EXAMINATION OF THE ORGANS OF CIRCULATION.

Inspection of the Præcordial Region.

The contraction of a heart which is structurally sound, normal in position and acting quietly, usually manifests itself externally as a slight elevation of the tissues in the fifth intercostal space, between the parasternal and mammillary lines; it is synchronous with the systole, does not raise the skin above the general level of the ribs, and is perceptible only over a small, circumscribed area, 1½—2, or at most 2½ ctm. in breadth. The normal impulse, commonly called the *apex-beat* * of the heart, never passes, to the right or left, beyond these limits. In children, however, it is not always situated in the fifth, but sometimes rises as high as the fourth, intercostal space, the diaphragm being drawn upwards with greater force by the lungs; in children also it not unfrequently passes a short distance (almost 1 ctm.) over the mammillary line towards the left side. On the other hand it occasionally happens, though relatively seldom, and only in the aged, that the heart's impulse is seen in the sixth intercostal space; in this case the displacement is due to diminution in the attractive force of the lungs and in the elasticity of the large vessels springing from the heart.

The situation of the apex-beat varies with the rise and fall of the diaphragm in the movements of respiration and on turning over towards the left side. The influence of respiration in altering the position of the heart's impulse is noticeable only on making a very deep inspiration, when the apex sinks, sometimes even behind the sixth rib, so that, the opposition to the transmission of the stroke of the heart on the chest-wall being increased, the heaving impulse can no longer be felt; in expiration it mounts again to its normal level. In quiet respiration

* By the term *apex-beat* is understood not only the impulse of the actual apex, but also that of the lower segment of the heart. The proper *apex* does not lie in the fifth intercostal space, but behind the sixth rib, and is also covered in front by a tongue-shaped process of pulmonary tissue connected with the lower border of the left lung.

no change takes place in the situation of the cardiac impulse. Lying on the left side brings it a little over the left mammillary line, occasionally as much as 2 ctm.; on turning on the right side no displacement, or the very slightest, is observable.

The apex-beat is not always visible, but is generally perceptible to the finger pressed deeply into the intercostal space; it escapes the eye also when the heart is acting very feebly, when the chest-wall is rigid and covered by a thick layer of fat and powerfully developed muscles, when the intercostal spaces are narrow, and when the heart, during full inspiration, is overlapped by the margin of the lung. (For the pathological conditions which cause the disappearance of the apex-beat, see p. 213).

In addition to the apex-beat, and occasionally also when it is wanting, a *diffuse* impulse, or undulation of the tissues in the præcordial region, may be observed, more particularly when the heart's action is abnormally strong.—A certain amount of vibration is also often felt between the third and sixth costal cartilages and over the lower portion of the sternum; it arises from the systolic tension of the mitral and tricuspid valves, and is designated the *valvular impulse*.

Vivisection teaches that the finger placed on *any* part of the exposed heart's surface experiences a distinct shock on the occurrence of each contraction; but in normal circumstances, from the relative position of the heart and lungs, only the stroke of the *apex* is felt. The entire base of the heart is covered by lung, which renders the transmission of its impulse to any distance a matter of difficulty; and this difficulty is increased by the backward movement of the base at each systole, by the resistance offered by the ribs, and by the thickness of the thoracic parietes (including the pectoral muscles and adipous tissue) in that region. The apex, on the contrary, is in immediate contact with the chest-wall, is formed chiefly by the powerful muscles of the left ventricle, and lies behind the yielding soft parts of the intercostal space; the most important consideration, however, is that this portion of the heart is tilted forwards at each systole, so that the tissues over it are of necessity raised along with it.

If the forementioned conditions, described as unfavourable to the occurrence of a visible impulse over the base of the heart, be removed, the apex-beat is accompanied by a distinct base-beat; this is the case in children with thin and yielding chest-walls, in

all cases in which the heart, in consequence of retraction of the anterior margin of the left lung (from atrophy), is in close contact with a large part of the thoracic parietes, and in all hypertrophies of the heart, especially of the left ventricle.

