A lecture on the diagnosis of disease of the heart / by George W. Balfour, M.D.

A LECTURE

ON

THE DIAGNOSIS

OF

DISEASE OF THE HEART.

BY

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A LECTURE

ON

THE DIAGNOSIS OF CARDIAC DISEASE

GENERALLY,

WITH SPECIAL REFERENCE TO THE VALUE OF THE INFORMATION DERIVABLE FROM

THE SYMPTOMS AND THE PHYSICAL SIGNS.

GENTLEMEN,—In gathering together the prominent facts in relation to the circulatory system in any case, few things are more striking than the frequently apparent irrelevance of the symptoms in those who are otherwise ascertained to be examples of serious cardiac disease, if we perhaps except the equally remarkable fact, that cardiac symptoms are frequently complained of when no cardiac disease exists. Thus, should a patient come to you complaining of palpitation, irregular action, or of his heart generally, you may at once assure him, without much fear of being wrong, that his heart is all right, that he is only weak, nervous, and probably dyspeptic. Almost the only exceptions to this rule are old hospital patients, who, from having been so often lectured over, are almost as well acquainted with their own special lesion as an average practitioner.

Of course, if pain be a predominant symptom, that is usually referred to the region of the heart, but not always to the organ itself; and we continually meet with cases in which serious valvular lesion has existed for many years altogether unnoticed by the patient, even although he may have been leading an active and laborious life. Such a lesion is said to be mute; that is, it has been perfectly compensated, and it never asserts itself as a disease until that compensation has been ruptured by accident, or by the gradual advance of those organic changes which are inseparable from it. In the one case the ruptured compensation is reparable, and comparative health may be restored; in the other, it is irreparable, though judicious treatment may prolong life and postpone the inevitable end.

A truly cardiac patient—one suffering from actual disease of the

heart—as a rule comes to you complaining, not of that organ, but of one or other of the secondary results of his lesion. He complains of breathlessness or of dropsy, either or both of which may result from that lesion if it be uncompensated, or if the compensation be ruptured.

If the patient complain of shortness of breath, as is often the case, you will find that this cardiac breathlessness presents certain distinctive features wherein it differs from pulmonary breathlessness, the most striking of these being the perfect tranquillity of the breathing while the patient is at rest, at the same time that any exertion at once produces so anxious a desire for more air as can be expressed by no fitter term than the *air-hunger* of the Germans. The amount of lesion is not to be measured by this breathlessness, but its seriousness, as dependent upon the degree in which the compensation is ruptured, may certainly be so. The patient may only puff considerably in going up a hill or ascending a stair, or his shortness of breath may be so great as speedily to compel him to call a halt on attempting either of these feats; or it may be so extreme as to prove distressing on making such perfectly trifling exertions as merely sitting up or turning in bed. At the same time there is no true dyspnœa, or difficult breathing properly so called; there is no obstruction either to inspiration or expiration; there may even be no curtailment of the air-space in the lungs from any cause whatever; the breathing while the patient is at rest is perfectly quiet and natural: yet such is the difficulty—from cardiac causes—of getting the blood aerated, that the slightest exertion produces such a gasping inquietude as is extremely characteristic. This is one form of cardiac asthma, as it is termed; now and then we have another, in which the breathlessness, though not dependent upon exertion, is yet equally independent of pulmonary lesion. In this case the patient wakes gasping and alarmed from his first sleep; he has palpitation, occasionally pain (angina), almost always irregular action of the heart, which is always feeble; now and then the patient is sick, and sometimes vomits a mouthful or two. This form of cardiac asthma is mostly senile in character, and associated with muscular degeneration rather than with valvular lesion. It frequently arises from some slight gastric derangement, which reflexly affects the enfeebled heart in an injurious manner; and it is often the beginning of the end to those affected—the first intimation that the "pitcher is broken at the fountain," and that death has already seized the very citadel of life.

Such patients, however, never come to you—you are always sent for to see them; and I have only mentioned this affection now to illustrate the fact, that exertion is not always necessary to produce cardiac breathlessness, and that even in this case the panting is characteristic, while the absence of pulmonary lesion marks its cardiac origin.

To produce so-called cardiac breathlessness, however, it is not

necessary to have actual cardiac disease. Breathlessness depends upon imperfect aeration of the blood, and in the absence of pulmonary lesion may depend upon lesion of the heart or of the blood itself. Even though a patient, then, presents all the characteristic symptoms of cardiac asthma, we must not therefore set him down as certainly labouring under cardiac disease: he may be only anæmic. But inasmuch as anæmia and cardiac disease frequently coexist, the assured presence of the former, evinced by the bloodless condition of the lips, gums, etc., does not exclude the latter. The presence of a breathlessness having the characteristic symptoms described, makes us certain that we have to do with a hæmic or a cardiac lesion; which it is, we must determine by further inquiries.

Dropsy depending upon cardiac disease always[1] begins across the instep, and gradually fills up the lower extremities, the face and upper parts of the body remaining free. But inasmuch as the œdema of simple debility commences in the same position and pursues the same course, such a dropsy cannot be accepted as certainly indicative of the existence of cardiac disease, but must only be received as a hint that possibly the heart may be affected.

When a patient, then, who breathes easily when at rest complains of breathlessness on exertion, or of swelling of his feet, with or even without marked breathlessness, we suspect the heart to be the organ at fault, and we proceed to confirm or to set aside that suspicion by further investigation.

In further examining the condition of our patient, we first feel both radial pulses simultaneously, noting whether the arteries are firmer or more tortuous than usual (atheroma). If there be a marked difference between the two radial arteries, we feel both brachials simultaneously; if these be equal, the difference between the two radials is due to irregular distribution. If the brachials differ, in all probability there is some abnormal physical cause to account for it—possibly an aneurism, the mode of detecting which we shall afterwards describe. Should the radial pulses be equal and regular, but small and feeble, we elevate the wrist to a level with the head, if the patient be standing or sitting; if lying, we elevate the arm to its full length perpendicularly to the body. Should the pulse then become extinguished, or nearly so, the patient is anæmic, and possibly anæmia is his sole disease; but we must never under any circumstances rely upon one symptom, however apparently trustworthy, but merely note it as an aid and a guide in our further investigation. Should the pulse, after elevation of the arm, remain still small and feeble, but distinct, the cardiac disease, if present, is mitral. Irregularity of the pulse confirms this suspicion; extreme irregularity points to the probability of the affection of the mitral valve being constriction rather than dilatation. Should the small, feeble, and possibly irregular

[1] "Almost invariably," says Walshe, any exception being excessively rare. Diseases of the Heart, p. 302: 1862.

pulse remain not only distinct after the elevation of the arm, but become more so, the systolic impulse being followed by such a sudden and complete collapse as to render the impulse apparently more marked, then we have to do with a double lesion, a mitral and also an aortic regurgitation. This form of pulse is, however, not always well marked, in many cases is not easy of detection, and is therefore not to be relied upon, unless the collapse is distinct. In simple aortic regurgitation, however, the peculiar sensation conveyed to the finger, and well known by the terms hammer or Corrigan's pulse,[1] is usually well marked, and frequently so greatly increased by elevation of the arm as to become almost painful, and wholly unmistakable.

We see, then, that while certain general symptoms indicate with greater or less probability the existence of cardiac disease, the examination of the pulse alone may not only confer more or less certainty on the suspicions thus aroused, but may even enable us in some degree to predicate the nature of the lesion.

We next examine the state of the patient by the INSPECTION of his thorax and neck, noting first whether there is any undue pulsation in the carotid arteries, or in the tracheal fossa; whether the veins are enlarged, and whether they pulsate or not. And to determine this in anæmic patients is sometimes a matter of some nicety, especially if they be young, as the veins are then small, and apt to be hidden by the subcutaneous fat, which is always more abundant than in older patients. When the individual is lying flat, a mere flicker at the root of the jugular is of no importance, as it is found in most healthy people. A simple undulation in the jugular is a sign of considerable congestion of the right auricle, with propagation through it of the systolic impulse of the ventricle, the valves at the root of the jugular remaining intact. But when we have distinct systolic pulsation propagated into the jugular veins, usually most evidently into the right one, and made most plain by pressing up the blood in the vein to the middle of the neck with the finger, when the vein is seen to fill from below by regular pulsations synchronous with those of the heart, then we know that we have to do with dilatation of the right side of the heart of some standing, which has rendered incompetent the tricuspid valve as well as the venous valves at the root of the neck, and must therefore have interfered injuriously with the systemic circulation.

Visible venous pulsation is therefore invariably a sign of considerable dilatation, with or without hypertrophy of the right ventricle, and its distinctness may be accepted as a measure of the persistence and degree of that dilatation; but visible arterial pulsation is occasionally found, especially in the peripheral vessels, in

[1] *Vide* Edinburgh Medical and Surgical Journal, vol. xxxvii., for April 1832, p. 227 and p. 229, where the phenomena referred to are described for the first time by Corrigan.

the normal condition, and is still more marked when these vessels are atheromatous and tortuous, as they frequently are in advanced age; but if we confine our inspection simply to the carotids, the tracheal fossa, and the brachials, and if we find well-marked, excessive, and symmetrical pulsation there, in the erect or semi-erect position, this will invariably be found associated with regurgitation through the aortic valves, and with coexistent dilatation and considerable hypertrophy of the left side.

