AUSCULTATION OF THE HEART.

The opposite qualities, normal and pathological, of the sounds developed by the action of the heart, are known as *sounds* and *murmurs*.

The Sounds of the Heart.

In normal conditions each contraction of the heart gives rise to *two sounds*, audible over the whole præcordia, and separated from each other by a short pause. The first of these, the systolic sound, is exactly synchronous with the systole of the ventricles, while the second, the diastolic sound, corresponds with the beginning of the diastole of the heart; then follows a pause, the cardiac pause, which lasts till, with the next contraction of the heart, the same phenomena are repeated in the same order.

The sounds are not of equal intensity at all points in the præcordial region: the first sound, that associated with the cardiac systole, is more clearly accentuated at the apex of the heart and over the lower portion of the sternum, than that occurring during the diastole, the rhythm of the sounds at these parts being trochaic; the second is, on the contrary, the more accentuated of the two on the second intercostal space close to the sternum, at both its right and left margins, and the rhythm of the sounds is here iambic. The systolic sounds at the four points just indicated are exactly coincident with each other, as are also the diastolic sounds which succeed them; there are four systolic sounds, therefore, and four diastolic,—eight in all.

These four points on the surface mark, as will afterwards be shown, four centres in or near which the different sounds of the heart originate; *at the apex*, accordingly, will be heard the *sounds produced at the mitral valve, at the lower end of the sternum those connected with the tricuspid valve, in the second left intercostal space, close to the sternum, those proceeding from the pulmonary artery, and in the second right intercostal space, at the edge of the sternum, those generated in the aorta.*

If the heart be displaced downwards, from sinking of the diaphragm, to the extent of one intercostal space for instance, the parts at which its various sounds will be audible will naturally be found at a level corresponding to the abnormal situation of the organ,—one interspace lower; if it be dislocated in any other direction, to the right or left, the apex-beat constitutes a general guide to the points at which the sounds will be heard; where there is no cardiac impulse the points must be sought for at which the sounds are loudest.

The regions described do not correspond absolutely to the anatomical position of the valves of the heart and the large vessels, but it is to them that, as is proved by many physiological and pathological observations, the acoustic phenomena are most freely conducted, and there that they are loudest. This is due chiefly to the anatomical relations of the heart and lungs.

1. Sounds emanating from the *mitral* valve are not looked for directly over the valve (in the second left intercostal space, close to the sternal insertion of the third left costal cartilage), as here the latter lies behind air-containing lung-tissue, a bad conductor of sound; they are auscultated rather at the apex of the heart, which is free of lung and in immediate contact with the chest-wall, and towards which experience shows that sonorous vibrations coming from the mitral valve are transmitted with greatest intensity.

2. In the same way the sounds of the *tricuspid* valve are not to be sought for exactly over their point of origin (behind the sternum, at the level of a line drawn obliquely from the sternal insertion of the third left rib to the fifth right costo-sternal articulation), but somewhat lower down, on the lower portion of the sternum.

3. The *pulmonary sounds* are most clearly conducted to the ear placed precisely over the anatomical site of the pulmonary artery,—in the second left intercostal space, at the sternal insertion of the third left costal cartilage.

4. Sounds developed in the *aorta* are loudest, not just over the orifice of the vessel (in the second left intercostal space), but in the second *right* intercostal space, in the direction of the ascending aorta. As the aorta at its origin completely covers in the root of the pulmonary artery the sounds produced in these vessels are necessarily intermingled, and would be indistinguishable from each other were it not for the fact that those generated at the aortic valves are propagated most energetically in the direction

taken by the current of blood in the ascending aorta, along the course of the vessel towards the second right interspace; at the latter point, therefore, aortic sounds should be auscultated.

It is thus possible to define with sufficient precision the four regions in which the sounds of the heart take their rise, by observing carefully the direction in which these sounds are most perfectly conducted. That this transmission always takes place towards the parts indicated above is proved, provided that the relations of the heart to the lungs be not altered—by the *abnormal* heart-sounds, the cardiac murmurs, to be discussed further on.

