Structural diseases of the heart considered from the point of view of prognosis / by W.H. Broadbent.

LUMLEIAN LECTURES

STRUCTURAL DISEASES OF THE HEART

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STRUCTURAL DISEASES OF THE HEART

CONSIDERED FROM THE POINT OF VIEW OF PROGNOSIS.

Delivered before the Royal College of Physicians of London.

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STRUCTURAL DISEASES OF THE HEART CONSIDERED FROM THE POINT OF VIEW OF PROGNOSIS.

MR. PRESIDENT, CENSORS, AND GENTLEMEN,—When your friendly compulsion and that of my then colleagues on the Censors' Board imposed on me the duty of delivering these lectures, my first impulse was to take up a subject which was one of the earliest to engage my attention, and in which I have never ceased to take a deep interest—that of chemical constitution and therapeutical action. In this, however, I have been anticipated by Dr. Lauder Brunton in his valuable Croonian Lectures, and I had, moreover, undertaken to give an address on Therapeutics before the British Medical Association in which I could not ignore this question. With regard to the nervous system, again, the ground had been effectually cut from under my feet by Dr. Hughlings Jackson and Dr. Ferrier, and it would have been the height of presumption in me to challenge comparison with the admirable and suggestive courses of lectures in which they have so recently instructed the college and the entire medical world. I have had, therefore, to fall back upon the subject of heart disease.

More than twenty years ago, in 1866, I read a paper before the Harveian Society on Prognosis in Valvular Disease, and in 1884 I took this as the subject of the Harveian Lectures before the same society. I have naturally had continually before my mind all this time the affections of the walls and cavities of the heart from the point of view of prognosis, but the question is one of such difficulty and complexity that I have never felt to have obtained so complete a grasp of it as to be in a position to place my conclusions before the profession. If I make the attempt now it is not from choice but of necessity, and I ask your indulgence for the many imperfections which you cannot fail to detect.

The muscular walls of the heart are liable to changes of various kinds, some of which constitute diseases which shorten life and give rise to much suffering. Of these structural alterations, some are extremely common—hypertrophy, dilatation, fatty degeneration; others rare—cancer, syphilitic gumma, abscess, aneurysm, localised fibrotic induration. We shall concern ourselves only with those which are comparatively frequent; the others, obscure as well as uncommon, are very seldom recognised during life, and a diagnosis is only made when an exceptionally clear case comes under the notice of an exceptionally acute observer.

Even when the common and familiar affections—hypertrophy, dilatation, and degeneration—only are taken into consideration, we find ourselves on much less secure ground than when dealing with lesions of the valves. The latter we can localise with great confidence; and knowing, partly by experience, partly by the application of mechanical principles, their effects and tendencies, we can, by making out how far such effects are manifest, form an opinion as to the probable course of the symptoms and as to the future of the patient.

On examination after death, again, we can understand the connection between the lesion and the symptoms, and can follow the sequence of secondary changes in the heart and vessels which are set up by the original valvular defect. In the case of structural changes, on the contrary, we are often left in some degree in the dark even by a *post-mortem* examination. In one patient fatty degeneration has apparently proved fatal at so early a stage that the naked eye characters of the condition are scarcely perceptible, and it is only by the microscope that its existence is definitely established; in another the change has proceeded so far that the fingers sink into the pale greasy walls, and the muscular fibres have almost disappeared, so that it is scarcely conceivable how the heart has been able to impress any movement whatever on the blood, or how life has been sustained through the intermediate stages of disintegration. So with regard to dilatation, there is no fixed relation between the degree of enlargement of the cavities and thinning of the walls found after death and the interference with the circulation observed during life. One man will live for years with a heart which has reached the extreme limits of dilatation, while another succumbs when it is but moderately advanced. If, therefore, we could make out with great exactness the dimensions of the heart, the size of its separate chambers, and the thickness of their respective walls, which is no easy task, we could not on these grounds alone compare one case with another, and decide upon the relative danger.

