of honour to students, and the directors and approvers of schools and teachers, but that we should become the centre of medical research, the mainspring of all the developments of medical science. And now that we can do it, why should we not do it? Why should we not have buildings and accessories, imposing in their grandeur and adequate in their accommodation, laboratories, complete in their fittings, instruments and materials, rooms for conferences, and proper places for the storing of records and results? Why should not our Fellows and Members who are moved with the desire to advance medicine and surgery, by inquiry and experiment, be presented with as great facilities as are to be found in any Continental city or university—such as are so profitably enjoyed by Pasteur, Ranvier, Brown-Séquard, and their colleagues and assistants—advantages hitherto unapproached on this side of the channel, but which our transatlantic brethren are not slow to emulate? I feel that to support the honour of our profession, to fulfil our duties as the representatives of surgical science, justly to carry out the intentions of our late president and benefactor, we ought to be magnificent in architectural and constructive outlay, active in personal work, and liberal in the encouragement and help we proffer to those who come to make use of the opportunities which our College will give, and promote the object for which we are all so deeply concerned and so heavily responsible.

I am encouraged to augur the completion of such a scheme as this, for rendering original investigation in the future infinitely more easy, precise, and valuable, than the surgeons of the past ever hoped for, and to believe in the accomplishment of its designs, when I think that the will of such meetings as this must be obeyed; and feel the conviction that the men of Birmingham will be among the first to avail themselves of it.

I have spoken to you of the past and the present. The future is in your hands; and I appeal to you, as men typical of the thought and action of the profession, actuated by the highest moral sentiments, seeking and mutually guarding your honour as a body; chasing out, with instinctive repugnance, that which, ignominious in principle or practice, has accidentally intruded itself among you, and conscious that—by assuming the highest functions of your calling, in the investigation of national interests, in the promotion of public good by giving counsel to princes and legislators, by such a devotion of your energies, by such a sacrifice of personal consideration as is involved in bettering the condition of the people, in shielding them from disease, and in the prolongation of their healthy lives—you will add lustre and dignity to the private confidence, gratitude, and sympathy, which the profession has already gained by its power over actual suffering and sickness, and by the skill, conscientious vigilance, and humane tenderness with which you make that power manifest.

"I speak as unto wise men, judge ye what I say."

HEALTH OF DERBY.—Mr. Iliffe devotes much space in his annual reports to an examination of the circumstances attending the prevalence of zymotic diseases, and the measures adopted for restricting their diffusion. As regards the compulsory notification of infectious disease, Mr. Iliffe is convinced that each successive year brings with it a greater confidence in its power for good, and also a deeper and fuller conviction of its appreciation by the general public. There were, in all, 187 deaths registered in 1882, and 144 in 1883, from the seven principal zymotic diseases. The experience gained during the prevalence of typhoid fever pointed in the direction of danger from immediate personal infection; and, in view of this, Mr. Iliffe suggests whether more pressure should not be used to secure the removal of sufferers. The absence of a separate hospital for the reception of cases of scarlet fever was largely responsible for the fatality recorded from this cause in 1882—a fact which is prominently brought before the authority for serious consideration. The sanitary work carried on of late years has been of a general rather than of a special kind. A large number of houses have been provided with town water, and in many others the sink-drains have been disconnected from the main sewers. The town is full of cesspools, middens, and ashpits; and, until some action is taken to secure that thorough reconstruction of the whole system of sewerage which is so urgently required, little can be done towards improving the present state of things. The general death-rate was 18.7 for 1882, and 18.3 for 1883.

PRESENTATIONS.—Mr. John Charles Davies, of Rhosllanerchrugog, has been presented with an illuminated address, a gold watch, and a dinner-service of forty-eight pieces, on the occasion of his marriage.—Mr. George Henry Cressey, upon leaving Lowestoft, has been presented with a gold ring, and a stationery- and writing-cabinet, by the brothers and friends of the "Star of the East" Lodge, M.I.O.O.F., to which he had been medical officer for several years.

