DISSECTING ANEURYSMS*

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A DISSECTING aneurysm may be defined as the lesion produced by penetration of the circulating blood into the substance of the wall of a vessel, with subsequent extension of the effused blood for a varying distance between the layers. In most cases the aorta is involved, though dissecting aneurysms can form in the pulmonary artery and in all grades of arteries down to the small perforating arteries of the brain. The sac communicates with the original lumen through a rupture, or ruptures of the inner layers of the wall, and though in a few cases it remains as a hæmatoma it more usually ruptures either to the exterior or back into the lumen. In the latter event, the original symptoms may disappear and the patient recover, with an additional endothelial-lined channel through which the blood circulates. Exceptionally the sac becomes completely obliterated.

In the 16th and 17th centuries, a number of authors described cases of rupture of the aorta with local dissection of the coats, but they did not seem to appreciate the real nature of the occurrence; to them, it was the initial event in the formation of saccular aneurysm. As an example, Nicholls in 1761, published his "Observations concerning the body of his late majesty" George II, in which he says: "in the trunk of the aorta, we found a transverse fissure on its inner side, about an inch and a half long, through which some blood had recently passed, under its external coat, and formed an elevated ecchymosis. This appearance showed the true state of an incipient aneurysm of the aorta".

Maunoir in 1802 was the first to clearly suggest dissection of the arterial coats by blood. The first to actually use the term "dissecting aneurysm" was Laennec; he used it in 1826, but as if it were an already accepted term. Elliotson in 1830, gives a clear description of dissecting aneurysm. Pennock, 1839, was the first to demonstrate that the dissection takes place between the laminæ of the media.

Peacock, at the middle of the 19th century, collected a large series of cases and also did work on the cadaver to prove that dissection took place along the media. He blamed disease of the media, possibly rheumatism, as the cause. During the latter half of the century, there was a great deal of theorizing on the causation, particularly the mechanical factors involved, and also the histological changes in the media. Such men as Boström, Adami, Flockemann, von Recklinghausen, Thoma, Schede, etc., dealt with the subject. The basic principles of the subject were well established, or at least suggested, a century ago. Sherman makes a thorough analysis of the subject in his valuable monograph, and several other authors in recent years have contributed their findings in series of cases. Most of the newer work has brought out the importance of idiopathic medionecrosis of the aorta as a predisposing condition for rupture and dissection.

It has been realized of late that this condition may occasionally be recognized clinically, and though little can be done in the way of treatment, it is important to differentiate it from coronary occlusion for prognostic reasons. Glendy, Castelman and White[6] presented an interesting paper in which the clinical aspect is stressed, and features of the differential diagnosis pointed out.

The present report deals with a series of 7 cases examined at autopsy in the Pathological Department of the Montreal General Hospital from 1925 to 1941.

These occurred in a total of 5,380 autopsies for the period, which is a ratio of 1 in every 768 autopsies. This is fairly well in accord with the incidence found in other series.

CASE 1

(Autopsy No. 26-111). A man, aged 57, with a history of inadequately treated syphilis and chronic pulmonary tuberculosis, developed a lung abscess following pneumonia, in April, 1926. The patient was up and about the house on June 18th, feeling quite well. On June 19, he felt pain in the right upper abdomen, which radiated to the right flank. There were spasms of dyspnœa and coughing, and distress about the heart. Blood pressure 142/76. A pleuro-pericardial friction was audible. Death occurred on July 1.

Autopsy.—The pericardial sac was filled with massive blood clot. The heart was greatly enlarged, weighing 550 grm. There was thickening of the cusps of the mitral and aortic valves. Just above the aortic valve was a sclerosed mass, and above this a ruptured area which admitted the finger. Microscopically, the aorta showed considerable atheroma, and some small round-cell invasion of the intima and adventitia.

Comment.—This case illustrates the most common site of primary rupture in the ascending aorta, with external rupture into the pericardium. The extent of the dissection was not described. The possible rôle of syphilis and tuberculosis in damaging the aorta is to be considered; no degeneration of the media was noted.

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CASE 2

(Autopsy No. 37-89). An unidentified woman who died suddenly after being sick with "bronchitis". Autopsy disclosed a dissecting aneurysm of the first part of the aorta with rupture into the pericardium. The aorta showed marked atherosclerosis. Microscopic sections showed a split in the outer third of the media filled with blood clot, marked atheromatosis of the intima, and slight degeneration of the media, a loss of muscle and collagenous connective tissue.

