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The clinical features of coronary thrombosis

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of

CORONARY THROMBOSIS

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CONTENTS

INTRODUCTION

There has always been divergence of opinion as to the causes of degenerative changes in heart muscle. From the time of Morgagni(15) ateriosclerotic changes in coronary arteries have been recognized. Many authors consider these arterial changes as primary; others do not attach such great importance to their presence. Much of the older literature may be discarded in the light of modern research, but there remains a mass of excellent studies, and while a complete analysis of this literature would be of value it has been considered beyond the scope and the intention of the present paper.

Coronary thrombosis has been defined as an obstruction generally acute, of a branch of one of the coronary arteries resulting in infarction and death of the heart muscle in the area supplied by the occluded vessel.(19) It is by far the most serious result of atheromatosis and arteriosclerosis. The working out of the details which comprise the complete clinical picture of this important malady, as well as for emphasizing its frequency and significance, was chiefly done by American clinicians. Dock in America, recognized the condition during life in one patient and reported the case with autopsy confirmation as long ago as 1896.(19) The earliest adequate description of the clinical symptoms and physical signs, however appeared in the German literature in 1910 from the pens of two Russians.(16) In 1912, Herrick(21), in America first laid stress on the fact that coronary thrombosis was a clinical entity quite clearly recognizable during life. Despite these reports and descriptions dating back to more than

thirty years ago, medical interest in the condition does not seem to have been aroused extensively until about 1918. It will be the scope of this paper to review only the clinical aspect which this disease presents.

SYMPTOMATOLOGY

(a) The typical clinical picture

It is deemed appropriate at this point to rehearse the symptoms and findings of a typical attack before taking up those features which are less constant but none the less important. A typical case would present symptoms somewhat as follows:

A man in the prime of life between fifty and sixty years of age comes in generally with a previous story of angina pectoris. Frequently, however, it may be a man who had never had any serious illness and who had always enjoyed good health, who has recently noticed that he had occasional pains over his heart. Often the complaints, even with a previous history of angina pectoris, may be regarded lightly by the patient so that no attention at all is paid to them. These pains would be more than a mere discomfort. They might be stabbing or gripping or burning in character. They are usually situated beneath the sternum and sometimes below the ensiform process.(19) The attack of coronary thrombosis, unlike the typical anginal attack, often is not precipitated by effort. It frequently occurs during rest, while sitting quietly in a chair at a dinner table or during sleep. To be sure, ordinary or more unusual effort is also often found to be a precipitating factor. When the attack takes place the patient is generally quickly aware that something terrible is happening. If he previously experienced anginal attacks he knows that this is different from anything that he formerly has had, or if a patient recovers from one or more attacks of

coronary thrombosis he well recalls these spells and the very dates when they occurred.(7)(16)

If death does not occur we learn that the patient is taken with a severe pain, generally in the chest, although not infrequently in the abdomen. The pain is constricting or squeezing in character, and may radiate in the customary fashion to the neck, throat, shoulder, back, or arms, more often to the left than to the right. The attack may be collapsing in character. Occasionally the patient actually falls, may become unconscious but more frequently has a sense of extreme weakness.(15) This symptom of shock has given rise to the description of the ashen gray countenance with beads of perspiration of most writers. He often gasps for breath and becomes slightly cyanosed. There frequently is vomiting at the onset of the attack, and it is this feature and the general feeling of distress that makes the patient, the family and the physician believe that the attack is one of acute indigestion, especially when it ends fatally, for then it practically always is incorrectly used to describe an attack of coronary thrombosis.

Shortly after the attack has begun the patient presents the picture of one suffering terrifically and seems to be in shock. He looks pale, is cold, and the skin has moist ashen gray color. The pulse will be found to be of small volume and rapid. There generally has occurred a fall in blood pressure and this may continue to fall for several hours during the subsequent days. Both the fall in pressure and the rise in heart rate are in marked contrast to what happens during an attack of angina when the blood pressure is apt to rise and

the pulse rate remains essentially the same. In a small number of instances the pulse may remain slow at the start and begin to rise after several hours or on the following day or so. Changes in the blood pressure may likewise be delayed. Sometimes very quickly or at other times only slowly, edema of the lungs may develop. Occasionally the presenting feature is a patient with acute pulmonary edema. (7) Some cases show no fixed behavior of the pulse. Usually it is rapid, weak, with extrasystoles perhaps, or block or spells of tachycardia or fibrillation. In other cases it is fairly regular or full.(21) Drop in blood pressure is the rule but there are cases in which the blood pressure remains up remarkably well and others in which variations may occur--a systolic pressure today of 120, tomorrow 180.(13) Dyspnea may be an early and marked manifestation or it may appear only later. In most instances a moderate number of moist rales can be heard at the bases of the lungs. Along with difficult breathing, however, there may be a moist and wheezing rales, cough sometimes with blood tinged expectorate and marked cyanosis.(21)

In a short while the liver may become slightly enlarged from congestion and tender to pressure. In fact, the upper abdomen has, at times, a board-like rigidity similar to that which is seen in surgical conditions. At this time the only striking feature in examination of the heart is the quality of the sounds which become very weak. This is especially true of the first heart sound as heard at the apex, which becomes very faint and at times actually inaudible. Very commonly there is a distinct gallop rhythm and an alternating pulse.(7)

It has been clearly established that painless acute obstruction occurs and at times when a large artery is concerned. In other cases the pain may not appear until several hours after the onset of the attack. The diagnosis of these painless cases may be difficult yet is usually possible if we recognize that in place of pain, other symptoms may be present such as abrupt heart failure with dyspnea, cyanosis and other phenomena. Acute pulmonary edema occurs so frequently in cases without pain, that one questions how often the cause of this condition may be due to cardiac infarction. An attack of severe dyspnea with severe shock, cold sweat, rapid irregular pulse and falling blood pressure are sufficient to make a tentative diagnosis of coronary occlusion.(9)

During the course of the first day or two several important developments occur. The pain gradually diminishes though the patient complains of feeling weak. There may have been actual shortness of breath, and at times Cheyne-Stokes breathing is noted. In most cases a slight fever develops. This can easily be overlooked as the mouth temperature may be normal or even subnormal while the rectal temperature may register 100' or 101'F. This discrepancy between the mouth and rectal temperature is probably dependent on the state of shock and dyspnea that is present. The skin and mucous membranes may actually feel cold while a true fever is present.(7) In other cases the temperature may not rise or it may go to 102' or over. It may rise early or only after three or four days. At the same time a slight or a marked leucocytosis is apt to be found. Herrick(21) reports as having seen

cases with counts of 30,000 to 40,000. Both of these features are probably dependent on the degree of infarction of the heart muscle and very likely represent a constitutional reaction to infarcted tissue. In a similar manner, sterile infarcts in other parts of the body, such as the spleen or kidney, even when unassociated with infection, are generally accompanied by fever and leucocytosis. The fever and leucocytosis in coronary thrombosis can develop as soon as several hours after the attack and thereby can be helpful in early diagnosis.(30) In one of Sutton's (15) patients who was seen six hours after onset, the rectal temperature was 96'F. He died suddenly fifteen minutes later from rupture of the left ventricle.

