatation of both right ventricle and right auricle and also of the left ventricle. The myocardium is of poor quality and shows widespread fibrosis particularly in the papilliary muscles and on the inter-ventricular wall of the left ventricle. The aortic valve cusps are thickened. The other valves show nothing of note. The coronary vessels in their course appear fairly healthy, but the orifice of the left coronary admits a probe with difficulty, while the orifice of the right coronary cannot be determined either from the aortic aspect or by tracing the coronary from its distal end. The first part of the aorta and the arch shows very well marked syphlitic aortitis. The descending and abdominal portions of the aorta show only slight patchy atheroma.



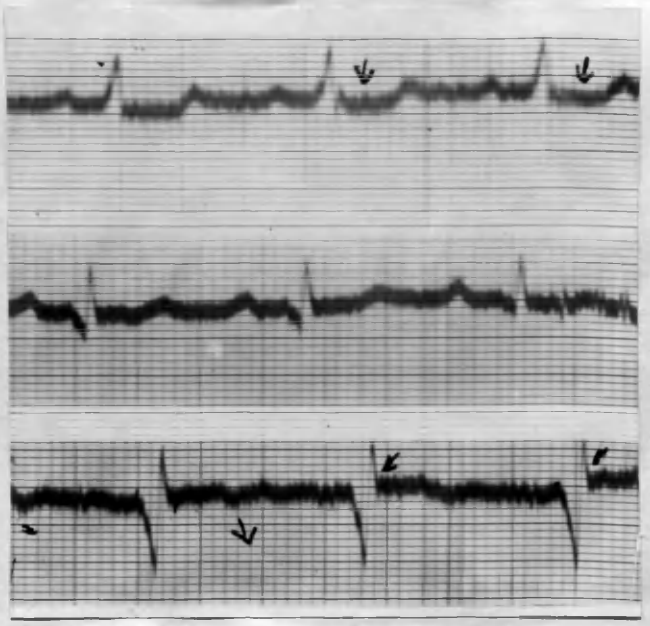

S.V.C.
A.
P.A.


FIBROSIS OF
MYOCARDIUM


R.
P.V.
I.V.C.
L.
P.V.


FIBROSIS OF
MYOCARDIUM

Case No. 13.



PR = .24. Depressed RT in I

Elevated RT in III

Cowan Wave in III +

QRST = .32.

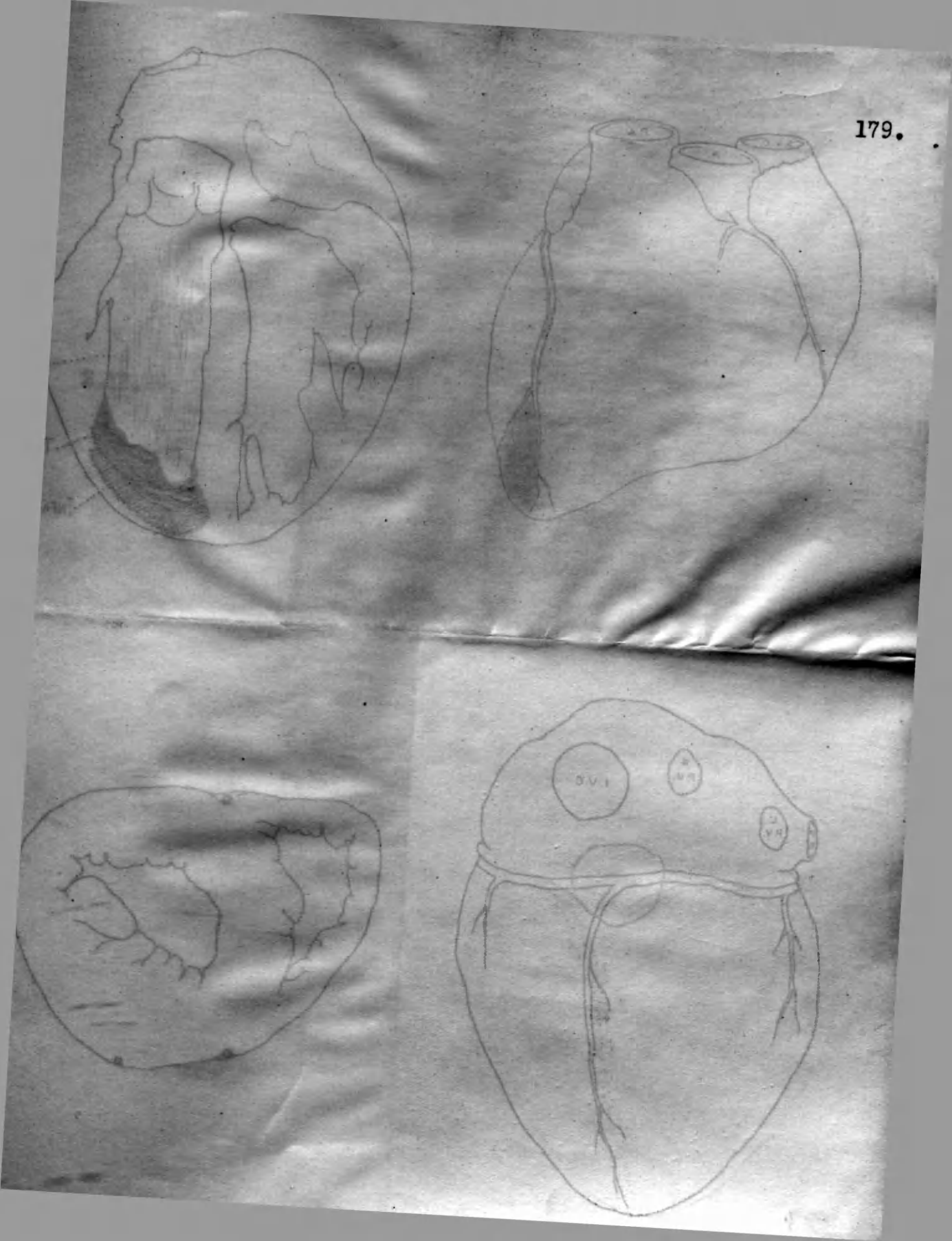
Thickening of R in I and S in III.

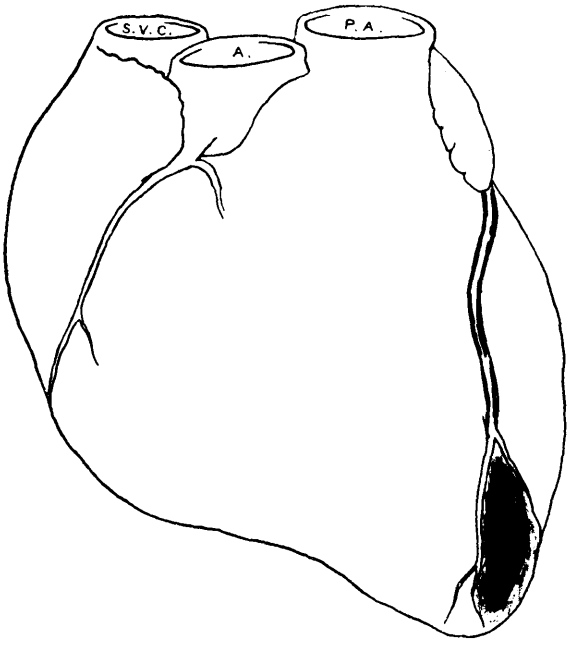
Case No. 14.

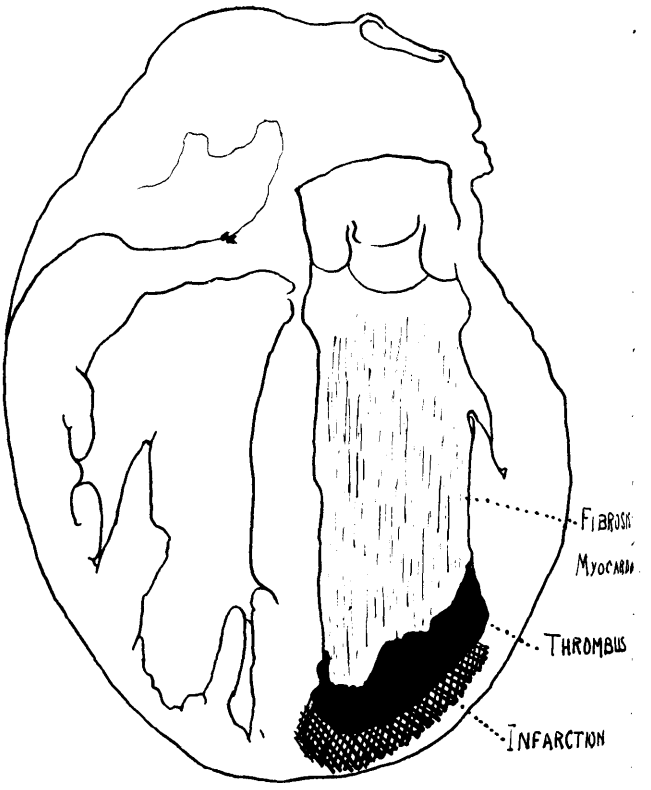
Post Mortem Summary.

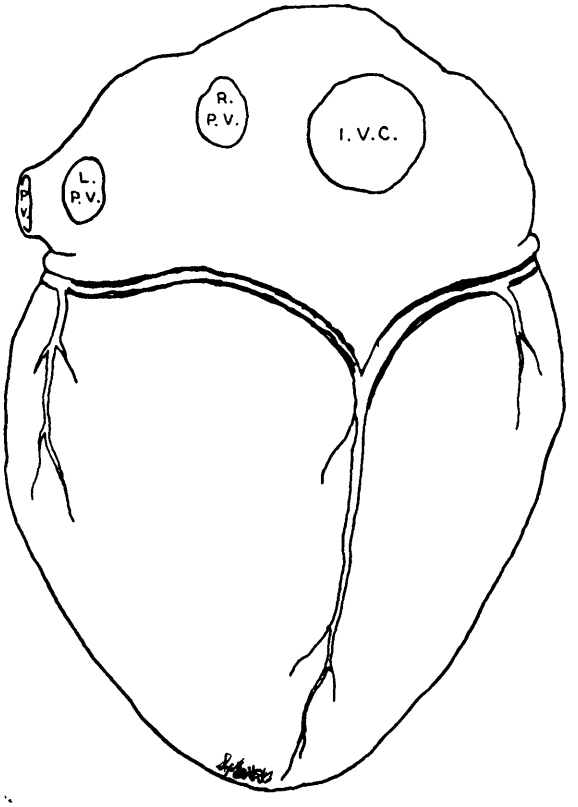
The heart was greatly enlarged being hypertrophied and dilated. The left ventricle was principally affected. Near the apex and just to the left of the septum there was an area which was congested, flattened and soft. On section, the muscle was paler than the rest and a large thrombus had recently formed on it.

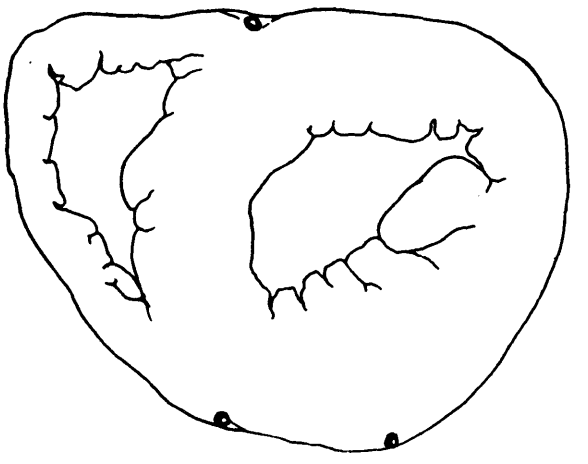
Aorta - Syphilitic Aortitis of long standing. Coronary orifices are narrowed but admitted probe. Fibrous patch on septum behind the undefended space and below it - not in position to interfere with Bundle of His.


SVI


S.V.C.
A.
P.A.

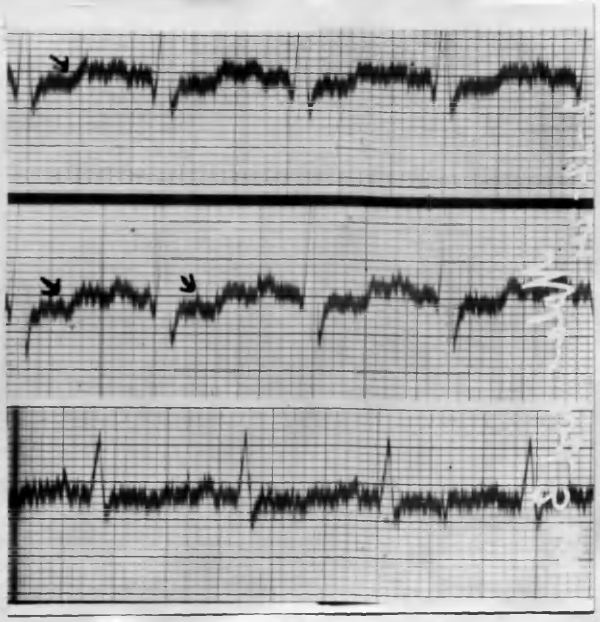

FIBROSIS
MYOCARDIUM
THROMBUS
INFARCTION


R.
P.V.
I.V.C.
L.
P.V.



Case No. 14.

2.12.29.



PR = .16 P poor in all leads.

T Diphasic in I and II.

Pulse rate 115. QRST = .32.

Fine somatic tremor.

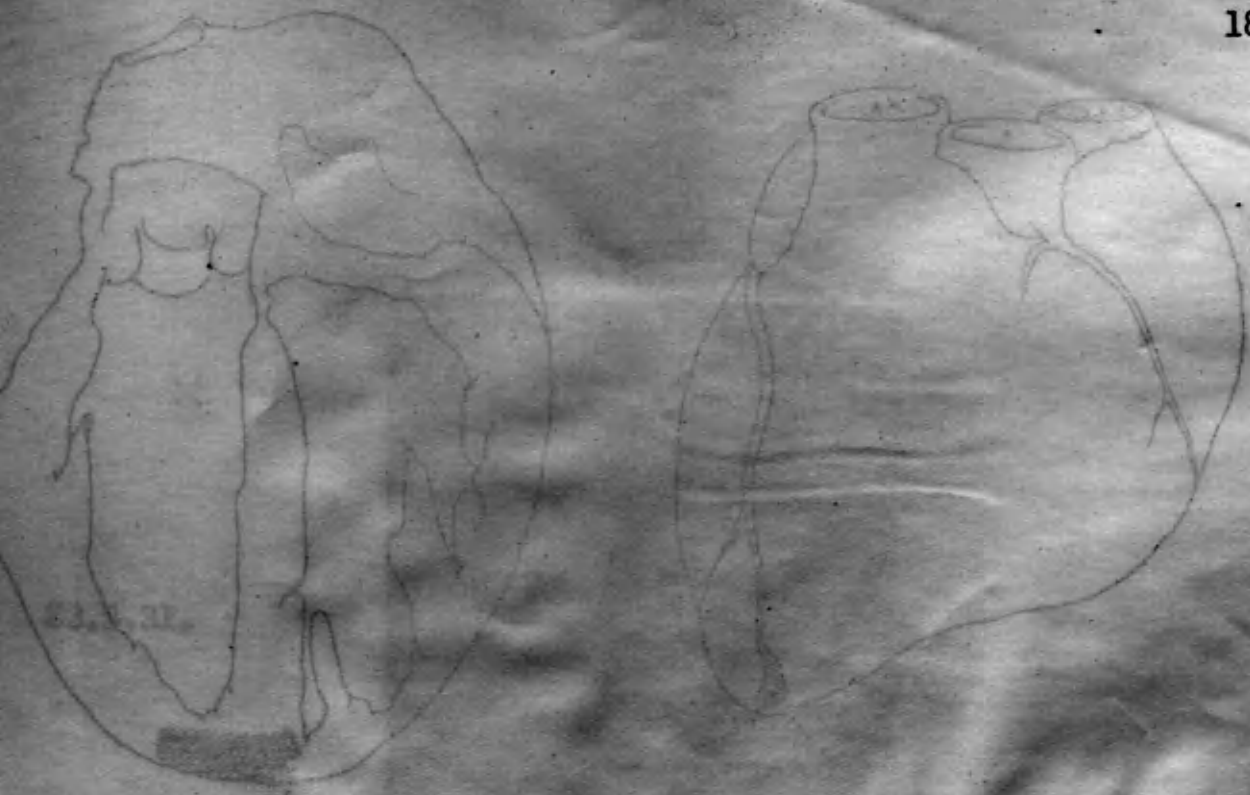
Case No. 15.

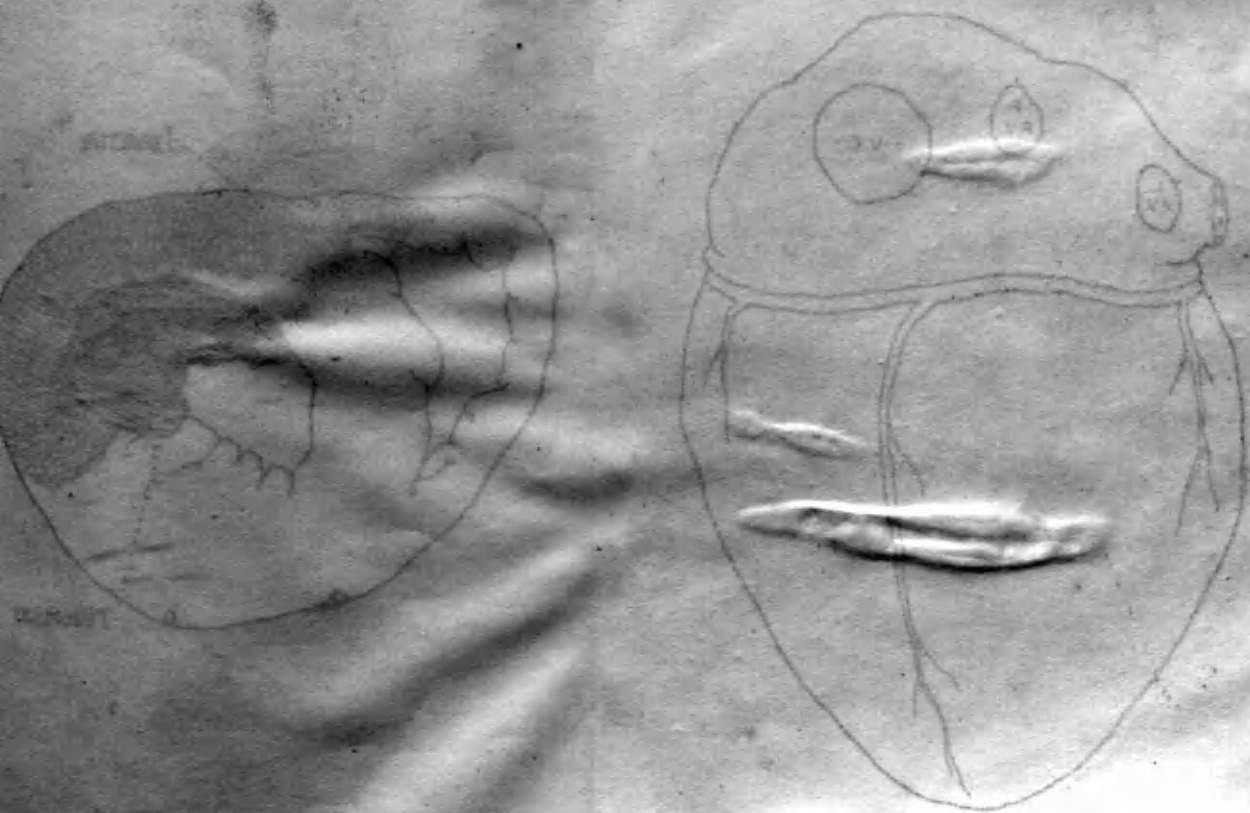
Post Mortem Summary.

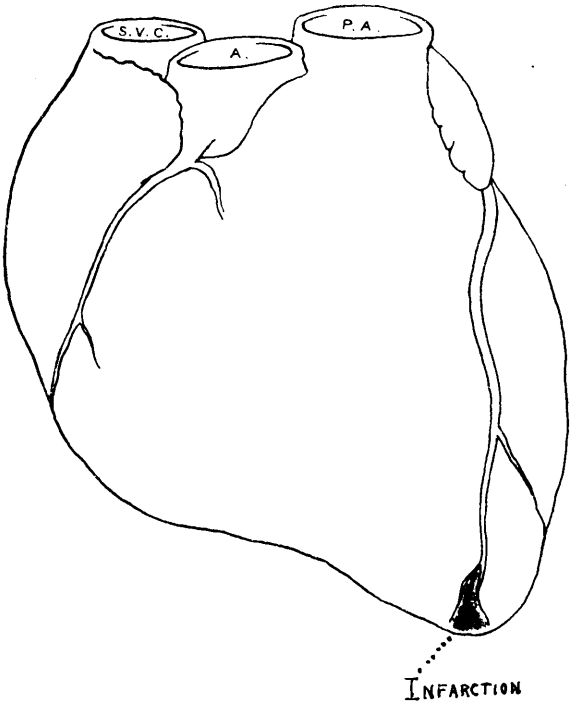
Heart 21 ozs.

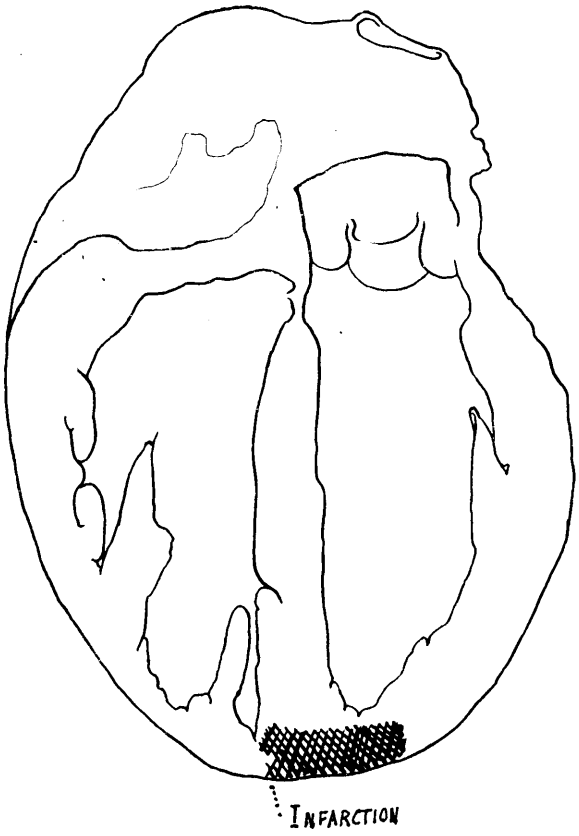
The organ is enlarged, soft and flabby in consistence. There is a patch of old pericarditis over the anterior aspect of the right ventricle. The posterior wall of the left ventricle towards the base feels extremely soft and is markedly congested showing evidence of a recent infarct. One cut was made through the heart about an inch below the auricular - ventricular groove and through the middle of the infarct. The section showed that at this area the whole of the posterior wall of the right ventricle was involved in an infarction of some duration a month ? with a recent infarction at the periphery of the older lesion. The old infarction appears yellowish white, while the recent is red in colour. It extended into the inter-ventricular septum for about half an inch. Both ventricles are hypertrophied and dilated. There is slight thickening of the aortic valve cusps. The aorta shows arterio-sclerosis and patchy atheroma. The coronary vessels show a marked degree of patchy atheroma, and the branches of the right conorary artery are especially thickened.

