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Coronary thrombosis

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CORONARY THROMBOSIS

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Coronary thrombosis did not assume the importance of a clinical entity until the last decade, before that time it was considered as a very severe form of angina pectoris which resulted fatally and therefore of pathological interest only. The literature of the latter half of the 19th century contains many accounts of fatal angina pectoris where coronary thrombosis was found post-mortem. Yet it was not until 1910 that any real attempt was made to establish cardiac infarction as a clinical entity. Obratzow and Strachesko in this year described three cases, confirmed by necropsy, in which two cases had been diagnosed correctly during life. They appear to have been the first to establish coronary thrombosis as a clinical entity distinct from angina pectoris. Herrick (1) in 1912 described the syndrome quite accurately, and built up by means of autopsy control the clinical picture now recognized as due to interruption of the coronary circulation. Starting with the experimental work of Smith (2) (3) on coronary artery ligation in animals the effect of coronary thrombosis on electro-cardiograms has been studied by Herrick (4), and Pardee (5) first recognized a curve caracteristic of the acute stage

of myocardial infarction in man. Since the recognition of coronary thrombosis as a clinical entity a voluminous and valuable literature has grown from the writings of many men in an attempt to familiarize the profession with the various features of coronary thrombosis. (6) (7) (8)

The literature is filled with descriptions of the symptomatology and methods of diagnosis but until recently not much has been said concerning the etiology and prognosis. The earlier literature did not contain much in the way of treatment, (because it was the common belief that nearly all cases resulted fatally, Herrick (1) was the first to point out the fallacy of this belief), however, in the past few years many good articles have appeared in the literature concerning the management and treatment.

This short paper on coronary thrombosis will deal for the main part on etiology, prognosis and treatment. No attempt has been made to make this a statistical study. I realize that such a study is impossible with many cases of coronary thrombosis not reported or correctly diagnosed and no systematic study made of them. It is my aim to ascertain the thoughts of men especially interested in this disease on the subject of its etiology and prognosis, and method of treatment.

ETIOLOGY.

"There is at the present time no one specific disease

that is a frequent precursor or that is in any intimate way a causative factor in the production of coronary sclerosis and thrombosis." (8)

I. Age and Sex

Coronary thrombosis occurs with the greatest frequency during the latter years of life, chiefly between the ages of fifty and seventy. That coronary thrombosis is not confined to old age is shown by the review of the literature by Smith and Bartels (9) in which they found twenty-one cases of coronary thrombosis with occlusion in which the patients were less than forty, on which necropsies were performed, and nine cases of probable coronary thrombosis with occlusion in which the data were insufficient to allow a positive diagnosis. From this they conclude that "myocardial infarction resulting from coronary thrombosis is relatively rare if patients are less than forty years of age." In this series there is one patient aged 18 and two patients aged 12 years.

Ramsey and Crumrine (10) report a case of coronary thrombosis in an infant four months and eight days old, revealed by autopsy in descending branch of the left coronary artery. They note a case reported by Hughes and Perry (11) of coronary thrombosis in an infant seven weeks old.

It is generally conceded by all observers that coronary

thrombosis is much more frequent in males than in females, the ratio being 3 or 4 to 1. There is no explanation for this that is satisfactory. The greater amount of physical work performed by men has been cited as a factor. The role of tobacco has been greatly discussed but no definite proof that it is at fault is available. In the past the habit was confined chiefly to men but perhaps since women have taken it up the next generation will show the effect, if any, of tobacco on them in the production of coronary thrombosis. It is said that coronary thrombosis is found in a great many men who have been very active in sports and it is therefore probable that general physique and strength and activity of the male make them more prone to this disease than women.

The following table given by Bramwell (12) shows the age and sex incidence in three series studied:

	Bramwell	Levine	Parkinson and Bedford
Age			
Over 70	3	14	10
50-59	17	99	68
Under 50	4	32	22
Sex			
Man	18	111	93
Female	6	34	7
Total	24	145	100

II. Race seemingly plays a very small role if any.

III. The role of infection in the production of coronary thrombosis is generally conceded to be very slight especially

in large series of cases. (7) (8). Wilson in a late article says that it seems to play an important role.

