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THROMBOSIS OF THE CORONARY ARTERIES

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In a paper¹ on certain clinical features of sudden obstruction of the coronary arteries, read six years ago before the Association of American Physicians, I called attention to the fact that while such obstruction as by a thrombus was very often suddenly fatal, it was not necessarily so; for the coronary arteries were not in the strictest sense terminal arteries; there were often anastomoses capable of reestablishing circulation in the infarcted area to such an extent as to enable the heart to functionate for a time at least. This had been shown by anatomic study of the coronaries, by necropsy observations by the pathologists, by experimental ligation of these vessels, and by clinical experience controlled by post-mortem examinations.

A tentative grouping of the cases of coronary thrombosis based on clinical symptoms was made:

1. Cases of instantaneous death, a group graphically described by Krehl, in which there is no death struggle, the heart beat and breathing stopping 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

1. Herrick, J. B.: Clinical Features of Sudden Obstruction of the Coronary Arteries, *J. A. M. A.* 59: 2015 (Dec. 7) 1912.

ordinarily not recognized, due to obstruction in the smallest branches of the arteries.

It was Group 3 whose clinical phenomena were discussed, as illustrative of which a few cases were briefly cited, including one with necropsy findings.

These cases of coronary thrombosis are, as it seems to me, of commoner occurrence than is generally supposed, and that is the justification for repeating some of what was contained in the former paper and for adding reports of other cases and for touching on

some recent experimental work that has a direct bearing on this subject.

CLINICAL SYMPTOMS

Most of the patients are middle-aged or elderly men. The heart and blood vessels in many show the evidence of arterial and cardiac sclerosis; the blood pressure may be high. In others no sign of such change is to be made out. In fact, in two of my three cases with necropsy, the only significant vascular sclerosis was in the coronary arteries, the hearts were of normal size, and there had been no hypertension during

life. In one there was slight involvement of the beginning of the aorta. Previous attacks of angina pectoris may have been experienced. If this has been the case, the patient will describe the attack due to the coronary thrombosis as of unusual severity. Often there is no assignable cause for the attack such as is commonly noted in the typical paroxysmal angina—walking, a heavy meal, undue excitement, etc.—though in some cases these exciting factors seem to provoke the attack or, at least, greatly to aggravate it when it has started. The painful seizure is usually more enduring than in ordinary angina, the spell lasting many minutes or several hours, or a status anginosus developing. The location of the pain, as in the classical angina, is com-

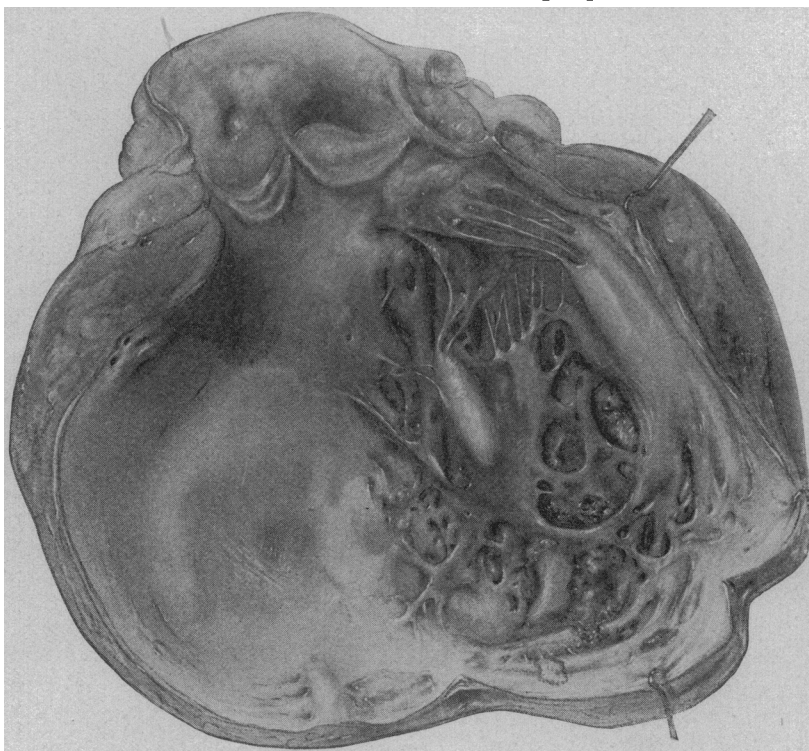


Fig. 1 (Case 3).—Changes on the endocardial surface of left ventricle.

