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## ORIGINAL ARTICLES.

### ON THE CLINICAL STUDY OF THE HEART SOUNDS.

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The volume of the transactions of the American Medical Association for 1858, contains a prize essay by me on the "Clinical Study of the Heart-Sounds in Health and Disease." The term heart-sounds in this essay, used in its conventional sense, was limited to the normal sounds of the heart, and their abnormal modifications, the term, as thus limited, not embracing adventitious sounds, these being distinguished as heart murmurs. The following quotation is from the introductory remarks in that essay: "The clinical study of cardiac affections, which has been prosecuted of late years with so much assiduity and success by observers in different countries, has had reference more especially to the murmurs. On the normal sounds and their abnormal modifications, comparatively small attention has been bestowed. In the course of investigations, having reference to the diseases of the heart, I have been led to think that these sounds have not hitherto received that consideration which their importance claims. Impressed with the belief that a field for clinical research is here open, which requires only proper cultivation to yield results, possessing not only interest, but much practical utility as regards our knowledge of the heart in its physiological and pathological relations, I have for some time been engaged in inquiries directed to the subject. My design in this essay is to contribute the fruits of my researches, and to indicate some of the questions which are to be satisfactorily settled by an accumulation of facts developed by clinical observation."

This quotation is not entirely inapplicable to the subject at the present time. After twenty-six years, it may still be said that here is a field for clinical study not yet fully cultivated, and that the heart-sounds do not now receive at the hands of medical practitioners a consideration commensurate with the importance of their practical bearings. My object in this paper is to supplement the essay to which I have referred, by the results of some further study of the subject, with a view, more especially, to bring it again to the attention of clinical observers.

It is customary to consider the heart sounds as

consisting of two in number, to wit: the first or systolic, and the second or diastolic sound. For convenience in regard to clinical study, it seems to me desirable to recognize five distinct sounds, two of which are diastolic, and three systolic.

The two diastolic sounds are referable, respectively, to the aortic and the pulmonic orifices of the heart. They may be studied separately, as is now generally admitted, by auscultating with the stethoscope, at the left and at the right second intercostal space close to the sternum. These two sounds may be called simply the aortic and the pulmonic.

Of the three systolic sounds, one emanates from the mitral, or the left auriculo-ventricular, and another from the tricuspid, or the right auriculo-ventricular orifice. The third systolic sound has no connection with the orifices of the heart, but is produced by the impulsion of the organ against the thoracic wall. These three sounds may be designated the mitral sound, the tricuspid sound, and the sound of impulsion. The facts stated in my prize essay in 1858 seemed to me then, and they seem to me not less now, to demonstrate the existence of these three distinct elements of the so-called first sound of the heart, and that each may be studied separately. I refer to the essay for the facts, not reproducing them in this paper. I will, however, state where the three sounds are to be separately studied.

Auscultating with the stethoscope (the binaural is to be preferred) over the apex, the intensity, the greater length and the distinctive quality, (which may be called *booming*<sup>1</sup>) of the so-called first sound, as compared with the second sound, (the pulmonic and aortic sound combined) are due to impulsion, that is, the movements of the apex upon the thoracic wall. The sound of impulsion is, therefore, to be studied in this situation. As a rule, this sound does not extend beyond a limited area around the apex. The mitral sound is heard, distinct from the sound of impulsion by moving the stethoscope to the left of the apex in the fourth intercostal space, for a distance sufficient to eliminate completely those characters of the so-called first sound which are referable to impulsion. The maximum of the intensity of the mitral sound is at the situation where the characters of impulsion are eliminated, and generally this situation is but a little removed to the left of the apex-beat. The tricuspid sound is heard distinct from the sound of impulsion, at the interior border of the heart, above the

<sup>1</sup>The word *booming* was applied by Walshe to the distinctive quality of the first sound as heard over the apex. This now has been applied, but inappropriately to the second sound.

xiphoid cartilage. Its maximum of intensity is at the situation where the characters of impulsion are eliminated<sup>1</sup>.

Not going further into details relating to the existence of the five sounds, and the manner in which they are to be studied separately, I proceed to offer remarks on the clinical study of each one of these sounds under a distinct heading.

