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Clinical Features of Sudden Obstruction of the Coronary Arteries

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Obstruction of a coronary artery or of any of its large branches has long been regarded as a serious accident. Several events contributed toward the prevalence of the view that this condition was almost always suddenly fatal. Parry's writings on angina pectoris and its relation to coronary disease, Jenner's observations on the same condition centering about John Hunter's case, Thorvaldsen's tragic death in the theater in Copenhagen with the finding of a plugged coronary, sharply attracted attention to the relation between the coronary and sudden death. In Germany Cohnheim supported the views of Hyrtl and Henle as to lack of considerable anastomosis, and as late as 1881 lent the influence of his name to the doctrine that the coronary arteries were end-arteries; his Leipsic necropsy experience, as well as experiments on dogs, forced him to conclude that the sudden occlusion of one of these vessels or of one of the larger branches, such as the ramus descendens of the left coronary, meant death within a few minutes. Others emphasized the same view.

No one at all familiar with the clinical, pathologic or experimental features of cardiac disease can question the importance of the coronaries. The influence of sclerosis of these vessels in the way of producing anemic necrosis and fibrosis of the myocardium, with such possible results as aneurysm, rupture or dilatation of the heart, is well known. So also is the relation of the coronaries to many cases of angina pectoris, and to cardiac disturbances rather indefinitely classed as chronic myocarditis, cardiac irregularities, etc. It must be admitted, also, that the reputation of the descending branch of the left coronary as the artery of sudden death is not undeserved.

But there are reasons for believing that even large branches of the coronary arteries may be occluded—at times acutely occluded—without resulting death, at least without death in the immediate future. Even the main trunk may at times be obstructed and the patient live. It is the object of this paper to present a few facts along this line, and particularly to describe some of the clinical manifestations of sudden yet not immediately fatal cases of coronary obstruction.

Before presenting the clinical features of coronary obstruction, it may be well to consider certain facts that go to prove that sudden obstruction is not necessarily fatal. Such proof is afforded by a study of the anatomy of the normal as well as of the diseased heart, by animal experiment and by bedside experience.

The coronaries are not so strictly end-arteries, i. e., with merely capillary anastomoses, as Cohnheim and others thought. By careful dissections, by injection of one artery from another, by skiagraphs of injected arteries and by direct inspection of hearts made translucent by special methods, there is proof of an anatomic anastomosis that is by no means negligible.

Jamin and Merkel's† beautiful stereoscopic skiagrams show the remarkably rich blood-supply of the heart, with occasional anastomoses between vessels of considerable size. The possibility of injection of one coronary artery from the other is admitted even by those who deny that

†Jamin and Merkel: Die Koronararterien des menschlichen Herzens in stereoskopischen Röntgenbildern, Jena, 1907. Extensive bibliographies are contained in the articles by Thorel (Lubarsch-Ostertag's Ergebnisse, ix, Abt 1), and in Amenomiya Virchows Arch. f. path. Anat., 1910, cxcix, 187). I repeat only some of the more important references and add new ones.

such injection proves more than a capillary non-functioning anastomosis. Amenomiya,[1] by injecting hearts of young persons, showed naked-eye anastomoses in the subepicardial tissue. He feels that Hirsch and Spalteholz[2] have nearly cleared up the question as to the relation between the heart muscle and disease of the coronary artery from the anatomic standpoint. Hirsch says that in dogs the anastomosing vessels are functionally competent, and Spalteholz says that in man the vessels are nearly the same as in dogs, rich in anastomoses even in those of considerable caliber. The latter investigator, by a method of injection and treatment of the heart so as to make the muscle transparent, shows to the naked eye that there are anastomoses of considerable size.

Among others who are on record as believing that there are non-negligible anastomoses may be mentioned Haller, Huchard, Orth, Michaelis, Langer, Legg, West. All recognize, however, that there are individual differences, and also that though the heart may show rich anastomoses, these are not necessarily functional, i. e., that an artery which anatomically is not a terminal artery may yet be such functionally.

