in a more satisfactory condition, and had the operation been performed at an earlier period, I consider there is good reason to think that he would have recovered, as the strangulation was relieved without the slightest injury to the gut, sac, or surrounding textures.

ARTICLE III.—*The Premurmuric Stage of Aortic Valvulitis.* By J. MILNER FOTHERGILL, M.D., Edinburgh, M.R.C.P.L.

(*Read before the Harveian Society of London, November* 7, 1878.)

THE importance of the early recognition of changes in the valves of the heart will be admitted by all. The question, then, arises, "Are we in all cases compelled to wait for the presence of a murmur before we feel certain that the aortic valves are the seat of disease?" To this, I believe, a negative answer may be returned. The cause of the second sound of the heart is so simple, that the difficulties are not great in comprehending how modifications of it may be of value, long before a murmur is developed. Tension of the semilunar valves is the cause of the second sound. Changes of blood pressure in the arteries, as well as structural changes in the cusps, will cause such modifications in the character of the second sound as shall possess a distinct diagnostic value. It is the consideration of these modifications, along with their associated conditions, that I ask for to-night. I do not expect full agreement with my views—and if I did, I certainly should not get it. Still, the subject is one which is worthy of investigation, in order to ascertain how far different observers agree, and on what points they differ. Through such discussion alone can any certitude of knowledge be obtained; and the Harveian Society is a most appropriate place for the consideration of questions relating to the circulation. We may, then, profitably discuss the question, "Are there other indications than a murmur, of commencing aortic valvulitis? are we always compelled to wait for the more remote and brutal answer of a murmur?"

In my opinion this is not always necessary; there are associations and modifications of sounds which teach us that the aortic valves are entering upon a pathological process before the morbid changes are sufficiently pronounced to develop a murmur. If so, it is well that they should be generally known and recognised, not only for such purposes as insurance, but in the interests of the patient. Further, too, our measures of treatment will be influenced by our knowledge of the causation and progress of aortic valvulitis. We know and recognise the fact, that an inflammatory storm of rheumatic endocarditis may light up a growth of connective tissue corpuscles in the mitral vela; which growth may progress, and too frequently does, after the storm which started it has passed away. Every

reader of Latham's instructive work knows that such valvulitis may be static or may be progressive—the prognosis being widely different in such case. Where the valves are but thickened, or their edges merely roughened by the interstitial growth, a mitral murmur may tell of the by-past storm for many years after it has occurred; and yet the patient may only be conscious of the injury done, on effort. The muscular compensation for such limited injury is easily made and maintained for long; the individual being but little the worse. We are not yet so certain that aortic valvulitis may be arrested, and the condition be rendered stationary; but it is probable that such may be the case in certain fortunate individuals. I know a faint aortic systolic murmur first heard by me in 1870, which is no louder at the present time than then, the gentleman presenting the appearance of perfect health; and, since 1870, has been passed as a first-class life in one of the most selective of our insurance companies. He at one time went in strongly for athletics of all kinds, but on marrying changed his mode of life entirely. To this is due, in my opinion, the static conditions of his aortic mischief.

A brief consideration of the associations of aortic valvulitis will help to elucidate the subject It may be occasioned by rheumatic endocarditis; but this form does not concern us here. There the murmur is soon audible. The associations of aortic valvulitis ordinarily are severe toil, especially certain forms of it, and the high blood pressure of lithiasis—of gout or chronic Bright's disease.

Aortic valvulitis is very common among men who wield a heavy hammer in foundries and ironworks, and as such is rife in Leeds, where my attention was first drawn to it by Dr Clifford Allbutt. Not only were there many such cases amidst the numerous patients of the Leeds Public Dispensary, affording plenteous opportunities for studying the disease, but I went to several foundries and examined these "strikers" when at work. After the severe exertion of striking during each "heat," *i.e.*, during the time the iron was sufficiently hot to be forged on the anvil, the circulation was much excited, and the heart beat violently. The "striker," in order to deliver deliberate and powerful blows, has to attitudinize himself —has to posture, in fact—and this entails his setting almost every muscle in the body into action when the blow is given; just as an oarsman has to fix his muscles when he makes a powerful stroke of the oar. In each case the trunk and legs are fixed, in order that the arms may be used more efficiently. Wardrop, many years ago, pointed out that many muscles cross arteries so as to compress the vessel somewhat when contracted; this he called "the musculo-cardiac function" of the circulation. When, then, these general efforts are frequently and persistently made, certain changes in the circulation are instituted. There is some hypertrophy of the left ventricle, with accentuation of the aortic

