ARGUMENTS

IN FAVOUR OF THE THEORY OF

DILATATION OF THE HEART AS THE CAUSE OF CARDIAC HÆMIC MURMURS,

AND OF

THE APPENDIX AURICULI SINISTRI BEING THE PRIMARY SEAT OF THIS MURMUR.

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THE valvular lesions which but a few years ago were almost the only recognised diseases of the heart, which seemed so difficult to diagnose, and to threaten life so seriously and mysteriously, are now-a-days readily discovered, differentiated, and their influence as mechanical hindrances discounted. All the interest and the mystery is now transferred to the state of the cardiac muscle and its nervous relations. And of all the problems we are thus brought to face, none is so simple as, and few more important than, the actual condition of the chlorotic heart, which has long been misunderstood.

I have been led to adopt the theory of dilatation of the heart as the cause of cardiac hæmic or chlorotic murmurs, because it seems to be the only one capable of reconciling the various discrepant hypotheses regarding the origin of these murmurs.

This theory is fraught with most important instruction on the one hand, and with a no less momentous warning on the other; because, if it be true, there is no longer any real distinction between functional and organic murmurs; and a murmur can only be looked upon as functional when it is found to have been curable. Moreover, if functional murmurs are really due to dilatation, as these murmurs are known to disappear—to become cured—in by far the larger number of cases, we thus obtain the important generalisation that dilatation of the heart is a curable disease. The curability of such affections becomes merely a question of degree; they are more or less curable, according to the length of time they have persisted, and the extent to which the dilatation has progressed.* This is the lesson with which this theory is fraught, and it involves the warning; because, if so-called functional murmurs are due to dilatation, and dilatation becomes incurable by persistence and extension, it is surely a most important matter that we should all take care that none of our cases of functional disease become organic by neglect. I have adopted the theory of cardiac dilatation as the cause of cardiac hæmic murmurs for three reasons: first, because it alone is capable of explaining rationally all the discrepancies in the prevalent theories of these murmurs; second, because it is thoroughly consistent with the results obtained by experiment; and third, because it is perfectly consonant with clinical experience.

1. *The theory of cardiac dilatation explains in a rational manner all the discrepancies in the prevalent theories as to the causation of hæmic murmurs.* The aortic orifice is the classic position of the hæmic murmur as described by Hope,† Bellingham,‡ Potain,§ and others, and of late reverted to by Marey.|| But this hypothesis is totally at variance with the now well known fact that the hæmic murmur, at its first appearance, though it is a basic murmur, is not propagated along the aorta or into the carotids, and has its position of maximum intensity in the neighbourhood of the pulmonary artery. On the other hand, Marshall Hughes's¶ idea, that the pulmonary artery is itself the seat of the murmur, is untenable, because there is no possible cause of murmur in the pulmonary artery which is not equally operative at the aortic orifice, or indeed more so; while the universally recognised basic position of the primary hæmic murmur completely excludes Parrot's** theory, that it originates at the tricuspid orifice. Stokes††

* How glad an "Eureka" this is to Senac's doleful wail: "A mesure qu'on pénètre dans les maladies du cœur, la médecine paraît plus stérile; que peut-on espérer des médicaments, par exemple, dans les dilatations du cœur?"—*Traité de la Structure du Cœur, de son Action, et de ses Maladies*. Paris: 1749.

† *A Treatise on Diseases of the Heart*, etc. London: 1839. P. 106, and p. 389.

‡ *Diseases of the Heart*. Dublin: 1853.

§ *Dictionnaire Encyclop. des Sciences Médicales*. Tome iv, p. 392.

|| *Physiologie Médicale de la Circulation du Sang*. Paris: 1863, p. 479.

¶ *Guy's Hospital Reports*. Second series, vol. vii, 1857, p. 161.

** *Archives Gén. de Médecine*. Sixième série, tom. viii, 1866, vol. ii, p. 158.

†† *Diseases of the Heart and Aorta*. Dublin: 1854, p. 533.