All during this time the patient's general condition and appearance may have changed very little. Those who early develop marked pulmonary edema, within a day or so will have shown a striking improvement. A few develop increasing evidence of failing circulation, such as edema of the legs and liver. Most, however show very little of importance except as mentioned above. As to the exact incidence of a pericarditis and the typical friction rub there is a divergence of opinion. It occurs over the area of infarction and is more often heard the more closely the heart is examined, but that does not mean that even most of these patients need show this sign. It will in some measure depend on the site and extent of the heart muscle involved. Since the greater number of cases involve the ramus descendens and circumflex branches of the left coronary artery, the pericarditis is usually present on the anterior aspect of the heart. Wearn(9) found a pericardial friction only twice in nineteen cases,

and Levine(7) found it in 13.8% of his cases. These variations in opinion are unquestionably due to the conditions under which the patient was seen. Like all pericardial friction it is likely to be evanescent, and its finding depends upon frequent examination and perhaps the "lucky chance" of examination at the right time. In one case in which an infarction of the wall of the left auricle was also found, the friction was heard at the base.(4) The fever and leucocytosis gradually disappear after several days and in some instances the patient looks and feels perfectly well. Even under such apparently favorable circumstances, the outlook may yet be grave as death can come very suddenly.

In an uncomplicated case after the pain has subsided, the patient remains weak, but an uneventful recovery takes place. The temperature and leucocytosis gradually disappear after a variable number of days. The pulse slowly returns to a normal rate and the blood pressure, which had fallen with the attack, frequently remains low permanently, although it rises somewhat from the extreme levels that obtained during the early days. In others the blood pressure again becomes elevated. The various types of irregularities of the heart will be discussed later, all disappear as well as the evidence of slight circulatory congestion, such as rales in the lungs and tenderness of the liver. The time for the apparently normal state to be resumed varies in different patients from a day or two to a few weeks, depending on the severity of the injury.

At any time after the onset a great variety of complications may develop of which a more detailed discussion will be taken up below. Whereas very little attention is necessary

for those patients whose course is simple, much will depend on the proper recognition of the complications. Although most of the cases do not present the picture that is generally seen in serious congestive heart failure, i.e., marked pitting edema of the legs, ascites, hydrothorax, etc., yet some of the atypical cases have these signs and need treatment directed at them.

The physical signs that are present are in most cases of no particular significance, except for the general appearance of the patient, and this is of great importance. He presents the picture of agitation and shock. Prostration is usually a feature. He gives evidence of extreme pain and apprehension. The skin is cold, moist, pale and has a peculiar ashen appearance. Beads of perspiration break out over the body. There may be slight or marked cyanosis of the skin and lips. He is apt to be restless and even thrash around seeking relief. Sometimes the pain is so extreme that the patient will actually be tearing the flesh of his chest in the attempt to drag out the demon that is crushing him.(19) (16) (7)

Examination of the heart usually reveals enlargement, which is sometimes rather marked. In spite of this, the systole is often so feeble that the apex impulse can be neither seen or felt. In some the apex beat will be made out definitely, and it is then apt to be found beyond the left nipple line. The rhythm of the heart will be dominantly regular in practically all the cases, although after the onset of the attack almost any type of irregularity may come and go. According to most authors, murmurs are present in around 50% to 60%. Of these practically all are systolic in character.

Rarely an aortic diastolic murmur is heard.(19)

The presence or absence of murmurs, however, is not nearly so important as is the quality of the heart sounds. This is particularly true of the first heart sound at the apex. The sounds are almost always muffled or distant. One turns from seeing dyspnea, cyanosis, collapse to look upon the chest and find there no signs of tumult or effort. The throbbing present in health may be absent. On occasion the first sound may be found to be entirely absent, while a distinct second sound may be heard. The significant change is the muffling of the sounds, but particularly of the first sound. This may be very faint and slapping and be entirely lacking in the normal, sustained muscular quality. In addition, the rhythm may have a peculiar foetal quality, and what is still more important is the great frequency of gallop rhythm. This is present in the majority of cases and comes with the acceleration in heart rate. It practically always disappears if a satisfactory recovery is made and the heart slows down to normal. This gallop is not necessarily due to a delay in the conduction of beats from auricles to ventricles although such disturbance in conduction does at times occur. On closer examination, a pericardial friction rub may be found. When present it is a very valuable diagnostic sign, but the friction rub is often very transitory and sharply localized so that it is detected clinically in not over 15 to 20 per cent of cases. A distinct pulsus alternans is another frequent finding and may be made out by palpating the radial pulse, while taking the blood pressure, or occasionally on auscultation of the heart. Of the above findings the evidence of hypertrophy and the presence of a systolic murmur are of

little importance as they are insufficiently distinctive, whereas the extreme muffling of the heart sounds, the gallop rhythm, the pulsus alternans, and the pericardial friction rub are of considerable aid in diagnosis. (32)(16)(7)(19)

Not infrequently pain is referred to the abdomen, in which case its differentiation from an acute abdominal insult may be extremely difficult or impossible. With enlargement and tenderness of the liver, the upper abdomen may become quite rigid and since nausea, vomiting, jaundice, fever and leucocytosis may all occur in coronary thrombosis, there may be simulation of acute cholecystitis, acute gallstone colic, ruptured gastric or duodenal ulcer, acute pancreatitis and acute intestinal obstruction.(9) A further point in the examination is the presence of rales in the lungs. In almost all cases on record, rales were present at one or both bases of the lungs. In the milder cases they may be few in number, but in the more severe the rales can be generalized. The findings of localized rales, pain in the chest, cough, fever and leucocytosis, are so much like what are found in pneumonia that this diagnosis is sometimes wrongly made.(7)

Discovery of one of the critical signs, such as pericardial friction sound, an unusually low blood pressure, especially if hypertension has been previously suspected, or changes in the electrocardiagram, in these atypical cases becomes very significant. Discovery of a previous history of angina pectoris is also important for if this condition has been present, a slight temperature and leucocytosis coming on after an increase dyspnea, or an attack of weakness, together with minor changes in the quality of the sounds, or rhythm of the heart, may lead to the proper diagnosis of coronary thrombosis.

Furthermore, although severe pain in the chest is probably the most important clinical feature of coronary thrombosis, it may be entirely absent. In such cases, very marked dyspnea, out of proportion to other evidences of circulatory insufficiency, is apt to be the dominating feature. In others, the clinical picture may be entirely one of pulmonary edema.

The above is a survey of as typical a case as can be given. Yet there is no fixed clinical picture of sudden obstruction of a coronary artery any more than there is of obstruction of a cerebral artery. There is no infallible symptom or sign on which we may pin a diagnosis; none that may be regarded as pathognomonic; none that may not be irregular or absent. Even the supposedly reliable electrocardiogram may fail to show a change. The variations in the details of the picture must depend in a measure on the suddenness with which occlusion occurs, it s measure of completeness, the size and location of the artery and the degree to which collateral circulation may work to repair the mischief wrought in the area robbed of its blood supply.

(b) Character and distribution of pain

The pain of coronary occlusion is similar to the pain of angina pectoris but more severe and more prolonged. The patient may be held in the grip of almost unendurable agony for hours or even days and morphia may be powerless to relieve his suffering.(32) Older authors have graphically described the condition under the term status anginosus. It will be described variously by different patients. It may come on while at his usual occupation, at meal time, while the patient is at rest, or even asleep in bed. He suddenly begins to feel a dull,

heavy pain at the sternum or across the front of the chest. He will often think it is indigestion, but the pain persists and grows to such an intensity in a few minutes or an hour that he fears that he is in the clutches of something more serious and even at grips with death.(40) To some it will be a terrible pressure or load on the chest or throat. To others it will feel as if the chest were in a vise. The pain is generally most severe at the onset and in the course of several hours or days it gradually disappears. After this in some cases there is no pain whatever; in others it remains as a mild lump in the chest. In other patients, the pain may become intermittent, lasting for a few hours and then returning on the following day.(31) Development of pericarditis does not necessarily bring on more pain. Generally at this time the severe pain has already disappeared, or if there remains the dull ache, it is not particularly altered when the friction rub is heard. Very frequently there is an uncomfortable ache in the arms, more often in the left arm. It often extends directly through the chest to the back or up towards the neck and jaws, sometimes producing a clutching sensation in the throat with a sense of constriction. The most frequent site for pain is between the two nipples, but almost any portion of the chest from the epigastrium up may be involved.(9)

The pain may become a relentless pain and in contrast to angina pectoris, last for hours or days. By this time the patient is so anxious and distressed, that he may be unable to be still because of his agony, and thrashes restlessly in bed or paces about the room.(40) The agony may become so extreme that the patient will be crying for death to end

his agony. Relief is not obtained by giving nitrites. In some instances the pain may be associated with a feeling of a lump under the breast bone, which if expelled, would end the attack. There are a few cases in which the pain can hardly be described as severe and may consist of a dull ache or peculiar gnawing sensation in the chest which does not prevent the patient from continuing his work. Cases have been reported who have experienced pain only in the left interscapular and no where else.(32) Hardly an author who has written on coronary thrombosis, has failed to mention the frequency of epigastric pain. These cases often show tenderness, rigidity and distention and resemble an abdominal accident. Then too, there are the cases described above who are apparently free from pain.

