to the second objection, we must remember that there often is a deficient supply of lime in the water, and that in consequence of this, we may be sure that there is a deficient quantity of it in any food the child may have, either from the mother or cow; that the mal-assimilation to a certain extent is dependent upon the presence of excess of lactic acid in the milk, which has generally been kept for some time, and that lime tends to counteract this; and, again, we have reason to believe that the phosphate of lime tends to control defective and perverse nutrition which is present in rickets; and as a proof of its efficacy I may recall what I have already stated:—1st. That rickets has been produced in animals by feeding them on food free from phosphate of lime. 2nd. That fractures purposely produced in animals united much more readily in those to which the phosphate of lime had been given than in those to which the remedy had not been given.

Cod-liver oil has been strongly recommended as a remedy in this disease, and is a very valuable one. It should be commenced in very small doses, for it is very apt in these cases to give rise to acidity, to derange the stomach, and produce griping pains and diarrhoea. I think that we are too fond of giving cod-liver oil, not only in this disease but in others, in too large doses, in doses which the tender stomach cannot tolerate, and that if we commenced by giving small doses, then increased the quantity if it is well tolerated, we should find that our patients would derive much more benefit than they do, and that the injurious effects of the remedy upon the intestinal canal would be avoided. If oil globules are found in the stools, it is a sign that all the oil prescribed is not digested, and the excess is hurtful.

Wherever there is much growth or nutrition going on, wherever there is rapid cell growth in health or in disease, there is abundance of fat, showing that it is necessary for its promotion. In these respects it is very like the phosphate of lime, but frequently it cannot be tolerated. Fat must then be given in other forms, as cream &c. Among the rich the disease is produced in a somewhat similar manner. In this disease patients should not be encouraged to walk early. Let the general health be improved by diet, medicine, fresh air, baths, &c., and let the bones solidify before the weight of the body is allowed to be put on the lower extremities or the upper part and spine upon the upper extremities, or the bending of bones will be increased. Of course, I say nothing about the surgical treatment of such disease. The compound syrup of the phosphate of iron is a remedy which may be prescribed with great advantage, more especially if the lips are pallid, as they generally are in these cases.

ON THE

MITRAL PRESYSTOLIC AND A MITRAL DIASTOLIC HEART-MURMUR.

BY AUSTIN FLINT, M.D.,

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THE LANCET of Jan. 27th, 1883, contained an abstract of a clinical lecture by me on the Occurrence of the Mitral Direct or Presystolic Murmur without Mitral Lesions. In connexion with that lecture a specimen was exhibited in which aortic lesions with valvular insufficiency existed without any mitral lesion. In the case which furnished this specimen, a well-marked mitral direct or presystolic murmur had been observed by others as well as by myself. In 1862 I reported several cases in which a well-marked mitral presystolic murmur had been observed, and after death no mitral lesions were found, free aortic regurgitation having existed in all these cases. So far as I know, instances of a presystolic murmur without any mitral lesion had not been previously reported, and I have not met with a report of similar instances in medical literature up to the present date. In the lecture referred to, and in other publications, I have ventured to explain the mechanism of the murmur when produced without mitral lesions. Dr. Balfour, in his work on Diseases of the Heart, after stating that a presystolic murmur always denotes constriction of the mitral opening, refers to my observations in a footnote, as follows:—"I have no hesitation in making

this statement, notwithstanding that Professor Flint says, 'I have recorded three cases in which this (presystolic) murmur was loud, and the mitral valve was found, post mortem, normal. In each of these cases there were aortic lesions, rendering the aortic valves insufficient.'¹ Obviously Flint had not timed these murmurs correctly, for a murmur depending on aortic regurgitation is always diastolic, and, therefore, has a rhythm very different from a presystolic murmur. But even without timing these murmurs, they have each of them such a distinctly different character, that no one perfectly acquainted with the presystolic murmur could ever mistake a diastolic aortic murmur for it."² In another place³ the author refers to me in connexion with the discrimination of a presystolic murmur, as follows:—"Nay, want of precision in this matter has led even a man of so much experience as Professor Flint to describe as presystolic murmurs which must have been diastolic in rhythm."

