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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 Leipzig 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, 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 anastomosis that is by no means negligible.

Jamin and Merkel's† beautiful stereoscopic skiagraphs 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 such injection proves more than a non-functioning anastomosis. Amenomiya, examining hearts of young persons, showed naked anastomoses in the subepicardial tissue. He feels that Haller and Spalteholz<sup>2</sup> have nearly cleared up the question 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, in anastomoses even in those of considerable caliber. The latter investigator, by a method of injection 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, V. All recognize, however, that there are individual differences, and also that though the heart may show anastomoses, these are not necessarily functional, and 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 experience in disease of the coronaries with autopsy findings support this.

Much of the earlier experimental work on the heart in animals, obstructing the coronary arteries by ligature clamps or artificial emboli, gave promptly fatal results. Among those who worked along this line and reached these conclusions may be mentioned Erichsen (1818), Panum (1862), von Bezold, Samuelson (1880),

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

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

2. Hirsch and Spalteholz: Koronararterien und Herz, Deutsche med. Wochenschr., 1907, No. 20.

and Schulthess-Rechberg (1881), G. Sée, Roche-  
e and Roussy (1881), Bettelheim (1892),  
ker, and, to some extent, Michaelis. The work  
nheim<sup>3</sup> attracted particular attention and his con-  
as as to end-arteries, irreparable injury, and ces-  
of the beat of both sides of the heart within two  
es from the time of shutting off the coronary  
ation confirmed and elaborated the conclusions of  
arlier experimenters, and was in turn confirmed  
e French writers just named, by Bettelheim and  
s.

t soon dissent was heard from various quarters  
many of Cohnheim's results, and among other  
s as to the sudden death following the ligations.  
aelis found that the injury from ligation in rabbits  
not so serious or irreparable as in dogs. Fenoglio  
Drouguell, in 1888, found that some dogs might

Porter showed that after ligation of one or two  
branches of the coronary artery a dog might live  
s or days. More than half his animals lived after  
ion of the descending branch of the left coronary.

Trey, at the Congress for Internal Medicine in 1891,  
that he doubted the sudden stopping of the heart  
ohnheim taught; he believed that clearly the greater  
t should attach to those observations in which the  
was borne without harm; and that the stopping  
t was not a necessary consequence of the  
of a large coronary branch. Hirsch in  
dogs and two apes had no sudden deaths from ligation.

Bickel,<sup>4</sup> under Orth and Amenomiya, had a dog  
nineteen days after the ligation of the descending  
ch of the left coronary; he killed two dogs, one  
e eighth and the other on the seventeenth day after  
on. Kölster ligated smaller branches; his dogs  
, and when killed at intervals of several weeks  
ed the progressive changes of fibrosis of the myo-  
ium. Imperfect technic, by which damage was  
to the heart muscle and pneumothorax produced,  
ered as a partial explanation, at least, for the more  
ly fatal results obtained by Cohnheim and others.  
er and Matthews<sup>5</sup> call attention to the better results  
e ether as an anesthetic is employed rather than  
e or other drug. With ether they were able to  
large branches, many of their dogs living several  
s.

perimentally, then, sudden death, even late death,  
t a necessary consequence of obstruction of even  
branches, such as the descending branch of a  
ary artery.

ere are numerous autopsy observations, frequently  
helpful clinical history, that show directly or by  
ence the existence of efficient anastomoses, and the  
y of the heart at times to survive the obstruction  
coronary or some large branch. Some of these  
ctive cases may be mentioned. Pagenstecher, on  
nt of an accident, ligated the descending branch  
e left coronary artery and the patient lived five days.  
el has seen hearts with complete obstruction of  
artery, with fibrous or calcified myocardium, and  
o symptoms during life, the patient dying of some  
isease. I have seen the descending branch com-  
occluded with an extensive fibrous area in the  
entricular septum and at the apex, the latter  
smallly dilated, where the process was clearly one

of long standing. West<sup>6</sup> 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 sup-  
plied by this artery. A portion of this thrombus had  
become detached and had embolically plugged the left  
coronary, resulting in sudden death. From the symp-  
toms 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 scat-  
tered, 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 right and left coronaries. Merkel<sup>7</sup>  
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<sup>8</sup>  
in a case of gradual occlusion of the right coronary  
artery was able to demonstrate a direct opening of the  
finer branches of the left coronary into the end of the  
right.

Spaltholz says that we all know cases of stoppage of  
large vessels without large infarcts resulting. Reckling-  
hausen 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 myocar-  
dium. By injection he found a round-about anastomo-  
sis between the right and left coronary arteries. Sam-  
nelson cites the case of a patient living five hours after  
obstruction, Huber one of a patient living several days.  
Aschoff and Tawara<sup>9</sup> 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<sup>10</sup> 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 experi-  
ments and from anatomic study, that there is no in-  
herent reason why the stoppage of a large branch of a co-  
ronary 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, recov-  
ery ensues.