A LECTURE

ON

VALVULAR LESIONS OF THE HEART.

Delivered at the City of London Hospital for Diseases of the Chest.

By J. MILNER FOTHERGILL, M.D.,

One of the Physicians to the Hospital.

In the present consideration of the subject, the matter of the diagnostic value of murmurs, and of each individual murmur, will not be discussed, because this is already known to all of you. The characters of each murmur, by which, in all probability, the form of valvular disease is indicated, are too well known to need any reference to them here; but there are some further points to be considered, which are of interest to the practitioner, anent valvular lesions.

In the lower forms of life, the heart is a mere pulsatile muscular sac; but, as the higher forms are reached, direction is given to the blood-current (caused by the contraction of this hollow muscle), and regurgitation is prevented, by the mechanism of valves. When any injury is done to the cardiac valves, enlargement of the muscular sac behind the seat of injury takes place—a change known as "compensatory hypertrophy"—by which the defect in the valve is compensated more or less perfectly. If the valve-lesion be but a small one, the less of this compensating hypertrophy is required; and the less this, again, the more easily and the longer is it maintained. This is a matter as important in each case as the correct determination of the seat of the lesion. Having made up your mind as to the seat of the lesion, the next matter is to calculate the extent of that lesion—whether it be large or small. It may be so small as practically to amount to little, being only felt upon exertion. While a quiet life is maintained, little is known of the injury done to the valve. If the lesion be a large one, its consequences will be proportionate to it. The more extensive the injury, the greater need for compensatory hypertrophy; the more mutilated the valvular apparatus, the more the heart is reduced to its primitive condition of a mere pulsatile sac. Such change was well marked in a case alluded to by Dr. Broadbent in his recent Harveian Lectures on "Prognosis in Heart-Disease." He said: "When the disease is established early in life, even extreme damage to the valves may be so far compensated by dilatation and hypertrophy that the subject may live for many years, as was illustrated by a case in which acute rheumatism, at the age of 14, in 1868, left behind aortic regurgitation, attended with a maximum amount of dilatation and hypertrophy, together with secondary mitral regurgitation and exaggerated signs of every kind, which the patient survived for fourteen years, earning his livelihood as a greengrocer. He died suddenly in May 1882, during convalescence from slight subacute rheumatism, when the aortic valves were found to be practically non-existent, and the heart weighed 4½ ounces." This was a very remarkable case, which, by Dr. Broadbent's kindness, I saw several times with him at St. Mary's Hospital. Despite his severe valvular injury, this patient was a very active fellow. How came this about? Because of the immense compensatory hypertrophy his system built up, and maintained for years. To meet the valvular lesion, the heart had grown to something like five times the weight of the heart of an ordinary man, namely, 4½ ounces. This case will give you a vivid idea of the effects of a large valvular lesion upon the muscular walls of the heart—where the organism is equal to setting up compensation. The case was a very unusually marked instance of a large lesion in the valves compensated by a huge muscular growth; and the immense development of the muscular walls endowed the patient with a corresponding vigour.

I wish to bring under your notice some illustrations of valvular lesions of varying gravity. In the tall man, you see an instance of a comparatively small lesion, mitral insufficiency, easily compensated, enabling him to follow his work as a fine tool-maker, but at times needing a little digitalis to brace up the heart-walls. From time to time, the muscular wall yields a little, and requires the invigorating effect of some agent which increases the energy of the muscular contractions. But the mischief in the valves does not progress; and as long as the compensatory hypertrophy in the right ventricle can be maintained, so long the man is capable of earning a living. When, and under what circumstances, the compensation will fail, is unknown

to me; probably it will not be for a considerable time, and then will be caused by some failure in the nutrition, or in the general health. In the next case, you see another instance of a non-progressive valvulitis, a small lesion, easily compensated, and enabling the owner of this heart to lead a pleasant life, provided exertion be avoided. Both cases have been under my observation now for five or six years, and have undergone no change for the worse during that time. In the next case, you see an illustration of an extensive lesion. Here the patient is comfortable so long as quiet is maintained; in the recumbent posture she is very comfortable, but moving about at once tells that the injury done to the mitral valves is extensive. The features also tell this by their hue—the dark hue of venous congestion.

