

say that I have been unable to obtain the pamphlet of Capellini, which is entitled *Dissertation sur les Effets des Odeurs*, and which was published in Paris (date unknown to me). While we cannot fail to recognize the important relation of olfaction to the imaginative faculty, and the frequency with which it serves as the connecting link between associated ideas, and while the above case illustrates the psychical element in the *excitation of the paroxysm*, it must not be considered that the affection itself is a disease of the imagination, a purely psychological phenomenon dependent solely upon a deranged mental impressibility. For both our present knowledge of the affection and the history of the case itself, militate against and destroy such a supposition. Indeed, we should distinguish carefully between a disease having a definite clinical history and subject to recognized pathological law, and a mere perversion of the perceptive faculty, although the latter may occasionally act as an exciting influence in the production of the paroxysms of the former. The chief lesson to be derived from the study of this particular case (*i. e.*, so far as the psychical element is concerned) is that it opens our eyes to the fact that the association of ideas sometimes plays a more important rôle in awakening the paroxysms of vasomotor coryza than the alleged vital property of the pollen granule.

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MITRAL STENOSIS.

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CONTRACTION of the mitral orifice is, on many grounds, the most interesting of the valvular affections of the heart. It is common and at the same time dangerous, standing, in point of danger, next to aortic incompetence; being, indeed, the more serious of the two in early life, so that it has a very practical interest. Its clinical history, again, presents peculiarities, some of which have long been recognized, while others have not yet received adequate notice. The special claim of this condition, however, upon our attention, arises from the fact that it presents greater difficulties in diagnosis than any other disease of the valves. It was the last of the valvular lesions to be associated with distinctive physical signs, and it is still not unfrequently entirely overlooked by physicians, while more commonly it is diagnosed as mitral incompetence, which is a far less serious affection. The physical signs are, in fact, extremely varied, and the attempt to elucidate their significance, and especially to attach diagnostic and prognostic meaning to

some of the combinations of modified sounds and murmurs, is the chief object and justification of this paper. Being a contribution to this particular end, historical and other references to the writings on the subject can only be incidental.

A remarkable fact is the relative frequency of its occurrence in women. This comes out whether the basis of the estimate is post mortem or clinical. Of 53 patients dying in St. Mary's Hospital, and examined after death, 38 were females and only 15 males, 72 and 28 per cent. respectively. Of 81 cases collected by Dr. Hayden, 54 were females and 27 males, 66.6 and 33.3 per cent. Dr. Dyce Duckworth in 80 cases found no fewer than 63 women—i. e., 78.75 per cent. It cannot be said that any satisfactory explanation of this great disproportion of women affected by mitral narrowing has been given. It is true that rheumatism is more common in girls than in boys, but were this the only reason, there ought to be a general predominance of valvular disease of the heart in women, and not of this particular condition. Scarlet fever and measles, which are accountable for a certain proportion of the attacks of endocarditis, leading to lesion of valves, are still further from affording the required explanation. Possibly, the greater liability of girls to anemia at the period of puberty may be one factor in the greater incidence of mitral stenosis in the female sex. Dr. Goodhart has pointed out how anemia may give rise to valvular disease, and Dr. Sanson, in his *Lettsomian Lectures* (1883), has shown that it is in the insidious and not in the acute forms of valvulitis that mitral stenosis is brought about. These two facts fit together very well into a conjectural explanation of the predominance of this affection in females, especially when it is borne in mind that anemia and chlorosis are attended with augmented arterial tension, which is in turn attended with increased backward stress on the mitral valves, a recognized cause of insidious damage.

A point on which there is less unanimity of opinion and, therefore, less conclusive evidence, is as to the special liability of children to mitral constriction. Dr. Hayden found reason to believe that such liability existed. Dr. Dyce Duckworth's statistics, on the contrary, do not support this view. The late Dr. Fagge, although he relates no case under ten, came to the conclusion that in children the mitral valve is especially prone to slow inflammation, leading to constriction, and Dr. Sanson speaks of seeing many cases under the age of seven. I have had two deaths at eleven, after protracted illness, and have had more cases of advanced mitral stenosis in children than of any other serious form of heart disease. It is, in my opinion, the most dangerous form of valvular lesion occurring in early life, and that in which there is least chance of compensatory changes sufficient to neutralize the effects of the obstruction to the circulation.

The subjects of mitral stenosis, up to a very advanced stage, have not the look of heart disease; they are neither pallid and anxious looking, as in aortic disease, nor livid and dusky, as in mitral incompetence, but often have a good bright color and cheerful expression, the lips perhaps being of too crimson a shade of red. Not unfrequently, up to the moment when, from some cause or other, serious symptoms have set in, and again after recovery from these, the patient is unconscious of embarrassment of the circulation, and is capable of ordinary work, even when this necessitates going up and down stairs. On the other hand, overexertion may be attended with a distressing sensation of constriction of the chest, so that the patient has to stand still for a moment, and feels as if he were about to die, while hæmoptysis is not uncommon; child-birth and attacks of bronchitis, or pneumonia, etc., bring on serious danger.

Mitral stenosis, moreover, is the form of valvular disease which, independently of actual inflammation or of ulceration, most frequently gives rise to arterial embolism. This is not, as a rule, the result of detachment of vegetations from the valves, but is due mainly to the formation of fibrinous coagula between the *musculi pectinati* of the auricle or in the auricular appendix; or, more rarely, between the *earnae columnæ* of the ventricle, which are shaken out of their beds by exertion or emotion and carried into the circulation. The stagnation which allows of such separation of fibrin may occasionally lead to the production of a large mass of solid coagulum in the atrium of the auricle, which either lies free in the form of a round ball or is attached to the wall of the cavity.

Another condition more frequently met with in mitral constriction than in any other form of valvular affection is great enlargement of the liver with true pulsation of this organ. This is not to be confounded with the jog communicated by the hypertrophied right ventricle, which is much more common. True hepatic pulsation is not a conspicuous phenomenon, but has to be sought for; it is not easily seen and it is scarcely to be felt at all. In order to bring it out, the hand must be pressed upon the enlarged liver at a part remote from the epigastrium and watched, when a gentle but considerable heave will be seen as the organ is distended by the reflux of blood into the hepatic veins. After repeated attacks of congestion the liver often refuses to yield to distention, so that in the later stages of the disease the hepatic enlargement may be less marked than at an earlier period, the result, no doubt, of that form of cirrhosis which is due to chronic congestion.

Related probably to the congestion of the liver is the occurrence of fluid in the peritoneal cavity before œdema is present in the legs, which is not uncommon; or the œdema will disappear speedily with rest in bed while ascites remains for a time; whereas cardiac dropsy, as is well

known, usually begins in the connective tissue, and ascites is late in its appearance, and is associated with extreme œdema.

The pulse of mitral stenosis is interesting. Different observers describe it as regular or irregular, according, no doubt, to the stage of the disease upon which the attention has been fixed. It is, according to my experience, almost invariably regular until the heart is obviously failing, unless the obstruction is complicated by regurgitation, or by valvular affection of the right side of the heart, or by intercurrent pulmonary affections. When irregularity comes on, it is usually at first inequality in the force of the beats, with perhaps slight loss of time in those which are weaker, and not any marked disturbance of the rhythm. Then some of the heart beats will fail to reach the pulse, no doubt from inadequate filling of the ventricle; the action of the heart may thus continue to be regular while the pulse is irregular. When, however, the heart is staggered by some serious pulmonary complication, the irregularity in its action, and, consequently, the irregularity of the pulse, may be indescribable, and it may be impossible to time either sounds or murmurs, or to determine their relation to the systole or diastole. But this is not constantly met with, even when bronchitis, or pneumonia, or pulmonary apoplexy supervenes. Many cases go on to a fatal termination with little or no derangement of the rhythm of the heart, and it is when there is regurgitation as well as obstruction that extreme irregularity is most likely to occur. The pulse, of course, follows the heart, sometimes, however, exaggerating its irregularity, mentioned as above; sometimes, on the other hand, disguising the extreme confusion revealed by the stethoscope. In some rare instances there is only one beat of the pulse for every two beats of the heart, the contraction of the left ventricle at every alternate systole being inadequate to raise the aortic valves. On listening to the heart under these circumstances, the aortic second sound is absent, the rhythm, as expressed by the sounds, being one-two—one, one-two—one. A weak pulmonary second sound may or may not be present—occasionally it is loud. Or there may be coupled heart-beats, one following the other, like an echo, accompanied by first and second sounds of quite different character, so that it appears as if the two ventricles were acting alternately, or one beat may be accompanied by a murmur at the apex, the other not. This curious modification of the rhythm of the heart, however, is not peculiar to mitral stenosis, and when it occurs in this disease it is often, though not invariably, distinctly attributable to digitalis.

