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Cystic degeneration and medionecrosis of the aorta

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CYSTIC DEGENERATION AND MEDIONECROSIS OF THE AORTA

by

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"A LIVE PROBLEM IS USUALLY BETTER

THAN AN ANSWERED ONE".

PREFACE

At first glance one might think that the subject chosen for my senior thesis was rather a waste of time. It is not very much in prominence in books of pathology. Therein lies my reason for choosing this subject; that, and the fact that my advisor interested me in the possibilities of discussing this peculiar lesion from an etiological and pathological standpoint.

The etiology and the pathology of this abnormality of the human aorta is not clearly understood. However, it is not surprising when competent pathologists did not recognize the lesion until Erdheim in 1929 gave a very thorough description of the degeneration as he saw it in routine examination of serial sections of the aorta. The old saying, "-nothing new under the sun-", is true in medicine too. Medionecrosis of the aorta is not a pathological entity peculiar to this twentieth century of ours. Recognition of this lesion, it seems to me, is an indication of the progress that is being made in all fields of medicine today. Although greener pastures always seem to be far afield, and great men are those of whom we read in history, we must not forget that great men and history are being made right now. It would be a privilege to talk to Erdheim and hear from his own lips the story of the tedious struggle to find some clue to the sudden rending apart of the human aorta.

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For many years the diagnosis of "acute indigestion" was put on many a patient who, we now know, died of circulatory failure due to impairment of the heart muscle. Likewise, dissecting aneurysm or rupture of the aorta has been called various and sundry names clinically. Among the more common misnomers are:

1. coronary occlusion
2. rupture of the heart muscle
3. cerebral accident

It is not surprising however, for it is true, the patient presents much the same picture clinically. Roberts (12) suggests that one should be able to diagnose the particular heart condition ante-mortem for the following reasons. There is an intense systolic thrill and murmur over the aortic area, also an aortic diastolic murmur and other signs of aortic regurgitation which should indicate an abnormal transverse shelf of dissected media. He admits rupture of the aortic valve cusps will be hard to differentiate; aortic stenosis and insufficiency may be excluded on the basis of history. Pulse pressure and tracings and roentgenologic findings will also be helpful in diagnosing aortic stenosis and insufficiency. A consideration of the patient and the possibility of,or known history of, lues, hypertension, kidney disease, or extensive arteriosclerosis will be helpful in at least suspecting the

rarer lesion of medionecrosis as the cause of dissecting aneurysm of the aorta. The prognosis is very poor in the case of dissecting aneurysm as opposed to coronary occlusion, with which it is so often confused. It is very helpful to the doctor, to be able to tell his patients what they may expect, or in cases of this kind the relatives. And although the truth may be hard, still the doctor who is able to predict what the outcome will be with some degree of certainty, will at least have the confidence of his patients.

As mentioned before the case of a dissecting aneurysm and the case of coronary occlusion are much alike. The patient may or may not be straining at some form of exertion. In many instances the victim will be talking leisurely to some friend, or will be walking along the street. Suddenly, there is a severe tearing sensation in the chest and a pain which at times radiates to the back, and on occasion into the arms and legs. At other times the individual may be perfectly quiet and even in bed resting when they suddenly collapse and faint away. (12) Sometimes the regain consciousness and even improve, or they may remain in stupor and never regain consciousness. (8). This fact, and the fact that if they do regain consciousness, one hesitates to examine such a sick patient, is the reason why this lesion is

not better understood.

Erdheim described the pathology of medionecrosis in 1929. (Erdheim, 1930, (3)). Until that time many were inclined to believe that the normal aorta occasionally was ruptured spontaneously. Bostrom (8) and(1), examined the aortas of 178 individuals which were autopsied and came to the conclusion that since he found no pathology present, the normal aorta was capable of rupturing spontaneously. However, we may now question his results, since we do not know the type of individuals examined, and whether or not a thorough investigation was made, such as serial sections of the entire area in which the rupture occurred. (1) Levinson working in Erdheim's Pathological Institute in Vienna reported six cases with this type of degeneration in every case. He pointed out that the process may be limited in extent, and apparently seems to be very insignificant, and unless serial sections are made from one end of the tear to the other, one may easily miss the area where the medionecrosis occurs. Levinson made 50 to 65 serial sections in order to find the pathology, and he found in nearly all cases the tear of the rupture or the dissection extends further than the pathology itself. Many investigators since Levinson have found more numerous pathological changes in

non-ruptured aortae.

At this time it would be well for the reader to know what we mean by medionecrosis or cystic degeneration of the aorta. Moritz presents the best description in English, combined with the original description of Erdheim. (3), (8). This particular lesion was found on a male patient, 50 years of age who collapsed suddenly while resting. It was thought that he had had a cerebral accident.

