Concerning aneurysms : the Gordon Bell memorial lecture / by Oskar Klotz.

CONCERNING ANEURYSMS

THE GORDON BELL MEMORIAL LECTURE

by

PROFESSOR OSKAR KLOTZ

(*Reprinted from the University of Toronto Studies, Pathological Series, No. 7*)

ered before the Winnipeg Medical Society
Winnipeg, Manitoba, April 1, 1926

CONCERNING ANEURYSMS

THE GORDON BELL MEMORIAL LECTURE

by

PROFESSOR OSKAR KLOTZ

(Reprinted from the University of Toronto Studies, Pathological Series, No. 7)

Delivered before the Winnipeg Medical Society
at Winnipeg, Manitoba, April 1, 1926

CONCERNING ANEURYSMS

By Oskar Klotz

I am much honoured in being asked to give the Third Gordon Bell Memorial Lecture, which has been established by your society to commemorate the name of a very worthy member of our profession. At first, upon receiving your invitation, I was reluctant to accept the honour, feeling that there are many worthier who could better perform the duties incumbent upon the speaker, and also it seemed that I would be "carrying coals to Newcastle" or "owls to Athens", when here amongst your own group of active young men you have such excellence for the exposition of advanced medicine. This happy situation which exists amongst you,—the spirit of gaining a wider knowledge of our human ills; the interest in extending the field of Public Health; the development of methods for the teaching of the undergraduate; and the desire for co-operation for the best interest of medical practice,—can be traced with little difficulty to the man for whom this lectureship was founded. The time has been too brief, since his loss to your community, to require a review of his active life, but we cannot forego the occasion to comment upon his versatility,—one that was not superficial, but was founded upon a personal experience in many subjects. Coming to Manitoba in the early days, when railroads had scarce disturbed the quiet of the plains, when only the hardy stock with venturesome spirit and unconquerable grit hewed their way to those recent frontiers, Gordon Bell, although educated first in Ontario and at the University of Toronto, completed his education in the newly established Manitoba Medical College. His interest did not centre upon the practice of medicine, but he gained a versatility in the various branches of medicine, serving as superintendent to

the Hospital for the Insane, subsequently entering upon special studies in the diseases of the eye, ear, nose and throat, and then forsaking clinical practice to become engrossed in the laboratory sciences of Pathology and Bacteriology. As the professor of Pathology and Bacteriology, none was more popular with the student body of the University than Gordon Bell, and in the capacity of Provincial Bacteriologist his advice was sought both by the profession and by the administration of the province, to lay the foundation for the future health of the people. His attractive personality and his lovable character endeared him to a host of friends not alone in Winnipeg and Manitoba, but throughout Canada and abroad.

The memory of Gordon Bell will long remain with you, and will carry stimulation and encouragement to all who learn of his life. His influence will be carried from generation to generation, and will stand, to each of the younger group, as an example of a man unselfishly serving his fellow-men. Gordon Bell possessed that rare quality of combining scholarship with human interest. He could enter upon the discussion of the minutiæ of medical problems, be these in practice or theory, but he never failed to lead the thought back to its practical bearing and its application to the prevention of disease. His sympathies for those in distress were deep, and much of his good work amongst the poor and in the rural districts is contained in no records save in the hearts of the people.

I have chosen for my topic of discussion the time-worn subject of aneurysm, not because our literature is wanting in discussions upon this topic, nor because I bring to you any startling or novel ideas, but rather because this subject has burdened me for many years, crowding me with viewpoints which have heretofore not been recorded. Much concerning aneurysm is well known, and has been handed on from decade to decade in a stereotyped fashion, to the exclusion of other matters which bear a worthy importance. Save for a few general studies upon aneurysms or upon special types of them, the literature contains mostly records of cases

studied clinically or at autopsy. Most of these records have added but little to our knowledge of the disease, save to draw our attention to special points which interested the observer. For upwards of twenty years the study of the arteries and of aneurysm has attracted my attention, and I have been interested by observation and trial to learn something concerning the processes of disease underlying the development of arteriosclerosis on the one hand and aneurysm on the other.

Galen appears to be the first author in antiquity from whom we learn of aneurysm, and his knowledge was limited to pulsating enlargements occurring underneath the skin. His familiarity chiefly centred about vessels which had been injured and about which a pulsating hæmatoma had developed which he found could be compressed under the hand, causing the disappearance of the mass. By experiment he found that it was unwise to attempt incision into these tumours without preliminary ligation, for he states that the blood is spouted out with much violence and can scarcely be arrested. Furthermore, he observed that when an aneurysm is compressed within the fingers, a peculiar thrill is felt within it, caused by the rush of blood. Galen also encountered vascular dilatations in the absence of injury which he concluded to be of the same character as traumatic aneurysms. It is probable that he confused venous dilatations and arterial aneurysms in his descriptions.

Some centuries later Ætius commented upon the frequent occurrence of aneurysm in the neck occurring in women during parturition and these aneurysms, he found, had signs similar to those described by Galen. Here again, however, it is not clear whether his reference is not to the enlarged and vascular thyroid rather than to the true aneurysm. He, however, also observed that true arterial aneurysm resulted at the bend of the elbow on account of the unskilful manipulation of blood vessels during blood-letting. Upon the development of an aneurysm a soft compressible tumour was noticed under the skin which was made to disappear by pressure, but upon removal of the finger it again returned.

In some of these aneurysms he noted the coagulation of blood within portions of the sac so that it became hard and irreducible. Both Galen and Ætius recommended the ligating of the artery on both sides of the aneurysm and dividing midway between them.

Another of the ancients who paid particular attention to aneurysm was Antyllus, the surgeon, a contemporary of Celsus, who lived about the middle of the second century. He found he was able to distinguish between two kinds of aneurysm; one arising through traumatism of the arteries during operation or accident, and the other being a local dilatation of spontaneous origin. Antyllus attempted various means for the cure of aneurysm and recommended ligating the vessel, both above and below the dilatation, followed by an incision over the enlargement, the opening of the dilated vessel and the removal of the coagulated contents. He warned against the greater danger in operating upon aneurysms of the neck than upon those of the extremities, and he did not favour the attempt at the removal of the sac. The work of Antyllus found favour for many centuries and little new was learned concerning the nature of aneurysm nor of the methods of cure. The knowledge of this disease remained limited to the aneurysms which were close beneath the surface and particularly upon the extremities. The commonest aneurysms encountered by the early surgeons were within the arm, where venesection had been performed and a hæmatoma had persisted from a leaking vessel. These aneurysms have little in common with the true spontaneous dilatation with which we are to-day concerned, and these early studies have only a historical value.

Fernelius (1542) recognized the difference in the character of the lesion arising from injury on the one hand, and occurring spontaneously on the other. He pointed out that in those cases where an injury had broken a vessel and permitted the escape of blood, the tumorous enlargement was partially composed of coagulum within a cavity and fluid in its centre. Furthermore, Fernelius recognized not only the development of aneurysm in the external and superficial

vessels, but also in "the internal arteries especially in the chest or about the spleen and mesentery where a violent throbbing may frequently be observed". Fernelius was the first to bring to our attention the presence of aneurysms in the deep tissues and, although he did not describe the nature of the aneurysmal dilatation within the thorax, we presume that he was referring to an aneurysm of the aorta. Some of them he associated with the presence of syphilis: "Venus vénérien forme des aneurysmes dans les arteries." A contemporary of Vesalius, Paracelsus, introduced the use of mercury in the treatment of syphilis in 1553.

The recognition of internal aneurysm during life was first made by Vesalius, who described its presence in the thoracic and abdominal aorta. Vesalius was able to demonstrate the lesion clinically and verify the diagnosis by dissection after death. His findings were very convincing, and he seems to have had the opportunity of studying completely a number of cases.

It was, however, Paré (1561) who appreciated aneurysm, its severity and mode of origin. To him we owe the first clear description of aneurysm in its different forms. He applied the term to all types of dilatation of the arteries, whether occasioned by wound or of unknown cause. He stated that aneurysms may arise by anastomosis, diapedesis, rupture, erosion or injury. "They may occur in any part of the body, but principally in the throats of women who have difficult labours; for these hold their breath forcibly so that the artery dilates and bursts." Paré also recognized the clinical manifestation of a bruit, which may be perceived either by the ear or by touch when slightly compressing the tumour. He also observed that thrombosis was very apt to occur within the cavity of the dilated vessel. Paré was very skeptical respecting the treatment of the larger aneurysm; for the smaller ones he advised ligation. Aneurysms which occur in internal parts are incurable, "happening frequently in those that have had the pox and been several times sweated for it, by which means their blood has been very greatly heated, and rendered thin; which occasions that

which is contained within the arteries to escape, and to dilate the body of the vessel, sometimes to the size of the fist. This I have observed in the body of a man named Belanger, a master tailor, living on the bridge of St. Michael, near the sign of the Cock, who had an aneurysm of the artère veineuse of which he suddenly died, in consequence of the artery bursting while he was playing at tennis. On dissecting him, I found a large quantity of blood effused within the thorax, the body of the artery dilated to the size of the fist, and its lining membrane quite ossified."

It would appear that in the days contemporary with Paré, some of the aneurysms described were venous varices. These observations were for the most part made clinically, some of them being dealt with by surgical means. A clear differentiation between pathological processes arising upon the arterial and venous channels of the extremities was not made. This confusion is particularly suggested by the frequency with which the aneurysms were observed upon the extremities. It is, of course, to be recognized that the arterio-venous aneurysm described by William Hunter was a distinct type which, although relatively infrequent to-day, was not uncommon in the days of blood-letting. The majority of the cases had been accidentally produced in the region of the elbow. Here the knife was carelessly thrust through both artery and vein so that the force of the arterial blood found a communication and exit into the venous channel.

Paré's belief that aneurysm was so common in the neck of pregnant women was based on a mistaken diagnosis, in which the vascular enlargement of the thyroid with its evident pulsation was the misleading feature. It is very difficult to estimate the frequency with which aneurysm appeared in Paré's time and to judge the kind and position of the various aneurysms seen by this observer. No doubt, some were of the traumatic variety received through injury in battle, in which Paré had no small experience. Others, and particularly those upon the arms, may have resulted from careless venu-puncture, as we have suggested above.

But with all this, when we remember that Paré's most active life was spent during that period when syphilis had spread and was still spreading over the continent of Europe in a truly epidemic form, such as we have not seen it since, it would appear that not a few of his cases were of luetic origin. This sixteenth century epidemic was of much greater virulence than is seen in the disease to-day, the present generation having the advantage of an immunity gained through four centuries of syphilization. The high mortality in the early stages of the disease, the extensive necrosis of bone, and the wide skin ulcers are not the common features of syphilis to-day, but the disease is more insidious and the tissue changes are slowly progressive, extending over years of time,—in other words, the very character of the syphilitic disease of to-day permits just such lesions as we find in the large arteries and which eventuate in aneurysm.

Even after Paré's description of aneurysm, there appeared to be some confusion as to the actual nature of the tumour. Sennertus (1628) again drew attention to the differentiation between subcutaneous and interstitial blood effusions and aneurysm. He attempted an explanation for the mode of origin of aneurysm and indicated an appreciation of the structure of arteries. An artery, he said, is made up of two coats; the external one being thin and soft, and the internal one thick, dense, and hard (media and intima). If, as a result of overstretching, the inner hard membrane be ruptured or opened by a wound, it would not readily coalesce, but the outer coat being thin and soft is readily distended by blood seeking to escape. He pointed out that such dilatation of the external coat readily becomes adherent to the skin which, when in a state of tension, is impossible to distinguish in its deeper parts. Thus, if such an aneurysm be cut into, the knife descends through the skin into the blood sac.

The history of aneurysm, because of the intimate relation between its important types and syphilis, follows closely the spread of lues through all countries. It is impossible to consider aneurysm as it is known to us to-day without an

understanding of the part played by syphilis through all ages. Yet in this we are hardly correct in attempting to associate the aneurysm of Galen with any specific disease, when the fact is borne in upon us that syphilis was an unknown disease at that time. It is probable, and the reports of Galen bear this out, that the aneurysms recorded in his time were traumatic in origin or were associated with forms of arterial disease other than syphilis. The information regarding aneurysm prior to the close of the fifteenth century is very meagre. That aneurysm was known and occasionally encountered there is no doubt, but its frequency appears to have been small and its importance was insufficient to lead to much comment. The aneurysms of the first fifteen centuries of the Christian era appear to have been mainly those attached to the peripheral vessels, and no comment is made respecting those important tumorous masses of the thorax which are of greatest interest to-day.

Almost suddenly with the beginning of the sixteenth century, aneurysm received new prominence. In part we may ascribe the greater interest to the more acute observations of the new medicine beginning with these times. However, all of the new impetus of this study cannot be ascribed to the renewed interest in medical studies, for concurrently with the extended observations on aneurysm, there appear the discussions on a new and mysterious disease spreading as a plague through Europe.

Shortly after the return of Columbus in 1493 to Spain, Diez de Isla, a physician of Andalusia stated that he had treated some of the ship's company for a new transmissible disease which had appeared among the sailors a year previously and while still in America. For some years following, this physician claimed to have had an opportunity of seeing the spread of the disease and of studying many cases in Lisbon. He collected his observations in a published work which appeared between 1510 and 1520. In this treatise he referred to the malady as the "disease of the Island of Española", and commented upon the fact that the disease was unknown in Europe before the year 1493. He found

that the members of the ship's crew observed the presence of the disease in the natives of the newly discovered land. The pilot, Pinzon, of the returned vessel had himself contracted the infection and was treated by Diez de Isla. This report is corroborated by contemporary Spanish writers, especially Las Casas, who was in America a few years after its discovery. In his royal report to Charles V, he stated that the disease was contracted from Indian women by the Spaniards with Columbus and was brought by them to Spain. Bishop Las Casas accompanied Columbus on his second voyage and remained in Haiti for a number of years. While in the new world he wrote his *Historia Generale de las Indias*. In this he stated that "there were, and still are, two things which at the beginning were very dangerous to the Spaniards. One is the disease syphilis, which in Italy is known as the 'French evil'. It is, however, known for certain that it came from this island, either when, with the return of the Admiral Don Christobal Colon with the news of the discovery of the West Indies, the first Indians arrived, whom I saw myself in Seville, or it may be that certain Spaniards were already tainted with this disease at the time of their first return to Seville."

"I took the trouble on several occasions to interrogate the Indians of this island as to whether this disease was of great antiquity, and they answered that it was present there before the first Christians came."

Other early chroniclers of Spanish-America offer similar evidence of the existence of syphilis among the Indians in the pre-Columbian period. The Indians were familiar with the symptoms and course of the disease and had well developed methods of treatment for it. It was, furthermore, observed that the disease was much milder among the natives than among the newly infected Spaniards who contracted it from them.