Here are also two aortic regurgitant cases. One is in an early stage, but with a very bad prognosis, being linked with a "gouty heart;" in the other, the mischief is far advanced, with a dilated aorta and bounding carotids. By contrasting these cases with one another, you will be able to readily estimate the extent of injury inflicted in each case. And, remember, the more extensive the lesion, the greater the need for compensating hypertrophy. What does this tell us? It tells us this: that, when we find great muscular growth, we have to deal, in all human probability, with an extensive valvular lesion. Especially is this the case with aortic valvulitis, and most of all with aortic regurgitation. When there is great enlargement of the left ventricle, then the inference is that there is a considerable backward current through the aortic valves. When you detect a fine regurgitant murmur, with only slight enlargement of the left ventricle, you are entitled to assume that the mutilation of the aortic valves is but small. Whether it is slight but progressive; or but slight, and, still more, static, is an important matter, of which something will be said further on. Stenosis of the aortic valves never leads to that massive hypertrophy of the left ventricle which is found with a considerable regurgitant current through injured aortic valves. Consequently, when you encounter a huge left ventricle, you recognise that you have to deal with an extensive injury of the valves, admitting a backward current flowing through them.

Such, then, is the significant change when the aortic valves are incompetent. But when the injury is situated at the mitral orifice, it matters little whether it be stenosis or insufficiency (in which mitral mischief contrasts with aortic disease, for in the latter it matters a good deal), then the resultant changes are not so obvious. In some, the enlargement of the right ventricle is readily detected by physical examination; in other cases, the lung covers the right heart so completely as to obscure the case, and the evidence is not to be found upon percussion, but rather by the physiological test of the effect of effort. Take out your watch, and count the respirations during perfect bodily quietude. Then let the patient make an effort; if the lesion be considerable, the respiration will quickly tell of the damming of the blood in the lungs, and the lessening of the breathing-space—necessitating more rapid respiration. The accentuation of the pulmonary second sound will also be of value in helping you to form your estimate of the case. The greater the block at, or rather behind, the mitral orifice, the fuller the pulmonary circulation, and the more forcible the closure of the pulmonary valves, and, consequently, the louder the sound so produced.

It is from these data that you are to decide upon the extent of a mitral lesion, rather than from any mere loudness of the murmur. The mere loudness or faintness of a murmur carries with it very often no diagnostic or prognostic significance. Indeed, congenital mitral obstructive murmurs, of no significance, are often very loud. Having, then, found your murmur and located it, the next thing to be done is to calculate its significance. It may mean a large lesion, a small lesion, or no lesion at all. To determine this question of significance, you must proceed on the lines just laid down. If this last were more insisted upon, we should not encounter so frequently cases where a grave prognosis has been indulged in without sufficient justification. Valvular lesions of the heart are not a mere matter of murmurs, their presence or their absence. Indeed, there are cases where it is possible to feel absolutely certain that a mitral lesion exists, though no murmur can be detected; just as a loud murmur may exist in life, and no morbid change to account for it be discoverable after death. It is as well, however, in practice, when a murmur is heard, to proceed on the hypothesis that it tells of valvular mischief, and not to dismiss this view until the evidence against it is convincing. Having found your murmur, determined its seat, and its extent, your next aim is to ascertain whether it is static, and non-progressive; or whether it is progressive, that is, going on from bad to worse at a measurable rate.