second sound. Not uncommonly there is some palpitation, and the heart's action is readily disturbed; there is excited action on slight effort. In such cases the occupation is abandoned, and some lighter form of labour adopted, and then the heart quiets down and no further stage is reached. I have examined several ex-strikers without finding any remaining evidences of the strain to which their heart had once been subjected. But in the majority of cases hypertrophy is developed, and a powerful ventricle distends the elastic arteries excessively, and the rebound is in proportion to the distension; the aortic valves are driven violently together, or in other words, the tension on them is excessive, and valvulitis is the result. This slow parenchymatous inflammation in the valves goes on along with hyperplasia of the muscular walls, and is a nutritive change, which may in the first place strengthen the valve cusps; but certainly the contractile tendency of pathological connective tissue mutilates the cusps, and renders them in time incompetent to close the ostium in the aortic recoil.

During this time the sounds are modified; the high tension to which the valve cusps are subjected produces accentuation of the aortic second sound. We are all agreed about that. But the question arises, Is there, in some cases at least, an intermediate stage betwixt the accentuation of the aortic second sound and the development of a murmur? This intermediate stage producing a muffling of the aortic second sound, as the thickened edges are brought together.

Walshe says: "The second sound at the base is rendered dull, and comparatively clanging by fibro-fatty thickening, without insufficiency of the sigmoid valves. Like the first, the second sound may be murmurish, temporarily or permanently. Very trifling insufficiency will probably thus modify its quality." Fuller writes: "The second sound becomes dull, and has its pitch lowered by want of contractility in the arteries, and also by thickening and impaired elasticity of the semilunar valves. In certain cases in which the elasticity of the arteries is great, and thickening or rigidity affects the free edges of the valves, the sound assumes a clanging character." Da Costa says, in his admirable work on Medical Diagnosis, too little known in this country: "The second sound is not so liable to be changed as the first. It is rendered somewhat duller by a thickening of the semilunar valves, and, on the other hand, more ringing when they are thin." While Hope writes, "There are exceptions to the production of systolic murmur by disease of the aortic valves. Thus I have frequently seen them slightly thickened by fibrous or steatomatous degeneration without producing murmur; though in other cases, a degree of disease apparently not greater has been productive of the phenomenon; whence we must conclude that a certain accidental configuration of the diseased parts, in addition to the amount of disease, is requisite for the development of a murmur."

From these different authorities, it will be seen not only that the aortic second sound may be modified by disease without the condition capable of producing a murmur being reached, but even that considerable disease may exist without a murmur being developed. This gives weight to the indications of aortic valvulitis other than a murmur. An advanced condition is reached when the ominous sound of a murmur is heard; it is doubly important, then, that we should be familiar with the other and earlier evidences of disease of the aortic valves. As to the absence of murmur in aortic regurgitation, the following extracts from a case published in the *British Medical Journal*, 30th March 1872, by W. T. Gairdner, of Glasgow, is very instructive:—"A regurgitant murmur was present, and disappeared—a very significant fact diagnostically. *January* 20.—Double aortic murmur. On January 30 it was found that the presumed murmur of aortic regurgitation had entirely disappeared, leaving only the apex prolongation of the first sound as formerly described. P. M., 10th of February. The aortic valves presented very well-marked and even extensive disease. There was a prominent rough mass of vegetation on the left fold of the sigmoid valves, while the posterior fold was perforated." It would seem, then, as Hope stated, some peculiar configuration is necessary, as well as disease, to cause a murmur. Further, this case shows that even extreme disease may not give out a regurgitant aortic murmur. The more important, then, is it to appraise carefully the other indications of aortic change!

I think we will all agree that when hypertrophy of the left ventricle is found along with accentuation of the aortic second sound, the suspicion of aortic valvulitis coming on at no distant date must be entertained. Probably the presence of constitutional syphilis renders the danger still more imminent. Whether the fact that early changes in the free edges of the aortic cusps modify the character of the aortic second sound can be demonstrated or not, is yet in dispute. The statements of the leading authorities given above render it very probable that an intermediate stage, characterized by muffling of the second sound, is in some cases developed betwixt the stage of accentuation and the unmistakable and significant production of a murmur.

