

## THE PATHOLOGY OF CARDIAC INFARCTION.

BASED UPON A STUDY OF SIXTY-FOUR CASES.

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Until about twenty years ago cardiac infarction was rarely, if ever, diagnosed before death, and hence the description of this condition was confined to the reports of pathologists, with rather meager clinical details. Following Herrick's classical paper in 1911, there has been a long list of excellent contributions to the subject, dealing with its clinical, electrocardiographic, and pathologic phases. Today this clinical entity is generally well known. Our medical students at once recognize the typical cases with acute onset of severe substernal pain, in patients past 50 years of age, who present signs of shock and cardiac failure, pericardial friction, slight fever, leucocytosis, and certain changes in the electrocardiogram. The variations from this textbook picture have been emphasized, viz.: cases without pain, but with marked dyspnea, instances with the features of an acute surgical condition of the abdomen, the occurrence in relatively young adults, etc. As pointed out by Barnes and Ball of the Mayo Clinic a few months ago, the recent communications dealing with the pathologic details of cardiac infarction have been relatively few. There is a lack of details relative to the incidence and situation of infarction in a large series of cases. This study was undertaken with this particular point in mind, and also with the idea of correlating the pathologic findings in this series of 64 cases with the clinical picture.

A more accurate use of diagnostic terms, as applied to cardiac infarction, is desirable on the part of the clinician. Since one can not possibly differentiate arteriosclerotic narrowing from actual thrombosis of the coronary artery at the bedside, and since both may cause cardiac infarction, it is evident that the term coronary thrombosis is not sufficiently comprehensive. Coronary occlusion with cardiac infarction appears to be both more inclusive and more accurate.

*Lantern Slide No. 1.* The material upon which this study is

based comprises 64 cases derived from 1424 consecutive post-mortem examinations made by the Pathological Department of the Albany Medical School and Hospital—an incidence of 4.3%. The only cases excluded were those of cardiac infarction due to emboli from endocardial lesions and a few cases of diffuse coarse fibrous scarring of the myocardium, which were not considered typical. All of the hearts showed gross evidence of cardiac infarction. There were 41 males and 23 females in the series. A large majority of all cases occur between the ages of 50 and 79. A tabular arrangement by decades of 113 cases (Barnes and Ball 49, our series 64) shows that 84 cases or 93% fell in the three decades following the 50th year of life.

*Slide 2.* (Coronary arteries.) *Slide 3.* (Coronary arteries.) *Slide 4.* (Location infarcts.) Barnes and Ball have emphasized that infarction of the heart is almost completely confined to the left ventricle, and that there are three different portions of the ventricle which may be involved. The site depends upon which of the three main arterial branches is obstructed.

*Location No. 1. "Anterior Apical."* The apex and anterior portion of the left ventricle are involved. This is almost invariably due to occlusion of the anterior descending branch of the left coronary artery. In rare instances it may be due to the occlusion of the main trunk of the left coronary. Roughly, the apex and adjoining  $\frac{1}{3}$  of the anterior surface of the left ventricle are involved. The interventricular septum is frequently affected, but the right ventricle almost always escapes.

*Location No. 2. "Midventricular."* The obtuse margin of the left ventricle about midway between base and apex, although the infarct may occur at the base and occasionally well down toward the apex. The circumflex branch of the left coronary is usually responsible for this type.

*Location No. 3. "Posterior Basal."* The posterior surface of the left ventricle in its basal three-fifths, the adjacent position of the interventricular septum and very rarely a small border of the adjacent right ventricle are involved. Occlusion of the main trunk of the right coronary usually is the cause of this type of infarct, although occasionally the circumflex branch of the left coronary may be involved. With variations in the blood supply the infarct may extend

down to the apex or out to the obtuse margin of the left ventricle occasionally.

