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Donald A. Limbeck

-- INTRODUCTION --

Due to improved methods of diagnosis and better surgical technique, more cases of successful suture, with recovery, of stab wounds of the heart are being recorded in the current literature. It is the intention of this paper to discuss the subject of stab wounds of the heart, primarily from the standpoint of electrocardiograms obtained, and to compare these findings with a case the author was privileged to observe for a period of ten months.

-- REVIEW OF LITERATURE --

GENERAL --

Block in 1882 first attempted surgical repair of a traumatic heart wound but was unsuccessful, and Rehn in 1896 reported the first successful attempt.[14]

Bigger[22] reports that approximately one-tenth of one percent of all patients admitted to the surgical service of a noted southern hospital over a nine year period were cases of traumatic heart wounds. Recent literature[21,22,28,29,49,51,53,60,63,78] gives a range of mortality of these traumatic heart wounds of from twenty-two to sixty-one percent and even to one-hundred percent in cases of pulmonary artery and aortic wounds.

Brickner[30] states that approximately thirty percent of cases of cardiac wounds die pre-operatively,

thirty to forty percent die during surgery, and thrity to sixty percent recover. However, throughout the past two decades, the mortality has gradually decreased.

Generally speaking, the diagnosis of cardiac wounds [15,20,29,49,52,79,84,87,111,112] is not difficult if the possibility of such injury is considered. Such a patient usually presents a fairly characteristic picture, including a majority of the following: 1) history of chest wound, 2) a variable "symptom-free" period in which he may walk or drive amazing distances before collapsing, 3) circulatory collapse quite beyond that due to obvious external blood loss, 4) faint and distant-sounding heart tones, 5) weak or absent peripheral pulse, 6) low blood pressure, 7) increased venous pressure, as high as two to three hundred millimeters of water, 8) marked dyspnea, 9) pallor and / or cyanosis, and 10) eventually coma. Failure to see cardiac pulsation under fluoroscopy with or without the characteristic "water-bottle" configuration is at least tentative evidence of cardiac wound with actual or impending tamponade.

Beck [15] lists the well-known triad of symptoms of acute cardiac compression as follows: "1) A falling arterial blood pressure, 2) A rising venous pressure, and 3) A small quiet heart."

Maquire and Griswold[79] state that the most frequent sites of injury of the heart, in order of their frequency, secondary to penentrating wounds of the chest are the right ventricle, left ventricle, auricles, and the aorta.

Treatment of a cardiac wound is generally considered a surgical emergency[18,19,21,36,49,50,51,54,58,87,101,102,127] and operation is performed at the earliest possible moment. There are, however, some authors[24,110] who advocate non-surgical relief of the tamponade by aspiration first, provided the major presenting clinical syndrome is tamponade alone, and surgery later if the tamponade recurrs or if the clinical picture fails to improve.

Singleton[123] discusses the cause of death in heart wounds and groups them into four categories; 1) tamponade, 2) hemorrhage, 3) foreign body, and 4) infection, in that order of frequency of occurrence. Tamponade is by far the most common complication and usually occurs early. Hemorrhage, however, results in the highest mortality when it occurs as a complication. If the wound is large, exsanguination occurs and results in fairly rapid death, whereas if the wound is smaller, tamponade is the more probably result. This is frequently life-saving because death is slower and thus

allows time for surgical intervention.

CASES WITHOUT ELECTROCARDIOGRAMS --

Numerous other authors [1,3,13,15,16,18,19,23,25,27,30,32,36,37,40,50,62,66,67,76,88,101,113,115,118,129,134,135,140] have reported cases without electrocardiograms which deal with some other aspect of the problem such as diagnosis, treatment, mortality, etc. They are listed for reference but will not be discussed further here.

CASES WITH ELECTROCARDIOGRAMS --

Electrocardiograms in cases of cardiac wounds were first reported in 1924 [41,128] but are rare before the 1930's. In most instances only the three major limb leads were described. There are only twenty-two papers prior to 1946 recording the use of chest or augmented leads [90] and only slightly over one hundred cases where in electrocardiograms were recorded at all. There is an amazing lack of long-term studies or complete series of electrocardiograms on anyone patient. The article by Solovay, Rice and Solovay [128] is one of the more complete reports prior to 1941 and Noth's review of the literature and report of twenty-three cases [90] is the most extensive up to 1946. Since 1946 less than six papers [2,38,70,90,105] in the literature available re-

porting electrocardiograms in cases of cardiac wounds could be located.

Solovay and his co-workers[128] summarize the pertinent electrocardiographic features of "twenty-two previously published cases, which is all that could be found in the literature" prior to 1941. "In fifteen of seventeen cases----where electrocardiograms of the first week were taken, a T_2 type of curve appeared. The R-T segments were elevated in the first two leads or in all leads and generally most marked in lead two. In one case (a reported case) this was rapidly followed by inversion of the T-wave in all leads as early as the third day, which by the sixth day was showing signs of becoming upright again. In some of the patients the R-T elevation in lead three was very slight and was followed by transient T-wave inversion in that lead, which usually disappeared by the end of the first week, These curves occurred early and were transient, as occurs typically in pericarditis."

"Tracings made two-to-four weeks post-operatively---were characterized generally by bow-shaped R-T segments ---and by inverted T-waves. These reverted to normal in from two-to-six months in most cases. In wounds of the anterior surface of the left ventricle, with or without involvement of branches of coronary arteries serving

this region, a late T, type of "curve with inversion of T-waves in lead one or leads one and two were found. In those patients with injury to the right anterior ventricle wall without damage to coronary vessels supplying the left ventricle, T-wave inversion in all three leads was present". One case was "complicated by purulent pericarditis, which may have produced sufficient inflammatory destruction of the superficial layers of the myocardium to cause the T-wave changes.----the remainder of this group of cases----suggests that an injury of the anterior wall of the right ventricle may in its later stages produce T-wave inversion in all leads."

"Those patients with wounds of the anterior right ventricle wall close to the interventricular septum with ligation of the descending branch of the left coronary artery showed a late T, type of tracing, probably because the bulk of the muscle in the infarct of the anterior left ventricle wall was much greater than that damaged in the injury of the right ventricle itself."

The authors therefore conclude, similarly to Noth [90], that the "electrocardiographic alterations of the first week or ten days following suture of the heart wound are due to pericardial inflammation which in most cases is of the serofibrinous variety. The T-wave inversion which is found two weeks to a month later, we

believe, represents late evidence of destruction of a localized area of the myocardium by the original traumatizing agent, subsequent to suturing and infarction if a coronary vessel was ligated."

"The Q wave changes in the case reports herein reviewed did not give any clue as to the location of the damaged muscle.----these Q waves were probably due to change in position of the heart, because of manipulation at operation and because of post-operative effusions and adhesions in the pericardium and pleura."

In summary Solovay and his co-workers conclude;

1) "The sutured wound of the heart wall is a localized area of myocardial injury, necrosis, and cicatrization. Ligation of a coronary vessel, generally the descending branch of the left coronary artery, is a frequent complication. Serofibrinous pericarditis occurs in almost all cases post-operatively."

2) "In injuries involving the anterior wall of the left ventricle or arteries supplying this region electrocardiograms taken from several minutes to a number of hours after operation may show the T, type of picture with elevation of the R-T segment in lead one and depression in lead three----."