(c) Atypical features of coronary thrombosis

The symptoms of coronary thrombosis vary chiefly with the size of the infarct and the previous condition of the heart. An infarction may explain the sudden occurrence of auricular fibrillation, flutter or heart block. It may determine the onset or accelerate the progress of congestive heart failure in the course of hypertension. Apart from these complicated cases, it is now quite certain that a cardiac infarct may for some unexplainable reason lead to dyspnea and failure without pain. It may also be one of the causes of certain syncopal attacks with shock and collapse as are seen in elderly patients.(32)(40)

It has already been remarked how frequently the pain of coronary occlusion is epigastric in nature. The patients may attribute their symptoms to indigestion and tell in detail how abdominal distention precipitates the pain which quickly

disappears as gas is eructated.(4) They beseech the physician to rid them of gas or to prescribe some remedy which will expell it. Also flatulence, distention, nausea and vomiting appear frequently and occasionally there is diarrhea and jaundice.(32)

Vasomotor phenomena are often present. Blanching and sweating the most commonly observed, have been mentioned. Sweating is sometimes profuse, with sweat actually pouring from the body. Wearu(41) mentions the occurrence of transient flushing associated with a sensation of heat.

Patients as a rule remain conscious, although when there is extreme myocardial insufficiency, they may rapidly become unconscious and remain so until they die. Fainting may occur after slight exertion and occasionally the patient has convulsions. Convulsions may be associated with heart block.(32)

It is very likely that some of the characteristics of the acute form may be lacking when thrombosis of the coronary arteries takes place slowly or when cardiac infarction develops as a result of gradual narrowing of the arteries without any true thrombosis. The acuteness and type of symptoms will depend in a great measure on the rapidity of the mechanical occlusion. Wearu(41) has shown how the thebesian vessels may maintain a very extensive circulation through, and has thereby explained how hearts with both coronary arteries completely occludes, could function satisfactorily. In these instances the occlusion must have been gradual.

In cases of so called cardiorenal disease with progressive heart failure, it is not uncommon to find at autopsy, infarction of the heart even with ventricular mural thrombi, without an actual occlusion of the coronary arteries, merely as a result of gradual narrowing of the vessels.(7) The

diagnosis may be difficult, as these cases at no time show an acute episode. One or more of the various findings, however, should be present even in the absence of any attack of pain and without any fall in blood pressure.

When a sudden hemiplegia develops in a patient whose blood pressure is not markedly elevated, the diagnosis of cerebral hemorrhage is too often made. In some cases of this type, the hemiplegia is due to an embolus and not to a hemorrhage, and more careful search for evidence pointing to coronary thrombosis should be made. Occasionally there is very little to help in making a diagnosis. One of Levine's(7) cases showed only hypertension, cardiac enlargement and circulatory insufficiency with a large amount of albumin in the urine. At autopsy an infarct of the left ventricle with a mural thrombus was demonstrated. The ante mortem diagnosis was here impossible with the available data.

(d) Type simulating an acute surgical abdomen

The term "acute indigestion" frequently appearing in the public press as a cause of death is an example of the indefinite and inadequate medical nomenclature. Among patients suffering from acute coronary thrombosis, there is a small group, but none the less an important one, in which the diagnosis is particularly difficult. This refers to those whom present the picture of an acute surgical abdomen with collapse, upper abdominal pain and nausea with vomiting. Several years ago attention was called to the differential diagnosis after observation of two cases.(28) Coffin and Rush(43) state that "in recent years, surgeons have called attention to the 'acute abdomen' but have neglected to stress the possibility of acute obstruction of the coronary arteries causing

abdominal symptoms which may be identical with intra abdominal emergencies." Friedenwold and Morrison(35) call attention to a group of cases of coronary thrombosis which bear a close resemblance to some serious abdominal surgical emergency. Mohler(33) reported two cases in 1933. Both of these cases had a collicky pain in the lower sternal region, muscular rigidity, nausea and vomiting with symptoms of shock. One patient was jaundiced. In both cases it was necessary to resort to the electrocardiogram for a diagnosis. Jennings(38) has reported three such cases. Many have noted acute abdominal symptoms in relation to thoracic disease, but have not referred to coronary thrombosis in particular.

Although quite often a history quite typical of angina pectoris precedes the attack of coronary thrombosis. Occasionally no such warnings are present and the very first indication of any serious trouble is the fulminating attack of pain. Further, this pain can be entirely confined to the upper abdomen or at least may be localized there during the first twelve hours or so.(9) In cases of this kind, vomiting is not at all rare, and in addition, there can be jaundice, marked rigidity and tenderness in the eipigastrium and in the upper right quadrant. Fever and leucocytosis frequently occur. With the above findings present in the same patient, it may be difficult to avoid making a diagnosis of gall stone colic, ruptured gastro-duodenal ulcer, acute pancreatitis, acute appendicitis or acute intestinal obstruction. All of these conditions have been diagnosed in patients with coronary thrombosis. In some cases, patients have been operated and a large number of these die on the operating table. Under such circumstances, every feature of the entire problem that

may throw any light upon the diagnosis becomes important. It may be the marked muffling of the heart sounds, presence of a gallop rhythm, alternating pulse, a pericardial friction sound, or a previous history of indigestion on effort that will direct the physician to the coronary arteries.(7) Minor consideration like the family history, may be useful in the diagnosis.

If electrocardiograms are obtainable, they may offer most valuable information. At times, changes occur that are so characteristic that no other data whatever would be necessary in making a positive diagnosis. It is also important to bear in mind that although these patients complain of pain in the upper abdomen, either at the same time or only a few hours later, they generally complain of a tightness of the chest of a type that is peculiar to coronary disease. Another helpful point is the fact that patients with coronary thrombosis are much more apt to have dyspnea than those with an acute surgical abdomen. It follows that a patient with coronary thrombosis may be excruciating pain in the abdomen, marked rigidity and tenderness in the upper abdomen, nausea, vomiting, slight jaundice, fever and leucocytosis, and yet have no surgical lesion. Waiting a short time may make a doubtful diagnosis more certain and prevent an unnecessary surgical operation on a patient to whom such a procedure could prove dangerous.

(e) Changes in blood pressure

It is now quite well known that the blood pressure falls in coronary thrombosis. With the onset of the attack, and in severe instances, for days after this fall is quite conspicuous. To recognize this constant and important

symptom, it is necessary to know the pressure level before the onset of the accident. A systolic pressure of 130 mm. Hg. may mean nothing as an isolated observation, but it acquires considerable importance if previously it has regularly averaged over 200 mm. Hg. Such observations have been frequently made. At times the pressure is well below 100 mm. Hg. At other times it is too low to be accurately determined. At still other times, the pulse completely fails and no radial movement can be detected. This fall in pressure is one of the most important symptoms of coronary occlusion. In angina pectoris, there is always a rise in pressure with the paroxysm, although the rise may be of short duration. Exceptions to this rule are very rare.