Comment.—The site of the aneurysm is again the usual one; the split occurred characteristically in the media, and slight degeneration of this coat was found.

CASE 3

(Autopsy No. 37-193). A man, aged 59, was playing ball with his sons one evening, when he experienced a sudden severe pain in the right scapular region. This occurred about 7 o'clock. He was taken to the out-patients' department at once. The pain was now present in the right hypochondrium, as well as the scapular region. Blood pressure 160/85. Morphine failed to give relief. About 11 o'clock he cried out, saying that something had broken in his back, and complained of pain in the lower back. His colour was ashen, and he was covered with perspiration. The right lung field was dull to percussion. Death occurred at 11.46 p.m.

Autopsy.—A large extrapleural hæmorrhage of the right chest, displacing the lung forward and inward, was found. A transverse slit through the intima and media of the aorta was located 2 cm. distal to the opening of the left subclavian artery. The wall was split upwards for 1½ inches, and downwards for 2 inches. The heart was not enlarged, and the coronary arteries were not diseased. The aorta showed marked arteriosclerotic changes; a plaque lay at the anterior edge of the slit. The histological findings were dissection of the inner third of the media, slight round-cell perivascular infiltration of the media, and degenerative changes in the media, including fatty infiltration.

Comment.—The clinical and pathological findings are readily correlated. Primary rupture produced the first severe pain; limited dissection in the ensuing hours was associated with steady uncontrollable pain, and the final event of rupture into the extrapleural space produced the subjective sensation "of breaking of the back". It may be noted that the blood pressure remained high. Rupture in the region of the isthmus of the aorta is the second common site.

CASE 4

(Autopsy No. 40-98). A man, aged 62, who had been attending the medical out-door for six years, was known to have arteriosclerotic heart disease with shortness of breath and pain in the chest. There were a number of admissions to the ward for episodes of congestive failure. The highest blood pressure recorded was 155/100; the heart was found to be enlarged 11 cm. to the left of the midline. In December, 1939, the final admission to hospital, he complained of precordial pain on respiration and palpitation. There was also frequency, pyuria, and hæmaturia. In January, 1940, there were several episodes of hæmoptysis, and groups of petechiæ appeared in the skin. In May, pressure sores developed, and one became infected. Blood urea began to rise, and he died in coma.

The pathological diagnoses were bilateral suppurative pyelonephritis, gangrenous pyelitis and ureteritis; patchy gangrenous cystitis; chronic suppurative prostatitis; moderate arteriosclerosis of the aorta; coronary sclerosis; a large white thrombotic vegetation on the tricuspid valve; passive congestion of lungs and abdominal viscera; old pulmonary tuberculosis, and "healed" dissecting aneurysm of the aorta.

The aorta was double-barrelled in the descending portion, the false channel lying posterior to the aorta, and separated from it by a common wall. It did not surround the aorta. The primary rupture lay just distal to the left subclavian artery; a button-hole like slit with rolled edges, opening into the false channel. This channel extended down to the level of the renal arteries where there was a re-entrance rupture into the lumen. The lining of the aneurysm was smooth, and showed atheromatous like degeneration. One intercostal artery crossed the false channel, appearing as a cord.

Histological study showed marked medial degeneration; there is muscle atrophy with crowding of the elastic lamellæ, and in some areas sharp interruptions in the elastica. Sections of the aneurysmal wall showed an inner layer of fibrous tissue with elastic fibrils, a middle layer composed of 4 to 8 elastic lamellæ from the original media, and an outer thickened layer of dense layers of collagen.

