Intrathoracic aneurysm / by F. de Havilland Hall.

The Lumleian Lectures

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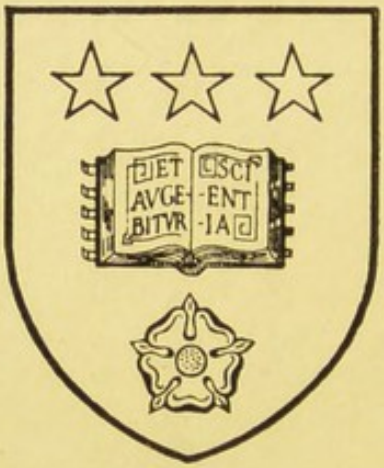

The Lumleian Lectures

ON

INTRATHORACIC ANEURYSM

Delivered before the Royal College of Physicians of London
on March 6, 11, and 13, 1913

BY

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The Lumleian Lectures

ON

INTRATHORACIC ANEURYSM.

LECTURE I.

Delivered on March 6th.

Mr. President, Fellows and Gentlemen,—My first duty is to tender my grateful thanks to the Censors' Board for selecting me for the post of Lumleian lecturer. On referring to the annals of the College it will be seen that the Lumleian lectures were the first lectures to be founded in the College, and there is on record the steps that were taken "for the better celebration of this most solemn lecture." At their inception the surgical side of the great science of medicine was chosen as the proper object of the lectures; it will be, therefore, not altogether unsuitable if I devote some portion of the time to the consideration of what surgery can do for the alleviation of intrathoracic aneurysm.

In selecting my subject, intrathoracic aneurysm, I was influenced by the fact that from the commencement of my medical career I was attracted by the difficulties in the diagnosis of this disease and by the tragedy which so often accompanies its progress. You have before you a disease which usually strikes down men in the prime of life, sometimes they die quite suddenly without any warning, except perhaps some vague pain or discomfort, or, on the other hand, you may see a poor being suffering the "throbs of fiery pain" which attend the gradual erosion of the ribs or vertebræ, and this anguish may be protracted for months, so

that death comes at last as a merciful release. Finally, there is its all but certain progress to a fatal termination, with just sufficient hope to tantalise the patient should he be conversant with the facts of the disease. In all medicine I know nothing more pathetic than what is seen in cases of aneurysm.

I will preface my remarks by saying that the scope of the lectures is limited to the discussion of the saccular variety of intrathoracic aneurysm chiefly from a clinical point of view.

Etiology.

The two prime factors in the production of an aneurysm are some degenerative change in the wall of the artery and strain. This is borne out by the fact that aneurysms are most frequent at that period of life when the greatest bodily vigour coexists with the commencement of atheroma. There can be no doubt that among the most important conditions giving rise to aneurysm is the endarteritis of syphilitic origin. Out of 89 male deaths from thoracic aneurysm in the Westminster Hospital there was a history of syphilis in 34, and in 9 female deaths a history in 4—i.e., a total of 98 deaths, with a syphilitic history in 38. The probability is that this does not express the full importance of syphilis in the production of aneurysm, as the difficulty of obtaining a correct history in hospital patients is well known. When the Wassermann test comes to be employed as a matter of routine in every case of aneurysm, we shall be in a better position to assign to syphilis its true place in the causation of aneurysm. Overwork, especially under unfavourable conditions, gives rise to degenerative changes in the vascular system and to localised patches of atheroma. The effect of alcohol in producing aneurysm is rather more doubtful. It is generally agreed that it is a potent cause of degenerative changes in the arteries, but this is usually accompanied with loss of tonicity in the arterial wall, and there is little tendency for the changes to be circumscribed. The beer-drinker who imbibes huge quantities of fluid keeps his vessels at a high tension, and if

he be also a hard worker then doubtless there is a liability to aneurysmal dilatation. If, as is often the case in these circumstances, syphilis be thrown in, the man is extremely likely to develop an aneurysm.

The statistics I am about to quote strongly support the usually accepted view that thoracic aneurysm is most common during the period of great bodily activity coinciding with the commencement of degenerative changes in the vessels. The following tables illustrate this point :—

Table of Age Incidence at Death (Dr. Oswald Browne's figures).

Age at death.	Men.	Women.
Under 35	22	2
35 to 55	82	15
Over 55	18	1
Age not given...	10	0
	132	18

Only one case occurred before the age of 25, a girl aged 15, and only two over 65, both men. These figures may be compared with those extracted from the post-mortem records at the Westminster Hospital.

Age at death.	Men.	Women.
Under 35	19	2
35 to 55	64	7
Over 55	5	0
Age not given...	1	0
	89	9

Only two cases occurrred before the age of 25, both men aged 24 years, and only one case over 65, a man aged 68 years. The average age at death (Westminster Hospital cases) for men is slightly over 43 years ; for women almost exactly 43 years. As might have been expected, the syphilitic cases occur at a somewhat earlier period. The age incidence

at death is almost exactly the same for men and women—i.e., 40 years. The figures are, of course, few, but they are suggestive.

A specimen in the museum of St. Bartholomew's Hospital, taken from a boy 6½ years of age, is an example of at how early an age an aneurysm of the aortic arch may occur. Death was due to rupture into the pericardium. The aneurysm was probably owing to localised septic aortitis, the boy having suffered from middle-ear suppuration.[1]

As regards the sexual proclivity of thoracic aneurysm, it would appear that it is a comparatively rare disease in females. All the cases I have seen in private have occurred in males, and in my hospital practice the cases of thoracic aneurysm in women have been of very infrequent occurrence. Between the years 1884 and 1910—both included—there were admitted into the wards of the Westminster Hospital 160 men and 28 women in whom the diagnosis of thoracic aneurysm was made. According to Lebert,[2] the proportion of males affected compared to females is as 10 to 3. Dr. Oswald Browne's figures give 122 to 18. From the post-mortem records at Westminster Hospital the figures are 89 males to 9 females. The last two sets of figures combined work out nearly 8 males to 1 female. The most reasonable explanation of this discrepancy is the greater strain to which men are liable, and this will especially explain the almost complete exemption of women in the upper ranks of society.

As Dr. Drummond[3] has pointed out, it is recognised "that certain diseases are more prevalent in some parts than others." This is certainly the case in regard to the occurrence of thoracic aneurysm in the Tyneside district. When examining for Durham University at Newcastle-on-Tyne I had the opportunity of observing this fact. From his large experience in the disease Dr. Drummond's conclusions are naturally of great value. As regards strain from heavy and laborious occupation being the chief factor in the causation of thoracic aneurysm, he says: "A careful examination of upwards of 300 examples has convinced me that this view is incorrect. For instance, out of a group of 145 cases in which the point was specially noted, 64 only had been engaged in heavy employment; in 81, including 14

women, the question of strain could be eliminated." His inquiry shows history of syphilis in 102 out of 145 cases. In his opinion, "Aneurysm of the thoracic aorta is undoubtedly a syphilitic disease, though aortic strain from hard work, where the tissues are already degenerated by alcoholic excesses, must certainly predispose." In more than 40 per cent. of his cases a strong alcoholic history was obtained.

Dr. Lee Dickinson [4] laid great stress on hypoplasia as a cause of aneurysm, and has put on record four cases in which this condition was apparently an important factor in the development of the aneurysm. He pointed out that women are notoriously more liable to hypoplasia than men, and it would seem that the rapid course which aneurysms run in women and their tendency to rupture externally may be dependent upon this peculiarity of the aortic wall. It may also possibly account for the occurrence of aneurysm in young children, which is not susceptible of explanation by ordinary causes.

Situation of the Aneurysm.

From an analysis of 150 cases of thoracic aneurysm dying in St. Bartholomew's Hospital [5] in the 30 years intervening between October, 1867, and May, 1897, and which were submitted to a post-mortem examination, the following figures indicate the portion of the aorta involved in the aneurysm: ascending portion of arch, 58; transverse portion of arch, 35; ascending and transverse portions combined, 19; descending portion of arch, 21; and descending thoracic aorta, 17; total, 150. The statistics of Crisp, Lebert, and Myers show the following figures: ascending aorta, 159; arch of aorta, 113; and descending thoracic aorta, 49; total, 321. The following are the statistics of Westminster Hospital: ascending portion of arch, 28; transverse portion of arch, 21; ascending and transverse portions combined, 10; descending portion of arch, 6; more than one portion of arch, 9; descending aorta, 18; and multiple aneurysms, 6; total, 98. These figures would apparently indicate that the force of the ventricular systole is a potent factor in the

production of aneurysmal dilatations, as the frequency of the disease diminishes as the distance from the heart increases.

Aneurysms of the ascending part of the arch of the aorta, which come to the surface and give rise to a tumour, almost invariably arise from the right or convex side of the vessel, and tend to enlarge towards the right. Dr. Gee states that he has found only one reference to a state of things contrary to that mentioned above as being the rule; and he quotes Oppolzer[6] when speaking of aneurysm of the ascending aorta as follows: "That as to the situation of the tumour to the right of the sternum, this is undoubtedly the rule in the majority of instances, inasmuch as the aneurysms arise likewise, as a rule, from the convex side of the arch. But in exceptional cases they spring not from the convex but from the concave wall of the aorta, and then the said tumour is found, not on the right, but on the left side of the sternum." Dr. Gee[7] has himself recorded a case in which a low, rounded, smooth tumour was seen stretching from the second to the fifth ribs on the left side between the sternum and the nipple line. Systolic pulsation was palpable, and a blowing systolic murmur and a weak second sound were audible over the tumour. Dr. Gee considered it probable that the signs were produced by an aneurysm of the pulmonary artery, but at the necropsy the aneurysmal sac was found to be formed by dilatation of the whole of the ascending part of the arch of the aorta.

The case I have just narrated illustrates the view that we are not justified in inferring the place of origin of an aneurysm from the place at which it appears on the chest. As another example of this difficulty I may point out that an aneurysm arising from the descending portion of the aorta has, nevertheless, come to the surface against the upper ribs.

Of aneurysms at the sinuses of the aorta I have had but little experience. I recorded[8] a case of a patient with four distinct aneurysms in the arch of the aorta, one of which was of this nature. Just above the aortic valve, corresponding to the sinus of Valsalva, was an aneurysmal cavity of the size of a small Tangerine orange. The valve allowed slight regurgitation. In his article on "Aneurism at the Sinuses of the Aorta" Sir Richard Douglas Powell[9] remarked: "The

diagnosis can rarely be made with certainty. It is almost impossible to single out from among the symptoms which may be accounted for by the many attendant lesions, those peculiar to an aneurism rarely exceeding the size of a filbert, and buried in the base of the heart." A diastolic murmur is frequently met with in these cases, but it is often inaudible, and comes and goes without any definite reason. In my case it is quite certain that there was no diastolic murmur while he was attending as an out-patient, but soon after his admission a loud diastolic murmur was audible, in the production of which this aneurysm took, I believe, a leading part. This view is in agreement with that given in Fagge and Pye-Smith:[10] "If the aneurism forms a pouch in the sinuses of Valsalva the valves become involved, their closure may be prevented, a diastolic bruit be produced, and all the other signs and symptoms dependent upon regurgitation occur. These may, indeed, be the only symptoms, and none exist indicative of the presence of an aneurism. The sac never grows to any great size, and usually bursts into the pericardium and causes instant death."

A symptom occasionally noticed is a single or double murmur at the pulmonary orifice; this is met with when the aneurysm develops in the direction of the pulmonary opening, and thus by increase in size distorting that orifice.[11]

A very unusual, indeed I might almost say unique, point of projection of an aneurysm is that recorded by Dr. Churton.[12] During life the swelling extended from the second to the fourth rib and from the mid-axilla to the nipple line, so that the aneurysm projected into the right axilla. At the necropsy the aneurysm was found to lie wholly in front of the lung and to have compressed the lung. An aneurysm of the descending portion of the arch may appear externally, to the left of the spinal column, as in a case reported by Dr. John Brunton.[13] The swelling was noticed between the vertebral border of the left scapula and the spinal column, the scapula being pushed outwards. Death occurred by rupture into the left pleural cavity, and at the necropsy a large aneurysm was found communicating with the posterior wall of the aorta.

The ex stence of several aneurysms in the same patient is a well-recognised fact. The physician is usually satisfied in being able to diagnose the presence of one aneurysm, but he should always bear in mind the possibility that more than one may exist. In a patient, a man of 36, whose case I read before the Medical Society of London,[14] I definitely recognised and recorded the presence of two aneurysms, but at the post-mortem examination four distinct aneurysms in the thoracic aorta were found.

Aneurysms of the arch of the aorta, especially when of large size, may cause considerable displacement of the heart in a downward direction, so as to give rise to epigastric pulsation. Dr. Hugh Walsham [15] has shown by means of the fluorescent screen that the heart occupies nearly a transverse position in the chest in many cases of aneurysm of the aortic arch. This position of the organ tends to raise rather than to lower the apex, unless there be considerable hypertrophy of the left ventricle, which is the exception in aortic aneurysm. Dr. Walsham claims that, in the absence of other physical signs of aneurysm of the aorta, this transverse position of the heart may be of great diagnostic value. My colleague, Dr. R. G. Hebb, has for many years demonstrated and taught at the Westminster Hospital that aortic aneurysm is not a cause *per se* of hypertrophy of the left ventricle of the heart. Dr. J. Calvert [16] has carefully analysed Dr. Oswald Browne's cases, and he has come to the conclusion that, so far as the records of St. Bartholomew's Hospital are concerned, there is no evidence in favour of aortic aneurysm being a cause of hypertrophy of the left ventricle. I think it may be taken for granted that some other explanation of the hypertrophy in these cases can be found. There is usually no dilatation of the ventricle, provided the aortic valve remains competent; so that a large aneurysm may exist within an inch of the aortic ring, and yet the left ventricle may be normal.

The chief characteristics of aneurysms affecting the different portions of the thoracic aorta require mention. Aneurysms of the intrapericardial portion of the arch may be so small as to elude the most careful examination, and they may cause unexpected death by rupture and hæmor-

rhage into the pericardium. Aneurysms of the first part of the arch of the aorta beyond the attachment of the pericardium were designated by Broadbent the aneurysms of physical signs, as they usually come to the surface and yield very definite indications. The physical signs are first manifested in the second right interspace, and the signs advance in a downward direction, so that later on they may be clearly recognised in the third space. Aneurysms affecting the transverse and descending part of the arch were termed by Broadbent aneurysms of symptoms as owing to pressure on the trachea, œsophagus and other structures they present marked symptoms, but physical signs may be equivocal. Pulsation of an aneurysm of the transverse arch may sometimes be felt in the episternal notch ; an aneurysm of the descending portion of the arch may come to the surface in the left infraclavicular region, or may even appear in the left interscapular region.

Aneurysms involving the descending thoracic aorta may well be called aneurysms of latency, as they often exist without definite symptoms or physical signs, and death may take place quite unexpectedly by rupture into the pleural cavity. When aneurysms of this portion of the aorta give rise to any symptom it is usually pain of an excruciating nature, due to pressure on, and erosion of, the vertebræ. As a result of the destruction of the vertebræ paraplegia may occur. Other symptoms sometimes met with in aneurysm of the thoracic aorta are dyspnœa and cough due to pressure on the root of the lung, and dysphagia from compression of the œsophagus. Owing to the depth at which the aneurysm is seated physical signs are usually conspicuous by their absence. As a result of pressure on the left bronchus there may be defective entry of air over the left lung, and, as Dr. Newton Pitt has pointed out, a diffuse bronchiectasis, with the expectoration of a large quantity of mucus, may result. If the aneurysm attain to a large size there may be impairment of resonance on percussion to the left of the spine, and a pulsating tumour has even been felt.

In opening a discussion on the Diagnosis and Treatment of Aneurysm of the Aorta at the Medical Society of London in December, 1889, Sir Richard Douglas Powell [17] made the

following statement: "The essential phenomena of aneurysm are, firstly, those derived from the pressure of a tumour upon surrounding parts; secondly, those indicative of that tumour being a vessel tumour, a diverticulum from the main artery. If in attempting to make a diagnosis we reverse this logical order of reasoning, and attach primary importance to the evidences of arterial disease and insufficient weight to the presence or absence of those of tumour, there are many clinical snares and pitfalls awaiting us." These wise words should always be borne in mind when examining a patient with symptoms or signs suggestive of aneurysm.

Symptoms.

The symptoms met with in intrathoracic aneurysm may be grouped as follow: (I.) Pain; (II.) Respiratory symptoms; (III.) Laryngeal symptoms; (IV.) Circulatory symptoms; (V.) Dysphagia; (VI.) Paraplegia and Hemiplegia; and (VII.) General symptoms.

I.—*Pain.*

Pain is one of the earliest and most constant of the symptoms of aneurysm. It may be of three kinds: (1) anginal, (2) that due to pressure, and (3) neuralgic. It is important to bear in mind that angina pectoris of the most typical character may owe its origin to aneurysm, otherwise the physician may be content with recognising the symptom and fail to discover its cause. As in other forms of angina, the pain may vary from slight discomfort in the cardiac region to the severest form of angina pectoris, in which the sufferer experiences acute pain in the præcordium, extending down the left arm, with an awful sense of constriction in the chest and the fear of impending death. In aneurysm, also, the distribution of the pain is the same as in angina due to other causes—i.e., it most frequently extends down the left arm, but the right arm may be affected; indeed, the pain may radiate to any part of the body.

In considering the effects produced by pressure of the growing aneurysm, it must be remembered that pain may be the only symptom to direct attention to the insidious malady, which is fraught with such terrible consequences to its victims. Deep-seated and persistent pain in the chest and back is most commonly due either to aneurysm or to a malignant growth, and it will sometimes puzzle even an experienced physician to decide with which of these two conditions he has to deal. The advice given by Sir Thomas Watson [18]—that Nestor of medicine—whose lectures, I am afraid, are seldom read in these scientific days, should be kept in mind: "When an adult person makes constant complaint of certain morbid feelings in a part, the probability is that he *has* something the matter, and we must investigate the case with what helps we can get." Fortunately since his days science has placed in our hands resources for investigation of which he could never have dreamt. A patient who has persistent pain in the chest which cannot be assigned to any definite cause should be X-rayed, and this method of examination will usually suffice to clear up the diagnosis.

The pain resulting from pressure of the aneurysmal sac upon the vertebræ is frequently of an agonising character, so that the unfortunate sufferer is at times tempted to put an end to his misery. Yet, on the other hand, we may come across cases in which the aneurysm in its onward march has eaten through the ribs, and appeared as an external tumour on the surface of the chest, though the patient may deny that at any time he has experienced pain—certainly not severe pain. I am at loss to explain the reason for this great difference, but that it exists I am sure. Dr. Sutton [19] gives a notable example of such a case: "There was a man lying in the ward with a big pulsating mass which I thought was an aneurysm, and the largeness of the pulsation made me suspect that the aneurysm had burst. I thought that the inexperience of the clerk might prevent his getting at the history rightly, so I took the notes myself. 'I was well,' said the patient, 'till a few weeks ago, when I was jammed by a cart against a load of bricks.' But an aneurysm had eroded his vertebræ for months and months, and the accident

had burst it. How obscure!" I can but repeat after Dr. Sutton, How obscure! Why in one case the pain should be so severe and so persistent, and in another case, apparently quite similar, pain may be entirely absent, is a problem I am unable to solve. As a rule, the comparative painlessness which attends the eroding by an aneurysm of the sternum and ribs is in striking contrast to the prolonged anguish of pressure on the spine.

