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CAUSE OF DEATH IN ANEURISMS OF THE THORACIC AORTA WHICH DO NOT RUPTURE; REPORT OF FIVE CASES.

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OWING to the frequency with which incomplete post-mortem examinations have been made in cases of thoracic aneurism, statistics are not entirely satisfactory. Haro and Holder¹ have collected from medical literature 953 cases of aneurism of the arch of the aorta. Among their conclusions these writers make the observation² "that in a large proportion of cases death did not ensue from rupture but from pressure by the growth." In another place they say:³ "Death occurs as a result of aneurism of the aorta either from rupture of the aneurismal sac, from pressure of the sac upon important nerves and bloodvessels, or from the secondary changes which take place in these tissues and in other vital organs as a direct or indirect result of such pressure."

From various data in their article I have made the following tabulation of the number of cases in which it was stated that death occurred without rupture.

Ascending arch, 570 . . .	Unruptured, 93 = 16 per cent.
Transverse " 101 . . .	" 20 = 19 "
Descending " 110 . . .	" 17 = 15 "
Unclassified, 169 . . .	" 24 = 14 "
Total, 953 . . .	" 154 = 16 "

I have also made a tabulation of the cases in which rupture is definitely stated to have occurred.

Ascending arch, 570 . . .	Ruptured, 339 = 60 per cent.
Transverse " 101 . . .	" 39 = 38 "
Descending " 110 . . .	" 72 = 66 "
Unclassified, 169 . . .	" 74 = 44 "
Total, 953 . . .	" 524 = 55 "

Browne,⁴ in a series of 173 cases, found that only 84, or 49 per cent., were due to rupture, a smaller percentage than that among the cases reported by Haro and Holder. Sibson⁵ made an analysis of 584 reported cases of aortic aneurism and of 296 museum specimens. Of the total number of 880 cases 703 were instances of aneurism of the thoracic aorta. Of these latter 358 ruptured, or 51 per cent.

Haro and Holder	. 953 cases.	Ruptured, 624 = 65 per cent.
Sibson	. . . 703 "	" 358 = 51 "
Browne	. . . 173 "	" 84 = 49 "
Total	. . 1829 "	" 966 = 53 "

The inference seems almost as reliable that nearly one-half of the cases of aneurism of the thoracic aorta die without rupture of the sac.

Turning, now, to the analysis of the 154 cases collected by Haro and Holder, of which it was stated that death was not due to rupture, I have made the following tabulation of the causes assigned for death by the various observers: *

Obstruction to air-passages	66
Dyspnea	26
Suffocation	10
Pressure on trachea	27
Pressure on bronchus	2
Edema of larynx	1
Exhaustion	50
Affections of lungs and pleura	28
Apoplexy of lung	3
Congestion of lungs	2
Pneumonia from pressure	2
Pneumonia	1
Pleuripneumonia	1
Abscess of lung	1
Bronchitis	2
Serous pleural effusion	14
Pleurisy	2
Pericardial affections	8
Serous pericardial effusion	7
Pericarditis	1
Pressure on the vena cava superior	1
Collapse	1
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It is clear at a glance that this information is vague and unsatisfactory. The largest single item is death from exhaustion, comprising practically one-third of the cases. In too many of these cases, I fear, the reporter thoughtlessly said "exhaustion" without any special effort to see if there were a better explanation. Suffocation, credited with ten deaths, leaves the mind guessing as to the exact way in which it was caused. Pressure on the trachea, credited with twenty-seven deaths, is an adequate cause of death if the pressure is extreme, but moderate

* It may be noted again that the faults in this table are due to the imperfect report of cases, and not to the excellent compilation made by Haro and Holder.

pressure does not necessarily cause death, though doubtless it may be one of the factors in producing that result. Dyspnea is fatal not in itself, but from the condition of which it is a symptom. It may signify trouble in the respiratory organs, in the circulation, or irritation of certain parts of the nervous system. The twenty-six deaths caused by dyspnea might certainly be more carefully analyzed. It is a noteworthy fact that any complication involving the heart is not once mentioned specifically as a cause of death in these cases, though recognized by all the leading authorities as holding an important place in this connection. Only one case is mentioned of pressure on the great vessels about the heart--another well-recognized factor in the fatal results. Undoubtedly more careful observation would have shown that many of the cases classed under the heads of exhaustion, dyspnea, suffocation, etc., were really deaths due to disturbance of the circulatory apparatus. It is not an exaggeration to say that many of these supposed explanations do not explain, and I think it is evident that there is need of a closer scrutiny of the fatal cases which do not rupture and a more thoughtful consideration of the real cause of death.

Let us consider briefly some of the problems to be thought out in this connection. We may divide the cases into three classes:

I. Cases in which an aneurism of the arch of the aorta exists, but in which it is not the important factor in the fatal result.

II. Cases in which death ensued from disease or disturbance of the circulatory system not caused by direct pressure of the aneurismal tumor.

III. Cases in which death was due, directly or indirectly, to pressure of the aneurismal tumor upon organs of vital importance.

Class I. comprises those cases which die from some intercurrent disease whose origin cannot properly be ascribed to the aneurism. Diseases of other parts than the thoracic organs or circulatory apparatus need no special discussion, and even some diseases of the thoracic organs may be so obviously due to other causes than the aneurism that its existence is plainly an unimportant incident. On the other hand, it will not be easy in other cases to decide how far the aneurism may have been a contributing factor in the causation of such disease. This is illustrated by the following case:

CASE I.--Male, aged fifty-one years, stationary engineer. His family history and previous history are unimportant, except that when twenty years old he was thrown from a locomotive and injured his back so that he lost the use of his legs for one month, and that he had two attacks of pneumonia nine and seven years ago. No history of syphilis could be obtained. Alcohol was not used to excess. On December 31, 1897, he was assaulted by two men, strangled, and beaten until insensible. It is probable that the strangulation, together with the violence of his struggles, may have been the immediate cause of the giving way of the

arterial wall. Certain it is that as a result he suffered from broken cardiac compensation for several months.

