local application of opium in some form is beneficial, and the pain will in many instances be greatly alleviated by brushing the surface with a solution of nitrate of silver (ten to twenty grains to the ounce). The *cachectic ulcer* is best treated with alteratives. Iodide of potassium should be given in five-grain doses or more three times a day, and it is often advantageous to administer some ferruginous preparation. These ulcers are particularly improved by a nutritious, not a stimulating, diet, and thus cod-liver oil and wholesome puddings of a farinaceous kind do more good than ale, stout, wine, or spirits. In the *phagedænic*, the *sloughing*, and the *sloughing phagedænic*, tonics are imperatively demanded, but due care must be taken to clear the *primæ viæ*. It is a grave mistake to over-stimulate patients suffering from this form of ulcer, for the increased heart's action tends to increased local congestion, and hence the sloughing process is rather favoured. Opium may be given with great advantage, and in many cases I have seen marked benefit from keeping the part in a warm bath for twenty-four hours. The *callous ulcer* is one in which strapping may be most usefully employed, and I think that Baynton's plan is as good as any. He says, writing in 1799, that "the straps should be two inches wide, and long enough to encircle the limb, and overlap at the ends to the extent of three or four inches. The plaster should be one inch below and two or three inches above the diseased part." I have seen benefit follow the use of blisters to these callous ulcers, and have observed a marked improvement after the application of Friar's balsam (the tincturæ benzoini comp.) Lateral incisions to release the edges of the ulcer are, in some cases, very desirable. A bandage evenly applied is very serviceable in this and in all ulcers where general support is required. The *specific ulcers*, as you may suppose, require specific treatment, which, from want of time, I am unable to include in the present lecture.

The importance of dressing ulcers properly cannot be over-estimated. Whether lotions or ointments be used, the lint should be cut no bigger than the wound, and it is best to apply several small pieces, which should be placed in the more excavated parts, instead of using one larger piece; thus the ulcer comes more immediately under the influence of the remedy employed. If the ulcer be undermined the lint should be introduced carefully, and not packed roughly under the overhanging parts. I have a high opinion of the *lotio hydr. nig.* (black wash), which I use quite irrespective of the syphilitic character of a wound. Further, I may add that if the case is one that is likely to improve by strapping, it is unnecessary to use other local applications at the same time, for it is obvious that if lint were placed on the ulcer, and then strapping applied, the lint would of necessity become puckered up. Strapping, then, deserves its own place in the treatment of ulcers, and is of immense value in cases in which the ulcer, whatever its precise physical character, is not very painful, and which has assumed a somewhat chronic character. I show you on this patient how to apply the straps. The centre of the plaster is placed on the leg exactly opposite the seat of the ulcer, and the two ends brought forward and made to overlap. Each successive strap covers half or two-thirds of the preceding one, and they should all lie parallel and equidistant as far as possible. It is, however, not always easy to effect this, especially in an enlarged knee-joint, in which, owing to the contour, it will often be found necessary to deviate slightly from the ordinary rule of practice.

In the necessarily brief remarks that I have now offered I have endeavoured to simplify the subject of ulcers, and have done my best to show that ulcers, like other diseases, must be treated on common-sense principles; for example, the inflamed ulcer must be soothed, and the weak ulcer must be stimulated, and so on. Our duty is to alleviate local pain by suitable topical applications, and to improve the constitution by proper remedies. In attending to these two points, we place our patients in the best position for ultimate recovery.

An epidemic of typhoid, affecting twenty families in the west-end of Edinburgh, and already fatal to several persons, is traced by Dr. Littlejohn to a dairy in which a person was suffering from the fever.

ON THE POSITION AND MECHANISM OF THE HÆMIC MURMUR.

By GEORGE W. BALFOUR, M.D.,
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INORGANIC CARDIAC MURMURS—that is, murmurs audible over any part of the cardiac area, and occurring independently of any irremediable mechanical alteration of the organ—have been classified by authors under two heads: hæmic and dynamic. Hæmic murmurs have been regarded by all authorities as systolic in their rhythm; and by most, as basic in position; and as produced by the passage of an abnormal blood through a perfectly normal heart, after a fashion which has been variously explained. Dynamic murmurs, on the other hand, though also systolic in their rhythm, yet have their position at the apex, are regurgitant in character, and are supposed to be produced by a perturbed or defective action of the cardiac muscle itself, independent of any abnormality of the blood, which, however, probably always coexists.