Microscopic: Multiple sections were taken, both transverse and longitudinally at the site of the rupture. The intimal change was minimal. The internal elastic membrane or lamella was intact although irregularly thickened. The intima was smooth, no degenerative change other than the presence of homogeneous, faintlystaining, basophilic, intercellular substance. The subendothelial tissue was increased in amount. Some calcification was noted along the internal elastic lamella and some finely dispersed fat droplets in the homogeneous intercellular matrix. The media was not uniformly thin and had been split by dissection of the aneurysm.which had split four to five lamellae. Homogeneous basophilic substance was noted throughout the media. It was more abundant in the middle and inner third of the media. In certain areas mu cle and

connective tissue were entirely missing. When the chromotrophic substance had replaced the cellular elements, the elastic fibrils were destroyed or showed projecting spurs and rough ends. Where several of these lamellae had come together there was a small cyst formation. In the abdominal aorta the most marked changes were noted under the atheromatous plaques. Fibroblasts were attempting to fill in the defect. The new axis of repair might be transverse or oblique. This would, of course, impair the best function of the aorta since the flow of blood would necessarily be somewhat impeded. There was no adventitial thickening to compensate for the weakness of the media. The vasa vasorum showed some intimal proliferation. This was an interesting finding since (Robertson, 1929, (13)) Koster has drawn attention to the relationship between vasa vasorum and the localization of arterial disease. Klotz felt senile degeneration of the media was the result of cell starvation noticed usually at a distance from the vas vasorum and the intimal change.

Up to the present time there has been no definite work on the etiology of medionecrosis of the aorta. There have been many conjectures in passing as to the cause, but no one has set himself to the task of finding out what the basis is for this peculiar pathology.

Perhaps one reason for this is the fact that after rupture or dissection occurs, it is usually too late to do much in the way of extensive examination of the patient. He certainly is too sick to stand much in the way of laboratory tests and other functional examinations. The history that one might obtain from the family is not too reliable and is rather difficult to get at the time the patient is critically ill.

With this in mind the author has collected a few cases from the literature and puts them down and calls attention to the points of similarity and offers some criticism to cases in which this lesion might have been a factor had it not been overlooked. Included in these cases will be two which the writer was privileged to see at the Nebraska Methodist Hospital, Omaha, Nebraska; the final anatomic diagnosis made in these two cases were by Dr. Charles P. Baker, Pathologist, Nebraska Methodist Hospital.

Moritz sites the case of a 50 year old white male who collapsed suddenly and died. His blood pressure was not known. Another case came to autopsy with numerous defects in the outer coat of the media. The blood pressure had been recorded at one time as 210 systolic and 120 diastolic. This patient too, died suddenly while resting, and the clinical diagnosis of coronary occlusion was made. Moritz found that the chromotrophic material was

more abundant in the blood vessels of older individuals, and this material had a definite affinity for fat and calcium. To further substantiate these findings, Moritz examined the aortae of 70 adults dying from other causes than rupture or dissecting aneurysm. This same type of degeneration was found, and in some, cases, very extensively, especially under the atheromatous plaques of the intima. The chromotrophic, perielastic substance increases with age. This raises the question as to whether or not this might be a disease of old age or a step in the process of growing old. But why is it noted to such an extent in some younger individuals, and why does it account for a case of dissecting aneurysm in some cases and perhaps a very extensive pathological lesion be noted in another individual who dies from some cause far frmoved from the vascular. system.

Moritz calls attention to the fact that medionecrosis is usually the most severe at the site of the rupture, and the site of the rupture is most often the ascending portion of the aorta. He also suggests the possible etiological agents as being:

1. adrenalin poisoning.
2. avitaminosis.
3. involutional type of growth.

It would seem that the common occurrence of this lesion in young individuals would rule against it being any kind of an involutional growth. All of the cases which were studied had cardiac hypertrophy. Three had valvular disease of one kind or another. Five had chronic renal disease. Three had known clinical hypertension. It will be noted how often these same findings are present in the next few cases that are reviewed.

(Roberts. 1939, (12) Roberts reports on a young man who was brought to the hospital following an attack of collapse which closely resembled a coronary attack with decompensation. The patient remained in the hospital for a few days; he sat up very suddenly one day, as if frightened by something, and then fell over dead. This patient was known to have a luetic infection. He had been treated extensively for a long period of time. However, it was not felt that the drug therapy had anything to do with the medionecrosis which was noted at autopsy.