But perhaps the most interesting point about the pulse is that the artery, which is rather small, as would be expected, is full between the beats, and presents the characters of moderately high tension—i. e., it can be rolled under the finger, and is not easily flattened. In my experience this modified high-tension pulse is almost constant, and it points

to resistance in the capillaries, but the causes of such resistance is not readily perceived. It may be due to reflex contraction of the arterioles, or the blood may be charged with impurities as a result of imperfect elimination, consequent upon its sluggish movement, which is, perhaps, the most probable explanation, or there may be backward pressure from the veins which makes itself felt through the capillary network.

Another fact which has been impressed upon me, in the course of my observation of the various forms of heart disease, is the late stage at which general dropsy supervenes in uncomplicated mitral stenosis. Not unfrequently the subjects of it are free from œdema at the moment of death, and very commonly it is absent when the symptoms arising from the condition of the heart are most urgent; it may set in suddenly after these have lasted for some time. Ascites, again, may occasionally be present independently of œdema, or it may persist after œdema has subsided, occurrences which are altogether exceptional, if not unknown, in other forms of heart disease. When dropsy comes on comparatively early, regurgitation will usually be found as well as obstruction, and the incompetence of the valve may not improbably play a greater part in the production of the symptoms than narrowing of the orifice.

I have met with an extreme degree of dropsy only when there has been tricuspid stenosis, as a complication of the mitral stenosis, and have come, in the diagnosis of this complication, which in most cases does not reveal itself by separate physical signs, to rely upon blueness and lividity of the countenance, and waterlogging of the tissues and cavities.

This conclusion with regard to dropsy, which is a simple expression of my experience, receives confirmation from an examination of 53 cases, abstracted for me by Dr. Phillips, from the post-mortem records of St. Mary's Hospital, and of the 67 cases related by the late Dr. Fagge in his interesting paper on mitral stenosis in Guy's Hospital reports. Of the 53 St. Mary's cases, anasarca was present in 11, and slight œdema in 1, but in 2 out of 11 there was tricuspid stenosis, and in 7 more mitral incompetence, or some other complication, which in most instances was of itself competent to give rise to dropsy, leaving only two or three to be the effect of mitral stenosis.

In 47 of the cases collected by Dr. Fagge a post-mortem examination was made, and in 7 of these a detailed account of the symptoms and physical signs is given. In 2 out of the 7 there was no dropsy from beginning to end; in 3 others there was fugitive and slight œdema, while the patient was up and at work; swelling of the abdomen preceding, in 1 of these, the pitting of the ankles; in 1 case the dropsy appears to have been marked, but was relieved; in another, it was apparently considerable for a time; but even here it subsided, although present at death.

In this, the only one which at all followed the ordinary course of

cardiac dropsy, it is to be observed that the heart weighed twenty-six ounces, which is an unusual weight in uncomplicated mitral stenosis, and the aortic valves were diseased. In 40 cases the post-mortem notes are brief and the clinical records imperfect, and dropsy is mentioned only in 8, while out of this number 2 were complicated by tricuspid stenosis; 1 by aortic disease, the change in the mitral orifice being comparatively slight; 1 by renal disease, leaving only 4 instances in which uncomplicated stenosis had given rise to dropsy. It is, of course, possible that in some of the 32 cases in which no mention is made of dropsy, this may have been due to omission, but in 11 the history implicitly excludes the condition.

In the remaining 20 cases no examination was made after death, but the clinical history is carefully given, and 15 had no dropsy, and 1 only occasional œdema. Another, however, had an enlarged abdomen. Of the remaining 4, 1 had severe ascites only, which disappeared after paracentesis; in another, ascites appears to have preceded general dropsy; and in the remaining 2, in which cardiac dropsy followed the usual course, a systolic murmur of a kind indicative of mitral regurgitation coexisted with the physical signs of stenosis.

The comparative infrequency of dropsy as an effect of obstruction at the mitral orifice, appears to me to suggest an explanation of the paradox which Dr. Walsh so forcibly puts forth as the outcome of his observation, with regard to the relation between heart disease and dropsy, to the effect, namely, that while universal experience demonstrates the causation of dropsy by heart disease, something beyond and in addition to any one or any group of cardiac lesions is required for the production of the dropsy. His conclusions cannot be summarized without losing much of their weight, due as much to his vast authority as to the concentrated force of the propositions on which they are expressed.

"(1) Mitral regurgitation or obstruction or aortic regurgitation or obstruction may severally exist and for a lengthened period, without systematic dropsy supervening.

"(2) Mitral regurgitation and aortic regurgitation may coexist for years, and yet no dropsy occur.

"(3) Both of these propositions (1 and 2) hold good, whether notable hypertrophy do or do not exist behind, or in connection with the obstruction.

"(4) Simple hypertrophy of the left ventricle may reach the highest point without systematic congestive effects of any kind arising.

"(5) Dilated hypertrophy, even of the left ventricle, may last for years without any such effect ensuing, provided the dilatation be not in notable excess.

"(6) The heart may be in a state of advanced fatty metamorphosis, the pulse feeble and infrequent, the encephalic and respiratory functions exhibit the singular perversions attending a high degree of that disease, the entire organization betray functional languor and inactivity, and yet even the prætibial integuments fail to pit in the least under pressure.

"(7) Or the heart may be soft and flaccid, and the pulse persistently frequent, feeble, and irregular in force and rhythm, and yet no systematic congestions occur.

"(8) The natural relationship of width of the arterial orifices, and also of the auriculo-ventricular orifices, may be materially perverted, without the least systematic dropsy arising until the closing days of life.

"(9) Tricuspid regurgitation, where the right ventricle is in a state of dilated hypertrophy, as shown during life by swollen and pulsating jugular veins which fill from below, and as shown after death by actual examination, does not necessarily produce dropsy."

These are, he says, incontrovertible; he adds:

"I cannot, then, see how the conclusion is to be avoided that something beyond, and in addition to, any one or any group of the cardiac conditions referred to is required in order, as a matter of necessity, to entail the occurrence of dropsy."

And again:

"The existence of some active cause beyond and independent of the heart is further shown by the facts that there is no direct relationship between the amount of heart disease and of dropsy; that dropsy comes on suddenly, sometimes from extraneous causes, the state of the heart remaining, as far as ascertainable, in precisely its previous condition; and that dropsy diminishes and increases, comes and goes, either spontaneously or through the influence of treatment, while the organic changes in the heart remain permanent and unmodified."

The propositions are indisputable, but the difficulties arising out of them appear to have their origin in the implied condition that the effusion of serum into the tissues and cavities of the body is the effect simply of venous stasis and of consequent slowing of the circulation through the capillaries. But it is not a question merely of obstruction in the veins, but of pressure in the capillaries, and if *vis a tergo* in the arteries is wanting, the condition under which the exudation takes place does not arise. Of course, whenever the resistance to the return of blood to the heart is such that the pressure in the arteries is unable to overcome it, actual arrest of the onward movement of blood in the capillaries occurs, and life ceases at once; but, short of this, and subject to the production of the degree of intravascular pressure needed to keep up the nutrient outflow into the intertextural spaces, the movement of blood in the capillaries may be very languid. Given a retarded circulation through the capillaries produced by venous obstruction, the occurrence of dropsy will depend on the pressure of blood in the arteries, and in mitral stenosis the conditions are such as to forbid any augmentation of it. Followed to its source, the pressure in the circulation depends, ultimately, on the left ventricle. Increased resistance in the capillaries is met by the increase of strength and vigor given by hypertrophy, and when disease affects its outlet, compensation is attained by hypertrophy with or without dilatation.

Under certain conditions the left is reinforced by the right ventricle, under all conditions, in fact, in which the pressure in the pulmonary circulation is increased and the right ventricle is hypertrophied, except when such augmented pressure is intercepted by a narrowed mitral

orifice. But in mitral stenosis the left ventricle is not hypertrophied, and, in consequence of the narrowing of the orifice, can get no help from the right, beyond such as is afforded by the more efficient filling of its cavity. The amount of blood entering it is, in advanced cases, probably less than normal, notwithstanding the increased pressure in the pulmonary circulation, and can scarcely at any time exceed the normal. It is not, moreover, propelled into the arteries with increased force. Although then the veins may be full, and there may be every appearance of backward pressure in the capillaries, the real source of pressure in the capillaries is wanting.

