Aortic Stenosis.

Narrowing or stricture of the aortic orifice is not nearly so common as insufficiency. The two conditions, as already stated, may occur together, however, and probably in almost every case of stenosis there is some leakage.

Etiology and Morbid Anatomy.—In the milder grades there is adhesion between the segments, which are so stiffened that during systole they cannot be pressed back against the aortic wall. The process of cohesion between the segments may go on without great thickening, and produce a condition in which the orifice is guarded by a comparatively thin membrane, on the aortic face of which may be seen the primitive raphés separating the sinuses of Valsalva. In some instances this membrane is so thin and presents so few traces of atheromatous or sclerotic changes that the condition looks as if it had originated during fœtal life. More commonly the valve segments are thickened and rigid, and have a cartilaginous hardness. In advanced cases they may be represented by stiff, calcified masses obstructing the orifice, through which a circular or slit-like passage can be seen. The older the patient the more likely it is that the valves will be rigid and calcified.

We may speak of a relative stenosis of the aortic orifice when with normal valves and ring the aorta immediately beyond is greatly dilated. A stenosis due to involvement of the aortic ring in sclerotic and calcareous changes without lesion of the valves is referred to by some authors. I have never met with an instance of this kind. A subvalvular stenosis, the result of endocarditis in the mitro-sigmoidean sinus, usually occurs as the result of a fœtal endocarditis. In comparison with aortic insufficiency, stenosis is a rare disease. It is usually met with at a more advanced period of life than insufficiency, and the most typical cases of it are found associated with extensive calcareous changes in the arterial system in old men.

When gradually produced and when there is not much insufficiency the dilatation of the left ventricle may be slight, though I think that in all cases it does occur. The walls of the ventricle become hypertrophied, and we see in this condition the most typical instances of what is called concentric hypertrophy, in which, without much, if any, enlargement of the cavity, the walls are greatly thickened, in contradistinction to the so-called eccentric hypertrophy, in which the chamber is greatly dilated as well as hypertrophied. There may be no changes in the other cardiac cavities if compensation is well maintained; but with its failure come dilatation, impeded auricular discharge, pulmonary congestion, and increased work for the right heart. The arterial changes are, as a rule, not so marked as in aortic insufficiency, for the walls have not to withstand the impulse of a greatly increased blood-wave with each systole. On the contrary, the amount of blood propelled through the narrow orifice may be smaller than normal, though when compensation is fully established the pulse-wave may be of medium volume.

Symptoms. Physical Signs.—*Inspection* may fail to reveal any area of cardiac impulse. Particularly is this the case in old men with rigid chest walls and large emphysematous lungs. Under these circumstances there may be a high grade of hypertrophy without any visible impulse. Even when the apex beat is visible it may be, as Traube pointed out, feeble and indefinite. In many cases the apex is seen displaced downward and outward, and the impulse looks strong and forcible.

Palpation reveals in many cases a thrill at the base of the heart of maximum force in the aortic region. With no other condition do we meet with thrills of greater intensity. The apex beat may not be palpable under the conditions above mentioned, or there may be a slow, heaving, forcible impulse.

Percussion never gives the same wide area of dulness as in aortic insufficiency. The extent of it depends largely on the state of the lungs, whether emphysematous or not.

Auscultation.—A systolic murmur of maximum intensity at the aortic cartilage, and propagated into the great vessels, is present in aortic stenosis, but is by no means pathognomonic. One of the last lessons learned by the student of physical diagnosis is to recognize the fact that this systolic murmur is only in comparatively rare cases produced by decided narrowing of the aortic orifice. Roughening of the valves, or the intima of the aorta, and hæmic states are much more frequent causes. In aortic stenosis the murmur often has a much harsher quality, is louder, and is more frequently musical than in the conditions just mentioned. When compensation fails and the ventricle is dilated and feeble the murmur may be soft and distant. The second sound is rarely heard at the aortic cartilage, owing to the thickening and stiffness of the valve. A diastolic murmur is not uncommon, but in many cases it cannot be heard. The pulse in pure aortic stenosis is small, usually of good tension, regular, and perhaps slower than normal.

The condition may be latent for an indefinite period, as long as the hypertrophy is maintained. Early symptoms are those due to defective blood-supply to the brain, dizziness, and fainting. Palpitation, pain about the heart, and anginal symptoms are not so marked as in insufficiency. With degeneration of the heart-muscle and dilatation relative insufficiency of the mitral valve is established, and the patient may present all the features of engorgement in the lesser and systemic circulations, with dyspnœa, cough, rusty expectoration, and the signs of anasarca in the lower part of the body. Many of the cases in old people, without presenting any dropsy, have symptoms pointing rather to general arterial disease. Cheyne-Stokes breathing is not uncommon with or without signs of uræmia.

