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Coronary thrombosis

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Senior Thesis On

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This paper is to be on coronary thrombosis but since this condition is considered a phase of coronary disease, therefore closely related to angina pectoris, I shall not attempt to draw a sharp dividing line and confine myself solely to coronary thrombosis. Neither shall I, thruout this paper, have breaks in it stating that here the introduction begins and here it ends. Symptoms are on page fifteen and treatments are on page twenty-two. For personally I would read a paper where the whole thing reads as a story and is not broken into integral parts. It seems to me that this method furnishes the reader with a more complete picture of the disease.

(6) Coronary disease is one of striking interest to the student because of its spectacular onset and because many famous men have died of it. John Hunter, Charcot and others. In nineteen hundred and twenty seven, one hundred and twenty five physicians died of angina pectoris, eighty seven died of coronary thrombosis. When we read statements like these your enthusiam like mine should be aroused to study more about the disease.

Probably one of the first thoughts that runs thru the physicians or medical students minds, when coronary disease is mentioned is why that is what John Hunter died of, and of course all of us are acquainted in some way with the

numerous discoveries that this great man made. He is themman who also innoculated himself with gonorrhoea andssyphilus so as to better learn the symptoms and treatment. (I2) He died at sixty years of age and it is now recognized that instead of ha having repeated attacks of angina pectoris. He suffered from coronary occlusion as well as angina pectoris and cerebral arteriosclerosis.

Since the disease is so closely allied in our minds to the name of John Hunter. It can hardly be amiss at this time to give John Hunters classical and graphic description of coronary occlusion. This should be of interest not only from a historical standpoint, but also to give us at first hand, from a keene observer, a good description of what constitutes an attack. (I2) The first attack assailed him in the spring of I773.

Sir Evarard Home records it as follows. Having met with something which very forcibly affected his mind, he was attacked at ten o clock in the fore-noon with pain in the stomach, about the pylorous; it was the sensation peculiar to those parts and became so violent that he tried change of position to procure ease; he sat down, then walked, laid himself down on the carpet, then upon chairs, but could find no releif. He took a spoonful of tincture of rhubard with thirty drops of laudanum, without the slightest benefit. While he

was walking about the room, he cast his eyes on the looking glass, and observed his countenance to be pale, his lips white, giving the appearance of a dead man; this alarmed him, and led him to feel for his pulse; but he founed none in either arm. Several physicians of his acquaintance were then sent for: Dr. William Hunter, Sir. George Baker, Dr. Huch Saunders, and Sir. William Fordyce. All came, but could find no pulse; the pain still continued and he found himself at times not breathing. Being afraid of death soon taking place if he did not breath, he produced the voluntary act of breathing, by working his lungs by the power of will. In this state he continued for three quarters of an hour, in which time frequent attempts were made to feel the pulse, but in vain; however at last, the pain lessened, and the pulse returned, although at first but faintly and the involuntary breathing began to take place. While in this state, he took Maderia, brandy, ginger ecte, but did not believe, them of any service, as the return to health was very gradual. In two hours he was perfectly recovered.

The next illness of importance came about three years later and appears to have been an inflammation in the arteries of the brain, accompanied by the feeling of being suspened in the air and by the sensation that the room was going around. On its subsidence vomited and felt relieved. These symptoms recurred the following day with sensations of sight, hearing, smell and taste extremely acute or heightened. His pulse was

generally about sixty, and weak, and a small degree of heat on the skin, especially on the hands and feet. He remained in this state about twelve days. The ataxia persisted during the convalescent period.

In I785 an attack of gout ushered in the third serious illness and was followed by a sensation of the muscles of the nose being in action, attended with an unpleasant sensation in the left side of the face, lower jaw, throat and down the left arm as low as the ball of the thumb. After these had continued for a fortnight they extended to the sternum. Giving the feel of the sternum being drawn backwards toward the spine as well as that of oppression in breathing; attthese times the heart seemed to miss a stroke and upon feeling the pulse the artery was very much contracted, often hardly to be felt. He next was seized with a pain in the region of the heart itself; and last of all with a sensation in the left side, nearly in the seat of the great end of the stomach, attended with considerable erucations of wind from the viscus. Several weeks later the most violent attack came which quite exhausted him and he sank into a swoon or doze to awake with confusion in his head which went off in a few days.

In December, I789, he suffered a total loss of memory with complete recovery within a half hour. This was followed in two weeks by giddiness and ataxia objects had lost their true direction, a perpendicular for instance seemed to lean to the left

and objects were also smaller than the natural recollection of them and appeared to be at an unusual distance. His recovery from this indisposition was less perfect then from any of the others. He never lost entirely the oblique vision, his memory was in some respects, evidently impaired, and the spasms became more constant. He never went to bed, without their being brought on by the act of undressing himself. The least ertion in conversation after dinner was attended by them.

