ON THE DETERMINATION OF THE CAUSE OF DEATH AND SOME CASES OF SUDDEN DEATH.

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ON THE DETERMINATION OF THE CAUSE OF DEATH AND SOME CASES OF SUDDEN DEATH.

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The determination of the cause of death is one of considerable importance, at the same time frequently a difficult and perplexing question to solve. The cause of this difficulty lies in the number of complicating circumstances which enter into its production. Death is never a simple, single phenomenon occurring suddenly during an individual's life. It is the result of a chain of events which finally ends in a climax; the latter, by syncope or asphyxia, initiates the general extinction of all the phenomena of life.[1]

If, therefore, we investigate the problem in detail, we see that ultimately the immediate cause of death is only the last link of a chain and may well be separated from the remote. But the knowledge of the former may not necessarily allow us to form an opinion about the latter. A physician, for instance, may be called to a patient suffering from the symptoms of a well marked œdema of the lungs. The patient is acutely ill and may die with it. Here, the immediate cause of death is apparently perfectly plain, but no scientific physician or recorder of vital statistics would be satisfied with this knowledge. Indeed, it may on further investigation even appear

[1]For a discussion of the definition of the conception of death, see Jores, Feststellung der Todesursache aus dem Leichenbefund in *Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie*, xiii Jahrgang ii Abteil., 1909, and Orth, Was ist Todesursache? in *Berliner klinische Wochenschrift*, No. 10, 1908.

doubtful whether this œdema, and this applies to a large number of the so called immediate causes of death, could be regarded as the actual cause, and not as a terminal accompaniment of the fatal catastrophe. For we have learned to regard œdema of the lungs, as well as that of the meninges, in many cases at least, as agonal phenomena.

Again, every one knows that ultimately death is ushered in by a permanent cessation of the heart and respiration, the so called *atria mortis* of the older writers. Whatever may be the processes leading to death, the immediate initiation of the phenomenon depends on the stoppage of one or the other of these processes. To declare, therefore, that a person dies of heart disease or of œdema of the lungs, or of apoplexy, is not only insufficient, but hardly goes beyond an observation which almost every one is able to make.

Such diagnoses, however, are frequently made, even by physicians, in cases of so called sudden deaths, that is, when a patient appears to enjoy health when stricken, or, having suffered from a long continued disease, suddenly dies. Not infrequently, in such cases, even the immediate cause of death may have been so obscure that a physician is unable to form any opinion and to a much less extent about the processes which have led up to it. In this connection it must be remembered that the external examination of the body after death is very rarely able to give us any conclusive evidence, and occasionally hardly a clue to what may have been the immediate *atrium mortis*. An apodictical verdict of the cause of death, after a view of the body, would seem strange to one technically trained, did we not appreciate that such diagnoses are often made as a matter of convenience to the family and to the physician.

I will not touch here upon the purely scientific and the every day criminal medicolegal aspect of the situation, but there are essentially two very im-

portant practical questions which can be truthfully answered only if every circumstance surrounding and leading up to the death of a patient is known. The first is with regard to useful, trustworthy vital statistics. Every one who does or sees autopsies frequently and is acquainted with the present methods of signing death certificates, based on clinical and frequently superficial clinical observations, will be impressed with the fact how far we still are from really trustworthy vital statistics. Questions of the first magnitude and involving the health of the community to the greatest extent can hardly be answered to-day on the strength of the statistics in our possession: for instance, whether tuberculosis is really making a very rapid increase or decrease; how tuberculous infection enters the body and spreads within it; many questions about cancer, about syphilis, and the relationship of these to each other; about the diseases of the heart, about Bright's disease and many others. Any one who has any knowledge of the difficulty of making even accurate post mortem anatomical diagnoses, and reconstructing from autopsy the natural history of a disease, will admit that no clinical evidence alone will ever be sufficient for useful, scientific records. This is particularly emphasized in some diseases like tuberculosis and cancer and during epidemics in which a strong tendency toward a particular clinical diagnosis exists.

Secondly, an exact knowledge of the processes leading to the death of an individual demands consideration in their relations to the previous life of that individual and his surroundings. These relations are daily acquiring greater interest and importance, even on this side of the water, for within them centres information about two very important questions; first, the factors surrounding the remote origin of the disease and influencing its course, exact knowledge of which would naturally influence prophylaxis and treatment; secondly, and of imme-

diate practical value, its relation to the liabilities and obligations of State, municipalities, and employers to the individual. They affect the individual's rights in the community, his rights towards pensions, insurances, etc. They may show how cer-

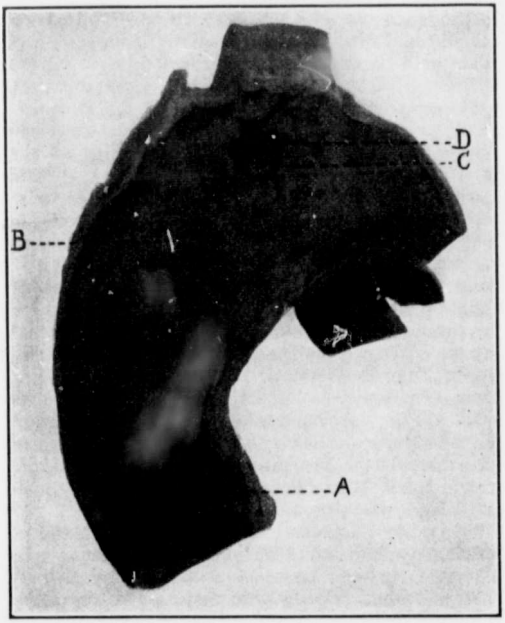

FIG. 1.—*A*, aortic cusps; *B*, principal seat of aortitis; *C*, ulceration; *D*, perforation.

tain environments either produce or materially influence the course of a disease. In other words, an exact determination of the genesis of the cause of death, or better, the natural history of a disease,

forms the necessary basis of a social pathology and, if properly applied, should form the foundation of social legislation. One appreciates how pathology in this respect goes far beyond the application of well established pathological facts to the welfare of one individual alone, and this branch of pathology will form in the future one of the most important biological studies.