Inspection of the chest in its normal condition reveals for the most part a perfectly symmetrical state of the thoracic walls, on both sides of the sternum. These walls gently rise and fall rhythmically with the in- and expiration, the only thing breaking the monotony of this gentle undulation being the tap of the cardiac apex—averaging four taps to each respiratory wave—which is visible between the fifth and sixth ribs, about two inches from the left edge of the sternum.[1] Any deviation from these appearances is abnormal, though possibly not of much import. Thus, in many cases, the precordial region is more prominent than the similar region to the right of the sternum. Frequently this is of no consequence, yet it may be associated with enlargement of the heart, or with pericardiac effusion if the intercostal spaces be effaced. Of itself, it is a sign of little importance; and in ascertaining its presence, we must be careful not to be misled by any rachitic bulgings of the ribs, or even by the more than usually distinct pulsations of the heart in children or in meagre individuals, which may apparently simulate a bulging. This latter simulation is readily corrected by more careful inspection, supplemented by palpation and measurement. In the former, the spinal column is either found to be curved, or, at all events, the costal arch is deformed and depressed posteriorly when it bulges in front. Bulgings dependent upon arterial aneurisms are almost invariably above the third rib, and appear as mere local tumours. Depression of the precordial region is much more rare than its elevation, and is the result of previous pericarditis, and the indication of adhesion of the visceral and parietal portions of the pericardium. We must distinguish between a permanent and general depression of the cardiac region and those rhythmical depressions of the intercostal spaces which occur over the apex, or even over a more extended portion of the heart's surface, which are often the result of adhesions of the pericardium, not only to the heart, but also to the pleura, and through that to the walls of the chest, but which are sometimes, especially in thin-walled chests, the simple result of atmospheric pressure depressing the intercostal spaces at the moment of the cardiac contraction, where no adhesions exist; this form of rhythmical depression being invariably associated with some degree of enlargement—not always hypertrophy—of the heart and consequent displacement of the lung.

[1] Those who have worked much with female patients will appreciate the discarding of the nipple as a fixed point.

Alterations in the position and extent of the apex beat are also readily appreciable by the eye. These must be noted accordingly, to be afterwards more fully investigated by palpation. Pulsation is also frequently seen in the epigastric region. Sometimes this is associated with absence of the apex beat from its usual position, and is to be regarded as one form of its displacement—a displacement which may be brought about in various ways, the commonest of these being dilatation of the right ventricle, by which the left ventricle is pushed backwards, the right one communicating its impulse to the lower part of the sternum and to the liver, which is then seen to pulsate in the scrobiculus cordis. It may be doubted if such pulsations are ever visible in a perfectly normal condition of the heart and neighbouring organs. Assuredly, they are often seen where no actual cardiac disease exists; simple dilatation of the right ventricle is invariably more or less present when pulmonary congestion exists, even from such simple and temporary causes as strenuous exertion or bronchial catarrh; and whenever dilatation of the right side exists to any considerable extent, epigastric pulsation may be seen. It is rendered more perceptible by any cause which may effectively conduce to the transmission of such impulse to the abdominal walls. Thus it is sometimes favoured by the existence of effusion in the pericardium, but especially by the occurrence of enlargement of the liver, which is so frequent a concomitant of dilatation of the right ventricle. Indeed, in such circumstances, the systolic impulse of the venous regurgitation itself is occasionally so great as to induce actual pulsation of the whole liver, a pulsation which is then visible, not merely in the scrobiculus cordis, but throughout the whole right hypochondriac region;[1] an extent of hepatic pulsation which is not, however, always distinctive of great venous regurgitation, as it is occasionally seen as the result of the impulse communicated by a large aneurism lying immediately above the liver.[2] There are other pulsations in

[1] This form of hepatic pulsation was first described by Allan Burns, in his Observations on some of the most Frequent and Important Diseases of the Heart, Edin. 1833. At p. 265, he quotes a case from Senac, in which direct pulsation was communicated to the epigastrium by a vena cava inferior, the size of a man's arm. At p. 266, he also mentions a case in which he states that epigastric pulsation was produced by repercussion from solidified lungs.

[2] Extract of a letter from a late physician labouring under aneurism of the thoracic aorta projecting through the sternum:—"I am satisfied that your view of the origin of the liver pulsations from the aneurism mainly is the correct one, however discouraging to myself that may be. But I have this to say, on the more cheerful side, that if an *enlarged* liver has had nothing to do with them (as Dr —— says), and which by decreasing in bulk has caused them to be less felt, the very great decrease in them which has occurred since I began the iodide, is very favourable to the conclusion, that under its action the sac must have contracted considerably. Early in February, before I had reason to suspect any increase of the liver, the pulsations had the effect of expanding the opposite sides of the hypochondria at each beat, an effect which is not now perceptible, though I am supposing the liver to be larger, in which, however, I may be mistaken." Dated 3d May 1870.

the epigastric region not depending upon cardiac impulse, such as the ordinary pulsation of the abdominal aorta, which occasionally becomes visible through the great emaciation of those previously of full habit, or which may become visible by their more energetic transmission to the abdominal surface by some overlying tumour. Now and then this pulsation is actually aneurismal in character: much more frequently it is simply neurotic, and yet limited to the abdominal aorta. Under some of these circumstances we can readily understand that the pulsation extends downwards along the course of the aorta; under others, as when propagated upwards by a solid tumour, it is obvious that it may not only be limited in its longitudinal propagation, but may even extend transversely. Inspection directs the attention to these pulsations indicating to an experienced eye their probable nature, which can only be accurately determined by other means of exploration. In rare instances, a pulsatory movement is also communicated to the epigastrium through the movement of the heart's apex during the ventricular systole, pulling upwards an adherent pericardium, diaphragm, and liver.[1] This movement is, of course, exactly the reverse of that in ordinary epigastric pulsation. As this extensive adhesion is, as a rule, only the result of a severe and extended inflammation, affecting uniformly the whole surface of the heart, we can readily understand how, in such circumstances, a universal undulatory movement may be perceived, in which, when the heart's action is at all rapid (over 90 beats per minute), it may be difficult to say what parts of the motion are systolic and what are diastolic. In these circumstances, the variations in time between the movements of any two parts are readily rendered visible by attaching to each, by means of a pellet of beeswax, a bristle carrying a small paper flag; and when the pulse is over 90, this is the only way in which such differences can be ascertained with any certainty; and it is a means of attaining certainty of diagnosis often of much importance, especially when we have pulsations visible above the fourth rib, which may possibly be either aneurisms of the aorta or pulsations of the auricle. A comparison of the movements of a flag on the doubtful point with one over the apex, will settle the matter at once, as the beat of the auricles always precedes that of the ventricle, while, however near the heart an aortic aneurism may be, its pulsations can never precede those of the ventricles, but must succeed them by an interval which is more or less appreciable. The determination of this fact, we shall afterwards see, may be a matter of some importance in the diagnosis of cardiac lesions.

Palpation confirms the information obtained by inspection, and adds somewhat to it. On placing the hand over the cardiac area, in many people with thin chest walls, we can distinctly perceive the alternate movements of auricular and ventricular systole, with each

[1] A remarkable instance of this will be found detailed in a note on p. 214 of Copland's Dictionary of Practical Medicine, vol. ii.

corresponding diastole; and, of course, we can also readily appreciate any pathological change in their movements. Great increase in their force indicates hypertrophy; but a diminution of that force is by no means to be regarded as a certain indication of atrophy, dilatation without hypertrophy, or even of cardiac debility from any cause,—though it may be a sign of one or other of these phenomena, as well as of great pericardiac effusion, pulmonary emphysema, or even simple thickening of the thoracic walls. In many of these cases, especially in pericardiac effusion, but more or less in all, the cardiac shock may be rendered perceptible to the hand, by causing the patient to sit up and lean well forwards. In this case, the readiness with which it can be appreciated, and the position in which it is felt, must be carefully noted. Forcible pulsation above the fourth rib, and within the cardiac area, may possibly be aneurismal, but most commonly depends upon dilatation and hypertrophy of the appendix of the left auricle. Forcible pulsation chiefly to the left of the cardiac area below the fourth rib, with depression of the apex beat, indicates dilatation and hypertrophy of the left ventricle; while pulsation beneath the lower part of the sternum, with disappearance of the apex beat, reveals dilatation with or without hypertrophy of the right ventricle, the extent of dilatation being to some extent measurable by the amount of epigastric pulsation, the degree of hypertrophy being denoted by the force of the pulsation. When the whole heart is hypertrophied and dilated, a more or less violent shock may be felt over the whole of the cardiac area; and where the hypertrophy is great, the shock may be double—the first forcible and systolic, the second less forcible and diastolic,—the result of the rebound of the enlarged heart from the posterior thoracic walls.

Displacement of the apex beat may be produced in the normal state by inclining the patient to the left side, when it may pass to the left a little farther even than a point equidistant between its normal position and the axillary line; and by inclining the patient to the right, the apex beat becomes faint or disappears, the right side of the heart being then felt to give a distinct pulsation in the epigastrium. In pericardiac effusion, the apex beat may be displaced upwards as far as the fourth interspace, and slightly outwards to the left. In pleural effusion on the left side, it may be turned quite round the other way into a similar position on the right side, and pleuritic effusion on the right side may displace it towards the left. In hypertrophy, with dilatation of the left ventricle, the apex beat passes downwards and to the left. In a similar condition of the right ventricle, it may entirely disappear from its normal position, a diffuse impulse at the lower end of the sternum revealing the cause.

When the left lung is retracted from the base of the heart from any cause, the pulsation of the pulmonary artery between the second and third ribs, close to the sternum, may be distinctly felt, and even the click of the semilunar valves perceived, because sounds consist of vibrations sufficiently rapid to become appreciated

by the ear, and can always be both felt and seen if the propagating medium be suitable. Hence friction, the result of the rubbing together of the two layers of the pericardium when roughened by lymph, is easily perceived by the hand, and even the vibration of valvular murmurs are readily enough appreciated. When rough, these vibrations have received the name of the purring thrill—*fremissement cataire*—because the sensation felt resembles that perceived on placing the hand on the body of a cat in the act of purring. Abnormal pulsations along the course of the thoracic aorta are usually confined to the ascending and transverse portions, and may be the result of simple displacement of the vessel, as occasionally happens in rickety chests, or they may be the result of aneurismal dilatations, and must be sought for in the tracheal fossa, as well as between the ribs along the course of the aorta, especially during expiration, when such pulsations, if faint, are most readily felt.

From all this we see that a very great amount of information may be obtained from palpation, the whole diagnosis frequently depending to a great extent on the information thus received. The points to be specially noted are—1*st*, The position of any perceptible pulsation; 2*d*, Its extent and force; 3*d*, Its rhythm, whether systolic, diastolic, intermittent, or irregular; and, 4*th*, Whether any vibratile sensations are perceptible over the seat of pulsation, and if so, what is their apparent rhythm.

