are plates of what he calls polymenorrhœic stratifications, which exactly resemble the two clots I have described. He says polymenorrhœa, by which he means profuse menstruation, may produce stratifications in the uterus.

Dr Grailly Hewitt, in his book on Diseases of Women, says, "Coagula may form within the uterine cavity in connexion with uterine hæmorrhage of all kinds, after labour, in consequence of the presence of polypi, cancer of the uterus, profuse menstruation, etc.

"Coagula, not recent, may present a tolerably firm, dense, grayish, fibrinous-looking surface. The want of organization in the mass, and the presence of blood-corpuscles, would assist in the diagnosis of the nature of the substance. The centre of the mass, moreover, generally exhibits a clot of a darker colour comparatively unaltered, which was the original nucleus of the formation."

Again he says, "In some rare cases coagula of some size have been expelled from the uterus unconnected with the previous occurrence of conception."

In the Pathological Society's Transactions, vol. xv. page 169, a case in point is recorded.

The information derived from these two authors shows that it was ignorance on my part which led me to suppose that I had observed a class of cases not before described. Nevertheless, though I cannot strictly claim originality in this paper, I am convinced, from experience in connexion with a medical society of which I am a member, that the existence of such cases is not so well known amongst medical practitioners as it ought to be. Ignorance of the fact that such cases do occur may throw suspicion upon those who are perfectly void of offence, and blast the reputation of many a virgin.

Nothing can be more annoying than to harbour a suspicion which you dare not mention; and if this communication should relieve any one from such a position, it will not have been made in vain.

ARTICLE V.—*Clinical Lectures on Diseases of the Heart.* By GEORGE W. BALFOUR, M.D., F.R.C.P.E., Physician to the Royal Infirmary.

II.—*On the Murmurs and other Physical Signs Distinctive of Mitral Stenosis—continued.*

GENTLEMEN,—You have learned, I hope, from the previous lecture, that the essence of the cardiac affection in mitral stenosis is obstruction to the onward current of the blood at the mitral valve; and we deduce this from the fact that the only murmurs

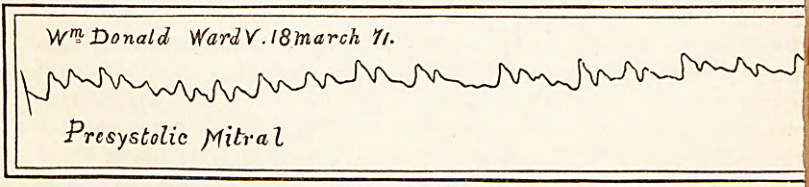
specially distinctive of this form of valvular lesion are those which, from the position at which they are best heard, are known to originate at the mitral valve, and which, from the time during which they are heard, are recognised as occurring occasionally during the ventricular diastole, but specially during that part of the pause occupied by the auricular systole. Now, these murmurs, as I have already described them, are perfectly distinctive of mitral stenosis, and when once the existence of one or other of them has been recognised, disease and deformity of that valve may be predicted, with a perfect certainty that it will be found after death. Unfortunately these murmurs are not always present, and sometimes, though occasionally audible, they are not always to be heard at the period of examination, that very time when it is of the greatest importance that a cardiac lesion should be recognised if present. In most cases the diastolic portion of the murmur is entirely wanting, and in many even the auriculo-systolic portion seems, if we can trust a hospital history of two or three years at least, never to have been audibly present at all; but, besides this, each portion of the murmur may vanish temporarily for a longer or shorter period, and this sometimes in a most remarkable and unaccountable manner. Further, as a necessary result of the condition of the valve in the most usual form of mitral stenosis (the diaphragmatic valve) we have regurgitation of the blood backwards into the auricle. In perhaps the larger proportion of such cases this regurgitation is unaccompanied by murmur; in a smaller proportion we have both the auriculo-systolic and the ventriculo-systolic murmurs constantly present; in a much smaller proportion we have these murmurs presenting themselves in an irregular fashion, both murmurs being present for one or two beats, the auriculo-systolic or the ventriculo-systolic, chiefly the latter, recurring alone for an irregular number of intervening beats; while in a certain number of cases, the exact proportion of which is as yet unknown, we have the ventriculo-systolic murmur alone and constantly persisting.

Such, then, are the variously complicated forms of murmur which may be heard in cases of mitral stenosis. It would be easy to narrate to you cases illustrative of each variety, but it is quite unnecessary, and would only oppress your memories with unnecessary details. So far as murmurs are concerned, those only are distinctive of mitral stenosis which have been already described, the essential part of them being the presystolic murmur; whenever that is recognised as persistent or can even be picked out as occasionally recurring amid a complication of other murmurs, the case is clear and wants no further proof. When, however, no presystolic murmur, or no murmur at all, can be detected, we are forced to unravel the case by a careful investigation of the various subsidiary phenomena evolved during the cardiac action, as well as of the relative condition of the different cardiac cavities, and the mode of propagation of any murmur which may be present. In this way we shall very frequently

be enabled to detect the stenotic condition of the mitral valve even where no murmur at all is present, or where the only murmur audible is one of regurgitation. And this branch of the inquiry is of all the greater importance that it is on the data obtained from it that our prognosis must in every case be founded; the mere diagnosis of mitral stenosis, so readily made in some cases, affording but one, and that in many respects the least important, element in such an inquiry.