Diagnosis.—With an intensely rough or musical murmur of maximum intensity at the aortic region and signs of hypertrophy of the left ventricle, a thrill and a hard, slow pulse of moderate volume and fairly good tension,

a diagnosis of aortic stenosis can be made with some degree of probability, particularly if the subject is an old man. Mistakes are common, however, and a roughened or calcified valve segment, or, in some instances, a very roughened and prominent calcified plate in the aorta, and hypertrophy associated with renal disease, may produce similar symptoms.

Let me repeat that a murmur of maximum intensity at the aortic cartilage is of no importance in itself as a diagnostic sign of stenosis. Roughening of the valve, sclerosis of the intima of the arch, and anæmia are conditions more frequently associated with a systolic murmur in this region. Seldom is there difficulty in distinguishing the murmur due to anæmia, since it is rarely so intense and is not associated with thrill or with marked hypertrophy of the left ventricle. In aortic insufficiency a systolic murmur is usually present, but has neither the intensity nor the musical quality, nor is it accompanied with a thrill. With roughening and dilatation of the ascending aorta the murmur may be very harsh or musical; but the existence of a second sound, accentuated and ringing in quality, is usually sufficient to differentiate this condition.

Mitral Incompetency.

Etiology. — Insufficiency of the mitral valve results from: (*a*) Changes in the segments whereby they are contracted and shortened, usually combined with changes in the chordæ tendineæ, or with more or less narrowing of the orifice. (*b*) As a result of changes in the muscular walls of the ventricle, either dilatation, so that the valve segments fail to close an enlarged orifice, or changes in the muscular substance, so that the segments are imperfectly coapted during the systole—muscular incompetency. The common lesions producing insufficiency result from endocarditis, which causes a gradual thickening at the edges of the valves, contraction of the chordæ tendineæ, and union of the edges of the segments, so that in a majority of the instances there is not only insufficiency, but some grade of narrowing as well. Except in children, we rarely see the mitral leaflets curled and puckered without narrowing of the orifice. Calcareous plates at the base of the valve may prevent perfect closure of one of the segments. In long-standing cases the entire mitral structures are converted into a firm calcareous ring. From this valvular insufficiency the other condition of muscular incompetency must be carefully distinguished. It is met with in all conditions of extreme dilatation of the left ventricle, and also in weakening of the muscles in prolonged fevers and in anæmia.

Morbid Anatomy.—The effects of incompetency of the mitral segment upon the heart and circulation are as follows: (*a*) The imperfect closure allows a certain amount of blood to regurgitate from the ventricle into the auricle, so that at the end of auricular diastole this chamber contains not only the blood which it has received from the lungs, but also that which has regurgitated from the left ventricle. This necessitates dilatation, and, as increased work is thrown upon it in expelling the augmented contents, hypertrophy as well.

(*b*) With each systole of the left auricle a larger volume of blood is forced into the left ventricle, which also dilates and subsequently becomes hypertrophied.

(*c*) During the diastole of the left auricle, as blood is regurgitated into it from the left ventricle, the pulmonary veins are less readily emptied. In consequence the right ventricle expels its contents less freely, and in turn becomes dilated and hypertrophied.

(*d*) Finally, the right auricle also is involved, its chamber is enlarged, and its walls are increased in thickness.

(*e*) The effect upon the pulmonary vessels is to produce dilatation both of the arteries and veins—often in long-standing cases atheromatous changes; the capillaries are distended, and ultimately the condition of brown induration is produced. Perfect compensation may be effected, chiefly through the hypertrophy of both ventricles, and the effect upon the peripheral circulation may not be manifested for years, as a normal volume of blood is discharged from the left heart at each systole. The time comes, however, when, owing either to increase in the grade of the incompetency or to failure of the compensation, the left ventricle is unable to send out its normal volume into the aorta. Then there is overfilling of the left auricle, engorgement in the lesser circulation, embarrassed action of the right heart, and congestion in the systemic veins. For years this somewhat congested condition may be limited to the lesser circulation, but finally the right auricle becomes dilated, the tricuspid valves incompetent, and the systemic veins are engorged. This gradually leads to the condition of cyanotic induration in the viscera and, when extreme, to dropsical effusion.

Muscular incompetency, due to impaired nutrition of the mitral and papillary muscles, is rarely followed by such perfect compensation. There may be in acute destruction of the aortic segments an acute dilatation of the left ventricle with relative incompetency of the mitral segments, great dilatation of the left auricle, and intense engorgement of the lungs, under which circumstances profuse hæmorrhage may result. In these cases there is little chance for the establishment of compensation. In cases of hypertrophy and dilatation of the heart, without valvular lesions, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme and lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy, which cannot be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic Bright's disease the hypertrophy of the left ventricle may gradually fail, leading, in the later stages, to relative insufficiency of the mitral valve, and the production of a con-

dition of pulmonary and systemic congestion, similar to that induced by the most extreme grade of lesion of the valve itself.