Rheumatic infections are the most common specific cause of heart disease. In Levines (8) series he found "only three patients who had a past history of rheumatic fever and those attacks occurred 33, 49 and 60 years before death from infarct of the heart." Slater (13) reports three cases of coronary involvement in acute rheumatic fever.

IV. Relationship to Angina.

Most patients have had previous angina. Levine (8) states that the "great majority of patients had definite angina pectoris antidating the attack." Of Parkinson and Bedfords (7) series only 38% had had no previous angina.

White and Bland (14) says "coronary thrombosis is a frequent complication of angina pectoris."

White (15) gives the incidence of coronary thrombosis in angina pectoris as more than 26%.

Hubble (16) describes a prodromal angina which he says is more severe than simple angina and not so severe as that in occlusion but comes on more and more frequently and becomes more severe each time, furthermore the pain is relieved by the nitrites only for the period of their pharmacological action, then return with even greater severity. These attacks he interprets as a warning of impending occlusion.

In some cases in which angina has not been present before an attack it may be after recovery, i.e. an attack of coronary

thrombosis may initiate a typical course of angina pectoris and vice versa no further attacks of angina pectoris may be suffered after coronary thrombosis.

V. Diabetes.

The part played by diabetes in the development of coronary thrombosis has been commented upon by various writers. Levine (8) in his series of 145 cases found "34 or 23.7% in whom glycosuria was found, or where it was definitely known that diabetes had existed previously." He feels that diabetes is second only to hypertension as a distinct disease entity as an etiological factor in coronary thrombosis. He did not find that diabetes altered the age at which coronary thrombosis occurred nor did it affect the prognosis. Other writers, White (15) feels that there is probably no great relationship existing between the two conditions but that they are coincidental in this age group.

Parkinson and Bedford (7) found diabetes in 10% of their series of 287 cases.

Much has been said as to the relationship of diabetes to the production of arteriosclerosis but so far as I can find there is no definite evidence of this relationship. Fitz and Murphy (17) in their article find that "general arteriosclerosis is fairly common in diabetes and the coronary arteries are not infrequently involved." I believe that the

view of this matter to be held is that insofar as diabetes is an etiological factor in arteriosclerosis it is also an etiological factor in coronary thrombosis.

VI. Hypertension and Arteriosclerosis.

Levine (8) states, "A previously existing hypertension is probably the most common etiological factor in the development of coronary thrombosis."

Most observers make some comment similar to that of Levine on this subject, very few report a previous normal blood pressure in cases which have been seen before the attack. White and Bland (14) found hypertension in 50 of their 200 cases.

Hamman (18) stresses the frequency of the association of coronary occlusion and arteriosclerosis giving the figure as 90% of the cases. Other observers have also noted this frequency and at autopsy sclerosis of the coronary arteries has been found in a very large percentage of the cases. In a large number of individuals there was very little evidence of arterial disease except in the coronary arteries.

VII. Syphilis.

That syphilis plays some role in the production of coronary thrombosis is a definite fact but that that role is small is readily seen by reading the reports of the various series of case reports. Hamman (18) says that "it is an uncommon cause of coronary occlusion because syphilis causes

extensive disease at the root of the aorta but seldom invades the coronaries."

Warthin (19) concludes from his study of autopsy reports from 1909-1929 that:

1. "Active syphilitic lesions of the larger coronary branches are infrequent. They rarely produce occlusion of the vessel, or lead to thrombosis or myocardial infarction.

2. "Arteriosclerosis of the coronaries, coronary occlusion, coronary thrombosis, myocardial infarction and angina pectoris are more frequent in the latent syphilitic than in the non-syphilitic. Syphilis predisposes secondarily to coronary and aortic sclerosis and their resultant cardiac pathology."

VIII. Heredity.

Musser and Barton (20) in a review of the literature find more and more attention being paid to the hereditary factor in the production of coronary disease by other observers. They are inclined to think that heredity plays a large part in the causation of this condition, especially in the younger group of individuals.