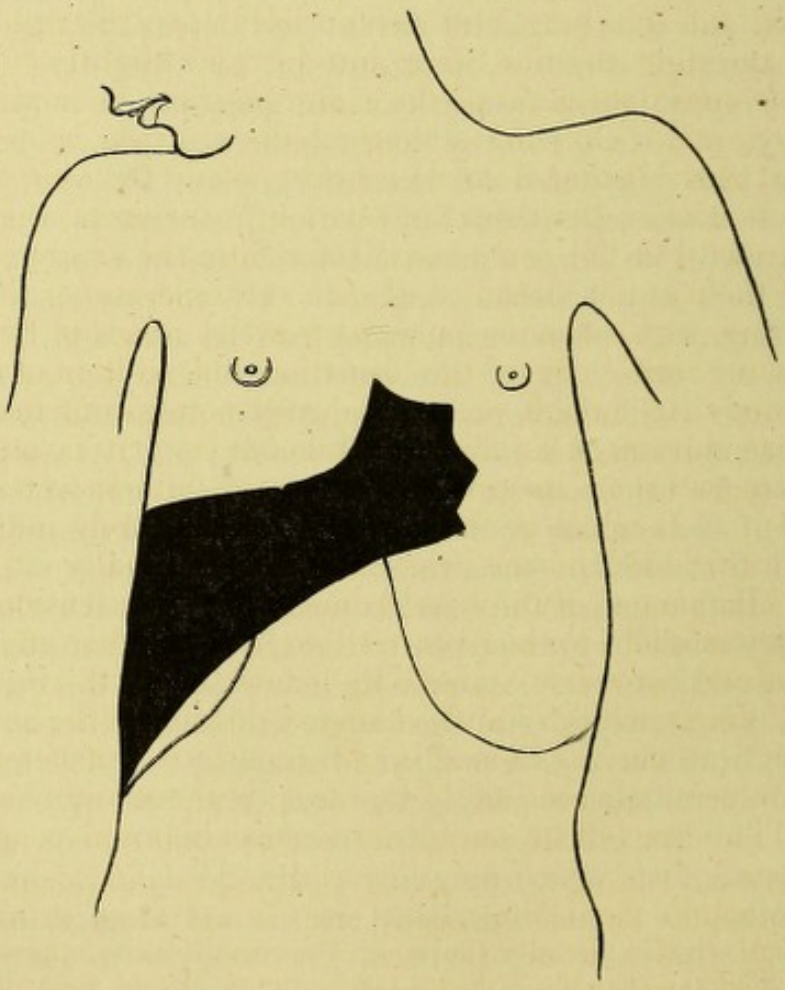
Having thus registered all the information to be obtained from the inspection and palpation of our patient, we now proceed to PERCUSS him, and from this we procure information of a totally different character from any hitherto obtained. Up to this time we have been dealing with phenomena which can be seen and felt, but by percussion we can map out the entire cardiac organ, or rather we can accurately limit the extent of the cardiac dulness, even in those parts where movement is unseen and unfelt; and, as we shall by-and-by see, we thus possess ourselves of a most important link in the chain of facts which connects those visible and tangible phenomena with that hidden cause, the condition of which it is our object to elicit. Percussion of the heart is usually reckoned rather a difficult and unsatisfactory procedure; in reality, if we set about it in a right way, and correctly interpret its indications of the condition of the heart, it is perhaps one of the easiest problems which arise in the varied applications of this method of diagnosis. All that we can learn from percussion is simply the size, form, and position of the precordial dulness, but we can learn these accurately, and, as already said, these are facts which may materially aid us in our inquiry.

Percussion, in medical parlance, is the art of ascertaining the condition of the internal organs of the body, as to resonance, by percussing or tapping upon the surface of the body just exterior to the organ whose state we wish to examine. It is obvious that this condition may be modified, first, by the amount of air contained beneath the part percussed—the size of the resonant cavity; and,

second, by the physical condition as to tension and structure of the parts on which we operate; and, in the living body, all of these circumstances may be variously modified. The heart itself, and the large bloodvessels with which it is immediately connected, contain, in the normal state, no air, and are never sufficiently tense to be themselves thrown into sonorous vibrations by percussion; when struck, therefore, mediately or immediately, they give forth no sound at all, or are simply said to be dull on percussion. The heart and large vessels are, however, in their natural positions, surrounded on three sides by the lungs; which are normally filled with air, and are therefore resonant; while, on the fourth side, the heart rests upon the liver, like itself, a dull, non-resonant body, from which we can, however, separate it more or less perfectly by measures presently to be described.

On percussing over the left side of the thorax, the veriest tyro can at once distinguish a more or less triangular area of complete dulness (Fig. 1[1]), which varies in size and shape in each indi-

Fig. 1.



vidual, and indicates the size of that portion of the heart in direct contact with the thoracic walls; the magnitude of this is often

This figure is a diagramatic representation of the cardiac and hepatic dulness of Fig. 4—organs healthy—of Sibson's Illustration of the Morbid

taken as an indication of the actual size of the heart itself, but it obviously only betokens the comparative degree to which the heart is uncovered by the lung, and as that may depend either upon increase of size of the heart itself or diminished size of the lungs, the information conveyed is of no positive value whatever. This superficial dulness, as it is termed, being of no value in estimating the size of the heart, it is evident that we must have recourse to mapping out the entire area of the cardiac dulness, both superficial and deep, to use for the nonce those anatomical terms which are so constantly applied to the acoustic phenomena, which, as Auenbrugger[1] first pointed out, ought to be explained and described solely by reference to the physical condition of the parts implicated, and not by any reference to their anatomical position, that being a matter not immediately apparent from the sounds elicited, though it may be deduced from them by a process of ratiocination.

The heart and large vessels occupy the centre of the chest, extending from the upper border of the third rib to the lower end of the sternum, and almost entirely occupying the space between the sternum and the vertebræ. In this position the ventricles encroach more upon the left lung, and the auricles upon the right one, the whole of the right cavities lying anterior and slightly to the right of the left ones; the axis of the right ventricle in relation to the pulmonary artery being almost vertical, the ventricle being broadest at the part most distant from the artery; while the axis of the left ventricle is almost horizontal in relation to the aorta, the ventricle being narrowest at the part most distant from the artery; in consequence of the peculiar formation of each ventricle, and of the anterior position of the right one, the pulmonary artery arises in front and to the left of the aorta. From the formation and position of the heart, it is obvious that, though we can and may percuss out the whole of the cardiac dulness, this is quite unnecessary; it is only of importance to ascertain its greatest extent of dulness vertically and transversely. Increase of the vertical dulness rarely indicates any alteration in the size of the heart itself, but is usually either due to hepatic enlargement, readily ascertained by an extension of these exploratory methods to the liver itself, or to pericardiac effusion; the former dulness, as a rule, extending below the sixth rib, the latter above the third; while a simple change of position of the heart, which may arise from various causes, is indicated by a transference of the normal dulness upwards or downwards, without any change in its extent. The apex beat, except in certain abnormal conditions, is,

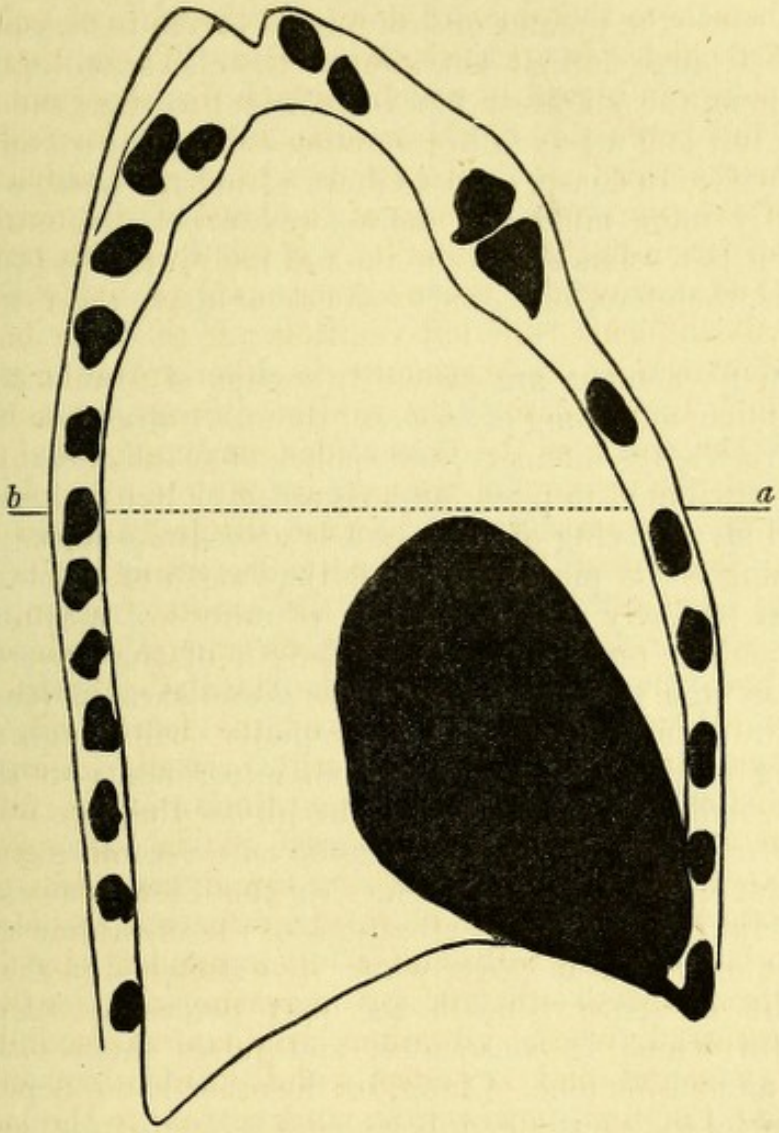
Anatomy of the Organs of the Chest, published in the twelfth volume of the "Transactions of the Provincial Medical and Surgical Association." It has been selected because the area of cardiac dulness approaches more nearly in configuration the ordinary conventional idea of such dulness than that of the other figures. How much this may vary within the limits of health may be seen by a reference to the other figures taken from bodies with healthy organs.