Orth mentions an example which may be cited in this connection in order to illustrate how intricate problems may arise: A person has suffered an accident by receiving a blow in the region of the stomach. Some time later, cancer of the stomach develops in this person. The cancer ulcerates and perforates into the peritonæum, causing a purulent peritonitis; death occurs with the symptoms of a weakened heart. We have to answer in the first place, what is responsible for the death? The peritonitis undoubtedly induced heart weakness and heart death, and the former in turn depends on the perforating carcinoma of the stomach. But, it may well be asked, what relation has the blow to this whole complex? Can everything perhaps be traced to the blow as a cause? On the other hand, if it can be demonstrated that the carcinoma existed at the time that the blow was delivered, may not the blow stand in a causal relation to the perforation and the following peritonitis and heart death?

To take another example: A person may be engaged in an occupation which requires constant bending over and considerable pressure just over the præcordial area. He suddenly collapses and dies. Autopsy discloses rupture of the heart as the result of a hæmorrhagic infarction. The questions here are somewhat similar. Did the pressure of occupation perhaps cause a circulatory condition in the heart favorable to the production of the infarct, or did it at least contribute towards it?

In the second place, if it appears that the infarction cannot be directly attributed to the injurious

habit of occupation two other questions must be settled: Has occupation directly caused rupture of the infarct, or at least has it prevented the proper healing of it? To-day we are in the habit of dismissing such questions from our thoughts in the

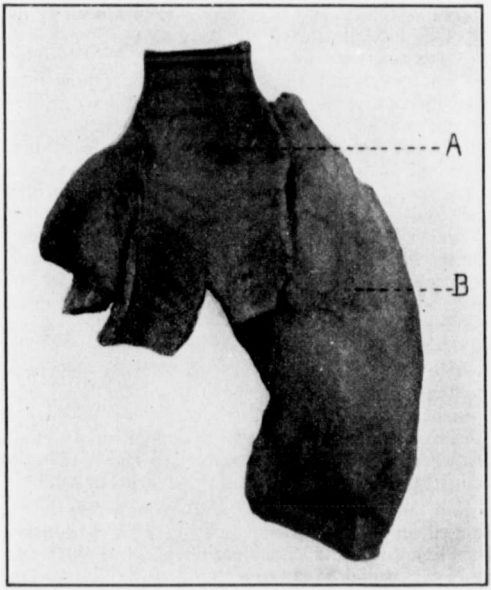

FIG. 2.—Same as Fig. 1, from the tracheal side. *A*, perforation into the trachea; *B*, gelatinous, thickened adventitia of aorta.

study of individual cases, as being of no practical bearing on the case or being uncertain. But, properly collected and critically accumulated, such factors may acquire a very great importance regarding the origin and termination of the disease,

consequently regarding their prevention and treatment and our social obligations.

These cases are comparatively simple, for we meet in them a chain of well connected symptoms

FIG. 3.—Inflammatory granulomatous infiltration around the vasa vasorum of the adventitia with endarterial obliteration and thickening; magnified 75 times.

and circumstances. Frequently, however, conditions are more complicated and not so definitely connected. This, for instance, is the case in causes which enter into the production of the much talked

of arteriosclerosis, its distribution throughout the body, and subsequent manifestations. It is commonly assumed that alcohol is a potent factor in its production, as well as in the production of other diseases. A careful analysis of trustworthy records, however, fails to disclose a direct relation of alcohol to these diseases and the fact that they frequently occur in certain alcoholics living under peculiar, complicated conditions of life only emphasizes that we must look for other, at least additional, factors in the production of these cases. Again, angina pectoris is a disease which appears particularly in the well to do. In charity hospitals it is almost entirely unknown, although the diseased condition to which angina pectoris is usually attributed exists in these cases as well as in the others. But what factor determines its occurrence in one case and not in the other? These questions, although generally neglected to-day, are really of the greatest importance, and the urgency to solve them will become greater as our social conditions on this side of the water become more complicated, as the population increases, and the interdependence of the people and their affairs becomes of greater consequence. In Europe they have received for some time careful attention.

There exists not infrequently what may be termed a competition of lesions in the body which may enter into the clinical picture and cause of death. As to such complicated cases, the question of the individual relations of the lesions to each other and to the cause of death must be settled, and whether the presence of one may or may not have taken an active part in producing the other and hastening death.

Here we are confronted by two problems: What is the relationship of the pathological processes and what kills the patient? Take, for instance, a patient with a cancer of the pancreas and, as is not infrequent, accompanying diabetes. In such an in-

FIG. 4.—From adventitia. *A*, gumma with fusion of cellular elements; *B*, a typical Langhans giant cell; *C*, circumvascular infiltrations surrounding gumma; magnified 125 times.

dividual severe jaundice, grave anæmia, cachexia, and nephritis often develop. In such a case, the relationship of one to the other and its contribution towards causing death is of greatest consequence to the scientific statistician. Again, in cases of metastases the secondary lesions may overshadow the original one. This, for instance, may be the case in a small annular carcinoma of the gut, which may not produce any symptoms at all, but may cause enormous metastases which kill. During life the diagnosis of the primary lesion may be impossible.

These examples are perhaps sufficient to illustrate how important it is that the determination of the cause of death should go beyond the immediate symptoms and findings preceding it and should in every way endeavor genetically to reconstruct the processes which have led up to the fatal catastrophe. For these reasons the objective anatomical findings are of fundamental importance for scientific as well as for practical use. While the clinical history is certainly indispensable for the correct understanding of a case, equally indispensable is knowledge of the morphological changes which can with certainty be disclosed only at autopsy.