It appears to me that a clear recognition of the reason for the comparative absence of dropsy in mitral stenosis tends to remove the necessity for any active cause beyond and independently of the heart.

Before describing the physical signs, it will be useful to review briefly the morbid anatomy and physiology of mitral obstruction. The characteristic effect upon the heart, a dilatation of the left auricle, with more or less thickening (sometimes thinning) of its wall, and great hypertrophy with some dilatation of the right ventricle. The left ventricle is not correspondingly enlarged, and may retain its normal size while the right, by its growth, displaces it backward, so that no part of it appears on the anterior aspect of the heart, and its apex is no longer in contact with the chest wall.

One fact which immediately arrests the attention is the great difference in the weight of the heart and the dimensions of its cavities in different cases. This is due chiefly to the varying degree in which the left ventricle takes part in the hypertrophy undergone by the right.

As a rule, the heart is not very large; it is smaller than in mitral incompetence and *a fortiori* much smaller than in aortic disease. Weights of ten and twelve ounces are common, and fourteen or fifteen will represent about the average. On the one hand, I find weights of seven and seven and a half ounces in adults, and on the other of seventeen and twenty-two ounces. Such differences are not merely capricious and accidental; they have some significance if we were only able to trace it. It is true that similar diversities are common in all forms of heart disease, and are to some extent accounted for by varying nutritional energy, but this is not a complete explanation. Other and often more influential factors, especially as regards their effect in mitral stenosis on the degree of hypertrophy of the left ventricle, enter into their production, and an attempt to follow them out cannot be otherwise than instructive.

Hypertrophy, by means of which the injurious effects of valvular imperfections on the efficient pumping of the blood through the heart are more or less neutralized, is no longer regarded as a vague conservative effect of nature, but is seen to be the response of the muscular

structure of the heart to increased work thrown upon it. Now, when the mitral orifice is narrowed, it is the left auricle and right ventricle only which are called upon to exert increased force, since there is no obvious cause of increased resistance in the systemic circulation. The same may be said when the valvular lesion is mitral incompetence with regurgitation; but here another element of change comes in which makes a difference between obstruction and incompetence. The high pressure in the pulmonary veins and left auricle, which is a result of the damming back of the blood and of the increased force of the right ventricle, causes a forcible inrush into the left ventricle during diastole; and this, so long as the orifice remains of the natural size, must distend, and in the long run dilate its cavity, taking effect, as it does, during the unresisting period of the ventricular rhythm. But an increase in the capacity of the cavity multiplies by so much the force required to expel its contents, and this constitutes a demand for hypertrophy. We have, then, as a result of mitral incompetence, dilatation, and more or less hypertrophy of the left ventricle, but the hypertrophy here is required as compensation for the dilatation, and not to overcome any direct effect of the impairment of the valvular apparatus. In extreme stenosis of the mitral orifice, the pressure which thus affects the ventricle is intercepted; there is scarcely time for it to be adequately filled during diastole, still less for any distending effect to be produced. We see, then, how it is that the left ventricle does not increase *pari passu* with the right. Such, however, is not always the case. The left ventricle is often dilated and more or less thickened, and here our reasoning appears to be at fault. But the difficulty disappears on reflection. Not uncommonly the change in the valves which glues them together and narrows the orifice, interferes at the same time with their apposition, and permits of regurgitation. Such regurgitation may, indeed, be for a long time the predominant result; and, in point of fact, incompetence often precedes considerably the establishment of obstruction. We have here abundant cause for differences in the condition of the walls and cavities of the heart found after death, and, it must be added, for variations in the clinical history, and especially for diversity of physical signs.

But even without regurgitation, it seems probable that in the early stages of the process which ultimately results in great constriction of the mitral orifice, the pressure in the pulmonary circulation may become considerable before the communication between the left auricle and ventricle is so far blocked as to prevent the ventricular cavity from being filled, or even distended, during the period of diastole. There may thus have arisen at an early stage dilatation and hypertrophy, which are utterly inexplicable by the conditions found at death, and we must look back along the whole line of morbid changes in order to understand their final product.

PHYSICAL SIGNS.—As has been already said, these are varied, and sometimes perplexing; but it has appeared to me that they afford a means of estimating approximately the degree of constriction which the mitral orifice has undergone. The contraction does not take place all at once, but increases by slow degrees through many months or years, and it is to be expected that corresponding change in the physical signs will accompany this change of mechanical conditions. The physical signs are not the same in a given case from beginning to end, and by following the modifications of the sounds and murmurs which gradually supervene, I have been led to recognize three stages of the disease.

The heart is not usually greatly enlarged; the apex is displaced to the left and sometimes also downward, but it is found, as a rule, not far from the normal situation.

The hypertrophy of the right ventricle, and the dilatation of the left auricle give rise to an extension of dullness outwards along the fourth and third left spaces, and the dilatation of the right auricle causes dullness up to or beyond the right border of the sternum. The apex beat is not well defined, and in advanced cases is frequently accompanied by a sharp and often powerful shock felt on palpation, which, however, is not a thrust; the impulse of the right ventricle is powerful, lifting the lower left costal cartilages, and making itself seen and felt in the epigastrium.

The systole of the left auricle has been said to communicate an impulse perceptible in the third space an inch or an inch and a half from the edge of the sternum, but this is a phenomenon which I have never seen.

Changes in the dimensions of the heart, however, have not the same direct relation to the degree of valvular mischief in mitral stenosis as in other valvular diseases, and it is by means of auscultatory signs that the division into stages is effected.

The pathognomonic sign of mitral stenosis is usually given as a presystolic murmur heard over a limited area to the inner side of the apex beat. It is not a smooth blowing murmur, but has a rough and vibratory character, and is often accompanied by a thrill perceptible to the hand at the same spot. Corroborative evidence is afforded by accentuation of the pulmonary second sound, the result of backward pressure in the pulmonary circulation, and, not unfrequently, by want of synchronism in the closure of the pulmonary and aortic valves, giving rise to reduplication of the second sound.

These are, in effect, the signs in the first stage, but another important note must be added, viz., that the second sound is audible at and beyond the apex. With such a combination of signs the diagnosis is extremely easy; a murmur heard near the apex and followed by a first and second sound can only be presystolic. If further aid were wanted, it would be

afforded by the character of the murmur, which, as has just been said, differs remarkably from other murmurs; it is not blowing and smooth, but vibratory or, in some instances, rumbling. And again, the way in which it runs up to, and suddenly ends in the first sound, which tends to become short and loud, is highly characteristic.

In this stage, that is when with the presystolic murmur both first and second sounds are well heard to the left of the apex, I have never known serious symptoms to arise from the condition of the heart, and I have seen illnesses of different kinds, even serious attacks of bronchitis, passed through without the intervention of embarrassment of the circulation. It is very rarely that patients are admitted into hospital presenting simply the signs above enumerated, but they are frequently met with in outpatient practice, and in consulting-rooms.

The second stage is marked by the disappearance of the second sound at the apex and by the short, sharp character of the first sound, which also usually becomes very loud; the first sound, in effect, comes to resemble a second sound. Mistakes in diagnosis may now be easily made. In mitral stenosis, at this stage, and in mitral incompetence there is alike heard a murmur followed by a short, sharp sound; but in the former the murmur is presystolic in time, and the sound is the modified first sound, while in the latter the murmur is systolic and the sound is the second sound. Very slight attention would, in most cases, suffice to prevent any confusion between the two; but an apex murmur is liable to be set down as the familiar systolic murmur of regurgitation without further investigation, and thus mitral stenosis, the most serious of the diseases of the valves, at a period, too, when symptoms may be impending, is taken for incompetence, which is attended with less danger than any other of the valvular affections. To bear this source of error in mind is to avoid it; but cases are sometimes met with in which, from absence of cardiac impulse and from the similarity between the sounds, it is not easy to follow the rhythm of the heart and time the murmurs and sounds. Flexible stethoscopes are here at a disadvantage as compared with the rigid wooden instrument, which communicates to the ear and hand, not only sound, but a sense of shock which at once indicates the moment of the systole, and this when there is no impulse perceptible to the hand. It has been recommended to place the finger on the pulse and to ascertain which sound coincides with the systole, but no guide could be more fallacious. Time is lost between the heart and the wrist, and not always to the same amount; the heart-beat and the pulse are never exactly synchronous, often alternate at equal intervals, and may go beyond this in their want of correspondence. The carotid pulse is a safer guide, but it is not easy for all observers to coördinate tactile and auditory impressions. The most trustworthy method of determining the relation of sounds to the cardiac rhythm is to find a spot in the region

of the base where the first and second sounds are unmistakably recognized, and then from this point to follow the sounds, step by step, toward the apex, when it will be found which of them it is that disappears, or which maintains some distinguishing peculiarity.

