DISEASES OF THE MITRAL VALVE.

MITRAL INSUFFICIENCY

Definition.—A diseased condition of some of the structures constituting the mitral valve; or a defect at the left auriculo-ventricular orifice, preventing the normal closure of the orifice during the systole of the left ventricle, and occasioning a backward flow of a portion of the output into the left auricle.

Morbid anatomy.—(i.) In the chronic stages of rheumatic endocarditis.—The curtains of the mitral valve are thickened and comparatively rigid; the neighbouring endocardium is also denser and more opaque than the normal, especially in the portion extending from the great anterior flap of the mitral valve to the base of the aortic semilunar valves. Many of the chordae tendineae, together with their columnae carneae, are thickened and shortened; there are often adhesions between the curtains, the cords and the columns, as well as in some cases between these and the endocardium of the wall of the ventricle. In some instances the chordae tendineae, especially the finer cords which are inserted near the free border of the curtain, are lengthened instead of shortened; probably this is due to yielding under the pressure of the blood upon the under surface of the mitral flap, so that the edge of the latter is inverted into the auricle during the systole of the ventricle. Whether the chordae be shortened or lengthened, the result is an imperfect apposition of the curtains at the time of ventricular contraction. The endocardium lining the left auricle is also thicker than normal, especially at the ring bounding the auriculo-ventricular aperture. From this ring extends a whitish or milky patch of the fibrously transformed endocardium into the auricle above and the ventricle below. Such thickening may involve the structures subjacent to the endocardium, and tend to narrow the orifice, though the signs may be entirely those of mitral insufficiency and not those of obstruction. Durozies (14) says, that if the orifice be large enough to admit the passage of the thumb the signs will be those of insufficiency, and not of stenosis. Much, however, depends on the condition of the internal surface; if this be smooth, as in many cases it is, there will be signs of mitral insufficiency only; if rough, there will be those of stenosis in addition.

The thickening of the endocardium is due to fibrous proliferation of the original inflammatory exudation, a process of development of connective tissue extending into surrounding structures. Repeated attacks of endocarditis affecting the already diseased tissue cause further thickenings and retractions; the thick fibroid material compresses the blood-vessels, and tends to induce degeneration. Fatty degeneration is not often observed, but calcareous change frequently, even in the
case of young children. The calcified portion of the valve structure may act as a mechanical irritant producing inflammatory or necrosing changes in the tissues adjacent. A fragment of the calcareous or the necrosed material may become detached and form an embolus. Very rarely a change of the firm fibrous material into cartilage has been found (72).

(ii.) In the chronic forms of ulcerative or septic endocarditis.—The valve curtains, the cords and columns, or the endocardium of the ventricle may show the lesions of ulcerative or septic endocarditis, the tissues in the affected areas being destroyed by necrosis. Usually the ulcerated surfaces are covered with large vegetations. These changes in a large majority of the cases of ulcerative endocarditis—about three-fourths of the total—are found on valves previously diseased. In all such cases some of the forms of pathogenetic micro-organisms are to be discovered. It is to be borne in mind, therefore, that on the chronic morbid products at the mitral orifice a destructive disease which has no relation with rheumatism may be engrailed. In a minority of the cases the necrosing changes are slow; there is evidence to show that the process may be arrested in some areas, cicatricial tissue covering the portions showing loss of substance.

(iii.) In rupture of the mitral valve.—The valve curtains, cords, or columns may be ruptured. It is improbable that such an accident can occur from strain where the structures had been previously healthy. Post-mortem evidences of the rupture of a tendinous cord are not infrequent; an occurrence which has sometimes changed fairly compensated mitral inadequacy into a hopeless disablement (72). In the majority of cases I think it probable that the rupture is due to ulcerative changes. In some of them it seemed to have been due to the direct irritation of a calcareous plate or firm fibrous band operating during the movements of the ventricle; in others ulcerative endocarditis has affected the rupture. In the case of a curtain of the valve there may be first aneurysmal pouching, and secondly perforation. A vegetation on the curtain, if it induce softening of the endocardial surface, brings about a yielding under the blood-pressure within the ventricle, and a pouch is formed which projects into the left auricle; further pressure may cause rupture (perforation), when of course the valve is no longer competent.

(iv.) In papilliform endocarditis.—A form of chronic endocarditis is sometimes observed in which there are small, firm, warty outgrowths from the surface; these are fibrous proliferations of the endocardium, usually attached by a broad base but sometimes pedunculated. They are covered by smooth endothelium to which fibrin does not adhere; the scleros changes of rheumatic endocarditis are not associated with them. They have been most frequently observed in cases of chorea; Lancea Louis has found them also in alcoholism and in malaria. I have seen an example in a case of tuberculosis. Sometimes in newly-born infants small spherical outgrowths are observed on the free border of the mitral; they are probably hematoma due to rupture of blood-vessels situated under the most superficial layer of the endocardium (36, 44); usually they disappear
in the first few months of life, but in some cases they may initiate the warty excrescences above described (Crueilhier). I have considered it probable that in some cases of chorea, determined by sudden fright, similar ruptures of intra-valvular vessels with subsequent fibrous warty transformations occur.

(ν) In dilatation of the left ventricle.—There may be considerable dilatation of the ventricle, and yet the mitral curtains be quite competent to close the aperture. In many cases, however, when there is no disease of the structures constituting the valve, the cavity is so greatly dilated that it is demonstrably impossible that the aperture between auricle and ventricle could be adequately closed during the ventricular systole. Amongst the post-mortem associations of the latter condition are the following: (a) there may be disease at the aortic orifice causing obstruction or regurgitation or, as very frequently is the case, the combined lesion. The ventricle has become hypertrophied and dilated on account of the abnormal pressure to which it has been subjected, and the dilatation continues and progresses until the mitral curtains are no longer capable of closing the auriculo-ventricular orifice; (b) the signs of chronic disease of the kidneys (chronic interstitial nephritis) may be found. In some cases there is great hypertrophy of the muscular wall of the ventricle; in others dilatation, even at early periods of the disease, preponderates over hypertrophy. Microscopical investigation has shown that the causes for the changes in the cavity and the walls of the left ventricle are complex. The obstruction to the general arterial circulation due to the thickening of the arterioles in various situations causes abnormal intra-ventricular pressure during systole and thus there is a mechanical cause of dilatation; but the muscle of the ventricle also suffers from the process of disease. The morbid changes in the ventricular wall have been described as an excessive proliferation of the connective tissue (3, 16, 29), a special quasi-inflammatory affection of the smaller branches of the coronary arteries — endarteritis and periarteritis (13, 26, 33, 39), or a fibrosis extending to the general connective tissue, but starting from the arterioles and capillaries — arterio-capillary fibrosis (69). The muscular fibres are altered, the transverse striae are obscured, some fibres are atrophied and encroached upon by the fibroid tissue, others are hypertrophied. Similar changes are sometimes noted in the walls of the ventricle in persons at and after middle age, when there are no signs of chronic Bright’s disease. (c) As a sequence to inflammation of the pericardium, the pericardial surfaces being found adherent. An excess of fibroid tissue not only extends amongst the muscular bundles and fibres, but also compresses the blood-vessels; this is especially seen after general rheumatic disease of the heart (carditis) in children; the left ventricle may be extremely hypertrophied and dilated so that the mitral valve is incompetent, and yet there may be no sign of endocarditis affecting the structures of the valve. Dilatation of the left ventricle to the extent of mitral incompetence is also observed occasionally after rheumatic fever in childhood, with no evidence of pericarditis or endocarditis. (d) In
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syphilitic affections of the ventricle the muscular fibrils have probably been weakened by myocarditis. In rare cases small gummata have been found in the wall of the ventricle; in other cases of fibroid material, probably the sequelae of syphilitic endarteritis, and obliterations of the vessels, have been seen (c) in Graves' disease, and other kinds of long-continued morbid acceleration of the heart's contractions, such as tachycardia. In some such cases the left ventricle has been found so hypertrophied and dilated that the mitral curtains were incompetent; it must be remembered, however, that in many of the fatal cases of these diseases the ventricular cavity had not been dilated, and the muscle of the heart was quite normal. The dilatation of the left ventricle must be regarded only as an occasional sequel of the disturbance of the nerve-mechanism of the heart.

(vi.) In degenerations or transformations of the structures of the left ventricle.—In a large number of instances the various forms of degeneration of the heart—fatty, fibroid, and granular—are associated with dilatation of the left ventricle; and the mitral regurgitation, which is a feature of their history, is thus explained. In a minority there is no such dilatation. In fibroid degeneration bands of firm fibrous tissue, replacing more or less the muscular fibres, are observed, on section of the ventricular wall, to spread out in certain tracts; they often extend into the musculi papillares, and some of these may be wholly transformed into fibrous tissue. In granular degeneration, "dissociation segmentaeva" (30), the heart-muscle is observed to be disintegrated, and to present the appearance of an aggregation of fine particles; the cement substance which normally binds the fibres together being softened. This morbid condition is to be demonstrated in the musculi papillares. In fatty degeneration pale spots or streaks are observed on section, not only in the wall of the ventricle but in the papillary muscles also; then microscopic examination shows the absence, in greater or less degree, of the proper muscle elements, and the presence of minute oil globules. These transformations or degenerations may be the consequences of obliteration of the arterioles. In cases where there is sudden and recent infarction of these vessels the appearances are those of the softening known as myomalacia cordis. Where the process has been more chronic, fibrous transformations or fatty changes are observed. In some cases the two forms of transformation, fatty and fibrous, are seen together.

In another form of fatty degeneration of the heart-muscle the pale spots and the mottlings indicating the areas of metamorphosed muscular fibres are scattered throughout the ventricular wall and the fleshy columns, having no relation with any tract of vascular supply. These constitute the majority of cases known as "fatty heart." In some they are associated—especially in fat persons and drunkards—with infiltration of fatty tissue amongst the muscular bundles. Fatty degeneration of the muscular fibres of the heart is found also in chlorosis, anæmia, and blood-deteriorations. In some of these cases there has been evidence of mitral regurgitation. If there be local degeneration resulting from the obliteration of arterioles the condition leads to a fatal issue. In
the majority of cases of fatty degeneration in anemia the heart regains its structural integrity, and any consequent mitral insufficiency disappears. When the heart thus recovers it must be inferred that many of the diseased fibres actually disappear, the fat which is the result of their disintegration becoming absorbed, whilst new formation of normal muscular tissue takes place. In cases in this category, where death does not occur from the fatal forms of anemia, careful search should be made for disease in other organs, and the formula, "death from fatty degeneration of the heart," should not be delivered too hastily.

(vii.) In some cases in which there has been strong evidence of mitral regurgitation during life the heart has been found on post-mortem examination to present perfectly normal appearances. The pathology of such cases will be considered later.

In mitral insufficiency from all causes the left ventricle is dilated and its muscular walls hypertrophied. The dilatation and hypertrophy proceed hand in hand, and both are the direct and salutary results of the regurgitation through the mitral orifice. As the late Dr. Herbert Davies pointed out, the process whereby, in sequence to mitral insufficiency, the cavity of the left ventricle becomes enlarged and the muscular tissue hypertrophied should not be considered morbid. The enlargement may be in just such degree that the amount lost to the aorta by the leakage into the auricle is compensated; and the increased driving power of the ventricle is precisely regulated to deliver the normal supply to the great artery (12).

In mitral insufficiency the left auricle is dilated and hypertrophied, and the endocardium lining it is thicker and more opaque than normal. In some chronic cases the muscle of the auricle wastes, and is replaced by fibrous tissue. The pulmonary veins also may be much dilated. Occasionally in chronic cases globular fibrinous coagula are found adhering to the lining membrane, and projecting from between the fleshy columns and trabeculae into the cavity of the ventricle or the auricle. These thrombi are firm and dense in their external portions, and often soft and fluid in their interior; cysts thus formed may rupture or become detached, and their fragments may cause embolism of systemic arteries. In some cases the coagula undergo fibrous and calcaceous transformations.

The right auricle and ventricle in cases of mitral insufficiency are also found dilated and hypertrophied. Hypertrophy is found to preponderate in the earlier stages, dilatation in the later. The wall of the ventricle is in some cases found thick and leathery, in others thin and flaccid. The tricuspid valve may be incompetent on account of extreme dilatation of the ventricle. The globular thrombi, described as sometimes visible in the left cavities, are much more commonly observed in the right. The detached coagula cause embolisms of branches of the pulmonary artery. The dilated condition of the right chambers of the heart is obviously associated, with general venous engorgement. In the heart itself the coronary veins are turgid and dilated.
The pericardium may show signs of disease, recent or remote, and there may be fluid effusion in the pericardial sac.

Morbid anatomy of other organs in mitral insufficiency.—The lungs in cases where there has been long-continued mitral regurgitation are found engorged with dark blood, and their fibrous tissues abnormally dense. The lung is tough; the capillaries of the alveoli have become dilated and varicose, their walls thickened. Patches showing the signs of broncho-pneumonia may be scattered throughout the toughened lung. Blood escaping into the surrounding connective tissue produces brownish pigmentation (brown induration of the lung); it may transude into the alveoli, causing the tinged sputa and haemoptysis observed in some cases. The lining membrane of the bronchi often shows extreme engorgement, and blood exudes from the surface. The blood-tinged sputa, therefore, may be derived from the lung capillaries or from the bronchial mucous membrane. The lower lobes, or the more dependent portions of the lung in chronic cases, become engorged, dense, and often oedematous. In many cases there are multiple pulmonary lesions, with evidence that these arose at different dates. Effusions into the pleura may have caused collapse of various portions of the lung. The signs may indicate that local pulmonary infarctions have occurred in different areas at various dates. There may be the blood-clot and prominence of the pleural surface indicating a recent embolism of a branch of the pulmonary artery (pulmonary apoplexy); the sites of old infarctions may be indicated by pigmented inductions of portions of the lung-tissue, with, perhaps, some depression of the pleural surface corresponding to the indurated portion. In cases of comparatively recent embolism the corresponding area of the pleura may be covered with the yellowish exudation of pleuritis. All pulmonary apoplexies, however, are not due to infarction. The abnormal strain of the pulmonary artery may lead to degeneration of the vessel and dispose it to rupture. Old adhesions of the pleura or of pleura and pericardium are often observed. In many cases there is fluid effusion in the pleural cavities.

The stomach manifests greatly dilated veins; its mucous surface shows much congestion; the venules are often varicose; mucus, tough or fluid, is seen in abundance. The liver is enlarged; the intra-lobular capillaries are very greatly dilated and their walls thickened; on section it shows the characteristic appearances of “nutmeg liver,” the dark brownish-red stellate spots marking the centre of each lobule on the yellowish ground formed by the bile-stained liver-cells. The bunch of greatly dilated capillaries in the centre of the lobule encroaching upon the hepatic cells may cause atrophy or fatty degeneration of the latter, some brown pigment granules being seen amongst them. The most marked signs of venous engorgement with increase of bulk of the liver are seen in cases in which tricuspid incompetence has followed mitral insufficiency. It is to be remembered that the size of the liver in such cases may become greatly reduced soon after death, the organ being partially emptied of blood by gravitation.
The *spleen* in mitral regurgitation may be enlarged from passive hypersemia, its connective tissue being much increased and causing it to feel much firmer than under normal conditions. In some cases it shows infarctions old or recent. When recent, wedges of hard tissue with their bases at the circumference (that is, the capsule) are felt on manipulation. Old infarctions are indicated by shallow depressions of the surface of the viscus.

The *intestines* show venous engorgement. In some cases embolisms of the small arteries supplying the intestinal wall have been found, with consequent necrosis of the bowel. The veins of the mesentery are engorged. The glands within the abdomen are enlarged and congested.

The *kidneys* are abnormally firm from cyanotic induration; the pyramids are especially engorged; blood may exude from the glomeruli into the tubules. In some cases they show on section pale, wedge-shaped, recent infarctions, their base towards the cortex and their apex towards the hilum; or deep depressions of the surface, with cicatrificial tissue visible on section, may indicate the situations of old embolisms. There may be much fibrosis in these kidneys.

The *peritoneal cavity* may be more or less filled with ascitic fluid.

The *membranes of the brain and spinal cord* may show much venous engorgement. Signs of embolism of the cerebral arteries are found in some cases.

The *subcutaneous tissue* generally, especially in the lower extremities, may be found infiltrated with dropsical fluid. In some cases patches of the superficial layer of the epidermis are raised in large bullae. In other chronic cases the fibrous elements of the skin are thickened—there is a brawny œdema.

**Mechanism of mitral regurgitation.**—In normal conditions of the structures, after the filling of the ventricular cavity from the auricle the muscular wall of the ventricle immediately contracts; the musculi papillares do not begin their contraction until after an appreciable interval, then these muscles act with sudden energy, drawing down the mitral curtains and completely closing the auriculo-ventricular aperture, the apposed curtains presenting a convex surface in the auricle; the energetic tug of the papillary muscles gradually ceases and they relax, whilst the muscle of the ventricular wall remains contracted (54). The contraction of the muscle of the ventricle has a direct effect upon the auriculo-ventricular aperture. Before the beginning of the systole of the ventricle this orifice is circular; during the period of systole the contraction of the surrounding muscular fibres causes it to become narrower and of oval form (35, 37). At the acme of systole the auriculo-ventricular orifice has an area not much more than half that which it presents in diastole (35). The shape of the papillary muscles is such that in the complete contraction of the ventricle they are accurately applied to each other (63).

The ventricular systole, therefore, consists in a series of co-ordinated
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rhythmic movements. There may be many causes of disturbance of the normal association and sequence of these actions, the result of which is insufficient closure of the mitral orifice and reflux into the left auricle occasioned by the ventricular systole. (a) There may be such structural disease in the curtains, cords, and attachments of the valve that due apposition is impossible. (b) The fibrous ring to which the flaps of the valve are attached at their circumference may be so much thickened that the muscles at the base of the heart are unable to compress it sufficiently to cause accurate closure by the curtains during ventricular systole. (c) The insufficient narrowing of the auriculo-ventricular aperture during systole may be due to no structural alteration of the ring, but to enfeeblement of the muscle of the ventricle. (d) The ventricle may be so greatly dilated, and with it the fibrous ring to which the mitral curtains are attached, that these latter fail to meet at their borders during the period of contraction of the ventricle. (e) The papillary muscles may be so enfeebled by disease that they fail to perform their function of approximating the valve curtains. (f) Owing to disturbance of the nervous mechanism the movements may not be performed in their due association and sequence.

Consequences of mitral regurgitation.—It is probable that in cases in which very small amounts of blood are regurgitated into the auricle from the left ventricle the consequences are inappreciable. The mechanical results are directly proportioned to the amount of reflux. The immediate effects may be regarded as simultaneous upon the left auricle and the left ventricle. The auricle is distended in proportion to the force of the ventricle and the amount of fluid regurgitated. The auricular wall becomes dilated, and its muscle, subjected to abnormal stimulus, hypertrophied. The left ventricle, receiving during its diastolic expansion an abnormal quantity of blood from the dilated auricle, is subjected to unusual pressure; the muscle yields and its cavity becomes enlarged. Such increase of capacity is a necessity if the normal supply to the aorta is maintained. In systole it is called upon for more work, in order to deliver an adequate amount into the aorta. Hypertrophy of its muscle ensues, and is a favourable condition. The effect of the regurgitant stream is manifested upon the right chambers of the heart. The current impelled by the right ventricle, which in normal condition should flow unimpeded through the pulmonary vessels, is met by the reflux current from the left ventricle. The capillaries of the lung, the branches and trunk of the pulmonary artery, and the right ventricle itself, are thus subjected to abnormal strain. The effects are hypertrophy of the muscle and dilatation of the cavity of the right ventricle. Hypertrophy of the right ventricle also is essentially favourable, for the more vigorous action antagonising the back flow into the left auricle helps the delivery of an adequate supply to the aorta.

The maintenance and the failure of compensation.—If the changes in the cavities and in the myocardium thus sketched out are nicely balanced, a condition of restored equilibrium ensues; thus a stationary lesion of
compensated mitral regurgitation may persist for long periods, the subject thereof presenting neither morbid sign nor symptom. An adverse change, however, may be effected by many causes: the dilating strain upon the left cavities may impair the muscular power of the left auricle and ventricle; renewed disease of the endocardium may increase the degree of valvular imperfection; intercurrent diseases may affect the structural integrity of the cardiac muscles, vessels, or nerves; affections of the lungs (from extrinsic causes, or from causes, such as embolism, intrinsic to the cardiac imperfection) may induce direct and mechanical as well as indirect and enfeebling difficulties. The result of any of these interferences is a break of compensation—a failure of the cardiac forces of circulation; the supply to the aorta and thence to the tissues becomes inadequate, then the muscle of the left auricle and the ventricle becomes more and more enfeebled, their constituent structures degenerate, and their cavities contain more and more residual blood. The force of the right ventricle now fails, and both right auricle and right ventricle become engorged with venous blood; the systemic veins are dilated, and the tissues suffer from venous stasis. The hepatic veins (which are in such immediate relation with the inferior cava), being destitute of valves, are especially congested, and their engorgement becomes manifest in enlargement of the liver. As the distension of the right ventricle continues, the right auriculo-ventricular orifice may become so much dilated that the tricuspid valve becomes incompetent to close it; then the pulsatile action of the right ventricle is communicated to the valveless hepatic vein, and thus to the liver, as well as to the veins of the neck, if the walls of these have been sufficiently dilated to render their valves incompetent. The interference of the general and the lymphatic circulations at varying stages of this period of failing compensation may induce dropy.