Histologic study showed focal accumulations of chromotrophic material between the elastic laminae of the media throughout the vessel. The material contained vesicles and some of the adjacent ones had coalesced so they formed large cysts. Intima and adventitia appeared normal, as did the vasa vasorum, and there were no perivas-

cular cellular infiltrations. At the point of the rupture through the intima into the media the chromotrophic accumulations were more numerous and showed more cyst formation. Some of the cysts could be seen with the naked eye, and measured from five to six mm. in length by one to two mm. in width. The elastic laminae were ruptured near the edges of the cysts. Short pieces of elastic tissue were seen free in some of the cysts. Some of the larger cysts lying just under the tear in the intima showed evidence of collagenous formation and also one could note the presence of a few irregularly fibroblasts which showed some evidence of healing by attempting to strengthen the elastic laminae. There was no evidence of elastic proliferation, and in the areas of collagenous proliferation there was often marked evidence of secondary necrosis. Again, cystic degeneration was most marked in middle third of muscular coat and this was the area in which splitting most often occurred. A nother interesting observation was the fact that the cystic degeneration was seen to end abruptly at the openings of the coronary arteries. There were no changes of atherosclerosis or syphilis so one could assume that the degeneration was the result of some other etiological agent. Again hypertension was a known factor present, and (Robertson, 1929, (13)) it raises the question as to what part the hypertension may play in causing the

process of degeneration. In studies on the aorta of the dog, the vasa vasorum supplying the wall of the aorta arise from the lumen of the aorta itself. In hypertension the lumen may be occluded or at least reduced somewhat in diameter. This naturally would interfere with the blood supply to the arterial wall and thereby reduce the nutrition and the consequent healthy state of the vessel wall.

Roberts, 1939, had another series of cases, eleven in all, three of whom it was proved at autopsy had cystic degeneration of the aorta. All were young women between the ages of 23 to 35, and all were in the last months of pregnancy. The question may be raised here as to whether or not there is some hormonal inbalance present in these pregnant women. There was no mention of a toxemia of pregnancy with high blood pressure and kidney damage that accompanies it so often. There is the possibility that the pregnant woman produces a toxim in her body that could cause the degeneration of the arterial wall, the same as the alleged toxin that produces the "morning-sickness" of pregnancy.

(Perry, 1936, (10)) Although, as we have mentioned before, medionecrosis is a relatively rare lesion, it is highly possible, as suggested by Perry, that many so-called heart cases in younger individuals might turn out to be dissecting aneurysm if autopsy would have been permitted.

Perry further suggests this interesting comment. Atherosclerosis affects the intima and inner layers of the media. Syphilis attacks the adventitia and outer coat of the media. Perhaps medionecrosis is the disease of the middle third of the medial coat of the aorta. In atherosclerosis there is rupture through the diseased intima and dissection inside of a healthy outer coat. IN syphilis there is the herniation through a diseased outer coat and the formation of a saccular aneurysm. With medionecrosis there may be dissecting aneurysm or there may be rupture. It would be difficult to say which way the balance of the scale would fall. Perry did not refer to the lesions which he saw as medionecrosis, but he said there was degeneration and necrosis of the medial coat. There was no inflammatory process present, and he thought the presence of some fibrous tissue was evidence to the fact that the blood supply to that particular area was not adequate for the other types of tissue to exist.

Klotz and Simpson, 1932, (5). Call attention to the fact that the inner two-thirds of the media has no blood supply which is in direct communication with it. When there is a disturbance in the vasa vasorum, marked changes are noted in the aortic wall supplied by the vessels affected. These men also found that death from dissecting aneurysm in young people was hard to correl-

ate, and these facts were commonplade to all cases:

1. cardiac hypertrophy
2. evidence of high blood pressure
3. chronic nephritis

These investigators felt that there must be some other reason than high blood pressure to account for the muscle necrosis and degeneration, and also for the disappearance of elastic tissue. Furthermore, the lesion was noted in young people from the ages of eight to 30. In this same paper, two men, Babes and Mironescu reported that they found focal degeneration and necrosis in relation to the vasa vasorum in the media. They noted degeneration and the formation of cysts and some hemorrhage, the vasa showed evidence of endarteritis.and thrombus indicating a faulty blood supply to the medial coat.

Gsell studied a number of aorta and found the focal degeneration in the area of the rupture. Wiesel found peculiar focal necrosis in the vessel wall of a young patient with concomitant acute infection. These lesions in the medial coat had no relationship to the plaques found in the intima.

Thus, these earlier reports and some to come later seem to correspond to the lesion as described by Erdheim.

No inflammation and very little attempt at repair. We are not confusing mucoid degeneration and necrosis of the media, since the dead material is absorbed without the evidence of any tissue reaction whatsoever. Klotz and Simpson also call attention to the sudden collapse of a young girl 23 years old, and in the latter months of pregnancy. She gave no history nor signs of any heart trouble of any kind. Examination of the heart revelaed no inflammation of the muscle nor of the aorta. Another case which they present is interesting from the point of diagnosis. Some authors contend the peculiar heart sounds in the presence of a certain history, should at least make one be suspicious of the difficulty involved and the etiology and the pathology to be expected. A white male, 45 years of age, and without evidence of lues collapsed suddenly. On examination he was found to have a blowing systolic mitral murmur. This should have been a clue to a shelf of media which protruding into the lumen and causing the murmur. He also had a bloodpressure of 210 systolic and 140 diastolic, and evidence of some kidney damage. Another patient, 54 years old, died suddenly while taking a bath. At post the heart was found to be enlarged and the corrugated appearance to the aorta turned out to be the depressions over the areas of ne-

crosis in the medial coat of the aorta.