3) "During the first week the electrocardiographic picture is that of pericarditis, regardless of the lo-

cation of the wound or whether a coronary artery is ligated. There is an elevation of the R-T segment in the first two leads or in all three leads, generally most marked in lead two. This is called the T_2 type of tracing."

4) "From two weeks to one month after operation, T-wave inversion is fully developed and may last from two to six months or more before reverting to normal. In anterior left ventricle injury, the inverted T-waves are in lead one or leads one and two. In anterior right ventricle, the T-waves are inverted in all leads----."

Noth[90] reports a series of twenty-three patients with cardiac wounds and gives an excellent review of the literature. In his series, five were wounds of the right ventricle, eleven of the left ventricle, one of the left auricle, one of the pericardium and branch of the right pulmonary vein, and five were not operated. The electrocardiograms cover a period of time from eighteen days to three years and the first electrocardiograms range from one day post-operatively to the thirteenth day post-operatively. Only two cases have pre-operative electrocardiograms. The average number of electrocardiograms per patient was seven but ranged from one to twenty. In two cases only the three standard leads were taken; "in five a single precordial lead (IV F) also was obtained on one or more occasions; in

four the precordial leads V_2, V_4, and V_6 were obtained in addition to the standard limb leads; in one there were three standard and six precordial leads (V_1-V_6); in eleven a total of twelve leads (three standard, V_1-V_6, and augmented unipolar leads from the extremities) were taken on one or more occasions."

In this series Noth reports "definite evidence of pericarditis in seventeen patients." Ten of the patients revealed electrocardiographic patterns "classified as evidence of non-specific myocardial damage." These patterns included "depression of the R-T segment in one or more leads,----inversion of T-waves----in leads other than those reflecting the localized myocardial damage,----auricular fibrillation,----prolonged Q-T interval,----defective intraventricular conduction,---- and prolonged auriculoventricular conduction----."

Of this series, Noth states that "the most common finding was deep persistent inversion of the T-waves in one or more of the precordial leads.----in sixteen cases S-T segment elevations and in eleven cases T-wave inversions characteristic of pericarditis were observed."

The R-T segment of five of eight patients with electrocardiograms during the first twenty-four hours post-operatively revealed evidence of pericarditis, in some cases "as early as eight hours post-operatively."

Noth states that "by the second day and thereafter during the first two or three weeks, the characteristic upwardly concave elevations of the R-T segment (indicating pericarditis) were almost uniformly present." These characteristic alterations of the RS-T segment occur most frequently in leads one and two and "disappear in from eight to eighteen days, averaging thirteen days."

T-wave patterns in Noth's series of patients tended to be elevated early, coincidental with RS-T elevation. The T-wave elevations level off in a few days and by the start of the third week when the RS-T elevation is subsiding, the T-waves are almost uniformly inverted. Noth states that T-wave inversion as early as the first week "may indicate localized ventricular injury since pericarditis rarely causes inversion at this time, particularly if elevation of the RS-T segment is present concurrently."

Only three of eight patients examined at a later interval of five to thirty-six months were found to have no definite objective evidence of residual cardiac damage, and all three gave "unmistakable evidence of neurosis." The remaining five had abnormal electrocardiograms. In two of the latter group the only objective abnormality was "clinically insignificant" pleuropericardial adhesions under fluoroscopy, while the remaining three revealed definite objective evidence of cardiac

damage.

Noth states that the "uniformity of electrocardiographic patterns" in the "early intermediate stage" (second post-operative day to start of third week) of his series was "due to the practically universal presence of pericarditis." He also concludes that cases of T-wave inversion during the first week are evidence of "non-specific myocardial damage" and "due to shock and anemia which are most marked at this stage," or to direct effects of the wound, rather than to pericarditis in which T-wave inversion appears later. Of the "late intermediate stage," (third week to the third month) he states that there is some evidence of persisting pericarditis and increasing evidence of myocardial damage but doubts whether one could attribute definitely the electrocardiogram pattern to either cause, because of overlapping. Of the "late stage," (three months or more) he states that "the three standard leads in approximately fifty percent of patients with persistently abnormal tracings correspond very well with the previously described "late T, pattern" of left ventricle wounds and in most instances with the "late T_2-T_3 pattern" of right ventricle wounds.----the later emergence of these patterns indicates that they were present earlier but usually indistinguishable from the changes due to pericarditis."

In summary Noth states:

1) "During the first twenty-four hours----changes due to pericarditis frequently appear but usually at this time do not obscure the RS-T segment and T-wave patterns of myocardial infarction due to laceration or ligation of the coronary artery."

2) "During the early intermediate period the effects of pericarditis predominate and cause strikingly similar findings in the majority of cases, regardless of the location of the wound."

3) "During the late intermediate period the electrocardiographic effects of pericarditis may persist. This makes the T-wave pattern unreliable at this time for locating the site of the wound."

4) "The consistant finding of abnormalities of the T-waves limited to one or more of the precordial leads----in late tracings of patients with left ventricle wounds amplifies the previously described "T_1 pattern" ----."

5) "The failure to find differences in the electrocardiographic patterns resulting from wounds of the heart, or between those with and without interruption of a sizeable coronary artery is usually due to the obscuring effects of pericarditis in most tracings except those taken either very early or after several months have elapsed."

Herve and Forers Sarabia, one of the authors quoted by Noth[90], reports "elevated concave R-T segments," representing electrocardiographic evidence of pericarditis, within the first twenty-four hours in better than eighty-five percent of his cases. They persisted for approximately eight days and gradually decreased to the isoelectric level. T-waves ordinarily positive and possibly slightly exaggerated in leads with elevated R-T segments gradually flattened and in seven-to-nine days became inverted, remaining so thruout the second week in approximately eighty-five percent of the cases reported.

VanderVeer and Norris[133] in reviewing fourteen cases also note the presence of electrocardiographic features characteristic of pericarditis. The "typical finding is elevation of the RS-T segment in leads one, two, and three, usually most marked in lead two." They conclude that "----electrocardiographic changes in many cases of stab wound of the ventricles suggest acute pericarditis rather than a single anterior lesion of the myocardium.", and also that "----a normal chest lead, the absence of a reciprocal action of the RS-T segment in leads one and three, and no well developed Q pattern differentiate this condition from acute myocardial infarction." In another paper[132] the same

authors state that "in cases of acute pericarditis, the changes in the electrocardiogram are generally caused by subepicardial myocarditis."

This view is in essential agreement with the conclusions of Barnes[7] regarding myocardial infarction complicated by pericarditis. He reported a number of cases in which there was early R-T elevations in all leads due to pericarditis.

Similarly, Koucky and Milles[74] report a case in which the take off preceeding the T-wave is elevated in the first two leads (Pardee type curve). Electrocardiograms on the fifteenth post-operative day show inverted T_1 and T_2 and isoelectric T_3, and are normal in five months. They stated that the electrocardiograms varied little, despite the location of the wound on the anterior surface of the heart or involvement of the coronary arteries, and that the "primary change is a rise in the take off of the T-wave with a gradual return to the isoelectric line, accompanied by inversion of the T-wave within ten days." They also concluded that the "----electrocardiographic changes noted are---- generally----purely myocardial in origin and are dependent on the degenerative changes that occur in the muscle fibers involved."