Diagnosis. — The chief sign by which the diagnosis of the insufficiency of the mitral valve is to be made is a physical sign obtained by auscultation—a systolic murmur heard at the apex of the heart, or having a maximum intensity in this situation. It is an essential preliminary that the position and outline of the apex be determined by palpation and percussion. The abnormal sound is often in some degree musical, varying in different cases from a very low to a very high pitch; in some it may resemble the sound of a whispered "who," in others a musical note of varying pitch and quality, and in no inconsiderable number a shrill whistle. In many it has the sound as of a puff of steam. A characteristic to be especially noted is that it fades off gradually, and does not come to a sudden, abrupt stop. The murmur begins with the systolic contraction of the ventricles; this may be determined, at the time that auscultation is practised, by the observer placing his finger over a point where the apex beat is to be felt; or, if this be impracticable, over one of the carotid arteries in the neck. The bruit may be very short, ceasing at an early portion of the systole, or may be prolonged nearly throughout the whole of the systolic contraction, ceasing just
before the second sound. It may wholly replace the first sound, or the
dull sound of valvular tension may be heard to precede it, when it "tails
off" from the first sound. The murmur may be of very slight
intensity, and may be localised at the exact apex, or it may be audible
over the whole precordia with maximum intensity at the apex. In some
cases it is audible from the apex in a line which extends into the
left axilla, and then it often has another area of audibility at the back
between the spine and the angle of the left scapula. In other cases
the conduction is to the left border of the sternum above the ensiform
cartilage, and the cartilages and the interspaces as far as the second
left costal cartilage. I have observed cases in which the explanation of
this conduction of a systolic murmur has appeared to me to be afforded
by discovery at the necropsy that the disease was chiefly confined to
the anterior flap of the mitral valve with the attached chordae and the
papillary muscles. Firm fibroid or calcareous material conducted the
vibrations towards the septum ventriculorum, whence they were trans-
mitted to the sternum and superficial parts adjacent. It is probable that
conduction towards the axilla and the back may indicate an implication
of the posterior flap in the disease.

It has been thought by some observers (41, 1, 53) that mitral
regurgitation may be evidenced by a systolic murmur in the second
left intercostal space, not quite close to the sternum but about two cen-
timetres to the left of it; the murmur being due to vibrations communicated
by the reflux current to the left auricular appendix (1). Many con-
siderations seem to me to render this view untenable. The left auricular
appendix, as has been pointed out by Russell, Byron Bramwell, and
others, does not approach the surface at the spot where the murmur
is audible; in many necropsies it has not been visible on an anterior
view of the heart; when seen it is at least an inch and a quarter to the
left of the left border of the sternum, and is for the most part on the
posterior aspect of the heart (55). It is more probable that the vibra-
tions of the reflux current, if transmitted to the auricle, would be audible
at the back. Duroziez has used this argument to explain the audibility
of the murmur of mitral regurgitation at the back:—"L'oreillette gauche
placée en arrière contre la colonne vertébrale transmet en arrière le
souffle formé à la mitrale." I consider it most probable that, when the
murmur of mitral regurgitation is audible in the second left interspace,
it is by means of vibrations communicated to the great anterior flap of
the mitral valve, or to the morbid structures in contiguity therewith.

The chief practical difficulty in the diagnosis is that of discriminating
a murmur due to mitral insufficiency from one to be ascribed to the
influence of the movements of the heart upon the portions of lung in
front of it and around it. Cardio-pulmonary murmurs have been
described by many observers (51, 64, 28, 18, 42, 48, 40). Of these
Potain has made a careful and elaborate study.

The first sound of the heart to the right of the apex and over much
of the area occupied by the right ventricle is often observed to be rough
under conditions in which cardiac disease has no part. Such rough sounds have been referred to many causes which it seems unnecessary to discuss. As a rule they are readily to be distinguished from murmurs due to mitral insufficiency, because they are not heard at the exact apex nor over the situations mentioned as those to which a mitral regurgitant murmur is conducted.

In some cases, however, the difficulties are greater. In order to make the distinction clear, certain steps should be methodically taken. First, the relation of the murmur to the movements of respiration should be observed. The cardio-pulmonary murmur is usually much influenced by the respiratory movements; for the most part it is intensified both during expiration and inspiration, especially during the latter; but it often becomes inaudible at the end of an expiration. If, therefore, rhythmical crescendo and diminuendo in the sound of the murmur are heard during the respiratory acts, it is probable, though not certain, that the murmur has its cause in the lung outside the heart.

The position of audibility of the murmur must be carefully noted. Cardio-pulmonary murmurs are not heard at the exact apex of the left ventricle, but over a small area at the level of the apex to the right and to the left. Instead of corresponding exactly to the centre of the outline of the apex of the left ventricle, as does that of mitral insufficiency from organic causes, these murmurs have their maximum from a quarter of an inch to an inch and a quarter away from the point of apex beat. Above the exact apex there is a doubtful zone, where a precise diagnosis cannot readily be made; but if a systolic murmur has its site of maximum audibility exactly over the apex, it must be ascribed to intra-cardiac causes.

The rhythm of the murmur must be determined. A cardio-pulmonary murmur does not replace the first sound. The valvular flap is heard, and the murmur is observed to occur subsequently, after an appreciable interval, and to cease before the second sound; it is manifested during a portion only of the ventricular coaptation, and is meso-systolic (Potain).

In the next place, auscultation should be practised in various positions of the patient. A cardio-pulmonary murmur, as a general rule, is very evident when the patient is recumbent, diminishing in intensity, and
even disappearing when the sitting or erect position is assumed. In a minority of cases this rule is reversed. It has been shown by Cuffer that though the bruits which have their causes outside the heart are in the greatest degree modified by changes of position, yet systolic apical murmurs, due to organic mitral disease, are sometimes similarly influenced. Potain says that if the change from the dorsal decubitus to the sitting position causes the complete or almost complete disappearance of the murmur, it can be confidently ascribed to extra-cardiac causes; the same may be said when a murmur well marked in the erect position disappears on recumbency. On the other hand, it is not true that every murmur which is uninfluenced by changes of position is necessarily organic.

Potain has adduced a great amount of evidence to show that the cardio-pulmonary murmur is caused by an aspiration of some of the alveoli of the lung produced by the cardiac movements. When the heart
is distended in diastole certain portions of the adjoining lung are compressed against the thoracic wall, and the air is squeezed out of them. When the systolic recession ensues the comparatively airless tongue of pulmonary tissue quickly becomes inflated, provided always the muscular contraction is accomplished rapidly.

Estimation of the degree of mitral insufficiency.—When the amount of blood regurgitated into the left auricle at each systole is very small, there may be no physical sign to indicate the existence of any lesion, other than the systolic murmur having the characters and areas of audibility already described. In the cases where the amount is sufficient to disturb the normal physical conditions within the chambers of the heart, there are signs which indicate, in greater or less degree, the amount as well as the existence of imperfection. In the attempt to make this estimation, in the first instance the second sound of the heart should be carefully observed. If, in any case in which a murmur indicating mitral regurgitation is manifest, the second sound, as heard in the second left intercostal space or the second and third left intercostal spaces, is noted to be of a sharp, loud, metallic, or tympanitic character, or by its loudness ("accentuation") to contrast with the second sound heard in the course of the aorta and great vessels of the neck, as well as in the positions below the third interspace as far as the heart’s apex, it must be concluded that the regurgitant stream, antagonised by the adequate force of contraction of the right ventricle, causes abnormal pressure in the pulmonary artery and the vessels of its circuit. This sign, as Skoda pointed out, indicates a compensated mitral insufficiency; when the right ventricle becomes feeble or the tricuspid valve inadequate, the accentuation of the pulmonary second sound is no longer heard. The observation of an accentuated pulmonic second sound, with no sign of pulmonary embarrassment, no abnormality discovered by auscultation, except the murmur of regurgitation through the mitral orifice, and no physical signs of dilatation of the muscular chambers of the heart, will indicate a moderate and not an extreme degree of mitral insufficiency.

Any deviation of the ventricles, and auricles from the normal should be noted and considered. The left ventricle should be investigated by palpation and percussion. In cases of mitral regurgitation, the apex may be felt to lift the finger of the observer considerably below the normal fifth interspace, and in a greater or less extent to the left; so that it may overpass the vertical mid-thoracic line, and be palpable in the axilla. The forcible heaving or thrusting movements of the ventricle constitute a measure of the degree of hypertrophy of the muscle. In young subjects the ribs and cartilages corresponding to the area occupied by the ventricles may be bulged forwards and prominent. It is very rarely that a systolic thrill is to be felt over the apex. The rhythm of a thrill must be carefully noted—ore felt near the apex is nearly always presystolic, and pathognomonic of mitral stenosis. Determination of the outline of the left ventricle by percussion adds to the information obtained, and indicates the shape and position of the apex, when
these are not perceptible on palpation. The line of dulness or deficient resonance on percussion, indicating the outline of the left ventricle, may be found to extend to the left of the mammillary or mid-thoracic line, even as far as the axilla at the level of the seventh rib, and thence in a line inclining upwards to the level of the second left intercostal space. The upper limit of deficient resonance has been found above the second rib (19).

At post-mortem examinations, even when there is clear evidence of much hypertrophy and dilatation of the left ventricle, the latter is generally observed only as a mere margin to the left of the right ventricle on an anterior view of the heart; the left auricle is often invisible on inspection of the front, and only discovered on so turning over the heart that a back view is obtained. It must be remembered, however, that the conditions during life differ from those observed after death; the heart-muscle contracts in rigor mortis; nevertheless, it is no doubt correct that the left auricle and left ventricle occupy but a small portion of the left border of cardiac dulness.

In cases in which a notable accentuation of the pulmonic second sound and the physical signs of enlargement of the left ventricle are manifested with no evident deviation of the right chambers from the normal, it may be inferred, that though regurgitation through the mitral orifice may be considerable, the lesion is compensated by augmented force of the right ventricle.

For the due estimation of the extent of the lesion the right cavities must be carefully explored. Palpation may detect a forcible heaving of the right ventricle to the left of the ensiform cartilage. Percussion parallel in direction with the long axis of the sternum may indicate a line of deficient resonance extending to the right of the mid-sternal line in various degrees in different cases. The dulness exceptionally extends two and a half inches from the median line; it delimits the right border of the auricle.

In some cases I have found on plessiometric percussion that the right border of dulness does not meet the line which indicates the upper border of the liver at a right angle; but, from one to two inches above the liver, a sloping line of dulness extends from the auricular border to meet the liver dulness an inch to an inch and a half to the right of the sternum. There is a wedge-shaped area of deficient resonance to the right of the vertical line which indicates the limit of the right auricle. I believe this to be due to a distension or dilatation of the venous cavae as they open into the auricle; it is only observed in cases of great dilatation of the right cavities. The upper limit of dulness may reach as high as the lower border of the second right costal cartilage. The extent of the dulness from right to left may be determined by percussion over the first part of the sternum in a horizontal direction; this line crosses the sternum to the second interspace on the left side. Such a line of dulness over the sternum at the level of the second rib still indicates the right auricle, which may even encroach on the second interspace on the left side. The
remainder of the upper limit of dulness is due to the right ventricle and the pulmonary artery.

The evidence of the outline of the heart obtained by percussion must not be accepted without the due estimation of causes of fallacy. Distension of the stomach with air will cause a tilting of the ventricles to a higher plane, and a dislocation towards the right of the right chambers. The content and consequent bulk of the right auricle and ventricle vary with the varying turgescence of the liver. Such distension may be protracted and due to a lasting or temporary and evanescent morbid congestion; for it is well known that the liver presents great variations in bulk even during brief periods of time. A dilatation of the blood-vessels within the abdomen (that is, in the splanchnic area) also may reduce the content and consequent bulk of the right auricle and ventricle when there is no obvious change in the volume of the liver. Another cause for reduction in the observed size of the right cavities is expansion of the lungs. In such cases there are two causes of a recession of the area of deficient resonance indicating the bulk of the heart; namely, the inflated air-cells of the tongues of pulmonary tissue overlapping the heart which give rise to a clearer note on percussion, and the augmented volume of blood circulating in the pulmonary blood-vessels which reduces the content of the heart chambers.

The apparent bulk of the heart, as determined by the means of physical diagnosis, may be temporarily increased by congestion of the vessels of the pericardium and neighbouring pleura as well as of those of the coronary blood-supply; and there may be fluid exudation into the spaces of the surrounding tissues.

The testimony of many observers has shown that the bulk of the heart may be much reduced in a brief space of time—in some cases, as in acute rheumatic diseases, without relation to the therapeutic means adopted; in others in response to special methods of treatment, such as baths and certain methods of muscular exercise.

When in a case manifesting the murmur of mitral regurgitation it is found that the right chambers are persistently dilated, and especially if physical signs of tricuspid incompetence be present, it must be inferred that the degree of valvular imperfection is great and the muscle of the heart gravely approaching failure.

The investigation of the bulk of the liver is also important as a guide to the estimation of the degree of valvular imperfection in a case manifesting the murmur of mitral insufficiency. When there are signs of dilatation of the right chambers of the heart, and the liver is felt as a thickened rounded tumour below the right costal margin, it must be inferred that the mitral valve is gravely incompetent—the imperfection is still greater if the liver be felt to pulsate.

Important evidence is afforded by the observance of the characters of the pulse. If in a case manifesting the systolic apex murmur and other physical signs of mitral insufficiency, the hand of the observer applied to the precordia is sensible of a forcible ventricular contraction, whilst the
radial and other arterial pulses are found to be small and weak, the inference is legitimate that much of the volume of blood which should have been delivered into the aorta is lost by regurgitation into the auricle. The pulse of a slight mitral regurgitation differs inappreciably from the normal: when the lesion is considerable the volume is small and the tension low. The sphygmograph often shows dicrotism when the evidence of impaired tension is not obvious to the finger. Not infrequently, even when compensation is maintained, the low-tension pulse presents marked fluctuation of the base line which shows that the normal correlation between circulation and respiration is disturbed.

The cardiogram in a case in which there is free mitral regurgitation sometimes presents special features. There is a pronounced dip or notch in the upper part of the tracing giving the summit a forked appearance.

It would seem probable also that the relative durations of the systolic and diastolic periods, as expressed in the cardiogram, are altered; the diastolic period being relatively shortened. In compensated mitral regurgitation in many cases neither cardiogram nor sphygmogram presents any notable deviations from the normal.

Irregularity of the pulse is not, in my experience, a characteristic of mitral insufficiency.

Clinical groups of cases of mitral insufficiency.—Group I. Mitral insufficiency the result of rheumatic endocarditis.—It will be convenient to consider this group in two divisions: the first of children, the second of adults.

In children of twelve years of age and under, who have suffered either from a well-marked attack of rheumatic fever, or from repeated attacks,
or from one attack with subsequent subacute manifestations, it is in the highest degree probable that the signs of insufficiency of the mitral valve will be observed. Such insufficiency is nearly always due to the sclerous alterations at the left auriculo-ventricular orifice and to a retraction of the valve curtains, the cords and the muscular columns which are the results of the progressive morbid changes of rheumatic endocarditis (9). These, however, are not the only changes in such cases. Pericarditis usually coexists; the layers of pericardium become united; oftentimes throughout their whole extent, by adhesions. The muscle of the heart is inflamed and infiltrated, and rapidly becomes extremely hypertrophied. The whole heart participates in the rheumatic inflammation; there is general cardiitis (Sturges), the result of which, though life may be prolonged for months and years, is a crippling of the heart while such life lasts. In the course of development of this severe heart disease subcutaneous rheumatic nodules are frequently observed (Barlow, Warner, Cheadle). Such severe general rheumatic heart disease is rarely met with in children under six years of age, it is most common between the ages of six and twelve years. As a general rule, of the children admitted into hospital for acute or subacute rheumatism 50 or 60 per cent are discharged with valvular disease, the most frequent form of which is mitral insufficiency. This, however, by no means represents the full effect of rheumatic endocarditis as a cause of the valvular imperfection, for the cases discharged without evidence of such disease are often found, after the lapse of months, or perhaps years, during which no rheumatic phenomena have been manifested, to present undoubted evidence of mitral regurgitation. The process of the changes in rheumatic endocarditis is slow and is not necessarily betrayed by symptoms.

In a considerable number of cases of mitral insufficiency in children no evidence of rheumatism is to be obtained. For instance, in a series of 118 cases of mitral regurgitation under my observation I found an absence of any evidence of rheumatic association in 40. In 8 of these there appeared to be a definite relation in sequence to scarlatina, in 6 to measles, and in 3 to scarlatina and mumps. In 13 cases there was no evidence of any antecedent disease to account for the valvular imperfection. Post-mortem evidence showed that the morbid changes in these were identical with those observed in cases known to be rheumatic (69).

In the cases in which there is no evidence of rheumatism the child may be brought under notice for a disorder of nutrition—especially wasting and anemia—or for a disturbance of respiration, such as cough and dyspnoea, the results or concomitants of the heart disease; or for an affection of the nervous system, such as chorea, epilepsy, or hemiplegia. In some of them there is cerebral embolism, the plug being derived from the diseased endocardium. Not infrequently the valvular disease is discovered by accident. No notable discomfort may be caused by the movements of the child in play nor on running upstairs; and Henoch says that in many cases the disease is first discovered by the mother observing the violent
motion of the heart when she strips the child to give the bath. The evidence points to the conclusion that a form of endocarditis which has the essential characters of the rheumatic may occur in infancy and childhood without any other manifestations of rheumatism (17). Endocarditis then may occur as a solitary expression of the rheumatic disease (Archibald Garrod). [Vide art. "Acute Rheumatism of Childhood," vol. iii. p. 42.]

The symptoms observed in childhood during the progress of uncompensated mitral inadequacy are very varied. The age of the child has some influence in regard to these. As a general rule, the signs in infants and very young children are chiefly those of inanition,—emaciation, anaemia, and deformity of the thorax. There are in many cases frequently-recurring attacks of bronchitis or broncho-pneumonia, cough being a prominent symptom. In children after the age of four years symptoms more directly indicating disorder of circulation become manifest: bleeding at the nose may be cited as one of these. Difficulty of breathing becomes apparent, and in some cases most distressing orthopnoea. Precordial pain and discomfort are severe symptoms in some cases, and these may be associated with lumbar pain. Palpitation may be a distressing symptom. Dropy is by no means uncommon, but it rarely follows the gradually ascending course usual in the adult; the oedema is either more general, or more variable in the sites of its manifestation. In cases with oedema or ascites albuminuria is a frequent complication: this may be transient and due to venous congestion, but in the majority of cases it is dependent on the coexistence of inflammation of the kidneys, and is a sign of dangerous import. In the later stages of the disease vomiting and diarrhoea may be observed as most serious indications; haematemesis occurs in some cases. A marked anaemia, occasional vomiting, restlessness followed by apathy, and partial unconsciousness are symptoms which in many cases mark the weeks or days preceding the close of life.