is the only British authority who recognises the possibility of the hæmic murmur being occasionally mitral in position; but Walshe* discards this, as being utterly inconceivable. Thus we have each of the four orifices of the heart described in turn by most competent authority, as the position of the hæmic murmur, and each of them discarded as incompetent because inconsistent with actual facts in individual cases. Considering the unanimity which prevails amongst all cardiac authorities in regard to organic murmurs, there can be but one possible explanation of this extraordinary discrepancy as to the hæmic murmur; and that is, that it is a murmur which may be audible in all the various positions described. It is impossible to accept any explanation that does not keep this in view and provide for it; and it is equally impossible to accept any explanation inconsistent with the ordinary laws of sound. It may seem absurd to make the latter stipulation; yet the laws of the propagation of sound are so incompatible with the theories propounded in regard to the hæmic murmur, that this very contrariety is in itself no trifling argument against the so-called functional character of the murmur. Thus Hayden,† who is the most explicit of all writers as to the propagation of the hæmic murmur, describes this sound as produced within the blood by friction of the blood-corpuscles against one another, by friction at the arterial outlets, and by the vibration thus produced in the walls of the heart and arteries. Yet he states that this murmur is best heard at mid-sternum, and "within a diffusion area of three to four inches"; that, though it may be heard here and there over the arteries, it is never propagated continuously along them, and that it is never heard at the apex. Nevertheless, he acknowledges that an organic murmur, produced, as he supposes, by friction at the mouth of the aorta, is propagated along the arteries, and may be heard at the apex. According to Hayden, then, a functional murmur differs from an organic one in that it is not propagated according to the ordinary laws of sound. It is never propagated along the medium in which it is produced; but, having selected a point of exit, it passes readily and directly outwards through the overlying heterogeneous media, within which it slightly radiates or diffuses itself in some inexplicable fashion. Unfortunately, the immutable laws of physics forbid the acceptance of any such dogmatic statement. That it has been made by so intelligent and so generally trustworthy an observer as Hayden, is only a further proof that the hæmic murmur is a varying murmur, and that, for want of a proper clue, he got bamboozled in attempting to follow and explain its different modifications.

2. *The theory of cardiac dilatation, as the cause of the hæmic murmur, is thoroughly consistent with the results obtained by experimental investigation.* When the late Dr. Marshall Hall was engaged in investigating the effects of loss of blood upon the system, he observed that, during the reaction subsequent to such loss, the action of the heart was "accompanied by a peculiar noise resembling that of the saw or of the file, termed by the French 'bruissement', and very discernible on applying the ear to the chest."‡ This "bruissement" Marshall Hall distinctly recognised as identical with the chlorotic murmur, and accepted its artificial production by loss of blood as a clear proof of the purely hæmic character of that murmur.§ Hope, who assisted Marshall Hall in making these experiments, adopts most explicitly the views of that author, and describes the murmur as due to the attenuation of the blood and the increased velocity with which it was circulated.|| We need not now inquire whether attenuation of the blood or increased velocity of the circulation can ever be, of themselves, a source of murmur, because we have learned since that time that those phenomena, occurring in animals repeatedly bled, are always accompanied by a condition of heart now universally recognised as an efficient cause of murmur. For Beau, investigating the production of the murmurs following bloodletting, which he also acknowledged to be chlorotic in character, found that they resulted, not from the anæmia primarily produced, but from the secondary spanæmia, to which he gave the name of serous polyæmia.¶ Beau also found that this serous polyæmia is invariably associated with a dilated and hypertrophied condition of the heart; and that, in dogs and rabbits killed by a gradually exhausting hæmorrhage, the cavities of the heart are not only manifestly dilated, but the heart itself, freed from clots, weighs from one-fifth to one-third more than the heart of a similar animal destroyed by one suddenly fatal gush of blood from a severed carotid.**

* *Diseases of the Heart*, etc. 4th ed. London: 1873, p. 86.

† *The Diseases of the Heart and Aorta*. Dublin: 1875. P. 248, and p. 252.

‡ *Medico-Chirurgical Transactions*. London: 1832. Vol. xvii, p. 257.

§ *Principles of the Theory and Practice of Medicine*. London: 1837. P. 370.

|| *Op. cit.*, p. 100.

¶ *Archives Générales de Médecine*, 4ième série, tome ix, 1845, pp. 145, 149, 151, 169, etc.