The pain caused by a thoracic aneurysm eroding the vertebræ has been mistaken for the pain of indigestion. The severity and persistence of the pain in the former instance should prevent this mistake being made. In some cases sensitive areas will be detected on the chest wall. The neuralgic pains met with in cases of aneurysm radiate up the neck, down the arms, and all over the chest, and Dr. Graham Steell[20] has drawn attention to "the occasional *occipital* pain suffered by patients the subjects of aneurysm of the arch." In two instances he has known this symptom to be so severe as to be the most prominent of the case. In a few cases herpes zoster has occurred. Very severe pain in the chest attends the formation of a dissecting aneurysm. Aneurysmal pain due to pressure is more complained of at night, and is relieved by change of position, whereas the anginal type of pain is worse during the day and aggravated by exertion.[21] Dr. Drummond[22] is of opinion that the position of the aneurysmal sac may frequently be diagnosed from the distribution of the pain, and he has given a diagrammatic explanation of his view on the subject. It has been suggested that the very pronounced effect that large doses of iodide of potassium have in relieving the pain of aneurysm might be of diagnostic value in recognising the aneurysmal origin of the complaint.

II.—*Respiratory Symptoms.*

Dyspnœa is a very common symptom in aneurysm, and it may be brought about by various causes. If accompanied by stridor it is highly suggestive of aneurysm. Two kinds of dyspnœa are to be recognised—i.e., a paroxysmal dyspnœa

and a gradually increasing difficulty of breathing. The former is probably due to implication of the vagi or recurrent laryngeal nerves, or to compression of the small branches of the pneumogastric forming one of the pulmonary plexuses; and the latter to direct pressure upon the trachea or bronchial tubes. Reflex irritation of the vagi will cause most alarming and paroxysmal attacks of dyspnœa due to spasm of the adductors of the vocal cords, precisely resembling attacks of laryngismus stridulus occurring in childhood. As Gairdner [23] long ago pointed out, pressure of an aneurysm upon the pulmonary plexus of nerves may give rise to symptoms very closely resembling those met with in spasmodic asthma. The possibility of the presence of an aneurysm should therefore be borne in mind, in the event of the sudden occurrence of an asthmatic paroxysm in a previously healthy man. Occasionally pressure upon the trachea will give rise to dyspnœa with paroxysmal exacerbations resembling asthma. This is probably due to variations in the fulness of the aneurysmal sac. Dyspnœa may also result from congestion of the lungs, from pressure on the vessels in the root of the lung, or from a dilated condition of the bronchi. Inflammatory changes in the pleura may occur by extension from the aneurysmal sac, and a pleural effusion may be the result, giving rise to persistent dyspnœa. Secondary changes in the heart, as a result of aortic regurgitation, may also cause dyspnœa. The sudden onset of dyspnœa in a patient in whom an aneurysm may or may not have been suspected may be due to the rapid dilatation of some portion of the aneurysmal sac already in contact with the trachea; this may bulge into the air-passage, and may even be recognised by the aid of the laryngoscope.[24] The character of the stridor may be of assistance in deciding as to the cause of the dyspnœa. If stridor be heard only with inspiration, the difficulty of breathing arises from paralytic stenosis of the glottis, whereas if the obstruction be seated in the trachea there will be stridor with both inspiration and expiration.

In November, 1900, I opened a discussion on Aneurysm at the Medical Society of London by reading notes on a case which had been under my observation for nearly five years.

The remarks on cough which I made on that occasion give very much my present view of this symptom : "Too much value should not be attached to any one symptom in the absence of other corroborative symptoms and signs, but I do think that a harsh, brassy cough occurring in a man over 50 with rather rigid vessels, with a history of gouty symptoms, of syphilis, and of hard work, should certainly lead to the careful consideration of the possibility of an aneurysm being the cause. Pressure upon the trachea, bronchus, vagus, or recurrent nerve by any kind of tumour may give rise to a similar cough, but I think that it is of more common occurrence in cases of aneurysm than in the case of other intrathoracic tumours."

In the X ray examination we have an almost certain means of detecting an aneurysm, so that the existence of a cough such as I have just described should lead as a matter of course to the patient being screened. When an aneurysm comes in contact with the trachea or bronchus there is always a tendency to cough, hence this symptom is particularly frequent in aneurysms involving the transverse and descending parts of the arch.

The characteristic cough of aneurysm has been described as ringing, brassy, rough, guttural, or compared to a leopard's growl. The cough may be harsh and dry, or it may be accompanied by a mucous expectoration, sometimes tinged with blood. If bronchiectasis be present the sputa will be purulent. In the early stage of pressure on the recurrent laryngeal nerve, the voice is little if at all altered, but when both the abductor and adductor muscles are paralysed and the cord is in the cadaveric position, the voice has a muffled, hoarse tone, which is readily recognised by the practised ear, so that the paralysis may be suspected as soon as the patient begins to speak. Hæmoptysis is frequently met with in cases of intrathoracic aneurysm—indeed, it may be the first symptom to direct attention to the presence of an aneurysm. It may be very profuse, the patient dying from actual loss of blood or suffocation, or the expectoration may be merely streaked with blood.

The causes of pulmonary hæmorrhage due to aortic aneurysm are of three kinds : (1) Direct—i.e., where there is

a leak from the aneurysmal sac into the air passages; (2) indirect—where the hæmorrhage is the result of pressure; and (3) secondary cardiac disease. The hæmoptysis of direct origin may at first be very slight, merely an oozing of blood into a bronchus or the trachea, the opening being closed by a clot. But there generally comes a time when a profuse gush of blood occurs, causing sudden death. Cases, however, are on record in which after repeated small losses of blood the hæmoptysis has ceased, and yet at the necropsy evidence of direct communication between the aneurysmal sac and the air passages has been found. In the second form—the indirect group—there may be (*a*) pressure on the air passages; (*b*) pressure on the blood-vessels; and (*c*) pressure on the lung substance. Wherever the pressure is exerted the effect is to cause congestion of the tubes or the lungs. It is this congestion which is so often responsible for the irritable and paroxysmal cough of aneurysm, the sputa being frequently blood-tinged. In the third kind—secondary cardiac disease—the hæmoptysis occurs, just as it does in primary heart disease, from engorgement of the pulmonary circulation. One of the most common valvular defects, secondary to aneurysm, is dilatation of the aortic ring with consequent aortic regurgitation. The occurrence, therefore, of hæmoptysis in a patient of the age and under circumstances to justify a diagnosis of aneurysm, should always lead to a careful consideration of the possibility of the hæmorrhage being of aneurysmal origin, especially if a paroxysmal cough be associated with it. Indeed, the appearance of a slightly stained sanguineous expectoration may be the precursor of a more abundant hæmoptysis to be followed by rupture. The expectoration is sometimes rusty, like that met with in pneumonia, or it may resemble the prune-juice expectoration of pulmonary apoplexy.

III.—*Laryngeal Symptoms.*

Probably on account of the fact that I was for many years in charge of the throat department at the Westminster Hospital, and that I have had a large number of throat

patients sent on to me in private, I have been much impressed with the frequent association of paralysis of the left recurrent nerve with intrathoracic aneurysm. In many instances patients have been sent to me by their medical advisers, or have come to me of their own accord, suffering from hoarseness, and on laryngoscopic examination the left vocal cord has been found in the cadaveric position. It must, however, be borne in mind that, in cases of intrathoracic trouble, the physician should not wait for the onset of hoarseness, but that he should carry out a laryngoscopic examination as a matter of routine, having regard to the fact that, in accordance with the law enunciated by Sir Felix Semon, the abductor fibres of the recurrent laryngeal nerve succumb to the effect of pressure or disease before the adductor fibres, consequently the vocal cord, at an early stage of the disease, may be in the position of adduction, and in this event the voice is practically normal.

In an out-patient under my care at the Westminster Hospital,[25] I had the opportunity of observing the changes which occur as the result of pressure on the left recurrent. At first, from commencing paralysis of the abductor muscle, the cord was less vigorous in its movements ; later, all power of abduction ceased, and the cord was found in the middle line ; still later the adductor muscles were implicated and the cord was seen motionless in the cadaveric position. If, therefore, a patient comes complaining of hoarseness, or if there be obscure signs of intrathoracic trouble, the discovery of left recurrent paralysis would be strong evidence in favour of aneurysm, and no other diagnosis should be seriously considered until all the points in favour of aneurysm had been carefully investigated.

I do not know of any reliable figures on an extensive scale as regards the frequency with which laryngeal paralysis is met with in cases of intrathoracic aneurysm. In 35 consecutive cases of aortic aneurysm from my private case-books the recurrent laryngeal nerves were affected in 22 patients. The conditions were as follows : in 2 patients there was impaired movement of the right vocal cord ; in 5 impaired movement, usually abductor paralysis, of the left vocal cord ; in 14 the left vocal cord was seen to be

in the cadaveric position; in 1 case there was bilateral abductor paralysis. In 1 patient, who was too ill to be examined laryngoscopically, the voice strongly suggested recurrent paralysis. In the remaining 12 cases the larynx was not affected. Tansk's[26] experience in the clinic at Pest is very similar. He noted hoarseness in 22 out of 41 cases of aneurysm; paralysis of the recurrent, usually on the left side, was seen in 19 cases; bilateral paralysis was said to be very rare. The number of cases in which I have observed laryngeal palsy in aortic aneurysm is quite out of all proportion to its occurrence in the practice of physicians with a less special experience.

If we except aneurysms of the first part of the arch of the aorta, in which the physical signs are usually quite distinct, in aneurysm affecting the remaining portion of the arch of the aorta there is no physical sign so constant and of so great diagnostic importance as the cadaveric position of the left vocal cord. A case I brought before the Medical Society[27] illustrates this point. At the left apex there was dulness to the second rib, and the resonance was impaired to the third rib; there was also dulness over the left apex posteriorly. There was deficient entry of air over the left lung, but there were no abnormal breath sounds. The apex of the heart was in the nipple line, 3 inches below the nipple. There were no murmurs when the patient was first seen, but later a loud diastolic murmur was audible. The absence of a pulse in the left radial artery was suggestive of aneurysm, and the cadaveric position of the left vocal cord practically clinched the diagnosis. Had this latter condition not been looked for and the condition of the left pulse not recognised, I can readily conceive that a diagnosis of old phthisical mischief at the left apex might have been made; indeed, this very mistake was made by a candidate to whom I had given the patient as a clinical case for examination. The post-mortem examination showed that there was at the junction of the second and third parts of the arch an aneurysm having an external circumference of 9 inches. The sac was lined with laminated fibrin 1¼ inches in thickness. Its outer wall was closely adherent to the apex of the left lung. Both the left pneumo-

gastric and recurrent laryngeal nerves were lost in the wall of the sac.

Paralysis of the left recurrent nerve, then, not only points very clearly to the probability of the existence of an intra-thoracic aneurysm, but it also indicates the position of the aneurysm as being usually seated on the transverse or descending part of the arch of the aorta. While implication of the left recurrent nerve is very commonly present in aortic aneurysm, the right recurrent is much less frequently affected. In 35 consecutive cases from my private practice the right recurrent, as I have already mentioned, was affected only twice as against 19 times for the left nerve. The reason of this great difference depends upon anatomical considerations; the left recurrent nerve turns round under the aortic arch and is therefore particularly liable to pressure; the right recurrent nerve winds round the subclavian artery and is, therefore, situated at a considerable distance from the arch of the aorta. Should, however, the sac of the aneurysm involve the innominate artery, the right recurrent nerve may be implicated, or, as in an extremely interesting case communicated by Dr. Scanes Spicer[28] to the Laryngological Society, the aneurysmal tumour had displaced the lower part of the trachea backwards and to the right in such a way, that the convexity of the deflected trachea pressed on the right recurrent and pneumogastric nerves, causing paralysis of the right vocal cord. Again, as Sir George Johnson[29] suggested, centripetal irritation of the trunk of the left vagus may act on the nervous centre, and through it upon the nerve-supply to the laryngeal muscles on the right side. A case of Sir Anthony Bowlby,[30] in which an aneurysm of the aortic arch compressed the left pneumogastric and recurrent laryngeal nerves and the trachea, and in which there also existed abductor paresis of the right vocal cord, is a good illustration of Johnson's theory, as at the post-mortem examination the right pneumogastric and recurrent laryngeal nerves were found to be quite free from all pressure.

IV.—*Cardiac Symptoms.*

Among circulatory symptoms may be mentioned pain of an anginal character—to this I have already referred—and throbbing, but, as a general rule, it is quite astonishing how little the heart seems to be affected by the presence of an aneurysm unless there be coexisting disease of the aortic valve. When dilatation of the heart occurs, usually in connexion with aortic regurgitation, dyspnœa, palpitation, and irregular action of the heart often result. Pericarditis is a comparatively rare complication of aortic aneurysm; it occurred once in the 98 cases at Westminster Hospital; it may be brought about by pressure of the aneurysmal sac upon, and irritation of, the pericardium. Adhesion between the aneurysmal sac and the pericardium is not uncommon, and death in these cases may result from rupture into the pericardium. The occurrence of rupture is attended by agonising pain, speedily followed as a rule by syncope and death.

V.—*Dysphagia.*

In aneurysms affecting the third part of the arch and the descending aorta the patient may experience difficulty in swallowing, due to pressure of the sac upon the œsophagus. It is very important to bear this in mind before any attempt be made to pass a bougie or other instrument down the œsophagus, otherwise sudden death may occur from rupture of the sac. Owing to the sac ulcerating into the œsophagus the patient may at first vomit small quantities of blood, until at last death takes place suddenly from a gush of blood. Hæmatemesis is, however, not nearly so common as hæmoptysis. Dysphagia may also be of reflex origin from spasm of the pharynx. Pressure on the pneumogastric nerves setting up irritation may give rise to various dyspeptic symptoms, such as vomiting and flatulence. In like manner irritation of the phrenic nerve may cause hiccough.

VI.—*Paralytic Symptoms.*

Paraplegia is a rare complication of aortic aneurysm, but it may occur as a result of erosion of the vertebræ and direct pressure of the aneurysmal sac upon the spinal cord, or it may come on suddenly as a result of pressure by the dislocated vertebræ. A specimen in the Westminster Hospital Museum [31] (No. 696) illustrates the first condition. Here the bodies of the eleventh and twelfth dorsal and first lumbar vertebræ have been almost completely destroyed, together with the intervertebral cartilages. The patient, a man, aged 45, had suffered from severe and paroxysmal pain in the back, followed by wasting and loss of power in the legs. The knee-jerks were absent. The cause of the paraplegia was not diagnosed during life.

A case recorded by Dr. H. G. Turney and Mr. C. A. Ballance [32] is an example of the sudden onset of paraplegia in a man, aged 35, due in all probability to pressure by the dislocated vertebræ. The aneurysm had eroded the sixth dorsal vertebra, and had practically destroyed the seventh. There was considerable destruction of the eighth dorsal vertebra and erosion of the ninth. The difficulty of the recognition of the aneurysmal origin of these cases of paraplegia is very great. Indeed, in these deep-seated aneurysms I fail to see how it is possible to do more than suspect the existence of an aneurysm. As the authors I have just quoted say, "the chances seemed to be in favour of a growth." My own experience would strongly support this view. I have seen quite a number of cases in which persistent pain in the back has eventually proved to be due to malignant disease. Hemiplegia, with or without aphasia, may result from the detachment of a thrombus from the wall of the sac. In one of my private cases left hemiplegia was noted 24 hours before death occurred.

VII.—*General Symptoms.*

The pain and consequent loss of sleep and the general misery caused by an intrathoracic aneurysm usually result in considerable loss of flesh. Emaciation may be more directly brought about by pressure on the œsophagus preventing the entry of food into the stomach ; or an enlarging aneurysm may compress the thoracic duct causing engorgement of the absorbent vessels and glands, and leading to great interference with nutrition. In some cases the loss in weight is rapid.

The sister for many years in charge of the male medical wards at the Westminster Hospital, a nurse of large experience, has pointed out to me that there is something very characteristic in the position an aneurysmal patient assumes in bed. When he is at his ease he will sit up in bed with the knees drawn up, arms forward, shoulders slightly raised, and the head bent forward, and he will say that he breathes more comfortably in this position. This contrasts with the attitude commonly assumed by the advanced cardiac case ; he is usually more comfortable with the legs over the side of the bed and the head reclining on one side of the pillow.

Physical Signs.

It is a trite saying that more mistakes are made from want of looking than from want of knowing, and in no complaint is this more true than in aneurysm. In this disease the indications are frequently so obscure that a very careful and systematic examination is necessary to detect the existence of a deep-seated aneurysm, and in some cases, in spite of the utmost care, no certain diagnosis can be made. These remarks are my apology should the directions for examination of the patient be considered of too elementary a nature by the audience I am addressing. In the first place a very careful inspection should be made of the patient in a good light, so as to notice any lividity of the lips and the condition

of the pupils. Enlarged glands in the neck should be sought for as possible indication of malignant or tuberculous disease rather than aneurysm. Then the state of the pulse should be tested as regards tension, force, frequency, and regularity, and the two pulses compared in order to detect any inequality. The presence or absence of tracheal tugging or any displacement of the larynx should be noted. A careful laryngoscopic examination should be made, and the presence of recurrent paralysis, on the left side especially, should be regarded as presumptive evidence of aneurysm, unless the paralysis can be explained otherwise satisfactorily. The patient's chest should now be bared, and it should be examined while the patient is sitting and in the recumbent position. It is also advisable to look at the patient sideways, as by this means it may be possible to detect a swelling which might otherwise have been overlooked. Any tumour, bulging or flattening of the chest, or defective movement should receive attention. The situation of the impulse of the heart should be looked for and any unusual pulsation noted; these observations should be confirmed by palpation. On inspection any enlargement of the superficial veins can be recognised, and, if present, the direction of the blood current should be determined. Distension of the superficial veins may be brought about by compression of the great veins—i.e., of the superior or inferior cava, or of one of the subclavian veins. Where there is a direct communication between the aneurysmal sac and the superior vena cava cyanosis is very pronounced, often amounting to a purple or black discolouration.

In connexion with pressure on the veins the occurrence of œdema should be mentioned. It is more marked in cases of malignant disease than in aneurysms, but I have seen very extensive œdema resulting from the pressure of an aneurysm. In one of my hospital patients there was very great œdema limited to the left shoulder and arm. In extreme vascular obstruction from the pressure of a large intrathoracic aneurysm, gangrene of the fingers has occurred. Pressure upon the brachial plexus may give rise to wasting almost always confined to one arm. Clubbed fingers have been noticed in some forms of aortic aneurysm. Inspection

should not, therefore, be limited to the chest, but it should also embrace the head and extremities in its purview. In cases in which there has been pressure on the left bronchus, or where there is a pleural effusion, inspection may reveal loss of movement on the affected side.

Palpation affords valuable assistance in diagnosis, but it must be practised very carefully, as by rough handling thrombi may be detached and give rise to cerebral embolism. In aneurysm of the first part of the arch a pulsation may be recognised at a very early period of the disease; indeed, the patient himself may be the first to direct attention to this condition. Very commonly the pulsation has an expansile character. When the aneurysm springs from the transverse or descending parts of the aorta, pulsation is a late sign or may be wanting altogether. At times a pulsation, not otherwise recognisable, may be detected by grasping the chest anteriorly and posteriorly and exercising a certain amount of pressure. Dr. Herbert French [33] points out that in cases in which there is a doubt as to the presence of pulsation the "direct application of the ear to the part in such a way that the pinna is in uniform contact with the patient's skin, will sometimes bring pulsation to the notice very clearly when its amount, appreciable to the membrana tympani, is too slight for the hand to detect; this applies particularly to deep-seated intrathoracic aneurysms."