The more serious symptoms of his present trouble date from April 1, 1901. His first serious attack of dyspnea occurred on April 19th. This attack resembled angina pectoris in the nature of its distress and anxious suffering. It also suggested to the attending physician emphysema with an asthmatic attack. There was marked respiratory dyspnea, "rattling breathing," and stridor. There was very little cough. On the night of April 25th, after drinking a certain amount of liquor, while walking on the street, he "felt queer," rapidly choked up, lost consciousness, and did not awaken until some hours later at the hospital.

At this time the physical examination showed the pupils equal, with normal reaction. Pulse was irregular, slow, of good volume and tension. The walls of the radial arteries were sclerotic. The right border of cardiac dullness was $3\frac{1}{2}$ cm. to right, and the left border 10.4 cm. to left of the median line; upper border at the third rib. The apex was in the fifth interspace, 10 cm. to left of median line. The heart sounds were faint. No murmurs were detected. The lungs were negative. No signs of aneurism were detected. He improved and left the hospital April 27th at his own request.

On April 28th he had two attacks of dyspnea, similar to his first attack. On April 29th he had two attacks; on April 30th one attack. His next attack was on May 12th. Four attacks followed in the next two days, and the final attack began on the night of May 15th.

He was brought to the hospital in a semi-conscious condition. The peculiar crowing respiration and hoarse cough at once attracted attention, and suggested the possibility of aneurism. A tracheal tug was found. Dullness in the second interspace extended 5 cm. to the right of the median line, but there was no impulse, thrill, or bruit detected. The right radial pulse was entirely obliterated, the left was regular and of only fair strength and volume. The heart was the same as at the previous examination, but weaker. Percussion showed fair pulmonary resonance throughout the chest. Auscultation showed exaggerated, crowing respiration, with many coarse bubbling and fine moist rales. He did not improve, the pulmonary edema increased, and he died early the next morning.

The autopsy showed a sacculated aneurism of the transverse arch of the aorta, and a dilatation of the ascending arch; chronic endocarditis of the mitral valve; hypertrophy and dilatation of the left ventricle; general chronic passive congestion; arterio-sclerosis; lobar pneumonia; acute congestion, and edema of the lungs.

The heart muscle appeared of good quality and showed no fatty changes microscopically. There was nothing noteworthy beyond a moderate thickening of the mitral valve and chordae tendineae and moderate hypertrophy and dilatation of the left ventricle.

The aortic orifice measured 8 cm. in circumference; one centimetre higher the aorta measured 9 cm., and six centimetres beyond the valve it measured 11 cm. At nine centimetres from the valve a sacculated dilatation began, extending backward from the transverse arch. The dilated aorta merged into the aneurismal sac without a definitely marked border on the side toward the heart. The distal edge of the sac had a definite rounded border. At this latter point the aorta measured 6 cm. in circumference, and continued of this size to the diaphragm.

The opening from the aorta into the sac measured 4 to 5 cm. in diameter, and 14.5 cm. in circumference. The circumference about the sac and aorta was 15 cm. The intima of the aorta and the lining of the sac showed irregular thickened patches, but no calcification.

The posterior wall of the sac was in contact with the trachea just above its bifurcation. The wall of the sac was eroded at this point over an area 4 by 2 cm., and here the rough tracheal wall and the cartilaginous rings could easily be felt. The tracheal wall was somewhat thinned at this place. The trachea was somewhat flattened antero-posteriorly, but at the post-mortem examination there was no definite bulging of the aneurism into the lumen of the trachea.

The cavity of the aneurism contained fluid blood except for a small amount of fresh red clot. The left pneumogastric nerve ran down the side of the sac anteriorly, but seemed perfectly free. The right pneumogastric nerve was not in direct relation to the sac.

The vessels of the neck arose from the anterior wall of the sac. The left carotid and subclavian arteries appeared normal. There was no opening to the innominate artery from the sac. Where the opening should be was a smooth, rounded thickening with a very small depression. The smallest probe could not find a passage. On opening the innominate artery from its distal end it appeared normal until within one centimetre of the sac, when it suddenly narrowed and was completely occluded by a firm, reddish-yellow thickening beneath the intima on one side. The arteries and veins of the right arm appeared normal and were filled with blood.

An area of acute lobar pneumonia occupied about the upper fourth of the lower lobe of the right lung. It also extended to a small area at the root of the upper lobe, but did not involve the middle lobe. The remaining portions of both lungs showed intense acute congestion and oedema. Death was evidently due to this congestion and oedema of the lungs, which in turn was caused probably by the toxins of the pneumonic inflammation. The stage of development of the pneumonia indicated that it was about the fourth or fifth day, which suggests that the series of attacks beginning May 12th were induced by the pneumonic process.

What part did the aneurism play in causing the death of this patient? It certainly was not the immediate cause, yet it is hard to believe that it did not have an important contributing influence. In the first place the recurrent attacks of extreme and prolonged dyspnoea must have materially affected the general condition and strength of the patient. This reduced the resisting power of the system of an individual who was easily susceptible to pneumonic infection, as shown by the history of two previous attacks of pneumonia. It would seem probable, also, that the obstruction to the passage of air to the lungs might have induced changes in the lung tissue, rendering those tissues more susceptible to invasion by the pneumococcus. Finally, after the pneumonic process started, the frequent attacks of dyspnoea from the pressure of the aneurism on the trachea must have had a bad effect upon the patient's vitality in fighting the pneumonia. It is not clear how far the

last attack of dyspnoea was due to the aneurism, and how far to the general acute congestion and oedema of the lungs. When he was examined at the hospital pulmonary oedema was already well established, but he also had the crowing respiration and brassy cough of aneurism.