monly substernal, with frequent radiation to the arms and neck. But in many of the thrombotic cases the pain is beneath the lower sternum or even in the upper epigastric region; and there may be no radiation to the arm or neck. This epigastric reference of the pain, with the nausea and vomiting that frequently occur, often suggests to patient and physician some abdominal accident, such as acute pancreatitis, perforation of the gallbladder, or a gastric or duodenal ulcer. And these suspicions are strengthened by the fact that there are so frequently signs of shock and collapse—ashy hue of the face, clammy skin, and small, rapid, feeble pulse.

The heart is commonly rapid, even to 140 or more, though slow heart action has been recorded. There may be irregularities, such as extrasystoles or partial block. The pulse usually lacks in strength and may be almost imperceptible, though in some cases the strength is wonderfully well preserved. Blood pressure is lowered, and tends to grow lower in the unfavorable cases. The heart tone may be startlingly faint, both because of the weakness of the heart's musculature and because of an acute emphysema that may develop and mask the heart sounds. Over the infarcted area a pericardial friction is sometimes heard. The heart may reveal evidence of dilatation by its increased area of dullness and its mitral systolic murmur due to relative insufficiency of the valve. Râles in the bronchi, with other evidence of pulmonary edema, may be present. Passive congestion of the kidney may show in a trace of albumin in the urine. In some cases, cardiac edema of marked degree appears,

the albumin being large in amount, the legs badly swollen, and free fluid being present in the abdominal cavity. The mind is commonly quite clear. I have been surprised at the preservation of bodily strength that is often manifested. Patients occasionally walk about within a few hours after such a seizure, and within a few days may be outdoors trying to attend to business.

These symptoms will often enable one to make a reasonably certain diagnosis of acute obstruction of the coronary artery. As in so many other conditions, the first essential is to think of this condition as a possibility and to rid the mind of the notion that such a diagnosis is possible only at necropsy.

In the following three cases the diagnosis was confirmed by necropsy. In the first instance, the first I had ever recognized clinically, the diagnosis was decidedly tentative and hesitating. In the other two, as well as in several in which no necropsy has been permitted or in which the patients are still living, diagnosis has seemed definite, as clear, for instance, as in most cases of obstruction of a cerebral vessel. I give very brief abstracts: Case 1 is reported more in detail in the article of six years ago.

REPORT OF CASES

CASE 1.—A man, aged 55, was seized with severe pain low in the chest and epigastrium. He vomited. The physician,

believing, as did the patient, that the attack was of gastric origin, washed out the stomach; but relief came only with morphin. The man lived fifty-two hours. He was conscious to the last. His pulse from the first was rapid and very weak, the heart tones nearly inaudible. An acute emphysema with many moist râles developed. No abdominal or other than cardiac cause could be made out as an explanation of the condition. The temperature never was above 99.2 F. The left ramus descendens was found plugged by a thrombus at a narrow spot where there was a small pathologic mural roughening and thickening.

CASE 2.—A man, aged 62, while walking on the board walk at Atlantic City, was seized with a terribly severe pain in the lower precordia. He was helped to the hotel, but refused to call a physician while in Atlantic City or for the next two days while in New York, for, as he said, he knew from the severity of the initial pain, the great weakness and difficult breathing, that he would be ordered to a hospital, and he wished to get to his home in Chicago for his illness and, as he feared, his death. He lived over two weeks from the

onset of the attack. His heart when he reached Chicago was dilated, weak and somewhat rapid. His blood pressure became progressively lower. There were dyspnea, cyanosis and marked edema of the legs and albuminuria. Death was as in ordinary cardiac failure. Necropsy revealed recent plugging by a thrombus of the descending branch of the left coronary, with softening of the heart muscle at the left apex and the lower interventricular septum.