The absence of pulsation over a dull area arising from a subjacent aneurysm may be owing to the filling up of the sac with clot or simply to a feeble ventricular systole. The diastolic thud or shock, which I shall mention under auscultation, may be recognised on palpation. A thrill is not common in aneurysm and is not of diagnostic importance. When present it is almost always systolic, but Dr. Parkes Weber [34] has exhibited a patient with aneurysm of the first part of the arch of the aorta in whom a diastolic thrill could be demonstrated. Dr. Seymour Taylor [35] has, however, a high opinion of the diagnostic value of thrill. According to him, the existence of a thrill is a fairly conclusive argument that an aneurysm exists. He also regards it as a prognostic sign, as he thinks that the presence of thrill is evidence that the sac has no laminated

clot. On percussion the recognition of a dull area, where normally there should be resonance, is in itself of little value, but, taken in conjunction with other signs, it will materially assist the diagnosis. For instance, should there be dulness in the second right interspace, this would help to exclude disease of the aortic valve or mere dilatation of the aorta, and point to the presence of aneurysm. Less commonly dulness may be detected under the left clavicle. This was the case in a patient to whom I have already alluded. Very exceptionally an aneurysm of the descending thoracic aorta may come to the surface and cause loss of resonance on percussion in the interscapular region. The tricks played on percussion resonance by aneurysmal pressure on the main bronchus are very extraordinary. There may be at one time such distension of the lung with hyper-resonance on percussion as to suggest the existence of pneumothorax, and later, owing to the absorption of the air in the lung, the percussion note may become quite dull.

Various conditions resulting from aneurysm may give rise to dulness, as Dr. Newton Pitt pointed out in his Bradshaw lecture. The most important are collapse of lung, the so-called retention pneumonia, dilatation and filling of the bronchial tubes with secretion, fibroid thickening of the lung, pleurisy, and empyema. A single or a double murmur may be heard over the aneurysm, or there may be no murmur at all. The systolic murmur may be due to changes in the aortic valve or produced by the blood entering the aneurysmal sac. As regards the diastolic murmur, I am convinced that in the great majority of cases, if not always, it is the result of aortic regurgitation, and is not produced at the orifice of the sac. The narrower the opening into the sac from the aorta the louder, as a rule, will be the murmur. Hence, a loud murmur is of rather favourable import, as the narrower the opening into the sac the greater is the probability of cure.

By far the most reliable auscultatory sign is the accentuation of the aortic second sound, sometimes amounting to a distinct thud or shock. In opening a discussion on Aneurysm at the Medical Society of London in 1889 Sir Richard Douglas Powell,[36] in speaking of this sound, said:

"When once heard—and it is doubtless familiar to all here—it cannot be mistaken, and it is, I believe, absolutely diagnostic of sacculated aneurysm. It is, however, by no means heard in all cases of sacculated aneurysm, requiring certain conditions for its production. It is certainly not well heard, some deny that it is heard at all, except in aneurysm of the first or second portion of the arch; and thirdly, the aortic valves must be competent, or but very slightly defective." For the due recognition of this sign a rigid stethoscope is necessary.

Dr. Sansom[37] has pointed out that a systolic murmur, too feeble to be easily recognised in the ordinary manner, may be rendered distinctly audible by listening with a binaural stethoscope, the chest piece of which is placed in the patient's mouth with his lips closed over it. "Tracheal whiff may be heard either in the trachea or in the mouth; it is usually systolic, but may be double, and it is loudest when the patient is expiring slowly after a deep inspiration with the mouth wide open."[38] After careful auscultation over dull areas to detect the presence of murmurs or the diastolic shock, the condition of the lungs should be investigated. Feeble, absent, or stridulous breathing may be noted and bronchial breathing over dull areas, and added sounds due to bronchitis or congestion of the lungs may be heard. The presence of tracheal breathing or stridor, especially if combined with dysphagia, is very suggestive of aneurysmal pressure on trachea and œsophagus. Should one bronchus be compressed there would be diminished entry of air on the corresponding side of the chest, and in extreme cases there may be even entire absence of respiratory murmur over the affected side. If an aneurysm be the cause of pressure the left lung is the one generally affected. This is shown by the fact that out of 140 aneurysms collected from the post-mortem records of Guy's Hospital[39] 30 compressed the left bronchus, and 16 of these eroded into it, while only 5 compressed the right bronchus and none eroded it. Where the calibre of the bronchus is not completely obliterated the breathing may be of a stridulous character. As I shall mention under the head of Diagnosis complete

occlusion of the left bronchus from the pressure of an aneurysm has given rise to signs closely simulating those of pneumothorax. In cases in which aneurysmal pressure has caused retention of secretion in the bronchial tubes moist sounds will be present, especially after coughing. But the whole subject of bronchial obstruction was so ably treated by Dr. Newton Pitt in the Bradshaw lecture for 1910 that it is unnecessary for me to say more on the subject.

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LECTURE II.

Delivered on March 11th.

Mr. President, Fellows, and Gentlemen,—The condition of the radial pulses may afford suggestive information as to the presence of an intrathoracic aneurysm. The variations met with in the pulse are: (1) complete absence of one radial pulse; (2) inequality of the two radial pulses in volume, as felt by the finger or demonstrated by the sphygmomanometer; (3) difference in the character of the sphygmograms; and (4) delay in pulse at one or other wrist.

In spite of the complete absence of the pulse the hand may be quite warm, showing that the blood flows into it as before. The failure of the pulse may be due to disease at the mouth of a vessel arising from the arch; pressure on the subclavian artery by the aneurysmal sac; blocking of the artery by a clot; or lastly, to the effect of the cardiac systole being lost as the blood passes through the sac, much in the way in which the second bag of a spray apparatus causes the intermittent action of the first bag to become continuous. In a patient of mine at the Westminster Hospital, at one period of his illness, there was entire absence of the pulse in the arteries of the left arm. Towards the end of the case, however, a feeble pulse was felt in the left radial, due, I believe, to the fact that as fibrin was deposited in the sac, the wall became more rigid, so that the impulse produced by the ventricular systole was not dissipated in the sac, but acted on the blood.

If the innominate artery be involved in the aneurysm, the right pulse would be affected and become small or imperceptible; if the left subclavian artery, as in aneurysm of the transverse arch, then, of course, it would be the left pulse. But this is not invariable, so that when there is a difference

in the pulse the smaller pulse is not always on the affected side. The inequality of the pulse generally becomes much plainer if the arms be raised, as in testing the pulse for aortic regurgitation.

Dr. Drummond[1] advises that when there is any doubt as to the existence of an inequality of pulses the patient should be directed to inspire deeply, and then slowly to expire to the fullest extent whilst the observation is being made. It will be found that, as a rule, the movements of the chest in respiration affect considerably the size of the smaller radial pulse. A deep inspiration will often abolish the inequality and a forced expiration will exaggerate it. In the majority of cases the inequality is the result of the compression of the artery by the sac, which pressure is relieved by the expansion of the chest in inspiration. This was the explanation of a case recorded by Dr. John Hay.[2] The patient was a man, aged 36, with an aneurysm of the aortic arch. The pulse in the right radial and right carotid arteries disappeared during expiration, especially if the act were exaggerated. This was due to the innominate artery being compressed between the aneurysm behind and the sternum and costal cartilages in front, the lumen of the artery being almost occluded towards the end of expiration.

I have no personal experience of the use of the sphygmograph in the diagnosis of thoracic aneurysm. It will enable a definite record to be kept of the condition of the pulses, and if there be any difference between the two pulses the tracing will give more distinct information than the impression conveyed to the finger. The sphygmograph is an interesting and useful adjunct to diagnosis, but it is not indispensable. Digital examination of the pulses is a far less sensitive method than the use of the sphygmomanometer, so that slight degrees of inequality are frequently overlooked or misinterpreted if the finger alone be employed.

The observations of Dr. O. K. Williamson[3] show that, as a rule, the arterial pressure in thoracic aneurysm is normal or slightly above normal, whereas in mere dilatation of the aorta it is much higher. A slight difference in pressure between the two radials is fairly common to aneurysm and dilatation of the aorta, but a difference of upwards of

30 millimetres is strongly in favour of aneurysm as against dilatation of the aorta. In the differential diagnosis of aneurysm of the aorta from arterio-sclerosis or mediastinal tumour, a difference of pressure between the two sides greater than 20 millimetres affords strong evidence in favour of aneurysm. Apart from other causes, a frequent pulse may indicate tachycardia due to pneumogastric irritation from aneurysmal pressure.

The Pupils.

As Sir William Gairdner [4] has pointed out it is a curious circumstance that in the past so little attention was paid to myosis. The existence of a contracted state of the pupil in cases of aneurysm seems to have eluded the clinical shrewdness of Sir Thomas Watson, as I can find no reference to it in his lectures on the Practice of Physic. Gairdner credits Dr. Walshe with being the first to mention this symptom in connexion with aneurysm. Though Dr. Walshe [5] was the first to record a case of aneurysm with contracted pupil, Dr. MacDonnell, of Montreal, had previously observed a case of malignant tumour producing pressure on the sympathetic with consequent contracted pupil, ptosis, and epistaxis. Gairdner himself published a case of aneurysm projecting into the neck and accompanied by contraction of the pupil on the affected side.

It was not, however, until Dr. John Ogle,[6] in 1858, presented his exhaustive paper, "On the Influence of the Sympathetic on the Eye and its Appendages," that the subject received adequate attention. According to Dr. Ogle pressure upon the cervical sympathetic from aneurysmal and other tumours may result in a *contracted* state of the pupil; but if the pressure were so slight as merely to act as a stimulus to the dilator fibres of the pupil, then a *dilated* state of the pupil was produced. This view was universally accepted up to recent years, but in 1902 it was challenged by Dr. Cecil Wall and Dr. Ainley Walker.[7] They point out that in the majority of cases there is no evidence of implication of that portion of the sympathetic nerve trunk containing pupil-dilator fibres,

and they are of opinion that inequality of pupils, associated with thoracic aneurysm, is usually due to inequality of blood pressure in the ophthalmic arteries, a contracted pupil being associated with high blood pressure and a dilated pupil with low pressure. Though Dr. Wall and Dr. Walker have put forward a very ingenious explanation of the pupillary changes seen in thoracic aneurysm, they do not appear to have met all the arguments in favour of the inequality being due to sympathetic influence. That the cervical sympathetic is implicated in some cases is shown by the fact that, in association with a contracted pupil, there are to be noted drooping of the eyelid, recession of the eyeball, and an absence of sweating and flushing on the affected side.

As regards the frequency of alterations in the pupils I may quote Dr. Drummond's[8] figures: "In 87 cases in which the point was noted, they were affected in 34 and equal in 53; in 13 the right was dilated, in 9 the left contracted, in 7 the right contracted, and in 5 the left was dilated." Very little stress should be placed upon a mere inequality of the pupils in the diagnosis of an intrathoracic aneurysm, but a permanent unilateral myosis, with other evidence of involvement of the cervical sympathetic, should certainly receive due attention. As an aneurysm is so frequently met with in syphilitic subjects, it may be associated with tabes, and in this event the patient may naturally present the eye-reactions characteristic of this disease.

Tracheal Tugging.

Tracheal tugging was first described by Surgeon-Major Oliver,[9] but it received little attention in this country until the question was taken up by Dr. MacDonnell,[10] of Montreal. The directions given by Surgeon-Major Oliver for eliciting this sign are as follows. "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to the fullest extent; then grasp the cricoid cartilage between the finger and thumb, and use gentle upward pressure on it, when, if dilatation or aneurysm exist, the pulsation of the aorta will be distinctly felt transmitted

through the trachea to the hand. The act of examination will increase laryngeal distress should this accompany the disease."

Dr. MacDonnell points out, and gives cases in illustration, that tracheal tugging may be present when many other physical signs and symptoms are absent, that the sign is limited to aneurysms involving the transverse arch, and that direct pressure upon the trachea does not cause tracheal tugging. He maintains that "tracheal tugging is a very early sign in the history of the case. In all the cases in which it was present it was detected on admission." In some cases the downward tug of the aneurysm upon the larynx is so pronounced as to be visible. On the other hand, in one case the trachea and larynx, instead of being dragged down with each heart beat, were pushed upwards in sudden oscillations; the suggestion being that this was due to aneurysm arising from the convexity of the arch of the aorta.[11]

Though the existence of tracheal tugging is almost diagnostic of aneurysm, it may be caused very occasionally by anything which brings the aorta into abnormally close connexion with the left bronchus. Hence, it has been met with in cases of new growth occurring in this position. Fibrosis of the lung and pleural adhesions to the chest wall have also given rise to it. This sign cannot, therefore, be regarded as absolutely diagnostic of aortic aneurysm, but its absence in a case where the other symptoms and signs suggest the presence of a tumour in contact with the trachea and left bronchus should lead to the exclusion of an aneurysm as the cause of the pressure.

M. Boinet[12] has described a new sign occasionally met with in aneurysm of the arch of the aorta, "signe du larynx," the larynx being drawn downwards, backwards, and to the left, and being immobile. Attention should also be directed to any lateral displacement of the trachea as indicative of aneurysmal pressure.

Roentgen Rays.

In no department of medicine has the X ray method of examination done greater service than in the detection of intrathoracic aneurysm. It is not too much to say that the diagnosis of aortic aneurysm has been completely revolutionised since we have been enabled to throw a flood of light upon the contents of the thorax. It is now possible to detect many an aneurysm which would formerly have escaped observation, and we are also able by the X rays to exclude conditions such as pulmonary tuberculosis and new growths, which in the past frequently presented features of great difficulty in regard to differential diagnosis from aneurysm.

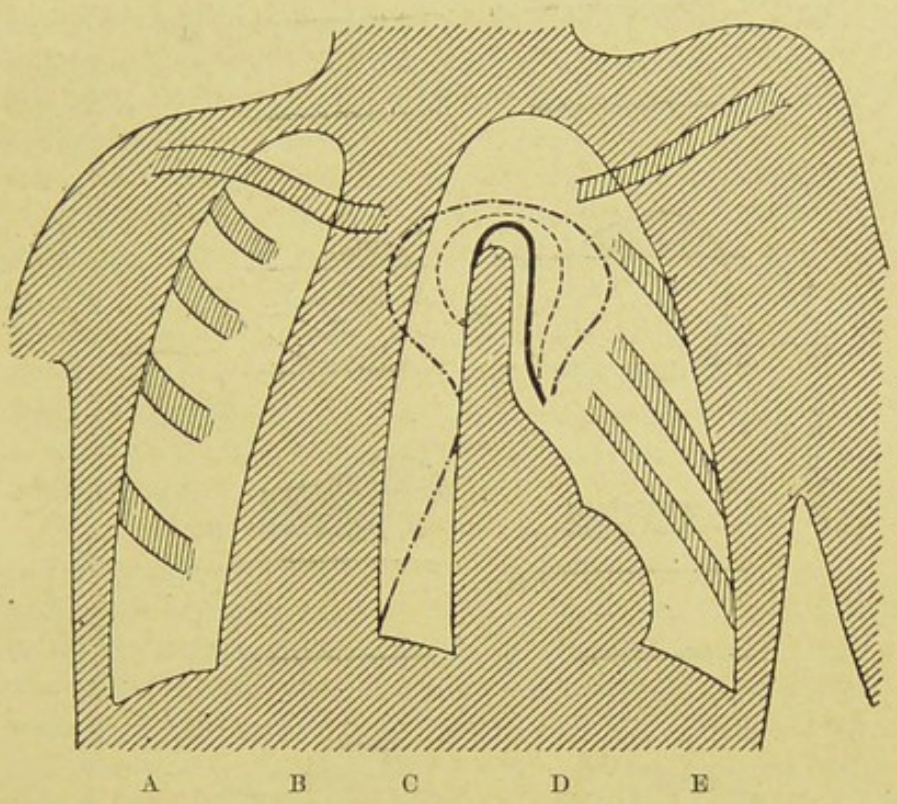
Thanks to the kind assistance of Dr. Hugh Walsham, I am able to show to you negatives of aneurysms which illustrate points in diagnosis. These are of great interest, but all radiographers are agreed that the screen examination gives more valuable information than can be obtained from an inspection of a skiagram.

To obtain a reliable result in any given case the anterior, the posterior, and the right anterior oblique examination must be employed. In order to demonstrate the appearances found in a right anterior oblique examination I would direct your attention to the diagram No. 1, which Professor Holzknecht has kindly allowed me to use.[13]

Diagram No. II. is also after Holzknecht. "No. 1 represents the normal aorta in the antero-posterior examination; the parallel lines represent the central opacity, and the shaded portion the portion of the aorta which projects beyond this central opacity—namely, the left lateral aortic bulge. In No. 2 the parallel lines represent the vertebral shadow, the shaded part the normal aorta seen in the right anterior oblique examination. Nos. 3 and 4 represent the appearances which may obtain in the case of a generally dilated aorta; Nos. 5 and 6 the appearance seen in aneurysm; Nos. 7 and 8 a small aneurysm springing from the under surface of the arch, which could not be detected

by X ray examination either in the anterior, or posterior, or right lateral oblique examination, the appearances presented being practically indistinguishable from those seen in Nos. 1 and 2, the normal aorta."

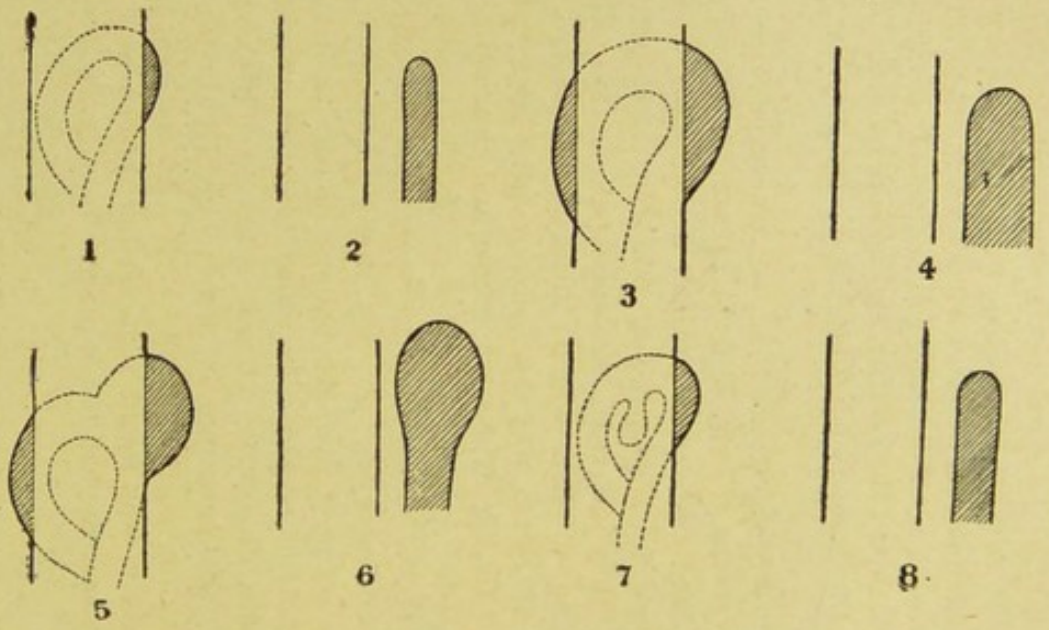
DIAGRAM I.



A. Clear area corresponding to right lung. B. Shadow of vertebral column. C. Clear middle space. D. Shadow of normal heart and aorta. E. Clear area corresponding to left lung; ——— dilated aorta; - - - - - - small commencing aneurysm; —.—.—., upper part, larger aneurysm; ——.—.—., lower part, position of dilated auricle.

As just pointed out, a small aneurysm in the concavity of the arch of the aorta may escape recognition by either the anterior or oblique method of examination. In nearly all other varieties of aneurysm an X ray examination will give

DIAGRAM II.



X ray appearances obtaining in the case of the normal and dilated aorta and of aortic aneurysms. (After Holzknecht.)

decisive information. Fluid blood is very nearly transparent to X rays, but clotted blood is comparatively opaque to the rays, so that if a dense shadow is cast by the aneurysm we can reasonably infer that deposition of laminated clot has taken place in the sac.