An attack in October, I792 was so violent that Home thought he would not live. The final attack which caused his instant death occured at St. Georges Hospital on Oct. I6.- I793 when Hunter angered at a meeting, restrained his feeling and left the room to drop over dead.

(23) It is in only the last twenty-three years that this condition has been recognized clinically and diagnosed ante-morten. Previous to that time it was only diagnosed at autopsy and clinically was not differentiated from angina pectoris or allied conditions. We find that tha writings of such great men as Sir. William Osler and Sir. James Mac. Kenzie failed to differentiate coronary thrombosis as a clinical entity from angina pectoris. Huchard after a study of one hundred and eighty-five fatal cases of angina pectoris concluded that the coronary artery was quite important and mentioned the frequency that coronary

thrombosis occured, ;; Yet he did not describe symptoms such as would allow diagnosing before death.

Dock was one of the first to report a case and diagnosed and proven at autopsy. (23) Obratzou and Strascheska probably gave the first satisfactory description of the clinical symptoms of the disease. These men were Russians and diagnosed correctly two of the three cases they published. They emphasized the importance of a triad of symptoms, dyspnoea, severe lasting retro-sternal pain, orthopnea and gastralgia. They also thought that coronary thrombosis was preceded by angina pectoris. They called attention to other findings such as gallop rhythm, cheyne stokes breathing, pericardial friction, distant heart sounds, mural thombosis and pale cyanosis.

American physicians did not pay much attention to coronary thrombosis until just in the last twenty years. During that time they have contributed a good deal to the clinical description of the disease.

Herrick in 1912 emphasized that coronary thrombosis was a clinical entity and could be recognized in life and it need not end fatally. Following Herricks publications interest warmed for five or six years. Then Herrick again published further data on the subject. That same year (23) Levine and Tranter published a report of two cases one of which was diagnosed ante-mortem. Libman in 1916 mentioned as aids in diagnosing a recent thrombosis the fact that there was often the development of a slight temper-

ature, a moderature leucocytosis and evidence of apatch of pericarditus all coming on within a couple of days following an attack of severe pain. Levine and Tranter next described cases wherein the disease when epigastric pain was predominant symptom along with leucocytosis and fever might simulate an acute abdomen thus causing a mistaken diagnosis and possibly an abdominal operation. (7) Dr. Blanton P. Seward described a similar case. (23) Following these publications extensive literature appeared on the subject. The more important articles were by Graham, Paullin, Levine, Longcope, Thayer, Wearn, Gordinier, Herman, Benson, Wolf and White. These men observed and recorded the symptons so that coronary thrombosis is now rather easily recognized. They brought additional symptons and signs such as the frequency of a pericardial friction rub, a marked diminution of the height of the waves in the electrocardiograms.

Levine noticed that patients with hypertension and angina pectoris before the attack of coronary thrombosis might become free from anginal attacks if recovery was attended by a permanent and distinct lowering of the blood pressure..

In spite of all the american literature at this time on the subject the English and French did not publish the same s sort of descriptions until I925. Smith and Herrick in this country next described some important changes in the"T" wave of the electro cardiogram which occured persistently in cases of coronary thrombosis. These changes were important?,,because they often served as

the only means of differentiating coronary thrombosis from gall stone colic; acute gastric ulcer, or some other abdominal condition. It was mentioned at this time that the "T" wave had a high take off from the "R" wave and it went thru rapid changes on succeeding days following a thrombosis. The "T" wave was found to be often sharply inverted.

(4) Under annatomy we must first consider that the coronaries are no longer regarded as end arteries on account of their numerous commumications, so it has been removed from the category of hopeless disease and placed in a more manageable rank. In early life there is thought to not be much difference between the right and left coronary distribution. With advancing years the left coronary gains the ascendency in number and distribution of its branches. The conductive system as well as the heart muscle depends upon the proper function of the coronary arteries for its nutrition, therefore any disturbance of the coronary arteries supplying the conductive system of the heart will impair its function. This will very frequently cause an alteration of the physiologic electrocardiogram. (9) Kugel describes a large anastomatic vessel which runs in the auricular walls and links up the left and right coronary arteries. This vessel is constant in its occurence, thought subject to variations in course. It supplies branches to the aortic cusp of the mitral valve, to the aortic valve, to the commissures and to the base of the aorta. Its intimate relationship with these structures suggests that it may play

a part in the pathogenesis of mitral and aortic lesions, commissural lesions, and possibly lesions at the root of the aorta.

As we know the pressure in these coronary vessels is maintained by the systolic and diastolic pressures.(I3) But as to the relative importance of the diastolic and systolic pressures there is a great difference of opinion. Anrep and King made experiments on heart-lung preparations. From these experiments they concluded that the coronary blood flow is not dependent on either the systolic or diastolic pressures singly. But it does closely follow the changes in the arithmetical average of the systolic and diastolic aortic pressures. They also found that changes in the systemic output and changes of the heart rate have within wide limits no direct effect upon the coronary blood. flow.

