having large muscles and an abundant covering of fat over the chest. They are further intensified by everything which increases the force of the heart's contractions,—mental excitement, physical exercise, the febrile state; in these circumstances *all* the sounds, both those connected with the ventricles and those emanating from the vessels, are alike influenced.

*Intensification of only one of the diastolic arterial sounds*, however, caused by hypertrophy of the heart, is very frequently observed, either in the region of the aorta or of the pulmonary artery. To determine whether there is actually any exaggeration of either sound it is only necessary carefully to compare the one with the other. Normally the second aortic sound is a very little louder than that of the pulmonary artery. Under pathological conditions the second sound at the two great arterial orifices varies in strength within very wide limits; the reinforcement of the sound may even be so considerable that the diastolic recoil of the blood against the semilunar valves, which gives rise to the sound itself, may be perceptible to the hand or to the eye as a distinct pulsation in the superficial parts corresponding to the situation of these vessels (see p. 238).

*Hypertrophy of the left ventricle*, from whatever cause arising, (excepting only that form which results from aortic insufficiency, as in such circumstances no sound is produced, but a diastolic murmur), *intensifies the second aortic sound.*

An *intensification of the second pulmonary sound* is the necessary consequence of *hypertrophy of the right ventricle*, to whatever cause the latter may be due, with the exception of those exceedingly rare cases in which the enlargement is connected with insufficiency of the pulmonary valves, when the diastolic sound is suppressed and replaced by a diastolic murmur. The most notable increase in the loudness of this sound takes place in hypertrophy of the right side of the heart from mitral insufficiency or stenosis of the mitral orifice, or from a combination of both lesions; it is as a rule more marked in the young than later in life.

The degree to which the second pulmonary or aortic sound is exaggerated in intensity is generally directly proportionate to the enlargement of the corresponding ventricle.—When, from fatty degeneration of the muscular substance of the heart, the contractile power of the hypertrophied ventricle begins to fail and to



be inadequate to fully compensate for valvular defects, the strength of the second arterial sound also diminishes.

It was Skoda who first drew attention to exaggeration of the second pulmonary arterial sound as a positive and unerring indication of mitral valvular lesion (in the stage of compensation). The same phenomenon is observed, though less strikingly developed, independently of defect of the mitral valve, in every variety of congestion of the pulmonary circulation, as this commonly leads to hypertrophy of the right heart (*e.g.*, bronchial catarrh of long duration accompanied by vesicular emphysema). It is in mitral affections, however, that it is found in its most marked form, as in such cases the consecutive hypertrophy of the right heart reaches its maximum.

The increased loudness of the second arterial sound is the more distinctly audible the fewer and the less important the obstacles to the transmission of the sounds to the walls of the chest; it is thus, other things being equal, most declared when the anterior border of the lung recedes and exposes entirely the base of the heart, as so often happens when the heart is very considerably hypertrophied in young persons, the base of the organ being then in immediate contact with the front of the chest. If, on the contrary, the heart be completely closed in by pulmonary tissue, whether from adhesion of the anterior margin of the lung in such a manner that it cannot retreat or from emphysematous distension, the second pulmonary arterial sound may be of merely its normal strength notwithstanding the presence of very decided hypertrophy of the right side of the heart.—It is further a common experience to find this sound of greater intensity when the upper lobe of the left lung is consolidated and excavated, and that without any enlargement of the right heart; the first pulmonary sound then also appears to be somewhat louder than the first aortic sound. In such cases, however, the pulmonary sounds are not in reality louder than those originating at the aortic valves, they are merely carried to the surface with greater precision through the solid lung parenchyma. Moreover, in those instances in which the consolidated portion of lung shrinks in volume a large part of the *base of the heart* lies immediately behind the thoracic wall.