Chlorosis, and the chlorotic cardiac murmur, is the typical example of the one class; chorea, and the apex murmur that so often accompanies it, the typical example of the other class. In the one set of cases the blood lesion is primary and paramount; in the other set the blood lesion is essentially secondary, though not always actually so, and it forms but one link in the chain of consecutive causes of the morbid phenomena. The essential lesion of the blood in both of these classes is deficiency in its nutritive qualities; and, in the latter class, we have superadded the exhaustion consequent upon almost incessant muscular action, or upon co-existent or pre-existent febrile disorder. This state of matters, as we all know, produces an enfeebled, relaxed, and flabby condition of the muscles, which, of course, is not confined to the external muscles, but extends also to the hollow internal muscles, which suffer proportionately to the work they have to do. In these conditions the heart necessarily participates, and we are not therefore surprised to learn that in fevers it sometimes becomes as limp as a piece of wet paper (Stokes¹), nor that in serious cases of chlorosis it is found to be considerably dilated (Bamberger,² Friedreich,³ Wunderlich,⁴ Stark,⁵ &c.) Beau, in 1856, associated fevers of various kinds with chlorosis and other forms of hydræmia as causes of dilatation of the heart, pointing out that it was rational to attribute this dilatation to that peculiar alteration of the blood which is common to all these different forms of disease.⁶ Naturally he was the more inclined to adopt this view, as, so long ago as 1845, he had experimentally proved that death following the hydræmic condition of blood induced by repeated venesections, in dogs and rabbits, was accompanied by a dilated condition of the heart.⁷ Dilatation of the heart, however, is, as was first pointed out by Stokes,⁸ and subsequently by Gairdner⁹ and Bristowe,¹⁰ a very frequent cause of systolic apex murmur. Stark has shown that the dilatation of chlorosis may be accompanied by relative inadequacy of both the mitral and tricuspid valves, and that murmurs of regurgitation may thus be heard at either of the auriculo-ventricular orifices; while Parrot¹¹ has given it as his opinion that the true hæmic murmur is invariably a murmur of tricuspid regurgitation. The chlorotic or typical hæmic murmur is thus shown to be invariably accompanied by a condition of heart which may give rise to a dynamic apex murmur; while, in the opinion

¹ Quoted from Louis, *Diseases of Heart and Aorta* (Dublin, 1854), p. 368.

² *Lehrbuch der Krankheiten des Herzens* (Wien, 1857), S. 89 and S. 246.

³ Virchow's *Handbuch der Spec. Path. und Therapie* (Erlangen, 1861), Bd. v., S. 298.

⁴ *Handbuch der Pathologie und Therapie* (Stuttgart, 1856), Bd. iv., S. 534.

⁵ *Archiv der Heilkunde* (1863), p. 47; and *Gaz. Hebdom.* (1863), p. 262.

⁶ *Traité d'Auscultation* (Paris, 1856), p. 355 and 365.

⁷ *Archives Gén. de Méd.*, 4ème sér., t. ix., p. 151, &c.; and *Gaz. Hebdom.* (1863), p. 263.

⁸ *Op. cit.*, p. 271.

⁹ *Ed. Monthly Journal* (July, 1856), p. 59.

¹⁰ *British and Foreign Med.-Chir. Rev.* (1861), vol. xxviii., p. 215.

¹¹ *Archives Gén. de Médecine*, 6ème sér., t. viii. (1866), vol. ii., p. 159.