A number of observers claim to have found remains of human bones of pre-historic people showing characteristic lesions of syphilis. Some of these have been obtained in Colorado, California, and Arizona. It appears, however,

that these specimens have not been thoroughly identified, neither as to their origin nor as to the nature of the disease. It is possible that some of the exostoses and osseous erosions were not of a luetic kind, but the result of a more acute infection. In those cases in which the syphilitic nature of the bony lesions appeared more definite, there is lacking evidence indicating the antiquity of the bones. The same criticism has been offered regarding evidence of syphilis in Europe in the pre-Columbian time. There is no straightforward documentary evidence of the time when this disease appeared amongst the European people. Likewise the pathological evidence of syphilis of the bones has never been brought forward from early periods. Some claim that lues existed among the Romans and people of the Middle Ages. The critical study of the available material, however, and the adverse criticism of Virchow discounted their claim. The undoubted marks of syphilis upon bones dating from the sixteenth century stand in sharp contrast to the findings of the previous times. This was later substantiated by Ruffer who, in examining the arteries of mummies dating from the eighteenth dynasty onwards, and also the bones of the Coptics up to the period 200 A.D., was unable to demonstrate lesions suggestive of syphilis.

The sudden rise and wide spread of syphilis immediately following the return of Columbus in 1493 is so sharply defined that we are at least justified in stating that the disease for the first time became a menace to the public health in a manner that had never occurred before. From Barcelona it spread through the communities of Spain and soon was found in the army of Charles VIII of France. It was in 1494 that Charles began his conquest of Naples, wandering with his army of adventurers through Italy. The success of his expedition permitted his mercenary group to carry on a very loose existence amongst the conquered people. Within a year, however, Charles was again driven from Naples and after partially disbanding his forces he returned to France. Syphilis appeared in Italy in the wake of this lawless group and within another year, in 1495, it

was found in Switzerland and Germany. It spread to Holland in 1496 and to England in 1497. Wherever the disease made its first appearance, its virulence appears to have been high. It was truly epidemic and was thought by the people to be a heaven-sent plague. In many places laws were made banishing the infected from their midst. Thus within a period of less than ten years this pestilence had found firm root in Europe and had spread to a remarkable degree among all classes of people. The literature is rich in description of the many odious deformities which resulted from the infection, and syphilis appears to have been one of the most important maladies of the sixteenth century. We are, therefore, not surprised to find that aneurysm, particularly the internal aneurysm, was brought to the attention of the reawakened medical world. Syphilis could not have made its appearance in Europe at a more favourable time to bequeath us accurate accounts of epidemic lues and its many secondary processes. Vesalius, Fernelius, Paré, and Fracastorius were all attracted to a discussion of the new and mysterious malady and found evidence, during the first plague, of its association with internal aneurysm. Fracastorius, the Veronese physician (1530), labelled the new disease with the name "syphilis", in a poem which he dedicated to Cardinal Bembo.

There are not a few who deny the American origin of syphilis. The record of Las Casas is discounted as the exaggerated ramblings of an octogenarian priest who was unable to differentiate venereal disease. His suggestion that the Indians found a cure in the wood of the guiac tree lends support to the belief that more than one disease was included in his discussion. The Codex of de Isla is also brought into question, both as to the date of its publication, and the veracity of his statements claiming intimacy with the sailors of Columbus. Then again, it is claimed that an authentic letter of Peter Martyr, dated 1488, speaks of the "Morbus Gallicus". About this document there has also arisen a heated argument concerning the correctness of the date. However, all this discussion about the value of

evidence in antiquarian documents is in no way solving the question. Not until a more systematic study of skeleton-remains, dating from the early centuries, is undertaken, will greater light be thrown upon the problem. It is possible, as has been suggested by some, that syphilis in its milder or attenuated form (for mercurial treatment in other diseases was known prior to the sixteenth century), was carried to America by the adventurous crew, and being implanted into a new and non-immune race, displayed itself in a virulent form, and was returned to Spain with its renewed pathogenicity. What is of particular interest is that, contemporary with the discovery of the new world, Europe suffered a most devastating epidemic of syphilis, and with it there was an occurrence of aortic aneurysm such as had never been seen before.

Lancisi (1728), and subsequently Morgagni (1761), observed that those affected with lues venerea were peculiarly subject to diseased arteries. It is not uncommon, however, to find that these and other authors confused the various arterial lesions, viewing as the same process the nodular, scarred, steatomatous, and ulcerated lesions found on the inner arterial wall. The influence of lues in bringing about serious defects of the vessel was admitted by Scarpa, who further stated that "so great a disorder as ulceration of a large artery ought certainly to be preceded by a state of disorganization or excessive relaxation of the artery, more particularly in the part corresponding to the seat or root of the aneurysm, since aneurysms are observed to arise in persons in whom it is impossible in any way to trace the origin to external violence applied to the artery or to an increased impetus of the blood circulating in it." It was clear in his own mind that an aneurysm must necessarily have a diseased artery preceding it, for, as he later pointed out, the force of the blood stream alone is insufficient to alter seriously the shape and character of the arterial wall.

Scarpa gave an excellent description of the appearance of the nodular endarteritis of lues, but as he then proceeded with a preconceived theory of the formation of aneurysm,

he utilized the atheromatous aorta, truly not luetic, to demonstrate his point. The steatomatous degeneration with its cheesy contents renders the intima brittle, and being so slightly united to the underlying muscular coat is easily detached from it. True ulcerations are seen associated with this pathological lesion, and these are the points where weakening of the wall may lead to an extravasation of blood beyond the artery. Scarpa believed that all aneurysms were beyond the arterial coats, and that the sac containing the extravasated blood was composed of the loose cellular tissue in which the artery was embedded. Under no circumstance did he find remnants of the arterial coat in the wall of the sac. Scarpa recognized the fact that the presence of aneurysm led to bone absorption.

The discussion on aneurysm by Lancisi (1728), the distinguished Roman physician, is much the best of those of older writers. He approached the subject systematically, dealing with its distribution, pathological anatomy, classification, and etiology. He appears to be the first one to have analysed carefully the manner of development and the part played by the contained blood of arteries acting upon diseased vascular walls. During his time the more frequent occurrence of aneurysm within the thorax led him to formulate principles which are more applicable to the problem of aneurysm at the present time. He realized that there were many factors brought into play for the development of aneurysm. Quite remarkable is the freedom with which he discussed the physiological activity of the thorax, heart, and blood-stream as having a bearing upon the development of a preternatural dilatation of vessels. He too indicated that the classification of aneurysms had been poorly defined. He found fault with the opinion of former writers in not agreeing upon the cause of the affection. Thus at one time, they looked upon it as depending upon wounds of the heart or arteries, either penetrating into their cavities or merely implicating their external tunics; at another time arising from contusion, compression, or erosion, or from too violent an impulse of the blood: all of these causes, he pointed out,

did not act in the same way, but some upon the substance of the heart, and others upon that of the arteries. Anatomically he divided aneurysms into two types, true and false; true are those "that arise from causes which immediately weaken the texture and the power of resistance of the heart and arteries". Some of them result form external causes, such as wounds and contusions; and others from an internal debility of the tissues occasioned by disease. He discussed at some length the manner in which contusions and trauma upon the arteries may bring about aneurysm and he, furthermore, discussed "depraved states of the fluids" appearing in scrofulous, scorbutic, and syphilitic diseases. Lancisi believed that he was able to differentiate aneurysms of the different modes or origin, and stated that "a venereal aneurysm may be known not only by the suspicious connection and the appearance of syphilitic infection in other parts of the body, by which it has been preceded, but more especially by the manner in which any particular vessel becomes affected by aneurysm". He reported a number of cases of aneurysm in which the syphilitic nature of the disease was undoubted.

Lancisi concurred in the belief with Paré that mercury might easily bring about aneurysm. He advised against inunctions in cases of aneurysm even when evidences of syphilis were present. He was firm in his belief that false aneurysms are primarily dependent upon the impulse of the blood exceeding the capacity and ordinary powers of resistance of the arteries. This doctrine he propounded at length, illustrating his contention by various examples. Spasm of the arteries, thrombosis, or compression of the walls may all, he believed, have an important influence upon the circulation predisposing to aneurysm.

A. Monro (1733), in discussing the formation of aneurysm, depicted the manner in which traumatic lesions were associated with their occurrence. He pointed out that, when the external tunic of the artery was removed and a certain amount of the middle coat was also taken with it, there was a tendency for the remaining tissues of the arterial wall to

bulge at the site of injury. In his researches he was able to find only two or three specimens in which the wall of the aneurysmal sac consisted of the annular muscular fibres, and this he believed was the only criterion whereby true aneurysm could be distinguished.

In 1749 Haller made the remark that "aneurysms of the aorta near the heart are no longer of rare occurrence". The cases which he reported appear to have been of a luetic kind.

At about this time aneurysms appear to have been fairly common in England, for William Hunter (1757) reported his observations upon a number of them. He recognized three types of aneurysm: (1) true, by dilatation; (2) false, by rupture; (3) mixed, that is, partly by dilatation and partly by rupture. He gave as his opinion that aneurysm of the aorta was incurable. Though he recognized clearly the association of syphilis with aneurysm, he did not think that this was the only cause of the aortic disease.

Hunter appears to have been the first to recognize clearly the development of the arteriovenous aneurysm produced through blood-letting. During the operation of venu-puncture, the knife is by accident driven through the vein walls into the underlying artery. This, he found, led to the permanent establishment of a communication between the artery and vein. Hunter also described the varicose aneurysm in which the dilated artery assumes a very tortuous course. It is not clear that this type of arterial change should merit a distinct name, since the majority of his cases appear to be nothing else than the irregular dilatations and sinuous course of sclerotic arteries. He spoke of the serpentine appearance of the thoracic aorta of a woman, as well as of similar characters in the peripheral arteries of the extremities.

A very interesting series of aneurysms was observed by D. Monro in 1760. Some of the lesions he recognized as arising in association with injury, while the internal thoracic type he believed of spontaneous origin. His observations upon these aneurysms, each of which was carefully dissected

in its minutest part, are very instructive in indicating the care with which pathological processes were studied with the crude instruments then available. Each layer of tissue was unfolded with patience to observe its relation to the surrounding structures. In this way the descriptions of the walls of aneurysms are almost as clear as those available to us to-day where the microscope is used to reveal the intricacies of tissue architecture. Monro, by such careful dissections, was able to show that true aneurysms may exist with the dilatation of all the coats of the arteries, or that the aneurysm may possess in its covering only portions of each layer. He stated: "That all the coats of arteries are sometimes dilated is plain to ocular demonstration from the dissection of the aneurysmal sacs in the case of John Parker. It may seem surprising that I should quote so particularly this case for a fact now so generally looked upon as common. The reason of it is that, although we have many histories of cases of true aneurysms related, yet I have not found one of all those I have examined where the different coats of the arteries have been traced by dissection, continued over the whole circumference of the aneurysmal sacs. Haller is the only good anatomist who says he observed all the arterious coats, particularly the muscular, in an aneurysm. But then he gives no figures of these coats, nor is he particular in the history of their dissection."

Monro could offer no common cause for the majority of true aneurysms. "Strainings of the body, an increased momentum of the blood, and a stop put to its free circulation through some of the large vessels, has been alleged to give rise to several." He found, however, that "sometimes a scorbutic or venereal taint, or some other acrimony in the blood has been accused", and he himself felt that "there must either be a particular disposition of the blood, which renders it capable of softening or relaxing the arterial fibres, or some disorder in the fibres or vessels affected, before an aneurysm can be produced." He found, however, that there was nothing very definite to which the cause of aneurysm might be ascribed.

Monro, reviewing the reports of aneurysms up to his time, found that hundreds of them had appeared in literature. Most of the authors, however, gave very meagre descriptions of the sac, and few of them undertook careful dissections. Not a few comments were made by various authors as to the cause, but in the majority of instances the words of former writers were carried forward without the addition of material information. He commented upon the statement by Ætius, Fernelius, and Paré that aneurysms are common in the neck, claiming with Heister that this did not conform with subsequent observation. It may be that the pulsating tumour which was observed in the neck of pregnant women, and to which Ætius applied the term "bronchocele", was in truth an enlarged thyroid.

Monro remarked upon the frequency of aneurysms of the aorta near the heart, being "almost equal to, if not greater than, those in all other parts of the body together". These conclusions were arrived at by a study of the cases reported by Lancisi and subsequent writers. As these reports were made in the post-Columbian period the statement still holds at the present time. It is still true to-day that aneurysms of sufficient importance to attract attention are for the most part located within the chest; nor has the type of aneurysm described by Monro changed in any manner. The mode of death was possibly more frequently by rupture in his time than in ours. The present-day intensive antiluetic treatment has held in check the progress of early aneurysm, whereby the increased burden of the circulation has been borne by the heart.

Comments made by Baillie (1789) upon aneurysm are very clear, and although he did not associate its development with lues, it is unquestionable that the specimens which he described were of this kind. "The most frequent situation of aneurysm within the cavity of the thorax is at the arch of the aorta. In this disease the arch of the aorta is much enlarged beyond its usual size, sometimes forming a uniform tumour; sometimes a large swelling which arises out of the arch of the aorta by a neck or narrower portion;

and sometimes smaller swellings arise out of the larger one. Where the swelling is uniform in its shape, there is reason to believe that there has been a considerable dilatation of all the coats of the artery. Where the swelling arises from the arch of the aorta by a neck or narrower portion, the inner and middle coat of the artery have been burst or ulcerated, and a dilatation takes place in the outer coat, which is strengthened by the condensation of the parts immediately surrounding it. The coats of the artery, both at the place where the aneurysm is formed, and near it, are considerably altered from their natural structure. The coats of the artery in the neighbourhood of the aneurysm are often found to be very irregular in their texture, being in some places transparent and thin, in others thick and opaque, and there is sometimes the appearance of a double internal membrane. The same sort of structure is also sometimes to be found in the coats of the aneurysm itself."

"The reason why aneurysms take place more frequently in the arch of the aorta, than in any other part of the arterial system is its curvature, which exposes it to the full impetus of the blood propelled by the strength of the left ventricle. Aneurysms hardly ever happen in the pulmonary artery because there is no arch formed by the pulmonary artery, and the blood readily passes by two large branches into the substance of the lungs."

"Aneurysms in the arch of the aorta, as well as in every other part of the arterial system, happen much more rarely in women than in men. This arises from two causes. The one is, that women, from their sedentary life and temperance, are less liable to an increased impetus of the blood, occasioned by excited circulation; the other is, that the arteries in this sex appear to be less liable to these diseased alterations of structure, which predispose to aneurysm."

Scarpa in his "Treatise on Aneurysm" (1804) stated in the most certain and unequivocal manner that there was only one kind of form of this disease, viz., that caused by a solution of continuity or rupture of the proper coats of the artery, with effusion of blood into the surrounding cellular

substance; that aneurysm, in whatever part of the body it was formed, and from whatever cause it arose, was never occasioned by the dilatations, but by the rupture or ulceration of the internal muscular coats of the artery.

Corvisart (1812), in describing a case of aneurysm of the aorta, said: "The ventricular orifice on this [left] side was large. We saw on the mitral valve vegetations analogous to venereal excrescences." But he made no suggestion that these may have caused the aneurysm.