In a few instances a *double impulse*, accompanying with more or less regularity each systole, has been observed (Skoda, Bamberger, Leyden). It occurs in aggravated cases of mitral insufficiency, and arises from the non-coincidence of the contractions of the two ventricles. To the *first* of these strokes alone the pulsation in the arteries corresponds, with the second it is wanting. This non-simultaneous contraction may be explained in the following way: when the mitral valve is markedly incompetent the overfilled right ventricle is unable to empty itself completely during the systole, and the next instant (during the diastole) is again distended with blood and so excited to renewed contraction; the left ventricle, on the other hand, takes no part in the second phase of the contraction of the right heart, as it contains at this stage but a small quantity of blood, or if it does act in concert with the second contraction of the right ventricle, it does so with greatly diminished force.—It is quite conceivable that the so-called abortive contractions, that is, those which are so feeble as to produce no arterial pulse, often associated with grave mitral lesion, arise from the non-simultaneous contraction of the two ventricles; though if this assumption were correct a double impulse should accompany the cardiac systole, which is not actually the case.

Cause of the Heart's Impulse.

During the diastole the intracardiac pressure is equal at all points on the inner surface of the ventricular walls, but when the heart contracts and the blood is thrown into the great vessels this pressure suddenly becomes less over the heart's outlets than at the part diametrically opposite to them,—the apex of the heart; the latter, therefore, in consequence of the *recoil* so generated, moves downwards and forwards at each systole. It is the operation of the same force, the sudden development of a difference in pressure, that sets in motion Segner's water-wheel, that produces the recoil which follows the discharge of firearms, and that causes a freely-suspended cylinder, filled with water and provided with an escape-pipe at its lower end, to move backwards, in a direction opposed to that of the jet of water, when the stopcock is opened (Gutbrod, Skoda). But the recoil-theory explains only the impulse of the apex of the heart, and not that of the base; the latter arises from the *hardening and swelling of the heart at the beginning of the systole* (Arnold, Kiwisch, Ludwig).

It appears to me, therefore, that the theory of Gutbrod and Skoda and that advocated by Arnold, should not be regarded as antagonistic: each, taken *alone*, is capable of explaining only certain of the pathological phenomena dependent on the heart's action; taken *together*, they satisfactorily explain them *all*.

The essential appearances seen on exposing the pulsating heart by vivisection are: *locomotion downwards and forwards* combined with rotation from the left side towards the right, and *increase in thickness* during each systole; *all* these factors must be taken into account in studying the causes of the cardiac impulse, no theory being free from objection which is founded on only one of them. While it is generally admitted that the impulse of the base of the heart is due simply to systolic increase in thickness and firmness of the cardiac tissue at that part, authorities are far from being unanimous in their explanations as to the manner in which the downward and forward movement of the *apex*, which gives rise to the apex-beat, is brought about. That the apex of the heart actually does move in the direction described, and at the same time rotates from left to right, has been frequently demonstrated, both in animals and in men suffering from thoracic fistula in the præcordial region. Bamberger teaches that the change in the position of the heart is the result of the systolic stretching of the aorta and pulmonary artery, and Kornitzer that the movement of rotation depends on the somewhat spiral arrangement of these vessels as they spring from the heart,—the elongation which they undergo making this spiral to turn slightly round on its vertical axis, carrying the heart with it. But Kornitzer's further hypothesis that this rotation has the effect of tilting the apex of the heart forwards, and should therefore be ranked as the special cause of the cardiac impulse, has not yet been satisfactorily proved. The same objection applies to another theory, more recently introduced by Aufrecht, which finds the cause of the heart's impulse in the *systolic flattening of the aortic arch*, and which seeks to explain exclusively on this ground all the physiological and pathological phenomena connected with the heart's beat; the observation that with each systolic discharge of blood into the aorta a diminution of the curvature of that vessel takes place, the aorta itself rising and the base of the heart sinking, is essentially corroborative of Bamberger's theory that the locomotion of the heart downwards is produced by the systolic stretching of the vessels, Aufrecht, however, holding that the pulmonary artery takes no share in the production of these changes. But whilst Bamberger does not attempt to found any doctrine as to the cause of the heart's impulse on this systolic depression of the heart by the elongation of the great vessels, Aufrecht argues that to it is added a forward movement of the organ, and that these together give rise to the impulse. The two theories thus agree so far, in regarding the injection of the blood into the arteries as the cause of

the apex-beat (the latter, indeed, instantly disappears, as Hiffelsheim and Jahn have shown, on cutting off the supply of blood to the chambers of the heart), but differ concerning the manner in which this cause operates.—The view which has found most wide acceptance is that advanced by Gutbrod and adopted by Skoda, based on the physical principle of recoil.