1. *The Aortic Sound.*—Incompetency of the aortic valve is generally, if not invariably, separated by a regurgitant murmur. It will, however, be admitted that this murmur, whatever may be its intensity and its characters in other respects, is, in itself, evidence only of the fact of incompetency. The murmur furnishes no criteria for determining the extent to which the valve is damaged. The incompetency may be so slight as to be innocuous, or it may be either small, moderate or great. Information on these points is furnished by either the unaffected intensity of the aortic sound, or the degree of its diminished intensity, and, in some instances, its absence. The importance of the study of the aortic sound, with reference to the gravity, or otherwise, of aortic lesions, is recognized now more than it was twenty-six years ago; but it is still inadequately appreciated by many practitioners. The abnormal weakness of the aortic sound may be estimated by comparison with the pulmonic sound, if the latter be neither intensified nor weakened by morbid conditions pertaining either to arterial tension of the pulmonary artery, or to an increased or diminished power of the right ventricle. A more reliable comparison is with the power of the left ventricle as represented by the sound of impulsion and the apex-beat. The co-existence of increased power of the left ventricle with notable feebleness or absence of the aortic sound, is especially significant as denoting considerable or great damage of the aortic valve.

Murmurs are sometimes spoken of as if they were modifications of the heart-sounds. The latter are always distinct from the former, the murmurs being either added to, or replacing the sounds. If the aortic sound be present, owing to its distinctive valvular quality, it may be recognized, notwithstanding the intensity or other characters of an associated murmur. Rarely, if ever, is the sound drowned by a murmur, however loud or rough, or musical.

The aortic sound is weakened by other causes than lesions affecting the aortic valve. Weakness of this sound is proportionate to the diminution of blood sent into the aorta with the systole of the left ventricle, in consequence of either mitral obstruction or regurgitation, or of both combined. The significance of weakness of this sound is greater in proportion as it is in contrast with the force with which the left ventricle contracts. As evidence of the degree of obstruction or of regurgitation, or of both com-

bined, weakness of the aortic sound, although more regarded now than twenty-six years ago, does not as yet receive the attention which its clinical importance claims.

Abnormal increase of the intensity of the aortic sound is referable to two causes, namely: increased power of the systole of the left ventricle, and increased blood-pressure or tension of the systemic arteries. With regard to the first of these causes, there can be no question as to its agency. Abnormal loudness of the aortic sound is a sign of hypertrophy of the left ventricle, and, to a certain extent, a criterion of its degree, assuming, of course, integrity of the aortic and the mitral valve. Attention has of late been directed especially to an increased intensity of this sound as a criterion of arterial tension, and this latter condition, at the present time, holds an important place in pathology. Clinical observations have led me to distrust the value which some observers appear to attach to the intensity of the aortic sound as evidence of arterial tension. The intensity of this sound varies considerably in different healthy persons. Its relative intensity, as compared with the pulmonic sound, in health, is variable. Exceptionally it is less intense than the pulmonic sound.<sup>1</sup> It is certain that in some cases in which arterial tension is supposed to be an important pathological element, namely, in cases of contracted kidneys, the intensity of the aortic sound is not increased.

The following case is illustrative of this fact:—A patient, about 50 years of age, had had three attacks of uræmic convulsions. The quantity of urine was large, of low specific gravity, slightly albuminous, and contained hyaline and granular casts. He had no cedema except slightly below the eyes. The heart was a little enlarged, and there was no cardiac murmur. The pulmonic sound was distinctly louder than the aortic, the difference in pitch showing that the louder sound was the pulmonic and not the aortic.

In this instance, arterial tension was to be inferred, but, notwithstanding slight enlargement of the heart, and the absence of any evidence of a pulmonary affection, the aortic sound was apparently not increased in intensity.

The recent experiments by Dr. Charles L. Roy, reported in the *London Lancet*, Jan. 26, 1884, show that abruptly cutting off the supply of arterial blood to considerable portions of the body, by ligating large arteries, produces but an extremely slight effect upon the blood-pressure or arterial tension.

Reliance on the intensity of the aortic sound, irrespective of hypertrophy of the left ventricle, as evidence of arterial tension, or on the weakness of this sound as evidence that arterial tension does not exist, must, as it seems to me, lead to erroneous conclusions.