According to Hamman (32) the blood pressure at times falls so low that urinary secretion is impaired and may for a time even be suppressed. Experiments on animals have shown that systolic pressure must fall below 40 mm. Hg before urinary secretion stops. (32)

In levine's (28) cases, 58 patients were known to have a high blood pressure before the attack, or some of the readings during the attack indicated a hypertension. The average systolic pressure was 191 mm. Hg. and the diastolic 110 mm. Hg. Six patients were definitely known to have had a normal blood pressure before the attack. It is obvious that the fall in the blood pressure that commonly is seen, is apt to be more marked if a previous hypertension existed, than if the pressure was normal. One case was known to have had, a short while before the thrombosis, a systolic pressure of 200 and diastolic 110. When seen two hours after the attack, the readings were systolic 160 and diastolic 90; a few days later the

readings were 120 systolic and 80 diastolic. The following day the systolic pressure had fallen to 110. The average patient with a systolic pressure around 150 to 160 following thrombosis, will show a pressure of around 95 to 105 systolic.

Rarely the blood pressure is but little affected for several hours, when it slowly falls; more rarely, a high blood pressure may be maintained throughout the attack. It may be possible in some of these cases that the blood pressure was quite previous to the attack. In many cases that recover, in whicha fall of blood pressure occurred, the pressure remains at a lower level thereafter, even while the patient returned to full activities.(7) There are, however, other instances in which shortly after the first few days, the blood pressure rose again as the patient improved. The recovery in blood pressure may be considerable but not complete.

In most instances, the fall in blood pressure is quite rapid with the onset of the attack. When death does not occur during the early days, the fall in pressure may either take place immediately, so that the first reading is the lowest, or it may gradually fall during the first 12 to 24 hours. During the process of recovery, the blood pressure is apt to rise slightly from the lowest figure, but in most cases does not return to the high level that existed before the attack. As a general rule, the patients who do best are those who show a marked fall with only a slight subsequent increase in the blood pressure. It is very striking that in some cases where recovery is satisfactory, the blood pressure remains low even when they have returned to their normal mode of living, and may not rise over a period of several years.(7) Another interesting feature is that with these lower pressures,

the patients who suffered from angina pectoris before the attack of thrombosis, thereafter were practically or entirely free from anginal attacks.

(f) The development of fever and leucocytosis

Fever may be considered as one of the most constant symptoms of coronary thrombosis. At the time of onset, the temperature is either normal or subnormal. Within twenty-four to forty hours after onset, the temperature may rise from one to three degrees, and continue for from three to ten days. It, however, has been noted a few hours after onset. During the early hours of the attack, the patient may be in collapse, feel cold, show a clammy moist skin and be quite dyspneic. In some cases the mouth temperature may read 97' or 98' when the rectal temperature for some reason or other would show a temperature of 100' to 102'. This is an important point that may be overlooked and may be rather critical in deciding whether an attack of chest pain is due to angina pectoris or to coronary thrombosis.

The duration of the fever is quite variable. It may last from one to several days, or as occasionally happens, it may continue for one to two weeks. The most general temperature is around 100' but on occasion it may rise to 101' or 102'. In a few cases it may go higher. The disappearance of the fever is gradual, usually without any recurrences when the patient's progress is favorable. There are a few rare instances when the fever is absent throughout the entire illness. The cause of the fever is not definitely known, but it is presumed to be due to absorption from the infarcted area. Its extent probably depends on the amount of cardiac

tissue involved. It has been seen clinically that whenever infarcts of parenchymatous organs occur, even in the absence of infection, a fever and leucocytosis may develop.(9)(32)(7)

Closely following the temperature change there is an increase in the number of leucocytes usually from 10,000 to 30,000. This change is quite constant and its presence is one of the important findings in coronary thrombosis. The count becomes elevated early and may remain above normal for several days. Occasionally the leucocytosis lasts 10 to 14 days. Libman and Saks(41) in a case reported in 1931, state that a leucocytosis may develop as early as one and one quarter hours after the onset of symptoms. The increase in white count is accompanied by a distinct increase in the polymorphonuclear ratio, which rises to 80 per cent and sometimes even to 90 per cent. The leucocytosis lasts as long as the fever does, and at times lags a day or so longer. This presence of a leucocytosis is one of the confusing factors in distinguishing an attack of coronary thrombosis from an acute surgical abdomen.

Foster(45) in 1933 reported a case of proven coronary thrombosis in which the temperature went to 107 degrees. It began at 101 degrees on the first day and gradually reached its peak on the fifth day, when death occurred. The white count reached a maximum of 23,800. This is the only case found on record with such a high temperature.

(g) The occurrence of pericarditis

The pericardial friction rub that is sometimes heard over a cardiac infarct has been definitely established as an important aid in the diagnosis of coronary thrombosis. Acute pericarditis not infrequently develops and the only clinical evidence of

this is the presence of the friction rub. To hear a pericardial friction under circumstances that suggest the occurence of coronary occlusion, is practically to establish the diagnosis.(32) There is, however, a great difference of opinion about the frequency with which a rub can be detected. Gorham(47) heard it in five out of six instances examined by him. Wearn(41) discovered a pericardial friction rub in only two of nineteen cases. Levine(7) gives 13.8 percent of patients as showing this sign. Parkinson and Bedford(9) reported that the condition exists in 13 per cent of patients with coronary thrombosis.

When present, a pericardial friction rub is first heard over the lower precordium in the vicinity of the left nipple and towards the sternum. It is a two and fro murmur and differs only slightly from the similar sounds heard in rheumatic pericarditis. It is not apt to be as loud nor is it apt to last as long. In the latter condition, it first appears and is generally best heard near the third left sternal border. Here it is best heard more frequently further down and out near the left nipple. Although the friction rub when present, is first heard one to several days after the onset of the attack, it may become audible in a few hours.(7) The signs of compression of the left lower lobe of the lung that are frequently made out below the angle of the left scapula, in cases of rheumatic pericarditis, are never present in this condition. Pericardial effusion is very rare, and the development of the pericarditis has not produced additional symptoms like pain on elevation of the temperature and pulse.

Gorham(47) states that pericardial friction rubs frequently occur. He described them as usually of light intensity,

developing in the first few days, transitory in nature, often recurrent, and localized over a small area. It may be heard at one visit, missed at the next and heard again the following day. Unless listened for repeatedly and with great care, it may easily escape detection.

When a pericardial friction rub is present, it is one of the most valuable bits of confirmatory evidence we can obtain; when it is absent, the diagnosis of coronary thrombosis is by no means contradicted.(32) In general, a friction rub indicates a fairly extensive area of cardiac infarction, but by no means signifies a hopeless prognosis.

(h) Changes in the urine

The previous history of patients with coronary thrombosis very infrequently gives evidence of renal difficulty. Following the attack, the blood pressure may fall so low that urinary secretion is impaired and may even be suppressed. This condition was first noted by Hamman(32) who observed a scanty urinary output during the 24 hours following the accident. He also reports a patient who had suppression for nearly 48 hours.The most important and constant urinary finding, however, is glycosuria. These conditions associated together, are now being recognized as a definite clinical syndrome.(49)

The possibility of a relationship between coronary disease and diabetes was suggested in 1922 by Levine(28) who found that in a series of cases of angina pectoris, seven per cent of the patients had previously shown a well defined diabetes. The same writer in 1929 in analysing a series of 145 cases of coronary thrombosis, found either glycosuria or a history of diabetes in 23.7 per cent. In some of these cases, there was only a transient glycosuria during the early days

of the attack; in others there was a large amount of sugar in the urine and it was considered that a well established diabetes was present. Between these extremes there were cases in which no appreciable amount of sugar appeared in the urine, even in the days immediately following a coronary occlusion, in spite of the fact that diabetes had been found previously and had been controlled by dietary measures. Some consider the relationship of glycosuria as a causal one. It seems more probable that the two conditions are the result of atheromatosis of the coronary and pancreatic vessels respectfully. It is difficult to imagine how glycosuria per se could determine the onset of coronary thrombosis.