It is regrettable that an unfortunate attitude is taken even by some physicians, who hold that in plain clinical cases autopsy is hardly necessary. How mistaken such an opinion is one may judge from a recent case at this institute: A body was sent from the hospital with definite diagnosis of extensive inoperable carcinoma of the uterus. The clinical diagnosis was apparently so clear that no one thought of any other possibility, and at autopsy the lesion appeared as an ulcerating necrotic growth involving the body of the uterus and all annexa, with pretty complete adhesions of all the structures of the pelvis, so that dissection was difficult. Even then, the probable diagnosis of carcinoma of uterus was entertained, but on closer inspection the lesion turned out to be an extremely

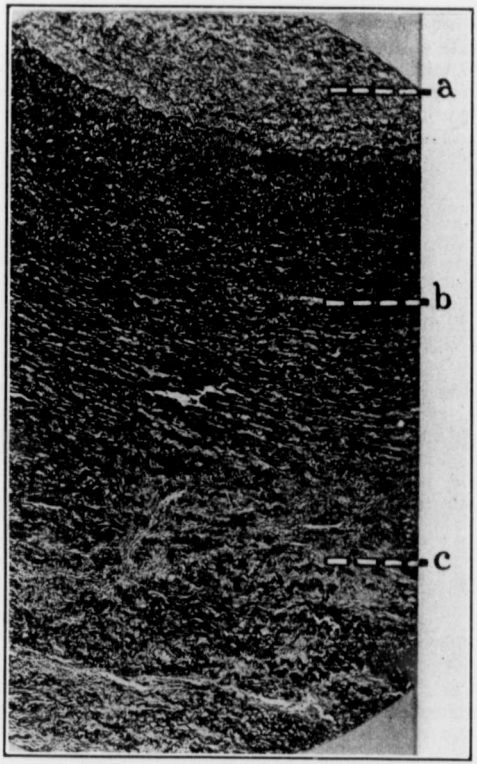

FIG. 5.—Section through intima and media of the aorta stained for elastic fibres. *A*, intima well differentiated from media with moderate fibrous and delicate elastic thickening and some necrosis of its elements; *B*, internal portion of media relatively well preserved; *C*, adventitial portion of media, with destroyed elastic and muscular elements replaced by cicatricial tissue.

extensive tuberculosis of the genital organs, and no cancer.

An absolutely exact clinical diagnosis hardly exists, and even after death a suggestive influence of former experiences may be strong enough to overcome the necessary impartial observation of the case.

In this way we are daily in the habit of arriving at erroneous conclusions until a better observer shows us that an accepted idea was based on insufficient observation.

In the following cases, which I have selected and which have come to autopsy in the Russell Sage Institute of Pathology during the last months, it is well illustrated how important anatomical post mortem evidence is for the correct understanding of the case, what extensive and severe lesions may exist in an individual without manifesting themselves in any symptoms sufficient to attract the patient's or the physician's attention, how certain symptoms may be misleading in clinical diagnosis, and finally, how apparently very sudden deaths are only the result of long continued disease, and in the strict sense of the word were not sudden, but very gradual.

These examples are interesting in themselves, and I may be allowed to add some epicritic remarks.

Cases coming to autopsy with the statement of sudden death may, for convenience sake, be grouped under the following headings: First, those which present certain abnormal developmental or acquired anatomical conditions which in themselves do not give a satisfactory explanation for the sudden death, although we assume an intimate relation on account of the large number of times they are associated with otherwise unaccountable or trivial causes of death. This group is exemplified particularly by the so called status lymphaticus, thymus enlargement, or congenital narrowness of the aorta.

The *modus operandi* is not certain here, and is explained only on very theoretical considerations.

To the second group belong cases which show distinct evidences of serious diseases, although the direct cause of the sudden termination of the disease is left uncertain. To this category belong a

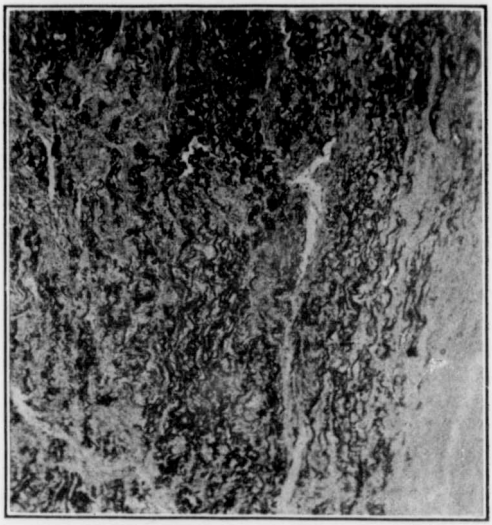

FIG. 6.—From another outer portion of the media, showing marked elastic tissue destruction with cicatricial replacement. Magnified 125 times.

good number of cases of Bright's disease, heart lesions, aneurysms, brain tumors, etc. In these it is impossible, unless we again assume theoretical considerations, exactly to state why the disease should terminate suddenly fatally at a particular time.

In this connection, however, it is important to

emphasize thoroughness in autopsy. For the sake of relatives, friends, and time, autopsies are rarely as complete as they should be in order to obtain the fullest necessary information. But one should bear in mind fully the necessity of examining the brain, the cavities of the skull, the middle ears, and the tonsils. The importance of examining the bones and joints, which in most permitted autopsies cannot be investigated, at least with great exactness, should also be remembered. I speak of this particularly, because a few times we have in this institute been uncertain about apparently obscure ante mortem signs, which were finally explained later, in the course of the autopsy, when an opportunity was given to examine all of these frequently neglected structures.