Among the most remarkable of these subsidiary phenomena we must reckon irregularity of rhythm, which, always present, I believe, in a greater or less degree, becomes so marked in some cases as to constitute a diagnostic phenomenon of considerable importance. The cause of this great irregularity is not very evident; it does not depend upon dilatation of the left ventricle, which in pure mitral constriction is always rare and never excessive; it is often just as little marked in cases of great debility with dilatation of the right ventricle, as when we have the left ventricle, hypertrophied from secondary causes, acting well to the last, and the right ventricle comparatively unaffected. On the other hand, general debility seems to have some effect in developing it, and it is always lessened by means directed towards improving the general health and cardiac power, but it is chiefly the coexistence of pyrexia which produces its most striking development, and that quite independent of the amount of stenosis present; and it occurs in other forms of cardiac disease under similar circumstances, though never in so marked a degree; its true cause, therefore, would seem to be some lesion of innervation. Of course, in mitral stenosis the elements of irregularity, as I have already described them in connexion with the reduplication of the second sound, are so persistently present that it is no wonder that they should become pre-eminently manifest on the occurrence of any disturbing cause. The following diagram (fig. 1) graphically represents this extreme irregularity in a sufficiently marked manner.

Fig. 1.



CASE 6.—The patient William Donald, aged 37, admitted into Ward V. on 10th March 1871, was a labourer recently working in a gaswork where he was much exposed to sudden alternations of heat and cold. He had always enjoyed good health till four weeks ago, when he caught a severe cold; since then he had been fever-

ish and ill, with gradually-increasing debility, some cough, and bloody expectoration. On admission he looked somewhat exhausted, his breathing was hurried, his pulse 120 and extremely irregular. On percussion the cardiac dulness was found to be normal, the apex beat being in the usual position. On auscultation over the mitral area, the first sound was found to be of slightly thumping character, impure, but unaccompanied by any bruit; no thrill was perceptible over the apex beat. Between the second and third ribs on the left side close to the sternum, the second sound of the pulmonary artery was heard greatly accentuated. Between the second and third ribs on the right side close to the sternum, the aortic second sound was found to be weakened. At the base generally, but most distinctly about mid sternum on a level with the fourth rib, marked reduplication of the second sound was to be heard. On percussing the lungs, over the left lung anteriorly, the upper portion was found to be natural, and the lower lobe somewhat dull; posteriorly there was a small dull patch about the centre of the scapular space, from the lower border of the scapular space the lung was dull. On the right side posteriorly there was dulness at the lower border of the scapular space, otherwise percussion over the right lung was normal; over the dull portions respiration was more or less obscured; over the other parts vesicular mingled with occasional coarse crepitating rattles. The expectoration was catarrhal, largely mixed with pure blood.

The diagnosis in this case was bronchial catarrh, occurring in a person affected with mitral stenosis, and culminating in pulmonary apoplexy.

The history of an ordinary febrile attack, accompanied by cough, catarrhal expectoration, and occasional coarse crepitation over the chest, persisting four weeks after seizure, sufficiently confirmed the first part of this diagnosis. The copious expectoration of blood, coupled with the existence of dull patches, over which the respiration was obscured, also confirmed the latter part of it.[1] While I based the coexistence of mitral stenosis upon—1*st*, the slightly thumping character of the impure first sound; 2*d*, the extreme irregularity of the pulse, which is so marked in no other form of cardiac disease that I am acquainted with; 3*d*, the reduplication of the second sound, which, though it is found under other circumstances, is never so persistent as in mitral stenosis; and, 4*th*, the weakened character of the aortic, and the strongly accentuated character of the pulmonary second sound. The first of these phenomena duly recognised by a practised ear is, I believe, quite pathognomonic of mitral stenosis, and is almost invariably associated with more or less irregularity of the pulse, though that is not always so extreme as it was in the present case; while the persistence of the third pheno-

[1] The occlusion of the bronchi passing through the hæmorrhagic masses preventing the development of bronchial breathing in such cases, thus in so far confirming Skoda's theory of the mode of origin of this phenomenon.

menon described aided greatly, when coupled with the two former, in confirming the diagnosis, which was further strengthened by the subsidiary, and, from a diagnostic point of view, comparatively unimportant phenomena of pulmonary accentuation and hæmoptysis.

After admission, the patient gradually fell into a state of low muttering delirium, from which he was roused by the moderate exhibition of stimulants and the free use of digitalis. For a couple of days subsequently he seemed to rally, and faint hopes of ultimate recovery were beginning to be entertained, when symptoms of subacute peritonitis set in, and in two days afterwards he died, on the 20th March, ten days after admission, the cardiac symptoms remaining unaltered to the close. At the dissection, the small intestines were found matted together by recent lymph, they were congested and friable, and the omentum was adherent to their surface, three ounces of bloody serum being found in the recto-vesical pouch, etc. The heart weighed 17½ ounces; the right auricle was distended, the left auricle flaccid; both ventricles distended with black clots; the right ventricle was somewhat dilated, its auriculo-ventricular opening admitted seven fingers; the left ventricle was somewhat hypertrophied, its cavity not dilated; the aortic and pulmonary valves were competent; a small vegetation was attached to the corpus aurantii of the posterior cusp of the aortic valve. On laying open the left auricle several vegetations were seen encroaching on the auriculo-ventricular opening, one of them quite cartilaginous but movable, the other softer; the mitral orifice admitted two fingers easily, its aortic cusp was shortened and thickened, especially at its free margin. Each pleural sac contained about a pint of fluid. On the right side there were many adhesions, on the left only a few at the apex. The upper lobe of the left lung contained two dark-coloured solid hæmorrhagic masses just beneath the pleura, one two inches and a half from the apex on the posterior aspect of the lung, the other two inches and a half from the interlobular fissure on its anterior aspect; while its inferior lobe contained four similar masses, all just in contact with the pleura, two as large as an orange, the other two the size of a walnut; the largest was in the interlobular fissure, and extended down to the base, the second largest was in the posterior aspect of the lung. The apex of the lower lobe of the right lung contained four hæmorrhagic masses.