The clinical manifestations of coronary obstruction  
will evidently vary greatly, depending on the size, loca-  
tion and number of vessels occluded. The symptoms and

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

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

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

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

10. Huchard: *Traité clinique des maladies du coeur, Second Edition, p. 500.*

Bettelheim and Schulthess-Rechberg: *Ueber die Folgen der Arterienverschliessung für das Herz, Virchows Arch. f. path. Anat., lxxxv, 503.*

Porter: *Ibid.* cited by Amenomiya (See Note 2).

Pagenstecher and Matthews: *Effect on the Heart of Experimental Occlusion of the Left Coronary Artery, Arch. Int. Med., June, 1891, p. 476.*

end-results may also be influenced by blood-pressure, by the condition 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<sup>12</sup> 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 the causes for the pains are lacking and that the changes of the myocardium that is later found may have originated in obstruction of the twigs and such obstruction in small vessels

der Verschluss der Kranzarterien; in Nothnagel's

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 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. Occasional 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 soft-

ing found at autopsy were similar to those in the case 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 twenty-four hours after the accident, though no autopsy could confirm 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 attack was described as the severest and most prolonged in the experience of the patient. Morphine alone did not give 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 in quality and rate, though dyspnea, râles, edema of the legs, albumin, increased area of cardiac dullness, 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 had compelled him to stop after walking two or three blocks. Three days before he had had a moderately severe angina. Twenty-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 morphine 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 wholly abdominal.

When I saw him his color had returned and he was ruddy-checked. 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 heart 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 abnormally small on account of overlying lung. The liver dullness 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 digitalin 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 at 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 cyanotic type. The patient seemingly dozed during the day, 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 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.<sup>12</sup> 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<sup>13</sup> describes the case of a man of 54 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 during the attack was slow. This may be so in the human case. The question of disease in man. In Hammer's

12. Fraentzel: Krankheiten des Herzens, Bonn, 1885.  
13. Engelhardt: Ein Fall von Herzruptur, Wchnschr., xxxv, 1910, No. 10, p. 838.  
14. Hammer: Wien. med. Wchnschr., 1878, No.

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 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<sup>15</sup> 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<sup>8</sup> recognized "pericarditis vera nilam" in one of his cases and found "pericarditis vera nilam" softened area. In one of his cases the patient lived five days from the onset of the attack, with faintness, small pulse, and especially at the time the heart sounds reached to the surface, there was a pericardial friction. Fluid was in the pericardial sac. This certainly is a case of coronary obstruction, but the nature of the lesion is not described. This pericarditis, in agreement with some of the experimental work on 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 leads to rupture of the heart or is to produce an extensive weakened fibrous area that will ultimately yield to 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." Balfour<sup>16</sup> styles it—a mode of death occupying from an hour to a week, illustrated by one of his cases in which death occurred one week after the obstruction, was found post mortem. In one instance in which I believe the anginal seizure was thrombotic or dilatation of the heart, with orthopnea, dropsy, etc., followed the seizure. Death here was, as in cardiac failure, of various causes. Some of the dogs of Miller and Balfour died in this way several weeks after the ligation of the coronary artery. In cases in which the heart slowly weakened over 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, the ashy color, cold sweat and other signs of collapse may lead one to 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 and enlarged heart, a history of previous angina, typical re-

15. Osler: Lancelan Lectures on Angina Pectoris, *Lancet*, London, March 12 and 20, and April 9, 1910.

16. Balfour: Clinical Lectures on Diseases of the Heart, Edinburgh, 1898, pp. 310 and 328.

The standard of public drinking-places may be classed in much the same way as the eating-places, with this exception: even in places of the highest class, glasses and bottles are only occasionally sterilized. In lieu of the foregoing conditions I would suggest the following:

1. The enactment of laws relative to the sanitary condition of public eating- and drinking-places, which shall provide for (a) the personal hygiene of owners and employees and (b) sanitary precaution on the part of owners and employees.

2. The owner of each and every public eating- and drinking-place shall be licensed under such law, such license to continue in force as long as there is no violation thereof.

3. Any and every glass, cup, dish and other eating- or drinking-vessel or utensil used in any hotel, saloon, restaurant, drug-store, soda-fountain, or other place of public refreshment in the state shall be sterilized in steam or boiling water after being used by any patron and prior to any further use thereof in eating or drinking by any other patron; and no such glass, cup, dish or other eating- or drinking-vessel or utensil after use by one patron in eating or drinking, shall be offered or be permitted to be used by any other patron without having first been thoroughly cleansed and then sterilized in steam or boiling water.