** *Op. cit.*, p. 156.

But, as a dilated heart is now-a-days universally recognised as an efficient cause of murmur,* it only remains to show—

3. *From the results of clinical experience*, that the attenuated blood of chlorosis—the serous polyæmia of Beau—is accompanied by a condition of heart similar to that produced by repeated bloodlettings; and that the cardiac hæmic murmur appears and disappears in a manner consistent with this theory. Wunderlich† and Friedreich‡ have described the chlorotic heart as dilated; while Beau,§ Bamberger,|| and Stack¶ have adopted the more rational view, that it is hypertrophied as well as dilated; and Dr. Heitler of Vienna, one of the most recent writers on cardiac disease, speaks of the dilated hypertrophy of the chlorotic heart as a well-known and acknowledged fact.** Beau has distinctly pointed out that the chlorotic murmurs are not due to anæmia, but to spanæmia—serous polyæmia††—a condition in which the blood is not diminished in amount, though its nutritive and oxygenating properties are very much lessened. The primary murmur in such cases is the venous hum, depending upon abnormal friction between the spanæmic blood and the venous walls, and the formation of sonorous fluid veins at certain favourable points within the venous lumen. The next phenomenon observed is an accentuation of the pulmonary second sound, unquestionably due to increase of the intrapulmonary blood-pressure, the only possible cause for this being obstruction to the onward flow of the blood; and, in a disease such as chlorosis, this can only be due to loss of tone and contractile force in the cardiac muscle, which in this respect suffers no less than the external muscles, in all of which this loss of tone is so obvious. Less able to do its work, and with no less work to do, the heart slowly dilates, as it always does in similar circumstances; and it also slowly hypertrophies, as it must do to prevent things from coming to an absolute standstill. The dilatation is due to loss of muscular tone or elasticity; the hypertrophy to that natural law by which a hollow muscle hypertrophies when opposed to an obstacle with which it is still able to cope. And that the heart, under these circumstances, is still able to cope with its troubles, is due to the fact that it always works well within its powers, and has a large reserve of energy to call upon when required. As the heart dilates, the mitral and tricuspid valves become incompetent, and give rise to murmurs. The regurgitation through the tricuspid gives rise to undulations in the jugular veins; and the abnormally large ventricular blood-waves give rise to systolic murmurs in the pulmonary and aortic areas, which are also propagated along the carotids, and may be heard on the slightest compression in every artery in the body.

Thus, we have murmurs in every area of the heart in advanced chlorosis—the primary indication of cardiac lesion being an accentuated pulmonary sound, due to loss of tone of the left ventricle. And I am sure that we all agree with Hayden, that the primary cardiac hæmic murmur is basic; that it is not propagated in any special direction, but radiates round the pulmonary area, in the neighbourhood of which lies its position of maximum intensity. As has been already pointed out, there are no causes of murmur operative at the pulmonary orifice, which are not at least as active at the aortic opening; so that a murmur in the one position would certainly be accompanied by a murmur in the other, which would of course be propagated along the aorta, and more or less distinctly into the carotids also. This murmur cannot, therefore, be pulmonary; and, indeed, on careful examination, its position of maximum intensity is found not to be over the pulmonary artery at all, but from one to two inches to the left of the sternum, in the second interspace. In this position I never fail to detect, at least during expiration, some faint pulsation; and occasionally this pulsation is so great as to form a tumour, which has been made to record its own movements graphically;†‡ and which, at least upon one occasion, has been mistaken for an aneurysm by a very competent physician.§§ Both Walshe and Hayden recognise “percussion-dulness in the second left interspace,” with pulsation in the second, or second and third, left interspaces, about an inch from the sternum, as physical signs indica-

tive of dilatation of the left auricle;* and Dr. Gibson's graphic records very distinctly prove the auricular source of the pulsation in those few cases in which such records have been obtained. There is therefore no room for doubt that in these special cases, and it is plainly to be inferred that in all cases presenting similar if less well-marked characteristics, this murmur is propagated outwards from the dilated appendix of the left auricle.

In mitral stenosis, it is no uncommon thing to have the appendix of the left auricle pulsating in the second left interspace; and it is still more common to have a basic systolic murmur, with a position of maximum intensity precisely similar to that of the basic murmur—viz., one inch and a half or two inches to the left of the sternum. This murmur, Naunyn† has distinctly proved to depend upon mitral regurgitation, the fluid veins impinging on the tense auricular wall, and throwing them into sonorous vibrations; these are conveyed to the chest-wall by the auricular appendix which lies in contact with it at the base of the heart.