2. *Pulmonic Sound.*—Forty or more years ago, Skoda called attention to an increase of the intensity of the pulmonic sound as a sign of hypertrophy of the right ventricle, in cases of mitral lesions involving

<sup>1</sup>To obtain the maximum of intensity of the mitral and the tricuspid sound, the stethoscope is to be moved gradually from the borders of the heart until the sound becomes purely valvular, that is until the characters which belong to impulsion are eliminated. It is easy to find the situations where the purely valvular sounds have their maximum. These situations vary somewhat in different persons in health, and still more in cases of disease. To designate certain circumscribed places as mitral and tricuspid ones, as is done by some writers, is therefore inaccurate and leads to error.

<sup>1</sup>In these exceptional instances, the relatively less intensity of the aortic, as compared with the pulmonic sound, may be due to a transient excitement of the heart's action. As will be stated under the head of the pulmonic sound, the intensity of this sound is greater than the aortic when the heart is functionally excited.

either obstruction or regurgitation, or both combined. The intensity of the pulmonic sound is undoubtedly increased by hypertrophy of the right ventricle, but there is another and perhaps a more important factor in the production of this sign, to which attention has been more recently directed, namely, increased blood pressure within the pulmonary arterial system. That notably increased intensity of the pulmonic sound may be due to this factor, irrespective of hypertrophy of the right ventricle, is certain—a fact which explains the persistence of the sign after the right ventricle, in connection with mitral lesions, has become weakened by dilatation.

The pulmonic sound is more or less intensified whenever there is obstruction to the free circulation of blood through the lungs, the co-existence of hypertrophy of the right ventricle not being essential. It is a sign present in cases of pneumonia, capillary bronchitis, asthma, pulmonary oedema, pleurisy, with effusion and hydrothorax. To a certain extent, the degree of increased intensity may be taken as a criterion of the amount of obstruction.

Increased functional activity of the heart intensifies the pulmonic, more than the aortic, sound. The greater intensity of the pulmonic sound is referable chiefly to increased blood pressure within the pulmonary arteries. When the heart is excited by either active exercise or a moral emotion, the pulmonic sound is especially intensified. More blood is propelled into the lungs than can readily pass through the capillaries. If the accumulation of blood in the pulmonary vessels exceed a certain limit, there is dyspnoea; but within this limit the blood pressure is sufficient to intensify the pulmonic sound.

I am not prepared to say how much significance pertains to diminished intensity of the pulmonic sound, as a sign of tricuspid regurgitation, or of the very rare lesions causing tricuspid stenosis, and, also, of weakness of the right ventricle from dilatation or from fatty degeneration. My colleague, Professor Janeway, has called my attention to the frequent absence of the pulmonic sound when, from lesions at the aortic orifice, the aortic sound is abolished. I am not prepared to explain this clinical fact. Of course, damage or destruction of the pulmonic valve must either diminish the intensity of the pulmonic sound or abolish it; but lesions producing these effects are so rare, that the clinical study of this sound, in connection therewith, is not easily pursued, and has but little practical importance.

3. *Mitral Sound.*—The mitral sound, disassociated from the sound of impulsion, is short and purely valvular, like the pulmonic or the aortic sound. Its intensity varies considerably in different healthy persons.

Insufficiency of the mitral valve is represented by a murmur, which, like an aortic regurgitant murmur, simply denotes the existence of lesion, without giving information concerning the importance of the lesion, that is, whether the regurgitating volume of blood be small or large. An abnormal weakness of the mitral sound and its absence, are signs which supply, in a measure, this information. These signs thus have a practical value in connection with mitral lesions.

This statement applies equally to mitral regurgitant and to mitral obstructive lesions. Weakness or absence of the mitral sound associated with either a mitral diastolic or a presystolic murmur, shows that the obstructive lesion either interferes to a greater or less extent, or abolishes the valvular movements which produce the sound.

Exclusive of mitral lesions, the mitral sound may be weakened in consequence of feebleness of the systole of the left ventricle from dilatation or fatty degeneration. Overfilling of the left ventricle as a consequence of aortic regurgitation, has the same effect, irrespective of the force with which the left ventricle contracts. The explanation is, that, under these circumstances, the excursion movements of the mitral curtains may be slight or *nil*, owing to the fact that at the time of the ventricular systole they are not only floated away from the inner surface of the ventricular wall, but are closely in coaptation with each other. When this is the case, the tension of the curtains produced by the pressure of the intra-ventricular blood in the systole, occasions little or no sound.