Bates and Talley[10], Purks[106], Elkin[54], Davenport[41]

Cole[36], and Porter and Bigger[104] all have reported similar cases and are in essential agreement with the electrocardiographic pattern as reported above by Solovay[128], Noth[90], Vanderveer[133], Koucky[74] and others.

Harvey and Scott[64] report a case of purulent pericarditis in which the pericardium was aspirated repeatedly and finally was opened and drained surgically. The RS-T segments showed a markedly elevated take-off in all three leads which was most marked in leads one and two. These gradually decreased following aspiration but never completely disappeared. The T-waves, at first elevated, gradually decreased, after two-three days, in leads two and three and finally became isoelectric in lead three in two weeks. Small Q_2 and Q_3 waves persisted thruout.

Kapp and Grishman[70], in more recent literature, have reported a case of gunshot wound of the heart wherein electrocardiograms revealed: "deep Q waves in leads two, three, CF_2, CF_3, and CF_4. The T-waves were inverted in CF_3 and CF_4, low in CF_5, and isoelectric in CF_6. Augmented unipolar extremity leads revealed a deep Q wave in AV_F, a slight depression of the S-T segment in AV_L, and inversion of the T-wave in AV_L. Multiple chest leads recorded with a unipolar central

terminal revealed deep Q waves in V_2, V_3, V_4 and small Q waves in V_5 and V_6. The T-wave was semi-inverted in V_3 and inverted in V_4."

Crastnopal, Goldberger and Marcus[38] point out that "signs of myocardial injury may remain in the unipolar extremity leads or multiple precordial leads even when the standard leads have returned to normal."

Beck[14] notes the high incidence of pericarditis, pericardial effusion and pericardial adhesions as complications in cases of cardiac wounds.

Bellet and McMillan[17], Vanderveer and Norris[133], Barnes[7,9] and numerous other authors[11,14,30,43,56,57,60,81,103,119,128,133,140,143,144] are also aware of the role of early pericarditis in similar cases of traumatic heart wounds and are in essential agreement with the electrocardiographic features of the condition as outlined above by Solovay[128], Noth[90], and others and below by White[139], Wood[143], and others.

In view of the fact that so many authors have noted the pericardial-like electrocardiogram pattern in cases of heart wounds, the following brief descriptions of electrocardiograms typical of pericarditis are included for comparison.

Wood[143] in 1937 made the first extensive report of electrocardiographic findings in pericarditis and

noted early and transient elevation of the RS-T segment in all leads and especially in lead two, calling this pattern the "T_2 type pattern." He concluded that there is an early stage, with elevation of the RS-T segment, and a later stage, with inversion of the T-wave with or without upward convexity of the RS-T segment, but he emphasizes that the "important feature of these changes is that they occur in all three leads and especially in lead two."

White[139] cites two distinguishing criteria for pericarditis; 1) "the same QT segment and T directions are usually consistantly found in all the classical leads in contrast to their opposite directions in leads one and three in myocardial infarction, and 2) the Q waves are not exaggerated in pericarditis as they tend to be in either lead one or three after acute coronary occlusion.

The case reported by Mustard, Solovay, and Ford[85] agrees with Wood's "T_2 pattern" of early pericarditis, as does that of Purks[107] who also reports the presence of a "Coronary T-wave" in a case of purulent pericarditis wherein the RS-T segments were markedly elevated and of the "plateau type". These were followed by slightly negative T-waves in leads one and two. Lead three showed a convex upward RS-T segment with an isoelectric

T-wave.

Scott, Feil, and Katz[120] also report the occurience of a "coronary type" pattern in their case of pericardial effusion. Porte and Pardee[103] report similar electrocardiographic patterns.

Davenport[41] ligated the interventricular branch of the left coronary artery and vein in the suturing of a cardiac wound and reported that the "striking feature of the electrocardiogram was the negative T wave deflections in the three deviations." Fowler, Rathe, and Smith[56] ligated branches of the left coronary artery and obtained uniform alterations of the electrocardiogram.

The T-wave was increased in size, approaching in magnitude the height of the R deflection, and usually becoming negative in twenty-four hours. The RS-T segment commonly showed a high take-off from the descending limb of the R wave. The QRS amplitude in all leads was generally decreased, as early as the second day. They concluded, therefore, that: "----the alteration in the T deflection was associated with a lesion of the myocardium. We believe that electrocardiographic changes of this character are indicative of a myocardial lesion and feel that these findings may be helpful in the diagnosis of occlusion of the small branches of the coronary arteries----."

Bean[11], in reporting a bullet wound of the heart with ligation of the anterior descending branch of the left coronary artery, concluded that the changes in the electrocardiogram were due to the combined effects of pericarditis, operative trauma (including the apex suture[14] used for stability during operation) and the bullet wound.

Davenport and his co-workers[42] reported a series of electrocardiograms following a cardiac wound which revealed RS-T and T-wave changes similar to those described above as well as the T_1 type seen in coronary occlusion. Parkinson and Bedford[100], Caviness and Turner[33], McGuire and McGrath[81], and numerous other authors[8,47,57,59,61,68,69,83,86,94,108,116,136,138] have reported essentially the same electrocardiographic pattern in cases of cardiac infarction and following wounds of the heart.

Zerbini[144] in his case of heart wound with coronary ligation failed to show the electrocardiographic changes described by Davenport[42] and other writers. He concludes his case with a diagnosis of pericardial effusion, with electrocardiograms similar to Scott and his co-workers[120,121].

Many experiments have been performed on laboratory animals in which coronary vessels have been ligated and/

or the heart has been subjected to a stab or lacerating wound. Barnes and Mann[9], for example, found that despite strict asepsis mere opening of the pericardium resulted in extensive pericarditis (thus accounting for the complicating electrocardiographic features in cases of cardiac wounds); and Fowler, Rathe, and Smith[56] reached similar conclusions.

Hermann and Schwab[65] produced artificial tamponade in dogs and goats and obtained elevation of the RS-T segment in all leads, and tracings with upward convexity of the RS-T segment and negative T-waves. The authors attribute the RS-T elevations to "acute decrease in coronary circulation" resulting in general myocardial ischemia. Scott, Feil, and Katz[121] expressed the same thought. The negative T-waves which subsequently appeared were attributed to "subepicardial myocardial involvement." In the healing phases of the pericarditis a "progressive T-wave negativity involving the leads in order and retrogressing similarly is the usual finding. The cause for these changes is apparently the inflammatory infiltration of the subepicardial myocardium."

Scott, Feil, and Katz[120] reported essentially similar electrocardiograms clinically and were able to reproduce them experimentally in a series of pericardial effusions[121] where in they reported three general types

of electrocardiographic abnormalities:- 1) "The S-T segment of this group, which resembles the clinical cases, usually remained unchanged in duration but rose distinctly above the isoelectric level; the T-wave became small, usually inverted, but occasionally remained upright. 2) The S-T segment in this group was shortened but usually remained at the isoelectric level, and the electrocardiogram was dominated by an upright, broad, tall and round T-wave. 3) The S-T segment in this group was also shortened. As a rule, the level of this segment of the curve rose above the isoelectric level, but the striking feature was the developement of a deeply inverted and peaked T-wave."