Coombs (21) states "The familial incidence of angina pectoris is well known." He mentions a family of father and four children, all of whom died of coronary disease. He says in explaining this familial tendency, "there are two possible interpretations of this familial incidence. One is that it

is nothing more than an example of the well-known liability of certain families to arterial degeneration, a liability which is apt to be particular as well as general since it includes a predilection for certain vessels. For example, there are families whose members all die of cerebral hemorrhage. Another suggestion is that aberrations of the coronary arteries which, as Hadfield has pointed out, may throw an undue measure of responsibility on the one trunk, may run in families."

A great number of cases (22) (23) (24) have been reported in the literature calling attention to the frequency with which coronary thrombosis occurs in the same family so I believe that it is just to ascribe to heredity a large share of the blame in evaluating the etiological factors.

IX. Physical type, work and weight.

Levine (8) describes, "the typical patient is a well set person, somewhat over weight, often of considerable physical strength who enjoyed unusual good health. The patients often give a history of having been extremely active and having indulged vigorously in sports or their ordinary work. One is impressed by the fact that athletes seem to succumb to vascular disease at surprisingly early years of adult life."

There is a generally accepted belief that angina pectoris and coronary disease are peculiarly common to men

of intelectual habits and to men who have held positions of responsibility and importance. On the other hand it is said to be uncommon among laborers and the whole group of poor patients in the free dispensaries and wards. Hamman (18) attributes this difference to the mental ability of the patients to appreciate the sensations rather than the lack of the sensations in any class. He says, "Therefore, angina pectoris will always be a disease of the refined, intellectual classes, but the dramatic manifestations of coronary occlusion will occur equally often in all."

A consideration of the weight of individuals with coronary thrombosis shows according to Levine (8) "that coronary thrombosis does not commonly occur in thin people." Vascular disease is more frequent in fat people after 40 than in thin ones and Du Bray (25) states that coronary sclerosis is frequently associated with obesity.

The above being true one would conclude that obesity in some way favored the occurrence of coronary thrombosis.

Various other factors have been mentioned from time to time by various authors. Among them are embolism which everyone agrees is a rare cause. Thrombo-angiitis obliterans is another rare cause. Luten (26) in a recent article analyzes some various factors which he considers as contributing factors in coronary occlusion. He mentions among these vasoconstriction saying, "The evidence that the precipitating

cause both of angina and of thrombosis frequently is referable to the gastrointestinal tract is impressive. In case reports of both, references to indigestion and to the relationship of onset to the preceeding meal are conspicuous."

Hewlett (27) attributes "vagus slowing of the heart to reflexes that arise from the abdominal viscera."

Luten (26) reports that in two of his four cases evidence of occlusion immediately followed the drinking of cold fluid. Several experiments have been carried on to ascertain the effect of cold on the heart and especially the coronary circulation (28) and the electrocardiogram (29). In these it was found that the coronary circulation is diminished and a negative T wave is produced similar to that following ligation of the branches of the left coronary.

It is readily conceivable that this reflex vagus stimulation or direct stimulation of the heart by cold after eating may account for a slowing of the blood in the coronary circulation sufficient to form a thrombus on any sclerotic changes which might be present. The fall in blood pressure which is known to take place normally at night may account for the night or early morning attacks.

Symptomatology and Physical Findings

I will not attempt a special discussion of the various symptoms and findings in coronary thrombosis in this short

paper but will give an account of the typical case.

There are different clinical manifestations in coronary thrombosis varying from no discomfort whatever to sudden death depending on the size and location of the affected artery.

Herrick (30) grouped the cases of coronary thrombosis on the basis of the clinical symptoms as follows:

1. "Cases of instantaneous death, a group graphically described by Krehl, in which there is no death struggle, the heart beat and breathing stop at once.
2. "Cases of death within a few minutes or a few hours after the obstruction. These are the cases that are found dead or clearly in the death agony by the physician who is hastily summoned.
3. "Cases of severity in which, however death is delayed for several hours, days or months, or recovery occurs.
4. "A group that may be assumed to exist, embracing cases with mild symptoms, for example, a slight precordial pain ordinarily not recognized, due to obstruction in the smallest branches of the arteries."