In the form of mitral insufficiency attended with general carditis the prognosis is bad. The pericardial adhesions and the consequent hypertrophy and dilatation of the whole heart are a constant menace, and prevent satisfactory treatment. On the other hand, an uncomplicated mitral insufficiency in childhood often has a favourable issue, and the results of treatment even when the severe symptoms of threatened failure are present are often very satisfactory. Henoch considers that children recover from rheumatic endocarditis better than adults, and that in them the valve is more likely to regain its structural integrity. Cadet de Gassicourt has given his opinion that, whilst in the adult the valvular thickening increases, and the sclerosis at the auriculo-ventricular opening becomes more and more considerable, so that incurable disease remains, in the child there is more probability of absorption of the morbid products: then, the obstacle being removed, the growth of the cardiac muscle ceases to be exaggerated, and the heart, at one time too large for the child, comes by degrees into due proportion with the needs of the adult (71).
In the treatment of mitral insufficiency in the child when compensation fails and the symptoms are those of progressive cardiac enfeeblement—the condition being one of chronic disease uncomplicated by acute rheumatism—the following are the chief points to be observed:—(i.) Rest in the recumbent position, or in the semi-recumbent with the shoulders supported, must be maintained as much as possible. (ii.) Precordial pain and discomfort or difficulties of respiration call for the application of warmth to the chest by warm moist flannels, spongio-pilina, or the jacket poultice. On some occasions a digitalis poultice may with advantage be substituted for the ordinary linseed meal poultice: this is made by boiling two ounces of digitalis leaves in a pint of water for ten minutes, about two ounces of linseed meal being gradually added until the proper consistence for a poultice is attained. The mass is of course to be spread upon suitable material and applied in the usual way. (iii.) Means for inducing good general nutrition are of the first importance. A child with mitral incompetence is often intensely anaemic. Cod-liver oil, by itself or in an emulsion, or in combination with some of the iron preparations, is very beneficial. In some cases small doses of arsenic (Fowler’s solution), with tincture of nux vomica or liquor strychninae, succeed better than iron. In not a few I have seen a plan of supplementary alimentation by nutritive enemas turn the scale towards amendment. One of the best of such enemas is made by shaking together in a bottle one egg, an ounce of hot milk, and an ounce of cod-liver oil, and administering very slowly through a large soft rubber male catheter, with a funnel attached and held at a sufficiently high level, or by an india-rubber enema tube. The administration should be twice or three times daily. (iv.) Cardiac tonics are to be prescribed with judgment. In some cases rest, carefully regulated diet, and the tonic methods just mentioned suffice, and all agents which directly influence the cardiac rhythm are unnecessary or even injurious. Of all cardiac tonics digitalis is of the greatest value; it is especially so when dyspnoea is a marked feature. The drug may be given in the form of the tincture in doses of from one to five minims, or the infusion, ten minims to one drachm, or the leaves in powder, one-fourth of a grain to half a grain, repeated three times a day. There is some difference of opinion whether the administration should be continuously for long periods or in larger doses with omissions for several days. In some instances digitalis is not well borne, and in children this intolerance is usually shown by the occurrence of vomiting; it should be omitted whenever vomiting appears. In cases when digitalis administered by the mouth seems to be inert, rapid improvement may follow the hypodermic injection of digitaline \( \frac{1}{100} \) to \( \frac{1}{25} \) of a grain for a child of from six to twelve years of age. In any case such hypodermic injection should not be repeated for at least forty-eight hours. As an alternative to any preparation of digitalis caffein citrate dissolved in water or in the ordinary saline mixture, in doses of from one to three grains three times a day, may be given. The administration should not be continuous, but for a period of four to six days, with similar periods
of suspension; for all cardiac tonics, though tending at first to increase
the excretion of urine, by their prolonged action often tend to diminish it.
In cases where as a consequence of mitral regurgitation the right cavities
of the heart are much dilated—especially when the tricuspid valve is
rendered incompetent—digitalis and other cardiac tonics may be powerless for good. Their inefficiency is readily to be explained, for it must
be remembered that their action is on both ventricles, and that they
augment the force of the right ventricle as well as that of the left: now
increased action of the right ventricle means so much the more reflux into
the general venous system and further disasters. In many cases where
there is such distension of the right cavities (an occurrence which may
supervene as an acute phase in a case of chronic mitral insufficiency), the
relief of venous pressure by leeching is a most valuable auxiliary to
treatment. One or two leeches may be applied to the precordia, and
the leeching may be repeated on several occasions at intervals of two or
three days. Exceptionally, half-a-dozen leeches may be applied at the
first. I have often observed that digitalis, which has been powerless for
good before the application of leeches, proved of great service thereaft
(58).

Dropsy, in cases of chronic mitral insufficiency in the child, may be
transient, and yield to the medicinal treatment already sketched out; or it
may become a far more serious symptom. There may be general anasarca,
and pronounced ascites and effusion may rapidly take place within the
pleural cavities. In a considerable proportion of cases desquamative
nephritis is manifested in the course of the mitral disease. In the treat-
ment of such cases, sponging of the skin with hot water made alkaline with
sodium carbonate, the child being afterwards wrapped in a hot blanket, is
often a more practicable and efficient measure than the administration of
a hot-air bath or a vapour bath. Purgatives, as compound jalap powd
er, are essential; at first calomel may advantageously be administered with
Saline diuretics are to be combined with digitalis and decoction of
broom. The removal of all traces of dropsy in the child is sometimes
rapid. In some cases medicinal means fail. As a rule, punctures of the
skin of the lower extremities and the use of Sputhey's tubes in the treat-
ment of dropsy in the child are not to be recommended; there is a
danger that restless movements may cause chafing and irritation. If there
be ascites, paracentesis abdominis should be performed; sometimes rapid
convalescence follows this operation. Sedatives and medicines to procure
sleep must be used with caution, but in many cases they are indispen-
sable.

In mitral insufficiency, the result of rheumatic endocarditis in the adult, we
find associations differing from those in the cases of children. In adult
life the occurrence of general carditis and the implication of pericardium,
endocardium, and myocardium in the rheumatic disease are much less
common. In this sense the disease is less formidable than in the child.
On the other hand, repeated storms of endocarditis in the adult increase
the sclerosis at the mitral orifice and the imperfection of the curtains,
cords, and columns; the thickened fibrous structures tend also in progressive degrees to undergo degeneration and calcareous transformation. The already diseased endocardium may be attacked by pathogenetic microorganisms; the endocarditis may be septic. This is especially probable in women after parturition, and in both sexes when there are dangers of septicaemia; but the disease may arise insidiously without traceable infection. The causes of overstrain, both physical and mental, which affect the adult warrior in the battle of life adversely modify the conditions. Emotions disturb the rhythm of the heart and tend to spoil the compensation. Severe physical efforts may rupture curtains or cords already diseased. Diseases of various forms may alter the nutrition of the heart-muscle. There are probably many forms of disease affecting the coronary arteries and their branches within the heart; arteritis and periarteritis occur in many forms of infectious disease, and notably in syphilis. Arterio-sclerosis involves the coronary arterioles (especially in chronic Bright's disease), and the larger branches in the later periods of adult life become affected by atheroma. The result of all such morbid alterations of the walls of the arteries is an impairment of the force of the cardiac muscle with subsequent degenerations. Intercurrent diseases of the lungs, again, may rudely interrupt a compensation hitherto satisfactory. In some cases causes of inflammatory irritations are imported from without. In others infarctions or so-called pulmonary apoplexies are both consequences and causes of cardiac failure. Any considerable interference with the function of the lungs imposes a direct obstacle to the work of the right ventricle. It is the energy of the right ventricle that, by impelling an abnormally large volume of blood through the pulmonary vessels, and thus antagonising in the left auricle the regurgitant stream from the left ventricle, is the effective agency of compensation.

The symptoms in the adult of a failure of the compensatory conditions in cases of insufficiency of the mitral valve are briefly, and in an approximative way chronologically, difficulty of breathing, especially upon effort, but also paroxysmally; cough, with physical signs of oedema of the bases of the lungs, and often of localised consolidations; and dropsy, gradually extending from the more dependent portions of the body. From all such symptoms and from the epiphomomena of embolism and infarction, pulmonary and systemic, there may be recovery. When the limits of possible restoration of the powers of compensation are reached, the picture is one of suffering and sadness. The recumbent position is intolerable, the lower limbs are persistently oedematous and their integuments indurated; the countenance wears the hue of combined sallowness and lividity, the expression is one of anxiety and of a restless craving for sleep, alternating with a feeble, helpless wandering of mind; there is abdominal discomfort from a large and tender liver; the arterial pulse becomes feeble and nearly imperceptible, and by slow degrees, with occasional awakenings to the reality of suffering and distress, life becomes extinct.

In the treatment of a case of mitral insufficiency in the adult, when
compensation is failing, rest is of the first importance. A practitioner to prescribe digitalis or other cardiac tonics in a routine fashion for patients who manifest morbid heart symptoms is a dangerous error. Rest, careful dieting, and judicious purgation may turn the scale towards recovery, even when dropsy, and signs of much venous engorgement of the viscera, have supervened (see case by Dr. Vivian Poore, 46). In a large proportion of cases, however, the difficulties are not to be thus surmounted, and recourse must be had to drugs, whose influence is especially upon the forces of circulation; of these digitalis is the chief. Digitalis may be administered in the form of the powdered leaves, the infusion, or the tincture. One grain of the powdered leaves is equivalent to one-third of an ounce of the infusion and to eight minims of the tincture. The leaves may be administered in doses of half a grain to a grain and a half three times a day in wafer cachet or pill, alone or combined with other agents such as mercury, iron, or aloes, or other aperients. The infusion may be given in doses of a quarter of an ounce to half an ounce, or the tincture from five to thirty minims.

In many cases the daily administration of digitalis can be continued for long periods, for a considerable number of months at any rate; but great care must be taken to ascertain that the patient is perfectly tolerant of the drug, and at the outset of this treatment the effects must be noted daily: the treatment should not be continued for more than three or four days without the control of a skilled observer. Digitalis has a complex action. It has a tonic effect upon the pneumogastric nerve, whereby its power of moderating and slowing the heart's movements is increased; but further, it increases the energy of the myocardium by a direct effect upon the neuro-muscular mechanism of the heart itself. It also augments the contractility of the walls of the arteries by an influence upon the vaso-motor centres and upon the local nervous mechanism of the muscular coat of the vessels. The good effects of digitalis are manifested by its so lengthening the diastolic pause that the ventricles become more completely filled, and deliver ampler blood-waves into the general arterial system. The arterties, when moderately contracted, do not impede the blood-flow; in fact, a larger amount of blood traverses the circulation in a given time. The ventricles, emptying themselves more completely, the previously dilated heart diminishes in volume. The beneficial effect of digitalis is also shown in the production of diuresis. Neither the heightened arterial pressure nor the augmented urinary outflow produced by the drug is, however, by any means constant. Variations of arterial blood-pressure under the action of digitalis have been noted by many observers to be quite independent of the slowing effect upon the heart; indeed the diuretic results are confined almost entirely to those cases that manifest edema. It seems probable that the fluid absorbed from the lymph-spaces, drawn within the capillaries on account of the augmented rapidity of the circulation, and carried to the renal capillaries, so stimulates the kidneys as to provoke diuresis. When there is no effused lymph to be absorbed, diuresis does not result;
in fact, the urinary outflow in some cases diminishes even to arrest, and there may be hematuria. Digitalis is contra-indicated when nausea, vomiting, and diarrhoea form part of the symptoms, and when the pulsations of the heart are rendered inordinately slow. When the administration of comparatively small doses is continued too long there may be a sort of chronic poisoning; the signs are pallor of surface, coldness, and, sometimes, attacks of faintness; it would seem that cerebral ischaemia is thus produced (Duroziez, 14). I am of opinion that the dangers of the prolonged administration of digitalis are too often ignored. Certain effects of digitalis may persist long after cessation of the administration. Abnormal retardation of the heart's contractions has been noted ten days after omission of the drug (Raven), three weeks (Potain), 28 and 29 days (Duroziez). The practical rule should be that average doses of the preparations of digitalis, repeated at intervals of four hours, should not be continued, in the earlier stages of treatment, for more than three days; then the drug should be suspended for a like period. It is only when a patient manifests a perfect tolerance that the protracted administration should be permitted.

The employment of digitaline is preferred by many physicians, especially by the French. It is to be remembered that the various digitalines vary greatly in strength; that of Nativelle has about fifteen times the strength of the digitaline of Homolle, weights being equal. Potain prescribes for a case of cardiac failure with dropsy one milligramme of Nativelle's crystallised digitaline. This may be administered in one dose, or, if tolerance be doubtful, it may be divided into four or five doses given in as many days. After the administration there is often profuse diuresis. There should be no readministration for many days—the interval may be from ten days to three weeks; renewed acceleration of the pulse is to be taken as an indication for repetition of the treatment.

Digitaline may be administered hypodermically. When satisfactory effects have not followed administration by the mouth, I have seen excellent results follow the hypodermic injection of digitaline in the form of a solution of the discs of Savory and Moore. Each disc contains $\frac{1}{100}$ of a grain. The dose should not exceed two discs ($\frac{1}{50}$ grain).

In cases in which the right chambers of the heart are much distended the abstraction of blood is indicated. A bleeding from the arm to the extent of six or eight ounces coincidentally with the administration of digitalis, or subsequently to it, will often turn the scale towards recovery.

In some instances of mitral insufficiency, the consequence of rheumatic disease, the treatment by digitalis entirely fails; there seems to be no good effect upon the left ventricle, the right cavities continue to dilate, dropsy increases, and the drug in combination with ordinary diuretics fails to increase the outflow of urine. Other cardiac tonics and various combinations of these may then be tried.

Caffein or its citrate may be given in doses of from three to five grains
every four hours, but, as in the case of digitalis, it is better that it should not be used for more than three days continuously. I prefer to administer it in the ordinary saline mixture (solution of ammonium acetate). Its action is in many points similar to that of digitalis, but it has a much less effect in retarding the pulse and also in causing contraction of the arteries. Its diuretic influence is decided, and, unlike digitalis, it stimulates the renal epithelium to the excretion of solids. I have found the diuretic effect to persist after the suspension of the drug. In some cases this result is coexistent with good and rapid recovery from all distressing symptoms; but diuresis may occur and persist, and yet the result be unfavourable. The drug very rarely induces insomnia; I have more frequently observed that by lessening the dyspnoea it has promoted sleep. In some subjects, however, it produces agitation, headache, vomiting, purging, and sleeplessness. The combination of digitalis and cafftein may act more favourably than either drug alone (4).

Theobromine, in the form of the sodio-salicylate (diuretin), may be substituted for cafftein. It is to be administered in doses of 15 grains six times in the twenty-four hours. It is freely soluble in water. It has a stronger diuretic action than cafftein, and does not cause nervous agitation and sleeplessness. The diuretic effect is manifested between the second and sixth days of its administration.

Strophanthus may be administered in the place of digitalis, cafftein, or theobromine. It is given in the form of tincture (two to ten minims, in chloroform water or with alcohol), or of tabella, each of which is equivalent to two minims of the tincture. The dose may be repeated every four hours; the same care in watching effects and suspending the administration at intervals of a few days should be used as in the case of digitalis. The action of strophanthus upon the heart by the way of the vagus and through the local neuro-muscular mechanism resembles that of digitalis (Fraser, Popper, Bucquoi); but, according to Roy and Adami, it stimulates the contraction of the papillary muscles to a far greater degree than that of the ventricular wall; these observers have shown that on repeating the dose so that the more pronounced toxic action of the drug is manifested, the papillary muscles become notably weakened, and even their power of contraction annulled. Fraser concludes that strophanthus acts upon the heart more forcibly than digitalis, but on the calibre of the arteries infinitely less. It has often a very favourable effect upon the difficulties of breathing, and, used with care, is an efficient and useful substitute for digitalis; but it is not without its dangers. Its protracted use may cause dyspepsia with diarrhoea and wasting (Lemoine), and there are some probabilities that it may lead to sudden death in the course of its administration for heart disease (Gottlieb). I cannot doubt that the protracted injudicious administration of digitalis and strophanthus—especially in those who absorb these drugs without skilled medical supervision—has often been productive of dangerous and fatal results.

The other substitutes for digitalis, Adonis vernalis, Cactus grandiflorus, etc., are not of proved importance. Spartheine has no notable advantage
over the broom tea (decoclitm scoparii) which contains it; the latter is useful as a diluting agent for the heart tonics already considered. Convallaria majalis will be considered in reference to the therapeutics of mitral stenosis.

Treatment of dropsy.—By the means already indicated, together with the administration of such purgatives as produce watery evacuations—one of two purgative doses of calomel are often of service in the early stage of treatment—may suffice to remove all traces of dropsy and to restore compensation. In other cases where the dropsy does not disappear the mechanical removal of the effused fluid may be necessary. Incisions by a lancet or punctures by a needle may be made into the skin of the lower extremities, the limbs being wrapped in flannels or other absorbent material to take up the fluid which copiously drains away; or the fine trochars and canulas known as Southey's tubes may be used. In either case the skin should be previously sponged with alcohol, ether, or an antiseptic solution. The former plan is to be preferred in the case of a delirious or very restless patient; the latter when the patient is tranquil enough to allow the fluid to flow gradually through the fine flexible tubes into the receptacle underneath the bed for many hours. The trochar should be inserted very obliquely beneath the skin; the opening of the canula should be at the extremity (and not at the sides), and the flexible exit tube in the portions nearest the inserted canula should be fixed to the skin of the leg by strips of adhesive plaster; it should also be arranged so that it does not kink and obstruct the flow. It is best, when the anasarca is considerable, that two canulas with tubes attached be inserted into each lower extremity. When ascites exists, the fluid within the abdomen may be drawn off by the slow process of draining through a small canula and fine tube, or by the more rapid process of paracentesis abdominis. I prefer the more speedy withdrawal by a comparatively large trochar. When ascites coexists with general anasarca it may be a question whether draining the subcutaneous tissue or tapping the abdominal cavity should be first performed. When the abdomen is not much distended the former should be practised first, for after the draining the intra-abdominal effusion may become absorbed. When the ascites is considerable paracentesis abdominis should take the precedence. Effusions within the pleural cavity should be withdrawn at once.

Agents for producing sleep or calming nervous agitation are of high importance in the treatment of the failing heart of mitral insufficiency. In some cases chloralumide has been useful, as it is always a harmless hypnotic. It may be given in doses of from 20 to 50 grains in wafer cachet or in weak spirituous or acidulated solutions. Each draught should be made up separately. I prefer a combination of 20 or 30 grains of chloralamid with 30 minims of dilute hydrobromic acid with a drachm of syrup of orange flowers and an ounce of pure water, administered at bedtime. Another harmless agent is urethane (ethyl carbamate), which is freely soluble in water, the solution having a saline but by no means unpleasant taste. In doses of 15 to 20 grains at bedtime I have found it
induce a calm, natural sleep lasting in a case of severe cardiac failure for more than five hours, the patient being manifestly refreshed on waking. Paradehyde is perhaps a little stronger as a hypnotic. It may be administered in doses of from 30 to 90 minims in diluted syrup or in almond mixture, or in capsules (each containing 40 minims); it has a powerful and unpleasant taste.

In a considerable number of cases manifesting distressful symptoms of dyspnoea and insomnia no agent succeeds so well as morphia. By far the best way of administering it in cases of cardiac disease is by hypodermic injection. The solution of the acetate or the hydrochlorate or the solution of morphia and atropia may be used. The first dose should be small—one-sixth or one-fourth of a grain—but this may be increased subsequently to half a grain. Care should be taken that the administration shall not become habitual.

In regard to diet the aliments in the condition of failing compensation in mitral insufficiency should be very simple. Milk is the best of all foods, but in some cases is hardly tolerated. In the gastric crisis accompanying the failing heart there is often a complete disinclination for food. Then peptonised milk or milk gruel may be swallowed in sipping fashion, the patient being never permitted to take a distinct meal, nor a particle of solid food (Sir Wm. Roberts). In such cases I have seen great benefit follow the administration of peptonised enemas or the cod-liver oil milk and egg enema already mentioned in the treatment of children. Brandy, if given at all, should be in teaspoonful doses with milk and wine only. Sherry, marsala, or tokay may be given in jellies. At the subsidence of the crisis, as soon as milk can be well borne, an all-milk dietary, especially if there be dropsy, should be prescribed until convalescence.

The diet and hygiene during the stage of comparative convalescence will be considered with the third group of cases.

Group II. Mitral regurgitation in chorea.—In the majority of cases of chorea a systolic murmur, having the characters which indicate regurgitation through the mitral orifice, is manifested at some period of the disease or throughout its whole course. In a large section of such cases the signs and symptoms are such as to leave no room for doubt that the imperfection of the valve has been caused by rheumatic endocarditis. In many instances of chorea there has been antecedent rheumatism; the proportion varying, according to the beliefs of individual observers, from 8 per cent (Hughes) to 30 per cent (Pye Smith), 31 per cent (A. E. Gartrod), and 32 per cent (Sir Andrew Clark). There is a consensus of opinion that about one-fourth of all the subjects of chorea are or have been rheumatic. In many also of those who have personally shown no evidence of rheumatism there has been a family tendency to the disease. The doctrine has been formulated that chorea is in all instances a rheumatic affection (Roger); other observers (Stephen Mackenzie, Barlow, and Cheadle) have estimated that in from 45 to 75 per cent of the cases there are sufficient evidences of rheumatic tendency; it may be
concluded, therefore, that in the majority of cases chorea is a phase of rheumatism. It must be allowed that in many of the cases the diagnosis of rheumatism (reposing as it necessarily does on the statements of unskilled observers, with whom as a matter of common experience almost every painful affection is rheumatic) can be by no means precise. If causes of fallacy be excluded we may perhaps take it as a fair working hypothesis that about half the total cases of chorea are rheumatic, and that the endocardial murmurs manifested in these patients are due to structural disease of the valves, the result of the rheumatic form of endocarditis. In this section of the cases the mitral incompetency which is the concomitant of the disease is to be estimated and treated—when any failure of compensation renders such treatment necessary—according to the rules already laid down. The therapeutics of chorea are discussed elsewhere.