of some, the so-called hæmic murmur is always of this character. It is true that the choreic, or true typical dynamic, murmur has been alleged to be caused by "disordered action of the muscular apparatus connected with the valve" (Walshe¹²). But while clonic spasm of one or other of the *musculi papillares* might give rise to an intermittent murmur, there is some difficulty in recognising such spasm as an efficient cause in producing a mitral murmur of such uniformity as the ordinary curable choreic mitral murmur possesses. Besides, "there is no good proof that involuntary muscular organs participate in the muscular disorder" (Kirkes¹³); while "the admitted coexistence of rhythmical action of the substance of the heart with this alleged tetanic and irregular contraction of the papillary muscles, which, as proved by dissection, are directly continuous with the fibres of the ventricular walls, constitutes a still stronger objection to this theory" (Hayden¹⁴), especially if we "bear in mind the unity of nerve-centres, and the community of nerve-distribution enjoyed by both portions of the same fibres."¹⁵ Moreover, the extension of this explanation to the dynamic murmur of weak and fatty hearts, in which there is not the slightest evidence of nervous disturbance—as has been done by Walshe—is quite inadmissible; while the explanation of "parietal debility of the left ventricle,"¹⁶ adopted by Hayden, seems to be the only one applicable to both classes of cases, is perfectly sufficient, and is consonant with all the facts of these cases. But "parietal debility" is merely another expression for that muscular relaxation resulting in dilatation, which, as we have seen, is so universally recognised as a tolerably constant concomitant of various forms of fevers as well as of chlorosis. Thus we have the dynamic apex murmur of chorea associated in its cause with that occurring in various forms of fever, as well as in certain cases of chlorosis. Chlorosis, in its severer forms, is therefore, and has long been, acknowledged to give rise to a dynamic apex murmur, of a perfectly similar character with that so often occurring in the course of chorea, the eruptive and other fevers, and originating in a precisely similar manner—namely, in relative inadequacy of the auriculo-ventricular valves, the result of dilatation of the ventricles consequent on parietal debility. It is of consequence to remember that this dilatation is not active, in which case it would rather be a sign of increased than of diminished ventricular power; but that it is passive, the gradual result of residual accumulation in the cavity of a hollow muscle too feeble fully to discharge its ordinary function. It is also important to remember that dynamic regurgitation in its results differs in no respect from regurgitation due to organic disease, but, equally with the latter, is always followed by pulmonary congestion, as well as by all the usual consecutive results of obstruction to the circulation originating in the left heart, all of which may be detected with greater or less ease in proportion to the amount of regurgitation present, and the time it has persisted.

Bearing all these facts in mind, we are tempted to inquire how it is that the hæmic murmur of chlorosis becomes, in its later stages, an apex murmur of regurgitation, after having been in its earlier stage a basic murmur—apparently of obstruction. To this query there is, I think, but one reply, and that is that, all through, the murmur is due to ventricular dilatation, and the changes in its position of maximum intensity are due to alterations in the heart itself. I am quite aware that alterations as to the position and textural condition of the media overlying and surrounding the heart, bring about many remarkable changes, both in regard to the production and the propagation of murmurs. Into these, however, it is at present unnecessary to enter, except in regard to one or two points subsequently to be referred to.

In chlorosis the prime evil, so far as the heart is concerned, is unquestionably the spanæmic condition of the blood, however that may have been brought about. In this state the great defect is the diminution of the cellular element, the serum remaining more or less normal; sometimes it is increased in amount, but it is never diminished, though occasionally its constituents are more or less

altered. Thus, even when at rest, the heart has at least as much work to do as usual, and sometimes it has more, while it is much less able to do it; and whenever any exertion is made the breathlessness due to the state of the blood necessarily calls upon the heart for more than usual effort, which it is less than usually able to exert. The result of this is easily foreseen; residual accumulation commences, and gradually increases, in the left ventricle, for it is there that the strain primarily falls, and it is the left ventricle which suffers first and most in these and similar cases, as we learn from Stokes¹⁷ and others. By and by the dilatation increases so much that a relative inadequacy of the mitral valve is established, and regurgitation is set up. We must not forget, however, that during all this time a similar process of congestion, from residual accumulation, has been gradually going on in the left auricle first of all, next in the lungs, and then in the right ventricle, terminating, of course, in the systemic venous system, where all such processes naturally end. This process is essentially slow and gradual, but it is constant, and proceeds *pari passu* with the changes in the left heart, the passage to the right heart being probably not long delayed by the distensibility of the pulmonary vessels. It is important also to remember that, in accordance with a well-known law, some degree of hypertrophy is set up in probably every case, but this must necessarily be imperfect, and must lag much behind the tendency to dilatation.

