II. CHRONIC VALVULAR DISEASE.

Aortic Incompetency.

Incompetency of the aortic valves arises either from inability of the valve segments to close an abnormally large orifice or more commonly from disease of the segments themselves. This best-defined and most easily recognized of valvular lesions was first carefully studied by Corrigan, whose name it sometimes bears.

Etiology and Morbid Anatomy.—It is more frequent in males than in females, affecting chiefly able-bodied, vigorous men at the middle period of life. The ratio which it bears to other valve diseases has been variously given from thirty to fifty per cent.

Among the important factors in producing this condition are: (*a*) Congenital malformation, particularly fusion of two segments—most commonly those behind which the coronary arteries are given off. It is probable that an aortic orifice may be competent with this bicuspid state of the valves, but a great danger is the liability of these malformed segments to sclerotic endocarditis. Of seventeen cases which I have reported all presented sclerotic changes, and the majority of them had, during life, the clinical features of chronic heart-disease.

(*b*) Acute endocarditis. This does not produce aortic incompetency unless the process passes on to ulceration and destruction, under which circumstances it is often found, and may cause a rapidly fatal issue. Simple endocarditis associated with the specific fevers is not nearly so common on the aortic as on the mitral segments; so also with rheumatism, which plays a less important *rôle* here than in mitral valve disease.

(*c*) By far the most frequent cause of insufficiency is the slow, progressive sclerosis of the segment, resulting in a curling of the edge, which lessens the working surface of the valve. This may, of course, follow acute endocarditis, but it is so often met with in strong, able-bodied men among the working classes, without any history of rheumatism or special febrile diseases with which endocarditis is commonly associated, that other conditions must be sought for to explain its frequency. Of these, unquestionably strain is the most important—not a sudden, forcible strain, but a persistent increase of the normal tension to which the segments are subject during the diastole of the ventricle. Of circumstances increasing this tension, heavy and excessive use of the muscles is perhaps the most important. So often is this form of heart-disease found in persons devoted to athletics that it is sometimes called the "athlete's heart." Alcohol is a second important factor, and is stated to raise considerably the tension in the aortic system. A combination of these two causes is extremely common. A third element in inducing chronic sclerotic changes in these valves is syphilis. Cases are rarely seen in which other factors must not be taken into account, but the association is too

frequent to be accidental. That syphilis is capable of inducing arterial sclerosis is, I think, acknowledged, although the way in which it is done is not yet clear. It is interesting to note with what frequency this form of valve disease occurs in soldiers. I was struck with this fact in the Philadelphia Hospital, to which so many veterans of the civil war are admitted. I was in the habit of enforcing upon my students the etiological lesson by a mythological reference to Bacchus and Vulcan, at whose shrines a majority of the cases of aortic insufficiency have worshipped, and not a few at that of Venus.

The condition of the valves is such as has already been described in chronic endocarditis. It may be noted, however, how slight a grade of curling may produce serious incompetency. Associated with the valve disease is, in a majority of the cases, a more or less advanced arterio-sclerosis of the arch of the aorta, one serious effect of which may be a narrowing of the orifices of the coronary arteries. The sclerotic changes are often combined with atheroma, either in the fatty or calcareous stage. This may exist at the attached margin of the valves without inducing insufficiency. In other instances insufficiency may result from a calcified spike projecting from the aortic attachment into the body of the valve, and so preventing its proper closure. Some writers (Peter) have laid great stress upon the extension of the endarteritis to the valve, and would separate the instances of this kind from those of simple valvular endocarditis. I must say that I have not been able to recognize clinical differences between these two conditions, though anatomically we may separate the cases into two groups—those with and those without arterio-sclerosis.

(*d*) And, lastly, insufficiency may be induced by rupture of a segment—a very rare event in healthy valves, but not uncommon in disease, either from excessive strain during heavy lifting or from the ordinary endarterial strain in a valve eroded and weakened by ulcerative endocarditis.

Relative insufficiency of the sigmoid valves, due to dilatation of the aortic ring, is a rare condition. It is said to occur in extensive arterial sclerosis of the ascending portion of the arch with great dilatation just above the valves. I have myself never met with a pure instance of the kind, for in such cases I have always found the valve segments involved with the arterial coats. In aneurism just above the aortic ring, relative insufficiency of the valve may be present.

It would appear from the careful measurements of Beneke that the aortic orifice, which at birth is 20 mm., increases gradually with the growth of the heart until at one and twenty it is about 60 mm. At this it remains until the age of forty, beyond which date there is a gradual increase in the size up to the age of eighty, when it may reach from 68 to 70 mm. There is thus at the very period of life in which sclerosis of the valve is most common a physiological tendency toward the production of a state of relative insufficiency.

The insufficiency may be combined with various grades of narrowing, but the majority of the cases of aortic insufficiency present no signs of stenosis. On the other hand, cases of aortic stenosis almost without exception are associated with some grade, however slight, of regurgitation.