But there is proof not only of anatomic connection between the two coronaries, but that in certain instances, at least, such connection is of functional value. Experiments on lower animals and the clinical experiment of disease of the coronaries with autopsy findings show this.

Much of the earlier experimental work on the lower animals, obstructing the coronary arteries by ligatures, clamps or artificial emboli, gave promptly fatal result. Among those who worked along this line and reached these conclusions may be mentioned Erichsen (1842), Panum (1862), von Bezold, Samuelson (1880), Cohnheim and Schulthess-Rechberg (1881), G. Sée, Roche-Fontaine and Roussy (1881), Bettelheim (1892), Kronecker, and, to some extent, Michaelis. The work of Cohnheim[3] attracted particular attention and his conclusions as to end-arteries, irreparable injury, and cessation of the beat of both sides of the heart within two minutes from the time of shutting off the coronary circulation confirmed and elaborated the conclusions of the earlier experimenters, and was in turn confirmed by the French writers just named, by Bettelheim and others.

But soon dissent was heard from various quarters as to many of Cohnheim's results, and among other things as to the sudden death following the ligations. Michaelis found that the injury from ligation in rabbits was not so serious or irreparable as in dogs. Fenoglio and Drouguell, in 1888, found that some dogs might live. Porter showed that after ligation of one or two large branches of the coronary artery a dog might live hours or days. More than half his animals lived after ligation of the descending branch of the left coronary. Von Frey, at the Congress for Internal Medicine in 1891, said that he doubted the sudden stopping of the heart as Cohnheim taught; he believed that clearly the greater weight should attach to these observations in which the ligation was borne without harm; and that the stopping of the heart was not a necessary consequence of the obstruction of a large coronary branch. Hirsch in eight dogs and two apes had no sudden deaths from ligation. Bickel,[4] under Orth and Amenomiya, had a dog live nineteen days after the ligation of the descending branch of the left coronary; he killed two dogs, one on the eighth and the other on the seventeenth day after ligation. Kölster ligated smaller branches; his dogs lived, and when killed at intervals of several weeks showed the progressive changes of fibrosis of the myocardium. Imperfect technic, by which damage was done to the heart muscle and pneumothorax produced, is offered as a partial explanation, at least, for the more rapidly fatal results obtained by Cohnheim and others. Miller and Matthews[5] call attention to the better results where ether as an anesthetic is employed rather than curare or other drug. With ether they were able to ligate large branches, many of their dogs living several weeks.

Experimentally, then, sudden death, even late death, is not a necessary consequence of obstruction of even large branches, such as the descending branch of a coronary artery.

There are numerous autopsy observations, frequently with helpful clinical history, that show directly or by inference the existence of efficient anastomoses, and the ability of the heart at times to survive the obstruction of a coronary or some large branch. Some of these instructive cases may be mentioned. Pagenstecher, on account of an accident, ligated the descending branch of the left coronary artery and the patient lived five days. Thorel has seen hearts with complete obstruction of the artery, with fibrous or calcified myocardium, and yet no symptoms during life, the patient dying of some other disease. I have seen the descending branch completely occluded with an extensive fibrous area in the interventricular septum and at the apex, the latter aneurysmally dilated, where the process was clearly one of long standing. West[6] cites several cases in which at autopsy complete obstruction of one coronary was found, yet the patients had long survived this serious lesion.

Chiari, in a 32-year-old nephritic, found a sclerosed right coronary plugged by a thrombus, with resulting scattered patches of myomalacia cordis in the areas supplied by this artery. A portion of this thrombus had become detached and had embolically plugged the left coronary, resulting in sudden death. From the symptoms and the autopsy findings the thrombus in the right artery had formed at least two days before. The fact that the softened patches in the myocardium were scattered, with normal tissue between, and that the heart functionated fairly well until the left artery also was obstructed, leads Chiari to infer that anastomoses must exist between the

1. Amenomiya: Ueber die Beziehungen zwischen Koronararterien und Papillarmuskeln im Herzen, Virchows Arch. f. path. Anat., 1910, cxcix. 187.