In Class II., which includes cases of death from circulatory disturbance not caused by direct pressure on the bloodvessels, the chief doubt will arise in determining how far in cases associated with arterio-sclerosis death may have been caused by that disease rather than by the aneurism itself. Arterio-sclerosis may cause death independent of the aneurism, as a result of changes in the nutrition of vital organs, by rupture of the diseased arteries, by the plugging of the arteries from thrombosis or embolism, or by its combined effect on the work and the nutrition of the heart. If death occurs from embolism, its source may be the aneurism itself, since coagulation of blood is favored in the aneurismal sac, and the danger of embolism is thus increased. Or, again, the aneurism may be an important factor in causing death by its effect upon the heart.

What effect may an aneurism of the aorta have upon the heart? It has been claimed by some that it causes hypertrophy of the heart. 'Oliver' has shown that this is not necessarily the case. His conclusion is based on statistics gathered by Dr. Oswald A. Brown from the cases occurring in St. Bartholomew's Hospital in thirty years. Of 124 cases, 68 showed no hypertrophy; in 17 the hypertrophy could be satisfactorily explained by other causes; and in the remaining 9 hypertrophy could be explained as probably due to other causes. In arterio-sclerosis we have a very common cause of heart hypertrophy, so that in many cases the aneurism and the heart hypertrophy are to be regarded as common results of the same cause and not as one dependent upon the other. One form of aneurism, however, certainly has a direct effect upon the heart. I refer to aneurisms situated close to the aortic valve and involving a general dilatation of the aorta. If such an aneurism enlarges enough it will by its stretching cause dilatation of the aortic orifice, from which follows aortic regurgitation with its results. Case II. belongs to this group.

CASE II.—Male, aged forty-four years, choreman, came to the outpatient department of the Boston City Hospital, February 18, 1899. Family history is unimportant. He had syphilis twenty-five years before. He had used alcoholic drinks moderately.

His chief complaint was of pain and a feeling of pressure in the precordial region, increased by exertion. He had suffered from this about one week. His trouble, however, began at least a year before, at which time he began to have a severe boring pain in the back of the chest, together with dyspnoea on exertion. At the time of the examination he was not hoarse, but he had had several attacks of hoarseness in the last few years.

Inspection showed the pulsation of the carotid arteries more marked and jerky than normal. On the right side of the neck, just above the

inner end of the clavicle and also in the episternal notch, a marked pulsation was seen. Palpation showed that the pulsation in the episternal notch had a distinct lateral thrust from the right, indicating its origin from the innominate artery and not from the arch of the aorta. A thrill was plainly felt just outside the sternomastoid muscle above the clavicle. The right radial pulse was weaker than the left, but there was no perceptible difference in time. A slight but distinct tracheal tug was made out. The pupils were equal. The apex-beat of the heart was felt in the sixth interspace in the left mammary line.

Percussion over the lungs was normal. Relative dullness of the heart extended to the left mammary line, four and seven-eighth inches to the left of the median line, and on the right its border was one inch to the right of the median line. The area of dullness over the great vessels was increased in breadth. On the left, at the base of the heart, dullness extended two inches from the median line, diminishing to one and five-eighth inches higher up. On the right the distance was one inch from the median line at the base of the heart, increasing to one and three-eighth inches just below the clavicle. The line of dullness bounding the great vessels on the right, together with the abnormal pulsation beneath the inner end of the right clavicle, made the diagnosis of aneurismal

FIG. 1.

Fluoroscope examination.
Fastform Aneurism of ascending, transverse,
and descending arch.

dilatation of the innominate artery practically certain. The breadth of the area of dullness over the vessels just above the base of the heart, with the tracheal tug and the associated aneurism of the innominate artery, made it highly probable that an aneurism existed in the ascending and transverse parts of the arch of the aorta. Physical examination gave no signs of the involvement of the descending arch.

Confirmation of the diagnosis was sought in an X-ray examination, which was very kindly made for me by Dr. F. H. Williams. The accompanying diagram shows the outline of the shadow cast by the heart and great vessels as shown by the fluoroscope.

The point of special interest in the X-ray examination lies in the shadow with a curved outline above the left base of the heart. This shadow extends upward three inches from the base of the heart,

and outward to the left one and one-quarter inches beyond the origin of the great vessels. Distinct expansile pulsation was seen in this shadow just following the systolic contraction of the ventricle. It evidently shows an aneurism of the aorta extending from the transverse arch to the descending arch. A distinct outline of the innominate aneurism could not be obtained, perhaps owing to its relation to the shadow of the spine.

By the fluoroscopic examination we made certain the existence of an aneurism of the arch of the aorta, whereas before we had great probability rather than certainty; we also learned that this aneurism extended to the descending arch, of which fact the ordinary physical examination did not give even a clue, and we were able to outline the size of the heart more accurately than by percussion.

Auscultation of the lungs was normal. At the aortic area a systolic and a diastolic murmur were heard. The systolic murmur had its maximum intensity at the aortic area. It was transmitted upward along the aorta and could be heard faintly in the left carotid and subclavian arteries. Just at the inner end of the right clavicle the systolic murmur was markedly reinforced, and had an intensity even greater than at the aortic area (a condition which was interestingly explained by the post-mortem findings), and the murmur was quite loud in the right carotid and subclavian arteries. The diastolic murmur could not be heard in the left carotid or subclavian arteries, but was faintly audible in the corresponding vessels of the right side. Its maximum intensity was at and just above the aortic area. It was not carried downward beyond the anatomical position of the aortic valve (the junction of the third left costal cartilage with the sternum), and at this point the second sound became distinct and somewhat accentuated. A slight systolic murmur was also heard at the apex, but was not transmitted far in any direction. It was not clear whether this was the aortic systolic murmur transmitted or not.

The diagnosis was aneurism of the aorta, involving the ascending, transverse, and descending portions of the arch; aneurism of the innominate artery, and regurgitation at the aortic valve. The patient was ordered to take iodide of potash, beginning with ten grains three times daily, and he was especially cautioned against sudden or marked exertion.

On February 23d, five days later, he returned. He felt much better. The pain in the shoulders and upper chest was much less, but he still complained of the precordial pain. Physical examination was the same, except that the radial pulse was fuller and apparently stronger. His condition was apparently improved. The dose of iodide of potash was increased to fifteen grains.