The intensity of the mitral sound, of course, other things being equal, is proportionate to the extent of the excursion movements of the mitral curtains. If the stethoscope (binaural) be placed directly upon the heart of an animal of considerable size, (*e. g.*, the sheep), the circulation being maintained by artificial respiration, the mitral and the tricuspid sounds are extremely loud and purely valvular, that is, without any sound of impulsion. Both the mitral and the tricuspid sounds have somewhat of a similar intensity in certain cases of anæmia when the heart is excited by exercise or mental emotion. The explanation, as I suppose, relates to the comparatively small quantity of blood within the ventricle when the ventricular systole takes place, together with the quickness and force with which the ventricle contracts.

All who are familiar with the mitral presystolic murmur, must have remarked an intensity of the sound which occurs at the termination of the murmur, that is, the mitral sound. This presystolic murmur, with the rough, vibratory character which it usually has, I believe denotes adherence of the mitral curtains to each other, leaving a bottom hole-like contraction, the mitral curtains remaining flexible. Now, owing to the contracted orifice, the left ventricle, at the time of its systole, is poorly supplied with blood. The movements of the mitral curtains, which I have called the excursion movements, are considerable and the curtains remaining flexible, the conditions are favorable for intensity of the mitral sound. Contrary to the statements generally made by writers, a mitral regurgitant murmur, under these circumstances, is not infrequently wanting. If a mitral regurgitant murmur be present, and the mitral sounds have a notable degree of intensity, it is certain that the regurgitant stream of blood cannot be large, or, in other words, the mitral incompetency is not great.

4. *Sound of Impulsion.*—In healthy persons the sound of impulsion generally predominates over the mitral and the tricuspid sound, at the apex and over

the body of the heart. The intensity of this sound varies much in different healthy persons. The abnormal modifications of this sound are, increase of its intensity, on the one hand, and on the other hand, either diminished intensity of the sound or its absence. It is to be noted that this sound is not like the valvular sounds endocardial but exocardial; and, like the exocardial murmurs, however loud the sound may be within the præcordia, it is not transmitted beyond the præcordial region. As we can imitate a pericardial friction murmur by placing the palm of the hand over the ear and rubbing gently the dorsal surface of the hand with the finger, so we may imitate the sound of impulsion by pushing the finger with more or less force instead of rubbing gently; and by light percussion with the finger, the sign, known as metallic clic (*tintement métallique*), is perfectly imitated.

It is the increased intensity of the sound of impulsion which gives to the first of the two heart-sounds (as they are commonly reckoned), its loudness, its length, and its booming quality, in cases of cardiac hypertrophy. The sound of impulsion predominates over the mitral sound, whereas, the latter is intensified and predominates when the action of the heart is functionally excited.

Diminished intensity of the sound of impulsion is denoted by diminution of the length and of the booming quality of the so-called first sound of the heart, the mitral sound predominating. The sound of impulsion is lost when the mitral sound is alone heard over the apex and body of the heart. Diminution and extinction of this sound, are signs of weakness of the ventricular systole from dilatation, fatty degeneration and the softening incident to certain infectious diseases. Clinical observers long since attached practical value to these signs, but their importance is perhaps not yet sufficiently appreciated by practitioners in general. Their value in the treatment of fever as bearing on the use of alcoholics and other cardiac stimulants, were especially emphasized by Stokes.

Removal of the heart from contact with the chest wall, in cases of pericarditis with effusion, abolishes the sound of impulsion, the mitral and the tricuspid sound alone accompanying the ventricular systole; and when the sound of impulsion is wanting, as a rule the aortic sound at the apex is louder than the mitral. The sound of impulsion is inappreciable in listening to the foetal heart; the so-called first sound is short and valvular like the second sound.

5. *Tricuspid Sound*.—Weakness and the abolition of the tricuspid sound in consequence of lesions affecting the tricuspid valve and orifice, are not signs of much practical value, owing to the infrequency of lesions in this situation.

Enfeebled action of the right ventricle from fatty degeneration, must occasion abnormal weakness of the tricuspid sound. A thrombus of considerable size formed within the right ventricle, may weaken and extinguish this sound. When the right ventricle is overfilled with blood at the time of the ventricular systole, the tricuspid sound must be weakened, for

the same reason that the mitral sound is weakened when the left ventricle is overfilled.