Klotz and Simpson presented another case of a pregnant woman in the last stages of pregnancy who had sudden pain in the interscapular region which would make one think of a gall-bladder attack. On physical examination there was a soft presystolic murmur and a harsh systolic murmur in the second interspace. This patient too, died suddenly while resting, several days after the initial episode of acute pain and collapse. One reason for mentioning the collapse of these individuals several days after the initial attack is this. When medionecrosis is present there is not apt to be any amount of recovery, while with atherosclerosis and the lesion noted in lues, there may be some recovery, and there are cases on record in which the patients were apparently completely well and got up and went on about their daily work; of course, being careful to not overdo to any extent. In the case of another pregnant mother, the aorta showed some patches of mucoid degeneration, but also, patches of muscle and elastic tissue necrosis. At the same time there was a report of the same kind of changes noted in the renal, splenic and coronary arteries.

In summarizing the cases of Klotz and Simpson they noted patchy and diffuse necrosis between the middle and outer third of the media. There was no evidence of

inflammation or fibrosis, and the overlying intimal change did not seem to have any correlation with the pathology in the media. Thus the possibility of infection or atherosclerosis playing a part in this degenerative process seems to be ruled out. The vasa seemed to show endarteritis at times, but these two men did not feel that the vasa had any connection with the degeneration in the media. The elastic fibers present looked like the elastic fibers found in the aortae of senile individuals who have died from couses other than those pertaining to the vascular system. The dissection always seems to follow the plane of most extensive necrosis, and this is usually the outer border of the middle third of the medial coat. Muscles fibers seem to be more extensively damaged than the elastic fibers.

Klotz and Simpson state that Wiesel feels some bacterial toxin may be responsible. Adrenalin and nicotine have been suggested, but there is no evidence to lend much support to these theories. Renal toxins have been thought of, and liver products have been mentioned. There is not enough evidence here to site these two waste baskets of the body definitely as the source of our problem. It may be that the vasopressor action of epinephrine which causes constriction of the blood vessels may be a part of the picture here. We have noted on nearly

all of the cases examined so far that there was often a history of high blood pressure. It has also been suggested that nicotine may be the etiological agent that is to blame. We do know (Goodman and Gillman. 1940, (19)) that nicotine has a tendency to cause the production of more epinephrine in the human body. It is true, that there is vasodilatation but if there is constant stimulation, then the state of paralysis which must occur in order to have vasodilatation never has a chance to occur. The patients studied thus far were not questioned as to their personal habits of smoking, but it may be assumed that a large percentage of them did smoke some, since the number of people who do not smoke, especially men, is in the minority. A dietary deficiency plus some toxin has been suggested, but all of the cases reviewed do not seem to present this complication at all. In most cases the individual was in apparently very good health.

(7) McGeachy and Paullin report on three cases of the correct diagnosis of dissecting aneurysm before death. In all cases they found a media with degenerative changes in the media as a result of the sclerosis of the vasa vasorum. They also had a young woman 32 years of age who gave birth to a stillborn child. While in the hospital she had an attack of heart trouble but improved enough to go home. This by the way is the only one that has been recorded as having improved enough to go home.

However, she was back in a few months with the same complaints, and died very suddenly one day while resting in bed.

These two men find the lesion more often in white males, and they find that syphilis is the cause for the dissection in very few. This leaves only atherosclerosis, and medionecrosis as the causes for weakening of of the aortic wall.

Glendy, Cattleman, and White, (3) found medionecrosis in two patients who also presented the clinical picture of hypertension. These men think that more and more we are coming to find medionecrosis aortae idiopathica cystica as a common lesion in dissecting aneurysm of aorta. They refer to the cysts as "faults", and state that the etiology is still unsettled. In reviewing some of the work by Moritz they state that Moritz found the condition in ten percent of 70 adult aortae examined. In this paper too, it was suggested that it might be some sort of involutional change, but brought up the question as to why it affected so many young individuals.

Cystic degeneration has been produced experimentally in rab its by the use of adrenalin. This fact and the fact that so many of these patients have hypertension may add some light to the subject. Perhaps the adrenal glands are stimulated somehow to over-activity which in

turn acts as a noxious agent to the aortic wall when present in such large concentrations. Also the vasoconstriction activity of the drug makes for a high blood pressure, and this in turn is hard on the weakened wall of the aorta. It sets up a vicious cycle as it were.

Ressnick and Keefer 1925, (11) had a case of dissecting aneurysm in a negro laborer. They could find no reason why the aorta should have ruptured. There was no evidence of syphilis nor of any marked atherosclerosis. This was at a time shortly before Erdheim did his monumental work with serial sections of the aorta. We raise the question here as to what else could have been the cause for rupture? As noted before, the lesion is an easy one to miss, and was easily overlooked in former times.