There are many views given as to the etiology of coronary thrombosis. (8) Don C. Sutton and W. W. King with their experiments on dogs found that compression of the coronary vessels and immediately adjacents tissues caused pain without exception, sometimes salivation, vomiting in one case, a disturbance of respiration, acute dilatation of the heart and various changes in the electrocardiogram, so we may conclude that whatever will cause a constriction must be an important etiological factor i. e. arteriosclerosis. (I6) Starling thinks angina, a symptom complex due to reflex nervous causes. Coronary disease is the same only a

difference in the amount of pain. (I4) Herrick states in discussing etiology of coronary thrombosis, we wonder does infection play a leading part many authors think not. He believes that recen ts infections may cause an acute arteritus or a former infection may perhaps sensitize the vessel wall. In either case it is suggest ed thrombosis may be favored. Are some of the cases encountered to-day of the crop that was sown by the influenza pandemic of I9I8 Why is thrombosis rare in rheumatic hearts though rheumatic mural changes in the vessels are not unknown; perhaps not unusual? Why does syphilus spare the main coronary trunks? What of thrombosis in the veins of the heart? Herrick suggests the whole problem needs further study. Is there more coronary thrombosis to-day or was it just unrecognized many years ago. Possibly because people live longer in our present days therefore more of them live to develop arterial changes.

The essential lesion seems to be a narrowing of the vessel due to disease of the arterial wall that is generally rough from calcification, Marie said that coronary thrombosis always is due to a disease of the artery, "It"is never spontaneous"; yet other factors may enter. The sluggishness of the blood current; qualitative quantitative, physical and chemical changes in the blood; variations in its viscosity or its contents of albumen, alterations in the number of platelets or their fragility, whether changes in the food, in the air we breathe, especially in our large cities with their automobile and factory fumes may have an influence, is a

matter of surmise. So far no definite facts along this line are known.

The question is sometimes asked, why are the coronaries oftener the seat of arteriosclerosis and thrombosis then other arteries in the body. In answer,it is said that circulatory conditions in the heart are unique. The ceaseless motion of the coronaries, their constant subjection to extreme active and passive stress and pressure, their unusual kinks and tortuosities may help to explain a striking development of sclerosis with consequent thrombosis. Yet it is not clear why an artery that is almost never quiet should so readily develop thrombosis. These are all interesting speculations.

It has been established that painless attacks may occur. Normally certain areas of the heart are not only less vital than others, indifferent or silent they heve bean called, but also less sensitive. At autopsy fresh infarcts are sometimes found associated with multiple areas of fibrosis that speak for previous obstruction of small branches, yet no pain has bean noted, no pain even announcing the recent infarction. There has evidently been a very gradual and progressive narrowing of the artery by sclerotic processes. The area irrigated by the artery has become relatively inactive, relatively anesthetized by destruction of vessels, nerves and functioning muscles, so that a painful response to the new obstruction is lacking. The final complete obstruction comes with a sudden shock, the element of

surprise is lacking as the heart is in a sense prepared for the supreme insult. Abrupt heart failure with its dyspnea and other phenomena may be present, but pain may be lacking. These are the "substitution symptoms" in the hypersensitive described by Libman. As Obratzow and Strascesko expressed it, dyspnea may be the pain equivalent.

(23) Levine states as to etiology that no particular disease precedes. There is not evidence that it is due to infection in fact the converse seems to be true, as it is often found in patients that have had very few of the common illnesses. Angina pectoris usually ante-dates an attack although sometimes the angina has been of such a mild nature that it has not been recognized as such and the history was only brought out by close questionong. But cases of coronary thrombosis do exist in which a history of preceding angina is not elicted.

Hypertension is probably the closest related etiological factor to coronary thrombosis. Diabetis is second to this. True diabetis may occur in the history of probably seven percent. Glycosuria occurs in probably twenty- three percent. Sugar may occur more frequently in the urine during attacks because of the great pain and fear. This could possibly be called a transient emotional glycosuria which generally disappears a few days following the attack unless a true diabetis existed previously

In the cases studied by Levine those showing a history of diabetis did not develop coronary thrombosis at any

earlier age then others, the average age being fifty-eight and one-tenth years. Iwwould expect diabetis to influence attacks of coronary thrombosis earlier in life and also to influence the prognosis making it more unfavorable but it did neither in the cases studied.

Insulin has increased the life of diabetics to such an extent that in living longer they are more likely to develop vascular disease. Vascular disease more a disease in older life. So our problem in the diabetic besides just treating his diabetis becomes one of anticipating vascular disease and treating accordingly. Altho diabetis did cause the development of coronary thrombosis earlier in life nor did it influence the prognosis materially, it does indicate the type of person having a vulnerable vascular system.

As has already been stated hypertension is probably the most commom etiological factor altho there are cases wherein a pre-existing hypertension cannot be proven so it is thought to not be a necessary pre-requisite. Often times during an attack the blood pressure is sub-normal so if the patient does not know of a previous hypertension it cannot be said to have existed unless there is retinal sclerosis or the blood pressure rises again later, showing that it probably previously was high.