I am inclined to think such an intermediate stage is furnished by the patient present for examination. He is a young man who came under my care eighteen months ago at the West London Hospital. He was a striker, and he had excited action of his heart and some hypertrophy. At that time his aortic second sound was clearly and loudly accentuated. I do not think any one will say that such is now the case. I insisted upon his changing his work, which he has done. It may be possible to raise the hypothesis that, as his heart is quieting down, the accentuation is declining. I am inclined to think otherwise, and once or twice recently have thought I could detect a fugitive murmur.

However, we have had him here, and he has been examined by several gentlemen. A watch will be kept upon him, and if a murmur should develop itself he will be brought here again for further examination. If a murmur should be manifested after a time, the intermediate stage will almost be proved.

Such, then, are the associations of aortic valvulitis in comparatively young persons. There are—(1) hypertrophy of the left ventricle; (2) accentuation of the aortic second sound, about which we all agree; and, later on, (3) a muffling of the aortic second sound, which is not generally admitted, though the great authorities quoted above countenance the hypothesis. Such, then, are the associations of the premurmuric stage of aortic valvulitis in young persons with a history of sustained effort.

In elderly persons we find aortic valvulitis of a chronic nature developed along with other vascular changes in the subjects of gout or chronic Bright's disease. Here there is contraction of the minute systemic arterioles, with a rise of blood-pressure in the arteries. Then develops hypertrophy of the left ventricle, and the high blood-pressure in the arteries is kept up. The pulse is hard and incompressible, and the tension on the aortic valves on the aortic rebound is great, and the sound produced is accentuated. These are the associations of aortic valvulitis in the aged or elderly. Again, we see that the tension in the aortic valves is the cause of trophic changes in them. When we meet with an elderly person presenting the evidence of high arterial tension, with a firm incompressible pulse, not obliterated by pressure of the finger during the diastole, and an hypertrophied left ventricle, and still more when the arteries are becoming atheromatous, we may reasonably suspect that changes in the aortic valves may ensue. If the tone of the valve-sound alter from accentuation to muffling, we may reasonably conclude that disease is present, and that a murmur will in time be heard. Accentuation may be present for years, and does not necessarily involve valvulitis or the production of a murmur. But if muffling come on, then we may be certain, or all but certain, that the case is entering upon a new phase, and that disease is actively progressing. If this stage of muffling can be demonstrated, it will be advantageous from a prognostic point of view, as well directing the treatment to be adopted, to arrest the disease at least, if it cannot be brought to a standstill.

I sent an old man lately to Dr Sansom of the London Hospital, in whom I thought muffling of the aortic second sound was present, along with some cardiac hypertrophy and atheromatous arteries. Amidst other disease the condition was somewhat obscure. Dr Sansom wrote me: "I think the second sound, though feebly heard, has a dull leathery character." Six weeks later he wrote me: "The aortic second sound is very dull, valves probably thick." My interpretation of the signs is, that amidst other vascular change the aortic valves are becoming

diseased; while Dr Sansom is inclined to think that the aortic valves were altered by an attack of rheumatic fever thirty years ago, and that the valves are in a static, non-progressive condition. Either hypothesis is possible; but in either case dulness of the aortic second sound is admitted as being "probably" connected with some thickening of the valves.

In April 1874 I saw a gouty lady, and my notes of her contain the following passages: — "Arteries atheromatous; hard and tortuous. Left ventricle hypertrophied (apex a rib down); aortic second sound not clear; probably the free edges of the valves are thickened." This month she was examined by an able provincial physician, who writes me: "Mrs ———'s heart is as you heard it, only with now some little irregularity; there is hypertrophy, and the second sound somewhat indistinct, *but absolutely no murmur*. She has had on more than one occasion most suspicious 'breast pang.'" Further observation of this case is then necessary. Indeed, extended observation of such cases by several observers will be requisite, before it can be held as proven that there is an intermediate stage of muffling betwixt the accentuation of the aortic second sound and the ominous and significant diastolic murmur, which tells that disease of the aortic valves is unmistakably present.

In a case I saw in private lately, the patient had been examined by his son, a medical man, three years ago, and then there was marked accentuation of the second sound at the aortic valves. Now there is a distinct to-and-fro murmur at the aortic area, showing that the valves have become so altered that there is now both obstruction and regurgitation. Whether there was any intermediate stage in the case is unknown. But that a distinct accentuation of the aortic second sound preceded the development of the murmur is beyond question.