Hypertrophy of the right ventricle intensifies the tricuspid sound. This is a better sign, by far, of hypertrophy of the right ventricle, than an increased intensity of the pulmonic sound, inasmuch as the latter involves, as an additional factor, increased blood pressure within the pulmonary vessels. In the rare instances of notable hypertrophy of the right ventricle resulting from congenital stenosis at the pulmonary orifice, the tricuspid sound must be considerably intensified. While I am writing, I have before me a specimen in which the pulmonary orifice is contracted to the size of a goose-quill. The right ventricle is hypertrophied, so that its walls are as thick as those of the left ventricle in healthy subjects, and the walls of the left ventricle are not much thicker than those of the right ventricle in healthy subjects, the normally relative size of the two ventricles being thus reversed. The tricuspid valve is normal. The specimen was from the case of a patient, aged 19 years, who died of scarlet fever, and who complained of no subjective symptoms denoting cardiac disease, except panting on active exercise. A very loud systolic murmur had been observed, and it was supposed to be a mitral regurgitant murmur. The patient was not under my observation, but it is certain that the tricuspid sound must have been much intensified; and this sign, taken in connection with the probable absence of the pulmonic sound, inasmuch as the segments of the pulmonic valve were perfectly rigid, would have been valuable as indicating the true seat of the valvular lesion.

In conclusion, the chief object of this paper, as stated at the outset, is to bring again, after the lapse of twenty-six years, to the attention of the profession, the importance of the clinical study of the heart-sounds, as distinguished from the heart-murmurs. As a motive for the preparation of the paper, I confess a desire to refer to my essay to which was awarded a prize by the American Medical Association at the meeting in Washington in 1858. I believe I may assume to have been, at that time, a pioneer in the clinical study of the heart-sounds in health and disease. This study, since that date, has not received from writers and practitioners that attention which its importance claims. If I mistake not, the majority of those who avail themselves of the information to be obtained by auscultation, do not take pains to interrogate separately the sounds emanating from the heart, but, for the most part, limit their auscultatory observations to the murmurs. In the latest French work on the diseases of the heart, published in 1883, the heart-sounds are hardly recognized as furnishing any important signs in diagnosis.<sup>1</sup> Having said this, I would add that in the German work on pathology and therapeutics by Eichhorst, published in 1883, and in the article by Guttman in the Real Encyclopædia, 1881, abnormal modifications of the heart sounds are fully recognized among the data for the diagnosis of cardiac affections. I do not doubt that the time will come

<sup>1</sup> Traité clinique et pratique des maladies du cœur, per Michael Peter. Paris, 1883.

when all clinical observers will consider the practical information to be derived from the heart sounds as not less essential than that furnished by the murmurs, taken in connection with the signs obtained by percussion and palpation.

#### DISCUSSION.

Dr. Richard McSherry, of Baltimore, Md., said:

Mr. Chairman: As an indication of changes in medical acquirement, I may say that when I was a student of medicine in Philadelphia—not very recently—I heard a distinguished professor say he knew nothing of diseases of the heart. The gentleman was very distinguished, certainly not less known to fame, in his day, than his successor, the present honored president of our great Association, but he had not kept up with the progress of his time, for even then the works of Arenbrugge, Corvisart, and Lænnec had taught new and important lessons to the medical world, with which the rising doctors were quite familiar. They at least could not afford to cast ridicule on the “conjuring rod,” as the stethoscope was derisively called by some of the “Conscript Fathers” of the profession.

The pulse had been the pre-eminent guide, especially in accordance with the teachings of Bordeu and his followers, from whose investigations and speculations, our fathers were led to believe that through it, they could not only take the measure of the circulation, but that it was a gauge of all diseases in all their modifications. Sphygmics, without the aid of the sphygmograph, had almost the attention since given to auscultation and percussion. The learned doctor of the last century, with watch in one hand while the fingers of the other rested on the radial artery, seemed to find in the throbbing of the artery as reliable an indication of the condition of his patient as of the time of day from the hands of his ponderous time piece.

Now the pulse was, and is, a good indicator, but yet like any other one source of information in regard to the vital machine, it may lead to many fallacies, so many, that its real importance came to be underestimated.