In this connection I may cite the case of a middle aged woman who had been in the City Hospital for a long time with a nephritis. Toward the end of her illness she developed an irregular, remittent temperature. Suspicion was entertained during life that this was due to an arthritis of one of her knees. At autopsy, however, this knee was found intact. While still considering some other possible explanation to account for the fever, other joints were investigated and a purulent (gonorrhœal) arthritis was found in the opposite knee, where it had not been suspected.

The third group, finally, is represented by cases of a type of which I intend to present a few, and which not only show evidences furnished by the last two groups, but very definite lesions, which in themselves demonstrate the *atrium mortis.*

CASE I[2]. A man, forty years old, was admitted to the City Hospital with multiple, small ulcers on his forehead of some six weeks' duration. His history showed that he had had a hard chancre seventeen years ago, and, a year and a half later, skin eruption and mucous patches. The present ulcers, about four in number, were not painful, but discharged freely a purulent, thick fluid. Physical examination showed nothing else of importance except old scars on his legs. There was no fever. Under large

[2]This and the subsequent two cases were reported by Dr. Detwiller to the New York Pathological Society at its meeting of December, 1909.

doses of potassium iodide and hypodermic solution of mercury salicylate, he felt sufficiently well after nine days to ask for his discharge. Before this, however, he was suddenly seized, while still in the ward, with a severe hæmorrhage from the mouth. When the house physician arrived a stream of bright red blood, described as being the size of a lead pencil, was pouring from his mouth. A few seconds later the patient died before anything could be done for him.

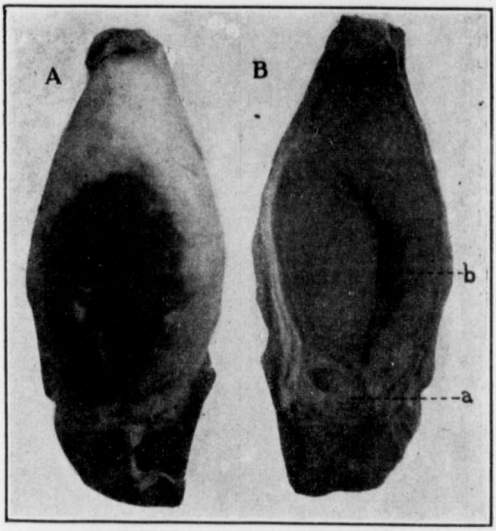

FIG. 7.—*A*, external view of the aorta above valve, showing hæmorrhagic infiltration of adventitia (dissecting aneurysm). *B*, *a*, aortic cusps; *b*, rupture.

The clinical diagnosis remained necessarily uncertain, although a ruptured aneurysm seemed possible. At the same time, the entire absence of any previous clinical signs or symptoms made even this diagnosis uncertain. One also had to think of ruptured gastric ulcer, or œsophageal varicose veins, against which, however, was the fact that the blood was not vomited, but literally

poured out of the patient's mouth. At best, the clinical diagnosis, then, remained dubious.

Autopsy disclosed the rather interesting lesion shown in Fig. 1. The origin of the aorta showed only a few white, slightly elevated, spots and streaks. The arch, however, was extensively puckered with diffuse, irregular, whitish elevations and depressions and distinct cicatricial con-

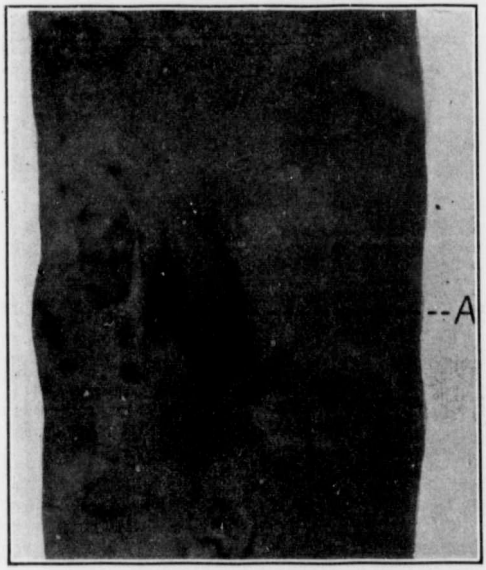

FIG. 8.—Atheromatous and calcareous portion of the aorta with (probably syphilitic) scar formation; *A*, irregular aperture in the long axis of vessel with loss of intima and media, perforation through adventitia into posterior mediastinum and right pleural cavity.

tractions. Occasionally the intima showed patchy, yellowish discolorations over this whole area. Typical atherosclerotic necrosis with calcification was absent. The surrounding adventitia throughout the course of the aorta was whitish, thickened, and gelatinous. Most important

with regard to his sudden death, however, was the presence of a deep, annular, punched out ulcer, situated at the height of the arch where it crosses and is intimately connected with the trachea. The ulcer was nearly circular, its edges somewhat undermined and irregularly thickened. The base of this ulcer was formed by the underlying and involved trachea. It showed a perforation, four millimetres in diameter, through which the fatal hæmorrhage had occurred (Fig. 2).

The aorta itself showed no special aneurysmal dilatation of its arch, and measured only 8.5 centimetres in its interior circumference. Of particular notice is the fact that the rest of the aorta was free from disease. The stomach was filled with semiclotted blood and the lungs were in a state of acute emphysema and filled with aspirated blood. The liver showed a typical large gumma and very deep scars, but smooth atrophy of the base of the tongue was missing.