To sum up, then, it may be regarded as proven that a condition of accentuation of the aortic second sound commonly precedes the murmur which indicates disease of the aortic valves. An attempt has been made to demonstrate that an intermediate stage, of which the main sign is muffling of the aortic second sound, occurs betwixt these two conditions, with what success your decision will say. I leave the matter in your hands.

The practical importance of being able to recognise commencing aortic valvulitis, without having always to wait for the unmistakable evidence of a murmur, is distinct enough. We know well, from the history of mitral disease arising in an attack of acute rheumatism, that a certain injury may be done to the mitral flaps which may remain static and stationary, non-progressive; where the patient may live for years, scarcely crippled, and severe effort alone reveals the impairment of the valves.

The evidence of such a static condition of the aortic valves is not so convincing from its magnitude. But it is very desirable

that we should always be on the alert to suspect commencing aortic valvulitis, when we find a clearly accentuated second sound along with hypertrophy of the left ventricle. If the patient be a young man, leading a very active life, like a university oarsman, then we can recommend a quiet life, and so, probably, in many cases bring the aortic mischief to a standstill. If he be a striker, or a bargee, we can advise a change of occupation, which will lessen the demand upon the aortic valves, and so, at least, retard the valvulitis. If the patient be a middle-aged or elderly person, and along with an accentuated aortic second sound we find an hypertrophied left ventricle, and a pulse full and incompressible, then we can suspect aortic valvulitis as a very probable consequence, and can do much to stave it off or retard its progress by bearing its causation in mind. Excess of nitrogenised waste is the cause of the high arterial tension which starts atheroma in the arteries, and a growth of connective tissue in the aortic valves, the consequences of which are so dire. One can do much for such a condition. The amount of meat in the dietary must be reduced, and potash, with or without colchicum, taken regularly. Cleanse the blood, and the subsequent condition—the "gouty heart," with its associated pathological changes—will be brought to a comparative standstill. If the looming mischief can be detected before such progress has been made as mutilates the valves and so produces a murmur, the brighter the prospects of our treatment. It is very important, then, to note the indications of commencing disease, and to make out, if clinically possible, the premurmuric stage of aortic valvulitis. Even if the intermediate stage of muffling cannot absolutely be demonstrated, still, if accentuation give way to muffling or a dull second sound, we have good and valid reasons to fear that such trophic changes are going on in the aortic valves as will before long furnish the dreaded and significant murmur.

Dr Broadbent, after a few remarks on the case presented to the Society by Dr Fothergill, proceeded to congratulate him on his interesting and valuable paper, with a great part of which he entirely agreed. He concurred with the author in the importance he attached to accentuation of the aortic second sound (which, strangely enough, Dr Balfour, writing in 1876, had said could not occur from any general systemic cause) as indicative of undue strain on the valves, which frequently gave rise to inflammation, and ultimately to insufficiency. But while recognising this accentuation of the aortic second sound as an antecedent of regurgitation, and admitting its significance, he could not accept the intermediate stage in which the sound became dull. His attention had for many years been fixed upon the effects of strain on the aortic valves. He had not had the opportunity of watching these in "strikers" or athletes, but he had had under observation many

patients in whom it was due to retained nitrogenized waste and to kidney disease; and he had seen cases proceed, on the one hand, to angina pectoris, and on the other to regurgitation, but had never noted any such change in the character of the second sound. On the contrary, he had met with cases, and had at that moment one under observation in St Mary's Hospital, in which there was co-existence of a diastolic murmur and of an accentuated aortic second sound audible at the right sterno-clavicular articulation and in the neck. Of course when the leakage is considerable the second sound disappears, as there is not the requisite tension for its production; but a very narrow chink may give rise to a murmur, while the valves are for all practical purposes competent and quite capable of producing a second sound. He believed that the expectation of a change from the sharp accentuation of the second sound to a sound more or less dull, rested on an imperfect theory of the production of the second sound. At one time it was commonly supposed to be produced by the click of the valves as they came together, but now the prevailing idea was that it was due to the tension of the valves. The late Dr Sibson, however, had shown conclusively that it was not the tension of the valves alone which gave rise to the second sound, but of the arterial walls and valves together; and that the character of the sound was determined more by the condition of the walls of the vessel than by the valves. Dr Broadbent had had few additions to his knowledge of the functional action of the heart more fruitful in its applications than this. It was impossible on any other grounds to explain the modification of the aortic second sound produced by dilatation of the ascending aorta; this was something more than accentuation. It was not easy to convey an idea of a sound by descriptive terms, but it had been called booming or voluminous, and the change was what might be expected from the tension of a wider cylinder with thinner walls. The valves were not carried into position by the refluent blood, but took up a position covering the orifice in virtue of the disposition of the elastic fibres in their structure, and were ready for the recoil of the column of blood, and so long as they were competent, they and the whole root of the aorta would be thereby rendered suddenly tense, and together yield the second sound, the character of which would be determined not simply or predominantly by the condition of the valves, but also by the condition of the arterial walls. There was, moreover, he considered, no reason to suppose that the valves were specially thick just before they became incompetent, and allowed of regurgitation; on the contrary, as the valvulitis is exceedingly chronic, and the insufficiency is due to consecutive contraction, they would certainly in most instances have been more thick and soft some time previously.