[1] Inventum Novum, §§ 17, 18, Scholia.

from the formation of the heart, the part which extends farthest to the left, and, being, as a rule, perceptible to the touch, only requires to be percussed out in those exceptional circumstances where the true apex beats beneath a rib, and not in an interspace. The right auricle is, of course, that part of the heart which extends farthest to the right, and being extremely dilatable, and readily influenced by any obstacle to the onward flow of the blood, transverse dulness about the level of the fourth rib comes to be an important indication of some obstacle to that onward flow, and therefore of enlargement of the heart chiefly in its auricular region. These, therefore, are the chief points in regard to which we look for important information from the percussion of the cardiac dulness. Increase of dulness above the third rib indicates, as a rule, pericardiac effusion. Increase of the transverse dulness at the level of the fourth rib indicates obstruction to the circulation. If the apex beat be displaced to the left and downwards, the obstruction is probably aortic, and has primarily influenced the left ventricle; if the apex beat be not displaced downwards, the obstruction is either mitral or pulmonary in its origin. In percussing the cardiac dulness, we must always remember that, so far as the three sides surrounded by the lungs are concerned, the passage of the percussion note from clear to dull is not abrupt, but transitional, because the heart upon all these three sides is covered by a gradually decreasing layer of lung. Practically, we find, in percussing the cardiac region, that the percussion note from above downwards ranges from clear and full just above the upper border of the third rib to perfectly dull somewhere below the lower border of the fourth rib, the note becoming gradually less full, but still perfectly clear, until it reaches the limit of dulness. As this diminution of fulness depends upon a lessening of the air-space, Skoda has applied to it the term *leer* or empty, the percussion sound becoming gradually emptier by the filling up of the resonant air-space from below, while it may also become gradually duller—more muffled—by filling the air-spaces from above,—that is, by increasing the density of the medium percussed, whose vibrations originate the sound, a perfectly empty sound and a perfectly dull one being, of course, synonymous; the first, however, remaining clear to the last, while gradually becoming emptier until perfect emptiness and perfect dulness coincide; while a muffled or originally dull sound may be full at first, but may gradually become emptier till perfect dulness and perfect emptiness coincide. The first form of dulness, the emptying of the sound, depending upon a gradual diminution of the air-space; the second form, the muffling or dulling of the sound, depending upon the gradually decreasing capacity of the percussed medium for undergoing sonorous vibrations. A percussion sound becomes, therefore, gradually emptier, less full, the duller or more muffled it becomes; but it by no means necessarily grows duller by becoming emptier, because a sound may be very

empty, and yet perfectly clear. It is obvious that, in this signification, the terms full and empty are equivalent to greater or less intensity of sound, an intensity in this instance depending upon the quantity of sound reaching the ear. A glance at the accompanying diagram (Fig. 2[1]) will show that, in the normal condition, we have

Fig. 2.



above the third rib (along the line *a b*) a larger air-space—a larger resonance box, and therefore a fuller sound producible on percussion

[1] This figure is a diagramatic representation of Fig. 2, Tab. 5 of Fasciculus 2 A of Pirogoff's "*Anatomia Topographica Sectionibus per Corpus Humanum congelatum triplici directione ductus illustrata.*"—Atlas, Petrop., 1859. The section passes at the upper part through the left sterno-clavicular articulation, at three Paris lines from internal margin of the head of the left clavicle. The lowest rib anteriorly is the seventh, cut through ten lines from the sternum; posteriorly, it is the twelfth rib, cut through nine lines from its vertebral extremity. It will be observed that, in this figure, the lung extends lower than ordinarily, and that perfect dulness would not commence till the lower edge of the fifth rib, instead of the fourth rib as usual.

—than below the third rib, and that the air-space gradually lessens, and, consequently, the percussion note gradually becomes less full, as we approach the lower border of the fourth rib, continuing to be clear up to the very last. In most works upon the subject, we are told to percuss more forcibly below the third rib, in order to elicit what is termed the *deep dulness;* but the idea of deep dulness is a composite conception with which acoustics have only a relative connexion; all we require to ascertain is simply the size of the resonance box—the greater or less fulness of the percussion sound; and in the average normal condition of the chest-wall and the contained organs, the slightest tap is sufficient to elicit the difference between a full and a less full or emptier percussion sound. When the chest-walls are denser or less vibratile, and the percussion sound consequently more muffled, a more forcible percussion may be required; but this refers equally to the full and to the less full sound; is the same, therefore, as regards percussion above the third rib and below it.

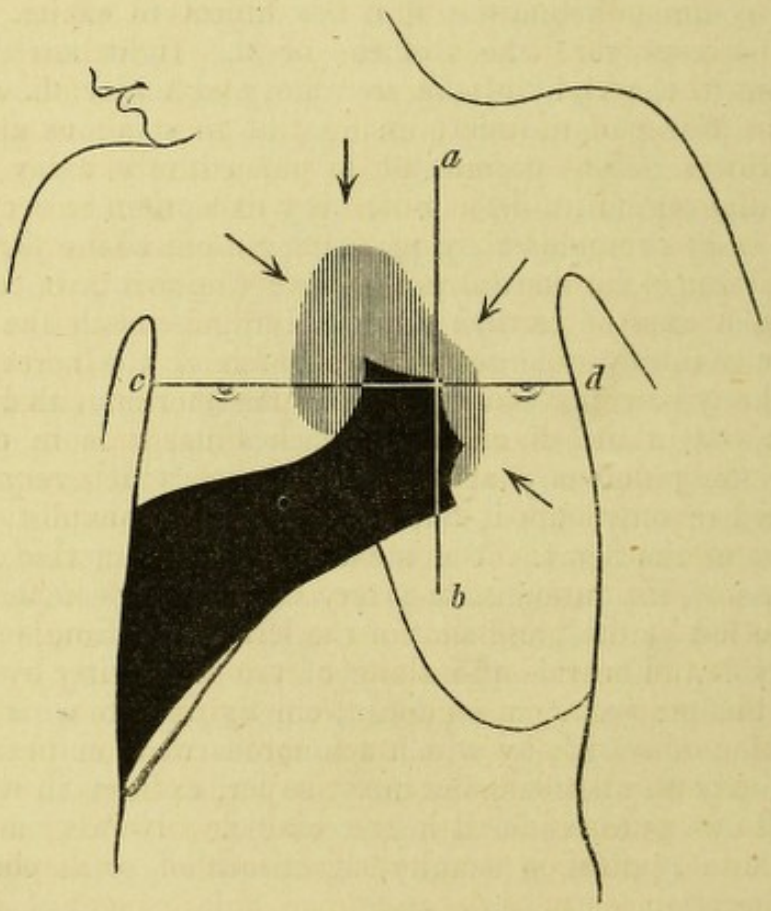
Acoustic phenomena are generally spoken of in relation to their intensity, pitch, and clang or *timbre.* By many observers the phenomena just referred to intensity, and spoken of as full or empty sounds, have been attributed to pitch, and regarded as low or high sounds; and indeed an emptying of the sound is necessarily accompanied by a heightening of its pitch. Because the length of a column of air which most perfectly resounds to the vibrations of a tuning-fork is exactly equal to one-fourth of the length of the sonorous wave produced by the fork, and this wave diminishes in length as it increases in rapidity, increase in rapidity being equivalent to heightening of pitch, as we learn from experiments on the syren. Hence the shallower any air space, the higher the note with which it most perfectly consonates, a percussion note becoming emptier becomes therefore also higher in pitch. Not that there is any alteration in the general rapidity of the vibrations of the membrane percussed because the resonance-box over which it is stretched is shallow, but because the air space beneath the part percussed selects those flutters with which it consonates, and raises them only to the dignity of an audible tone. Clang, on the other hand, depends upon the mingling of the overtones or harmonics belonging to the vibrating body itself with its fundamental note, and therefore varies with the nature of the structure thrown into sonorous vibrations by percussion—varies, therefore, as we percuss the intercostal tissues, the ribs, or the sternum—varies even with the structure of the plexi-meter or plessor employed. Nay, more, clang, mingled with resonance, has its influence in altering the percussion notes in those cases where, from emaciation of the chest-wall and size of the plexi-meter, the latter rests upon two ribs, leaving an air-space of varying size beneath. To avoid the confusing influence of clang, and, to some extent, also, that of varying tension or elasticity, in estimating the exact quality of a percussion note, it is advisable, in

percussing from above downwards, to compare rib with rib, and interspace with interspace; and, in like manner, in percussing transversely, we must carefully percuss along a rib or along an interspace, and avoid shifting indefinitely from one to the other. Both pleximeter and plessor must be as much as possible free from clang themselves, and the former must also be capable of close and accurate application to either rib or interspace; and in these respects no instruments are comparable to the forefinger of the left hand for a pleximeter, and the first two or three fingers of the right hand as a plessor, the only objection to their use—and it only applies to class teaching—being the comparatively less intensity of the sound produced; but this difficulty is easily surmounted by the use of a Winterich hammer as a plessor, by means of which an adept can, without injury to the finger pleximeter, produce a percussion note of perfectly sufficient intensity. A less skilful party using such a hammer is apt not to tap smartly enough; he may hit hard enough —there is not much likelihood of failure in that respect; but he does not lift the hammer quickly enough, and he therefore muffles or stops the sonorous vibrations it is his object to excite. It occasionally happens that the dulness of the right auricle is not appreciable to the right of the sternum, and under these circumstances the clang of the sternum may be so sonorous as—in percussing transversely—apparently to do entirely away with all cardiac dulness; but, unless under very exceptional circumstances, we can remedy this matter by percussing from above downwards; and by eliciting the sternal note above the aorta, we can at once map out the area of cardiac dulness lying beneath the sternum. We must also never forget that the arch of the aorta does not extend like a bow transversely across the sternum, as Piorry has figured it, but that, as anatomy teaches us, it is in close contact with the pulmonary artery and the heart at every point, so percussion can only map it out as a somewhat rounded projection at the base of the heart. We must also remember that, as a rule, the dulness of the innominate artery is scarcely perceptible, while that of the left carotid, and that of the left subclavian, is altogether imperceptible. For this the clang of the ribs lying over them is partly to blame; but it is, no doubt, chiefly due to what is called the inflection of sound, by which a sonorous wave embraces a non-resonant body on all sides, and may, so far, extinguish the dulness of a small one as to render it inappreciable. Hence, marked dulness in these regions is usually significant of some considerable morbid alteration.