From these preliminary explanations it is obvious that in chlorosis we may have a murmur at all the four cardiac orifices; but I am quite certain that most observers will agree with Hayden that its most ordinary position is the base of the heart, and that it has no "definite line of propagation, as is the case with organic murmurs in this situation."¹⁸ This absence of any distinct line of propagation up the aorta, innominate, and carotid, of course at once renders untenable the classic hypothesis of Hope,¹⁹ Bellingham,²⁰ Potain,²¹ &c., who hold that the cardiac hæmic murmur is always aortic in its origin. Marshall Hughes,²² feeling the force of this objection, was, I believe, the first to suggest the orifice of the pulmonary artery as the source of the murmur, forgetting that under ordinary conditions any purely hæmic, intracardiac cause of murmur must act at least as forcibly at the aortic orifice as at the pulmonary. And Marey did not improve matters by suggesting that the basic murmur was due to the formation of fluid veins at the aortic orifice, by the lowering of the tension within the aorta itself,²³ because most observers are agreed that the murmur is at least as loud at the pulmonary orifice as at the aortic, while the accentuated pulmonary second indicates an increase of tension within that artery—a condition also proved to exist by the gradual development of dilatation of the right ventricle. The cause, therefore, supposed to favour the production of the murmur at the aortic orifice is wholly inoperative at the pulmonary orifice, where nevertheless the murmur earliest makes its appearance and is usually loudest.

This basic position of the chlorotic murmur in its early stage of course excludes Parrot's supposition of its being always tricuspid in its origin; nevertheless, I acknowledge that a greater or less dilatation of the right ventricle, as well as a more or less evident undulation or pulsation of the jugular veins, are always present, though I think that most observers will agree with me that a systolic tricuspid murmur is the rarest of all the murmurs which may be found in connexion with chlorosis, unless, indeed, we regard the murmur not unfrequently found in the mitral area in advanced cases as of tricuspid origin, which there are some good reasons for doing.

We must not also forget that although the reasons already stated preclude the idea of the origin of the chlorotic murmur in its commonest form at either the aortic or the pulmonary orifice from ordinary causes, yet it might possibly arise at the pulmonary orifice from compression, during the ventricular systole, of the artery uncovered by retraction of the lungs, as has been pointed out by Quincke.²⁴ In all cases, however, where the murmur

¹² On Diseases of the Heart and Great Vessels, 3rd edit. (London, 1862), p. 96.

¹³ Medical Times and Gazette (1863), p. 637.

¹⁴ Diseases of the Heart and Aorta (Dublin, 1875), p. 268.

¹⁵ Hayden, loc. cit.

¹⁶ Hayden, op. cit. p. 278.

¹⁷ Op. cit., p. 372.

¹⁸ Hayden, op. cit., p. 252.

¹⁹ A Treatise on Diseases of the Heart (London, 1839), p. 106.

²⁰ Diseases of the Heart (Dublin, 1853.)

²¹ Dictionnaire Encyclop. des Sciences Médicales, t. iv., p. 392.

²² Guy's Hospital Reports, second series, vol. vii., 1861, p. 161.

²³ Physiologie Médicale de la Circulation du Sang (Paris 1863), p. 479.

²⁴ Berliner Klinische Wochenschrift, 1870, No. 21, p. 250.

depends solely upon this cause, and the lungs, as in chlorosis, are texturally sound, full inflation of them causes the murmur completely to disappear. But this is not the case in chlorosis; a full inspiration with subsequent holding of the breath certainly makes the murmur somewhat fainter, but never causes it to disappear.

Besides, however, all those causes of basic murmur already shown to have no influence in the production of the chlorotic murmur, in its early stage at least, there is one other of more recent discovery which in its essential nature, the position of the murmur to which it gives rise, and its mode of propagation, exactly fulfils all those conditions which we find associated with the chlorotic murmur.