This author distinguished between true and false aneurysm of the aorta: true, if all the coats of the artery are dilated; false, if one or more coats are ruptured and the others dilated. He thought that the greatest number of aneurysms belong under true; some also to both classes. He studied two cases in which he found no opening from the tumour into the artery, and so he concluded that aneurysms, instead of forming from the inside out, form from the outside to the inside by a sort of corrosion of the same coats. "Besides," he said, "the aneurysmal sac, instead of being furnished by one or several dilated arterial coats, would be wholly formed by an adhering fibrous membranous sac, but extraneous to the coats of the artery."

He attributed the formation of true aneurysms of the aorta to two chief causes: "To the first is referred whatever belongs to the derangements of the circulation, as (1) the augmentation of the impulse of the blood, (2) the obstacle to the progress of the blood beyond the part that is dilated. To the second, whatever may induce or favour any debility, or disorganization in the parietes of the vessel, as violent efforts, blows applied upon the chest, the translation of morbific matter, and its attachment to the parietes of the vessel."

Lobstein (1833), who is the author of the term "arteriosclerosis", believed that true aneurysm was not caused by the lateral pressure of the blood, but followed in the wake of arteriosclerosis. According to Scarpa, all aneurysms without distinction arise through rupture of the arterial coats following upon their degeneration, "whether ulcerous,

steatomatous, fungus, or squamous," and that the sac is formed by the dilatation of the external coat of the artery. In other words, he believed that the aneurysm was not contained within the true arterial wall, but only within a small portion of its exterior. Lobstein disagreed with this, inasmuch as the theory was not applicable to diffuse or fusiform aneurysms. Dubois and Dupuytren found that the internal coat of the artery was always extended over the inner surface of the aneurysmal sac. Lænnec, in the early part of the nineteenth century, recognized the great damage which was done to the middle coat of the artery and observed the disappearance of the muscular tissue in its structure. His means, however, to obtain a thorough understanding of the extensive destruction which takes place in syphilitic aortitis were limited.

The important credit, however, falls to the medical officers of the British Army of impressing on the medical profession the intimacy between syphilis and aneurysm. These men, Crisp in 1847, Lewer in 1862, Aiken in 1866, Lawson in 1868, Myers in 1870, and Welch in 1876, presented irrefutable evidence by the demonstration at autopsy of the common presence of aortic disease in known syphilitics and by showing the stages of development of the aneurysmal pouches in these luetic aortas. Syphilis amongst soldiers had always been accepted as high in incidence, but it had never been so forcefully shown that the infected groups had a definitely high percentage of internal aneurysm. The work of Crisp was of particular importance, as it was the first monograph in the English language dealing with the subject in a general manner and based upon a large number of cases. Even at this time (1847) Crisp was able to collect 551 cases of spontaneous aneurysm from the British medical journals between the years 1785 and 1847. The cases were carefully analysed as to the distribution upon the arteries, the age periods of occurrence, sex, and history of syphilis. Crisp agreed with the view of Morgagni that the aneurysms of the popliteal artery, which were so frequently met with in his day, occurred mostly amongst postilions and cavalry-

men. He believed that a frequently recurring injury in the posterior cavity of the knee had its direct effect upon the artery and was the reason for the occurrence of the aneurysm. Lewer reported a case of multiple small aneurysms upon the thoracic aorta occurring in a young man of twenty-five who at the time was presenting signs of secondary syphilis. Aiken (1866) of the Army Medical School found lesions in the aorta in seventeen young syphilitic soldiers in a total of twenty-six syphilitics who had died. The aortic lesions, he stated, are a "cicatrical-like loss of substance of the inner coats, small local dilatations of the artery, and in several cases aneurysmal expansions, one as large as an orange which proved fatal."

Inspector-General Lawson found a marked increase in the incidence of aneurysm amongst the soldiers at Aldershot between the years 1860-1868. His observations led him to believe that less importance must be placed upon the rôle played by the degenerative processes of the common arterial diseases in respect to the appearance of aneurysm. He concluded that "it would be going too far to discard the influence of this degeneration altogether, but certainly aneurysm, as I have met with it among soldiers, seems frequently to exist quite independent of that form of disease of the arteries, and destruction of large portions or even the whole three coats may take place by an acute process, and without a trace of atheroma in the neighbourhood."

Subsequently in 1875, the paper presented by Francis H. Welch, assistant professor of Pathology at Netley, added the closing words to the evidence linking clinical syphilis with the development of aneurysm. He reported a study of thirty-four fatal cases of aortic aneurysm in soldiers at Netley. The average age was thirty-two; the youngest was twenty-six. In the majority of cases the first part of the thoracic aorta was involved, and in five the aneurysms were multiple. In over fifty per cent. there was a definite history of syphilis, while the aorta in all cases had a similar and characteristic picture. He could find no ground for the claim that aneurysm had any relation to constriction of

the chest or arterial obstruction, but believed that the aorta must needs be damaged in its several coats before dilatation took place. And he added, "While no dilatation need ensue under ordinary arterial pressure or such as might be present under civil exertion, yet under the forced exertions of army exercises, with chest constriction, it would be difficult to understand how such a crippled tube as a degenerate and weakened aorta could resist the extra internal pressure." With the number of cases of aneurysm and syphilitic aortitis which passed through his hands, Welch clearly distinguished the specific aortic disease from the ordinary arteriosclerosis and nodular endarteritis. The puckered scars and the radiating grooves coursing over the thickened inner coat of the artery served to distinguish this as a distinct type of disease. A few years later Malmsten collected 101 cases of aneurysm in one of the Swedish hospitals and found that eighty per cent. of these had a syphilitic origin. Subsequently Heller found syphilis to be present in eighty-five per cent. of the cases of aneurysm, Pansini in eighty-four per cent., and Rasch in eighty-two per cent. There are others who during these years of controversy failed to support by clinical evidence the syphilitic nature of aneurysms in as high a percentage as given by the above authors. Heller indicated that at the autopsy only three per cent. of cases having had syphilis showed aneurysm, two per cent. possessed cerebral lesions, two per cent. showed cardiac lesions, and about one per cent. had recognizable changes in the lungs.

The importance of syphilis was thus well established by the latter part of the nineteenth century, although in some quarters the part played by arteriosclerosis, high blood pressure, anomalies of structure, and other infections, was still maintained to be equal to its influence in the origin of aneurysm. The microscopic differentiation of the syphilitic lesion from other forms of disease had not been made and the causation of the infection was not yet discovered. However, the therapeutic test as shown by the French authors, Fournier, Vallin, Verdie, and others, as well as the reports

by Muelhaus and Schmidt in Germany, gave further support to the syphilitic nature of the process.

The above studies have been made in relation to aneurysm of the aorta, and do not refer to the less frequent and less important aneurysms arising in the peripheral branches of the arteries. The latter were found to have a variable etiology, in which syphilis played no very important part.

An important work which is frequently overlooked is that of Barkow, who published three unwieldy volumes in 1869, 1871, and 1872. In his work he described the tortuosities and the dilatations of arteries and illustrated his work profusely. These volumes, in fact, represent the first work wherein care was taken to draw the arterial system in detail and represent the characters of the arterial lesions as they were found in the cadaver. Although this author did not discuss the importance of syphilis as observed by him, his drawings show syphilitic lesions of the aortic wall in many of the cases where distortions and dilatations occupied their course. Barkow, moreover, found that aneurysm was not only a lesion of human arteries, but also of lower animals, particularly the horse. It is unfortunate that this author, with his wide experience in the study of aneurysm, did not accompany the excellent plates by a discussion of the nature of the lesion and by an analysis of his entire series, as to the incidence, distribution, and manner of development of these aneurysms.

In 1885 Doehle, a pupil of Heller, made a definite contribution to the knowledge of aneurysm. He carefully studied a case in which the aorta was markedly involved in a disease process and there was present a saccular aneurysm. In studying the lesions of the aorta in its various portions, he was able to find various grades of destruction and varieties of reaction, but amidst these he found that in the areas of advancing disease, there were lesions which had all the characteristics of a syphilitic process. Doehle described these in detail and compared them with the gummatous reactions found elsewhere. This work was the beginning of the better understanding of the aortic lesions arising in syphilis, and

was also the beginning of our appreciation of the destructive quality of the syphilitic process whereby aneurysm was made possible. Doehle followed up this report, some ten years later, by a confirmatory analysis of two new cases. At the same time, Backhaus repeated the work of Doehle, and confirmed it in its details. Backhaus indicated that the reactions in the intima of the syphilitic arteries differ from the ordinary arteriosclerosis inasmuch as they seldom show fatty changes and rarely give rise to calcification. These authors were the first to state that the syphilitic process upon arteries is a lesion quite distinct from that arising from other causes, and is to be separated from the nodular endarteritis previously described by Virchow. It is true that although Doehle and others found true gummata within the aortic wall, and evidences of gummy necrosis, this is not the usual finding, for in syphilitic aortitis the lesion is of the nature of a low-grade inflammatory reaction with a perivascular infiltration by lymphocytes invading the media, but seldom giving rise to the specific inflammatory reaction which is recognized as a gumma. Giant cells in the lesions of the aorta are unusual.

It is interesting to note that Koester, in 1875, described a type of mesarteritis associated with nodular endarteritis. Koester believed that all types of nodular endarteritis and particularly those of the aorta, arise in consequence to inflammatory processes localizing in the middle coat of the arteries. It is possible that a number of cases examined by him were truly syphilitic in character, but it is also probable that other instances showing nodular endarteritis were not syphilitic in origin, but were related to other infections, as is recognized to-day in rheumatism, typhoid, and bacteræmias.

Finally, in 1904, Chiari reviewed the work of antecedent authors and repeated the studies upon endarteritis of different sources. He concluded that there was one variety of endarteritis which was always secondary to a mesarteritis. In this group, the syphilitic lesion was most conspicuous, and to it he gave the name "chronic productive mesarteritis".

Chiari's work was a step in advance, in that he recognized a primary and a secondary endarteritis, and also inasmuch as he emphasized the importance of the medial inflammation in the one group. Furthermore, whereas Koester had drawn attention to the presence of a mesarteritis in certain cases of "arteriosclerosis", the differential diagnosis between the luetic type and other arterial infections remained uncertain. Chiari brought out forcefully the characteristics of the macroscopic lesion as well as the differential points in the microscopic picture. A review of the important modern work which appeared up to the investigations of Chiari are fully detailed in the monograph by Benda. In 1905 Schaudinn and Hoffmann reported the discovery of the treponema pallida in syphilitic lesions.

Types of Aneurysm.—In our discussion upon aneurysm we include only the dilatations which occur in the course of an artery. It has always been the practice since the time of Lancisi to divide aneurysms into the two large groups: (1) the true aneurysm, and (2) the false. The latter group are of little importance, for they represent hæmatomata which often are only temporary, but at other times are of longer duration and develop a lining membrane which is continuous with the vessel with which they communicate. The true aneurysms are pouchings of the arterial wall in which one or more coats of the vessel are maintained. It was at one time considered essential to have all three coats present in aneurysmal dilatation to permit of the use of the term "true aneurysm". This, we realize, is not necessarily the case inasmuch as secondary processes may lead to the destruction of certain portions of the arterial wall, depriving it of one of its coats. Such is not uncommonly the case in the massive globular or saccular aneurysms where the wall has been stretched to its maximum and atrophy and disease have led to the disappearance of the media. As long as the cavity of the aneurysm is bounded by the tissues of the arterial wall, an aneurysma verum exists.

These true aneurysms assume a variety of shapes and

bring about great distortions of the lumen of the vessel. In some instances, the wall of the artery has been weakened equally in its circumference, and the local dilatation is uniform. This may give rise to a bottle-shaped enlargement of the vessel, or at times it is smoothly elliptical, the afferent portion of the artery gradually passing into the greatest dilatation of the cylinder and then again reducing its diameter to meet with the normal circumference at the distal extremity. These are spoken of as the "fusiform aneurysms". And then again there are the curious tortuous dilated arteries which affect not only a local portion of the vessel structure, but come to occupy considerable stretches of its extent as well as progressing along the branches of the main vessels so as to form a tangled mass of dilated arteries and spoken of as a "circoid aneurysm". This type is not uncommonly of congenital origin, and it would appear that its beginning is in relation to an inherited weakness of the vessel wall. Of this type we need offer but little discussion. Curiously enough, Crisp was able to collect as many as forty-five cases from the early literature. I doubt whether the literature of the last few years will offer as many. Finally we have also to deal with the dissecting aneurysms wherein a pocket has formed within the arterial coats themselves so that the blood lies between two strips of the arterial wall, bounded on the outside by the outer border of the media and adventitia, and on the inside by the inner two-thirds of the media and the intima. These aneurysms are unique and differ essentially from all other varieties. The most important of all is the saccular aneurysm or some variety of it.

As these aneurysms, the saccular, the fusiform, the circoid, and the dissecting type, differ in their appearance and in the locations in which they are found, so too it is to be noted that the causes leading to their development are different. For this reason it is not possible to determine upon an etiological factor of aneurysm as applicable to all types, although it is found that some of the conditions leading to the development of aneurysm may bring about changes giving rise to more than one variety.

The subject of aneurysm is so large that we will be forced to confine our remarks respecting the nature and distribution of this disease to the more important types. Aneurysm may make its appearance in every vessel in the body, but the incidence in which it occurs in certain regions is so striking that it is advisable to deal with the commoner groups, and not to attempt to enter upon an analysis of the less frequent varieties. Furthermore, the etiological factors which, in themselves, determine the position of the aneurysm vary greatly in the frequency with which they act upon the arterial wall, and are not to be weighed with equal importance in our study of aneurysm. The lesion has a greater importance to the physician than to the surgeon, although in the past, when aneurysms upon the external parts of the body were more frequent than they are to-day, the reverse was the case. Internal aneurysms, those found within the thorax, the abdomen, and the skull, are much more common than ones appearing in the extremities or upon the surface areas of the body. As we will point out later, there is good reason why this change has taken place in modern times, and why the condition is one of the serious problems of internal medicine. The surgeon to-day is rarely called upon to deal with aneurysms of the brachial, popliteal, or carotid arteries, and although surgical methods and technique have been vastly improved over those of a century ago, we can claim little advance in the surgical treatment of these cases.

Inasmuch as aneurysms are the outcome of definite lesions, and in the majority of cases the result of a parasitic attack upon the arterial wall, the incidence of aneurysm varies very much in different countries, in relation to the frequency of specific infections. The statistics which are available indicating the frequency of aneurysm in various districts are not very reliable, as we find authors differing in their reports obtained from the same country. Nevertheless, a general idea can be gained from the autopsy statistics of large hospitals.

Aneurysms resulting from Syphilis.—Naturally the frequency of syphilitic aneurysms will bear a direct relation to the incidence of syphilis amongst the people. In estimating the prevalence of syphilis we are confronted with the divergence of the figures which are offered on the one hand upon clinical examination, and on the other through the Wassermann reaction. Furthermore, if we compare this evidence with that gained at the autopsy table, we again find a great variation in the results obtained. Based upon clinical evidence alone, it was stated that syphilis in the German army prior to 1907 was present in 19.8, in France in 28.6, in Austria in 54.2, in Russia in 62.7, in the United Kingdom in 68.4, and in the United States in 167.8 individuals per thousand. At the Johns Hopkins Hospital in 1922 it was found that the Wassermann reaction was positive in 11.8 per cent. of all admissions. At the Toronto General Hospital in 1923 the Wassermann reaction was positive in 6.4 per cent.; in 1924, 4.8 per cent.; in 1925, 5.3 per cent. of admissions to the medical wards (Detweiler). On the other hand, McNeil reported a positive Wassermann reaction in 34 per cent. of negroes in the Southern States, while Lynch found that a positive Wassermann reaction may be obtained in 65 per cent. of negro women. Other reports from the Southern States give an even higher incidence of syphilis among the coloured people, and in Brazil, both at Rio de Janeiro and Sao Paulo, the percentage of positive Wassermann reactions was over 70.