It is not, perhaps, of much consequence that an explanation should be given of the modification of the first sound and of the disappearance of the second sound, which are believed to characterize the second stage of mitral obstruction; but an explanation, however imperfect, or even the discussion of a point, may serve to keep it before the mind. The short, sharp first sound to which the presystolic murmur runs up, often with increasing intensity, the importance and significance of which were emphasized by Dr. Fagge, is considered by Dr. George Balfour, who has also done much to bring out the importance of this modification, if I rightly understand him, to be a sort of climax to the murmur, while the murmur is present, and to represent the remains of it when it is no longer heard; but this scarcely seems to be adequate, or, indeed, at all tenable, since the first sound is systolic, and marks, therefore, the arrest of the current of blood from the auricle into the ventricle, which gives rise to the murmur. It might again be plausibly attributed to the state of the valve, interposed like a membranous diaphragm between the two cavities, but this again does not bear critical examination. No very satisfactory explanation, perhaps, can be given at present; there are reasons, however, for ascribing the modified sound rather to the ventricular wall than to the valve: this impression, indeed, is forcibly suggested by the shock communicated to the hand. In hypertrophy the increase in thickness of the parietes modifies the first sound in a reverse direction, rendering it dull and prolonged; in dilatation, on the other hand, it becomes short, sharp, and loud, and comes to resemble that of stenosis, often to a remarkable degree; in palpitation, again, the first sound is very commonly extremely short and sharp.

The suggestion arising out of these considerations is that the abrupt first sound of mitral stenosis may be the result of the imperfect distention of the left ventricle, to which this valvular condition gives rise; the muscular walls at the first moment of contraction meet with no resistance, and acting rapidly are suddenly brought up, and made tense when resistance is encountered, and so produce the sharp first sound. In palpitation, the brief diastolic interval, not giving the ventricle time to fill, has a similar effect.

The disappearance of the second sound at and outside the apex is probably explained as follows: The second sound heard here, in the normal state of the heart, is that of the aortic valves. Of this I have satisfied myself by prolonged observation, the second sound at the apex, and that at the right margin of the sternum, from the third costal cartilage upwards, almost invariably corresponding in intensity and char-

meter, the aortic second sound, indeed, being not unfrequently better heard at the apex than at the base, while that produced by the pulmonary valves, even when accentuated, is not well conducted to the apex of the heart, and is not heard beyond it, unless, indeed, it is audible over a large part of the chest. There are two reasons, then, why, in an advanced stage of mitral stenosis, the second sound should not be heard at the apex; first, that as the left ventricle does not enlarge, it is overlapped by the right, which monopolizes the apex and displaces the left ventricle from all contact with the chest wall, thus preventing it from conducting to the surface the aortic second sound; next, that the aortic second sound is itself enfeebled in consequence of the diminished output of blood from the ventricle; the aorta is not distended, to the normal degree, and the recoil to which the second sound is due is correspondingly weak.

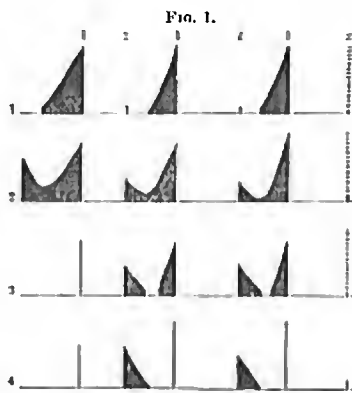
While the altered first sound in which the presystolic murmur ends abruptly, and the absence of the second sound at the apex are, together with the accentuated pulmonary second sound, and the want of accord between this and the aortic second sound, which gives rise to reduplication, the chief indications of serious narrowing of the mitral orifice, there are certain modifications of the murmur not unfrequently met with in advanced stages of this condition, which must be described.

Up to the present, the presystolic murmur has been spoken of as just preceding and running up to the first sound, when it corresponds with the auricular systole, and is the result of the propulsion of blood by the auricle into the ventricle. It is thus a short murmur only, but may be longer—*i. e.*, may begin sooner, when the auricle is hypertrophied and its systole is more powerful and protracted. This is not all, however, for the murmur may occupy the entire diastolic interval, and often follows immediately upon the reduplicated second sound, so that it seems to begin with this reduplication. It will then correspond, not simply with the auricular systole, but also with the active dilatation of the ventricle which takes place on its recoil from the systole. The diastole of the left ventricle is often regarded as a mere passive yielding to the distending force; first, of the blood entering it under pressure from the auricle and pulmonary veins, and then of increase of this pressure by the auricular systole, but when the naked heart is carefully watched, while in action, it will be seen that in its rebound from the extreme contraction of systole, the left ventricle dilates, and exercises a vigorous suction action which partially empties the auricles and causes the appendix rapidly to recede. When, again, the heart is removed by a rapid sweep of the knife, separating the ventricles from the auricles, and the pulsating ventricles are immersed in water, this active dilatation draws in sufficient fluid to yield a copious jet from the aorta and pulmonary artery. The first part, then, of the prolonged murmur often heard when the mitral orifice is greatly constricted is produced by the sucking

in of blood by the ventricle, the last part by the forcing in of blood by the auricle.

It should be added that the first part of the murmur has not always the marked vibratory character of the presystolic murmur proper, but still less has it the soft, blowing character of an aortic diastolic murmur, with which it corresponds in point of time; it may, perhaps, best be described as rumbling.

A further modification arises as follows: In a murmur occupying the entire diastolic interval, attention will usually detect a diminution of its intensity midway, and this may be carried so far that the murmur is cut in two, and the diastolic and presystolic portions are separated by a brief interval. Not only so, but from time to time there is an entire disappearance of the proper presystolic part, so that the obstruction at the mitral orifice is represented only by a true diastolic mitral murmur, which, however, is not likely to be mistaken for the diastolic murmur of aortic regurgitation, as it is almost invariably low pitched and rumbling, and often begins with a reduplicated second sound, instead of being smooth, blowing, and high pitched. Such a diastolic mitral murmur, moreover, is not heard, or certainly will not have its maximum intensity, at the same points as the diastolic aortic murmur, although I have heard it an inch or more beyond the right edge of the sternum in the right fifth space. It may, perhaps, be well to enumerate these varieties of murmur produced by the passage of the blood through a narrowed mitral orifice, representing them diagrammatically. They will be:



The double dotted lines represent the reduplicated second stage.

(1) The common presystolic or auricular systolic murmur, which may be short or long. (2) A murmur occupying the entire diastolic interval,

usually diminishing in intensity midway. (3) This murmur cut into two parts: diastolic and presystolic. (4) The diastolic part alone surviving. It cannot be said of these modifications of the obstructive murmur that they specially mark an advanced state of constriction, or that they have any very definite significance, but they are met with only in the second stage, and belong to the period of symptoms, being often associated with evidences of embarrassed circulation. In a young subject they would be of serious importance.

The third stage is marked by the disappearance of the presystolic murmur altogether, so that the sole remaining sign of the condition of the valve, present at or near the apex, is the loud, short, sharp first sound, with or without a systolic tricuspid murmur. This sharp and loud first sound is not unlike that heard in dilatation, with thinning of the left ventricle; but the absence of the second sound to the left of the apex constitutes a diagnostic difference, since this is distinct in dilatation. The absence of murmur and of any very considerable hypertrophy and dilatation may lead to the valvular disease's being entirely overlooked; instances have, indeed, come under my observation in which the heart has been pronounced normal on the eve of embolism or pulmonary complications.

No careful observer who has devoted much attention to the study of mitral stenosis has failed to notice the fact that the presystolic murmur is sometimes absent in cases in which an advanced stage of this condition is found after death. But it is not recognized and taught that this is extremely frequent. It is, in my experience, so common as to constitute a stage in the disease, and a large proportion of the cases of mitral obstruction which are brought into hospital to die would not be diagnosed if the presystolic murmur were looked upon as a pathognomonic *sine quâ non*, since it may never be present.