Barnes and Ball found that infarcts in their series could be classified as follows: Type I, 28 cases; Type II, 8 cases; Type III, 24 cases. Our figures show: Type I, 54 cases; Type II, 4 cases; Type III, 11 cases. (The apparent discrepancy in the total number of cases is due to the fact that more than one infarct occurred in several instances.) The above authors state that their study "serves" to correct the impression that an overwhelming majority of cardiac infarctions occurs as results of occlusion of the anterior descending branch of the left coronary artery. It is no longer justifiable to designate the anterior descending branch as the "artery of coronary occlusion." Levine in his series found: Type I, 39 cases; Type II, 4 cases; Type III, 2 cases.

From our observations we are inclined to believe that a larger series than 49 cases must be studied before accurate conclusions may be drawn regarding this point. Parkinson-Bedford: Type I, 24 cases; Type II, 10 cases; Type III, 18 cases.

*Slide 5.* The method we have employed of examining the heart for the presence of cardiac infarction is very important and is worthy of description.

The size and color of the pericardial sac are first noted. If enlarged and dark in color hemopericardium due to rupture is indicated. On opening the sac the position and character of adhesions are noted. If localized they may offer a clue as to the site of the lesion; if easily broken they point to a recent infarct; if tough they indicate an older one. If no adhesions are present a slightly turbid fluid containing flecks of fibrin may be found in acute infarction.

After removing the pericardial fluid, or in the case of hemopericardium, the large blood clot enveloping the heart, the organ itself is carefully examined *in situ*. Loss of normal luster of the visceral pericardium means a fine deposit of fibrin, which usually indicates the site of the infarct. A fresh infarct gives a reddish mottling to the epicardial layer above it, while an old fibrotic one produces in this layer a white, wrinkled, slightly depressed appearance. An aneurysmal dilatation may be seen in old infarcts.

If rupture has occurred its location may be demonstrated by firm pressure, blood being made to flow through the tear.

Palpation may reveal an area, somewhat fluctuant, of lessened resistance, and easily dimpled in either an old or recent infarct. If a thick mural thrombus is present in the ventricle, there may be board-like rigidity.

Ante-mortem thrombosis is best determined before fixation in any preserving fluid. Microscopic examination should be the final check however, and may be carried out after the heart has been hardened. The usual method of opening the coronary arteries with fine scissors should not be employed, as a small thrombus may be crushed or dislodged. It is far better to use a small sharp scalpel and to lift off small wedge-shaped segments of the roof of the vessel, along as much of the vessel as is necessary. This will leave the thrombus undisturbed in its original position.

The heart should not be opened in the fresh state once the presence of an infarct is determined. It should be hardened in Kaiserling's fluid for ten days. The long single incision will usually reveal the infarct and ventricular contents better than several small cuts. The proper exposure is made by a long-bladed knife, starting from about the mid-line of the apex, keeping to the left of the inter-ventricular septum and finishing just to the left of the base of the aorta.

There are two points in this technique to be emphasized: *First*, to fix the heart by suspension in Kaiserling's fluid so that pressure of its own weight or the proximity of other specimens will not disturb it; *Second*, to open the heart by a single incision, extending through the middle of the infarct and continued through both anterior and posterior walls of the heart.

*Slide 6.* Photograph showing thrombus in anterior descending branch of left coronary artery about 3 cm. from its ostium. It is to be noted that the thrombus or arteriosclerotic occlusion of the coronary artery producing cardiac infarction usually occurs in the first few cm. of the vessel, while the area of infarction is regularly a considerable distance distal to it. Note infarction in this case, previous slide, was at the apex.

In our series there were 25 instances of actual thrombosis with arteriosclerotic narrowing, such as this shown in the slide, and 39 cases in which no thrombosis was demonstrated, but in which vascu-

lar narrowing alone occurred. Both types of occlusion result in typical infarcts.

*Slide 7.* A microscopic section of a thrombosed coronary artery with canalization.

*Slide 8.* Colored plate showing a thrombus, arteriosclerosis of coronary artery, infarct, mural thrombus of ventricle, and acute pericarditis.