It was Lænnec who laid the foundation of our present knowledge of the acoustic phenomena developed by the action of the heart; he named the sounds *normal cardiac murmurs*, in contradistinction to the *abnormal cardiac murmurs*. The term *heart-sounds*, as distinguished from *heart-murmurs*, was introduced by Skoda.

Differences in pitch in the heart-sounds are very common. Thus, the second sound at the base is frequently higher than the first, and when the heart is acting with great energy its pitch may often be accurately determined. At the apex the first sound is most usually deeper and duller than at the lower part of the sternum, where it is sometimes exceedingly loud and clear; this is obviously to be explained by the facility with which sound is conducted through the solid tissue of the sternum. The sounds also present great variety in timbre, being sometimes clear and ringing, at other times dull and without proper musical character; even in the same person this timbre may change rapidly with the varying force of the heart's contractions.

The *rhythm* of the sounds at the apex and over the lower portion of the sternum (that is of the sounds of the left and right ventricles) is not invariably trochaic, nor is it always iambic at the roots of the great vessels; often it is the reverse of this, trochaic over the vessels and iambic at the apex, or in both situations iambic, or in both trochaic. These differences depend on the occasional *predominance of the first sound*, when the rhythm is at all points trochaic, or *of the second sound*, when the rhythm becomes iambic. These modifications have no particular significance.

If the action of the heart become irregular from any cause the rhythm of its sounds is also lost; in the severer forms of irregularity, indeed, it is a matter of some difficulty to distinguish between the systolic and diastolic sounds.

Physical Cause of the Heart's Sounds.

The *first* sound arises from the *tension of the auriculo-ventricular valves*, due to the shock of the mass of blood thrown against them during the cardiac systole; the *second* sound is caused by the *tension of the semilunar valves of the aorta and pulmonary artery* at the instant that the blood projected into the vessels is driven backward by the elastic recoil of the arterial walls.

During the period of rest the auriculo-ventricular valves hang loosely down into the ventricular cavities, but are floated upwards when the latter are filled with blood and belly out in the direction of the auricles, shutting off the lower from the upper chambers of the heart; the short auricular systole, which precedes by an instant that of the ventricles, renders them somewhat tense, but to such a slight degree as to give rise to no audible sound. Sound is produced only by the more marked tension which results from the systole of the ventricles, and which is caused partly by the pressure of the blood against the valves, and partly by the contraction of the papillary muscles connected with the valves by the chordæ tendineæ.

The theory above stated, first propounded by Rouanet (1832), is supported by many physical analogies and pathological observations. Every elastic membrane (like a cord of cat-gut, for instance) emits a sound on being suddenly brought from a state of relaxation into one of extreme tension; the sudden tension of such membranes, therefore, as the cardiac valves, may naturally be supposed to be attended by similar phenomena. Experimentally, also, a feeble, dull tone may actually be obtained from a tense mitral valve by directing against it a forcible stream of water either from the apex of the heart (O. Bayer) or through the aorta, the semilunar valves of the latter being previously removed (Landois).

The mitral and tricuspid valves must also give out sound, each independently of the other, but both at the same moment, both ventricles contracting simultaneously.

Further proof of the accuracy of Rouanet's theory is furnished by certain pathological conditions. Thus, if the mitral valve lose its smoothness of surface and delicacy of structure from pathological deposits of any kind, from retraction or inflammatory adhesion of the tips of the valvular segments, &c., in such a way that it becomes totally or partially incapable of being put thoroughly on the stretch, the systolic sound at the apex of the heart undergoes change, or disappears completely and is replaced by a murmur. Over the lower third of the sternum, however, the first sound remains perfectly pure and clear so long as

the tricuspid valve retains its smoothness,—which it usually does.