Schwab and Hermann, in another article[119], found essentially the same thing, as did Anderson and Starbuck[2].

Foulger and Foulger[55] showed experimentally that pericardial effusion and increase in intrapericardial pressure may result in decrease in voltage in all leads and "developement of, and increase in, negativity of the T-waves, this latter change extending even to the production of T-waves of the coronary type----." An intrapericardial pressure of "from three to eight mm. of mercury is sufficient to produce an obviously negative T-wave. The negative T-wave may resemble in all respects

that seen in coronary occlusion."

Inasmuch as many of the above mentioned clinical reports [8,41,56,57,69,81,94,107,116,120] mention "typical coronary patterns", "plateau type", "Pardee type pattern", "T_1 pattern", etc. as prominent features in the electrocardiograms, the characteristic changes described by Pardee [98] should be reviewed; "The characteristic changes appearing a day or two after the obstruction are as follows: the QRS group is usually notched in at least two leads, and usually shows left ventricular predominance. The T-wave does not start from the zero level of the record in either lead one or lead three the from a level not far removed from it, and in this lead quickly turns away from its starting point in a sharp curve with the short straight stretch which is so evident in normal records preceeding the peak of the T-wave. The T-wave is usually of larger size than customary and accordingly shows a somewhat sharper peak. The T-wave is usually turned downward in lead two and in one other lead." Pardee [97] also states that the "significant feature (of a coronary) is 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. If this feature is only present in lead three it cannot be

considered significant unless associated with a downward T-wave in lead two.----downward T-wave in lead one or lead two or both was only considered significant when the patient was not under the influence of digitalis at the time of taking the record.----the coronary T-wave seems to be a quite special sort of abnormality of the electrocardiogram occurring in about one third of all patients who give symptoms suggesting coronary narrowing or infarction.----the coronary T-wave here described is probably due to the secondary reaction of repair about the ischemic area."

Barnes[6,7] agrees with this when he says "the most characteristic feature of the RS-T segment changes following acute coronary occlusion is that an elevation of the RS-T segment in lead one is accompanied by a depression of the RS-T segment in lead three." At the same time, however, Barnes also states in his conclusions that the "electrocardiograms of patients in whom acute coronary occlusion is complicated by pericarditis differ from the type of RS-T changes associated with uncomplicated acute coronary occlusion. The typical feature of the electrocardiogram seen in coronary occlusion associated with pericarditis in its early stages consists of elevation or upward rounding of the RS-T segment in all leads."

Barnes[9] produced similar electrocardiographic experimentally in the dog following ligation of various branches of the coronary arteries. Like Fowler and his co-workers[56], Barnes believed that electrocardiograms could be used in locating the site of injury. He noted that when branches of the left coronary artery were involved, the characteristic RS-T segment deviations were in lead one while the RS-T segment of lead three was a "trough-like depression". When the branches of the right coronary artery were involved, the typical elevation of the RS-T segment was in lead three with the reciprocal RS-T depression in lead one. Smith[124, 125, 126] made similar observations in T-wave changes, as have Clarke and Smith[35], and Otto[96]. Crawford and his co-workers[39] obtained similar localizing effects when respective arteries were experimentally ligated, but Stewart[130] states that the "----exact correlation between lesions and electrocardiographic signs is impossible." Oppenheimer and Rothschild[95] also conclude that characteristic variations of the RS-T segment and T-waves warrant a diagnosis of cardiac infarction but do not specify the site of the infarction.

Wearn[137] observed a series of ten patients with coronary infarction and concluded that "disturbances of

the T-wave and a diminished amplitude were the most constant findings;" and that in one only "was the T-wave found to come off the R wave as described by Pardee[98]." Rothschild and co-workers[114] also observed a series of infarcted patients and divided their electrocardiographic evidence into two stages; the first, ranging from six and one-half hours, at the earliest, to approximately ten days or two weeks, is a stage of "abnormally elevated RS-T segments above the base line"; the second, ranging from about thirty hours to several months is a stage in which the "T-wave may be inverted and assumes a characteristic form, ie, the first limb is curved, the apex peaked, and the second limb is rather straight."

Katz and Kissin[73] conclude from their studies that there are "----three types of changes occurring in lead four which are "specific" for recent coronary occlusion: a) a positive "humped" S-T segment with a negative coronary T-wave, b) a negative "humped" S-T segment with a positive coronary T-wave, and c) a diphasic coronary T-wave which is transient."

Other conditions may produce confusing electrocardiographic evidence. Shearer[122] reports a case of lobar pneumonia showing a characteristic "plateau RT" where the take off of the RS-T segment is high on the

descending limb of the R wave and is characteristic of the early stages of coronary thrombosis. Master and his co-workers [80] report characteristic "coronary T-waves" in a series of forty-five lobar and seven bronchopneumonia cases. DeGraff and Wible [44] produced a similar electrocardiogram picture by digitalis, stating that "except for the fact that the T-wave comes off the S wave instead of the R, it is precisely the type of curve described as characteristic of left coronary occlusion." Kountz and Gruber [75] showed in animals that "when the oxygen saturation of the arterial blood fell below fifty percent of the normal, the animals showed in their electrocardiograms changes similar to those observed clinically in coronary occlusion."

Wood and White [142] report in a case of uremia, the occurrence of electrocardiographic "----changes in the T-wave (diphasic or inverted) of lead two----." They attribute the changes to a toxic, digitalis-like effect on the heart muscle.

Randles and Fradkin [109] report a case of angina pectoris wherein "electrocardiographic changes resembling those observed after a myocardial infarction" are presented. Wilson and Johnston [141] report much the same; "----increase in size of the S deflection in leads two and three and pronounced downward RS-T dis-

placement in leads two and three----. The RS-T displacement----is similar in magnitude and in kind to that seen immediately following sudden occlusion of the anterior descending branch of the left coronary artery. There is one difference. Following occlusion of the artery mentioned the RS-T displacement is usually definitely discardant, ie, it is upward in lead one and downward in lead three. In the three cases of angina pectoris described, however, the RS-T displacement is either concardant (downward in all three leads) or so inconspicuous in lead one as to make its classification as concordant or discordant difficult."

Deep Q waves do not necessarily indicate myocardial distress or disease. Here again, other conditions may simulate the "coronary deep Q wave." Pardee[99] states that elevation of the diaphragm, hypertension, pregnancy, rheumatic heart disease and deep expiration will all give a large Q_3. Edeiken and Walferth[48] Meek and Wilson[82] and Ziskin[145] all agree. Krumbhaar and Jenks[77] state that a certain percentage of normal children will show a large Q_3. Pardee[99] however, also states that between fifty and eighty percent of patients with deep Q_3 will also present other evidence of organic heart disease. DeMay[45] correlates the most useful criteria for differentiating QRS changes

due to coronary artery disease from similar changes due to other causes, and cites Durant's[46] "criteria "C" " as follows: " 1) an initial downward deflection in lead three having an amplitude at least one-half as great as the largest QRS deflection in any lead, 2) an initial downward deflection in lead two at least one-fourth as large as R_2, 3) left axis deviation or a normal axis deviation, and 4) T-wave inversion in leads two and three, but not in lead one."