Nearly all observers, however, are agreed that some cases of chorea are non-rheumatic. It is well known that a sudden shock or terror may be the precursor of chorea: such a cause may operate in a case undoubtedly rheumatic, but, in common with many other observers, I have seen many cases of chorea where a sudden and violent emotion preceded the attack in a person who showed no sign of rheumatism, nor any proclivity thereto. Dr. Stephen Mackenzie's statistics showed rheumatism and fright to be nearly equal, numerically, as antecedents of chorea (38). Observers are generally agreed that emotional and mental disturbances have a large share in the immediate causation of the disease (A. E. Garrod). "The only immediate cause of chorea that can be traced with any frequency is emotion, usually fright, rarely mental distress" (Gowers, 20). The heart affection, in Dr. Stephen Mackenzie's statistics of cases of chorea, was associated with rheumatism in 50 per cent; whilst in 35 per cent no such association was recorded. In non-rheumatic chorea I consider that the symptoms and signs of mitral insufficiency differ from those in the rheumatic cases. In some of these, careful examination for many days may detect no evidence of valvular disease; then a soft and slightly pronounced systolic murmur, localised at the position of the heart's apex, may become audible. There is no accentuation of the pulmonic second sound; the ventricles do not become dilated; yet the murmur, having its original characters, persists for several years. At later periods it may become completely inaudible. The late Sir Andrew Clark held that the murmurs of mitral regurgitation so frequently observed in cases of chorea disappear, in the great majority of cases, within eight or nine years of the attack (10). These clinical features greatly differ from those of mitral insufficiency due to rheumatic endocarditis. The evidence of morbid anatomy completes the distinction. In cases of fatal chorea wherein a soft, apical, systolic murmur has been observed during life, the left auriculo-ventricular orifice on its auricular aspect has been found studded and fringed with small, firm outgrowths having the signs of papilliform elevations of the endocardium. These outgrowths are firm to the touch, and are not detached by rubbing with the finger. The endocardium is smooth over them. They do not
begin, as in rheumatic endocarditis, with a change in the epithelium and an attachment to the roughened surface of fibrous caps, but they are firm outgrowths showing fibrous hyperplasia. Their formation is not followed by the sclerous changes, the widely-spread fibrous proliferation, the retractions of valve curtains, cords, and columns so frequent in rheumatic endocarditis. On the other hand, they interfere but little with the closure of the orifice in systole, and in process of time, the endocardium remaining quite smooth, they come to have no pathological significance whatever. It seems to me probable that they may be the immediate results of a sudden overstrain and rupture of the terminal arterioles distributed to the valve structures. The immediate symptoms induced by terror or by any sudden mental shock is a blanching of the surface of the body, a contraction of the arterioles, a stimulation or over-action of the sympathetic nerve mechanism. The effect on the heart at first would seem to be arrested action, afterwards palpitation. In the case of the delicate arterioles of the endocardium of the valves the result might well be ruptures:—mimic hemorrhages, followed by thickenings analogous to those observed after the experimental production of overstrain in animals (Roy and Adami).

In cases of chorea in which there is no evidence of failure of compensation, but only a systolic murmur at the apex to indicate some incompleteness of the closure of the mitral orifice during the ventricular systole, all treatment by cardiac tonics, or by means specially directed to the valvular imperfection, is unnecessary, and probably mischievous. The therapeutic methods adopted should be those for calming the tumult of the nervous system and for ministering to a healthy nutrition.

Group III. Mitral insufficiency the result of dilatation of the left ventricle.—This group must of necessity be subdivided. In some cases the dilatation of the ventricular wall is from mechanical causes. This can be traced in the case of disease of the aortic valves, which has caused obstruction, regurgitation, or the combined lesions. For long periods no murmur is heard at the apex, but later the systolic bruit of mitral regurgitation becomes audible, and the case, which formerly presented no such signs, begins to manifest the venous congestion, the rising dropy, and the forms of dyspnea of mitral disease. A similar sequence may be observed in chronic Bright's disease with arterio-sclerosis. The left ventricle may for long periods show signs of hypertrophy; then signs of dilatation are manifested more or less rapidly; later the murmur and the signs of mitral insufficiency are observed. The ventricle has become hypertrophied, or dilated, and hypertrophied, from the resistance in the aorta and the

1 Witness the words of the poets:

I could a tale unfold whose lightest word
Would barrow up thy soul, freeze thy young blood,
Make thy two eyes, like stars, start from their spheres,
The knotted and combined locks to part
And each particular hair to stand an end,
Like quills upon the fretful porcupine.

SHAKESPEARE.

Obstupui, susteruntque comae; vox vocibus laesus.

VIRGIL.
peripheral vessels on account of the thickening and contraction of the smaller arteries. The intra-ventricular overstrain continuing and increasing—because of the augmenting arteriole-obstruction—the left ventricle yields to such an extent that the mitral curtains fail to coapt during ventricular systole.

In another set of cases there may be none of the ordinary signs of chronic Bright’s disease, nor of thickening of the walls of the systemic arteries; and yet, in patients who have manifested no signs of rheumatism, nor of endocarditis, the physical signs show dilatation of the left ventricle and finally mitral insufficiency. In some of these it is found after death that there have been arteritis and periarteritis in the vessels of the heart itself; in others atheroma of the coronary artery of the left ventricle and tracts of degeneration, molecular, fibrous or fatty, corresponding to the area supplied by the branches of the artery. In another subsection, these patients being usually obese and often alcoholic, there is fatty infiltration amongst the cardiac muscular fibres, and the left ventricle yields because of the imperfection of its muscle. In yet another subsection in this group the heart becomes dilated to the degree of incompetency of the mitral valve from a morbid affection of the nervous system. Probably the nervous influences disposing to dilatation of the left ventricle have been too much overlooked. I have traced a rapid and extreme dilatation of the left ventricle coincidently with signs of neuritis of the vagus. In several cases the complete signs of dilated ventricle and mitral insufficiency have come on in the course of Graves’ disease; these will be considered hereafter.

It is obvious from these considerations that dilatation of the left ventricle with mitral insufficiency, apart from structural disease of the valve, may be the result of various and complex morbid states. It must be remembered that these complex morbid conditions may coexist with structural disease the result of rheumatic endocarditis, which has already been discussed.

These considerations must have their due weight in questions of treatment. In cases of arterial obstruction in the subjects of chronic Bright’s disease, and often in patients after middle life, digitalis and all forms of cardiac tonics fail, or even do positive harm. In such cases good may result from the administration of arterial relaxants, and with these digitalis may often be associated. Dr. Balfour considers that digitalis cannot be safely given in cases of senile heart without a simultaneous unlocking of the arterioles. The cardiac tonic, therefore, should be combined with iodide of potassium or sodium, or with a nitrile, such as nitrite of ethyl (nitrous ethyl), nitrite of sodium, or nitro-glycerine. In cases in which there is reason to suspect thickening of the walls of the arteries—in the general arterial system, or in the heart itself—a long course of the iodides is to be advised. Digitalis may be also administered for periods of two or three days at long intervals. Trinitrine should be prescribed if any sign of intolerance of the iodides be noticed; or if these seem to be inefficacious, it may be administered in one-minim doses
of the one per cent spirituous solution; or in the form of tablets in which \( \frac{\text{15}}{\text{16}} \) grain of nitro-glycerine is combined with chocolate. For continuous administration I prefer very small doses (\( \frac{\text{13}}{\text{16}} \) grain) three times a day. A combination with amyl nitrite is in some cases a distinct advantage, for example, nitro-glycerine \( \frac{\text{100}}{\text{16}} \) grain, amyl nitrite \( \frac{\text{1}}{\text{4}} \), menthol \( \frac{\text{1}}{\text{10}} \) grain, capsicium \( \frac{\text{1}}{\text{4}} \) grain, with chocolate to form a tablet (Pharmacopoeia of the Westminster Hospital).

When a case in this group shows signs of marked cardiac failure, such as severe dyspnoea and dropsy, complete rest in bed should be enjoined. Before the administration of any cardiac tonic it is well that purgatives be administered. A dose of calomel, three to five grains, is a good beginning; or the patient, having abstained from liquids for some hours, may take two to four drachms of sulphate of magnesia in hot water (Matthew Hay). A considerable watery discharge may rapidly reduce the oedema. The patient should be cautioned against getting out of bed, or even assuming the sitting position during the relief of the bowels, lest syncope be thus induced. The trunk should be supported by pillows and the bed-pan used.

In cases in which dropsy is not extreme, massage may be of great advantage. The muscles of the extremities and of the thorax should be gently kneaded. Abdominal massage should be practised with caution; to dilate the vessels within the splanchnic area may induce anaemia of the brain. Massage of the extremities aids the venous circulation, quickens the function of the absorbents, and tends to bring about a more deliberate and efficient ventricular systole.

In the grave conditions of failure of compensation it is best that the diet be exclusively milk, diluted with barley-water or peptonised. Small quantities should be swallowed at a time. Milk is a notable diuretic, and in the dropsical stages it should form the staple diet. All strong extracts of meat, which contain many products of retrograde metabolises, are to be forbidden; but chicken or veal broth and jellies may be permitted in some cases. In the stages of recovery three to six pints of milk may be taken in the twenty-four hours.

When the patient begins to be able to take some walking exercise, and the probability of resuming ordinary avocations comes into consideration, the question of limitation of the ingestion of fluids has to be settled. Oertel permits only 34 to 36 ounces of water, including that contained in the solid food, per diem. The best proportions of food are said to be, about 1 ounce of fat, 3\( \frac{1}{4} \) ounces of carbohydrates, and not less than 5 ounces of proteids. A cup of tea morning and evening, about half a pint of claret, from 8\( \frac{1}{2} \) ounces to rather more than a pint of water, and a little over 3 ounces of soup, should constitute, besides that contained in the solids, all the fluid taken during each day. The solid diet should be rich in nitrogen—for example, bread 4 to 5 ounces, meat or fish 6 to 7 ounces, with 5 ounces of chicken or game, one or two eggs, a little salad, cheese, etc., and 3\( \frac{1}{2} \) to 7 ounces of fresh or cooked fruit (43).
As compensation is recovered, and during its maintenance, systematised muscular exercise is a valuable therapeutic means. Stokes, in 1854, said that "the symptoms of debility of the heart are often removable by a regulated course of gymnastics, or by pedestrian exercise even in mountainous countries such as Switzerland or the Highlands of Scotland or Ireland" (66). This opinion sounded the note of reaction against the routine practice of a long series of years of keeping a patient who presented any sign of heart disease in the most complete muscular repose attainable. Supposing that active disease be not going on in the cardiac tissues, a "coddling" policy, whereby the heart muscle is kept at a minimum exercise of function, is contrary to sound physiology and good practice. Saeterburg of Stockholm and Zander used gymnastics in the treatment of diseases of the heart, and described their experiences, which appeared to be very favourable in the period between 1862 and 1872. The Swedish system for the promotion of good physical development—the chief exponent of which was Professor Ling—became an important agency for preventive as well as curative treatment; the essentials being a forced action of the voluntary muscles for given periods. The order proposed by Ling for these exercise movements was (i.) respiratory, (ii.) lower extremities, (iii.) upper extremities, (iv.) abdomen, (v.) trunk, (vi.) movement of lower extremities repeated, (vii.) respiratory movement repeated. In the Zander system mechanical appliances were used for the special exercising of certain groups of muscles. Oertel in 1884 extended the doctrine and practice, and advocated, in a regulated and graduated manner, the promotion of vigorous muscular effort in mountain-climbing. The effort of ascending a hill is much more potent for good than that of walking on level ground. There is an increased flow of venous blood to the right side of the heart; the lungs become more fully expanded, the channels of the pulmonary circulation to the left auricle are more free, and the volume of blood delivered to the arteries by the left ventricle is greater. The perspiration causes a reduction in the volume of the fluid blood, and a relative augmentation of the haemoglobin. The lymphatics are stimulated to their task of absorption. Many cautions, however, are necessary in the prosecution of this plan of treatment. If the efforts induce unduly rapid breathing, the patient should at once come to a rest and make deep inspirations. It seems to me that the plan is only good when, with the increased muscular effort, there is no considerable increase of the breathing-rate—the lungs must be adequately but not rapidly, imperfectly, and deceptively inflated. No effort must be sudden. It is the sudden overstrain, such as occurs in running to a railway station, that kills. Again, great caution must be exercised in sending cardiac patients to considerable altitudes. Dangerous and fatal symptoms have occurred even at moderate elevations above the sea-level.

The climbing of hills is not to every patient a possible method of treatment. Systematised gymnastic exercises exclude the necessity of hill-climbing. The exercises recommended by Dr. Schott of Nauheim
are known as resistance gymnastics (Widerstandsgymnastik). The patient, loosely and lightly clothed, is instructed to breathe quietly, and to make certain movements which are gently resisted by a skilled attendant, who uses for this purpose the palms of the hands, without grasping or constricting the limbs. The movements made are (a) various flexions and extensions of the forearm and upper arm; (b) movements of the lower extremities, the patient maintaining his position by resting his hand upon a chair; (c) flexions, extensions, and rotations of the trunk upon the hips. A short interval is enjoined after each movement, during which the patient sits down; the exertion should be only moderate in degree, and should cause no flushing nor pallor, nor quickened breathing.

It is not possible in all cases for a patient to have the assistance of a skilled attendant, yet much good often results from a course of systematic movements executed without such aid. These should be (a) exercises of the arms and coincidently of the upper thorax muscles, (b) of the legs both in walking and with the body at rest, (c) flexions and extensions of the trunk; thus movements are communicated to the abdominal viscera. No heavy weights, such as clubs or dumb-bells, should be used, and the muscles of one side of the body should not be exercised disproportionately to those of the other. So far as the movements of the upper extremities are concerned, these may be accomplished by the patient, standing erect or in the sitting position with spine straightened, holding lightly in the hands a rod or cane, and lifting this by deliberately calculated actions to the fullest extent above the head the rod is then brought down behind the shoulders, the chest being thus thrown forwards. The position of the rod is to be always maintained at right angles to the spinal column; the movements are to be repeated slowly and deliberately until there is a slight sense of fatigue.

The effect of exercise of the voluntary muscles is an accumulation of blood in their vessels of supply, and a corresponding derivation from congested areas—for example, from the right chambers of the heart and engorged veins (34). “The vessels which supply the muscles of the body are capable of such extension that when fully dilated they will allow the arterial blood to pour through them alone nearly as quickly as it usually does through the vessels of the skin, intestines, and muscles together” (Lauder Brunton, 7). The conditions, however, induced by muscular exertion are very complex. There are alternate contractions and relaxations, the former compressing the blood-vessels, the latter freeing these channels; concurrently there are increased activities of the absorbents and reflex nerve-stimulations. In the movements of the trunk upon the lower extremities another set of factors comes into play. The alternate compressions and relaxations of the abdomen affect the blood-supply to the abdominal viscera. The tendency must be in the main to cause the vessels in the splanchnic area to dilate and so to co-operate with those of the muscles in relieving any turgescence of the right cavities of the heart.

The use of baths and bathing in the treatment of ill-compensated mitral
insufficiency can be very useful. In years past there has been no doubt too great fear lest a patient presenting the signs of mitral regurgitation should catch cold; thus the ablutions have often been insufficient. The use of cool or cold water has been proscribed, and possibly hot baths have been too freely indulged in. The effect of a hot bath is evident to ordinary experience—causing dilatation of the vessels of the skin it may induce cerebral anemia with symptoms of faintness. The debilitating effect of repeated hot baths is well known. On the other hand, the invigorating effect of cold tub in those who can bear the shock, and of cool sponging in those who are more susceptible, are matters of common experience. For a long period the sending patients to any health resort for a course of treatment formed no part of the therapeutics of heart disease. Beneke in 1859 and 1861, and Groedel in 1878, adduced evidence to show that the baths of Nauheim, near Frankfort, in Germany, were beneficial in increasing the force of the heart and in restoring compensation in cases of valvular disease. Dr. L. Blanc in 1886 recommended the course of treatment at Aix-les-Bains by douches (temperature about 90° F.), together with skilled massage; and he cited 52 cases of mitral regurgitation in which this plan was pursued: in 15 of these all signs of disease disappeared, in 21 there was improvement, and in 16 the signs remained stationary (5). The chemical constitution of the water of Aix-les-Bains has probably but little to do with its therapeutic effect as used externally in these cases. Its chief value lies in its soft, unctuous quality, due mostly to the presence of organic matter (baréigne), which, when at the agreeably warm temperature at which it is used, adapts it admirably for the douche-massage. The therapeutic conditions of the employment of the Nauheim waters are more complex. These come from hot springs (temperature 83° to 100° F.), and are charged with saline matters, chiefly chlorides of sodium and calcium, and free carbonic acid gas. In marked feebleness of heart, and generally in the earliest stages of treatment, the patient takes a saline bath from which the carbonic acid has been allowed to escape; the duration of the bath is six to eight minutes, the temperature of the water being 95°. A rest of an hour is enjoined after each bath. The periods of immersion are increased during the course of treatment to twenty or thirty minutes, and the temperature is lowered by degrees to 85.5° F. The water used is allowed to retain its carbonic acid in less or greater proportion, as it is exposed for longer or shorter periods to the air, or used as the Strombad foaming with its full content of the gas. The effects of the various agencies thus put in force have been studied experimentally by Dr. R. F. C. Leith and others (31). In regard to temperature, simple thermal baths at 90° F. or under commonly tend to reduce the pulse-rate by five or seven beats a minute. The effect of the addition of sodium chloride to the bath is generally to emphasise the change in the pulse, and to make the bath more agreeable to the patient; when the bath is charged with carbonic acid gas (Sadow's effervescing tablets being used) the pulse-rate is further reduced, whilst the force of the heart's action is increased; the
pleasantry and buoyancy of the bath are also enhanced, and the patient experiences an agreeable sensation of warmth. The result of a bath at a temperature below body-heat is contraction of the cutaneous vessels of the area immersed, higher temperatures cause their relaxation; the lymph-circulation is necessarily modified, the internal vascular conditions are changed, dilatations of the vessels occur in various regions—notably in the vascular districts of the brain, and probably there are some rhythmic alteration of dilatations and contraction. Furthermore, there are reflex effects upon the vaso-motor and cardio-inhibitory centres. When the bath contains free carbonic acid gas the fine bubbles adhering to the skin protect the body from the colder surrounding water, and constantly impinging upon the surface stimulate the cutaneous nerve-endings. Probably also some of the gas permeates the skin; carbonic acid has been shown to be a notable and valuable local anaesthetic (Ozanne).

The effects of the combined treatment by baths and muscular exercises as carried out at Nauheim are said to be increased strength of the pulse with diminution of its abnormal frequency, decreased rate of respiration, together with fuller inspirations and greater ease and comfort in breathing, and diminution in the size of the dilated heart. There is sufficient testimony to show that in a large number of cases there has been a great improvement in the subjective conditions. The evidence is less generally conclusive as to the reduction in size of the heart. From examination of a considerable number of outlines purporting to be those of the heart before and after the Nauheim treatment, I am of like opinion with Dr. G. V. Poore, Sir William Broadbent, Dr. Leith, and Dr. Herschell, that many are the results of a fallacious plan of physical examination, and cannot be held to represent with any degree of accuracy the size and position of the heart (24, 31, 45). On the other hand, there is a very high probability that in some cases the situation and shape of the heart have become changed, and the right chambers reduced in volume. Careful observations have shown that the bulk of the heart may greatly change under varying conditions within very short periods of time. In the case of mitral disease, whilst the patient has been at rest, and when no special therapeutic means could be invoked as causes, I have observed signs of very considerable variations in the bulk of the heart in less than twenty-four hours (61). Sir W. Broadbent says: "That a diminution in the volume of the heart may take place under the influence of saline baths and certain movements there can be no doubt, but such diminution is an occurrence which is perfectly familiar to all who are in the habit of noting the changes in the size of the heart under other methods of treatment or from various causes. In a heart dilated from over-exertion, for example, the apex beat may often be felt to come in for half an inch towards the normal situation, when the patient is simply made to walk two or three times across a room" (6). Not only the positions of the apex (Leith), but also the outlines of precordial dulness, have been found to vary at intervals during the day. Heitler considers from his observations that there are rhythmic changes
in the volume of the heart, the pulse remaining unaffected by these (22). All these considerations must have their due weight, and too much reliance must not be placed on the evidence derived from the ordinary means of physical examination as to the space occupied by the heart at a given time. The concurrence of signs,—the evidence of rational as well as of physical diagnosis,—however, shows that a combination of judicious bath treatment and physical exercises may be a valuable agency for good in cases of mitral insufficiency with failure of compensation.

One factor in the therapeutics of a health resort must not be overlooked. The change in surroundings must produce an effect upon the higher attributes of the nervous system—the will, the emotions, and the intellect. It is no slight advantage for a patient to be taken away from the little worries of home to a place where, with clear sky and pure air, there are facilities for systematic self-management, a prescribed and regulated dietary, and the associated hope and faith inspired by the favourable experiences of others. Mental and emotional impressions can strongly influence the trophic nervous mechanism of the heart. It is true that there is a reverse to this picture. Patients are sometimes deceived by false hopes and fallacious arguments; persons, for example, the subjects of mitral insufficiency, well compensated and causing no adverse symptoms, have been persuaded by so-called friends that calcareous incrustations and fibrous thickenings about their heart-valves would by the operation of a certain “cure” disappear as crystals dissolve in water. Long and arduous journeys have been undertaken by those who were totally unfit to leave the comforts of their home, and there has followed a sad awakening from the delusive dream. These agencies are potent for good or for evil, and every case in which the use of them is contemplated, must be careful considered.