The two most important objections to the Gutbrod-Skoda theory are the following: 1st, Bamberger, Kürschner, Scheiber, and others, maintain that the physical principle of recoil is not applicable to the heart, as those parts on which the force of the recoil should fall are precisely those which by their contraction generate the original force; a recoil, therefore, at the apex would be overcome by the counter-pressure arising from the contraction of the heart from the apex towards the base.

This counterpressure, however, is in part neutralized by the downward movement of the heart at each systole, but chiefly by the fact that the heart, as Skoda points out, does not contract simply from the apex towards the base, in the direction opposed to that of the recoil, but *concentrically*, so that the increase of pressure on its walls is everywhere *equal;* in this case the cardiac parietes are placed under exactly the same conditions as the sides of a cylinder filled with water, —they are subjected to a uniform pressure.

2ndly. The second objection rests on the observation that that point on the inner surface of the heart which is diametrically opposite to the arterial orifices does not coincide with the apex, but with a spot on the wall of the right ventricle (Scheiber); the recoil from the orifice of the pulmonary artery takes effect on the side of the right ventricle, that from the aorta exactly on the apex of the heart, so that the resultant of these two forces passes through a point situated somewhat to the right of the apex. It is only in cases of unequal hypertrophy of the different parts of the heart that these relations are appreciably disturbed.

Skoda meets this objection by the statements that the aortic and pulmonary orifices do not lie in the same plane; that the heart may, on account of the intricate disposition of its muscular fibres, be considered as a single-chambered tubular organ, the lower part of which, diametrically opposite to the arterial orifices, suffers a certain amount of pressure from the current of blood flowing from it into the great vessels, the area so affected being equal to the sum of the transverse sections of the pulmonary and aortic openings; and that this pressure is distributed over the surface of the lower segment of the heart, which assumes a conical form during the systole; and he concludes that it is not essential to the general accuracy of his theory that the force of the recoil should light *exactly* on the *apex*. Jahn has also recently shown that the resultant of the lines of recoil from the aorta and pulmonary artery falls precisely on the apex of the heart, as indicated by Gutbrod and Skoda.

Hiffelsheim has demonstrated the recoil experimentally in a caoutchouc heart filled with water, discharging into an artificial aorta; he found that the force of the recoil is proportional to the volume of fluid contained, to the thickness of the walls of the artificial heart, and to the diameter of the aortic orifice.

On p. 214 have been mentioned certain clinical facts which admit of satisfactory explanation by means of the Gutbrod-Skoda theory only.

Pathological Alterations of the Normal Cardiac Impulse.

These affect the situation, force, and extent of the impulse.

Alteration in the *situation* of the apex-beat arises from *displacement* of the whole heart, which is very frequently the result of change in the position of the diaphragm,—when the latter is depressed the heart sinks, when elevated the heart rises.

Depression of the whole diaphragm may be caused by bilateral emphysema of the lungs, lowering of one-half by the presence of gas or fluid in one of the pleuræ. The dislocation of the heart in pulmonary emphysema takes place downwards and to the right, in effusion into the left pleura, when the fluid is moderately abundant, simply downwards, or when the quantity is excessive, to the right side, sometimes even as far as the right mammillary line; pyo-pneumothorax of the left side has the same effects, though not usually to such a marked degree. On the absorption of the exudation, should the left lung fully expand as the fluid is removed, the heart, if it have not formed adhesions to its new surroundings, returns gradually to its normal position.

If the displacement of the heart towards the right be but slight the apex-beat remains at the natural level or, if the diaphragm be forced a little downwards by the weight of the fluid, sinks to a trifling extent; the position of the heart with regard to the direction of its axis usually continues normal, however, that is, the apex is still the part which lies furthest to the left. But as the heart approaches the sternum its axis becomes more and more vertical; if it be thrust beyond the middle line of the sternum the apex rises, from the greater elevation of the *middle* segment of the diaphragm,—the part over which the apex must, of course, pass when pushed over into the right side of the thorax; the impulse may then be felt in the fourth right intercostal space. The apex forms now the portion of the heart situated furthest to the right, as its movement is not hampered by its anatomical relations, whilst the base, from the manner in which it is bound down by the great vessels, is less capable of being altered in position.—Very copious effusions into the *right pleura* push the heart beyond its normal limits towards the left side, sometimes even as far as the axillary line. In most

cases also in which the heart is displaced by pleuritic exudation the apex-beat is considerably augmented in force, on account of the greater resistance offered by the fluid to the emptying of the right ventricle; the pulsation not only of the apex but commonly also that of a large portion of the heart's surface may then be seen.