Schattenberg and Ziskind,1938, (16) are not able to add much to our knowledge of the problem. They quote the findings of Oppenheim, Peacock, Shennon, Gsell and Erdheim. All of these men feel that cystic degeneration occurs more often in the older individual. We cannot overlook the fact however, that many young people die each year with this same condition, and we call it "heart trouble". These two authors (16) site two cases, one, a 23 year old colored male, and a 56 year old woman. Both had degeneration in the ascending aorta, and the

microscopic picture showed the absence of muscle or elastic fibers, and cyst-like areas taking the place of the degenerated tissue.

Roesler, Gifford, and Betts, 1937, (14) had a patient in which they diagnosed dissecting aneurysm correctly before post-mortem. From the clinical sign and the physical examination, they felt sure it was a lesion based on a wide spread atherosclerosis. This is the first mention we have of some attempt to try to differentiate. the lesions by the examination and the history. Roberts feels that sometimes, at least by the process of elimination, one may get some idea of what pathology could ne expected.

Hamburger and Ferris, 1938, (4) present two cases of men around the half century mark who, on physical examination, presented rather unusual diastolic murmurs. At autopsy, there was no tear in the intima to account for the murmur, and the aortic valves and cusps seemed to appear compabible. They could find no evidence of any degree of atherosclerosis or of syphilis to account for the amount of tearing and dissection which was present. They did observe a slight amount of ecchymosis around the coronaries in both cases.

Norris, 1934, (9) found five dissecting aneurysms in 1461 autopsies. The rupture usually occurred two

centimeters above the aortic cusps. The cause for the dissection included the following:

1. atherosclerosis
2. high blood pressure
3. nephritis
4. coarctation
5. meso-aortitis
6. trauma

We question the fact that not one case seemed to include medionecrosis, and yet all of the patients were under 45 years of age, and there was one pregnant mother 32 years of age who did not present a history of any hypertension.

Arenberg, 1932-33, (1) finds the reason for some dissecting aneurysm to be a very interesting one. One case in particular, white male, 50 years of age. The man was known to be a heavy drinker. and died suddenly. At post the dissection was seen to extend between the adventitia and the media. The author ran 22 serial sections in order to discover if there might not be some reason for the weakening of the arterial wall. He did not find a logical explanation. Perhaps more sections would have proved fruitful. At this time Arenberg reviewed some of the literature and found that Letterer, Osler, and Oppenheim explained the dissection on a

mechanical basis. Lynn described a woman 29 years old, who had a dissection during labor. He reports the post was not very satisfactory due to the haste to return the body to the relatives, and the poor light given for post exam. Arenberg notes that Rokitansky in 1838 studied eight cases from an etiological and pathological standpoint; he ended by saying he felt something was missing from the picture. He suggested a developmental abnormality, which we are led to say may not be too far from wrong. As we will point out later on some work done on the blood supply to aortic wall. Woodward 1875 said that rupture could occur in the normal arteryor aorta. Ames and Townsend 1897, were the first ones to have their feet on the ground, and although they did not find a definite lesion, they felt that there must be some underlying pathology to account for the rupture or the dissection of the aorta. It was well into the 1900's that Erdheim and Cellina did their work on foci of suppuration in media, and medionecrosis and mucoid degeneration.

In 1926, Gsell made serial sections in eight cases of spontaneous rupture and found what he called medionecrosis angiomalacia. He felt that chronic suppurations or toxic processes in the body such as chronic nephritis might have something to do with the formation of this lesion in the aortic wall

Wood, Pendergrass, and Ostrum, 1932, (17) in their work feel that the intima must be weakened in order to have a dissection, but they also feel that the media is weakened too. They suggest chronic alcoholism, but admit it has no support. Syphilis is accused, but the healing and binding affect which we find with lesions of syphilis is against that dread disease. Congenital weakness is mentioned, and the old stand-bys atherosclerosis and hypertension. These authors site the work on dogs in which it was found that epinephrine used over a long enough period of time had a degenerative effect on the tissue of the aortic wall of the dog, especially the elastic tissue. Their case studies included for study included those of a man whose only positive history as far as we are concerned is a little record of hypertension, and a woman who admittedly drank a small amount on occasion. Both were close to the age of 40.

Here again, we see the old complaint hypertension creeping in, and the long-suspected irritant being accused openly, alcohol.