Symptoms.—During the development of the lesion, unless the incompetency comes on acutely in consequence of rupture of the valve segment or of ulceration, the compensatory changes go hand in hand with the defect, and there are no subjective symptoms. So, also, in the stage of perfect compensation, there may be the most extreme grade of mitral insufficiency with enormous hypertrophy of the heart, yet the patient may not be aware of the existence of heart trouble, and may suffer no inconvenience except perhaps a little shortness of breath on exertion or on going up-stairs. It is only when from any cause the compensation has not been perfectly effected, or having been so is broken abruptly or gradually, that the patients begin to be troubled. The symptoms may be divided into two groups:

(*a*) The minor manifestations while compensation is still good. Patients with extreme incompetency often have a congested appearance of the face, the lips and ears have a bluish tint, and the venules on the cheeks may be enlarged, which in many cases is very suggestive. In long-standing cases, particularly in children, the fingers may be clubbed, and there is shortness of breath on exertion. This is one of the most constant features in mitral insufficiency, and may exist for years, even when the compensation is perfect. Owing to the somewhat congested condition of the lungs these patients have a tendency to attacks of bronchitis or hæmoptysis. There may also be palpitation of the heart. As a rule, however, in well-balanced lesions in adults, this period of full compensation or latent stage is not associated with symptoms which call the attention of the patient to an affection of the heart.

(*b*) Sooner or later comes a period of disturbed or broken compensation, in which the most intense symptoms are those of venous engorgement. There are palpitation, weak, irregular action of the heart, and signs of dilatation. Dyspnœa is a marked feature, and there may be cough. There is usually a slight cyanosis, and even a jaundiced tint to the skin. The most marked symptoms, however, are those of venous stasis. The overfilling of the pulmonary vessels accounts in part for the dyspnœa. There is cough, often with bloody or watery expectoration, and the alveolar epithelium containing brown pigment-grains is abundant. Dropsical effusion usually sets in, beginning in the feet and extending to the body and the serous sacs. The liver is enlarged, and there are signs of portal congestion, gastric irritation, and catarrh of the stomach and intestines. The urine is usually scanty and albuminous, and contains tube casts and sometimes blood-corpuscles. With judicious treatment the compensation may be restored and all the serious symptoms may pass away. Patients may have recurring attacks of this kind, but ultimately the condition is beyond repair and the patient either dies of a general dropsy or there is progressive dilatation of the heart, and death from asystole. Sudden death in these cases is rare.

Physical Signs.—*Inspection.*—In children the præcordia may bulge and there may be a large area of visible pulsation. The apex beat is to the left of the nipple, in some cases in the sixth interspace, in the anterior axillary line. There may be a wavy impulse in the cervical veins which are often full, particularly when the patient is recumbent.

Palpation.—A thrill is rare; when present it is felt at the apex, often in a limited area. The force of the impulse may depend largely upon the stage in which the case is examined. In full compensation it is forcible and heaving; when the compensation is disturbed, usually wavy and feeble.

Percussion.—The dulness is increased, particularly in a lateral direction. There is no disease of the valves which produces, in long-standing cases, a more extensive transverse area of heart dulness. It does not extend so much upward along the left margin of the sternum as beyond the right margin and to the left of the nipple line.

Auscultation.—At the apex there is a systolic murmur which wholly or partly obliterates the first sound. It is loudest here, and has a blowing, sometimes musical in character, particularly toward the latter part. The murmur is transmitted to the axilla and may be heard at the back, in some instances over the entire chest. There are cases in which, as pointed out by Naunyn, the murmur is heard best along the left border of the sternum. Usually in diastole at the apex the loudly transmitted second sound may be heard. Occasionally there is also a soft, sometimes a rough or rumbling presystolic murmur. As a rule, in cases of extreme mitral insufficiency from valvular lesion with great hypertrophy of both ventricles, there is heard only a loud blowing murmur during systole. A murmur of mitral insufficiency may vary a great deal according to the position of the patient. It may be present in the recumbent and absent in the erect posture. In cases of dilatation, particularly when dropsy is present, there may be heard at the ensiform cartilage and in the lower sternal region a soft systolic murmur due to tricuspid regurgitation. An important sign on auscultation is the accentuated pulmonary second sound. This is heard to the left of the sternum in the second interspace, or over the third left costal cartilage.

The pulse in mitral insufficiency, during the period of full compensation, may be full and regular, often of low tension. Usually with the first onset of the symptoms the pulse becomes irregular, a feature which then dominates the case throughout. There may be no two beats of equal force or volume. Often after the disappearance of the symptoms of failure of compensation the irregularity of the pulse persists.