On the other hand, in those rare cases in which the tricuspid valve has been deprived of its elasticity, the systolic sound over the lower portion of the sternum is superseded by a murmur, while at the apex it is still distinctly heard provided that the mitral valve be intact.—

But tension of the auriculo-ventricular valves, though the principal, is not the only cause of the first sound of the heart; this depends to a certain extent also on the *muscular contraction* of the ventricles. Every large muscle, when contracting vigorously, especially under electric stimulation, generates sound (Wollaston), usually of a quality so distinctly musical that its pitch may be determined with precision (Helmholtz). This takes place not only in all striped muscles, but also in the heart. The most weighty evidence in favour of this view, however, is presented in the experimental fact that the heart, removed from the body and emptied of blood, emits a distinct sound at each contraction (Ludwig and Dogiel), although under these conditions the tension of the auriculo-ventricular valves is reduced to a minimum.

And further, in the domain of the pathology of the heart many phenomena are observed which tend to support the doctrine that muscular contraction plays a part in the production of the first sound. Thus the systolic sound is found not to be invariably abolished by the presence of even very marked and advanced changes in the mitral valve; and, on the other hand, it may become indistinct and feeble, even though the mitral valve preserve its normal smoothness and general structure, if the *muscular substance* of the heart be affected, as in myocarditis, fatty degeneration, and other diseases which diminish the contractile power of the organ.—There are also other circumstances which lend countenance to the theory that the action of the cardiac muscle is concerned in the generation of the first sound,—the facts that in certain conditions the duration of this sound is longer than can be satisfactorily accounted for by the transient tension of the auriculo-ventricular valves, and that a simple state of tension of the mitral valve, produced artificially (by water-pressure) in the heart extracted from the body, gives rise to a sound having no resemblance to that heard in the living subject. Nevertheless,

the part taken by muscular contraction in the development of the systolic sound of the heart is but a very subordinate one: it must not be inferred,—solely on the experimental ground that the heart even when empty gives a sound in some respects similar to the normal first sound in the living subject,—that muscular contraction is the essential factor in the causation of the sounds and the tension of the valves but a secondary element, as such a conclusion is plainly at variance with the fundamental phenomena presented in cases of cardiac valvular lesion.

It is now no longer necessary to discuss or refute in detail the numerous theories that formerly prevailed regarding the cause of the first sound of the heart; the only question that calls for particular notice is concerning the greater or less share taken by muscular contraction in the production of this sound. Till comparatively lately the sudden tension of the auriculo-ventricular valves was almost universally accepted as the sole cause of the systolic sound, the arguments advanced by certain of the earlier authors (Lænnec, Williams, Hope, &c.,) to show that the cardiac sounds are simply muscular, meeting with but partial acceptance; recently, however, Ludwig and Dogiel, basing their views on experimental research, have again ascribed to the cardiac muscle the principal rôle.

I have repeated Ludwig and Dogiel's experiments, and in doing so pursued the following method: the circulation was controlled by opening the thorax (artificial respiration being carried on) and enclosing the great vessels entering and leaving the heart in a ligature, in such a way that the current of blood could be arrested or restored at pleasure. Like Ludwig and Dogiel I found that the empty heart, at each contraction, certainly emits a sound which, as might have been expected from the feebleness of the heart's action in such circumstances, is of far less intensity than the normal heart-sound; I have to state positively, however, that *this sound differs essentially in character from that of the normal heart, it is duller, is toneless* (*without timbre*), and wants also the flapping quality which distinguishes the ordinary first sound.—Another source of error in these investigations remains to be pointed out, namely, that in the empty heart also the musculæ papillares continue to contract, and that therefore a certain, though probably slight amount of tension of the auriculo-ventricular valves is brought about. There is thus the possibility to be borne in mind, that this valvular tension may be sufficient to give rise to a sound which, though feeble, goes to diminish still further the relative importance of muscular contraction in the causation of the first sound.—The only really satisfactory proof that the contraction of the cardiac muscle has any claim to be regarded as an influential factor in the origination of the first sound would be the production of evidence to the effect that the sound is heard even after total *destruction* of the auriculo-ventricular valves; it seems impossible, however, to perform such an

operation on the living animal without at the same time setting up such grave disturbance as would inevitably vitiate the result of the observation.