Microscopical examinations of sections taken from the arch of the aorta showed a characteristic granulomatous aortitis. Throughout the thickened gelatinous adventitia were, partly diffuse, partly localized circumvascular lymphocytic infiltrations. The vasa vasorum themselves showed endarterial proliferation, frequently leading to obliteration of their lumen (Fig. 3). In some places, circumscribed granulomatous formations, made up of lymphocytic and immature fibroblastic and some Langhans giant cells, were plain. (Gummata; Fig. 4). There was an irregular tendency to cell fusion and necrosis, although definite caseation did not occur. This granulomatous infiltration freely invaded the media, leading, particularly in its lower and middle portions, to circumvascular inflammatory foci with patchy tearing and disorganization of its elastic and muscle tissue, which was replaced by cicatrices. On the other hand, the intima, which was well separated and distinct from the media, showed moderate fibrous and delicate fibrillar elastic thickening, and in places degenerating fibres (Figs. 5 and 6).

The case, therefore, may be regarded as a typical specific granulomatous aortitis, which commences in the form of circumvascular infiltrations of the adventitia, becoming more diffuse and extending into and destroying the media, leads to the formation of cicatrices. The intima is not attacked. For, as I will discuss in a moment, the thickening and hyperplasia of the latter cannot be brought in direct causal relation to the specific aortitis. Over the trachea the process had produced extensive loss of

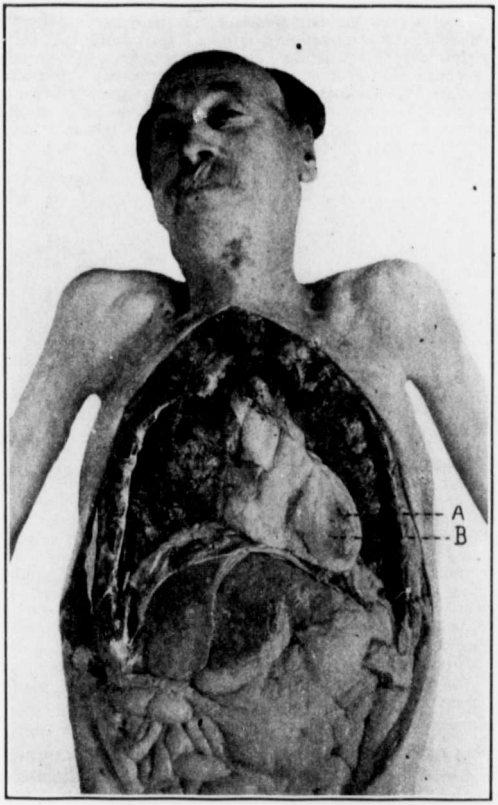

FIG. 9.—Blood removed from the pericardium and the heart lifted and turned on its axis so as to expose the border of the left ventricle; *A*, rupture; *B*, surrounding hæmorrhagic infarction with partly yellowish white necrosis.

substance and had formed deep, retracting scars with a deep ulcer and, by invading the trachea, this had led to perforation into its lumen.

That this process was due to syphilis appears certain, in spite of the fact that a search for spirochætæ pallidæ was made in vain. For we have not only a definite history, but also very conclusive morphological evidence in the aorta and various other parts of the body. The case is of a more general interest, because it is clear cut and emphasizes, first, the difference between ordinary arteriosclerosis and the granulomatous (specific) aortitis. Whatever view we may take with regard to the origin of the ordinary arterio- or atherosclerosis, whether we place it first into the intima or as a primary weakening into the media, it is certain that its characteristic feature is a noninflammatory hyperplasia of the intimal elastic and fibrous tissue, which is characterized by the formation of thick, coarse lamellæ and obliterates the distinction between intima and media.[3] These changes are associated with definite nutritive disturbances which lead to characteristic fatty disintegration of the intimal and medial tissue with the formation of the atheromatous ulcers, which undergo calcification. Contrasting these changes with what we find in this case, we appreciate that we are dealing with two entirely different lesions in genesis and character.

The specific syphilitic lesions of the aorta have only recently received more careful attention and been separated from those of arterio- or atherosclerosis, although the syphilitic affections of the smaller vessels have received much earlier recognition. Heller, and his pupils, particularly Doehle in 1885 and 1895, Backhaus, Moll, and Isenberg described cases of granulomatous mesaortitis, which they regarded as syphilitic in origin. Later, Heller gave before the German Pathological Society, in 1899, a

[3]This is particularly important, because in inflammatory aortitis clear distinction between intima and media remains and the newly formed elastic fibres are thin and delicate. (See Fig. 5.)

general review of the subject and pointed out particularly its relationship to the formation of aneurysms. Finally Chiari, in an extensive discussion on the syphilitic aortic lesions before the German Pathological Society in 1903, and Marchand before the German Congress of Internal Medicine in 1904,

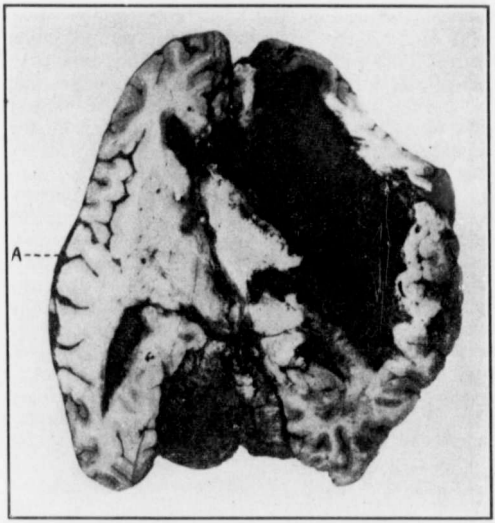

FIG. 10.—Extensive idiopathic hæmorrhage into right side of brain with complete destruction of the basal ganglia, blood flowing into the anterior and posterior cavities on either side and becoming subpial; *A*, marked subpial hæmorrhage, evidently from intracranial pressure on the opposite side.

recognized the lesion as specific and separated it from arterio- or atherosclerosis.