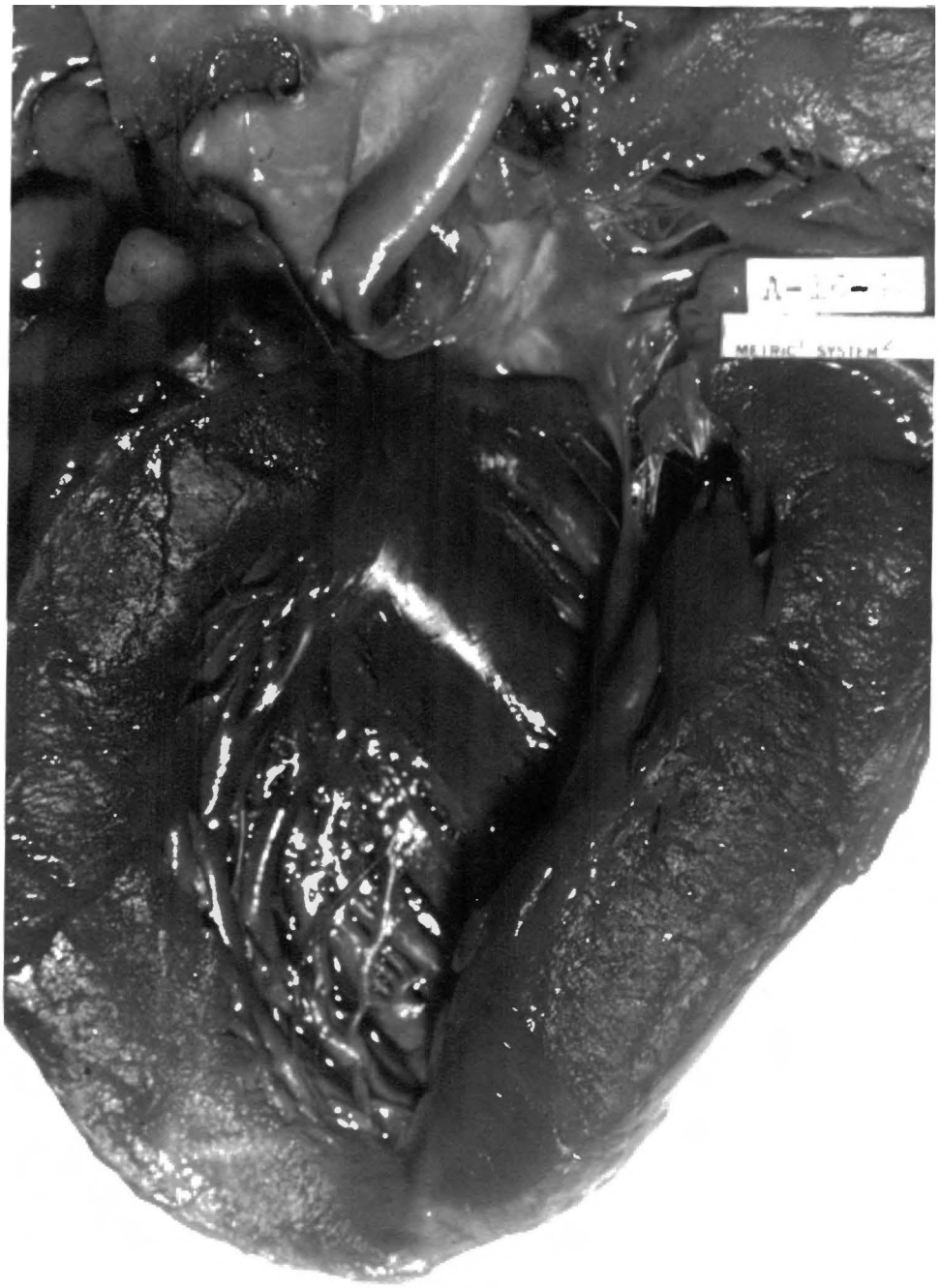
We were fortunate to see two cases of dissecting aneurysm in Nebraska Methodist Hospital, Omaha, Nebr. The one, a 29 year old white male, had had a heart lesion for some time, and on entrance to the hospital,

the physical examination revealed the same heart sounds and murmurs that, apparently, are typical for, or should suggest to one the possibility of a shelf of media extending out into the lumen. The other case was of a white female, 49 years of age who had a history of hypertension. She had had pneumonia twice before, just previous to her entrance to the hospital for her heart. The attending physician had taken care of her for sometime for her high blood pressure, and it is interesting to note here, this same doctor is taking care of members of the same family now for high blood pressure. We mention this only to bring out the possibility of an inherited weakness. Typical medionecrosis was noted on examination of the microscopic sections, and a picture of each heart and portion of the aortic wall is included for examination.

Lyman,1931, (6) did some work with diptheria toxin on the aortae of rabbits. He found degeneration in the medial coat, and it was most marked in the arch of the aorta and in the thoracic portion of the aorta. The result was thinning and formation of an aneurysmal sac. The lesion starts in the mid-third of the medial coat. It appears as cloudy-swe ling, with degeneration and necrosis of the muscle fibers. The elastic fibers undergo degeneration and lose their elasticity.