Fig. 1 is from a patient who was sent to me, as his symptoms were very suggestive of a thoracic aneurysm. Physical examination, however, gave no support to this view. To clear up any doubt on the subject I requested Dr. Walsham to X ray the patient, and as will be seen from the skiagram, the aorta is considerably dilated, but there is no aneurysm.

Fig. 2 is a good example of an aneurysm affecting the ascending portion of the arch of the aorta; in Fig. 3 the ascending and transverse portions are involved. Figs. 4 and 5 show an aneurysm of the transverse portion of the arch; they are from the same patient, and illustrate the increase in size which aneurysms undergo. The skiagram of No. 5 was taken five months later than that of No. 4. In Fig. 6 the transverse and descending aorta is implicated.

Fig. 7 is the skiagram of a man aged 55. This was evidently an aneurysm of very rapid development, as a few weeks before I saw the patient he had been examined by a very able physician, who failed to detect an aneurysm. The patient died two months after his first interview with me. The aneurysm is seen to involve the transverse and descending portion of the arch. At the post-mortem examination it was found that further development of the aneurysmal process had taken place.

Modes of Termination.

In speaking of the mode of termination of intrathoracic aneurysm I must mention the possibility of cure, though, as I have already stated, this termination is seldom met with. In the rare cases in which cure of an aneurysm takes place this is effected in one of two ways: either by the deposition of laminated fibrin in the interior of the sac, or by the outer

coating of the sac getting more and more supported externally, by fibrous change taking place in the tissues around the aneurysm.

That a cure, either spontaneous or after treatment, of a large thoracic aneurysm does take place is proved by the number of instances in which, at a post-mortem examination, an aneurysm has been found filled with firm decolourised clot. I need only cite two instances which have appeared in THE LANCET recently. 1. A patient who had been under Mr. Tufnell[15] for aortic aneurysm died a violent death. The aneurysm was found filled with solid clot. 2. Sir Thomas Oliver[16] has recorded a case of an aneurysm arising about one inch above the aortic valve, and extending almost to the margin of the left carotid and subclavian, which was found to be filled with firm decolourised clot—the man had died of renal disease.

The extreme difficulty of estimating the effect of treatment is illustrated by a case which I showed at the discussion on aneurysm at the Medical Society in December, 1889.[17] I then claimed that the improvement in the symptoms and physical signs, which the patient exhibited, was due to some amount of consolidation having taken place in the sac, but at the post-mortem examination, six months later, the wall of the aneurysm was found to be very thin and there was no laminated fibrin.[18]

A fatal termination may be brought about in the following ways:—

I. Rupture: (*a*) Internal—(1) into serous membranes; (2) into mucous membranes; (3) into blood-vessels; (4) into the subcutaneous tissue. (*b*) External.

II. Pressure upon trachea.

III. Pressure upon œsophagus.

IV. Exhaustion due to long-continued suffering, or arising from repeated small losses of blood.

V. Heart failure—frequently in connexion with aortic regurgitation.

VI. Anginal attacks.

VII. Cerebral embolism.

VIII. Pulmonary tuberculosis and other complications.

Fig. 1.

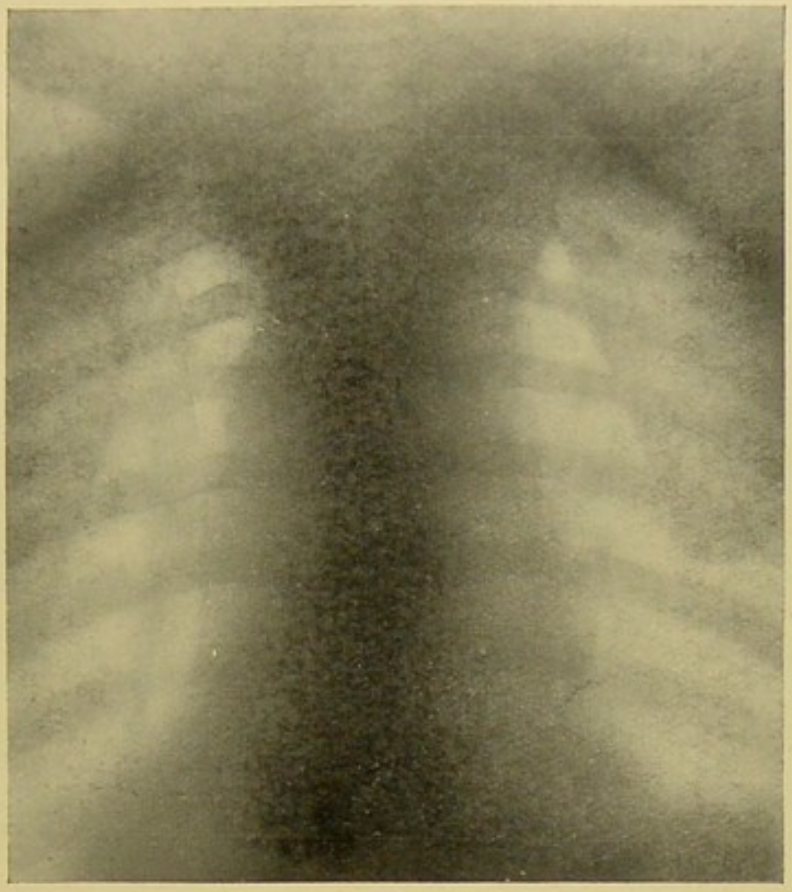


Fig. 2.

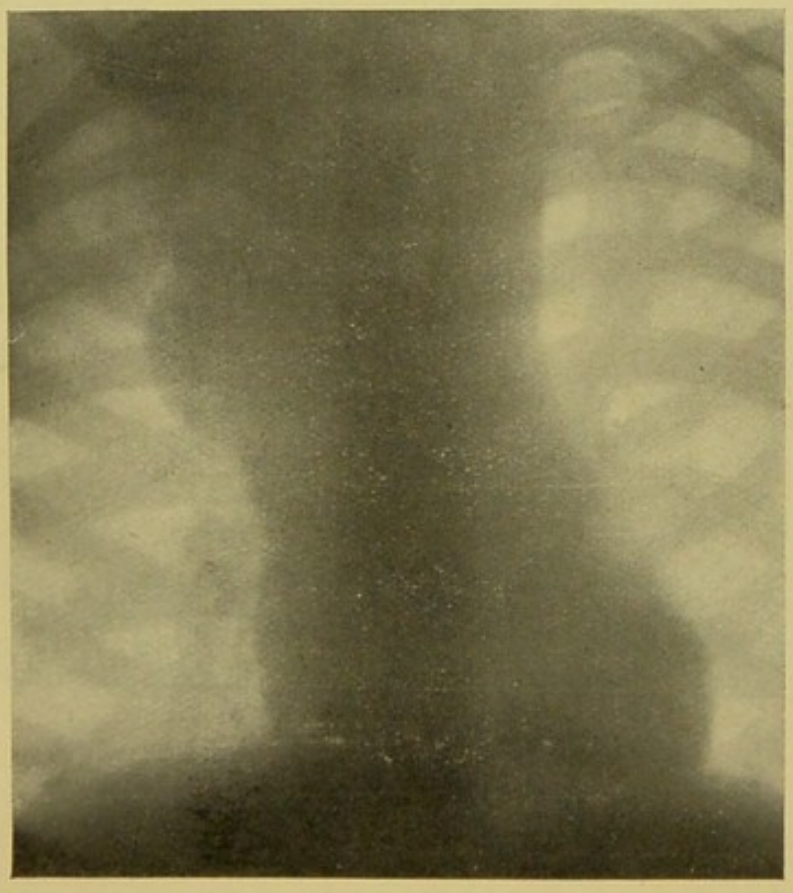


FIG. 3.

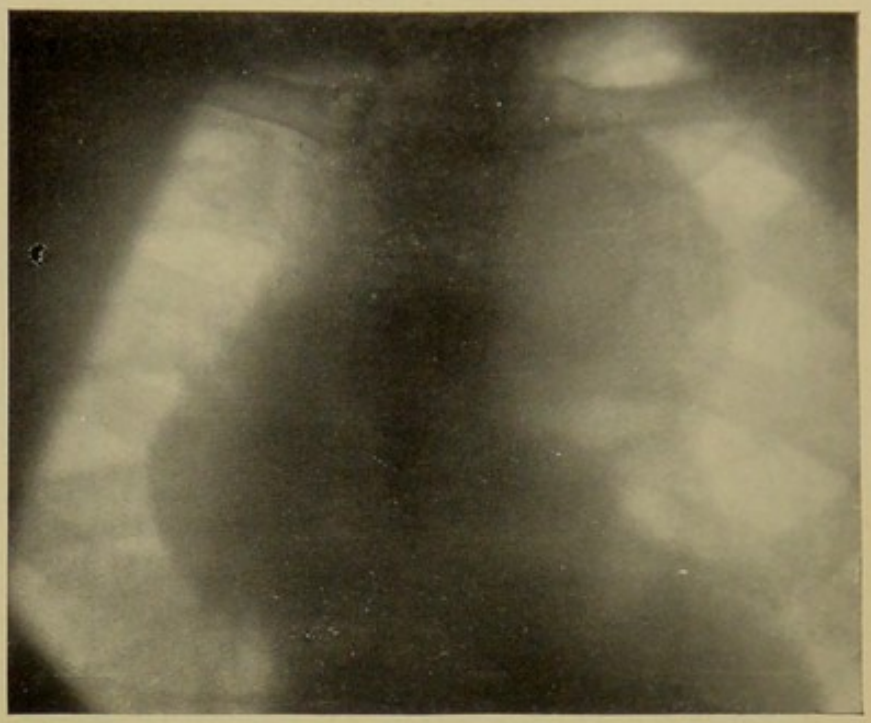


FIG. 4.

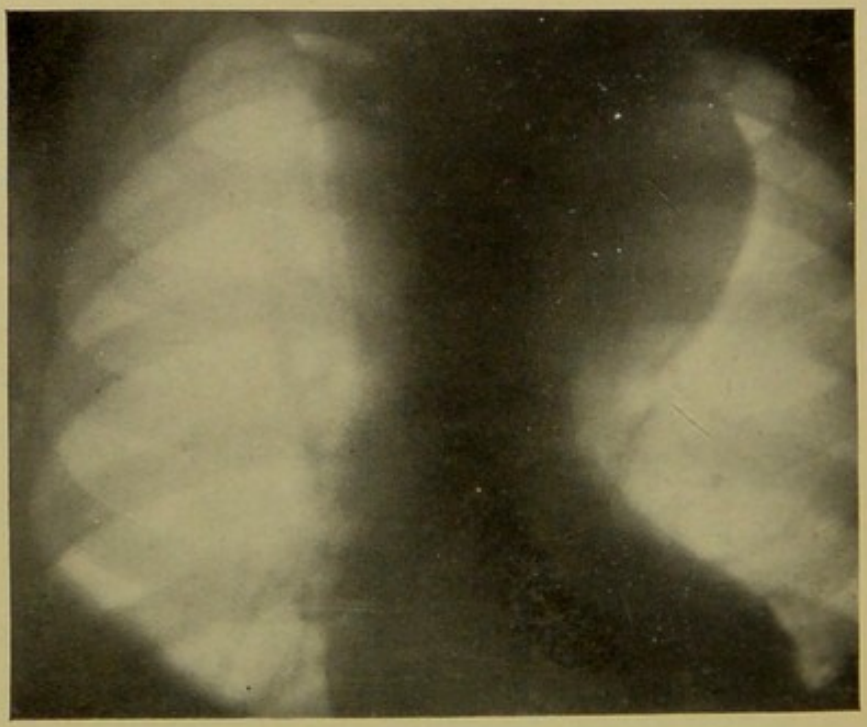


FIG. 5.

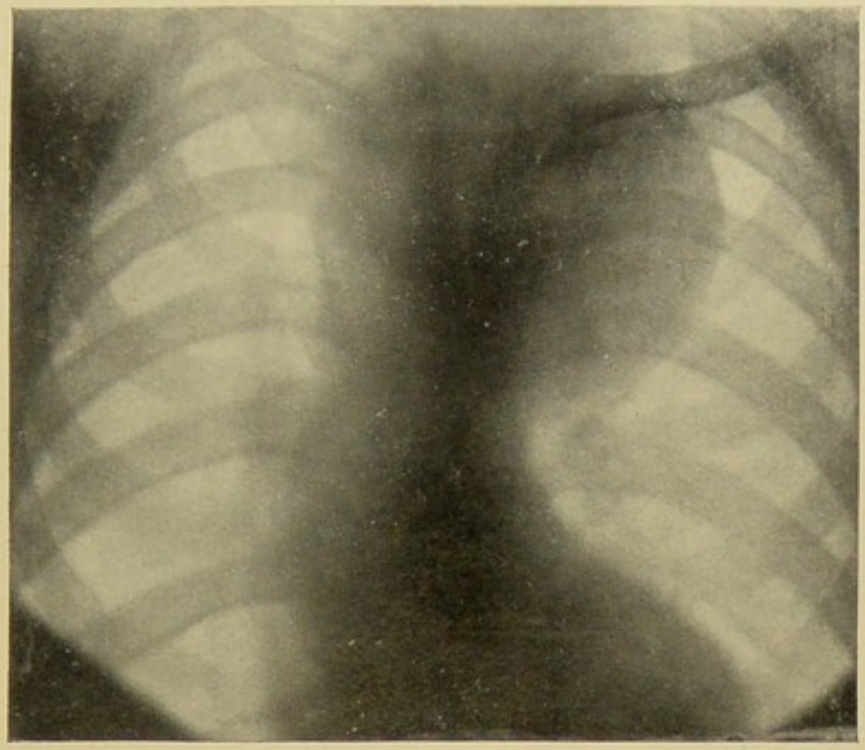


FIG. 6.

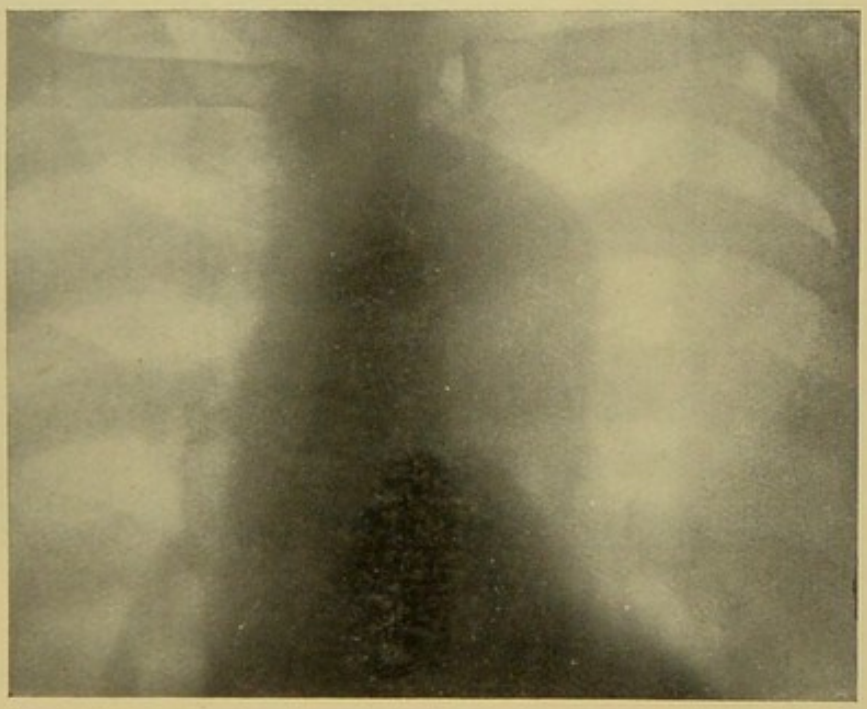


FIG. 7.

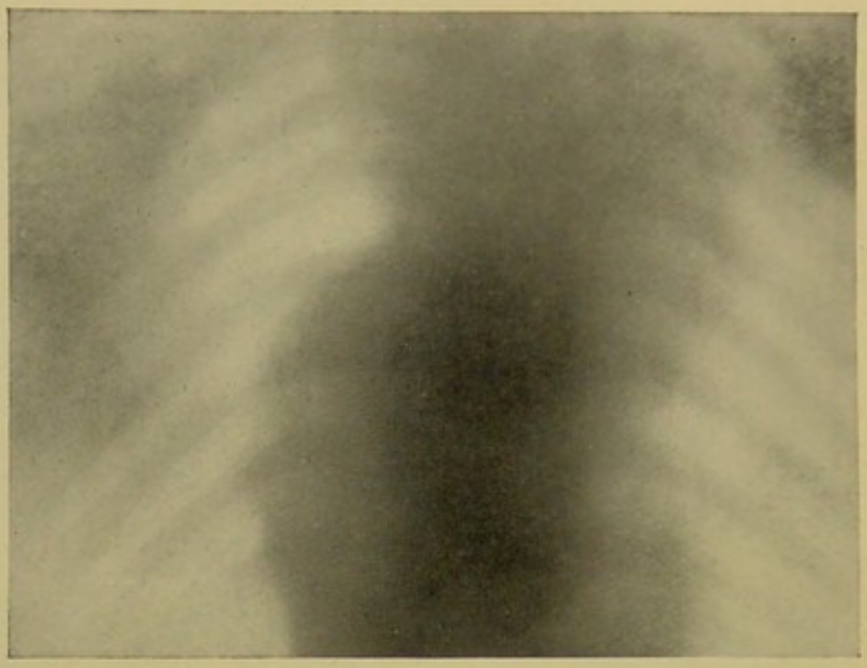
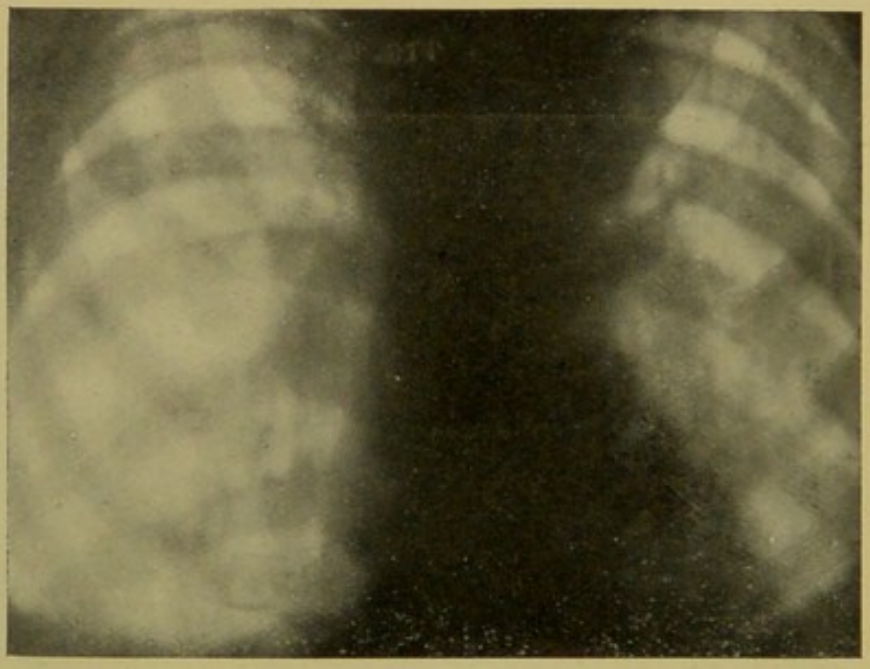


FIG. 8.