And now your acquaintance with pathology comes into play. Your studies in the dead-house are worthless, unless they can be made to

possess a practical value; and, in the matter at present under consideration, your knowledge of morbid processes is your lighthouse. If you merely look at the mutilated valves, and the extent of the injury, and the amount of mural change found therewith in the dead-house, your pathological studies will be of comparatively little use to you. Let me tell you why. There are two well recognised forms of valvulitis. The first, common in youth, is an active process, doing a certain amount of damage; but often without tendency to go on from bad to worse. The injury is done, and our hopes lie in compensating hypertrophy, that is, in the growth of the muscular fibrillæ. The second, belonging to advanced life, is a slower, chronic, and progressive valvulitis. To which form does a case before you belong? In other words, in a case of a mitral lesion in an elderly person, is the case one of old static change, where the muscular hypertrophy is wearing out? or is it one where a comparatively recent change has been set up, with a tendency to go from bad to worse? It is a matter of no slight moment to distinguish betwixt the two; nor is this always done successfully. Of course, your common sense will incite you to plant one or two incisive questions upon the patient. "Have you been short of breath for long?" you ask. If you find that the patient has been unequal to much exertion for many years, but has not become perceptibly worse of late years, then the presumption is strongly in favour of an old standing static lesion. But if the answer be otherwise, and to this effect—"It is only lately I have found my breath getting short"—then the probability is that you have a new morbid departure before you. You are, however, not to rest satisfied with such a presumption; you must proceed to verify your view. And here your pathological knowledge comes in again. There is valvulitis and valvulitis. There is an acute form of endocarditis closely linked with acute rheumatism, but not confined to it. Here an acute inflammatory storm passes over the serous membrane, accompanied by a growth of connective tissue corpuscles in the structures beneath the serous membrane. Such is the ordinary history of the mitral disease found in young subjects; and in older persons, as these said young folks advance in years. At other times, but infinitely less frequently, the aortic valves are the seat of the mischief. Sometimes, both sets of valves are involved. The great matter of interest is not the inflamed serous membrane, but the growth of connective tissue corpuscles beneath it. Why? Because pathological connective tissue has an inherent tendency to contract. That is why. These connective tissue corpuscles contract in time, and, in so doing, distort the valve-curtains and their attachments. To how great an extent this distortion will attain is a matter, to speak broadly, of the amount of this neoplasm. If the cell-growth be small, the resultant distortion will be limited. If considerable, a large injury will be the result. Whether the mutilation will be limited, and the mischief be static, or the distortion will progress, is a matter of constitution. It was terrible, in the dead-house of Vienna, to find how swiftly the necropsy followed the original disease. In English patients, I am glad to say the interval is widely greater as a rule. But the constitution, or the irritability (relative) of the tissues is not the sole factor in the result. The line of treatment adopted has great influence over the result. This may fitly be termed the preventive treatment of valvular lesions of the heart.

While there is cell-growth going on beneath the serous membrane, it is clear that no avoidable strain should be thrown upon the valve-curtains, or the fibrous cords attached to them, in order to limit this cell-growth. Perfect rest in bed ought to be maintained until it can be reasonably assumed that no active cell-growth remains. By so restricting the cell-growth, you limit the resultant mutilation of the valves. Instead of giving tonics, and trying to get the patient up and about, common sense tells us, if we know anything of pathological processes, that absolute quiet should be maintained until the day of the development of these connective tissue corpuscles is over. When this growth has ceased, and the mischief is practically done and ended, then the condition of the muscular walls calls for our attention. But each matter in its true position, and order of precedence and sequence. It is enough to make one despair of the reasoning powers of one's species when one contemplates the routine-treatment of cases of acute endocarditis. The nature of the morbid process is forgotten, the lessons of the dead-house cast to the winds; and, with good intentions yet murderous action, a tonic is given as soon as the active symptoms have apparently passed away, and the patient is allowed to leave his bed. I have just used the word "apparently" advisedly. The articular symptoms have passed away, and the murmur caused by the flow of the blood-current over the inflamed serous surface may have disappeared; but you know, and ought to remember, that the cell-growth in the fibrous structure of the valves does not cease so quickly. Time must be allowed for this to be brought about; while the tension