It must not be understood that the third stage, as here defined, is necessarily attended with serious symptoms. While this is the rule, patients may for a long time have only the modified first sound described and yet suffer little inconvenience. The principal justification for taking the disappearance of the presystolic murmur as a mark of a distinct stage in the clinical history of mitral stenosis is that, very commonly, when pulmonary complications set in, or other serious symptoms arise, the presystolic murmur is lost, and that it again becomes audible when these subside and the patient improves. It is a matter of repeated and familiar experience for cases to be admitted into hospital on account of serious symptoms with only the short sharp first sound spoken of and to leave with a loud presystolic murmur.

The probable cause of the disappearance of the murmur is the establishment of tricuspid regurgitation; this, at least, is conspicuous in a considerable proportion of cases. The giving way of the tricuspid valve,

and the occurrence of considerable reflux into the right auricle make it impossible for the right ventricle to sustain the same high pressure in the pulmonary circulation and in the left auricle, as was present previously. There is not, consequently, sufficient force in the current through the left auriculo-ventricular orifice, even when reinforced by the auricular systole, to generate sonorous vibrations. There being no valves, the contractions of the auricle will drive the blood backwards into the pulmonary veins as well as onwards into the ventricle, unless the pressure in the pulmonary circulation is adequate to resist the reflux.

The tricuspid regurgitation may or may not be attended with a murmur, but it will be manifested by a true jugular pulsation, and perhaps by hepatic pulsation. If a tricuspid murmur is present, it may become a source of confusion and error in diagnosis. The maximum intensity of the murmur is usually in the tricuspid area over the costal cartilages, just to the left of the ensiform, and over the lower end of the sternum; but not uncommonly the murmur is heard up to the apex, and perhaps even beyond it, so that it may easily be taken for a mitral murmur.

But another source of possible error arises out of the tricuspid reflux. It has already been repeatedly stated that accentuation of the pulmonary second sound is one of the important evidences of mitral stenosis, and this, of course, is due to the high pressure in the pulmonary circulation. When, therefore, the tricuspid valve gives way, and the pressure in the pulmonary artery is diminished, the second sound will cease to present the intensifications previously observed.

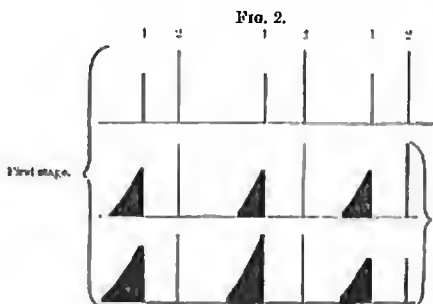
There has been so much of digression and discussion in the preceding account of the three stages of mitral stenosis, that it will be well to recapitulate the signs by which they are believed to be indicated, and these signs may at the same time be made more clear by diagrammatic representations of the sounds and murmurs.

The normal heart sounds may be represented as follows: the short, thick mark standing for the heavy first sound; the taller thin mark for the sharp second sound.

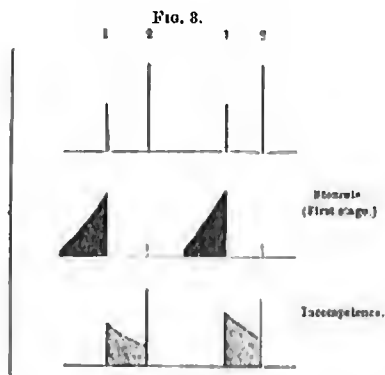
The presystolic murmur attending the first stage of narrowing, then, will be indicated by shading running up to the first sound, which may be made coarse, so as to typify its vibratory character. But the first sound will itself be undergoing a modification tending to render it short and sharp and like the second. The mark representing it, therefore, will somewhat resemble that representing the second, as here shown. Up to this point the recognition of the presystolic position of the murmur in the cardiac rhythm is perfectly easy; followed, as it is, by two sounds, it cannot be misinterpreted.

The second stage is marked by the more or less complete disappear-

ance of the second sound at the apex, while at the same time the first has become more than over short and sharp, so as quite to resemble the normal or accentuated second. That is, we hear at the apex a murmur, followed by a sharp sound; the murmur being presystolic, and the sound



the highly modified first. But in mitral incompetence, also, we have a murmur followed by a sharp sound, only the murmur here is systolic and the sound the second. The same words, it will be seen, may be used to describe both, and the systolic murmur and second sound of mitral regurgitation being the more common and familiar of the two, it is easy



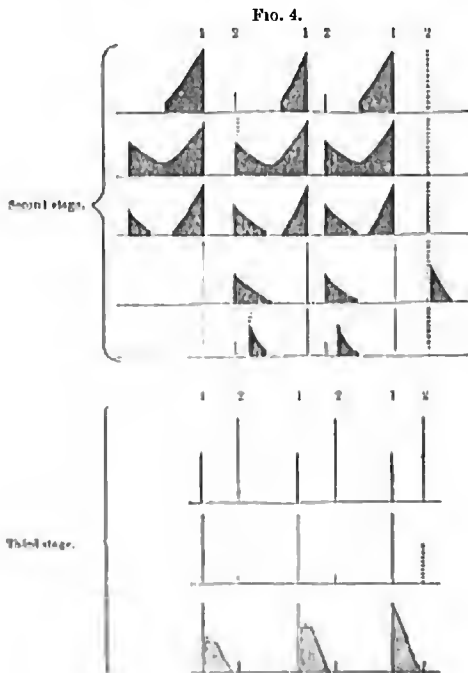
to take the presystolic murmur and modified first sound of stenosis for them. Such a mistake is, in fact, extremely common; but it will not

be made if the least attention is given to the character of the murmur, which, in regurgitation, is blowing or musical, and begins with an accent; while in stenosis it is vibratory, and ends in an accent.

These differences are illustrated in the accompanying diagrams, in which fine shading represents the smooth blowing systolic murmur (Fig. 3).

The variations in length and character of the presystolic murmur have already been represented, but they may be reproduced here (Fig. 4).

Finally, the presystolic murmur is no longer heard, and the most advanced narrowing of the mitral orifice is indicated only by the loud,



To illustrate the kind of contrast between the vibratory presystolic and the blowing systolic murmur.

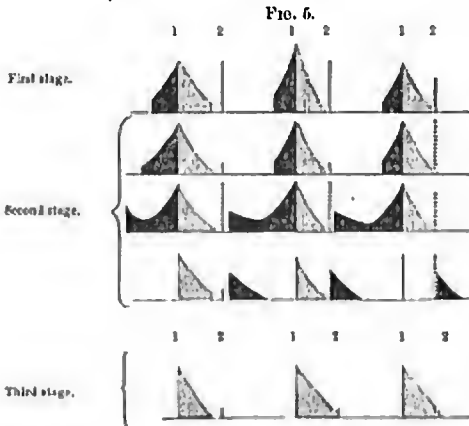
short, sharp first sound, which may, however, be accompanied by a tricuspid systolic murmur shown by the horizontal shading.

In the foregoing account of the sounds and murmurs attending constriction of the mitral orifice, incompetence of the valve, not na-

commonly associated with it, has been left out of sight for the sake of simplicity in description. It is possible, and, indeed, probable, that in some instances regurgitation takes place without giving rise to audible murmur, and when the heart is examined after death, it sometimes seems as if the irregular or thickened margins of the narrowed orifice could not possibly have come into the close apposition required to prevent leakage, while no systolic murmur has been noted during life.

In such cases the diagnosis of regurgitation as a complication of constriction can scarcely be made. There may, in fact, be no regurgitation even when the valves are not competent. As has been already stated, the course which the blood takes on the contraction of the ventricle will be determined by the direction of least resistance, and in mitral stenosis the fluid pressure in the aorta is a minimum, while the pressure in the auricle and pulmonary veins is a maximum; there may consequently—we may even say that if the circulation is to be sustained, and life is to go on, there must, in some cases, be such resistance in the auricle that no reflux of any moment into it can take place, however incompetent the valves may be. We need not, perhaps, therefore concern ourselves either with the diagnosis or prognosis of mitral incompetence complicating stenosis, which is not revealed by a murmur.