As the heart rarely rises above the level of the third rib, and if it does so, it rises, as a rule, equally on both sides, so it is obvious that Piorry's line of oblique dulness, from apex to base, is of no special practical value; and, from all we have said, it follows as a corollary that there are only two lines of percussion of any practical importance—viz., a vertical and a transverse one. The first of these

must be thrown so far to the left as to be uninfluenced by the aorta and the pulmonary artery, and I have been in the habit of placing it at one inch to the left of the left edge of the sternum. When, percussing from above downwards from the lower edge of the left clavicle, we have in the normal condition first the clear, full sound of the lung down to the upper border of the third rib; beneath that, a gradually emptying but clear sound down to the lower border of the fourth, or upper border of the fifth rib; and, beneath that, perfect dulness till we reach the tympanitic sound of the stomach or intestine. Any deviation from this is abnormal, and its explanation must be sought by other methods of investigation. In like manner, the transverse dulness in the line of the fourth rib, which is that mainly affected by distention of the right auricle, must be carefully mapped out, percussing from without inwards on each side—that is, from the lung to the heart; because in that way alone we can best appreciate comparative shades of dulness, and also eliminate the clang of the sternum. The accompanying diagram (Fig. 3[1]) exhibits the per-

Fig. 3.



cussion dulness of the normal cardiac area, with the lines of practical importance (vertical, *a* to *b;* transverse, *c* to *d*) specially indicated. To separate the lower part of the heart from the liver is by no means an easy matter. It is said that we can occasionally

[1] This figure refers to the same subject as Figure 1. Percussion is to be made in the direction of the arrows.

make out a line of clearness between the two. I have never observed any such line of clearness, and, as the heart invariably rests upon the diaphragm, unless separated by fluid, I do not clearly see the mechanism by which this line of clearness may be produced. Any change of pitch between the cardiac and hepatic dulness is impossible, and can only be produced by upward conduction of the tympanitic note from the stomach or intestines. It follows, then, that we can only separate the heart from the liver approximately, by ascertaining the position of the apex beat upon the left side, and also the highest point of hepatic dulness upon the right side, and joining the two by a straight line. This line of separation can never be absolutely accurate, but is usually sufficiently so for all practical purposes.

It is sometimes of importance to map out the aortic dulness, so as to ascertain if it be increased in any direction; and this is by no means a difficult task, if we set about it in a right way. As I have already said, it is impossible to separate the aortic dulness from that of the heart: the only part in which this might be possible is occupied by the pulmonary artery, the dulness of which, however, differs in no respect from that of the aorta. It is therefore absurd to map out the aortic dulness, as Piorry and his followers have done, as an independent arch crossing the chest above the area of cardiac dulness. The normal aortic percussion is indicated in the foregoing diagram (Fig. 3), in which it will be seen to occur simply as a more or less rounded bulging, rising out of and inseparable from the general area of cardiac dulness, and only known to be aortic from its position above the vertical limit of cardiac dulness as indicated by the upper limit of the dulness in the line *a b*. Increase of this dulness in any direction may depend upon morbid enlargement of the aorta; but in the diagnosis of these enlargements, percussion dulness is only one of many physical facts which require to be collated and reasoned upon in estimating the probabilities for and against the assumption of the existence of any such morbid condition.

Absence or diminution of the precordial dulness in the rarest of cases is caused by the presence of air in the pericardium, less seldom by atrophy of the heart, and infinitely more frequently by increased extent of the pulmonary resonance from emphysema. Increase of the area of cardiac dulness is a much more common phenomenon. This may depend upon effusion into the pericardium, in which case the dulness is pyramidal, with the base downwards; and in this case the cardiac pulsation is annulled, enfeebled, or displaced. Or the increased dulness may depend upon enlargement of the heart, with or without hypertrophy; in these cases, the pyramidal shape of the dulness is less marked, but its broadest part is always above. When hypertrophy predominates, the cardiac shock is increased; when dilatation predominates, it may be almost annulled. But we must never forget that these conditions may be variously modified, and that our diagnosis, to be correct, must be based, not

upon one or two facts, but upon all the phenomena which can be ascertained in regard to the physical condition of the heart.

The next and last method[1] which we can employ in ascertaining the physical condition of the heart is AUSCULTATION, by which we understand the art of ascertaining the physical condition of the mechanism of the heart by the sounds produced during the passage of the blood through its several cavities.

If we place our ear over the cardiac area during the progress of the circulation, we become conscious that it is accompanied by sounds alternated with silences. If we listen over the heart of an infant, we distinguish only a uniform ticking, in which the sounds are alike in intensity, and the silences similar in duration; but, as we gradually extend our experience by listening to the hearts of older individuals, we find that, as we approach adult life, these sounds, with their intervening silences, assume a peculiar rhythm, which the ear thus educated very readily appreciates; and we also discover, that while the silences remain the same in every position, the sounds vary somewhat, according to the part of the cardiac area in which they are heard. Thus, when listening over the apex beat, we distinguish two pauses or intervals of silence,—a long pause and a much shorter one; immediately succeeding the long pause, we hear a dull, prolonged sound, followed by the short pause, and this succeeded by a much shorter and sharper sound, which is immediately followed by a renewal of the long pause, the accent in this situation being upon the prolonged or first sound following the long pause, making what is called a trochee in prosody—"– ⏑." On the other hand, when we listen over the base of the heart, we find that, while the relation of the sounds to the silences remains the same, the accent in this situation falls upon the second sound instead of on the first, so that, following the long pause, we have, instead of a trochee, an iambus—"⏑ –." We see thus, that though the cadence of these sounds alters with the position in which they are heard, they can, in the normal adult, be readily enough recognised to be first and second by their relation to the periods of silence; but whenever the heart's action becomes rapid from fever, debility, or any other cause, the long pause is diminished, and the rhythm of the sounds approaches that of the infant, so that, when the pulse beats more than 90 times a minute, it is almost impossible to distinguish with accuracy what is first and what is second, and if for any reason it is desirable to do so, we must employ a double stethoscope, and by placing one end over the heart's apex, and the other over the base, a little practice and a little care will enable us readily enough to distinguish what is first from what is second, by a due attention to the slight difference in the character of the sounds, and by a careful appreciation of the position in which each sound is most distinctly heard. Apart from the alterations in rhythm,

[1] For, of course, in the practical exploration of the heart, we exclude both the cardiograph and the sphygmograph, the use of which can never become general as a means of clinical examination by an ordinary practitioner.

which are due to the rate of cardiac action, the heart sounds vary in distinctness in each individual; and experience has taught us, that in nervous hearts, which are usually thin-walled, and in dilated hearts, which are always thin-walled, the first sound is particularly loud, clear, and distinct; while, in hypertrophied, and therefore thick-walled hearts, the same sound is always muffled and indistinct, the second sound not being affected by this cause. Of course we cannot securely base our diagnosis of the state of the cardiac walls upon the distinctness or indistinctness of the sounds; but this phenomenon supplements and confirms the information we obtain in other ways. The second sound also varies in distinctness and in intensity, but always from causes which are extracardiac.

Besides the alterations in rhythm depending upon alterations in the rate of cardiac action, and alterations in distinctness depending upon hypertrophy, dilatation, or some extracardiac cause, we find these sounds occasionally replaced by noises, *bruits*, or murmurs, as we term them, which are totally different from the sounds they displace, and which vary with their causes.

Physiologists teach us that the first sound coincides with the contraction or systole of the ventricles, and is composed of several ingredients, of which the shock of the heart's apex on the walls of the chest, the bruit which accompanies muscular action, and the sound produced by the closure of the auriculo-ventricular valves, are the most important. But for all practical purposes the last is all-sufficient, as we find that whenever these valves are from any cause —such as dilatation of the ventricle—rendered incapable of closure, the valves themselves being healthy, the first sound is almost entirely replaced by a murmur which more or less completely obscures the muscular bruit, and, to a less extent, the shock. The second sound, on the other hand, as it immediately follows the ventricular systole, must coincide with the ventricular diastole, and therefore with the moment of time when the arterial semilunar valves are closed by the recoil upon them of the blood compressed by the arterial systole; and, accordingly, we find that when these valves are from any cause rendered incapable of closure, the second sound is more or less completely replaced by a murmur according to the degree of imperfection present.

The heart, however, is a double organ, with two auriculo-ventricular openings and two large arteries springing from it; and it is of importance to determine which side is affected. But it so happens that all the cardiac valves lie so close together, that a superficial area of half an inch square will include a portion of all the four sets of valves *in situ*, while an area of about one quarter of an inch will include a portion of all except the tricuspid.[1] It is obvious, therefore, that it is impossible to differentiate the sounds or murmurs produced by one valve from those produced by another by merely listening with an ordinary stethoscope over the place of origin. By the rhythm alone we can readily distinguish which is first and

[1] Walshe on Diseases of the Heart, 3d edition, 1862, p. 6.

which is second; but to differentiate a right first or second sound from a left first or second sound, we must take means to separate the one from the other; and this we do by taking advantage of the facts that sounds produced in one medium lose in intensity in passing into another, and that sounds produced by any fluid in motion are invariably transmitted in the direction of the onward current.[1] Hence we find that sounds produced in any one cavity of the heart are heard with most distinctness over that part of the thoracic wall at which the given cavity approaches the surface most closely; thus, the only point at which the left ventricle directly impinges on the chest-wall is where the apex beat is felt, and that is precisely the spot where the first sound produced in the left heart by the closure of the mitral valve is most distinctly heard: a space of about an inch in diameter around the apex beat is therefore termed the mitral area. Nearly the whole of the right ventricle is uncovered by lung, and impinges directly on the lower part of the sternum; and at this part, especially along the left edge of the sternum, where it is joined by the cartilages of the fourth, fifth, and sixth ribs, the right first sound produced by the closure of the tricuspid valve is best to be heard, and the triangular space covering the position of the right ventricle is therefore termed the tricuspid area. In the normal condition, the two ventricles act simultaneously; and the two sounds differ so little from one another, that it is impossible to differentiate clearly the one from the other. When, however, from any cause, these sounds become irregular or replaced by bruits, the differentiation by means of a reference to these areas of audition is readily enough made, though, of course, there are always plenty of confirmatory proofs.