Naunyn's views have been accepted by Paul Niemeyer,‡ Gerhardt,§ and all recent authorities. The basic position and auricular character of a mitral regurgitant murmur, in certain cases, is therefore a well acknowledged fact. That the primary cardiac hæmic murmur is of this character seems to be proved by these facts: (1) in chlorosis the heart is dilated and hypertrophied, and the primary murmurs are certainly not arterial in character; (2) the position of maximum intensity of the primary hæmic murmur and of Naunyn's murmur is precisely the same; and (3) the graphic record of the pulsation present in both classes of cases is similar; and, as it is known to be auricular in the one, it is most likely to be auricular in the other also, especially as it is certainly neither ventricular nor belonging to the conus arteriosus.¶ The dilated character of the heart in chlorosis is further proved by the apex-beat being either entirely absent, or only faintly perceptible; not in the usual position, but just beneath the lower part of the sternum. This is due to dilatation of the right ventricle, which dilates *pari passu* with the left ventricle, and, like a water-cushion, separates it from the chest-wall, leaving the dilated appendix of the left auricle the only part of the left side of the heart in contact with the chest-wall. The peculiar position of the primary hæmic murmur is no doubt largely, if not wholly, due to this. Shortly after the appearance of the primary hæmic murmur, a tricuspid murmur with jugular undulation is found to be developed. This is naturally accompanied by a pulmonary, and also of course by an aortic systolic murmur; the active cause in the production of both of these murmurs being the large blood-wave sent on by the dilated and hypertrophied ventricles, as was first, I believe, pointed out by Beau.¶ The aortic murmur is propagated into the carotid arteries; and about the same time, we also have a systolic murmur in the mitral area; but this is probably due to the right ventricle being now so dilated, that its apex occupies the position of the apex of the left ventricle in health.

It is only rarely, and chiefly in a peculiar class of cases, that we have an opportunity of observing the gradual development of the hæmic murmur from area to area; but we all have frequent enough opportunities of tracing the involution of this murmur; and, if carefully traced, it will be found to die off precisely in the reverse order to that which I have described, the venous hum being the last to disappear.

With a short auricular appendix, it is, of course, quite possible that the primary hæmic murmur may be pulmonary, or aortic, or possibly tricuspid in character. I have never seen any such case, but, were it to occur, it would strengthen and not invalidate my position. My argument is, that the cardiac murmurs of chlorosis are formed in a dilated and hypertrophied heart, and that the primary position of the murmur is, in by far the larger proportion of cases—if not in all—to be found over the left auricular appendix, where it comes up from behind just to the left of the pulmonary artery. And, having shown this theory to be consistent with physics, with the results of experiment, and with clinical experience, I now leave it with my professional brethren, assured that, if not now, at least ere long, it will be universally adopted.**

Dr. CLIFFORD ALLBUTT (President of the Section) said they must feel themselves peculiarly fortunate in having had this paper read in

* Vide Gairdner's *Clinical Medicine*, Edinburgh, 1862, p. 592, p. 597, and p. 600, note; also *Ed. Med. Jour.*, July 1856, p. 55, for a paper by Dr. Gairdner entering fully into the literature of the subject; also Dr. Bristowe, in the *British and Foreign Medico-Chirurgical Review* for July 1861, p. 215; and Dr. McDowell, in the *Dublin Quarterly Journal*, vol. xiv, 1852, p. 352; vol. xvi, 1853, p. 76; and vol. xvii, 1854, p. 90.

† *Handbuch der Pathologie*, Stuttgart, 1856, Band iii, s. 647; Band iv, s. 534.

‡ *Krankheiten des Hersens*, Erlangen, 1867, s. 172.

§ *Archives Générales de Médecine*, 41ème série, tome ix, 1845, p. 169 and p. 451, etc.

¶ *Lehrbuch der Krankheiten des Herzens*, Wien, 1857, ss. 88 and 246.

‡ *Gazette Hebdomadaire*, 1863, p. 262.

§ *Wiener Medicinische Wochenschrift*, June 1882, s. 654.

¶ *Op. cit.*, p. 145 and p. 169, etc.

†† *Vide Lancet*, September 1877, p. 418; and *Ed. Med. Jour.*, October 1877, p. 299, and May 1878, p. 1012.

§§ *Vide Balfour, Clinical Lectures on Diseases of the Heart*, 1882, p. 176.

* Hayden, *Op. cit.*, p. 576. Walshe, *Op. cit.*, p. 31.

† *Berliner Klinische Wochenschrift*, April 1868, p. 190.

‡ *Handbuch der Percussion und Auscultation*. Erlangen: 1870. Band ii, Abtheilung 1, S. 140.

§ *Lehrbuch der Auscultation und Percussion*. Tübingen: 1871. S. 283.

¶ *Vide Ed. Med. Jour.*, August 1882, pp. 121, 128, and 129.