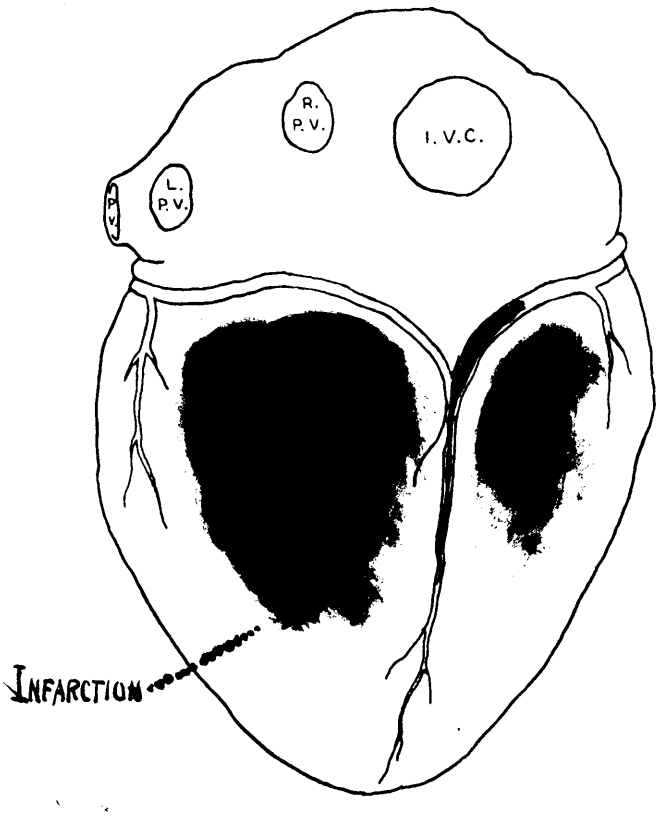
Further examination showed that the posterior wall of the left ventricle at the base was also involved. See Diagram.

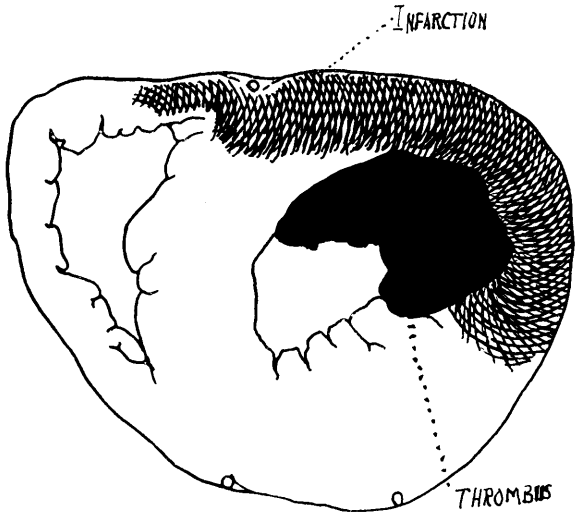





S.V.C.
A.
P.A.
INFARCTION

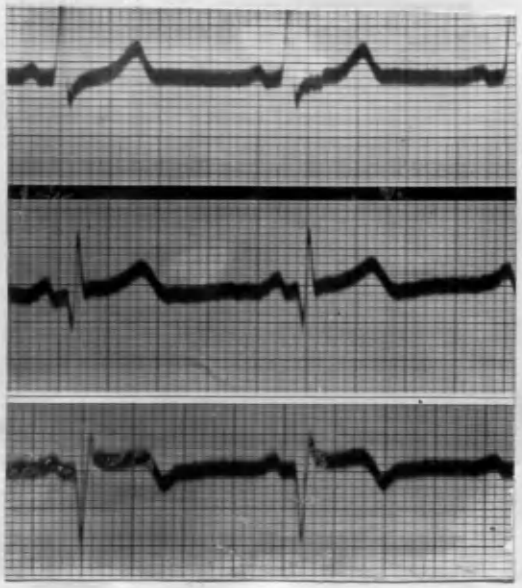

INFARCTION


R.
P.V.
I.V.C.
L.
P.V.
INFARCTION


INFARCTION
THROMBUS

Case No. 15.

23.3.31.



Left Ventricular preponderance.

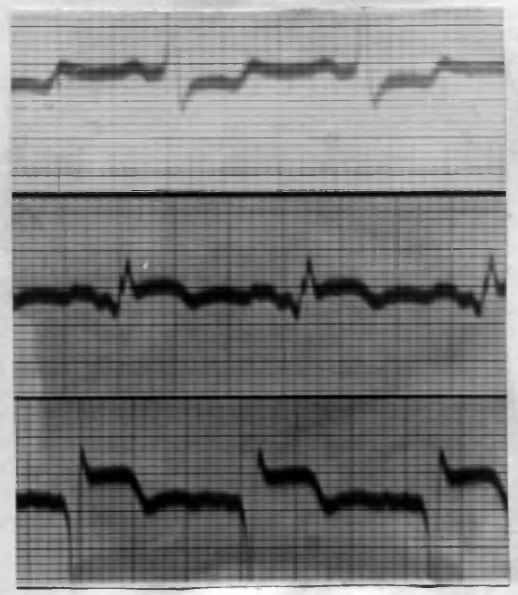
RT elevated in II and III.

depression in I.

Note amplitude of Q Wave.

Case No. 15.

29. 3. 31.



RT depression with diphasic T in I.

RT elevation more marked in II and III.

Splintering of QRS in II.

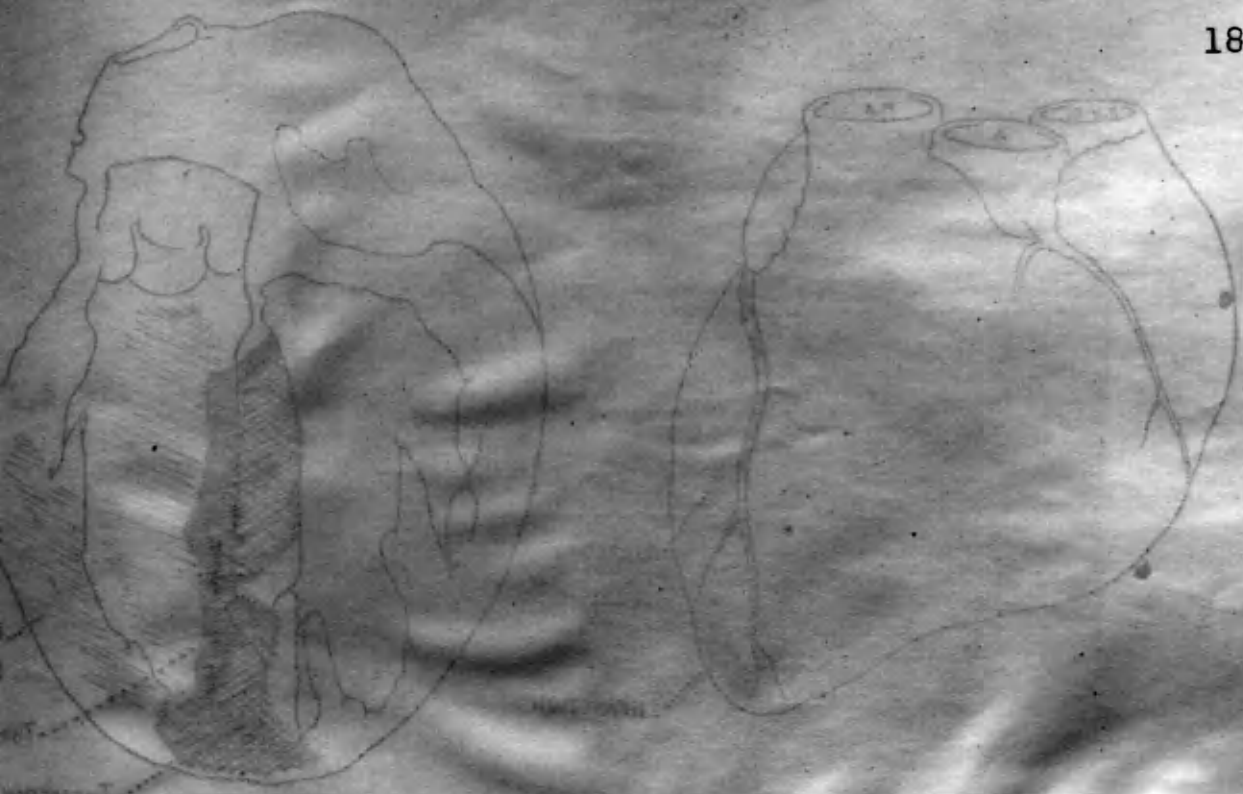
Case No. 16

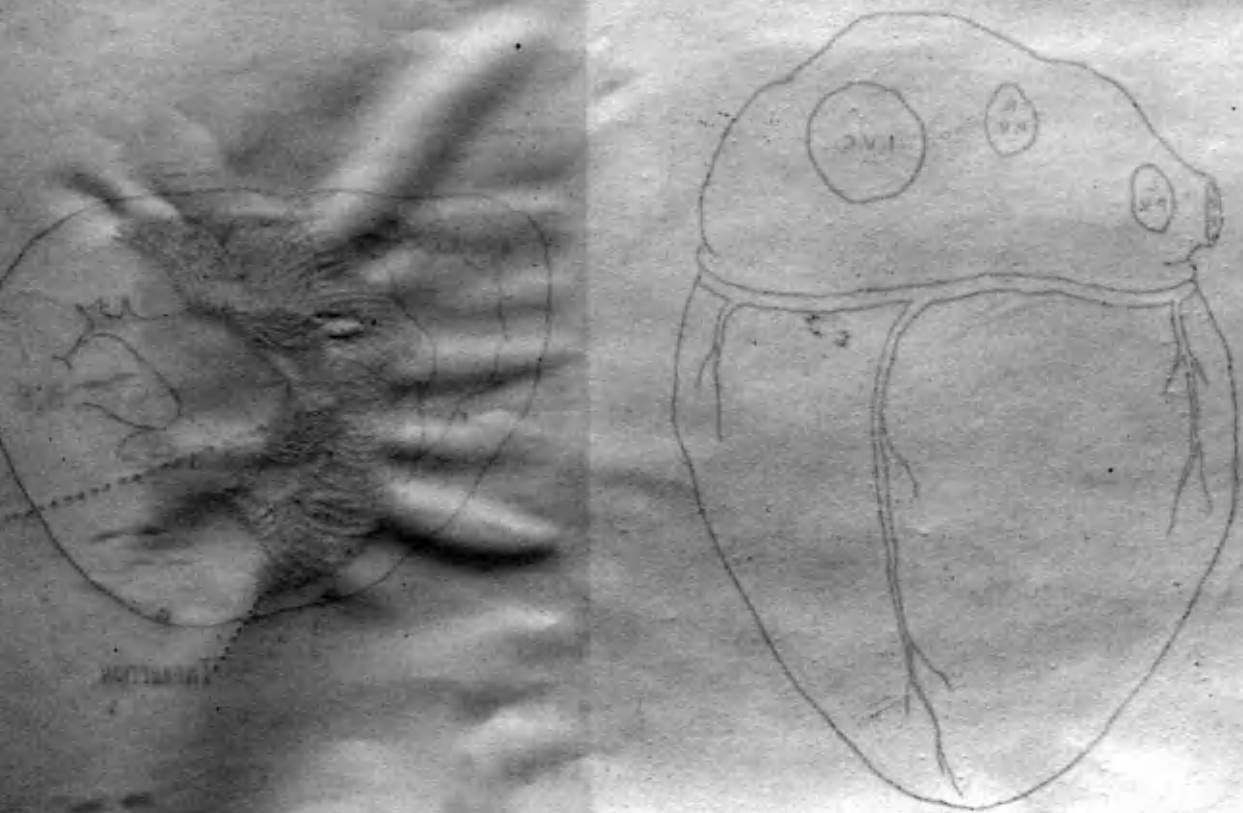
Post Mortem Summary.

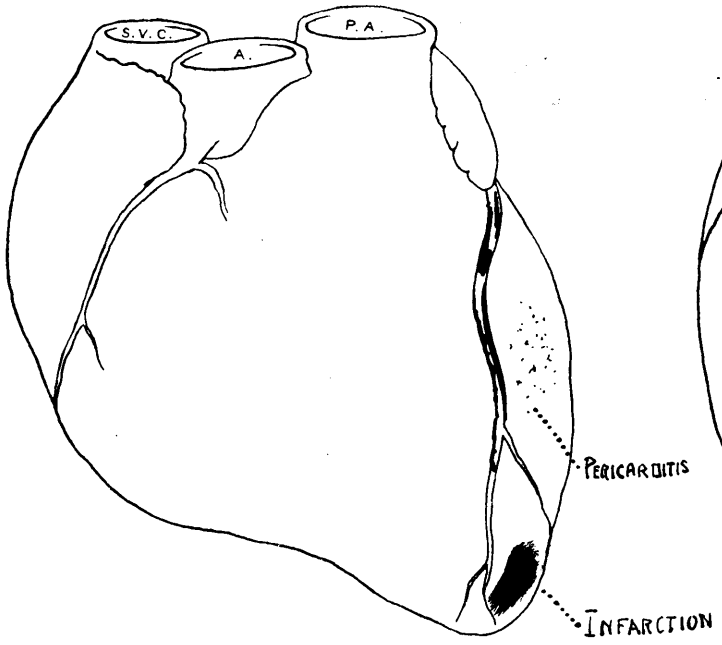
Heart - 18 Oz.

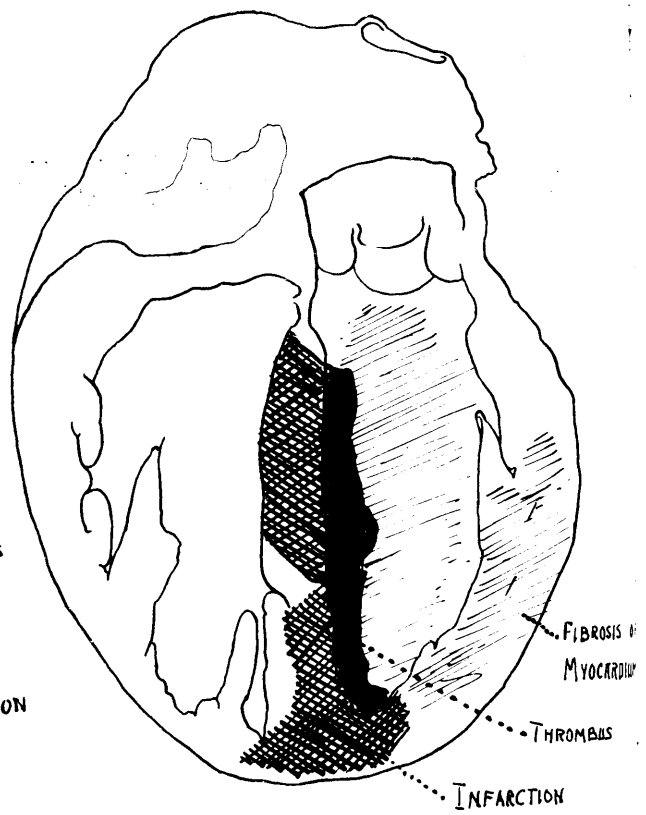
The organ is much enlarged showing considerable dilatation of the right ventricle. There is a patch of recent pericarditis on the anterior wall of the left ventricle midway between apex and base and corresponding to an area of fibrosed myocardium. The myocardium is of poor quality and in places the wall of the left ventricle feels exceedingly thin, notably in the line of the descending branch of the left coronary midway between apex and base; at the extreme apex of the left ventricle where there is definite bulging of the wall; and on the posterior aspect of the left ventricle in the line of the descending branch of the right coronary. On section the myocardium in these areas shows extreme fibrosis and at the apex there is definite recent infarction with adherent thrombus. The aortic valve cusps are slightly thickened from primary sclerosis. The other valves show nothing of note. The aorta shows patchy atheroma and arteriosclerosis. The coronary vessels show well marked atheroma. The lumen of the descending branch of the left coronary artery is completely stenosed by recent thrombus overlying an atheromatous patch ½" from its orifice. The transverse branch of the right coronary artery is completely blocked about 1¼" from its orifice by old-standing thrombus and atheroma.

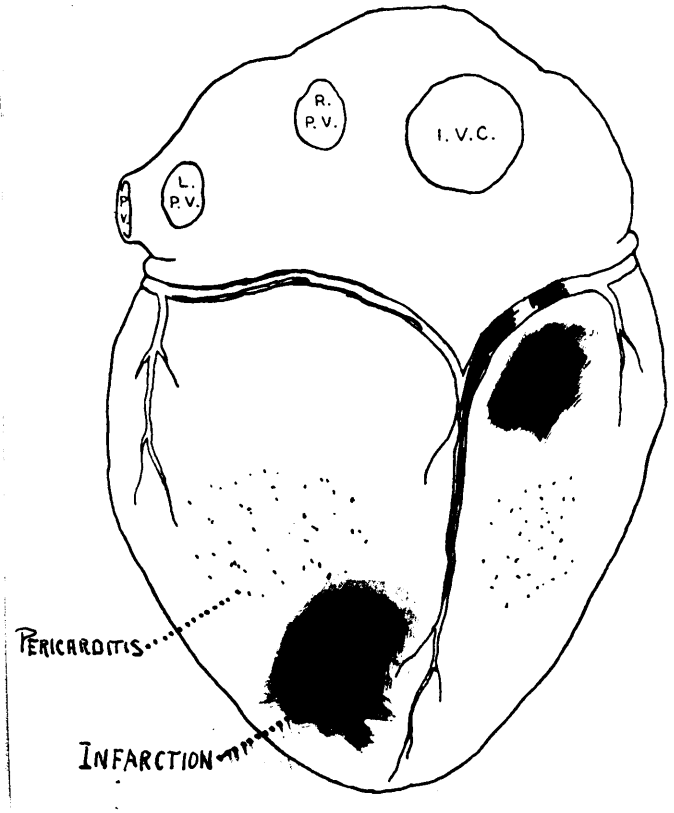
Further examination showed recent infarction involving apex and left side of septum. There were two recent infarctions on posterior wall, right ventricle at base and left ventricle at apex. See Diagram.

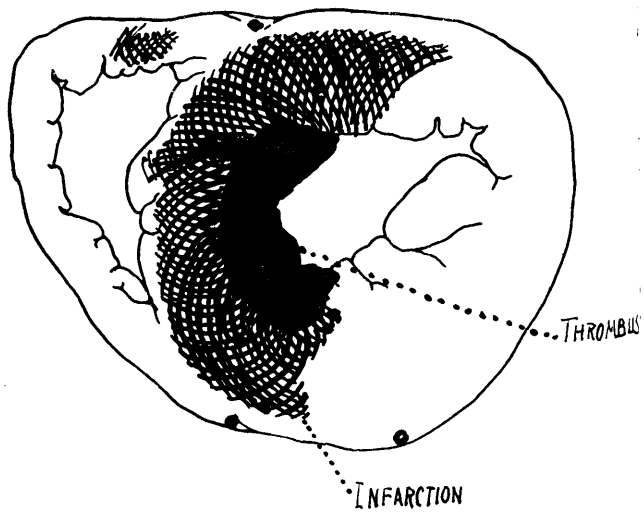





S.V.C.
A.
P.A.
PERICARDITIS
INFARCTION

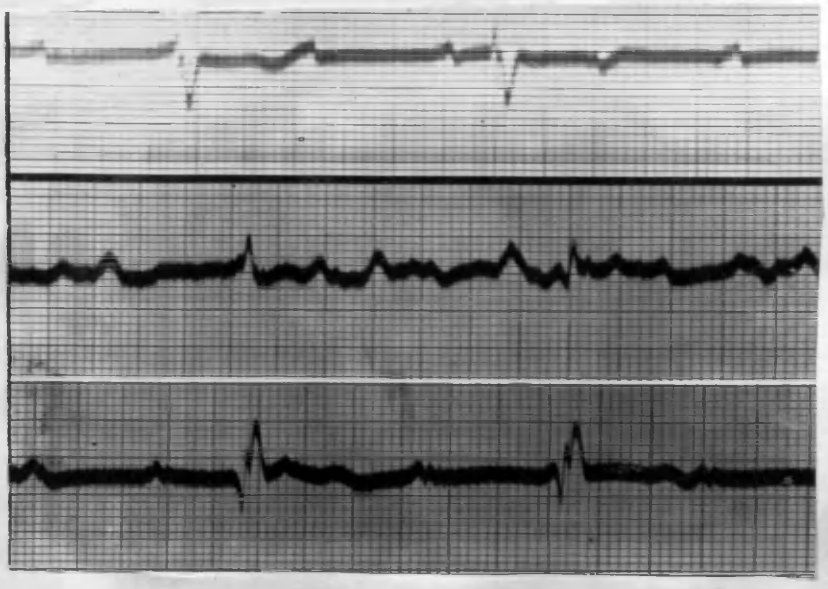

FIBROSIS OF MYOCARDIUM
THROMBUS
INFARCTION


R. P.V.
I.V.C.
L. P.V.
P. V.
PERICARDITIS
INFARCTION


THROMBUS
INFARCTION

Case No. 16.

1.6.31.