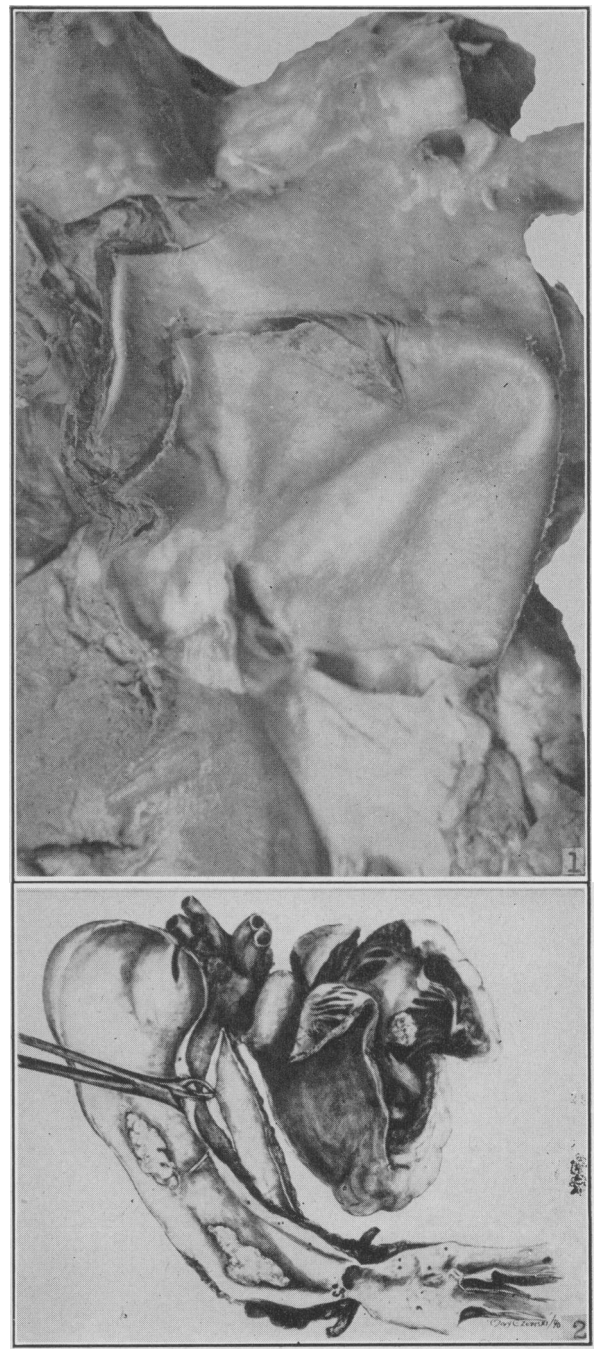

Fig. 1. (Case 7).—Primary rupture in ascending aorta. **Fig. 2.** (Case 4). — "Healed" dissecting aneurysm of the descending aorta and vegetative endocarditis of the tricuspid valve.

Comment.—The clinical history does not point to any definite episode when the actual rupture and dissection occurred. Secondary rupture back into the lumen prevented an immediately fatal result, and allowed the formation of an edothelium-lined channel through which circulation took place, though re-entrance to the lumen does not always obviate external rupture. "Healed" dissecting aneurysms are compatible with many years of life.

CASE 5

(Autopsy No. 40-256). This case concerns a sailor, aged 46, who developed symptoms of peptic ulcer in February, 1939. In April, 1939, following severe burns of the legs, he vomited blood. About the same time, he had precordial pain and palpitation with signs of congestive failure. He was able to return to work until February, 1940, when there was a severe hæmatemesis. He was admitted to the Montreal General Hospital again in September, 1940, with signs of auricular flutter and heart failure. The blood pressure was 184/128. The heart was enlarged to the left, 14.5 cm. from the midline. On November 25, he had a little precordial pain radiating to the left shoulder. The final episode occurred on December 14. He was awakened at 5 a.m. by pain in the throat, which passed off in a half hour, to be replaced by tingling pain beginning in the left hip and descending down the left leg. At the same time, there was milder pain in the right hip and leg. The patient was unable to move his left leg owing to the severity of the pain. During the rest of the day he felt very poorly, and his pulse was weak and irregular. At 2 p.m. he became very cyanosed and dyspnœic, and died in 25 minutes, about 9½ hours after the onset of the pain in the throat.

Autopsy.—The pericardium was distended by a massive hæmorrhage, 1,200 c.c. by volume. The heart was hypertrophied; the coronary arteries were patent. The aorta revealed a dissecting aneurysm extending from the base of the aorta to the bifurcation of the common iliac on each side; the false channel lay posteriorly to the aorta as far as the bifurcation, but dissection completely encircled the iliacs. It contained recent blood clot. The primary rupture was L-shaped, 2.5 cm. in length, and lay 2.5 cm. above the aortic valve in the right posterior position. The external rupture into the pericardium could not be found. The aorta showed moderate atherosclerosis and no evidence of syphilis. There was no peptic ulcer or other lesion to explain the hæmatemesis.