**Group IV. Mitral insufficiency from anaemia.**—A systolic murmur over the apex of the heart is heard not infrequently in the subjects of the various forms of anaemia; in some cases it is also audible at the back internally to the angle of the left scapula. Dr. A. G. Barrs found an apex systolic murmur alone in 13 out of 115 cases of anaemia. In 60 examples of chlorosis Potain observed a murmur, which he considered to be cardio-pulmonary, in nine cases above the apex, and in one case near the apex. Byrom Bramwell and Stephen Mackenzie have recorded cases of apex-systolic murmurs in cases of pernicious anaemia. I have myself found an apex systolic murmur in 7 per cent, and coexisting murmurs at the apex and over the site of the pulmonary artery in 9 per cent of cases of anaemia. The first question to determine is whether a bruit having such characters be due to causes operating externally to the heart itself. Potain describes all the murmurs heard in the neighbourhood of the heart which are causally related with anaemia and chlorosis as cardio-pulmonary; he finds that they do not begin with the systolic contraction of the ventricle as organic murmurs do, but are meso-systolic (occupying a portion only of the systole), that they are soft and superficial, greatly modified by the act of respiration, that they
are influenced by the attitude of the patient, so that they sometimes disappear when the recumbent is changed for the erect position, and that they vary from day to day. He considers that chlorosis tends to the production of cardio-pulmonary murmurs by influencing the nervous system, and so enhancing the cardiac excitability. When in a case of anemia a systolic murmur is heard at or near the situation of the apex, it is of importance (a) to determine by palpation and percussion the position of the apex beat of the outline of the left ventricle, and the relation of the observed murmur to the area thus determined; (b) to consider the various signs already noted which differentiate the cardio-pulmonary from the organic mitral murmur. A certain proportion may be found to answer to Potain's criteria of non-organic murmurs. I can have no doubt, however, that in some cases the apical murmur is due to veritable mitral regurgitation; first, because it has the site and characters identical with those due to organic causes, and, secondly, because it may be followed by all the symptoms of failure of compensation in mitral insufficiency. I have observed an apical systolic murmur to arise in a healthy woman after profuse uterine hemorrhage (from fibroids), severe dyspnea with abundant dropsy to follow, and ultimately complete recovery to take place, with the disappearance of all the physical signs of disease (56).

From the well-known association of fatty degeneration of the muscular fibrillæ of the heart with anemia, it must be inferred that the mitral insufficiency is caused, the valvular apparatus being normal, by the resulting enfeeblement of the myocardium. The incompetence may be from impairment of the muscle of the ventricular wall or of the musculi papillares, or of both. Positive dilatation of the left ventricle has been described by some observers (Goodhart, Stephen Mackenzie, Niemeyer). In these cases the incompetency of the valve is readily explained by the passive dilatation of the auriculo-ventricular orifice; on the other hand, the ventricle, and the heart generally, have been found by other observers to be abnormally small (Duroziez, Potain). I have observed cases in which there have been the physical signs of mitral regurgitation in anemia when the outline of the heart has been markedly smaller than the normal. The regurgitation in such cases may be explained by enfeeblement of the papillary muscles. In fatal cases of anemia these muscles have been observed to be profoundly affected by fatty degeneration.

The treatment of cases of mitral insufficiency, the result of anemia, is practically the treatment of the form of anemia which is the proximate cause. Though there may be very extensive fatty degeneration of the myocardium, there is good evidence that there frequently occurs a "restitutio ad integrum"; new and healthy muscular fibrillæ being developed. The good effects of tepid and cool baths in such cases may be briefly mentioned; the use of baths, spongings, and spinal affusions of cool or even cold water has been a routine practice with many physicians, myself included, in cases of anemia. The occurrence of a systolic murmur at the apex is no contra-indication to this mode of
treatment. The carbonic acid and saline baths, such as those of Nauheim, so much extolled of late, have been used very successfully for many years at Schwalbach, in co-operation with the internal administration of ferruginous water, in the treatment of anaemia. The modes in which such baths influence the heart and blood-vessels have been already discussed.

Group V. Mitral insufficiency in Graves' disease and allied affections.

—Murmurs in the precordial area are heard in a large number of cases of exophthalmic goitre. In the majority of these the maxima of the murmurs are over the base of the heart, and especially over the pulmonary artery. In a minority the systolic bruit is heard over the situation of the apex. I found an apex-systolic murmur in six out of a series of twenty-nine cases of Graves' disease. In some of these the bruit varied much and the diagnosis of mitral insufficiency was doubtful. In one case, however, that of a lady who had previously shown no sign of rheumatic change in the valve, the onset and course of the disease were carefully watched, and there could be no doubt of the establishment of mitral insufficiency. The disease was initiated by a sudden fright: after violent palpitation the pulse-rate rose to 160 per minute, a systolic murmur became evident over the apex, and general dropsy supervened with the usual signs of failure of compensation in mitral regurgitation. Complete recovery, however, succeeded, and the murmur disappeared, health being maintained for at least thirteen years after the acute manifestations (60). It is probable that in such a case the insufficiency of the valve was due not to endocarditis but to a disturbance of the nerve-mechanism of the heart. In some cases of Graves' disease dilatation of the left ventricle has been indicated during life and proved at the autopsy. In others the heart has been found to be quite normal. In one case of Graves' disease in a man, observed by myself, the dilatation was shown chiefly in the right chambers; the signs of tricuspid regurgitation were manifested in well-marked systolic venous pulsation in the neck. The evidence pointed strongly to the conclusion that the morbid conditions of the heart advanced step by step with the exophthalmic goitre, and that there was no pre-existing disease of the heart. I have found that dilatation of the heart has been by no means commensurate with the rapidity of its action. In cases of extreme tachycardia the outline of the heart has remained normal, whilst in the case of Graves' disease in the man, where the rapidity of the heart's action was far less, there occurred distinctly progressive hypertrophy and dilatation of the left ventricle. I consider it probable that the insufficiency of the mitral valve, which occurs in a minority of cases of exophthalmic goitre—structural valvular disease being excluded—has a like pathogeny with that which obtains in anaemia. The valve curtains fail to coapt in some cases on account of dilatation of the ventricle; in others because of enfeeblement of the papillary muscles, or faulty correlation between these muscles and those of the ventricular wall. In the treatment of these cases, supposing that there are signs of failure of compensation, the rules already laid down may be followed;
but another therapeutic agency demands consideration—the employment of electricity.

The treatment of the cardiac symptoms occurring in the course of exophthalmic goitre is notoriously unsatisfactory. The rapidity and irregularity of the heart's contractions in the majority, and the dilatation of the cavities in the exceptional cases, are not favourably influenced by digitalis or any form of cardiac tonic. Only those agencies which tend to calm the nervous perturbations can be relied upon. Yet I think that there is good evidence that patient and systematic electrification, carried out in such a manner that the pneumogastric nerve and the surrounding nervous elements can be directly influenced, is of therapeutic value. The interrupted current (faradisation) as well as the continuous galvanic current were employed by the late Professor Charcot and by Vigouroux. I have not found benefit from the treatment by the interrupted current, the immediate effects of which have indeed been objected to by many nervous patients; but I consider that in the employment of the continuous current the results have been good (8). The current should be weak—two to four milliamperes as given from three to eight Leclanché bichromate or chloride of silver cells; the anode should be placed at the nape of the neck, just above the vertebra prominens, and the cathode on the groove external to the larynx and trachea. The current should be allowed to pass for from six to ten minutes three times a day, the cathode, which may be moved over the skin, without lifting and re-applying, towards the clavicle, being adapted to each side of the neck alternately. This treatment in cases of Graves' disease manifesting severe and distressing cardiac symptoms has seemed to me more efficacious than any other, and a considerable number of patients have completely recovered. Although in many cases the heart-rate is often reduced after each application it is long before continuous improvement is obtained. I have seldom seen much amendment under six months of treatment.

The continuous galvanic current may also be of value as an aid to treatment in cases of failure of compensation in mitral insufficiency other than that which is manifested occasionally in Graves' disease. I have employed it in cases of chronic endocarditis undoubtedly of the rheumatic form, and it has seemed to turn the scale towards recovery. I have recorded the case of a young man who suffered from rheumatic endocarditis involving the mitral and aortic valves, and in whom extremely severe symptoms occurred during seven months. At a time when the signs were very grave the constant galvanic current from eight Leclanché cells was employed in the manner already described. Improvement soon ensued, the abnormal rapidity of the pulse was diminished, strength returned, and but for the warning note of a murmur indicating aortic regurgitation, the patient became a strong, well-nourished man (62). Poitain writes concerning electrification of the vagus: "Its efficacy is not limited to the thyro-cardia which accompanies exophthalmic goitre. We have been able to apply it advantageously in cases of cardio-arterial disease accompanied by marked excitability of the heart where heart remedies had absolutely
failed. It was always applied in the form of the constant current (descending), the positive pole being applied over the sides of the neck, and the negative on the anterior surface of the chest with an intensity varying between 10 and 15 milliampères" (47) It is probable that in the constant galvanic current we have a valuable therapeutic means for the treatment of some cases of mitral insufficiency.

A. Ernest Sansom.

REFERENCES

MitrAL STENOSIS

Definition.—A morbid condition of the structures at the left auriculo-ventricular aperture, causing a constriction of the latter and an obstruction to the normal flow of the blood from the left atrium to the left ventricle.

Morphology.—The appearances of the mitral valve and the structures adjacent to the orifice in mitral stenosis may conveniently be considered as they are manifested (a) in infancy and childhood, (b) in maturity and advanced life.

(a) In infancy and childhood the comparatively slight degrees of obstruction at the mitral orifice are marked by a ring of vegetations—in some cases friable and easily detached, in others sclerosed and firmly fixed—situated around the orifice on its auricular aspect. The fibrous structures subjacent to the vegetations are firmer than the normal, the thickening frequently involving the mitral curtains, the chordae tendineae, and the musculi papillares. In a more advanced stage the marginal portions of the curtains are joined by fibrous adhesions. At a still later stage the two curtains are so completely fused together that the valve presents the form of a hollow cone or membranous funnel, the wider portion of which is at the auriculo-ventricular orifice, and the narrower
points downwards within the ventricle near the apex of the heart. The funnel form of mitral stenosis, and the smooth polished membrane, regular in its conformation as a hollow cone, have suggested that the malformation of the valve is a congenital anomaly. It is undoubtedly true that in rare cases such an obstruction of the mitral orifice has been found in association with congenital malformation. In a case of this kind recorded by Parrot, the aorta and pulmonary artery were united in a single trunk. In one of my own cases the aorta arose from the right ventricle, and there was a communication between the ventricles. In these, and in all cases the records of which I have examined where the mitral orifice was found on post-mortem examination to be obstructed in infants who died shortly after their birth, the vegetations of endocarditis were found. In one of my cases, a babe of two months, a ring of granulations was found encircling the mitral orifice, and the valve was thickened. I consider that mitral stenosis, as observed in these cases, is not a congenital malformation, but the result of intra-uterine endocarditis—the smooth and regular conformation of the funnel constituted by the cohering curtains of the valve being due to the even pressure of the fluid blood both on the auricular and ventricular surfaces during the rhythmic movements of the heart. The terminal aperture of the funnel, by which the blood issued into the ventricle, may be extremely small, allowing the passage of nothing thicker than a goose-quill.

The fibrous thickening of the valve, of the chordae tendineae—which may be much shortened as well as thickened—and of the musculi papillares is in some instances very dense; in one patient, a girl aged eleven, these structures presented the characters of cartilage. Though the "funnel" form of transformation of the valve is by far the more common in childhood, the "button-hole" form is sometimes observed; it has been noted in the case of a boy aged seven (Hayden). The auriculo-ventricular orifice as seen from the auricular side then presents the form of a slit or chink, or a crescentic opening in the firm, thick, fibrous septum of the welded valve-structures. The division of cases of mitral stenosis into the "funnel" and "button-hole" forms, first made by Sir R. Douglas Powell, is a very practical one from the point of view of morbid anatomy. In some cases, however, the auriculo-ventricular aperture on its auricular aspect presents a very irregular form. It may be surrounded by thickenings and nodosities, and the opening may have a puckered appearance resembling, as a French observer has aptly said, the normal anus.

(b) In adults and in persons of advanced years the "button-hole" form of mitral stenosis is observed with much greater frequency. In childhood the proportion is about one "button-hole" to eight "funnels"; in adult age and later life twenty-five "button-holes" to one "funnel." The associations with the rheumatic form of endocarditis are abundantly manifested in the necropsies of cases showing constriction of the mitral orifice in adults. I have seldom, I think I may say never, observed cases of chronic endocarditis or repeated endocarditis affecting the mitral valve—whether
the signs during life have indicated combined stenosis and regurgitation, or regurgitation only—without the necropsy demonstrating that the left auriculo-ventricular orifice was more or less constricted, and the surrounding fibrous ring firmer than the normal.

In many instances in adult age and later life the fibrous material is infiltrated with calcareous salts, the resulting plates having the hardness and general characters of bone. In rare cases the curtains of the valve have been found normal, whilst calcareous plates have been observed in the adjoining muscular wall of the ventricle. These may be associated with atheromatous changes, or may represent syphilitic gummata which have become calcified. In a case of chronic interstitial nephritis the vegetations surrounding a stenosed mitral orifice have been found to contain urates (Lanceaeeaux). Dr. Goodhart, on an analysis of the post-mortem records of 192 cases, showing the changes of chronic interstitial nephritis, found that about one-fourth of the whole number presented either thickening or contraction of the mitral valve. Dr. Newton Pitt observed, on examination of the records of the post-mortem department of Guy’s Hospital, that the cases of mitral stenosis in the subjects of granular kidney were to those not manifesting renal lesions in the proportion of three to one. In many cases in this category atheroma of the aorta was also found, more rarely atheroma obstructing the coronary arteries. Huchard has designated the cases as “rétrociissement mitral artério-scléreux.” In some instances, as in a case of my own, chronic fibrotic changes have been found in various situations—in the pleure, the lungs, the capsules of the kidneys, the liver, the spleen, and the intracranial membranes. In this last, which was that of a woman aged 52, the mitral valve presented the funnel form of stenosis. This form is exceptional in the subjects of chronic renal disease, but other such instances have been recorded. It is obvious that the funnel form of transformation of the mitral valve, the so-called “pure mitral stenosis” of Duvoizez and other French observers, is found not only in childhood (when it simulates a congenital malformation), but also in advanced life. In some cases it is certainly associated with rheumatism; in others such association is not proved; but it may be found in the subjects of chronic renal disease and of arterio-sclerosis.

The left auricle in cases of this affection is frequently hypertrophied and dilated. In some cases the cavity is greatly enlarged, but the walls are thin. In a child of nine years old I have found hypertrophy so far advanced that the muscle was a quarter of an inch thick (the normal being about 3⁄8 of an inch); in another case, that of an aged woman, it was as thin as an ordinary visiting-card, almost destitute of muscle, and lined with laminated coagula. The appendix of the auricle is usually the portion which manifests hypertrophy in the greatest degree. When opening the pericardium the heart is viewed in position, the hypertrophy of the auricle is in some cases very striking; instead of being flaccid it stands out firm and muscular. On section it does not collapse, and pronounced reticulations mark its internal surface. In other cases, when
dilatation preponderates, the capacity of the auricle is increased, in some cases enormously. The pulmonary veins are also greatly dilated. In my own records of 40 cases of mitral stenosis at all ages observed after death, the left auricle was found dilated in 18, dilated and hypertrophied in 10, and hypertrophied without notable dilatation in 3. Dr. D. W. Samways (21), who examined the register of necropsies at Guy's Hospital for four years, found that in 70 cases of mitral stenosis the left auricle was hypertrophied in 36. In 36 cases of well-marked stenosis—the mitral orifice admitting only one finger or the extremity of a finger—the left article was hypertrophied in 26, dilatation coexisting in 14. In 3 cases only was there dilatation without hypertrophy. In the cases of less pronounced stenosis the state of the auricle was precisely noted in 11 only, and of these 5 showed dilatation without hypertrophy. The conclusion is probably correct that hypertrophy is the rule; with the hypertrophy some dilatation nearly always coexisting. When compensation fails, the muscle becomes enfeebled, and dilatation progressively increases.

The endocardium lining the auricle is usually thickened; in some cases all over—the probable cause then being the excess of blood-pressure to which it is subjected, and in many cases in patches by chronic endocarditis or atheromatous change. The posterior wall of the auricle is most frequently thus affected. On the internal surface of this part of the auricle coagula are frequently observed. These are sometimes stratified and composed of alternating layers of coloured and colourless fibrin closely adherent to the endocardial surface. In some cases the whole auricle, thus distended with layer upon layer of coagula, resembles an aneurysm (Potain and Rendu).

The vegetations observed on the lining membrane of the auricle may be sessile or pedunculated—warty, globular, or polypoid (Coats). The warty vegetations are simply coagula of fibrin on the diseased surfaces of the endocardium. Globular thrombi are found especially in the auricular appendage, and between the muscular bundles; in rare cases they almost fill the auricle. Their external portion is smooth and tough; on section they are found to contain a creamy fluid. Polypoid thrombi are more rare; they are attached by a pedicle to the wall of the auricle or to the auriculo-ventricular ring. Some, like the globular thrombi, are masses of firm fibrin; others are hard and calcified. Thrombi at the left auriculo-ventricular aperture are found with greater frequency in mitral stenosis than in mitral regurgitation. They may be detached and become emboli, which are arrested at some point in the arterial channels; or one or more may persistently block the aperture; or, again, one may obstruct the orifice, in the manner of a ball-valve, during certain periods of the cardiac cycle.

The pulmonary veins are in some cases much dilated; their coats may be thickened and atheromatous. Dr. James Barlow of Liverpool has described well-marked atheroma of the pulmonary veins in cases of mitral stenosis.

The left ventricle in the majority of cases presents characters which do not obviously differ from the normal; its cavity is not enlarged; in some
instances its capacity is less than the normal. In the cases of young children the smallness of the left ventricle is striking; in some of these patients the whole heart is correspondingly diminished in size, the lungs are small, and the thoracic capacity reduced. On account of the imperfect blood-supply to the ventricle the whole organism has been impoverished (Wilks), and the entire economy has suffered from arterial starvation. In other cases the contrast with the large and muscular left auricle is very obvious. In about three-fourths of those which I observed after death the wall of the left ventricle was not hypertrophied. When hypertrophy is manifest, as in the remaining fourth of the cases, there is usually an obvious concurring cause—in the young pericardial adhesions, in the old chronic renal disease or arterio-sclerosis. Globular thrombi are sometimes found in the interstices between the musculi papillares of the left ventricle remote from the valve.

With the exception above noted, when death has occurred in the period of childhood, the right cavities are dilated in marked degree, and the walls of the right ventricle and right auricle are hypertrophied. The hypertrophy is often evidenced by the massive muscular columns in the ventricle and the thick interlaced muscular bands in the auricle. The orifice guarded by the tricuspid valve is usually abnormally wide; the valve in some cases is competent to close this orifice, in others its incom- petence is obvious; indeed, cases have been recorded of such dilatation that auricle and ventricle appeared to form one enormous cavity.

Thrombi are observed in the right auricle and right ventricle in many cases; the surfaces of endocardium, on which they are formed, are not necessarily diseased. Such thrombi, when they become detached, plug the larger or smaller branches of the pulmonary artery. Their inception is no doubt due to the retardation of the blood-flow. The chain of consequences is as follows:—Obstruction at mitral orifice, abnormal tension of the walls of the left auricle, auricular hypertrophy and dilatation, obstruction to blood-flow from pulmonary artery to pulmonary veins, increased labour of right ventricle, tension of its walls, hypertrophy and dilatation of right cavities.

In some cases of mitral stenosis vegetations are observed on the tricuspid valve, and these are evidently the results of endocarditis. An induration of the structures at the right auriculo-ventricular aperture may take place, and lead to a series of morbid changes producing a stenosis of the tricuspid aperture closely resembling that of the mitral. Tricuspid stenosis is nearly invariably associated with mitral stenosis, and the morbid changes producing it are more recent in the right heart than in the left. When mitral and tricuspid stenoses coexist, the tendency to the formation of thrombi and emboli in the right cavities is more pronounced than when mitral stenosis exists alone.

In some cases the right auricle have been found greatly dilated; the inferior in greater degree than the superior vein cave.