Skoda mentions that in cases of dilatation and hypertrophy of the right ventricle, dependent upon incompetence of the mitral valve, a systolic murmur is often audible in the pulmonary artery; this, he adds, is "perhaps" dependent upon softening (*auflockerung*) of the internal coat of the dilated artery.²⁵ So far as I know, Skoda was the first to point out the existence of this murmur, which has long since been recognised as of no infrequent occurrence in mitral regurgitation, whether accompanied by stenosis or not. If stenosis be present, it is somewhat remarkable to hear, as we often do in such cases, a presystolic murmur over the apex, and a systolic one in the region of the pulmonary artery. Skoda's idea as to the causation of this murmur was not long maintained; it was not difficult to show that the internal coat of the pulmonary artery in these cases is not softened, and that its condition in those cases where this murmur had been present does not differ from that in those other cases where this murmur had been absent. Meyer,²⁶ who next took up this question, endeavoured to show that this murmur was produced by the propagation of the vibrations of the mitral valve to the wall of the pulmonary artery. But, supposing this to be an efficient cause, it is difficult to see why these vibrations should not be propagated to the aortic wall which is attached to the same fibrous ring, rather than to the pulmonary artery which is attached to the same fibrous ring with the tricuspid valve, and belongs, as it were, to a different heart. If it were produced in this way, we ought to have the systolic murmur loudest in the aortic area in these cases of mitral regurgitation, instead of in the pulmonary area; but this we know not to be the case. Bamberger supposed this systolic murmur, audible in the pulmonary area in cases of mitral regurgitation, to be due to an abnormal relaxation of the coats of the pulmonary artery. It may be well to give his own words. He says this murmur "seinen grund höchst wahrscheinlich in der durch die andauernde Erweiterung bedingten Erschlaffung der Gefäßshaute hat."²⁷ And I especially quote Bamberger's words, because Naunyn has said—"Bamberger und ihn folgend Gerhardt geben an; es sei das bei mitralis insuffizienz in der gegend der Pulmonal Klappe hörbare systolische Geräusch eine folge der abnorm hohe Spannung, in welche die wandung der Pulmonalarterie unter solchen Umständen versetzt würde." I need not say that Bamberger's own view is utterly devoid of any rational foundation whatever; while, on the other hand, it is unnecessary to discuss the opinion ascribed by Naunyn to Gerhardt, as he,²⁸ as well as Paul Niemeyer,²⁹ have now both adopted the view originally propounded by Naunyn himself.³⁰ By a careful examination of these cases, Naunyn appears to have distinctly made out that the position of maximum intensity of the murmur in them is not over the pulmonary artery at all, but one or two inches to the left of it, and in the same plane, just where anatomy teaches us that the appendix of the left auricle comes up from behind. Naunyn therefore regards this murmur as propagated to the chest wall from the left auricular appendix; and he supposes the vibrations which constitute the murmur to be conveyed to the auricular wall by the fluid veins formed by the regurgitating blood at the auriculo-ventricular opening, which carry with them the vibrations originating at that orifice to the auricular wall

upon which they impinge. I entirely agree with this explanation given by Naunyn; and though he has not specially referred to the chlorotic murmur as originating in this way, it is not, I think, difficult to show that the history of the development of this murmur, as well as its primary position of maximum intensity, fully confirm this theory of its causation.

It is a difficult matter to explain why a murmur of regurgitation, which always originates at the auriculo-ventricular opening, should be at one time audible at the apex and at another at the base. But some approximation at least to an explanation may be attained if we consider that sounds are only audible vibrations, that in fluids these vibrations originate only at some part where the stream is constricted, and are not only communicated to the constricting walls at the point of origin, but are also carried with the fluid veins and transferred by them to any solid upon which they may impinge. In these circumstances it is obvious that a murmur of mitral regurgitation may be audible either at the apex or the base, according to the relative readiness with which its vibrations are transferred to the chest-wall in either situation. If the vibrations are powerful, they are readily propagated to the ventricle, and make their appearance as a distinct murmur at the left apex, which under most circumstances of mitral regurgitation comes more directly in contact with the chest-wall than the auricle—at first, at all events,—and for long continues to do so, because the cardiac muscle in these cases being fairly firm, consecutive dilatation is only very gradually developed. It is quite otherwise in chlorosis. In this disease residual dilatation is the essential lesion of the heart, and the murmur only becomes developed as a late result. By this time the left auricle, which very early commences to dilate, has become so enlarged that the appendix is frequently visible as a pulsating tumour between the second and third ribs on the left side, while the left apex is gone, separated from the chest-wall by the dilated right ventricle lying in front. How trifling a dilatation of the right ventricle is sufficient to produce this displacement of the left apex, even in the normal state of the heart, may be readily ascertained by anyone who places his hand over the ventricular area and holds his breath. In a very few seconds the left apex will be found completely to disappear, and that without any perceptible development of the impulse of the right ventricle. So it is in chlorosis; the right ventricle speedily becomes sufficiently dilated to displace the apex beat without its own impulse being developed in any marked manner, though that ultimately follows in due course. By this time, or soon after, the left ventricle is so dilated that during its systole the edges of the mitral valve are unable to meet, and regurgitation results. The fluid veins thus formed are of low tension, the vibrations originating at the auriculo-ventricular orifice have little force, so that they are propagated but feebly to the wall of the left ventricle, which in its turn, from its deep-lying position, transmits them with difficulty to the chest-wall, where they become audible only as an impure first sound. On the other hand, these vibrations readily pass with the fluid veins to the wall of the left auricle on which they impinge, and the auricular wall being already somewhat tense from unusual dilatation, and being also, from the same cause, in more than usually close apposition to the chest-wall, these vibrations are readily transmitted to it, and become audible as a soft murmur in the area of the auricular appendix, around which it is propagated to an extent commensurate with its own loudness and the resonance of the chest-wall.