Histological study showed atherosclerosis, and medial degeneration of mild degree. The split was in the outer third of the media. No microscopic lesion of syphilis was found.

Comment.—The primary rupture took place in the common site, and likewise the rupture into the pericardium. This external tear into the pericardium may be difficult to find; it is frequently hidden by one of the auricular appendages. Very extensive dissection occurred within a few hours; the pain in the legs was undoubtedly associated with dissection of the common iliacs. The attacks of precordial pain which occurred previously likely arose in the heart, though there was no gross myocardial damage; the electrocardiogram showed evidence of it.

CASE 6

(Autopsy No. 40-92 Western Division). The patient was a man of 52, suffering from severe hypertensive cardiovascular disease with cardiac and renal failure. For six years, he had had headaches, breathlessness, and attacks of substernal discomfort following exertion. He had had bilateral splanchnic neurectomy performed in two stages, January and March, 1939, at the Montreal Neurological Institute, but this had afforded only temporary relief. Blood pressure before operation was 220/100. In November, 1940, he was admitted complaining of headache and failing vision. Blood pressure was 226/140. The apex beat of the heart was in the axilla. The cerebrospinal fluid was found to contain blood. The patient died very suddenly while lying in bed.

Autopsy.—The pericardium was filled with blood, 1,000 c.c. in volume. A 3 cm. linear tear was found in the outer coat of the intrapericardial aorta. The primary rupture was placed transversely in the ascending aorta, 6 cm. in length. The dissection extended distally over the arch and the upper third of the descending thoracic aorta, and also involved the roots of the arteries of the neck. The aorta showed marked arteriosclerosis with calcification and ulceration. The heart was extremely hypertrophied (780 grm.); there was no old or recent infarction, but the coronary arteries were hardened, calcified, and reduced in size. There was a large recent hæmorrhage in the right occipital lobe of the brain; it was in close apposition to the posterior horn of the ventricle, without actually rupturing into it. The fluid in the ventricles was bloody.

Histology.—The aorta showed marked intimal thickening. The media in the region of the dissection showed infiltration of round cells and polymorphonuclears.

Comment.—Cerebral hæmorrhage associated with dissecting aneurysm has been reported in a number of cases. One may only speculate on a causal connection.

CASE 7

(Autopsy No. 41-70 Western Division). This patient, a woman of 61, was running for a street car, at 2 p.m., when she felt an excruciating pain in the left chest that ran through to the interscapular region. She was taken to the hospital by taxi, and brought in in a state of shock. The blood pressure was 92/62. The pain persisted in spite of morphine. About 7.30 p.m. she suddenly became cyanosed, pulseless, and the respirations were gasping; death occurred in five minutes. The duration of the illness was 5½ hours.

Autopsy.—The pericardium contained about 200 c.c. of blood, mostly clotted. The external tear was not evident. The primary rupture was in the ascending aorta, 3 cm. above the aortic valve; it was placed transversely, and 5 cm. in length. Dissection was limited in extent, reaching only 7 cm. distal to the aortic ring. There was no atheroma at the site of the tear, though distally it was marked in degree. The heart was moderately hypertrophied, but showed no infarction. The coronary arteries were thin walled and patent.

Histology.—The media showed marked degenerative changes; muscle atrophy, crowding of elastic lamellæ, and interruptions in the elastica. Dissection occurred in the middle third of the media. There was associated arteriosclerotic changes.

Comment.—Here, the primary tear was associated with sudden physical exertion—a common finding. The primary and external rupture occurred in the common sites. The microscopic studies showed well marked idiopathic medionecrosis of the aorta.

CAUSATION

1. *Mechanical injuries.*—These are really rare factors. A few cases have been reported, *e.g.*, blow on chest, crushing injury, bomb explosion. External injuries more usually cause rupture of the aorta without dissection. On the other hand, severe or even moderate physical strains are frequently recorded, *e.g.*, severe muscular exertion while working, hurrying to an appointment, the passing of a stomach tube. Then, cases of mental strain, such as a quarrel or epileptic fit (here combined with physical strain) are known. But, again, cases with no increased strain are reported, occurring during sleep. Of our cases, one occurred in a man of 59 while playing baseball, and another, a 63-year old woman was

seized with pain while running for a street car.