The post-mortem appearances, which are extracted from the pathological records of the Royal Infirmary, thus completely confirmed the diagnosis.

In the following very similar case the diagnosis was confirmed, fortunately for the patient not by any pathological research, but by the subsequent development of those bruits which are recognised as pathognomonic of the condition diagnosed.

Case 7.—A. M., aged 31, admitted into Ward V. 23d March 1871. This patient had not previously suffered from rheuma-

tism, nor from any serious illness, and had been always able for his work up to the commencement of his present attack, which dated only five days back. He had then been seized with shivering and slight febrile symptoms, accompanied almost from the first with hæmoptysis. He was somewhat breathless, with hot skin, some cough, copious bloody expectoration, pulse 120, extremely irregular, its sphygmographic tracing precisely resembling that of the former case. On examination his heart was found normal in size, the apex beating between the fifth and sixth ribs, slightly within the nipple line. The heart's action was extremely irregular, and on placing the hand over the apex beat, a thrill running up to the apex beat was occasionally, but not always, perceptible. On auscultating over the mitral area, the first sound was found to be somewhat impure and slightly thumping in character, but no bruit was audible. The pulmonary second sound was accentuated, the aortic somewhat weakened, the two sounds not being simultaneous, but presenting the phenomenon of a reduplicated second sound, most distinctly audible about mid-sternum, opposite the end of the fourth rib. The auscultation and percussion over both lungs was normal, except at the back, posteriorly in the infra-scapular regions, where over both lungs some dulness existed, and the vesicular murmur was much weakened, almost extinct. The diagnosis in this case was slight febricula, complicated with mitral stenosis and pulmonary apoplexy. He was treated with tincture of digitalis and squill, ten minims of each in water four times a day. Under this treatment the hæmoptysis gradually lessened, the pulse became somewhat more regular, and fell to 90, and after about a fortnight's treatment an ordinary presystolic murmur was heard in the mitral area, and a diastolic murmur became audible at the end of the fourth left rib, just where it joined the sternum, the reduplication of the second sound continuing to the last. He was sent to the Convalescent Hospital, and at the end of three weeks again readmitted for a slight exacerbation of his cardiac symptoms, but without hæmoptysis. The murmurs continued as just described, till he was discharged relieved, and declaring himself fit for work.

These cases exhibit in a marked manner the extreme importance of great irregularity of the pulse and cardiac action as a symptom of mitral stenosis, especially when associated with hæmoptysis; and though extremely suspicious under all circumstances, these symptoms may, I believe, be regarded as quite pathognomonic when accompanied by tolerably persistent reduplication of the second sound, particularly if associated with presystolic thrill, notwithstanding the absence of all murmur. The presence of pulmonic accentuation, though never absent in these circumstances, is really a phenomenon of comparatively slight importance in specializing the diagnosis, as it depends entirely upon pulmonic congestion, which may be the result of so many various lesions.

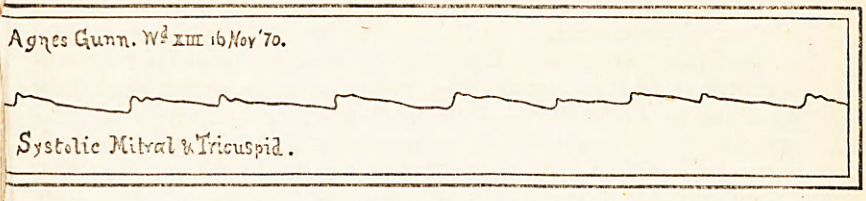
Apart, however, from any presystolic murmur or from any extreme irregularity of the pulse, mitral stenosis is occasionally accompanied solely by a systolic mitral murmur, and it is always a matter of interest, and sometimes of importance, to determine the exact nature of the lesion on which this murmur depends. The two following cases, besides the great individual interest which they possess, are of importance as showing the data upon which this diagnosis may be founded.