This observation was made about 1 p.m. The patient was found not long afterward on a door-step not far from the hospital. He looked sick and distressed, and was very weak. He told the person who found him that he had been at the hospital for treatment. This person assisted him to arise, but finding he was too weak to walk, he left him and hurried to the hospital. An ambulance was sent at once. The patient was carried directly to the ward, and was seen immediately by the house physician. He was in collapse, pale, and with no radial pulse. His mind was clear. He did not respond to stimulants, but grew steadily worse and died in about ten minutes. This was about one hour after he was seen at the out-patient department. It was naturally supposed that the aneurism had ruptured.

At the autopsy the following pathological conditions were found: Fusiform aneurism of the aorta and of the innominate artery; general arterio-sclerosis, with calcification and atheroma; general chronic passive congestion; atelectasis of the lungs.

The aneurism had not ruptured. The heart was enlarged, somewhat

distended with blood, and the right auricle was especially full and prominent, extending further to the right than normally. The aneurismal sac was not tensely distended, but rather relaxed. The aneurism was of the fusiform type, involving the whole of the arch. There was also a fusiform aneurism of the innominate artery, extending from its origin at the aorta to its bifurcation.

The circumference of the aortic valve was 8.5 cm.; six centimetres above this the circumference of the aorta was 13½ cm.; at the innominate artery it was 13 cm.; at the left subclavian artery, 10 cm., and at the first intercostal branches, 7 cm. The opening into the innominate artery had slightly thickened walls and measured 1.1 cm. in diameter. The artery gradually increased in size as it ascended, until it reached a diameter of 2½ cm. From this point the enlargement gradually diminished to the division into the carotid and subclavian arteries, which were both normal in size. There was a marked general arteriosclerosis, with calcification and some atheroma, involving the whole extent of the aorta.

The autopsy, then, thoroughly confirmed the diagnosis, and showed that the extent of the aneurism had been accurately recognized. Moreover, the shape of the innominate aneurism gave an adequate explanation of the peculiar intensity of the systolic murmur near the clavicle, namely, that a new murmur was here created as the blood rushed through the narrow opening into the larger space of the dilated innominate artery beyond.

Death came from the cardiac complication. The existence of aortic regurgitation had been recognized clinically by the aortic diastolic murmur, the enlargement of the left ventricle downward and outward, and the characteristic jerky pulsation of the carotids. At the autopsy the aortic cusps were practically normal, but there was dilatation and slight hypertrophy of the ventricle, and the aortic orifice was somewhat stretched, sharing in the dilatation of the aorta just above. This is shown by comparing the measurements of this case with the normal measurements (Mallory and Wright).

	Normal	Case 11.
Circumference of aortic orifice,	7.7- 8 cm.	8.5 cm.
" mitral "	10.4-10.9 "	10 "
" pulmonary orifice,	8.9- 9.2 "	8 "
" tricuspid "	12 -12.7 "	12 "
" ascending aorta,	7.4 "	8.5-13.25 "
" pulmonary artery.	8 "	? "
Thickness of left ventricular wall,	0.7- 1 "	1.2 "
" right "	0.2- 0.3 "	0.4 "

Here we find the measurements of all other orifices corresponding pretty closely to the minimum figures of the normal table, while we find the aortic orifice nearly one centimetre larger than the minimum figure of the normal table.

The autopsy also showed a general chronic passive congestion of the organs. This suggests that the systolic murmur heard at the apex was due

to mitral regurgitation. This regurgitation was secondary to the dilatation of the left ventricle, as the mitral curtains and chordæ tendinæ were normal. The oedema of the lungs, the distention of the cavities of the heart, and the clinical story of the rapid failure and death all point to failure of the heart muscle as the immediate cause of death. The muscle seemed to be in good condition, and microscopical examination failed to find fatty degeneration. The coronary arteries were patent. We have to fall back on the well-known clinical fact that death is apt to ensue suddenly in aortic regurgitation—a fact for which we are not yet always ready to furnish a satisfactory explanation. We are apt to think of death from aortic regurgitation as more rapid than this, which lasted somewhere between one-half and one hour. But Balfour⁴ has called attention to the fact that ingravescent asystole occurs in aortic regurgitation, and may even be much more prolonged than in this case.

Our story in this case would seem to be clear, and the sequence of events as follows: Syphilis, arterio-sclerosis of the aorta, fusiform aneurism of the arch, resulting dilatation of the aortic orifice, aortic regurgitation, dilatation of the left ventricle, mitral regurgitation, general passive congestion, and death from asystole accompanied by oedema of the lungs. The responsibility of the aneurism for the death is indirect.

This case illustrates so well the view of fusiform aneurisms held by Sir Douglas Powell that I will quote a few of his remarks on the subject. He says: "Clinically the phenomena characteristic of aneurism are scarcely ever observed except in association with the encircled form, the so-called 'fusiform aneurism' being merged, as regards prognosis and treatment, in the class of heart disease with which they are most closely connected. These general dilatations involve, for the most part, the first part of the aorta, and are associated with valvular lesions and secondary cardiac dilatations and hypertrophies; they give rise, with rare exceptions, to no pressure signs; they do not tend to cause death by rupture or by compression of vital parts, but rather through cardiac failure, angina, or syncope. The treatment is that appropriate to heart disease."

Class III. includes by far the greatest number of cases. It is true in the vast majority of cases of aneurism of the aorta that if death is not due to rupture it is due, directly or indirectly, to pressure.

In its course from the left ventricle to the diaphragm the thoracic aorta lies in close relation to many important structures. It is worth while, in the study of the effects of pressure by the aneurismal sac, to briefly recapitulate those organs which may be affected.

The origin of the aorta is imbedded in the structures at the base of the heart. In front lie the conus arteriosus of the right ventricle and the auricular appendage of the right auricle. The right auricle and

the vena cava superior are in close contact on the right. Around the left the pulmonary artery winds from the front, while behind lie the left auricle and the right pulmonary artery.