Exaggeration of the second arterial sound is of much less frequent occurrence in the aorta than in the pulmonary artery, as simple and uncomplicated hypertrophy is less common on the left side than on the right, and as, further, it is precisely in the affection which most often causes enlargement of the right ventricle,—aortic insufficiency,—that the diastolic sound is masked by a diastolic murmur. Almost the only disease of the heart, therefore, which results directly in hypertrophy of the left ventricle and intensification of the second aortic sound is simple contraction of the aortic orifice unattended by lesion of the aortic valves,—a condition which is on the whole seldom met with. The most exquisite examples of exaggeration of the second aortic sound are observed in hypertrophy of the left heart from atrophy of the kidneys or arterial sclerosis, as in these cases the valves and the orifice of the aorta remain perfectly normal.

### Enfeeblement of the Sounds of the Heart.

This is frequently noticed, within physiological limits, in perfectly healthy persons and in those whose chests are protected by a thick covering of fat. Pathologically it proceeds from one of three causes: from the mere feebleness of the heart's action, —in cases of general debility, therefore, in convalescence from severe acute diseases, and in fatty degeneration of the cardiac muscular substance; from the presence of some obstacle to the clear transmission of the heart-sounds to the chest-wall, as when the margin of an emphysematous lung comes in front of the heart, and when the latter is separated from the thoracic parietes by pleuritic or pericardial effusion; or from masking of the sounds by loud râles in the adjoining parts of the lungs.

*All* the cardiac sounds are weakened in the conditions just named. But there are also cases of cardiac valvular lesion in which the second arterial sound alone is effected; the sign, however, is of no great diagnostic importance. Thus, the partially-filled state of the aortic system which accompanies extensive and advanced mitral disease (insufficiency and stenosis) leads to enfeeblement of the second aortic sound; and in the rare cases in which the tricuspid valve becomes incompetent the diminution in the quantity of blood in circulation in the pulmonary system lessens the intensity of the second pulmonary arterial sound.

### Heart-Sounds altered as regards their Purity.

The sounds of the heart, particularly the systolic ventricular sounds, are frequently neither so accentuated nor so markedly flapping in character as those heard in health; they become impure, and in extreme instances lose more or less completely the qualities of a sound proper, so much so that one is sometimes in doubt whether to regard them as sounds or murmurs. Sounds of this doubtful kind often merge into unmistakeable murmurs when the heart is excited to more powerful action.

The slighter degrees of impurity of the sounds may have their origin in comparatively unimportant changes in the auriculo-ventricular and arterial valves; thus a merely trifling diminution in the structural delicacy of the valves, a slight thickening of



their substance, perhaps also the want of absolute uniformity of tension and vibration in the various valvular segments, and other similar but not yet thoroughly known conditions, may be sufficient to produce such a result.

*Impurity of the Sounds*, when not associated with other cardiac anomalies, particularly with changes in the cardiac impulse or in the volume of the heart, has not usually any special diagnostic value.

### Heart-Sounds of Ringing, Metallic Quality.

These are observed when air gains access to, and accumulates in the pericardium (*pneumopericardium*), in left pneumothorax, and when there are large air-filled cavities in those parts of the lungs which are in immediate proximity to the heart. In all these cases a metallic timbre is added to the heart-sounds, by consonance in the air-spaces through which they are conducted (compare the metallic respiratory murmur and râles, pp. 139, 156, *et seq.*).

The heart-sounds acquire a ringing metallic timbre of the most exquisite quality in pneumopericardium. The air may enter the pericardial sac from without through wounds, or from within through pathological communications established between parts containing air and the sac, as from perforation of the œsophagus or stomach, or the bursting of a pyopneumothorax or pulmonary cavity into the pericardium, or, as in one case I saw in Traube's clinique, the gas may be given off by a pericardial exudation.

In left pneumothorax the sounds have not invariably the metallic character; it is wanting particularly when the heart is much displaced towards the right side. The amount and tension of the air in the pneumothoracic cavity modify this sign in many ways. It is only under specially favourable circumstances also that pulmonary cavities situated near the heart communicate to the sounds a metallic timbre.

Whilst in the pathological conditions just mentioned *all the heart-sounds* have the ringing quality, in cases of *atheromatous* degeneration limited to the initial portion of the aorta the second aortic sound alone is frequently of this character, so long as the valvular segments at the root of the vessel remain perfectly intact.