CASE 8.—Agnes Gunn, æt. 18, admitted into Ward XIII. on 27th October 1870, complaining of dyspnœa, cough, and spit. There is no history of rheumatism, but she had suffered from chorea about two years ago. About twelve months ago she was admitted with symptoms similar to the present into Ward XI. under Dr Laycock, and in the record of her case it is stated that there was then a mitral murmur succeeding and partially replacing the first sound, and that the tricuspid dulness was increased. At the date of admission under my care, her face was observed to be full, puffy, and rather livid, her expression dull and heavy, yet not free from anxiety. There was some swelling of the feet and legs, great dyspnœa, considerable hard cough, and copious watery expectoration. On passing the hand over the lower part of the sternum, the right side of the heart was fouud to beat with a slow heaving pulsation, the left apex being only faintly felt in its normal position between the fifth and sixth ribs, the impulse between the third and fourth interspaces on the left side being felt to be greater than normal. On percussion, the cardiac dulness was found to extend at one inch from the sternum from the upper border of the third rib down to the liver dulness; and on a level with the fourth rib, from one inch to the right of the sternum transversely across for a distance of six inches. On auscultation in the mitral area, the apex beat is found to be accompanied by a slight thump, followed by a bruit which replaces the first sound; the second sound is distinctly audible at the apex; this systolic murmur is lost as we pass to the left, and, about the middle of the infra-axillary space, is replaced by an impure first sound, which continues audible on auscultating round to the base of the scapula. Over the tricuspid area, a loud blowing murmur completely replaces the first sound. At the base, between the second and third ribs on the right side, an impure first sound is heard followed by a feeble second; between the second and third ribs on the left side, the first sound is also impure, and followed by a distinctly accentuated second sound. There is no venous pulsation in the neck, but the veins are somewhat small, empty, and obscured by the general turgidity. The pulse is 76, small, weak, and somewhat irregular; its character is graphically represented in the annexed diagram (fig. 2). She continued under treatment, mainly with digitalis and squill, till the 29th December, when she was discharged, relieved from her more urgent symptoms.

In this case the main elements relied on in proof of the existence of mitral stenosis were — 1*st*, The slight thump preceding the

systolic bruit, and constituting, as it were, an imperfect and abortive attempt at a first sound, a phenomenon which in my experience is

Fig. 2.



never absent in such cases of mitral stenosis as have no presystolic murmur attending them, quite apart from the existence of any systolic murmur, and which very possibly is in some cases merely the tail of the rough auriculo-systolic murmur, which it phonetically exactly represents, but it is too sharp and short for my ear, at least, accurately to time; in other cases it undoubtedly is due to the imperfect closure of the mitral valve, as can be accurately enough ascertained by timing it by the carotid pulse, the thump in such cases being more prolonged. 2*d*, The fact that the soft systolic murmur was not distinctly propagated past the middle of the infra-axillary space. This is, according to my experience, an invariable accompaniment of mitral stenosis; not that the systolic murmur is altogether lost, but that the remnant of the first sound is so much more distinctly propagated, that it speedily becomes merely an impure first sound, in which the systolic blow is more or less distinctly to be recognised according to the amount of regurgitation present. In the pure regurgitant murmur this is never the case; the murmur may become fainter as we pass to the angle of the scapula, but it never assumes any of the elements of a first sound. These two phenomena are to me distinctive, and are never observed save when mitral obstruction is present. As corroborative phenomena, we had the early occurrence and rapid development of dilatation of the right side of the heart, proving the obstacle to the onward flow of the blood, an obstacle still further evinced by the diminished arterial pulse (*vide* upstroke in fig. 2), the weakened sound of the closure of the aortic valves, and the persistence of the pulmonic accentuation, notwithstanding considerable tricuspid regurgitation, as evinced by the loud systolic tricuspid murmur. This proof derived from the state of the right side is even more strong in cases in which there never has been any coexistent bronchitis, as is occasionally observed.[1] And even in this case, though there was evidently great pulmonary congestion and copious expectoration of watery fluid, yet the absence of rhonchi and the rarity of

[1] Especially in Ellen Harkins, admitted into Ward XIII. on 20th April, and discharged improved on 17th June 1871. In her we had all the percussion signs of tricuspid dilatation with distinct jugular pulsation, but no tricuspid bruit, and an entire absence of any history of bronchitis.

crepitation showed that this condition was much more allied to the general turgescent œdema of the body than to true bronchitis; that it was, in fact, more an œdema of the lung than a true catarrhal condition, and therefore all the more valuable a proof of obstructed circulation.

On 27th March 1871, Agnes Gunn was readmitted for an exacerbation of all her symptoms, induced about a month previously by going on a cold day from the wash-tub to an outside well while scantily clothed. Her cough, shortness of breath, watery expectoration, and cardiac distress were much increased; the jugular veins, though still small, were now seen to pulsate distinctly synchronously with the heart, and her face, neck, and limbs, but especially her lower ones, were very œdematous. The condition of her cardiac organ was much as formerly, with this exception, that now the apex beat was very distinctly to be felt, and the result of this was that the impulse at the lower part of the sternum was apparently lessened. This was explained to be probably due to increased hypertrophy and dilatation of the right ventricle, which had pushed the left wholly backwards, the right apex being now the only one to be felt. It is obvious that this was the only explanation admissible in the face of an increasing cardiac affection, now fast becoming serious, and so largely implicating the right heart from the first, it was impossible to conclude that a left ventricle, defectively nourished and unable to hold its own from the first, could recover itself and again re-assert its position apart from any well-marked evidence of improvement in the onward circulation, which was entirely absent, while the view taken was perfectly consistent with the ordinary progress of such cases, though the change effected is only rarely observed in such an extreme degree. Gunn remained in the ward till her death, on the morning of 12th July. During this period she had an attack of hemi-chorea, the sufferings induced by it being rapidly subdued by hydrate of chloral and full doses of arsenic; but it persisted for many weeks as a slight twitching of the thumb and forefinger of the right hand, finally disappearing, however, many weeks before death. This choreic attack, however, rapidly broke down her cardiac power, and she never rallied, dying under symptoms of pulmonary apoplexy, with gradually increasing cardiac asthenia. The autopsy was made on 13th July, thirty hours after death. Her body was moderately fat, her legs very œdematous, her face and lips livid. On opening the thorax, the pericardium was found to be distended by from 15 to 20 ounces of very slightly turbid serum. The heart was considerably enlarged, weighing 16½ oz.; the right ventricle concealed the left, and formed the apex of the heart; it was much dilated and its walls hypertrophied, measuring half an inch in thickness; the tricuspid valve was slightly dilated, admitting five fingers easily; on its right cusp, near the free margin, there was a small vegetation about the size of a millet seed, soft and elastic; the right auricle was greatly dilated, not hyper-