On the other hand, a specificity of the lesion was denied by some like Orth, Ziegler, Baumgarten, and, particularly, Mönckeberg who, while not denying the process, had been unable to convince himself of its

syphilitic nature in most of his material. The occurrence of genuine gummata in the aorta he considers doubtful, as the necrosis attacked, not the granulomatous tissue, but only part of the aortic wall.[4] However, in our case the localized infiltrations with giant cells show plainly, not only necrosis of the aortic wall, but a similar tendency on the part of the granuloma cells, and therefore present the features of the typical gumma. (Fig. 4.)

Mönckeberg and Ziegler and others refer a number of the cases of mesaortitis, not to syphilis, but to coccal infection. It must be admitted that in certain, especially late cases, it is impossible to form a definite opinion. For, when a process has gone on to scar formation or when the mesaortitis is more diffuse without characteristic gummatous formations, one cannot decide whether it results from a syphilitic or other infection. A similar difficulty applies to cases associated with advanced arteriosclerosis. For these reasons I regard the case here presented as valuable because it conforms in location, morphological evidence with history, the absence of a complicating arteriosclerosis, and the certainty of excluding tuberculosis, with the picture of true gummatous aortitis, a mesaortitis gummosa.

A practical point of interest is one to which Heller drew attention, the great importance which must be attributed to syphilis in the production of aneurysms and perforations of vessels, on account of its distinct, destructive tendency in the media and adventitia. This, as very much greater than in arteriosclerosis, makes the granulomatous aortitis of much greater danger than the uncomplicated arteriosclerosis.

Clinically the case is interesting, to observe how extensive lesions may occur in the aorta and progress to fatal termination without manifesting symp-

[4] Herxheimer, Zur Aetiologie und pathologischen Anatomie der Syphilis in *Ergebnisse der allgemeinen Pathologie und pathologischen Anatomie*, xi, 1, 1907.

toms or signs allowing us even to suspect their existence.

The next two cases are similar in character, although the lesions are somewhat different.

Case II. Man, large, well developed, fifty-nine years old. Practically no previous history was obtainable. He was in the institution for no particular complant, except that he was homeless and generally weak. No serious difficulty had been suspected in this case, but he died very suddenly one night in bed. The immediate circumstances surrounding his death were therefore not available.

The autopsy showed the pericardium filled with blood. The first portion of the arch of the aorta showed a slight general dilatation. Along its right margin, just immediately above the cusps, and while the aorta was still within the pericardium, there were found two small, slitlike apertures, situated in the longitudinal axis, being about three millimetres long and separated by thin fibrous tissue, about two millimetres broad. The adventitia around these apertures showed an extreme diffuse hæmorrhagic infiltration. These apertures communicated with the interior of the aorta, and through them the hæmorrhage into the pericardium had taken place. The interior of the aorta showed a rather irregular, puckered thickening with whitish scars and occasionally raised, yellowish white nodules. A long slit, three centimetres in length, extending through the intima and media, commencing immediately below the valve, and which contained the previously mentioned communications through the adventitia, was found. (Figs. 7 A and B). The case is, therefore, an example of extreme, peculiarly linear destruction through media and adventitia, leading to their complete rupture, with hæmorrhagic infiltration of the adventitia, and necessarily consequent perforation into the pericardium. In this case also the other parts of the aorta showed no evidence of disease.

Case III. The third case combines a (probably syphilitic) granulomatous mesaortitis with arteriosclerosis. I need not discuss it in full, because we covered these questionable points in Case I. It concerns a woman, fifty-seven years old, who had been in hospital with a clinical diagnosis of asthma and cardiac insufficiency. She died very suddenly during the night in bed.

At autopsy was found a right hæmothorax. The aorta showed throughout very extensive atheromatous and calcareous changes, although the latter were not particularly numerous. In addition to this there were many translucent looking scars, some also with radiating linear furrows. In the lower portion of the thoracic aorta, one centimetre above the opening in the diaphragm, a slitlike aper-

ture situated in the long axis of the vessel, and communicating with the posterior mediastinum and right pleural cavity was found. (Fig. 8). This rent went completely through the intima and media to the adventitia, where a small dissecting aneurysm had formed, which had ruptured.

The question arises, in all of these three cases, why rupture occurred at a particular time. In the first case, this may be somewhat easier to answer, for the reason that the patient considered himself well and was about to leave the hospital. It may be that physical effort in his preparations for departure precipitated the fatal issue. The other two cases, however, occurred apparently when the patients were far from even making ordinary physical efforts. They were, however, not observed during that time, and it may be possible that the ruptures occurred really not while they were asleep, but after their awakening, and during a possible effort to leave the bed.

Case IV. Man, well developed, described as apparently healthy, sixty-four years old, who had been an inmate of the City Home (alms house), and had been seen to become suddenly pale and fall over. He was dead immediately. In this case no clinical diagnosis could be attempted.

At autopsy the pericardium was distended with fluid and recently clotted, dark red blood. A clot weighing 300 grammes was removed from it. On lifting the heart, the left ventricle presented an irregular rupture, 1 by 1.5 centimetre in length along its lateral aspect. This was surrounded, partly slightly posteriorly and more anteriorly, by an irregular necrotic (infarcted) area, about 1 by 1.5 centimetre in diameter (Fig 9). The upper part of the anterior coronary artery, which was very sclerotic, contained a recent, but firmly adhering thrombus, while lower in its course there existed a complete obliterating endarteritis. Further dissection disclosed marked general arteriosclerosis, healed calcareous tubercles in the upper lobe of the right lung, extensive caseating tuberculosis in the upper lobe of the left lung, right hydrothorax, pleurisy with effusion on the left side, and cyanotic induration of liver, spleen, and kidneys.