### Reduplication or Division of the Heart-Sounds.

The systolic and diastolic sounds may each be broken up into two distinct parts; if these be separated from each other by a short pause they may be spoken of as *reduplicated* cardiac sounds, or if

the one passes into the other without any appreciable interval, as *divided* sounds. Reduplication and division, however, are terms which are generally used synonymously, as both forms commonly owe their existence to the same causes, and are apt to pass insensibly into each other even in one and the same patient. For instance, the pause which comes between the two elements of the sound disappears when the heart's action is accelerated, and the properly double sound becomes a divided sound. The closer the two parts approach to each other the less clear and pure is the quality of the sound. The rhythm of the heart-sounds when the first is reduplicated is anapæstic, when the second sound is so affected, dactylic.—In some rare cases the sound falls into three very short portions.—Division of the first ventricular sound is relatively most frequent, the next most common phenomenon of this kind being doubling of the second arterial sound.

Reduplication of the *first* ventricular sound occurs in perfectly healthy persons, but is then never permanent,—it comes and goes, disappearing entirely as the force and rate of the heart's action are increased. It is noticed temporarily also in diseases of the heart, but cannot be said to be characteristic of any particular affection. It is connected sometimes with mitral and sometimes with tricuspid disorder, and most probably originates in *non-synchronous tension* of the individual segments of the auriculo-ventricular valves. This explanation does not necessarily involve the assumption of the non-simultaneous contraction of the ventricles, though such an occurrence is not impossible in certain cases, marked by a double cardiac impulse (see p. 208); the irregularity in tension of the valvular segments may quite justifiably be ascribed to the absence of perfect uniformity in the contraction of the papillary muscles. When the heart begins to act more *powerfully* these muscles contract regularly and energetically, the tension of the valves with which they are connected becomes uniform, reduplication of the sounds vanishes, and the two sounds become *one*.

Two other explanations, much less probable than the one here adopted, have been advanced to account for doubling of the first sound.

1. It is explained by some on the supposition that the tension of the arterial walls produced by the entrance of the systolic blood-wave is later in point of time than the tension of the auriculo-ventricular valve. But in the first place, the interval which separates these two phenomena



is so exceedingly short that the formation in this way of two systolic sounds, often marked off from each other by a very appreciable pause, is scarcely possible; and in the second place, even in health there is always such an interval, though it is not directly measureable, between the systole of the heart and the expansion of the aorta and pulmonary artery, whilst the occurrence of a double first sound is far from common and is only periodic in those in whom it does appear.

2. Reduplication of the first sound has further been said to be due to the non-simultaneous contraction of the two ventricles, a similar irregularity being thus established in the tension of the mitral and tricuspid valves. But in these circumstances the second arterial sound should also always be doubled, for if the ventricles do not contract in concert arterial expansion, and consequently the diastolic closure of the semilunar valves, should likewise be non-simultaneous. Moreover, both portions of the divided systolic sound are exactly alike in intensity and timbre, which would not be the case if they were propagated from different ventricles. And lastly, this theory fails entirely to explain those rare cases in which the first sound is split up into three parts (*trommelschlag*, drum-beat).

In view of the fact that reduplication of the systolic sound of the heart is not very rare even whilst the organ itself is healthy, no very decided diagnostic signification can be attached to the sign. I have on several occasions observed that it could be artificially produced by pressing firmly over the apex of the heart in those in whom there was already some impurity of the first sound. Potain has also, in cases of hypertrophy of the heart consequent on granular atrophy of the kidneys, met with a variety of double first-sound (*bruit de galop*) in which besides the two normal sounds a third was heard, coming immediately before the first sound and separated from it by a short pause; this additional sound was therefore presystolic. It is quite conceivable that this presystolic sound, which I too have noticed in a number of instances of hypertrophy of the heart, is caused by the contraction of the hypertrophied auricles. The auriculo-ventricular valves are to a certain extent rendered tense even at the end of the diastole, that is, in the presystole, but this tension is normally so feeble that no sound results; but if the walls of one of the auricles undergo hypertrophy as the consequence of some valvular lesion, the corresponding auriculo-ventricular valve is put more sharply and thoroughly on the stretch by the contraction of the auricle and in this way the conditions necessary to the production of a presystolic sound are realised. The systole of the heart is then followed by another sound, dependent on the much fuller tension of the valve, and considerably louder and clearer in tone than the presystolic sound in question. — I have frequently heard doubling of the first sound associated with a systolic murmur; occasionally also I have observed this sign at once develope into a marked systolic murmur when the heart's action was increased in force.