In my series of one thousand autopsies carried out at Pittsburgh, I encountered aneurysm in thirty-one cases. In sixteen of these, the aneurysm was located in the aorta; the remaining fifteen were variously distributed along the peripheral arteries. Of the sixteen aneurysms of the aorta, two were multiple, two were acute mycotic in origin, two were in the sinus of Valsalva, and one was in the abdominal aorta. In three out of the fifteen aneurysms upon the peripheral arteries, the lesions were multiple, while the remaining were distributed as follows: splenic three, hepatic two, cystic two, mesenteric two, iliac two, femoral one, ductus arteriosus one,

intercostal one, pancreatic one, posterior tibial one, renal one. In two of the cases showing multiple aneurysms the lesions were of the nature of a periarteritis nodosa.

There is undoubtedly a great divergence in the frequency of syphilis in different countries, and although syphilis is the most important factor having to do with the development of aneurysms in the aorta, one cannot immediately conclude from the high proportion of syphilis as to the distribution and frequency of aneurysms in different races. With the aid of the Wassermann reaction, latent syphilis is diagnosed much more commonly to-day than it has been in the past; and as aneurysm develops only after a period of years following the primary infection, it becomes possible to undertake active treatment of the syphilitic cases so that they do not progress with the dire results. Aneurysm is not only dependent upon the incidence of syphilis, but also upon the want of care with which treatment is instituted when the disease is recognized. Communities having a high proportion of syphilitic infection may, to some degree, control the development of aneurysm through active treatment. Symmers reported a case of syphilis arising in a doctor who received intelligent treatment for a period of three years, but who at death showed a definite syphilitic aortitis.

Syphilis as a disease has its peculiarities, the most striking of which is that the tertiary lesions are the most important and are insidious in their development, occupying a period of years before producing serious injury. This is particularly true of the arteries, for it is rare to find the syphilitic process injuring the arterial wall during the early period of its occurrence. A few cases have been reported of syphilitic aortitis making its appearance during the first year of the infection. The virus of syphilis tends to localize in a number of tissues and remain latent or become slowly progressive. Warthin has shown that the heart is not uncommonly involved by the infection which persists for long periods of time. The same, however, is also true of the arteries and particularly the aorta, where the injury becomes recog-

nizable only after a period of years and aneurysm is not evident before ten or more years. In Cummer and Dexter's series of cases there was an average of 17.2 years' interval between infection and the appearance of symptoms; in Longcope's series an interval of 16.4 years, and in Denke's the interval was 20 years. It is important too to appreciate that, although in its early manifestations, syphilis shows lesions becoming rapidly necrotic, the later effects of the virus commonly fail to show the intensity of the reaction, and true gummy necrosis is often wanting in the slowly destructive lesions.

The aorta is one of the common points for the localization of the infection, and Symmers found that out of three hundred and fourteen cases showing a positive Wassermann reaction, 55.7 per cent. showed a syphilitic aortitis. He, furthermore, found that of these three hundred and fourteen cases of syphilis, aneurysm was present in forty-five. Larkin and Levy, after studying the pathological lesions in the aorta in Wassermann positive cases, came to the conclusion that it was highly probable that about ninety per cent. of individuals dying with a positive Wassermann, granted that the infection was not recently acquired, have, from a histological point of view, a luetic aortitis; that sixty per cent. of them die from their aortitis; and that ninety-four per cent. of patients suffering with aortitis give a positive Wassermann reaction. Citron found ninety per cent. of them positive. Obendorfer found that a syphilitic aortitis was present in 6.89 per cent. of all autopsies. A few cases of syphilitic aortitis of congenital origin have been recorded (Weissner, Bruns, Klotz, Nixon), and a single case of aneurysm of the abdominal aorta resulting from congenital syphilis was described by Nixon.

Of the cases showing syphilitic aortitis at autopsy, over thirty per cent. showed saccular aneurysm or the early stages of pouching and dilatation of the aortic wall. Schroetter found aneurysm in only 230 of 19,300 post mortems carried on in Vienna over a period of ten years. Eppinger reported 22 aneurysms in 3,149 post mortems, while at Guy's

Hospital, in a series of 18,678 autopsies, 325 disclosed the presence of aneurysm. These reports were compiled prior to the routine performance of the Wassermann reaction, and one is unable to compare the incidence of this lesion to the frequency of syphilis.

Although the treponema pallidum has been repeatedly demonstrated in the aortic lesions and in the tissues of the wall of the aneurysm (Wright, Benda, Reuter, Klotz, Schmorl, Longcope), the organism is not always easily found. This parasite does not permeate the injured areas in the aorta, nor does it persist in the old scarred areas. We have discovered it mainly in the advancing borders, grouped in small clusters about the inflammatory reactions near the vasa vasorum. Many times it is necessary to prepare repeated blocks of tissue before a successful demonstration is made. Symmers failed to find any evidence of the treponema in thirty cases of syphilitic aortitis which were studied. However, this same author obtained a positive Wassermann reaction in 78.5 per cent. of cases (seventy in all) which showed undoubted syphilitic aortitis at autopsy. No doubt a certain number of the negative Wassermann reactions were due to active treatment.

The common age at which aortic aneurysm makes its appearance lies between thirty and fifty years. Before the age of twenty, aneurysms are relatively rare, and those which do make their appearance are usually not of syphilitic origin. The fourth and fifth decades have an almost equal number of cases of aneurysm, and after the age of fifty the numbers slowly decline, but are maintained high because of the appearance of another type having no relation to syphilis, the dissecting aneurysm. In Longcope's series of sixty-three cases, the majority of them were young, eighty per cent. being between the ages of twenty and thirty years. This is unusually early. Syphilitic aortitis in its severe form is usually encountered five or more years after the initial infection and the aneurysm which occurs with it appears at a still later date. Most commonly the first appearance of the lesion of syphilis in the arteries is found in the ascending

aorta along the inner and posterior wall. At this point a small perivascular reaction is found in the vessels entering the aorta from its outer surface and, seemingly extending from the tissues of the mediastinum, invades the outer and middle coat of the artery. The reaction progresses very slowly, bringing about a destructive lesion which particularly affects the elastic tissues and the musculature of the wall. At first these lesions are almost inconspicuous, forming narrow paths of destruction along the tender nourishing vessels of the aorta. They remain confined to the outer coats until the local reaction has led to inflammatory accumulations which are followed by the development of new vascular channels penetrating deeply into the middle coat and close below the intima. These early lesions cannot be detected from the inner side of the vessel wall, and show no evidence of their presence save by microscopic examination. Subsequently and in response to the presence of the low grade inflammatory process of the middle coat of the aorta, the intima responds in a nodular proliferation over the site of the injury.

From this local focus the virus is disseminated into the neighbouring parts, and at each new location it tends to follow the small vessels in the adventitia and the media. Thus the virus follows the ramifications of the nutrient arteries, and gradually involves more and more the first portion of the aortic wall. In a previous study (Klotz) upon the manner of localization of the virus of syphilis upon the aorta, I was led to conclude that it entered the tissues by way of the lymphatics from the mediastinum. It is important to note that within mediastinal tissues themselves, a syphilitic process is rarely encountered. Furthermore, the virus as it appears in these structures seems to have lost some of its virulency, and does not tend to give rise to necrotic processes as seen in the earlier lesions. In this respect the virus somewhat simulates that which Levaditi has described for infections of the nervous tissues. In the latter, this author has found that the virus has changed its character to the extent that it will no longer give rise to the classic cutaneous

lesions, but will produce diffuse reactions within the brain. In syphilitic aortitis it is likewise found that gummy necroses are unusual, and ulceration upon the aortic wall is only rarely encountered. The typical lesion of syphilis which we observe at the autopsy table in specific aortitis is the late reaction occurring in the intima after much damage has been done in the media. The intimal reaction is entirely proliferative and in itself has no specific character indicating a syphilitic infection. We have, of course, come to appreciate that an endarteritis is the usual sequel to the presence of the treponema in the arterial wall, but a study of these arteries will show that the wall has received its damage in the outer coats, and that the intima has not been injured in necrosis, but has responded in an overgrowth.

Thus syphilitic aortitis is first to be looked for in the ascending aorta. Next, most commonly we find that the lesion lies in the arch and, following this in lesser frequency, within the thoracic aorta. In almost all instances where the arch or the thoracic aorta, or both, show a syphilitic process, we find that the earliest damage has occurred close to the heart. It is remarkable to note that in the majority of cases of syphilitic aortitis within the thorax, the lesion seldom goes beyond the diaphragm. It is common to find a syphilitic aortitis spreading through the aorta within the chest and coming to an abrupt ending at the point where the aorta passes through the diaphragm. It would appear that the virus does not find ready access to the lymphatic channels lying beyond this structure. It is likewise interesting to observe that a syphilitic aortitis involving the arch frequently leads to an extensive nodular overgrowth about the mouths of the vessels entering the neck and yet fails to enter these vessels themselves. I have repeatedly seen healthy carotids and innominate artery attached to a seriously diseased aorta.

The abdominal aorta is but seldom involved in a syphilitic process. When it does occur its most common site of development is about the coeliac axis, which is the point where the abdominal lymphatics centralize about the aortic

wall and encircle it in a rich network of lymph channels. Occasionally we observe the primary and only location of syphilitic aortitis in this vicinity, but it is more common to observe the dual location, one in the thorax and the other in this restricted area in the abdominal aorta. At Johns Hopkins Hospital, Osler saw only sixteen cases of abdominal aneurysm in a total of over 16,000 admissions to the medical wards; at Vienna, in over 19,000 autopsies only three cases were reported; while at Guy's Hospital in an almost equal number of autopsies, fifty-four cases were recorded. In 1911, Nixon collected two hundred and thirty-three cases of abdominal aneurysm in the literature, of which one hundred and fifty-two had ruptured, either into the abdominal cavity or into the retroperitoneal tissues. This number of aneurysms of the abdominal aorta were not all of syphilitic origin, some of them being the outcome of acute infections or erosions of the arterial wall through the presence of infectious processes in the vicinity. Nixon reported a case of abdominal aneurysm appearing in a girl of twenty and due to congenital syphilis. This is the only case of this kind on record.

The reason for the development of aneurysm following upon the presence of syphilis in the aorta is entirely dependent upon the weakness induced in the arterial tissues. The stretching of the wall is, of course, the direct outcome of the blood-pressure within the vessel, but, as the normal arteries are provided with a resisting strength equal to six or seven times the normal blood-pressure, it is necessary that a sufficient damage is done in the sustaining tissues before the dilatation occurs. This is one reason why the development of aneurysm is long delayed, as the destructive process by syphilis goes on gradually and does not have its effect quickly, as is observed in connection with the acute mycotic aneurysms. Under the ordinary conditions of the appearance of a syphilitic aneurysm there is no increased blood-pressure, although the effect of the pressures which are commonly observed in individuals may make themselves felt when, through physical strain, the blood-pressure is for a short

period greatly increased in the central vessels. This is not uncommonly observed in individuals who, having suffered syphilitic aortitis for many months, suddenly are aware of the vascular weakness when, upon undertaking heavy work, they suffer a sudden onset of pain and discomfort. At these times the vessel wall is put under greater tension and some of the fibres within the muscular coat give way. As Benda stated, every aneurysm is a pulsion dilatation of an artery. In general, however, the aneurysm enlarges but slowly, advancing only in the relative proportion in which the balance between the strength of the vessel and the blood-pressure contained within it is undone.

The syphilitic process in the arterial wall is scattered in small foci over variable stretches of its extent, and these foci, as they enlarge, fuse with each other to give rise to larger patches of tissue destruction and fibrosis. It is to be remembered, however, that there are always areas of normal tissue lying between these involved areas in which little or no influence by the virus of syphilis has been produced.

The treponema pallidum can be demonstrated in the involved tissues along the course of the vasa vasorum. At times difficulty is encountered in finding the microörganism, but if search is made along the advancing lesion, and not in the areas which have suffered destruction and fibrosis, one can usually succeed in finding fair numbers. It is our belief that the syphilitic infection is never absent from the tissues of specific aortitis and aneurysm in untreated cases, and it has also been stated that the treponema is dealt with with greater difficulty in syphilitic aortitis than in the majority of the syphilitic processes of internal organs. Strenuous treatment may fail to eradicate the infection from the aortic wall, but whether this is due to the peculiar location of the infective agent lying in a tissue poorly vascularized, or whether the long habitation of the organism in these peculiar tissues have given to it a new and resisting character, we are unable to say. In this respect the unsatisfactory result in the treatment of syphilitic aortitis

somewhat resembles that encountered in syphilis of the nervous system.

The spirochaete within the wall of the aorta is not rapidly disseminated to new areas. The organism does not appear to multiply very rapidly, and only small collections are found in the active foci. Usually the organisms lie within a short radius of the progressive lesion, and do not tend to infiltrate great distances beyond it. It is uncommon to observe the presence of the spirochaete in the tissues of the intima, except in those cases where the small branches of the vasa vasorum have penetrated beyond their normal reach through the inner two-thirds of the media and have entered the deep layers of the intima. This extension of the vascular channels is quite unique in syphilitic aortitis, for in other regions we do not observe the progressive extension of the capillary circulation along with the progress of the granulomatous reaction. In general, granulomata do not cause the greater vascularization of the infected tissue; but in this instance we have an exception, and it is almost always found that the tissues of the aorta show a greater penetration by fine blood vessels than they do normally. This increased vascularization is present both during the early stages of the inflammatory processes when the aortic wall is thicker than normal, as well as under those conditions where the damage has led to a thinning of the arterial structures and the development of aneurysm.

Although in our discussion upon aneurysms resulting from syphilis we have up to the present confined ourselves to the observations upon the aorta, it is to be noted that similar lesions passing through similar stages of reaction may be found elsewhere. Aside from the aorta, however, the smaller vessels are less frequently attacked. Here and there it is found that the syphilitic process has not limited itself to the aortic structures, but has advanced along the vessels of the neck and has led to much destruction of the tissues of the carotids and the innominate artery. I have never seen a case of localized syphilitic disease of any one of the vessels of the neck without a neighbouring syphilitic

aortitis. The same is also true of the occasional aneurysm of the coeliac axis where a syphilitic process present in the abdominal aorta has advanced into this artery with destructive effects. The nodular endarteritis accompanying the syphilitic mesaortitis may at times be so marked as to occlude the mouths of the small vessels arising from the aorta, and instances have been observed with advanced stenosis of the mouths of the coronary arteries or even almost complete occlusion of as large vessels as the innominate artery (Darling and Clark).