trophied; the aortic valves were competent; the left ventricle slightly hypertrophied, not dilated; the opening of the mitral valve was so extremely contracted as only to permit the point of the little finger; it was very much thickened, and its cusps glued together by their margins; on the auricular surface of each cusp there was a row of small vegetations like millet seeds; the left auricle was slightly dilated, and its walls much hypertrophied, being about twice their natural thickness. The left lung contained two large and recent hæmorrhagic clots, presenting on section a dark venous surface; one of these was situate along the anterior margin of the superior lobe, extending from the apex downwards for a distance of four inches, while it measured transversely one inch and a half; the other was situate in the inferior lobe, near its anterior margin, and was about the size of a large orange; its circumference was pretty sharply defined. The right lung contained no recent extravasations, but in the inferior lobe, immediately beneath the middle of its external surface, there was a yellowish-gray patch of a wedge shape, with its base to the pleura, evidently the remains of an old hæmorrhage; over the surface of this lung, and especially at its anterior margins, there were some emphysematous patches. The liver weighed 4 lbs. 2 oz., was much congested, and slightly cirrhotic, the lobules being very distinctly differentiated by the interlobular cellular tissue. The spleen weighed 5 oz., was of firm texture, and contained two hæmorrhagic infarcti, each about the size of a walnut, tough in texture, and of a bright yellow colour. The kidneys were healthy, and weighed each $5\frac{1}{2}$ oz. The dissection in this case, therefore, completely confirmed the diagnosis during life, while the marked hypertrophy of the left auricle, in contradistinction to the simply dilated condition of the right auricle, in spite of the great preponderance of regurgitation on the right side, seems an evident physiological proof of the influence of obstruction in originating the disease—a proof which, I may add, is never wanting in any case of mitral stenosis, though, as a further evidence of its importance, it is always found to vary in degree according to the amount of obstruction present.

The next case which I shall quote is also very instructive as to the signs of stenosis where only the murmur of regurgitation is present. Case 9.—Alex. Milne, a printer, aged 17, admitted into Ward V. on 28th November 1870, complaining of palpitation, cough, shortness of breath, and pain in the cardiac region. His history was, that similar cardiac symptoms had troubled him from the age of six, and that to these a cough had been superadded about a year ago; since this time the pain in the cardiac region had been much worse; the patient was pale and anæmic-looking, he has never had rheumatism, his family history was unimportant. On inspection, greater pulsation than natural was visible over the cardiac region, especially at its lower part; the apex beat was distinct between the fifth and sixth ribs, $2\frac{5}{8}$ inches from the left edge of the sternum; on percussing downwards one inch from the left edge of

the sternum, cardiac dulness was found to commence at the upper edge of the third rib, and extended down to the liver dulness; on a level with the fourth rib the transverse dulness did not commence till the left edge of the sternum, but extended to the left for a distance of fully three inches; on auscultating over the mitral area, the first portion of a first sound was heard, followed by a loud systolic bruit, and closed by an imperfectly-heard second sound; this systolic murmur is propagated to the right, and is heard distinctly over every part of the right ventricle, followed by a reduplicated second sound, but is not so distinctly propagated to the left, becoming very faint after passing the centre of the infra-axillary space; over the right ventricle a slight but distinct heaving is conveyed to the ear by the stethoscope; between the second and third ribs, on the left side, distinct accentuation of the pulmonary portion of the second sound is audible, the second sound being here reduplicate, as it is also heard to be between the second and third ribs on the right side, where the aortic portion is heard tolerably natural. Over the jugular veins a hæmic murmur is audible, but no pulsation is visible. The pulmonary physical signs were normal, and the cough left him entirely after a short residence in the Infirmary. His digestive system is normal, except that he has little appetite for food. The urine is, however, smoky, contains numerous blood-corpuscles, a few small granular casts, and one-sixth of albumen.

The peculiar character of the first sound, the mode in which the systolic murmur was propagated, and the persistent reduplication of the second sound, gave me reason to state that in this case also we had to do with mitral constriction; while the tricuspid bruit and evident dilatation, with hypertrophy of the right side, gave a seriousness to the prognosis, which was not lessened by the persistent hæmaturia and the anæmic condition of the patient.