On the other hand, there is, both in health and in disease, a marked difference between the aortic and pulmonic second sounds, and it is often of the greatest importance to differentiate the one from the other, and clearly to recognise the distinctive characteristics of each. As already mentioned, the aorta and pulmonary artery originate very close to each other, the pulmonary valve lying about the middle of the third left cartilage, one half being to the left and the other to the right of the left edge of the sternum, which exactly divides it in two. From its point of origin, the pulmonary artery rises to the lower edge of the second left cartilage, where it divides into its two great branches going to the right and left lungs; this, therefore, is the place where it is nearest the surface of the chest, and the second interspace, or the sternal end of

[1] Sound is reflected, inflected, and refracted like light, and is readily conveyed to almost any distance in smooth tubes; the difficulty which it finds in passing from one medium to another, even though these should be only strata of the atmosphere of varying densities, is sufficiently indicated by the curious statement that battles have been lost for want of reinforcements which were actually waiting within what was thought to be earshot for the sound of the cannon to indicate the moment of advance.—*Vide* Tyndall "On Sound," London, 1869, p. 23, etc.

the third cartilage, is the position in which the pulmonary second sound is best heard. On the other hand, the aorta rises a little below, behind, and to the right of the pulmonary artery, its valves corresponding to the lower edge of the third left cartilage, behind and to the right of the pulmonary valves, and it passes upwards, forwards, and to the right, till it reaches the upper border of the second right cartilage, when it passes obliquely backwards and to the left, forming what is termed the transverse portion of the arch of the aorta. At the second right cartilage, therefore, the aorta is nearest the surface of the chest, and the arterial walls and the blood-current coincide in readily conveying in this direction the resonant vibration which results from the closure of the aortic semilunar valves; in this position, therefore, the aortic second sound is most readily differentiated from the pulmonary one. In the normal condition the aortic second is heard to be louder and more distinct than the pulmonic one; both vessels are, however, normally covered with lung, and as from various causes, congenital or morbid, one or other may become uncovered and thus brought nearer the surface, its corresponding sound may then be heard more distinctly than is normal, and the ordinary condition may then be either intensified or reversed; but this does not happen without the occurrence of other phenomena which enable us to detect and explain the source of the abnormality, as we shall by-and-by have occasion to see. But besides this greater distinctness of the one sound more than the other, depending upon mere alteration in the anatomical relation of the parts, we have occasionally an alteration in the character of one or other of these sounds depending upon intrinsic causes. To this peculiar alteration of the second sound a variety of terms have been applied: it has been called booming, ringing, clanging, pumping, cavernous, and accentuated; and, perhaps, accentuated or booming are the most unobjectionable expressions we can employ to define this particular change in the second sound, in which the element of tension seems united with an increase of distinctness—a combination which, however, must be heard to be properly understood. This accentuation of the second sound may be heard either at the second right or at the third left costal cartilage; it may, therefore, be either of aortic or pulmonic origin. Its occurrence in the pulmonary artery was long ago pointed out by Skoda as an important aid in the diagnosis of mitral disease; but it is not only in mitral disease that the presence of an accentuated pulmonary second is of important significance, for this accentuation is constantly present in every form of cardiac disease involving obstruction to the onward flow of the blood, and is the most persistent of all the acoustic phenomena indicative of cardiac disease, being frequently the only thing markedly abnormal to be detected. In the absence, therefore, of any pulmonary disease capable of producing congestion, persistent accentuation of the pulmonary second sound is to be regarded as invariably indicative of cardiac valvular lesion. The

mode in which this accentuation is produced is very simple. The pulmonary circulation is a closed circulation, and, like the water in a Bramah press, the blood within the pulmonary circuit presses equally in all directions; any obstruction to its onward flow, therefore, produces not only congestion of the lungs, but also a uniform increase of the tension throughout the whole of the pulmonary circulation, often accompanied, if long continued, by slight dilatation of the pulmonary artery, and always by a closure of the semilunar valves with an exaggerated force proportionate to the hindrance it has met with. In the aorta the case is somewhat different, but the explanation of this is equally simple. The systemic circulation, though also anatomically a closed circulation, is yet incapable of acting like a Bramah press, because of the many local conditions which may influence it in various ways. The portal circulation itself cuts off a varying yet always considerable amount of blood from any participation in general hydraulic influences; while each of the extremities, as well as every gland, has a circulation which, the experiments of Claude Bernard have taught us, may be entirely separated as regards acceleration or retardation—congestion of veins or arteries—from the general circulation, by means of local nervous influence. It is quite otherwise with the lungs. Here we have solely to do with the circulation through two large glands, whose circulation is so united as to make them in that respect but one, so that though nervous influence may either accelerate or retard this through the lungs generally, or through only a part of either of them, as in the case of local inflammation, yet the circuit is so short, and the vascular connexion so intimate, that this influence is not merely felt locally, but is exerted on the pulmonary circulation generally, though it may vary in degree according to circumstances. The result, however, of the systemic circulation being compounded of many local circulations in which the blood tension is continually varying, is completely to neutralize the effect of these local alterations on it as a whole; while the variety of causes which may produce these alterations, and the variety of times and positions in which they act, is such as to render any general alteration impossible. Accentuated closure of the aortic semilunar valves, from a general or systemic cause, is therefore not to be thought of; it can only arise from some local cause. But the only local causes possible are, first, increase of arterial tension, which, to be effective in this matter, must be general; and this, as we have just seen, is impossible. And, second, increase of the weight, and consequently of the volume of the blood closing the valves, irrespective of the arterial force acting upon it; and as increased amount of contents involves increased size of what contains it, we arrive at dilatation of the ascending aorta chiefly, and partly, also, of the innominate artery, as the sole active causes in producing accentuated closure of the aortic semilunar valves. Accentuation of the pulmonary second, unaccompanied by disease of the lungs, is therefore an invariable

sign of some cardiac lesion; while accentuation of the aortic second is as invariable a sign of aortic dilatation, when these accentuated sounds are heard in their normal positions, the third right and the second left costal cartilages.

Outside of the normal area of cardiac dulness, as depicted in Fig. 3 (p. 1072), we occasionally hear the normal cardiac sounds more distinctly than is usual, and this may depend either upon increased resonance from condensation of that part of the lung over which they are heard, or upon an aneurismal bulging of the aorta at that part. In the latter case, we may either hear the normal cardiac sounds louder than usual in the given position, or if the cardiac sounds be abnormal, these abnormal sounds may be heard more distinctly than they ought to be; or the second sound alone may be heard accentuated in this abnormal position, or this abnormally placed accentuated second sound may be preceded by a localized systolic bruit, or it may be more rarely replaced by a localized diastolic bruit. But all these sounds, when heard outside of the normal area of cardiac dulness, indicate disease of the lungs or of the aorta, if they be identical with those heard within the cardiac area; if they vary from them, then they indicate disease of the aorta in one or other of its various forms, and in any case have only to be noted for further examination. In the normal condition of the heart, the sounds and silences succeed one another in the manner described,—each ventricular systolic sound being accompanied by an arterial pulse perceptible in the radial arteries. But it sometimes happens that these sounds and silences succeed one another with perfect regularity over the cardiac area, and yet the radial pulse intermits; this is an evident sign of cardiac debility, and must be noted, that its cause may be inquired into. At other times the intermission extends to the cardiac action itself, and this also must be noted for further inquiry. At still other times the cardiac sounds and silences, and also the radial pulse, occur with extreme irregularity; and this also must be carefully noted, that its cause may be subsequently inquired into.

But there is still another form of irregularity in the cardiac sounds which may be a symptom of disease, but which may also occur in perfect health, and that is reduplication of these sounds. In its most extreme and rarest form we have, instead of two, four sounds,—two first and two second sounds; the two beats acting separately and not simultaneously as usual. More usually, however, we have three sounds instead of two; and the rarest form of this is when the first sound is reduplicated, which is extremely seldom to be heard with any distinctness. A reduplication of the second, however, is a matter of common occurrence not only in disease but even in health. It is produced by a more rapid action of one ventricle compared with that of the other, and is heard as a *bruit de rappel* either at base, apex, or both. It may be vocalized by the syllables ta-ta, and closely resembles the sound of a hammer which strikes the anvil, rebounds, and strikes again, remaining motionless.

Reduplication of the second sound is of frequent occurrence in mitral constriction where there is a physical cause capable of preventing the filling of the left ventricle, and which therefore permits the right one to get the start of it. Reduplications of the cardiac sounds in disease are often permanent; in health they are more frequently fugacious, appearing one instant and disappearing the next. They are merely abnormal exaggerations of a phenomenon which, with care and appropriate appliances, may be detected in every one; reduplication of the first sound occurring at the end of the expiration or commencement of the inspiration; reduplication of the second sound occurring at the end of the inspiration and commencement of the expiration. These normal reduplications depend upon the variations in the pressure produced by the respiratory movements at the origin of the arterial and venous systems. The first sound becomes reduplicate because excess of blood-pressure in the heart retards the closure of the tricuspid valve; and the second sound is reduplicated because excess of pressure in the aorta accelerates the occlusion of the aortic valves.[1]

Within the cardiac area, as already remarked, murmurs may take the place of one or other of the normal sounds, or may occupy the time of one or other of the silences, or may even occupy the whole period of a cardiac pulsation, or any portion of it. Such murmurs may be of endocardiac or of exocardiac origin, and as endocardiac murmurs are most frequent, and are chiefly due to valvular lesions, it is a good rule to endeavour first of all to associate all murmurs with a valvular origin, and only after exhausting this hypothesis to proceed to determine the probabilities in favour of their extracardiac origin, or of their dependence upon some intracardiac cause apart from valvular lesion.