Our great master, Lænnec, attached little importance to it by reason of its great uncertainty, saying the ignorant could deduce from it all sorts of indications, while the cleverest could get from it but equivocal conjectures. In truth we know it may deceive us, taken alone, therefore we only rationally use it in conjunction with various other indices of physiological or pathological conditions. It used to be, more than now, a guide in regard to blood-letting, but an improved acquaintance with the central organ of the circulation showed sometimes a strong pulse with a weak heart, and again a vigorously acting heart with a weak pulse (*fictitiè debilis*) and it is from the state of the central organ, and not from the action of the arteries, that we may draw conclusions, *pro* or *con*, in regard to depletion.

We can herein find a scientific guide to practice more reliable than the empirical.

We may now begin to contrast the acquirement of

the professor of the earlier half of the century, with that of the professor of the latter half, the one declaring that he knew nothing of the diseases of the heart, the other showing that no diseases of any other organ or viscus are subject to equally exact diagnosis. I know of no more striking evidence of the progress of medical science.

But although diagnosis is practicable, it is still environed with difficulties. We cannot get patients to correspond exactly with elastic models, which represent one age, one size, and average chest walls. Of two successive patients, one may be an enormously fat elderly lady, and the next a slim youth whose chest walls have but a fraction of an inch between the ear of the auscultator and the heart of the patient.

There are so many accidental perturbing causes, too, to make diagnosis not impossible but difficult. This may be particularly perplexing when there is great functional disturbance with an early stage of organic disease. I was called, a few years ago, to Mrs. S., from New York, at a Baltimore hotel, who had come to reside in our city. I found the lady suffering with great dyspnoea, due to pulmonary congestion with evidences of mitral insufficiency and regurgitation. She appeared to be alarmingly ill. She had passed the menopause by some months as I learned upon inquiry. I prescribed according to the needs of the case, and when questioned by some lady boarders upon leaving the house, I spoke of disease of the heart. In a day or two she was greatly relieved, but in the meantime there occurred a flow rather to be called metrorrhagia than menorrhagia. The great general perturbation subsided rapidly, and upon its subsidence the heart's action showed comparatively little disturbance. I heard from some friends afterwards that my diagnosis had been hardly commented on by the inquisitive critics of the house. I soon lost sight of the lady, who took another medical attendant. About two years subsequently, I saw announced in the morning papers the sudden death of Mrs. S., after a few hours' illness. Whether there was another pulmonary congestion or other cause of death, I never learned, nevertheless I could not doubt that organic disease of the heart was the efficient cause.

It appears, according to Porah's statistics, that in gravo-cardiac disorders, as they are called, more than two-thirds of the cases are those of mitral stenosis, mitral insufficiency, or the two combined. Women affected thus are in great peril from pregnancy and child bearing, and they are advised to refrain from marriage.

“Landouzy mentions the case of a young girl who had been in the hospital under his care, and whom he had advised not to marry, but who disregarded his advice, married, became pregnant, and after a miscarriage, died suddenly in an attack of asystole.” (*Medical News*, April 10, 1884.) In Mrs. S.'s case, although the mother of several children, I presume the perturbation of her general organism was equivalent to that of pregnancy and miscarriage.

We have anæmia (spanæmia) modifying heart sounds, thus it is urged that an elastic contraction of

the orifice of the aorta in anæmic subjects causes a morbid systolic sound at the base of the heart, where no valvular disease can be detected upon post-mortem examination.

Childhood and old age produce their modifications. The evidences of hypertrophy and dilatation are often observed in growing children, which may pass away entirely about the time of puberty, a fact observed by Laennec, and which, from his own observation, induced Dr. Flint to urge caution in regard to giving a prognosis unfavorable and alarming in such cases.

The sharp accentuation of the second sound without valvular disease, or any disease, except in some remote organ, is a matter of perpetual observation. In one of our dispensary patients, but a short time ago, this sign led to an examination, *inter alia*, of the state of the kidneys. A moderate attack of nephritis, with albuminuria, was soon detected, and soon relieved. The cure of the primary disorder corrected the morbid heart sounds which had caused investigation as to the seat of remote disease.

Old age and deep-seated infirmities greatly modify sounds and impulse. I shall never forget my surprise, in my early professional life, upon finding a greatly enlarged heart upon the post-mortem examination of an old man-of-war's man, whom I had seen a few days before death without diagnosing such degree of hypertrophy.