2. Hirsch and Spalteholz: Koronararterien und Herzmuskel, Deutsch. med. Webnschr., 1907, No. 20.

3. Cohnheim and Schulthess-Rechberg: Ueber die Folgen der Kranzarterienverschliessung für das Herz, Virchows Arch. f. path. Anat., 1881, lxxxv, 503.

4. Cited by Amenomiya (See Note 2).

5. Miller and Matthews: Effect on the Heart of Experimental Obstruction of the Left Coronary Artery, Arch. Int. Med., June, 1909, p. 476.

6. West: Tr. Path. Soc., London, 1882, xxxiv, 67.

right and left coronaries. Merkel[7] drew the same inference as to anastomoses from the patchy character of the lesions in the heart of a woman of 76 years, there being normal muscle between the softened areas. The left coronary was the seat of the obstruction. He also saw in a man of 37 the left coronary closed, with nourishment through the right artery. Dock[8] in a case of gradual occlusion of the right coronary artery was able to demonstrate a direct opening of the finger branches of the left coronary into the end of the right.

Spalteholz says that we all know cases of stoppage of large vessels without large infarcts resulting. Recklinghausen and Fujinami found this condition in man, as Hirsch had in dogs and monkeys; i. e., smaller infarcts than the distribution of the vessel would lead one to expect. Galli saw complete closure of the entrance to the right coronary artery yet no change in the myocardium. By injection he found a round-about anastomosis between the right and left coronary arteries. Samuelson cites the case of a patient living five hours after obstruction, Huber one of a patient living several days. Aschoff and Tawara[9] saw a patient live fourteen days, "with nearly complete infarction of the parietal wall of the left ventricle." In several cases of angina pectoris cited by Huchard[10] the patients lived many hours after the onset of the final attack, which autopsy showed was due to a thrombotic closure of an artery. Osler refers to the fact that the patient may live for some time after obstruction. Krehl expressly states that in man the more or less sudden occlusion of an entire coronary artery, or at least a large branch, such as the descending branch, is compatible with a continuance of life.

One may conclude, therefore, from a consideration of the clinical histories of numerous cases in which there has been careful autopsy control, from animal experiments and from anatomic study, that there is no inherent reason why the stoppage of a large branch of a coronary artery, or even of a main trunk, must of necessity cause sudden death. Rather may it be concluded that while sudden death often does occur, yet at times it is postponed for several hours or even days, and in some instances a complete, i. e., functionally complete, recovery ensues.

The clinical manifestations of coronary obstruction will evidently vary greatly, depending on the size, location and number of vessels occluded. The symptoms and end-results must also be influenced by blood-pressure, by the conditon of the myocardium not immediately affected by the obstruction, and by the ability of the remaining vessels properly to carry on their work, as determined by their health or disease. No simple picture of the condition can, therefore, be drawn. All attempts at dividing these clinical manifestations into groups must be artificial and more or less imperfect. Yet such an attempt is not without value, as it enables one the better to understand the gravity of an obstructive accident, to differentiate it from other conditions presenting somewhat similar symptoms, and to employ a more rational therapy that may, to a slight extent at least, be more efficient.

