An Address

ON

THORACIC ANEURYSM.

DELIVERED BEFORE THE CLEVELAND DIVISION OF THE
BRITISH MEDICAL ASSOCIATION AT ORMESBY
HOSPITAL, MIDDLESBROUGH.

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WHEN you did me the honour of inviting me to give a lecture, and suggested as a title Thoracic Aneurysm, I felt that this was a subject which we in this particular part, the north-east coast of England, had many opportunities of seeing and treating, and, therefore, of expressing an opinion upon. Thoracic aneurysm may be one of the simplest diseases to diagnose—it may also be one of the most difficult. There is a type of aneurysm which is recognized by physical signs and another mainly by symptoms. Occasionally the disease is present without giving rise to any complaint on the part of a patient. A ruptured aneurysm is occasionally found at the *post-mortem* examination of persons who have died suddenly. Men are more liable to thoracic aneurysm than women. The proportion is given as nine males to one female, but in the Newcastle Infirmary it is probably nearer twelve than nine. It is a malady that affects men in the early part of the prime of life. Taking at random 17 cases of thoracic aneurysm from our infirmary patients I find that 12 were men between the ages of 35 and 45, 4 were between 45 and 55, while 3 of the men affected were between 55 and 60. It is during the strenuous part of a man's life that the diseased wall of the aorta yields before the strain to which it is exposed, the change commencing in the intima but outstripped in the course of time by the middle coat becoming the more affected.

Aneurysms are spoken of as *fusiform* and *sacculated*. In a fusiform aneurysm a segment of an artery is more or less regularly distended throughout its circumference, and it is spindle-shaped; but a sacculated aneurysm, formed as it is by a dilatation of a particular portion of the wall of an artery, is often in appearance not unlike a diverticulum of the intestine. It is in sacculated aneurysms that the middle coat of the vessel is found to have become thinned out in many instances to disappearance, and in which the interior is lined by laminated clotted blood, the layers next the wall of the aneurysm being pale and stratified, while internal to this the blood clot is red and soft. Sometimes, as in the drawing I show you, a crack occurs in the intima of the aorta and penetrates into the middle coat, often separating it into two layers, between which the blood forms a channel for itself which extends over several inches of the aorta, recommencing further on, so that practically speaking the greater part of the abdominal aorta may thus be canalized, and to appear on section as if composed of two vascular tubes, one contained within the other. This is known as the *dissecting* form of aneurysm. It is unattended by any special signs by which we can diagnose it during life. As it is to the sacculated form of thoracic aneurysm that we are going to give our attention to-day, I shall for the present dismiss fusiform aneurysm by saying that when this exists in the ascending and transverse parts of the arch of the aorta the dilatation generally extends into the large vessels arising from the arch, whereas in sacculated aneurysm this is not the case.

There is not the least doubt that in a very large percentage of the cases there is a history of syphilis. In at least 50 per cent. of the patients, probably more, syphilis has been the principal cause; and, when we add to this the influence of strain when at work and of alcohol, we have a triad of causes which when co-operating explain the origin of nearly all cases of thoracic aneurysm. A specific endarteritis or aortitis, therefore, is the most frequent precedent. In nearly all cases there has been some pre-existing disease of the walls of the aorta—of the middle coat in particular. There are, however, other causes than syphilis. Sudden strain and injury, by taking the heart and blood vessels, so to speak, unawares, may induce aneurysm, but whether these *per se* without previous disease of the walls of the aorta can cause aneurysm affords material for discussion.

The fact that a traumatic rupture of the external coat of the aorta may be followed by aneurysm is proof that injury and strain may be followed by protrusion of the remaining walls of the vessel; besides, aneurysm of the smaller arteries is known to be the consequence of embolism of these vessels by a clot rich in micro-organisms carried thither from the heart. We must not close our minds to the operation of other causes than syphilis. Horses occasionally suffer from thoracic aneurysm. I am not referring to the aneurysms met with in these animals in the branches of the mesenteric arteries, and due to the perforation of these vessels by the larvae of the strongylus, but to the rarer forms of aneurysm of the fore part of the aorta, and towards which strain has been the principal cause. It has been experimentally demonstrated by Josué that in even such vegetable feeders as rabbits atheroma of the aorta can be induced by the injection of suprarenal extract. The pathological changes induced in the aorta by means of adrenalin are in all respects similar to those which are observed in man. For the production of thoracic aneurysm there are required a diseased condition of the walls of the aorta, high arterial tension, and strain. Towards the first, toxæmic conditions of the blood other than those due to syphilis can contribute.