Upon referring to the text which was then my guide, I found the following explanatory passage:

"The heart's impulse in hypertrophy and dilatation is almost lost in some cases of pleurisy, asthma, œdema of the lungs, or of congestion before death." (*Laennec*). The modifying conditions were all there, and as I had had no opportunity of tracing the progress of disease with the patient, but only saw him when near his end, the oversight seemed comparatively pardonable.

Besides the proper heart-sounds, it may not be amiss to refer to sounds induced by the heart's action indeed, but extraneous. Thus, the clinician often notices puffs or whiffs, heard when the contraction of the ventricles forces air out of the portions of lung lying between the heart and the thoracic walls.

There is a condition in which all of the five normal sounds may be heard, but not in the right place. I had an opportunity of seeing, a little more than a year ago, a case of dextro-cardia under the care of my friend, Prof. Chew, at the University of Maryland Hospital.

This was an illustrative case of traction from the left to the right side. The patient, a sailor, had been under my care some months previously with effusion in the right pleura, and moist râles in the right lung. The heart was not then displaced. When subsequently re-admitted into the hospital, on inspection, the intercostal spaces on the right side were depressed, the ribs drawn together, and the lower part of the chest wall sunken in. Measurement showed that the semi-circumference of the thorax was one inch less on the right than on the left side. The impulse of the heart could be seen and felt near the right nipple, the apex beat being a little to the right of the mammary

line. There was perfect dullness on percussion when I used the hammer and pleximeter over the right side; respiratory murmur was replaced on that side by subcrepitant and bronchial râles audible as far as the base of the chest, and at two points the sounds indicated the formation of vomicæ. On the left side there was a somewhat exaggerated percussion note, and the respiratory murmur was harsh and puerile, though intermingled to some extent with moist râles. \* \* \* Reasoning then by the power of exclusion, we have remaining as the most probable cause of the phenomenon that to which I think it should be assigned, viz.: the contraction of the right lung and right pleura, the lung being adherent to the chest wall; and as it had undergone contraction, together with the thickened pleura it had drawn upon the mediastinum, and thus effected the displacement of the heart. (For the full account of this rare and interesting case see Prof. Chew's report in Trans. Med. and Ch. Faculty of Md., 1883.)

Every one has probably heard various neurotic (perhaps sometimes choreic?) sounds different from any ordinary physiological or pathological sounds which are difficult to express or to explain. Among these anomalies, one gentleman informs us that he has heard with a robust and nervous girl over the heart above and to the left, a noise very like the clucking of a young chicken, synchronous with the systole. The sound was loud enough to be heard by the bystanders.

In the case of a vine-dresser, an ex-soldier, at 41, there was a hard intermittent sound in the precordial region, not coincident with the systole, loud enough to mask all the normal sounds of the heart, and to be heard at some metres from his person. It began suddenly about a month before the examination. The man thought some living animal had taken up its abode within his chest. The nearest approach to the sound that the auscultator could suggest would be expressed by the lettres grrouou, 75 times in a minute, in a low but harsh childish voice.

No change was made by acceleration or suspension of the respiration.

The reporter of these two cases attempted no explanation, saying that such phenomena would have to be classed with so many others of a transient nervous, or anomalous character, which are never explained even by autopsy. (*Dictionnaire Annual 1867, from Jour. de Med. de Brux. par M. Putégnat.*)

Mr. Chairman, when I consented to make remarks upon Prof. Flint's paper, which he was to offer to this Section, I knew perfectly well that little room would be left for addition or subtraction by commentators. This must be my apology for a rather free handling of the subject, instead of adhering closely to the text presented by so eminent an authority.

Dr. J. S. Lynch, of Maryland, referred to the importance of alteration of the aortic sound in Bright's disease. He thought that a large part of the booming character of the first sound was due to the vibration of the aortic walls at each systole of the heart. He could not understand why increase in the blood pressure in the large arteries should not increase the intensity of the aortic sound. Dr. F. had stated

that the intensity of the pulmonary sound was increased by increase of blood pressure in the pulmonary arterial system. Why should not the same result follow increased pressure in the systemic arteries?