The following is a description of the events occurring in a typical case of coronary thrombosis.

Prior to the onset of an attack the patient may have considered himself in excellent health, and as in many cases reported to have recently passed a rigid examination for

life insurance.

The onset of an attack of coronary thrombosis is usually abrupt and dramatic. There may or may not have been previous anginal attacks, the attack usually has no relationship to exertion and may come on while the patient is sleeping. The pain comes on suddenly is boring or constricting in character and is located in the region of the sternum. The pain may be retrosternal, substernal or precordial, or may be referred entirely to the epigastrium. The pain may radiate over the chest, to one or both arms, to the neck and back. The patient realizes at once that this attack is different from any that he has experienced before. The pain unlike that of angina pectoris, fails to pass off in a few seconds or minutes, and is not relieved by the nitrites, nor does it respond to morphine in the ordinary doses. The pain persists for hours or even days. In a certain per cent of the cases there may be no pain but in its stead there is severe dyspnea.

The patient is usually quite apprehensive and restless; he may be sitting up in bed or in a chair or tossing from side to side. The facies are those of extreme suffering, the complexion is ashen grey or pasty, and cold beads of perspiration stand out. Dyspnea is marked and some degree of cyanosis is present. Nausea and even vomiting are present, associated with the expulsion of gas.

Examination reveals a pulse which is rapid and very weak, sometimes imperceptible and often irregular. The blood pressure falls and this is regarded as a strong diagnostic feature.

The heart may be enlarged. Cough is usually present and productive of a frothy, blood-tinged sputum. Coarse rales are usually heard quite early. The heart sounds are always faint and of poor quality, the muscular element is almost entirely lost, the first sound especially being almost inaudible.

If the patient survives the immediate attack, or the first 24 hours, a low fever develops, the temperature rising to 100 or 101 degrees or more. Leucocytosis also develops, the average count being 10,000 to 15,000 but it may be higher.

Robinowitz, Shookhoff, and Douglas, (31) observed in 10 cases "that the sedimentation time was definitely shortened, and that this change appeared later in the disease than did the fever and leucocytosis, and persisted for some time after the temperature and blood count had returned to normal." They say, "the persistence of a rapid sedimentation rate beyond the time when the temperature and blood changes had disappeared made it, for us, a better index of the progress of healing of an acute myomalacia due to coronary occlusion than the temperature and leucocytosis."

They suggest that if these results are found to be a constant finding in further study that the procedure may be a valuable aid in diagnosis, prognosis and treatment of acute coronary occlusion.

A pericardial friction rub may be heard in some cases and is regarded as pathognomonic of myocardial infarction. This friction rub is said to be present in a varying proportion of cases by different observers, it is transitory and is therefore easily missed if the doctor is not there to look for it.

The value of the electrocardiogram in the diagnosis has been stressed and to sum up the principle changes as described by various investigators, (5) (32) (33) (34) (35)(36)(37).

There are changes which are said to be more or less characteristic of thrombosis, such as inversion of the T wave in the first, second or third lead; high level of the R T interval (Pardee signs); and prominence of the Q wave in lead three. The important diagnostic feature of the electrocardiogram is the rapid successive changes that occur in the shape and direction of the T wave from day to day.

In ten or fifteen days after an attack, in thrombosis of a branch of the left coronary, the T wave tends to become sharply negative in the first and possibly the second lead, while at the same time the T wave in lead three goes up.

In case of thrombosis of the right coronary opposite changes occur in leads one and three that is, the T wave in lead three is inverted and goes up in lead one.

As the lesion progresses to chronicity the take-off becomes lower and at the end of the upwardly rounded curve appears a gradually deepening V shaped inversion, this is the characteristic of the later stages of coronary occlusion. As healing and recovery take place the R T interval approaches the isoelectric line and the T wave becomes less inverted and in some cases a normal electrocardiogram may be re-established. The serial changes in the electrocardiogram are in this way an aid in prognosis.