A murmur is, however, often present, and we may thus have a systolic apex murmur superadded to any of the signs enumerated—i. e., in the



first stage, at and near the apex, will be heard a double murmur, presystolic and systolic, the former vibratory or rumbling, and leading up to a

short first sound, the latter smooth and blowing in character, beginning with the first sound and followed by the second; in the next stage the double murmur presystolic and systolic, divided by the sharp first sound, is still present, but the second sound is no longer heard at and to the left of the apex. In the third stage there is only a smooth systolic murmur, headed by a sharp first sound; the presystolic murmur, too, is more liable to be lost where there is incompetence as well as stenosis. Almost everything specially characteristic of obstruction therefore disappears, and the murmur only of regurgitation remains. Graphic representations of the sounds and murmurs of the different stages when a systolic murmur is present may be added. No interpretation of them will be needed beyond a glance at the corresponding diagrams on the preceding pages.

The account here given of the murmurs resulting from combined mitral obstruction and regurgitation will certainly seem too simple to those who are familiar with the difficulty of analyzing and recognizing them in practice, and of determining the place of each murmur and sound in the cardiac rhythm. This very difficulty may, if the observer is experienced, constitute a diagnosis, for in no other heart affection does such an extraordinary confusion of sounds and murmurs arise. Sometimes listening at the apex and at the base it seems as if there were two separate hearts beating at a quite different rate.

So long as the presystolic murmur is distinct, no serious uncertainty need arise as to the diagnosis. The significance of the absence of the second sound at the apex is, perhaps, obscured, while regurgitation as well as obstruction will give rise to accentuation of the pulmonary second sound. Still the presence of a presystolic murmur reveals the existence of stenosis and the additional gravity belonging to this condition will be recognized. But when, as frequently happens, there is no presystolic murmur, the case is greatly altered; we have, so far as murmurs are concerned, only a systolic mitral murmur as our indication for both stenosis and incompetence. The recognition of stenosis under these circumstances is not easy, but it is most important that it should be effected, since the pulse may be regular and the heart will not be greatly enlarged, conditions which would make the prognosis of incompetence alone favorable, whereas stenosis and incompetence together constitute a grave combination. The absence of the second sound outside the apex will usually be of great value in identifying the stenosis, but, if the incompetence have preceded it, dilatation of the left ventricle resulting therefrom may conduct the aortic second sound to the chest-wall. Here the modification of the first sound again comes to our aid. Regurgitation through the mitral orifice, it will be remembered, tends to destroy the first sound; stenosis, on the other hand, tends to shorten and exaggerate it, and prevents it from being lost; when, therefore, a systolic

apex murmur begins with a first sound, the question of stenosis, associated with the incompetence, should at once present itself to the mind; usually the sound will have the short, sharp character so often dwelt upon, or some marked peculiarity, and may be accompanied by the peculiar shock felt at the apex; but, even when this is not the case, we may arrive at a diagnosis of the existence of constriction, though it must often remain uncertain to what extent the narrowing of the orifice has gone, or which is the predominant effect, obstruction or regurgitation. It is when both effects are present that we have the extraordinary fluctuations in the physical signs; at one time a presystolic at another a systolic murmur only, and at another both, while the second sound will be reduplicated one day and single the next. It may be conjectured, with some plausibility, that when the pressure in the left auricle is well sustained, its systole is capable of generating a presystolic murmur, and the resistance to regurgitation is efficient; when, on the other hand, it falls below a certain point there is reflex and systolic apex murmur, while the contraction of the auricle does not drive the blood through the opening with sufficient force to produce sonorous vibrations. In intermediate states of pressure both murmurs may be heard.

PROGNOSIS.—Mitral stenosis stands next to aortic regurgitation among valvular affections in the order of gravity. The average age at death, however, as deduced from 53 cases abstracted from the post-mortem records of St. Mary's Hospital, is higher than I expected. This was found to be 33 for males, and 37 or 38 for females; one woman dying at the age of 68 had slight, another dying at 61 had extreme narrowing of the mitral orifice.

A suggestive inquiry is, why mitral stenosis is so serious a disease, and why, especially, it is attended with greater danger than mitral incompetence. One reason will be that the effect of obstruction at this point is not so easily neutralized by compensatory changes. Just as in the case of regurgitation, the compensating influence is the increased pressure in the pulmonary circulation and the hypertrophy of the right ventricle. This antagonizes incompetence of the mitral valve in two ways, by resisting the reflux into the auricle during the ventricular systole, and by mere rapid and complete filling of the ventricle during diastole. Now in stenosis the only way in which the high pressure in the auricle and pulmonary veins can neutralize the effects of a narrowed orifice is by increasing the rapidity of the current through it, while the ventricle is relaxed. But the diastole only lasts a certain period, and when the constriction has reached a point at which the orifice will only admit, say the tip of one finger instead of three entire fingers, the ventricle cannot possibly be properly filled in the time available, even under pressure; the compensation, therefore, is inadequate. In aortic stenosis the left ventricle can take its own time in discharging its contents

through the narrowed orifice, the systole is prolonged; there is no corresponding prolongation of the diastole in mitral stenosis. While, however, it appears to be obvious that the provision for compensation falls short of that which comes into operation in other forms of valvular disease, on the other hand, since the narrowing is often found to have attained an extreme degree—such a degree as would in the absence of repeated experience have been thought quite incompatible with adequate circulation of the blood, and since the patients have lived through all the minor stages of the process, taking many months for their evolution, we cannot look upon the nature of the difficulty—*i. e.*, obstruction, and the difficulty of overcoming it by compensatory processes, as constituting the entire explanation of the greater relative danger. It has seemed to me that when once adhesion between the flaps of the valves has set in, there is a tendency for the adhesive process to continue, so as to encroach, progressively, more and more upon the channel. Changes, therefore, initiated by acute rheumatic endocarditis, would in this case be progressive; not, however, simply by continued shrinking of fibrous tissue or organized exudation, but because chronic inflammation is kept up by the friction and strain resulting from the narrowing of the orifice, and gradually glues the margins of the valves together. The process may be compared to the readhesion which it is so difficult to prevent after division of the web between the fingers.

The important point in prognosis, however, is the comparative prospect of life in individual cases, and it is in the estimation of this that the recognition of the different stages is of service.

We have, let us say, in a patient not complaining greatly of cardiac or respiratory distress, mitral stenosis indicated by a presystolic murmur and thrill, with a more or less sharp first sound in the usual situation; there is also an audible second sound at and to the left of the apex. This second sound shows that the disease is in the first stage and that the narrowing is not considerable. It is assumed that the pulse is regular. We should, of course, define the position and note the character of the apex beat, and endeavor to make out the size and form of the heart, directing attention specially to the existence of dulness to the right of the lower sternum and along the third and fourth left intercostal spaces, so as to bring into evidence any distention of the auricles. We may not be able to draw any trustworthy conclusions from the amount of hypertrophy and dilatation, but information on the point is indispensable. The aortic and pulmonary second sounds would be scrutinized and compared; accentuation of the latter will be present, and the degree of loudness and predominance of the pulmonary second sound will serve to indicate the degree of pressure in the pulmonary circulation. Variations in the intensification of this sound, however, move within narrow limits, and they are influenced by so many conditions,

that inferences based exclusively or even predominantly upon them would lead to error. Reduplication of the second sound, showing loss of synchronism between the aortic and pulmonary valves in their closure, is evidence of actual derangement of the cardiac mechanism; but, as it can be produced in a state of health merely by holding the breath, it need not necessarily have any great significance. An accentuated pulmonary and a reduplicated second sound then, while corroborating the diagnosis of stenosis of the mitral orifice by exhibiting effects resulting from it, are not necessarily of immediate serious prognostic import. Nor will the existence of a systolic apex murmur, indicating mitral regurgitation, add greatly to the gravity of the condition, in the absence of symptoms. So long as the second sound is not extinguished at and beyond the apex, the question as to danger relates rather to the future than to the present, and with regard to this, we have to fall back upon our knowledge of the history and tendencies of the disease. The degree of constriction actually reached is probably not dangerous, but it tends to increase, and if there has recently been an attack of acute rheumatism, still more if the patient is liable to frequent recurrence of subacute or slight rheumatism; if, again, the patient is marked by anæmic tendencies, there is every reason to apprehend continuous aggravation of the valvular affection. The younger the patient, the greater the fear of this; partly, perhaps, because of the greater liability to rheumatism. Later, there is the possibility that the mischief may be of old standing and stationary.