The lungs generally present the appearances—congestions, consolidations, brown and pigmentary degenerations and scleroses—already de-
scribed in mitral insufficiency. In cases of stenosis, however, hemorrhagic extravasations in the lungs, and infarctions of the pulmonary artery, are observed to a greater extent and with greater frequency. Not seldom there are signs of pulmonary infarction, old and recent. From an analysis of the post-mortem appearances in 36 cases of mitral stenosis, I find that infarctions of branches of the pulmonary artery, or so-called pulmonary apoplexies, were observed in 22 instances. In rare cases a coagulum, evidently detached from the auricle, has plugged the pulmonary artery itself. Cases of mitral stenosis have been recorded in which an extremely dilated left auricle has compressed the left bronchus to such extent as to reduce its calibre to a mere chink (Friedreich). In no inconsiderable number of cases of mitral stenosis the lesions of tuberculosis have been found in the lungs. In the case of a woman aged 29, observed by myself, the necropsy showed a very narrow mitral aperture, with much thickening of the adjacent structures; both lungs were studded with tubercles, some miliary and others yellow and softening. I observed a well-marked case also under the care of my late colleague Dr. Sutfoë. These are the only cases of tuberculosis in association with mitral stenosis which have come under my own eye, but according to Potain the coexistence is frequent. In 35 autopsies, in which mitral stenosis was demonstrated, tuberculous changes were found in 12 instances. Taking the cases recorded by Teissier, Kidd, and other observers, I find a total of 31 in which the association of mitral stenosis with tubercle was proved after death: of these cases 11 presented also the signs of tricuspid stenosis or of endocarditis affecting the tricuspid valve, and 5 others manifested disease of the aortic valves. Uncomplicated mitral stenosis, therefore, was present in 16 cases only. Potain has stated his opinion that the occurrence of mitral stenosis in the course of pulmonary tuberculosis is so frequent that there seems to be a causal relationship between the two diseases. Teissier has gone much farther than this; he considers that some form of tuberculosis is the cause, direct or hereditary, of the “pure” form of mitral stenosis. Nevertheless, his own observations agree with those of Letulle, that the search for bacilli and for any lesion demonstrably tuberculous in the diseased structures surrounding the mitral orifice has always been fruitless. To ascribe the origin of the fibrous thickening to an attenuated tuberculosis seems to me an extraordinary example of special pleading. A more tenable hypothesis, in my opinion, is that in some cases the anæmia resulting from the delivery of an insufficient volume of blood from the imperfectly supplied ventricle, especially in the case of coexisting aortic disease, disposes to the tuberculous invasion; and in others the failure of the right ventricle, or the obstruction to the supply to the pulmonary artery in the cases of concurrence of tricuspid stenosis, disposes to tuberculosis of the lungs; for it is to be remembered that in stenosis of the pulmonary artery, where there is a like physical impediment to the blood-current to the lungs, pulmonary tuberculosis is almost invariably the mode of death.

The stomach, liver, spleen, and other abdominal viscera in mitral
stenosis show, for the most part, the appearances already described in mitral insufficiency. Embolisms and their consequences are much more frequent in mitral stenosis. Taking post-mortem evidence alone, I find that embolism is most frequently observed in the arteries of the brain and the kidneys, and these in equal proportions. Next in order of frequency are pluggings of the splenic arteries. In a small minority of cases the arteries of the pancreas, stomach, and intestines have been blocked by emboli.

In the cases in which emboli have obstructed the intra-cranial arteries the infarctions have been found almost invariably in the vessels of the left hemisphere. In seven out of eight cases the left middle cerebral artery was the vessel occluded; in two cases the anterior cerebral artery also was plugged. The resulting softening was found chiefly in the frontal and parietal convolutions and in the corpus striatum. In a case recorded by Hallopeau, in which the left vertebral artery was blocked by an embolus, softening of the left eminentia teres was observed. According to the evidence which I have obtained, fatal cerebral embolisms, which are the result of the chronic conditions of mitral stenosis, are invariably left-sided. In cases in which acute endocarditis, especially infective endocarditis, has supervened, the limitation to the arteries of the left hemisphere is not so decided. When there is necrosis of the tissues adjacent to the valve there are often multiple emboli. The clinical evidence in cases of mitral stenosis sometimes indicates a lesion of the right hemisphere, but the emboli which are fatal—probably slowly formed and comparatively large—are those which plug the arteries of the left hemisphere. There can be no doubt that the well-known physical explanation of their occurrence in the arteries of the left hemisphere is correct. The left carotid has its axial current in the same direction as that from the ascending aorta; the stream, therefore, carries the disintegrated coagulum most readily through the aorta into the left common carotid, the internal carotid and the middle cerebral, the current continuing in these vessels without deviation. If the embolism be large, it is sufficient to block not only the trunk of the middle cerebral artery, but also that of the anterior cerebral at its bifurcation with the former. If small, the embolism may be only in one of the branches of the middle cerebral. The right hemisphere is practically immune, because the right carotid, arising from the innominate, is placed at such an angle with the aorta as to lie off the axial current.

The working of the heart in mitral stenosis.—In the slighter forms of obstruction the mechanism is precisely that obtaining in the sclerous form of mitral insufficiency. The orifice may be so narrowed as to admit only two fingers or even the thumb only; but the thickened curtains of the valve are retracted, and the physical signs, symptoms, and consequences are those of mitral regurgitation.

The conditions are characteristically different when the mitral orifice is so narrowed or obstructed that the outflow from auricle to ventricle is seriously impeded; and when, as may be inferred with great probability,
there is no regurgitation at the time of the systole of the left ventricle. The most pronounced effect in such case is upon the left auricle. The muscular wall may be greatly hypertrophied, while the diameter of the chamber remains not notably greater than the normal. Or, again, the auricle may be greatly enlarged, so that in some cases its capacity is more than double the normal; its muscle in some cases is hypertrophied, in others atrophied, even so far as to be represented only by a few muscular fibrille scattered through a shell of fibrous tissue. Observers have differed as to the relative preponderance of hypertrophy and dilatation in the auricle. Potain and Rendu consider that, suffering as it immediately does a "contrecoup" on account of the obstructive lesion, the left auricle dilates and hypertrophies simultaneously, and these

![Diagram](image)

changes are never wanting in mitral stenosis. It is obvious that the muscular auricle is strong enough to inject its blood-content forcibly into the ventricle even though the mitral orifice be considerably stenosed. I have myself found, in the case of a child, the muscular wall of the auricle as thick as that of the right ventricle. Cases have been recorded in which the left auricle has maintained life for a long time when the left ventricle, converted into a completely calcified chamber, had been incapable of any active contraction (Burns, Gérard).

It has been generally considered that the auricle ceases its active contraction before the systole of the ventricle begins. This was the doctrine deduced from the graphic records obtained by the experimental methods of Chauveau and Marey in the horse. Subsequent investigation, however, has demonstrated that the auricular systole may continue after the commencement of the contraction of the ventricular muscle, both auricle
and ventricle continuing to contract simultaneously until the moment when the sigmoid valves are opened and blood begins to be expelled from the ventricle into the aorta. Potain considers that the auricle is in action from the beginning of its systole until the precise moment of closure of the auriculo-ventricular valves—that it is this muscular contraction of the auricle which ordinarily causes the propulsion of the heart's apex against the wall of the chest, and that thus it plays a notable part in the production of the impulse which is felt by the hand applied over the situation of the apex beat (Fig. 66). In stenosis of the mitral aperture this lifting of the apex by the force of the contracting auricle may be greatly exaggerated. In investigating cases of mitral stenosis by the cardiograph, I have repeatedly observed that in some the eminence which alone can be ascribed to the auricular systole has contributed to the general elevation due to the systole of the ventricle; one of the most remarkable is here figured (Fig. 67). It is to be remembered that the pen of the cardiograph is guided by the apex of the left ventricle; the record of the auricular systole is written by an impulse communicated from the auricle to the ventricle. It is obvious not only that the auricle contracts in a manner

![Cardiogram in a case of mitral stenosis. Auricular systole (a) greatly exaggerated and contributing powerfully to the elevation completed by the systole of the ventricle.](image)

much more powerful and much more prolonged than under normal conditions, but also that it contributes in very marked degree to the general elevation which is completed by the systole of the ventricle. Professor Potain has entirely corroborated my observations and conclusions. Dr. D. W. Sámways (19) has advanced the ingenious hypothesis that the abnormally powerful contraction of the left auricle prevents regurgitation in compensated mitral stenosis. He shows from mechanical and experimental data that the force of the auricle, seeing that its active contraction is continued until the aortic valves are opened and a free outflow is permitted into the aorta, is adequate to prevent any reflux during the ventricular systole. It seems to me very probable that this view is correct. It affords a good explanation of the post-mortem appearances when a contracted mitral orifice, evidently of slow pathogenesis, is accompanied by a very small left ventricle. If mitral regurgitation had occurred in such a case the ventricular cavity would in all probability have become dilated. Yet in the early stages of the transformation of the mitral orifice it would seem that such regurgitation would have been inevitable unless prevented by some cause apart from the sclerosis of the structures at the periphery of the valve. A compensatory hypertrophy of the muscular wall of the auricle—whence an abnormally prolonged
and powerful auricular systole—occurring early in the morbid process
would explain not only the absence of the characteristic signs of mitral
inadequacy during life, but the absence of hypertrophy and dilatation of
the left ventricle observed after death.

It is obvious that the enhanced force of the auricle, evidenced by the
muscular hypertrophy, is an important, if not the chief factor in main-
taining compensation during the survival—many months or many years
it may be—of the subjects of mitral obstruction. It is equally certain
that it is not the only factor, for hypertrophy of the right ventricle may
be looked upon as a constant sequel of mitral obstruction. Dilatation in
most cases accompanies the hypertrophy, but for long periods the tricuspid
valve is competent to close the right auriculo-ventricular orifice.
Abnormal pressure is thus maintained in the pulmonary blood-circuit.
The hypertrophied right ventricle co-operates with the hypertrophied
left auricle in augmenting the force by which the blood is urged through
the narrowed mitral orifice. In the later stages of the affection, however,
the right ventricle may become dilated on account of the exaggerated
blood-pressure to such degree that the tricuspid is no longer competent,
and there is reflux into the great veins. Compensation is then no longer
maintained. The failure of compensation, however, in a given case may
be not by failure of the right ventricle, but on account of enfeeblement
of the left auricle. We have seen that the auricular cavity may be
enormous, but with practically no effective muscle in the wall. The
evidence, especially the deposition of layer upon layer of fibrin, shows
that failure has been slow and life has been prolonged without any active
participation of the auricle in the work of the circulation. The kinetic
energy of the right ventricle must have operated with the elastic recoil
of the distended auricle after its injection by the right ventricle, and the
suction power of the left ventricle during diastole.

In a case of compensated mitral stenosis we may thus summarise the
work of the heart—Systole of the ventricles. Left unimpeded, quantity
delivered minus or else sufficient for the needs of the organism. Right
abnormally forcible, thus distending the pulmonary veins and the left auricle.
Left auricle over-distended after right ventricular systole; this distension
in greater or less degree relieved immediately on diastolic relaxation and
suction action of ventricle; its own elastic recoil probably aiding the
inflow into the ventricle in the earliest stages of diastole. Probably
muscular contraction of the pulmonary veins a concurring cause; pos-
sibly such contraction in the manner of a sphincter preventing reflux
from the auricle into the pulmonary veins; the proper auricular systole
following and, being abnormally forcible and protracted, contributing to
produce the apex impulse.

Diagnosis.—The diagnosis is in many cases easy, in some attended
with considerable difficulty; at any rate all the ordinary means of physical
investigation should be put in force.

Inspection may reveal no signs. The apex beat may be invisible or
observed in the normal situation—if displaced to the left, causes external
to the heart being excluded, the explanation may be enlargement of the right cavities or a general increase of bulk of the heart due, in the early periods of life, chiefly to adherent pericardium; in the later periods to the hypertrophy and dilatation of the left ventricle accompanying arteriosclerosis. In some cases the precordial region over the right ventricle is rendered prominent and visible; pulsation is seen below the ensiform cartilage. In any case where there is this prominence over the right ventricle, whilst the left ventricle is not observed to pulsate to the left of the normal position, mitral stenosis is prima facie more probable than mitral insufficiency. Inspection of the veins of the neck may show a pulsation in the venous sinus just above the right clavicle coincident with the systole of the right auricle; or, when the tricuspid is incompetent, a definite pulsation of the jugular veins coexistent with ventricular systole.

Pulsation may be observed in the second interspace, or second and third left intercostal spaces near the sternum, and if a vibrating flag or lever be affixed over the spot of pulsation and another over the visible apex beat of the heart, it may be seen that the movement of the former (auricular) is distinctly in advance of that of the latter (ventricular). Thus there may be evidence of abnormal force of the left auricle. It must be remembered that this is capable of demonstration only in rare cases; it has not been observed in adults but in children only.

Palpation may reveal some very important evidence or may be negative. In a case of marked mitral stenosis of long standing a heaving impulse may be found over the position of the right ventricle, under the false ribs to the left of the ensiform cartilage, whilst there may be no palpation-signs of a forcible ventricular systole abnormally to the left. Palpation may thus confirm inspection in indicating that the right side of the heart is enlarged and the right ventricle hypertrophied, whilst the left ventricle does not show these abnormalities. Any such deduction, however, must be made cautiously, for the left heart may be more enlarged than the signs indicate, as it may be covered by inflated lung-tissue. There is one sign obtained by palpation to be observed in a considerable number of cases of mitral stenosis which, provided it has certain essential characters, may be regarded as almost a crucial sign of the affection. This is thrill—‘frémissement cataire.’ The feeling of vibration communicated to the finger lightly laid in the intercostal space close to the point of the apex beat or slightly to the right thereof may be fine or coarse, protracted throughout the whole diastole and ceasing (usually) at the instant of the shock of the apex beat, but sometimes very shortly after the commencement of this event, or occupying a very brief period just before the systole of the ventricle. It can best be timed by the finger of the observer’s free hand placed over the carotid artery, when the thrill is found to cease at the moment of the carotid pulse. If in the case investigated there be well-marked signs of incompetency of the aortic valves, it is to be borne in mind that the diastolic-presystolic thrill may be present without mitral stenosis. Such cases are, however, rare; in a larger number mitral stenosis coexists with the disease of the aortic valves.
In the absence of signs of aortic-valve disease a well-marked diastolic-presystolic or presystolic thrill when observed in the apex region is nearly always indicative of mitral stenosis. It is important that thrill be investigated in varying positions of the patient. Vibrations which are scarcely felt when the patient is in the recumbent position may become much more marked in the sitting posture with the body bent forwards. The observation, however, does not excuse the omission of all other ordinary means of investigation. It is to be remembered that thrill may be absent at some periods and present at others during the observation of a case. Sometimes it is absent when the patient is at rest, and developed after exertion or when the arms are elevated. Percussion is chiefly of importance to determine the outline of the heart; it gives more precision to the evidence obtained by inspection and palpation, and when disproportionate enlargement of the right chambers of the heart is thus indicated, this method of investigation is valuable for diagnosis not only of the nature of the affection, but of its extent and significance. The signs obtained by auscultation are of chief importance in the diagnosis of mitral stenosis—they are murmurs, double shock-sound during the period of ventricular diastole (reduplication of the second sound), accentuation of the pulmonic second sound, loud and sudden snap at the acme of ventricular systole, and inaudibility of the second sound at the heart’s apex. The murmur characteristic of mitral stenosis is that known as the presystolic murmur. It is generally of rough quality, vibratory or bubbling. Potain states that it rarely has the characters of a blowing sound (souffle); most frequently it is snoring or rolling. It may begin almost immediately after the second sound of the heart, be prolonged through the whole period of ventricular diastole, become reinforced towards the end of this period in a “crescendo” manner, and end with a sudden tap or snap. This terminal tension sound is in some cases coincident with the impulse of the apex as felt by the finger; in others it is noted to occur very shortly after the first shock of the impulse; but it is always synchronous with the pulse felt in the carotid artery. The sound of the murmur may begin long before the proper systole of the auricles (see Fig. 69)—it may therefore be correctly designated diastolic-presystolic. The evidence leaves no room for doubt that the reinforcement towards the close is coincident with and due to the muscular contraction of the auricle. Sir Wm. Gairdner uses the term “auricular systolic” (A. S. murmur) to denote the bruit. Whether the term “presystolic” or “auriculo-systolic” be used, it must be remembered that the active muscular contraction of the auricle is not the only force on which the murmur depends. In some cases the bruit is not prolonged throughout the periods of diastole and presystole (the entire diastolic murmur of Bristowe), but is audible as a short murmur closely following the second sound (early diastolic murmur), or isolated with a pause before or after (mid-diastolic or meso-diastolic murmur). Usually these disjointed murmurs are found in a case which at some periods of observation manifests the more typical presystolic murmur.

The sudden snap which generally terminates the murmur is peculiar
and characteristic. In some cases it is observed without any bruit leading up to it. It is evidently an unusually short and sudden first sound of the heart; if in any case it be observed in the near neighbourhood of the apex—in some cases I have noted it at the back under the angle of the left scapula—mitral stenosis should be suspected and the concurrent signs searched for. The cause of this phenomenon is not definitely settled. It closely resembles the sound of sudden tension which may be imitated by abruptly stretching a piece of moist membrane. In the left ventricle of some hearts with stenosed mitral aperture observed after death, in which the phenomenon had been manifested during life, it would seem that there are no structures likely to give rise to this sudden sound of tension at the moment of contraction of the ventricle—the mitral curtains being thick and leathery, the chordae shortened, and, with the papillary muscles, forming thick fibroid bands; the muscle of the ventricle not obviously differing from the normal, and the ventricular cavity small rather than large. On the other hand, the tricuspid valve is seen to be thin and membranous, and it seems probable that to its sudden tension by a forcible right ventricle the loud snap may be ascribed.

Another very important auscultatory sign for diagnosis is the double sound heard during the period of the diastole of the ventricles. This phenomenon, which vividly recalls the "postman's knock," has been generally named the reduplicated second sound. To avoid speculation as to its mode of production we may be permitted to call it a double-shock sound in diastole. It may be manifested in the neighbourhood of the apex or at the base of the heart. When audible in the neighbourhood of the apex of the heart and not over the base the double-shock sound indicates an early stage of mitral stenosis. This view which I enunciated in 1880 was confirmed by Chedele. As a sign of mitral stenosis in later as well as earlier stages, it has been noted by many observers (Potain, Rouche's, Gerard, Phear, Boyd). The explanation of the mechanism of this sound first recorded by myself has been for the most part accepted. It is not a true doubling of the second sound, and cannot be ascribed to the asynchronous closure of the semilunar valves of the aorta and the pulmonary artery, but is of mitral origin. It is a sound of tension due to the first inrush of blood into the ventricle, such inrush being more sudden and forcible than under normal conditions from the increased blood-pressure in the left auricle due to the constriction of the mitral orifice. Potain, Rouche's, and other French observers have described this sound as the "claquement d'ouverture de la mitrale." Potain thus explains the mechanism of the sound. The opening of the mitral valve is normally noiseless; but in the subject of mitral stenosis the valve curtains at the moment when they separate, moved by the blood-wave that enters the ventricle, are abruptly checked by the adhesions of their free borders; the sudden tension which results produces the sound, which is the more dull as the normally thin curtains have become more dense and have lost their elasticity.

When the double-shock sound is audible over the base of the heart
and not in the close neighbourhood of the apex the problem of its cause by no means admits of a ready answer. It is undoubtedly over the base of the heart that the double sound, when manifested in mitral stenosis, is heard in the majority of cases. The diagnostic value of the sound is very great; the double sound either at base or apex is found in more than one-third of all cases of mitral stenosis. The generally accepted view of its mode of production is that the semilunar valves of the aorta and the pulmonary artery respectively do not close in normal synchronism, but those of one vessel coapt in advance of those in the other according to the relative degrees of blood-pressure. The objection to this hypothesis I take to be that it involves an admission of an asynchronous action of the two ventricles which physiologists are not able to accept. The sound of tension of the aortic valves cannot be produced until the left ventricle begins its diastolic expansion; if this sound be followed by that of the tension of the sigmoid valves of the pulmonary artery, it follows that the diastolic expansion of the right ventricle is not synchronous with that of the left, but is in all cardiac revolutions delayed. Pottain has, however, more recently minimised or overcome this difficulty by advancing the following hypothesis: Premising that the precession of the two sounds of tension is aortic in the earlier, and pulmonic in the later phases of the disease, he considers it probable that when the obstruction at the mitral orifice is slight, but yet sufficient to bring about some difficulty in the entry of blood into the left ventricle, the aspirating power of the latter in diastole is augmented ("elle est moins aisément satisfaite"), and the semilunar valves, drawn upon with more force than ordinarily, close more rapidly. Later, when the obstacle at the left auriculo-ventricular orifice has notably impeded the circulation in the lung, and the right ventricle has become hypertrophied, the over-pressure in the pulmonary artery compels the semilunar valves of this vessel to close more forcibly and more rapidly at the beginning of ventricular diastole. For my own part, though there is room for much difference of opinion, I think it more probable that the phenomenon has a similar cause at base and apex of the heart. The first element of the double shock-sound is the normal second sound often accentuated as to its pulmonary artery component; the second element is a sound of tension produced by the forcible entry of blood into the ventricle, the shock being communicated either to the wall of the ventricle or to the anterior curtain of the mitral valve close to the aortic cusps, and thence to the sternum and especially its left border.