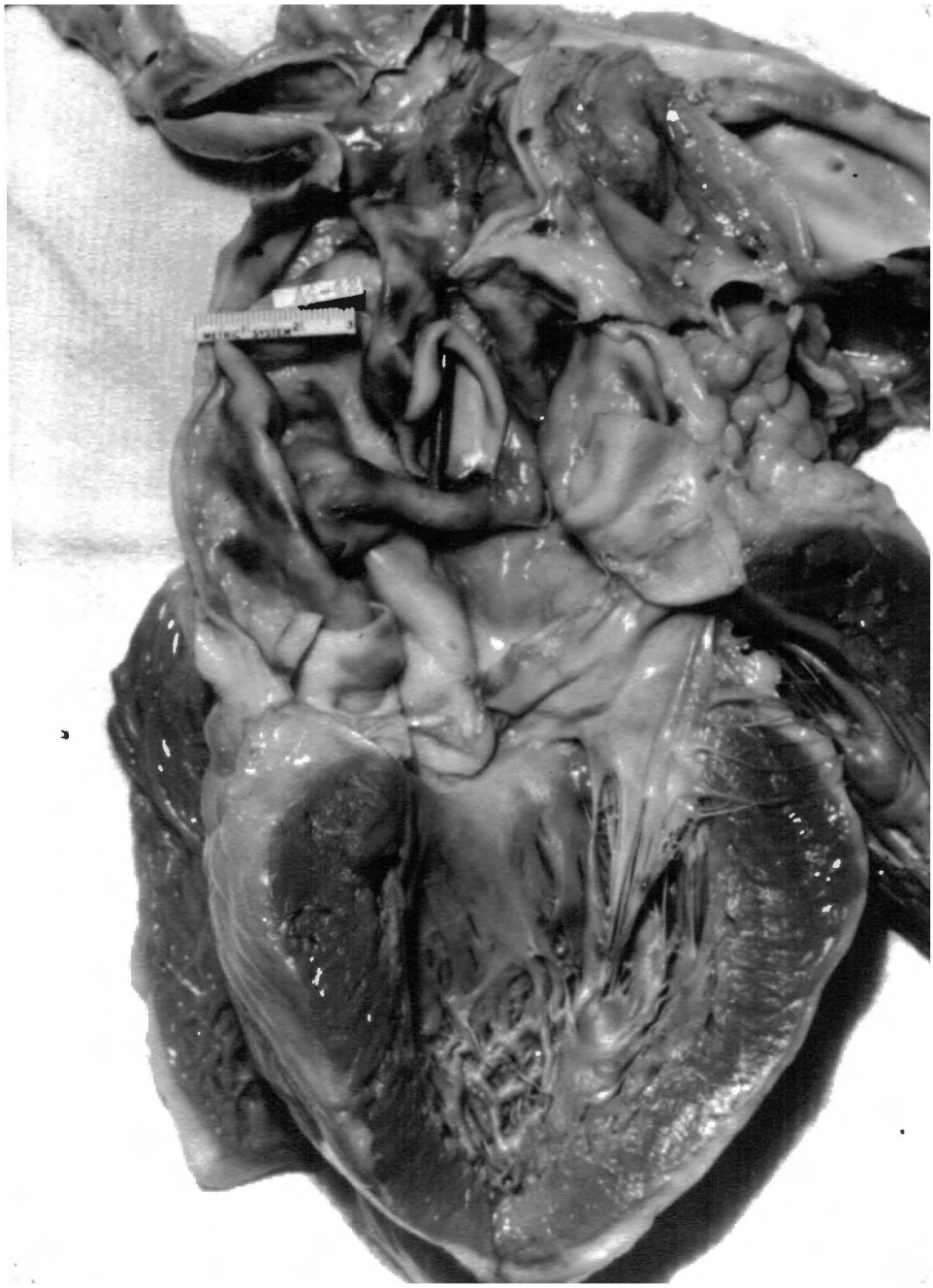
Patient 49 White female

Heart can be seen to be markedly enlarged and the area where the blood ripped through the intima and media may be seen in the upper left of the picture. The chordae tendinae and the papillary muscles are not remarkable.



Patient 29 years old, white male.

The heart is markedly enlarged. The probe seen in the upper center of the picture may be seen coming through what was left of the aortic lumen. Directly below the centimeter rule and a little to the right may be seen the dissected media which was torn away from the intima, and could be easily separated manually. Dissection occurred out into the innominate on the right, and into the subclavian on the left.



It was also noted that the change in young animals was different from the change observed in older animals.

Further evidence that the blood supply to the aorta may be involved comes from Woodruff, 1926, (18) who found that the vasa vasorum run through the media the intima, and do not end in the media. So, although it may appear that the media is well supplied with nutrient channels, in reality it is the intima which receives the main channels from these vessels. It was also noted that some of the blood supply to the aorta was through vasa which received their openings in the lumen of the aorta, and these, of course, are subject to much variation in constriction and dilatation from various chemicals in the blood. If the small opening to just one of these vasa became occluded, the area of media supplied by that particular vessel would suffer for proper aeration and removal of toxic products of metabolism. The result would be degeneration and necrosis.