on the valves must be reduced to a practical minimum by keeping the blood-pressure low. Rather the patient should be kept on chloral for a week or ten days, than be allowed to get up. Instead of such rational treatment of valvulitis, what do we usually see? So soon as the active symptoms have disappeared, the patient is allowed to sit up, is probably given a tonic, and, possibly enough, even some digitalis, or other agent increasing the energy of the ventricular contractions; and medical man and patient alike congratulate themselves on the satisfactory progress made. Very likely; but what do we too often find as a consequence? Before long, a murmur is once more audible, the significant indication that the cell-growth is contracting, as is its nature, and permanent mutilation of the valves has been established. It is of no good to be repentant now; just as it is useless to lock the stable-door after the steed has disappeared. The patient, whose progress was so satisfactory, is now the subject of a valvular injury, which will, if at all large, cripple him or her through life. If the mischief wrought be not extensive, it is not because of the judicious action of the unreflecting medical attendant. Whatever the extent of the lesion, the time usually comes when the mischief becomes static; and then our hopes rest on the setting up of compensatory hypertrophy; but it is infinitely more desirable to attempt to limit the valve-mutilation at the time when this may be done. The lesion may be so small that a murmur may only be heard when the heart is a little distended by the effect of some effort; that is, the valve-curtains are still competent to close the ostium ordinarily, but admit of a leak when the ostium is enlarged by a distended ventricle; or no murmur may be audible until the ventricle yields, from malnutrition or disturbance of the general health; and when the heart is once more well fed, it is no longer heard. It may be more extensive, and the murmur permanent, yet permitting a light occupation to be followed; or, as in the case of the girl just seen, it may be so large as to be incompatible with any exertion.

But, whether great or small, the injury is done, and is non-progressive in the bulk of cases. Nothing can be done to restore the original condition of the valves, or to make them again competent. For the rest of the patient's life, the maintenance of the compensatory hypertrophy is the object of our aims and hopes. This may be managed for years under favourable circumstances, and especially if the injury occur in early years; but the day does come when the heart-wall begins to finally yield, and the symptoms consequent upon muscular failure manifest themselves. There is no change for the worse in the valvular lesion, but there is a very decided change for the worse in the heart-wall.¹

Widely different is the course when the inflammation of the valves is of the sclerosing character found with the gouty heart; while the mitral valve is the seat of the mischief usually in acute valvulitis, the aortic is rather the seat in chronic inflammation. But there are numerous exceptions in each case.

In this chronic valvulitis, you find first a hypertrophied left ventricle, and a tight pulse with a loud second sound, due to the forcible closure of the aortic cusps. This condition may go on for an apparently indefinite time, but at last a tell-tale murmur is detected. The high blood-pressure has produced an atheromatous change in the arteries, and therewith a like morbid change in the aortic valves, in the case of the man I had hoped to have shown you. I have watched him from a time anterior to any murmur till the present, when a very distinct lesion has been developed. Usually, I say, such endarteritis is found at the aortic orifice; but, in some cases, it is seated at the mitral orifice. When you detect a faint mitral murmur in a person of mature years, the next point to determine is this—does this murmur indicate a chronic progressive lesion, one where advancing valve-change calls for corresponding hypertrophic growth to compensate it; or does it tell of an old standing static lesion? I said before, you plant an incisive question or two upon the patient; but that is not enough; you must also observe. You are taught, in your educational course, the consequential changes found with each valvular lesion.

Now you must reflect upon what has gone on anterior to the development of the valvular lesion. Say it is a case of mitral stenosis—the most perplexing, perhaps, of all the forms of valvulitis in the gouty heart. In cases of mitral stenosis, the result of an acute endocarditis, you are told in your text-books, "The left ventricle is small, and disposed to become atrophied." But, instead of that, you find an unmistakably large ventricle giving a powerful impulse. Why? Because this hypertrophy preceded the development of the valvulitis.

¹ The time of life when mitral valvulitis is set up is a matter of moment. The older the individual, the less the power to set up that hypertrophic growth—that compensation in which our hopes are founded. The younger the patient, the more easily it is established.