The causes of death, in cases submitted to post-mortem examination at the Westminster Hospital, are as follows (98 cases):—

Rupture	41	Pneumonia and broncho-pneumonia	7
Œdema of larynx	1	Other causes (tuberculosis, gangrene of lung, pericarditis, cancer of stomach) ...	4
Pressure on trachea	1		
Cardiac failure	44		

Modes of termination of private patients (35 cases):—

Rupture	6	Cerebral embolism	1
Died suddenly (? rupture) ...	6	Cause of death not stated ...	4
Pressure on trachea	4	Lost sight of	2
Lung affections	6	Alive and well	2
Exhaustion and cardiac failure	4		

From Dr. Oswald Browne's statistics it appears that out of 58 aneurysms affecting the ascending part of the arch the disease terminated by *rupture* in 18 cases; in 9 instances into the pericardium, in 4 into the right pleural cavity, in 2 cases the aneurysm ruptured externally, and once into the right lung, pulmonary artery, and left pleural cavity respectively. Out of 35 aneurysms affecting the transverse portion the disease terminated by rupture in 12 cases. Three times into the left pleural cavity and into the left bronchus respectively, twice each into the trachea and into the pericardium, and once each into the œsophagus and externally. Out of the 19 instances of aneurysms affecting both the ascending and transverse portions of the arch the disease terminated by rupture in 4 instances—once each into the right lung, right pleural cavity, left pleural cavity, and trachea.

In the third or descending portion of the arch there were 21 cases, and rupture occurred in 16 instances. In 8 cases into the left pleural cavity, in 3 into the œsophagus, 3 into the left bronchus, and once into the right pleural cavity and right lung respectively. In the descending thoracic aorta there were 17 cases, and rupture occurred in 14. In 7 cases into the left pleural cavity, twice into the right pleural cavity, thrice into the œsophagus, once into the left bronchus, and

once into the subserous connective tissue lying to the right of the spinal column.

From the figures I have given it will be seen that the most common termination of aneurysms of the descending part of the arch and of the descending thoracic aorta is rupture into the left pleural cavity; this occurred 15 times out of 38 cases of aneurysm.

Dr. Oswald Browne's Statistics.

—	Ascending portion of arch.	Transverse portion.	Ascending and transverse portion.	Descending portion of arch.	Descending thoracic aorta.
Pericardium	9	2	—	—	—
Right pleural cavity	4	—	1	1	2
Externally	2	1	—	—	—
Right lung	1	—	1	1	—
Left pleural cavity	1	3	1	8	7
Pulmonary artery	1	—	—	—	—
Left bronchus	—	3	—	3	1
Trachea	—	2	1	—	—
Œsophagus	—	1	—	3	3
Subserous tissue	—	—	—	—	1
Total number of cases of rupture	18	12	4	16	14
Total number of cases of aneurysm	58	35	19	21	17

From this table we get the remarkable figures that out of 112 aneurysms affecting the first two portions of the thoracic aorta rupture occurred 34 times, whereas in 38 cases of aneurysm of the two remaining portions of the aorta, rupture occurred 30 times—i.e., more than three times as frequent.

Summing up Dr. Oswald Browne's figures, and arranging them in order of frequency, they come out as follows. They

may be compared with cases reported by Dr. Kelynack [19] and with the Westminster Hospital series.

	Dr. Oswald Browne.	Dr. Kelynack.	Westminster Hospital.
Into left pleural cavity... ...	20	7	5
,, pericardium	11	13	9
,, right pleural cavity ...	8	1	5
,, left bronchus	7	—	5
,, œsophagus	7	3	6
,, trachea	3	2	2
,, right lung	3	—	2
Externally...	3	3	2
Into pulmonary artery	1	—	1
,, subserous tissue	1	—	—
,, left lung	—	1	1
,, right bronchus	—	1	—
,, superior vena cava ...	—	1	—
,, left auricle	—	—	2
,, lung	—	—	1
Total	64	32	41

Combining Dr. Oswald Browne's cases with those from the Westminster Hospital it will be found that rupture occurred in rather over 42 per cent. of the cases submitted to a post-mortem examination.

The difference in the way in which an aneurysm ruptures into a serous cavity compared with rupture through a mucous membrane is very remarkable. When the rupture takes place into a serous cavity, as the pleural cavity or the pericardium, there is almost always a considerable rent and death occurs speedily, often, indeed, almost instantaneously. After this manner died a medical man, who was under my care in the Westminster Hospital. He was talking to the night nurse, being apparently quite comfortable, but before she reached the end of the ward he was dead. At the necropsy a rupture into the pericardium was found. In another case, where rupture took place into the pleural cavity, the patient cried out for help, but before the nurse could reach the bedside he was dead.

On the other hand, note what often occurs when an aneurysm bursts into the trachea ; there is a little gush of

blood, followed by cessation of the bleeding, then another gush, and perhaps one or two more, finally the patient may be choked by a great gush of blood. In some cases of rupture, especially into a closed space such as the mediastinum or subserous tissue, there may be acute blanching with coma, and not syncope, preceding further loss of blood and death.

Though rupture of a thoracic aneurysm is usually immediately fatal, a few exceptional cases have been recorded. A notable example is that of the celebrated surgeon, Liston, who had a considerable hæmorrhage, suggestive of rupture of an aneurysmal sac. He died five months later, but not from hæmorrhage, though there had been slight discharges of blood in the interval. At the necropsy the trachea was found to be perforated in three or four places and portions of clot were discovered projecting through the openings from the sac, partially blocking them, and so preventing further severe hæmorrhage.

Sir Felix Semon [20] has recorded a case of aneurysm of the thoracic aorta with repeated perforations of the œsophagus. Even rupture into the pericardium, in which instantaneous death is the rule, is sometimes compatible with life for a time. This is shown by a case reported by Sir Samuel Wilks,[21] in which the opening was closed by inflammation, and the patient lived exactly a month after the onset of the pericardial symptoms.

Rupture into the superior vena cava is a comparatively rare mode of termination. The combined figures of Dr. Oswald Browne, Dr. Kelynack, and the Westminster Hospital give 138 cases of rupture of an intrathoracic aneurysm, and there is only one example of rupture into the vena cava. Death usually occurs very speedily, but Sir William Gairdner [22] reported a case which lasted eight days; Dr. Syers's [23] case lived a month; but the most remarkable of all is a case reported by Dr. Laurence Humphry.[24] In this instance the patient lived for nearly 18 months after the onset of the urgent symptoms, developing large veins over the chest with a downward current.

A rare mode of termination is by rupture of the sac into the subcutaneous tissue of the chest wall. Such a case has

been described by Dr. Raussier and Dr. Houel.[25] A woman, 60 years of age, had a large pulsatile tumour in the upper and anterior part of the right chest. The aneurysm gave way suddenly into the subcutaneous tissue after attempt to cough; the patient experienced a very painful tearing sensation, and the tumour increased in size. Next day the swelling extended into the axilla and there were severe attacks of pain in the branches of the brachial plexus. The patient died three days after the rupture, apparently in consequence of the escape of the large quantity of blood into the cellular tissue. At the necropsy it was found that the aneurysm had developed from the ascending aorta.

As a rule, rupture of an aneurysm externally—i.e., through the skin—is almost invariably attended by sudden death, but, as in a case mentioned by Gairdner,[26] the external hæmorrhage may be arrested. The patient ruptured an aortic aneurysm externally opposite the cartilage of the third rib. He lost so much blood that he fainted; then the bleeding stopped; he lived for four months without any recurrence, and died from typhus fever.

Dr. Oswald Browne's tables illustrate the tendency of aneurysms of the transverse arch of the aorta to exert direct pressure on the trachea, as this effect was noted in 13 out of 35 cases of aneurysms of this part of the aorta, and in 15 death arose from asphyxiation due to increasing pressure on the trachea. Among my own cases there are four which I think may be attributed to direct pressure on the trachea. In two cases my opinion was especially asked as regards the question of tracheotomy, and the correctness of the diagnosis was confirmed by a post-mortem examination. In respect to the other two cases, in one the presence of an aneurysm of the transverse arch was verified by an X ray examination, but in the remaining case the evidence is less convincing. In all four cases death took place from asphyxia; in one case death was preceded by epileptiform convulsions.

The difficulty of diagnosing aneurysmal pressure upon the œsophagus is very great, inasmuch as the pressure is usually effected by a sac springing from the descending part of the arch, or from the descending thoracic aorta. In these situations the aneurysm is deep-seated, and the physical

signs are difficult to make out. Take, for example, the case of a lady aged 63, who was sent to me by her medical adviser on account of difficulty of swallowing (and particularly as regards solids), with loss of flesh. On making a laryngoscopic examination I found loss of movement in the right vocal cord. I could not, however, detect any physical signs of aneurysm. The patient died from hæmatemesis. If the vocal cord paralysis had been left-sided I would certainly have made the diagnosis of aneurysm, but as it was I am doubtful. This is a type of a class of cases I have frequently seen, and in the absence of an X ray examination or a necropsy it is often impossible to say whether malignant disease or aneurysm was the cause of death. Skiagram No. 8 is an example of malignant disease of the œsophagus, which was mistaken for an aneurysm until the X ray examination was made.

I have come across several cases in which death has resulted from the exhaustion due to the long-continued suffering which so often accompanies erosion of the vertebræ. The first case in my list of private patients was an example of this sort, as was also the case I brought before the Medical Society of London in December, 1900. This I regard as a fairly common cause of death in aneurysm. I have not met with death resulting from repeated small losses of blood.

In four of my cases death was attributed to exhaustion and cardiac failure. Out of 159 cases collected by Dr. Drummond,[27] in which the cause of death was determined, in 27 the fatal result was due to valvular disease. Where aortic regurgitation exists death is due rather to degeneration of the heart's substance consequent upon the valvular defect than to the aneurysm itself.

I have no doubt, in my own mind, that some of the deaths which have been ascribed to angina pectoris are in reality due to anginal attacks occurring in patients with an aneurysm which has not been diagnosed. One case of the sort, which occurred upwards of 30 years ago, has remained impressed upon my memory. The patient, aged 40, was an officer in the Life Guards, who was brought to me by his usual medical attendant. Though the physical signs were

not conclusive as to the presence of an aneurysm, I have little doubt that one existed, and that the awful attacks of angina from which the patient suffered were due to the aneurysm. This was the first case to suggest to me the possibility of severe angina pectoris being caused by an aneurysm. I have seen other cases which would justify the same conclusion. I would therefore venture to suggest that in every case in which the patient complains of anginal pains the question of the presence of an aortic aneurysm, as the underlying cause, should be carefully considered.

One of my patients died as a result of cerebral embolism giving rise to hemiplegia, and in the case of another it is stated that the patient died with hemiplegia and lung trouble, so that it is quite possible that the hemiplegia and lung trouble were both of embolic origin.

Curiously enough, lung conditions causing death seem to have been usually frequent among my cases. For instance, there is a death from bronchitis, three from pneumonia (one case being of a septic nature), and, finally, a death from influenza complicated with pleural effusion. One case, which I have not included in my list of aneurysms, died from phthisis. I myself think that this was a case of pulmonary tuberculosis supervening upon an aneurysm, but unfortunately it occurred before the days of X rays. It illustrates the difficulty, to which I shall allude later on, in diagnosing aneurysm where there is disease of the lungs.

Diagnosis.

To the careful and experienced physician, as well as to the neophyte, there comes at times a rude awakening, and he finds, greatly to his chagrin and regret, that he has failed to make a correct diagnosis. In this connexion two cases of aneurysm come to my mind.

In the first case I was saved from making a gross blunder by examining the patient laryngoscopically. At the end of a busy morning's work a medical man came into my consulting-room, saying that he had caught cold and had a cough. I was about to order a simple cough mixture and

to ask him to call again, when I recognised that he was hoarse. A laryngoscopic examination showed the left vocal cord in the cadaveric position, and a pulsation under the left clavicle convinced me of the presence of an aneurysm. Knowing that he was a locum-tenent out of work I advised his admission into Westminster Hospital. He came in two days later, and on the night of admission, while talking to the nurse, he was seized with acute pain in the chest and died within a few seconds. The necropsy showed rupture of a large aneurysm into the pericardium, and yet prior to his visit to me the patient was doing his ordinary work quite unconscious that he was seriously ill.

The fatal termination of the second case came to me as a shock at the time, but subsequent consideration has convinced me that the diagnosis of an aneurysm in this particular case was almost impossible. I doubt whether even an X ray examination would have revealed the true condition of affairs.

A stout-built, healthy-looking man, aged 46, a solicitor, consulted me on account of a swollen condition of his face. He stated that the swelling was first noticed the morning following a heavy dinner, the eyelids being especially affected. Physical examination of the chest revealed nothing amiss. The urine contained a trace of albumin. From the patient's appearance and the account he gave of himself I came to the conclusion that he lived too well and that his kidneys were affected. He paid me a second visit five days later. I heard nothing more of the patient until 11 days after his last visit, when I received a telephone message from the coroner to say that while playing at bridge the patient suddenly fell off his chair and expired at once. Death was found to be due to the rupture into the pericardial cavity of an aneurysm seated 2 inches above the aortic valve, the pericardium being distended with blood clot. A puffy or swollen condition of the face is not unfrequently present in cases of aortic aneurysm, but apart from this symptom there was nothing to direct my attention to the state of the patient's aorta.

The rupture into the pericardial cavity of an aneurysm, originating from that portion of the aorta covered by the

pericardium, is a possibility that should be borne in mind in cases of sudden death occurring without any distinct indication as to its cause.

In contra-distinction to these deep-seated aneurysms we may meet with aneurysms coming to the surface, so as to be clearly recognisable by sight and touch, and yet not giving rise to any symptoms. Sir B. W. Richardson[28] has recorded such a case. The patient had a tumour the size and shape of half a moderate-sized orange just above the right breast; the skin over it was tense and of a bright red colour. Except for the tumour the patient had not any unhealthy symptoms. At the necropsy, two months later, an aortic innominate aneurysm was found. The aneurysmal sac may sometimes attain an enormous size and compress the lung in front of it, and yet give rise to hardly any of the characteristic physical signs of aneurysm, so that there may be great obscurity in diagnosis.

A small aneurysm on the transverse arch of the aorta may easily escape recognition. A ringing cough may be the only symptom. Tumour, murmur, pain, dyspnœa, and other evidences of intrathoracic pressure may be absent, and the pupils, pulses, and vocal cords may afford no help in the diagnosis.

My predecessor in this chair, Dr. Percy Kidd, has drawn attention to the association of aneurysm with tuberculosis. He reported five cases in which this combination was found post mortem. In three no signs of phthisis were noted during life; one died in the out-patient department before being examined and the remaining case was only one day in the hospital. This report should emphasize the importance of carefully investigating all the organs of the body, and of not allowing the diagnosis of aneurysm to prevent the recognition of other ailments. On the other hand, I would point out that pressure of an aneurysm on the root of the lung may result in retention of secretion in the bronchi, thus giving rise to bronchiectasis, the symptoms and physical signs of which may, unless great care be taken, cause an erroneous diagnosis of pulmonary consumption to be made.

I have already mentioned a case in which impairment of resonance at the left apex, really due to an aneurysm, was

regarded as evidence of pulmonary tuberculosis. In cases of this sort the presence of a few râles and hæmoptysis may increase the difficulty of diagnosis. The absence of tubercle bacilli from the sputum should awaken suspicion that the disease is not tuberculosis, and an X ray examination would almost certainly settle the diagnosis. Skiagram No. 9 is taken from a patient in whom the presence of an aneurysm was suspected, but the X rays showed clearly that pulmonary tuberculosis was the cause of the trouble.

There can be, I think, no doubt that some of the cases of death from profuse hæmoptysis, which have been attributed to phthisis, were really due to the rupture of an aneurysmal sac into a bronchus. On the other hand, retraction of the lung from old phthisis, usually on the left side, may leave the vessels of the heart uncovered, and the pulsation of the exposed vessel may simulate an aneurysm. The pressure of an aneurysm upon the pulmonary branches of the vagus may give rise to paroxysmal attacks of dyspnœa, which, without the aid of X rays, might be difficult to distinguish from spasmodic asthma.

An empyema may be the result of extension of the septic mischief from the bronchi to the pleura in a case of aneurysm. In these cases too rapid withdrawal of the fluid has been followed by rupture of the sac of the aneurysm. A pulsating empyema may very closely resemble an aneurysm. At the commencement of my medical career a friend of mine had a pulsating tumour of the chest wall. He was seen by some of the most distinguished men of the day, but there was no consensus of opinion, some maintaining that the tumour was aneurysmal, others that it was an empyema, and one surgeon went so far as to say that he was prepared to incise it then and there. The patient died suddenly, but unfortunately a post-mortem examination was not permitted.

The existence of advanced emphysema by masking the dulness of the aneurysmal sac will sometimes render the diagnosis difficult. This occurred in one of my patients. Fortunately in this case the recognition of the cadaveric position of the left vocal cord gave the clue to the diagnosis, otherwise I am afraid that there would have been great difficulty in arriving at the true state of affairs. The skiagram

FIG. 9.

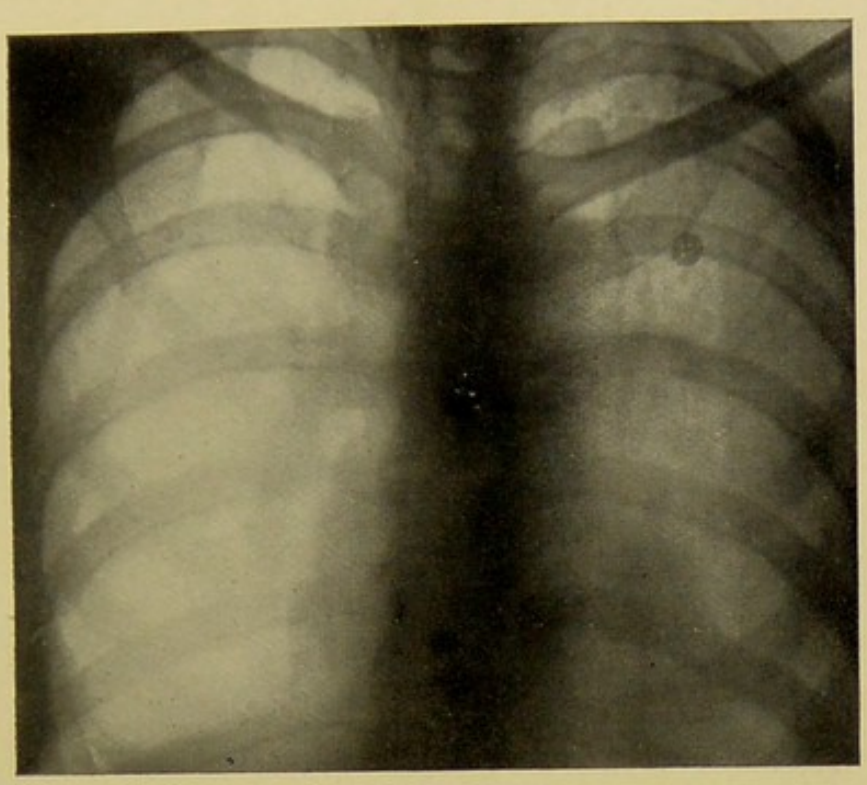


FIG. 10.