‡ *Loc. cit.*, 1845, p. 158; and *Traité d'Auscultation*. Paris: 1856. P. 366, etc.

** For an account of the position and mechanism of the hæmic murmur, *vide also the Lancet*, September 1877, p. 383; and Balfour's *Clinical Lectures on Diseases of the Heart*, etc. London: 1882. P. 165, etc.

the section. Until that afternoon, his own mind had been in a state of perfect mist as regarded hæmic murmurs. It had very early occurred to him that the explanation which attributed them to the condition of the pulmonary orifice was entirely out of the question, and he wondered that even its original observer was not soon possessed with its untruth, because in no kind of way did it correspond to the conditions they should expect to find if the murmurs were formed in the pulmonary artery. Now they were asked, and he thought very properly asked, to take some *vera causa*, the most likely they could get, and push it forward as far as they could go. That was, he thought, the only way in which they could get at the truth in these things. It would be very difficult, as Dr. Austin Flint had said to him, and was scarcely courteous to the writer, to discuss off-hand the physical side of the question, to which he had given such attention and pains. But, from the clinical side, they might offer some remarks. In referring to the occurrence of dilatation of the heart with cure, Dr. Balfour had touched a subject on which he (Dr. Clifford Allbutt) had long intended to publish some results of his own experience, and he was extremely glad to hear one of the first authorities of the day state that as a fact. He had not the slightest hesitation in saying that cases of morbid degrees of dilatation—that was to say, showing positive disease, many of them of advanced years, fifty or sixty years old—recovered under diet, careful nutrition, and the use of iron. He believed, therefore, that not merely what they called functional dilatation in young ladies, and after hæmorrhage and so on, but what he should call morbid dilatation, associated with fatty degeneration—he did not like the term—did recover, even when attended with hæmic murmurs. Then, as regarded the position of these murmurs, he had always supposed a very large number of them were mitral regurgitant. He should like to ask Dr. Balfour what he thought about the appearance of hæmic murmurs in cases in which, as far as he knew, the heart was not dilated. Perhaps Dr. Balfour would say it was dilated. But he (Dr. C. Allbutt) would say that, in cases where the heart was degenerated, as in progressive pernicious anæmia, his impression was that the heart was not dilated, but simply atrophic. What had also struck him was the absence of chlorotic murmurs in other cases of dilatation, and so on, not of anæmic causation. He was under the impression that a great many cases of dilated heart would be still found in which there were no basic murmurs. There was something in the peculiar ways in which the dilatations were brought about, namely, from anæmia, and spanæmia, and so on; and if brought on in other ways, such as valvular disease, and so on, there were not murmurs further than those changes would account for. One case of his, if singular, would be a mere curiosity, and therefore of little importance; but, if any other member had met with similar cases, it might be important. He believed that he had once met with a case of murmur corresponding to the second interspace across the sternum in anæmia. It was the case of a man about thirty years of age, labouring under a great deal of anxiety from his affairs, exceeding anæmic, and very much broken down indeed. He had given him a very definite opinion that he had aortic valvular disease, with aortic regurgitation. But, on recent examination, when he was very altered in appearance and health, all traces of murmur had absolutely disappeared. He would be obliged if members would describe any other case of the kind that had occurred.

Dr. AUSTIN FLINT (New York), in commenting on Dr. Balfour's paper, said that hæmic murmurs were frequently met with in apparently healthy persons.

Dr. THEODORE WILLIAMS (London) could not entirely agree with Dr. Balfour as to the cause of chlorotic murmurs being a dilated state of the right side of the heart. If it were so, would not this appear with increased cardiac dulness? Again, the contrast between the appearance of chlorosis and that of the ordinary dilated right side of the heart with the livid tint and gorged capillaries was as marked as it could be. Dr. Williams thought that some cardiac hypertrophy often followed prolonged anæmia, the condition producing greater difficulty of circulation.