Cruickshank (49) has reported two cases of coronary thrombosis with glycosuria. The most outstanding facts in these cases were (1) the widespread distribution and severity of the atheroma, which affected not only the coronary circulation but also that of the kidneys and pancreas. (2) That a glycosuria so severe as to require 40 units of insulin per day for its control could clear up so rapidly in the face of progressive deterioration of the cardiovascular system.

Parsonett and Hyman(34) in a series of 89 cases, reported 22 patients with definite diabetic symptoms. Of this group, 7 had severe coronary seizures following administration of the initial first day dose of insulin.

It is important to appreciate that glycosuria is a common occurrence during an acute attack of coronary thrombosis, that it may be transitory, and that it need not indicate any important diabetic state. It may prove to be a concomitant of the shock and terrific pain that exists with this condition.

Another feature of the urinary findings is the presence of albumin, casts and cells. During the first few days while the acute process is going and there is fever and pain, the urine may resemble what one finds during an active nephritis. These changes are also transitory and when recovery occurs, the urine returns to a normal state. Scanty urine and practical suppression of the urine during the first day or two, probably results from the shock and the markedly diminished arterial blood pressure. In an occasional case quite a different condition may occur. There are some instances where in the wake of the coronary thrombosis, hematuria develops. This takes place when emboli are dislodged from the mural thrombus that commonly forms within the cavity of the left ventricle adherent to that portion of the heart that becomes infarcted. Such emboli occasionally lodge in the kidneys and here produce secondary renal infarcts. In this manner, gross or microscopic hematuria may result from which recovery can take place.

(i) Disturbances in the rhythm and mechanism of the heart

Upon examining the heart in coronary thrombosis, the most conspicuous and impressive feature that at once attracts attention, is the remarkable contract between the sumptoms of myocardial insufficiency that have been observed and the entire lack of local manifestations of cardiac disease.(32) The usual throbbing present in health may be absent. It is also a striking fact that coronary thrombosis almost invariably occurs in patients who previously have had an essentially normal heart rhythm. A corollary of this observation is that it almost never develops in a patient who previously had persistent

auricular fibrillation. The former has an apparent protective influence over the latter and one may with safety, predict that a patient with persistent auricular fibrillation is most unlikely ever to get coronary thrombosis.

With the development of coronary thrombosis, almost any form of cardiac irregularity may be found. The frequency with which they are observed may depend entirely on the care and frequency with which the patient is studied, for many of the disturbances are very transient, lasting only hours and may not be detected except by the more graphic representation of the heart, like the electrocardiograph.(28)

Cardiac infarction may leave existing murmurs unaffected, or, a systolic murmur previously noted may disappear with the enfeeblement of the beat; or, again, a systolic murmur may make its first appearance. There is nothing of importance in these manifestations nor of help in diagnosis. Gallop rhythm often comes on to betray embarrassed heart action and at times the sounds take on the quality and rhythm designated embryocardia or tic-tac rhythm.

Only a small proportion of the cases show constantly a regular pulse. Of the irregularities, premature beats are the most common, the ventricular form being much more common than the auricular. Inasmuch as these occur commonly in normal individuals and with almost any type of heart disease, they are not of any particular significance. In Levine's (7) 86 cases, there were 35 in which extrasystoles occurred and of these 19 were fatal and 16 recovered. Auricular fibrillation, auricular flutter, heart block, pulsus alternans and paroxysms of tachycardia may frequently occur. Ventricular tachycardia,

otherwise rare, is not uncommon in cardiac infarctions, and its appearance is ominous, since it is followed by ventricular fibrillation.(32) In 14 cases reported by Parkinson and Bedford (9) cardiac infarction induced abnormal rhythm as follows:

	Cases
Paroxysmal Tachycardia	2
Paroxysmal Fibrillation	5
Paroxysmal Tachycardia and Fibrillation	1
Paroxysmal Flutter	3
Paroxysmal Flutter and Fibrillation	1
Partial Heart Block	1
Complete Heart Block	1

Although relatively rare, an important development in an acute attack of coronary thrombosis, is heart block. Any degree of auricular ventricular conduction disturbance may be found during the early days.(28) Simple delay in the conduction time of impulses, resulting in a regular heart rhythm without blocking of beats or actual heart block of varying degrees, is not uncommon. Sudden halving of the heart rate is not an ingrequent event. This results from the blocking of every other impulse from the auricles. At other times only an occasional drop beat may be found. The occurence of complete heart block is very infrequent. Levine (7) has reported two of such cases, both of which died. Frothingham (50) has reported one such case in which the patient recovered. Ball (1) has reported a case of complete A-V dissociation and states further that 93 percent occur in the right coronary and 7 per-

cent in the left. The presence of complete A-V dissociation may be a valuable diagnostic criterion in differentiating between left and right coronary artery thrombosis. Although attacks of syncope may be explained in some instances as a result of heart block and the accompanying temporary standstill of the ventricles, other patients may become unconscious as a result of the extreme state of collapse and the insufficient circulation.

Paroxysmal tachycardia is a clinical syndrome characterized by periods of an unusually rapid heart rate of sudden origin and termination.(23) Electrocardiographic studies have shown that the rapid heart rate is associated with an abnormal point of origin of the cardiac impulse. It has also been shown that the ectopic point of origin may be either in one of the auricles, as is usually the case or in one of the ventricles. Differentiation of the two types can usually be done by means of electrocardiographic records.

Paroxysms of auricular fibrillation may be quite common in the early days of the attack. They may occur with much greater frequency than has been noted, as such attacks are apt to last but a short time and are often not observed. Levine(7) noted 34 cases of auricular fibrillation in 58 patients with coronary thrombosis. In these cases, the heart rhythm suddenly became rapid and grossly irregular and there was present a distinct pulse deficit. The rate of the heart generally is about 140 or 150. Rarely the patient may develop symptoms of marked dyspnea or circulatory difficulty. Usually, however, they are unaware that any unusual changeshas taken place. In some cases the pulse may jump from 80 to well over 100 as the patient sits up in bed. (32) In most instances the attack lasts only from

one to several hours. Development of auricular fibrillation apparently does not alter the prognosis in coronary thrombosis except in cases where the permanent form develops.

Paroxysmal ventricular tachycardia is much rarer but in some ways a more important disturbance. Its association with coronary disease was first emphasized by Herman and Robinson (23) who reported on four cases all of whom were observed with heart rates from 170 to 228 per minute. In only one case, however, was it definitely proven to exist with coronary thrombosis, but it was probable in the other three. Parkinson and Bedford (9) have reported on two such cases in whom electrocardiographic tracings were taken. Herman (17) presented six cases three of which were proven at necropsy to have coronary thrombosis. All had severe anginal attacks and presented pulse rates from 170 to 250 per minute and characteristic electrocardiographic findings of paroxysmal ventricular tachycardia. One out of the six cases was still living after the attacks. In all of the above reported cases, there were symptoms of severe myocardial damage in contra-distinction to the other arhythmias. The average rate seems to run from 150 to 225.