Dr Stephen Mackenzie said—I think many important points are

raised by Dr Fothergill's communication. It is clear that, in all except the acute inflammatory cases, valvular changes precede for a longer or less period the murmuric stage of heart disease; that the valvular defect not being suddenly produced, will probably give some evidence of its condition before a positive murmur is produced; and that it is of the greatest clinical importance to be able to foresee the probable issue of events, before the existence of a bruit makes it obvious, by recognising the premurmuric signs. The latter have been given, I believe, correctly, as far as they go, by Dr Fothergill, and on the foundation he has laid, others may raise a superstructure rendering it still more easy to detect the premurmuric stage of aortic valvulitis. I can confirm Dr Fothergill's observations as to the sequence of events in chronic aortic valvulitis due to strain. One observes the increased ventricular impulse and prolonged first sound, the accentuated second sound, and the hard pulse of increased arterial tension. In some of such cases, on being followed up, the other signs remaining the same, a sound is noticed, at first too indistinct to call a bruit, tailing off from the still accentuated second sound; later the second sound is wholly replaced by a bruit, of course diastolic. It seems to me that these signs indicate the sequence of events occurring in the aorta and its valves. First, the chronic inflammation (due to strain, syphilis, and other causes) leading to sclerosis and loss of resilience of the aorta, and inducing hypertrophy of the left ventricle; and next, as the organized inflammatory products in the valves contract, the valves are rendered *gradually* incompetent to retain the blood on the aortic recoil. In all of such cases, however, as indeed I may almost say in all cases of aortic regurgitation, the first sound is prolonged and altered, never having the pure shock sound of health. This is in accordance with post-mortem experience, for on examining valves diseased by rheumatic endocarditis or by atheroma, the valves have lost their pliancy, and offer a certain, though not necessarily great, resistance to the entrance of blood during systole. In this connexion, I may mention that I believe Dr Fothergill's statement as to the respective changes produced in the cuspid valves under the two conditions of rheumatic endocarditis and atheroma to be correct, and would add, that in the former the cusps have a tendency to curl towards the ventricles, in the latter towards the aorta. After what Dr Fothergill has said, and from what others have observed, I think it is clear that one is not justified in assuming absence of valvular disease from the absence of murmur. This is true as regards the aortic orifice; is it less true as regards the mitral? One sees, not very rarely, cases of acute rheumatism, where, during the acme of the attack, there is a murmurish first sound, or even a soft systolic murmur at the apex. As the attack progresses the murmur or altered first sound disappears, and gives place to a sound indistinguishable from that of health. A few

weeks or months later, when the patients return to report themselves, as they invariably should do, a murmur is present, and persists. In such cases, as Dr Fothergill points out, one must assume the valve swollen and infiltrated with inflammatory products, thus giving rise to the primary temporary murmur, and, as the products of inflammation become organized, they shrivel and distort the valve, after a period of some weeks or even months, and thus produce the permanent bruit. More careful examination of such cases may yet reveal to us some signs indicative of the "inter-murmuric stage" of mitral valvulitis. What is of great pathological importance is, that in those cases of chorea, sometimes seen, in which no murmur can be detected when the patient is first seen, and yet a mitral regurgitant murmur becomes manifest, and remains permanent, it is possible that there may be valvulitis—and if so, of course, some of its remote consequences—in a premurmuric stage. Such cases admit, I am aware, of other explanation, such as that of Dickinson, that it is the consequence of the choreic affection of the heart; or that it is also, like the chorea, a rheumatic manifestation of the attack. I think, however, the point is one of great importance, and hope Dr Fothergill will help us to discover the premurmuric stage of mitral valvulitis.