In fifty-eight cases studied by Levine THe average systolic was one hundred and ninety-one Mm. The average diastolic

one hundred and ten Mm. The average age fifty-eight and five-tenths years. Thirty nine males and nineteen females. This is a larger proportion of females than in the total group studied. This shows that a previous hypertension occurs more in the female although the disease itself occurs more often in the male. Hypertension did not effect the immediate prognosis. It was definitely known that only six of all the cases Levine studied, (I45) had a normal blood pressure previous to the attack although others may have had. There was other evidence of arterial disease such as histories of intermittent claudication and occassionally occurence of hemiplegia. Syphilus as a causative factor can scarcely be considered as it occured in only three of eighty-nine cases in which a Wasserman was taken. But the average age of occurence in these three, was fourty-five years; showing that it maybe a factor wherein early attacks may occur.

Rheumatic fever seems to be of no importance as an etiological factor for only three of the cases studied gave a history of rheumatic fever. It occured 33-49-and 60 years previous. Post-mortem examination showed no disease of the valves. Cases with coronary disease rarely give a history of rheumatic fever. It is true also that rheumatic fever cases rarely develop coronary disease.

Contrary to this, Slater (I7) cites three cases to illustrates the point wherein the occurence of acute coronary closures during a very active rheumatic infection. Closures

occured during the active stage of the infection for the reason that the larger branches of the coronary system was involved by the rheumatic virus.

Levine seems to think that hereditery and a physiological constitutional diathesis may be a factor in the etiology of early vascular disease and coronary thrombosis. In his case studied he found that rather frequently the patient gave a strong family history of coronary disease also the patients were commonly apparently healthy, well built, strong, stocky, slightly overweight individuals. Coronary thrombosis was seldom fond in a slender individual.

Extreme mental exertion was thought to be only a minor factor in etiology.Hard physical labor seemed to be a stronger etiological factor in coronary disease. Almost three times as many men had coronary thrombosis as women in the group studied by Levine. Why there should be this difference in the sex incidence has never been determined. It might be because the men are engaged in more strenous physical labor, or because they use tobacco, but there is no proof that either of these is a factor.

(I8) Wm. D. Stroud, Philadelphia beleives in hereditery as a big factor. He cites several family histories showing how succeeding histories of coronary disease developed in the same families. He believes mental strain to also be a big factor because the placid, imperturable Chineese almost never have it.

In a family with a history of the degenerative

types of cardio-vascular disease as the usual cause of death, an individual is born with a hyper irritable vaso moter system i. e. a "spasmogenic aptitude" This individual is exposed to the necessary environment which may consist of:

(I) Frequent emotional upsets.

(2) Infections (tonsillar) dental or other strept. infections.

(3) End products of improper diet.

(4) Insufficient intestinal and renal elimination.

(5) Excess of coffee, tea ecte.

(6) Obesity or diabetis.

(7) Excessive or prolonged physical effort.

(8) Long hours of nervous tension and mental concentration and inadequate vacations. Coronary sclerosis develops followed by coronary occlusion.

(I9) Then Dr. H.W. Ratke sums up the etiological evidence well by stating that the etiology of coronary disease is as yet not definitely known.

In discussing the symptoms and coronary thrombosis I believe it is well to first discuss those of angina pectoris, because it is usually the preceding symptom complex.

(5) The primary symptoms of angina are a sudden onset of pain of varying degrees, usually related to effort or taking more rarely occuring in sleep is characteristically substernal most frequently under the upper third rarely under the lower

portion of the sternum; is not pre-cordial as loosely stated by many. Radiation may be to left arm and shoulder, left side of neck, jaw, less often to right arm or both arms. The attack may subside suddenly or slowly leaving the parts hyperesthetic for a day or two. John Hunter spoke of the sensation as though the breast bone was being crushed, with pain in the throat, face, left side of jaw, and inability to swallow during the attacks. A peculiar dread or fear, often a calamitous sensation of impending death.

Secondary or incidental symptoms, (A) various vaso motor phenomena, salivation, polyuria, sweats, pallor.

(B) More or less gaseous eructation and distention.

(C) Lessened systolic output, judged by the quality of the pulse, arrythmic.

(D) Sudden death.

Some cases may be so mild as to scarcely arrest attention.