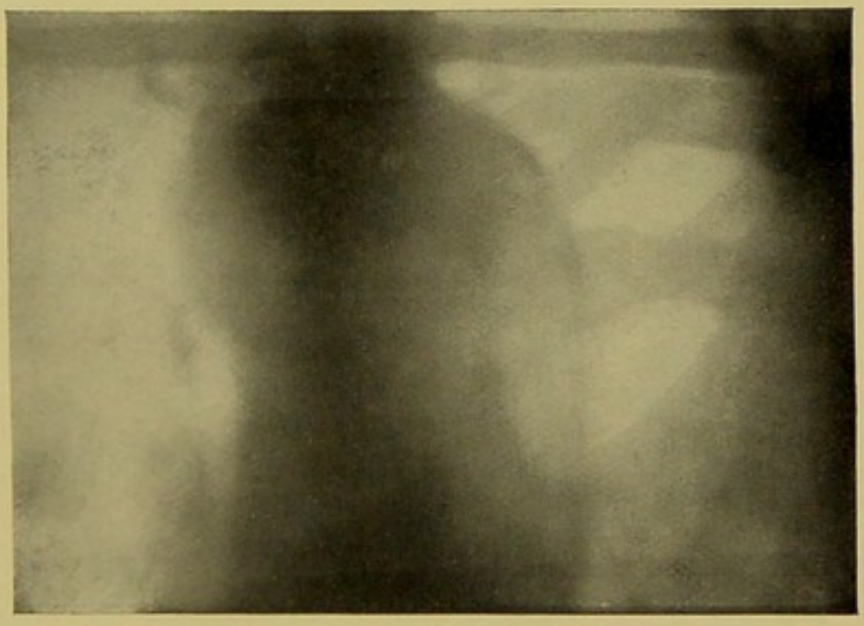


FIG. 11.

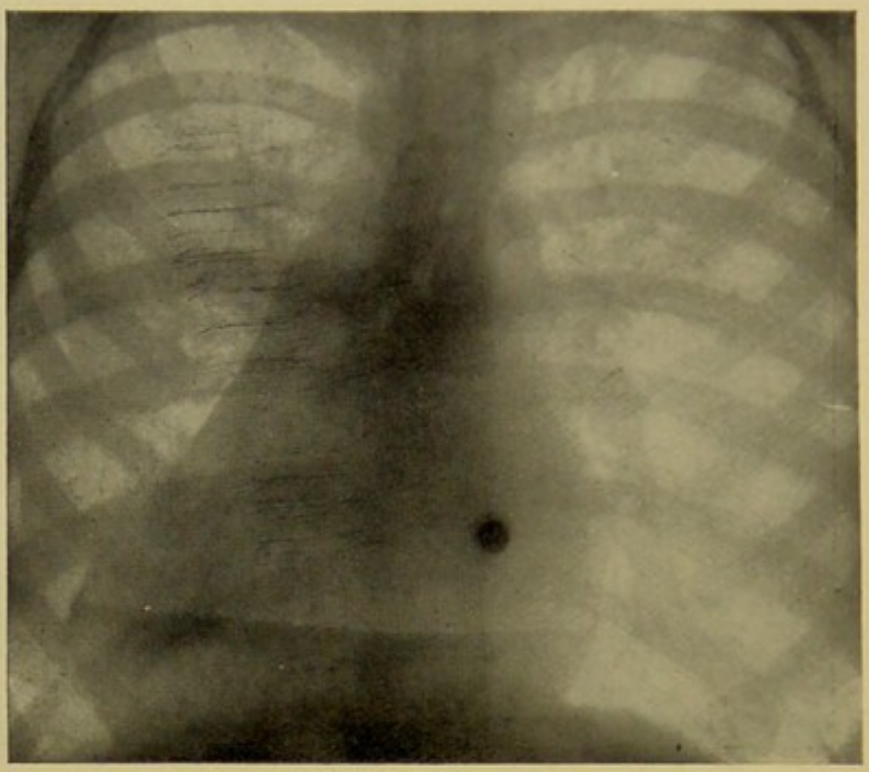
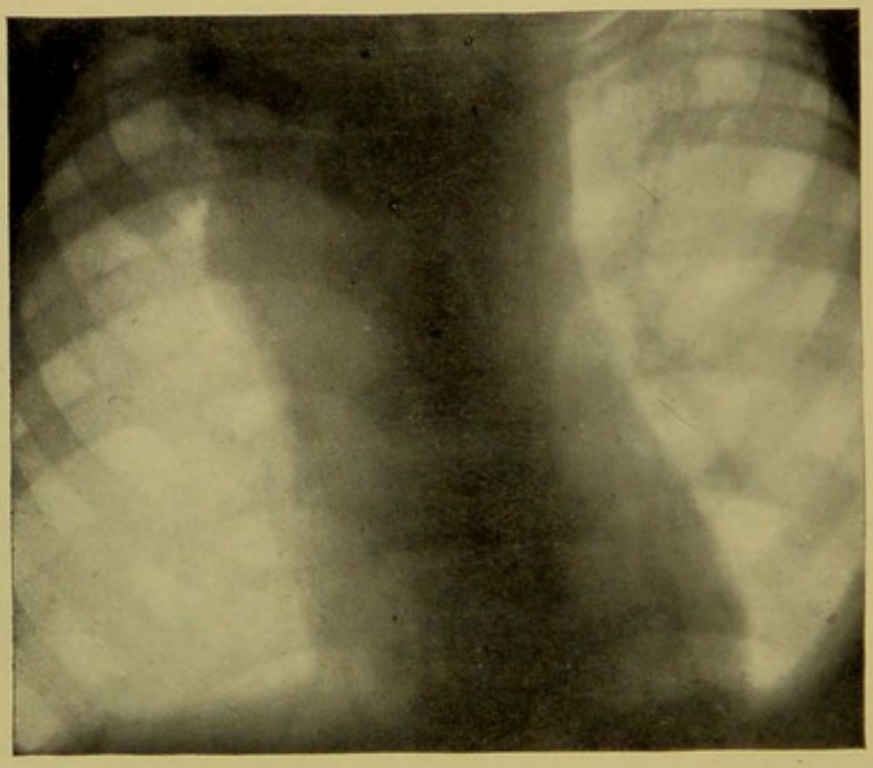


FIG. 12.



(No. 10) confirmed the diagnosis which had been already made of an aneurysm affecting the transverse and descending portions of the arch of the aorta.

The possibilities of error in the diagnosis of aneurysm seem to be almost limitless. One would hardly have thought that, given the physical signs of pneumothorax, the question of aneurysm would have to be considered, but Dr. Newton Pitt[29] has brought forward six cases, all of which were submitted to a post-mortem examination, in which compression of the left bronchus by a saccular thoracic aneurysm produced such over-distension of the lung as to give rise to the suspicion that the patient had a pneumothorax. It therefore behoves the physician not to be too confident in a diagnosis of pneumothorax until he has put out of court over-distension of the lung due to compression; and on the left side an aneurysm is the most common cause of compression. Dr. Walsham states emphatically that an X ray examination of the chest will discriminate between an over-distended lung and a pneumothorax. "In the pneumothorax the shadow of the collapsed lung can always be seen, while in the over-distended lung, of course, it is never so seen."[30]

As I have already mentioned, I am fully convinced of the diagnostic value of complete paralysis of the recurrent laryngeal nerve on the left side giving rise to what is termed the cadaveric position of the corresponding vocal cord. I am, therefore, quite in accord with what appeared to be the general feeling of the speakers who took part in a discussion on this subject at the Laryngological Society[31] in 1905. One of the speakers said that he had come to the conclusion that the great majority of cases of paralysis of the left vocal cord were due to aortic aneurysm where no other cause could be found for it. Indeed, he went so far as to say that he believed fully nine-tenths of such cases of paralysis of the left cord were due to that cause. While not going quite so far as to say that nine-tenths of the cases of left recurrent paralysis are of aneurysmal origin, I would certainly agree that the great majority of the cases are of this nature. I can, however, recall numerous cases in which malignant disease of the œsophagus has involved the nerve and caused

paralysis. Three other cases come to mind in which there was difficulty in deciding as to the cause of the paralysis.

A medical man, 30 years of age, consulted me with the history that eight days previously he became suddenly hoarse. The day before the onset of hoarsensss he had had pain in his throat, but he had had no difficulty in swallowing, and he was not feverish. On laryngoscopic examination I found the left vocal cord immobile in the cadaveric position. I could not detect anything abnormal in the lungs or heart ; the left pulse was larger than the right ; the pupils were equal. It was before the days of X rays, so that I could not call them in to assist me in arriving at a diagnosis. In consultation with Sir Felix Semon it was agreed that peripheral neuritis was the probable cause of the paralysis. The subsequent course of events confirmed the correctness of the diagnosis. Fourteen years later I met the patient in consultation, and I was glad to find that he was enjoying vigorous health.

The second case was a young lady, 19 years of age, who consulted me in June, 1912, on account of hoarseness and choking when she drank. These symptoms had existed two months. The left vocal cord was found in the cadaveric position. The cardiac dulness was greatly increased, especially to the right ; systolic and diastolic murmurs were audible in the aortic area, and presystolic and systolic in the mitral. The right pulse was much larger than the left, and the right pupil was also larger than the left. From the skiagram No. 11 [32] you will see that there is great dilatation of the right side of the heart, but no evidence of an aneurysm. The paralysis of the left vocal cord must be due to stretchiug and atrophy of the corresponding recurrent laryngeal nerve. The inequality of the pulses and pupils, together with the laryngeal paralysis, would certainly have suggested the possibility of an aneurysm, in spite of the age and sex of the patient, had it not been for the X-ray examination.

In the third case, which was under my care in the Westminster Hospital (before the days of Roentgen rays), there was a double aortic murmur and a mitral systolic murmur. As the left vocal cord was in the cadaveric position a diagnosis of aneurysm was made. At the post-mortem

examination there was no aneurysm, simply great dilatation of the left side of the heart.

The last two cases illustrate the difficulty that the combination of valvular disease with enlargement of the heart and the presence of left recurrent paralysis may give rise to in diagnosis. Fortunately, however, we can now usually clear up the question by means of an X ray examination.

Sir James Risdon Bennett, whose admirable Lumleian Lectures in 1871 did so much to direct attention to intrathoracic growths, truly said, "*The differential diagnosis between aneurysm and malignant tumours is sometimes* beset with difficulties."[33] He pointed out that unlike aneurysms which take a definite course in their development, in malignant tumours there is the utmost diversity, not only in the original situation of the growths as regards their anatomical relations, but also as regards the direction they take in their development. The superficial veins are more likely to become distended from the pressure of a cancerous growth than by an aneurysm. He says that speaking generally he believes "that pain is a more prominent and severe symptom in connexion with aneurysm than with intrathoracic cancer."[34] Another difficulty at an early period of the case is the existence of a cardiac bruit in the presence of deep-seated cancerous disease at the root of the lungs. In this condition the diagnosis of aneurysm might very well be made.

Sir J. R. Bennett's next statement draws attention to a most important point. "A marked distinction between the progress of an intrathoracic aneurysm and cancer is the well-known tendency of the former to produce *absorption of bone and external tumour.*[35] Like all general rules in medicine it has exceptions. Sir J. R. Bennett himself quotes a case recorded by Mr. Holmes[36] in which both external tumour and absorption of bone resulted from the outward growth of a malignant mediastinal tumour. Still the fact remains that while absorption of bone and external tumour are comparatively common in aneurysm, they very rarely occur in the case of new growths. Owing to the deep-seated position of the thoracic aorta an aneurysm of this part of the aorta seldom comes to the surface, so that dulness to the left of

the sternum is more likely to be due to a new growth than to aneurysm.

It may be stated that as a general rule thoracic aneurysm occurs in men under 55 years of age, who usually present the appearance of health ; whereas malignant disease, though it may occur in early life, is more common in advanced years, and it is frequently associated with emaciation and pallor.

The signs of an intrathoracic tumour in a woman should awaken suspicion of a new growth in the first instance. Anæmia, cachexia, and irregular pyrexia are in favour of a malignant neoplasm, as is also the presence of enlarged glands. The course of the disease will sometimes assist in the diagnosis. In cases of intrathoracic tumour there is a steady progression from bad to worse. This can readily be understood, if it be borne in mind, that neoplasms, occurring in the mediastinum, are almost invariably sarcomata or carcinomata of rapid growth, and associated with this active development are emaciation and constitutional disturbance. Contrariwise, patients suffering from aneurysm are liable to alternations of improvement and retrogression. But there are exceptions to this course, and I remember the case of a patient, suffering from a malignant growth in the mediastinum (verified by a necropsy), in whom the improvement, which ensued after the administration of iodide of potassium and the enforcement of rest, was so great as to confirm the idea that an aneurysm was the real cause of the trouble. The paroxysmal tendency of the symptoms in cases of aneurysm needs to be emphasised ; this is especially to be noticed as regards dyspnœa.

In distinguishing intrathoracic aneurysm from an intrathoracic growth, it should be borne in mind that the dulness in the case of the former is less irregular, and there is not so much resistance on percussion. In aneurysm there is a greater likelihood of the pupils and pulses being unequal. There is also a greater tendency to pressure upon the trachea and left recurrent laryngeal nerve, as evidenced by cough, hoarseness, and stridor. In new growths, as has been already pointed out, there is a greater likelihood of pressure upon veins, causing enlargement and tortuosity of the superficial

veins and œdema of the upper extremities and chest wall. The most common cause, however, of great venous distension is the obliteration of some deep veins due to thrombosis.

Murmurs are naturally much more common in aneurysms, but they are by no means unknown in new growths. In a doubtful case an X ray examination will usually clear up the difficulty. It did so in the patient from whom this skiagram No. 12 was taken. Aneurysm was suspected, but, as will be seen, a new growth (lymphadenoma) was present.

The combination of the signs of loud tracheal breathing and dysphagia is so suggestive of the presence of an aneurysm that their occurrence should entail the most thorough investigation, and especially the use of Roentgen rays, before an adverse decision is arrived at. But even with the help of an X ray examination the aneurysm may be overlooked, as I think would have happened in a case recorded by Dr. Martin,[37] where an aneurysm of the size of a small walnut projected backwards from the descending part of the arch and pressed itself between the trachea and œsophagus. Into this latter the sac, at its lower portion, had opened; while at the same time it had by its upper portion still exercised pressure upon the œsophagus above; and so the whole stream of blood, which should have passed down the descending aorta, had been pumped into the stomach—not a single drop having been allowed to come upwards to the mouth.

The dysphagia caused by the pressure of a thoracic aneurysm upon the œsophagus may give rise to the suspicion of malignant stricture. A history of syphilis would point to aneurysm. The pain caused by stricture is comparatively slight, whereas that due to aneurysm is boring, persistent, and severe. I have seen the recurrent laryngeal nerves involved in several cases of œsophageal stricture due to malignant disease. The cadaveric position of the right vocal cord would certainly be in favour of malignant disease. As regards the left side, the aneurysmal sac which compresses the œsophagus is usually too low down to catch the left recurrent, so that the cadaveric position of the left cord is more likely to be due to malignant disease than to aneurysm. Then it should be remembered that pressure upon the left

bronchus with all its results is a very common effect of aneurysm of the descending thoracic aorta.

In the early stage of the erosion of the vertebræ by an aneurysm the pain and tenderness thereby produced may lead to an erroneous diagnosis of other diseases affecting the spine.[38] The two most common are tuberculosis and the invasion of a malignant new growth. On the present occasion it must suffice to mention the difficulty, and to suggest that the presence of Wassermann's reaction would be in favour of aneurysm. The X rays should, of course, be employed; careful investigation of the back should be carried out, and any bulging of the ribs should suggest the presence of an aneurysm.

It seems to be probable, though I can bring forward no actual proof, that the thoracic aorta may pulsate and give rise to the suspicion of an aneurysm, much in the same way as occurs in the abdominal aorta. The most graphic account of the latter condition we owe to the pen of Sir James Paget.[39] He pointed out that this mimicry is most frequent in the abdominal aorta, but he said that it was not very rare in the subclavian, innominate, and carotid arteries, and is sometimes not easy of diagnosis. In patients complaining of pulsation in the chest, if the innominate, carotids, and subclavians, in the absence of aortic regurgitation, are all found to be throbbing, then we may suspect that the sensation of pulsation in the chest may be of a functional nature. Aortic insufficiency may very closely simulate aneurysm; the greatly enlarged left auricle and widened aorta may cause increased dulness.

Should the aneurysm fail to reach the chest wall there may be, in the absence of an X ray examination, considerable difficulty in recognising its existence. If, however, it be of a large size it may be possible to diagnose it by the extent and intensity of the bruits or sounds which are audible over the chest. Take, for instance, a case in which there are stridulous breathing of a tracheal character and dysphagia. Here we have the evidence of pressure upon the trachea and œsophagus. This may be due either to aneurysm or a new growth. If the heart sounds or bruits are widely transmitted over the chest, the presumption is

almost certain that we have to do with an aneurysm. In the absence of this diffusion we should naturally think of a new growth.

In considering the difficulties of diagnosis the question of an abnormal arrangement of the aorta must be borne in mind. Dr. Herringham [40] has recorded "a case where a right aortic arch passed behind the œsophagus to the left side, and becoming dilated, killed the patient by slow compression of the trachea." The occurrence in this case of paralysis of the right recurrent laryngeal nerve with equal pupils, together with dyspnœa and stridor evidently due to something compressing the trachea, presented a grouping of symptoms which it was difficult to explain. At the necropsy it was found that the right recurrent laryngeal nerve, turning under the aortic arch instead of the subclavian, had become thickened and adherent to the dilated artery. This explained both the right-sided laryngeal paralysis and the escape of the fibres of the second dorsal nerve to the eye.

Among the specimens in the exhibition of aneurysms at the discussion on the Surgical Treatment of Aneurysms by the Surgical Section of the Royal Society of Medicine [41] was one which illustrates a very rare difficulty in diagnosis—a difficulty, indeed, which seems almost insuperable. An aortic aneurysm was so closely simulated by a dilated pulmonary artery that the left common carotid artery was tied, and apparently with benefit. Death occurred years afterwards from phthisis, and the aorta was found to be unaffected. The specimen is in the Museum of St. George's Hospital.

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LECTURE III.

Delivered on March 13th

MR. PRESIDENT, FELLOWS AND GENTLEMEN,—The *prognosis* in intrathoracic aneurysm must always be a most anxious one. I would hardly like to say that a complete cure is impossible, but with one or two doubtful exceptions—I say doubtful, because the cases were observed before the X ray method of examination was introduced—I have not seen a case which I could consider a permanent cure.

Treatment by rest and other measures will usually remove pain and other unpleasant symptoms, and cause a great modification in the physical signs; but, as is often shown by the post-mortem examination, while the original aneurysm has become consolidated, another aneurysm has developed, and this, which frequently escapes recognition, may be the cause of death. As might have been expected, an aneurysm progresses less quickly in people who can lead a quiet life and who are of a placid disposition. It has been stated that aneurysms run a more rapid course in women than in men, but I have not been able to verify this statement. The presence of aortic regurgitation is a very unfavourable sign, as is also advanced degeneration of the blood-vessels.

To show the frequency of sudden death in aneurysm I may quote Dr. Kelynack's statistics; out of 32 cases of rupture reported by him six were observed in medico-legal investigations, the subjects beings brought dead to the hospital. Six of my 35 private patients died suddenly, in addition to 6 who died from rupture of the aneurysm, 4 of whom are definitely stated to have died suddenly, and possibly the other 2 also may have died suddenly.

The duration of life after the recognition of an intrathoracic aneurysm is, in my experience, usually limited to

less than 4 years, though in very exceptional cases life may be prolonged for 6 or 7 years.

I have carefully investigated the notes of 35 cases of aneurysm I have met with in private practice. Excluding all doubtful cases and cases in which I have not been able to trace the final result, and excluding also a patient who was alive 10 years after I had made a diagnosis of aneurysm, I find that the average duration of life in 27 cases works out to a little over 2 years and 8 months. There are 2 cases in which death apparently occurred nine months after the appearance of symptoms suggestive of aneurysm, and 1 case in which death was delayed for seven years. Owing to the extreme difficulty which often presents itself in fixing the exact date of the onset of symptoms, I feel convinced that my figures underestimate the duration of life, and I think that about three years would be nearer the mark. In some cases the symptoms of the aneurysm started so definitely after a well-recognised strain that one can be quite sure as to the duration. In one patient who was under my observation nearly the whole time the strain occurred four and a half years before death.