The variations in the results are to be accounted for in part by variations in the freedom with which anastomosing branches occur. Presumably, too, symptoms will vary with the vessel or branches occluded. It is conceivable that with occlusion of the right coronary the symptoms might be different from those following obstruction of the left artery; systemic edema might be a consequence of the former condition and pulmonary edema of the latter. These points are, however, by no means settled either by experimental or clinical observation. The condition of the remaining vessels as to patency and presence of sclerosis must play an important part in deciding how much they are capable of doing in the way of compensatory nutrition to the anemic myocardium; the strength of the heart itself, as determined, perhaps, by old valvular or myocardial disease, would also have its influence. And presumably a sudden overwhelming obstruction, with comparatively normal vessels, would be followed by a profounder shock than the gradual narrowing of a lumen through sclerosis which has accustomed the heart to this pathologic condition and has perhaps caused collateral circulation through neighboring or anastomosing vessels to be compensatorily increased. The influence of the vessels of Thebesius is also not to be overlooked in this connection; compensatory circulation through these accessory channels may be of considerable importance in nourishing areas of heart muscle poorly supplied by sclerotic or obstructed arteries.

Attempts to group these cases of coronary obstruction according to clinical manifestations must be more or less unsatisfactory, yet, imperfect as the groups are, the cases may be roughly classified.

One group will include cases in which death is sudden, seemingly instantaneous and perhaps painless. Krehl[11] has emphasized the peculiarities of the sudden death of this type, the lack of terminal respiratory agony, of distortion of the features, of muscular contractions.

A second group includes those cases in which the attack is anginal, the pain severe, the shock profound and death follows in a few minutes or several minutes at the most.

In a third group may be placed non-fatal cases with mild symptoms. Slight anginal attacks without the ordinary causes (such as walking), perhaps some of the stitch pains in the precordia, may well be due to obstruction of small coronary twigs. Such an interpretation of these phenomena is, however, only a surmise based on the fact that other causes for the pains are lacking and that the patchy fibrosis of the myocardium that is later found at autopsy may have originated in obstruction of the sclerotic vessels; and such obstruction in small vessels may well have produced symptoms differing chiefly in degree from those caused by obstruction of larger arteries of the heart.

In a fourth group are the cases in which the symptoms

7. Merkel, H.: Ueber den Verschluss der Kranzarterien des Herzens, Festschrift für Rosenthal, Leipsic, 1906.

8. Dock, George: Notes on the Coronary Arteries, Ann Arbor, 1896.

9. Aschoff and Tawara: Die heutige Lehre von deu pathologisch-anatomischen Grundlagen der Herzschwäche, p. 56, Jena, 1906.

10. Huchard: Traité clinique des maladies du coeur, Second Edition, p. 560.

11. Krehl: Der Vetschluss der Kranzarterien; in Nothnagel's System, xv, 369.

are severe, are distinctive enough to enable them to be recognized as cardiac, and in which the accident is usually fatal, but not immediately, and perhaps not necessarily so. It is to the clinical features of this group that attention is directed in what follows.

By way of introduction, I give in outline the history of a case, experience with which acutely attracted my attention to this subject.

CASE 1.—*History.*—A man, aged 55, supposedly in good health, was seized an hour after a moderately full meal with severe pain in the lower precordial region. He was nauseated and, believing that something he had just eaten had disagreed with him, he induced vomiting by tickling his throat. The pain continued, however, and his physician was called, who found him cold, nauseated, with small rapid pulse, and suffering extreme pain. The stomach was washed out and morphin given hypodermically. The pain did not cease until three hours had passed. From this time the patient remained in bed, free from pain, but the pulse continued rapid and small, and numerous râles appeared in the chest. When I saw him twelve hours from the painful attack his mind was clear and calm; a moderate cyanosis and a mild dyspnea, were present. The chest was full of fine and coarse moist râles; there was a running, feeble pulse of 140. The heart tones were very faint and there was a most startling and confusing hyperresonance over the chest, the area of heart dulness being entirely obscured. The abdomen was tympanitic. The urine was scanty, of high specific gravity, and contained a small amount of albumin and a few casts. The temperature was subnormal, later going to 99 F. Occasionally there was nausea and twice a sudden projectile vomiting of considerable fluid material. This condition remained with slight variations up to the time of death, fifty-two hours after the onset of the pain, though at one time the râles seemed nearly to have disappeared. A few hours before death the patient described a slight pain in the heart region, but said it did not amount to much. A remarkable circumstance, and one that occasioned surprise in those who saw the patient and who realized from the almost imperceptible pulse and the feeble heart tones how weak the heart must be, was the fact that he frequently indulged in active muscular effort without evident harm. He rolled vigorously from side to side in the bed, sat suddenly bolt upright, or reached out to take things from the table near by; and once, feeling a sudden nausea, he jumped out of bed, dodged the nurse and ran into the bathroom, where he vomited; and yet he seemed none the worse for these exertions.