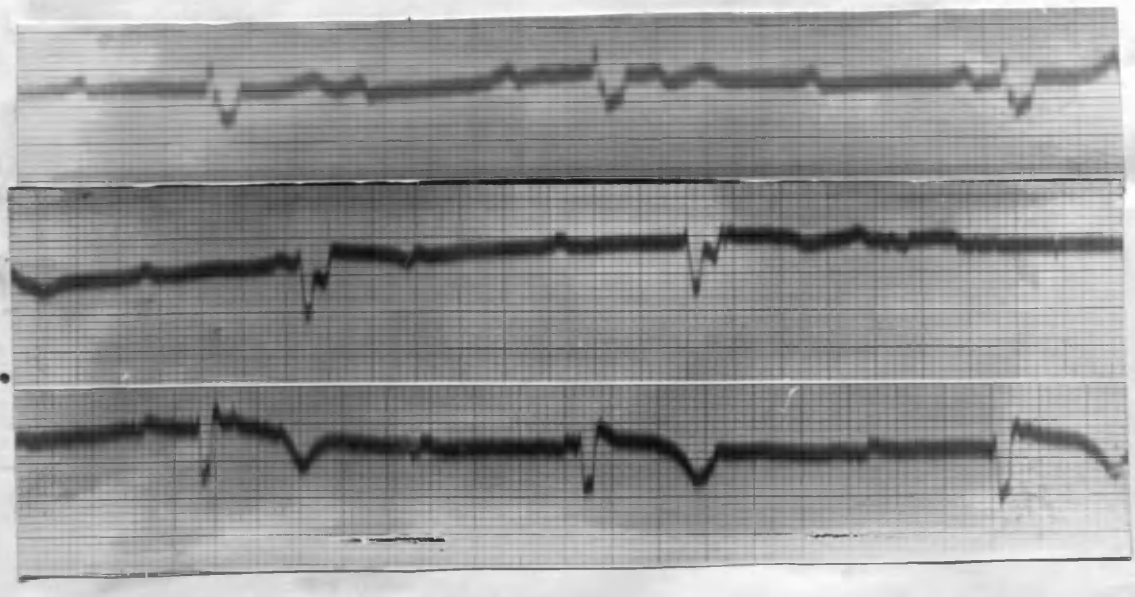


Full Heart Block

QRS broadened and notched.

Left Bundle Branch Block?

3.6.31.



Full H.B.

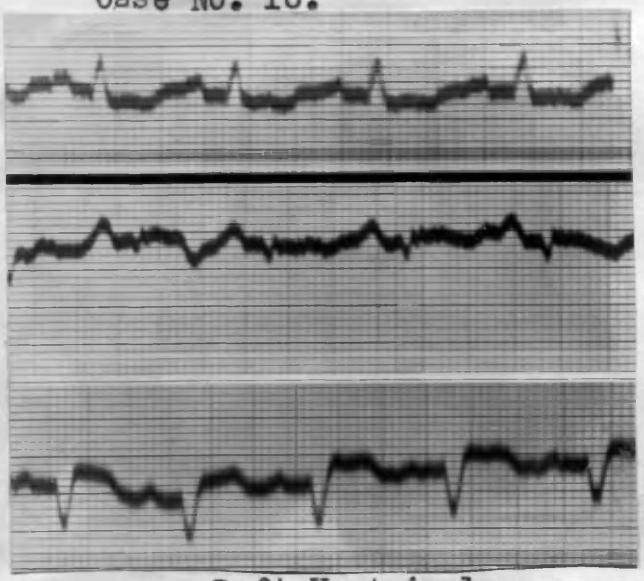
RT elevation in II and III.

T in III Sharply depressed.

Note change of QRS in II.

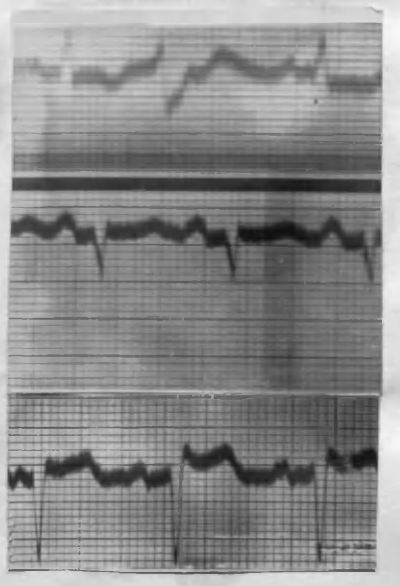
Case No. 16.

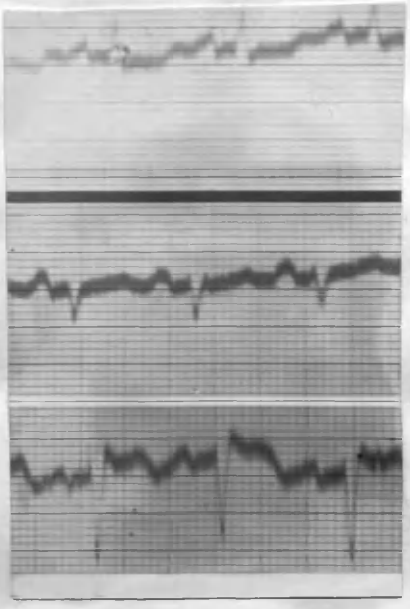
4.6.31.



Left Ventricular preponderance.
RT elevation in III.
Depression in I
Small amplitude QRS in II

6.6.31.





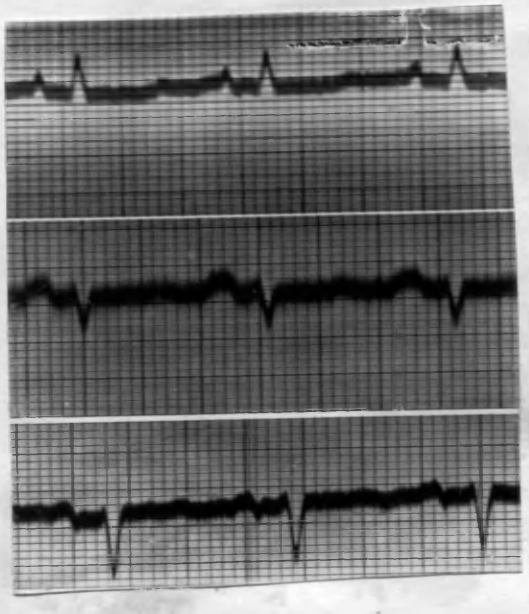
8.6.31.

Persistence of RT elevation in III

Ventricular extra systole Lead I (8.6.31).

Case No. 16.

12.6.31



Left ventricular preponderance.

Poor T wave in all leads.

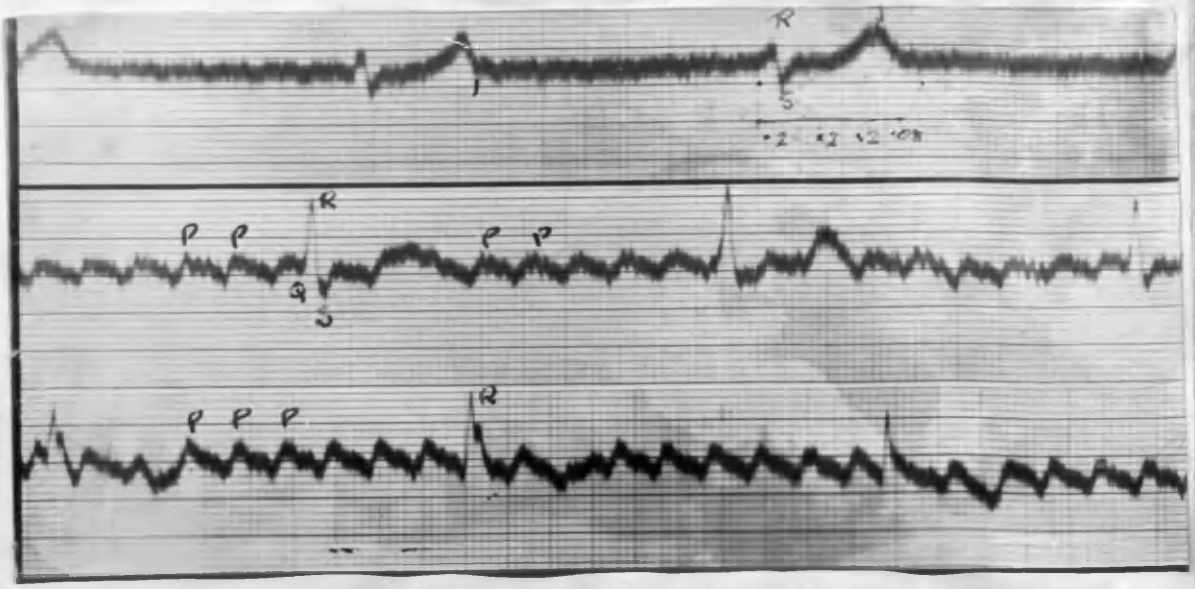
Case No. 68.

Post Mortem Examination.

There is a slight excess of fluid in the pericardial sac. The left ventricle is greatly hypertrophied, and the right considerably enlarged. The aortic cusps show considerable primary sclerosis with some calcareous infiltration at their bases. The other valves appear normal. The myocardium is unduly dense in consistence, but no naked eye evidence of fibrosis or old infarction can be determined. There is some subendocardial fibrosis at the introventricular septum, and muscular papillaris over the left side. The coronary vessels are sclerotic and rather dilated with a few atheromatous patches. The aorta, particularly in its abdominal portion, shows an advanced senile atheroma with extensive areas of ulceration and calcification.

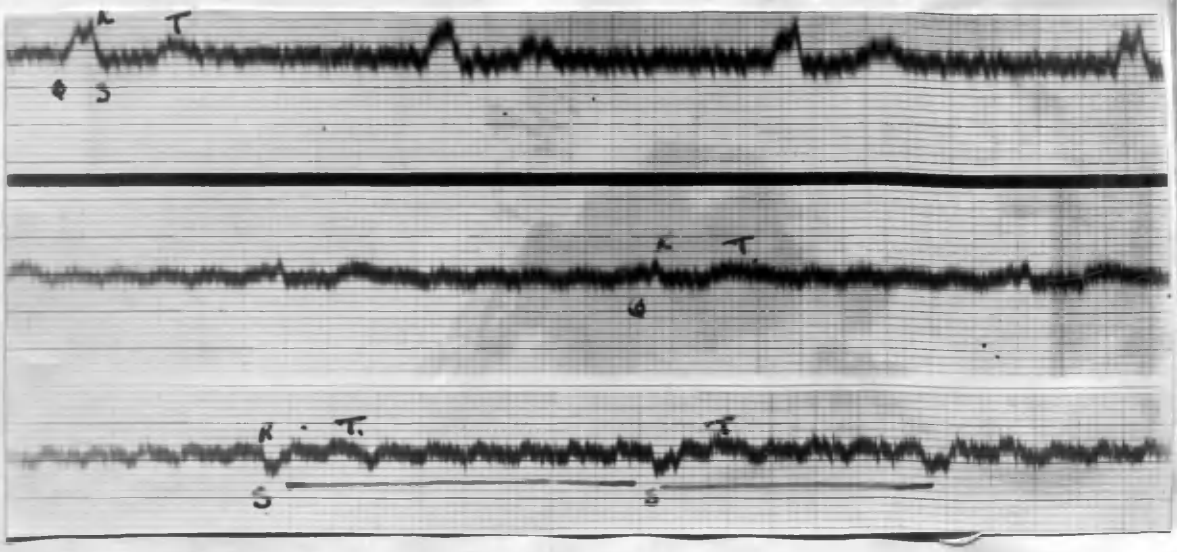
Microscopic examination shows small recent infarction in the region of and involving the Bundle. There is much fibrosis of this region.

2.8.29



Auricular Flutter.
Splintering of QRS.
QRST = ·68. (Full H.B.)

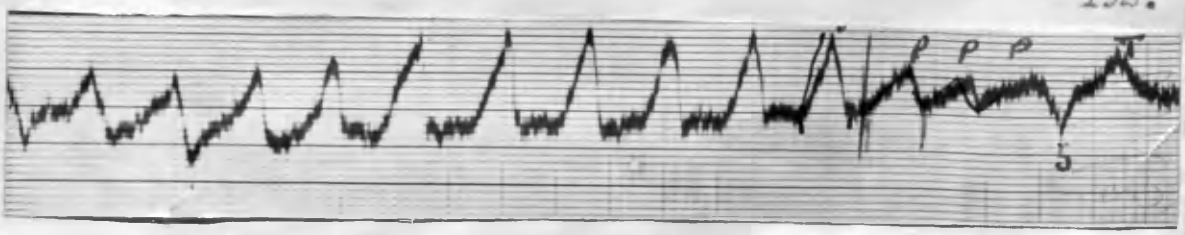
9.8.29.



Auricular Flutter.
Right Bundle Branch Block.
or
Recurring Left Ventricular Extra Systole.

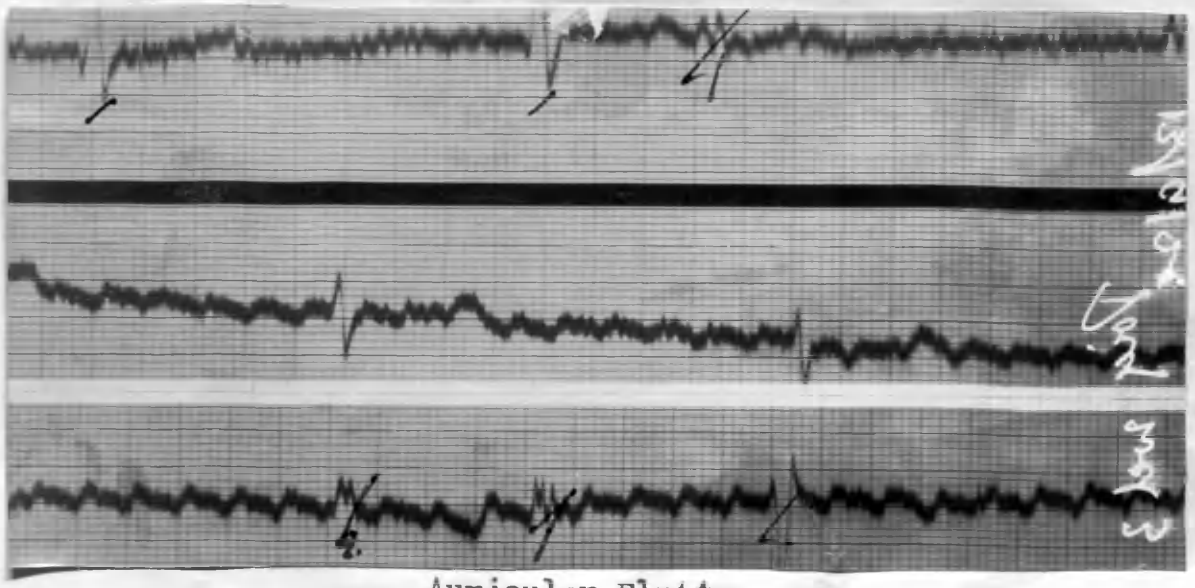
II only.

28.8.29.



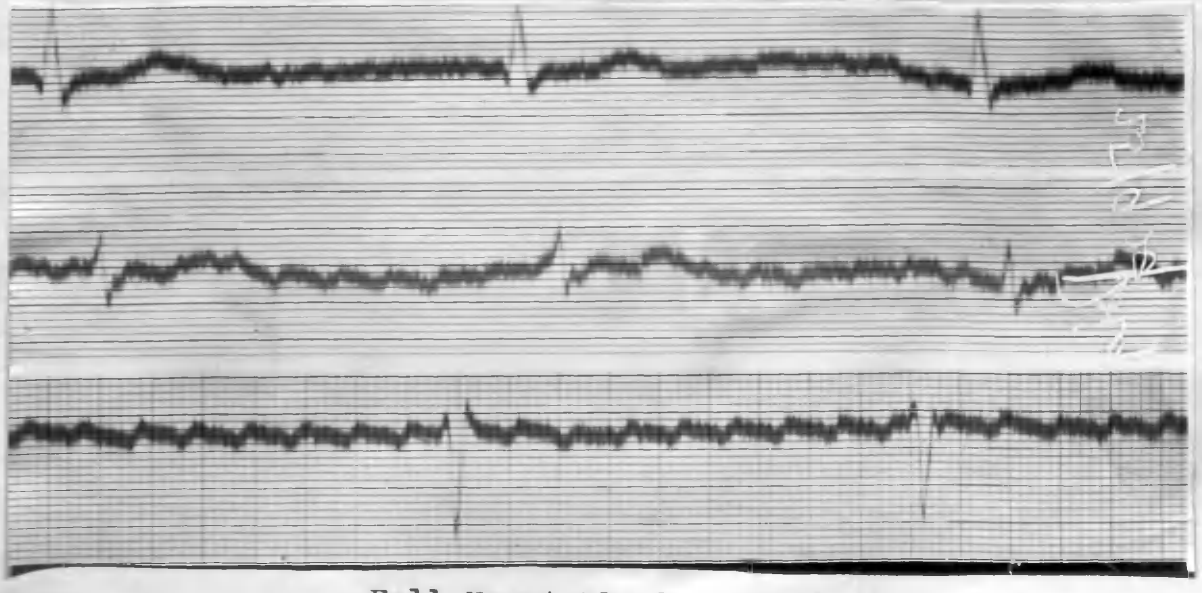
Taken immediately after Stokes Adams Seizure when pulse rate at wrist was rapid. (Termination of Ventricular Paroxysm?).

13.9.29.



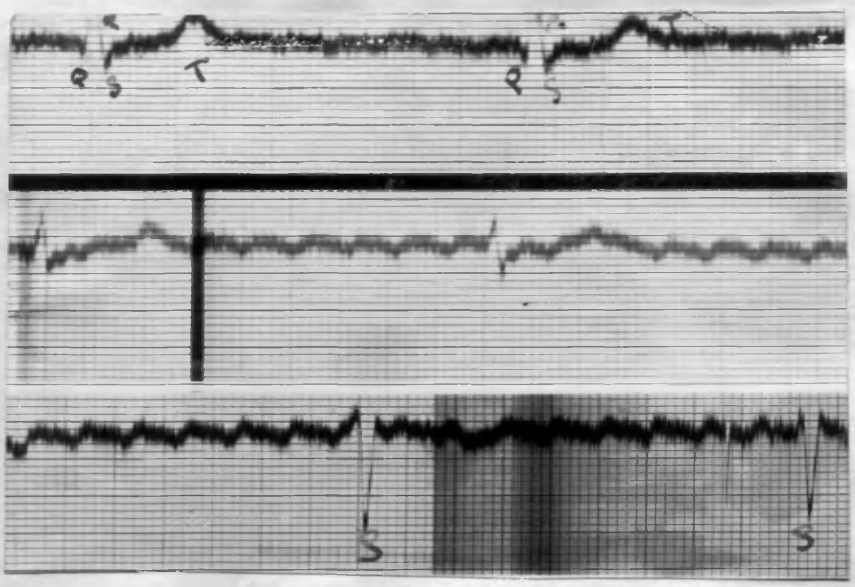
Auricular Flutter.
Note change in QRS in III - Left ventricular preponderance.
Ventricular extra systoles in I and III

25.9.29.



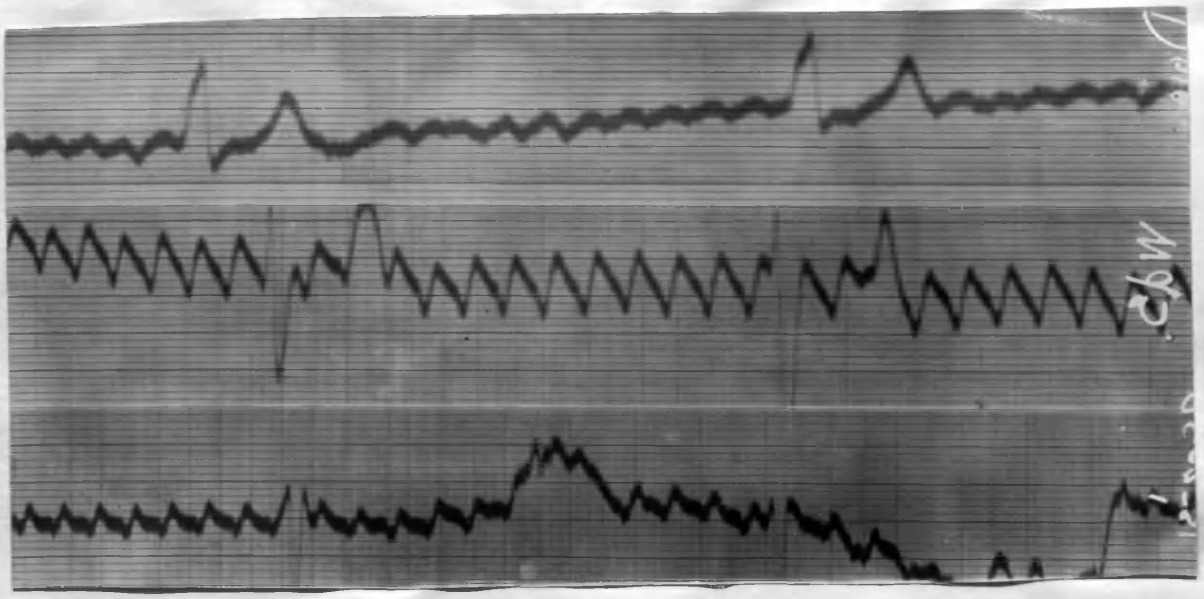
Full Heart Block - Auricular Flutter.

15.10.29.