Distant from the aorta we occasionally encounter the syphilitic lesions of the vessels of the brain, but the incidence to-day is less frequent than was reported years ago. It is probable that many of the cases of military aneurysm of the cerebral arteries were accredited in the past to a syphilitic process. These lesions, which are not uncommon in nodular endarteritis, are not necessarily of luetic origin. Likewise, the nodular endarteritis which is found along the course of the coronary arteries of the heart bears no relation to syphilis. It is true, of course, that the small arterioles in the vicinity of the primary and tertiary ulcerating lesions of syphilis suffer an endarteritis which may almost completely obliterate their lumina; and a similar reaction is encountered in the vessels of the placenta when congenital lues is found. Other than these, however, it is quite uncommon to observe syphilitic processes and aneurysm about the peripheral arteries.

In a series of 1000 aneurysms collected from the literature of the last twenty-five years, there were 696 males, 278 females and 26 in which the sex was not stated. Of these 1000 cases, 695 occurred in the aorta, 133 in the brain and 202 in other regions. Of the aortic aneurysms 587 were within the thorax, 85 were abdominal, and 23 showed the presence of an aneurysm both within the thorax and the abdomen. Of these 1000 cases of aneurysm, only 94 had appeared in individuals under the age of twenty and only a few of these were of syphilitic origin. The highest incidence of aneurysm of the aorta, which as we have said is the common one resulting from syphilis, lies between the ages of thirty

and sixty. In our series the number of cases was fairly uniformly distributed in the fourth, fifth, and sixth decades, being 152, 166, and 136. This is in agreement with the reports of others (Crisp, Paul, Hare, Lemann, Boyd, Symmers, Longcope, and others).

Rupture of the aneurysm with severe hæmorrhage is the most common cause of death. In our series this fatal ending was noted in 55.8 per cent., while Boyd, in reviewing a series of 4000 cases of thoracic aneurysm, found rupture to have taken place in 52 per cent. of the cases. The manner of the rupture and the direction in which the blood escapes varies much with the location of the aneurysm. When the aneurysm has developed upon the first portion of the aorta, a large portion of it is found to project within the pericardial sac and rupture into the pericardium is not an infrequent event. On the other hand, the development of the aneurysm may have given rise to adhesion to the pulmonary artery or to the vena cava, or have been projected against the right or left auricle, so that with its subsequent enlargement and erosion, the escape of the blood under high pressure is forced into these viscera. In well over one-third of the cases the blood escaped into the pericardium.

In others again, the aneurysmal dilatation presses upon the left bronchus, the trachea, or the œsophagus, and the erosion of the contiguous tissues allows the entry of blood into one of these channels. Or again, the rupture has occurred into the pleura, or the lung, or has escaped upon the external surface by erosion through the thoracic wall.

Although in the majority of cases the rupture comes on suddenly and leads to immediate death, there are others in which the erosion of the tissues allows, for a time, the leakage of blood into the surrounding parts. Slow hæmorrhage may exist for days and weeks, giving rise to blood-stained sputum or a leakage on to the skin surface. In others again, hæmorrhage may appear in small quantity, and then come to a conclusion through the presence of a thrombus which partly fills the cavity of the aneurysm.

Another form of aneurysm to which less attention has

been given, and to which a minor importance may be ascribed, is that which is commonly spoken of as the "fusiform aneurysm". This preternatural dilatation or tubular aneurysm occurs most commonly in the aorta, and is present in every case of syphilitic aortitis. It is sometimes associated with the presence of a saccular aneurysm arising from some portion of the diseased aortic wall, but not infrequently it occurs by itself, involving considerable stretches of the aortic structure. These fusiform aneurysms nearly always have their origin in a syphilitic aortitis wherein the aortic wall is diffusely involved in this specific disease. Diffuse dilatation of its structure occurs, and as there is no particular area in which excessive weakness is present, the condition remains as a generalized diatation without the presence of a saccular mass. The thoracic aorta is nearly always the site of the lesion, and it is infrequently found in the abdominal aorta.

Attention to the fusiform aneurysm was long ago indicated by Crisp, Andral, Hodgson, and others, some of whom referred to it as a preternatural dilatation of the artery. On the other hand, many dissertations dealing with aneurysm have failed to give a place to this particular type. The manner of development of this diffuse dilatation is similar to that of the other types of syphilitic aneurysm save that the primary syphilitic lesion of the aortic wall has remained diffuse and has not led to an individual localized weakness of its structure. This type of aneurysm may go unrecognized for many years and even escape detection before autopsy. However, with the advent of radiographic studies of the thorax, the abnormal size of the aorta is often discovered when clinical evidences were lacking. Here again, however, with the nature of the primary lesion of the vessel and the secondary widening of the aortic structure, the heart suffers the greatest inconvenience, and death from cardiac incompetency is the most outstanding clinical evidence. As these diffuse dilatations most commonly involve the ascending aorta and its arch, it is common to have the process of dilatation beginning at and involving the aortic ring so that

incompetency of the aortic valves usually accompanies the process. Although the aortic valves exhibit clinical incompetency, it is not common to find that the leaflets themselves are involved in organic change. Of recent years much discussion has been introduced into the literature respecting the frequency with which the aortic valves are attacked by the syphilitic process in association with the syphilitic aortitis. In 1902 Fabris pointed out that syphilitic aortitis not uncommonly leads to a dilatation of the aorta or to a thickening and retraction of the aortic cusps as a result of the downward extension of the inflammatory process, and that, in either event, a condition of aortic insufficiency is produced. Others have confirmed the evidence of clinical insufficiency of the aortic valves (Saathoof, Moenckeberg, Longcope). Longcope found that thirty-four per cent. and Symmers that twenty-seven per cent. of "uncomplicated cases of chronic aortic valvulitis" were of syphilitic origin. This has not been our experience; in fact, the reverse has been found at autopsy, wherein a marked syphilitic aortitis with its diffuse dilatation or the presence of a saccular aneurysm has led to great distortions of the main artery from the heart, yet the aortic leaflets themselves were free from the specific disease. Moreover, in a number of instances, we have found that in the presence of the syphilitic aortitis, the aortic valves were subject to a secondary infection of an acute type, and through this new lesion the incompetency was further exaggerated. A functional incompetency of the aortic ring due to a dilatation of its structure is commonly associated with syphilitic aortitis.

Syphilitic aortitis, as we have indicated before, attacks the aorta very severely and progresses from its initial point of attack in the ascending aorta both towards and from the heart. The lesion may not advance into the sinuses of Valsalva behind the aortic leaflets, but at other times it does progress into these pouches, though rarely does it produce definite lesions of the leaflets themselves. The nodular masses of the intima resulting from syphilis border the mouths of the coronary arteries, though here again the process does

not enter the tissues of these vessels, but usually comes to a conclusion at the edge of the aortic tissues themselves.

The incompetency of the aortic valves is almost always the result of a dilatation of the ring in association with the dilatation which occupies the aorta immediately above it. If careful examination be made in those cases in which syphilitic aortitis is suspected or demonstrated by the X-ray study, it will be found that the arch of the aorta is palpable directly beneath the sternal notch and the pulsations of the aorta are easily recognized at this point.

From what we have said above, it is clear that syphilis plays an important rôle in the development of aneurysms of the aorta, and what we will have to say in the succeeding discussion will indicate that aneurysms from other causes play a much less important rôle in arterial diseases.

Aneurysms resulting from Acute Infections.—Acute infections occurring in the arterial wall leading to severe damage with aneurysmal pouching occur more frequently than was formerly suspected. In recent years the literature has contained many references to the presence of acute inflammatory processes and aneurysm. The infections vary in kind and differ in the position of the arterial wall which is affected. This group of aneurysms is commonly spoken of as the "mycotic" form and may be associated with acute heart disease, acute sepsis, acute tonsillitis, or with an acute inflammatory process localized in the neighbourhood of one of the vessels. They are more commonly seen in young individuals in whom the acute infectious diseases are more commonly encountered than in the elderly. In 1851, L. Koch described a case of ruptured aneurysm of the mesenteric artery associated with acute endocarditis, and two years later Tufnell found a similar association in a case of popliteal aneurysm.

Those diseases possessing an acute bacteræmia, during some phase of their course, almost always show the presence of an inflammatory process in the first portion of the aorta in locations similar to that which is selected by syphilis. This

is true particularly for acute rheumatic fever, in which one may constantly demonstrate the presence of an acute periarteritis and mesarteritis in the wall of the ascending aorta and to some degree in the transverse arch of the aorta. In these cases it is to be observed that the inflammation follows the course of the nutrient vessels and brings about a disturbance of the tissues in the immediate neighbourhood. In acute rheumatic fever the lesion induced by the virus of the disease is of the nature of a non-suppurative inflammation which forms small foci of injury along the course of the vasa vasorum. In the majority of instances these injuries become healed by fibrosis before the wall of the vessel is severely damaged, and it is not common to find aneurysmal pouching taking place. Nevertheless, these cases of acute rheumatic fever which have had their recurrences cause a considerable fibrosis in the wall of the aorta, and the vessel is found to be slightly dilated. At other times, however, these areas of injury may be the site of localization for the non-hæmolytic type of streptococcus, when it is found that the injury becomes rapidly progressive and the aorta suffers a local dilatation of considerable extent. Saccular aneurysm has been reported by a number of authors, and we have seen an example in a child of five years. The French authors have also described an acute aneurysm following in the wake of acute influenza. It is their belief that the micro-organism causing this disease is also the cause of the aneurysm, although in no case has the nature of the infecting organism been clearly demonstrated.

On the other hand, associated with the intense infections due to the streptococcus and staphylococcus, one observes similar localizations of the infection upon the aortic wall giving rise to small suppurative processes which rapidly destroy the tissues in small areas. These lesions are quite remarkable inasmuch as the extent of damage which may be demonstrated in the specimen is quite circumscribed and occupies an area less than 0.5 cm. in diameter. The area, however, has suffered complete suppurative destruction of the supporting tissues of the adventitia and the media, and

the intima alone remains to support the pressure of the circulation. It is not uncommon in these cases to find that the intima eventually gives way, not through erosion by bacterial destruction, but by a tear caused by the blood pressure upon the weakened wall. These tears are unique, presenting themselves as sharp linear ruptures or stellate fractures of the intima passing through to the softened aortic wall. The name "aneurysm" is in these instances a misnomer, for true pouching of the wall is not demonstrated. However, the lesion is usually spoken of as the "acute mycotic aneurysm with rupture". In these instances the infection invades the arterial wall from the outer surface, leading to a localization of the bacteria within the fine ramifications of the nutrient vessels and the subsequent development of a small abscess. In the unruptured state the inner surface of the aorta shows little change, although occasionally a thrombus may localize over the area of injury. McCrae reported such a case in an individual suffering from a severe streptococcus septicæmia. Other cases have been reported by Bostroem, Sella, Endenhinzen, Morgan, and others.

The mycotic aneurysms of the above variety are most commonly seen in the aorta and it is not common to find these types in the small vessels of the body. The mode of development of this type of aneurysm is obscure, and Eppinger, Bostroem, and Thoma, each of whom has discussed the problem, are not in complete agreement. Two possibilities have been considered: (1) that the infection has become implanted upon the surface of the intima leading to erosion or ulceration of the surface, spreading into the media and finally completely destroying the middle coat and the adventitia, and (2) that the infective agent has set up a primary mesarteritis by way of the vasa vasorum with consequent destruction of the middle and outer coat of the vessel and a final rupture of the inner membrane. Eppinger's own words are, "In a mycotic-embolic thrombosis [of the vasa vasorum] there occurs an acute peri- and mes-arteritis leading to complete solution of the continuity of the elastica

and damage to the intima, and finally to the bulging of the recently inflamed, but yet resistant adventitia to form an aneurysm."

Whether the aneurysms which develop in association with periarteritis nodosa have a similar mode of origin by the invasion of bacteria through the vasa vasorum is not clear. Periarteritis nodosa is undoubtedly an acute infectious process which involves the mesenteric, hepatic, renal, or coronary arteries of the heart. The lesion found is quite peculiar, inasmuch as necrosis of the arterial wall is a prominent feature of the disease. Another type of acute infectious aneurysm is found in association with acute valvular disease. This lesion (other than the embolic) occurs in relation to the presence of acute aortic endocarditis and is found to be present upon the first portion of the aorta. At times it is seen that the infectious process of the heart valves has advanced by direct continuity from the valve leaflets on to the aortic wall. A continuous deposit of exudate links the two inflammatory processes to each other. Bacteria can be demonstrated in all portions of the exudate, and in that part lying upon the aortic wall an erosion of the tissues extending from the intima outwards is found to take place. The erosion may become progressive and simulate the ulcerative process which is not uncommonly seen upon the aortic valves. This continuous destruction of the aortic wall again leads to a defect sufficient to allow the vessel wall to stretch and form a local pouch. However, the pouch is never free and empty, but it is always found to be filled with the soft granular exudate similar to that which lies on the surface of the valves and on the aorta itself. If the ulcerating process does not continue beyond the strength of the vessel, rupture may not occur. Where, however, the ulceration is more extensive, rupture takes place during the acute phase of the disease.

Sometimes it is found that in these cases of vegetative endocarditis a continuous deposit does not associate them with the aneurysmal mass lying 1 or 2 cm. above the aortic ring. In these instances it would appear that the bacteria

arising from the infected valves have become localized upon the surface of the aorta, and have given rise to a destructive and inflammatory process similar to that in the heart. In other words, it does not appear to be necessary that the infection be gained by a continuous growth of the bacteria and their products over the surface of the aorta, but that implantation of the infection may take place a short distance from the infected valves. The majority of these types of acute aneurysms is found in the first portion of the aorta, but occasionally they are met with in the abdominal part. At times it is difficult to determine the actual mode of development of these distant aneurysms, inasmuch as the extent of the destruction has obliterated the evidence indicating an approach from the outside by way of the vasa vasorum or from the inner surface by implantation upon the intima.

The destructive process in the aortic wall giving rise to aneurysm and associated with acute vegetative endocarditis is not necessarily a suppurative one,—in fact, it is usually not such, but like the original infective process on the heart valve, is the outcome of the localization of the streptococcus viridans. Differing from the luetic type of the disease, it is most difficult to trace even remnants of the aortic wall into the boundary of the aneurysm. The tissues about the sac are fused by the inflammatory products, and firmly adherent to the inner surface is a pulpy infected thrombus.

The acute aneurysms developing through the presence of infectious processes along the lateral border of the arteries we need hardly discuss; the mode of development is obvious. Occasionally the initial lesion lies within a lymph gland which has undergone suppuration or suffered a tuberculous infection, and through its proximity to the arterial wall the destructive process entered the arterial tissues. Those arising through tuberculosis are more commonly found within the thorax in relation to the lymph glands bordering the descending thoracic aorta. A few instances of a similar infection have also been noted in the abdominal aorta, either in the region of the outlet of the coeliac axis or below the

level of the renal arteries where the lumph glands have close contact with the lower portion of the abdominal aorta.