Necropsy (Dr. Hektoen).—The heart was of normal size, but both coronary arteries were markedly sclerotic, with calcareous districts and narrowing of the lumen. A short distance from its origin the left coronary artery was completely obliterated by a red thrombus that had formed at a point of great narrowing. The wall of the left ventricle showed well-marked areas of yellowish and reddish softening, especially extensive in the interventricular septum. At the very apex the muscle was decidedly softer than elsewhere. The beginning of the aorta showed a few yellowish spots, these areas becoming less marked as the descending part was reached. An acute fibrinous pericardial deposit, which showed no bacteria in smears, was found over the left ventricle. (The pericarditis probably explains the slighter pain complained of a few hours before death.) There was marked edema of the lungs. In other respects the anatomic findings were those of health.

A colleague personally related to me the case of a man of 60 who three days after a severe anginal seizure, felt well enough to walk on the street, though with some dyspnea. He died suddenly on the fifth day. The obstruction in the left coronary, and the muscular softening found at autopsy were similar to those in the case just described.

Since my attention has been called to this condition, I have seen five other cases that I am convinced were instances of coronary thrombosis, the patients living many hours after the accident, though no autopsy control confirms this opinion. All were men beyond 50. In all there was some evidence of peripheral arteriosclerosis; all had had previous anginal attacks. In all the final attack was described as the severest and most prolonged in the experience of the patient. Morphin alone had given relief. In all the sudden development of a weak pulse, with feeble cardiac tones, was a striking feature; the pulse was generally rapid. Dyspnea and cyanosis varied in degree. Râles, moist and dry, were usually present. Emphysema was present to a moderate degree in two of the five. Only one patient left his bed after the attack. His pulse showed great improvement as to quality and rate, though dyspnea, râles, edema of the legs, albumin, increased area of cardiac dulness, etc., showed failure of the heart muscle. From the time of the seizure, i. e., the time of the obstruction, to death was in one case three days, in one seven, in two twelve, and in one, twenty days.

One of these cases is, it seems to me, a typical one of this sort and, though necropsy is lacking, I venture to give the history.

CASE 2.—The patient was a man of 65, of exemplary habits. His health had been good up to three years before, when he noticed at times a tight feeling in the precordia on walking. For the past three months typical anginal seizures often compelled him to stop after walking two or three blocks. Three days before he had had a moderately severe angina. Thirty-six hours before I first saw him, in the night he made a noise, awakening his wife. For a few seconds he was, perhaps, unconscious. He complained of unbearably severe pain in the upper stomach region; the pain did not radiate. He was nauseated and belched gas freely. His physician saw him inside of twenty minutes and gave sodium bicarbonate, which was vomited. The pain continuing, a hypodermic injection of morphin was necessary. The patient was pale, covered with cold sweat and had a small, rapid pulse. His appearance was that of collapse. His distress seemed to him largely abdominal.

When I saw him his color had returned and he was ruddy-cheeked. He complained of extreme weakness. His mind was clear. There was a little cyanosis, and respiration was somewhat labored. There were numerous râles in the chest. The pulse was 110 and small. The heart tones were faint; there was no murmur. The heart was a trifle enlarged, as it had been for some years. The area of cardiac flatness was decidedly small on account of overlying lung. The liver dulness was but a narrow band along the costal margin; the edge of the liver could be palpated. No spleen could be made out. The urine contained a distinct ring of albumin and a few granular and hyaline casts. There was a doubtful faint trace of bile. On digalen and nitroglycerin there seemed to be some improvement in the quality of the pulse.