All my 27 cases occurred, as I have already stated, in my private practice; they were almost all of them sufficiently well endowed with this world's goods to be able to take care of themselves, as in every case they had been warned of their condition; and as all the cases had been submitted to treatment more or less carefully carried out, it was to be expected that they would last longer than hospital patients, who generally have to resume work when quite unfit for any active exertion.

Sir William Osler[1] states that "the average duration after discovery is from 18 months to 2 years," but he does not mention from what class of patients his statistics are derived.

A case I showed at the Medical Society[2] in April, 1911, illustrates the slow progress of some cases. The patient was a man of 43 years of age, a wire-drawer. He was originally admitted into Westminster Hospital under my care in July, 1905, placed on low diet, kept at absolute rest, and given iodide of potassium in moderate doses. He improved greatly under treatment, but has taken iodide off and on ever since.

He was able to continue at work—fairly laborious—the whole time. In March, 1910, he had a slight attack of hæmoptysis. When shown to the society the physical signs of an aneurysm of the first part of the arch of the aorta were very distinct.

A somewhat similar case is recorded by Dr. Gibson.[3] The patient was a man, 39 years of age, whose occupation involved a very considerable amount of physical exertion, who lived for 12 years after the diagnosis of an aneurysm of the ascending portion of the arch of the aorta, and who died eventually from heart failure. On two occasions he was an in-patient, and was treated by means of absolute rest, restricted diet, and iodide of potassium.

The most remarkable instance of the preservation of life under threatening conditions which I have come across is a case reported by Dr. T. R. C. Whipham.[4] A man, 40 years of age, was admitted into hospital with a rounded tumour in the first and second right interspaces, the skin over it being tense and discoloured. He was under observation for full 15 years, and during the whole of that time he had a large external tumour, though the aneurysm had probably existed a year or so longer. With the exception of about 18 months spent in hospital the patient was able to carry on his occupation of builder's foreman. Death occurred from external rupture of the sac.

These three cases afford good examples of the great difficulty which often attends the prognosis in cases of aneurysm.

Life Assurance.

My experience in life assurance has taught me that thoracic aneurysms are not infrequently overlooked, and that consequently they represent one of the causes of early death after assurance.

In a paper I read before the Life Assurance Medical Officers' Association in 1907 [5] on Statistics of the Rock Life Assurance Company I made the following remarks :—

> There were 6 deaths from aneurysm of the thoracic aorta and 1 from aneurysm of the abdominal aorta. In addition to these cases there were 3 deaths recorded from rupture of the aorta—these were

probably due to aneurysm. Two of the claims due to death from aneurysm of the thoracic aorta occurred after only two premiums had been paid. One of these was for a policy of £10,000. The deaths occurred at the ages of 47 and 51 respectively. These two cases illustrate the difficulty of the recognition of aneurysm of the aorta, as from what we know of the clinical history of this disease aneurysm was almost certainly present at the time of the examination, death only very rarely occurring within two years from the development of symptoms and physical signs of the disease. All physicians with large clinical experience will know how difficult is the diagnosis of an aneurysm, even when one has the aid of the patient in mentioning symptoms. It is in these doubtful cases that the Roentgen rays are so useful in clearing up the obscurity of the diagnosis, but it is a counsel of perfection to expect that the X rays should be employed in examination for life assurance.

Taking the 10 cases of aneurysm and rupture of the aorta together, the average age at entry was 51·5 years, with an average duration of life of 16 years, the expectation being 19·28 years, so that the aneurysm cases were very unfavourable from a pecuniary point of view. I would therefore urge the importance of carefully investigating any man of 40 years and upwards in whom there is a ringing second sound in the aortic area.

These remarks, made five years ago, are in accord with my present views on the subject.

Medical Treatment.

While I was attending the practice of St. Bartholomew's Hospital I was much impressed by the teaching of the late Dr. James Andrew, whose great diagnostic skill and general excellence as a physician were universally admitted by all who had the privilege of working with him in the wards of the hospital. He was particularly keen on the recognition and treatment of intrathoracic aneurysm.

Tufnell's Method of Treatment.

Dr. Andrew was one of the first in London to adopt Tufnell's plan of treatment, of which he thought highly, as the following remarks show:—

But although the instances of complete consolidation must always be few, those in which relief from distress and an increase in the strength of the walls of the sac are obtained are very numerous.[6]

He insisted on the observance of the following rules as being of the utmost importance :—

Place the patient at once upon the minimum diet, and forbid even the slightest movement which can be avoided.

The room in which he lies must be as quiet and secluded as possible.

No treatment by drugs is to be attempted at the same time.

Listen to no complaints of thirst so long as the pulse and temperature are normal, or nearly so, and the whole allowance of solid food is consumed.[7]

These are most admirable rules, and time has only confirmed their utility. But before mentioning additional rules I must direct attention to his third rule—i.e., that in reference to the use of drugs. This, I believe, refers almost solely to the use of iodide of potassium. Increasing experience in the treatment of aneurysm has brought home to me that Dr. Andrew was right in advising that iodide of potassium should not be given while the patient is undergoing the Tufnell treatment, because the iodide so increases thirst that it is impossible to limit the amount of fluid as laid down by Tufnell. This rule has no reference to the occasional use of drugs, such as aperients for the constipation, which the dry diet almost inevitably produces, or of opiates for the relief of pain.

As regards the diet to be employed I cannot do better than quote Tufnell's[8] directions, which are as follows :—

The diet, under ordinary circumstances, must be confined to three meals served at regular intervals, and restricted to the following in kind and amount—viz., for breakfast, two ounces of white bread-and-butter, with two ounces of cocoa or milk. For dinner, three ounces of broiled or boiled meat, with three ounces of potatoes or bread, and four ounces of water or light claret. For supper, two ounces of bread and butter, and two ounces of milk or tea, making in the aggregate ten ounces of solid and eight ounces of fluid food in the 24 hours, *and no more*.

He adds in a footnote :—

In some irritable constitutions this restriction in diet will be irksome, and the patient becomes intolerant and restless. Here, instead of attempting to persist in the withholding of food, the appetite should be indulged *to the satisfying of the patient* (so as to keep him tranquil), *but no more*.

In practice it will be found that very few will, or indeed can, put up with so rigid a diet as Tufnell laid down, and I have found that it has been almost always necessary to increase the fluid to 12 or even 16 ounces in the 24 hours—I have usually found that 12 ounces of solids were sufficient to satisfy the patient.

After having settled the diet, there comes the very important question of the duration of the treatment. According to Tufnell two months or ten weeks at least are necessary, and during this time the patient should be kept absolutely at rest. When I have proposed, either to hospital or private patients, that they should undergo this method of treatment, I have considered it my duty to put plainly before them that they must submit to at least six months' complete rest, with a very restricted diet for the first six or eight weeks. After this period I have generally considered it desirable to relax somewhat the restrictions as to diet, though still most careful to maintain absolute rest of body during the remaining four months of treatment.

One of the most important factors for success, if not the most important, is the due selection of the patient. In the first place, he should be of a quiet, placid temperament. Two of the cases I had in private illustrate the difference of behaviour in this respect. One, a man aged 37 years, went through an eight months' course quite cheerfully and happily, and was easily amused by trifles. The second case was a man aged 55 years, in other ways a good subject for the treatment, but his restlessness and irritability were so great that I had to advise that the treatment should be discontinued after a trial of three weeks. Then the social position of the patient has to be considered. I think that it is almost cruel to put a working man to the strain and discomfort involved in this method of treatment if at the end he must return to hard manual labour, whereas a person whose occupation is of a sedentary nature might be offered the chance of cure. Patients under 55 years do better than older patients. Men who have drunk freely, who have very degenerate vessels or with a syphilitic history are not good subjects for the treatment. On the other hand, the history of a distinct strain or injury as the cause of the aneurysm is a favourable indication.

The "aneurysm of physical signs"—i.e., the aneurysm springing from the first part of the arch of the aorta—is more suitable for this treatment than the "aneurysm of symptoms." It is recognised earlier, and the progress of the case can be watched in a more satisfactory manner. In aneurysm affecting the transverse arch of the aorta, where there is pressure upon the trachea or œsophagus, prolonged rest in bed in the recumbent position is usually not well borne, and may have a prejudicial effect; even rupture with a fatal hæmorrhage may result. A saccular aneurysm, with a comparatively small opening from the aorta into the sac, is the most suitable kind for treatment. This usually implies that the aneurysm has existed some little time, so that the sac has become larger in proportion to the opening. A fusiform aneurysm does no good under Tufnell's treatment.

The presence of aortic regurgitation puts the treatment out of court. In the first place, the quickness with which the aorta is emptied prevents the slowing of the circulation in the sac, which is essential if coagulation is to take place, and secondly, as in thoracic aneurysm with aortic regurgitation death commonly occurs from heart failure, it is very important that nothing should be done which would tend to check the conservative hypertrophy of the left ventricle.

Constipation is generally a trouble. This may be remedied by calomel in combination with colocynth and hyoscyamus, or by cascara sagrada, or by a glycerine or olive-oil enema. As I have already mentioned, pain may require the exhibition of morphine. Sleeplessness is frequently present. A small dose of chloral (10 grains) or chloralamide (20 grains) with or without bromide of potassium may be given. Sulphonal or trional I have also found useful. Veronal I have avoided since I came across cases in which comparatively small doses have had untoward results.

For palpitation and increased frequency of the pulse I have ordered tincture of aconite and tincture of strophanthus. But as far as possible the less drugs the patient has the better he will stand the treatment, the explanation possibly being that the cases which respond best to the treatment do not require any assistance from medicines.

When the rigid confinement to bed ceases the patient

should be allowed to return to a more normal state only very gradually. He should first be allowed to sit up in bed ; after he has done this for three or four days, he may be placed on the side of the bed and his legs allowed to hang down. After he has accustomed his circulation to this change of position for a day or two, he may be assisted out of bed on to a couch, then into an armchair, and finally he may be allowed to walk. During these various experiments attention should be paid to the pulse, and if its frequency be much increased the patient's progress as regards movement must be checked for a time.

As regards the result of the Tufnell treatment, I do not think that we have advanced much further than the position taken up by Dr. Vincent Harris[9] upwards of 30 years ago, who sums up a very able paper on the subject by saying that "although there was almost certain relief of symptoms the cures were rather rare."

Based upon an entirely different principle to Tufnell's plan of treatment is that recommended by Dr. Bezly Thorne,[10] which he describes as follows :—

> The treatment by mineral baths, liberal diet, and free water drinking is directed towards the increase of arterial lumen and the diminution of peripheral resistance, with free elimination of uric acid and other blood toxins, and consequent diminution of hydrostatic pressure on the walls of the aneurysmal sac, arrest of degenerative processes, and repair of the cardio-vascular tissues.

He also advocates "regulated walking exercise taken twice daily." This line of treatment is naturally only applicable in the very early stage of aneurysm.

The Iodide Treatment.

I suppose that in no disease, excepting, of course, tertiary syphilis, has iodide of potassium been more employed and given in larger doses than in aneurysm, and most authorities are agreed that the immediate effect of its administration is often very striking, but I have yet to be convinced that iodide of potassium is capable of curing an aneurysm. More-

over, it is difficult to separate the effect of the iodide from the rest, which usually has been enjoined at the same time. The excellent result following the use of this drug must have been noticed by all hospital physicians. Under its influence the pulsation in the tumour may be seen to lessen, and eventually to cease entirely ; cough and dysphagia are no longer troublesome ; the agonising pain, which previously had kept the patient awake, disappears as though by a charm, and frequently in the course of two or three weeks' treatment the patient feels so much better that he is anxious to leave the hospital. The after-history, however, is usually that the improvement lasted some months—indeed, in some cases upwards of a year; but sooner or later there is a return of the old symptoms, and eventually death from rupture of the aneurysm or gradual exhaustion.

Of all the symptoms produced by aneurysm pain is the one upon which iodide appears to exercise its greatest effect. Some patients as long as they take the salt are free from pain ; omit it and the pain returns. This holds good not only for patients in bed, in whom the rest may have a great deal to do with the cessation of pain, but it also applies to patients who are walking about.

Iodide of potassium was at first employed more or less empirically, but when its favourable action came to be recognised attempts were made to explain its action. The first explanation propounded was that the cases in which it succeeded were of a syphilitic nature, and that therefore its virtues were due to its anti-syphilitic properties ; but, as Dr. Balfour has pointed out, this has only made its action still more mysterious. He has strongly advocated the theory that iodide of potassium owes its curative virtue in aneursym to its power of lowering the blood tension. He has pointed out that large doses are not necessary and are sometimes injurious, and by large doses he means doses over 20 grains. He believes that the failures in the use of this drug, which were generally accompanied by an unduly quickened cardiac action, were most likely dependent on too great a lowering of the blood pressure; because this would not only account for the excited action of the heart, which had an unfavourable

influence upon the aneurysm, but would also permit the blood to flow away too rapidly to provide for the due nutrition of the tissues.

Dr. Balfour regards the cure of an aneurysm as depending upon the hypertrophy of its coats, "which cannot be effected without a certain amount of blood pressure, neither can it occur if the blood pressure remains at normal, for all experience teaches us that the effect is then one of gradual though slow dilatation, with pressure corrosion of all the surrounding tissues." He continues:—

> Hit the happy mean, and the effect is that the arterial coats behave like a hollow muscle, which hypertrophies when opposed to an obstacle with which it is able successfully to cope. To hit this happy mean and ascertain the proper dose the patient is put to bed without any other treatment, and his pulse-rate ascertained night and morning for a few days. So soon as his average pulse-rate in recumbency is ascertained he is put upon 10-grain doses of iodide of potassium every eight hours. If the pulse-rate remains unchanged the dose is increased to 15 grains every eight hours, and every week an increase of 5 grains each dose is made till the pulse-rate begins to rise. In this way it has been ascertained that 15 grains to a dose is seldom able to be exceeded, while it has often happened that 10 grains has been found to be the most advantageous quantity.

Dr. Balfour further points out that with a proper dose a marked improvement can generally be effected in three months instead of six months, the usual time before accurate dosage was resorted to. He first enunciated these views in 1876,[11] so that the main credit of establishing the iodide of potassium treatment on a firm basis must be assigned to him.

As I have already stated, the diet of patients on the iodide of potassium treatment should not be much curtailed, they do better with a fairly liberal allowance of food, and it is quite impossible, even if desirable, to limit the amount of fluid, as the salt causes great thirst in some people, and iodism may be produced unless a fair supply of liquid be taken. At first the patient should be kept in the recumbent position; as a rule two or three months will suffice; after this period the patient may be allowed up on the sofa for a few hours daily and by degrees he may take gentle exercise. From the patient's point of view there can be no doubt that

the iodide of potassium plan of treatment is infinitely preferable to the Tufnell system. What we want are some definite statistics as to the comparative advantages of these two methods of treatment, and unfortunately at present there are none available. Of one thing in regard to these two plans of treatment I feel certain, that it is unwise to attempt to combine them. If the Tufnell plan of treatment be adopted do not give drugs. If it be decided to try Balfour's method of giving iodide of potassium, do not limit rigidly the amount of solid and liquid nourishment, otherwise I am afraid that the patient will fall between two stools.

The excellent effect which commonly attends the administration of iodide of potassium would seem to point to the possibility of a syphilitic element in the case, and to indicate the desirability of the Wassermann reaction being tried in every case of aneurysm. If a positive reaction be obtained a vigorous antisyphilitic treatment should be instituted, but it is not advisable to employ salvarsan.

The Gelatine Treatment.

The gelatine plan of treatment was introduced by M. Lancereaux, and at a meeting of the Academy of Medicine in Paris in July, 1904, he stated that among 1000 injections of solution of gelatine he had not observed any mishaps. The solution he advises consists of 25 parts of gelatine and 7 parts of chloride of sodium dissolved in 1000 parts of water. He injects 200 grammes at a time, and the process is repeated every four or five days. If the gelatine be of good quality and thoroughly sterilised at a high temperature M. Lancereaux maintains that the treatment is in no way dangerous, and has yielded very favourable results.

Two years later (July, 1906), in a paper communicated to the Académie de Médecine,[12] M. Lancereaux stated that about 1200 injections had been made by himself and his assistants without the occurrence of a single case of tetanus. He maintains that the efficacy of this method of treatment is indisputable, and he points to the relief

of pain which follows after four or five injections as a proof of this. In every case of aortic aneurysm perforating the sternum, covered only by thin and stretched skin, which he has treated he has noted commencing contraction of the tumours after 10 injections, and total induration after from 30 to 40 injections. M. Lancereaux goes on to say that these good results cannot be attributed solely to rest in bed, as he has watched cases in which patients kept for long periods in bed obtained no benefit from treatment until they received injections of gelatine, and he has had other cases in which after the injections the aneurysmal tumours contracted, notwithstanding the absence of complete rest.

In contrast to this optimistic view of the introducer of the gelatine treatment I would cite Sir James Kingston Fowler's experience at the Middlesex Hospital.[13] Twelve cases of aneurysm were treated by subcutaneous injection of gelatine, and he did not consider that this series of cases established a superiority of the gelatine method over the treatment by rest and diet, combined with the administration of iodide of potassium. In one of the 12 cases, however, a strikingly superior result was obtained, and there was no doubt as to the complete cure of an innominate aneurysm in a man aged 38.

Dr. Guthrie Rankin, who has employed the treatment at the Seamen's Hospital, where aneurysms are of frequent occurrence, writes to me as follows:—

> The general impression the treatment leaves in my mind is that it is very useful and palliative, that in some cases it almost produces cure, and that if careful methods are adopted in its preparation there is evidently no risk of evil consequences. One striking fact about its use is the rapid relief which it affords to the pain of aneurysm; of this I am quite sure.

The apparatus employed consists of a glass flask with a vulcanised rubber cork through which pass two tubes, one reaching nearly to the bottom of the flask and the other just projecting beyond the cork. To the former is connected an indiarubber tube attached to a hollow platinum needle, with which the puncture is made. The shorter glass tube

is connected with a pump, and in it is a bulb containing cotton-wool to filter the air as it is driven into the flask. Everything must be carefully sterilised, and the gelatine is kept fluid by the flask being placed in a warm-water bath. The skin over the buttock is then rendered aseptic, and the injecting needle is plunged deeply into the muscle of the buttock. The injection is made slowly, about a quarter of an hour being occupied in the process. After the injection the patient should be kept at rest. Sir James Kingston Fowler begins with 50 c.c. of a 2 per cent. solution and gradually increases the amount to 100 c.c. and 4 per cent. respectively, although as much as 250 c.c. of a 2 per cent. solution may be used at first without unfavourable symptoms. Occasionally the injection is followed by smart reaction, the patient may have a rigor, and his temperature may rise to 103° or 104° F. Though very alarming at the time no evil may result from it. When this plan of treatment was first introduced into this country two deaths from tetanus occurred, but if the gelatine be carefully sterilised, and strict aseptic precautions be taken in carrying out the injection, the risk of tetanus may be regarded as *nil*.

As regards the results obtained by the gelatine plan of treatment, it may be said that though benefit has resulted in a considerable number of cases, it has not justified the claims that were made on its behalf when it was first introduced, as there is no definite evidence of the cure of a thoracic aneurysm by this means. The two classes of cases in which its employment seems particularly indicated are cases in which an aneurysm has perforated the chest, and in which there is a danger of external rupture, and cases in which there is great pain.

The calcium salts, such as the chloride and lactate, have been employed in the hope that they would favour coagulation of the blood in the sac, but I have not been able to discover any evidence that they have a beneficial action—with the exception of a case mentioned by Dr. Oswald Browne in the discussion on Aneurysm at the Medical Society of London in 1900.