Various other circumstances may combine to facilitate this mode of propagation of the murmur of mitral regurgitation, but the conditions described are certainly efficient, and are more or less present in all cases in which this mode of propagation is observed.

By and by, however, without there having been any disappearance or diminution of the basic murmur, the impure first sound at the apex becomes changed into a loud, blowing murmur. And this may occur in two ways: first, the dilatation and subsequent hypertrophy of the left ventricle may be so increased that the vibrations originating at the auriculo-ventricular orifice become forcible enough to be transmitted to the chest-wall in the region of the apex; or, second, the right side may become so dilated that its apex occupies the normal site of the left apex, and a tricuspid

²⁵ Abhandlung über Percussion und Auscultation, 3rd Ed. (Wien., 1844), S. 223.

²⁶ Virchow's Archiv, Bd. iii., Heft 2, S. 277.

²⁷ Op. cit., S. 93.

²⁸ Lehrbuch der Auscultation und Percussion, 2nd Ed. (Tübingen, 1871), S. 233.

²⁹ Handbuch der Percussion und Auscultation (Erlangen, 1870), Bd. ii., Abtheilung i., S. 140.

³⁰ Berliner Klinische Wochenschrift, 1868, No. 17, S. 189.

may thus be mistaken for a mitral murmur. Both kinds of cases may be met with, and require to be differentiated one from the other.

There is no doubt, also, that cases occasionally do occur in which we have—besides the auricular and any other murmur which may be present—a distinct systolic murmur originating in the aorta and propagated along the arteries; and there is every reason to believe that the same thing may occur in the pulmonary artery, quite apart from any external source of pressure in either case, and from internal causes alone. This will be found to occur at a somewhat advanced period of the disease, when the primary dilatation of the ventricles has been followed by some degree of hypertrophy; so that, as Beau³¹ has pointed out, a larger wave than usual is thrown into the arteries, not only therefore producing vibration waves in the arterial coats themselves, but very possibly also forming fluid veins at the arterial orifices, from the relative constriction produced there by the abnormal friction of the chlorotic blood—a relative constriction which, in some cases at all events, may be supplemented by some slight dilatation of the aorta itself, due to relaxation of its tissues. For the production of the aortic systolic murmur, therefore, in such cases, we have the possible occurrence of two efficient causes, sometimes one of them, and at other times the other, predominating; while of course similar causes must coexist in similar relation to the pulmonary orifice, and be followed there by similar results.

The sequence of the cardiac phenomena in chlorosis is entirely in accordance with the pathological view just propounded. Whatever may be the ultimate cause of the chlorosis, *spanæmia* is its first evident symptom, and of this the earliest sign is a *venous hum—bruit de diable*—in the right jugular vein. As the result of this *spanæmia*, the cardiac muscle becomes relaxed, and its cavities consecutively dilated from residual accumulation. The earliest indication of the delay of the blood, resulting from this residual dilatation, is *accentuation of the pulmonary second*. This is speedily followed by a *systolic murmur* in what I have termed the *auricular area*, an inch or two to the left of the pulmonary area, and in the same plane where a distinct pulsation is usually both to be felt and seen. This murmur radiates of course all round its position of maximum intensity, but is most readily propagated to the right from the resonant property of the sternum. With this murmur there is always associated *an impure first sound in the mitral area*, and in many cases this gradually passes into a *systolic murmur*. When the chlorosis is cured these murmurs disappear, precisely in the reverse manner to that in which I have described them as arising, every trace of the auricular pulsation vanishing long before the venous hum ceases to be heard. This systolic auricular murmur coincides in time with the systole of the ventricles, its rhythm is ventriculo-systolic, its position basic, differing in both, *toto cælo*, from the so-called presystolic murmur, which is apical in position, and auriculo-systolic in rhythm, coinciding with the time of the contraction of the auricles; the former murmur is also soft in character and high in pitch, the latter rough and low. In chlorosis this auricular murmur is always present, though it may be associated with other murmurs, which, as I have shown, may arise in this condition at all the four cardiac orifices. Whenever we have *spanæmia* we have a tendency to residual dilatation, and consequently to the development of this murmur, which therefore may be heard in chlorosis, chorea, all the exanthematic and other fevers, in syphilis, and all other diseases known to be accompanied with *spanæmia*, as well as in the exhaustion following excess in tobacco-smoking or sexual indulgence. Where the residual dilatation is the result of extraordinary or unusual exertion—heart strain as it is termed, which may be the result of arterial atherosclerosis alone, the extra exertion being confined to the cardiac muscle—this murmur is often the only detectable sign of the commencement of what may, if neglected, terminate in serious cardiac disease; the only other indications of an affection which may prove so serious being uneasy feelings in the left breast, which are too often pooh-poohed as nervous, air and exercise being recommended for a heart already unable to perform its ordinary lifework. As dilatation is the most serious affection that can befall the heart, and as this