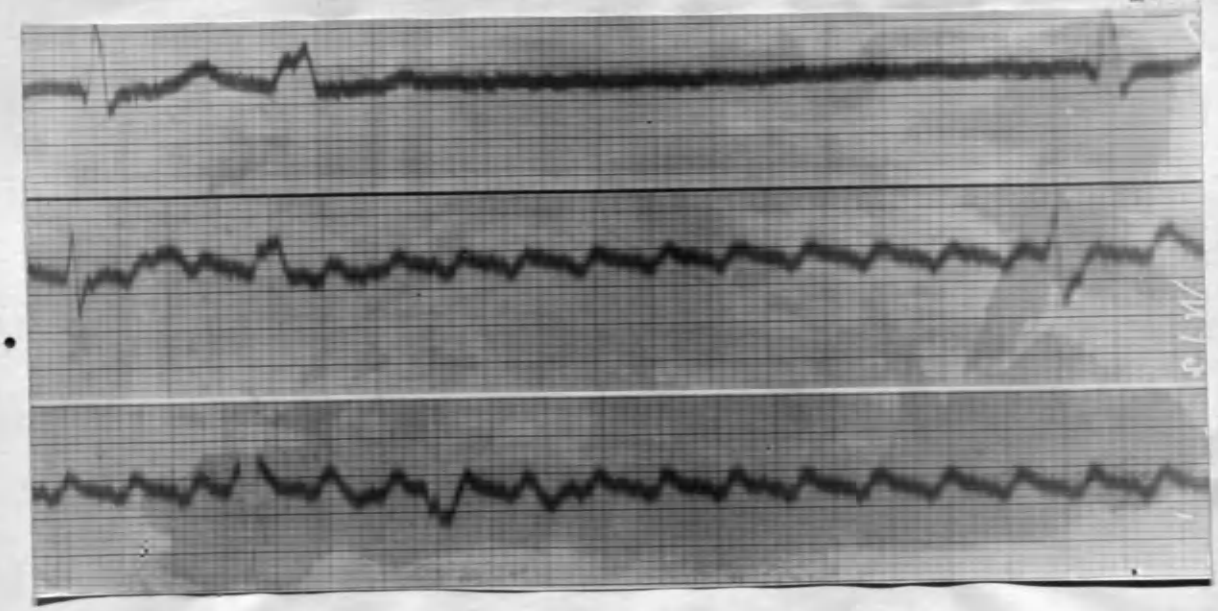


Auricular Flutter.
Full Heart Block.

28.2.31.



Auricular Flutter.
Full Heart Block.

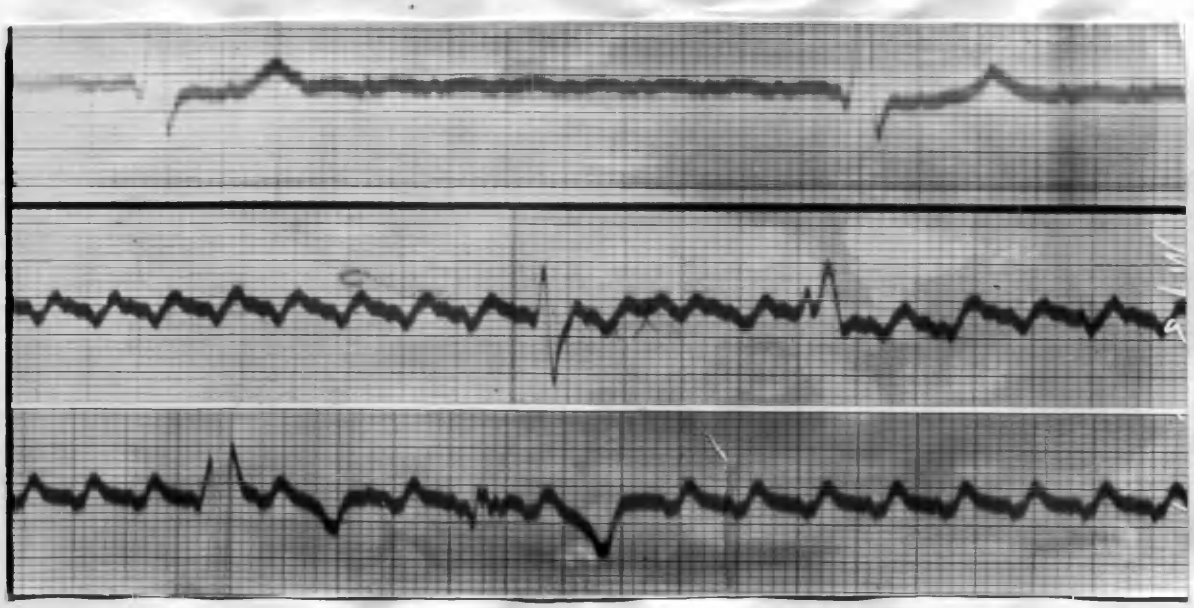


9.3.31.

Auricular Flutter.

Full Heart Block.

Rhythm upset by regularly recurring Ventricular extra systoles.



31.1.31.

Note T wave sharply inverted in III

in normal QRS and in Extra Systoles.

Case No. 17.

Post Mortem Summary.

The heart was enlarged. There was old pericardial adhesions over the apex. A large infarct virtually an aneurysm involved almost the entire wall of the left ventricle. Nearly the whole of the cavity was filled with laminated thrombus. The lumen of the descending branch of the left coronary artery ¾" from its orifice was completely obliterated by a calcified patch. The transverse branch could be felt as a hard cord running round the left side of the heart. The orifice of the right coronary was narrowed but the vessel otherwise was fairly healthy, and infarcts of all ages, some of them gangrenous, were scattered through both lungs.

A recent embolus was present in the right femoral artery.

Liver nutmeg and shrunken.

Kidneys - Acute pyelitis and gravel in calyces.

Case. No. 18.

Post Mortem Summary.

The heart was enlarged.

Descending branch of left coronary was visible and palpable.

Transverse branch of right coronary was palpable.

Myocardium - showed mottling from infarcts and considerable fibrosis. The cavity at the apex was filled with well formed thrombus adherent to the wall.

Case No. 19.

Post Mortem Summary.

Heart was enlarged. There was marked atheroma of aorta and coronary orifices were stenosed but not occluded. Right auricle and right ventricle were dilated. Heart muscle was soft. This softness was more definite at the apex of left ventricle. There was no macroscopic evidence of occlusion of the coronaries.

Case No. 20.

Post Mortem Summary.

Syphlitic aortitis. Stenosis of orifice of left coronary artery.

Area of recent pericarditis at apex. The myocardium under this was soft and looked haemorrhagic.

Further examination showed infarction involving the arterior part of left ventricle at the apex.

CASE HISTORIES.

Case No. 1.

Admitted 13. 1. 27.
Died 24. 1. 27. Age 56 years.

On Tuesday (11.1.27) when walking home at dinner time (12.30 p.m.) patient began to have severe pain down the sternum. He walked on and the pain became worse.

The following day the pain moved round the sides and up the shoulder blades. He was unable to sleep owing to the presence of the pain and could not take food. He vomited yellowish material, very sour to the taste.

Patient was admitted to hospital on 13.1.27.

NOTES ON ADMISSION. Patient was a well nourished and well developed man but pallor was marked. He had attacks of breathlessness accompanied by increased severity of the pain. These were not reduced by sitting up. The mucosae were fairly well coloured.

Respirations varied in rate, being more rapid when the pain was worse. Chest expansion was poor owing to the pain on taking a breath. There were some rales at both bases.

The pulse was weak and easily compressible. Apex beat was in the 4th Interspace 9½ cm from middle line.

Case No. 1 (Continued).

It was not easily palpated. The cardiac sounds were distant. There was pericardial friction over the praecardium, most marked at the apex and heard outside the area of cardiac dullness to the left.

Blood Pressure 100/80.

The tongue was thickly furred, brown in the centre and white at the sides. The abdomen was full and slightly rigid. There was no abdominal tenderness. Liver and spleen were not palpable.

Cardiac sounds became progressively weaker and on 18.1.27 were almost inaudible. On the same day sputum contained fresh blood and dullness with diminished breath sound was detected at the base of left lung.

On 22.1.27 he developed an erysipelas of the left side of face with severe pain.

He sank and died on 24. 1. 27.

Temperature. Subnormal throughout.

Blood Pressure.

13. 1. 27	100/80.
15. 1. 27	95/70
16. 1. 27	90/75
17. 1. 27	100/82
18. 1. 27	108/78
19. 1. 27	82/70
21. 1. 27	110/75.

W.R. Negative.

Case No. 2.

J.McL. Aged 64.

Admitted 5. 8. 29.

Died 7. 8. 29.

Patient was stoutly built man. His complexion was rather pasty. There was marked cyanosis. He was a little dazed but answered questions sensibly. He stated that on Sunday evening 4/8/29 he was compelled to give up work because of intense epigastric pain and feeling of pressure and choking in the chest. He was able to walk home with rests at intervals. During the night he was sick and vomited some green and yellow material. Pain was continuous until his admission at 7-30 p.m. on 5/8/29. He was able to walk part of the way to the Infirmary. At 11 p.m. he had a severe attac of pain, breathing was difficult and he was sick and vomited a little (about 1 oz. greenish material).

Pulse was rapid, soft and low volume. There was irregularity due to occasional extra systole. Cardiac area was not greatly enlarged. Cardiac sounds were of very poor quality, the 2nd sound being almost inaudible. There was no friction. There was a pulsation in the jugular veins. Tongue was furred He was inclined to be constipated. He had no gastric disturbances but for a year previous exertion had tended to induce epigastric discomfort. His appetite had always been good.

Case No. 2 (Contd).

Abdomen was lax. There was no general or local tenderness or rigidity. Liver and Spleen were not palpable. Chest was clear in front. Back was not examined.

The following night dyspnoea was troublesome, and pain over praecordium was constant.

The patient was asleep during the night of 7/8/29 when it was noticed that he was a little restless, and that his breathing was disturbed. His colour suddenly became very grey, and he expired in about 5 minutes very quietly.

Temperature was elevated to 99°F on 2nd and 3rd days.

Blood Pressure	102/44	5/8/29.
	90/40	6/8/29.
	82/50	7/8/29.

Case No. 3.

Report by Dr. J.F. Cowan. 204.

Andrew McAlpine, 59. Vii. 89 67. 18.ii. -9.iv.30 Death p.m.

A healthy man who had had attacks of angina on exertion for about a year before admission. These were accompanied by breathlessness, and were more frequent when he felt cold. Recently the attacks had been induced more easily, and they had sometimes wakened him from sleep. Palpitation occurred at times. For the last six months he had taken a bus to his work, though he only lived a short distance away from it.

On 17.ii. after supper, he was preparing to go to bed when another attack, the most severe that he had experienced, ensued. He vomited on several occasions. In the early attacks the pain radiated to the ulnar side of the left arm, but on this occasion affected both arms.

On admission he was evidently suffering from severe pain which persisted for more than twenty four hours after admission, notwithstanding the administration of morphia. Subsequently his attacks were minimal and of short duration.

But on 26.ii a severe attack ensued about 9-20 a.m. and continued until the afternoon. The pain was mainly epigastric, and radiated into the chest and the left arm as far down as the wrist. His pulse which had been running about 80 and was quite regular, was now over 100 and had lost notably in volume and strength. The cardiac sounds were very indistinct. Two hours later the pain was lessening

and the pulse had regained some of its former volume and strength. The pain passed in the afternoon and he had a good night.

Another fairly severe attack occurred on 4.iii. but after the middle of the month the attacks lessened in frequency and in severity and he was very cheery and well. He told J.M.C. on 1.iv. that he had never been so free from pain since the onset of his illness.

On 9.iv., at the visiting hour his wife and a friend whom he had not seen for long were visiting him, and he was rather excited at the meeting. Another attack supervened, he became very blue and pulseless and died almost instantaneously.

Pulse. 120 on admission slightly fell and was running at 80 on 25th.

25th. 84/108.

27th. 88/110.

28th. 72/96.

Since then in vicinity of 80.

Temperature.- 99 to 99.6. 1/9.

22nd. 100.2

26th. 98/100.

27th.

28th.

4/4. 98.8

Blood Pressure.;- Systolic 99.5.

	Systolic.	Diastolic.
27th.	92	70.
28th.	90	68.
1/3.	85	60
After 7/4.	95/105	60/80.

Case No. 4.

Thomas Gibson.

The clinical history of this case was described by Dr. J. Norman Cruickshank in British Medical Journal, April 11th 1931.

Case No. 5.

R.C. Aged 58. Leatherworker.

Admitted 20.8.28. Died 21. 8. 28.

Eighteen months before, patient had severe attack of praecordial pain when running for a car. Pain was severe for several days. He resumed work after fourteen days. Pain, breathlessness and swelling of feet recurred in cold weather.

On 20. 8. 28 patient collapsed on his way home from the bowling green. Pain was continuous over sternum. He was sick and vomiting. The following day patient complained of pain over praecordium radiating into the axilla. His left arm was numb. He was ashen grey and orthopnoeic. Pulse was regular but weak. Cardiac sounds were poor. Respirations were 30 per minute. Pain was relieved by morphine.

At 2 p.m. patient vomited and became cyanosed. Pain and sickness recurred an hour later and he became very cyanosed and gradually sank and died at 8.30 p.m.

Temperature was subnormal throughout.

Blood pressure - 110/80 mm. Hg.

Pulse Rate was over 100 per minute.

Case No. 6.

J.D. Aged 64. Craneman.

Admitted 22.6.27 Died 29. 7. 27 (1-30 a.m.)

On 11.6.27 patient had an attack of pain of great severity in left side of chest when returning from work, By 17. 6. 27 pain had improved sufficiently to allow him to resume work. The following day it returned but was not constant. Vomiting gave temporary relief.

On 20.6.27 he had an attack of severe pain in left hand and arm accompanied by intense dyspnoea. Two days later he was admitted to hospital.

On admission expression was anxious. Pulse was regular. Apex beat was not palpable. Cardiac sounds were weak but pure.

On 7. 7. 27 patient became delirious with praecordial pain. Faint praecordial friction was audible. Urine contained albumin and blood.

He gradually went downhill from that date.

On 16.7.27 both legs were oedematous.

A week later there was effusion in both sides of the chest.

At 12.5 a.m. on 29.7.27 he became breathless and cyanosed and died within an hour.

Case No. 6 (Continued).

Temperature ranged from 97° - 99.4°F.

23. 6. 27. Temp. 99.4

28. 6. 27. 99.2

29. 6. 27. 99.4

2, 3 & 4. 7. 27. 99

Pulse 124 on 2nd July. Continued between 130 - 100.

B.P. 120/60. July 4th 140/90. 10th, 105/70.

W.R. - ve.

Case No. 7.

J.B. Aged 65. Meat Van Driver.

Admitted 20. 8. 28.

Eight months before, he had a severe attack of praecordial pain with sickness and breathlessness.

He resumed work after three weeks.

Four months later, he was compelled to give up work because of increasing breathlessness. Pain was constant in praecordium and epigastrium.

In July feet became swollen and oedematous.

20.8.28. Pulse irregular - extra systoles.

Cardiac sounds poor. Duplication of 1st sound at apex.

P. 55/120. T. 97/normal. R. 30.

23.8.28. O.E. Fundi normal.

4.10.28. Pulse rate high, 110 per minute.

Lungs clear. Flatulence troublesome.

11.10.28. Patient collapsed suddenly when reading and died.

Temperature - Varied from normal to sub-normal.

Blood pressure.		
	20.8.28	120/80.
	25.8.28	140/110.
	15.9.30	110/70.
	10.10.30.	110/70.

W.R. - ve.

Report from Dr. J.M. Cowan.

Robert McMaster - 44 - Admitted 19.5.29, died 8.VI.29.

The story is imperfect. He seems to have been a healthy man who took ill suddenly on 31.XII.28 with acute abdominal pain in the left side of the abdomen. He was admitted into hospital on 2.1.29 and a laparotomy was performed.

He left hospital on 18.1.29, and returned to work a week later. He was, however, short of breath upon exertion and this steadily increased in severity. He suffered from attacks of pain in the abdomen, and from a cough which was most troublesome at night. His appetite was impaired, but he had no discomfort after food or vomiting. In March his symptoms became exaggerated and he had to cease work. The abdominal pain became constant, and the dyspnoea on exertion more severe.

On admission he was orthopnoeic, with cold blue extremities and minimal oedema. The pulse was frequent, weak and irregular (E.S.) the area of cardiac dullness increased to the left side, and the cardiac sounds were distant, soft and probably impure. There was some dullness at the right base with deficient R.M. and rales were audible at both bases. J.M.C. thought that the trouble was

primarily pulmonary. Improvement followed and at the end of the month he was lying low in bed, with no discomforts, but he was still breathless on any exertion. On 5.vi. he had a short attack of pain over the lower part of the cardiac area accompanied by some breathlessness. On 6.vi. he was very well and free from discomfort, but he still required 3 pillows and although his respirations were charted 20-22, he became short of breath when talking or excited. The heart was smaller, and the sounds were pure but rather indistinct. The second sounds had no special accentuation. The chest was clear.

"J.M.C. was inclined to look upon the case, on admission, as a "pulmonary" one, but this opinion requires revision, as it is quite clear now that there was a considerable cardiac element at that time. His chest is WELL SHAPED. The history is quite unreliable".

At night he had an attack of acute pain in the epigastrium.

He became breathless, pale and cyanosed. The breathing was frequent and laboured and he had great discomfort. He had a restless night, and in the morning was worse. The pulse was imperceptible at the wrist and 100 at the heart. (It fell to 80 at night). The pain which was now experienced in the epigastrium and lower thorax was severe.

He died at 10 p.m.

The temperature throughout was subnormal.

The pulse rate was not very frequent, running about 100 on admission and then falling and touching 60 on 22.V, rising again on 22, 23.V. to similar levels, falling to normal on 24, 25.VI, and thereafter running about 90.

The respirations were always between 20 and 30 p.m. until 7.VI, when they rose to over 40 p.m.

The urine was generally clear, occasionally showing small trace of albumin.

Case. No. 9.

Report from Dr J.M. Cowan.

Name, James Wilson,	Ward 7 Book 78 Case No. 43.
Address, 108 Widson Terrace	Admitted, 24.4.29.
Age, 56.	Died, 27.4.29.
Occupation, Steelworker.	

Complaint . A "night's vomiting" followed by pain over the heart for two days and then 14 days less severe pain in the chest and abdomen accompanied by gasping breathlessness.

6.4.29. Well and at work.

7.4.29. (Sunday). At 10-30 p.m. he became violently sick whilst in bed.

Vomiting consisted first of food and later greenish material. This was replaced by retching which continued until the following morning. He had no pain until Monday. Pain developed about the praecordium. Pain was stabbing but did not radiate. It was increased by movements. At the same time he became very breathless.

Pain now in epigastrium and dull in type became less intense in a day or two. Breathlessness continued.

Patient was not confined to bed but was definitely orthopnoeic.

24.4.29. Very distressed and breathless. Lips blue. Fully conscious. Pulse very soft and irregular. Uncountable. Extremely orthopnoeic. No oedema. Cyanosis ++.

Case No. 9 (contd.)

25.4.29. Urine - albumen. Praecordial friction not heard. Rales at both bases.

26.4.29. Vomited and extremely restless. Abdomen distended with flatus. Oedema of feet and legs. Later at night he rested a little but had a recurrence of his restlessness and died suddenly 7-15 a.m. 27.4.29.

Case No. 10.

S.M. Age 40. Steel Worker.

7. 6. 27.
19. 6. 27.

Two years ago when out walking, had a sudden attack of substernal pain, which radiated to left shoulder. Since then he had not felt well and he was treated in Ruchill Hospital 1926 - February 1927 for Pulmonary Tuberculosis.

On 27.5.27 when out walking, he developed intensive abdominal pain - vomiting temporarily relieved pain. He was constantly sick until admission 7.6.27.

He was orthopnoeic and expression was anxious. Breathing was rapid and shallow. There was cyanosis of lips and ears. Feet, legs and thighs were oedematous.

Pulse was rapid but regular. Heart was enlarged downwards and to the left.

A loud systolic murmur was audible at apex and base with conduction into axilla and vessels of the neck.

Two days after admission, friction was detected at 2nd left costal interspace, and on 16.6.27 pulse had become shotty and capillary pulsation was present. Apical systolic was now less and a diastolic murmur was audible at aortic area.

He died suddenly whilst asleep on 19.6.27.

Case No. 10. (contd.).

Temperature. Was normal to subnormal throughout.

Blood Pressure.

7. 6. 27	130/60
14. 6. 27	160/60
18. 6. 27	140/60

W.R. +ve.

Case No. 11.

J.K. Aged 57.

Admitted 8.5.28.
Died 11.6.28.

15 months before admission patient was awakened with severe praecordial pain and breathlessness which lasted 3 hours. After a month in bed he felt fairly well but exertion caused recurrence of pain.

About a month before admission to hospital he became breathless even on the slightest exertion and was unable to sleep lying down.

On admission he was orthopnoeic and breathing was rapid and shallow. There was cyanosis of extremities and back and face were oedematous. Pulse was regular and arteries were hard and tortuous. Heart was enlarged downwards and to the left and there was soft systolic murmur at apex.

Urine contained albumen and many hyaline casts were seen on microscopic examination.

Patient's condition gradually deteriorated. Oedema increased and breathing became cyclic.

On 11.6.28 he had a rigor and died in a few moments.

Temperature. Normal throughout.

Blood Pressure. 9.5.28 180/120.

21.5.28 142/120.

W.R. - ve.

Case No. 12.

J.B. Aged 55.

Admitted 6/6/30.
Died 13/7/30.

He stated that he was feeling perfectly well until Sunday 4th May when at 10 p.m. he felt his head was going to burst.
He had no vertigo.
He sat down and found that he was panting for breath.
He recovered in a few moments and went to bed. He had pain below the breast bone. This pain persisted for two days.
He felt his pulse and found that it was jumping irregularly.
Note on Admission :- Patient is a well developed muscular man. Complexion is slightly cyanotic. The tongue is clean. He is edentulous. There is no oedema. Cardiac action is constantly irregular and the rate by auscultation is about 120. A short inconstant systolic murmur is audible between the apex and foot of sternum. The arteries show slight but definite degenerative changes. The lungs are clear.
The urine contains a small quantity of albumen.
Patient remained breathless with a distinct tendency to cyanosis and on 30/6/30 there was slight oedema of the lumbo sacral region.

On 1/7/30 there was a little moist rale at the left base.

Case No. 12 (Contd.)

Oedema and cyanosis gradually increased.

Patient died on 13/7/30.

Temperature remained normal except on 12/7/30 and 13/7/30 when it reached 100°F.

Blood Pressure was constant at 160/90.

W.R. - ve.

Case No. 13.

D.F. Aged 52. Woodman.

28.11.29 - 4.12.29.

Patient had been short of breath on exertion for several months. Two months before admission he was in hospital at Dunoon. Shortness of breath was troublesome and he required four pillows. Since that time patient had been more or less confined to bed - the slightest exertion produced dyspnoea. He had slight transitory attacks of praecordial pain. This could not be associated with exercise or taking of food. There had been no substernal pain or palpitation.

Temperature normal. B.P. 150/70.

On admission patient was well developed, sallow complexioned man. His face was rather haggard. There was very marked oedema of both feet and legs and some ascites.

Liver was enlarged and palpable.

Pulse was of fair quality and regular.

V.S. and V.D. murmurs were audible at aortic area. The diastolic murmur was very variable in intensity.