*Slide 9.* Microscopic section of parietal pericardium with fibrinous exudate. This exudate is sterile, often confined to a small area, and it is conceivable that it might readily be absorbed in some instances, leaving no trace behind it of involvement of the pericardium. This perhaps explains the transient and localized character of the pericardial friction rub.

*Slide 10.* Colored plate of chronic adhesive pericarditis with parietal aneurysm. In the 64 hearts there was post-mortem evidence of pericarditis in 24 instances (16 acute pericarditis, 8 chronic adhesive form), or 37.5%. A friction rub was heard during life in only six of these cases—9.4%. Barnes and Ball found pericarditis in 22.5% of their hearts. No statement as to the frequency of the friction rub was given. Evidently pericarditis is much more common at autopsy than are the signs of it detected during life.

Pericarditis occurred in our series in 56% of the cases with actual thrombosis, plus arteriosclerotic narrowing, and in only 28% of the cases without thrombosis.

*Slide 11.* Microscopic section of heart muscle, showing necrosis and infiltration with polynuclear cells from an infarcted area. The fever and leucocytosis seen in acute cases are readily explained when one sees this picture.

*Slide 12.* Infarction of the interventricular septum of extreme grade, producing an aneurysmal bulging which stopped just short of rupture.

*Slide 13.* Anterior-posterior view of the septum, showing the muscle tissue reduced to a thin band and covered by a thrombus.

Repeated electrocardiograms of this patient during life failed to reveal any striking abnormality. It is interesting to note how extensive the pathologic change may be in the septum which carries the

bundle of Hiss, or in the left ventricle itself, without producing significant electrocardiographic findings.

Our series contained only a relatively small number of patients on whom electrocardiograms had been made, so that no deductions can be drawn from these. Barnes and Ball, however, in 35 cases of cardiac infarction report characteristic electrocardiographic changes in 11 cases, abnormal but not pathognomic in 19, and normal tracings in 5 instances. Rupture of the septum may occur and has been diagnosed during life.

A clinical diagnosis of rupture of the interventricular septum was made in 1928 to my knowledge by Dr. V. H. Norris on a patient admitted to the Columbia University Division at Bellevue Hospital. In a recent personal communication he kindly gave me the details of the case with permission to refer to it. The patient gave a typical history of coronary occlusion, with two previous attacks. The diagnosis was confirmed by physical examination and by the electrocardiogram. In addition a loud systolic murmur was heard all over the precordium, of maximal intensity at the fourth left interspace, near the sternum, and transmitted to the left as far as the posterior axillary line. Over this space a thrill was felt. Two members of the visiting staff independently interpreted the murmur as due to a defect in the septum, secondary to a cardiac infarct. The autopsy findings were as follows: Pericarditis. At the apex of the left ventricle a bulging sac, 3 cm. in diameter, with evidence of recent myocardial injury. Mural thrombus at apex of left ventricle. Through the interventricular septum near the apex a small opening, 5 x 6 mm., elliptical in shape. Left coronary contained a thrombus occluding the lumen.

Another similar case occurred on the Division last summer, autopsy showing a small perforation in the septum near the apex. A somewhat similar murmur is described as having been heard. This was interpreted by the house physician as being due to a defect in the interventricular septum, although the visiting physicians who saw the case did not agree with this interpretation.

*Slide 14.* Hemopericardium—Rupture of the left ventricle occurred in six cases of our series. It is to be noted that the rupture never takes place through the protective mural thrombus, but rather

to one side of it where the heart muscle has become markedly thin and fibrous.

*Slide 15.* A cross section of the same heart, showing thrombosis of the anterior descending branch of the left coronary artery. Five of the six cases of rupture were due to actual thrombosis of a coronary vessel—four times the anterior descending and once the circumflex branch of the left coronary artery.

A cross section at a lower level reveals the point of rupture.