O. Bayer adopts the theory that the sound is muscular, adducing certain clinical and pathological facts in support of that view. Thus, he states that very frequently in severe acute and chronic diseases he has noted various changes in the first sound, especially enfeeblement and diminution in its distinctness, while, as was demonstrated by *post mortem* examination, not the slightest trace of valvular affection existed; there was invariably present, on the other hand, a more or less considerable degree of alteration of the muscular structure of the heart, (particularly fatty degeneration of the primitive bundles and albuminous infiltration) occasionally plainly visible to the naked eye, at other times to be detected only with the aid of the microscope.

In all these cases a much less forced conclusion appears to me to be indicated, namely, that the modifications of the cardiac sounds referred to are due rather to *non-uniformity in the vibrations of the valves* (see p. 287). There are also on record various instances in which, notwithstanding the fact that the cardiac muscular tissue was the seat of extensive degeneration, and thus quite incapable of originating any sound, the sounds of the heart were normal during life, and could be caused only by tension of the unaffected auriculo-ventricular valves (Bamberger).

It is further often asserted, in favour of the muscular theory, that mitral lesions grave enough to render the valve absolutely *insufficient* do not always cause the first sound of the heart to disappear; in such conditions it is only masked by the systolic murmur, through which it can always still be heard on careful auscultation.—I find, as the result of my own personal experience in nearly 200 cases of mitral insufficiency, that in these circumstances the systolic sound very rarely continues to be audible; and I would also submit that even when it does persist, notwithstanding the existence of a systolic murmur, we have in this fact no absolute proof of its muscular origin. In the first place, it is never so loud as that heard over the right ventricle, (the tricuspid valve being intact); and further, the mitral valve may, if there still remain certain portions of its tissue not invaded by the degenerative process, preserve its power to vibrate at those parts, and may in this way be enabled to emit a sound; or a systolic sound may be conducted from the right ventricle to the apex of the heart, a consideration which would explain the persistence of the sound along with a systolic murmur even in those cases in which the mitral valve is degenerated in its whole extent.

There still remains to be mentioned another phenomenon, observed in the domain of cardiac pathology, which admits of no possible explanation on the supposition that the first sound is muscular, and which is compatible only with the valvular theory,—the *absence* of the systolic sound at the apex when the aortic valves are to a high degree incompetent. Were the first sound due to muscular contraction it should still be heard in such cases, or should even be louder than usual,

inasmuch as the insufficiency of the aortic valves leads to hypertrophy of the left ventricle. Now in hypertrophy of the left ventricle from any other cause, as from disease of the kidneys or sclerosis of the aortic system, the first sound is clearly marked, frequently intensified; it is unquestionable that such enlargement of the heart not only does not render its sounds weaker, but actually renders them *stronger*,—though some have indeed held, on the contrary, that a hypertrophied ventricle is less capable of producing a sound than one of strictly normal development. The absence of the first sound in aortic insufficiency, however, is, as Traube has shown, readily explained by the valvular theory of the origin of the sounds. Thus, during the diastole the auriculo-ventricular valve, the mitral for example, is in a state of relaxation; at the end of the diastole it is put slightly on the stretch by the contraction of the left auricle, as has been already stated on p. 263. This slight degree of presystolic tension (*initial tension*) gives no sound; an audible sound is produced only by the firmer tension which results from the systole of the ventricle (*final tension*). The greater the difference between the initial and the final tension of the auriculo-ventricular valves the louder will be the sound, just as in any other membrane which, previously slack, is *suddenly* and violently tightened. If, on the other hand, the difference between initial and final tension become less, the vibrations of the valve become less ample and the sound consequently more feeble; and if the difference be diminished still further, absolutely no sound is heard. Diminution of the difference between the initial and final tension of the mitral valves of precisely the nature described, is observed in cases of aortic insufficiency: the initial tension increases, as at the end of the diastole of the left ventricle the blood rushing back from the aorta presses on the valvular segments, while the final tension is lessened, as in this affection the aorta, into which the left ventricle projects its contents, soon loses much of its normal elasticity and tension.