(23) In going in to see a patient during an acute attack of coronary thrombosis. We are likely to get first a history of antecedent attacks of angina pectoris. The patient usually is in extreme pain. His chest feels like it is in a vise and he may be beating at it to get the pain out. He usually gives the appearance of one in extreme shock. He will be pale, is cold and the skin has a moist ashen gray color. The attack may have come on while he was perfectly

quiet. It doesen't have to follow exertion as in angina pectoris. The patient may have been asleep, eating a meal or resting in a chair. It usually comes on suddenly and the patient knows that it is different from anything experienced previously. If a sufferer of angina he knows this is different. Oftentimesthe attack is initiated with vomiting and pain in the abdomen then the case is frequently called one of acute indigestion. The blood pressure is usually found to have dropped. In this it differs from angina attacks where the blood pressure rises. The heart is rapid, but the pulse is small. The first heart sound is often inaudible at the apex. The second is faint. There is often rales at the base of the lungs. The liver soon becomes palpable and tender. There is often cheyne-stokes breathing usually there is a rectal temperature and a slight leucocytosis. A pericardial friction rub is heard in about 20% of the cases. This is thought due to the formation of an infarct in the area blocked off by the thrombosed artery. This infarct involves the pericardium so that a friction rub is produced. The necrosis may extend towards the endocardium and cause a local mural thrombus. If the case is uncomplicated the patient may slowly return to normal. His blood pressure even though high previous to the attack may remain low afterwards. He may also now be free of future angina attacks. The rhythm of the heart is often regular there may be a systolic murmur. The heart sounds are almost always muffled or distant. There may be a gallop rhythm. The most

important of the heart findings are the extreme muffling of the heart sounds, gallop rhythm, pulsus alternans and pericardial friction rub. These findings are an aid in diagnosis.

Patients later may develop edema of the legs and free fluid in the abdomen. In a very few cases the attack may not be accompanied by pain but by extreme dyspnea. A diagnosis in a typical case such as this has to be made on the other findings.

The pain in the typical attacks has been described as feeling like the chest is in a vise. Others describe it as a feeling like a ton of bricks has hit the chest. Feeling of a terrible pressure on the chest or the chest feels as though it was in a death grip. The pain gradually lets up over a period of ½ hours or days. In some, when the pain disappears a dull ache remains. The pain may occur intermittently for a period of a week. But all attacks last longer than an attack of angina. The pain radiates the same course as angina. It most commonly appears first between the two nipples. As has been mentioned cases may simulate an acute abdomen, gall bladder colic, perforated ulcer ecte. Symptoms are excruciating pain in the abdomen, nausea, vomiting, slight jaundice, fever and leucocytosis.

Fever and leucocytosis usually comes soon after the attack. The fever may be missed if the temperature isn't taken rectally. The patient may feel moist and cold and mouth temperature only 96-97 while the rectal will be 100-101.

The fever and leucocytosis seem to depend on the amount of infarctionpresent. It lasts from one to two weeks. The infarcted tissue probably liberates toxic products causing the leucocytosis and fever. The presence of leucocytosis is one of the most findings in coronary thrombosis.

The urine during an acute attack may give the findings of an acute nephritus, albumen and casts. Sugar commonly appears in the urine at this time and may confuse the case with a diabetic acidosis or pancreatitus.

It is surprising that coronary thrombosis almost never develops in hearts previously having a persistent auricular fibrillation. The usual history is one of a normal heart rhythm, therefore it is practically safe to predict that a patient sick with chronic myocarditus or auricular fibrillation will not develop a coronary thrombosis. Neither will they develop angina pectoris.

Altho the rhythm of the heart is normal to begin with, and remains so essentially during attacks of angina pectoris, it is quite otherwise when coronary thrombosis developes. With coronary thrombosis most any cardiac irregularity may be found. Premature beats are frequently found during any acute attack but since these often occur in normal individuals they are of no significance here. All sorts of disturbances of conduction between the auricles and ventricles may occur such as partial

or complete heart block, dropped beats ecte. Paroxysms of auricular fibrillation sometimes occur during the first few days of the attack. Ventricular tachycardia frequently appears at this time. Usually if auricular fibrillation does appear it soon clears up whether treatment is instituted or not. Paroxysmal tachycardia is more often associated with grave coronary thrombosis. The heart suddenly becomes rapid with a rate of about 150-200 and remains essentially regular. This differs from auricular fibrillation in that the heart altho fast remains absolutely regular, in auricular fibrillation it is very irregular and fast. To further differentiate ventricular flutter from auricular flutter or fibrillation we may say that the former is not influenced by pressure on the vagi nerves while the latter are. Also in ventricular tachycardia slight irregularities in the lenght of the heart beat and a varying intensity of the first sound may be heard at the apex. A curious clicking or reduplication sound may be heard with the various cycles. Gallop rhythm is another form of disturbed rhythm often found in the acute stages of coronary thrombosis. But since this rhythm is also associated with other conditions little significance can be attached to it here except the fact that it is additional evidence.(23)

(23) As to prognosis and modes of death, it is difficult to say whether the patient will die suddenly in an attack, will last a few hours, days,weeks, or recovers. There is no way

of determining the outcome. Some cases of anginas may be walking the streets and suddenly drop dead from a sudden coronary thrombosis. Sudden fibrillation of the ventricles during an attack may prove fatal. Complete heart block or general failure of the circulation may kill the patient. The shock and lowered blood pressure may enfeeble the circulation soas to cause death. The patient may survive until the fifth or tenth day and then suddenly die due to rupture of the infarcted area of the heart. Peripheral embolic and mural emboli of the heart may be the cause of death. One group of patients may recover from the acute attack but shortly after that temporary improvement a progressive failure of the circulation of the congestive type occurs. The heart may become quite irregular, the patient develop increasing dyspnea,peripheral edema, cheyne stokes breathing and other evidences of circulatory insufficiency.