It may be concluded, therefore, that sudden increase of blood pressure generally caused by some physical or mental strain is sufficient, even if of moderate degree, to determine the rupture of the media which leads to the formation of a dissecting aneurysm, but only when the vessel wall is diseased. This sudden rise has to be regarded only as the immediate exciting cause of the primary rupture. Both Sherman and MacWilliam believe, however, that the abrupt diastolic recoil is more important as a cause of primary rupture than is any increase of blood pressure caused by more deliberate systolic propulsion.

2. *The inflammatory theory.*—Syphilitic mesaortitis: this is unusual; as absence of frank syphilitic disease of the aorta is generally regarded as one of the outstanding differences between ordinary aneurysm and dissecting aneurysm.

In well developed syphilitic aortitis with fibrous replacement and interruptions crossing the laminæ of the media there is a tendency to localize the sac and prevent extension, but in some cases dissection is extensive. In this connection, syphilis may weaken the wall in another manner, by toxic necrosis of the muscularis in the absence of infiltration or adventitial changes.

Rheumatic aortitis: degenerative changes here may play a part. Rheumatic disease is only rarely found associated with dissecting aneurysm.

"Dissecting aortitis": this is a condition described by Babes and Mironescu in 1910, in which there are inflammatory degenerative changes in the media with new vessels that give rise to small hæmorrhages, that do not originate from the lumen of the vessel itself. Splits develop and ultimately the intima ruptures and dissection follows.

3. *Degenerative theories.*—Atheroma: It was commonly held among some earlier pathologists, including Virchow, that the cause of dissecting aneurysm was atheromatous ulceration with dissection of the blood through the floor or the edge of the ulcer into the layers between intima and media, or media and adventitia.

In examining a large number of cases, the prevalence of atheroma is striking, in 50% of a large series. But the intrapericardial aorta, where the primary rupture usually occurs, is not the commonest site. The patches usually appear distally, in the transverse part, and become more numerous in the descending aorta. In only 4 of Shennan's 218 recent cases did the dissection begin in an atheromatous ulcer.

Leary and Weiss (1940) reported a case of dissecting aneurysm in a rabbit which originated through an atheromatous ulcer, experimentally produced.

Medial degeneration: anatomically, the parts of the aorta farthest from the nutritive supply from lumen or vasa would be expected to suffer more from any harmful agencies, *i.e.*, the middle layer or inner two-thirds.

Of the three constituent elements of the media, the elastic laminæ are probably the most important. Clothing them on either surface is wavy fibrous connective tissue. The smooth muscle fibres pass obliquely between the laminæ, and are inserted at either end into the connective tissue. In systole, the aorta dilates under control of the tone of the smooth muscle, allowing the strain to come gently on the elastic laminæ, and preventing sudden jerking. The connective tissue acts as a check to prevent over-stretching. At this stage, the muscle passes into active contraction and initiates the contraction of the elastic membranes which is powerful.

Delicate connective and elastic tissue fibrils are other important structures, passing across the interlaminar space and encircling the muscle fibres in corkscrew fashion. These act to prevent free movement of the laminæ on each other.

Toxic or nutritional changes tend to involve the muscle and connective tissue primarily, as one would expect, and secondarily, the elastic tissue suffers and becomes increasingly liable to loss of retractile power, to friability, fracturing, or granular disintegration. Shennan emphasizes the importance of "faults", especially in the middle layers of the media, and of atrophic changes sometimes with complete disappearance of the muscle fibres in extensive areas associated with degenerative changes in connective tissues and elastica.

One should beware of appearances that are not truly abnormal, such as fenestrations, and also artefacts. Then again, there are the relatively physiological age-changes of fatty alterations of the connective tissue, muscle atrophy, and even slight changes in the elastica. Still another factor to be considered is the nutritional loss adjacent to recent dissection, seen as large areas devoid of nuclei.

In old healed aneurysms, the reparative processes obscure the degenerative changes of the

recent type, but degeneration occurs in the organized fibrous wall too.

In all 15 (except 1 syphilitic) of Shennan's own cases, marked medial degeneration was found in relation to the primary rupture, and in 6, numerous "faults".

Erdheim described the lesion, and Moritz[3] first discussed it in the American literature under the label: medionecrosis aortæ idiopathica cystica. They spoke of necrosis developing focally in areas the seat of chromatropic or mucinous degeneration, and tearing of the elastic elements with and without cystic degeneration.