Accentuation of the pulmonic second sound is a sign to be noted in mitral stenosis as in mitral regurgitation. The cause—over-pressure in the pulmonary artery—occurs in both morbid states, though from differing causes. In mitral stenosis, however, the irregular rhythm of the heart in many of the cases prevents a due appreciation of this accentuation; the sound then is very loud in some cardiac cycles, in others feeble or almost inaudible.

Another auscultatory sign to be noted in a section of the cases is
inaudibility of the second sound of the heart at the apex. This extinction of
the second sound at the apex is usually manifested in the later stages of
mitral stenosis (Broadbent, Acland); its causes are—(a) a diminution
of blood-supply to the aorta, and consequent feeble recoil against closed
aortic valves (it is the aortic element of the second sound that is audible
over the heart's apex); (b) the enlargement of the right auricle and ventricle
which, coming more and more to the front, displace the left ventricle, the
chief conductor of the sound.

In the latest stage of mitral stenosis the presystolic murmur may
be inaudible, the second sound absent, and the short and sudden first
sound, to which attention has been already called, the only notable
auscultatory sign. More frequently, however, in later as well as
in earlier stages, a systolic murmur is to be heard in the neighbour-
hood of the apex. This murmur may have the ordinary characters
of that of mitral insufficiency, audible over the apex and at the back
under the angle of the left scapula, or may be a short systolic "puff"
having a very limited area of audibility, but over the site of the apex. It
may coexist with the presystolic murmur, which in such case is usually
heard for the most part slightly to the right of it; or it may be heard
when no presystolic or diastolic-presystolic bruit is audible. Nearly
always in these cases the sudden tap indicating the first sound is heard
over some part of the apex region. In another section of the cases the
systolic murmur has an area of audibility to the right of the apex,
encroaching more and more on the tricuspid region, and in some instances
localised at the base of the ensiform cartilage, that is, the area of a tricuspid
regurgitant murmur. It has been considered (and the contention has a
great show of validity) that in some of the cases in which a systolic
murmur has been ascribed to regurgitation through the mitral orifice
the real cause of the phenomena has been tricuspid reflux (Sapp, loc. cit. pp. 64 et seq.). In some instances, however, there are
two areas of audibility of the systolic murmurs, when it is most
probable that there is regurgitation through both mitral and tricuspid
orifices. If the hypothesis be correct, that the abnormally powerful
muscular contraction of the left auricle prevents regurgitation in the
compensated stages of mitral stenosis, it is probable that some such
regurgitation is inevitable when compensation fails and the auricular
muscle has become feeble.

It is to be noted that a very marked irregularity of the heart's rhythm
is by no means infrequent in mitral stenosis, and that this irregularity
may modify all the physical signs already described. The murmurs, the
so-called reduplications of normal sounds, the snap sound, and the thrill
may be observed in some cardiac cycles, and may be absent in others.
The irregularity of rhythm is evident to the auscultator. Such irregu-
larity may be entirely due to disturbances of the nervous mechanism,
and may be quite independent of structural changes in the heart; but
when signs of organic valve disease coexist with it, mitral stenosis is the
lesion in the great majority of cases.
Cardiographic evidence.—The use of the cardiograph has in many instances afforded valuable evidence not only for the diagnosis of the condition of mitral stenosis, but for the elucidation of some of the difficult problems connected therewith. The chief signs recorded by the cardiograph have been—

(a) An abnormal magnitude of the elevation denoting the auricular systole. It has been shown in some cases that the power of the auricle is sufficient to lift the ventricle in a pronounced manner (Fig. 66). This can only occur when the narrowing of the mitral orifice is not considerable, the auricle being hypertrophied.

(b) An increase in breadth of the auricular eminence, the summit of which is seen to be broken by undulations, a condition felt by the finger as thrill. (c) Repeated elevations denoting rise and fall of pressure during ventricular diastole, not necessarily indicating any muscular contractions of the auricle, but probably expressing graphically the interruptions of the flow of blood through the diseased valve-structures which are audible as a rolling or bubbling murmur. (d) Fine serrations in the diastolic and
presystolic periods audible as harsh murmur, and due to the causes already considered. This form of cardiogram denotes a considerable degree of stenosis contrasting with (c), in which the orifice is only moderately constricted. It has already been noted that in some cases (e) the cardiogram in mitral stenosis differs in no appreciable way from the normal. It would be legitimate in such case to infer that the stenosis is slight in degree.

Sphygmographic evidence.—In a large number of cases of mitral stenosis the sphygmograph indicates a very notable irregularity; this irregularity may be observed when the lesion is compensated and the patient appears to be in perfect health. In some instances in which the

![Diagram of sphygmograms]

**Fig. 71.**—Sphygmograms in mitral stenosis. A, In stage of compensation; man aged 44, observed during five years. B, Case manifesting typical presystolic murmur; no signs of failing compensation; patient in good health. C, Mitral stenosis with failure of compensation (tricuspid regurgitation, pulsating liver). D, Late stage of extreme mitral stenosis (female aged 17). E, Regular anacrotic pulse; mitral stenosis in a female aged 41, with rheumatic antecedents.

rhythm of the heart is apparently regular, a slight exertion serves to provoke the irregularity. The administration of digitalis may produce or increase it, but it is often found in cases in which the drug has not been administered. The most frequently observed form of irregularity is that evidenced in the sphygmogram by a repeated elevation in the downstroke. There may be two or three of such elevations before the baseline is reached. It is evident that these excursions from the downstroke contain all the elements of a complete pulsation effected by the ventricle. They show that after a comparatively effective emptying of the ventricle there may be repeated systoles following at very brief intervals. In late
stages of the disease, the irregularity may be extreme. The irregular pulse of mitral stenosis has been noted by many observers (Ralfour (2), Makomed, Foster, and others). I consider that the sphygmographic indication of irregularity in a case in which compensation appears to be perfect may aid in the differentiation between mitral stenosis and mitral insufficiency, for the latter lesion during the stages of compensation is not attended by irregularity of the pulse unless there be some coexisting neurosis.

In another large series of cases the sphygmograms show a perfect regularity in the heart's rhythm. Many observers have considered such regularity to be the rule in mitral stenosis (Hayden, Fagge, Broadbent). The up-stroke of the tracing indicating the volume of the artery is inconsiderable, and the indications are that the vessel is full between the beats. Sir W. Broadbent (5) considers that this modified high tension pulse is almost constant in mitral stenosis, and indicates resistance in the capillaries. Such resistance may be due to contraction of the arterioles consequent upon the overloading of the blood with impurities arising from defective elimination or, possibly, from the backward pressure in the veins effected through the capillary network, or from the contraction of the entire arterial system upon a diminished supply of blood from the imperfectly filled left ventricle.

Practically the observation of a heaving impulse of the right ventricle without signs of dilated left ventricle, together with the evidence of a pulse having the characters above stated, may have a valuable bearing in the diagnosis of mitral stenosis. That the arteriole resistance is in some cases increased is proved by the anacrotic form of pulse which is sometimes observed (see Fig. 71, E). The association with arterio-sclerosis, well proved in a section of the cases, is in these a sufficient explanation.

Some difficulties in the diagnosis.—Although the presystolic murmur and the thrill observed in the positions mentioned close to the heart's apex are indications of mitral stenosis, in the great majority of cases they are not absolutely pathognomonic.

Austin Flint was the first observer to show that a murmur having the characters of that of mitral obstruction could be produced in cases of insufficiency of the aortic valves in the absence of mitral stenosis. These observations were confirmed by many observers. I have shown that the presystolic thrill of mitral stenosis also can be exactly simulated under conditions of aortic regurgitation. Dr. Phear (15) has carefully analysed the records of forty-six cases in which there was presystolic apex murmur without mitral stenosis; in twelve of these, thrill, presystolic or diastolic, was present. In seventeen of the cases the aortic valves were incompetent; in twenty the pericardium was adherent; in the remainder there was no valve-lesion, but in some of these there was dilatation of the left ventricle. The hypotheses which have been adduced to explain these phenomena are the following: (i.) That in the cases of aortic regurgitation the regurgitant stream tends to lift the great anterior mitral curtain, and so to obstruct the mitral orifice at the end of diastole as to impede the current from the
auricle; (ii.) That the mitral valve is thrown into vibration by the two currents, the regurgitant from the aorta and the direct from the auricle, such vibrations lasting until the commencement of ventricular systole; (iii.) That in the absence of aortic valve disease, but in the presence of adherent pericardium, vibrations may be set up by the current propelled from a dilated and hypertrophied auricle into a ventricle whose muscular walls are deficient in their normal nerve-tone; (iv.) That shortening of the chordae tendineae, or dilatation of the left ventricle, may bring about a virtual narrowing of the aperture through which the blood passes from auricle to ventricle, the auricular muscle continuing to be sufficiently powerful to generate a fluid vein. It must be admitted that these opinions are for the most part conjectural, but the fact remains that in some cases the physical signs have led most competent and careful observers to an erroneous diagnosis of mitral stenosis. The practical lessons I take to be the following:—In cases where the concurrent signs indicate dilatation of the left ventricle, and where the previous history tells of an antecedent pericarditis, we must be cautious in interpreting a presystolic murmur as pathognomonic of a stenosed mitral orifice. In all cases careful investigation must be made into concurring signs of incompetency of the aortic valves. If the murmur of aortic regurgitation be absent from the base of the heart and the line of the sternum, it may yet be found alone at the apex, and may then closely simulate the murmur of mitral stenosis. In such case, however, according to my experience, the terminal tension sound, the tap or snap, is not marked—the sound is dull. All available means, including the use of the cardiograph and sphygmograph, should be used to effect the differentiation.

It must be remembered that aortic insufficiency and mitral stenosis may coexist, and the diagnosis of the combined lesion may present great difficulty. Dr. F. J. Smith found on examination of the post-mortem records of the London Hospital evidence of the combined lesions in thirty-nine instances. Uncomplicated aortic insufficiency was to aortic insufficiency plus stenosis as 88 to 39. The association of the two valvular affections therefore is not very rare, and the diagnosis of such association can only be made with an approach to certainty when there are decided physical indications of each separate morbid condition.

Clinical groups of cases of mitral stenosis, their symptoms and treatment.—Group I. Cases associated with rheumatism.—The intimate relation between mitral stenosis and rheumatism is shown by a large series of cases. In some of these the rise and progress of the endocarditis, the cause of the obstructive lesion, can be traced by clinical observation. The patient may show all the signs of acute rheumatism, an occurrence comparatively rare in children, the acute symptoms being often very slightly pronounced, though in some instances they are fully manifested, and then usually the first sign of implication of the valves is the systolic murmur of mitral regurgitation. The child in the course of months or years may suffer from repeated attacks of acute rheumatism, and after
a longer interval the systolic murmur is preceded by a presystolic murmur, the other signs of mitral stenosis concurring. In some such cases, and in course of time, the murmur of mitral regurgitation becomes replaced by that of mitral stenosis. Many such instances have come under my observation (27). In other cases the presystolic murmur of mitral stenosis after repeated attacks of rheumatism has been very decidedly modified—it has been followed by a systolic murmur. The significance of such a change it may be difficult in some cases to estimate. The murmur may be very loud, and heard in the left axilla and at the back: if so, there can be no doubt that it is due to regurgitation from organic disease. Or it may be heard over a very restricted area, not conducted to the axilla, but just over the apex itself. In such case the auriculo-ventricular orifice may not be widened by any retraction of curtains or columns, but the anatomical lesion may be stenosis nevertheless, and the auricular muscle have become weak; therefore regurgitation, which previously had been prevented, is now permitted. Or the murmur may be observed to the right of the position of the apex close to the tricuspid area; in such case the probability of tricuspid regurgitation must be borne in mind.

In some cases rheumatic phenomena are declared, not in the early stages of the affection, but subsequently, during the observation of the case. For instance, a girl of fourteen, without any rheumatic antecedent—though there was hereditary tendency thereto on the mother's side—manifested a prolonged systolic and a short presystolic mitral murmur. There were no rheumatic phenomena for thirteen months when polyarticular rheumatism appeared. At that time a marked thrill was felt at the apex; a grating presystolic and a prolonged blowing systolic murmur were heard, and the heart was enlarged, especially as regards the right chambers. The autopsy showed a funnel-shaped transformation of the mitral valve and a ring of small vegetations (recent rheumatic endocarditis) encircling the auriculo-ventricular orifices. This affords one of many pieces of evidence that the rheumatism which is associated with mitral stenosis may be attended for long periods by no obvious symptoms.

A sign of the advent of the structural change in the valve inducing obstruction at the mitral orifice is a double shock sound heard during the period of ventricular diastole, and resembling a doubling of the second sound over the apex of the heart. I noted this simulated doubling of the second sound at the apex in a large number of cases which eventually manifested all the usual signs of the lesion. Dr. Cheadle found "33 cases with presystolic murmur, and 24 with reduplicated second sound at the apex, indicating commencing stenosis out of 273 cases of organic heart disease in children" (8). He adds: "There can be no question as to the connection of this morbid sound with early mitral stenosis, and of its clinical significance." Potain has confirmed these observations, ascribing it, as I do, to causes affecting the mitral valve. The first element is the normal second sound heard at the
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apex, the second element occurring soon after it, the "changement de l'ouverture de la mitrale."

In a large number of cases the clinical signs of association with rheumatism are insignificant. The evidence of many which I have analysed shows that in a considerable proportion the origin and progress of the morbid changes in the valves and the adjacent structures are insidious and gradual. The disease which initiates these is not independent of rheumatism, but is often unaccompanied by pronounced rheumatic phenomena. The endocarditis which results in mitral insufficiency is more violent and more obviously associated with ordinary acute rheumatism; that which induces stenosis is more protracted and symptomless, giving rise to a gradual welding of the curtains and a slow formation of fibrous tissue which, under the even pressure of the blood within the auricle and the ventricle, tends to the production of a smooth septum. This septum becomes gradually thicker, for it has to bear the chief strain of the auricular pressure—not the ventricle, as in the case of mitral insufficiency.

When the acute signs of rheumatic endocarditis have passed away, or when, in the absence of any obviously acute manifestation, the obstructive lesion has been gradually induced, compensation enduring for protracted periods may ensue. Such compensation is a simpler matter than in the case of mitral insufficiency, for an increase of power in the muscle of the right ventricle and of the left auricle only is necessary to maintain it; enhanced force and increased capacity of the left ventricle not being also required as in the structural lesion inducing mitral regurgitation. The left ventricle may deviate but little from the normal, and a strong right ventricle, aided by a hypertrophied, or at least not enfeebled, auricle, will urge a sufficiency of blood through the narrow orifice.

The symptoms of failure of compensation differ in many points from those in cases of mitral insufficiency. In the latter the signs are more uniform—the dyspnoea of effort, or the paroxysmal dyspnoea progressively increasing in intensity, the gradual oncome of dropsy, and other signs which have already been considered are evidenced; in mitral stenosis, on the other hand, the symptoms are more erratic, the accidents of the disease predominate, and it is these rather than the gradual heart failure that have in the greatest degree to be reckoned with.

One of the earliest symptoms to attract attention in cases of mitral stenosis is epistaxis; Duroziez has noted this, and I confirm his observations. Probably we are not told of this symptom in many of our cases in hospital because it is considered trivial. In some, though in a less proportion than I should have imagined, there have been complaints that the patient is soon "out of breath." Percordial pain and distress are noted, however, in a considerable number of patients, and in some of these palpitation. My notes show these symptoms in 21 out of 54 cases. Hæmoptysis was recorded in nine of these cases; it occurred in the course of the lung affections in many more. The most frequent of all the induced morbid states is that evidenced by dyspnoea,
cough, and other symptoms referred to the lungs. In some cases there is a general bronchitis; but in the great majority there are signs of a localised pneumonia, in the course of which the sputa are frequently blood-stained. The bronchitis can be referred to the general venous engorgement of the lungs, but the localised consolidations are proved, by morbid anatomy as well as by clinical evidence, to be due to infarctions of branches of the pulmonary artery. These occur with the highest degree of frequency in mitral stenosis; and in at least half the cases I have observed they have been manifested at some time of the life-history. The haemoptysis and the lung signs often suggest the probability of pulmonary tuberculosis; but in the vast majority of cases this is negatived. I have mentioned, however, the fact of its occurrence in a small minority; so that investigation should be made for tubercle bacilli in the sputum, and the other related signs should be duly weighed. Other symptoms which occur in the course of mitral stenosis, increasing the dangers of the disease and adding new difficulties to its treatment, are those due to embolic infarctions of the systemic arteries. These will be considered in the next group of cases. In only a few cases are they clinically observed in the spleen, though morbid anatomy teaches that this is a very frequent site of embolism. Probably the symptoms thus occasioned pass in many cases unnoticed and unknown. It is otherwise when an intra-cranial artery is thus blocked—then the danger of the condition is proclaimed. It is to be remembered that these embolisms—whether in the pulmonary or in the systemic circulation—very rarely occur in mitral stenosis from detachment of the vegetations of acute endocarditis, but most frequently from plugs passively formed within the chambers of the heart. Frequently, therefore, they are the first manifestations of disease, and not symptoms developed during an acute or subacute illness. They occur both in the cases which are obviously associated with rheumatism, and those which present no such evident relation. Of course they tend further to disturb compensation, though in many cases there is recovery for long periods.

Generally speaking, in the cases of mitral stenosis oedema is not nearly so marked a symptom as in the cases of mitral insufficiency. A fugitive and slight oedema occurs in many of them, but general dropsy rarely until the final stages, when the right chambers of the heart have become dilated and the tricuspid valve incompetent; and many patients die before this stage is reached. Sir W. Broadbent notes that great enlargement of the liver with true pulsation of this organ is more frequently found as a consequence of mitral stenosis than of other valvular affections; and it is not uncommon to find fluid in the peritoneal cavity before oedema of the feet and legs. The oedema will disappear with rest in bed while ascites remains for a time; whereas cardiac dropsy in mitral and tricuspid insufficiency begins, as a rule, in the connective tissue of, the most dependent parts (6). My own observations confirm these conclusions. In the rheumatic group of cases the influence of sex in the disposition to the obstructive mitral lesion is well marked and difficult to explain. Of
264 cases of all forms of mitral stenosis collected by Sir Dyce Duckworth, 177 were female and 86 male. In Hayden’s cases the proportion of females to males was two to one. In Broadbent’s list of 53 cases examined post-mortem, 38 were females and only 15 males. Sir Dyce Duckworth concluded that in 70 per cent of the cases of mitral stenosis tabulated by him there was a certain or strong presumption of rheumatic antecedents; and he considered this estimate of the relation to rheumatism to be rather under than over the mark. In regard to my own cases, in 17 autopsies of children manifesting mitral stenosis in conjunction with pericarditis or endocarditis, which I judged to be of the rheumatic form, 10 were female. Of 35 children under 12 years clinically observed, 22 were female; of 31 adults with mitral stenosis in distinct association with rheumatism observed by myself, 18 were female. It would appear, therefore, that the preponderance of cases in the female sex in my own experience is not so great as in that of other observers. It must be remembered that I have taken those only in which I considered the rheumatic association to be strongly accentuated: the groups of cases not decidedly associated with rheumatism will be considered hereafter.

Prognosis.—I have found the average age at death of 61 patients with mitral stenosis to be 32.7. The late Dr. Hayden’s cases—42 in number—gave an average age of 37.8. Sir W. Broadbent states that the average age at death, deduced from 53 cases abstracted from the post-mortem records at St. Mary’s Hospital, was 33 for males and 37 and 38 for females; and he adds: “Mitral stenosis stands next to aortic regurgitation among valvular affections in the order of gravity.” I have records of 17 cases fatal before the age of 12 years, the average being 9.5 years; 10 of these at the age of 10. The association with rheumatism is shown by the fact that, in addition to the valve-lesion, in 14 of these either pericarditis or recent endocarditis of rheumatic characters were found in necropsy. The rheumatic associations of the majority of cases of mitral stenosis constitute a very great, if not the chief element of danger. The other causes of fatality will be pointed out in the consideration of the other groups. It must be accepted as a general proposition that the subjects of mitral stenosis (discovered at an early age) rarely survive the age of 40; the disease, therefore, when dating from childhood and adolescence, and in such cases having its origin, as I believe, in a rheumatic affection, is of grave significance.