COMMENT --

The general impression one obtains from the literature survey presented is that in the majority of cases of stab wounds of the heart a T_2 type of curve appears. The RS-T segments are elevated in almost all leads and usually most marked in lead two. These are usually followed by T-wave inversion in from three days to one month post-operatively and last a variable length of time, ranging from two to six months before returning to normal, depending upon the extent of the injury. Pericarditis, therefore, is an almost universal finding in the early post-operative period and the electrocardiographic T_2 type pattern of pericarditis effectively masks any evidence of localized injury. Generalized myocardial inflammatory changes (the so-called "sub-epicardial myocarditis") also contributes to this early

T_2 type pattern. The later T-wave inversion, in all probability, represents later evidence of a localized myocardial injury due to the traumatizing agent and a larger area of myocardial ischemia and inflammatory reaction due to the subsequent suturing. Coronary artery ligation, if present, overshadows the local injury due to the trauma and results in a T_1 or T_3 infarction type electrocardiogram, since the bulk of the heart muscle involved in the infarction is usually larger than that damaged by the traumatizing agent and the operative procedure.

Q wave and QRS changes are of little or no aid in localizing the site of injury, unless the injury includes damage to the intraventricular septum with resultant impaired impulse conduction, or definite coronary artery ligation in which case the Q wave changes are part of the typical T_1 or T_2 type pattern.

exceptionally deeply inverted T-waves or T-wave inversions during the first week may indicate localized myocardial damage, since pericarditis alone rarely produces such deep T-waves or T-wave inversions during the first few days post-operatively. However, non-specific myocardial damage due to shock, anemia, tamponade, and operative trauma may produce similar T-wave changes pre-operatively and during the first

few days post-operatively; hence T-wave inversions at this time are not conclusive evidence of localized injury. After the first week deep T-wave inversion is more indicative of localized myocardial injury.

Signs of myocardial injury may frequently persist in the unipolar limb leads or the multiple precordial leads even after the three standard leads have returned to normal.

Not all elevated RS-T segments are T-wave inversions are due to pericarditis, infarction, local myocardial injury, etc. Electrocardiograms with T-wave and RS-T segment changes typical of myocardial damage may also be found in such conditions as pneumonia, over-digitalization, reduced oxygen content of the blood, uremia, angina pectoris, and various types of acute intra-abdominal pathology such as acute appendicitis, acute cholecystitis, pancreatitis, ruptured viscus, etc. Deep Q waves may also be seen occasionally in hypertension, normal pregnancy, rheumatic heart disease, and deep expiration, although a deep Q wave, in by far the majority of cases, is indicative of myocardial damage.

CASE PRESENTATION

GENERAL

A nineteen year old mexican youth was admitted to the hospital at 10:50 A.M., April 24, 1949 with: 1) a

stab wound of the chest, and 2) severe chest pain and dyspnea. The patient had suffered the stab wound of the chest approximately thrity minutes previously during a fight, and had run two blocks before collapsing.

Upon admission to the hospital, the patient was in severe pain and respirations were labored. The patient's skin had a white pallor but there was no evidence of external bleeding. There was a two cm. laceration over the fifth interspace in the left mid-clavicular line. The chest was "clear to percussion bilaterally" but upon auscultation the breath sounds in the left base were diminished. The heart rate was rapid (140), there were no murmurs, and there was no diminution of heart sounds. Arterial blood pressure expressed in mm. mercury was 0 systolic and 0 diastolic, respirations were thirty per minute, venous pressure one hundred thirty-two mm. water. A diagnosis of laceration of the heart with cardiac tamponade was made.

Portable x-ray of the chest at this time revealed a "bottle-shaped heart" and fluid in the left lung field. The impression of the radiologist was "hemothorax, left chest and hemopericardium, probably due to a stab wound of the heart involving the pleura and pericardium."

The past history, family history and medical

history of the patient were negative except for a "goiter operation" four years previously.

The patient was given five hundred cc of plasma and five hundred cc of whole blood and emergency operation was performed by Drs. J.D. Bisgard and LEO ANDERSON under cyclopropane and local novacaine anesthesia. An additional five hundred cc of whole blood was given to the patient during the operation.

The chest was entered through a curved incision "starting at the second rib and extending parallel to the sternum and inferiorly curving laterally in the fifth interspace". Three inch segments of the fourth and fifth costal cartileges were reflected laterally to expose the pleura and pericardium. Upon entering the chest, the pleura was found to be "perforated in the fifth interspace in the mid-clavicular line and a moderate amount of sanquinous fluid was expressed with each respiration. An visualizing the pericardium there was no apparent distension; however when it was opened a moderate amount of blood was present. There was a three cm laceration in the left ventricle near the apex, which produced extensive bleeding with each systole. There was no evidence of fibrillation during the entire procedure. ----an anchor suture of #1 chromic was placed in the apex to lend stability. The laceration in the

heart was closed with four interrupted #000 silk sutures and the bleeding was controlled by this procedure. The opening in the pericardium was not sutured." The rib cartileges were replaced and a catheter was placed in the pleural opening. "The subcutaneous tissues were closed with continuous chromic #one. The skin was closed with interrupted silk #000. The catheter was withdrawn slowly and the skin was closed tightly over the opening."

The immediate post-operative condition was "good", blood pressure was one hundred ten systolic and sixty diastolic, and pulse one hundred twelve per minute. The patient was given three thousand units of tetanus anti-toxin and started on one hundred fifty thousand units of penicillin every two hours. Blood pressure ranged from ninety-eight to one hundred systolic and from fifty-four to sixty diastolic, pulse from one hundred ten to one hundred twenty and temperature up to one hundred degrees during the remainder of the day.

The following day, April 25, 1949, a thoracentesis of the left chest was done and four hundred thirty cc of dark red blood was removed. Two hundred thousand units of penicillin and one hundred fifty cc of air was injected into the left chest. Portable x-ray of the chest following the thoracentesis was reported as

"heart still globular in shape. There is less fluid in the left chest. The condition generally looks much improved."

Portable x-ray of the chest on April 26, 1949 showed slightly less fluid in the left chest than on April 25, 1949. Blood pressure ranged from ninety-two to one hundred ten systolic and from sixty to sixty-two diastolic, pulse from one hundred eight to one hundred fifty-six, and temperature from ninety-nine to one hundred four degrees during the next three days, after which the temperature remained normal and the pulse dropped to a range of seventy-two to ninety. Progress was good and there were no complications. Portable x-ray of May 3, 1949 revealed an "even density in the lower one-half of the left lung field, indicating the presence of fluid. The heart is not as large as on previous examination." Six-foot upright x-ray of the chest on May 11, 1949 showed further clearing of the fluid in the left lung field. Following an uneventful recovery, the patient was dismissed ambulatory May 21, 1949. Six-foot upright films of that date were reported as "heart and lungs essentially negative."

The patient has been re-examined at later intervals of one month, three and one half months, and nine and one-half months and on all occasions has presented no

no evidence whatever, subjective or objective, of any residual cardiac damage or impaired cardiovascular function. There has been no signs of any accompanying neurosis and the patient is well adjusted in his former occupation.