At a second visit the condition was much the same. There had been a few periods of more marked oppression in breathing, with some increase in cyanosis and weakness of the pulse.

At a third visit, Oct. 19, 1910, it was learned that the patient had had a bad night, with severe attacks of dyspnea. The pulse had been but barely perceptible at the wrist and beat 120 to the minute. At 5 a. m. both the physician and the patient himself had felt that death was at hand. The patient had rallied,

however, and when I saw him was conscious, with very feeble pulse of 110, and barely perceptible heart tones. He was extremely weak. Breathing was of the Cheyne-Stokes type. The patient seemingly dozed during apnea, yet answered questions. What I took to be a faint pericardial friction could be made out over the lower left sternal border. The patient said he was not in pain. He declared that he obtained relief from swallowing orange juice, which he repeatedly sipped. He remained in this condition for sixteen hours longer. From the onset of his severe anginal attack to death was seven days.

The instructive case of Professor Panum is described by Fraentzel.[12] For a few weeks Panum had noticed dyspnea and a tight feeling on going up stairs. May 1, 1885, coming home in the wind, he stopped often, and on reaching home had a sudden, severe, tearing pain in the precordia, running out to the left arm and fingers. The pulse became rapid, small and irregular. The patient broke out into a profuse sweat. He was nauseated and induced vomiting by tickling his throat. The physical findings are not accurately known. The mind was clear to the last. Death occurred suddenly about fourteen hours after the onset of symptoms. At the necropsy both upper lobes of the lungs and the middle lobe were found emphysematous. The left coronary artery was atheromatous, narrowed, and a white soft thrombus was attached to the wall. The musculature of the left ventricle was degenerated and softened and had ruptured just to the left of the septum.

Engelhardt[13] describes the case of a man of 51 in whom, after a thrombosis of the left coronary artery with suddenly developing gastric and abdominal symptoms, there was an illness of eight days, with fever, meteorism, vomiting, oppression, and then in a tachycardial attack rupture of the anterior wall of the left ventricle, with hemopericardium. Death twelve hours after the rupture. The symptom-complex resembled the picture of the abdominal-pectoral vascular crises (Pal).

A study of cases of this type shows that nearly all are in men past the middle period of life. Previous attacks of angina have generally been experienced, though, as shown by my first case, the fatal thrombosis may bring on the first seizure. The seizure is described by patients who have had previous experience with angina as of unusual severity, and the pain persists much longer. In some instances there has been no definite radiation of the pain, as to the neck or left arm, though this may have been a feature of other anginal attacks, and the pain, as in these two cases, may be referred to the lower sternal region or definitely to the upper abdomen. Cases with little or no pain have been described. In Chiari's case pain is not referred to, the patient though with slow, irregular and weak pulse being out of bed. The obstruction of the right coronary was, as Chiari says, "so to speak, latent." Thorel also refers to a painless case. Some of Huchard's cases with obstruction did not show anginal pain. Nausea and vomiting, with belching of gas, are common. There may be tympany. Ashy countenance, cold sweat and feeble pulse complete the picture of collapse. The attention of the patient and the physician as well may, therefore, be strongly focused on the abdomen, and some serious abdominal accident be regarded as the cause of the sudden pain, nausea, collapse. The cardiac origin may be the more easily overlooked when there has been no previous typical angina, and when, as may happen (Case 1), there is no arteriosclerosis manifested peripherally and no enlargement of the heart to be made out.