Recently, certain clinical criteria have been brought out which may be an aid to diagnosis of paroxysmal ventricular tachycardia at the bedside (53) as most patientssuffering from coronary thrombosis are cared for in the home, differentiation from paroxysmal auricular tachycardia may become important. In auricular fibrillation, the rhythm is absolutely regular, while here it is essentially regular. A further point that differentiates the two disorders is that, whereas in auricular tachycardia the sounds have a constant and similar character, in ventricular tachycardia, the quality and intensity of the first

sound heard at the apex is apt tovary with occasional heart cycles. The heart sounds for periods of several seconds may be identical, when suddenly a clicking sound is heard, a muffling, or an accentuation of the first sound takes place. Besides these two auscultatory features, another distinguishing point is the failure to produce any effect upon the tachycardia either by direct vogal pressure or pressure on the eyeballs. These latter procedures, as is well known, frequently exhibit auricular tachycardia.

In gallop rhythm there is no change in the rhythmicity of the heart or in the conduction of impulses, but there is a definite change in the quality of the heart sounds. This condition is present in the majority of cases of coronary thrombosis and if careful ascultation is employed during the earlydays following an attack, it is not unlikely that in most patients it can be heard. It is generally best heard in the vicinity of the left nipple and unlike a splitting of the first or second heart sounds, it is a true canter or gallop. The heart rate at this time is generally elevated to about 100 to 120. The presence of a gallop rhythm may seem to be of only minor importance due to its presence in numerous other conditions, but in cases with obscure symptoms any detail may be enough to swing the diagnosis in the proper direction.

(j) Changes in sedimentation time

The rate of red cell sedimentation has commonly been used as an index of the systemic reaction to infection. It has been shown that a rapid sedimentation rate, if present, might be used in the diagnosis of coronary occlusion in patients giving a history of an attack at a time when the leucocytosis, temperature and other systemic manifestations had abated.

The red cell sedimentation time was studied by Rabinowitz and Shookhoff (2) in 10 patients who presented sufficient clinical symptoms and laboratory data to make probable the diagnosis of coronary occlusion. It was observed in these cases that the sedimentation time was definitely shortened, that this change appeared later in the disease than the fever and leucocytosis, and persisted for some time after the temperature and blood count had returned to normal. The persistence of this rapid sedimentation rate seemed to make it a better index to the healing of an acute myomalacia due to coronary occlusion than the temperature and leucocytosis.

(k) Modes of death and types of recovery

A large percentage of patients with coronary thrombosis die as a result of that condition. There is a great variability in the time that death occurs and in the mechanism of fatal events. Death is frequently sudden and often occurs when the general condition has improved and the patient seems on a fair way to recovery. From the moment that the attack begins, death may occur at any time until a period of several weeks has elapsed, although the cause of death is quite different at times. Fulton (46) observed a series of 20 cases in which 11 died. Seven of them died suddenly and four more gradually. The shortest time from onset until death was about two hours; the longest four years. In Parkinson and Bedford's (9) series of cases, eight recovered sufficiently to lead a normal active life, 33 were able to lead a comfortable restricted life and 25 were completely invalid from pain or failure. White (59) in a series of 62 cases of coronary thrombosis of which one-half were alive at the time of reporting, found the average survival period was

20 months. There are many instances in which ambulatory patients, without warning, either while at rest at home, or at their work, suddenly cry out and fall dead instantly. Fulton (46) reports 3 cases which died suddenly without warning while at rest in bed. In many such cases no pathology is found at autopsy. Sudden fibrillation of the ventricles can be offered as a satisfactory explanation of such a sudden death, where no pathology is found. (61) It can also explain the sudden death that occurs some days after the onset of the attack, when the patient was apparently doing fairly well, in which rupture of the heart did not occur and no embolic phenomena had taken place. In fact, ventricular fibrillation explains quite well such unexpected fatalities, but the hypothesis does not lend itself to proof as rarely is an electrocardiogram taken at that time to confirm this theory. (57)

The deaths which occur as a result of coronary thrombosis have been divided by Fulton(46) into three groups. The first includes those cases in which the immediate cause is more or less remote from the original lesion. Not infrequently after an artery becomes occluded, an intracardiac thrombus is formed at the site of the infarct. If this thrombus becomes loosened some disturbance of the circulation occurs. Death in these cases is not strictly speaking a cardiac death. In the second group, the damage to the heart as a result of cutting off its blood supply may lead to progressive weakening of the heart muscle so that death occurs as it does in any other instance of gradual myocardial failure, or there may be sudden rupture of the heart wall. In a third group are those cases in which the heart ceases to beat because of some interference with the mechanism by which the heart beat is maintained in its orderly se-

quence and efficiency, resulting, e.g., in heart block or ventricular fibrillation.

The chief causes of sudden death are heart rupture either because of a softening infarct or a ventricular aneurism; embolism of the pulmonary artery; ventricular fibrillation; and heart block. Whether death occurs as a result of vagus inhibition is a question very difficult to demonstrate. However, a majority of the sudden or instantaneous deaths are unaccompanied by pain.(7)

The importance of ventricular fibrillation as a cause of sudden death is not very generally recognized. In experimental observation it has been noted as the terminal event and especially when a coronary or some branch is obstructed.(20) The first disturbance may be manifest as ventricular extrasystoles, followed perhaps by ventricular tachycardia, culminating finally in ventricular fibrillation. Heart rupture and pulmonary embolism may be demonstrated post-mortem. It is a difficult matter to demonstrate with certainty that heart block has or has not been the cause of death and as for ventricular fibrillation, it leaves no evidence whatever after death as to whether or not it has been present. (46)

When the patient survives long enough for a physician to attend him, general failure of the circulation may result within one to several hours. The patient then presents the picture of profound shock and may actually be unconscious. This can occur even when the heart is contracting regularly. Another type of unconsciousness is the one that results occasionally with actual temporary standstill of the heart and complete heart shock. Death may occur much in the same way as it does in the ordinary case of Adams-Stokes disease from a prolonged heart block.

When block does not occur, and the patient present the picture of extreme shock and low blood pressure, death may follow merely from the results of an enfeebled and insufficient circulation.

After the first day or two, marked and rapid improvement may take place so that the patient can enjoy a sense of complete well being and freedom from symptoms. Despite this, death may occur very unexpectedly between the fifth and tenth day from rupture of the heart through the infarcted area. Rupture may, however, not be the explanation. In these, death may be explained on a basis of ventricular fibrillation or heart block. Further complications which may develop shortly after the initial attack are peripheral emboli. Occasionally emboli are dislodged from mural thrombi and reach the brain, limbs or other important organs. Death may occur from the accompanying gangrene of the leg and the additional burden of the patient's vitality, resulting from the new infarcted blood vessel, or from a cerebral embolus with its trail of hemiplegia, unconsciousness and impairment of the function of the higher centers. Although it seems reasonable to suppose that emboli might be detached at any time, even months or years after the formation of the mural thrombus, and no doubt explains some obscure cases of sudden hemiplegia and the like in vascular patients, this phenonema is rarely seen after the sixth week.

The patient may also, after recovering from the immediate effects of coronary thrombosis, develop a progressive failure of the circulation of the congestive type. The heart may remain regular or after one or more spells of transient auricular fibrillation, it may become permanently irregular. There develops increasing dyspnea, peripheral edema, Cheyne-Stokes breathing and the other evidences of circulatory insufficiency.

When this occurs, death usually takes place in one to three months. In some instances, a fair degree of cardiac strength is restored, the patient becomes ambulatory, remains free from pain, and carries on with a limitation in his activities for years. It is in this last group of ambulatory patients, that instances of true aneurisms of the ventricles are found. (28)

Patients who recover from an attack of coronary thrombosis differ considerably as to their future health entirely apart from the actual duration of life after such an attack. There are three general types of recovery that are fairly distinct and quite different from each other. In one the patients suffer from typical angina pectoris for a variable period to time before the attack of coronary thrombosis. With the attack there is a well marked fall in the blood pressure which with recovery, is usually maintained at a distinctly lower level. In these cases when the patients become ambulatory, the angina pectoris does not return. This type illustrates the most favorable kind of recovery for the patients are in some ways in better health after than before the attack. Several of these patients become quite well and active three to five years after their attacks.