It must be understood that while a good aortic second sound at the apex, and along the right edge of the sternum, from the second space upwards, has not in my experience been met with, when serious results of mitral stenosis have been present, or impending, a contradiction of this particular indication by symptoms must always be accepted. No one indication is infallible.

When the second sound is lost at the apex and feeble over the aorta, there may still be a complete immunity from symptoms, under the ordinary conditions and circumstances of life; but there is no capacity for the adjustment of the circulation to deviations from these. We are not, therefore, to be thrown off our guard by the good looks of the patient, and by absence of complaints. Loss of the aortic second sound, associated with a short, sharp, abrupt first sound at the apex, and a reduplicated second sound, the pulmonary element in which is markedly accentuated, warrants a serious prognosis, and all the more when the presystolic murmur is modified in the way described, or is absent. Inquiry will usually elicit evidence of embarrassment of the heart on exertion, and this may assume a serious degree at any moment. An occasion for some extra exertion arises, which may be only slight; the muscular contractions drive the blood in the veins toward the heart,

which is the first step in the acceleration of its movements rendered necessary by exertion; the blood, however, cannot be passed more rapidly through the narrowed mitral orifice, and cannot, therefore, be forced through the lungs; the right cavities of the heart become over-distended, and the patient is at once brought to a state of extreme distress and great danger. In some cases relief is obtained by hæmoptysis, in others this does not occur, or it fails to relieve, and we have dyspnoea, congestion of the liver, and other associated symptoms.

A similar result may be produced by a comparatively slight attack of bronchitis or other pulmonary affection, by any febrile condition, or by anxiety or excitement. The right side of the heart is habitually over-taxed, and any acceleration of the circulation brings about distention of its cavities, with insufficiency of the tricuspid valve, and systematic venous obstruction. On the occurrence of such consequences the presystolic murmur will usually disappear, returning if recovery takes place.

Before symptoms have arisen, the considerations already enumerated as bearing on the question of the probable increase of the obstruction will apply with greater force, since the constriction is assumed to have proceeded further. Recent rheumatism, acute or slight, and any trace of gradually increasing tendency to shortness of breath will be reasons, the one for apprehension, the other for certainty, of progressive aggravation of the state of the valve, while childhood, so far from being favorable, is often the reverse. When this period of life is passed the condition of the heart and of the patient may remain much the same for years.

Anæmia, or obvious debility, will add to the gravity of the condition. Real strength, as tested by power of endurance, and capacity for exertion, will have a contrary significance. In some cases the end is brought about by the constriction reaching a point incompatible with life, in others it is due to want of power on the part of the heart and system to cope with the difficulty.

When symptoms have set in, and we are called upon to form an opinion whether a patient, who is suffering from dyspnoea, pulmonary congestion or apoplexy, venous obstruction, jugular pulsation, enlarged liver, dropsy, and other effects of overdistention of the right side of the heart, will recover from the attack, the first element in the mental calculation will be the severity of the symptoms. But recoveries are witnessed from conditions apparently so desperate that scarcely any combination of unfavorable symptoms can be pronounced absolutely hopeless, if it is the first time the patient has suffered from a similar attack. The number of times, then, that complications have arisen in the case, and the readiness with which they have been provoked, become considerations of the first importance. If the patient has had repeated attacks, and if a very slight cause has sufficed to set up serious symptoms, the danger is very great. It has seemed to me an unfavorable sign when, under such circumstances,

the liver does not enlarge; this is evidence of cirrhosis from previous congestion, and a reservoir for venous blood is lost. Advanced dropsy is always a very serious matter in mitral stenosis.

A complication of mitral stenosis sometimes met with is tricuspid stenosis. Tricuspid regurgitation is extremely common; it arises out of the mechanical and hydraulic conditions set up by the narrowing of the mitral orifice, the damming back of the blood in the lungs, the consequent high pressure in the pulmonary circulation and resulting stress on the right ventricle; it does not create a new difficulty, but serves as an indication and measure of the old difficulty. With regard to stenosis of the tricuspid orifice the case is different; it is not the direct outcome of antecedent mitral obstruction, and when present it adds a new source of obstruction to the circulation. What it is that gives rise to it can only be a matter of conjecture. The reason why the valves of the left side of the heart are so much more frequently the seat of disease than those of the right is apparently that the strain upon them is so much greater in consequence of the higher pressure in the systematic circulation. In mitral stenosis, the strain upon the pulmonary and tricuspid valves becomes probably quite equal to the normal strain upon the aortic and mitral valves, and this perhaps may account for the occurrence of tricuspid valvulitis and stenosis, especially when there are recurrent attacks of rheumatism.

However this may be, it is a fact of clinical observation that narrowing of the tricuspid orifice is more commonly met with in association with narrowing of the mitral orifice than with any other valvular affection of the left side of the heart, and when it supervenes it constitutes a most formidable complication.

It is consequently very important that it should be recognized early. This I have not found to be an easy task. A perceptible tricuspid murmur is described, and interesting cases in which it has guided to a correct diagnosis are related. I have myself heard what I believed to be a pre-systolic tricuspid murmur; but, on the other hand, I have frequently had cases under my care in which tricuspid narrowing has been diagnosed during life, and demonstrated after death, without any such murmur having been recognized although repeatedly and carefully sought for.

The grounds, then, on which the existence of obstruction at the tricuspid orifice may be inferred are a degree of blueness of the face and lips unusual in uncomplicated mitral disease, and liability to coldness and dusky lividity of the extremities. There will be great distention of the jugular veins, but less pulsation in them. Dropsy also becomes a more marked feature, and indeed in my experience extreme general dropsy in which all the tissues of every part are laden with fluid is not met with as an effect of obstruction at the mitral orifice unless there is also tricuspid narrowing.

TREATMENT.—The treatment of mitral stenosis is conducted on the same general principles as obtain in other forms of heart disease.

Considering first the important period before the disease is so far advanced as to give rise to serious effects, and symptoms are absent or induced only by overexertion, the damaged organ must be protected from any undue strain at the same time that nutrition of the tissues generally is kept up. The great difficulty is to reconcile these two conditions, which are more or less conflicting; but this only affords an opportunity for the exercise of the common-sense and clinical sagacity which are indispensable in the physician. It would be as mischievous to the patient as it would be insulting to the medical man to lay down strict rules. All that is necessary is to offer such suggestions as arise out of the special tendencies of the particular form of disease, which must be adapted to the enormous diversity of individual cases, constitutions, and habits. We must, however, look before us, and not wait for the recognition of such tendencies till symptoms compel our attention to them.

The patient's avocation and mode of life generally should be interfered with as little as possible, unless his habits are such as are distinctly injurious. Restrictions which go beyond the mark may of themselves impair the health, or they may render the sufferer's life unnecessarily anxious and miserable, or they may provoke reckless reaction.

Common-sense precautions must be taken against rheumatism and catarrh, or other effects of cold, but there should be no cultivation of a susceptibility to changes of temperature by excessive care. Flannel or other woollen material should be worn next the skin, sitting or standing with wet feet or in damp clothes must be avoided, as must draughts and hot, close rooms. It will be desirable to send a patient out of a climate or district conducive to rheumatism, and to forbid an occupation attended with much exposure.

In the matter of exercise there should be no violent exertion, and fatigue should not be carried to the point of exhaustion. Any effort which causes pain in the chest, or marked dyspnoea, must be looked upon as harmful, and must be avoided. Short of the above, the more the patient can be out of doors, and the more exercise he can take the better, and it will often be found that by beginning gently he will easily attain a rate of walking which, if attempted in the first instance, would have brought him to a standstill; or he may mount an incline without distress which at his ordinary pace would have been impossible. He may even be permitted to bathe and swim if experience shows that he bears it well. It is not prudent to send patients suffering from this or any other form of heart disease to mountain resorts. The effects of the diminished atmospheric pressure cannot be foreseen in individual cases; it is apt to set up palpitation, and should unfavorable symptoms supervene, removal—which is imperative—may be difficult and dangerous.

We would gladly add, were it of any use to do so, that anxiety and depressing emotions ought to be excluded from the patient's life.