Mr. MACALISTER (Cambridge) described how Professor Ludwig and Dr. Hesse, of Leipzig, had lately succeeded in overcoming the great mechanical difficulties in the way of determining the exact form of the heart in systole and in diastole. By a method of great ingenuity, they at length were able to obtain casts of the still living dog's heart in its systolic and in its diastolic configuration. These showed clearly the hitherto misunderstood relation of the *ostia venosa* at the beginning of the systolic stroke to that at the end of it. The area of the base in systole was only one-half of the area in diastole. The *ostia*, which were round and wide in diastole, were oval and oppressed in systole. The valves even in the healthy heart were incompetent to close such orifices as were seen in diastole. The great share of the work of clos-

ing these orifices was done by the contracting heart itself; the opening which the valves were called upon to close were already in great part contracted by the approximation of their borders. It was thus conceivable that anything which interfered with the nutrition of the heart as a muscle might impair its power to perform its share in closing the orifices. The valves were structurally sound, but they were called upon to close larger orifices than in health. This was not necessarily because the *ostia* were dilated; it was because they were insufficiently contracted. Mr. MacAlister urged this as a help to the understanding of how regurgitation might be produced without valvular lesion and without real dilatation, but simply from malnutrition and imperfect contraction of the heart-muscle. He exhibited casts of the heart in support of his view.

Dr. BROADBENT (London) agreed with Dr. Balfour in his account of the effects ultimately produced on the heart by chlorotic anæmia, that there might be dilatation of the ventricles and tricuspid, and mitral regurgitation; but he was unable to accept his explanation of the murmur heard at an early stage of the disease in the left second space an inch or more from the edge of the sternum. He had been acquainted with Dr. Balfour's views for some time, and had taken much trouble to test them by careful observation, with the result that up to the present moment he felt bound to reject them. His first difficulty was this, Dr. Balfour said that the murmur of chlorosis heard in the left second space was due to mitral regurgitation, but he gave no explanation of the fact that systolic murmurs much louder than this, undoubtedly due to mitral regurgitation, audible at the apex, and round to the angle of the scapula, were not heard in this situation. If a mitral murmur not loud enough to be heard at the apex were audible over the auricle, some reason ought to be given why the louder murmur, audible at the apex, was not present over the auricle. There were, no doubt, cases in which the systolic mitral murmur of valvular disease was heard higher up in the chest than usual, and up to the left second space, but this was when there was an unusual degree of dilatation and hypertrophy of the left ventricle, causing it to be exposed along the left border of the heart; and the rule was, that systolic apex-murmurs, however loud, did not reach the second space. Another difficulty was, that he had never seen after death the auricular appendix, or any part of the auricle near the surface of the chest as high, as the second space. The appendix came round the pulmonary artery just at its origin from the conus arteriosus, which was at the level of the third space; in no stage of chlorosis was there any reason to suppose that there was excessive dilatation of the auricle, and he did not believe that, as a matter of fact, the auricular appendix ever lay beneath the chest-wall at the point in question. Dr. Balfour had spoken of auricular pulsation felt in the same situation, *i.e.*, in the left second space, in mitral stenosis, as a reason for concluding that a mitral regurgitant murmur might be heard here; but he had examined every case of mitral stenosis he had seen since the publication of Dr. Balfour's book, and had never been able to satisfy himself of its presence. He could not, indeed, understand how pulsation of the auricle could be felt through the chest-wall. In assisting Dr. Sibson in the experiments by which he determined the form of the heart in systole and diastole, he had had the opportunity of watching the heart in action for hours in dogs and other animals; and, if the finger were applied to the ventricle of the exposed heart, it was powerfully thrust out during the systole; but, applied to the auricle, it received no appreciable push. The auricular appendix, again, the only part of the auricle which reached the chest-wall, was at the moment of the auricular systole suddenly and rapidly withdrawn. It must be borne in mind that, in those cases in which auricular pulsation was supposed to occur, there was extremely high pressure in the pulmonary artery, which would make the pulsation in the vessel powerful and extensive. Dr. Balfour had quoted Dr. Gibson's traces as conclusive evidence of auricular pulsation, and it might be said that instrumental records could not deceive. Dr. Broadbent, however, had seen enough of instrumental investigation to know that it required careful checking, and that the personal equation of the observer had to be taken into account. He had read Dr. Gibson's paper without being at all convinced, rather the contrary, of the occurrence of auricular pulsation. For his part, Dr. Broadbent saw no difficulty in accepting the pulmonary artery as the seat of the murmur heard in the left second space. It was not always the case that the seat of maximum intensity of a murmur was immediately over the point at which it was produced, as was seen in aortic regurgitation and mitral regurgitation; and it might very well be that a murmur arising at the pulmonary valves would be better heard a short distance along the vessel. He had sometimes been inclined to think, however, that the murmur under consideration might arise at the bifurcation of the artery, more especially as it could often be heard upwards and to the left for some distance. There was one point in the history of the development of