Reduplication or division of the *second arterial sound*, audible over the aorta and pulmonary artery, occurs sometimes physio-

logically, but more generally as a pathological condition. Its cause is *non-simultaneous closure of the pulmonary and aortic valves*. The two diastolic arterial sounds therefore do not coincide with each other, and one of the two heard over either of the arteries is thus invariably conducted from the other vessel. It is, nevertheless, not unreasonable to suppose that certain anatomical changes in the semilunar valves might cause the tension of these structures to take place in two distinct movements, and thus give rise to division of the second sound; this assumption would explain those cases in which the reduplication is confined to the sound proceeding from only *one* of the arterial orifices, while that arising at the other is either simple and undivided or very much enfeebled in its second element.

Reduplication of the second sound occurs most often, and is most fully developed, in *stenosis of the mitral orifice*. In my experience it is heard in almost a third of all such cases. It is most clearly defined over the lower portion of the sternum and near the apex of the heart, and is less pronounced in the region of the arterial orifices; it is, further, generally appreciable only when the heart's action is slow and tranquil, while if the heart be excited to quicker and more vigorous contraction the two sounds usually merge into a diastolic murmur: in some cases, however, they persist, notwithstanding the appearance of this murmur. The double diastolic sound associated with mitral contraction remains tolerably constant, and always returns even after a temporary disappearance.

Reduplication of the second sound of the heart in mitral stenosis is difficult to account for satisfactorily. Geigel ascribed it to non-coincidence of the closure of the arterial valves, the necessary consequence of the unusual difference in the quantity of blood contained by the aorta as compared with that in the pulmonary artery in cases of this affection; the aorta receives a relatively small quantity of blood on each contraction of the heart, is therefore but slightly distended, and accordingly contracts sooner than the pulmonary artery and its valves are sooner closed than those at the entrance to that vessel; the latter also is constantly overloaded with blood discharged by the hypertrophied right ventricle and is thus unduly distended, and its walls are eventually to a considerable extent deprived of their elasticity.

There are several circumstances, however, which seem to tell decidedly against this view: the broken diastolic sound is (so far as I have observed) certainly not loudest over the large vessels, but at the lower part of the sternum and near the apex of the heart, and is, further, absent in the more marked cases of mitral contraction, precisely the cases in which the conditions most favourable to the postponement of the closure of the pulmonary valve are present in the highest degree; division of the diastolic sound, also, is never met with in mitral *insufficiency*, notwithstanding the fact that it is followed by the same consecutive changes (hypertrophy and dilatation of the right ventricle, engorgement of the pulmonary circulation) as stenosis. It appears rather that the reduplication of the second sound originates at the narrowed orifice itself, as it so often vanishes completely on increasing the force of the heart's action, and gives place to a loud diastolic murmur; there is therefore nothing forced in the inference that the two elements of the phenomenon in question, which form a sound which is always more or less muffled or impure, are in reality the component parts of a murmur.—It has also been conjectured, on the other hand, that the first part of the divided sound is simply the diastolic pulmonary sound, and that the second is produced, towards the end of the diastole, by the contraction of the hypertrophied left auricle,—a theory which would yield a plausible explanation of those cases in which the reduplication remains even when the diastolic murmur is developed.

A doubled second sound is heard also when the *pericardium becomes adherent to the heart* (Friedreich); here the posterior wall of the chest, being first dragged inwards towards the vertebral column during the cardiac systole, springs back sharply to its original position during the diastole, the parietal vibrations so generated constituting a dull muffled sound following closely on the second ventricular sound.



### Cardiac Murmurs.

The murmurs heard over the heart, in pathological conditions, arise either *within* the heart or the initial portion of the great vessels, or *outside* the heart, on its outer surface; the first are named *endocardial*, the second *pericardial*, murmurs.