The food and drink may be liberal and varied, but they should be distributed as equably as possible among three meals, not one of the three being specially large. If the nourishment for the day is taken at one huge repast the vessels are at one time filled, perhaps to undue distention, by the absorbed products of digestion, and then gradually unduly depleted. Such extremes throw unequal work upon the heart, to which disease renders it less able to adapt itself. There is, again, the liability of the distended stomach to carry up the diaphragm and so directly embarrass the heart by pressure. While the inclination, digestive powers, and idiosyncrasy of the patient should be duly regarded, it is important that fruit and farinaceous and other vegetable articles and milk should enter into his diet so that it may not be too nitrogenous. Further, due regard must be had to the fact that excess of any kind beyond the actual requirements of the body tends to the accumulation in the blood of nitrogenized waste which may act as an irritant, and will certainly give rise to resistance in the arterio-capillary circulation, one of the influences most to be dreaded in the long run.

Constipation must never be permitted. Both the mechanical and the chemical effect of fecal accumulation are injurious.

Medicines, except for the correction of functional derangements, and the prevention or removal of anaemia or debility, are not required, and being unnecessary will, if given habitually, be injurious.

When symptoms set in, excluding embolism, which will not be here dealt with, they will be either pulmonary complications due to the effects of the obstruction in the pulmonary circulation, or they will be further effects of such obstruction upon the right side of the heart. The treatment will not be really different in the two cases—nor will it usually be possible to distinguish between them, since the additional stress thrown upon the right ventricle by the occurrence of pulmonary apoplexy or congestion will generally break down the valvular competency of the tricuspid.

The danger to life consists in the inability of the right ventricle to force the blood through the lungs. Both ventricle and auricle become extremely distended, so much so as to be almost paralyzed by overstretching of the muscular fibres in their walls. When the tissues of the heart have undergone degeneration, or when the right ventricle is worn out by protracted overstrain, death may be sudden, either as the result of an effort or motion, or without obvious exciting cause. The fatal termination is, indeed, very frequently abrupt and unexpected, but it is not sudden death while in apparent health, but in the course of illness. The symptoms of overdistention of the right side of the heart are sufficiently conspicuous and familiar: urgent dyspnoea, with more or

less duskiuess or lividity of the face, which, however, even at this period may not be extreme; cough, which may or may not be attended with hæmoptysis, and a small, weak, and irregular pulse, many beats of the heart not reaching the wrist, and in extreme cases scarcely any. The contrast between the weak pulse and the powerful impulse of the heart is very striking, but the impulse felt is that of the right ventricle only, and there is no proper apex beat. The jugulars are distended and pulsating, and an indication, not always looked for, but of the greatest importance and significance, is enlargement of the liver, which may be felt to extend below the umbilicus and across the epigastrium to the left hypochondrium; it is nearly always jogged by the violent contractions of the right ventricle, and may pulsate from reflux into the hepatic veins. Associated with this there may or may not be a slight amount of effusion into the peritoneal cavity; anasarca need not be present, though sometimes it supervenes suddenly while the struggle is at its height, or even when improvement has begun.

Under such circumstances the first and most important thing to be done is to relieve the right ventricle. There are three ways of effecting this: by venesection, cupping or leeching, and purging. Bleeding from the arm or jugular is the most prompt and effectual, and it is certain to come again into more general use as the profession becomes familiar with the remarkable results which may be witnessed in cases apparently hopeless; pulselessness, cold extremities, and the cold sweat of impending dissolution are not contraindications as long as there is power in the laboring right ventricle. While blood is abstracted on the one hand, brandy and other stimulants may be administered on the other; or in desperate cases ether or brandy, or both, may be injected under the skin. It is in private practice that the best opportunities for successful bleeding are met with. In the case of hospital patients there is usually a smaller reserve of strength, and the rest, warmth, care, and nourishment they receive, to which they are strangers outside, make such a difference in their favor that milder measures are sufficient. Leeches may therefore be employed, or cupping, with or without the abstraction of blood. A very suitable situation for the application of leeches or the cupping-glasses is over the enlarged liver. It is not supposed that blood is abstracted from the organ, but the pain usually present in the region of the liver and the sense of fulness and oppression are relieved. After leeches the bleeding may be encouraged by poultices. I have seen recovery so far as to allow of the patient's leaving the hospital after the application of eight leeches in a girl of thirteen, when not only was she cold, pulseless, and apparently moribund, but when stasis in the capillaries of the surface had actually begun, giving rise to large livid patches on the abdomen and elsewhere.

As an accessory to the abstraction of blood in the attainment of the

same object, viz., the diminution of the afflux of blood to the overdistended right ventricle, or as the principal means of effecting it, purgatives are to be employed. A mild purgative will be of no use; the purgation must be decided. Nor is it, in my opinion, a simple matter or purgation to be induced by any method which may happen to be convenient. A mercurial pill or powder appears, according to my observation, to have a greater effect in reducing the liver and relieving the right heart than more powerful purgatives of another kind.

As has been already said, there is no incompatibility between the depleting agencies just described and stimulants.

Stimulants, indeed, are often required, but until the right side of the heart is relieved, neither stimulants nor the remedies which act upon the heart will have a proper effect. They may, on the contrary, do harm; and much as the frequent and irregular action of the heart may appear to call for digitalis, the administration of this or similar drugs had better be postponed till the stress upon the right cavities of the heart has been relieved by the bleeding or purging.

Digitalis may then be given, at first with ammonia, ether, and belladonna; afterwards with nux vomica or strychnine; or, again, with tincture of iron. Convallaria, or caffeine, may be substituted for digitalis when the latter appears to disagree with the patient or fails in its effects. Caffeine, however, in my hands has been of much greater value taken at the same time with digitalis than in place of it, a profuse flow of urine withheld up to that time, coming on when the caffeine has been added. It is scarcely necessary to repeat, what is so well known, that diuresis is the most certain evidence of the beneficial effects of digitalis. But it may be added that it is not the increased secretion of urine which does the good. It is in itself only one of the various secondary results of the improvement in the circulation, showing, as it does, that pressure has been lessened in the venous and increased in the arterial side of the capillaries, and that the rate of movement through them is increased.

Other diuretics, squills, scoparium, juniper, the salts of potash, find their place as accessories or as alternatives, when digitalis and caffeine, or convallaria, have been taken so long that they begin to lose their effect.

In mitral incompetence digitalis may be given almost indefinitely, and patients often take it for years with obvious advantage, but such is not the case in mitral stenosis. Here the effects must be watched from day to day; at any time the action of the heart may all at once become disordered, many of its beats not taking effect on the systemic circulation, and failing to produce a pulse in the radial artery; the heart may be acting with fair regularity, and at the normal rate, when the pulse is rendered irregular in this way; or its rate may be slackened, and its rhythm disturbed, the effects of which will be exaggerated in the pulse.

It is not uncommon to have established under the influence of digitalis the apparently alternating systole of the two ventricles already mentioned, so that quite regularly there will be two beats of the heart for one of the pulse, pulso and heart sometimes alike acting regularly; but more commonly, the heart-beats being coupled, a weaker beat following a stronger one at a briefer interval, giving on auscultation the sounds one-two—one (silence) one-two—one.

This derangement of the heart's action is usually accompanied by a senso of oppression and distress in the region of the heart, and sometimes by nausea, and frequently the liver will be found to be swelling again. The digitalis should be at once suspended; afterwards a repetition of the mercurial aperient may enable the heart to bear it again, or its place may be taken by some of the other remedies enumerated.

AFFECTIONS OF THE EYE ACCOMPANYING MUMPS.

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THE implication, either concomitantly or by a process of metastasis, of other and distant organs during an attack of mumps, is a well-known peculiarity of that disease. The method of this transference is, I believe, one of the many questions pertaining to that obscure disease that yet remain to be solved by the general pathologist.

The testicle in man, and the mammary gland in woman, have been recognized from the earliest history of medicine as the objects of this metastasis of disease from the parotid glands; and within late years the ear has been discovered to stand in a similar unfortunate relation. That another important organ of senso was in danger from this usually mild and innocent affection I was not aware until the following case fell under my observation:

CASE I.—Frank W., a colored boy of fifteen, applied to me for treatment on July 25, 1885. He stated that the mumps had been going the rounds of his family, and that he had a slight affection of the *left* gland. On the third day of the disease he got wet, and two days after he noticed that his right eye was affected. On the fourth day of the eye trouble he applied to me. I found the upper lid much swollen, and he was unable to clovato it. On raising it I found the cornea intact, but there was a considerable clear chemosis of the conjunctiva at the outer and inner portions: the pupil was dilated, but not ad maximum, and there was paralysis of accommodation. There was diplopia in the upper, lower, and extreme left fields of fixation, and the upward and downward move-