auricular murmur is the earliest detectable indication of dilatation, I need not say that I regard it as perhaps the most important sign we possess of cardiac disease, all the more so that it is an early one, while I look upon its detection as a most valuable indication for treatment, a treatment which of course must be varied according to the other concomitant phenomena.

The auricular pulsation between the second and third ribs to the left of the sternum, which is so prominent a phenomenon in many cases of chlorosis, is sometimes so well marked that it is mistaken for serious arterial disease. A case of this kind was sent to me in 1869³² by the late Dr. Hughes Bennett as one of aneurism of the aorta. This case was one of the first which gave an impetus to my inquiries as to the causes of the murmurs and of the pulsations in the pulmonary area. Treated at first as a case of aneurism, this patient was subsequently recognised to be a case of simple chlorosis with auricular pulsation. She was discharged cured. I have seen her frequently since, and I saw her a few months ago perfectly well and free from all trace of murmur or pulsation. My present resident physician, Dr. George A. Gibson, has succeeded in obtaining tracings of this auricular pulsation by means of an ingenious adaptation of Marey's cardiograph, and his paper will fitly conclude what I have to say upon this subject at present.

[The paper referred to will appear in the ensuing number of THE LANCET.]

ON SYPHILITIC PHTHISIS.

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THE recent debate before the Pathological Society on the subject of Visceral Syphilis brought out in strong light this point at least with reference to pulmonary syphilis—that, except in the form of gummatous nodules or complicated with tubercular phthisis, syphilitic disease of the lung is of rare occurrence in the post-mortem room; and the united experience of pathologists made it evident that the recognition of the morbid effects of syphilis in the production of pulmonary disease is a matter of considerable difficulty.

Having made some clinical observations on the subject for some years past, I attended the meeting of the Society with much the same views that have been expressed by Dr. Robinson in his "Notes on Syphilitic Phthisis," in THE LANCET of May 5th last, hoping to find in the experience of the pathologists some corroboration and explanation of the observations, which it seemed advisable to withhold until confirmed and interpreted by pathological evidence; but although the subject has occupied attention for the last three years, the post-mortem appearances have been seen so seldom, while the clinical examples have been comparatively numerous, that the publication of such details as have been collected appears to be justified by the fact that this form of phthisis is very slow in action and is seldom *per se* fatal.

In order to be assured that the form of syphilitic phthisis described by Dr. Robinson was identical with that which I believed to be syphilitic, I wrote to Dr. Robinson to ask permission to examine some of his cases. This permission he very readily accorded, and he showed me such instances as he had then under observation. They presented all the characteristics which I have found common to such lesions, and thus showed that we were both agreed on the subject; the same results having been obtained by two observers from different platforms of observation.

Dr. Robinson has approached the subject from the experience of a military hospital. He has tabulated all cases of syphilis which presented subsequently signs of pulmonary disease, and deduced the characteristics of the disease from observations on syphilitic patients. The experience of a hospital devoted to pulmonary affections has elicited the same result from another point of

³¹ Op. cit., pp. 366 and 565, &c.

³² Ellen C—, admitted to Ward 13, Nov. 4th, 1869.