Some have endeavoured to explain the absence of the first sound in aortic insufficiency as owing to fatty degeneration of the muscular substance of the heart; but this view is negatived by the fact that it is not only in the later stages of the valvular affection, when the contractile power of the heart begins to fail, but also in its earlier stages, when the hypertrophy of the ventricle is *fully* compensatory for the valvular defect and the action of the heart regular and powerful, that the systolic sound is wanting. Moreover, in cases of genuine fatty heart, in which the degeneration of the primitive muscular bundles is unquestionably much more pronounced than when the structural alteration is consequent on aortic lesion, the first sound is generally clear, though possibly feeble.—Other less weighty objections to the valvular theory may be passed over without further discussion.—Although the whole question in dispute cannot yet be said to be definitely settled, the conclusion to which the present state of our knowledge seems to point is that *the first sound is essentially of valvular origin and only to a slight extent muscular.*

Cause of the Second Sound of the Heart.

That the second sound of the heart is produced, not within the chambers of the organ, but at the semilunar valves of the aorta and pulmonary artery by the recoil of the column of blood against these structures, and that it is only conducted towards the cavities of the heart, is placed beyond all doubt by numerous observations, experimental as well as pathological.

The second sound disappears totally on cutting off the flow of blood to the heart in living animals. This may be effected, (artificial respiration being kept up) by passing a ligature round the venæ cavæ close to the point at which they enter the right auricle; on lifting and thus tightening the cord circulation is stopped, on slackening the cord it goes on as before. Raising the ligature abolishes the sound completely, lowering the ligature restores it at once; but the first sound is found to persist, notwithstanding the arrest of the circulation, as the heart continues to contract as usual and the auriculo-ventricular valves are regularly thrown into a state of tension. Over the heart, also, removed from the body and still pulsating, only the first sound is heard, not the second; after the heart has ceased to contract, however, a second sound may be produced at the aortic valves by closing them abruptly by injecting a stream of water into the vessel with sufficient force. The greater the pressure exercised by this body of water the louder the sound obtained.

The ordinary physiological and pathological phenomena which pass under our notice every day are strongly confirmatory of the above conclusions. Thus, it is observed that the second sound is much feebler over the ventricles than opposite the arterial orifices, and it is only at the latter situation that it possesses its distinctive flapping character. While auscultating, if the stethoscope be slowly moved along the surface from the region of the apex towards that corresponding with the orifices of the great vessels the gradual intensification of the second sound may often be clearly traced.

But by far the strongest evidence that the second sound of the heart is generated at the semilunar valves is furnished by the following pathological observations. When, from hypertrophy of the *left* ventricle, the blood is thrown into the aorta with unusual

force the second sound becomes abnormally loud, in consequence of the more energetic recoil of the mass of blood against the aortic valves. When, on the other hand, the quantity of blood which enters the aortic system on each contraction of the heart is small, as in mitral insufficiency, and more especially in mitral contraction, the diastolic recoil of the blood against the aortic valves is less forcible and the second sound becomes much fainter, or occasionally even quite inaudible, over the left ventricle. This occurs also when the contractile power of the heart is from any cause lowered, as in cases of fatty degeneration, the general failure of strength which precedes death, and in the asphyxial stage of cholera. (In the last-mentioned affection there is sometimes not the slightest indication of a second sound at the apex, while it is still perceptible, though faintly, at the great arterial orifices.) Further, when the aortic valves are so altered in structure as to be no longer competent to perform their office efficiently, when therefore there is no second sound but a distinct murmur at the root of the aorta, the diastolic sound over the left ventricle is also wanting, nothing whatever being heard after the systolic sound, or only the feeble diastolic murmur conducted from the aortic valves. And finally, in cases of reduplication of the second aortic sound, the second sound over the left ventricle is also double, but weaker than over the aorta.