DIAGNOSIS.

The diseases that thoracic aneurysm may be mistaken for, and vice versa, are mediastinal tumours, malignant and other growths of the lungs, fibrosis of the lungs, malignant disease of the spine, and pulsating empyema. Thoracic aneurysm generally occurs in persons under 50 years of age, usually with all the appearance of health, whereas while in malignant disease the age may not be greater, there are frequently pallor and a certain amount of emaciation. It is only right to remember that when a thoracic aneurysm presses upon the thoracic duct the emaciation and pallor may be even greater and the loss of flesh much more rapid than in malignant disease of the lungs or mediastinum.

In the short time at my disposal it will be impossible to do more than give a brief review of some of the main points concerning the diagnosis and treatment of thoracic aneurysm, but, as the industries on the banks of the Tees are much the same as on those of the Tyne, and the habits of the workpeople are also practically the same, this is all that is required of me, since we are dealing with a disease that is common to the two districts, and one therefore with which you in Middlesbrough are just as familiar as I am. The labouring classes of the North of England work hard, they drink hard, and they are exposed to the vicissitudes of the weather, circumstances which all tend to induce structural changes in blood vessels.

SYMPTOMS.

If I were asked what I regarded as one of the earliest symptoms of thoracic aneurysm I would say *pain* in the chest—not always well defined or localized, usually worst at night and aggravated by exertion. When to the complaint of precordial pain, there is added a loud ringing accentuated second sound heard on auscultation over the aortic area in a healthy-looking man whose urine is free from albumen, the existence of thoracic aneurysm is extremely probable. There may be neither localized visible pulsation nor dullness detected on percussion, but to the hand placed over the upper part of the sternum there may be conveyed a distinct feeling of vibration which it is important to note. While the second sound of the heart is due to the closure and tension of the segments of the aortic and pulmonary valves there is also as a contributing element the shock that is given to the whole of the arterial tree, and as in aneurysm of the aorta the elasticity of the walls of the aorta is much impaired the shock or thud under these circumstances is much exaggerated.

Only a few patients can recall the particular day and the hour in which after some unusual strain, such as lifting a heavy weight, they were seized with pain in the chest and a sense of difficulty of breathing, symptoms which obliged the individual to rest for a few moments, or these were so slight as to be disregarded and, practically speaking, have been forgotten. In some such instances the sudden strain that dilated the aorta may also have produced aortic regurgitation either through injury of the valves or

by stretching the first part of the aorta. Here, too, the question arises, is it possible to have traumatic aortic regurgitation without pre-existing disease of the aorta or of the aortic valves? My answer is in the affirmative.

The pain in thoracic aneurysm may be explained in various ways. Apart from pressure upon adjacent parts and coexisting inflammation, increased tension of the walls of the aorta can of itself become a cause of pain either by stretching of the delicate nerves which run in the walls of the aorta or by a neuritis. Since pain is one of the earliest symptoms of thoracic aneurysm, its importance from a diagnostic point of view can be demonstrated by the administration of digitalis. This drug, administered under these circumstances, often augments the pain in the chest, since, by improving the force of the contraction of the left ventricle and by constricting the peripheral arteries, it raises the arterial tension. Increased stretching of the aortic walls or the heightened tension in a sacculated aneurysm must therefore be a cause of pain; and just as digitalis aggravates the pain by raising the arterial tension, so vascular depressants, such as iodide of potassium, relieve pain by taking off this pressure. As the aneurysmal sac grows larger it may cause pain by pressing upon sensory nerves, and involving them in the inflammatory exudation. When an aneurysm of the descending aorta presses upon the posterior spinal roots, or has become incorporated with the periosteum of the vertebrae and is eroding the spine, pain of an excruciating character is often complained of, and calls for the hypodermic administration of morphine. On the other hand, it is well to remember that intrathoracic aneurysm may reach a great size without giving rise to pain or any very distressing symptom.