### Endocardial Murmurs.

These are produced,

1. By those anatomical changes in the valves or arterial coats which give rise to incompetence of a valve, to contraction of a valvular orifice, or to dilatation of the roots of the great vessels, lesions which all cause some obstruction of the circulation; occasionally, however, they are produced also by anatomical changes which offer no impediment to the passage of the blood, such as the presence of deposit on the endocardial lining of the ventricles, &c.

2. By non-uniformity in the tension of valves and arterial walls, the anatomical structure of the heart being perfectly normal.

Murmurs dependent on the existence of actual obstruction of the circulation are termed *organic* murmurs, those occurring independently of such obstacle, and in a heart in no way altered in structure, *inorganic* murmurs.

*Organic* murmurs arise from *oscillation* (a whirling, eddy-like motion) *of the blood-current*, caused by the obstruction which embarrasses the circulation.

The physical cause of this oscillation in cases of stenosis of any of the cardiac orifices (arterial or venous) is the abnormal force with which the mass of blood is driven through the narrowed aperture; in valvular insufficiency it depends on the circumstance that a part of the blood on the further side of the affected valve regurgitates through the partially-closed opening (*e.g.*, from the left ventricle into the left auricle in mitral insufficiency, from the aorta into the left ventricle in aortic insufficiency), the backward wave thus coming into collision with the onward current; a similar movement takes place within aneurismal tumours of the aorta, the column of blood within the sac being thrown into commotion by each successive systolic blood-wave. All murmurs established in connection with disturbance of the circulation from organic causes, are thus primarily *murmurs pertaining to fluid;* unquestionably, however, these movements are often communicated also to the degenerated valves, and the murmurs are in this way greatly intensified.

Some anatomical alterations also, which do not present any hindrance to the transit of the blood, such as abundant and thick deposit on the inner surface of the ventricular walls, small tumours, &c., may occasionally give rise to murmurs; in this case, as in the others, they are to be referred to some commotion in the blood-stream.

Impediment to the circulation takes no share in the causation of *inorganic* murmurs; they are attributable solely to irregular, non-uniform vibration of the valves and arterial walls. *Uniform vibration yields sounds, non-uniform vibration, murmurs* (see p. 287).

Endocardial murmurs are always exactly synchronous with one or other of the two phases of the heart's movement, and are therefore either systolic or diastolic, or both, according as the embarrassment of circulation occurs only in the systole or in the diastole, or in both.



It is generally a matter of no difficulty to determine whether murmurs are systolic or diastolic, this being accomplished by noting the relation they bear to the cardiac impulse: systolic murmurs coincide with the apex-beat, diastolic murmurs come directly after it. But when the heart's action is very irregular (as is frequently the case in mitral valvular lesion when compensation begins to fail, and after the use of digitalis) or tumultuous, or so feeble that pulsation is no longer perceptible to the touch, the rhythm of the murmur can be ascertained by placing the finger on the carotid artery, the throb of which is synchronous with the impulse of the heart. Palpation of the radial artery is not to be trusted to for this purpose, as the pulse at the wrist is appreciably later than the heart's contraction. *Systolic* murmurs are further commonly distinguished from diastolic murmurs by being more *accentuated*, and usually also louder, as the force under the influence of which they originate is that supplied by the muscular contraction of the heart itself; *diastolic* murmurs, on the other hand, are generally *prolonged* and *not accentuated*.

Endocardial murmurs, both systolic and diastolic, are very variable in *character;* they are usually blowing or softly aspirated, but may also be of a rustling, sawing, scraping, grating, whistling, or singing quality. Not unfrequently a murmur presents several of these peculiarities simultaneously.

None of the properties just mentioned possesses any differential diagnostic signification; though the differences in the murmurs are *in part* due to the special form of degeneration in which the valves or the structures round the cardiac orifices are involved, all the various diseases to which these parts are subject set up the same hindrance to the circulation, and it is only the latter, the nature of the obstruction, that is the proper object of diagnosis.