ELECTROCARDIOGRAMS --

Electrocardiograms were taken on this patient two hours post-operatively, nine and one half hours post-pperatively, and then at daily intervals for seven days. Follow-up electrocardiograms were obtained at intervals of fifteen days, twenty-four days, forty-two days, two months, three and one-half months, and nine and one-half months. The three standard leads were obtained in all tracings and, in addition, six conventional pre-cordial leads (CF_1-CF_6) were also obtained.

The RS-T pattern on the first day shows progressive elevation, especially in leads one and two as well as in leads $CF_{2,3,4,5}$. During the first week this elevation remains essentially the same in the standard leads, but becomes progressively less elevated and developes a convex upward curvature in the chest leads. At the beginning of the second week the RS-T is decreasing toward the isoelectric level in all leads, and particularly in the chest leads. By the end of the second week the RS-T segment is only slightly elevated in leads

one and two and almost isoelectric in the precordial leads. A trace of elevation of the RS-T segment persists in leads one and two and chest leads $CF_{2,3,4,5,6}$ thruout the course of illness and is still present, to a minimal degree, in the follow-up electrocardiogram nine and one-half months after the original injury. The RS-T segment also retains a slight upward convex curvature in leads $CF_{3,4}$ in the nine and one-half months tracing.

The T-wave becomes progressively tall and peaked in all leads during the first day, but in less than twenty hours the T-wave shows a tendency to round off in both standard and precordial leads. By the second day, the T-wave is diphasic in lead CF_1 and almost isoelectric in leads three and CF_6. The T-wave is absent in lead three after the third day. By the fourth-to-sixth days, the T-wave has started to become negative in leads $CF_{2,3,4}$. At the end of the second week, the T-waves also have begun to dip below the iso-electric level in leads one and two and are quite prominently negative in leads $CF_{5,6}$. The end of the second week also marks the return of a small but positive T-wave in lead three. On the twenty-fourth post-operative day the T-waves are weakly positive in lead three, almost iso-electric in leads two, $CF_{1,6}$, weakly negative in lead one, and strongly negative in leads $CF_{2,3,4,5}$.

The electrocardiogram of two months shows T_1 to be slightly positive while T_2 and T_3 are definitely positive. The T-waves in leads $CF_{1,6}$ are beginning to become negative. The three and one-half months' tracing reveals a further progression of the changes present at two months. At nine and one-half months the T-wave is positive in all three extremity leads. Leads $CF_{1,3,4,5}$ have a definitely negative T-wave, whereas the T-waves in lead CF_2 are strongly positive and those of lead CF_6 are diphasic.

The QRS component of the electrocardiograms is markedly decreased in size in lead three immediately post-operatively and does not regain its normal size until the tracing of the second month. The first limb of the QRS complex in lead three is notched slightly at the baseline and remains so thruout all the series of electrocardiograms. The QRS complex is decreased in size in leads $CF_{4,5}$ the day of the operation and in lead CF_6 the following day. These changes persist in lead CF_4 until the nine and one-half month's tracing and in leads $CF_{5,6}$ thruout all the electrocardiograms. There is a slight notching of the QRS complex in lead CF_4 thruout all the electrocardiograms. This same notching is also found in lead CF_3 from the third day thruout the remainder of the tracings and in lead CF_5

from two hours post-operatively until the nine and one-half months tracing.

A prominent Q_1 was evident in the tracing taken two hours post-operatively and by nine and one-half hours a deep Q-wave in lead CF_6 was also present. A Q_2 wave developes the day after operation and a deep Q wave appears in lead CF_5 on the fifth post-operative day. The nine and one-half month's tracing showed a persistent deep Q wave in leads one, and $CF_{5,6}$.

The P-wave is normal and remains unchanged in the three standard leads. The P-R interval is within normal limits and remains unchanged in all leads. The rythm is a normal sinus rhythm in all tracings. The rate of one hundred eight to one hundred ten obtained in tracings of the first day decreased progressively to a rate of seventy to seventy-two in the tracings at nine and one-half months.

COMMENT --

The present series of electrocardiograms reveals a "Q_1 type pattern" which is partially obscured, within a matter of hours, by a "T_2 type pattern" due to a generalized pericarditis. This T_2 pattern shows signs of abatement by the end of the second week and a late Q_1 type pattern representing evidence of anterior myocardial damage is revealed again. This pattern is

characterized generally by bow-shaped RS-T segments in leads one and two which start at the iso-electric line and terminate in negative T-waves. The T-waves are also negative in all the precordial leads except lead CF_1.

The evidence of myocardial damage reflected in the electrocardiograms of the first day is not sufficient to be of conclusive localizing aid. It is only fair to point out that these electrocardiographic changes may be the result of shock, anemia, and operative trauma which would logically be most severe at this stage, as well as the result of the myocardial injury alone.

The only evidence of any localizing value is the predominance of RS-T segment and T-wave changes in leads one, two, and $CF_{3,4,5}$. The RS-T segment elevations in leads one and two are not associated with the reciprocal RS-T segment depression in lead three as is usually seen in anterior left ventricular infarction. This might have been anticipated, inasmuch as the initial injury did not damage the coronary vessels and they were not ligated during repair of the wound. The damaged area, therefore, was limited to the original myocardial injury plus a surrounding zone of ischemia and inflammatory reaction secondary to the suturing. Such an injury as this represents damaged myocardium

thruout the thickness of the ventricle wall but is a relatively small area when compared to the bulk of myocardium involved in an infarction. Thus, it seems logical to assume that the electrocardiographic evidence of such a wound would be less dramatic and have less localizing value than the electrocardiograms following infarction. However, the deep Q wave in leads one and $CF_{4,5}$, the decreased size of the QRS complex in leads $CF_{5,6}$, and the negative T-waves in leads $CF_{1,3,4,5}$ all point toward the conclusion that some type of anterior myocardial injury has taken place.

The T-waves are tall and peaked in the tracings of the first day but this change is transient and the T-waves rapidly round off and later become inverted in approximately two weeks. These early T-wave changes and the above-described elevations of the RS-T segment in leads one and two are seen frequently in pericarditis.

The residual elevation of the RS-T segment in leads one, two, and $CF_{2,3,4,5}$ and the comparatively more prominent Q wave in leads one and $CF_{5,6}$ are minimal in nature and are probably indicative of the scar formation which has taken place in the area of the original wound. The fact that these changes are still present almost ten months after the injury means that they represent what will probably be permanent electrocardiographic evidence of the injury.

The electrocardiograms here presented are in essential agreement with the findings of other authors in similar cases. Some of the tracings reported by Noth revealed evidence of pericarditis persisting later in the course of illness than was the case here.

-- SUMMARY --

1) A review of the literature on stab wounds of the heart with electrocardiograms is presented, and another case with electrocardiograms is reported.

2) Electrocardiograms taken pre-operatively or within the first two-to-ten hours post-operatively may show signs of localized myocardial injury. These changes may be due to circumscribed myocardial injury or they may be due to shock, anemia, tamponade, and operative trauma which is most severe at this stage. After the first day these changes are, for the most part, obliterated by a generalized pericarditis.

3) During the first two weeks post-operatively the electrocardiographic pattern is that of a "T_2 type" due to the almost universal presence of pericarditis in this early period, regardless of the site of the wound and whether or not there was involvement of the coronary arteries.