Dr Sansom.—I consider it a point of very high importance in the physical diagnosis of diseases of the heart, to take note of the characters of the second sound heard over the site of the aortic valves. We thus obtain not only indications as to the conditions of the valves themselves, but also criteria as to blood-pressure—points of the utmost importance as regards prognosis during the progress of any case. And when we hear a decidedly accentuated second sound which tells us of an arterial blood-pressure greater than the normal, we are able, I think, to form an opinion as to the pathological state of the valves according to the quality of the sound we hear. Of such sounds we may have a *slap*, a *boom*, or a *thud*, each having some intrinsic signification. I am entirely of opinion, therefore, that we are able to judge of pathological changes in the aortic valves which are not betrayed by murmurs. Obviously, however, this is a much wider proposition than that brought forward and sustained by Dr Fothergill. The proposition which we have to consider is this: The pathological changes in cases of disease attacking the structures at the aortic orifice are progressive; in the earlier stages of such disease the segments of the valve are altered, but yet are competent; it is contended that such stages of the disease can be recognised. It follows that in a given case it can be predicted that at a future stage the valve-segments will become incompetent, and regurgitation will ensue. The stage, then, called by Dr Fothergill, from its *clinical characters*, the "premurmuric stage of aortic valvulitis," may, from its *pathological characters*, be termed the "ante-regurgitant stage" of aortic disease.

In considering this proposition we must not fail to appreciate certain difficulties As I have said, I fully agree that even in the absence of murmurs we may be justified in arriving at the diagnosis of structural alterations of the valves. It is quite another thing, however, to say that the signs denote changes which must be progressive. Obviously the condition may be stationary, and our experience points to numbers of cases in which the signs do not vary throughout periods of years.

As an illustration of the difficulties which we have to encounter, I may refer to a case which Dr Fothergill has been kind enough to send me for observation. In this case a man of 69 evidenced palpitation, dyspnœa, with modified angina. The signs were aggravated after exertion, and at once gave rise to the supposition that they were due to cardiac disorder. There were evidences of hypertrophy and dilatation of the heart, but at my first examination I detected no murmur. Arterial atheroma was pronouncedly indicated by tortuous arteries, whose coats showed a want of uniformity to the touch. Moreover, there were pulsations in the arteries of the neck, more on the right side. The veins also were turgid and dilated, and, as I considered, exhibited a faint pulsation. Now, as regards the heart-sounds. Whilst the first sound at the apex was rather prolonged, the second sound at the base was scarcely audible. It could be recognised, however, and had nothing of a murmurish character—it was very dull. Let us here call attention to a point that I have found of great importance in diagnosis. When we hear a feeble second sound over the aortic valves we are by no means to conclude that there is a feeble reflux: the valves being thickened and leathery, may close with a sound that may be almost inaudible. In such case our tactile impressions aid the diagnosis, as in the instance of this patient, for the finger in the second right interspace perceived a distinct shock at the moment of the second sound. I concluded, therefore, as Dr Fothergill had concluded from his previous examination, that the aortic valves were altered and thickened; but yet I heard no evidences of their permitting any regurgitation.

But now another question presented itself. Was this an example of what might rightly be called "aortic valvulitis," or was it a chronic condition left by a former disease? I found on inquiry that the patient thirty years previously had suffered from rheumatic fever, and that he had suffered from joint pains more or less ever since We could not, therefore, exclude rheumatic endocarditis as a probable cause of the diseased conditions. I had the patient under observation in the London Hospital for a short time, and in the quietude of the ward there was evidence of what was not detected before—a decided systolic murmur at the apex. I felt quite certain that there was mitral regurgitation, with the failure of heart commonly observed in the late stages of the affection. I thought it the more probable hypothesis that

this disease was the legacy of rheumatism—that it was rheumatic endocarditis of slow evolution. Commencing in the mitral valve, rheumatic endocarditis tends to spread over the endocardium lining the left ventricle to the aortic valves, ultimately involving these also. Such might reasonably be supposed to have occurred in this case, the valves being thickened, but not incompetent. Atheroma, however, was undoubted, and from the sphygmographic tracings we thought there was some evidence of aneurismal sacculation. Atheromatous disease might, therefore, be a concurring cause with the rheumatic endocarditis. Of course, it is quite a legitimate hypothesis that all the conditions were the outcome of atheroma, though I myself should incline to the greater probability of the rheumatic causation.