On 4.12.29 patient complained of feeling tired.

In the afternoon he complained of feeling sick and died suddenly.

Temperature - normal.

Blood Pressure - 28.11. 29. 150/70.
2.12. 29. 120/40.

Leucocytes - 3.12. 29. 11600.

Case No. 14.

A.N. Aged 61. Watchman.

13.11.29. 4.12.29.

Patient had suffered from pains radiating down the arms. These pains were much more marked after a heavy meal or on exertion. There was no substernal pain.

Four months before he noticed that he was very breathless when climbing stairs.

Three weeks previous to admission dyspnoea was much worse. The slightest gradient caused him to feel praecordial constriction. He continued at work until 11th November when he was compelled to cease work because of extreme breathlessness. He was restless at night; had to sleep propped up; and was troubled with palpitation.

On admission patient looked older than stated age. He was slightly orthopnoeic. Breathing was definitely periodic. There was no oedema. There was considerable arterial degeneration. Carotid pulsation was visible. Radial pulses were equal, full and shotty. Short V.S. and V.D. murmurs were audible at the aortic area. Lungs were clear. Liver was just palpable and slightly tender.

On 1st December he felt short of breath. Pulse was rapid and irregular. The condition suggested Auricular Flutter.

Two days later breathing was definitely Cheyne Stokes with long periods of apnoea. Pulse was rapid but regular. There was rale at both bases.

On 4th December patient died suddenly.

Case No. 14. (Continued).

Temperature -	21 & 22/11	99.
Blood pressure -	150/60	13/11.
	120/50	15/11
	80/40	26/11
	120/40	2/12.

W.R. + ve.

Case No. 15.

Name. Mrs McLennan,

Address, 20 Grafton St. Townhead.

Age, 49 years.

Admitted 18.3.31.

Died 31.3.31.

Notes from Journal.

Complaint. Breathlessness and feeling generally out of sorts of nine or ten months' duration. Also a haziness over the eyes of a year's duration.

History of present condition. Patient has not been at all well for three years. Since then she felt generally out of sorts. For at least two years she has been troubled with a very severe thirst and used to drink pints of water during the day. At that time she passed a great amount of urine, and had to get up five or six times during the night, and this fact kept her from coming to hospital. In the last two years she has been complaining of marked weakness and has been gradually losing weight.

About 14 months ago she noticed a haze over her eyes and went to the Infirmary where she was found to have retinal haemorrhage. Since then she has never felt well.

About 12 months ago she felt a numbness and tingling in both feet and also in the left arm. This is still present.

Whenever she exerts herself she is very breathless, and she

Case No. 15 (Contd).

feels as if she is going to die. Her feet swell every night. During these attacks of breathlessness she has no pain. She has never been jaundiced. About a year ago, she had frequent attacks of vomiting, accompanied by pain under the sternum which went through to the back. This vomiting started about an hour after the taking of food and vomit consisted of stomach contents. She has never had heartburn or water brash. She is greatly troubled with constipation but her motions have never been black. She is greatly troubled with flatulence. About a month ago she had an attack of praecordial pain and breathlessness. Pain lasted several hours and was relieved by injection. She was sick and vomited during the night. Flatulence was troublesome. The following day she was very ill and breathless.

She has vomited every day for past three days and has brought up everything she ate. She has not been troubled with diarrhoea. She has a slight cough with a scanty spit.

19.3.31. Patient is a stoutly built woman, and has somewhat flushed cheeks. She states that she has frequent attacks of sub-sternal pain; sometimes very severe and on the night of the 15th, she had to have an injection to relieve it. The pulse is infrequent and tending to couple. Vessel walls are somewhat hard. There is a short aortic systolic murmur. The lungs are clear. The abdomen is normal. The urine

Case No. 15 (Contd.)

contains a good deal of albumen, sugar and much uric acid, with a few hyaline and granular casts.

Patient had an attack of breathlessness during the night. The attack came on suddenly. Palpitation was troublesome; she felt afraid of dying. There was no pain and the attack passed off gradually. Pulse at present, is regular.

29.3.31. Patient has had frequent attacks of breathlessness; pain in praecordium - passing up the left side of chest - she feels sick but does not vomit.

31. 3.31. Patient died suddenly today.

Temperature. 98°F.

Pulse. 96.

Respiration. 24.

Blood Pressure. 154/100.

W.R. - ve.

Case No. 16.

T.C. Aged 63.

Admitted 1/6/31.

Dismissed 14/6/31.

For four years previous to admission patient had suffered from recurring attacks of praecordial pain brought on by exertion and relieved by rest and amyl. nitrate. On 31/5/31 he was awakened by intense pain in the left lower thorax radiating upwards into the right breast. He stated that the pain which he experienced was quite different from his previous attacks of angina. It was situated in the gall-bladder region and above it, and also below the sternum. It did not radiate into the arm or the neck. Vomiting soon began and with retching was almost incessant. He sweated considerably.

On admission patient was orthopnoeic and slightly cyanosed. Tongue was coated and breath offensive. He vomited a little but there was no pain. The pulse was regular, very soft, and the cardiac sounds were of poor quality. On attempting to take the blood pressure he fainted with convulsive movements. At 12-30 a.m. on 4.6.1931 patient had a short attack of unconsciousness with stertorous breathing and failure of pulse. Seizure lasted 1½ minutes and was repeated at 1-30 a.m. Patient was better by morning. Pulse which had been irregular

Case No. 16 (Contd).

on previous day was now regular and of better quality, and cardiac sounds were better. There was no bile in the urine. There was no jaundice, but the stools were quite white.
The anterior or 1/3rd of the left foot and all the toes were discoloured and cold. No pulse could be made out in the tibials and dorsalis pedis.
On 5/6/1931 the foot was warm and the toes were still discoloured - there was some oedema. Patient's general condition is better. The pulse rate had risen and was almost regular. Colour was improved.
On 6/6/1931 patient was rather better. Irregularity of pulse was only very occasional. The conjunctivae were bilestained and the skin was a little yellow. There was no bile in the urine, but uric acid was present.
On 9/6/1931 there was marked oedema in lumbo-sacral region. He complained today of burning sensation in right foot, which was much healthier looking, the circulation having been slowly restored.
He had another Stokes-Adam seizure at 2-30 p.m. lasting for a minute and a half.
On 14/6/1931 patient who apparently had been doing well while talking to his son during the visiting hours suddenly fell back dead.

Case No. 16 (Contd.).

Temperature was normal except on last two days when it rose to 99°F.

Blood Pressure 85/65 on admission and thereafter whole course of illness except on 6/6/1931 when it reached 98/68.

Leucocytes.		
	2/6/31	13,200 per c mm.
	4/6/31	13,000 per c mm.
	8/6/31	9,200 per c mm.
	9/6/31	9,000 per c mm.
	11/6/31	14,000 per c mm.

W.R. - ve.

Case No. 17.

J.M. Aged 65. Quartermaster.

9.11.26. - 19.12.26.

Pain and catching of breath on left side of chest of six months duration. Pain on right side of fortnight's duration.

Pains in kidney regions of four days duration,

Six months before, whilst in Australia, patient had sudden attacks of pain over mid sternum and passing to the left side; it was accompanied by shortness of breath. Previously he had been troubled with pain at lower end of sternum produced by exercise.

He was under constant medical attention from the onset of illness.

The pain in kidney regions, four days before admission to Royal Infirmary, had been most severe, He had twice fainted and was unable to pass urine for 15 hours. He had a racking cough ever since admission to hospital in Australia and from time to time brought up blood stained sputum.

Patient was orthopnoeic. Respirations were rapid and wheezing. There was restrained cough. Colour was good but malar flush was noticeable. There was no oedema, skin was dry and there was no enlargement of lymphatic glands.

Case No.17 (Continued).

Sputum was muco purulent and mixed with red blood. There was marked hyperaesthesia of left axilla with dullness, diminished R.M. friction. Coarse friction was audible over the R. upper lobe. Cardiac sounds were weak and of poor quality. No adventitious sounds were heard. Pulse was regular.

X-ray of chest on 15.11.26 showed patches of tumour formation with heart dilated.

On 20.11.26 it was noticed that left arm was oedematous and there was prominence of left External Jugular Vein.

Larynoscopic examination showed both vocal cords moving. Left was sluggish.

Patient's progress was downhill.

On 3.12.26 right arm was oedematous and distended veins on left side of neck were thrombosed.

On 7.12.26 he had oedema of feet and legs.

He died at 6.25 a.m. on 19.12.26. Jaundice had been present during the last week and oedema of hands, arms legs and feet had been gross.

Temperature - Slight irregular pyrexia for a month - subnormal for last ten days.

Case No. 17 (Continued).

Blood Pressure	17.11.26.	100/70.
	24.11.26.	90/70.
	1.12.26.	100/70.
Leucocytes.	13.11.26.	21500 per c.mm.

W.R. - ve.

Case No. 18.

J.T. Aged 55 years. Railway Canvasser.

Admitted - 5.10.27.

Died - 9.10.27.

Five years before admission to hospital patient had a sudden attack of abdominal pain. Appendicectomy was performed. From that time patient did not feel well. He suffered from frequent attacks of epigastric discomfort, made worse by exertion.

Six months before admission these attacks became much more frequent. Heartburn and flatulence were now almost constant.

Patient was awakened from sleep six weeks before admission by intense praecordial pain. Pain started suddenly, radiated through to the back and down both arms to the finger tips. It was vice-like. He was very short of breath and felt that he would die. He felt nauseated, had giddiness, with some vomiting which relieved the pain. Pain lasted for six hours and as it was passing off patient perspired freely and had some palpitation. From that time praecordial pain recurred almost every week and was brought on by exertion.

Patient was a worn looking man. Cheeks were red - There were many injected venules. Pulse was small, soft and regular. Cardiac area was enlarged downwards and to the left.

Case No. 18 (Contd).

Cardiac sounds were pure but of very poor quality. Moist rale was present at bases of both lungs.

On 9.10.27 patient had slight pain in left side of chest about 5.45 p.m. This gradually increased in intensity and became agonising. Pain was partly relieved by morphine.

He died in extreme agony at 8 p.m.

Temperature was normal throughout.

Blood pressure - 100/75 5.10.27.

100/78 9.10.27.

W.R. - negative.

Case No. 19.

John Guy. Aged 61.

Was admitted 1.8.27.

On 31.7.27 patient was troubled with pain in the left arm and extreme breathlessness. He collapsed in Queen Street Station.

On admission he looked shocked, breathlessness was marked and he was cyanotic. Pulse was soft and irregular, the irregularity being due to extra systoles.

Heart sounds were of poor quality.

The patient died that day.

Temperature - Subnormal.

Blood Pressure - 80?/

Pulse rate - 110.

Case No. 20.

Edwin Hoddle. Aged 45.

Admitted 22.6.27.

On 13.6.27 the patient whilst out walking had a sudden attack of praecordial pain and breathlessness. He collapsed and was taken home, and was confined to bed from that date.

From 13th until his admission on the 22nd he was troubled with slight cough and occasional discomfort under the sternum.

On the morning of 22.6.27 he had an attack of severe pain in his chest and down both arms. He perspired freely. Pulse during the attack was regular.

Coarse praecordial friction was heard. Moist rales were present at both bases.

Heart sounds were soft and distant. Pain was relieved by morphia.

At 8 p.m. patient had another severe seizure and complained bitterly of pain. He lapsed into unconsciousness at 8.30 p.m. and died at 9 p.m.

Temperature - Subnorm 90/60.
Blood pressure - 90/60.
Pulse Rate - 110.

Gavin Allan,
63 Meadowpark Street,
Dennistoun. Age 64 years.

Admitted 4.9.28.
Died. - 5.9.28.

Present Illness.

When patient was on his way to his work this morning he collapsed. He had been feeling quite well and had taken quite a satisfactory meal before leaving home. Following his collapse he was assisted to gate. When seen there he was quite conscious, but very cyanosed. He complained of feeling sick, but no pain whatsoever was present.

Condition on admission.

P. 60. T. 97 R. 20 B.P. 80/60.

Patient was very cyanosed - his lips and ears were quite blue and there were blue injected venules on his cheeks. He complained of no pain but said he felt slightly sick. His skin was covered with a clammy sweat and his hands and ears were very cold. Pulse was regular but of small force and volume; the heart sounds were poor in quality but no murmurs were detectable. He complained of weakness in his legs but no paralysis was present. Knee jerks both active and pupils reacted to light.

Patient vomited shortly after admission and continued to do so frequently throughout the day. His general condition remained much the same. He complained of a numb sensation in his right leg from time to time. Both lower extremities tended to be cold and hands remained bluish.

During the afternoon he complained of general abdominal discomfort, and towards evening this seemed to amount to pain.

About 9 p.m. abdominal pain was severe and patient became restless. B.P.70/50. He had a great desire to sleep but could not on account of his pain. Pain became extreme. Skin became moist and clammy and respirations rapid.

Patient was quite conscious and was given Morphine gr.1/6 with Atropine gr.1/200. He died almost immediately at 12.20 a.m.

Case No. 22.

Name. Mrs. Isabella Chambers, Admitted 7.7.27.

Address, 8 Stanhope Street, Townhead, Glasgow. Died. 16.7.27.

Age 57.

In November 1926 the patient had an attack of severe pain in the upper part of the abdomen. It commenced during the night, and was accompanied by nausea, but there was no vomiting although the patient felt as if that would give her relief. She was unable to remain in bed with it, but walked about during that night, and the doctor was called in the following day. She stayed in bed for a fortnight and recovered to some extent, but never felt very well. From March 1927 she complained of pain in the same region. It was very slight at first, and only came on after exertion or about half an hour after food and lasted a short time. It commenced in the epigastrium, radiated through to the back and down the left arm. Pain increased in intensity and came on at night if the patient lay down. It was spasmodic in character, sometimes lasted for about an hour. Occasionally there had been vomiting of a clear fluid. The last attack of vomiting was on Saturday 2nd July 1927. Her appetite was poor, and she was troubled very much with flatulence and constipation, the bowels not acting for two or three days.

Case No. 22 (Continued).

She was worn in appearance and looked older than her stated age. The skin was moist and the general appearance did not suggest myxoedema. The cardiac dullness was increased and there was marked pericardial friction over the lower half of the sternum. A soft systolic murmur was audible at the apex and a double murmur at the aortic area. There was some resistance and slight tenderness in the epigastrium. The right kidney was palpable.

Respirations were regular in rate and rhythm. The lungs were resonant to percussion and the respiratory murmur was vesicular.

For some time after admission she suffered from delusions but was very quiet and not restless.

At 11.30 p.m. on 16.7.27 she suddenly said she was choking. She fell back unconscious and died 10 minutes later.

Temperature - Normal throughout.

Blood Pressure -	130/50	8.7.27.
	125/55	11.7.27.
	132/55	15.7.27.

W.R. + ve.

Case No. 23.

D.Mc. Aged 50. Journalist.

8. 3. 29.

Five days before admission he felt tight sensation in the chest and was compelled to rest for a little. He was not short of breath. Two days later he had a similar attack. The day before admission he rested in bed, but was free from any symptoms.

At 1-50 p.m. on the 8th whilst engaged at typing, he felt a very constricting pain in the chest. Pain radiated across the mammary regions. He was not breathless but collapsed because of the intensity of the pain.

On admission patient was pale and distressed. Pain was intense and unaffected by respirations.

Pulse was slow - poor quality. There were numerous "Dropped" beats. Cardiac sounds were soft but pure.

Pain was greatly relieved by Amyl nitrite. administration, and pulse became regular. Urine 1020. Clear.

At 11 p.m. pain was less. Patient complained of nausea and thirst.

The following morning at 4 a.m. patient vomited clear fluid. Pain persisted but was much less.

Case No. 23 (Continued.)

11.3.29. Over lower half of sternum a regular high pi'ched squeak was heard very superficial and synchronous with the cardiac beat. This had disappeared on one and a half hours.

Patient's progress was uneventful. He went home on 14.4.29 feeling very well.

12.5.29. Patient reported today. He feels very well. There was a short squeaking V.S. murmur.

B.P. 105-65.

Patient reported on 5.8.29 - 14.11.29 and 5.6.30. He complained on each of these occasions of mild anginoid pain on exertion; otherwise he felt well.

B.P. remained low 100-110/70-60.

He died suddenly whilst at work, at the end of March 1931.

Temperature - 99 on 9th.

Blood pressure - 145/84 8/3.
115/70 9/3
100/65 20/3/

Leucocytes - 19600 per cmm. 9.3.29
14800 " 12.3.29
10400 " 16.3.29
8800 " 19.3.29
7200 " 24.3.29.

W.R. - ve.

Case No. 24.

J.D. Aged 52.

Admitted 7/4/30.
Dismissed 4/6/30.

Patient felt perfectly well until a fortnight before admission, when, while lying in bed in the morning, he felt sick and on getting out of bed, fell but did not hurt or strain himself. He felt no ill effects at the time but an hour later had a dull pain in the epigastrium accompanied by flatulence. Passing of flatus did not relieve the pain and he did not vomit. At the same time he had a feeling of pins and needles in the hands and arms and felt loss of power in the arms.
After a dose of salts he felt relieved and next day went back to work.
He remained well until 6th April when on rising in the morning he felt giddy and had a return of the pain at the lower end of sternum.
The pain increased in severity and was made worse by deep inspiration and on movement.
He returned to bed and immediately vomited. The vomit was brownish green and was sour.
Hot fomentations were applied to the abdomen and the pain shifted to the chest.

Case No. 24 (Contd).

He vomited several times on the day before admission but this did not relieve his pain which persisted all day.
He was sent to hospital as acute gastric perforation.
The patient was a strongly built man of 52. Colour and nutrition were good.
He was unable to lie on the left side because of pain.
The tongue was coated and moist. Appetite was poor.
The pulse was regular. Cardiac sounds were pure but distant.
There was tenderness at the lower end of the sternum.
The abdomen appeared to be normal.
Note two days after admission :-
Two days after admission there was sore tenderness in the upper epigastrium but no rigidity. The pain was felt chiefly over the left lower chest especially when lying on that side. There was some dullness at the base of the left lung with diminished breath sounds. The cardiac sounds were distant but pure.
The pulse was regular.
On that day friction was detected below the left nipple and its presence was noted during the following twelve days.
Patient's condition gave cause for much anxiety for the first seven days. On the 14th, however, he felt much better

Case No. 24 (Contd).

and free from pain.

His progress was steady until the 22nd when he developed thrombosis of the right femoral vein. Notwithstanding this set back his condition again improved and the apical first sound which previously had been almost inaudible was of fairly good quality on 6/5/30.

On 10/5/30 pleural friction was audible in right axilla. Patient however, felt well and was free from pain.

Improvement was uninterrupted from this date and he went home on 4/6/30, feeling well and without symptoms even when walking.

Temperature. Irregular pyrexia reaching 100°F was present until 14/5/30.

Blood Pressure 100/40 on admission after which it remained constantly about 110/70.

Leucocytes.

14/4/30	8200	per c.mm.
15/4/30	10800	"
16/4/30	9800	"
17/4/30	10200	"
22/4/30	9800	"
26/4/30	9500	"
5/5/30	10400	"

W.R. -ve.

Case No. 25.

Admitted 6/6/31. Aged 64.
Dismissed 7/31

Patient had always been in good health until sixteen days before admission when whilst out walking he experienced a sudden acute pain, like a violent blow in the region of the middle of the sternum. The pain remained for twenty four hours in the same situation and was of severe intensity, gradually changing in character to a duller pain which flitted about the chest affecting different areas - the nipples, the region of the apex of the heart, the foot of the sternum.
About this time he developed a short cough with a dry spit which was difficult to bring up. Coughing caused pain in the chest, in the region of the sternum, the pain being severe in nature and making him catch his breath.
He had no attacks of breathlessness.
His previous health had always been exceptionally good.

Note on 7.6.31. Patient is a nervous man and looks older than his years. He has a frequent irritating cough which caused substernal pain. There is a little cyanosis of lips and lobes of ears, but face is not so grey and worn looking as when he was on his feet. He is still somewhat breathless.

Case No. 25 (Contd.)

Pulse is regular of moderate force and fairly high tension. The radial pulses are equal. There is no tracheal tugging and no substernal dullness. Cardiac dullness is a little enlarged to right. Apex is palpable with difficulty. Sounds are poor. 2nd aortic is accentuated. A short systolic murmur is present at apex and aortic area and heard faintly in neck. Pericardial friction is heard at the foot of the sternum.

The lungs are clear to percussion. Much rhonchus and a little rale are heard all over the back.

Abdomen is distended, but there does not appear to be any ascites. There is general tenderness, most marked in epigastrium which is rigid. An indefinite mass (?colon) is palpable in left hypochondrium.

There are no urinary symptoms.

There was steady improvement in the patient's condition and it was noted on 21.6.31 that cardiac sounds were now pure and of better quality and blood pressure rising. He went home on /7/31 free of symptoms.

Temperature was normal throughout.

Leucocytes	8.6.31	8,300
	9.6.31	8,400
	10.6.31	11,600
	12.6.31	9,000.

Case No. 25 (Contd.)

Blood Pressure	138/95	6/6/31
	125/78	7/6/31
	110/74	8/6/31
	132/98	15/6/31
	128/74	18/6/31
	170/122	26/6/31

W.R. - ve.

Case No. 26.

Mrs. E.
11 Cornfield Street,
Gallowgate.

Admitted 17. 2. 28
Discharged 4. 5. 28.

Age 49 years.

About six years before she had an attack of influenza lasting three weeks after which she suffered from debility and breathlessness. Two years later she had a second attack lasting fourteen days after which she became subject to attacks of nocturnal dyspnoea - these came on after a few hours sleep when she would waken with noises in the ears and "sounds" in her throat and gasping for breath; after two hours it passed and allowed her to sleep. These attacks came at regular intervals of about a week, and continued for two years, since when they were very infrequent although she became steadily more breathless on exertion. Breathlessness was most severe on rising in the morning and tended to be less severe as the day wore on. About a year before admission to hospital swelling began in the feet and gradually extended up the legs. It was worse at night and cleared up with rest. About November 1927 the thighs were involved and the abdomen became swollen; a little later the hands were involved. Her appetite was always good although she had occasional nausea. Her eyesight was not affected.

Case No. 26 (Contd.).

She had never been subject to cough during her illness except for three days prior to admission when she developed some cough and mucous spit.

On admission patient was orthopnoeic and dyspnoeic. There was much oedema of legs, thighs, lumbo-sacral region, left arm and hand and to a lesser extent of the right arm. There was a short apical systolic murmur and the sounds otherwise were of poor quality. There was a cough with a mucoid spit. There was much moist rale at both bases.