It is not the consequence of the valve-change; rather it is the cause of the valve-change. The powerful ventricle with the high blood-pressure has thrown such a strain upon the mitral valve-curtains, that a chronic inflammatory condition is set up; in other words, trophic changes are set up in the fibrous structures of the valve, corresponding to the trophic change in the muscle. Whether it is the aortic or the mitral valves, or both, which become affected, there remain the powerful ventricle and the high blood-pressure, with consequent strain upon the valves; and bad goes on to worse. Possibly, too, there is a tendency to degenerative change established in the system. There may be atheromatous change in the valves, or an infiltration of earthy salts, the so-called ossification of the valves. But, whatever the pathological process, there are the concomitants of the gouty heart to be taken into consideration, in determining the nature of the lesion. The murmur, eloquent enough as to the fact of a valvular lesion being present, is dumb, is aphasic, simply as to the nature of the pathological process. The stethoscope tells of the mischief wrought, but can say nothing as to how it came about. For the last, something more is essential.

From what has been said here, if properly thought over, marked, learned, and inwardly digested by further thought on it, you will find the difficulty of deciding in each case before you what form of valvular change is present not insurmountable in many instances.

You have now determined (1) the seat of the lesion; (2) its extent; and (3) the form of valvulitis. But something more still remains for careful consideration, and that is the condition of the muscular wall; a very important matter diagnostically, prognostically, and therapeutically; for, as said before, nothing can be done to the mutilated valves. You may find a weakened wall with a recent acute valvulitis, where, if you feel fairly certain that no cell-activity remains—fairly morally certain—you may stimulate the vigour of the ventricular contractions, while improving the general nutrition. You may detect a by-past valvulitis, leaving a distinct measurable lesion behind it, which nature has met by a compensatory muscular growth, endowing the patient with the capacity to toil—met so well as to call for no aid from medicine. At other times the extent of the muscular change will tell of the amount of the valve-lesion; there is great hypertrophy, without much capacity for exertion. And in such case you must not expect that the massive hypertrophy will be long maintained. I said before that the nature of the lesion is comparatively unimportant in mitral disease; but it is widely different with aortic disease. When the valvular change at the aortic orifice is that of stenosis, then the ventricle has merely to deal with an obstruction to be overcome, requiring a comparatively small amount of hypertrophy, which is easily maintained for years; often many years. But with aortic regurgitation there is a new distending force in action, a very serious matter. The ventricle is no longer filled by a current welling in by the mitral orifice; there is a current driven backwards by the arterial recoil, through the defective valves. The ventricle yields before this new force, and this dilatation is more or less arrested by muscular growth. If the regurgitant current be but a thin riband, and the new force be met by a limited muscular growth, the case moves but slowly onward; sometimes so slowly that progress is not measurable. There is aortic regurgitation, and aortic regurgitation. There is the question of the extent of the lesion, and the proportionate muscular counter-growth. When the lesion is a large one, the resultant changes will be proportional to it. If it be a large mitral regurgitant lesion, the backward current is diffused over the pulmonary vascular area; the greater the current the more the pulmonary congestion, and the demand upon the right ventricle. When there is a large aortic regurgitant current, its distending force dilates the left ventricle, evoking hypertrophic growth for its arrest. And now let me impress upon you a broad rule, namely, the less the muscular growth requisite to compensate the valvular lesion the longer will it be maintained; the more massive the mural change the sooner it will wear out. Let that sink into your minds.

But, beyond the question of the amount of growth in the muscular wall of the heart, lies that of the structural condition of the muscle. As long as the structural integrity of the muscular fibrillæ is maintained, all goes well. But in massive hypertrophy, in no long time the coronary vessels become tortuous, or atheromatous, with a diminished lumen, and then a sufficiency of blood is no longer forthcoming for the requirements of the tissues of the heart-wall, and surely mural decay follows. In other words, fatty degeneration of the heart-wall is afoot, and failure of the heart in diastole first looms up, and then advances to the inevitable end; the case either ending suddenly, or the softened wall begins to yield, and the condition of dilatation extends with corresponding loss of vigour in the ventricle, and the usual sequelæ of a failing heart. When the muscular wall of the heart

decays and yields; then the nature of the valvular lesion, whether static or progressive, ceases to be of much importance; the mural failure is then the object of our thoughts. If there be really fatty degeneration spreading through the heart-wall, then all our efforts will in no long time be futile to stave off the inevitable failure of the ventricle in diastole. But let me impress upon you this fact, that the heart, as a muscle, is liable to be affected by malassimilation, or general defective nutrition, and may be temporarily weakened in consequence. Such a condition I have described as "Heart-Starvation;" a term which holds its own, despite the not very polite remarks of one critic, who perhaps wrote in haste.