The electrocardiogram is also of great assistance in the discovery and identification of the various arrhythmias which occur with great frequency in the first few weeks following an attack.

Any form of cardiac arrhythmia may be met with in coronary thrombosis. Premature beats are frequent in the acute stages of the attack, as also is gallop rhythm.

Auricular fibrillation is also quite common early.

Any degree of heart block may be met with, complete heart block is not so very common but must be recognized and dealt with as an emergency.

Paroxysmal ventricular tachycardia is rare but is important as its recognition and proper treatment may

save the patient's life. Levine (8) gives the following features which distinguish this condition: "In auricular fibrillation the rhythm is absolutely irregular while here it is essentially regular. It is important to bear in mind that slight irregularities in the rhythm may be heard on careful auscultation over the precorduim. This point has been emphasized as an aid in distinguishing ventricular from auricular tachycardia. The rhythm, although in the main regular even for fairly long periods of time, will every now and then be interrupted by a slight pause which can be readily detected by the ear. A further distinguishing feature between this type of tachycardia and paroxysmal auricular tachycardia, or auricular flutter, is that it is absolutely uninfluenced by vagal pressure, whereas, in the latter two conditions such pressure frequently completely arrests the one and the other is temporarily slowed. A final distinguishing feature is that during ventricular tachycardia one may not only hear occasional slight irregularities in the length of the heart beats, but the intensity of the first sound of the heart at the apex may vary. A curious sudden clicking, reduplication or snapping sound may be heard with various cycles."

Diagnosis

Pain, shock and circulatory failure are cardinal signs of coronary thrombosis. These with the special findings mentioned, fever, leucocytosis lowered blood pressure and electrocardiogram when possible are sufficient to make a diagnosis.

Angina pectoris is the chief other cause of pain in this region and the following table as given by Parkinson and Bedford (7) sets forth the main points in differential diagnosis.

	Angina Pectoris	Cardiac Infarction
Onset	During exercise	Often during rest or sleep
Site of pain	Sternum, often mid-sternum	Sternum, often lower third
Attitude	Immobile	Restive, even walk about
Duration	Minutes	Hours or days
Shock	Absent	Present
Dyspnea	Absent	Present
Vomiting	Rare	Common
Pulse	Unchanged	Small, often rapid
Temperature	No fever	Fever follows
Blood Pressure	Normal or rise	Fall
Heart Sounds	Normal	Distant, sometimes gallop, or pericardial rub.

	Angina Pectoris	Cardiac Infarction
Congestive failure	Absent	Commonly follows
Electro-cardiogram	Often abnormal	Usually diagnostic

Pain, shock, rigidity of the abdomen nausea and vomiting may simulate gall bladder disease, ruptured gastric ulcer, appendicitis or some other acute surgical abdomen. Many cases are on record in which the patient suffering from coronary thrombosis was needlessly operated upon.

Cholelithiasis is most commonly implicated in the error in diagnosis. The gall stone patients are younger, females predominate among them, four to one. A previous history of angina is strong evidence in favor of coronary thrombosis. Attacks that go back over a long period of time probably are not coronary thrombosis. The pain in gall stones does not radiate to the arms, but to the back, and comes in attacks; while the pain is more constant in thrombosis. Signs of circulatory failure point to thrombosis, while jaundice may occur in either.

A perforated ulcer will produce more rigidity than is seen occasionally in coronary thrombosis. A patient with a sufficient thrombosis, severe enough, to cause the shock and collapse of a perforation would show many signs in relation to the heart and circulation. Perforation can be

diagnosed by fluoroscopic examination, by seeing the air bubble above the liver or outside the stomach. History of any previous cardiac disease is helpful.

Smith (38) calls attention to the following, "The precordial or upper chest pains from an intercostal neuralgia or the nerve root pressure from a mediastinal tumor, tuberculosis of the vertebrae, or an hypertrophic osteoarthritis of the spine may at times arouse suspicion or even closely resemble that of angina pectoris. Usually the history is different and the physical examination will point to other possibilities."