In cases of arterio-sclerosis accompanied by high arterial tension the left ventricle becomes enlarged, and there is heard over the aortic area a loud accentuated second sound. One consequence of the excessive strain imposed upon the ascending part of the arch of the aorta is that the wall of the vessel dilates, having been previously the seat of aortitis—a lesion which may silently extend downwards to the aortic cusps and induce aortic incompetence; or the mouth of the coronary arteries becomes involved, and, as a result of the malnutrition of the heart, the left ventricle dilates, or pain of the nature of angina pectoris is experienced.

Among the physical signs, dullness on percussion and pulsation seen or felt over a limited area are very important. When to these are added vascular bruits, the diagnosis of aneurysm is pretty complete, always barring the existence of such a growth as a very vascular sarcoma. So far as murmurs are concerned, a systolic murmur is the more frequent. It is due to the rush of blood from the aorta through the constricted orifice of the aneurysm throwing the blood in the aneurysm into eddies and inducing vibration of the walls of the sac. That vibration of the walls of the aneurysm contributes largely to the causation of the murmur is shown by the fact that if by any external adhesions the aneurysmal sac is bound down, or if the interior of the aneurysm is lined with thick layers of laminated clot, a systolic murmur may be entirely absent. In many large aneurysms there is no bruit heard at all, and such will sometimes be the case if the orifice between the aorta and the aneurysm is pretty large. If a systolic murmur when heard over an aneurysm is due to the inrush of blood and vibration of the walls of the sac, so a diastolic murmur might likewise be due to the sac of the aneurysm contracting and forcing blood out again into the aorta, but as a matter of experience a diastolic murmur heard under these circumstances is generally due to concurrent aortic regurgitation. It is the coexistence of aortic regurgitation, and not the presence of a thoracic aneurysm, that explains the hypertrophy of the left ventricle observed in these cases. To the hand placed over the seat of an aneurysm in the chest, even in the absence of a systolic murmur, there is frequently conveyed the sense of shock caused by the closure of the aortic valves and vibration of the aorta generally.

For clinical purposes not less than for anatomical, the arch of the aorta is divided into three portions—ascending, transverse, and descending. Aneurysms arising just above the aortic valves may be so small as to escape detection, and not reveal themselves until they rupture into the pericardium, when they cause death. In some of these

cases faintness on getting out of bed in the morning has been the only symptom complained of. It is from the convex side of the first part of the arch of the aorta that aneurysms most frequently spring, since this is the part of the aorta that has to bear the main force of the contraction of the left ventricle, and it is from this part of the aorta that aneurysms may reach fairly large size without causing serious distress. The aneurysm may grow forward and show itself as a pulsating tumour in the second and third right interspaces—part of the intercostal cartilages or ribs having become eroded. Large pulsating swellings may thus be found on the right chest with very little but the skin in front of them. These are the aneurysms of physical signs. Instead of coming forward the aneurysm may grow towards the right lung and project into the pleural cavity, compressing the lung and causing the respiratory murmur on this side of the chest to be extremely feeble; or the sac may grow towards the superior vena cava, and subsequently open into it, giving rise to an arterio-venous aneurysm, the distinguishing sign of which is the presence of a continuous souffle; it may grow upwards, and by pressing upon the right subclavian vein cause oedema of the arm; or it may press upon the right recurrent nerve, and cause paresis or paralysis of the right vocal cord. When aneurysms arise from the concave side of the first part of the arch they may present at the left border of the sternum, and cause displacement of the heart.