Still another group is one in which angina symptoms, whether existing before the attack of coronary thrombosis or not, are present after it. In such cases either the fall of blood pressure does not occur, was slight, or if it is considerable, it rises to a high level after the attack. Here the patient presents the ordinary problem of angina pectoris which limits their activities to some extent. This may be considered the next most favorable type of recovery.

Patients who show evidence of general circulatory insufficiency after an attack of coronary thrombosis comprise the major group. They may have had no such symptoms before the attack and in fact during the early critical days of acute thrombosis they might show no signs or symptoms that are different from those manifested by the first group, and yet as the days or weeks progress it becomes increasingly clear that the circulation is insufficient; dyspnea, edema, enlarged liver and hydrothorax develop which may or may not respond to the ordinary measures employed under such circumstances. In this group persistent auricular fibrillation may be found. Dyspnea, congestion of the liver and lungs, Cheyne-Stokes breathing, and the like when occuring during the first few days, although evidence of an insufficient circulation, by no means indicate that the patient will belong to this third group when recovery takes place. They are frequently only temporary and the patients showing these signs commonly recover most satisfactorily. When such symptoms are present a few weeks following an attack they have a more ominous significance. There will be an occasional instance in which recovery takes place after a cerebral embolus occurred, and here, the patient will be left with hemiplegia or a like residual.(7)

There is in addition a heterogeneous group which may continue to have anginal attacks of pain and still show evidence of congestive heart failure of varying degree; or as happens occasionally, a patient who was free from angina pectoris for some time after an attack may then develop either pain or dyspnea or other symptoms of an insufficient circulation. Those who become free from pain and well may develop congestive heart failure or angina pectoris in the future, or those who still have anginal attacks may have another attack of coronary throm-

bosis which, if not fatal, may serve to render the patient free from further pain. The great majority of patients, however, will fall into the general divisions as given above.

ELECTROCARDIOGRAPHIC CHANGES

The electrocardiogram is a record of the production of electricity by the heart muscle.(12) This electrical production is one of the functions of the muscle and is intimately connected with the contractions of its fibers. If the muscle be diseased it is not likely to produce the same sort of electric current as when it is healthy, so that the electrocardiographic diagnosis of myocardial disease has become possible through the correlation of special abnormalities of the record, with special types of pathological change. By this means and by animal experiment our knowledge has been increased, so that the electrocardiogram reveals with increasing clearness whether the contractions of the heart is normal or abnormal.(10)

Although the recognition of cases of coronary thrombosis developed as a bedside or clinical diagnosis before the use of electrocardiography was generally made, this latter proceedure has in recent years added materially to the criteria that we use in diagnosis. In fact, certain changes that occur in the electrocardiograms are now regarded as so characteristic that without any other evidence whatever a proper diagnosis might be made in some cases. Other changes have also come to light that are less distinctive but nevertheless decidedly helpful when coupled with further clinical data. Altogether electrocardiographic data as will be seen below are now of great importance both in diagnosis of coronary thrombosis during the acute stage of the disease, and in some instances enabling us to detect even a previous

attack of coronary thrombosis from which the patient has recovered.(7)(12)

In 1918 Herrick(13) published a case with a typical history of coronary occlusion whose record had a peculiarity of the T wave. This peculiarity was the presence in one or more leads, usually in only one, of a downward, sharply peaked, T wave with an upward convexity of the S-T or R-T interval. In 1920 Pardee's(10) attention was drawn to the possibility that the electrocardiogram might afford a means of confirming a clinical diagnosis of coronary artery acclusion. He reported a case and noted that if the peculiarity is only present in lead III it cannot be considered significant unless associated with a downward T wave in lead II, tho the downward T2 need not also have this upward convexity preceeding the peak.

In the original patient this peculiarity of the T wave persisted for months after the attack. He was lost sight of during the war and an autopsy was not obtained when he died in 1919. Herricks patient came to autopsy and was found to have the scar of an old infarct in the left ventricle toward the apex. In 1920 Smith(14) reported a case that had the descending branch of the left coronary artery tied to stop hemorrhage and records of this patient showed the typical T wave for many weeks.

In 1925 Pardee(10) reviewed a series of 150 adult patients from hospital and private practice and attempted to determine a relation between the clinical features and the electrccardiographic record. Thirty-six per cent of the patients with coronary diagnosis showed the special coronary T wave. Thirty-two percent though failing to have the

special T wave, yet had some other significant abnormality of the ventricular waves, so that sixty-eight per cent of these patients had abnormal waves.

As a rule the electrocardiogram after cardiac infarction is characteristic and occasionally it provides the only objective sign of cardiac lesion. The diagnostic feature is the absence of an iso-electric period between the R S and T waves so that a plateau type of curve occurs in which the R-T interval is either elevated or depressed.(9) Electrocardiograms taken within a week of the onset of symptoms of infarction usually show a deviation from the iso-electric line between R-T or S-T. The R-T segment of the curve starts from the R or S either above or below iso-electric level, and being maintained there results in a plateau shaped elevation or similar depression. This deviation above or below iso-electric level usually measures one-third mms. being equal to one millivolt.(11) Where the amplitude of the QRS deflection is relatively large the R-T deviation is greater than where the QRS waves are of low voltage. Often the R-T plateau is flat topped and descends gradually to the iso-electric level, which is reached at a point corresponding to the apex of the T wave in subsequent curves. As a rule the R-T deviation is best seen in leads I and III and is constantly in opposite directions. Elevation in lead I is about as frequent as is depression in lead I. R-T deviation may, however, be confined to a single lead, or may be most evident in leads I and II or leads II and III. Where R-T deviation is considerable the T waves, strictly speaking, are not evident. A positive R-T deviation is the precursor of a negative T wave, and a negative R-T deviation is the

precursor of a positive T wave.(10)

Within two or three weeks after the onset of infarction the R-T segment has usually returned to iso-electric level and the T waves are fully developed in all leads. As R-T deviation is usually in opposite directions in leads I and III it follows that the T waves in leads I and III will also be in opposite directions. These such curves have been divided by Parkinson and Bedford(11) into two main groups and have been called the T1 type and the T3 types of curves. The T1 curve being one in which the T wave was inverted in lead I, the T3 in which it was inverted in lead III.

T1 type: There is a deep, sharply pointed, negative T wave in lead I, often a lesser degree of T inversion or flattening of T in lead II and a sharply pointed positive wave in lead III. T inversion in lead II is less constant in this type of curve than in the T3 type. T inversion such as described may remain almost unaltered for months but some change in the direction of the normal usually occurs within three months. This return toward the normal may proceed until eventually the T waves become upright in all three leads or may stop short of this so that T remains inverted or flat in lead I. The change to a positive T occurs first in lead II, then in lead I while the upright T in lead III becomes blunted or flat. Since the T waves can return to normal it follows that a single record taken at an interval after coronary thrombosis may exhibit any stage of transition between the typical curve and the normal, though it is unlikely that the curve will have returned to normal except after an interval of many months.(11)(10)

T3 type: This type of curve is the exact reverse of the

T1 type. There is deep inversion of T in lead III, a lesser degree of T inversion in lead II and an upright T in lead I. T inversion in lead II is more constant in this type of curve than in the T1 type, and the amplitude of the inverted T is usually greater. In a proportion of curves exhibiting T inversion in lead III there is a large initial Q wave preceeding the R wave in lead III.(29) In later curves the R wave may diminish or disappear so that the Q wave would then be termed an S wave. Subsequent changes in the direction of normal, comparable with those described for the T1 type of curve, follow in the course of time. T becomes upright first in lead II and later even in lead III; all stages of transition between the fully developed curve and the normal may be recorded, but in a few there is little change.(11)

The association of QRS deflection of low valtage has been reported by a number of authors. Bearing in mind the fact that acute infarction of the heart is almost invariable associated with some degree of diffuse ischemic fibrosis of the myocardium we must not conclude that sudden coronary occlusion is the only factor concerned in the production of low voltage. The fact that in some cases exhibiting low voltage, a subsequent increase in size of the QRS wave does occur in one or more leads, does suggest that the acute infarction itself may be a factor. This is bourne out through the fact that Smith (14) produced low voltage curves in dogs by coronary ligation, though not constantly.