Of all the signs of cardiac disease, murmurs are those most usually confided in, and yet they are really those of least value; first, because, as already remarked, exocardiac murmurs may simulate those of endocardiac origin; and, second, because murmurs truly of valvular origin may disappear temporarily or permanently.[2] Thus we may have murmurs apparently of valvular origin which are really exocardiac in their origin; second, we may have murmurs truly of valvular origin, yet without permanent valvular lesion, which may disappear, leaving the heart uninjured; and, lastly, we may have murmurs truly of valvular origin which may disappear temporarily or even permanently, the valvular lesion still continuing. It is obvious, therefore, that murmurs cannot of themselves be accepted as certain indications of cardiac disease, even although we can positively connect them with the lesion of a definite valve, because that lesion may be temporary in its character

[1] Potain, "Note sur le Dedoublement Normaux des Bruits du Cœur."—*L'Union Médicale*, 1866.

[2] On the Variation and Vanishing of Cardiac Organic Valvular Murmurs, by W. R. Sanders, M.D., Professor of Pathology in the University of Edinburgh.—*Ed. Med. Journal*, Jan. 1869, p. 584.

and wholly unconnected with actual disease of the valve affected. To this there is but one positive exception, and that refers to the auriculo-systolic, the so-called presystolic murmur; though there is obviously a greater or less probability of any of the other valvular murmurs being also connected with actual disease of the valves affected. That probability, however, falls to be considered under the head of each separate valve; at present I have only to describe the acoustic phenomena audible on auscultation, to point out the means by which we determine whether these are valvular in their origin or not, and to state that no murmur, except the one already referred to, can ever be accepted as a definite sign of actual cardiac disease, but must be simply noted, to be afterwards duly considered along with the other information derived from inspection, palpation, and percussion, when we come to estimate the probabilities for or against the existence of any special lesion, for there is no royal road to diagnosis any more than to anything else that must be made, as Opie mixed his colours, "with brains."

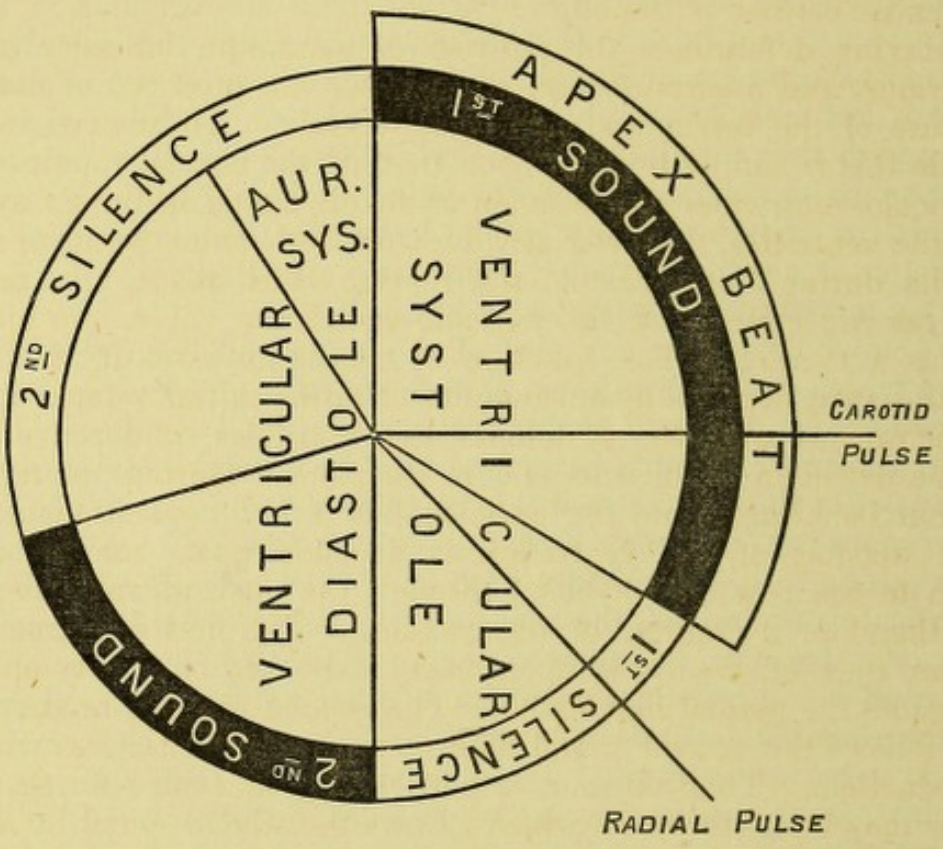
When we hear, over any part of the area of cardiac dulness, any sound which differs from those sounds we have already taught ourselves to recognise as those audible in the normal condition of the heart, our first care must be to ascertain by careful examination at what part of the cardiac area this peculiar sound is most distinctly to be heard; this we may term the position of maximum intensity. This position either coincides with one of the normal areas already described as the mitral, the tricuspid, the aortic, or the pulmonary area, or it does not. If the position of maximum intensity of this abnormal sound coincides with one or other of these normal areas, it most probably depends upon some lesion, temporary or permanent, of that valve whose normal sound is audible in that position, and which it either obscures, replaces, precedes, or follows. If it does not coincide with one or other of these normal areas, it is certainly not valvular in its origin, unless this position of maximum intensity be over the lower end of the sternum, or just to the right of the pulmonary area and in the same plane; and that any such murmur may be of valvular origin, it must be diastolic in the first position and systolic in the latter; the reasons for this being to be found in the anatomical relations of the parts, and in the mode in which sound is conducted.

Our next care must be to determine the actual rhythm of the murmur or abnormal sound, that is, its positive relation to those several physiological acts which constitute a cardiac pulsation. That we may be able to do this, we must first of all have a clear conception of those physiological acts which make up what we call a cardiac pulsation; and, secondly, we must be able to recognise them. The accompanying figure (Fig. 4, altered from Gairdner[1])

[1] To Dr Gairdner we owe the first attempt to represent a cardiac pulsation diagramatically. This diagram, which is altered from his, is merely an attempt to give more physiological accuracy to it. The apex beat and the two sounds

represents diagramatically the several acts of a cardiac pulsation in the normal adult heart, in which a long sound precedes a short silence, followed by a short sound, and that again by a long silence. When the heart beats under 90 per minute, there is never any difficulty in making out the several relations of these sounds and silences, however slow the heart's action may be. When, however,

Fig. 4.



the heart's action is over 90 per minute, it approaches more or less to the uniform tic-tac of the infant's heart, and the more nearly this is the case the more equal are the two silences, and the greater is the difficulty experienced in determining what sound is long or first and what is short or second. To ascertain this with precision, we must either wait until rest and other agents have slowed the heart's action to 90 or fewer beats per minute, or we must depend upon our accuracy in the use of a double stethoscope, knowing that under all circumstances the first sound is always relatively long at the

are represented as occupying, as they do, appreciable intervals of time during that pulsation. The heart's action is represented as continuous and without actual pause or rest, though at times soundless, while the carotid pulse is placed in the position which Valentin says it ought to occupy in relation to the commencement of the apex beat, and the radial pulse is placed in more nearly its usual relation both to the carotid pulse and the apex beat. The foot pulse is nearly synchronous with the radial. According to Mr A. H. Garrod, the difference between the two, with a pulse beating 75 per minute, amounts to 0·0012 of a minute.

apex and short at the base, while the second is exactly the reverse; or we may determine what sound or murmur coincides with the apex beat or carotid pulse, that being certainly coincident with the ventricular systole; remembering that any reference to the radial pulse is irrelevant and misleading, as even in the normal condition it is separated from the apex beat by an interval which may amount to about one-sixth of a second, and that this interval may be increased in disease to the half, or even to the whole period of an entire cardiac pulsation.

Having determined the position of maximum intensity of the murmur, and ascertained its rhythm, we are prepared to state the nature of the lesion upon which it depends. All that we require to do this is simply to remember that in the normal condition the auriculo-ventricular valves on both sides are closed during the systole of the ventricles, the arterial valves on both sides being opened; while during their diastole the reverse takes place, the arterial valves are closed and the auriculo-ventricular valves are opened. Thus a murmur, whose position of maximum intensity is in the mitral area, depends upon some defect in the mitral valve: if it be synchronous with the systole of the ventricles, it depends upon some defect in its closure, and is therefore a murmur of regurgitation backwards into the auricle; but if it be synchronous with the diastole of the ventricles, it depends upon some obstruction to the flow of the blood through the open mitral valve, and is therefore a murmur of obstruction. The first of these murmurs runs off from the apex beat, and more or less completely replaces the normal first sound. The second murmur accompanies or follows the second sound, but in no respect interferes with its production. That murmur, or portion of a murmur—for the murmur may be continuous—which follows the second sound by an appreciable interval, and whose position of maximum intensity is not only placed in the mitral area, but which is but little audible out of that area, immediately precedes and runs up to the first sound, (is therefore presystolic); but a glance at the diagram (Fig. 4) shows us, what we indeed already know, that the systole of the ventricles is immediately preceded by the systole of the auricles, and that what we term presystolic is truly auriculo-systolic in its rhythm. This murmur is not only a murmur of obstruction, but a murmur of obstruction to direct cardiac action, and, like all such murmurs, is always rough. It is the only cardiac murmur invariably associated with actual disease of the valves affected.

These three murmurs may exist separately, as occasionally they do, or any two of them may coexist; and sometimes the whole three are present, and then we have a murmur audible in the mitral area, and running through the whole period of cardiac action, the first sound being either much altered or entirely replaced; while the second sound, though not always audible at the apex, always exists, the pulmonary second being greatly ac-

centuated, unless the two second sounds are heard separately, the one immediately following the other, as what I have already mentioned as a reduplication of the second sound, in which case the accentuation of the pulmonary second is always modified.