Rottino[5] reported 12 cases of dissecting aneurysm studied carefully by the serial block method. In all 12 he found some degree of medial degeneration with muscle loss, crowding of elastic membranes, degeneration of collagen and elastic fibres, and formation of small fibrous scars. He has shown small areas of regeneration of muscle in foci devoid of elastic tissue. As to the distribution of the lesions, they were found in the ascending aorta and arch consistently, chiefly in the middle and inner thirds of the media, and only rarely in the descending aorta. The same author studied the aorta by the serial block method in 210 routine autopsies, and in 92 he found medial degeneration. The distribution of the lesion was similar to that in the aortas showing dissecting aneurysm. There were no clinically demonstrable symptoms associated with these lesions.

Vasa vasorum: according to Tyson, dissecting aneurysms may be the result of disease of the vasa vasorum. They may become obliterated by arteriosclerosis or inflammation, with resultant medial hæmatoma, and thus start dissection without a primary intimal tear.

Association with coarctation of the aorta: the congenital changes in the aorta concerned in rupture and dissection of its wall are chiefly those found at the isthmus. The isthmus may be completely occluded or show any degree of stenosis.

In 200 collected cases of coarctation of the aorta, Maude Abbott found dissecting aneurysm in 42. In 35 of these the rupture was in the ascending aorta, 5 near the coarctation, and 2 in the heart. The aorta above the stenosis is frequently atheromatous; usually patchy and of mild degree.

EXPERIMENTAL WORK

The earlier experimental work dealt mostly with attempts to split the coats of the aorta by injecting fluid under pressure. For example, Pennock (1839) succeeded in splitting the media by introducing a fine hollow needle between the laminæ and injecting water. This was accomplished in an apparently normal aorta as well as a diseased one.

Jores in 1902 and Josué, began the experimental work which has a direct bearing on the degenerative changes in the aorta wall, producing arteriosclerosis by injections of adrenalin. The modes of action were: (1) increased blood pressure; (2) chemical action as a muscle poison, and (3) constriction of vasa vasorum to produce anæmic necrosis. If amyl nitrite were given along with adrenalin, antagonizing its action on the blood pressure, one still got the poisonous effect on the muscle.

A number of authors have claimed that they could produce dissecting aneurysm with adrenalin in rabbits. Erb (1905) had one that dissected in the outer third of the media; there were necrotic foci in the media, and no atheroma. Bennecke believes that dissecting aneurysms can occur spontaneously in rabbits, however. Leary and Weiss,[4] more recently, (in 1940), state that spontaneous dissecting aneurysm is unknown in rabbits. They say that about 35 to 50% of normal rabbits show medial necrosis with a tendency to calcification, and that the lesion produced with adrenalin is more severe. These changes can also be produced by feeding vitamin D for prolonged periods, and in this case the vasopressor element is lacking. In the course of experimental work on the production of atherosclerosis by prolonged cholesterol feeding, they obtained a dissecting aneurysm which originated in an atheromatous ulcer. Adequate cholesterol feeding will produce arteriosclerosis in 100% of rabbits, and in normal rabbits examined, it was found to occur in less than 1%.

PATHOGENESIS

In most cases the primary rupture is brought about by a sudden increase in blood pressure, due to physical or mental stress. This acts upon the already diseased aorta to produce the primary rupture. Now the most advanced degenerative changes do not always occur at the site of the rupture, and in fact are fairly widespread, and so an additional factor, the mechanical, must be considered. The great majority of

primary ruptures occur in the first part of the aorta, as we have seen, and so the forces in play must be examined.

First, the mechanical influence of a high systolic blood pressure acts in elongating and distending the aorta in its ascending part. Moreover, the direction of the blood stream is more or less suddenly altered as it passes from ascending to transverse part, and again from transverse to descending. Hence one would expect the greatest strain would be along the outer curvature of the arch and at the junctional turning points, but the primary ruptures do not occur there usually. And, after all, the systolic propulsive force is exerted chiefly longitudinally, parallel to the axis of the lumen and will have a greater effect in elongating the vessel if there are irregularities, *e.g.*, atheromatous plaques, on the inner surface to increase frictional resistance. These irregularities are not common in the ascending aorta.