Aneurysms of the transverse portion of the arch, even when small, will give rise to serious symptoms if they grow from the posterior wall, for, owing to the confined space, the trachea and oesophagus are readily pressed upon, and respiration and deglutition unpleasantly interfered with. It is when an aneurysm arises in this particular position that the sign known as "tracheal tugging" is best marked. Cough is frequently a distressing symptom; it is paroxysmal. Inspiration is attended with stridor. When the aneurysm grows forward there may be dullness over or at one side of the manubrium sterni, or the tumour may grow upwards and appear in the episternal notch. Frequently the left recurrent laryngeal nerve is pressed upon, and there occurs paralysis of the left vocal cord. To this subject I will return. In a female patient recently under my care the right sterno-cleido-mastoid muscle was extremely tender on pressure compared with the left; and in one of my male patients with aneurysm of transverse part of the arch, the right sterno-mastoid muscle was much smaller than the left. Signorelli of Rome states that he too found a distinctly attenuated condition of the left sterno-mastoid muscle in a patient in whom an aneurysm arose from the junction of the transverse and descending portions of the arch of the aorta, and which he attributed to an ascending degeneration of vago-spinal nerve fibres proceeding to the larynx. Aneurysms springing from the transverse part of the aortic arch may press upon the left bronchus, and by diminishing the quantity of air entering the left lung give rise to enfeebled respiratory murmur, or the thoracic duct may be encroached upon and starvation be slowly produced. The close proximity of the sympathetic chain of ganglia renders them liable to pressure, whereby when the pressure is slight, so as only to cause irritation, the pupil of the eye on the same side is dilated, but if the pressure is such as to cause paralysis of the sympathetic the pupil becomes contracted owing to the action of the third cranial nerve.

Aneurysms of the descending thoracic aorta when they pass backwards become incorporated with the periosteum of the vertebrae and cause erosion of the bones. There is induced a rarefying osteitis with absorption of the bone. When the erosion has extended into the spinal canal the cord is pressed upon and there is paralysis of the lower limbs. Pain, often of an excruciating character, is experienced during the process of erosion. Sometimes a pulsating swelling appears to the left of the spinal column.

Let us revert for a minute or two to the distressing symptom of paralysis of the vocal cords due to a thoracic aneurysm pressing upon the recurrent laryngeal nerves. On the right side the nerve winds round the subclavian artery, on the left round the arch of the aorta. With the exception of the tensor muscles of the vocal cords, the crico-thyroids, which are supplied by the superior laryngeal nerves, the motor mechanisms concerned with voice—namely, the abductors and adductors of the vocal

cords—are innervated by the recurrent laryngeal nerves. Risien Russell has shown that the recurrent laryngeal nerve can be split up in its peripheral length into three distinct bundles—one of which supplies the abductors of the vocal cords, another the adductors, whilst from the third no motor effects are obtained. The interesting point, therefore, is that it is the same nerve that goes to the abductors and adductors, only the fibres are differentiated in their course. These fibres come from the vagus and spinal accessory nerves. During inspiration and expiration the vocal cords form an isosceles triangle the sides of which move very little during the two phases of respiration. After death the cords occupy the cadaveric position, that is, only half their previous width. The larger width of the glottis during life is due to the abductor muscles being endowed with greater tonus, a kind of special reflex whereby, as Sir Felix Semon¹ maintains, the glottis is kept open during life throughout both phases of ordinary respiration. When during life the glottis is closed, there is dyspnoea, the breathing is quick and shallow, or there is deep and laboured inspiration, accompanied by audible inspiratory stridor. The narrowest part of the respiratory tube is the larynx, for by the introduction of the phonatory apparatus its width is reduced one-third, so that if the narrowing were not counterbalanced by some compensatory arrangement of nature, there would not be sufficient space for the entrance of air into the lungs when any extra demand was made upon the breathing powers. All this is obviated, Sir Felix Semon states, by the abductor muscles of the larynx being provided with the tonus already mentioned, so that the glottis is always kept sufficiently open for breathing purposes. When the recurrent laryngeal nerves are cut across, all the muscles supplied by them are paralysed, but owing to the removal of the abductor tonus the vocal cords tend to become drawn into the cadaveric position. In disease, when pressure symptoms develop slowly, the abductors always suffer more than the adductors, and tend to undergo atrophy and degeneration much more rapidly. Abduction paralysis represents the initial stages of complete paralysis of the recurrent laryngeal nerves. The abductor nerve twigs are more excitable to electricity than those going to the adductor muscles. In cases of pressure upon the recurrent laryngeal nerves, the function of the vocal cord that is first lost is always that of abduction. Whether this is due to the different manner in which the nerve fibres end in the abductors and adductors is a question we may well leave to laryngologists and others to settle. It is important for us to recognize the greater vulnerability of the abductor muscles, for once the abductor tonus is lost the cords are almost immediately drawn into the cadaveric position, and, as both sets of muscles are impaired, there occurs a paralytic contraction of the adductor fibres owing to the absence of antagonistic influences, and as the adductor shortening becomes permanent the cords are kept closed. The paralysed cord is drawn into the middle line, where it remains immobile, and yet, when only one cord is affected, all this may occur without giving rise to symptoms.