4) From two-to-eight weeks post-operatively the pericarditis pattern shows signs of regression and the

T-waves become inverted, giving a late Q_1T_1 type---- which is the most common----or Q_3T_3 type electrocardiographic pattern similar to those seen following infarcion.

5) Signs of myocardial injury may persist in the precordial or augmented leads after the standard leads have returned to normal.

6) Accurate localization of the site of injury is not conclusive unless Q wave and QRS complex changes indicative of septal injury with impaired impulse conduction or definite coronary interruption or ligation with the classical Q_1T_1 or Q_3T_3 type patterns is present.

7) The present case is in essential agreement with similar cases reported in the literature. There are generalized signs of myocardial damage the first day which are masked by a pericarditis type pattern that appears on the second day and lasts approximately two weeks. Following recession of the pericarditis pattern a late Q_1T_1 type of tracing is revealed. This Q_1T_1 type pattern gradually improves and at ten months the patient is asymptomatic, but the electrocardiogram on this date shows evidence of some residual myocardial damage in leads one and $CF_{5,6}$. It would be interesting to obtain electrocardiograms of this patient at eight-to-twelve month intervals for further observation.

Donald A. Limbeck

--- 1950 ---

FIG. 1 -- Portable x-ray of the chest upon admission to the hospital April 24, 1949, showing "water-bottle" contour of heart and hemothorax of left lung field.

FIG. 2 -- Portable x-ray of the chest following thoracentesis April 26, 1949, showing less fluid in left chest and a persistent "globular" shape of the heart.

FIG. 3 -- Portable x-ray of the chest May 3, 1949 showing gradual improvement of cardiac shadow and progressive clearing of the left lung field.

FIG. 4 -- Six-foot upright x-ray of chest at time of patient's dismissal from the hospital May 21, 1949. Both "heart and lungs essentially negative."

Films taken on April 25, 1949 and May 11, 1949 were not included because of their similarily to those here presented. Each of the omitted films represents a progressive pattern between the respective films that were selected.