cardiac changes in chlorosis, which Dr. Balfour had omitted to mention. He had spoken of the accentuation of the pulmonary second sound, but he had said nothing of the accentuation of the aortic second sound, which was equally constant. Dr. Broadbent thought this important, and that in it was the key to the production of the dilatation of the left ventricle, and to the difference pointed out by Professor Austin Flint between chlorotic anæmia and the anæmia of phthisis and cancer, in respect of venous murmurs. It was an unexpected thing that, in a disease such as chlorosis, characterised by debility, there should be high arterial tension, but such was the fact; and it was indicated not only by the accentuated aortic second sound, but also by the character of the pulse. Of course, the increased tension was relative rather than absolute. The high arterial tension implied resistance in the peripheral circulation, and this would throw increased work upon the heart, and so give rise to dilatation. With regard again to the presence of venous murmurs in chlorotic anæmia and their absence in phthical anæmia, if pointed to the same fact as the increased peripheral resistance and arterial tension in chlorosis, namely, that the primary change was in the blood; and there might be some relation between the two.

Dr. DRUMMOND (Newcastle-on-Tyne) said that he was in the habit of teaching that, in his opinion, the chlorotic murmur, heard as a systolic *bruit* in the second left interspace, was not due to mitral regurgitation, but was really the result of vibration of the pulmonary artery, which occurred on account of the sudden state of high tension into which it was thrown by a rapid systole. The fact that no aortic murmur was heard, was due to the difference to be found in the structure of the walls of the two vessels. Dr. Drummond asked Dr. Balfour to account for the appearance of the chlorotic murmur in cases of rapidly developed spanæmia due to hæmorrhage.

Dr. BALFOUR, in reply, thanked the members of the section for the interest they had taken in his paper. The subject was well deserving of it, as, from its relation to curable dilatation, it was, perhaps, the most important murmur they had to deal with. They would all remember what Senac had said—the longer and more carefully they studied diseases of the heart, the more difficult it seemed to make any practical use of their studies. What, for instance, could they hope from treatment in dilatation of the heart? Why this was, if he was right, the most curable of diseases. The President had referred to the presence of hæmic murmurs in cases of pernicious anæmia. This was constantly the case when spanæmia was present, especially in cases of gradually fatal hæmorrhage, and in such cases he had always found the heart slightly dilated and hypertrophied.* In phthisis and malignant disease, on the other hand, there was anæmia and no spanæmia. The hæmic murmurs were absent, and the heart was atrophied and not dilated. Even in spanæmia the murmurs occasionally disappeared before death, owing to loss of cardiac energy. The President had also referred to an interesting case of curable aortic regurgitation, the result of spanæmia. This was no doubt due to loss of tone in the arterial tissues, coupled with the high arterial tension constantly present in these cases, as Dr. Broadbent had pointed out. He himself had seen no case exactly the same, but temporary aortic regurgitation in somewhat similar conditions was not unknown, and he had even seen it occur in the pulmonary artery, apparently from excessive intrapulmonary tension. Dr. Flint had said that hæmic murmurs were often present in cases of apparent health, and no doubt this was so; but in all such cases the blood would be found to be really spanæmic and deficient in hæmoglobin. Dr. Williams had very properly pointed out that the signs of dilated right ventricle, as seen in cases of emphysema, were very different from those present in chlorosis. But the cases were widely diverse; the peculiar phenomena in emphysema were due to the limitation of the capillary area within the lungs, forming a positive obstruction to the onward flow of the blood, of which the dilated right ventricle was only one symptom. In chlorosis, on the other hand, the dilatation was solely due to loss of tone, there was no positive obstruction, and the conditions and signs were perfectly different. The dilatation in spanæmia, though greater and more permanent, was entirely similar to that trifling form which was readily brought about by holding our breath, when the apex-beat would be found to disappear, from the dilating right ventricle gradually pushing it back from the chest-wall. Dilatation of the right auricle might usually be detected by percussion; dilatation of the right ventricle could not be so discovered, but might readily be inferred from other physical signs. Mr. MacAlister's interesting communication did not invalidate, but rather confirmed his position. The mere mechanism of the dilatation was of little consequence; his object was to get the spanæmic heart acknowledged to be always dilated, and to use this as a standpoint for further investigation and as a basis for treatment. In re-