The *intensity* of murmurs is as variable as their quality. Sometimes they are so faint and soft that they are recognized only after long and attentive examination and by eliminating the respiratory sounds (causing the patient to suspend respiration); at other times they are so loud as to be heard not only in the præcordial region but also over the whole anterior, and occasionally also the posterior surface of the chest. In the latter case they may be audible even to the patient himself, and the

examiner may hear them while his ear is still a short distance from the chest-wall. Such a pitch of intensity is reached only by systolic, never by diastolic murmurs.

Amongst the conditions which influence the intensity of a murmur the most important is the *energy of the heart's action*. The more violent the whirling movement of the blood-stream at the degenerated valves or contracted orifices the louder the murmur so produced. A murmur, therefore, which is scarcely audible while the heart contracts quietly and regularly, may be transformed into one of a loud and distinctly-marked character when the action of the heart is exaggerated (as by walking rapidly or raising the arms frequently), and murmurs which before had no existence may by this means be at once developed. This sudden springing into notice under excitement is particularly characteristic of diastolic murmurs at a contracted mitral orifice, these, under ordinary circumstances, often entirely escaping the ear (see p. 289). Murmurs may also frequently be intensified by pressing firmly with the stethoscope on the surface of the chest in examining.—When the muscular power of the heart diminishes, especially from fatty degeneration of the cardiac substance, at the stage in which compensation fails, murmurs become weaker and sometimes almost completely disappear.

In those cases in which the murmur has its origin in some obstacle to the free passage of the blood the heart generally contracts with greatly increased vigour, as the right or left ventricle undergoes a certain amount of hypertrophy to enable it to overcome the difficulty with which it has to contend in keeping up the circulation. Such murmurs are accordingly usually loud. Inorganic systolic murmurs, on the contrary, not being caused by obstruction to the blood-current but merely by irregular vibration of the valves and arterial walls, conditions which never lead to hypertrophy of the heart, are almost invariably weaker than those determined by organic change.

The intensity of the murmur is by no means invariably proportionate to the *gravity* or extent of the anatomical lesion to which it is due; it not unfrequently happens that on post mortem examination very slight changes are found in the valves of those who, during life, had presented a very loud murmur, and *vice versâ*. For example, the intensity of the systolic murmur resulting from degeneration of the whole extent of the mitral valve is not necessarily greater than that generated when the



valve is *still in part intact*. Only such murmurs as proceed from a constricted orifice show any correspondence in intensity with the degree of contraction present.—On the other hand, the nature of the degenerative process which has taken place in the valves and orifices occasionally, but not always, has a marked influence on the strength of the murmur; very hard excrescences excite a louder, rougher murmur than a soft deposit.—Finally, all murmurs are *louder* when the patient *stands* or *sits* than when he is recumbent; in the latter position indeed the softer murmurs are sometimes entirely suppressed.

The *duration* of a murmur is very different in different cases; in some instances it is short, in others it takes up the whole of the systole or diastole, while if both periods of the cycle be occupied by murmurs the latter may be so prolonged as to leave scarcely any appreciable pause between them.

In general organic murmurs are of considerably longer duration than the normal sounds of the heart, as the latter arise simply from the short, transient tightening of the valves, while the former are produced during the whole of the time that the whirling commotion of the blood-current lasts. This is true especially of the diastolic murmurs; the ordinary diastolic sounds are heard only at the commencement of the diastole, but the murmurs often continue to near its end; in another class of cases, however, such murmurs appear only towards the close of the diastole.

For this reason the pauses observed between the sounds of the heart have been by some subdivided, the interval which elapses between systole and diastole being designated *perisystole*, while that which comes between the diastole and the following systole is divided into two parts, of which the first is termed the *peridiastole*, the second, which shortly precedes the systole, the *presystole* (Gendrin). This classification, except for the fact that it recognises the importance of the presystole, which will be found more minutely discussed on p. 290, is of no particular practical value, as it is not on the longer or shorter *duration* of a murmur that the diagnosis of any valvular or other anatomical lesion rests, but simply on the *existence* of the murmur as the prime fact, on *the exact point in the cardiac cycle* (in the systole or in the diastole) at which it makes its appearance, and on the consecutive changes found in the heart.