The direct effect of aortic insufficiency is the regurgitation of blood from the artery into the ventricle, causing an overdistention of the cavity and a reduction of the blood column; that is, a relative anæmia in the arterial tree. As an immediate effect of the double blood-flow into the left ventricle dilatation of the chamber occurs, and finally hypertrophy. In this way the valve defect is compensated and as with each ventricular systole a larger amount of blood is propelled into the arterial system, the regurgitation of a certain amount during diastole does not, for a time at least, seriously impair the nutrition of the peripheral parts. In this valve lesion dilatation and hypertrophy reach their most extreme limit. The heaviest hearts on record are described in connection with this affection. The so-called bovine heart, *cor bovinum*, may weigh 35 or 40 ounces, or even, as in a case of Dulles's, 48 ounces. The dilatation is usually extreme, and is in marked contrast to the condition of the chamber in cases of pure aortic stenosis. The papillary muscles may be greatly flattened. The mitral valves are usually not seriously affected, though the edges may present slight sclerosis, and there is often relative incompetency, owing to distention of the mitral ring. Dilatation and hypertrophy of the left auricle are common, and secondary enlargement of the right heart occurs in all cases of long standing. The myocardium usually presents changes, fibroid or fatty; more commonly the former in association with disease of the coronary arteries. The arch of the aorta may present extensive arterio-sclerosis and dilatation. In rare instances, usually the rheumatic cases, the intima is perfectly smooth, and the arch with its main branches not dilated. This condition may be found post mortem even when during life there have been the most characteristic signs of enlargement of the arch and of dilatation of the innominate and right carotid. I have even known the condition of aneurism to be diagnosed when post mortem no trace of dilatation or sclerosis was found, only an extreme grade of insufficiency with enormous dilatation and hypertrophy. The coronary arteries are usually involved in the sclerosis, and their orifices may be much narrowed. Although these vessels have been shown by Martin and Sedgwick to be filled during the ventricular systole, the circulation in them must be embarrassed in aortic incompetency. They must miss the effect of the blood-pressure in the sinuses of Valsalva during the elastic recoil of the arteries, which surely aids in keeping the coronary vessels full. The arteries of the body usually present more or less sclerosis consequent upon the strain which they undergo during the forcible ventricular systole.

Symptoms.—The condition is often discovered accidentally in persons who have not presented any features of cardiac disease.

Physical Signs.—*Inspection* shows a wide and forcible area of cardiac impulse with the apex beat in the sixth or seventh interspace, and perhaps as far out as the anterior axillary line. In young subjects the præcordia may bulge. On palpation a thrill, diastolic in time, is occasionally felt, but is not common. The impulse is usually strong and heaving, unless in conditions of extreme dilatation, when it is wavy and indefinite. *Percussion* shows a greater increase in the area of heart dulness than is found in any other valvular lesion. It extends chiefly downward and to the left.

On *auscultation* there is heard a murmur during diastole in the second right interspace, which is propagated with intensity toward the ensiform cartilage or down the left margin of the sternum toward the apex. In the majority of cases it is a soft, long-drawn *bruit*, and is of all cardiac murmurs the most reliable. It occurs during the time of, and is produced by, the reflux of blood from the aorta into the ventricle. In a large proportion of the cases there is also a systolic murmur heard at the aortic region, usually shorter, often rougher in quality, and which may be propagated upward into the neck. A common mistake is to regard this as indicating stenosis, whereas in the great majority of instances of aortic insufficiency there is no material narrowing, and the murmur is produced by roughening of the segments or of the intima of the arch. The second sound is usually obliterated, though in some instances both the murmur and the valvular sound may be distinctly heard. At the apex murmurs are also heard, either transmitted from the aortic orifice or produced at the mitral. In the majority of cases with aortic incompetency of high grade, the mitral orifice is dilated, and there is relative insufficiency of the valves. It can frequently be determined that the systolic murmur at the apex differs in quality from that at the base. A second murmur at the apex, probably produced at the mitral orifice, is not infrequent. Attention was called to this by the late Austin Flint, and the murmur usually goes by his name. It has a distinctly rumbling quality, is limited in area, and is sometimes, though not always, distinctly presystolic in time. The explanation of its occurrence, as given by Flint, is that in the extreme dilatation of the ventricle the mitral segments cannot during diastole be forced back against the wall, and, therefore, remaining in the blood current, they produce a sort of relative narrowing, and in consequence a vibratory murmur not unlike in quality the presystolic murmur of mitral stenosis. My experience as to the frequency of this murmur coincides with that of Lee.*

The examination of the arteries in aortic insufficiency is of great value. Visible pulsation is more commonly seen in the peripheral vessels in this than in any other condition. The carotids may be seen to throb forcibly, the temporals to dilate, and the brachials and radials to expand with each

* American Journal of the Medical Sciences, 1890.