It is particularly through the work of Eppinger that our attention has been attracted to another type of acute aneurysm. He has pointed out that not a few emboli arising upon diseased aortic valves contain bacteria and are capable of giving rise to an acute arteritis at the point where the embolus locates. Embolic aneurysm is almost always to be looked for in the small vessels. Curiously enough, the mesenteric artery has shown the greater number of examples due to this cause. In 1873, Ponfick reported eleven cases of heart disease associated with aneurysms in small arteries. Lindbom reported one case in the splenic artery, and one in the common iliac. Unger collected ninety-one cases of mycotic aneurysm in the peripheral arteries and referred to the frequency of the embolic type localizing in the mesenteric vessels, but found that many other arteries may be involved, depending upon the distribution of the infected embolus. Richey and Maclachlan have added two more cases, one in the superior mesenteric, and the other in the posterior tibial artery. In both cases there was an associated vegetative endocarditis. These authors point out that mycotic-embolic aneurysms are the arterial manifestations of subacute bacterial endocarditis. Undoubtedly the occurrence of these acute embolic aneurysms would be much more frequent save that the acute heart disease giving rise to them leads to an early fatal result.

The type of aneurysm to which I here refer as the embolic form is one in which a large embolus escapes from a vegetation usually found in the heart and migrates with the blood stream until brought to a vessel too small to pass. Thus the embolus usually rides at the point of bifurcation of an artery, and through the presence of the infecting bacteria gives rise to an acute arteritis extending outwardly from the intima with which it is in contact. Thus, when the vessel wall is severely damaged, an aneurysmal pouch occurs at a point immediately opposite to the location of the embolus itself. New thrombus is precipitated upon the

injured vessel and tends to fill the aneurysmal pouch. Under these conditions the vessel may become completely occluded at the point of embolus and aneurysmal formation. A careful search, however, will reveal the presence of a central embolic mass surrounded by a more recent laminated thrombus attached to the inflamed arterial wall. Other authors also use this term, "embolic aneurysm", to denote the type which, through the dissemination of bacteria by way of the blood stream, gives rise to an inflammatory reaction in and around the vasa vasorum in the outer coats of the large artery. The bacterial mass in the latter instance formed the embolus which is microscopic in size and which occludes a small capillary in the arterial wall. The aneurysm resulting from this primary lesion is the outcome of an acute destructive process arising in the outer tissues of its structure. These are sometimes spoken of as "mycotic-embolic" aneurysms (Eppinger). It is true, both types here referred to are embolic in character, but the name "embolic" is more applicable to that form in which a true embolus is found in the affected artery. Libman claims to have observed an embolic aneurysm in which infection was not present, but in which the damage to the artery was effected by a calcareous plaque cutting the intima.

It is interesting to note that the aneurysms of childhood are usually of the acute variety. Syphilitic aortitis and its sequelæ are unusual in the early years of life, while on the other hand acute infectious processes, particularly arising from the heart, are more common before the age of fifteen. It is not common that a diagnosis of aneurysm is made during life in these young patients, but occasionally, as has been reported by Endenhinzen, the aneurysm may have perforated into the œsophagus or into the bronchi. In a few instances the acute aneurysms of the smaller vessels within the abdomen or in the limbs may rupture into the neighbouring tissues.

Of eleven cases of aneurysm arising in children under fifteen years of age which have come under our direct observation, three were associated with endocarditis (two being rheumatic); two had a tuberculous origin; three were false

aneurysms resulting from trauma; two were congenital circoid aneurysms; and one was a congenital aneurysm of the sinus of Valsalva. The last case was reported in the literature by Dr. Goehring. The three cases associated with endocarditis occurred at the ages of five, seven, and eleven years. In two of them a true vegetative endocarditis was present upon the aortic valves, but in the third a granular verrucose mitral endocarditis was alone found. The cases of tuberculous involvement of the arteries presented lesions in the one instance upon the internal carotid and in the other upon the abdominal aorta. In the former, the boy suffered from an extensive tuberculosis of the lymphatic glands of the neck which were matted about the artery, and one of which had led to a caseous destruction of the adventitia and the carotid wall, so that a cavity containing blood was formed within the capsule of the lymph gland. A miliary tuberculosis had developed and had led to death. In the other instance, the wall of the aorta was firmly adherent to tuberculous lymph nodes at the root of the mesentery. Erosion of the aortic wall had proceeded with some rapidity and an opening had been excavated, large enough to admit the index finger. Apparently at the beginning the blood was withheld by the capsule and neighbouring fibrous tissue. Suddenly, however, and without warning, the mass ruptured, giving rise to an enormous hæmatoma in the retro-peritoneal tissues and between the layers of the mesentery. In both these cases arising through a tuberculous infection the organisms were demonstrated in the wall of the false sac. The three cases of traumatic aneurysm occurring in children showed nothing unique, other than that in one instance it had resulted through an accidental gun-shot wound; in the second, the child had been pierced through the thigh by a hay-fork; and in the third, the child had received a blow behind the knee in the popliteal space. In each instance a hæmatoma was the first sequel to the wounding of the artery, following which, after a week or two, the local blood mass became firm and contracted, but a cavity within it remained in communication with the injured artery.

Aneurysms resulting from Trauma.—Trauma of the arteries resulting in aneurysm is much more commonly seen in the peripheral vessels than in the aorta. It is quite unusual to have direct trauma acting upon the aortic wall, and when it does occur it is in the region of the lower portion of the abdominal aorta, which lies directly in front of the vertebral column, and which is open to direct blows received upon the abdominal wall. Occasionally such blows crush the tissues of the aorta against the bone and allow the development of a false aneurysm. The upper portion of the abdominal aorta is protected by the soft viscera, so that it lies beyond the reach of common injuries. This is still more true of the thoracic aorta which can only be reached by perforating instruments or projectiles penetrating the thoracic wall. On the other hand, indirect force may sometimes play an important part in leading to the tearing of the aortic tissues and the development of either a false or a dissecting aneurysm. The peculiar lesion which sometimes is found in the aorta, commonly spoken of as a spontaneous rupture of the aorta, is one in which the aortic tissues for one reason or another had an inherent weakness which allowed a tearing of the inner coats of the vessel with the penetration of the blood into a sac bounded on its outer side by the adventitia and the outer one-third of the media. These tears sometimes arise without the presence of any trauma being inflicted or any blow being received. At other times a similar lesion arises following a fall and doubling up of the body, wherein a sudden jar of the blood within the aorta thrusts great force against the arterial wall just above the opening from the heart. The extent of the tear varies much in the different cases, and the very nature of the lesion is such that we need not further consider it in the discussion of aneurysm. As, however, a similar event when acting upon the tissues of the aorta suffering arteriosclerosis takes place, the lesion consists not only of a tear of the inner lining of the wall, but is accompanied by a progressive separation of the middle coat of the aorta. This type we will discuss under "dissecting aneurysm".

In civil life traumatic aneurysm is an unusual occurrence and follows unique injuries of the external parts. Whereas in the long past the presence of traumatic aneurysms was not uncommonly observed in the bend of the elbow where the barber-surgeon performed his operation of blood-letting, this has disappeared from observation, and we rarely encounter aneurysms in this location. However, we still observe the cases of aneurysms close to the wrist and along the femoral and popliteal arteries as the result of injuries of various kinds. The reason for the development of aneurysm following upon a blow upon an artery lies in the fact that the arterial tissues are severely crushed, allowing either a sacculation of the injured area or permitting the escape of blood through a rupture in the vessel and leading to a haematoma and false aneurysm. Hedinger, who observed an aneurysm of the basilar artery of the brain follow upon indirect trauma, believed that any injury to an artery permits of the ready localization of infection, particularly syphilis, to take place at that point. This infection may cause the further degeneration of the vascular structure with the outcome of aneurysm. Such instances must be extremely unusual.

More important in late years has been the occurrence of aneurysm resulting from wounds received in war. Saigo reported the very common appearance of traumatic aneurysms during the Russo-Japanese War. These aneurysms were mainly of the false variety, developing upon the arms or legs and appearing within two to five weeks after the injury was received. Similar reports have been made by various authors during the Balkan War, and in all we find as many as two hundred and nine cases recorded by different authors (Bier, Subotitch, Haire, Haberer, and others).

The type of traumatic aneurysm as found in modern warfare is quite distinct, and it would appear that its relative frequency to-day is higher than in the past century. Modern warfare, with the use of various projectiles hurled at high velocity, has given us a type of wound different from those resulting from the military equipment of but a few years

ago. Wounds from projectiles result either directly by the passage of the bullet through tissue, or through the tearing by spicules of bone torn from the body framework. The modern projectile has undergone considerable modification from the round bullet or slug which was used with the musket. Now the small arm projectile consists of a neatly pointed casing of hard metal which is considerably longer than its diameter. When travelling through the air the pointed end remains in front and drills, by a twirling movement, its way through the air or any soft obstruction which it may encounter. The hole produced in tissue is much smaller than that obtained by a round bullet of the same weight. The point of entry is often much smaller than the diameter of the bullet, while the orifice of exit exceeds it. It is claimed that the majority of bullets travel in a horizontal plane and do not somersault. However, when the projectile is flying with an irregular motion, the damage to the tissues becomes excessive. Naturally the horizontal course of a bullet may be altered when passing from a thinner to a denser medium. Bony structures may cause a bullet to veer off and while changing its course, the bullet tears laterally an opening much greater than in the tissues of entry.

Much interest has recently been lent to the character of wounds, and the injuries to arteries induced by bullets. Makins has collected considerable statistics showing the manner in which projectiles lacerate the arterial walls. An interesting feature in the outcome of these wounds is that the frequency of secondary hæmorrhage is reduced in proportion to the opportunity to apply aseptic surgery. The wounds of arteries in which death from primary hæmorrhage was prevented through adequate clotting of blood, frequently gave way to secondary hæmorrhage as a result of infection. The repeated probing for bullets undoubtedly had its unfortunate consequences in introducing infection and disturbed the clot about the injured vessel. It is remarkable that a number of cases have been observed wherein arteries have been perforated and spontaneous healings of the

openings have occurred (Young, Brentano, Makins). Depending upon the position and fixation of an artery, it may be perforated by the slender projectile with a very small rent in its walls. The slight hæmorrhage permits of external clotting of sufficient strength to prevent fatal hæmorrhage. If this clot is not disturbed by infection or movement of tissues, organization brings about a repair so that the injury may barely be recognized.

However, the direct perforation of an artery is not as common as lateral lacerations or complete severance of the arterial trunk. Makins found that about fifty-eight per cent. of the arterial injuries are lateral wounds, fifteen per cent. perforations, and twelve per cent. complete division. Under any circumstance, these lesions may all show spontaneous repair by the organization of the extravasated blood. Commonly the function of the artery at the point of injury is seriously impaired, the lumen being occluded by clot. Secondary canalization of this must occur in a given number of cases just as it is found in thromboses of other origin. In a certain number, and particularly where the injury does not bring about a complete severance of the arterial trunk, but where a localized hæmatoma surrounds the artery, a permanent communication may remain between the arterial lumen and a cavity within the blood clot. Thus a simple type of false aneurysm is developed. Organization of the capsule of the blood clot gives strength to the false aneurysm, while its inner surface becomes lined by an endothelial coating from the intima of the artery. This is the usual form of war-wound aneurysm, and it is but rarely that an artery is injured only in its outer coats, leaving an intact intima which bulges like a mucosal diverticulum of bowel. I have never heard of nor seen the occurrence of such an accidental aneurysm. The like can, however, be produced by experimental means. Thus it is found, that if an artery be laid bare and its external tissues be carefully dissected away, a lateral bulging, amounting to a small saccular aneurysm may be developed. I do not believe, however, that it would be possible to recognize an

accidental aneurysm of this kind from a false one in which repair by endothelial tissues had reconstructed the sac.

Statistics on the distribution of traumatic aneurysms in different arteries are available from the South African, Manchurian, and Balkan Wars. In all there was a total of three hundred and eighty-three aneurysms recorded. The femoral artery was most often affected; and next in frequency were the popliteal, brachial, axillary, subclavian, and posterior tibial, in the order named. In one hundred and sixty-two of these cases, fifty-nine per cent. were localized or diffuse and forty-one per cent. were of the nature of arteriovenous aneurysms or aneurysmal varices. It is evident that with the proximity of the large venous channels to the main arteries of the extremities an injury common to both is not infrequent. Such injuries sealed by a common clot will resemble those discussed by William Hunter, resulting from careless venu-puncture. The primary clot which seals the openings between the closely approximated vessels is at the outset impervious to circulation. It is only after the process of organization and the outgrowth from each vessel of the endothelial lining that a communication between the vessels is established. Whether the new communication is more dependent upon a true canalization of the organizing blood clot or results through the rapid outgrowth of the endothelium from each vessel circumscribing the clot between the vascular openings and subsequently permitting the clot to be dissolved or removed by the circulation, is not clear.

One other type of traumatic aneurysm must be referred to. This is one which results from the operation of periarterial sympathectomy. This operation, which is one of stripping the outer coats from the artery in the hope of destroying the communication of the sympathetic vaso constrictors, is not altogether free from danger. Brandenburg, in a collection of one hundred and three cases of eight surgeons, reported three cases of secondary hæmorrhage and one of false aneurysm following this operation upon the femoral artery. Milko reported perforation of the femoral artery, and Matons had a similar accident occur following

sympathectomy upon the external iliac artery. Others report similar results with the development of hæmatomata and false aneurysms. In carrying out such operations upon animals, one may readily observe the ill-effects which are induced when an artery is completely lifted from its bed and its external coat is torn from it. In carrying out the operation of sympathectomy, it is necessary to exercise the greatest care so that the stripping of the vessel does not remove tissues too deeply and weaken its wall. We have observed in our animal experiments that when an artery is cleanly separated from its surrounding tissue and lifted free from its investing membranes, the vessel suffers irreparable damage inasmuch as the vasa vasora are torn from their arterioles of supply and the musculature of the vessel wall suffers from lack of nutrition. Small areas of necrosis like infarcts are produced in the media, and these are the regions of weakness. They may lead to small sacculations, or rupture occurs in their track, with serious hæmorrhage.

We still meet with a few cases of traumatic femoral, brachial, and popliteal aneurysm, but these are not nearly as common as in the past. These aneurysms were quite frequent a century ago, and Crisp reported two hundred and four out of a total of five hundred and fifty-one aneurysms. The incidence was high in the cavalry, and also amongst post-riders who received injuries in the saddle or blows while about their occupation.

Aneurysms resulting from Arteriosclerotic Changes in Vessels.—Formerly a great deal of stress was laid upon the factor of arteriosclerosis in bringing about aneurysmal dilatations. Many cases were reported in which it was believed that no other change than simple arteriosclerosis had caused the tissue damage and weakening. Lobstein, Koester, and others were quite insistent that aneurysm and arteriosclerosis went hand-in-hand and that, without the latter, the former seldom took place. Even Benda, as late as 1904, supported this view. After the differentiation between syphilitic arterial disease and arteriosclerosis had been made,

it became clearly evident that much of the discussion respecting the part played by arteriosclerosis in aneurysm had to do with syphilitic processes. Syphilitic aortitis always shows a marked endarteritis which, in its manner of development and in the tissues composing the thickened intima, does not differ from the endarteritis of non-luetic origin. Hence, it is true, the earliest plaques of syphilitic arteritis may be distinguished with difficulty from the chronic nodular disease, unless careful microscopic analysis be made of the entire structure of the artery wherein in the syphilitic form of the disease a true inflammatory reaction will be found to pervade the outer coats of the artery and its media also. It is upon the surface of this mesarteritis that the secondary endarteritis in syphilis occurs. In the chronic nodular endarteritis these reactions in the outer coats of the artery are absent or are very slight, and can be found only with difficulty.