Many other considerations of extreme importance will come into the estimate: the functional vigour of the muscular walls as well as their thickness, the liability to palpitation, the state of the vessels, the degree of peripheral resistance, the presence or absence of reflex irritation of the heart from gastric or other derangement. The question of prognosis thus becomes extremely complicated, and is beset with uncertainty. An element of chance or luck even comes in—the subject of advanced disease is at the mercy of the slightest accident; many a patient lives for years with a dilated or fatty heart who would be killed by an attack of influenza or by tripping over a stone or a mat. A serious obstacle to the attainment of the minute and definite knowledge which alone is of real use in arriving at a sure prognosis in structural disease of the heart is the fact that a very large proportion of the cases occur in private and consulting practice, and comparatively few come under observation in hospital. How often does it happen that a patient presents himself a certain number of times in the consulting room at irregular and perhaps long intervals, and then ceases to attend, and nothing more is heard of him. Or he is seen once or twice in consultation, and his medical attendant is too busy to inform us of his further progress or to report his death; still less has he time to obtain and make a *post-mortem* examination.

While, however, the great difficulty of prognosis in structural diseases of the heart is acknowledged, it is of the utmost consequence that such approximation to a forecast of the prospects of life as is possible should be an object of serious endeavour. The cases are numerous, far more numerous at and after middle age than those of valvular disease, and everything which has been said as to the importance of prognosis in general applies here. An early recognition of these changes, indeed, is often of greater service to the subject of them than in the case of valvular affections, since it reveals also the ten-

dencies which are in operation, and often at a time when they can be successfully combated by treatment.

Too commonly, however, no attempt is made to recognise the existence and extent of degeneration or dilatation. The symptoms due to derangement of the circulation force themselves upon the attention of the medical man, but no murmur being detected, the only diagnosis ventured upon is that of "weak heart," a vague term which covers the entire ground, from temporary functional debility to disease inevitably and imminently fatal. Such a diagnosis reacts unfavourably upon the mind of the observer who rests upon it, and makes him less exact and trustworthy, while it may be full of danger to the patient.

These considerations alone would justify an attempt to render the prognosis of structural diseases of the heart more definite, but a study of these affections from the point of view of prognosis also leads to closer observation, and to the recognition of the importance of details which do not force themselves upon the attention so long as diagnosis in the ordinary and limited sense of the word only is the object.

A further justification of the prominence given to prognosis is that the grasp of all the facts of the disease and of the individual case, which is necessary to the formation of a just forecast of the result, is the best guide for treatment, whether this may demand chiefly patience and caution, or must be energetic and prompt. Prognosis is not merely a well-instructed conjecture as to the ultimate issue, it is a deliberate judgment as to the processes and tendencies of the disease, and as to the constitutional soundness and strength of the patient. The foresight relates to the dangers which attend the attack, to the course it will run, and to the influences and contingencies which make for or against the sufferer. To this there has only to be added a knowledge of the therapeutic measures by which the tendency to death or structural injury can be antagonised, and by which the patient can be guided and helped, together with skill, courage, and promptitude in applying it, and we have all the requisites for successful treatment.

Treatment, therefore, will form a natural sequel and corollary to prognosis.

Hypertrophy.—This condition of the heart will not detain us long. The prognosis of cardiac hypertrophy, like the symptoms, is that of its cause, and the character and degree of hypertrophy are important, not so much in themselves or on account of danger likely to arise out of them, but as indicating the existence of some condition which has given rise to hypertrophy, and which is serious, possibly, in proportion to the hypertrophy which it has provoked.

The causes which give rise to hypertrophy must be enumerated. The left or right ventricle may be affected alone or predominantly, or both may have undergone change. The causes which bring about hypertrophy of the left ventricle are in the first place valvular diseases, stenosis, or insufficiency of the aortic valve, or mitral insufficiency. With these we are not now concerned. Next in frequency will be protracted high arterial tension. When high pressure in the systemic arteries is recognised, and especially when there are evidences of its having been habitual for some time, such as large, thickened, tortuous, radial, and temporal arteries, or a dilated ascending aorta, the underlying condition to which it is due must be identified, and according as this is found to

be renal disease more or less advanced, or gout, incipient or confirmed, or one or other of the affections, organic or functional, which are capable of inducing high arterial tension, will be both prognosis and treatment.

Left ventricle hypertrophy, again, may in rare instances have no other assignable origin than adherent pericardium, a condition which is difficult to diagnose, and with regard to which prognosis is extremely indefinite. Adherent pericardium may, however, be a factor in the causation of sudden death, and its recognition, therefore, is a matter of interest.