It can be shown equally clearly that the second sound heard over the *right* ventricle is transmitted from the semilunar valves at the origin of the pulmonary artery. If, as the result of hypertrophy of the right ventricle, the blood be driven into this vessel with greater force than usual it is thrown back with proportionately greater force against the semilunar valves during the diastolic period, and the second pulmonary sound is consequently intensified; over the right ventricle also the same sound is then considerably louder. If, on the other hand, the pulmonary valves become insufficient from degeneration,—a somewhat rare occurrence,—no second sound, but a diastolic murmur, is produced, and over the right ventricle also there is no second sound, but the feeble diastolic murmur conducted from the pulmonary valves. And finally, when the second pulmonary sound is double at its point of origin it has the same character over the right ventricle.

Cause of the First Arterial Sound.

The first arterial sound, which is heard in the second right and second left intercostal spaces, close to the sternal articulations of the third pair of ribs, and which is synchronous with the first ventricular sound, is to some extent to be regarded simply as the ventricular sound propagated towards the periphery; it *takes its origin chiefly, however, at the orifices of the aorta and pulmonary artery, and is due to the tension and sudden expansion of the arterial walls which take place when the blood is discharged into the vessels by the contraction of the heart.* Many pathological observations go to prove this. Thus, if the coats of the first part of the aorta undergo such changes (as from atheromatous degeneration) as deprive them of their property of uniform expansibility and of their power of entering into vibration, the arterial sound under consideration is not developed; instead of it is heard, at the aortic orifice, a systolic murmur, while the first ventricular sound remains unaffected. Similarly, the first sound at the pulmonary orifice disappears, and is replaced by a murmur, when the width of the opening is diminished or the inner surface of the vessel roughened by disease.—The first sound at the arterial orifices also continues audible when over the ventricles, generally over the left, the first sound is abolished or is represented by a systolic murmur.

But it is not merely in the central portions of the vessels springing from the heart that this systolic sound, arising from distension of their walls by the advancing blood-wave, is observed; it occurs also in their larger subdivisions, in the carotid and subclavian, and even, under certain pathological conditions, in still smaller arteries, at a greater distance from the heart. These sounds will be treated of when discussing the auscultation of the arteries.

It will thus be seen that *of the eight sounds* heard at the above-described four points in the præcordial region *to six only is a proper point of origination assignable: at each of the auriculo-ventricular valves one (systolic) sound is produced; in the aorta and pulmonary artery arise two sounds, one systolic and one diastolic;* the second ventricular sound is merely the transmitted second arterial sound.

These views regarding the parts at which the heart-sounds are developed, first announced by Rouanet and accepted (with some modification only with respect to the first arterial sound) by Skoda, have been almost universally adopted.

The diagnostic interpretation of the presence of the normal cardiac sounds is simply this,—that all the valves are faithfully executing their normal functions, and that therefore there is no obstacle to circulation within the heart itself. But the satisfactory performance of their functions does not necessarily imply the absolute anatomical integrity of the valves themselves; slight changes may nevertheless exist, and these are not unfrequently found on examining the bodies of individuals in whom the cardiac sounds during life were perfectly clear.

Physiological and Pathological Variations in the Character of the Heart-Sounds.

The sounds of the heart naturally present so much diversity of character, and pass from the domain of health to that of disease by stages so insensible, that it is frequently only by calling to our aid such other signs as are furnished by an exhaustive examination of the organs of circulation, that it can be determined whether or not these changes in the sounds are really due to anatomical lesion of the heart itself. These alterations affect the *intensity*, the *purity*, and the *timbre* of the sounds; *reduplication* or *division* of the sounds comes under the same category.

The Intensity of the Heart-Sounds

is very different in different individuals, even in the total absence of any excitement of the heart's action. The sounds are somewhat louder in the erect than in the recumbent posture, and are still more accentuated when decubitus is on the left side; they also acquire greater intensity during expiration and in the respiratory pause, as in inspiration the margins of the lungs creep forward and cover the heart and so prevent the free conduction of its sounds to the surface. The strength of the cardiac sounds is also considerably modified by the thickness of the thoracic parietes; other things being equal the sounds are therefore louder in children and in those who are emaciated than in persons