Hence, having separated the luetic type of arterial disease from those discussed under arteriosclerosis, we find that we have taken from the arterial group of diseases the most important lesion related to aneurysm. The remaining group, for arteriosclerosis is not a pathological entity, only occasionally offers those conditions which, in association with a normal or altered blood-pressure, advance to some type of aneurysm.

Three types of arteriosclerosis bear a relation to aneurysms of rare type or minor importance. The first type to which we will refer is the common form of medial degeneration and calcification which is observed in the peripheral vessels of the extremities. This type of arteriosclerosis (Moenckeberg type) is so commonly found in old age, diabetics, and in the vessels of the extremities which are particularly active. It is not uncommonly observed to be more marked upon the right side than upon the left in individuals who are right-handed and perform heavy manual labour. This type of sclerosis is not commonly found in internal vessels save in the arteries of the uterus. In observing the scleroses which occur in the vessels of the extremities and where a marked

beading can be recognized by the palpating finger, we find that the course of the vessel is occupied by numerous small dilated segments which give the pronounced character of the beading. These vessels suffering medial calcification have become rigid during the active dilatation of the artery and just prior to the time of calcification when the muscular tissue of the wall was suffering the primary degeneration, the lumen was dilated beyond the normal diameter. Thus the process of calcification occupying the areas of degeneration leads to a fixation of the vessel wall in an over-distended state. When removed at autopsy, these vessels have these multiple pouchings along the course of the artery, showing that the areas with the calcified rings are distended beyond the intervening portions which are more normal in character. Thus, multiple aneurysmal pouches arise during the course of development of these lesions. It is to be noted, however, these aneurysmal sacs are small and seldom attain a size beyond that of a wheat-grain. In a few instances, we have seen specimens where several of these pouches had coalesced with the development of a larger sac and sometimes even a fusiform dilatation. Ordinarily this lesion is overlooked at the autopsy table, and we do not attempt to record the incidence in which these minute saccules appear within the arteries of the extremities. It has been our experience, however, to find them more commonly in the course of the external iliac and the femoral artery than in the remaining portion of the arterial branches. However, they are also to be observed in the brachial and its subdivisions.

The mere presence of these small pouches along the course of the calcified vessels offers no serious alteration to the circulation. The condition of calcification inducing a rigidity of the vessel wall is of much greater moment than the minor dilatations which accompany the sclerosis. It is to be recognized that in this particular form of arteriosclerosis the lumen of the vessel is not narrowed, but in truth is enlarged, and, furthermore, the medial calcification bears no direct relation to the presence of an endarteritis. Many instances of Moenckeberg's arteriosclerosis will be seen in which the

intima has in no way been altered, while the media has suffered severe damage. However, on other occasions an endarteritis may precede, accompany, or follow the presence of the medial degeneration. Occasionally also the small pits and pouches occupying the sclerosed vessel will be the site of deposition of thrombi. At times these thrombotic masses remain within the pouches, and do not extend beyond the borders of the sac. At other times, however, the thrombus extends beyond the initial point of development, leading to quite a plaque of deposit and always adding a new danger for the development of embolism.

A second form of arteriosclerosis which may have a relation to the subsequent appearance of aneurysm is the nodular endarteritis accompanied by atrophy of the media. It has been repeatedly shown that nodular endarteritis of the small vessels, such as the coronaries of the heart, the renals, and the cerebral arteries, has an independent origin wherein the proliferative reaction seemingly begins through the presence of an irritant localizing upon the intimal tissues and having no relation to the presence of change in either the media or the adventitia. We have, of course, indicated above that when a mesarteritis, such as occurs in syphilis, rheumatism, sepsis, typhoid fever, and many other infectious processes develops, there is almost always an accompanying endarteritis. On the other hand, we have been able to show in some of our earlier work, and Andrewes of London has confirmed our results, that an endarteritis may be individual and progressive, resulting from the presence of chronic infection localizing directly in the surface tissues of the arteries. This type of lesion is commonly found in a great number of different arteries. A nodular proliferative process leads to a great thickening of the intima and often its most serious effect is the narrowing of the lumen of the artery in which it occurs. This is particularly true in coronary sclerosis and in the sclerosis of the renal vessels. In these arteries, as well as in others, the nodular endarteritis is found not only to lead to projection of a new tissue into the lumen of the artery, but also through the presence of the blood-

pressure contained within the particular artery, the nodule, being forced outwards, leads to a pressure atrophy of the neighbouring tissues of the media. At times we may observe a slight pouching of the artery opposite to the end-arterial thickening, but when this occurs in vessels as well supported as are the arteries of the heart, the kidney and other viscera, the weakness induced through the atrophy of the media is sufficiently supported by the restraining tissue surrounding the artery so that excessive dilatation is rarely observed. The matter, however, is quite different in relation to the cerebral arteries. This system of vessels from the vertebrals onward are poorly supported by surrounding stroma, for they are merely suspended by a loose net-work of connective tissue along their course at the base of the brain and through the cerebral sulci. When, under these circumstances, a nodular endarteritis has progressed to the extent of causing marked atrophy of the middle coat, no resistance is offered in restraining the blood-pressure by means of the neighbouring tissues to prevent a stretching of the arterial wall. Hence, in the vicinity of such advanced nodular endarteritis of the cerebral vessels, it is not uncommon to observe slight dilatations and even true aneurysmal pouches along the course of the diseased vessel. These pouches are not always present immediately opposite the nodular endarteritis, but, as has been demonstrated by Halsted in regard to experimental constriction of the aortic lumen, the pouch may appear upon the distal side of the artery. Our attention has been repeatedly called to the presence of miliary aneurysms on the cerebral arteries under these conditions. Occasionally such pouches are quite marked and are subject to hæmorrhage. In cases of cerebral apoplexy it is difficult to demonstrate the lesion in the artery from which the blood has escaped, but when search is made, a careful examination will be rewarded by the demonstration of one of these atrophic lesions associated with endarteritis. It is our belief that some of the cases which are referred to as being of syphilitic origin may be of this character. Hedinger has referred to cases of this kind wherein an individual

suffering from accident has led to the development of aneurysm and rupture in the basilar artery. It is obvious that a weakened artery having imposed upon it an increased blood-pressure may develop hæmorrhage, or when the individual is subjected to severe trauma, rupture of the aneurysm may take place.

It is true that an endarteritis of syphilitic origin occupying the vessels of the brain leads to similar progressive damage of the middle coat, and in them aneurysmal pouches, thrombosis, and hæmorrhage not infrequently arise. It is often difficult, however, in those cases in which syphilitic encephalitis is wanting, to come to a conclusion as to the actual mode of origin of the primary endarteritis. It has been our experience that unless the arterial wall shows inflammatory lesions outside of the intima, that is, a true mesarteritis, a diagnosis of a syphilitic process is established with difficulty. When we compare the endarteritis occurring in the smaller vessels with that which takes place in the aorta, we find a similar differentiation. The luetic aortitis with its marked mesarteritis is a lesion separate and apart from the chronic nodular endarteritis wherein an atrophy of the media may occur, but a destructive process of the middle coat is rarely present to a degree sufficient to allow marked dilatation to arise. The media of the aorta possesses a relatively greater strength than is present in any of its branches and the atrophy occurring opposite the nodular thickening of its inner coat produces some distortion of the vessel wall, but rarely of such degree that its strength is sufficiently impaired to allow local dilatation.

Finally we come to a third type of sclerosis wherein the vessel wall suffers weakening with the opportunity of aneurysmal dilatation. In this type spoken of as the "senile sclerosis of the aorta", an atrophy of the muscular element in the media progressively leads to a loss of these important tissues with the deposition of considerable quantities of fine granular calcareous salts and a change in the elastic qualities of the lamellae. The senile aorta is found to be thinner than normal; the media is compacted; the elastic

lamellae lie more closely approximated; and the intervening smooth muscle fibres have disappeared to a large extent. It is interesting to note that all portions of the media are not equally affected, but that the greatest change occurs in the middle layer of this coat, while both the inner one-third and the outer one-third are less intensely involved. In this process of degeneration the intima and adventitia may not have suffered any change. As the media constitutes the main support of the vessel's strength, this atrophic process weakens the structure and a diffuse enlargement of the lumen may take place. If one observes the aorta and its diameter in elderly individuals, one will note that both the thoracic and abdominal portions are wider than normal and the aortic wall is unusually thin. Thus varying grades of a fusiform enlargement are encountered.

On the other hand, also, another important change is present in these senile vessels (aorta) when the process of progressive atrophy has advanced so that the middle coat is thin and non-elastic. It is observed that the middle coat is readily split into two lamellae. The elastic elements which normally intertwine between the alternating layers of elastic and muscle fibres become extremely brittle and lose their strength. In the area of the middle portion of the media most severely affected in this process of senile atrophy, there is no longer the binding strength between the neighbouring layers, and if one grasps the inner and outer portion of the aortic wall between the fingers, one is able to split the aorta in its entire length into two portions, an inner one containing the intima and inner one-third of the media, and an outer one containing the adventitia and the outer one-third of the media. This was first described by Lænnec and termed by him "aneurysma dissecans". The tear through the aortic tissues has passed through the mid-portion of the media. It is under these conditions that the dissecting aneurysm takes place. The presence of a senile atrophy of the aorta offers a potential danger for the development of such a dissecting aneurysm, for the wall of the aorta may be readily split into the two portions as has

been described. To permit this splitting to occur it is necessary for an opening to be present between the lumen of the aorta and the mid-portion of the media. Such is not uncommonly contained in these elderly individuals in the presence of an atheromatous ulcer (erosion), which not uncommonly extends through the depth of the intima and through the inner border of the media. Under ordinary conditions without the presence of a sudden thrust of the blood-pressure upon this weakened portion, no effect is accomplished. When, however, an individual possesses the potential weakness of the media along with the atheromatous change of the intima or even at times a calcified plaque in the intima, and he has further added a sudden increase of blood-pressure by muscular exercise, excitement, or other cause, the blood may be forced to enter the opening through the intima and find its way to that portion of the media which is readily disrupted. Under these circumstances the blood separates the arterial wall into two layers, forming two complete tubes and frequently completely encircling the aorta and extending from the arch of the aorta to the bifurcation at the iliacs. The point of entry for the blood is more commonly seen in the arch and upper thoracic aorta than in its remaining extent. True dissecting aneurysm is almost always seen in individuals over fifty years of age and, up to the present, more than three hundred cases have been recorded. The process of dissection takes place suddenly and rapidly comes to its completion. It may not lead to death and not a few cases are on record wherein a dissecting aneurysm has persisted for several years of time, leading to a healing of the injured tissues sufficient to be compatible with an adequate circulation. Very few cases have been diagnosed during life. Adami reported two cases of dissecting aneurysm occurring in a man of sixty-three and in a woman of fifty-three. In the former the lesion arose one year before death and in the woman twelve weeks before the end. In each case the process of healing along the torn tissues of the aorta was well advanced.

The term "dissecting aneurysm" is often used improperly

in referring to various types of false aneurysm. This occurs when, through traumatic rupture of an artery or by the slow erosion of a primary saccular aneurysm, a leakage of blood takes place into the outer stroma of an artery and there, following the line of least resistance, forms an elongated hæmatoma. This may be observed along the descending thoracic aorta when the blood will lie between the aortic wall and the margin of the pleura, or along the abdominal aorta where the peritoneum and mesentery may form the outer boundary of the escaped blood. Furthermore, from the very character of the tissue changes occurring in syphilitic aortitis, I do not believe it is possible to have a dissecting aneurysm develop in such a diseased vessel. I am aware that such cases have been described in literature, but I must conclude that these were false aneurysms of the nature of lateral haematomata, and not true dissecting aneurysms in which the media is split into two layers.

It is to be remarked that a certain number of dissecting aneurysms have been described in individuals under forty years of age. In all, the literature contains about fifty cases of this kind. The majority of them are described under the heading of "spontaneous rupture of the aorta", wherein the individual has died immediately or a short time after the rupture has occurred. These spontaneous ruptures are quite interesting, inasmuch as in them there appears to be no primary lesion in the intima which will permit of the entry of the blood to the middle coat which possesses an inherent weakness. These cases show most commonly an annular tear of the intima and the inner portion of the media, a short distance above the aortic ring. In others the tear is branched, but in all of them the edges of the tear are clean-cut, as if produced by an instrument. Following upon the tear, the blood not uncommonly dissects a short distance of the aortic wall separating it into two lamellae similar to those which were described occurring in the senile cases. In these instances (and we have had an opportunity of examining carefully only one such case), there are occasionally found curious gaps and degenerations in the outer

portion of the media which have the appearance of nutritional necroses. The weakening of the aortic wall is localized, so that the process of dissection does not extend very distant from the point of rupture of the intima. In our own case it was found that the aorta, a few centimetres beyond the point of dissection, could not be split into the two layers.

These cases of dissecting aneurysm have been reported as occurring in youth up to forty years of age, and are distinct from the senile group. They usually die through either an immediate rupture of the outer layer of the dissecting aneurysm or through a perforation of that layer, within a few days. Inasmuch as this lesion arises most commonly in the ascending limb of the aorta, the fatal hæmorrhage takes place into the pericardial sac. The abdominal aorta does not suffer this peculiar lesion of spontaneous rupture with dissection in young persons.

Aneurysms occurring in Animals.—It has been observed that aneurysms make their appearance upon the arteries of animals, but often from causes quite distinct from those in the human. We have previously mentioned that Barkow noted the presence of aneurysms in horses. In these instances, however, he made no comment as to the nature of the injury nor the character of the lesion which brought about the dilatation. Barkow (1871) found, however, that all aneurysms in animals, being like those in man, are the result of a primary injury in the arterial wall and acted upon by the contained blood under pressure. Baer states that aneurysm in horses bears a direct relation to their work. Aneurysms in dogs have been reported by a number of authors and particularly occurring in the tropics (Trevor, Pecus, Pease, Bel, and Haythorn). Some of these have been due to the presence of a parasitic worm invading the arterial wall, most often the aorta, and leading to a localized destruction and inflammation of the tissues. Haythorn and Ryan described a series of four cases in which the spirocerca sanguinolenta was found in the adventitia and the outer portion of the media, leading to localized aneurysmal pouches

and sometimes hæmorrhage. These cases occurred in the southern portion of the United States. They also reported three cases of aneurysm in dogs in which the worms were not discovered in the tissues of the aneurysmal sac, but in which the evidence seemed to point to a similar parasite as the causative factor. This canine parasite is fairly widely distributed in some tropical countries.