There are other conditions which might be confused with coronary thrombosis but a careful history and weighing of the physical findings will usually lead to a correct diagnosis if the possibility of coronary thrombosis is borne in mind. The electrocardiogram is a very valuable aid in differential diagnosis and if possible it should be had in all suspected cases.

Manner of Death

Fulton (39) has discussed this matter at some length and summarizes his work by saying that the deaths which occur as a result of coronary thrombosis may be divided into three group:-

I. "The first includes those cases in which the immediate cause is more or less remote from the original lesion.

Not infrequently after an artery becomes occluded an intracardiac thrombus is formed at the site of the infarct. If this thrombus becomes loosened, some disturbance of the circulation occurs. If it is in the right heart and the main pulmonary artery is completely occluded death immediately ensues. If the embolus lodges in one of the branches, any degree of disturbance of the pulmonary circulation may occur. This will depend on the size of the vessel obstructed. If in the left, the result here too depends upon the size of the embolus and the artery which it finally occludes. The most serious obstructions are those involving one of the main coronaries, some of the cerebral arteries causing paralysis, the arterial supply of an extremity resulting in gangrene plugging of the renal artery or any large branch leading to any important organ. Death in the above group of cases is not strictly speaking a cardiac death.

II. "In the second group the damage to the heart as a result of cutting off its blood supply may lead to progressive weakening of the heart muscle so that death occurs as it does in any other instance of gradual myocardial failure, or there may be sudden rupture of the heart wall.

III. "We may include in a third group those cases in which the heart ceases to beat because of some interference with

the mechanism by which the heart beat is maintained in its orderly sequence and efficiency, resulting, e.g. in heart block or ventricular fibrillation."

The chief causes of sudden death are heart rupture either because of a softening infarct or a ventricular aneurysm; embolism of the pulmonary artery; ventricular fibrillation, and heart block. Most of the sudden or instantaneous deaths are unaccompanied by pain.

Prognosis:

The prognosis in an individual case of coronary thrombosis is hard to predict. Several large series of cases have been studied and from these certain conclusions have been drawn and the effect of certain factors noted.

A patient may seem to be well on the road to recovery and suddenly die, whereas, in other cases which are seemingly very severe and there is thought to be little hope for recovery the patient will survive and even become active again.

From the series of Parkinson and Bedford (7) the following is found on the course and prognosis, in one hundred cases: " 31 have died, one not traced, probably dead, and 68 still living. Twelve patients died within a month of the attack, 11 in 1 to 6 months, 7 in 6 to 24 months, and one after 11½ years, the average duration of life being 6 months in these 31 patients who died.

Of the 68 known to be still alive the duration of life after the attack has so far been longer than 5 years in 2 cases, 2 to 5 years in 9, 1 to 2 years in 19, 6 to 12 months in 16, 1 to 6 months in 20 and less than one month in 2, the average being just over 13 months. The average survival period for the whole series up to date is just under 11 months, but will be much longer when all cases have been followed to their termination. On pathological as well as on clinical grounds, we can confidently assert that a patient seen alive with coronary thrombosis is more likely to survive than to succumb to the attack."

Conner and Holt (40) give us some further interesting findings. Their series consisted of 287 patients, and of these, 117 were still living and 142 had died. They give the immediate mortality in the first attack as 16.2 per cent; the immediate mortality was said to be higher in cases with severe initial symptoms than when they are mild, yet about 1/3 of those who recovered had had severe initial symptoms.

Quite a number of their patients had more than one attack and this seemed to shorten the duration of life in some. They report one case in which the patient lived for 17 years and died in a second attack 18 years after the first one.

Levine (8) from his series of cases concluded that the younger patients are a little more apt to recover. White and

Bland (14) came to the same conclusion. They found the average duration of life after the attack for the 101 patients who were dead to be 1.5 years. The average duration to date for the entire series was 2.4 years. The patients who died were said to have "almost invariably died of heart failure."

Diabetes, hypertension and syphilis do not seem to have any effect upon prognosis.