The three important physical signs then of mitral regurgitation are: (*a*) systolic murmur of maximum intensity at the apex, which is propagated to the axilla and heard at the angle of the scapula; (*b*) accentuation

of the pulmonary second sound; (*c*) evidence of enlargement of the heart, particularly the increase in the transverse diameter, due to hypertrophy of both right and left ventricles.

Diagnosis.—There is rarely any difficulty in the diagnosis of mitral insufficiency. The physical signs just referred to are quite characteristic and distinctive. Two points are to be borne in mind. First, a murmur, systolic in character, and of maximum intensity at the apex, and propagated even to the axilla, does not necessarily indicate incompetency of the mitral valve. There is heard in this region a large group of what are termed accidental murmurs, the precise nature of which is still doubtful. They are probably formed, however, in the ventricle, and are not associated with hypertrophy, or accentuation of pulmonary second sound.

Second, it is not always possible to say whether the insufficiency is due to lesion of the valve segment or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary second sound, nor the hypertrophy assists in the differentiation. The history is sometimes of greater value in this matter than the physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart (in which the systolic murmur may be of the greatest intensity), and the instances of arterio-sclerosis with dilated heart.

Mitral Stenosis.

Etiology.—Narrowing of the mitral orifice is usually the result of valvular endocarditis occurring in the earlier years of life; very rarely it is congenital. It is very much more common in women than in men—in 63 of 80 cases noted by Duckworth. This is not easy to explain, but there are at least two factors to be considered. Rheumatism prevails more in girls than in boys and, as is well known, endocarditis of the mitral valve is more common in rheumatism. Chorea, also, as suggested by Barlow, has an important influence, occurring more frequently in girls and often associated with endocarditis. Of 110 cases of chorea which I examined at a period more than two years subsequent to the attack, 54 cases had signs of organic heart-disease, among which were 17 instances with the physical signs of mitral stenosis. Anæmia and chlorosis, which are prevalent in girls, have been regarded as possible factors. In a number of cases, however, no recognizable etiological factor can be discovered. This has been regarded by some writers as favoring the view that many of the cases are of congenital origin; but it is not improbable that with any of the febrile affections of childhood endocarditis may be associated. Whooping-cough, too, with its terrible strain on the heart-valves, may be accountable for certain cases. Congenital affections of the mitral valve are notoriously rare. While met with at all ages, stenosis is certainly more frequent in young persons.

Morbid Anatomy.—In a majority of instances with the stenosis there is some incompetency. The narrowing results from thickening and contraction of the tissues of the ring, of the valve segments, and of the chordæ tendineæ. The condition varies a good deal according to the amount of atheromatous change. In many cases the curtains are so welded together and the whole valvular region so thickened that the orifice is reduced to a mere chink—Corrigan's button-hole contraction. In other cases the curtains are not much thickened, but narrowing has resulted from gradual adhesion at the edges, and thickening of the chordæ tendineæ, so that from the auricle it looks cone-like—the so-called funnel-shaped variety of stenosis. The instances in which the valve segments are very slightly deformed but in which the orifice is considerably narrowed, are regarded by some as possibly of congenital origin. Occasionally the curtains are in great part free from disease, but the narrowing results from large calcareous masses, which project into them from the ring. The involvement of the chordæ tendineæ is usually extreme, and the papillary muscles may be inserted directly upon the valve. In moderate grades of constriction the orifice will admit the tip of the index-finger; in more extreme forms, the tip of the little finger; and occasionally one meets with a specimen in which the orifice seems almost obliterated, as in a case which came under my notice, which only admitted a medium-sized Bowman's probe.

The heart in mitral stenosis is not greatly enlarged, rarely weighing more than 14 or 15 ounces. Occasionally, in an elderly person, it may seem slightly if at all enlarged, and again there are instances in which the weight may reach as much as 20 ounces. The left ventricle is usually small, and may look very small in comparison with the right ventricle, which forms the greater portion of the apex. In cases in which with the narrowing there is very considerable incompetency the left ventricle may be moderately dilated and hypertrophied.

These changes gradually induced are associated with secondary alterations of great importance in the heart. The left auricle discharges its blood with greater difficulty and in consequence dilates, and its walls reach three or four times their normal thickness. Although the auricle is by structure unfitted to compensate an extreme lesion, the probability is that for some time during the gradual production of stenosis, the increasing muscular power of the walls is sufficient to counterbalance the defect. Eventually the tension is increased in the pulmonary circulation, owing to impeded outflow from the veins. To overcome this the right ventricle undergoes dilatation and hypertrophy, and upon this chamber falls the work of equalizing the circulation. Relative incompetency of the tricuspid and congestion of systemic veins at last supervene.

It is not uncommon at the examination to find white thrombi in the appendix of the left auricle. Occasionally a large part of the auricle is occupied by an ante-mortem thrombus. Still more rarely the remarkable