If hypertrophy is at any time produced by a mode of life entailing sustained muscular exertion, it is physiological and not pathological, and has no claims on our attention; unless, indeed, the initial change has been dilatation caused by violent and sustained effort, such as racing, the hypertrophy being secondary to this; but even here it is the dilatation and not the compensatory process which has to be considered. Hypertrophy of the right ventricle, as of the left, has its most common cause in valvular disease, in this instance chiefly mitral stenosis or insufficiency, and after valvular disease in conditions which give rise to obstruction in the pulmonary circulation.

Emphysema and bronchitis are the lung affections which most frequently give rise to hypertrophy of the right ventricle, but the hypertrophy is rarely dissociated from dilatation, which is the primary effect of the obstruction in the pulmonary capillaries. Collapse of a portion of the lungs, contraction of a lung from pleural adhesions, fibroid phthisis, or any condition which throws a considerable area of lung surface out of gear will in a certain degree give rise to overwork and consequent hypertrophy of the right ventricle.

The diagnosis of cardiac hypertrophy must be briefly described, if only for the purpose of pointing out the distinctions between it and dilatation. Taking first hypertrophy of the left ventricle—as regards the pulse, it is that of the condition which has given rise to the hypertrophy. When this has been arterio-capillary obstruction from renal disease, gout, lead poisoning, pregnancy, or other cause, the pulse will be that of high tension. The artery will be full between the beats, will not be easily flattened under the fingers, and can be followed some distance up the forearm like a cord. The pulse wave will not have a violent ictus, but will rise gradually and subside slowly, and as it makes little impression on the fingers it is sometimes described as weak, but when the attempt is made to arrest the wave very firm pressure is required. The description given applies strictly only when the artery is contracted and small, or of moderate size; when it is large, whether from physiological relaxation of its muscular coat or from distension by protracted high pressure within it, while the vessel can still be felt very distinctly between the beats, the pulsation is more conspicuous and abrupt.

Physical Signs.—On inspection, there may or may not be recognisable bulging over the cardiac area; the impulse is not extensive or violent in uncomplicated cases; the apex beat, if visible, is seen below the normal point, frequently in the sixth, sometimes even in the seventh, space, and is probably also displaced somewhat outwards; it is a circumscribed gentle heave. Occasionally one or more of the intercostal spaces above and to the inner side of the apex line may be seen to be retracted, but this is very rare, and is never

so well marked in hypertrophy without valvular disease as it may be seen in aortic regurgitation.

By palpation, which is always an extremely important part of the physical examination of the heart, the apex beat is further defined, and is felt as a powerful but deliberate thrust in the space, sometimes distinctly lifting the adjacent ribs. The more the fingers are pressed into the space, the more distinctly is the thrust recognised. When the flat of the hand is laid over the cardiac region, a general heaving impulse can usually be felt, but when the left ventricle is alone or mainly affected, it is not very conspicuous. As a rule, no impulse is felt to the right of the sternum except when the aorta is dilated, in which case pulsation may be felt when the fingers are thrust into the second and third spaces near the sternum. It must be added that sometimes neither apex beat nor impulse can be seen or felt when the hypertrophy is considerable. Mostly this is in deep-chested individuals with large, overlapping lungs, but occasionally hypertrophic enlargement takes a direction which carries the heart away from the chest wall.

Percussion maps out more or less accurately the enlargement of the heart downwards and to the left. This demarcation should be done with extreme care, but it must not be taken for granted that the outline drawn on the surface corresponds exactly with that of the organ, or gives a trustworthy idea of its size. To say nothing of the difficulty of defining deep dulness, the heart may enlarge backwards instead of laterally. The results of percussion must be correlated with all the other evidence as to the size of the heart.

Auscultation, besides teaching the character and intensity of the sounds, must be made to contribute to the information on this point by careful noting of the seat of maximum intensity of the sounds in the apex region.

The left ventricle first sound as heard at the apex is less distinct than in a normal state of the heart; either the mass of muscle enters into contraction less simultaneously, and the muscular tension being less sudden yields a duller sound, or the thickness of the walls masks the sound produced by the sudden tension of the valves and tendinous cords. The second sound, which at the apex is aortic only, is, on the contrary, usually louder than normal, and is often heard at or near the apex more distinctly than in the right second space.