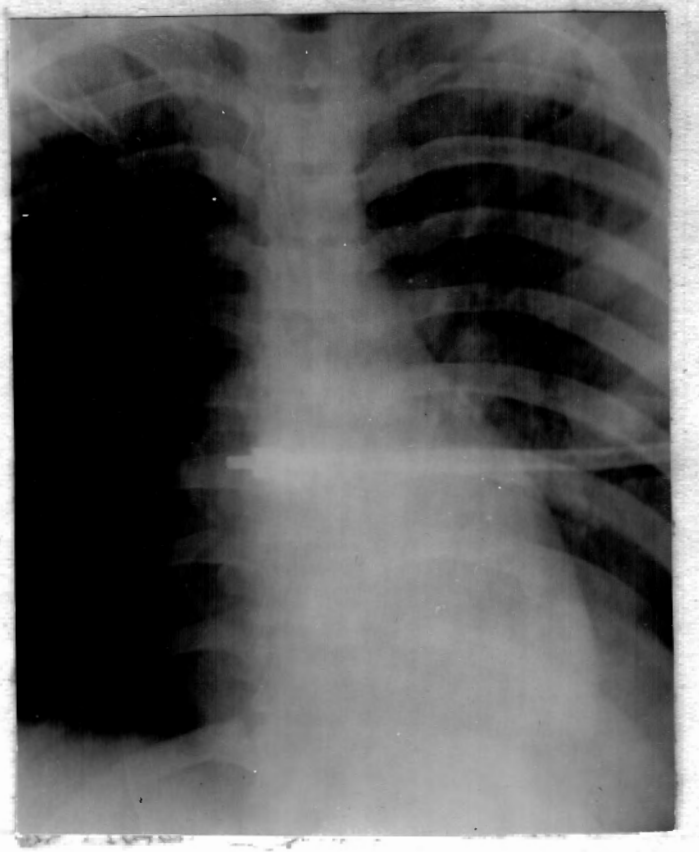


FIG. 1 - 4-24-49

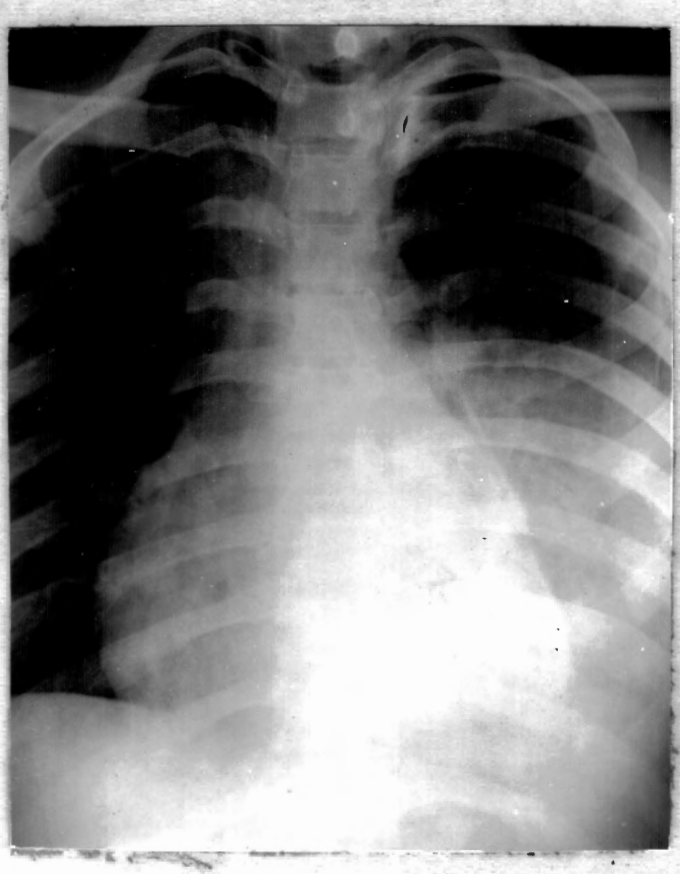


FIG. 2 - 4-26-49

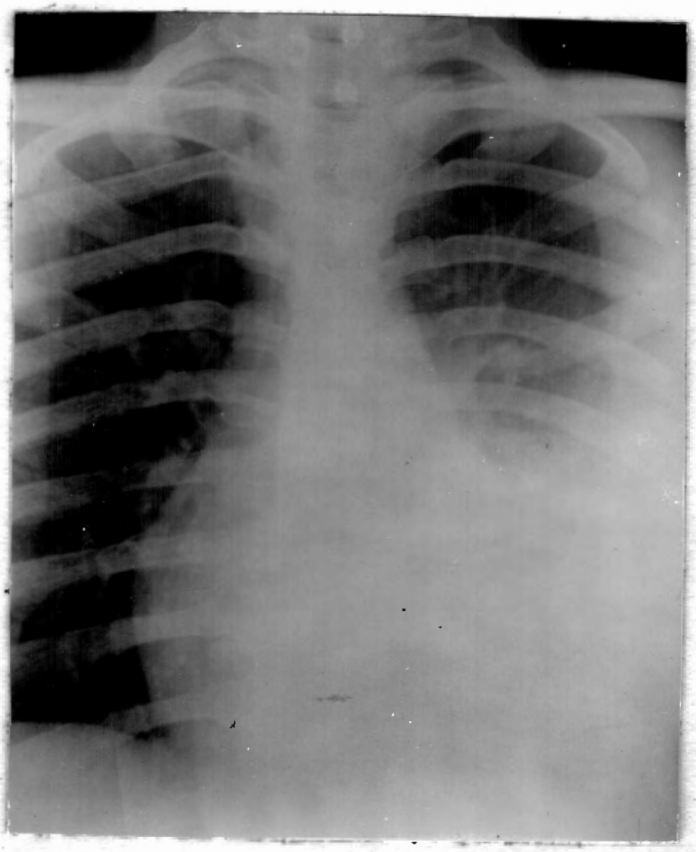


FIG. 3 - 5-3-49

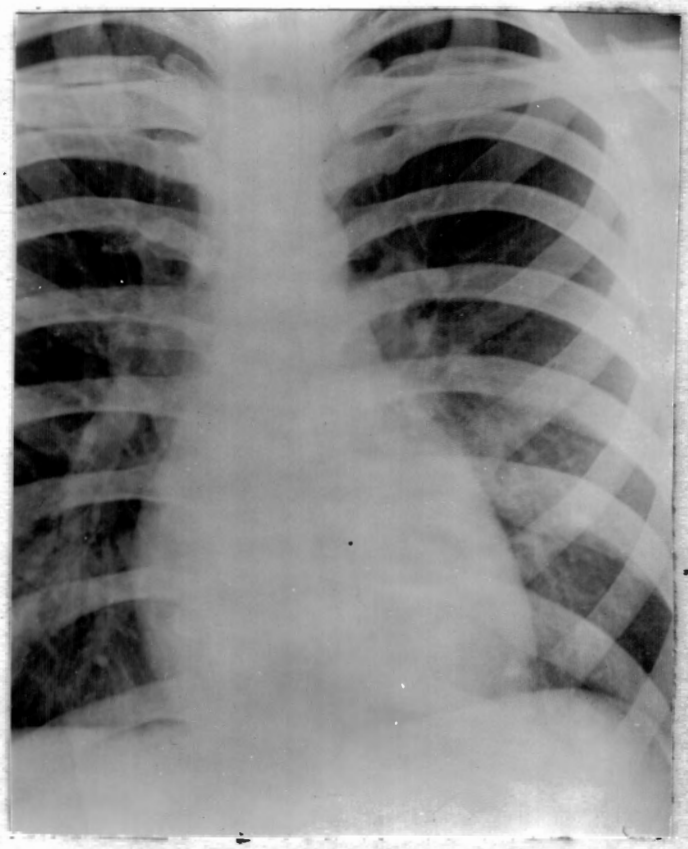


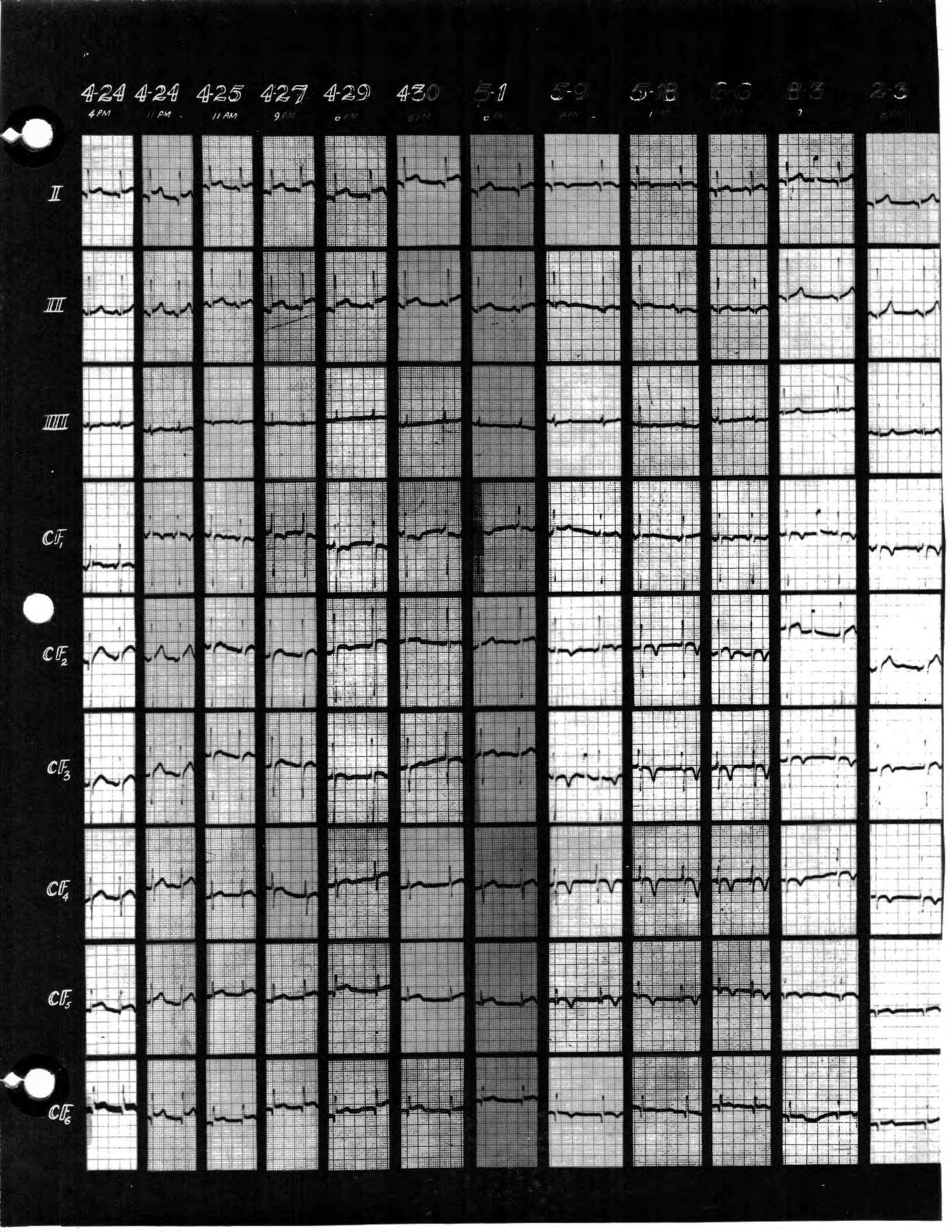
FIG. 4 - 5-21-49

The accompanying graph represents twelve of the fifteen electrocardiograms taken on the case presented.

The electrocardiograms cover a period of time from April 24, 1949 through February 3, 1950. In each case three standard leads and six (CF_1-CF_6) precordial leads were obtained.

Tracings of April 26, April 28, and June 29, 1949 were omitted because of similarity to other tracings or lack of significant changes.

See context for description and discussion of the electrocardiographic changes.


4-24 4PM
4-24 11 PM
4-25 11 AM
4-27 9 PM
4-29 6 PM
4-30 6 PM
5-1 6 PM
5-9 8 PM
5-18 1 PM
6-6 1 PM
8-3 9
2-3 2 PM
II
III
IIIIII
CF1
CF2
CF3
CF4
CF5
CF6

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