Even in these instances on proper treatment the patient may recover for a few months before death or he may actually become ambulatory and carry on bylimited activity for several years.

There are several types of recovery. The ideal one is when the patient, who has angina pectoris is hit by coronary thrombosis which causes his blood pressure to drop and it remains down. If his blood pressure remains down and he recovers the possibilities are that he will no longer suffer from attacks of angina therefore appears to be in better health then before. It is a good omen for the blood pressure to drop if it does not return to its former high

level.

Another group that recovers is the one where the patient shows definite signs of heart failure that he never had before. Ther is increasing dyspnea, enlarged liver, cheyne stokes breathing. This gets progressively worse until the patient dies. These symptoms when present a few weeks following the attack are most ominous. Cheyne stokes breathing may occur during the acute stage in the first few days and the patient yet recover and fall into the first group.

(2I) Dr. Howard Wakefield tells of cases which he calls the coronary origin of certain cases of angina pectoris. Previous to a patients coronary accident they could walk, run get excited, and apparently no matter of exertion would bring on distres in their chest. Some day the patient is brought down with a coronary accident which may be either mild or severe; he is laid upb for a variable time. If he survives the initial attack as many patients do, every time he exerts himself he has the distress of angina of effort.

(23) Electrocardiograohic changes of coronary ~~th~~ thrombosis may easily enough be diagnosed now from the available bedside data but additional evidence may be gathered with the electrocardiograph. It has been noted that in cases of coronary thrombosis the electrocardiograph showsdiminution in the amplitude of the waves. There is a difference of the interval between the

q. r. s. complex and the "T" wave . This is shown early in the attacks. The "T" wave comes directly off of the "R" wave on the down stroke. This may show in one of the leads, while in another lead there may be a low take off of the "T" wave from the up stroke of the "S" wave before it has returned to the base line. When these changes are marked they are practically pathogmonic of coronary thrombosis. In electrocardiography of rheumatic fever the q. r. s. and t. intervals may be missing, but the "T" wave does not come off as high or as low as in coronary thrombosis. uremia and pneumonia may show a similar one but here the distinction is that in coronary thrombosis the "T" wave has a peculiar rounded hump. These changes do not appear in all cases, so their absence does not rule out coronary thrombosis. Another feature is the fact that if electrocardiograms are taken daily they will be found to vary. This is not true of any other condition. These changes are sucessive so that if one sees a picture of a recovering stage he may predict that there was coronary thrombosis and it occured back about so long ago.

IN making a differential diagnosis it is important to first differentiate a speudo angina then a true angina. (22) In pseudo angina annatomic defects do not exist in the heart or aorta, whereas in true angina pectoris lesions exist to a recognizable degree. Practically all cases of pseudo angina

may be grouped under the heading of psychic and emotional phenomena. They are tremendously frequent, particularly now that the laity has become so conversant with medical or pseudo medical literature and because of the agitation which we ourselves have started for the purpose of acquainting the public with medicals matters. There are usually three cases of pseudo angina seen to one true one. These are usually easily differentiated for those with the spurious attack are obviously neurotics. If the physician can see an attack and carefully observe it mistakes will seldom be made because angina presents a typical picture.(I8) (I8) The following table will help to differentiate true angina from coronary thrombosis

Angina	Coronary Thrombosis.
onset----during exertion.	Often during sleep.
Site of pain --Sternum, mid-sternum	Sternum,often lower $\frac{I}{3}$ or epigastrium.
Attitude---immobile	Restive, even walks around.
Duration---minutes	Hours or days.
Shock---absent	Present.
Dyspnea---absent.	Often present.
Vomiting---rare	Common.
Pulse---unchanged.	Small often rapid and irregular.
Temp.---none.	Fever follows.
Leuk.---normal	Leucocytosis.
B. P.---normal or rise.	Fall.
Heart sounds---normal.	Distant sometimes gallop or pericardial rub.
Congestive failure---absent.	Commonly follows.
Electrocardiograph---often abnormal.	Often diagnostic.

(23) Levine states that in differential it is most important to differentiate from angina pectoris. The principal difference being in the amount and degree of pain. Pain in angina pectoris lasts only minutes and is not as severe, while that of coronary thrombosis lasts hours or days. Nitro glycerine or any of the nitrites usually give relief in angina. While in coronary thrombosis morphine may not even relieve it. Other events charactering coronary thrombosis are usually a gradual fall in blood pressure, an increase in heart rate, a leucocytosis, a fever, various irregularities of the heart, occasionally a pericarditus, embolic phenomena, evidence of shock, collapse and congestive failure, and the development of dyspnea and gastric symptoms occuring at times.