heart-beat. With the ophthalmoscope the retinal arteries are seen to pulsate. Not only is the pulsation evident, but the characteristic jerking quality is apparent. In the throat the throbbing carotids may lead to the diagnosis of aneurism. In many cases the pulsation can be seen in the suprasternal notch, and prominent, forcibly-throbbing vessels beneath the right sterno-mastoid muscle. The abdominal aorta may lift the epigastrium with each systole. To be mentioned with this is the capillary pulse, met very often in aortic insufficiency, and best seen in the finger-nails or by drawing a line upon the forehead, when the margin of hyperæmia on either side alternately blushes and pales. In extreme grades the face or the hand may blush visibly at each systole. It is met with also in profound anæmia, occasionally in neurasthenia, and in health in conditions of great relaxation of the peripheral arteries. Pulsation may also be present in the peripheral veins. On palpation the characteristic water-hammer or Corrigan pulse is felt. On the majority of instances the pulse wave strikes the finger forcibly with a quick jerking impulse, and immediately recedes or collapses. The characters of this are sometimes best appreciated by grasping the arm above the wrist and holding it up. On auscultation a double murmur may be heard in the carotids and subclavians when it is present at the aortic orifice. Occasionally in the carotid the second sound is distinctly audible when absent at the aortic cartilage. In the femoral artery a double murmur also may be heard sometimes, as pointed out by Duroziez.

Aortic insufficiency may for years be fully compensated. Persons do not necessarily suffer any inconvenience, and the condition is often found accidentally. So long as the hypertrophy just equalizes the valvular defect there may be no symptoms and the individual may even take moderately heavy exercise without experiencing sensations of distress about the heart. The cases which last the longest are those in which the sclerosis follows endocarditis and is not a part of a general arterio-sclerosis. Coexistent lesions of the mitral valves tend early to disturb the compensation. It has scarcely been sufficiently recognized by the profession at large that pure aortic insufficiency is consistent with years of average health and with a tolerably active life. I know several physicians with aortic insufficiency who have been able to carry on for years large and somewhat onerous practices. One of them since the establishment of insufficiency has passed successfully through two attacks of acute rheumatism. In large hospital practice, scarcely a month passes without the discovery of a case of aortic insufficiency in connection with some other affection.

With the onset of myocardial changes, with increasing degeneration of the arteries, particularly with a progressive sclerosis of the arch and involvement of the orifices of the coronary arteries, the compensation becomes disturbed. In advanced cases the changes about the aortic ring may be associated with alterations in the cardiac nerves and ganglia, and so introduce an important factor.

Headache, dizziness, flashes of light, and a feeling of faintness on rising quickly are among the earliest symptoms. Palpitation and cardiac distress on slight exertion are common. Long before any signs of failing compensation pain may become a marked and troublesome feature. It is extremely variable in its manifestations. It may be of a dull, aching character confined to the præcordia. More frequently, however, it is sharp and radiating, and is transmitted up the neck and down the arms, particularly the left. Attacks of true angina pectoris are more frequent in this than in any other valvular disease. Anæmia is also common, much more so than in aortic stenosis or in mitral affections.

More serious symptoms, as compensation fails, are shortness of breath and œdema of the feet. The attacks of dyspnœa are liable to come on at night and the patient has to sleep with the head high or even in a chair. Of respiratory symptoms cough may develop, due to the congestion of the lungs or œdema. Hæmoptysis is less frequent than in mitral disease. I have reported a case in which it was profuse and believed to be due to tuberculosis of the lungs, inasmuch as the patient was admitted in a state of emaciation and profound exhaustion. General dropsy is not common, but œdema of the feet may occur early and is sometimes due to the anæmia, at others to the venous stasis, at times to both. Unless there is coexisting disease of the mitral valve, it is rare in pure aortic incompetency for the patient to die with general anasarca. Sudden death is frequent; more so in this than in other valvular diseases. As compensation fails the patient takes to bed and slight irregular fever, associated usually with a recurring endocarditis, is not uncommon toward the close. Embolic symptoms are not infrequent—pain in the splenic region with enlargement of the organ, hæmaturia, and in some cases paralysis. Distressing dreams and disturbed sleep are more common in this than in other forms of valvular disease.

Here may appropriately be mentioned the connection between mental symptoms and cardiac disease, as they are oftenest seen with this lesion. An admirable account of the relations between insanity and disease of the heart is to be found in Mickle's Gulstonian lectures for 1888. In general medical practice we seldom find marked mental symptoms, except toward the close of the disease, when there may be delirium, hallucinations, and morbid impulses. It is to be remembered that in many heart cases this terminal delirium is uræmic. The irritability and peevishness sometimes found in persons the subject of organic heart-disease cannot, I think, be associated with it in any special manner. We do meet insanity, breaking out in patients with aortic and mitral disease, in the stage of compensation, which appears to be related definitely to the cardiac lesion. It is important to bear this in mind, for cases occasionally display suicidal tendencies. I have twice had patients throw themselves from the window of the ward.