Another critic in speaking of this matter says: "Dr. Milner Fothergill enters fully into the diagnostic symptoms of fatty heart, distinguishing, in his usual forcible language, between a fatty and a starving heart, to discriminate which is most important. Each is the effect of malnutrition: one, however, is a perfectly curable disease; whereas fatty degeneration of the heart, with atheroma of the arteries generally, and of the coronary arteries especially, is a truly senile change, which carries with it the gravest prognosis." Now, you will find that partial starvation of the heart-wall is the common cause why a patient, with a valvular lesion, comes for relief. The lesion has been there for years; no change has taken place in it. What, then, has made the difference? The nutrition of the heart has broken down; may be, because the demand made upon the crippled heart has been in excess of its nutritive powers; may be, because the assimilative processes have broken down. Whatever the cause, the heart cannot longer hold out, and the person is laid up, possibly with dropsy. In such cases (and they are common enough), it is usual to speak of the valvular lesion as the cause of the patient's break-down; yet the lesion has been there for years, and is unaltered. It is really the compensating hypertrophy which has become defective; it is the condition of the heart-wall rather than the mutilated valves which should command our attention, and give direction to our therapeutic endeavours. Where there is merely an ill-fed heart, rest, good nutrition, and appropriate remedies will in no long time restore the patient to his ordinary state, and allow him to go on again. But when the ventricle is yielding, because its muscle is the seat of a necrobiotic process, and there is fatty degeneration spreading through the muscular fasciculi, it is impossible to arrest, or even to appreciably delay, the downward progress of the case.

From what has just been said, I hope I have convinced you that, when you have determined the seat, the extent, the nature, of the pathological process in any valvular affection of the heart, there remains the associated condition of the muscular walls to be calculated. The detection of the presence of the lesion by its appropriate murmur is not all that is required. It is but the first step taken, the first rung of the ladder climbed; and much more remains to be done. You have not only to make sure that the murmur tells of actual change in the valves; you have to ascertain the extent of the lesion; its origin, and whether it is static or progressive; whether the muscular wall is well fed, or ill fed, or decaying. And all this can usually be done if the requisite attention and thought be given to the case. The stethoscope is invaluable; but other evidence is essential. That a murmur means diagnostically a valvular lesion, prognostically sudden death, therapeutically digitalis and iron, is a shallow view; which, however, obtains too largely. I trust that this lecture will leave all of you with a much wider grasp of the subject; indeed, if you had remained satisfied with that simple, but comprehensive, if erroneous, view, you would probably not have honoured me by your presence here to-day.

HEALTH OF CAMBRIDGE.—If Dr. Anningson's reports are brief, they are to the point. For 1881, the death-rate represented 15.7 per 1,000; for 1882, 16.2; and for 1883, 18.0 per 1,000. Scarlet fever was very fatal in 1882, the mortality, indeed, being higher than any previously noted. Dr. Anningson has little doubt that this disease is kept active by the migration of persons attending various fairs held in the town. Assemblies of this kind exercise a pernicious influence in the spread of infection; and, on more than one occasion, sanitary authorities have endeavoured, sometimes with success, to forbid them. The reiterated recommendations of the health-officer for the provision of a sanatorium seem at last to be approaching realisation, since a site has been selected and approved, and the boundary-wall built, and provision made in the rates for the current year for the cost of its erection.

SUPERANNUATION.—Mr. Robert Jones, late medical officer for the Conway District and the workhouse of the Conway Union, has obtained a superannuation allowance of £40 *per annum*.