4. No person, firm or corporation shall at any time or place keep, expose, offer or prepare for sale, or shall store pending sale, any article or substance of human food or drink in any place or premises which is not, at such time, screened so as to prevent insects from having access to such article or substance; and no person, firm or corporation shall at any time or place keep, expose, offer or prepare for sale, or shall store pending sale, any article or substance of human food or drink in any place or premises unless such article or substance is pure and wholesome and entirely free from contagious and infectious bacteria.

5. No drinking-vessel, used in common or to be used in common, shall be kept, exposed, provided or permitted in any railway station, public or private school, public playground, public park, public building or in any place to which the public are admitted.

6. No drinking-vessel used in common shall be exposed, kept, provided or permitted in any mill, factory, railway train, ship, boat, hospital, private or public institution, public works or in any place where a number of persons are employed or assembled.

7. No table-linen or coverings, towels, cloths, sponges, soap, combs or water used in common or to be used in common shall be exposed, kept, provided or permitted in any public eating- or drinking-place.

8. No person or persons shall be served food or drink at any public eating- or drinking-place until they shall have thoroughly cleansed the hands and face with soap and water, as provided for in Suggestion 7.

9. No person or persons known to be suffering from an infectious or contagious disease shall be served food or drink at or in any public eating- or drinking-place unless isolated from other patrons under proper sanitary conditions, in order that no other patron shall in any way be exposed or endangered in any manner whatsoever to said infection or contagion.

It may be argued that no sanitary system can supplant present conditions unless entailing great expense and inconvenience. To this I agree; but whatever is humane and promotes the progress of civilization is expedient and necessary. I would therefore suggest the following as a few of the many things that may be

used: sanitary paper cups, straws, cupless attachments on faucets, sanitary systems of drinking-fountains in mills, factories, stores, institutions, etc., bubbling fountains, which make cups unnecessary, paper napkins, individual towels, cloths, soap; installation of systems for rapid cleansing and sterilization of dishes, glasses, cups or other utensils, used in or about public eating or drinking places.

Most, if not all, of the state boards of health have condemned the public drinking-cup. The progressive cities have passed ordinances prohibiting the use of the public cup. St. Louis has taken advanced measures to enforce the cleansing of any and every glass, cup, dish and other eating- or drinking-vessel or utensil used in or at any public eating- or drinking-place, in running water or clean water. This ordinance is a great stride in the right direction, but falls far short of the mark.

Railway companies announce that passengers do not demonstrate any great desire for individual sanitary cups. Other large corporations maintain that there is no particular benefit derived from the use of the individual cup, and further that the added expense is necessarily great. The same argument comes from the individual in respect to slot machines, paper napkins, etc.

The solution of this great problem of sanitary control of public eating- and drinking-places depends on the elimination of two factors—avarice and ignorance. The mercenary motive of the individual, as regards hygienic and sanitary methods, predominates over his moral obligation to himself and his fellow men. Hence the sequence of his reasoning leads to antagonism of laws respecting sanitary science, basing a great deal on the aphorism that “the Lord giveth and the Lord taketh away.” The community reasons that the enactment of laws relative to the control of sanitary conditions brings on it added hardships in the form of increased tax-rate, higher cost of living, physical discomfort and infringement on its personal liberty.

The state, until a comparatively recent date, did not take marked interest in sanitary affairs. It does not at the present time consider seriously the individual. The most notable advances made by the state have been when great commercial interests were endangered. It is said that “Ignorance of the law excuses no one.” Such being the case, we have one of two ways of solving a serious problem. The laws must be enacted after the mosaic method and then enforced, which is a most plausible way, or the laws must lie dormant until the masses are educated or have reached a higher standard of civilization.

Realizing that any community that wishes to be prosperous must be healthy, sanitarians have taken the initiative in formulating laws to that end, and have accomplished more for their respective communities than has been achieved in this or any other country in the last 2,000 years. This may seem a broad assertion, but is it not true? The brilliant and noble work of Gorgas, White, Blue, Stiles, Sternberg, Wyman, Guiteras, Carroll, Lazear and associates who forged the chain enabling the United States government to have and to hold its many possessions, had the sanction and legal support of the government.

Then must we work and wait until we are in the face of a great commercial enterprise, or until we are in the throes of epidemics of typhoid, diphtheria, scarlet fever, small-pox, cholera, yellow fever or bubonic plague before we awaken to the necessity of legislative measures of sanitary control? No! there is one reality—our

duty. It demands of us as medical men and sanitarians to let the light of our knowledge shine and point out the way. Let us blot out the black pages of the last 2,000 years and take up the work that the ancient Babylonians, Jews and Egyptians so nobly began. It is for sanitarians to apply sanitary science to the affairs of civilization; the masses depend on sanitarians for leaders and teachers.