Patient's condition improved gradually and progressively during her stay in hospital and on her dismissal 4. 5. 28 oedema was no longer present, and lungs were clear. Apical systolic murmur was still present.

Case No. 27.

Vincent Sirus. Aged 47.

Admitted 21.5.31

Dismissed 7.7.31.

This case is of especial interest as blood pressure did not fall to any extent until 48 hours after onset. Patient stated that he went to bed perfectly well on the night of the 20th and was awakened in the early morning by a severe pain in the hypogastrium. He rose and his bowels moved. Thereafter an intense burning sensation was experienced in the sternal region and down the left arm to the elbow so that he could not move the arm. There was also a feeling of tightness in the chest. He vomited and felt better. Patient was a burly sunburnt man but face looked pinched and anxious. Pulse was regular and bounding. Cardiac sounds were soft and of poor quality but pure. Chest in front was clear. Tongue was furred, but he did not feel sick.

Examination of abdomen was negative.

Substernal discomfort persisted for several days and there was steady fall in blood pressure.

On afternoon of 17.6.31 he complained of severe pain over lower part of sternum, he felt sick but did not vomit.

Progress was practically uninterrupted from that date and on 29.6.31 he felt very well, pulse was regular, cardiac sounds were of better quality but the apical first sound was still poor.

Case No. 27 (Contd.)

Temperature was elevated to 99°F on 21st, 22nd and 23rd and normal thereafter.

Leucocytes	13,500	22.5.31.
	12,800	23.5.31.
	14,400	25.5.31.
	13,000	26.5.31.
	9,500	28.5.31.
		29.5.31.
	8,600	30.5.31.
	8,200	31.5.31.
	6,400	1.6.31.

Blood Pressure See Chart.

W.R. - ve.

Case No. 28.

Mrs. M. Aged 64.

Admitted 1.7.28.

Dismissed. 3.10.28.

For nine weeks prior to admission on 1.7.28 patient had suffered from frequent attacks of Angina Pectoris. She was a thin but well coloured woman who looked older than her years. When free from attacks she was able to lie in the dorsal decubitus without discomfort. Pulse was regular but of high tension. Heart was enlarged downwards and to the left. There was much apical hyperaesthesia. A rough systolic murmur was audible at the apex.

On 4.7.28 patient was delirious with praecordial pain which lasted two hours. She was not sick.

Blood Pressure which had been 180/96 on admission fell to 100/60.

Temperature was slightly elevated 99°F.

Pulse Rate rose from 68 to 112 per minute.

Following this severe attack patient had numerous anginoid seizures of short duration.

On 3.10.28 she went home where she died suddenly six weeks later.

Temperature. Normal throughout except on 5.7.28 when it reached 99°F.

Blood Pressure.	1.7.28	180/96.
	5.7.28	100/60
	18.9.28	140/90.

W.R. - ve.

Case No. 29.

E.F. Aged 61

Admitted 16/6/28

Dismissed 20/7/28.

Was admitted on 16th June after having collapsed on the street. For the previous week he had felt stiff and sore.

On the morning of his admission he took a good breakfast, but an hour or so afterwards when out walking he became short of breath and felt discomfort in the epigastrium. He became very weak and perspired profusely. He rested and was found unconscious by the side of the road.

On admission on 16th June. Patient was a man of weather beaten appearance. He complained of burning pain across the chest. He vomited twice since admission.

Pulse 60 p.m. infrequent, irregular and feeble. Cardiac sounds were almost inaudible.

Temperature 96·8°F. Respirations 22 p.m.

Blood Pressure 80/50 mm. Hg.

On 17/6/29 patient was cyanosed. He was free from pain.

Pulse was 54 per minute and regular.

Cardiac dullness was small. Sounds were pure but distant.

There was moderate emphysema with much bronchial catarrh.

Urine S.G. 1030. Albumin. ++.

Friction was audible in left axilla on the 20th.

Case No. 29 (Contd.)

He had improved considerably by 23rd. There was liquid rale in left axilla. Friction was no longer present.

Patient's progress was steady and without incident until his dismissal.

He reported again on 3rd December. He had been free from symptoms and marked fit for work by Insurance Referee.

Temperature was elevated to 99°F from 17th to 21st June.

Blood Pressure	80/50	16/6/28.
	114/78	21/6/28
	130/80	24/6/28
	110/70	8/7/28
	140/80	19/7/28
Leucytes	12800	18/6/28

W.R. - ve.

Case No. 30.

A.G. Aged 43 years. Engineer.

Admitted - 2. 5. 30.
Died -12. 5. 30.

Patient had lived a fairly strenuous life. He indulged in all forms of athletics as a young man, particularly rugby football.

For the last twenty years he had been troubled with indigestion - heartburn after food.

For the last two or three years he had had much mental worry.

On November 1928 whilst on holiday, and when passing through Biggar, he was seized by intense praecordial pain - which radiated down the left arm. He felt as if he were going to die. With rest and Amyl Nitrite the attack soon passed off. From that date he had not felt quite well. He had been troubled with flatulence after food, dyspnoea on exertion, and occasional praecordial discomfort. He was compelled to change his lodgings from three stairs to one stair up. On 24th March 1930 he had a very severe attack of pain typically anginoid in type and persisting for several hours. Pain came on at 8 p.m. and by the following morning he felt comparatively well and free from symptoms. Since that date he had not felt quite well; he took life easily and attended strictly to his diet.

Most of his discomfort was epigastric. Flatulence,

hiccough and fullness in the epigastrium were very troublesome. Dyspnoea was always associated with this epigastric discomfort and this had so impressed itself on the patient that he was convinced that his illness was entirely due to gastric upset.

Patient had been feeling out of sorts on the evening of 30th April 1930. He had some digestive disturbance - fullness in epigastrium and flatulence after a small meal. At 2 a.m. on 1st May, he was awakened by intense praecordial and epigastric pain. The pain, he stated very definitely was more epigastric than thoracic, was both boring and vice-like in character (he felt that "it was squeezing the life out of him"). There was some pain down the left arm. He felt sick, retched and vomited bilious material. At 7 p.m. he was restless and orthopnoeic. His face and lips were grey. The extremities were cold. Tongue and toes were clubbed. Nails were cyanosed. Tongue was furred. Temperature 97°F. Breathing was shallow but regular. Periods of extreme restlessness alternated with short periods of rest and comparative freedom from symptoms. Pulse was soft and easily compressed. There was occasional extra-systole. Pulse rate at wrist was 96 per minute. Cardiac sounds were slapping and of very poor quality but otherwise pure. There was no friction. There was a fair amount of rhonchus all over the chest in front - the

Case No. 30 (Contd).

the back was not examined. Liver dullness was not increased.

Blood pressure at 1 p.m. was 98/75 mm. Hg.

He had developed troublesome cough which caused discomfort and exhaustion. During the rest of the day he had repeated attacks of vomiting which, considering his condition, were curiously effortless. The vomited material was clear in colour (he was sipping water continuously). Pulse at one part of the day was of the alternans type.

On at least three occasions he became collapsed and practically pulseless but recovery was rapid - pulse gradually returning and increasing in force.

Temperature 11 p.m. 97°F.

Patient slept fairly well, in short snatches, during the night and on 2nd April at 8 a.m. was feeling better. Pulse was regular but soft. He was free of discomfort but looked ill. He stated that he knew he could bring on his pain with forced effort.

The day following admission patient was still extremely ill. His facies was somewhat sunken and his complexion earthy and cyanosed. Hiccough was constant and he had frequent attacks of vomiting.

On 7.5.30 the right foot and toes were somewhat blue and painful and no pulsation could be felt in the dorsalis pedis.

He sank gradually and on 8.5.30 his colour was

Case No. 30 (Contd.)

distinctly yellow with some staining of the conjunctivae but no bile was present in the urine. His spit was blood stained.

On the night of 10.5.30 he had several generalised convulsive seizures. He was unconscious for a brief period after each seizure but was soon fully conscious and spoke sensibly.

He died suddenly at 12.45 a.m. on 12.5.30.

Temperature was subnormal throughout.

Blood Pressure -	74/40	2.5.30.	
	70/40	3.5.30.	
	74/52		
	80/50	8.5.30.	
	80/58	10.5.30.	
Leucocytes -	15,800	5.5.30	per c.mm.
	15,800	6.5.30	"
	24,600	8.5.30	"
	19,000	10.5.30	"

Case No. 31.

Alexander Kerr. Aged 41.

Patient was admitted to Dr. Middleton's Wards on 17.3.30.
For thirteen months he had suffered from epigastric pain and discomfort, - occurring half an hour after food and unrelieved by taking more food.
He gave a previous history of two attacks of acute rheumatism, first at the age of 15 and again 10 years later. About a year prior to admission he had an attack of haemoptysis which lasted a week.
He was undernourished, pale and looked anxious. Apart from slight tenderness to palpation in region of umbilicus examination of abdomen was negative.
Pulse was rapid but regular. Cardiac area was within normal limits. Rough presystolic and soft systolic murmurs were audible at apex. The chest was clear.
X-ray examination on 21.3.30 showed irregularity of pylorus and first part of duodenum. There was no evidence of malignant disease.
On 2.4.30 patient complained of peculiar pain over the right side of chest radiating up into the neck and into both shoulders and arms. He felt breathless and respiratory rate was increased to 60 and over. Temperature rose to 101°F. The following day patient felt better. There was now a little dullness over the right base and air entry was poor. A few moist rales were present at left base. He made a good recovery and was dismissed on 30.4.30.

Case No. 32.

Mrs. S. 58. Housewife.

Admitted 18. 11. 28.
Dismissed 30. 12. 28.

Patient was in good health until 8th inst., at 12-30 p.m. when she became giddy. This was followed by burning substernal pain which radiated to the right wrist. Pain lasted eight hours. It has not returned but breathlessness has increased. Patient vomited several times at the onset.

Patient is undersized and somewhat orthopnoeic. Pulse is regular. The apex beat is not palpable. Cardiac sounds are poor in quality. There is a tendency to "triple rhythm" over the right ventricle. There is a little moist rale at bases. The abdomen is normal. Urine S.C. 1020. Clear.

Patient made an uneventful recovery. On 7.12.28 a short systolic murmur was detected at apex.

She went home at her own request on 30.12.28. On that date she felt perfectly well; pulse was regular and heart sounds pure.

Temperature. There was slight pyrexia 99°F for one day (20.11.29)

Blood Pressure.	18. 11. 29	120/70.
	20. 11. 29	100/60.
	8. 12. 29	110/60.
	30. 12. 29	120/70
Leucocytes Count.	18. 11. 29	11600 per c.mm.

W.R. + ve.

Case No. 33.

Mrs E.C. Aged 53.

Admitted. 17.4.29.
Dismissed. 10.6.29.

Patient had severe attack of pain in the back a fortnight before admission.

te on mission. A week ago she was awakened by intense pain at the lower end of the sternum. There was in addition a dull aching pain in the left flank.

There had been no sickness. The bowels were regular.

T. 100°F. P. 100. R.30.

Note on 18.4.29. Patient is comfortable. The tongue is coated and the throat congested. Mucosa are slightly cyanosed. Capilliary pulsation is visible. Pulse is regular and soft. V.S. and V.D. murmurs are present with greatest intensity at lower end of sternum.

O E Fundi normal.

Praecordial friction was detected on 20.4.29 and was still present on 25.4.29.

On 16.5.29 friction had disappeared but double aortic murmur persisted.

Patient was free of pain from admission. She gradually improved and was dismissed on 10.6.29. She was free of symptoms but signs of aortic involvement were still present.

Case No. 33 (Contd.)

Temperature. For first fourteen days there was irregular pyrexia.

Pulse Rate. Remained elevated throughout 120-100.

Blood Pressure.	18.4.29	100/50.
	25.4.29	175/95.
	16.5.29	185/130
	14.6.29	130/80.
Leucocytes Count.	25.6.29	14667 per C.mm.

W.R. -ve.

Case No. 34.

J.S. Aged 58 Porter.

Admitted 14/10/28.
Dismissed 23/11/28.

At 5 p.m. on 13/10/28 patient had a sudden attack of extreme breathlessness whilst going upstairs. He had no pain or sickness.

He was helped to bed and was able to sleep propped up.

On 14th he had a return of breathlessness and was admitted to hospital.

He was a somewhat weather beaten man and was a heavy drinker.

His face was grey and lips and extremities were cyanosed and cold.

Pulse was regular, rapid (130 per minute) and soft.

Heart sounds were pure but of very poor quality.

There was much moist rale at bases of both lungs.

Urine contained + + albumin. Granular and hyaline casts were seen on microscopic examination.

Patient improved rapidly after admission and urine was clear on the second day.

He went home at his own request on 23/11/28.

Temperature was normal throughout except on 15/10/28 when it was 99.7°F.

Blood Pressure	135/78	14/10/28
	105/62	21/10/28
	120/62	20/11/28

W.R. - ve.

Case No. 35.

W.G. Aged 73 years. Admitted - 30.9.27
Dismissed - 19.10.27.

Patient collapsed while out walking on 30.9.27. He was taken by ambulance to Glasgow Royal Infirmary. He was a thin, somewhat wasted little man. There were obvious signs of arterial degeneration. He was extremely breathless and cyanosed. Pulse was rapid, 120 per minute and soft but regular. Apex beat was neither visible nor palpable. Cardiac area was enlarged downwards and to the left. Sounds were soft and of poor quality. There was a soft systolic apical murmur. Lungs were clear. Examination of abdomen was negative. Urine contained abundant albumin.

Patient's general condition improved rapidly and urine was clear on fourth day after admission. His cyanosis persisted, however, and cardiac sounds remained of poor quality.

He went home on 19.10.27.

Fourteen days later - a month after his first attack - patient died suddenly.

There was no post mortem examination.

Temperature was subnormal on admission.

98.8 on 1.10.27. During the rest of his stay in hospital it was normal.

Blood pressure -	172/90	1.10.27.
	150/85	4.10.27.
	120/70	9.10.27.
	125/70	13.10.27.
	154/90	18.10.27.

W.R. - negative.

Case No. 36.

J. McC. Aged 58. Warehouseman.

Admitted 27/4/28.
Dismissed 17/7/28.

Patient complained of shortness of breath with occasional attacks of pain on exertion of about one year's duration.
On 25/4/28 he was awakened on three occasions by extreme pain and breathlessness. Pain radiated down both arms and hands.
Pain persisted and was unrelieved by local applications.
It was still present when he was admitted to hospital two days later.
Patient was well coloured with suggestion of cyanosis.
He was fairly comfortable and could lie down without distress.
There was no oedema. Pulse was regular and of high tension.
Heart was enlarged to the left. A systolic murmur was audible at apex and aortic areas. There was mild diffuse bronchitis with moist rale at left base.
Urine contained a haze of albumen.
Patient's progress was interrupted by attacks of pain on 18/5/28 and 11/6/28. There were of very short duration and he went home on 17.7.28 much improved.
Pulse rate on admission was 120 per minute but soon fell to 70 per minute.

Case No. 36 (Contd.)

Temperature was normal throughout.

Blood Pressure	230/130	27/4/28
	190/120	3/5/28
	160/118	2/6/28
	220/130	8/7/28
	235/130	16/7/28.

Case No. 37.

J.H. Aged 63. Tailor.

Admitted	12/5/28.	Readmitted	19/10/28.
Dismissed	30/6/28.	Dismissed	12/10/28.

Patient complained of pains in the praecordium radiating down the left arm; shortness of breath and giddiness of three years' duration. Patient was a well nourished man. He looked anxious. Colour was good. There was no cyanosis or oedema. Pulse was regular and soft., Cardiac dullness was much diminished. The note over the sternum was frankly tympanitic. Cardiac sounds distant and poor. Chest was clear. Examination of abdomen was negative. Urine was clear.

On 25/5/38 he had severe pain at level of fourth rib from nipple to sternum, this pain persisted for several hours. There was some hyperaesthesia over the upper part of the left pectoral region. Heart sounds were still distant but pure. Pulse rate was elevated to 90 per minute. He had several similar attacks during his stay in hospital but went home on 30/6/28 feeling better. Patient returned to hospital on 19/10/28. His condition was much as on previous admission. He remained in hospital for a month during which time he had frequent attacks of intense praecordial pain lasting from

Case No. 37 (Contd.).

minutes to hours. There was no improvement in his condition on dismissal.

Temperature was normal during the whole of his stay in hospital.

Blood Pressure	130/90	12/5/28.
	100/70	27/5/70.
	100/60	2/6/28.

Case No. 38.

James Robertson. Aged 63.

Admitted 3/5/31.
Dismissed. 6.7.31.

Patient had always enjoyed good health until a month before admission when, after working for a week, having been idle for nine months, he had a sudden choking sensation and had to stop work. He felt fairly fit two days later and resumed work but had to give up again owing to a similar attack. During the period from cessation of work until admission he had several attacks of breathlessness, and he noticed that these were apt to come on when his head was low, as in stooping. The slightest exertion, even walking a short distance, caused breathlessness and when such an attack occurred he had to stand still for a few minutes until it passed off.

Patient was orthopnoeic, cyanosed and looked worn and tired. There was slight oedema of the ankles and moderate oedema of lumbo-sacral region. Varicosity of veins of legs was marked with much pigmentation of both especially the right. An ulcer about the size of a penny was present on antria and outer aspect of right leg. Glands were palpable in both groins. The throat was congested, teeth very septic, tongue dirty and thickly furred and breath offensive. Pulse was completely

Case No. 38 (Contd).

irregular and of fairly high tension. Vessels were tortuous and easily palpated. Heart was enlarged to left and sounds were of very poor quality. No murmurs were heard, but shuffling to and fro friction was audible. There was much bronchitis, with loud rhonchi in front and rales at bases. Percussion note over right base was flat, breath sound was diminished V.R.+ and rales ++.

Liver was easily palpated but spleen was not enlarged. There was no abdominal tenderness.

Patient made steady progress until 26/5/1931, pulse became less irregular and cardiac sounds improved in quality. Apical systolic murmur was occasionally noted.

On that date he was not so well - oedema increased and liver became tender and enlarged.

Gradual improvement again took place and on 16/6/1931 oedema was almost absent and liver was no longer palpable. This improvement was maintained and he was dismissed at his own request on 6.7.31.

Temperature remained normal throughout.

Blood Pressure	3/5/31	140/110
	25/5/31	110/75
	11/6/31	140/110.
Leucocytes	5/5/31	7400
W.R.	- ve.	

Mr Allan. Age 58.

Was quite well until 17th July 1931. He became troubled with shortness of breath and praecordial discomfort on exertion or walking uphill. On examination on 1st August 1931 he complained of praecordial discomfort and shortness of breath on exertion. Heart was enlarged to the left. 1st sound somewhat muffled. Blood pressure 178/100. The electrocardiographic tracing was taken on 16.8.31

Notes from Dr David Smith:-

On 15.9.31 he had severe praecordial pain lasting six hours. He was sick and vomited.

On 16.8.31 blood pressure 140/90. Slight friction was heard.

On 17.9.31 praecordial friction was marked.

Leucocytosis 15,000.

On 27.9.31 patient felt better. Heart sounds had improved in quality. No friction. Blood pressure 150/95.

Case No. 40.

Mr Gallagher. Age 44.

Was examined on 4th April 1931. He gave a history of sudden attack of praecordial and epigastric pain, sickness and vomiting three weeks before whilst out walking. He was taken to the surgical department of Victoria Infirmary and detained for two days. Since that date suffered from pain and catching of breath; dyspnoea on exertion.

Was seen on 4th April pulse was regular and soft; blood pressure 120/86; heart sounds soft and rather distant. He had definite dyspnoea and tightness in his chest when walking upstairs.

Re-examined about a month later after having rested. Patient felt better and had less discomfort on exertion. Physical signs were unaltered.

Case No. 41.

R.R. Aged 45 years.

Admitted 23/11/30.

Discharged 23/13/30.

During July 1929 patient felt disinclined for work - he was depressed. He ceased work on 6th August. Three days after about 10-30 a.m. he had an attack of severe praecordial pain, palpitation and buzzing in his ears. He walked about to obtain relief. The acute attack lasted about five minutes but the pain persisted in the chest for 2-3 weeks. Pain did not radiate. There was no vomiting. He was off work for eight weeks.

In December he was compelled to cease work because of nervousness. At the time pain was not excessive. He rested until the middle of March, when he again resumed work. Four weeks later when leaving work he had a nervous seizure - he felt "something coming up from his feet" which made him powerless. He had not worked since and had not been free of symptoms. There was always discomfort and 'irritability' in his chest. He occasionally felt nauseated.

Patient was a fairly robust looking man. He lay comfortably in bed and complained only of this "cold in his chest".

He was nervous and appeared to be keen to discuss his symptoms.

Case No. 41 (Contd.).

Teeth were artificial - tongue was furred. Pulse was soft, somewhat collapsing in type - regular in rate and rhythm. There were no abnormal pulsations in the chest, neck or abdomen. Chest was well formed. Apex beat was neither visible nor palpable. There was no hyperaesthesia of chest wall. Cardiac sounds were soft, of poor muscular quality, but pure. There was no manubrial dullness.

Abdominal examination was negative.

During his stay in hospital, patient had frequent attacks of praecordial discomfort.

Patient reported from the home on 29/13/30. He complained of persisting pain in praecordium. Physical examination was negative. He was dismissed on 29/12/30 and has reported at frequent intervals since.

Praecordial discomfort and dyspnoea are still troublesome.

Temperature was subnormal throughout.

Blood Pressure remained constant about 128/75.

W.R. - ve.

Case No. 42.

D. Forbes. Age 47.

Was sent to the Medical Dispensary of the Glasgow Royal Infirmary on 19.3.30 with a provisional diagnosis of syphilitic aortitis.

His doctor stated that for the past month patient had been becoming progressively short of breath. Shortness of breath occurred on exertion, but in addition he had occasional attacks whilst in bed. There was no history of pain. He further stated that two days before sending him to hospital he had heard a rough double murmur over the aortic area.

Patient was stout, his cheeks showed many injected venules. He was short of breath. Pulse was regular and soft. Blood pressure 110/75. Cardiac area was enlarged. Apex beat ½" outside nipple line and 6th interspace. Heart sounds were very soft and distant. No murmur was detected at any area. An electrocardiographic tracing was taken at this date.

X-ray examination of the chest showed enlargement of the heart. Wassermann Reaction negative. Urine clear.