Bad prognostic signs are said to be ventricular tachycardia and heart block, the mortality in these cases being higher than the average, evidence of circulatory failure which does not start to abate in 24 hours or becomes worse in spite of treatment, a continued leucocytosis and pyrexia for more than a few days or a week, and extreme hypotension.

Parsons-Smith (41) summarizes the more noteworthy characteristics which he regards as definitely favourable in cases of coronary thrombosis, they are principally the disappearance or absence of the factors listed above as of bad prognostic significance. He says also that "Well marked serial changes in the electrocardiogram preceeding a gradual development of the pre-existing type of curves is a good sign."

Pathology:-

The pathology of coronary thrombosis is the process of infarction and a description of this process can be found in any good textbook of pathology, and in many articles in the literature so I will not describe it in detail.

The most frequent location of thrombosis is in the anterior descending branch of the left coronary. The process is one of extravasation of blood into muscle tissue, necrosis and early repair by connective tissue. The area is sharply demarcated from the uninvolved area. The appearance of the area depends upon the time elapsed between the attack and death. During the first day the muscle is red, due to extravasated blood, some necrosis starts in immediately but is not marked until about the 4th day until the end of the third week during which time it is the predominating feature. It is during this time that rupture is most apt to take place. Connective tissue repair begins in a small way as early as the sixth day but is not marked until after about three weeks and it takes about five or six weeks for good firm cicatrization.

Ball (42) in a review of the cases of heart block reported in the literature finds that in complete heart block the electrocardiogram shows that the right coronary artery is involved in 93% of the cases and the left in about 7%.

This is said to be due to the fact that the blood supply to the A-V node is derived in most cases from branches of the right coronary.

Treatment:

The proper management of a case of coronary thrombosis is a very important factor in the prognosis.

All authors have stressed the importance of immediate rest and quietness for the patient and also for long continued bed rest, five or six weeks and even longer during the period of the convalescence. During the early hours and days the patient should be spared any unnecessary movement or exertion and in some instances Brown (43) says it may be wise to leave the patient where he collapsed instead of moving him.

The pain is usually the most important symptom to be relieved and for this morphine in large doses, $\frac{1}{4}$ to $\frac{1}{2}$ grain or more, should be given. Sleep will also be promoted by the morphine and this is desirable. Rizer (44) has used oxygen and reports that with its use the pain and other symptoms quickly disappear and the necessity for using morphine is diminished; other writers report similar results and say it is especially valuable in relief of dyspnea and cyanosis.

Shock should be treated by the usual method, keeping the patient warm with blankets and hot water bottles. It is

generally agreed that stimulation should be avoided unless there is a very poor circulation. Caffeine, caffeine sodiobenzoate and similar drugs may be employed with benefit as they are said to strengthen the heart beat, stimulate the vasomotor center causing constriction, and stimulate the respiratory center causing an increase in alveolar ventilation. This produces an increase in the coronary flow and better oxygenation of the blood, both probably of value in lessening tissue necrosis. When a stronger stimulant is needed eight to ten minums of epinephrine may be given.

None of the vasodilators are of value and it is generally agreed that they may do harm by causing a further lowering of blood pressure, and an extension of the thrombus may occur.

The role of digitalis in coronary thrombosis is discussed at some length by Levine (45) and Kilgore (46). I find that it is generally agreed that digitalis may do considerable harm to a heart in this condition, i.e. so stimulate it that it causes rupture of the weakened infarcted area or so irritate the heart as to increase the chances of ventricular tachycardia or fibrillation; it also depresses the conductivity and may be the added factor necessary to produce block in an already injured conducting system. In general digitalis should not be used unless it

is indicated by auricular fibrillation or congestive heart failure.

Heart-block may be combated with oxygen, (43) but the usual method is to administer adrenalin 0.5 cc. intramuscularly and repeated as often as necessary; in some cases it is necessary to inject it directly into the heart. Barium chloride may be used but its value is still doubtful. For repeated Adams-Stokes syndrome Brown (43) says "barium chloride in thirty milligram doses may be given three or four times a day by mouth."