In thoracic aneurysm there is often detected inequality of the radial and temporal pulses—a circumstance that is sometimes of value from a localizing point of view. When an aneurysm springs from a part of the arch of the aorta, either before or after giving origin to the large vessels, there is not usually observed any difference in the pulses, although the pulse wave itself is frequently modified, for in the sphygmographic tracing some of the waves are obliterated and the apex of the tracing is lost. When an aneurysm involves the innominate artery, it is the right radial pulse that is weakened and modified in character, although, as in this black-and-white drawing of a case of fusiform dilatation of the innominate artery, from the upper part of which a small sacculated aneurysm arises, the left temporal pulse in the patient could scarcely be felt owing to the displacement of the trachea to the left and compression of the left carotid artery; yet the pulse of the right was readily appreciable. An aneurysm involving the arch of the aorta, and situated between the origins of the innominate and left subclavian artery, is almost sure to render the left radial pulse the weaker of the two, and to modify its character. By means of these drawings of aneurysms of the thoracic aorta of patients that were under my care, I am able to draw your attention to the fact that occasionally the mouth of one

or other of the large vessels springing from the aortic arch may become almost entirely blocked by firmly adherent clot, so that by this circumstance, and not the situation of the aneurysm, the weakness of the pulse on the corresponding side is to be explained. The inequality of the pulses, therefore, while in many instances a guide to the localization of the aneurysm, is not always a reliable guide.

The mode of rupture of aneurysms is an interesting study. While in some cases the rupture is sudden and death comes quickly when the hæmorrhage is severe, or from asphyxia when the blood has been poured in quantity into the trachea, the sac in many instances simply leaks and only a small amount of blood escapes. The classic case recorded by my old friend and teacher, Sir William Gairdner, of the man whose aneurysm presented in the neck above the episternal notch, and who, when the sac ruptured, held out at arm's length a bowl at the far end of the red arch to catch the blood, and who ultimately died not from aneurysm at all, is probably known to many of you. I have seen a few patients die suddenly from rupture of aneurysm, but in a very large number of cases of aneurysms that come to the surface the skin becomes thin, red and reddish-blue at two or three places, and while the sac seems on the point of bursting, and of giving rise to a fatal hæmorrhage, only a few drops of blood escape and continue to do so. This leaking goes on for days or the minute openings become blocked, and then, after a violent fit of coughing, a larger rupture occurs, and, a considerable quantity of blood escaping, the patient faints and recovers, but only to live in constant dread of a final recurrence which, in one sense happily for the sufferer, is not long delayed. What I have said of the mode of rupture of external aneurysms applies equally to those that open internally. Take the case, for example, of an aneurysm that has been pressing upon the trachea and which has opened into it. If you examine the mucous membrane of the trachea over the site of the aneurysm, it will be observed that there are several small openings in the plate of tissue separating the interior of the windpipe from that of the aneurysm, and through this cribriform plate the bleeding that caused death took place. In one of my patients, a woman, whose aneurysm pointed at the upper part of the sternum and which kept oozing for several days, the interior of the sac became septic. She ultimately died from a large rupture of the sac.

TREATMENT.