There are several other conditions that may simulate coronary thrombosis. A small group of patients may give signs of an acute abdomen. In all patients above fourty years with an acute abdomen the possibility of a coronary thrombosis should be considered. The distinguishing features have already been set forth in the above. At times suspicious findings on auscultating the heart or an electrocardiograph may be the thing which saves the patient from an abdominal operation. (7) Dr. Blanton P. Seward describes one of these typical abdominal cases and emphasizes the importance of recognizing them.

If there is a previous history of angina and along with a predominating pain in the abdomen there is also

a feeling of constriction in the sternum or a squeezing ache in the arms or the presence of some dyspnea during the attack may lead to a correct diagnosis of coronary thrombosis.

Pneumonia, diabetic acidosis and pneumothorax are other conditions that may be confused. To differentiate from pneumonia may be difficult because in both conditions we may find rales in the bases of the lungs, leucocytosis, fever, pain in the chest and cough. But the heart is seldom effected in pneumonia. The pain in coronary thrombosis is behind the sternum and is constricting. That of pneumonia is usually laterally and is worse on breathing.

Coronary thrombosis may be mistaken for diabetic coma because sugar often occurs in the urine following an attack, there is also shock and stupor. History should help rule this out. Also the presence of chest pain is unlikely in a true diabetic.

Some cases of coronary thrombosis may be so mild that the first evidence is hemiplegia. Whenever a hemiplegia occurs with a low blood pressure the possibility of a breaking off of a ventricular mural thrombus following coronary thrombosis and its lodging in the brain must be thought of.

Once in a great while there is a case of coronary thrombosis occuring without pain. Then it can only be determined

by the increasing dyspnea which is far out of proportion to the heart findings. A drop in the blood pressure and evidence from the electrocardiogram.

Prognosis: very difficult to predict. Sometimes patients with the most terrific attacks recover while those with mild ones apparently recovering die on the fifthor seventh day. So a guarded prognosis should always be given. In one series of cases that were studied the patients had an even chance to recover fifty per-cent lived. The average duration of life after recover was two years. The younger the patient the better his prognosis. Sex doesn't seem to alter the prognosis. Some few patients may recover and live as long as eight or ten years after most terrifying attacks. Previous hypertension or diabetis doesn't seem to alter the prognosis. The character and severity of the pain seems to be a poor guide to prognosis.

Treatment: must be based on principles only because the disease has not been recognized long enough for accurate observations over a long period of time to be made. Treatment then must be based on the known pathological physiology that occurs.

First and primarily we must treat the terrific pain that usually initiates an attack. Nitroglycerine which was probably formerly used will usually be useless now. Best to give large doses of morphine subcutaneously. First dose should be not

less then a quarter of a grain useless to give it by mouth. Give morphine as needed to control the pain. It should relieve it but often a dull sense of oppression in the chest remains. Yet with this the patient should be able to go to sleep. If at a late hour the pain still persists give more morphine. If it then has subsided but the patient is nervous and restless and respirations are not particularly depressed it may well to give even more morphine.

Altho the patient is in shock it is better not to stimulate the circulation unless the blood pressure is below a hundred or the pulse is barely perceptible then it is well to give large doses of caffeine sodium benzoate, strophanthin or adrenaline intramuscularly. Avoid stimulants as much as possible usually morphine is all that is necessary. Keep the patient covered with blankets warm.

(I) Richard Bodo has experimented on heart-lung preparations using different drugs for their effect on the coronary circulation. He finds that the digitalis group, caffeine and camphor all dilate the coronaries. In order of their effect we can list (I) caffeine (2) digitalis group (3) camphor only a slight effect. Amyl nitrite and sodium nitrite also increase the coronary flow. They had no heart tonic effect. Quinidine produced brachycardia and heart dilatation.

(3) Dr. Renner, New York states that mustard stupes are useful for the pain.

(23) When we consider the pathology present it is easy to see why stimulation should be avoided unless very early in the attack. There is a block present in a coronary vessel because of this block a portion of the ventricle supplied by the vessel suffers an insufficient amount of blood. This poor nutrition to the heart muscle causes it to degenerate an form an infarct. This usually occurs several days after the initial onset. A stimulant used then would cause stronger contractions of the heart which might dislodge emboli from the infarct or cause the heart to rupture thru it.

The value of digitalis is questionable here. Most a authors seem to think it alright to use early, but not after infarction has had time to take place. If decompensation or auricular fibrillation developes several weeks after the onset it is well to use digitalis.

Adams Stokes syncope rarely occurs but when it does a life may be saved by the use of adrenaline, another rare but serious complication that may occur is ventricular tachycardia. Any small rapid heart in already enfeebled circulation is particularly dangerous. Ventricular tachycardia may be only transient lasting but a few hours or it may be very troublesome lasting over a period of days. Quinidine is the proper treatment

for this condition.