Just above the heart the edges of both lungs separate the ascending arch of the aorta from the sternum in front. The pulmonary artery is on the left. Behind and below are the right branch of the pulmonary artery and the root of the right lung. On the right lies the vena cava superior and the right lung. It should be remembered that the pericardium extends up on the aorta from one to one and one-half inches above the base of the heart.

The relations of the transverse arch are perhaps the most important of all. The ascending arch has a certain freedom of movement, and after it emerges from the structures at the base of the heart it may become considerably enlarged without serious results to surrounding organs—it displaces rather than compresses them. The transverse arch has much less freedom of movement, its relation to surrounding parts is closer, and these parts cannot readily be moved aside. Enlargement here causes compression rather than displacement. Symptoms of pressure, therefore, develop early in the process of enlargement.

The transverse arch passes backward and to the left in front of the trachea and above the left bronchus. The bifurcation of the pulmonary artery is beneath the anterior part of this section of the aorta. Above lies the left innominate vein. Further back than the trachea lie the œsophagus and thoracic duct. The relations of the nervous structures to this part of the aorta are important. The deep cardiac plexus lies behind the aorta, between it and the trachea, and above the bifurcation of the pulmonary artery. This is the main centre for the nerves distributed to the heart. The superficial cardiac plexus lies between the aorta and the right pulmonary artery. The left recurrent laryngeal nerve passes down in front of the aorta, passes backward beneath it, and ascends behind it and in front of the trachea. In front also pass the left vagus and phrenic nerves. From the transverse arch are given off the innominate, the left carotid, and the left subclavian arteries.

The descending arch passes backward and downward to the left of the trachea, behind the root of the left lung, to the left anterior aspect of the dorsal vertebrae. As it passes downward the œsophagus and thoracic duct lie on its right. The left pleura and lung lie to the left and anteriorly. The descending arch of the aorta and the thoracic aorta below are held firmly in place by the intercostal branches which are given off from the aorta.

We may now turn our attention to the ways in which pressure from an aneurism upon the organs surrounding the aorta may contribute to a fatal result. It is clear that the results will depend partly upon the

size of the aneurism and partly upon the vital importance of the organ or organs pressed upon. It is also manifest that the size of the aneurism is of less importance than the place where the pressure is exerted. A striking case to illustrate this point is reported by Irvine.¹⁹ It was a case of double aneurism of the arch of the aorta. A relatively large aneurism of the ascending arch caused very little trouble, whereas death came because a small aneurism of the descending arch compressed the left bronchus and induced extensive changes in the left lung.

Before considering the effect on individual structures, however, let us note that death may come from the gradual wearing out of the patient's strength without any extreme pressure on vital organs. When an aneurism enlarges enough to press upon peripheral nerves pain results. Pain may also be of the nature of "referred pain," as in cardiac disease. Pressure on these nerves may also cause dyspnea or cough, especially of a paroxysmal type. The suffering from the pain, the dyspnea and cough, and sleeplessness due to any of these factors are the main causes of death in some cases, and such death may be properly called "death from exhaustion."

Pressure on the nerves may contribute to the death in other ways. Spasm of the glottis may result reflexly from irritation of the vagus nerve. And Gibson²⁰ says the same result "may possibly be an early symptom of interference with the motor nerve of the larynx," the recurrent laryngeal. To what extent pressure on the cardiac nerves or ganglia may exert an inhibitory or paralytic effect on the cardiac muscle is not perfectly clear. It is mentioned by some writers, and it may be a possible explanation of some of the cases where the most careful post-mortem examination fails to reveal any adequate explanation. This subject needs further investigation, as does also the question of what effect pressure on the vagus may have on the condition of the lung.

In considering the effect of pressure on the heart and the great vessels, we may practically disregard the left ventricle and the aorta, for it is from the left ventricle and through the blood in the aorta that the pressure is carried to the periphery of the aneurismal sac. We cannot expect this pressure to be greater than its source, hence it cannot compress the left ventricle or aorta. An aneurism arising at the root of the aorta may, however, by pressure distort either the aortic or pulmonary valve. In either case we get regurgitation into the corresponding ventricle as the chief factor in causing cardiac failure and death. Compression or obliteration of the innominate or left carotid or left subclavian arteries may occur. This leads to impaired nutrition of the parts supplied by these arteries, but the collateral circulation is so good that a fatal result is hardly to be expected from this cause alone, unless all three were occluded together, of which occurrence I have found no reported case. The supply of blood to the brain is, of course, the most

important item in considering obstruction of these arteries. Of the other branches of the general arterial system we need consider only the bronchial arteries—the nutrient arteries of the lung. Obliteration of one of these will cause gangrene of the part of the lung supplied by it, with fatal termination. This is a less frequent occurrence than was at one time supposed, as the bloodvessels of the roots of the lung are so situated that they are generally protected by the resistance of the bronchi.

Returning to the heart, we have to consider pressure upon the auricles, the conus arteriosus of the right ventricle, the pulmonary artery, and the pulmonary veins. In these cases we are dealing with obstruction of the circulation, essentially the same in its results as is found in valvular disease. Death may result in the same way as from broken compensation. Pressure on the vena cava superior is by no means uncommon. We get venous stasis of the upper part of the chest, the head, and the upper extremities. Collateral circulation may lessen this by furnishing a channel to the vena cava inferior, by anastomosis with its branches, or to the vena azygos if the pressure be above the point of entrance of that vessel into the vena cava superior. Here, again, the brain is the important organ to consider, and death by coma from the obstructed venous circulation is reported. (Edema of the glottis has also been reported)" as a cause of death from impeded venous circulation.

Case III. is an illustration of death caused by pressure interfering with the circulation. The point of application was the pulmonary artery.