These murmurs, though most common on the left side, may also be heard over the right heart, a systolic tricuspid murmur being very frequent, a presystolic tricuspid being somewhat rare, while a diastolic tricuspid is a possible, but, so far as I know, a hitherto unrecorded complication. And in saying this it is obvious that I mean a diastolic murmur over any part of the right auricle or ventricle, both the aortic and pulmonary second sounds continuing audible, and with entire absence of any symptom or sign of aortic regurgitation. This is a most important point, because it often happens that a diastolic murmur, originating in defective closure of the aortic valves, is heard loudest over the lowest part of the sternum, being conducted downwards partly by the descending current of the blood, and partly by the peculiar resonance of the sternum. We shall afterwards see that a diastolic murmur, of purely aortic origin, may have its position of maximum intensity over the lower part of the sternum—the tricuspid area, or in the mitral area, and that this depends upon the segment of the valve affected and has an important influence upon the prognosis, being always accompanied by absence or alteration of the aortic second sound, and, if uncomplicated, free from any of the symptoms and signs of mitral or tricuspid lesion, affording a well-marked example of the importance of looking on a murmur as merely one of many physical signs which must all be duly considered in estimating the probabilities in favour of the existence of any given lesion.

At the base of the heart the audible murmurs are apparently less complicated than at the apex, because here we have no presystolic murmur, the only possible murmurs being systolic and diastolic. But the causes of these are so various that the pulmonary region has somewhat deservedly received the name of the region of romance, from the various theories propounded in explanation of the murmurs occurring there; while the physical causes of aortic murmurs, and the physical relations of the aorta itself, are so complicated, and so efficiently modify the conduction of these murmurs, that though it is quite usual to say that a systolic murmur having its position of maximum intensity at the sternal end of the second right costal cartilage is due to aortic obstruction, yet this is by no means always the case; and in like manner, though a diastolic murmur audible in that position is most probably due to aortic regurgitation, yet its absence from that situation is no proof of the absence of regurgitation, because the murmur due to regurgitation may be, as already pointed out, only audible beneath the level of the aortic valves.

The important thing for us to remember is, that in noting the characteristics of a murmur the chief points to be determined are,

first, the position of maximum intensity, as probably indicating the valve affected; *second*, the rhythm of the murmur, as indicating whether it is systolic or diastolic; and, *third*, whether the valve sound normally heard in any given position is wholly replaced by the murmur, or only altered in duration and character. We must also remember that even the existence of a well-defined murmur is not by any means a certain indication of actual valvular disease, any more than is the absence of any murmur to be regarded as a sure proof of the non-existence of valvular disease. The characteristics of a murmur, and its relations to the normal sounds and silences, must merely be noted and afterwards duly considered in connexion with the other physical signs already commented on.

Endocardiac murmurs of other than valvular origin are occasionally, though rarely, observed. Sometimes these are due to congenital deficiency of some part of the septum between the two hearts; and in that case they only intensify those valvular murmurs which almost invariably coexist, and extend their area of audibility; or where no valvular lesion coexists, as in other endocardiac murmurs of still rarer occurrence, said to depend upon the existence of some vibrating substance, as a flake of lymph, upon one of the *chordæ tendineæ*, in an otherwise healthy and normal heart, the position of audition and the rhythm may be certainly the same as those of truly valvular origin, from which they can only be differentiated by a due consideration of the other physical signs; that is, by a judicious reference to those physical facts which, as I have already shown, indicate actual obstruction to the onward flow of the blood, and which point out with a certainty, the attainment of which is exactly proportionate to our skill in interpreting these signs, whether that obstruction is substantive or only dynamical. Exocardiac murmurs frequently simulate in their timbre those of endocardiac origin, and when they also coincide in rhythm with the systole or diastole of the heart, it may be difficult to distinguish such a murmur from an endocardiac one of non-valvular origin. As, however, exocardiac murmurs depend chiefly upon the friction of the roughened visceral and parietal portions of the pericardium upon one another, or of the pericardium upon the pleura, and as, unless under very exceptional circumstances, this friction is always greatest over the centre of the heart, and neither at its apex nor exactly at its base, we have a pretty safe indication of the exocardiac origin of the bruit in the non-coincidence of its position of maximum intensity, with the usual situations of that position in the case of murmurs which are of valvular origin. We have also the localization, more or less complete, of the bruit, which never passes to any distance from the area over which it is most audible, and which is not propagated by any blood-current, but simply radiates equally all round the position where it originates, usually possessing a distinctly superficial character; and we have the entire absence of any pulmonary

accentuation, or of any anatomical indication of obstructed circulation. The sound produced, also, as a rule resembles the crackling of parchment, the creaking of new leather, or a grating or mere rubbing sound, of greater or less intensity, but only very rarely in any respect simulates the blowing murmurs of valvular lesions. Whatever may be its timbre or its rhythm, it must, like all and every other sound heard over the cardiac area, be carefully noted as to its character, position of maximum intensity, rhythm, degree, extent, and direction in which it is propagated, to be afterwards submitted, along with the other facts ascertained, to a careful and discriminating scrutiny. The same may be said of those murmurs which are audible in various positions out of the cardiac area, and which owe their origin to the passage of the blood through the vessels. Such murmurs are sometimes heard in the arteries and sometimes in the veins. They are chiefly systolic, but sometimes diastolic, in rhythm. In the arteries these murmurs may be strictly localized, or may be audible over every part of the arterial system. In the veins, as a rule, they are audible everywhere, provided certain conditions are complied with, though they are permanently present in certain parts of the body where these conditions naturally exist. Such intravascular murmurs are of the most various significance, and can only be most briefly referred to now. Sometimes they signify serious lesion of the vessels themselves; at others, nothing worse than some slight deformity of the chest, the result of rickets; and at still other times, only an alteration in the condition of the blood itself, which may arise from the most various causes.

I cannot conclude this lecture without a cursory reference to the manner in which these murmurs are believed to be produced, because this is a subject in regard to which practical experiment may yet become a most fruitful source of improvement in diagnosis. The crude ideas of our forefathers, which have culminated in the notion that such murmurs are produced by the friction of the blood-current on its containing walls—an idea which the researches of Poiseulle and others have set aside, by showing that no such friction exists—have been replaced by a recurrence to the theory, first started by Corrigan, that such murmurs are due to the vibrations induced by eddies in the blood-column itself, a theory which has been reduced to scientific simplicity by M. A. Chaveau,[1] who has with great probability referred the causation of all murmurs, whether intracardiac or intravascular, to the sonorous vibrations of fluid veins, such as have been observed by Savart, and which are capable of being transferred, as Marey has experimentally shown, to the surrounding fluid within which the vein may be produced, and to the walls containing it. The roughness of the

[1] *Comptes Rendus de l'Académie des Sciences*, 1858: "Toute veine fluide est le siège de vibrations susceptibles de produire des sons, vibrations qui ébranlent aussi l'orifice d'ecoulement de la veine."—P. 841. The intravascular murmurs

murmur and the tactile perceptibility of the vibrations are in some degree proportionate to the force exerted in producing this sonorous vein, while its musical character must of course depend upon the rapidity of these vibrations.

But the exact manner in which these sonorous veins are produced, and the mode in which the production and conduction of their sonorous vibrations may be modified by evident or imperceptible alterations in the heart's action itself, and in the position of the patient himself, are problems which yet remain to be defined, and an accurate experimental investigation of these problems will certainly prove to be of the utmost importance in the accurate diagnosis of cardiac and vascular diseases.

of anæmia are due "aux vibrations de la veine fluide intravasculaire, vibrations qui se produisent toujours quand le sang pénètre avec une force suffisante dans une partie réellement ou comparativement dilatée de l'appareil circulatoire."—P. 933.

Fig. 5.

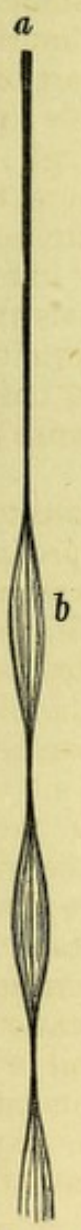
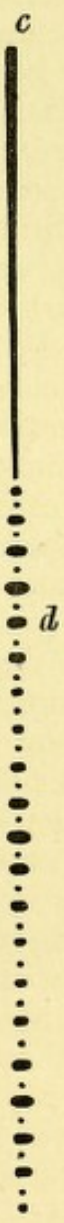


Fig. 6.



Felix Savart has described the phenomena presented by a liquid vein of water flowing through a circular orifice in the bottom of a vessel. The part of the vein *a b*, Fig. 5, is steady and limpid, presenting the appearance of a solid rod, which obstructs vision and wets the finger when passed through it. This decreases in diameter till it reaches a point (*b*) of maximum contraction, beyond which it is turbid and unsteady, marked by periodic swellings and contractions, does not wet the finger when passed through it, and does not obstruct vision, even though the fluid be mercury. At *b*, in fact, the liquid is no longer continuous, but resolves itself into a series of liquid spherules, which have an appearance of turbid continuity, from their rapid succession never permitting the primary impression wholly to fade from the retina till succeeded by a second. Fig. 6 represents this fluid vein illuminated by an electric flash, when the drops of which it consists are seen as it were motionless in the air, and the cause of the periodic swellings and contractions are at once apparent; for these spheroidal drops, when first detached, have their long axis vertical; when abandoned to their own molecular forces they seek to become spheres, and, like a pendulum in motion seeking to return to rest, the contraction goes too far, and they become flattened spheroids, which again elongate vertically; hence the appearance of alternate swellings and contractions. Savart traced the production of these pulsations to the orifice through which the vein passed, but did not regard them as the result of friction. Under moderate pressure they succeed each other sufficiently rapidly to produce a feeble musical note, the pitch of which may be fixed by allowing the drops to fall upon a stretched membrane.—*Vide* Tyndall "On Sound." London: 1869: p. 244, etc.

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