Some widening of QRS in one or more leads is quite common and is usually associated with definite notching and splintering. The term arborization block may be useful as a means of classification but that it represents any single

pathological entity is far from proven. It may be transient and has been suggested by Robinson(31) to be due to functional fatigue. It would seem that both widening and low voltage of QRS indicate abnormal myocardial function which may be due to permanent myocardial damage, to impairment of its conducting system or to temporary impairment of the myocardium.

In 40 cases of acute coronary artery occlusion studied by Master (6) definite changes in the P wave occured in 32 patients,(80%). These changes consisted for the most part in increase in amplitude of the P wave of at least 0.5 mm. and occasionally in notching or widening of the auricles complex. In 16 patients the P wave measured 2 mm. or more in height in some lead.

The change occurred more frequently in leads I and II, rather than in leads II and III, but always in lead II. It may be suggested that P wave changes in leads I and II are associated with left auricular dilatation.

The P waves were larger in the first few days of illness when cyanosis, congestion of the lung, enlarged liver, severe dyspnea or orthopnea were present, and became smaller when there was recovery from circulatory failure. The P waves were larger when RS-T changes were present and returned to normal when T wave inversions appeared.

There is evidence that the larger P wave early in acute coronary artery disease is indicative of a dilated auricle and that increase in size or change of shape of the P wave is one of the electrocardiographic signs of coronary artery occlusion.

Bohning and Katz (8) presented electrocardiograms of 10 cases with a clinical history typical of protracted transient coronary occlusion or recent origin. The most significant

findings were changes in the form of the S-T segment and the T wave: Either an elevation, depression or inversion. The most significant fact is that a definite change is present. Successive records usually show a rapid change in contour in the early stages of coronary occlusion. Not all curves in recent coronary cases can be fitted into the T1 and T3 types of Parkinson and Bedford.(11)

Attention has been drawn (8) to a large, upright, sharply peaked T wave whose limb and shoulders are symetrical and have their convexity pointing downward and toward each other, associated with an isolectric or negative S-T interval having a hump pointing downward. It is different from the non-specific tall T wave. This large, upright T wave is most commonly found in leads II and III of the T1 type and is as diagnostic a feature of the coronary occlusion type of curve as the inverted cone shaped T wave of which it is the inverse image. This characteristic has been designated as the upright coronary T wave. (8)

Barnes and Whitten (60) in a series of 12 cases studied in an effort to establish a differention of the areas involved, come to the conclusion that infarction of the left ventricle produced characteristic changes in the R-T segment. The essential change was considered to be the fact that the R-T interval fails to establish an isoelectric level. These changes can usually be classified as of types T and T3 of Parkinson and Bedford. Infarction limited to the anterior portion of the left ventricle, either alone or combined with infarction of the apex, or infarction of the apex alone, produces modifications of the R-T segment of type II, whereas infarction of the posterior portion of the left ventricle, with or without infarction of the apex produces modifications of the R-T inter-

val of type T3. In cases in which infarction occurred in both areas at successive intervals of time, there is a corresponding shift in the changes in the R-T segment, and the last change observed corresponds with the last portion undergoing infarction.

Katz and Kissin (51) out of a series of 16 cases reported 7 in which the only electrocardiographic changes characteristic of coronary disease were found in lead IV. In some cases an upright positive humped S-T segment with a negative coronary T wave was noted. In others, there is a negative S-T interval with a positive T wave. In still others a transient diphasic T wave or an inverted P wave have been described. (17) It may thus be advisable to take a record of lead IV in every case.

Experimental ligation of the coronary arteries of dogs has revealed that if the posterior coronary artery is ligated an inversion of the T wave is produced. Purks (18) has reported a case of accidental ligation of the descending left coronary artery of man. There followed changes in the T wave and an elevation in the takeoff in lead I and a depression of the R-T interval in lead III. These findings were noted 30 minutes following ligation and reached a maximum in 48 hours. In leads I and II, there was gradual descent in the R wave until it finally became isoelectric in 12 days, followed by inversion in 16 days and back to normal in 27 days. An increase in size was noted in the Q wave in lead III especially on the fourth and sixth days.

Many cases of coronary occlusion do not show characteristic changes in routine electrocardiogram. This applies to the so called " silent areas " of the heart where infarction may occur without producing a deviation of the S-T interval from the isoelectric line in any of the three conventional

leads. During some recent animal experimentation, Wolferth and Wood (17) found that typical electrocardiographic evidence of cardiac infarction could be recorded after the occlusion of arteries supplying hitherto silent areas. To do this it was necessary to apply the electrode to the anterior and posterior portions of the chest. They observed a striking deviation in the S-T interval from the isoelectric line. Since that time two cases have been reported in which the three conventional cardiac leads produced nothing of diagnostic evidence until the fourth day. Lead IV showed definite deviation of the S-T segment continously up until the eighth day.

Another feature in the electrocardiogram found by Levine (7) to be of some importance, is the presence or development of prominent Q waves in lead III. Their frequent appearance has been sufficient to require some comment. It is hardly likely that their appearance is accidental. The exact significance of this however, is obscure.

There are of course many other electrocardiographic abnormalities that occur in this disease, but are not different from those observed in other patients with degenerative myocardial disease who have not had coronary thrombosis. Such changes are bundle branch block, or evidence of intraventricular defective conduction as indicated by lengthening of the duration of the QRS complex. Caution must be used in interpreting the abnormal electrocardiogram, properly assigning certain changes to the chronic process in the heart muscle and other changes to the more acute events that follow coronary thrombosis.

The question of the electrocardiogram in coronary disease is far from settled.(13) It is not always possible to interpret the electrodardiographic findings. There is a great

difference to be noted between acute transitory and slow gradual permanent obstruction. As there are cases in which absolutely no changes can be detected, clinical findings must still be the main factor in diagnosis.

SUMMARY

1. The typical clinical features and certain of the atypical findings have been discussed. The pain may vary from a slight discomfort in the chest to an almost unbearable agony. It may be located from the upper abdomen to the upper sternum and throat. In a few cases the patient is entirely free from pain. In some instances the entire picture may closely resemble an acute surgical abdomen. The important features on examination are the appearance of shock, gallop rhythm, various irregularities in heart rhythm, occasionally a pericardial friction rub, rales in the lungs and sometimes liver engorgement. A fall in blood pressure is the rule but rarely may not occur. Fever and leucocytosis develops early in most cases but there are rare exceptions.

2. Changes in the electrocardiograms have been discussed and their value in diagnosis considered. Certain unusual features have been brought out.

3. Glycosuria frequently accompanies coronary thrombosis. At times the urine may be suppressed and it may contain albumin and casts.

4. In order to enable one to more clearly understand the course of each particular case it is necessary to have a knowledge of the types of death and of the types of recovery that are met with in patients with coronary thrombosis.

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