Under treatment his cough speedily ceased, his cardiac symptoms moderated, the pain ceasing, and even his kidney symptoms were modified, the tube-casts being no longer to be found, and the hæmaturia lessened, while his general health was much improved. He was sent to the Convalescent Hospital on 23d February. After being some time at home Milne became gradually worse, weaker, and more distressed by his cardiac symptoms, and he was in this state readmitted into Ward V. on 6th April 1871. His cardiac symptoms were unaltered, but his debility was greatly increased. His hæmaturia continued, and there was a good deal of general œdema. He was very shortly afterwards confined to bed, suffering much from orthopnœa; and after lingering on in this state he died exhausted on the 24th of May, having spitten a good deal of blood during the last few days of his life. At the autopsy there was found great general œdema of the body; the face, which had been livid before death, had become of a roseate hue; a quantity of froth exuded from the mouth; over both lungs there were numerous pleuritic adhesions; the left lung was congested and œdematous; the inferior lobe of the right lung was hepatized, the middle lobe

partially consolidated, and the base of the upper lobe also partially consolidated and wholly œdematous. The heart weighed 22 oz. The pericardium was universally adherent—the adhesions tough and fibrous; the right side was dilated and filled with dark clot, the tricuspid opening admitting 5½ fingers; the left side was excentrically hypertrophied and filled with a tough and partially decolorized clot; the mitral opening was constricted, admitting only one finger and a half, its cusps adherent and greatly thickened; the aortic valves were competent. The nutmeg liver weighed 3 lbs. 9 oz., the spleen weighed 12 oz., the kidneys weighed each 6½ oz.; they were enlarged, the capsules non-adherent, the surface a marbled-gray, studded with stellate vessels and hæmorrhagic spots, cortical portion speckled with yellowish opacities.

These cases, which might be largely increased in number, are sufficient to show the signs upon which I rely for the diagnosis of mitral stenosis where no murmur, or where only a murmur of regurgitation, is to be heard; they are, I believe, perfectly reliable, and are of importance, not only in regard to accuracy of diagnosis, but also for the prognosis, and still more for the treatment, of the case.

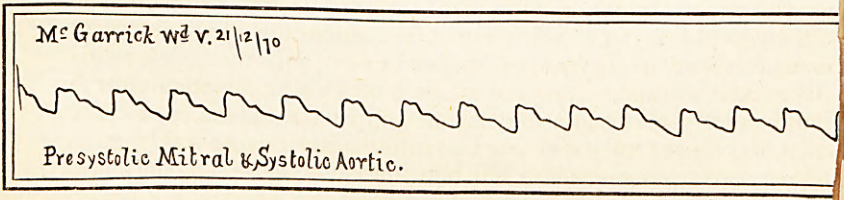
It will have been observed from the cases narrated, that a simple stenotic condition of the mitral valve leaves the left ventricle comparatively unaffected; but inasmuch as this is a rather rare state of matters, the left ventricle is usually found to be secondarily somewhat hypertrophied, this condition being due to the obstruction caused by hepatic cirrhosis as in Case 8, by disease of the kidneys as in Case 2, or by adherent pericardium as in Case 9, or by some obstruction in the aorta, as in the following case.

Case 10.—James M'Carrick, a brassfounder, æt. 18, first admitted into Ward V. on 25th October 1869, complaining of shortness of breath, palpitation, and hæmoptysis. Patient states that about a month ago, while wheeling a barrow up a hill, he lost his breath, was suddenly seized with a cough, and spat up some blood. The cough improved under treatment, but was always accompanied by expectoration of blood, and his dyspnœa on going up hill continued. Previous to this attack he had always been well, except that he has had measles, scarlatina, and enteric fever; he has never had rheumatism. His family history is good. He is short and slightly built, but his expression is natural, as is also his external conformation. His pulse is 86, and somewhat jerking. Heart's apex beats somewhat forcibly a little inside of the nipple line between the fifth and sixth ribs; on placing the hand over the precardial region a rough purring thrill is felt to precede and run up to the apex beat. One inch from the sternum the cardiac dulness commences at the upper edge of the third rib, and runs down to the liver dulness, and in the line of the fourth rib it extends from the left edge of the sternum to near the nipple for a distance of three inches. On auscultation over the mitral area a loud purring murmur is heard passing into a soft blowing one; the rough portion of

this murmur, tested by the carotid pulse, is auriculo-systolic in time, the soft-blowing portion ventriculo-systolic, and replaces the first sound, the two being separated by the impulse of the apex; a loud diastolic murmur is also heard, which becomes more distinct as you pass the stethoscope towards the base, where it is heard to follow the second sound, which is so faint that it almost seems to be obscure. In the aortic area there is a loud systolic bruit propagated into the arteries of the neck. The second sound is audible but somewhat weakened. In the pulmonic area the second sound is accentuated. A few bubbling râles were heard here and there over the lungs, otherwise his condition was normal, except that he had some *jactitatio e somno*. By 5th November his hæmoptysis had ceased, and he gradually improved till 7th December, when he was discharged.

On 16th December 1870 he was readmitted on account of increased cough, dyspnœa, and palpitation; no hæmoptysis. Since his discharge he has been constantly working, though obliged to be careful, and occasionally laid aside by dyspnœa for a day or two, but never for longer than a week at a time. On inspection a heaving impulse is seen and felt between the fifth and sixth ribs in a line with the nipple. This impulse is strictly localized, occupies a space two inches in width, and elevates the chest walls a full quarter of an inch; a purring tremor is felt over this region preceding the impulse. One inch from the sternum the cardiac dulness extends from the upper border of the third rib to liver dulness, and extends transversely from about one inch to the right of the sternum for a distance of five inches. On auscultation over the mitral area a loud purring murmur, auriculo-systolic in time, is heard running up to the apex beat, and followed by an impure first sound, no systolic murmur being now audible. In the aortic area a loud systolic murmur is audible, which is propagated into the arteries of the neck; the closure of the aortic valves is distinctly heard, but somewhat more feebly than usual, and followed by a faint diastolic murmur feebly audible over all the cardiac area, but most distinctly heard just where the fourth rib joins the sternum on the left side. In the pulmonic area, the systolic murmur is heard (propagated) followed by an accentuated second sound, and that by the faint diastolic murmur already described. The pulse is 84, small, soft, and without jerk, neither does it become jerky on elevating the arm; it is graphically represented as follows:—

Fig. 3.