Male, aged forty-two years, painter, came to the out-patient department of the Boston City Hospital in the latter part of September, 1900. He had diptheria eleven years ago. Otherwise he had been in good health, except for syphilis ten years before. He had used alcoholic drinks and tobacco to excess.

His present illness began about one year ago. He began to get short of breath on exertion, and also had vertigo and palpitation. For some months he had had pain at times in the precordial region, radiating into the shoulders. Of late the pain had been worse and more frequent, and he complained of increasing dyspnea and weakness.

On physical examination the cardiac area of dulness was practically normal. At the base of the heart was to be heard a peculiar murmur. It was loud and harsh in quality. Its point of maximum intensity was to the left of the sternum just above the pulmonic area. In rhythm it began just after the first sound—that is, it was a somewhat late systolic murmur—and continued just a trifle after the second cardiac sound; yet it was clearly all one murmur, and not a systolic running into a diastolic murmur. There was also a loud, ringing, markedly accentuated second pulmonic sound.

This peculiar combination is characteristic of a persistent ductus arteriosus. But it seemed highly improbable that such a case should live to this age without a longer history of cardiac trouble. The only other explanation which seemed at all likely was the existence of an

aneurism which compressed the pulmonary artery, and thus raised the tension in the pulmonary circulation. There was, however, no dulness, no pulsation, no tracheal tug, no hoarseness, no inequality of pupils, or any other physical sign to confirm the suspicion of aneurism of the arch of the aorta. He was asked to return for X-ray examination, but did not return till December 7th, when he was admitted to the hospital.

He was now much worse. He was weaker, had much more pain, more dyspnoea, had been unable to do any work for three weeks, and had had orthopnoea for two weeks. He had also become hoarse at these times. He was pale and had a slight cyanotic hue. The pulse was 80, regular, and of fair volume and strength. There was no perceptible difference in the two wrists. A distinct tracheal tug could now be felt.

There was now a slight bulging of the chest just to the left of the sternum, from the second interspace nearly to the clavicle. Over this area a slight pulsation could be seen; palpation revealed a distinct, coarse, rough thrill; percussion showed marked dulness; and on auscultation a rough, coarse, rumbling systolic murmur was heard. The second pulmonic sound was still somewhat accentuated, but was not as loud or clear-cut as before. Moreover, it was followed by a diastolic murmur, with its maximum intensity in the pulmonic area, transmitted downward and diagonally across the sternum to the right—following the location of the right ventricle and not the left.

This murmur seemed to me to indicate pulmonary regurgitation. It was now perfectly evident that we had an aneurism pressing front to the chest wall to the left of the sternum. This had in September been too small to be detected, but so situated as to press on the pulmonary artery and greatly increase the resistance in the pulmonary circuit. This obstruction had now increased so much as to cause regurgitation through the pulmonary valve.

As a matter of interest an X-ray examination was made in this case also. It showed the heart slightly enlarged to the left, but markedly enlarged to the right. Above the base of the heart was an abnormally broad shadow, extending especially to the left of the sternum and very closely resembling the shadow of the aneurism of the descending arch, as shown in Fig. 1. The outline is shown in Fig. 2.

This patient gradually failed, and died December 17, 1900. Toward the end the attacks of dyspnoea were more frequent and severe. He had to be propped up in an upright sitting position for the last forty-

FIG. 2.

Fluoroscopic examination.
Sacculated aneurism of the ascending arch,
growing forward and to the left, and compressing the pulmonary artery.

eight hours of life. During the last few hours he developed pulmonary edema and his pulse gradually failed.

The autopsy showed a sacculated aneurism of the aorta compressing the pulmonary artery; hypertrophy and dilatation of the right ventricle; hypostatic congestion of the lower lobes of the lung, and atelectasis of an area of left upper lobe of lung, which was compressed by the aneurism; chronic passive congestion of liver, spleen, intestines, and kidneys; fatty degeneration of the heart and kidneys.

On removing the sternum the mediastinum presented a roughly spherical mass about 10 cm. in diameter, projecting forward and to the

FIG. 3.

Interior of left ventricle and aorta, showing opening into aneurismal sac just above the cusps of the aortic valve.

left. The mass is continuous with the heart and vessels of the base, and lies anterior and to the left of them. It projects 8.5 cm. to the left of the median line at about the level of the second rib. On dissection the mass is found to be an aneurismal sac 11 x 9 x 7.5 cm., which communicates with the aorta by an oval opening 2.75 x 2 cm. in diameter. This opening is situated in the anterior wall of the first portion of the aorta 3 mm. above the upper margin of the aortic cusps. The opening is on the left anterior aspect, above the junction of the anterior and the left posterior cusps of the valve. The edges of the opening are smooth and slightly raised. (See Fig. 3.)

Inside the sac a rounded mass of firm, organized clot fills the cavity to within $\frac{1}{2}$ cm. of the opening. This mass becomes firmly adherent to the inner wall of the aneurismal sac at $\frac{1}{2}$ to 1 cm. from the edge of the opening, except at the posterior side of the opening. Here a flattened pocket extends 3 cm. in depth between the rounded sac on the outside and the rounded mass of clot on the inside. The portion of this pocket farthest from the aortic opening is directly opposite the sinus of the right anterior cusp of the pulmonary valve. It is evident that this pocket is so situated as to have caught the greatest force of the blood

FIG. 4.

Aneurismal sac laid open, showing layered clot within.

current entering the sac, and this fact has prevented the formation of blood clot at this point. The cause of the peculiar murmur heard in this case was the eddying blood current in this pocket of the aneurismal sac.

Elsewhere the whole aneurismal sac is filled with firm clot. An incision from the anterior surface of the tumor shows a firm, fibrinous, dark red, layered clot, everywhere adherent to the inside of the sac. (See Fig. 4.) This aneurism not only had not ruptured, but it presented that condition, filled with firm clot, which is called "cured."