In this, as in the other case cited, the aorta was free from evidence of sclerotic change. No other vascular lesions were found than the isolated patches of sclerosis in the coronary. It happened also that in each case the patient had only a short time before been carefully examined by competent physicians and had



Fig. 2 (Case 3).—Site of thrombi and fairly normal external appearance of heart.

been pronounced in good health as to heart, blood pressure, urine, etc.

CASE 3.—A physician, aged 42, who had never suffered from angina pectoris, had no acute or chronic infection so far as he knew, and who regarded himself as free from cardiovascular and renal disease, was seized, while in this seemingly good health, with a sudden, excruciating pain in the lower sternal

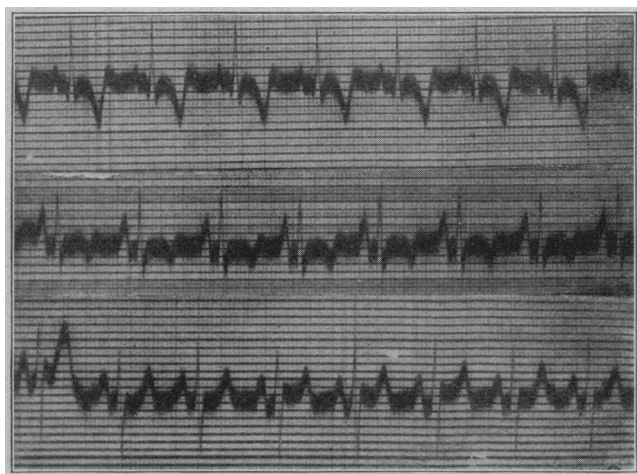


Fig. 3 (Case 3).—Electrocardiogram taken May 3, 1917, forty-one days after the coronary obstructive symptoms. Digitalis not used at this time.

region, which pain radiated to the arms and to the epigastrium. He was profoundly shocked, very weak and nauseated, the skin cold and clammy, the pulse rapid and thready. His colleagues who saw him thought he would die in a very short time. Morphine was necessary to control the pain. After a time he rallied, and in ten days was able to be about and even tried to do some professional work. But in a few days his weakness, precordial distress on exertion, dyspnea and irregular, rapid and small pulse caused him to give up. He rested, again improved, and came to Chicago. He was seen by my assistants, J. R. Greer and F. M. Smith, who ventured a diagnosis of coronary obstruction, a diagnosis that I felt was correct when I saw him a few days later. Electrocardiograms taken on these two occasions gave support to these opinions, a point to which reference will be made later. Rest and small doses of digitalis were ordered, and improvement followed. The summer vacation was spent on an island, where there was considerable violation of the injunction as to quiet and digitalis, the patient not only eating very heartily, but not infrequently going on strenuous walks, fishing and hunting trips, even though precordial pain, occasional attacks of dyspnea, and cardiac irregularity gave warning that the heart was far from normal. From an unduly severe strain of this sort he suffered for several days with dyspnea and arrhythmia; he contracted an infectious bronchitis and hastened to Chicago, where, in a deplorable condition, he went under my care in the Presbyterian Hospital, Sept. 26, 1917. The dyspnea and cyanosis were marked; he was sleepless and had Cheyne-Stokes breathing; the temperature rose to 102. The blood pressure was not high, the heart was slightly enlarged to the left, and there was a faint systolic murmur at the apex. After four days he developed signs of pneumonia; the respiration rose to 32 and the temperature to 105; there were bloody and rusty sputum, marked albuminuria and leukocytosis; he became delirious, and just as a distinct drop in temperature and a clearing of the mind made one wonder if recovery after all might be a possibility, he died suddenly. Necropsy disclosed pneumonia of the lower lobe of the right lung and several infarcts in other portions of the same lung.

The heart was large—the left ventricle at the apex, the lower interventricular septum and the papillary muscles, especially the anterior one, thinned, scarred and cutting like gristle. The descending branch of the left coronary and the large descending branch of the left circumflex were completely obliterated by old thrombi. The only sclerotic changes in the arteries

were a few small patches in the aorta close to the openings of the coronary arteries, and similar patches in the coronaries themselves. It may be added that no history or stigmata of syphilis were noted during life, and that the Wassermann test on the blood had been negative.