This case presents many points of interest: In the first place, the immediate cause of the sudden

demise was due to spontaneous rupture of the heart, induced by hæmorrhagic infarction of the heart muscle, which in turn must be traced to the endarterial obliteration of a large coronary artery. These cases are of particular interest, for they are apt to be confounded with traumatic hæmorrhages of the heart, and indeed the decision may not always be as easy as in this case, where evidence is quite complete.

In a traumatic severance, the cut or puncture is definite, straight, the surrounding musculature shows little hæmorrhagic infiltration, and, extremely important, the surrounding heart muscle and arteries are healthy. The spontaneous rupture, on the other hand, is irregular, indentated, torn, and occasionally tortuous. Of importance here is the surrounding hæmorrhagic infarction (myomalacia), degeneration and fatty metamorphosis of the infarcted area, and a diseased state of the uninvolved neighboring muscle fibres. In the smaller, recent infarctions, the thrombosed artery may sometimes be seen within the necrosing focus, and where they are extensive, as in this case, a larger branch will usually be found the seat of an obliterating endarteritis. In doubtful cases microscopic examination is absolutely essential to arrive at a trustworthy conclusion.

On the other hand, it is also important to know whether the infarcted area, once produced, leads to spontaneous rupture or whether external physical factors are essential. To answer this properly we must inquire somewhat into the genesis of the hæmorrhagic infarction of the heart.

The idea once prevalent that coronary arteries are endarteries, since the investigations of Spalteholz, has been superseded by the view that there exist anastomoses, not only between main coronary arteries, but also between their branches and extensions. And, indeed, the experimental investigations into the results of obstruction of coronary vessels have not been followed by uniform results.[5] Even

[5]Hirsch. In the *Verhandlungen der deutschen pathologischen Gesellschaft* for 1909. Spaltehölz's work *ibidem.*

where infarctions have occurred, a functional derangement has by no means always taken place. Jores has therefore properly called attention to the fact that sudden deaths in human beings occur when the infarction takes place in an already diseased, degenerated heart, which is unable to withstand sudden, additional, serious interference. That even extensive necrosis and loss of heart substance may occur in man without rupture or sudden death is illustrated by the extensive, healed scars which are occasionally detected after death in the substance of the left ventricle. In this institute, we have, for instance, possession of such a heart, which shows an almost entire replacement by fibrous tissue of the apex, extending well up into the left ventricle.

We, therefore, assume that previous degenerative disease of the heart muscle, associated with lack of sufficient nutrition, is responsible for failure of cicatricial replacement in certain infarcts and therefore the necessarily ultimate rupture. It is plain that this may then take place unaided by any outside influence, by mere force of the blood pressure and systole. The ante mortem observation in this case may be looked upon as corroborative of the purely spontaneous character of the rupture.

Of great interest from the standpoint of the physician is the fact that an individual may have such a far advanced disease of a large coronary artery followed by infarction, without any symptoms to himself or others, until heart rupture occurs.

Occasionally, particularly in the better to do classes, severe subjective symptoms seem to result from less extensive anatomical changes. This, in our experience, applies to other lesions as well and leads one to conclude that the subjective factor in certain groups of individuals modifies essentially the clinical picture of a disease.

Of similar interest in this case are the well defined, additional lesions, namely, extensive tuberculosis and pleuritis with fluid in both sides of the

chest. At the same time, the individual was considered by others as a healthy old man; evidently he himself cannot have been much troubled by the state of his body, for he had not complained or applied for examination and treatment.

The next cases are of different character and concern hospital patients who, while they were supposed to be suffering from chronic diseases, died suddenly and unexpectedly. In each of them the autopsy disclosed an unsuspected lesion which accounted for the sudden deaths.

CASE V.* A young man, twenty-seven years old, had been in the hospital for extensive syphilitic ulcerations of face and nose, which, under appropriate treatment, had healed with the formation of thick retracting scars. He had then been employed as a hospital orderly. During the last five months he developed symptoms of a syphilitic laryngitis, his voice had become husky and weaker, until he was able to speak only in a hoarse whisper. It was also noticed that he gradually developed difficulty in walking upstairs, and he had become subject to what were termed asthmatic attacks. During one of these attacks, which was precipitated by running up a flight of stairs, he became extremely cyanotic and died in a short time. The physician who saw him die thought of a possible ruptured aneurysm or acute dilatation of the heart.

Autopsy disclosed a granulomatous (syphilitic) laryngitis of a much greater extent than had been anticipated. The base of the tongue showed large swollen, hypertrophied papillæ. The epiglottis was completely ulcerated away, leaving only a small stump. The arytænoepiglottidean folds were extensively thickened by an irregular, nodular, and scarred growth. Even at the time of autopsy œdema of the glottis to a practically complete stenosis, existed, the walls of the chords were hard, firm, and thickened. The under surface of the larynx showed a similar destructive granulomatous growth extending to the fifth ring of the trachea.

Corroborative evidence of the syphilitic character of the lesions was found not only in the skin lesions, but also in typical gummata of the liver. The aorta, on the other hand, in this case presented no distinct lesions, only along its posterior wall fine, pale, linear markings.

The cause of death was then œdema of the glottis, due to extensive destructive syphilitic laryngitis. While the

*This case was also presented to the New York Pathological Society by Dr. Detwiller at its meeting of January, 1910.

latter had been known to exist during life, its extent and direct connection with the sudden death of the individaul had not been known, and indeed his last attack had rather led to a suspicion of a circulatory disturbance.

More surprising even were the findings in the two following cases:

CASE VI. Man, sixty years old, of large frame and relatively good nutrition. He had been in the hospital for some time with the diagnosis of, and under treatment for chronic nephritis and colitis, and was turned over to the pathological institute with that diagnosis. The staff was particularly anxious to know why he had died very suddenly, apparently without any warning.