The general direction of the growth of the aneurism has been to the left and forward. It has also extended upward, as two-thirds of the mass is above the opening from the aorta. Still more important is the growth of the tumor a short distance backward, crowding in between the aorta and the pulmonary artery, so that where the transverse arch should cross close above the pulmonary artery it is separated from it by an aneurismal mass 5 cm. in thickness. The arch of the aorta has been apparently pushed upward, backward, and to the right to accommodate this growth; but there has been little, if any, diminution of the lumen of the artery.

FIG. 5.

Interior of right ventricle and pulmonary artery. Cusp of pulmonary valve stretched taut over bulging surface of aneurism.

In the pulmonary artery, however, a different condition obtains. There has been marked flattening from the encroachment of the tumor upon its lumen. The tumor presents as an irregular rounded mass, projecting downward and to the left into the pulmonary artery and the upper part of the right ventricle. (See Fig. 5.) The right anterior cusp of the pulmonary valve is stretched taut over the rounded surface of the tumor. The tumor projects into the right ventricle just below this valvo, and the distance over its rounded surface is 4 cm. from the right ventricular wall to the upper edge of this cusp, and from this point to the end of the projecting mass in the pulmonary artery is 7 cm. The tumor mass as felt from the right ventricle or pulmonary artery is firm

everywhere except over the shallow pocket of the aneurismal sac already mentioned. This pocket lies behind the right anterior cusp of the pulmonary valve and part of the pulmonary artery just above it. Though yielding in the post-mortem specimen, it must have been firmly distended by the pressure of the aortic blood during life. The right anterior cusp of the valve must have lain flat over the surface, useless as far as its function was concerned.

The right ventricle was very much dilated, and the wall was hypertrophied to about four times its normal thickness. Death came from failure of the right ventricle, owing to the obstruction in the pulmonary artery and the regurgitation through the crippled pulmonary valve.

Pressure on the trachea or the primary bronchi is a frequent cause of death in aneurism of the aortic arch. This may act directly or indirectly. When it acts directly, death comes by suffocation, and the pressure is usually on the trachea just above its bifurcation. Death from this cause is rarely sudden; it is generally by slow asphyxiation, and the suffering is terrible. Sometimes the attack of asphyxiation is rather rapid in course, and unconsciousness ensues before there is much suffering, as in the following case:

CASE IV.—Male, aged thirty-nine years, laborer, entered the Boston City Hospital, December 6, 1900. Family history was negative. He had had typhoid fever when young, and remembered no serious illness since then. At twenty-seven he had syphilis. Used alcohol moderately, tobacco to some excess.

His present illness began in August, 1900, four months before, when he began to have occasional spells of difficult breathing. He felt an attack coming on, would then sit down, and soon lost consciousness. If severe, the attack lasted about one and one-half hour. The attacks were followed by great prostration. The attacks came on rather suddenly after exertion or excitement. He had one just before entrance to the hospital; another five days before, and another five weeks before that. The intervals between the attacks had been variable. In the attacks breathing was extremely difficult, and he got blue in the face.

In the intervals between attacks he had pain and discomfort in the precordia. There was some cough, but little expectoration. The voice had been husky since September.

Physical examination showed the pupils equal, the left vocal cord immovable, and the radial pulse equal and of good volume and tension. Tracheal tug was present. There was no edema. Examination of lungs was negative. Upper border of cardiac dulness was at the third rib. The right border was one and one-half inches to the right, and the left border five and one-half inches to the left of the median line. Apex-beat was in the fifth interspace. A soft, systolic murmur was heard at the base, loudest to the left of the sternum. The second pulmonary sound was accentuated. Physical examination was otherwise negative.

Up to January 21, 1901, a period of about six weeks, he had three attacks—all of them severe. The dyspnea was especially marked on expiration. He became cyanotic and lost consciousness. He recovered from these attacks, but was exhausted and weak for hours afterward.

The hoarseness had continued, but varied in degree. At times he had a metallic, barking cough. There was dyspnea on any marked exertion. He was able to be up and about the ward most of the time.

An X-ray examination had been made on December 21, 1900, and showed the following outline. (See Fig. B.)

From January 21st to April 18th he had five attacks at varying intervals. On May 3d he had an attack. On May 5th and 6th he had four attacks. On the night of May 7th he had an attack during which he died.

The autopsy showed the following: sacculated aneurism of the transverse arch of aorta, and dilatation of the ascending arch; obstruction

of trachea by pressure of aneurism; distention of lungs; chronic healed tuberculosis of lung; arterio-sclerosis; and hypertrophy of heart.

The heart was somewhat enlarged, weighing 480 grammes, the principal change being in the left ventricle. The muscular tissue was firm and dark red, but showed some fatty degeneration on microscopic examination. The aorta is apparently dilated, and a mass about the size of a lemon lay below the clavicular notch, extending slightly into the left pleural cavity. This was a sacculated aneurism of the transverse arch of the aorta, which had not ruptured.

The aortic orifice measured 8.5 cm. in circumference, and the ascending arch was somewhat dilated, measuring from 9 to 10.5 cm. in circumference. At a point 9.5 cm. from the aortic valve there is a rounded border forming part of the

Fluoroscope Examination.
Sacculated aneurism of transverse arch,
pressing on trachea.

opening of an aneurismal sac. This opening measured 8 by 9 cm. in diameter. This sac arose almost entirely from the posterior and inferior part of the transverse arch, while the innominate, left carotid, and subclavian arteries arose in front from the apparently normal aortic wall, and were practically normal. The sac was hemispherical in shape, and the greatest circumference of it and the dilated aortic wall was 22 cm.

The aneurism lay directly anterior to the trachea just above its bifurcation. The trachea was flattened and curved back by the pressure, and its lumen was encroached upon from the anterior and left side by the bulging wall of the aneurismal sac. Even with the sac empty the lumen of the trachea was only one-third of its normal size at this point.

The lungs were very much distended with air. When the sternum was removed they did not collapse, but bulged forward, nearly covering the heart. On cutting the bronchi the lungs collapsed to less than one-half their former size from the escape of air. There was an area of

healed tuberculous tissue occupying about one-third of the right upper lobe. Elsewhere the lung tissue was normal. The bronchi were normal. The bronchial lymph nodes were normal.