THE PATHOLOGY OF PULMONARY EMPHYSEMA.

Introduction to a Discussion in the Section of Physiology, and Pathology at the Fifty-Second Annual Meeting of the British Medical Association.

By D. C. McVAIL, M.B.,

Dispensary Physician to the Western Infirmary, Glasgow: Lecturer on Medicine, Western Medical School.

LAENNEC was the first observer who placed on anything like a certain basis the pathology of pulmonary emphysema. So far as mere anatomical description of the condition is concerned, nothing of importance has had to be added to what Laennec had told us. He wrote very accurately regarding the enlarged air-cells, showing that the enlargement affected more especially the cells of the surfaces, the margins, and apices of the lungs. He attempted more than mere description of the condition: he endeavoured to account for the abnormal size of the cells. He tells, concerning this, that in certain cases of dry catarrh there is secreted a very thick tenacious mucus; that little portions of this become lodged in certain of the smaller air-tubes; that inspiration (in his opinion), being a stronger force than expiration, keeps adding to the air in the air-cells beyond the obstruction, while the mere expiratory force can send little or none out of these same cells past the obstruction; and thus he supposed that, by an action that he compares to the charging of the air-gun, the vesicles beyond the little portion of tough mucus become more and more distended with air, until they attain to the sizes we see in the *post mortem* room. But, by and by, Louis pointed out that it was not in the parts of the lung that were actually suffering from bronchitic action that the emphysematous condition was chiefly found; he showed that, while the bases of the lungs were most prone to bronchitis, the condition of emphysema was, for the most part, located—as Laennec himself well knew—in the anterior surfaces, margins, and apices.

About 1835, Dr. C. J. B. Williams suggested that the emphysema in the portions of the lung, where it is found, is complemental, or vicarious, to other portions of the organ which from bronchitis had become obstructed or collapsed.

In 1850 and 1851, Dr. W. T. Gairdner greatly amplified and strengthened the vicarious theory of Dr. Williams, and gave it the permanent place it has since occupied. He strongly insisted that the inspiratory force caused the non-occluded cells so to dilate as to fill the space that was occupied by the collapsed cells, when they were in their normal state of patency. He further gave an explanation of the cause of pulmonary collapse, that up to the present time has been almost universally received, and concerning which I shall presently have something to say.

Some years prior to the time when Dr. Gairdner wrote, Fuchs and Mendelssohn had advanced the theory that expiration had the chief or only share in bringing about the emphysematous dilatation. They alleged that, in the strong expiratory act of coughing, while the glottis is partially or wholly closed, there is a very violent compression of the thoracic walls by the expiratory muscles, and chiefly by the expiratory muscles that act on the lower portions of the thorax; and by this means the compressed air will be driven with great force into the parts of the lungs that are not supported by the chest-walls, and so will dilate greatly the air-vesicles of the margins and apices of the lungs. This view was advocated, twelve years later, in this country, by Dr. Jenner, who, not knowing anything of Fuchs and Mendelssohn's work, argued the case in favour of expiration very much on the same lines, and so ably and lucidly, that all writers accord him on the matter a share of honour quite equal to that attaching to his two predecessors. He argued most resolutely against inspiration having anything to do with the vesicular dilatation. He has since, in his article in Reynolds's *System of Medicine*, changed considerably in his attitude towards inspiration as a cause of emphysema, although he still holds to expiration as being chief agent in the majority of cases.

Niemeyer, while adopting the expiratory theory as applicable to very many cases, and the vicarious view as being true of some, sought to explain a number of cases of emphysema where there was no collapse—cases of substantive emphysema—as being produced by a heightened negative inspiratory pressure. In England, he is supported by Dr. Waters, of Liverpool, who, quite independently of Niemeyer, explained, by the inspiratory force, cases of vesicular dilatation, where there had been no cough, and where there was no collapse of lung-tissue. But both these eminent writers limit this explanation to a comparatively small number of cases. Prior to Niemeyer, Freund ad-