This case, then, illustrates the difficulties of the subject under discussion. Suppose the question propounded in regard to it, "Is this a case of the premurmuric (ante-regurgitant) stage of aortic valvulitis?" I could only answer, that I must withhold my opinion until after many examinations. I should after some time be enabled to say, This is *not* a case of *typical* aortic valvulitis, because it is complicated by mitral disease. Again, I could not consider it a typical form of aortic valvulitis, because the history is so closely associated with rheumatism, and the probabilities of the occurrence of rheumatic endocarditis are so great. We have to remember that where the aortic valves are altered by *rheumatic* endocarditis it is almost invariably found that the mitral are affected also. Of the implication of both, I think we have evidence in this case. Typical aortic valvulitis is unassociated with rheumatism; its common causes are alcoholism, overstrain, atheroma, gout. The case under consideration, therefore, I cannot consider *typical*, though it is very probable that atheromatous aortitis may be a concurring cause.

From these criticisms I am by no means to be understood to imply, that the conditions so ably described by Dr Fothergill have no clinical status. On the other hand, I think a strong case has been brought forward as to their existence and their value, only I think exclusion will reduce them to comparative rarity. Given an example of a patient who shows a certain train of symptoms, whose history indicates a certain probability of the oncome of aortic valvulitis, and in whom auscultation demonstrates an altered second sound without murmur; then the considerations adduced by Dr Fothergill ought to lead us to a better scientific appreciation of the position than heretofore. A little longer, and the complete competence of the valves may become an only partial competence. Let us remember that such partial competence—a position only a little removed from that now described by Dr Fothergill—has been recognised as a discoverable lesion by Dr Hayden. A post-diastolic murmur at the base is regarded by Dr Hayden as evidence "either of a transitional state of the

valves, an early stage in the process of valvular disorganization, or of dilatation of the aorta without valvular disease. It is, therefore, of much value as an aid to precise diagnosis, and in determining the probable duration of life."[1] It will be of great value to us as practitioners if we can discover the diseased condition previously to the more complete disorganization when the fatal regurgitation commences; and still more important if, when discovering it, we can suggest any means of arresting the danger.

ARTICLE IV.—*Cases of Intra-Cranial Tumour.* By BYROM BRAMWELL, M.D., Physician and Pathologist to the Newcastle-on-Tyne Infirmary, Joint Lecturer on Clinical Medicine and Pathology in the University of Durham College of Medicine, Newcastle-on-Tyne.

(*Continued from p.* 605.)

CASE VIII.—*Female, æt.* 20. *Headache; Vomiting; Double Optic Neuritis, with Perfect Vision; Relief under Iodide of Potassium and Chloral; Convulsion; Death; Large Tumour pressing upon and causing Extensive Atrophy of the Ascending Frontal, Ascending Parietal, Inferior and Middle Frontal Convolutions, and of the Island of Reil, on the Right Side.*

M. D., æt. 20, single, shop girl, was admitted to the Newcastle-on-Tyne Infirmary on 5th January 1877, complaining of headache, vomiting, and giddiness.

Previous History.—When nine years of age she fell and hurt her head; the injury was a severe one, and was followed by vomiting. With this exception she has never been laid up until the present attack. It commenced three years ago with headache. Eight months ago the headache got worse, and she vomited occasionally. Two months ago she had to leave her situation. She says she has never had a fit, but has more than once tumbled off her chair when sitting, in consequence, she thinks, of giddiness. She has only menstruated once, and that was two years ago. She knows no cause for her illness. There is no suspicion of syphilis.

The family history is good.

Present Condition.—She is a well-developed and well-nourished girl, and, with the exception of a slightly coated tongue, is, as regards the circulatory, respiratory, alimentary, and urinary systems, perfectly healthy.

She seems intelligent, but her friends say that she is very much quieter than she used to be. The face at times has a somewhat dusky, congested hue. The eyes are unusually prominent, but have always been so. The headache is severe, and is worse at

[1] *Diseases of Heart and Aorta*, p. 238.