Patient reported at dispensary almost every week during the succeeding nine months.

Apart from frequent recurrences of intense dyspnoea there was no appreciable change in his physical condition during this period.

The electrocardiographic tracing remained unchanged.

Case No. 43.

Mrs. C. Aged 51.

Admitted - 8.5.27.
Died - 8.7.31.

Two months before admission patient had an attack of severe praecordial pain which radiated down the left arm. Following this attack she was breathless on exertion and palpitation was troublesome. Patient was a thin nervous woman with rather anxious expression.
She complained of constant praecordial discomfort.
There was oedema of the ankles.
Pulse was regular but of poor volume.
Cardiac sounds were of poor quality. A rough systolic murmur was audible all over the base.
Patient's condition gradually improved with rest and she was dismissed on 13.8.27 feeling much better. Systolic murmur persisted at the base.

Temperature. 99°F on admission - subnormal.

Blood pressure.

8. 5. 27	110/50.
25. 6. 27	95/50.
8. 7. 27	120/70.

Leucocytes Count 7000 per c.mm.

W.R. - ve.

Patient reported again a year later; she felt well but unable for hard work. Systolic murmur was still present.
On 1.2.30 she complained of frequent attacks of palpitation.

Pulse was rapid and irregular in force and rhythm (Auricular Fibrillation).

She was readmitted on 31.5.31 orthopnoeic and oedematous. Auricular Fibrillation was still present. Cardiac dullness was increased downwards and to the left, sounds were of poor quality and rough systolic murmur was still audible.

She gradually sank and died on 8.7.31.

Case No. 44.

F.H. Aged 52.

Had been sent to dispensary of Glasgow Royal Infirmary suffering from nervous debility. He complained of pain in his chest; shortness of breath on exertion.

Seen on 5.6.30; he gave a history that two months previously he had an attack of Influenza, sudden onset of pain in left side of his chest, shortness of breath and general weakness. He felt at that time that he was going to die. He was confined to bed for two weeks. From that date had suffered from dyspnoea and praecordial pain, the pain radiating at times down his left arm. Blood pressure 115/90.

Rest was advised and patient reported again four months later, feeling better but still complaining of tightness in his chest on exertion.

Case No. 45.

P.T. Aged 54.

Admitted 19/6/30.
Dismissed 24/6/30.

History. In 1917 he was buried for 10 minutes by a shell exploding and was crushed by the earth. Since then he had been a little breathless.
About six months before admission he took a stabbing pain over his left nipple which made him stop his work. The pain worked over till he felt it under his breastbone. The pain was not extremely severe. It lasted about five minutes and he eased off his work till it was over. This pain came back now and again but in the last three months it came on after any heavy exercise and passed down into his wrist of left arm to elbow leaving it numb. For months he had been breathless all the time - and when the pain came on the breathlessness was extreme.
On 19/6/30 patient had attack of severe pain in praecordium radiating to left arm and left side of chest. With onset on seizure the left hand became cyanosed and remained so till the pain passed off - about 20 minutes.
On 20/6/30 the patient's face was grey, his expression somewhat anxious. There had been no vomiting. The pupils were

Case No. 45 (Contd.)

equal and small but responded normally. Radials and brachials were thickened and the pulses were probably equal. The carotids were equal. The radial pulse was full, slightly shotty and regular.

There was slight capillary pulsation in nails and lips.

Apex beat was not forcible. The sounds were of poor quality. A systolic murmur was heard at the apex, and V.S. and V.D. soft at the lower end of the sternum. Air was entering both lungs equally.

On 24/6/30 after having burning pain under sternum all day - patiend died suddenly.

Temperature was normal throughout.

Blood Pressure	135/50	19/6/30
	100/30	20/6/30
Leucocytes	14800	20/6/30.

W.R. + ve.

Case No. 46.

F.L. Aged 43.

Took ill in January 1931 with severe pain in the left side of his chest.

Temperature was slightly elevated, 99°F. He was short of breath. He was confined to bed for 3-4 days and felt short of breath on exertion.

When examined three weeks later his colour was poor, he was short of breath on slight exertion and complained of a catching feeling in his chest.

Pulse was regular.

Heart sounds were of poor quality.

Blood pressure 120/95.

Patient improved considerably after prolonged rest in bed, but is still subject to praecordial discomfort on exertion.

Case No. 47.

P.H. Aged 73.

Was sent from Ophthalmic Institute for examination on 27.6.31.

Doctor stated that five months previously whilst convalescing from a minor eye operation he had a sudden attack of severe nocturnal dyspnoea. He was grey and collapsed, and it was thought that he would die that night. He remained orthopnoeic for five to six days and recovery was gradual. A further operation was contemplated.

Patient was a done old man. He made light of his troubles but was obviously short of breath on very slight exertion. His arteries were visible and palpable. Heart was enlarged downwards and outwards. First sound at mitral area was slapping in quality. There was a rough systolic murmur at the aortic area. Blood pressure 175/128.

J.C. Age. 62.

Was examined on 23.12.30.

He had been unfit for work for three months following pleurodynia. He looked weather beaten and complained of shortness of breath on the slightest exertion. He stated that his illness began three months before with a sudden attack of very acute pain in his left side while returning home from his work in the mines. He was sick and vomited and felt very short of breath. He went home to bed and early next morning was compelled to send for his doctor because of the persistence of the pain. Pleurisy was diagnosed. Pain lasted for two days. He was kept in bed for three weeks, but although up and going about he had not felt well. Pulse was irregular, the irregularity being due to frequent extra-systole. Arteries were hard and tortuous. Cardiac area was enlarged, apex beat 6th interspace. Blood pressure 142/100.

Case No. 49.

M. McC. Age 46. Farmer.

In the middle of January whilst at work in the morning he felt a tightness in his chest. No shortness of breath. Later in the day pain spread across his shoulder and down his right arm.

There was no epigastric pain.

He felt sick and vomited, and had to be assisted home.

He was examined by doctor. He was grey, collapsed, was short of breath and complained of pain and discomfort in the left side of his chest.

Pain persisted for 2½ days in spite of administration of morphia. No praecordial friction was heard.

Systolic blood pressure 135.

He was examined by me at the end of April. He complained of slight flatulent dyspepsia, but had no praecordial discomfort on exertion.

Pulse was regular and of moderate volume.

Heart sounds were pure.

Chest was clear.

There was no praecordial hyperaesthesia.

Blood pressure 132/86.

Case No. 50.

Mr A. Aged 61.

13. 7. 31.

For the past five years felt short of breath and has had a feeling of weight in his chest on exertion.

He was examined by a cardiologist at Easter 1931 and no abnormality was detected. Whilst on holiday two or three weeks later he had a sudden nocturnal attack of dyspnoea and vomiting. There was no pain. Theintense dyspnoea persisted for 2½ hours and he felt that he was going to die. Two days later he had an attack of pain at the right side of his chest made worse by deep breathing. His spit was haemorrhagic. He was in bed for a month.

Since that date his shortness of breath has been less and he no longer feels the "catching in his chest". He is now troubled with flatulent dyspepsia which gives rise to palpitation and he sleeps badly.

He is a plethoric man with many injected venules in his face. Pulse is regular, good volume, fairly high tension. Blood pressure 175/95.

He states"that for some years his blood pressure has been over 200".

Urine is clear. Heart sounds are soft. There is a short systolic murmur at the aortic area.

Patient died suddenly whilst at work (January 1932).

Case No. 51.

F.M. Aged 62.

Patient was examined on 7.9.30. He was considered to be suffering from debility following pleurisy.

Six weeks before he was awakened by sudden attack of pain in the left side of his chest. He felt very short of breath and it was thought that he would die that night. Next morning he was sick and sickness persisted most of the day. Pain was apparently relieved by brandy three hours after onset, but it tended to recur for brief intervals during the next day.

He was confined to bed for 10 days but was short of breath on the slightest exertion.

7.9.30. On examination he was dyspnoeic and looked ill. Extremities were cold and cyanosed. Pulse was regular and feeble. Cardiac area was enlarged, apex 6th interspace. Heart sounds were of very poor quality.

Blood pressure 154/108.

Electrocardiogram was taken on 9.9.30.

Case No. 52.

Mrs W. Aged. 58.

Was examined on 8.9.29.

She gave a history of having had two attacks of severe praecordial pain coming on at night and persisting till the following day. The pain on both occasions radiated down the left arm. She was dyspnoeic. The first attack occurred whilst abroad about a year before. The second attack occurred at home nine months later. She looked well and stated that she was well, except for a catching in her throat and chest on walking up a hill or going upstairs. Pulse was regular; blood pressure 130/95; heart sounds were soft but pure. There were no obvious signs of arterial degeneration

Case No. 53.

T.F. Aged 54.

Suffered from angina of effort since November 1930. He was examined by Dr David Smith at various intervals.

Date.	Blood Pressure.	Pulse Rate.	Urine.
8.12.30.	170/100	72	V.F.T.
15.12.30	160/100	78	V.F.T.
28. 2.31	200/105	68	V.F.T.
9. 3.31	160/100	80	Cloud.
	From this last date i.e. 9.3.31 Angina became more marked and dyspnoea developed.		
23. 3.31	140/100	90	Cloud.
15. 4.31	200/105	78	V.F.T.
	Patient was fairly well from 15.4.31 until 6.5.31 when dyspnoea and angina of effort again became very troublesome.		
6. 5.31	150/100	90	Cloud.
15. 6.31	140/100	92	Cloud.

Patient was admitted to Glasgow Royal Infirmary for observation and treatment on 22.6.31. On day of admission he had severe attack of status anginosus and died that day.

Case No. 54.

W.K. Aged 51.

Admitted	29/9/28	Readmitted	28/3/29
Dismissed	19/10/28	Dismissed	20/4/29.

Whilst walking slowly to work he had an attack of severe pain across the chest up to the left shoulder, and down the left arm. He felt sick during the attack and gaseous eructation brought temporary relief. He collapsed and was taken to hospital. On admission he was comfortable and free from pain, pulse was regular and had suggestion of bisferiens. Cardiac sounds were poor but pure. Chest was clear. Examination of abdomen was negative. During his stay in hospital patient was without symptoms and he went home on 19/10/28 feeling weak but much improved.

Temperature varied from subnormal to normal.

Blood Pressure	125/90	29/9/28.
	100/60	8/10/28.
	115/78	16/10/28.

W.R. - ve.

Patient remained well for one month, when he began to have heavy sensation in epigastrium after food.

On 21/3/29 he had a severe attack of pain in left Xiphoid, lasting one hour shooting to the angle of left scapula and

Case No. 54 (Contd.).

over the praecordium.

He had similar attacks on the following two days, the first lasting two hours, and the second five hours. During the attacks he felt sick and vomited.

He was readmitted on 28/3/29. Patient looked pinched and anxious. Although pain had been absent for four days vomiting was still present. Tongue was dirty and furred. There was hyperaesthesia of both subcostal areas and epigastrium. Pulse was regular but soft and easily compressed. Cardiac sounds were distant and of poor quality but pure. Chest was clear.

Patient's condition again improved during his stay in hospital and he was dismissed on 20/4/29, feeling well.

Temperature was normal throughout

Blood Pressure	115/58	28/3/29
	125/65	125/65

At a later date patient had another attack of pain. He was seen by two medical practitioners who diagnosed typical gall bladder colic. Laparotomy was performed and was negative.

Case No. 55.

Hugh Alexander - Age 33.

Admitted 1. 2. 30.

On January 22nd 1930 patient was troubled with flatulence and pain in the epigastrium after food. He continued at work until 29th January but was compelled to go to bed because of shortness of breath. Patient was admitted to hospital on 1.2.30.

Note on admission.

Patient is a pale faced man. He states that he has always been pale but has never been ill, and that he feels in his usual health except perhaps a little weaker. Teeth are unsatisfactory. Tongue is clean and much corrugated. There is a large polypus with purulent discharge in the right nostril and pus and crusting in the middle meatus of the left. Pulse is infrequent and regular, full and well sustained. The auricular pulsations in the jugular veins are at least double the rate of the regular pulse. The apex beat is slight. A systolic murmur is audible at all areas, more marked at the mitral and toward the base; a faint thud sound is audible.

From 8.2.30 until 15.3.30 patient had many attacks of transient loss of consciousness; in the milder

Case No. 55 (Continued.)

of these attacks patient became momentarily unconscious, whilst in the more severe there was twitching of the arms and body. During one of the attacks ventricular diastolic pause was 10.8 secs.

Patient went home on 7.5.30. He felt well and seemed to have benefited greatly from andrenaline and ephedrine. Full Heart Block persisted.

W.B.C. 11.2.30 19800

14.2.30 18600

B.P. rose from 130/60 on admission to 160/60 on dismissal. There were occasional attacks of mild pyrexia.

Patient reported on 4.10.30. He felt well but was troubled with indigestion and a little morning vertigo. Pulse was regular 38 per minute. B.P. 160/80.

In November 1930 patient died at home from an attack of acute lobar pneumonia.

Temperature - Irregular pyrexia.

1.2.30	130/60.
21.2.30	160/85
21.3.30	190/90.
12.4.30	170/85
2.5.30	170/70.

Case No. 56.

John Hannah. Aged 45.

Patient had been troubled with flatulent dydpepsia for some time. He had a sudden attack of epigastric pain, sickness, vomiting and collapse. He was confined to bed - a diagnosis of gastric ulcer was made by his medical adviser.
Two weeks later he was sent to the X-ray Department of the Royal Infirmary where it was suspected that the primary lesion was cardiac. As pain was now praecordial and he had definite dyspnoea on exertion he was referred by Dr. McLean for electro-cardiographic tracing. This showed abnormality associated with cardiac inferction.

Case No. 57.

R.W. Aged 47. Engineer.

Patient consulted me because of stomach complaints.
He wished some medicine to relieve persistent epigastric pain and vomiting. He looked pale and ill.
He gave a history of having been in perfect health until two days before when, sitting at tea just after his return from work, he was seized with violent substernal pain. Pain radiated downwards to epigastrium and upwards to the neck and down both arms. He vomited, and stated that he felt that if he did not vomit he would die.
Vomiting relieved the acute pain - which lasted 1 hour, 20 minutes, but there was persistent dull epigastric pain and food caused sickness almost immediately. He was short of breath on exercise.
Patient looked dyspnoeic even when at rest but denied any discomfort.
Pulse 42 p.m. and regular. The irregularity appeared to be of an irregular extra systole type. Heart sounds were of poor quality but pure. Arteries felt hard and arterio-sclerotic.
Blood pressure 105/65. Temp. 98°F. Resp. 26.
Chest was clear. Urine contained no albumin.
The next day, patient felt well. Pulse was regular 54 p.m., otherwise physical signs were as on previous day.

Case. No. 57 (contd).

He would not admit that he was ill and refused any suggestions with regard to treatment.

On the third day after consultation, 5 days after acute onset, he went out for a walk. Two days later, the seventh day of illness, he died suddenly whilst sitting in his chair.

Case No. 58.

M.McG. Aged 56.

9.7.31.

Nine weeks before had attack of severe pain in the left side of the chest when out walking. He felt as if life was being crushed out. He was very short of breath; and had to be assisted home. Pain did not radiate from the chest. Acute pain persisted for one week. From that date patient had been short of breath on slightest exertion and had recurrent attacks of praecordial pain. His general condition was fairly good. He was slightly puffy under the eyes. Extremities were cold and cyanosed; pulse was regular and soft. Blood pressure 122/82. Heart was enlarged to the left; apex beat was palpable 6th interspace. Heart sounds were soft and distant.

Case No. 59.

Mrs. N.

Was seen by Dr. Harrington in December 1928.
She suffered from marked hyperpiesis and myxoedema.
On 4th February 1931 she had a sudden attack of right sided hemiplegia.
Power had almost completely returned by the 16th.
On the first of March she had an attack of severe praecordial pain, felt sick, retched and vomited.
She looked collapsed, pulse was regular and soft.
Heart sounds were distant and of poor quality.
Blood pressure 180/135.
Pain was relieved with morphia.
Patient had frequent recurrences of pain on 3rd, 5th, 6th and 11th, pain lasting from ½ hour to several hours but always relieved by morphia. Blood pressure fell on the 4th to 140/115.
On the 14th blood pressure 175/130. On the 25th blood pressure 185/140.
Her progress following last attack of pain on the 11th was uneventful.
There was a leucocytosis varying from 8,000 to 14,000 during the whole course and on the 30th March W.B.C. was 9,000.

F.M. Aged 60.

Was troubled with flatulent dyspepsia for the past two years. He had no cardiac symptoms.

On the afternoon of 30th June he had an attack of acute pain in his chest passing down the left arm. Pain lasted for a minute or two.

He felt miserable all day and at night had a severe attack of pain in his chest. Pain persisted during the night. He felt as if he were being crushed by a vice. Pain did not radiate to the arms. He was not sick.

When seen on 1st July the patient was grey in colour, lips cyanosed, extremities cold. He complained of pain in his chest. Pulse was regular, soft, feeble. Blood Pressure 150/115. He felt better after morphia and three hours later was free from pain. Blood Pressure 145/110.

Patient remained free of pain but felt very weak. He was inclined to be restless but Blood Pressure fell steadily during the next three days - 120/90 - 100/85 - 90/70.

He died suddenly in his sleep at 2 a.m. on 6th July.

Leucocytes:-

2nd July	8,500.
3rd July	11,000.
4th July	9,500.
5th July	13,000.

Pericardial friction was present throughout.

Temperature from first to third day varied from subnormal to 99.5.

Case No. 61.

T.B. Age 33.

Seen at out-patients' department, Glasgow Royal Infirmary on 21.6.30. Complained of pain in the epigastrium 1½ hours after food. Pain relieved by food.
Flatulence and sour mouthfuls were troublesome.
He was sent to the X-ray Department for Barium Meal.
Shortly after the X-ray examination had been made he collapsed. He was soon pulseless and after a few sighing respirations he died. He was seen for the first time by A.W.H. just when he was on the point of dying.
A Fiscal Post Mortem was carried out and it was reported that deceased had advanced degeneration of the ascending part of the aorta with stenosis of the entrance of the coronary arteries. No lesion of stomach, duodenum or intestines was revealed at the post mortem.

Case No. 62.

Wm. Mcl. Aged 42. Carting Contractor.

For 2-3 years patient had suffered from pain in the lower abdomen on exertion and after food.

On Tuesday, 5th November, pain was very severe after his evening meal and he was slightly sick. Pain persisted and on 6th November became very acute, patient falling to the ground. He was violently sick - vomit contained bile. Palpation showed tenderness over the umbilical area and the right side.

Laparotomy - No apparent abnormality.

Pulse 72. Temp. 96. Resp. 22

B.P. 100/60.

Urine clear.

W.B.C. 9 5600.

Patient was a weather beaten man, looking much older than his years. There was considerable degeneration of the radials and brachials and to a lesser extent of the legs. The pulse was regular. The apical first sound was somewhat slurred at the commencement.

Patient reported again two months later. He felt well. There was little change in physical signs.

Case No. 62 (Continued).

Temperature - Subnormal.

Blood Pressure - 23.11.29 100/60.

26.11.29 100/60.

2.12.29 115/80.

W.R. - ve.

Case No. 63.

Mrs. M.N. Age 60.

26. 2. 39.

October 1927 Patient had severe pain in epigastrium and vomited a great deal. She became slightly jaundiced and "gall stones" were diagnosed. Jaundice disappeared soon but patient remained short of breath. In January her feet and legs became swollen.

Oedema has been progressive and within the last four weeks she has become orthopnoeic.

On Admission. Patient is stout. She is orthopnoeic and cyanosed. There is gross oedema of legs and lumbo-sacral region.

Urine - Trace of albumin.

Blood Pressure 120/90 155/95 140/80 160/90.

Much moist rales in both lungs.

Patient improved a little during her residence.

Pulse became irregular on 7.4.29 and on 9.4.29 (Extra systole ?).

Patient went home on 13.4.29 apparently slightly improved.

Two days later she collapsed suddenly and died soon afterwards.

Case No. 64.

J.P. Aged 54.

Was seen by Dr David Smith at Glasgow Royal Infirmary on 16.6.31. Seven days before he was awakened from sleep by intense praecordial pain. This pain lasted for some time. He was sick and vomited, and felt short of breath. He was allowed up on the fourth day of illness and since then had been short of breath on slight exertion.

He gave a previous history of transient hemiplegia six years before. Heart sounds were soft and of very poor quality. There were no adventitious sounds.

His medical practitioner was advised of diagnosis and treatment.

No further particulars could be obtained of this case.

Case No. 65.

J.W. Admitted 3. 7. 27. Aged 66.

Complained of extreme breathlessness of two days' duration. Four months previously his left leg was amputated for diabetic gangrene. During the operation he had sudden collapse and only recovered after massage to the heart. He remained fairly well after the operation and was sent to a Convalescent Home in June, that is three months after the operation.

On the evening of 1. 7. 27 he had a sudden attack of breathlessness. The following afternoon he had another.

When admitted to the Royal Infirmary he was dyspnoeic, complexion cyanosed, pulse was soft. Heart sounds poor.

Rales over both lungs behind.

Temperature on admission 99°F.

Blood Pressure 170/90.

Urine; Specific gravity 1020. No albumin.

Patient was free of symptoms the day following admission and had no further attacks of dyspnoea.

He went home on 14.7.27.

TEMPERATURE - 4th - 99° - normal.

BLOOD PRESSURE -

4. 7. 27. 170/90.

10. 7. 27. 170/80.

Case No. 66.

A.S. Admitted 25.8.30.
Dismissed. 20.9.30. Aged 52.

About 8 o'clock on the morning of admission patient had frontal headache, was sick and vomited bright yellow material. He stated that he had two separate cardiac attacks the previous day. The first came on about 11.30 a.m. - was ushered in by palpitation which was followed by nausea, sickness and vomiting. He felt faint, and lost consciousness for a short time. There was feeling of constriction of chest but no actual pain. By 2-30 p.m. he felt well but on attempting to do some work he had a second seizure about 3 p.m. similar in type to the first but more severe. He was admitted to the Wards at 6.30 p.m. and soon after admission had a fainting attack and was unconscious for a very short period. (Rate of Pulse was not noted). He was a well nourished rather weather beaten man who lay comfortably in bed free of symptoms apart from slight feeling of sickness. Tongue was furred. There was slight discomfort to palpation of epigastrium. Pulse was regular, of moderate volume, and easily compressible. There was slight pulsation in supra-sternal notch and carotid pulsation, particularly on the right side was visible. Apex beat was visible at nipple line in 5th interspace.