Robertson, 1929, (13), called attention to a piece of work by Bichat in which he found the vasa ending in the outer third of the media.

Meckel found the vasa going to the intima entirely. Crisp in his experiment found the vasa going from the adventitia to pierce the media; Koster, Ebner, and Meigs,

and Klotz found vasa only in the outer one-third of the medial coat of the aorta. The outer third of the media seems to be supplied by branches of networks. These penetrate at right angles and then spread laterally and longitudinally between the elastic lamellae. The largest and the most closely grouped are near the parent vessel; those more distant are fewer in number, have less anastomoses, have longer branches, and have to supply a greater area of vessel wall. Two sections from the human aorta showed minute nutrient vessels penetrating the intima and branching in the inner third of the media. Other vasa arise from other vessels of the aorta while passing through the wall. They enter at the junction of the media and adventitia. Again the outer third of the media is not penetrated directly, and seems to be the one portion of the medial coat which is not well supplied with blood. Klotz concluded that aneurysm was most frequent where vascularization was most abundant; senile changes of the media were the most abundant where the vasa vasorum was the least abundant.

An interesting case was reported by Niehaus and Wright, 1941, (20). One of the authors attended a negro porter, age 49 years, who suddenly developed a pain in the chest and back. He was taken to a hospital where he remained for a few hours. He dies suddenly while taking a drink of water.

He was examined by the doctor prior to death and the physica examination was so typical of dissecting aneurysms; the diastolic murmur was heard over the aortic area. The company for which this individual worked had a physical record through the years which included the blood pressure, also a history of the serological reactions taken each year. It was found th t at one time this particular case had had a blood Wassermann of four plus. The authors were surprised to note that in spite of the past history of luetic infection, there was no evidence of this fact when the body was examined carefully at autopsy. There was no thickening, retraction or sclerosis, and aortic ring was not widened. The aortic cusps were soft, thin, and free from any fibrotic changes or retraction. Dr. H. E. Eggers, Professor of Pathology at the University of Nebraska Medical College reported the following microscopic findings: sections were taken through the involved portion and what appeared to be normal vessel wall. The area from the normal wall did not show any evident pathology; the microscopic sections through the dissection showed evidence of necrosis of the media which extended as a band for short distance but widened gradually toward the center. It is interesting to note

here that Dr. Eggers calls attention to the fact athat as far as can be determined, the vasa vasorum appeared normal, and this was true in the area of the necrosis.

It was felt by the authors that medionecrosis of the aorta was the lesion responsible for the dissection, even in view of the fact of the history of four plus Wassermann which the patient presented. There was no evidence of syphilis in the sections, and it was felt by these men that the lesion of syphilis would not tend to produce the picture of dissection, since the characteristic picture is one of a binding process and reparative growth and a holding together of the various layers of the wall. Eggers found hyalinized vacuoles in the media which showed no inflammatory or reparative process. Again we are confronted with the problem of what caused the degeneration? The patient was examined yearly, and an accurate account kept of his blood pressure from 1923 to 1925. He was not checked again until 1932. At this time he had gone from 135 systolic to 230 systolic, and from 78 diastolic to 140 diastolic. It would be interesting to know what happened to aortic media at this time, and if this degeneration was alreay there for the increased blood pressure to tear asunder, or if the degeneration came with the increased pressure. The relationship of the longitudinal grooves to the destruction of the media

may have some bearingon the etiology, since these grooves do tend to make the lumen of the aorta somewhat smaller. The grooving effect was mentioned by Klotz and Simpson, and by Niehaus and Wright. The disfigurement may be due to the shrinkage of the medial coat as the result of the cysts and vacuoles which may be seen throughout the medial wall. These "faults" are naturally not as firm and solid as the muscle and elastic tissue which it has replaced.

SUMMARY

In conclusion, it must be obvious that we must consider medionecrosis as a more common-place lesion than we have heretofore. That it has not been recognized more often is not surprising. We have seen the difficulties incumbent on microscopic diagnosis, even in the most efficiently equiped laboratories.

The evidence offered by various workers should be a start in the direction of enlightenment as to the cause for this lesion. Likewise, the very convincing evidence as to the ability of the normal aorta to withstand much greater pressures than could possibly be reached in vivo, should strenghthen our belief that the normal aorta does not rupture spontaneously without some pathology.

Further, it is not without the realm of possibility to think that sometime we will be able to recognize the lesion, and to some extent be able to ascertain ahead of the laboratory-post mortem examination, what the lesion is.

It appears to the writer that work to determine the exact etiology, must center around the hypertensive patient, and some toxin, which is so potent as to cause degeneration and necrosis and inhibit and curtail reparative growth of any sort.

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