Shennan[1] therefore believes that it is the diastolic force which is important, the abrupt diastolic recoil meeting the resistance of the closed aortic valve. In diastole, on closure of the valve, the longitudinal force is largely converted into a transversely acting force with consequent lateral stretching and distension of the intra-pericardial aorta. Further, in the dilated portion which bulges above and below the ridge formed by the right pulmonary artery, there will be an extra drag on the wall along the line where it loses the support of that artery, and along this line, chiefly below the artery, there will again be a tendency to rupture. An additional factor is the rigid attachment of the pericardium at its reflexion.

At the moment of rupture the transversely acting force will come specially into operation and impel the blood outwards into the wall, and at the same time the longitudinal component will separate the edges of the tear. The edges of the tear will at first be pressed outwards, but if the transverse force is equalized by the persisting outer layer of the wall, the edges of the tear will return to their former position or even project inward. This provides obstruction to the blood, which will force its way obliquely into the rupture. The longitudinal force will now exert its effect causing the blood to pass parallel to the laminæ. During diastole the dissection will extend proximally, to its limit, which is the aortic ring, and during systole the dissection proceeds distally till the blood either penetrates to the exterior of the vessel or re-enters its lumen, or ceases to extend and forms a hæmatoma of the wall, with subsequent clotting *in situ*.

The other important location of primary tears is in the region of the ligamentum arteriosum. It is questionable if the ligamentum acting as a rigid band is the sole determining factor; in that case one would perhaps expect to find ruptures in the left pulmonary artery at the other end of the ligament. The more valid explanation seems to be that at the ligamentum one passes from the relatively free arch to the relatively fixed descending aorta, and that at every pulsation there is a hinge-like motion at the junction between the two. This implies an enhanced tendency to wear and tear, and to degeneration of the wall.

As to the factors involved in secondary rupture to the exterior or interior; this will depend largely on the severity of the initial transversely acting force and the resultant plane of dissection, on the presence of atheromatous plaques extending into the wall or other obstruction of an anatomical nature, such as an outgoing branch.

Re-entrance of the dissection into the lumen is by no means a safeguard against fatal rupture to the exterior. That organization and healing can take place is largely due to the circulation permitted by the distal re-entrance of the channel; in the outer part the tissues are well supplied with vessels from which organization can take place. There results a well-formed fibrous connective tissue covered by endothelium.

SUMMARY

Seven cases of dissecting aneurysm are reported. In the majority, 5 cases, primary rupture took place in the ascending aorta just above the aortic ring. The tear was not associated with an atheromatous patch in any one of them. In all 5 exit was due to external rupture into the pericardium with resulting tamponade.

The second common site of primary rupture is the isthmus of the aorta. Of the 2 cases presenting the tear in this location one terminated with an extrapleural hæmorrhage, and the other produced a re-entrance rupture in the abdominal aorta, and "healed".

Dissection was always in the layers of the media, and the extent varied from a few centimetres, to involvement of the entire length of

the aorta and the proximal part of its main branches.

Medial degeneration was found in 5 cases, but may well have been demonstrated in the other two by more extensive histological study.

Though arteriosclerotic changes were noted in the aorta in every case, there was no apparent relation to the primary rupture or the dissection.

REFERENCES

1. SHENNAN, T.: Dissecting Aneurysms, London, 1934, H.M. Stationery Office.
2. TYSON, M. D.: *Am. J. Path.*, 1931, 7: 581.
3. MORITZ, A. R.: *Am. J. Path.*, 1932, 8: 717.
4. LEARY, T. AND WEISS, S.: *Arch. Path.*, 1940, 29: 665.
5. ROTTINO, A.: *Arch. Path.*, 1939, 28: 1, 377.
6. GLENDY, R. E., CASTLEMAN, B. AND WHITE, P. D.: *Am. Heart J.*, 1937, 13: 129.

TUBERCULOUS ASCITES IN AN AGED NEGRESS*

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THE following case is reported to emphasize that tuberculous disease is not so extremely rare in the aged that it may be dismissed from consideration on the basis of age alone, as was done in this case, prior to exploratory cœliotomy.

CASE REPORT

An 82-year old negress was admitted on September 12, 1942, with three complaints: Abdominal distension for three weeks; "high blood pressure" for eighteen years; spells of giddiness and "shaking" for eight months. Ascites of unknown causation was found as the explanation of her abdominal distension. Ready confirmation of her claim of hypertension was obtained on examination (180/110). Her third complaint could have been explained on the basis of the hypertension, but was also considered in the differential diagnosis of the ascites.