Treatment.—The recognition of the rheumatic association of mitral stenosis is of much importance in treatment. In childhood and adolescence a slight febrile attack in the subject of mitral stenosis, or in one who presents signs of the advent of the lesion, should be held as a probable indication of a subacute rheumatism; and treatment by complete rest with the administration of salicin or the salicylates should be enjoined. If cough and difficulties of breathing are also present, symptoms of bronchitis or pneumonia, the systematic administration of ammonia in addition is valuable. The frequency of infarction of branches of the pulmonary artery in cases of mitral stenosis has already been pointed
The late Sir Benjamin Richardson advocated the frequent administration of liquor ammonis, well diluted, as a means not only of inducing fluidity of the blood, but also of dissolving a coagulum already formed. There may be differences of opinion whether such solution be thus possible; but there are many reasons in favour of the treatment. Besides increasing the alkalinity of the blood-plasma, ammonia is a valuable stimulant of the nervous mechanism of the heart and of the respiratory centre; and, by increasing the bronchial secretion and rendering it more fluid, it acts very favourably as an expectorant. I have witnessed a case in which death seemed impending from plugging of the pulmonary artery when the frequent administration of ammonia seemed to be the means of saving the patient’s life; and I have observed many cases in which there were signs of partial embolism when recoveries were very satisfactory. The best mode of administration in young subjects is the liquor ammonis fortior in doses of one to five minims, with liquid extract of liquorice well diluted with water; the dose being repeated—according to the urgency of the case—every half-hour, every hour, or every two hours until signs of improvement appear. It may then be continued every four hours for several days. Whether there be bronchitis from venous congestion or local consolidations of the lungs from infarcts, the ammonia treatment is valuable. It may be well to issue a caution against the use of digitalis during any febrile manifestation in these cases. I have found it worse than useless. The hemoptysis which may occur should not be treated by styptics or opium. As a general rule it is better that any haemorrhage which breaks out in the course of mitral stenosis should not be checked by drug treatment. A like medicinal treatment to that just mentioned may be put in force in cases in which precordial pain or distress is manifested in the subjects of mitral stenosis. It is to be remembered that pericarditis arises not infrequently in this connection, when the special treatment for this disease must be carried out. The occurrence of pericarditis or of lung complications of any kind may rapidly break the compensation in mitral stenosis; and inadequacy of the right heart, with dropsy and other signs of heart failure, may occur. In such cases the treatment should be as already described under mitral insufficiency. The symptoms, however, are frequently recovered from, and compensation is restored.

Whilst there are any indications of acute changes—of endocarditis, of pericarditis, of rheumatism, or of any symptoms attended by pyrexia—perfect rest in bed should be enjoined. It is otherwise, however, in convalescence, when it is to be presumed that sclerosing changes in the valve structures are going on. Then systematic exercises, gradual and tentative at first, should be recommended, for they fulfil important indications; they not only aid the venous circulation, but by expanding the thorax they tend to aspirate the heart, increase the outflow from auricle to ventricle, and perhaps prevent the imminent danger of the progressively increasing contraction of the auriculo-ventricular aperture. It may be urged that a danger of such exercises may be a detachment of
vegetation left by the rheumatic endocarditis; this is possible, but it is proved that the greater danger is the passive formation of thrombi within the heart in consequence of retarded circulation within it. The patient should be cautioned against violent movements, but there can be no doubt, I think, of the value of systematic exercises. Those have been considered in the treatment of mitral insufficiency. During convalescence from any acute febrile manifestation in the subject of mitral stenosis, the first method employed should be gentle massage and movements of the legs, the patient being quiet in bed; next in order the arms and thorax. Later, expansion movements of the thorax, made by the patient himself cautiously and deliberately, should be practised, with judicious intervals of rest. Concurrently, or, as I prefer, just subsequently to these movements, there should be sponging, first with warm and afterwards with cool water, followed by dry towel friction. Later the systematic muscular exercises, as prescribed by Ling and Schott, may be used. An excellent summary of methods of muscular movement is given by Dr. Lauder Brunton (7).

Although moderate exercise in the fresh air in the subject of fairly compensated mitral stenosis is salutary, sudden overstrains are dangerous. In some cases breathlessness does not come as a warning, and patients persist in overtaxing their strength. The subject of mitral stenosis should be protected from chills by suitable apparel, and no clothing is better than pure woollen. A light woollen night-dress is also to be recommended. Heavy overcoats and sealskins, which weigh down the shoulders and thus prevent good expansion of the thorax, are, in my opinion, to be deprecated.

The late Sir Andrew Clark, in a clinical lecture which was published after his death, gave some valuable hygienic rules for a patient with mitral stenosis. In the daily dietary fluids should be restricted, for after their absorption they distend the vascular system, and increase the bulk without increasing the nutritive value of the blood within the vessels. The ingestion of much liquid enfeebs the heart and increases the labour of the right ventricle and left auricle in the transmission of blood through the narrowed aperture into the left ventricle. The patient should have three good meals a day as dry as he can make them; over-eating and indigestible foods must be strictly guarded against. I consider it a good plan to advise that the two meals of the day of which meats form a portion should be taken without alcohol, and with a little pure water or toast water only; and subsequently to each of these a wineglassful of milk with two teaspoonfuls of good old brandy or whisky may be allowed. In some patients there is a slight appearance of jaundice, the liver is embarrassed; there is often constipation. There may be basic congestion of the lungs. Sir Andrew Clark said, "To relieve the lungs give something to relieve the bowels." Sulphate of soda and phosphate of soda, equal parts in powder, may be administered in doses of two or three teaspoonful dissolved in water in the morning, or a teaspoonful of sulphate of magnesia may be taken in hot water. Such aperients relieve the portal system, and so the right
side of the heart and the lungs. Mercurial purgatives are occasionally of service.

The routine administration of digitalis in cases of mitral stenosis is to be condemned. Very often it does harm. When once a patient manifesting the physical signs of mitral stenosis has recovered from any intercurrent disease which has disturbed the compensation, careful hygienic treatment and the administration of ordinary tonics are all that is necessary; all the special heart tonics should be avoided. When, however, the right heart begins to fail, or dropsy to appear, some special heart treatment becomes necessary. Even then in many cases the administration of digitalis cannot be advised with the same confidence as in cases of mitral regurgitation. In many it causes the heart's action to become irregular, or increases an already existing irregularity; in some it induces nausea and vomiting, in others precordial oppression. I have found convallaria to act more beneficially in these cases than digitalis; it favourably influences the irregularity, and acts as a powerful diuretic. The extractum convallariae fluidum in doses of 5 to 10 minims, or the tinctura convallariae in doses of 10 to 20 minims, may be administered every four hours, or three times a day, in adults. Strophanthus is useful in some cases, but, like digitalis, should not be continued for long periods. When there are serious symptoms of heart failure—the radial pulse small and irregular, whilst the right ventricle is felt to beat forcibly, and the veins of the neck are seen to be distended, and perhaps pulsating, the patient being pale or dusky and breathing badly—relief of the venous engorgement by venesection is a valuable means of treatment. The ordinary method of opening the vein in the arm and permitting the flow of about six ounces of blood is the best, but this is often objected to; if so, six or eight leeches may be applied over the epigastrium. In children the relief given by the abstraction of blood by two or three leeches is very well marked. After abstraction of blood digitalis and other heart tonics often act more favourably than they would have done before the relief of the venous engorgement.

Group II. Cases in which the disease is first declared by symptoms of lesion of the nervous system.—Not uncommonly a patient comes under medical care for a lesion of the nervous system which has suddenly shown itself and then the diagnosis of mitral obstruction is made for the first time. If rheumatic manifestations existed at any period of the previous history of the patient these were trivial and unnoticed. The physical signs indicate a pure mitral stenosis; there is no evidence of mitral regurgitation. In fatal cases, for the most part, the funnel form of mitral constriction is found. In many there is good reason, from the hereditary bent, or from the occurrence of some symptoms which suggest a rheumatic proclivity, to suspect that these insidious morbid changes had their origin in rheumatism; but it may not be so in all cases. It is possible that the hematomata of the delicate mitral flaps in infancy may be the starting-points of the fibrous proliferation; or vascular dilatations or hemorrhages from the fine vessels of the growing valve may be the
earliest changes. At any rate the only cause concerning which we have precise evidence is rheumatism.

The most characteristic among the severe lesions of the nervous system is right hemiplegia. In one of my cases, a girl of 10, the first detected sign was sudden paralysis of the right arm and leg; the child recovered completely from the paralysis, but died seven months afterwards after having manifested much precordial distress. Mitral stenosis was demonstrated at the autopsy, and there was universal adhesion of the pericardium. In another patient, a woman aged 22, who had never manifested any symptom of rheumatism, and who had no hereditary tendency thereto, sudden right hemiplegia occurred with aphasia. There were pronounced physical signs of mitral stenosis without regurgitation. The patient made a perfect recovery from the paralysis of motion, but complete aphasia persisted (28). In another of my cases, also a woman, left hemiplegia occurred; after full recovery from this lesion right hemiplegia came on suddenly; from this latter attack the recovery was but partial. In Duroziez's 43 cases of "pure" mitral stenosis in females, 11 manifested right hemiplegia with aphasia, and 4 hemiplegia without aphasia; there were no such cases in the male sex.

Another nervous disorder which may suddenly arise in subjects of the affection is hemichorea. In 38 cases of mitral stenosis I found 4 of hemichorea. Duroziez records a case of a woman, aged 24, with mitral stenosis declared by right hemichorea in which the convulsive movements of the limbs ceased, but chorea of articulation remained, so that the beginning only of each word was uttered. One of my patients, a boy aged 3½, was suddenly seized with epilepsy, the unconsciousness lasting twenty minutes. Nine months afterwards chorea became manifested; recovery took place, but after a second period of nine months another attack of chorea occurred; there were well-marked physical signs of mitral stenosis. In a boy, aged 5, who manifested presystolic murmur and thrill, a fit had occurred eighteen months previously attended with unconsciousness so profound that the child was thought to be dead; nine months afterwards chorea appeared. In another case, a girl aged 5, epilepsy occurred, and the attacks were repeated and severe. In a lad, aged 18, in whom I had, the opportunity of watching the physical signs of the gradual establishment of mitral stenosis, from the manifestation of a soft apical systolic murmur to that of complete and characteristic presystolic murmur, thrill, and doubled second sound, there occurred during his exercise in the garden a sudden unconsciousness, which was complete for a minute or two, but was not attended by muscular spasm.

It is, I think, reasonable to conclude that these sudden perturbations of the nervous system are caused by infarctions of branches of the intracranial arteries; in some instances this was positively proved by necropsies. It is clear that the consequences of such embolism may in some cases pass away completely; in others the plugging of the vessel is followed by necrosis of the nervous structures thus supplied.
In the treatment of such cases complete rest should be promptly enjoined. There is fair evidence that the ammonia treatment, as described in relation with embolisms of the pulmonary artery and its branches, may fulfil a useful purpose.

Group III. Cases presenting disorders of nutrition.—Children are not infrequently brought for treatment on account of their progressive wasting. The parents, or those who have charge of them, think they are "in a consumption." On removal of the clothing the emaciation is seen to be considerable; the ribs stand out and the intercostal spaces are sunken, except in some cases over the situation of the right ventricle, where there is a marked prominence; on further examination the physical signs of mitral stenosis are in full evidence. In those who have arrived at adolescence or adult life there are other signs of ill development. The patients are indisposed for exertion (though they seldom complain of breathlessness); they are unstable and infirm of purpose, are accounted very nervous, and in some instances are demented; they are frequently dyspeptic. The elucidation of the condition is in fatal cases made by the post-mortem examination; constriction of the mitral orifice, and the enlarged right chambers of the heart contrast with a small left ventricle and small aorta. The normal arterial blood-supply has been gradually diminished by the contraction of the mitral orifice, and has continued to be in minus quantity during the periods of development and growth. As Sir Samuel Wilks has pointed out, "The lungs are small as well as the chest, and the respiratory process is correspondingly lowered, and with this probably the whole body is impoverished. At all events, the organism is working with a diminished amount of blood" (30).

In young women—and in the great majority of such cases, even in childhood, the patients are of the female sex—there is frequently, though not invariably, an association with anaemia and chlorosis. The frequency with which a chlorotic patient has presented physical signs of mitral stenosis has been noted by many observers. Stokes in 1854 was the first to record this in describing the case of a young girl, aged 18, who was anaemic and chlorotic, and showed the physical signs of organic mitral disease, the precise form of the lesion being then undiscovered. Death occurred after the manifestations of anasarca and congestions of the lungs, and at the necropsy the funnel form of mitral stenosis was found, with an auriculo-ventricular aperture that scarcely admitted a goose-quill. This case may be regarded as an exemplary one. I have observed many instances of very marked anaemia, some not presenting signs of wasting, in which there has been well-marked physical evidence of mitral stenosis without regurgitation. Duroziez, who has given the notes of many cases, goes so far as to say that pure mitral stenosis is a feminine and a chlorotic malady (9). Teissier points out that a similar anaemia occurs, though more rarely, in the male subjects of mitral stenosis.

In any of the cases in this group haemoptysis may occur, and local
consolidations may be found in the lungs—the group of symptoms closely resembling those of pulmonary tuberculosis. In the great majority the diagnosis of pulmonary consumption is not justified; the symptoms are the accidents of the mitral disease itself. I have given my reasons for dissenting from the view that mitral stenosis can be considered as standing, even remotely, in any causal relation to tuberculosis; but I think it probable that the deficient arterial supply which is a consequence of the disease disposes to the occurrences of tubercular changes in the lungs in a small minority of cases, and the remote probability of this should be present in the mind of the observer. The presence or absence of tubercle bacilli in the sputa will settle the question.

In the treatment of this group of cases physical training should hold a first place. It is evidently of the highest importance that the blood-flow from the right to the left ventricle should by judicious means be increased. It is possible that if this be accomplished by systematic muscular movements and careful hygiene at an early period of the manifestation of the morbid condition, the insidious contraction of the orifice may be averted. The means to this end are frictions, massage, carefully planned muscular movements, baths and bathing, the selection of suitable climates, and the regulation of diet. Medicinally iron, arsenic, small doses of liq. strychninae, and cod-liver oil are the chief agents to be employed. The treatment of complications and of failure of compensation will be as in other groups of the disease.

Group IV. Cases associated with chronic renal disease and arterio-sclerosis.—As I have already stated, the association between mitral stenosis and chronic renal disease was first pointed out by Goodhart (11), and confirmed by Pitt in 1887. The observations were made chiefly from the standpoint of morbid anatomy, though Pitt contributed some clinical data. It was made clear that the cases demonstrating the coexistence of the two morbid states are by no means infrequent. Nevertheless Gérard and others hold that mitral stenosis, having its origin in arterio-sclerosis, is rare. I cannot doubt that the explanation of this apparent conflict is to be found in the fact that the cases demonstrating the conjunction of the diseases are most frequently found after death; they come under clinical observation with comparative rarity. The two morbid affections progress insidiously, and either the patient is suddenly stricken down with apoplexy, or some sudden complication which precludes any physical examination, or, if such examination has been possible, the physical signs were supposed to indicate some form of disease other than mitral stenosis.

I have notes of six cases observed by myself, in which, without any evidence of rheumatism or other predisposing malady, there have been signs which should bring them into the group under consideration. In three other cases aortic valvular disease was conjoined with the mitral. Several others could be regarded as mixed cases, these having rheumatic antecedents; but the subsequent evolution was after the manner of arterio-sclerosis. In my cases the most advanced age was 70, the only
male; the youngest was 35. The cases recorded by Blind (four) and Gérard (five) which should come into this group are nine in number, five men and four women, the oldest patient 67, the youngest 32. In the cases taken as examples there is no rheumatic antecedent; but in the majority the usual signs of chronic Bright's disease are present. The radial and other arteries are firm and incompressible; the usual hypertrophy of the left ventricle of the heart, however, is not manifested. In some of the cases, in addition to the signs of thickened arteries, there are obvious evidences of gout with deposits of urates in the joints and elsewhere. In some there are well-marked signs of arterial atheroma. There may be emphysema of the lungs or pulmonary fibrosis. Fibroid changes may occur about the viscera, the perivisceritis of Huchard. The origin of the disease is not to be traced, the progress is slow and imperceptible. The physical signs of mitral stenosis in many of the cases do not differ from those ordinarily observed—the presystolic murmur, the entire diastolic, or the early or mid-diastolic murmur, the sudden, loud first sound and the double shock sound in diastole. In some cases there is no presystolic murmur, but a systolic. This may be heard at the apex and the back, thus answering to the criteria of mitral regurgitation; in such case it is probable that the auricle has become dilated and weak. The diagnosis of stenosis can only then be made from the evidence of a heaving and enlarged right ventricle, contrasting with the absence of signs of enlargement of the left ventricle, perhaps also from the absence of any second sound at the apex (Broadbent). Exceptionally there is no loud, sharp, short, sudden first sound, but a dull sound as in the case of a hypertrophied left ventricle. In the cases manifested between the ages of 30 and 40, there have been the evidences of the gradual oncome of chronic renal disease with thickened arteries or undoubted gout with deposits of urates. There is no evidence of any pre-existing disease of the valve due to rheumatic or other causes; but there must be a remaining doubt whether any change in the valve preceded the fibrous proliferations intrinsic to the Bright's disease. It is, I think, improbable, seeing that the great majority of cases due to rheumatism are fatal before the age of 40, that chronic Bright's disease is a superadded factor, for if so the scene would be more speedily closed, and death would ensue. It is at first sight more likely that the changes are independent of rheumatism and due to a slow form of sclerosis.

In one case, that of a lady aged 52, I had opportunity of observing the gradual involution of the disease as evidenced by the physical signs and confirmed by post-mortem examination. There was at first no sign whatever of cardiac disease, but gradually all the usual signs of mitral stenosis were manifested. The urine showed normal characters for nearly the whole period of observation, and the case was observed during thirteen years. The symptoms were those of dyspepsia, with gradual implication of the nervous system, first evidenced by an epileptic attack and afterwards by dementia. The necropsy showed funnel transformation of the mitral valve, with much fibrous thickening of the surrounding
structures. There were chronic fibrinous thickenings in the pleura, the left lung, the spleen, the liver, the capsules of the kidneys, and the membranes of the brain. The granular changes in the kidneys were but slightly pronounced, and no doubt comparatively recent. The chief morbid change was the widely-spread fibrosis, the progress of which had been very gradual; and it seemed legitimate to infer that the stenosis of the mitral orifice and the fibrous transformation of the surrounding structures were due to a similar morbid process. This case is no doubt exceptional in that the fibroid changes in so many situations long preceded any signs of interstitial nephritis. In the majority of cases the evidence of chronic renal disease is well marked when the case comes under observation. The age 50 to 70 renders it improbable that obstruction of the mitral orifice from any cause had preceded the gradual evolution of the chronic renal disease with its attendant arterio-sclerosis. In all cases the progress of the disease must have been very gradual and insidious. In many there have been signs of cerebral disease; indeed, it is for symptoms indicating such disease that the cases usually come under notice. Epileptic seizures, apoplexy, dementia, or uremia, are the chief forms. In several instances the signs of albuminuria retinitis have been recorded. That the morbid changes have been slow and gradual is shown also by the post-mortem evidence. In the case of a woman of 65 there has been found a funnel transformation of the mitral curtains; just as observed in the cases in earlier life; but in the majority the button-hole form of mitral stenosis is manifested with great thickening and firm fibrous transformation of the papillary muscles.

The treatment in this group of cases is subordinate to that of the chronic renal disease and the attendant thickenings of the arteries. It is important to realise that the prognosis is very grave. When a patient manifesting the signs of mitral stenosis at whatsoever age presents signs of firm and thick arteries, and the urine is found to be continually of low specific gravity and occasionally albuminous, it is well that for a few weeks an entire milk dietary be enjoined. It may be a little difficult to convince a patient past middle age, whose stomach has been the receptacle of foods of many and various kinds far more than adequate to the needs of his organism, whose nerves of taste have been frequently and abnormally stimulated, and whose absorption of nutritive material and excretion of effete products have been after the manner of periodic and irregular thunder-showers which have deluged the land and blocked the drains, that he must return to the sweet simplicity of the earliest months of his life. Yet it is best so. It is the absence of the irritation to the arterioles caused by the complex albuminoids which turns the balance towards amendment. It may be necessary, however, to make some concessions. In the early morning, or on waking, the patient may take half a pint of milk with half an ounce of rum, or of cognac and an ounce of lime water. In some cases one to two ounces of fluid magnesia may be substituted with advantage for the aqua calcis. Three or four hours afterwards a second half-pint of milk may be taken flavoured with a little hot coffee; the third
half-pint, after a like interval, may be taken as a blancmange made with isinglass or gelatine. At similar intervals, during the remainder of the waking hours, the changes may be rung with the various flavourings; but no solids should be permitted other than light biscuits.

The total amount of milk taken in the twenty-four hours should be three to six pints. The total quantity of cognac or spirits of any kind should be limited to two ounces. To break the monotony of the purely milk diet, it is a good plan to allow occasionally a firm jelly fully flavoured with madeira, rum, kirschwasser, or chartreuse. One or two tablespoonfuls of isinglass are to be melted in very hot water, and the milk added thereto; the small quantity of gelatin thus mingled with the milk is sufficient to prevent any firm curdling of the casein in the stomach, the coagulum being rendered much softer and its digestion facilitated (25).

In regard to medicinal treatment, the rule of Dr. G. W. Balfour should be followed, that no cardiac tonic should be administered without a simultaneous unlocking of the arterioles. The therapeutic measures should follow the lines already described in the consideration of Group II. of cases of "Mitral Regurgitation." As Sir Wm. Broadbent has said, "Nitro-glycerine and other vaso-dilators may sometimes be given with good effect for many weeks or even months in conjunction with general tonics, such as iron, quinine and nux vomica." (5).

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REFERENCES

DISEASES OF THE MITRAL VALVE


A. E. S.