We know what the epidemics of the past have cost us. We know how much we have accomplished and how far we have advanced, but we are not left without a guide. It is true we do not have a Moses to lead us, but we have more in Hurty, Bracken, Evans, Dixon and their kind, great broad-minded men, unselfish, forceful and considerate of the humane advancement of sanitary propaganda. These men have taken the initiative in health work. Now let us have the hearty cooperation of every member of the American Medical Association—every person, it matters not his walk in life, interested in the progress of civilization, in securing for every state in the United States laws relative to the sanitary control of public eating- and drinking-places. It should therefore be our earnest endeavor and duty to make hygiene and sanitary science one of the basic principles of our civilization.

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## THE CONTROL OF CONTAGIOUS DISEASES IN A MUNICIPALITY \*

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DETROIT

Accepting the word "contagious" in its broadest sense, we have a subject before us which would make a better title for a book than for a discussion of this kind, and yet I desire at the start to say that I shall use the word "contagious" as synonymous with "infectious" and "communicable," and speak of contagious diseases as those diseases which are capable of transmitting infection from one person to another in one way or another.

If I had been asked ten years ago to give my opinion as to how to control contagious diseases in a municipality, I would have said "by immunization, isolation and disinfection"; but after more than ten years in this field of work, it would seem that the answer would be even more simple, namely, "by education." It is true to-day, as it was then, that some acute contagious diseases, principally small-pox and diphtheria, are controllable by immunization, the former by vaccination, the latter by the use of diphtheria antitoxin; but in order to get good results from these methods, it is necessary to have the people of a community so educated that they will submit to them readily and whenever the health officer finds it necessary for them to do so. Immunization is undoubtedly valuable in the control of some of the other diseases, among which may be mentioned typhoid fever, but it would be difficult to make any great use of this method in any municipality at the present time because of the lack of knowledge of this procedure on the part of the community. Isolation, the second method which has been in vogue for a number of years for the control of contagious diseases, is desirable and satisfactory when it can be employed, but it is necessary first to have knowledge of a case before it can be isolated. It was because

of the fact that so many cases of acute contagious diseases, particularly diphtheria, scarlet fever, measles, German measles and others of that nature were never reported and consequently escaped the attention of the public health officials and could not be isolated, that a system now known as medical inspection of school children was first introduced.

Medical inspection of schools, as it is better known, comprises at the present time not only the detection of contagious diseases among school children but also the detection and correction of physical defects and the study of mental defects; but it was originally planned for the purpose of detecting cases which otherwise escaped the attention of the authorities.

In considering the isolation of cases of contagious diseases, the question of hospitalization comes to our mind and with it the fact that many cases of contagious diseases cannot be properly isolated in private houses. I would not take the position that all cases of all contagious diseases, including typhoid and tuberculosis, should be hospitalized, but on the other hand, I think it is absolutely necessary for the success of the preventive measure known as isolation that in each municipality there should be a properly equipped and constructed municipal hospital for contagious diseases under the control of the board of health in which such patients as cannot properly be isolated in their own homes may be cared for. By having facilities for early reception of such patients, it will be possible to reduce not only the morbidity but also the mortality from contagious diseases.

Disinfection is the one method of the three that I have mentioned that has been bitterly attacked by some progressive sanitarians, among whom may be mentioned Dr. Charles V. Chapin, of Providence, R. I. In a paper<sup>1</sup> read before the American Public Health Association in 1910, Dr. Chapin took the position that "terminal disinfection," or disinfection of a room or house after a case of contagious disease has been terminated, really has no value, and he showed by a series of figures that in Providence, where "terminal disinfection" had been discontinued, the results obtained with regard to additional cases arising in the houses in which there had been an original case, were almost as good without "terminal disinfection" as when it had been performed. In spite of this opinion, and the opinion of other progressive men, the fact remains that "terminal disinfection," properly done, does disinfect; in other words, that formaldehyd used in sufficient quantities for sufficient lengths of time, in a room which has been properly prepared, does destroy such infection as may be within the room. Of course the method of disinfecting for the purpose of controlling the spread of contagious diseases should not be limited to terminal house disinfection by formaldehyd or sulphur, but should consist in a disinfection during the course of the disease of everything that has been known to have become infected. All materials which have no intrinsic value and which have become infected by contact with the patient during the course of the disease should be destroyed by fire, and whenever this procedure cannot be followed, we should resort to boiling, immersing in chemical solutions, or some other method equally satisfactory.

If it were possible to apply one or more of these methods, namely, immunization, isolation and disinfection, to all of the communicable diseases within a municipality, there can be no question but that the result

\* Read in the Section on Preventive Medicine and Public Health of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

1. Chapin, C. V.: The Value of Terminal Disinfection, Jour. Am. Pub. Health Assn., January, 1911.