As regards the treatment of thoracic aneurysm, rest in bed and quietude are essential, accompanied by a restricted diet. Cases of thoracic aneurysm unaccompanied by aortic incompetence are more amenable to treatment than those arising just above the aortic orifice and accompanied by aortic regurgitation, for in these there cannot be that calmness in the interior of the sac which is necessary for coagulation of the blood. The administration of iodide of potassium gives on the whole the best results. Pain and paroxysmal dyspnoea are frequently relieved by it. I have administered to some of my patients as much as 450 gr. of potassium iodide per diem, but there is nothing to be gained by giving such large doses. In some instances the results will not be so satisfactory, for when pushed beyond a certain limit potassium iodide lowers the blood pressure and quickens the action of the heart that the circulation in the aneurysm is too excited for the deposition of clot to occur. Since calcium salts are known to favour coagulation of the blood I have administered these in various forms, but without obtaining results as satisfactory as those from potassium iodide alone. Even with this potassium salt there are few real cures of thoracic aneurysm, although great relief is obtained. While in distal aneurysms spontaneous clotting of blood may occur that leads to cure, this, owing to the situation, is very seldom the case in thoracic aneurysms. I have had no experience of carrying silver wire into the interior of an aneurysm nor of needling. Several cases of aneurysm I have treated by the subcutaneous injection of gelatine into the tissues in the neighbourhood of the sac. Some of the patients complained of great pain in the part afterwards, while in others there was always a slight rise of temperature and considerable discomfort. In a few of the patients thus treated the pulsations of the aneurysm became less marked and the tumour became harder and shrunk. Several of

the patients left the infirmary much better, but the cure was never complete, for in some of them the sac ruptured weeks or months afterwards. Apart from the pain which the subcutaneous injection of gelatine causes there is another risk that has to be borne in mind. Tetanus followed gelatine injections in one of the London hospitals, and as it is extremely difficult to sterilize gelatine, this risk, although no doubt remote, must be borne in mind. I have had a limited experience of galvanopuncture; I have seen one case of subclavian aneurysm thus treated and in which for several months the result was satisfactory until, after friction caused by the workman carrying his spade on the affected shoulder, the aneurysm redeveloped and burst into the surrounding tissues, causing a haematoma which began to ooze through the skin, but in which the blood subsequently coagulated, and the aneurysm was finally removed. One of the difficulties of treating thoracic aneurysm is in getting the subjects of the disease to be patient under treatment. Rest in bed and the restricted diet in time become irksome to the patients, who, once they feel rather better, long for freedom. If, when the pulsation of the tumour has lessened and the sac has become smaller and harder, patients can be got still to carry out the rest cure, the results are, as might be expected, more satisfactory.

REFERENCE.

1 BRITISH MEDICAL JOURNAL, 1898, vol. i, p. 1. The Diagnostic Significance of Laryngeal Abductor Paralysis, Sir Felix Semon.

A Lecture

ON

AORTIC DISEASE.

DELIVERED AT THE LONDON SCHOOL OF CLINICAL
MEDICINE.

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FROM the point of view of etiology, this hospital furnishes an instructive object lesson in cardiac disorders. Its wards are never without cases of aortic disease; whereas mitral lesions, except such as are secondary, are seldom found. As you know, our patients are all seafaring men, who, whether as seamen or firemen, are, by the nature of their work, exposed to the wear and tear of an arduous life which involves irregularity of habits, both as regards food and sleep, hard physical work, and exposure to all varieties of weather and climate. Toilers on the deep are also liable to other dangers which militate strongly against the best interests of their health. They are not more intemperate than other classes of men—perhaps less so than many—for, in the nature of their occupation, they are perforce compelled, during the continuance of each voyage, to abstain from alcohol in every form. As a consequence, when they finally get into port, they are apt to rush to the other extreme, and, in the first gladness of being once more ashore, too frequently squander most of the wages they have so hardly earned by over-indulgence in alcoholic drinks which, as supplied to them, are potent and impure. The publican knows from experience that his sea-going customers like their drinks strong, and, by various methods of adulteration and faking, he succeeds in catering to their tastes. The sailor, therefore, who indulges in alcohol at all—and most of them do—not only takes it from time to time in excessive quantity, but he has also to accept it of villainous quality. The sailor, again, is no exception to the common rule that, under the influence of alcohol, he readily succumbs to the seductive attractions of Eve. His necessary continence throughout a voyage encourages a sudden lapse from virtue when the circumstances become propitious, and the Legislature is so little concerned for the welfare of those who go down to the sea in ships and on whom the commerce of the country so largely depends that it takes no trouble to supervise and regulate this traffic. In every seaport prostitution of the most degraded type is rampant, and of those who give way to temptation few escape without the penalty of venereal disease. It cannot be wondered that, under such loose regulations, syphilis is prevalent among seamen, and unfortunately its