Autopsy revealed a huge, nearly circular, cauliflower carcinoma of the stomach, reaching within 2.5 centimetres from the cardiac orifice and four centimetres from the pylorus. It covered about half of both the anterior and posterior walls of the stomach. Both orifices could evidently functionate properly, as the stomach was nearly normal in size. The surface of the growth, however, was ulcerating and hæmorrhagic, and there had taken place a recent extensive hæmorrhage from the growth.

The mesenteric glands showed metastasis, and there existed gray hepatization of the middle lobe of the right lung. The kidneys were large (200 grammes), and showed a degenerative nephritis.

Here again it is surprising to observe the character and extent of the lesion, which during life had certainly not produced sufficient symptoms to point to a more definite diagnosis. On the other hand, the anatomical location of the growth explains the lack of any interference with the motility of the stomach and the possible indefinite, gastric symptoms were ascribed to the nephritis. The terminal pneumonia had remained quite obscure.

CASE VII. Woman, seventy-four years old, had been in the hospital for some time with the clinical diagnosis of chronic nephritis. She had died very suddenly with what was given as œdema of the lungs.

Autopsy showed the stomach filled with a large amount of hæmorrhagic fluid. About three centimetres from the pyloric end, on the posterior upper surface of the stomach, a small, punched out ulcer, about 1.5 by 1 centimetre, had eroded through the muscular coat of the stomach. At the base of this ulcer is seen the opening of one of the gastric arteries. A probe passed easily through the opening along the course of the artery. The mucous membrane of the remaining parts of the stomach was considerably atrophied and showed catarrh. The kidneys showed arteriosclerotic atrophy.

Her principal lesion was then an active ulcer of the stomach which had eroded a large gastric artery. The immediate cause of death was hæmorrhage into the stomach.

CASE VIII. Woman, seventy-five years old, homeless, sat in the reception office of the hospital awaiting transfer. She suddenly collapsed in the chair unconscious with stertorous, labored respiration of 24, pulse 75. Left eye contracted, but both reacted to light. The left lid drooping, the left cheek sunken, the mouth drawn up, left arm and left leg flaccid, knee jerks absent, right arm and leg not affected. Blood pressure 220. Temperature 97.4° F.

After an hour, respiration changed to Cheyne-Stokes, blood pressure rose to 300, both pupils became dilated. Babinski present in right leg: slight movement to be elicited by mechanical stimulation in both legs. Pulse became weak and irregular. Œdema of lungs supervened and she died after tewnty-two hours.

Clinically, the case was regarded as a probable hæmorrhage in the brain. But, on account of the rapidity of the fatal termination and the difficulty in thorough examination, the extent and exact location could not be definitely given.

At autopsy was found extensive and massive recent hæmorrhage into the right brain substance, ventricles, both anterior and posterior cornua. It had led to complete destruction of the left basal ganglia, penetrated within the cortex, and became subpial. But subpial hæmorrhage was particularly marked on the left side along and posterior to the Sylvian fissure and over the cerebellum (Fig. 10). The superficial vessels on this side showed strong engorgement (*Encephalorrhagia permagna basis et corticis cerebri*). The convolutions had become flattened and were dry. The vessels at the base of the brain were very atheromatous.

In view of the anatomical distribution and extent of the lesion, it is safe to assume that there occurred on the right side rupture of a huge atheromatous artery (*lenticulo striate*), which with enormous force had crushed the brain substance on that side and broken into the left lateral ventricle from where blood flowed into the communicating cavities. It is interesting to know that a patient with such a hæmorrhage still lived twenty-two hours.

From a medicolegal standpoint, the destruction and enormous extent of the hæmorrhage with involvement of the subpia, particularly upon the opposite side, and cerebellum in a spontaneous hæmorrhage, is very instructive. It is impossible to con-

ceive a direct extension from one side to the other, particularly as there was only slight pial involvement on the right side. More probable is it, therefore, that the enormous intracranial pressure produced by the hæmorrhagic destruction of the right half of the brain led to such pressure on the necessarily distended superficial vessels of the left side as to produce a really traumatic pial hæmorrhage. This was probably so much more easily accomplished, as the vessels there were in a similar diseased condition and had undergone compensatory engorgement. The latter was still plain after death, as well as the evidences of pressure in flattening of the convolutions.

A review of these cases convinces one of the great importance of anatomical evidence supplementing the clinical, and that the determination of the cause of death and its genesis is a difficult and serious matter, which never must be done hastily, but, like the clinical examination, by one well trained and experienced, with great care and thoroughness. With Virchow we might say, almost with greater care and thoroughness, because what has been neglected in one clinical examination may, as a rule, be investigated during a subsequent visit; but in the determination of death and the processes leading to it, evidence presents itself only once, and anything omitted is irreparably lost.

On the other hand, one should also emphasize the necessity of scientifically accurate clinical records for the proper understanding of anatomical findings. No greater mistake can be made than to expect a pathological anatomist to arrive at correct conclusions without full knowledge of the clinical history. It is sometimes thought by those not well informed that the objective anatomical evidence ought to be separated and not biased by the largely subjective clinical data. But it is forgotten that we are not dealing with mathematical problems, but with a mass of evidence that must be grouped not

only in what appears to the investigator as essential and unessential, and in logical sequence, but, much like the work of an artist, plastically reconstructed. The pathological anatomist, therefore, like the artist who models a statue or paints a picture, must possess not only a knowledge of the component parts of a structure, but of the life history of the subject. Only then he can hope to impart to the creation of his own mind the truthfulness of an actual occurrence.

In the clinical diagnosis, the physician carries responsibility toward one individual. In the determination of the cause and genesis of death, this responsibility extends to the whole of the community.

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