As early as May, 1942, she had suffered vague abdominal pains, not related to meals. Her physician employed barium visualization of the gastro-intestinal tract in seeking an explanation, but no evidence of disease was found.

The patient was born in South Carolina and had lived only there and in North Carolina. She married at 15, bore ten children, had three miscarriages, and worked from childhood until the winter of 1941 as a field labourer. At 12 she had "pneumonia". Subsequently she had suffered no serious illness and had never consulted a physician until 1927, when intermittent headaches and giddiness of three years' duration caused her to seek medical counsel, whereupon the diagnosis of hypertension was made.

No one in her family was known to have had cancer, tuberculosis, lues, or heart disease. There was no history of alcoholism.

The patient had had no cough, sputum, hæmoptysis, or night sweats. There had been no chest pain, dyspnœa, cyanosis, or swelling of the ankles. Her appetite was good, but she had lost forty pounds in the last seven years. Fifteen years ago the patient had been advised not to eat meat because of her hypertension, and she had religiously followed this advice. There had been no jaundice, hæmatemesis, or melæna. The patient denied that she had ever had piles. Her bowel habits had been regular. No abnormal symptoms from other systems were found.

This 82-year old negress was stoop-shouldered and had moderate kyphosis of the dorsal spine. She could walk easily and balance well with her eyes closed and her heels together. The wizened face, thin and inelastic skin, shrunken breasts, dry and brittle nails, arcus senilis, and the ocular cataracts supported her claim of being an octogenarian. Her pupils reacted to light. There was no jaundice. No adenopathy was found in the cervical, axillary, epitrochlear, or inguinal regions. No pathological signs were elicited by examination of the chest. The heart was not enlarged to percussion and was otherwise also not remarkable. The abdomen was distended to the extent of a seven- to eight-month pregnancy but was soft (not doughy) and thin-walled, with physical signs of fluid (wave and shifting dullness). There were no dilated veins or caput medusæ. No tenderness and no abnormal masses were noted. The spleen and liver were not palpable and liver dullness was slightly reduced. There were no hæmorrhoids. The pelvic examination was non-contributory; normal senescent atrophy was noted. No scars were found on the lower legs and there was no œdema of the sacrum or extremities, although wrinkling of the skin just above the ankles suggested that there had been some ankle œdema.

On the basis of the above information the following tentative diagnoses were advanced: carcinomatous peritonitis; hypoproteinæmia; tuberculous peritonitis; cirrhosis or neoplasm of the liver.

Laboratory reports.—The urine was negative. There were 4,700 white blood cells, and a normal differential count. Erythrocytes numbered three million and the hgb. was 73% (8.2 grm.). The Kahn reaction for syphilis was negative. Serum proteins on September 19, 1942, were: total 6.58, albumin 1.79, and globulin 4.79%. An x-ray examination of the chest revealed a small, discrete, opaque nodule at the level of the third rib antero-laterally in the right lung field, near the periphery. The left lung fields were normal. The heart, aorta, and trachea were normal. "The patient has a healed primary tuberculous infection which at her age is of no clinical significance . . ." read the report. The tuberculin patch test was negative.

Course in hospital.—The patient was given bed-rest (with several hours up in a chair daily) and a high protein, high vitamin diet. Her weight (recorded daily) fluctuated between 105 and 110 lb. and was not significantly affected by the exhibition of theophylline. Between 4 and 8 p.m. every day, her temperature rose above 101° F., falling again below 100° F. by midnight. This rise was not accompanied by a commensurate increase in pulse rate.

The patient's advanced age seemed to exclude tuberculous peritonitis and favour the diagnosis of neoplastic disease. She had a fifteen-year history of low meat intake. This and the wrinkled skin about the ankles led to the presumption that the serum proteins might be low. The negative tuberculin patch test and the x-ray findings lent support to the prejudice against tuberculous peritonitis in this old woman. The inverted A/G ratio, the moderate anæmia and leukopenia, the daily spiking of her temperature record ("liver fever"), and even the "shaking" spells were considered explicable on the basis of hepatic damage from neoplastic disease. The diagnosis of cirrhosis was less favoured because of the absence of jaundice or evidence of collateral circulation, the negative history of alcoholism and lues, the

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