Levine (45) and Kilgore (46) both advise the use of quinidine 0.2 Gm. doses three times a day during the first two weeks to two months as a prophylactic measure against the occurrence of ventricular fibrillation. When paroxysmal ventricular tachycardia has developed quinidine sulfate will usually restore the heart to normal rhythm. The dose necessary to accomplish this varies from 5 grains to over 15 grains. Levine (45) says, "It is best to start with a small dose and increase the dose every four hours until the desired result is obtained."

In cases complicated by diabetes and glycosuria it has been urged that insulin should not be used as it increases the amount of work done by the heart and may precipitate anginal attacks or cause rupture of the heart and result fatally.

Likewise in syplilis, intravenous treatment should not be given but the treatment should be the same as in non-syphilitics. After convalescence is established treatment may be given with potassium iodide by mouth and mercury preparations by mouth or intramuscularly.

During the acute stages especially early it is impossible for the patient to take anything by mouth, it is necessary to keep up the body fluids and supply nourishment. This can be done by giving 30 to 40 cc of 50% glucose solution intravenously once in 24 hours and 500 to 1000 cc of normal saline with 5% glucose per rectum until they are able to take enough fluid and food by mouth. There is usually constipation early due to the morphine but this condition does not require attention and is said to be of value in that the patient does not have to exert himself in attending to the bowel movement. Enemas may be given after the first few days but no laxatives. The diet should be soft, easily digested nourishing foods.

When the period of bed rest is over the patient should be allowed to get up gradually and sit in a chair for increasingly long periods of time. A week or so should be spent in this process. A gradual return to former activities is based upon the condition of the heart. Graduated exercises serve a double purpose, they will enable the patient to discover how much effort he may put forth without feeling

pain, and they may possibly strengthen the heart muscle to some extent. The patient must realize his limitations and adapt his activities to them. He should lie down and rest several hours a day. If previously over weight he must keep his weight down. He should avoid sudden strains both physical and emotional. Excessive smoking, drinking and eating should be warned against. The degree of recovery and activity permissible vary in the individual case from those that are permanently crippled to those who return to their customary duties and remain in good health for years.

Special attention is called to the management of those cases in which the initial symptoms are mild and the clinical course is apparently favourable by Kilgore (46). In these cases it is essential that bed rest be continued long enough to allow firm healing of the infarcted area of unknown size. The time required for this healing to become strong enough to resist rupture or dilatation is not definitely known, but from four to six weeks should be given and in case the blood pressure is high a longer time. Possibly further studies of the electrocardiographic changes or the sedimentation time as suggested by Robinowitz, Shookhoff and Douglas (31) would be of value.

Congestive heart failure is to be treated as such, limitation of fluids, diuretics and digitalis.

Embolism, not resulting fatally, requires a longer period of bed rest and general supportive measures.

Conclusion:

1. Coronary thrombosis is most common between 50 and 60; in males than in females the ratio being about 3 to 1.

2. The chief etiological factors found were hypertension, arteriosclerosis, a familial tendency and previous agina pectoris.

3. The infectious diseases, syphilis and diabetes along with tobacco and alcohol play a minor role in etiology.

4. The role played by physical work and mental strain is hard to evaluate, but probably have a definite bearing.

5. Description of events in a typical case is given along with special findings; also some differential diagnosis. The importance of history and a careful evaluation of symptoms is stressed. One should always bear in mind the possibility of coronary thrombosis in patients of this age group with symptoms.

6. As to prognosis: the majority of patients seen alive after an attack will recover, the length of life varies from less than 1 month to 17 years in cases reported in the literature. Favourable and unfavourable prognostic findings have been given.

7. Good treatment is essential and consists of the following:

(a) Relief from pain and shock.

(b) Bed rest for from 4 to 6 weeks or longer.

(c) Avoidance of the use of the vasodilators and early stimulation.

(d) Use of digitalis only for auricular fibrillation and congestive heart failure.

(e) Quinidine in 0.2 gm. doses to prohibit or in larger doses to arrest ventricular tachycardia and fibrillation.

(f) Adrenalin for heart block.

(g) Graduated exercise after the period of bed rest and education of the patient as to his limitations.

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