The diaphragm may come to occupy an abnormally high position as the result of atrophy of one lung, or of atelectasis of a lung after absorption of pleuritic exudation of long standing; or it may be driven from below upwards by increase in the volume of the abdominal organs, particularly by hepatic, splenic, uterine, and ovarian tumours, and by ascites or meteorism. The heart rises higher in the thorax in proportion to the extent of the upward displacement of the diaphragm, and in extreme cases may pulsate in the third intercostal space.

Contraction of the left lung causes the heart to beat not only at a higher level but also frequently to the outside of the mammillary line, or even in the axillary line, as the mediastinum, like the diaphragm, is also dragged further into the diminished cavity of the thorax. In contraction of the right lung the heart, with the anterior mediastinum, passes over to the right side, sometimes as far as the right border of the sternum and even beyond it.

Congenital malposition of the heart exists in cases of *Inversion of the Viscera;* the heart is here found on the right side, beating in the fifth right intercostal space, and the cardiac axis is directed from above and behind downwards, forwards, and to the right.

The position of the apex-beat (or cardiac impulse) may further be modified by *increase of the size of the heart*, hypertrophy with dilatation.

When the *left* ventricle is hypertrophied and dilated it is chiefly in its *long* diameter that its size is augmented, so that the impulse is shifted downwards to the sixth, seventh, or even the eighth intercostal space; as the ventricle also gains sensibly in breadth the pulsation of the heart extends outwards beyond the mammillary line.

In hypertrophy with dilatation of the right ventricle it is principally the *breadth* of the heart that is added to; its impulse, accordingly, passes further to the right than normally, sometimes to the right margin of the sternum or even to the right mammillary line. In cases in which the dilatation of the right ventricle is moderate in amount the cardiac impulse to the right

is not so distinctly visible as it is to the left when the left ventricle is hypertrophied to an equal degree, the intercostal spaces becoming considerably narrower towards the sternum.

In dilatation of the right heart the cardiac impulse oversteps the normal boundaries also to the left and downwards; it reaches outwards to or even beyond the left mammillary line, and inferiorly often to the sixth intercostal space. Both these conditions are due chiefly to a change in the relation of the axis of the heart to that of the body: the heart, when the dilatation of its right half is considerable, assumes a less upright and more *horizontal* position.—In very pronounced cases of hypertrophy and dilatation of the left side of the heart the impulse, though most marked towards the left, is appreciable beyond its normal limits also towards the right.

The *force of the heart's impulse* presents great differences even amongst individuals who are in perfect health, and is found also, other circumstances being the same, to vary chiefly with the energy with which the heart contracts. The impulse may be so *weakened* as to escape detection either by eye or hand, or may, on the contrary, become so *forcible* as to elevate and throw into vibration a large part of the chest-wall.

Enfeeblement of the cardiac impulse, even to such a degree that no perceptible shock is communicated to the hand, takes place (apart from the physiological causes already mentioned on p. 207) in the following circumstances:

1. When the heart's action becomes less vigorous than in health. This may occur as the result of fatty degeneration of the cardiac muscular fibres, or of inflammatory changes in the latter, due to myocarditis or arising in the course of severe acute affections; or it may be owing to the prolonged influence of a high febrile temperature (from the so-called albuminous infiltration of the muscular fibres), or to abnormally feeble innervation of the heart. This disorder of the innervation may be of a transient character, as in an ordinary case of fainting, or it may be more lasting or recurrent, presenting itself frequently as a concomitant symptom in many diseases of the nervous system; and, finally, it usually appears shortly before death, indicating paralysis of the nervous system.

2. When the heart is separated from the chest-wall by any medium, which may either be interposed between that organ and the pericardium (—the presence of fluid, or rarely air, in the

pericardium), or which covers the heart (an emphysematous lung, for instance), or which comes between the heart and the thoracic parietes (such as pleuritic exudation, air in pneumothorax). Displacement of the heart from the causes just mentioned does not abolish the cardiac impulse, the latter being in such conditions usually visible at some other than the normal spot.

3. When the heart has contracted adhesions to the pericardium. In these cases, as the systolic movement of the heart downwards and forwards is rendered impossible, the impulse is absolutely wanting, and instead of the raising of the tissues by the apex-beat we have systolic retraction taking place in the region where the former is normally felt, (see p. 222).