Now, I should not take pains to substantiate my ability to discriminate a mitral presystolic from an aortic regurgitant murmur, were it not that upon this ability rests the validity of my statement (which I suppose to be original) that, in connexion with free aortic regurgitation, a mitral presystolic murmur may exist without mitral lesion. In view of this consideration I hope it will not seem indelicate for me to state some facts in order to show that I may fairly claim to be able to recognise, by its rhythm and distinctive characters, a mitral presystolic murmur. I was the first, as I believe, to recognise this murmur in my publication on this side of the Atlantic. I referred to it in a prize essay on "The Clinical Study of the Heart-sounds in Health and Disease," published in 1852. It was embraced among the organic murmurs in the first edition of my work on the diseases of the heart, published in 1859, and more fully described in the second edition published in 1870. In an article published in the *American Journal of Medical Sciences*, July, 1862, on "Cardiac Murmurs," after stating that for many years after I had begun to devote special attention to cardiac operations I committed the mistake of confounding it with the mitral regurgitant murmur, seven pages were devoted to an account of the mitral presystolic murmur. The differential points involved in the discrimination of the latter from an aortic regurgitant murmur were stated fully. The characteristic quality was described, and the name "blubbery" was applied to it. Dr. Balfour refers to this article, but either he could not have read it with care, or he had forgotten it when he assumed my inability to recognise this murmur. The murmur is described in the first edition of my work on the Practice of Medicine (1866), as well as in all subsequent editions, also in my work on Clinical Medicine (1879), and in the first edition of my "Manual of Auscultation and Percussion" (1876). Moreover, for more than twenty years in clinical teaching I have been accustomed to point out this murmur to medical students and practitioners. My field of observation has been large. It is not an exaggeration to say that more than five hundred examples have come under my observation. Several examples may, at any time, be found either within the wards or in the out-door department of Bellevue Hospital.

Claiming, therefore, that I am sufficiently familiar with the mitral presystolic murmur to recognise it, I claim, in opposition to Dr. Balfour's statement, that this murmur does not invariably denote mitral lesion, but that in certain cases, when associated with aortic lesion, giving rise to regurgitation, it may exist, its characters being well marked when the mitral valve is perfectly normal, and I refer to the cases which I have reported as proof of the correctness of this statement.

Now, as to the mechanism of the presystolic murmur when not dependent on mitral stenosis. My explanation assumes that the murmur, as it is usually heard, is produced by vibration of the mitral segments. The characteristic quality supports this view of its production. The terms vibratory and blubbery express the characteristic quality of the murmur. It may be exactly imitated by throwing the lips or the tongue into vibration with the expired breath. Moreover, this method of its production may be demonstrated by the following experiment. Take an indiarubber bag with thin walls, of the size of a small orange. The little bags which when inflated form the toy balloons for children are well adapted for this purpose. Attach the mouth of the bag to the efferent tube of Davidson's or Higginson's syringe. At

¹ Lectures on Diseases of the Heart. Philadelphia, 1870.

² Second edition, p. 165.

³ Page 116.

the other extremity of the bag make a button-hole opening. Place the bag and the efferent tube of the syringe in a basin of water. Insert the afferent tube of the syringe in the same basin, or in another containing water. Force the water by compressing the bulb of the syringe into the india-rubber bag, and through the opening, and apply the pectoral end of the binaural stethoscope upon or near the bag. The vibration of the walls of the bag as the water passes through the opening gives a perfect imitation of the presystolic murmur, and to the touch a *frémissement cataire*.

Attributing the presystolic murmur to the vibration of the mitral segments, it is easy to understand that this vibration takes place when the segments are adherent to each other, leaving a button-hole opening, the segments remaining flexible. The *frémissement cataire*, so often associated with the mitral presystolic murmur, corroborates the explanation of the production of the murmur. It is also, as it seems to me, easy to understand that vibration of the mitral segments may take place without mitral stenosis, if, when the auricular systole takes place, the left ventricle be already filled with blood. This ventricle is filled with blood at that time if, added to the blood, which flows into the ventricle from the auricle before the auricular systole, there be a large or considerable regurgitant flow from the aorta owing to insufficiency of the aortic valves. The mitral segments must then be brought into close coaptation, and they are made to vibrate by the current which is forced between them by the contraction of the auricle. It is under these circumstances that the presystolic murmur occurs without mitral stenosis. With reference to this explanation, Dr. Balfour remarks as follows:—"Flint's theory that the regurgitant blood floats up the segments of the mitral valve and produces a practical constriction of the opening, is obviously no explanation. In the normal state of the heart 'the segments of the valve at the end of the ventricular diastole are so close as to be nearly in contact.'⁴ According to Flint's theory a presystolic murmur ought to be a constant phenomenon not only in all cases of incompetence, but also in health."

The question is not what ought to be, but what is the clinical fact. In the foregoing quotation it is stated that at the end of the ventricular diastole the segments of the valve are so close as to be nearly in contact; but in cases of aortic regurgitation these segments are not nearly, but absolutely, in close contact. Physiological experiments show that the mitral orifice may be fairly closed by injecting a liquid from the auricle into the ventricle, having previously ligated the aorta and coronary arteries. The exact similarity of the murmur when it occurs without mitral lesion to the murmur when it represents mitral stenosis, shows the mode of production to be the same—namely, by vibration of the mitral segments. The condition of the left ventricle at the end of the ventricular diastole in health differs widely from the condition when there is free aortic regurgitation; but why the presystolic murmur is not oftener present than it is in cases of aortic regurgitation, I am not prepared to explain.⁵ If, however, Dr. Balfour will direct his attention to the occasional occurrence of the murmur without mitral lesion in some of these cases, I doubt not he will, in the course of his continued clinical experience, meet with examples.