According to the necropsy record made by Dr. Thacher of the Presbyterian Hospital, Oct. 8, 1917, the heart weighed 415 gm. The enlargement involved both sides of the heart. The external surface was fairly normal in appearance with the exception of a pale, depressed area 4 cm. in diameter midway between the base and apex of the left ventricle and a smaller similar area on the lateral surface close to the apex. The wall of the left ventricle near the apex and the interventricular septum were thin and of gristly consistency, cutting with marked resistance. The endocardium of the left ventricle had a pale, mottled appearance especially marked over the lower three quarters of the septum and the papillary muscles. The papillary muscles were smooth, contracted and firm, these changes being particularly marked in the anterior muscle. The greater extent of the fibrous changes in the endocardial and subendocardial tissues as compared with those on the external surface of the heart was striking. The left coronary artery contained an organized thrombus in the ramus descendens anterior 3 cm. from its origin. Also the first descending branch of the circumflex was occluded 1.5 cm. from its origin. The coronary arteries showed sclerotic plaques scattered throughout most of their course, these spots varying in size from 0.5 to 2 mm. in diameter. The aortic leaflets were thickened. There were a few sclerotic areas in the supra-sigmoid area of the aorta.

EXPERIMENTAL WORK

I wish to call attention briefly to certain experimental work that has a direct bearing on the question of obstruction of the coronary arteries. At my suggestion, Dr. Fred M. Smith² in the Presbyterian Hospital and Rush Medical College studied experimentally the coronary arteries in dogs.

Method.—He took an electrocardiogram of each dog when under ether. Then the desired branch of the coronary artery was ligated. Following the operation electrocardiograms were taken at intervals varying from a few minutes to many days or weeks. On all

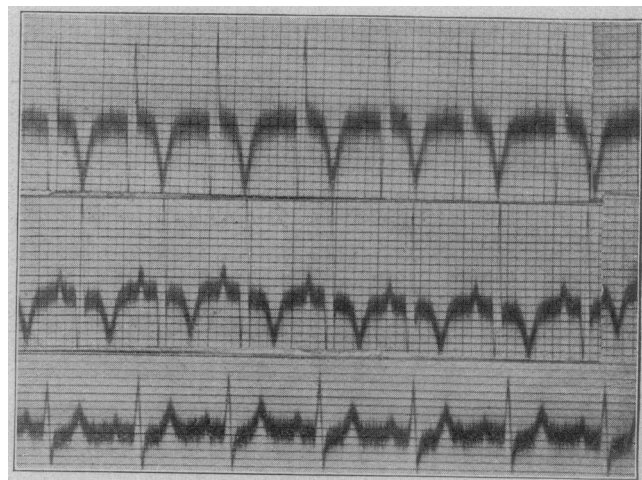


Fig. 4 (dog).—Electrocardiogram taken two days after ligation of anterior and posterior descending branches of the left circumflex artery.

dogs that died or that were killed a postmortem was made, and the gross and microscopic structure of the myocardial lesion was studied. Electrocardiograms were compared to see if there was uniformity in the tracings made after ligation of the same arteries and with the same lesions. Lastly, in human beings with

2. Smith, F. M.: The Ligation of Coronary Arteries with Electrocardiographic Study, *Arch. Int. Med.* 22: 8 (June) 1918.

symptoms suggestive of coronary thrombosis, the electrocardiograms were compared with those in dogs.

Results.—The results may be thus summarized:

1. Dogs may live days or months or may recover after ligation of branches of the coronary arteries, even branches of considerable size. This is contrary to the findings of Cohnheim, but is in accord with those of Porter, Miller and others.

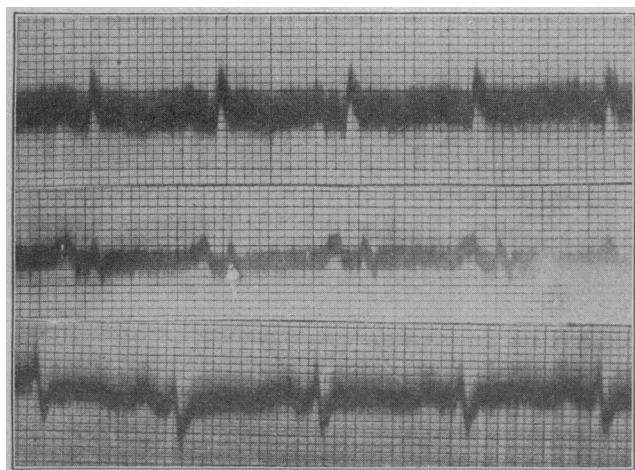


Fig. 5 (Case 3).—Electrocardiogram taken Sept. 27, 1917, 178 days after coronary thrombosis.