gard to Dr. Broadbent's remarks, he acknowledged that large auricles were somewhat rare, but they did occur, and even Dr. Broadbent would be sure to see one by-and-by. The late Professor Bennett had sent him his first large auricle as a case of aneurysm, and it was the accidental occurrence of several such cases together that led him to investigate the conditions under which they occurred. The reason, or at all events one reason, why the mitral murmur in chlorosis was audible at the base, and not over the apex, was because the dilated left auricle was the only part of the left side of the heart remaining in contact with the chest-wall, the left ventricle being pushed backwards by the dilated right side. The auricular murmur was not unfrequently present along with a loud mitral murmur, when the conditions were favourable; and, when it was absent, the physical condition and relations of the auricle would be found to be entirely different. The auricular pulsation was not due to the systole of the auricle, but to its distension by the regurgitant blood during the ventricular systole, as Dr. Gibson had pointed out, and as was very well shown both by the character and time of the tracings.* Dr. Gibson had enjoyed exceptional opportunities of observing and getting tracings from auricular pulsations; he had also obtained tracings from the *conus arteriosus* in a case of split sternum; and a single glance at the tracings from the auricle was sufficient to show that they were quite peculiar. Something no doubt might be said as to personal equation in regard to graphic tracings, but the tracings he referred to had all been taken by the same man; the equation was therefore the same for all, and he thought they might be reasonably accepted at least as a basis for further investigation. Dr. Drummond's idea, that the murmur was due to vibrations in the pulmonary artery was inconsistent with the fact that the position of maximum intensity was not over the pulmonary artery at all; it was, moreover, an old theory, which had more than once been adopted and been discarded.† His object in initiating this discussion was to bring out the truth, and to direct their attention to the probability of the murmurs of chlorosis being due to dilatation, the result of debility. He was satisfied that a careful consideration of the hæmic murmur from this point of view was well worthy of their most serious consideration, and he begged again to thank them for the attention they had already bestowed upon the matter.‡

* *Vide Lancet*, Sept. 1877; *Ed. Med. Jour.*, Oct. 1877 and May 1878, also August 1882.

† By Meyer, Bamberger, and Gerhardt; *vide* Naunyn's paper in the *Berliner Klinische Wochenschrift* for April 27th, 1868, p. 189. Von Dusch also adopted a similar view, *vide Lehrbuch der Herzkrankheiten*; Leipzig, 1868, p. 205.

‡ In Marshall Hall's paper in the *Medico-Chirurgical Transactions* for 1832, vol. 17, and especially in Beau's paper in the *Archives Générales de Médecine*, 4^{ième} série, tome ix, 1845, will be found a full account of the development of the hæmic murmur after bloodletting, showing the rapid way in which it sometimes develops, and the interesting way in which further bloodletting temporarily stops it.

SYPHILITIC INFECTION OF THE FINGER IN MEDICAL MEN.—Dr. Fessenden N. Otis communicates to the *Independent Practitioner* of March particulars of eight cases of syphilis contracted by physicians in making digital examination of the vagina of syphilitic women. The initial lesion of this form of syphilis is described as being uniformly a papule, "coming soon to be of a deep red colour, and presenting a superficial abrasion, becoming circular and deeper by a slow molecular necrosis; not by ulceration with formation of pus; the secretion thin and serous, and drying into a scab which is soon displaced by the fluid accumulating underneath." He also remarks "the entire absence of induration; in its place a slight, flat, juicy-looking, boggy swelling, or elevation, about like a small peppermint in size and thickness." As proof of the efficacy of treatment, which was continued in five of the cases for one and a half to two and a half years, he states that subsequently "eight healthy children have been born, and both they and the parents have continued free from any evidence of syphilis."

HYOSCYAMINE IN INSANITY.—Dr. Grieve, of the Colonial Lunatic Asylum at Berberce, reports that he has tried hyoscyamine in a few cases of insanity, but without encouraging results. Three negroes, two of whom were suffering from chronic mania and one from acute mania, took the drug. The preparation used was the crystal, and the dose to begin with was one-sixth of a grain twice a day. This produced marked physiological effects; the pupils were widely dilated, there were staggering gait ending in complete inability to walk, loss of power of articulation, and dribbling at the mouth, giving an appearance of total and helpless imbecility. Whilst this condition lasted, the patients were quiet, but as soon as it passed off noisiness and restlessness returned. So quickly was tolerance of the drug established, that the dose had in one case to be trebled in three days, and then lost its power over the patient, after which it was not considered advisable to push the treatment any further. None of the patients permanently improved in condition from the use of the hyoscyamine.

* One such case will be found narrated at p. 173 of Balfour on *Diseases of the Heart*. Second edition: 1882.