early manifestations are usually regarded so nonchalantly that, in most instances, the disease has reached its secondary or tertiary manifestations before its victims come under treatment. Climatic exposure, strenuous toil, alcoholic excesses, and venereal infections combine to undermine the sailor's health and predispose him especially to arterial disease and the many serious consequences thereof. Therefore it is that in a hospital such as this we find unusual opportunities for studying not only all varieties of venereal and alcoholic disease, but also the many secondary disorders for which one, or other, or both are primarily responsible.

It is remarkable that acute rheumatism is not frequently met with among seafaring men. One would expect that the constant exposure to cold and wet which is their almost daily experience would cause rheumatic fever to be a frequent ailment among them; but it is not. Chronic rheumatism is common enough, but the acute manifestations of the disease are comparatively rare. Sailors themselves tell us that they never catch cold or rheumatism from salt water; and though it is difficult to see why this should be, it is at least offered as an explanation of their immunity, and rather fortifies the opinion that acute rheumatism is a different disease entirely from chronic rheumatism, and that its proper position in nosology is among the infective diseases. This immunity from rheumatic fever is further testified by the fact I have already referred to, of the absence from these wards of mitral disease. Rheumatic endocarditis may involve any or all the valves of the heart; but we know that the mitral valves are specially susceptible to its influence, and in severe cases seldom escape some degree of impairment.

Aortic disease, which is the subject with which we are specially concerned to-day, may be a sequela of acute rheumatism or it may be the result of degenerative changes in the arteries or heart. I am able to show you a case of each variety, because, fortuitously, one of the patients before you has suffered recently from rheumatic fever, to which event he refers, and probably rightly, the commencement of the cardiac disturbance which has brought him here. The other man is an example of that form of aortic disease with which we are daily made familiar in this hospital. Time does not enable me to enter minutely into their histories, but it will suffice for our purpose that I should tell you that these two patients resemble one another in their habits and occupation. Both have indulged themselves bountifully in alcohol, both have had syphilis, and both have been exposed to the rigours and vicissitudes of a seafaring life. The main differences between them are that the one man is younger and has suffered from an acute rheumatic attack which lasted over several weeks and involved many of his joints; the other is older by twelve years and, so far as he remembers, has never been seriously ill, though now he complains of stiffness and pains in some of his larger joints, which he attributes to rheumatism. When you come to examine their physical condition, you will find that both patients have a double aortic murmur, and present the usual signs and symptoms by which we are accustomed to recognize this condition. I would specially draw your attention, as you look at the men, to the tortuous and locomotive arteries, the pulsation in the suprasternal notch, the heaving cardiac impulse extending beyond the nipple line, and the absence of cyanosis or dyspnoea. These facts tell us a great deal, because we may infer from them that the vessels are thickened, that the aorta is dilated, that the heart is hypertrophied, and that compensation is well maintained. On palpation the impulse is extensive and vigorous, and you will observe in this patient that two of the intercostal spaces between the nipple line and the sternum are depressed with each cardiac systole. This is usually ascribed to pericardial adhesions, and it is not impossible that such may be the cause in this instance, but it should not be forgotten that where we have as great hypertrophy as exists here, the effect of atmospheric pressure in filling up the space created by systolic contraction of the enlarged ventricle is enough to account for thoracic recession. Percussion shows an increased area of dullness in both cases, which extends, as the apex thrust tells us, mostly down and to the left. When we come to examine the pulse it is found to possess, in both men, the water-hammer characteristics which belong to aortic regurgi-