He had some cough when admitted, but was otherwise normal. Treatment again restored him to a state of comparative health, but not so perfectly as formerly, as during the past year he has repeatedly called for advice, and is now again in Ward V., having been readmitted on 25th November 1871. He does not seem to have grown any since he first came under treatment; he looks pale and thin, and complains much of debility; his cardiac phenomena are unchanged, except that the diastolic murmur is much more feeble and more distinctly localized to the spot indicated by the junction of the fourth rib on the left side with the sternum. This case is especially instructive in three respects—1*st*, It shows the influence of obstruction in developing hypertrophy behind it as exhibited in the greatly increased force and extent of the apex beat, distinctly that of the left ventricle as shown by the absence of any increased influence of the lower part of the sternum or of any systolic bruit developed there; 2*d*, It shows the influence of persistent mitral stenosis in developing dilatations of the right ventricle, evinced by the increased breadth of dulness; and, 3*d*, It shows in a very marked manner the non-importance of mere murmurs as signs of organic disease in comparison with the other physical signs. Here we had a loud systolic apex murmur which has completely disappeared, the physical cause of this unquestionably persisting, as revealed by the permanence of the presystolic murmur; but we had also a diastolic murmur so loud and extensive in its propagations as to simulate so fully aortic regurgitation that in the earliest report of the case it is referred "probably" to this cause,—the jerking nature of the pulse, due to pulmonary hæmorrhage, contributing an important quota to this "probably;" yet now it is restricted to a limited area directly over the mitral valve, and from the distinctly audible closure of both sets of sigmoid valves, the absence of every other sign of aortic regurgitation from the pulse (there being neither distinct delay nor jerking), or from the heart (dilatation of left ventricle in excess of hypertrophy), and its common occurrence as a mere symptom of mitral stenosis, as already described (p. 442, etc., *antea*), it is most probably to be referred to the latter cause alone. It might, indeed, depend on a mere opening—ruptured aneurism or other lesion—in the aortic valves; but in that case the increased hypertrophy of the left ventricle would rather have intensified than lessened the murmur, especially if we consider the comparative invariability of aortic regurgitant murmurs,[1] while the variablity of mitral murmurs, as evinced in this as well as in other cases, coupled with the persistent absence of other physical signs of aortic regurgitation, is a very strong argument in favour of its purely mitral origin.

Cases of mitral stenosis are of such frequent occurrence that I could readily multiply these narratives; I have selected a few only, the history of which seems to me to bear importantly upon

[1] *Vide* Dr Sanders on the Variations of Organic Cardiac Murmurs, *Ed. Med. Jour.*, January 1868, p. 566.

the diagnosis, and I have only in so far selected them as they happened to present themselves to my memory, for I possess many records of at least as much importance as those narrated. The next case I shall relate has a bearing on the prognosis respecting which sufficient data are still wanting.

CASE 11.—Andrew Ormiston, a miner, aged 18, admitted into Ward V., on 1st November 1869, complaining of shortness of breath, and pain in the cardiac region. Patient states that he has been ailing for about a year, but before that was always in good health, with the exception of having had chickenpox and scarlatina in childhood—in particular, has never suffered from rheumatism. He has been in hospital once during the past year for similar complaints. He is troubled with shortness of breath, aggravated by exertion or coughing; his expectoration is grayish, but sometimes bloody. He has occasional paroxysms of pain in the epigastrium, coming on about half-an-hour after taking food. He is small for his years, but of a ruddy countenance and fresh healthy appearance. He is pigeon-breasted, the chest being flattened laterally; on palpation a thrill is felt over the heart's apex preceding its impulse. Cardiac dulness extends from the upper edge of the third rib to the liver dulness, and transversely along the upper border of the fourth rib from the right edge of the sternum to the nipple, a distance of 4½ inches. On auscultating over the mitral area a well-marked rough bruit is heard preceding and running up to the first sound; the pulmonary second sound is accentuated, the aortic somewhat weakened. The accentuation of the pulmonary sound is markedly increased whenever he happens to have a cough, and also after exertion. Pulse 88, soft, and only slightly irregular. Respiratory system normal. Tongue clean, appetite variable, bowels regular, urine normal. During his residence in hospital his general symptoms varied according as he suffered from catarrhal complication or not, or from temporary dyspepsia. He was sent improved to the Convalescent Hospital on 15th December. He was recommended to give up mining, and if possible to procure some easy indoor occupation. For some time he took charge of a library; latterly he has been assisting in a grocer's shop. I have seen him frequently since his discharge, and once quite recently. He has grown considerably, and is now a well-grown, healthy-looking lad of his years, though quite unfit for the ordinary exertion of manual labour, his sufferings being always increased by any unusual exertion. The peculiarity and importance of this case is, that he is a contemporary of Cases 9 and 10, that he presents similar symptoms *quoad* the mitral valve itself, but has no complication cardiac or otherwise, that he has been enabled to take considerable care of himself, and that the result is, that he is still alive, and presents none but local symptoms of the cardiac disease under which he labours, showing that under even tolerably favourable circumstances uncomplicated mitral stenosis is not incompatible with life and a moderate share of health; but this, which might be

shown by the record of many similar living cases, is perhaps best proved, *quoad* life at least, by the comparative infrequency of mitral stenosis as a cause of death in the pathological theatre, compared with its frequency as a symptom of disease in the wards—a fact borne out by your own daily experience, coupled with that of the Pathologist of our Infirmary.