Dr. Balfour refers to a murmur produced by the current of blood from the left auricle into the left ventricle, in cases of stenosis, at the beginning of the ventricular diastole—that is, prior to the presystolic murmur. Peter, in his late work, describes this murmur as of two kinds—namely, *un bruit de souffle diastolique*, and *un bruit de roulement diastolique*, the latter being much more frequent in its occurrence.⁶ These murmurs are produced by the gravitation of the blood, owing to the situation of the auricle, and by a *vis a tergo* incident to auricular distension, before the muscular contraction of the auricle takes place. I have, as I believe, verified repeatedly these murmurs, mitral in their source, and preceding the presystolic murmur. They have been confounded with aortic regurgitant murmurs, from the fact that they are the same as regards rhythm. They are not, however, heard in the same situation as the latter—that is, at or just below the base of the heart. Their situation is at or just above the apex of the heart. The question arises,

By what name shall these murmurs be distinguished? The name mitral diastolic murmur, in contradistinction to the name mitral presystolic murmur, seems to me an appropriate and convenient designation.

NOTES OF THREE CASES OF ARSENICAL POISONING.

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IN my capacity of district medical officer to the parish of Liverpool I was called to see Thomas Higgins, aged forty-five, on Sept. 25th, 1883, as a probable case of fever. On examining him I found his temperature and pulse normal. He stated to me that he had diarrhoea and felt "sickish," and that he had been drinking for a couple of days. He smelt strongly of drink. I ordered him a full dose of castor-oil, to be followed by a mixture consisting of opium and solution of bismuth. I saw him again on the 29th, and found him weaker, and his temperature subnormal. I observed no direct evidence of drink on this second visit. In the interval between the visits he had taken miscellaneous food, and there was doubt as to his having taken the mixture, at least regularly. There was tenderness on pressure over the lower abdominal region. The patient complained of vomiting and "bad" diarrhoea. I gave directions to him to take a strict milk diet, and substantially continued the medicinal treatment. On Oct. 2nd, three days after my last visit, his wife called at the parish surgery, and told me that he had just died, and requested a certificate. I was surprised to hear of his death, as I had looked upon his ailment as a comparatively trivial one. I closely questioned her as to his symptoms before death. She stated that he had frequent "bloody stools," and that he died suddenly. I gave a certificate, assigning dysentery as the cause of death, although I had no definite opinion as to the exciting cause of the dysentery. On the evening of Oct. 3rd Patrick Higgins called on me, and stated that his brother, Thomas Higgins, had been heavily insured by his widow and her sister, Catherine Flannagan. On hearing this my suspicions were first aroused, and I directed the brother to call on the coroner, and to insist on an investigation being held. I called the following morning, and told the coroner that I had grave suspicions of foul play. The coroner declined to act himself, but gave me verbal permission to make a post-mortem examination to verify or set at rest my suspicions, and directed his beadle to accompany me. After examining the body as described below, with the assistance of Dr. Limerick, I gave directions for the funeral to be stopped, and placed a policeman in charge of the room. I may here mention some symptoms and circumstances immediately preceding death which were sworn to during the recent trial. The deceased spoke of figures on the wall and around his bed, and yet a few minutes before he died he refused a drink offered by Mrs. Flannagan, and subsequently took water offered by a stranger. He sat erect in bed whilst he drank it, and, having said "God bless you" to her, lay down and died. The vomiting, purging, and consequent thirst were marked symptoms for the last two days.

The post-mortem examination was made on Oct. 4th, fifty-eight hours after death. Rigor mortis had disappeared, and slight discolouration was noticeable. The conjunctivæ were free from any yellowish tinge. On opening the abdomen the following appearances were observed: The peritoneum over its entire surface presented its normal glistening character. On the anterior wall of the stomach, extending from the lesser curvature and nearer to the cardiac than to the pyloric opening, a reddish patch about four inches in area was seen through the semi-transparent walls of the stomach. The duodenum was intensely inflamed, and it was separated with difficulty from the pancreas and other adjacent structures. The intestines, which were remarkably free from contents, had patches similar to the one described in the stomach. There were six on the small and two on the large intestine, and these were scattered at irregular intervals over the tube. A most remarkable "cord-like" condition of the intestines was observed in parts for about two inches in length. There were three such cord-like con-

⁴ Pettigrew, Trans. Roy. Soc. Ed., vol. xxiii., Part 3, p. 796.

⁵ The explanation may be that for the production of the murmur without mitral lesion a certain degree of hypertrophy of the left auricle is requisite. Peter supposes that a presystolic murmur always implies more or less hypertrophy of the left auricle.

⁶ *Traité Clinique et Pratique des Maladies du Cœur*. Par Michel Peter. Paris, 1883.