Finally, it is sometimes observed that when stenosis of the aortic orifice or of the left auriculo-ventricular orifice reaches a certain degree of intensity *the apex-beat is absent ;* in such circumstances also *the impulse of the base* is, on account of the consecutive hypertrophy, much increased in force and spread over a larger surface than naturally.

The absence of the apex-beat in severe stenosis of the aortic orifice admits of very satisfactory explanation by means of the Gutbrod-Skoda theory of the causation of the normal impulse, and in the following way:—the force of the recoil depends, not only on the energy with which the ventricle discharges its contents into the arterial system, but also on the rapidity and volume of the current of blood, that is, on the diameter of the aperture of exit. When this diameter is encroached upon in stenosis the quantity of blood propelled from the ventricle in a given unit of time becomes less, so that the diminution in pressure at the aortic orifice is not so marked, nor is the systolic *difference* between the pressure at the aortic orifice and that at the apex of the heart so great, as under normal conditions; the recoil of the apex, therefore, that is, its systolic locomotion downwards and forwards, is also less, and may become so slight as to be no longer appreciable.

In like manner the want of the apex-beat in severe stenosis of the left auriculo-ventricular orifice may readily be accounted for; the constriction prevents the left ventricle being so completely filled with blood as it should be before its contraction, and the systolic difference between the pressure over the aortic orifice and that at the apex of the heart is consequently *abnormally small*.

The force of the cardiac impulse is increased by whatever strengthens the heart's contractions: in health, therefore, this takes place from mental excitement or violent physical exertion; pathologically it occurs in all febrile conditions, in inflammatory

diseases of the heart, endocarditis, pericarditis (in the latter, however, only so long as the exudation is insufficient in quantity to obscure the perception of the shock), in the various neuroses of the heart (which may exist independently of any other disease, or may complicate other morbid processes of the most diverse kinds), and in all those conditions which favour the transmission of the heart's impulse to the surface, such as condensation of the upper lobe of the lung, retraction of the margin of the left lung, &c. This phenomenon is met with most often and in its most aggravated form, as the result of increase of the muscular substance (*hypertrophy*) of the heart.

The impulse is the more forcible the more the hypertrophy of the muscular structure of the heart preponderates over the dilatation of its cavities; should the hypertrophy cease to increase, or should it even decrease, as it frequently does in the more advanced stages of the disease, from fatty degeneration of the muscular fibres, so that the dilatation comes to be a more prominent feature in the case than the hypertrophy, the impulse of the heart grows feebler; this is observed in the later stages of all cardiac lesions.

The heart's impulse is stronger in hypertrophy of the *left* ventricle than in that of the right. When the power of the *left* side of the heart is increased to a considerable degree the impulse takes on a *heaving* character, and in severe cases the greater part of the front of the left side of the chest is elevated and thrown into distinct vibration each time the heart contracts; in the diastole the raised portion returns sharply and with some force to its original position. Such a heaving impulse is never observed in hypertrophy of the right ventricle, as in the latter the thickening of the cardiac muscle is not so great as in the left ventricle; while the thickness of the hypertrophied wall of the right heart amounts only to $\frac{3}{4}$—1 ctm. or very little more, that of the hypertrophied left heart reaches 2—$2\frac{1}{2}$ ctm., or even slightly exceeds that measurement. In a large number of cases also an increase in the force of the cardiac impulse from hypertrophy of the right ventricle is not very readily detected by the eye, as the right side of the heart is not so favourably situated anatomically as the left for the conduction of its stroke to the surface (compare p. 212); it may, nevertheless, always be *felt*, by placing the hand on the lower part of the sternum.—Other things being equal the impulse of

the heart is most powerful in enlargement of the whole organ.—When the hypertrophy of either ventricle is considerable it may give rise to very marked *prominence* in the præcordial region, particularly in young persons, whose thoracic parietes yield readily to pressure.

The following is a short statement of the general causes of hypertrophy of the heart.

Hypertrophy of the heart is almost invariably the result of the existence of obstacles to the free circulation of the blood; to overcome these obstacles more energetic contraction of the heart is necessary, and this, as in every case in which a muscle is habitually called upon to exert its full power, leads to an increase in the bulk of the cardiac muscular substance. The development of hypertrophy is always preceded by *dilatation* of that ventricle which, on account of the resistance encountered in the systemic or pulmonary circulation, is unable to empty itself completely of its blood at each contraction. Hypertrophy is thus always associated with dilatation of the corresponding ventricular cavity. This is known as *excentric* hypertrophy.