Cohnheim found that in dogs the pulse after obstruction was slow. This may be so in the thrombotic obstruction of disease in man. In Hammer's case[14] the pulse dropped from 80 to 8 per minute, the patient living thirty hours from the onset of the symptoms that marked the closure of the right coronary opening. A rapid pulse is frequently seen however. The pulse may be irregular. A striking feature has been its weakness. In two patients I have seen a rapid, thready, almost imperceptible, radial pulse, of such a quality that if met with in pneumonia or typhoid fever it would have warranted one in presaging death within a few minutes or hours. Yet one patient lived forty hours and another four or five days with a pulse of this quality. Blood-pressure is low. The heart tones have been feeble—in fact, often startlingly feeble. Feeble contraction of the weakened, anemic heart muscle accounts for the weak pulse and the weak tones. Still another reason for the faint tones is found in the acute emphysema—*Lungenschwellung* and *Lungenstarrheit* of von Basch—by which condition the heart sounds are obscured by overlapping air-containing lungs. This also makes it difficult to map out the outlines of the heart and, coupled with the feeble apex impulse, may make such an examination for the size of the heart very unsatisfactory.

Dyspnea and cyanosis have been variable, at times much less than one would expect from the character of the accident and the quality of the heart's action. Râles, dry and moist, have been present in many cases, in some, as in my first case, largely moist, diffuse, not very large. Here there was a moderate amount of thin, frothy, slightly blood-tinged fluid expectorated, as in edema of the lungs, which condition was found at the autopsy. I mention this because some, with Cohnheim, contended that the conditions for edema would not be produced by coronary obstruction, as both right and left heart ceased beating simultaneously. Others, e. g., Samuelson, Bettelheim and Michaelis, found edema. My case shows such edema. Possibly the right heart may have remained relatively stronger than the left after the accident, and so Welch's condition for edema has been presented.

The weakness of the heart and the low blood-pressure will account for the scanty urine and the trace of albumin. A palpable liver may likewise owe its enlargement to passive congestion.

Nearly always the mind is clear—at times unusually clear—until toward the last. Some patients seem conscious, as is so common in angina, that they are face to face with death, but in none that I have seen has there been uncontrollable fear or the restlessness of fright. The seriousness of the accident seemed to be realized, but there was no panic. Perhaps the relief from the agony of the initial pain causes an unnatural mental calmness.

General weakness has been marked in some cases, in

12. Fraentzel: Krankheiten des Herzens, Berlin, 1892, iii, 51.

13. Engelhardt: Ein Fall von Herzruptur, Deutsch. med. Wchnschr., xxxv, 1919, No. 19, p. 838.

14. Hammer: Wien. med. Wchnschr., 1878, No. 5.

others not. One patient showed for more than a week an asthenia comparable to that of the terminal stage of pernicious anemia or Addison's disease. He hesitated to move in bed for the further reason that even turning on the side caused him the sensation as though the heart were giving out. Even slight movement caused some pain. His case is representative of the type of status anginosus. Obrastzow regards this as the usual manifestation of coronary thrombosis. My experience shows that such obstruction may be followed by a complete cessation of pain for hours, and even to the time of death. Some of these patients of the latter type will, if permitted, move freely or even get out of bed.

The occurrence of a serofibrinous exudate over the area of myocardial softening, with roughening of the pericardium, has been noted in several instances. This may explain a later precordial distress, as in Case 1. A fine pericardial friction, therefore, occurring several hours or a few days after the initial pain, may be confirmatory evidence of coronary obstruction. Osler[15] concluded, in one of his cases of angina, that the attack was probably associated with acute infarct of the ventricle, "as a pericardial rub was detected the next day." Dock[8] recognized this pericarditis *intra vitam* in one of his cases and found it post mortem over the softened area. In one of Leyden's cases in which the patient lived five days from the onset of symptoms of dizziness, faintness, small pulse, there was found myomalacia cordis, and especially at the apex, where a softened area reached to the surface, there was pericarditis; cloudy fluid was in the pericardial sac. This was almost certainly a case of coronary obstruction, though the occluding lesion is not described. This pericarditis is in keeping with some of the experimental work on lower animals, e. g., that of Bickel, who in his dogs killed nineteen and seventy days after ligation found localized pericarditic adhesions over the area representing the myocardial softening.