Expectant and Symptomatic Treatment.

Lastly, there comes the purely expectant and symptomatic method of treatment. Some physicians are altogether opposed to the rest cure ; they maintain that patients who are subjected to it are worse at the end of the treatment than when it was started, because their tissues had degenerated during their passive existence, and that they were consequently less able to resist disease.

Confinement to bed on a very spare diet has a most depressing effect upon some people, and therefore the cases which are to be submitted to this line of treatment should be selected with the greatest care, as I have already pointed out, not only in regard to the physical state, but also as regards the mental condition. To say that a patient with an aneurysm leaves the hospital, after a rest cure with a modified Tufnell diet, in a worse case than on admission, is not in accord with my experience, now extending over more than 40 years. Still, there are quite a number of patients with intrathoracic aneurysm who do better physically, and certainly mentally, if they are allowed to pursue their ordinary avocations, provided their work be not too strenuous for body or mind. We all must have met men who, when their grave condition has been clearly placed before them, have elected to continue their life work at all risks, and I am bound to say that my sympathy is with them, and that I myself would, under similar circumstances, feel inclined to follow their example.

In the event of a patient deciding to lead his ordinary life, he should be cautioned to take things as easily as possible, to avoid all great exertion, especially of a sudden nature, to eat with great moderation, and, as a general rule, to avoid alcohol. Tobacco may be indulged in with moderation, and by moderation I mean a quantity of tobacco not exceeding three ounces per week.

Medicinally, I have no doubt that these patients are benefited by iodide of potassium, especially if the blood pressure be high. I usually order from 5 to 10 grains with 15

to 20 minims of aromatic spirit of ammonia in chloroform water to be taken three times a day after food. This will often suffice to keep them comfortable.

For the relief of the agonising pain that is sometimes experienced in aneurysm no remedy has a more remarkable effect than bleeding. Dr. Pye-Smith's [14] experience on this point is decisive. He writes as follows :—

> I shall never forget the case of a man who for more than a week had not been able to lie down or enjoy more than broken and uninterrupted (*sic*) sleep. I found him the day after his admission to the hospital sitting up in bed, exhausted by want of rest and continually dropping asleep, but wakened again by the terrible pain he endured. The house physician had given him morphia in full doses without any benefit. He was bled from the arm to 6 oz., and before the blood ceased flowing expressed the relief he felt, and had fallen fast asleep on his pillow before the arm was bound up. His distress was never suffered to reach so advanced a point, and again and again on its return he was relieved by a similar or a still smaller abstraction of blood.

One such case is quite sufficient to prove the utility of venesection for the relief of the awful pain occasionally suffered by the victims of aneurysm. Venesection is to be preferred to leeches in these cases, as the relief experienced is more rapid and complete.

The application of an ice-bag suspended from above so as to avoid too much pressure upon the tumour will often have a beneficial result upon the pain.

For the relief of the anginal attacks met with in aneurysm iodide of potassium in 10 to 20 grain doses three times daily will usually effect much good. In cases where the iodide fails liquor arsenicalis in 5 minim doses three times a day will sometimes be found useful. For the acute anginal attacks the inhalation of nitrite of amyl, followed up by the internal administration of trinitine, is generally efficacious. Erythrol tetranitrate, in ¼ to ½ grain doses, may be given instead of trinitine, especially if the duration of the attacks be protracted. But in many cases there comes a time when the only drug to give relief is morphine subcutaneously. I prefer to give it in combination with atropine. I am of opinion that the atropine counteracts some of the injurious effects of the morphine and prolongs its action.

For the treatment of dyspnœa, especially when accompanied by lividity, there is no remedy which can be compared to the abstraction of blood. The blood may be removed either by venesection or by the application of leeches. The over-distended condition of the right heart is often greatly benefited by loss of blood, when there is pressure on the veins causing great venous engorgement.

Should an aneurysmal tumour on the surface of the chest begin to weep and ooze, the best application is styptic lint, kept in position by a broad piece of strapping, but, of course, this can only temporarily check the tendency to rupture.

Tracheotomy.

There is one question in regard to treatment which occasionally presents itself in a most dramatic form—viz., whether tracheotomy should be performed for the relief of paroxysmal and urgent dyspnœa?

In this connexion the older Fellows of the College will doubtless recall the case of the Earl of St. Maur. This case occurred before the laryngoscope was commonly employed in this country, but what Dr. C. J. B. Williams [15] wrote upwards of 40 years ago is still true to-day. After speaking of the life-saving effect of tracheotomy in cases of laryngeal obstruction, he goes on to say :—

> It is different with the dyspnœa caused by *intrathoracic tumours*. These produce not only *laryngeal* dyspnœa by pressure on the recurrent nerves which regulate the motions of the glottis, but also *tracheal* dyspnœa by direct pressure on the lower part of the windpipe itself. Whatever difficulty of breathing may be due to the pressure on the nerves going to the larynx may be relieved by the operation for a time; but that proceeding from direct pressure on the windpipe is beyond the reach of that or any operation, and if this direct pressure exist to any great degree it would render the operation fruitless. Further, it must be borne in mind that aneurysm, the commonest kind of intrathoracic tumour, may cause death by rupture and loss of blood, and in other ways besides by suffocation, and therefore that in such a case tracheotomy cannot be regarded in the light of a curative measure. Its power to give temporary relief will depend on how much the difficulty of breathing caused by the aneurysm is *laryngeal*, from pressure on the recurrent nerves only, and not *tracheal or bronchial*, from pressure on the windpipe or one of its branches. When there is

time and opportunity for careful examination, these distinctions may be made from the character of the physical signs, so as to determine beforehand whether the operation is likely to be beneficial or not.

I have ventured to quote at length the wise remarks of Dr. Williams, and which, though written in the pre-laryngoscopic days, still in the main hold good.

In the discussion at the Medical Society in 1900,[18] on the Diagnosis and Treatment of Intrathoracic Aneurysm, I propounded the question "For what conditions should tracheotomy be advised in cases of aneurysm of the arch of the aorta?" As my contribution to the discussion I then said :—

From what I have seen myself, and from what I have gathered from the literature of the subject, I am of opinion that it is but rarely that tracheotomy is likely to be of service in aortic aneurysm. The chief obstruction to the breathing is due to direct pressure on the trachea, even in those cases in which there is spasm of the adductors or paralysis of the abductors of the cords.

I also mentioned two cases in which I had been called in at the last moment, and in both I had decided that tracheotomy would not give relief. Both these patients died the day after I saw them, and at the necropsy there was found to be direct pressure on the trachea, and this was evidently the cause of the dyspnœa.

Sir William Broadbent, in the course of this discussion, said that "he had seen tracheotomy done, but without influence on the symptoms." On the same occasion Sir James Kingston Fowler mentioned the case of a man who had bilateral abductor paralysis from pressure of an aneurysm, and as a result suffered from most severe paroxysmal attacks of dyspnœa. Tracheotomy was performed between the attacks, and the patient, who had had no sleep for two and a half days, went to sleep immediately after the operation, and slept for nearly 24 hours continuously.

Of course, there can be no doubt that if the dyspnœa be due to spasm of the adductors or paralysis of the abductors, then tracheotomy will be an immense relief to the patient; on the other hand, tracheotomy is useless in cases of direct pressure on the trachea. But it is in cases of a combination of these two conditions—i.e., where there is a double

stenosis of the air passage, due to bilateral paralysis of the abductors and direct pressure on the trachea—that the great difficulty as to the advisability of tracheotomy arises. In these cases the respiratory excursions of the larynx will help in deciding. In two cases in which I was called in, at the last moment, to decide whether tracheotomy should be performed I decided in the negative on account of the absence of respiratory excursions of the larynx. Where the obstruction is at the glottis there is an up-and-down movement of the larynx; but where the obstruction is in the trachea this is absent. I think that the correct attitude to assume in these cases of double stenosis is to advise operative interference in the hope that the laryngeal condition is the chief cause of the dyspnœa. In this event the tracheotomy will add much to the patient's comfort. If, on the other hand, the pressure on the trachea be the principal cause of the trouble, then tracheotomy will be practically useless, as it was in the case of Earl St. Maur, but the operation would add but little to the patient's distress.

The possibility of passing a stiff rubber tube down the trachea, beyond the obstruction, should be borne in mind—an early example of this procedure is afforded by a case recorded by Sir J. R. Bennett.[17] The patient was a man under Mr. Bryant's care at Guy's Hospital with a goitrous-like tumour pressing on the trachea. Tracheotomy was performed, but no air passed out of the chest. A long perforated tube was then introduced through the cannula into the bronchus, when a rush of air immediately followed and was succeeded by natural respiration.

My present attitude towards tracheotomy is in favour of the performance of the operation in doubtful cases. The patient may receive only temporary benefit, but we substitute a quiet death for one attended with great suffering. To avoid the unpleasant sequel of Earl St. Maur's case the physician should clearly lay before the patient's friends the state of affairs, pointing out that tracheotomy offered a prospect of relief, but that it might only be very partial should there be any mechanical pressure obstructing the trachea at a lower level.

Before resorting to tracheotomy in cases of paroxysmal

dyspnœa the administration of chloroform might well be tried. If it relieved the patient operative interference would be uncalled for; but if not, then the operation could be carried out all the easier for the anæsthesia, and artificial respiration resorted to if necessary. Even after the performance of tracheotomy the administration of chloroform may be necessary to relieve the spasmodic attacks of dyspnœa due to irritation of the vagi.

Surgical Treatment.

The surgical treatment of intrathoracic aneurysm may be considered under five heads: (1) the ligature of the great vessels arising from the innominate artery or arch of the aorta; (2) the passing of wire—filipuncture—into the aneurysmal sac, with or without the passage of a galvanic current through the wire; (3) galvano-puncture; (4) Macewen's method of needling; and (5) the ligature of the neck of the aneurysmal sac.

Ligature of Vessels.

The treatment of thoracic aneurysm by ligature of vessels is not a subject on which I have much personal experience. Even among surgeons there is considerable difference of opinion as to the advisability of the operation. In a case of aneurysm of the arch, in which Mr. Christopher Heath [18] tied the left common carotid, he maintained that the aneurysm was practically cured at the time of death, and that the fatal termination of the case was due to the pressure effects of the consolidated tumour. At the meeting of the Clinical Society when this case was discussed Mr. Howard Marsh mentioned that in one case in which he tied the right common carotid and subclavian arteries for aneurysm of the arch, it appeared that the operation made the patient worse instead of better, for the sac rapidly enlarged. On the other hand, Mr. Holmes, in his lectures delivered before the Royal College of Surgeons of England in

1872, mentioned 7 cases in which the left carotid was tied for aneurysm of the arch ; 4 of these cases were successful, and all except one derived benefit from the operation.

The chief indication for the ligature of the carotid and subclavian vessels is pain, while aortic regurgitation and degeneration of the arteries are unfavourable to operation.

Most physicians will agree with Mr. Christopher Heath's [19] summing up of the 6 cases in which he tied the left carotid.

> The results, taken as a whole, are perhaps not very encouraging, but it must be borne in mind that in dealing with a practically incurable disease, a prolongation of life for even a few months may be worth attempting.

Introduction of Wire into Aneurysmal Sac.

The introduction of wire into the sac of an aneurysm—Moore's method [20]—has received from M. Verneuil the name of "filipuncture." Though this operation has been attended with very unsatisfactory results in aneurysm of the arch of the aorta, it has been more successful in abdominal aneurysm. I have seen only one case in which this treatment has been carried out. The patient was under the care of my colleague, Sir Bryan Donkin.[21] Mr. Macnamara passed a yard of silver wire into the sac through a curved tubular needle. The man died six days after the operation. In 1887 Sir Alfred Pearce Gould [22] operated on a patient of Dr. W. H. White. The case terminated fatally on the sixth day from sloughing of the tissues. In his report on this case Sir Alfred Pearce Gould mentioned six other cases of aortic aneurysm which had been treated by the introduction of watch-spring or steel wire. All the cases ended fatally, but in one there was temporary improvement.

Until the year 1903 the method of wiring was unscientific and was carried out with comparative disregard of aseptic principles. In this year Mr. G. H. Colt invented an instrument for the rapid introduction of a known quantity of sterilised wire into an aneurysm, with a minimum of disturbance. He has described and figured an improved instrument (No. 3).[23]

Dr. Dawson Turner's[24] experiments would suggest that surgeons should employ zinc wire for introduction into the aneurysm, which they intended to treat by electrolysis rather than silver or other kinds of wire, a larger and a firmer precipitate being deposited by this means.

A full account of the results of the method of treatment by wiring, with or withont electrolysis, will be found in the discussion which took place at the Surgical Section of the Royal Society of Medicine in May last. Mr. d'Arcy Power's[25] conclusions at this discussion are so much to the point that I will quote them *in extenso* :—

> The chief indications for the operation of wiring are: 1. An aneurysm of the ascending part of the arch of the aorta, especially if X ray examination in two planes at right angles to each other indicate sacculation. 2. Abdominal aneurysm in the usual situation—namely, at the origin of the cœliac axis. Both these types of aneurysm are usually saccular, and the louder the bruit the greater is the chance of a case being a suitable one for wiring, because the aneurysm is probably saccular and the opening of the sac small. Medical treatment is of no avail. [Rather a gratuitous remark.]
>
> The chief contra-indications are: 1. Rapid increase of pain or of pressure symptoms. These are probably indicative of an early and fatal termination. If these symptoms pass away under treatment the question of operation can be again considered. 2. The presence of a second aneurysm. 3. Sepsis. 4. Involvement of the transverse part of the arch. The cases recorded by Hodgson show clearly that the patient's discomfort is much increased by consolidation taking place in this situation.

Professor A. A. Eshner[26] has collected the records of 36 cases, in which an aortic aneurysm has been treated by the introduction of wire into the sac and the passage of a galvanic current through the wire. In 19—i.e., in about half the cases—death followed within a month of the wiring, the best result being that obtained by Professor Rosenstirn, whose patient lived nearly 12 years after the operation. One of Professor Eshner's patients lived four weeks, the other a little over seven months.

The risk run in passing wire into the sac of an aneurysm of the arch of the aorta is well illustrated by a case treated by Mr. H. A. Ballance.[27] He introduced silver wire into the sac, and passed an electrolytic current through the wire. The patient lived ten months after the operation in com-

parative comfort. At the necropsy loops of silver wire were found in the sac, and loops of wire had also passed along the arch of the aorta, as far as the origin of the left common carotid artery and down to the bottom of the left ventricle. It is certainly very remarkable that a foreign body was tolerated in the heart so readily and for so long a period.

A careful consideration of the prospects afforded by wiring an aneurysm, with or without electrolysis, has convinced me that the operation, though quite justifiable in cases of abdominal aneurysm, cannot be of nearly the same service in cases of intrathoracic aneurysm. In the former case the surgeon can cut down and expose the aneurysmal sac, and can form a fairly clear idea as to the amount of sacculation present, and the operation, moreover, can be carried out when the wall of the sac is still in a satisfactory condition. In the case of an intrathoracic aneurysm the surgeon would, indeed, be a bold man who would propose to submit to surgical treatment an aneurysm which had not definitely come to the surface. By the time that this has occurred there is almost invariably a thinning of the sac wall and the risk of rupture. In addition the surgeon cannot satisfy himself that the wire has filled the sac. I expect, therefore, that in cases of intrathoracic aneurysm the operation of wiring will be more or less confined to those desperate cases in which there is a definite pulsating tumour on the chest wall, with the prospect that at no distant date external rupture will occur. If wiring succeeds in prolonging life in even a small proportion of these cases, and very occasionally effects an apparent cure, then I think that the surgeon may reasonably claim that he has done all that could be expected of him.

Galvano-Puncture.—Formation of White Thrombi by Needling.—Ligature of Neck of Sac.

I should feel inclined to restrict the use of galvano-puncture to cases in which the aneurysm has made its way through the ribs or costal cartilages, and where external rupture appears imminent. Cases of this sort have from

time to time been recorded in which life has been prolonged in rapidly progressing cases, relief has been afforded to cough, paroxysmal pain, and pulsation, and finally death has taken place slowly and painlessly from gradual oozing from the thickened sac instead of by sudden rupture.

Sir William Macewen,[28] in a paper read before the Midland Medical Society in 1890, advocated the cure of aneurysm by inducing the formation of white thrombi within the sac. The instrument he employs is a cylindrical pin, tapering to a point, like an ordinary sewing needle, with rounded head at the other extremity. He gives the following directions for carrying out the treatment :—

> Before performing the operation the skin over the aneurysm ought to be carefully cleansed and rendered aseptic. The aseptic pin ought then to penetrate the sac and pass through its cavity until it comes in contact with the opposite side. It ought to touch and no more. Then one of two methods may be employed—either to move the pin over the surface of the inner wall so as to irritate its surface, or to allow the impulse of the blood-current playing on the very thin pin to effect the same object. ..... After acting thus for ten minutes at one part the point of the pin, without being removed from the sac, ought to be shifted to another spot, and so on until the greater portion of the internal surface opposite to the point of entrance has been touched ; this ought to be done in a methodical manner.

The pin should be allowed to remain in a few hours, though it has been kept in for 24 or even 48 hours. The operation may require to be repeated on several occasions at intervals of about a week. In his original paper[29] Sir William Macewen reports 4 cases—2 of intrathoracic aneurysm, 1 of the abdominal aorta, and 1 of the right femoral. One of the intrathoracic cases and both the remaining cases were apparently cured by the treatment. In the case of the aortic aneurysm which died, at the necropsy the sac contained a large amount of firm white thrombi. Death in this instance occurred from asphyxia due to pressure of the aneurysm upon the trachea.

Though many theoretical objections might be alleged against this plan of treatment, the cases which Sir William Macewen brought forward, though few in number, are so striking that I am astonished that it has not received more attention at the hands of surgeons. Sir William Macewen

wrote to me on this subject on Sept. 9th, 1912, as follows :—

I have had numerous successes since (i.e., 1890), patients living for many years and working as before, with care however.

Mr. Gilbert Barling[30] in his remarks at the discussion on the Surgical Treatment of Aneurysm, on the three principal forms of surgical treatment adopted in the case of aortic aneurysm, did not take an optimistic view of these methods.

He had never tried the introduction of wire, but he had tried electrolysis, and had been deeply disappointed with it, so that he was not likely to try it again. He had tried the operation of needling, but he did not think that the patient was either better or worse, and he was disappointed at the absence of improvement.

* * *

I think it doubtful whether surgeons in the future will attempt direct surgical interference in a case of thoracic aneurysm, but in view of the enormous strides that surgery has made of late years it seems impossible to limit their power of giving relief, if not cure. As an example of what has been attempted I may quote a case put on record by Tuffier.[31] The patient was a woman suffering from sacculated aneurysm of the first part of the arch of the aorta. As she was not more than 40 years of age, was in perfect general health, and was in imminent danger of rupture of the sac, Tuffier determined to ligature the sac close to its insertion into the aorta. This was done, but the aneurysmal sac was not removed. The omission was unfortunate, as the patient, who did well for some days after the operation, died on the thirteenth day from secondary hæmorrhage, due to gangrene of the sac and infection at the seat of ligature.

And here I must leave the question of the surgical treatment of thoracic aneurysm in the hands of the surgeons, hoping that in time to come they may be able to formulate some definite suggestions as to what kind of surgical interference is desirable and in what circumstances it should be adopted.

In concluding my lectures, I would again like to say how much indebted I am to Dr. Hugh Walsham for his generous

assistance in putting at my disposal his large experience in the radiography of aneurysm, and I have also to thank most warmly Dr. Braxton Hicks, assistant pathologist at the Westminster Hospital, for his careful investigation of the post-mortem records of the hospital, which enabled me to make use of the valuable information therein contained.

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