Case No. 66 (Contd.)

Cardiac sounds were soft but pure.

Patient's progress was uninterrupted and he was dismissed on 20/9/30 feeling well.

Temperature was subnormal throughout.

Blood Pressure	135/82	25/8/30.
	118/82	28/8/30.
	130/80	25/9/30.

Wassermann Reaction - Negative.

Case No. 67 (Contd.)

Temperature.	(2-26) - 2-28	97.2°F.
	(27-28) - 2-28	99°F.
	(4,5 and 6) - 3-28	normal.
Blood Pressure.	2.2.28	140/85.
	18.3.28	85/58.
	30.3.28	105/80.
	13.4.28	65/45.

W.R. - ve.

Case No. 67.

Mrs R. Aged 62.

Admitted 2.2.28.
Dismissed. 16.4.28.

For a year previous to admission she was short of breath on exertion. In October 1927 when out walking she was seized with violent praecordial pain - pain lasted for several minutes and she had to be assisted home. There was no sickness or vomiting. Two days prior to admission she had a second attack of praecordial pain which was relieved by injection of morphia. Although pain had subsided breathlessness persisted.

On admission, she was orthopnoeic, looked anxious and complained of "terrible weakness and shortness of breath". Facies was grey and extremities were cold. Pulse was irregular - (the irregularity being due to frequent extra systoles) and of poor volume.

Heart was enlarged to the left. Cardiac sounds were distant, poor quality, but pure. There was moist rale at the right base.

Patient had two attacks of severe pain during her stay in hospital-

27.2.28 - lasting 6 hours,
5. 3.28 - lasting 12 hours.

Pain was relieved on each occasion by injection of morphia.

No pericardial friction was detected.

She went home on 16.4.28 feeling better but died suddenly three weeks later.

Case No. 68.

Robert Doig. Aged 72 years.

Admitted 11.7.29.
Discharged 27.10.29.

Readmitted 27.2.31.
Died 21.4.31.

Note from Doctor.

This is an interesting case of complete heart block with Stokes Adams fits. His history is as follows :-

A year ago he was troubled with praecordial pain following an attack of bronchitis. His cardiac sounds were almost inaudible at that time, but his pulse rate was not affected. Some time later he stated he took a giddy turn while outside, and he had been troubled with dizziness and shortness of breath since. About three months ago I saw him again and found his pulse rate was 24. He was kept in bed and a day or two later typical Stokes Adams fits made their appearance. He was kept in bed for three months, the attacks becoming gradually less frequent. He was seen by Dr. John Cowan who took a polygraph tracing, confirming the diagnosis of complete heart block. Lately he has been allowed out of bed, his pulse rate varying from 18 to 30. Early this morning he took suddenly ill again with severe epigastric pain and vomiting and the fits have reappeared again. He seems to have developed some acute abdominal condition, with rigidity of the

Case No. 68 (Contd).

upper abdomen and very great tenderness, extending along to his gall bladder. Sickness has persisted and he is again very ill. He had a quarter grain of Morphine Sulphate at 7-30 a.m.

The patient was an old but powerfully built man. He looked very ill. He was extremely orthopnoeic. His breathing was typically Cheyne-Stokes.

After admission he had many typical Stokes Adams seizures and complete physical examination was impossible.

On 14. 7. 29 patient had an attack of sudden severe pain in right side of chest, radiating to the right shoulder at 2 a.m. It was accompanied by a feeling of tightness and breathlessness. It was very tense at 5 a.m. and by 7 a.m. had passed off. The heart sounds were extremely distant and feeble. No friction was audible over the heart or on right side of chest in front. Blood pressure 180/60 the following day.

The sputum contained blood.

The patient remained in a fairly comfortable condition considering his serious state on admission. He made steady progress and had only two Stokes Adams' seizures during the rest of his stay in hospital - (27. 8. 29 and 29. 8. 29). The second attack was peculiar. Patient looked pale that

Case No. 68 (Contd).

morning but stated that he was feeling better. Whilst talking, he suddenly became very pallid, respirations which had been quiet and effortless were now forced and sighing. The eyes rolled and he was unconscious for about a minute. Pulse rate at the wrist was 120 and apparently regular. Face became engorged and patient soon recovered, except for spasmodic twitchings affecting the hands and arms. Pulse rate was now 30 per minute. The patient went home on 26. 10. 29 improved but far from well. His feet tended to swell when he was up and he could only walk a very short distance with support from a nurse.

Since dismissal patient has been fairly well but on occasions his feet became swollen when he had been walking but had never been breathless. He was able to go away on holiday during the summer. On 17. 2. 31 he felt sick and vomited about a pint consisting of stomach contents and then green bile. He complained of a headache. For several days he was generally out of sorts. On 20. 2. 31 at 8 a.m. he had an epileptiform convulsive seizure when his breathing became very laboured and he became very cyanosed. He felt very hot and was bathed in a cold sweat, being "icy" cold to the touch. He held his head back and

Case No. 68. (Contd.).

gasped for breath with convulsive movements of all his limbs; these fits lasted only a few seconds. These attacks came on each day until he was readmitted to hospital.

Condition of admission. T. 96·8°F. P. 28. R. 32. B.P. 120/70. Patient was slightly cyanosed and cold on admission. His pulse was very slow, fairly regular but rather weak.

The area of cardiac dullness was not increased but liver dullness was enlarged and palpable. It was not tender on admission but had been previously and the skin in this region was erythematous due to blistering. The cardiac sounds were very frequent and poor in quality but no murmur could be detected. There was very slight oedema of his legs. He had several convulsions on the day of admission.

His progress was gradually downhill. Oedema increased, cardiac sounds became poorer in quality and liver became more enlarged and tender. Stokes Adams' seizures occurred almost daily.

On 19. 4. 31 he had a succession of very acute attacks During the periods of consciousness he showed evidence of mental deterioration with loss of knowledge of time and place. He gradually sank and died on 21. 4. 31 after having been practically unconscious for 24 hours.

B.P. 14. 7. 29 180/60.
27. 2. 31 120/70.

W.R. - ve.

Case No. 69.

Wm. McG. Age 51.

Patient was seen in consultation on 25th August 1928. He complained of severe and continued substernal pain. Pallor was marked. Pulse was poor but regular. Heart sounds were soft and of poor quality.
Blood pressure 110/65.
When he reported five months later, he looked a somewhat weather beaten but robust man. Apart from slight shortness of breath on exertion he felt well. Pulse was irregular - the irregularity being due to frequent extra systoles.

Case No. 70.

Dr. B. Aged 67.

Examined on 8/6/1931.

For past 30 years he had been subject to extra-systole without discomfort, but during the year prior to examination he had suffered from attacks of paroxysmal tachycardia lasting from a few minutes to two hours. Lately these attacks had become more frequent and he was having ?slight syncopal seizures.

His previous health was good. He was a total abstainer and smoked little.

Heart sounds were of poor quality but pure.

Blood pressure 200/110 ?

Case No. 71.

Dr. L. Aged 53.

Was examined on 20/5/31. He complained of pain on exertion. Pain was upper sternal and radiated up into the shoulder and neck. It was relieved by rest and aspirin. He had one attack of pain in bed which was relieved by aspirin. He was subject to fibrositis.

He had a similar attack a year before which lasted on and off for a fortnight. He was quite well after this attack and had worked hard, fish, bathe and play tennis without discomfort.

During the war he suffered from Leishmaniasis of neck and had beri-beri with oedema and cardiac weakness.

He was pale and excited.

Pulse was rapid and regular. Heart sounds were pure.

Blood pressure 145/90.

Three days later he became ill whilst getting his car ready. He was sick and vomited and suffered from praecordial pain; he died soon afterwards.

Case No. 72.

Miss McN. Age 84. Dr. I.

Patient had suffered from intermittent claudication and petit mal for some time. On 3. 1. 31 she took ill with vomiting, praecordial pain and collapse. Seen about 8 hours after, she was very ill, pallid, retching and vomiting. Pulse was very irregular. Cardiac sounds of poor quality. Blood pressure 90/? There was oedema of both lungs.

She died next day.

Case No. 73.

Mr. G. Age 57. Dr. D.

For some time patient had suffered from substernal discomfort on exertion.

On 13. 2. 31 whilst at a meeting in Glasgow he had sudden acute pain in upper abdomen. He went to lavatory but bowels did not move. He went to get bus but could not reach it and had to take a car home. Thereafter bowels moved but there was no relief to pain. Saw Dr. D. on Monday 16th - there was left sided pleurisy. On 17th a little blood was present in spit. He had no nausea, vomiting or retching but sweating and latterly dyspnoea. On 21. 2. 31 he was sitting up and pale but voice was strong . Pulse was 120/140, soft and irregular. B.P. 45/?. Sounds were of poor quality with ? friction over sternum. Spit was muco-purulent and there were rales at both bases. He died that night.

Case No. 74.

Mr. S. Age 60.

Patient was first examined on 15. 10. 29 when he had right hemanaesthesia. Pulse was regular but for extra systole. B.P. 225/110. There were soft apical systolic murmur. Ophthalmoscopic examination showed arterio-sclerotic changes.

He went a trip to Marseilles. On 14th April 1931 whilst on deck he felt extreme nausea. He went down to lavatory but did not vomit. He was very weak and undressed with great difficulty. Shortly after he was seized by great pain in lower 1/3 sternum which lasted until the next day. He vomited after medicine and pain was relieved. Three days later he collapsed while on deck. He came home rather ill and had one faint attack on 28th April.

When seen on 5th May 1931 he looked well. Colour was good. Pulse was irregular, the irregularity being due to auricular fibrillation. Cardiac sounds were poor in quality but pure. B. P. 110/70. Lungs were clear.

Case No. 75.

Mr. R. Age 57.

Patient had an attack of Coronary Thrombosis two years before he was seen by Dr. Cowan and made a good recovery. Blood Pressure was always low and down to 90.

In August 1930 he had cerebral embolism, since then had three epileptiform seizures.

When examined on 26. 5. 31 looked well but a little pale. Pulse was regular. B.P. 110/80.

Soft systolic murmur was audible at the apex.

Case No. 76.

Mrs. McD. Age 67.

For three months had not felt well, dyspnoea being present on exertion. B.P. 160. She was wakened on 28. 5. 31 by severe pain across chest. She felt very ill. There was some vomiting but no sweating. When seen, a few hours later she was very ill and pallor was ghastly. Pulse was soft, rapid and irregular. B.P. 115. Pericardial friction was present.

She died that night.

Case No. 77.

Mr. P. Age 66.

Patient had suffered from angina on exertion for several years. In 1929 he had an operation for gallstones.

On 18. 4. 31 at Islay he had an attack of severe substernal pain. Pain was very bad and accompanied by vomiting and retching. There was sweating. He struggled home from Islay to Ayr, and was sick all the time. He became very short of breath and had cough with blood stained spit and slight pyrexia. When seen on 22. 4. 31 he was orthopnoeic and there was slight cyanosis. Oedema of pulmonary bases was marked. Pulse was regular. Blood pressure 160/95. Pericardial friction was present. He was seen at a later date. He had been dyspnoeic and pleural effusion was detected. Aspiration showed this to be haemorrhagic in type. Tumour of lung was suspected but the previous history indicated the more likely diagnosis of infarction of lung and effusion secondary to myocardial lesion.

Case No. 78.

W.McL. Age 72.

This patient was examined in consultation in 1929, when he had a cerebral attack ? intermittent claudication. He had been very well until 19. 3. 31 when he had an attack of substernal discomfort, dyspnoea and slight pyrexia. In spite of this he actually went up to town. When seen on 23. 3. 31 he was very ill, pale and cyanosed. Pulse was irregular and of poor quality. Pericardial friction was present along sternum. There was oedema of both bases.

He died suddenly on 31. 3. 31.

Case No. 79.

Mr. M. Age 51.

Patient was an old athlete. On 8. 3. 31 while walking home from football match, he had an attack of sternal pain which passed into left arm. He was sick and vomited. When seen 4 days later he was delirious. Pulse was irregular. There were infarcts in both lungs. Temperature was elevated. B.P. 130/65.

He made a good recovery.

Case No. 80.

Mr. S. Age 64.

Patient had history of previous angina of 2½ year's duration. On 12. 5. 31 he had a typical attack of coronary thrombosis with intense pain substernal. He perspired freely but had no vomiting. Blood pressure fell from 160/100 to 115/90. Pulse was soft. Cardiac sounds were poor but pure. When examined again on 13. 6. 31 he had done well but for several attacks of collapse. Pulse was regular. Cardiac sounds were of good quality. B.P. 98/80.
Patient did well.

Case No. 81.

Mr. W. Age 51.

Patient had always been well until 29. 1. 31 whilst at business he had a sudden attack of praecordial pain followed by epileptiform seizure. During the attack it was noted that his pulse was extremely slow. When seen some time later pulse was regular. Cardiac sounds were soft but pure.
Blood pressure 140/75.
He made a good recovery.

Case No. 82.

Rev. Mr. N. Age 57.

Suffered from angina for one year previous to 7. 9. 29 when he had severe attack of praecordial pain lasting sixteen hours. Pain radiated down both arms. He did not vomit. Heart sounds were of poor quality. The following day he complained of abdominal pain. Pericardial friction was not present.

When seen on 3. 10. 29 patient was now much better. Pulse was regular and soft. Heart sounds were pure but of poor quality.

On 7. 2. 1931 patient felt well. There was irrerularity of pulse due to occasional extra systole.

Patient did very well.

Blood Pressure	3. 10. 29	108/50.
	7. 2. 23	145/75.
	10. 3. 21	150/80.

Case No. 83.

Mr. W. Age 52.

Patient had always enjoyed good health until October 1930, when he had an attack of Coronary Thrombosis complicated by double femoral thrombosis and infarction of lung.

On 16. 10. 30 he was seen in consultation by A.W.H. after having been seen by another consultant. When examined again by A.W.H. on 2. 3. 31 he felt very well. Pulse was regular. Sounds were poor. There were dilated veins on chest and abdomen.

Blood Pressure	16. 10. 30	103/65.
	2. 3. 31	120/65.

Case No. 84.

Miss F. Age 84.

Patient was a very active old lady who had been in bed with feverish cold and bronchitis. She wakened up early in the morning of 12. 2. 29 with upper abdominal pain, collapse, vomiting, sweating. Acute abdomen was suspected. When seen later in the day she was very ill and delirious. Pulse was of poor quality. Cardiac sounds were almost inaudible and pericardial friction was heard. She remained ill for some time. When seen again on 22. 1. 31 she was very well. Pulse was regular. B.P. 210/90. Cardiac sounds were pure. She made a good recovery and is going about as usual. 1931.

Case No. 85.

Mr. W. Age 84. Dr. I.

Patient had been golfing two rounds daily at Dunbar and came home on 25. 4. 30. The following day while hoeing in the garden he had an attack of pain in front of chest and down left arm. When seen by me six hours later, he had little or no pain. His face was grey and he was sweating. Tongue was dirty and breath foul. He had occasional vomiting. Pulse was regular. B.P. 145/90 ? There was well marked pericardial friction all over. Some rale was present at bases of lungs.

On 3. 5. 30 Dr. Ingram reported he was doing well.

When seen again on 4. 8. 30 he was very well and looking well. His exercise tolerance was good and he was at business daily. Pulse was regular. There was slight V.S. at apex, distant at aortic cartilage. B.P. 175/85.
Lungs were clear.

Case No. 86.

Mr. S. Age 52.

For some time, 2 weeks, had been troubled with a little praecordial pain on exertion. His feet swelled but he could play golf. On 3. 2. 30 he had a severe attack of substernal pain. Pain radiated down both arms. When seen the following day he was much distressed. There was intense pallor. Tongue was coated and breath foul. Vomiting was almost continuous. Pulse was fast and alternating. B.P. 110/80. Cardiac sounds were poor but pure.

He did very well.

Case No. 87.

Mr. L. Age 52.

Patient had suffered from angina for three weeks and had to stop frequently whilst out walking. On 5. 3. 30 he was awakened by severe pain - substernal and in upper abdomen. Condition was thought to be acute abdomen, but pain went down right arm. Vomiting was constant. When seen, his face was ghastly. He was sweating profusely and sick and vomiting. Pulse was imperceptible. Blood pressure was impossible to estimate.

He died one hour later.

Case No. 88.

E.B. Age 67.

Patient used to take a good deal of alcohol.

Pain was very severe on 5. 5. 30 - retrosternal and down inner side of both arms. There was no vomiting but a little retching. Sweating was profuse and alarm was marked. Pain was persistent. He was given morphine and was quiet all night but pain came back in the morning.

Two days later he looked all right but lips were slightly blue. Pulse was regular and soft. B.P. 128/92. Cardiac sounds were poor. There was some pulmonary catarrh.

On the 8th he felt comfortable but died quite suddenly at 3 p.m.

Case No. 89.

Dr. A.P.G. Age 50.

81.3.30. Since June 1929 he had been troubled with substernal pain on exertion and once or twice when at rest. Of late attacks had been more frequent and he stated that they radiated to the right elbow. He was at Crieff over the week-end but not feeling well came home. He was fairly well but at 11 p.m. 17. 3. 30 he had an attack of agonising pain over the lower sternal region as if he were "being crushed until he burst". Pain radiated to right arm and there was a little numbness in the hand. He began to spit much bloody mucus. He was given morphine with some relief. He was seen by me at 12 noon next day. He was restless. There was slight vomiting. Pulse was regular. soft and rapid. B.P. 108/80. Cardiac sounds were poor. Lips. were bluish. Colour was otherwise good. A good deal of moist rale was present over front of chest. He was sweating a little. At 11 p.m. his face was grey, ashen and sunken. Pulse was imperceptible. Breathing was laboured and he was apparently dying. 1 c.c. Strophanthone was administered.

On.19.3.30 he was much better. Pulse was soft and irregular but he was still a little cyanosed.

20.3.30. B.P. 100/80.

21.3.30 98/70

23.3.30 98/68

On 25.3.30 B.P. was 95/45. He was very well. Pulse

Case No. 89. (Contd.).

was regular. Colour was good. Lungs were clear. There was no cyanosis. Cardiac sounds were poor.

27.3.30. B.P. 92/65. Pulse was regular.

29.3.30. He died suddenly at 4 p.m.

Case No. 90.

Mr. K. Age 54. Dr.L.

Patient seen by me for glycosuria 8.3.27. when he had a very moderate mild diabetes (glucose curve). B.P. 160/90, up to 170/90.

On 19.3.29 he was very well. B.P. 158/83.

On 14.12.30 while out walking he had anginal symptoms. Six days later he had an attack of anginal symptoms. He was vomiting, retching and sweating. He was very ill. Pulse was soft and irregular. B.P. 118/80. Pericarditis was audible. Temperature was slightly elevated.

When seen again on 23.3.31 he was very well. 2½% glucose in urine. Pulse regular and cardiac sounds were of good quality.

Case No. 91.

Mr. A. Age 63. Dr. P.

Patient seen by me 3¾ years before suffering from high blood pressure. He did fairly well but for past year had slight angina on exertion, ceasing when at rest. Since May attacks were more severe but were relieved by amyl. nitrite. He came home from Millport on 8.9.30 and for two nights took attacks in bed.

He was seen by Dr. P. on 9.9.30 after attack of substernal pain which radiated down both arms and into throat. Dr. P. found B.P. 210/144. Attack followed. B.P. in attack 220/156. Two minutes after amyl. nitrite 210/130. Pulse was rapid about 120. He had another attack on 10.9.30. He was excited but had no vomiting or sweating. Flatulence was marked. Cough was troublesome. He was very flushed and excited. Pulse was regular 110-120. There was soft apical systolic murmur. B.P. 170/110. There was moist rale at left base.

He died in 10 days.

Case No. 92.

A.L. Aged 49. Admitted 30.12.31.
Dismissed 5. 1.32.
Insurance Accountant.

Patient had been in good health until 9 a.m. on 30th December 1931 when, after running for a bus, he had an attack of severe anginal pain; swooned and fainted. He was in a fainting condition for about a quarter of an hour.

On recovery he felt very weak and had constant gripping pain, behind the sternum and down both arms. He felt sick and vomited small amounts and had the feeling that if he could be properly sick he would be better.

Patient was a moderately well developed man, very grey haired and prematurely aged. There was slight cyanosis.

Pulse was feeble and irregular, the irregularity being due to frequent extra systoles.

Cardiac sounds were distant and of poor quality. No murmurs were audible. The chest was clear.

He steadily improved after admission and on 5. 1. 32 insisted on returning to his own home for treatment.

TEMPERATURE - normal throughout.

BLOOD PRESSURE -

31. 12. 31. 98/78.
5. 1. 32. 100/75.

WHITE BLOOD COUNT -

5. 1. 32. 5400. per c.mm.

Case No. 93.

H.G. Aged 44 years. Shopkeeper.

During the Christmas Season of 1931 he was working hard from 6 a.m. until late at night. Apart from feeling tired he was not unduly upset.

On the afternoon of December 27th, however, about an hour after running to catch a tramcar he was seized by sensation of constriction in his chest; he felt that he was dying and collapsed. He soon made a partial recovery but for two hours he was breathless, felt nauseated and vomited.

These symptoms were relieved by the administration of alcohol.

He was seen by me on the following day and stated that apart from a slight shortness of breath he was comparatively well. He had no pain and was quite definite that the sensation of constriction had at no time been painful.

His face was ashen grey and his lips were pale. Pulse was regular but rapid 100 per minute. It was of low tension and poor volume. Blood pressure 105/78. Cardiac sounds were almost inaudible but pure.

There were a few crepitations at both bases. Temperature was normal. Leucocytes 12000 per c.mm.

His convalescence was uneventful until the evening of 22nd January 1932 when he had a return of chest

Case No. 93 (Continued).

constriction and breathlessness. The attack lasted a few minutes. He had no sickness but complained of a feeling of numbness down the inside of both arms.

During the following two nights (23rd and 24th) he had similar attacks.

Since then he has remained well and been entirely free of symptoms.

Temperature was normal throughout.

Blood Pressure:-

28. 12. 31	105/78.
29. 12. 31 30. 12. 31 2. 1. 32.	110-105 / 84-78.
8. 1. 32. 15. 1. 32. 24. 1. 32. 25. 1. 32. 26. 1. 32. 27. 1. 32.	115-120 / 84-80.

White Blood Count;-

28. 12. 31.	12000
6. 1. 32.	11000
13. 1. 32.	13000
20. 1. 32.	9000
25. 1. 32.	10000
30. 1. 32.	10000.

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