Oxygen may be quite beneficial where there is an evident anoxemia, cyanosis and edema of the lungs.

Good nursing care is essential, absolute rest, freedom from visitors, give fluids freely as the patient may become dessicated from the marked perspiration. Milk is the best food to give. May also give other liquids and soft solid nourishment. The morphine given is likely to cause constipation, but it is probably better not to force movement for fourty-eight hours; then use enemata. The patient should remain in bed at least six to eight weeks.

(3) Surgical intervention such as section of the nervous sympathetics or block anesthesia has been decided against because of the high mortality.

(2) In summarizing we may say that the outstanding symptom is the occurence of sub-sternal pain, usually severe, often radiating and characteristically persistent. The signs are those of myocardial insufficiency, frequently with a sudden and striking fall in blood pressure; a febrile movement and leucocytosis as the process of infarction progresses, in some instances a pathognomic friction rub as the area of myocardial degeneration emerges externally and involves the pericardium and more rarely embolic phenomena in the greater or lesser circulations if the extension of degeneration is inward with the formation of intra cardiac thrombi.

There is also in coronary thrombosis an upward convexity of the S-T portion of the electrocardiogram with a"T" wave inversion. Auriculo-ventricular heart block and fibrillation of the ventricles are other disorders of heart action which may follow the failure of adequate coronary bloodsupply. In treatment all evidence points to the fact that morphine is probably the best to relieve the pain, anxiety and keep the patient quiet absolute freedom from exertion over a period of weeks should be carried out. Nursing care is very important, a light diet not necessarily liquid should be fed. Distention should be gaurded against by the use of enemas. The use of digitalis is debatable. Probably better to withhold until the need of stimulation exceeds the need for rest.

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Coronary Thrombosis.

Case Report---------- Patient George Avery.

Disp. # 37043.

Petersburg Neb. Farmer Male, Age 60. I-22-27.
Comp. Pain over pre-cordium, dull aching and radiates to the shoulder and down the left arm. Pain appears upon exertion. First noticed nine months ago. Began first at base of neck radiating down to involve the whole pre-cordium, was advised to discontinue tobacco which he has done for the last two months, without much relief.

Childhood diseases: mumps, measles, other illness: influenza, appendicitus with perforation, small pox.
Operations: Appendix no injuries

Cardiac: palpation

Eye, Ear, Nose and Throat Neg. Resp. Neg.
Gastro int. Neg. Genito urinary Neg. Neuromuscular Neg.

Habits Chews tobacco, gaining wt. Family Hist. Father died of Brights disease, age 66. B. none M. D. 55? W L.&W.

Phy. Exam. Pulse 95 B. P. I70/I09 Fine tremor. Teeth bad, Tonsils injected. Chest- Lungs Neg. Heart enlarged. Abd. Neg. Reflexes Neg. Diag. Angina, Focal Infection.

℞
Sat. Sol. Na. I. oz.I/2
I0 Gtts. in H_2O T.I.D.
Amyl Nitrate pearls to relieve pain.

Jan. 22 Urinalysis Straw, Clear, I0I2 Alb. Neg. Sug. Neg. Diac. Neg. Micro. Mucous.

Jan. 22-27 Hemo. 92% R.B.C. 5,390,000 W.B.C. I3,800. Polys 79% Lympho. 2I% Jan. 22-27 Wass. Neg.

Jan. 26 Has had much distress. B.P.220/I30 Almost went out when he took amyl nitrate ℞. Nitro Glyc. Gr. I/I00 No. I5 P.R.N.

Jan. 29- Nitro Glyc. Tab. made patient have a severe headache. Has had no anginal attacks, but noticed skip beats and so took one of the tabs. Rest and ice bag over pre-cordium. B.P. I95/I30 Feb.I0, I932. Comp. Vision failing, especially in right eye. Headaches just above the eyes. Repeated attacks of dyspnea, sense off oppression pain in the chest, over the pre-cordial region and radiates to the pit of the stomach and down inner side of both arms. Not relieved by medicine but somewhat by heat. Eyes react to light Pupils small, Ant. chamber deep Conj. inj. poor vision. Referred to eye. Diag. Arterio Scleroti retinitus, rt. hemianopsia. Two or three weeks before Thanksgiving,had a stroke and after that had no idea of direction This remained for a period of about two weeks. Along about Christmas, eyes became worse. Feb. I3, I932. Wt. I89 T. 98 Pulse 80 irreg. R/. Tinct. Dig. Oz.I
60 min. to-day and then 30mn. B.I.D.

Feb. I7 Improved but still fibrillating.

Feb. I7 In to neurology. During the last year patient has had periods of profound depression. May come on daily or at larger intervals. Patient feels there is nothing left worth living for. Has had suicide thoughts, but has never tried to carry them out. Insomnia.

Feb. 20-I932. Continue with digitalis no ill effects still some edema of the ankles. Patient next moved out of town, so was lost sight of.