Aneurysms have also been described in rabbits by Fischer, who, in carrying out experiments for the production of arteriosclerosis in these animals, observed that aneurysmal pouches in the aorta are not uncommonly seen in the areas which undergo degeneration. Inasmuch as the arterial lesion produced by Fischer, by means of adrenalin, simulates quite closely the calcareous degeneration of arteries as seen in man in Moenckeberg's arteriosclerosis, the aneurysms observed in animals have a similar character to those which we have described arising in the human cases. Other than the parasitic worm infections in dogs, however, there appears to be no disease in lower animals which tends to localize upon the arterial tissues like syphilis in man. Aneurysm in animals is not common and the factors of causation vary very greatly in the different species.

In a series of experiments carried out by C. C. Guthrie and studied by Klotz, Permar, and Guthrie, aneurysms were found to develop as end results of arterial transplants. As early as 1905, Guthrie transplanted vascular segments from one artery into the divided ends of another vessel. Some of these animals lived for many years, the longest surviving a period of eleven years, when death occurred from other causes. A common observation upon these arterial transplants was the presence of a local diffuse dilatation of the transferred arterial segment. It was found that this dilatation was constant in all of the experiments, whether the operation was undertaken using auto-transplants, hetero-transplants, or devitalized segments of arteries. In both the auto- and hetero-transplants the arterial segment was removed from its normal bed and transferred to its new location. By this operation all communication between the

vasa vasora and their arterioles of supply is interrupted and the new granulation tissue which develops in the area to which the transplant has been transferred does not adequately bring nutrition to the transplant, and a certain amount of degeneration and necrosis is established. This injury to the arterial wall is sufficient to allow the blood-pressure to expand the segment into a fusiform aneurysm. The same has been found when transplants of devitalized tissue (segments which have been placed in formalin for days or weeks) are utilized in place of living portions. The devitalized segment was found to serve adequately as a conduit for the maintenance of the circulation, but it represented only a foreign body which within its lumen became lined by a living endothelial layer growing from the proximal and distal portions of the living artery, and also became infiltrated by the cells of an inflammatory reaction along with the tissues of repair from the outer zone. In the end, the devitalized segment was replaced, to a considerable extent, with new fibrous tissue which made up the bulk of its wall. This inert transplant with its subsequent fibrosis was unable to withhold the normal blood-pressure and became dilated into a fusiform aneurysm.

Halsted collected a series of 716 cases of cervical rib in man, and found that aneurysm or dilatation of the subclavian artery was present in twenty-seven. These aneurysms appeared on the distal side of the constriction produced by the supernumerary rib. Various explanations have been offered for the presence of these aneurysms, such as (1) weakening of the arterial wall due to erosion or trauma, (2) intermittent pulse pressure, (3) vasomotor or vasa vasorum disturbance. In carrying out a series of experiments upon dogs, Halsted observed that the constriction of an artery, by means of a ligature or metal band, led to a lowering of the maximum blood-pressure on the distal side of the constriction while the minimum pressure was increased. The segment of artery lying proximal to the point of constriction tends to become reduced in its diameter as far as the first arterial branch and the vessel wall shows a marked cell proliferation.

This author believed that there is an unusual whirlpool in the dead pocket beyond the constriction and that this, along with the change in the pulse pressure, may be the chief factors in bringing about dilatation on the distal side of the constriction.

Summary.—Every enlargement or dilatation of the arterial lumen, when contained within the structures of the arterial wall (or part of them), and which remains in communication with the circulating blood, is an aneurysm. These aneurysms are variable in size and in shape, but all of them are the outcome of a primary weakening of the arterial wall followed by a stretching of the structures by the force of the blood-pressure.

The weakness of the arterial wall which gives way to aneurysm always affects the middle coat, regardless of the changes to be observed in the intima or adventitia. An uninjured media will not give way in dilatation even when the blood-pressure is markedly raised. Changes in the quality of the intima or adventitia, when not affecting the media, have no bearing upon aneurysm formation.

The changes involving the media and leading to aneurysm are of the nature of degenerations and necroses, which may or may not be accompanied by inflammation. These degenerations bring about a solution of continuity of the muscular and elastic tissue elements, either diffusely or in focal areas, with an inadequate replacement by new tissue substance to maintain the strength of the arterial wall. When, in some cases, the repair by fibrous tissue is equal to the loss of the functional tissue of the artery, an aneurysm may be averted or long delayed.

The blood-pressure plays only a secondary rôle, having an influence only after tissue injury has been done in the media. Every aneurysm is a pulsion dilatation.

The factors of the present day to which aneurysms chiefly owe their origin are syphilis and acute infections, while mechanical trauma and arteriosclerosis play a rôle of diminishing importance. Syphilis stands out preëminently the most

common and destructive disease of the aorta, in which aneurysm is the sequel. Proof is at hand to show the high proportion of tertiary syphilis localizing upon the aorta. The treponema pallidum lodges in the arterial tissues and remains to continue its damage. The organism appears to resist the effect of chemo-therapy. The arterial lesions, particularly in the middle coat of the aorta, become progressive.

The ascending aorta is the commonest site for the early localization of the virus of syphilis, but from this point the lesion progresses both towards the heart and towards the thoracic aorta. Evidence of the syphilitic lesion in the aortic wall usually does not appear before five years after the primary, and the evidence of aneurysm occurs at a still later period.

Syphilitic aortitis is always accompanied by some dilatation of the arterial trunk, and as this takes place close to the heart, regurgitation murmurs at the aortic orifice are not uncommon. True aortic valvular disease, resulting from syphilis of the aorta, has in our experience been rare.

The lesion of syphilis in the aortic wall is unique and differs from other inflammatory processes; but its mild grades bear a resemblance to the lesions encountered in acute rheumatic fever.

Aneurysms resulting from syphilis occur most commonly between the ages of thirty and fifty, and are five times more common in men than in women. Syphilitic aneurysm is rarely seen under twenty years.

Aneurysms of the abdominal aorta are unusual. Those of the syphilitic variety are usually found in the vicinity of the coeliac axis artery.

Aneurysms resulting from acute infections are commonly associated with infective valvular heart disease, pyæmia, or bacteræmia. The lesion leading to the weakened arterial wall begins either in an infected adventitia or implantation infection of the intima. The process is always very acute and rupture is the usual ending.

The majority of traumatic aneurysms are of the false variety; they occur upon the vessels of the limbs and sur-

face areas, and save in warfare, are becoming less frequent than in the past.

Dissecting aneurysms might be spoken of as the "aneurysms of old age". The senile changes of arteries, particularly of the aorta, predispose to the development of the dissecting aneurysm. Syphilis plays no part in the appearance of this type of aneurysm.

In over fifty per cent. of the aneurysms of the aorta, death takes place through rupture of the aneurysmal sac. Over thirty per cent. of the remaining number die of cardiac incompetence, and the remaining few die of intercurrent diseases or complications induced through the mechanical displacement of organs by the presence of the aneurysm.

BIBLIOGRAPHY

Adami	Montreal Med. Jour., 1896, 24, 31.
Ætius	De vasorum dilatatione, 1542.
Andral	Diseases of the Chest, Philadelphia, 1843.
Andrewes	Report of Med. Officer, 1911-12, Appendix B, No. 1.
Backhaus	Ziegler's Biet., 1897, 22, 417.
Baer	Frank. Zeit. f. Path., 1912, 10, 147.
Baillie	Morbid Anatomy of the Human Body, 1820.
Barkow	Dilatation and Tortuosity of Arteries, Breslau, 1869 and 1871.
Benda	Berlin. Klin. Woch., 1906, 43, 989.
Benda	Ergeb. d. Allge. Path., 1902, 8, 1906.
Benda	Ver. Deut. Path. Gesell., 1904, 6, 164.
Bier	Deut. Med. Woch., 1915, 41, 121.
Bostroem	Deut. Arch. f. Klin. Med., 188, 42, 1.
Boyd	Amer. Jour. Med. Sci., 1924, 168, 654.
Brandenberg	Med. Klin., 1924, 20, 532.
Busse	Virchow's Archiv., 1906, 183, 440.
Chiari	Ver. Deut. Path. Gesell., 1904, 6, 135.

Citron	Cited by Heitz.
Corvisart	Diseases of Heart and Great Vessels, Philadelphia, 1812.
Crisp	Diseases of the Blood Vessels, London, 1857.
Cummer and Dexter	Jour. Amer. Med. Assoc., 1912, 59, 419.
Darling and Clark	Jour. Med. Res., 1915, 32, 1.
Dencke	Deut. Med. Wochen., 1913, 39, 541.
Doehle	Inaug. Diss., Kiel, 1885.
Endenhinzen	Frank. Zeit. f. Path., 1914, 16, 150.
Eppinger	Arch. f. Klin. Chir., 1887, 35, 1.
Faber	Virchow's Archiv., 1904, 177, 508.
Fabris	Virchow's Archiv., 1901, 165, 439.
Fernelius	De morbis universalibus, 1645.
Fischer	Deut. Med. Woch., 1905, 31, 1713.
Fracastorius	Opera Omnia. Venice, 1574.
Galen	Opera Omnia. Venet. ap. Juntas, 1609.
Goehring	Jour. Med. Res., 1920, 42, No. 1.
Guthrie	Blood Vessel Surgery, Inter. Med. Monographs, 1912.
Haberer	Wien. Klin. Woch., 1915, 28, No. 18.
Haire	Deut. Med. Woch., 1914, 40, 1928.
Hall	Lancet, 1913, 1, 803.
Halsted	Jour. Exp. Med., 1916, 24, 271.
Hare and Holder	Amer. Jour. Med. Sci., 1899, 118, 399.
Haythorn	Jour. Amer. Med. Assoc., 1913, 60, 1413.
Haythorn and Ryan	Jour. Med. Res., 1917, 35, 411.
Hedinger	Corres-Blatt fur Sch. Aerzte, 1917, 47, 1393.
Heitz	Archiv. des Mal. du Coeur, 1915, 9, 315.
Heller	Virchow's Archiv., 1903, 171, 177.
Helmstedter	Inaug. Diss. Strassburg, 1873.
Hodgson	Diseases of the Arteries, London, 1815.
Hunter, Wm.	Medical Observations and Enquiries, London, 1757 and 1761.
Klotz	Jour. Path. and Bact., 1913, 18, 259.
Klotz	Jour. Med. Res., 1915, 31, 409.

Klotz	Jour. Med. Res., 1917, 37, 1.
Klotz	Amer. Jour. Med. Sci., 1918, 155, 92.
Klotz	Brit. Med. Jour., 1906, 1, 1767.
Klotz	Arteriosclerosis. Diseases of the Media and Their Relation to Aneurysm. Monograph University of Pittsburgh, January 1911.
Klotz and Guthrie	Annaes. Paulistas de Medicina ē Cirurgia, 1923, 11, 16.
Klotz, Permar and Guthrie	Annals of Surgery, 1923, 78, 305.
Koch, L.	Inaug. Diss. Erlangen, 1851.
Koester	Ges. f. natur. u. Heilk., 1875, p. 15.
Lænnec	Diseases of the Chest, Philadelphia, 1835.
Lancisi	De aneurysmatibus. Romae., 1728.
Larkin and Levy	Jour. Exp. Med., 1916, 23, 25.
Lawson	Army Med. Reports, 1868, p. 267.
Lemann	Amer. Jour. Med. Sci., 1916, 152, 210.
Lewer	Army Med. Reports, 1862, p. 512.
Libman	Trans. New York Path. Soc., 1905, 88, 91.
Libman	Trans. New York Path. Soc., 1905, 88, 481.
Lindbom	Mitteil. Grenz. Med. u. Chir., 1914, 27, 912.
Lobstein	Traité d'Anatomie Pathologique, Paris, 1831.
Longcope	Jour. Amer. Med. Assoc., 1910, 54, 118.
Longcope	Arch. Int. Med., 1913, 11, 15.
Makins	Lancet, 1913, 185, 1743.
Matons	Sem. Med., 1922, 2, 98.
Maclachlan	Amer. Jour. Med. Sci., 1920, 159, 525.
Maclachlan	Amer. Jour. Med. Sci., 1925, 170, 856.
Malmsten	The Etiology of Aortic Aneurysm, Stockholm, 1888.
McCrae	Jour. Path. and Bact., 1905, 10, 373.
McNeil	Jour. Amer. Med. Assoc., 1916, 67, 1001.

Milko	Cent. f. Chir., 1924, 51, 513.
Moenckeberg	Med. Klin., 1905, 1, 1027.
Monro, A.	Medical Essays and Observations, Vol. 2, Edinburgh, 1771.
Monro, D.	Essays and Observations, Physical and Literary, Vol. 3, Edinburgh, 1771.
Morgagni	De sedibus e causis morborum, 1761.
Morgan	Arch. des Mal. du Coeur, 1915, 9, 312.
Muelhaus	Inaug. Diss. Kiel, 1899.
Myers	Diseases of the Heart among Soldiers, 1870.
Osler	Brit. Med. Jour., 1909, 2, 1509.
Osler	A System of Medicine, 1908, 4, 448.
Puppe	Deut. Med. Woch., 1894, 20, 854.
Paré	Les œuvres d'Ambroise Paré, 1641.
Pearce	Arch. Int. Med., 1910, 6, 478.
Pease	The Veterinarian, London, 1901, 74, 134.
Pecus	Jour. de Med. Vet., 1902, 301.
Ponfick	Virchow's Archiv., 1873, 58, 528.
Reuter	Munch. Med. Woch., 1906, 53, 480.
Richey	Arch. Int. Med., 1923, 31, 232.
Richey and Maclachlan	Arch. Int. Med., 1922, 29, 131.
Ruffer	Studies in the Palaeopathology of Egypt, 1921.
Saigo	Deut. Zeit. f. Chir., 1907, 85.
Scarpa	Sull' Aneurysma. Pavia, 1804.
Schaudinn and Hoffmann	Deut. Med. Woch., 1905, 31, 1665.
Schmorl	Munch. Med. Woch., 1907, 54, 188.
Schroetter	Cong. f. Inn. Med., 1899, p. 246.
Sella	Ziegler's Biet., 1910, 49, 501.
Sennertus	Opera Omnia, 1650.
Shennan and Pirie	Brit. Med. Jour., 1912, 2, 1287.
Subotitch	Deut. Zeit. f. Chir., 1914, 40, 127.
Symmers and Wallace	Jour. Amer. Med. Assoc., 1916, 66, 397.

Thayer	Johns Hopkins Bulletin, 1904, 15, 323.
Thoma	Virchow's Archiv., 1888, 111, 76.
Tozer	Arch. des Mal. du Coeur, 1915, 9, 312.
Trevor	Proceed. R. Soc. Med., 1912, 5, 20.
Tufnell	Dublin Quart. J.M.S., 1853, 15, 371.
Vallin	Aneurysmes de l'aorte. Gaz. des Hopit, 1879.
Verdie	Aneurysmes de l'aorte. These. de Paris, 1884.
Virchow	Virchow's Archiv., 1851, 3, 427.
Virchow	Virchow's Archiv., 1858, 15, 217.
Warthin	Amer. Jour. Syph., 1917, 1, 693.
Warthin	Miss. Valley Med. Jour., 1917.
Warthin	Harvey Lecture, N.Y., 1917.
Welch	Trans. Med. Chir. Soc., 1876, 59.
Wright and Richardson	Report Mass. Gen. Hosp., 1909.