Dr. Garland, of Massachusetts, stated that it was often difficult to decide whether a murmur was coincident with, or replaced the cardiac sound. This is important to decide in diagnosis. He spoke of the importance of getting rid of the respiratory movements in trying to determine this point. A murmur will sometimes disappear when the breath is held for a moment. A deceptive murmur is sometimes heard at the base of the heart, to the right or the left of the sternum and up and down its border. It is systolic in time. The peculiarity of this murmur is that it disappears on full inspiration.

Dr. Donaldson thought it a question whether or not the impulse of the heart could produce a sound. Muscular contraction does not produce a sound, or at least not of sufficient force to be heard without very delicate instruments. Experiments have shown that the first sound is produced by the closure of the mitral and tricuspid valves. Where these have been prevented from closing the sound has been absent. In regard to presystolic murmurs, Dr. Leamy, of New York, has claimed with much plausibility that they are not really presystolic, but that they occur at the beginning of the systole.

Dr. Flint in concluding the discussion, said that many of the points advanced were in reference to the mechanism of the sounds and in regard to murmurs. These he had not touched on in his paper, and therefore he waived any consideration of them at this time. His paper was concerned solely with the clinical aspects of the heart sounds. In regard to the Germans having taught the subject in the same manner as he did, he would call attention to the fact that 26 years ago he had pointed out this plan. He could not explain why the blood pressure acted differently in the pulmonic and systemic arteries, but such appears to be the fact.

## THE MULTIPLE WEDGE PRINCIPLE IN THE TREATMENT OF ORGANIC STRICTURES OF THE URETHRA.

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It is now nine years since I published, in the *Medical Record* of New York, my application of the multiple wedge principle to the treatment of organic strictures of the urethra.

To quote from that paper: "It ought to be a self-evident proposition, that it is easier to introduce singly the component parts of a wedge, than to introduce it as a whole. Take, for example, a No. 12 conical bougie, even capillary at its point, and attempt to introduce it into a stricture: Where is the point of friction and resistance? Is it not around

the entire circumference of the stricture? Diminish the size of the instrument, and in a direct ratio, you diminish the amount of resistance. After having passed into a stricture one of these whalebone guides, the second has to overcome the friction and resistance of but half the circumference of the stricture, and the line of contact with the other bougie. Now that we have passed these two, we have a groove in front and behind them through which we can readily pass the third and fourth, having now to overcome the resistance of but one-fourth the circumference of the stricture and the two lines of contact with the instruments already in position."

Since that time, I have treated by this method some twenty-five or thirty cases with invariable success. There was not, in any case, marked febrile disturbance; but in some few cases there was sufficient constitutional irritation to cause me to suspend the treatment for a day or two.

It is my habit to give, as a prophylactic, full doses of quinine, say fifteen to twenty grains per day.

I will give in detail one case, as it fully illustrates my method.—Capt. H. D. F. presented himself for treatment of a stricture of twenty years' duration.

His urine was always voided with difficulty, and the act usually consumed from twenty to thirty minutes of his time.

There was marked cystitis, as evidenced by the abundant mucus and the decomposition of his urine. The fetor of his urine was so great as to necessitate the immediate removal of the "pot de chambre." His strictures had been ruptured twice during the year 1866, and once in 1867, with Holt's divulsor.

Since the last operation, he had wholly neglected himself. March 16, 1879, after a prolonged and tedious effort, I succeeded in *worming* through his stricture one of the smallest-sized French filiform bougies, and securely tied it in *situ*. Five days afterwards I succeeded in introducing a second by the side of the first, but the cicatricial tissue was so dense and unyielding, that it was not until the fifteenth day that I could pass the third.

On the twenty-fifth day I readily passed the fourth and fifth bougie.

On the twenty-eighth day I introduced the sixth.

At no time was there any hindrance to the passage of the urine.

Feeling assured that now the caliber of the urethra would admit a good-sized instrument, I attempted to remove the bundle of filiforms. In this I was disappointed, for a sufficient amount of concretion had formed to prevent their removal *en masse*. The string binding them together was cut, when they were readily removed, until the last (the first one introduced) which required some little force to withdraw it. Three inches of its distal end had been covered by a *phosphatic* concretion. About one-half the deposit had slipped off the bougie in its withdrawal. (In subsequent cases I have obviated this *contretemps* by removing from time to time those first introduced and replacing them by new instruments).

My patient was now so nervous that he would not permit me to use a sound.

On the twenty-ninth day, retention of urine having