Death is the result in nearly all of these cases. Yet it may be delayed for many days. More than this, there is, as has been shown by reference to experimental work, no intrinsic reason why some patients with obstruction of even large branches of the coronary artery may not recover. Experimental animals sometimes do. And, as already said, mild cases must occur, and one cannot pretend to say where the dividing line should be drawn between the mild obstruction of a coronary branch, whose recovery means a few fibrous patches in the myocardium, and the more serious one that in a few days is to lead to rupture of the heart or is to produce an extensive weakened fibrous area that will ultimately yield in cardiac aneurysm. Death may then be caused by rupture, by sudden asystole, or by gradual giving out of the weakened heart muscle—by "ingravescent systole," as Balfour[16] styles it—a mode of death occupying from half an hour to a week, illustrated by one of his cases in which death occurred one week after the obstruction, which was found post mortem. In one instance in which I believe the anginal seizure was thrombotic a dilatation of the heart, with orthopnea, dropsy, etc., followed the seizure. Death here was, as in cardiac failure, from other causes. Some of the dogs of Miller and Matthews died in this way several weeks after the ligation of the coronary. In cases in which the heart slowly wears out in the course of a few days, Cheyne-Stokes respiration, general asthenia, urinary scantiness, with mental apathy and exhaustion may be present.

Emphasis ought to be laid on the resemblance of some of these cases to surgical accidents. The sudden onset with pain over the lower sternal and epigastric region, the nausea and vomiting, the tympany, the feeble pulse, ashy color, cold sweat and other signs of collapse make one think of such conditions as gall-bladder disease, acute hemorrhagic pancreatitis, perforation of gastric or duodenal ulcer, hemorrhage into the adrenal capsule, etc. The dyspnea, hyperresonant thorax, obscured heart tones, may suggest pneumothorax or diaphragmatic hernia. In my first case, while the diagnosis made was that of cardiac accident, there were so many disquieting features that surgical counsel was called to make sure that some surgical accident, such as those enumerated, had not been overlooked. Details as to differential diagnosis need not be given. Where there is arteriosclerosis, enlarged heart, a history of previous angina, typical radiation of the pain to the neck and arm, the diagnosis will not be so difficult as where these suggestive aids are lacking. The bilateral character of the emphysema, the persistence of breath sounds, often with râles, the failure of the heart to be dislocated, will help exclude pneumothorax and diaphragmatic hernia. The absence of blood from the vomitus, the absence of peritonitic tenderness, a study of the temperature, the leukocytes, etc., will help in excluding subdiaphragmatic accidents.

Obrastzow[17] calls particular attention to this resemblance to surgical accidents which my own experience corroborates. Engelhardt's case also illustrates this point.

If these cases are recognized, the importance of absolute rest in bed for several days is clear. It would also seem to be far wiser to use digitalis, strophanthus or their congeners than to follow the routine practice of giving nitroglycerin or allied drugs. The hope for the damaged myocardium lies in the direction of securing a supply of blood through friendly neighboring vessels so as to restore so far as possible its functional integrity. Digitalis or strophanthus by increasing the force of the heart's beat, would tend to help in this direction more than the nitrites. The prejudice against digitalis in cases in which the myocardium is weak is only partially grounded in fact. Clinical experience shows this remedy of great value in angina, and especially in cases of angina with low blood pressure, and these obstructive cases come under this head. The timely use of this remedy may occasionally in such cases save life. Quick results should also be sought by using it hypodermically or intravenously. Other quickly acting heart remedies would also be of service.

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15. Osler: Lumleian Lectures on Angina Pectoris, Lancet. London. March 12 and 26, and April 9, 1910.

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17. Obrastzow and Straschesko: Zur Kenntniss der Thrombose der Koronararterien des Herzens, Ztschr. f. klin. Med., 1910, lxii, 116.