Hypertrophy (with dilatation) of the *left* ventricle arises from the presence of some *impediment to circulation in the aortic system*, hypertrophy (with dilatation) of the *right* ventricle from *obstacles in the pulmonary system*. Other conditions being similar, the thickening is usually the more marked the nearer the obstruction lies to the ventricle.

Hypertrophy of the *left* ventricle may be caused by atrophy of the kidneys, by sclerosis of parts of the aortic system, by *atheromatous degeneration* of the walls of the aorta, by *stenoses* of the aorta, by *aneurisms* of the aorta (when complicated by lesions of the aortic valves), by *insufficiency* of the aortic valves, and *stenosis* of the aortic orifice.

Hypertrophy of the *right* ventricle may be due

1. To *engorgement of the pulmonary circulation*. It is in this way, by impeding the return of the blood through the pulmonary vein, that *mitral lesions* (insufficiency and stenosis), cause enlargement; in order to enable the right ventricle to rid itself of the whole of its contents, notwithstanding the opposition presented by the overloaded condition of the pulmonary vessels, its muscular mass undergoes large increase.

2. To *obliteration of a large number of the pulmonary capillaries*. This arises from vesicular emphysema, atrophy of the lung from chronic interstitial disease, prolonged compression of the lung by pleuritic exudation, and from lateral and angular curvatures of the spinal column. In these cases it is inevitable that the right ventricle should undergo a certain amount of hypertrophy, that it may contract

with sufficient force to open up a pathway for the blood through the already reduced area in which it circulates in the lungs.*

3. To *valvular lesions at the orifice of the pulmonary artery* (stenosis of the conus arteriosus or of the ramifications of the pulmonary artery, insufficiency of the *semilunar valves* at the root of the same vessel,—both very rare affections).—Incompetence of the tricuspid valve is usually associated with hypertrophy of the right ventricle, which, again, is chiefly the result of the *concomitant mitral lesion*, and which, in uncomplicated cases, is exceedingly slight.

If the circulation be obstructed both in the aortic and the pulmonary systems, (as in cases of lesion of the aortic and mitral valves), hypertrophy of both ventricles takes place. Severe aortic lesions alone may occasion thickening of the right as well as of the left ventricle, as in the more advanced stages of aortic valvular disease congestion of the pulmonary circulation also appears.

The *auricles* also *undergo hypertrophy* when, from contraction of the auriculo-ventricular orifice, they are unable to empty themselves entirely, or when, from insufficiency of the auriculo-ventricular valves, the blood regurgitates at every systole from the ventricles into the auricles, so that the latter are kept constantly in a state of overdistension; in both affections the complete evacuation of the auricles becomes impossible, and conditions are presented which are favourable to the development of hypertrophy of these parts, (of the left auricle, for instance, in stenosis and insufficiency of the mitral valve).

Hypertrophy with dilatation of the ventricles may also arise *independently* of the existence of any mechanical hindrance to the circulation, and due simply to overaction of the heart. Though the truth of this doctrine has often in the past been held doubtful it has recently been firmly established on the strength of numerous and accurate observations. This form of hypertrophy without any trace of valvular lesion is developed especially after violent and prolonged physical exertion, as in soldiers as the result of long and fatiguing marches, &c., and amongst the labouring classes; its production is also sometimes favoured by undue indulgence in spirituous liquors and by immoderate smoking.

Extent of the Heart's Impulse.

An impulse whose breadth within the fifth intercostal space

* Bäumler has recently made the assertion that extensive, but more particularly complete *adhesion of the pleural surfaces* to each other gives rise to a disposition to the development of *hypertrophy of the heart*, as the free expansion of the various parts of the lungs is thus hampered or prevented and the elasticity of the lungs diminished. But as diminution of the elasticity of the pulmonary tissue impedes the outflow of blood from the pulmonary veins towards the left ventricle the pulmonary circulation becomes overloaded, and the work which the right heart has to perform is increased. There is thus produced a *disposition* to hypertrophy of the right heart, the occurrence of which in such cases is generally further favoured by other concomitant circumstances. —When at a later stage the congestion extends also to the systemic veins, and the left ventricle is thus called upon to contract more energetically, the left side of the heart also becomes enlarged, though not usually to such a considerable degree as the right.