It was clear that the distention of the lungs was due entirely to the pressure of the aneurism on the trachea. The pressure of the distended sac must have been sufficient to entirely prevent the exit of air, and the patient died of suffocation. It is not difficult to understand how the air passed by this obstruction on inspiration, but could not escape again. With the descent of the diaphragm on inspiration the heart and anterior part of the arch are drawn downward. This drew the aneurism away from the trachea enough to allow the entrance of air, but when this traction ceased at the end of inspiration the pressure was at once resumed, and the aneurism acted like a ball valve to prevent the exit of the air. The resulting dyspnoea caused more forcible inspiratory efforts, the entrance of more and more air which could not escape, and the extreme distention of the lungs resulted. With the complete distention of the lung no more air would enter, and respiration was brought to a standstill, and death ensued from suffocation.

Compression of one bronchus is not so likely to cause death from suffocation. It is rarely so sudden that the necessary supply of air cannot be obtained through the other lung. Death is more likely to come through secondary changes in the lung. How far these secondary changes may be the result of pressure upon the bloodvessels or nerves at the root of the lung has been a matter of dispute, and I do not think we are yet prepared to decide the question definitely. It is, however, certain that the mere mechanical occlusion of the bronchus may result in extensive and fatal changes in the lung tissue. This is due to the retention of secretions and the invasion of micro-organisms. Collapse and consolidation of lung tissue, a purulent bronchitis extending to the smaller divisions, and then to the lung tissue, and causing multiple foci of suppuration, and gangrene, are the manifestations of pulmonary disintegration which follow compression of the bronchus. Death in this case comes as an indirect result, not as the direct result of the pressure of the aneurism. Irvin⁹ has reported a case of this sort, and has called attention to the subject. Compression of one primary bronchus may, however, produce collapse and atelectasis of the corresponding lung without further secondary results, as is shown in Case V.

Finally, in considering the effects of pressure on the pulmonary tissue, we must note the occurrence of cases where the aneurism is so situated that it does not press on the main air passages, but upon the lung tissue itself. Here we get primarily a compression of the lung tissue, and may get secondary inflammatory processes in the tissue thus affected.

Pressure on the oesophagus may become so extreme as to prevent the passage of food and cause death by inanition. In less degree it may be

a more or less important factor in causing insufficient ingestion of food and imperfect nutrition, thus rendering the patient an easier victim to intercurrent disease of any kind. Compression of the thoracic duct may cause serious impairment of nutrition with similar relations to the fatal termination, but this is extremely rare.

The following case illustrates death by starvation from the pressure of an aneurism on the œsophagus.

CASE V. Female, aged fifty-seven years, seamstress, had always enjoyed good health before the present illness. In the fall of 1893 she first noticed a pain in the lower left axillary region, which also shot into the back and up to the left shoulder. This continued at intervals.

In the fall of 1895 she began to have partial aphonia and difficulty in swallowing. After a time solids could not be swallowed at all, but she could swallow liquids. By the last of January, 1896, a hacking cough had set in. Sibilant and sonorous râles were heard over both lungs. By March there was considerable expectoration, which was occasionally streaked with blood. By April dyspnea was caused by even slight exertion. She was very weak. She had been losing flesh since fall, and was now greatly emaciated. Even liquids were now vomited or regurgitated, and nutritive enemata were resorted to after April 5th. She failed gradually, and died quietly on June 2, 1896. No new symptoms had developed, and all the old ones had persisted.

During the latter part of her illness it was noticed that the respiration was very faint on the right side of the chest. Tuberculosis of the lung was thought of, but the diagnosis would hardly account for all the symptoms, and no tubercle bacilli could be found in the sputum. The probable diagnosis was thought to be a malignant tumor of the œsophagus. This would account for the obstruction of the œsophagus, the wasting, and the pain, and, by extension and pressure on the left recurrent laryngeal nerve and right bronchus would account for the aphonia and diminished respiration of the right lung. This clinical diagnosis represented very closely the autopsy findings, except that the tumor was an aneurism of the aorta instead of a malignant tumor of the œsophagus.

The autopsy showed a sacculated aneurism of the thoracic aorta; atheroma of the aorta; pressure of aneurism on right primary bronchus and on œsophagus; atelectasis of right lung; bronchitis of left lung, and all other organs normal.

The aneurism was a sacculated one, about three and one-half inches in diameter, arising from the upper part of the descending thoracic aorta. It had grown forward and to the right, crowding in between the œsophagus behind and the air passages in front. The œsophagus was compressed flat against the vertebral column. The right primary bronchus was so completely occluded by the pressure toward the front that the right lung had collapsed and was in a state of atelectasis, but no inflammatory change had resulted. The fourth to sixth dorsal vertebrae (inclusive) were eroded on their right side from the pressure of the sac. The aneurism was partly filled with lamellated clot, and had not ruptured. The thoracic duct could not be found. The anatomical relations of the aneurism, the absence of other pathological cause, and the very extreme emaciation showed that death came from starvation, of which the aneurism was the immediate cause.

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THE ASSOCIATION OF PULMONARY TUBERCULOSIS WITH
BOTH PRIMARY AND SECONDARY ENDOCARDITIS,
AND THE EFFECT OF VALVULAR DISEASE
UPON LUNG TUBERCULOSIS.*

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THE subject of the relationship existing between diseases of the endocardium and pulmonary tuberculosis is admittedly but imperfectly understood. The cases of endocarditis met with in tuberculosis of the lungs are clearly divisible into several classes, as follows: 1. Those due to the presence of tubercle bacilli within the heart—*endocarditis tuberculosa*. 2. Those that are secondary to tuberculosis or merely intercurrent, and caused by various organisms other than the tubercle bacillus. 3. Various forms of valvular heart disease that precede the tuberculous infection of the lung and are due to rheumatism and other etiological agencies.

A distinction between these different varieties has not, as a rule, been

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