2. After the ligation of special branches, the lesions produced in the muscle are fairly constant. These lesions are most marked in the endocardial and subendocardial tissues, that is, in the conducting region. These findings may be compared with those of Oppenheimer and Rothschild.

3. Following the ligation, numerous irregularities, such as extrasystoles, tachycardia, auricular fibrillation, auricular flutter and ventricular fibrillation, may result. Thus far it has been impossible to predict the type of irregularity that will follow a ligation.



Fig. 6 (dog).—Electrocardiogram taken thirty-four days after the ligation of the ramus circumflexus sinister. Compare this low voltage tracing with Figure 5.

4. There seems to be a fairly constant variation in the electrocardiogram following the ligation of a particular branch of the coronary.

5. There is some hope that the work may assist in interpreting abnormal human electrocardiograms. The thought has been that if it can be proved that with a certain artery obstructed there is a definite lesion in the heart muscle or in the conducting system, and if with

that lesion there is a definite electrocardiogram, may we not, when we encounter that abnormal electrocardiogram in the human being, particularly if he has had symptoms suggestive of coronary thrombosis, be able to state with a reasonable degree of certainty that the patient has had obstruction in a particular portion of the coronary system? May it perhaps be possible to localize a lesion in the coronary system with an accuracy comparable to that with which we locate obstructive lesions in the cerebral arteries?

NEED OF CONFIRMATION

This work needs confirmation as to the regularity of the results obtained and especially as to the interpretations and conclusions. Confirmation from necropsies is particularly desired. Patients with this condition do not present themselves very often. A large proportion with coronary obstruction die a sudden death, or are too ill to come to the office or to a hospital where they can have electrocardiographic tracings taken. And yet we have been able to take several suggestive tracings in patients in whom we believed these coronary thromboses to have occurred, and in one of the cases, Case 3, in which the patient lived five months after obstruction, the abnormal electrocardiograms gave helpful confirmatory evidence. Necropsy revealed the lesions anticipated.

TUBERCULOSIS ASSOCIATED WITH MALIGNANT NEOPLASIA

REPORT OF TWENTY CASES*

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To Rokitsansky¹ has been given the credit for teaching that tuberculosis and cancer are incompatible diseases; but since McCaskey,² in 1902, made the statement that he was strongly inclined to doubt that Rokitsansky had ever held to this extreme view, it may be well to quote direct from Rokitsansky's article on cyst formation:

Cyst formation, as a new growth, is rarely found concurrent with tubercle, either in the same organ or in the same organism generally.

With regard to tuberculosis and cancer, he said:

A similar antagonism, as shown from still more numerous observations, prevails between tubercle and carcinoma. Whenever their general correlation is susceptible of proof, cancer has seemed to succeed to tuberculosis, tubercle rarely to become developed after the extinction of cancer and its crisis.

Also:

A corresponding result of much interest is afforded by a comparison of the scale of frequency of cancer and tubercle, as well as of several special local relations of both. They are diametrically opposed to one another, as thus:

FREQUENT	RARE
Lung tubercle	Lung cancer
Ovarium cancer	Ovarium tubercle
Salivary gland cancer	Salivary gland tubercle
Stomach cancer	Stomach tubercle
Esophagus cancer	Esophagus tubercle
Rectum cancer	Rectum tubercle
Ileum tubercle, etc.	Ileum cancer, etc.

* From the Department of Surgical Pathology, Mayo Clinic.

1. Rokitsansky, C.: A Manual of Pathological Anatomy, London, Sydenham 1: 313-314, 1855.

2. McCaskey, G. W.: The Clinical Association of Cancer and Tuberculosis, with Report of a Case, Am. J. M. Sc. 124: 97-105, 1902.