You will remember, then, that mitral stenosis is to be recognised by the existence of a rough murmur preceding and running up to the apex beat and the carotid pulse, which is pathognomonic of this lesion; that this murmur may, and often does, commence as a soft, blowing, and sometimes musical diastolic murmur following the second sound, becoming rougher towards its conclusion, audible sometimes over the whole cardiac area, more usually, *quoad* its diastolic portion, just over the mitral valve where the fourth rib on the left joins the sternum; that any portion of this prolonged murmur may be absent, and that it may either be continuous or broken by a pause equivalent as to rhythm, though not as to duration, to the normal pause of the heart's action, the length of the pause being dependent on the amount of stenosis present, *i.e.*, the shorter the pause the greater the stenosis; that to be distinctive of mitral lesion a diastolic murmur must be unaccompanied by any signs of aortic regurgitation, must have a clear second sound preceding it, and be accompanied by no jerking thrill of the pulse increased or produced by elevation of the arm, and no dilatation of the left ventricle evinced by depression of the apex beat; the distinctive signs of pulmonary regurgitation are unknown, but the affection is so extremely rare that it may be practically excluded. That extreme irregularity of the pulse associated with pyrexia is indicative of the possible existence of mitral stenosis, and this is corroborated by the existence of a thump preceding or accompanying the apex beat, by hæmoptysis, signs of enlargement of the right side of the heart, and accentuation of the pulmonary second sound. That a simple systolic apex bruit to be indicative of stenosis of the mitral valve must be accompanied or preceded by a more or less evident thump, the bruit ceasing at or about the middle of the infra-axillary space; being there replaced by a more or less impure first sound, the diagnosis of stenosis in such cases being confirmed by great irregularity of the pulse, and by great accentuation of the pulmonary second sound, and especially if accompanied by evidence of tricuspid regurgitation, and the other signs and symptoms of dilatation of the right heart.

Persistent reduplication of the second sound, though in itself comparatively unimportant, is always to be regarded as strong confirmatory proof of mitral stenosis when it accompanies any of the phenomena just described.

The etiology of mitral stenosis is as yet wholly unknown. This much alone is certain, that in the vast majority of cases it is wholly unaccompanied by any preceding history of rheumatism.

The prognosis is equally uncertain. Simple, uncomplicated, mitral stenosis is not a disease of itself very fatal to longevity. Judg-

ing from my own experience, I should say that a deduction of one-third from the mean future expectancy of life is quite within the mark, and more likely to be exceeded than the reverse. Any cardiac complication, however, will materially reduce this expectancy, while intercurrent, though apparently irrelevant, complications are also of course liable to produce a similar effect. Comparing it therefore with other cardiac diseases, my own experience would lead me to place it in regard to life expectancy as second in the roll—aortic obstruction, mitral obstruction, aortic regurgitation. In purely hospital practice, aortic regurgitation is by far the most fatal of cardiac valvular diseases. So far as expectancy of life is concerned in this disease, however, the experience of private practice is required to correct that obtained in hospitals; and mitral regurgitation being the order of fatality in which such diseases ordinarily follow each other in a pathological register, consecutive disease of the right side of the heart being the true test of expectancy of death in all except aortic regurgitation, in which, as I shall by-and-by show, certain causes exist which lessen the value of the symptoms of this affection, or rather render their absence of less importance in prognosis.

The treatment of this disease I shall reserve till I come to speak of the treatment of valvular heart affections generally.

ARTICLE VI.—*On the Treatment of Club-Foot.* By THOMAS ANNANDALE,, F.R.S.E., Surgeon to the Royal Infirmary, and Lecturer on Clinical Surgery.

FURTHER experience in the treatment of club-foot has led me to modify and improve the apparatus described and figured in the Journal for February 1869. The two principal obstacles encountered in the treatment of talipes varus by means of a simple apparatus not carried above the knee are—1*st*, The difficulty of retaining the appliance in position, owing to the constant movements of the parts and restlessness of the patient. 2*d*, The difficulty of acting effectually upon the inversion of the anterior portion of the foot, and, at the same time, preventing the rotation inwards of the whole leg.

Finding that my former apparatus was not thoroughly efficient in overcoming these difficulties, I have endeavoured, during the last two years, to perfect it, and have, by means of the improved appliance, obtained the best possible results.

The adaptation to the sole-plate of a soft leather boot, lacing in front, was the first improvement to my former apparatus in regard to the better fixing of the foot; but as even this was not completely efficient, I took advantage of a suggestion which has been formerly made (not by myself), that the two feet should, in some way, be secured together. This latter addition has proved most valuable, not only in restraining the movements of the parts, and so prevent-