#### SUMMARY.

1. "Coronary occlusion with cardiac infarction," is a more accurate and comprehensive term than coronary thrombosis.
2. A method for the post-mortem examination of the heart with cardiac infarction has been described.
3. The exact location of the cardiac infarcts in a series of 64 cases has been tabulated and compared with those reported in three other large series.
4. Acute or chronic pericarditis was found post-mortem four times as frequently as was the friction rub during life. Due to its transient and localized nature the rub is probably often not discovered at the bedside.
5. Two instances are mentioned in which the diagnosis of rupture of the interventricular septum was made and confirmed at autopsy.
6. In our series of cases actual thrombosis plus arteriosclerotic narrowing produced pericarditis and rupture more frequently than did arteriosclerotic narrowing alone.

#### REFERENCES.

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#### DISCUSSION.

DR. JOSEPH T. WEARN: It was formerly the impression, I believe, that a majority of infarction resulted from occlusion of the anterior descending branch, and it is just such a study as Dr. Gorham's which eventually puts the house in order and shows us quite clearly that impressions are not always correct.

The other point that I think is a very interesting one is the question of collateral circulation. We frequently see this in occluded coronary arteries, a

space in the apex of the heart that literally amounts to almost the whole apex; that is to say, the left anterior descending branch will be occluded two or three centimeters up from the apex. Now, if that occlusion is complete, the whole apex should be infarcted, but we rarely see that as a result of an occlusion at that point. When it heals, one sees an infarct about the size of a quarter, and this undoubtedly is due to the collateral circulation that is set up.

One frequently sees, for instance, a thrombus or an infarct on the surface of the heart not extending down to the chamber, or vice versa in the chamber not extending to the surface, and these can be readily explained by the collateral circulation that takes place.

One other point that I think is of interest and worth mentioning is one or two clinical pitfalls. These I have seen in the past ten years. Two of them resemble exactly the picture of an occlusion where there are thrombi in the left auricle that blocks the mitral valve. In each of these instances the diagnosis could be made by X-ray or fluoroscopic study of the chest. The clinical picture of leucocytosis, fever, sudden failure was so complete that in each of the instances I missed the diagnosis.

A second picture that resembles it very closely is the sclerotic aneurysm of the aorta near the aortic valve. I have seen three of these now, all told, that presented the characteristic pain, precordial, radiating down the arm, with leucocytosis, fever and all of the findings that one is accustomed to meet in coronary occlusions, with eventual rupture of the aortic aneurysm or the second aneurysm, followed by sudden death.

DR. JOSEPH S. PRATT: I would like to ask the cause of the embolism in the six cases of this series?

DR. PORTER: I think that when one uses the pathology of coronary sclerosis and tries to fit in entirely the end results of coronary sclerosis as being thrombus formation, he is confronted with certain difficulties which appear in the light of most published reports as being insurmountable. Coronary sclerosis, with gradual occlusion of the vessel, is a slow, insidious and progressive lesion, and yet we are all impressed with the suddenness of occlusion with no preceding history in many of the patients who die from myocardial infarction.

For the past two or three years we have been selecting a certain group of patients largely from the coroner's cases who have died suddenly from coronary occlusion without even a period of two or three minutes of illness after the occlusion. We have made serial sections of these vessels, and there is one lesion which is quite outstanding in the majority of these cases, namely, a very diffuse arthritis. It is very difficult to evaluate this finding, but I am wondering whether there is not some X factor which is a precipitating cause, and that of a coronary occlusion is the end result of arteriosclerosis plus this X factor, and this X factor may be an acute arthritis.

The origin and nature and the pathogenesis of this acute arthritis I do not care to speculate on. It is just a striking finding if one studies those acute cases.

DR. GORHAM: In answer to Dr. Pratt's question as to the cause of em-

bolism, we have not completed our studies of this group of cases. However, I might say that these cases all show evidence either of rheumatic or bacterial endocarditis. The type of infarction seen is quite dissimilar from that that I have shown you. It consists in multiple small areas of fibrosis. We felt that these cases were not typical and, therefore, could be excluded from this series.

Dr. Porter's suggestion brings to my mind the point that in two cases, not included in this series, we found evidence of thrombosis in smaller vessels microscopically without any gross evidence of change in the large coronary vessels and without marked infarction. These cases were also not included.