When the first sound is prolonged and muffled, and especially when it can be described as "impure" (a very objectionable term) or murmurish, careful examination will usually reveal that it is reduplicated, the two first sounds of the right and left ventricles not coinciding. This indicates that the left ventricle is no longer quite equal to the extra work imposed upon it, and marks the supervention of a tendency to dilatation.

At the base of the heart the left ventricle first sound is still less distinct than at the apex, and is, indeed, frequently quite inaudible, while the accentuation of the second is rendered more evident by the absence of a first sound. As has already been said, however, the aortic second sound may be even more distinct at the apex than in the aortic area. When this sound is not only accentuated but low pitched and ringing, the root of the aorta is more or less dilated, and the sound will be heard for some distance to the right of the edge of the sternum, perhaps over a great part of the chest and along the spine.

The sounds of the right ventricle undergo no modification of sufficient importance to require notice.

Various symptoms are described as resulting from hypertrophy of the heart—discomfort from the violence of the impulse, or actual pain in the region of the heart, tenderness on pressure in the neighbourhood of the apex, throbbing sensations in the head and neck, pulsatile noises in the ears, or audible pulsation in the carotid and other arteries. The action of the heart may be unduly frequent, or too easily excited, or abrupt and irritable, or irregular with falterings and bounds, which are very disturbing to the subject, and the heart may be prone to palpitation. There may be a sense of respiratory oppression, with sighing and desire to fill the chest with air.

Some of the symptoms are simply the result of the size of the heart and of the vehemence of its beat; others are due, not to the hypertrophy itself, but to its cause, whether in the valves or in the vessels, or in external influences which have given rise to it; others, again, are common to various affections of the heart, functional or organic, besides hypertrophy. They have no such direct or definite bearing on prognosis as would warrant a discussion of their significance, though a sustained frequency of the pulse is an unfavourable sign.

The question of prognosis in relation to hypertrophy mostly resolves itself into this: whether the compensation which it establishes is adequate and efficient, and how far it promises to be durable. The danger that the hypertrophy may go beyond the requirements of the occasion which has called for it does not, in my opinion, need consideration.

Compensation is efficient when there are no symptoms of embarrassment of the circulation, and when the heart responds to all ordinary calls upon it without undue shortness of breath or respiratory distress. The effects of exertion are an important criterion, due allowance being made for the greater liability to breathlessness which is natural to some individuals, or is produced by bodily conformation or results from a sedentary mode of life.

But the sounds of the heart usually give notice when it is overtaxed by the resistance to the onward movement of the blood. The interval between the first and second sound may be prolonged, the systole requiring more time than under ordinary circumstances to complete itself. So long as the normal proportion between the systolic and diastolic pauses is not disturbed there is no indication that the heart is unequal to its work or is suffering from the stress put upon it: but when the systolic pause is lengthened at the expense of the diastolic, so that the sounds are equidistant, the period of repose and reconstitution of the muscular fibres of the ventricle is shortened, and their nutrition must in time suffer. During systole the blood is squeezed out of the walls of the heart, and it is during the diastolic relaxation that it obtains free access to the cardiac fibres.

Another evidence that the heart is yielding to the strain of overwork is reduplication of the first sound. That is, as I have argued elsewhere, due to want of synchronism between the two ventricles in the act of contraction, or rather in arriving at that point in their contraction when their valves and tendinous cords and muscular walls are all made tense. This reduplication is at first very slight, the two sounds are separated by the briefest possible interval, and are distinct one from the other only at one spot, just to the inner side of

the apex; elsewhere there is only a slurring or prolongation of the first sound. Later the sounds of the right and left ventricles are quite distinct, and the duplex first sound is recognised over a considerable area, usually in the direction of the ensiform cartilage, but sometimes upwards. Sometimes the dislocation of the ventricular first sound is so considerable that it is a task of extreme difficulty to identify the sounds of the heart at all, and associate them with the systole or diastole respectively so as to say which is first and which second. The most striking and confusing examples are met with in aortic stenosis, but they occur also in high arterial tension. The prognosis becomes serious when the first sound is broken up in any very considerable degree.

But when the cardiac hypertrophy has been brought about by high arterial tension, whether associated with renal disease, or gout, or other cause, there must be taken into consideration the possibility that the powerful heart may rupture diseased vessels in the brain, and if the arteries are conspicuously degenerated it is better for the patient that the heart also should undergo some degree of degeneration, and fortunately this usually takes place.

Treatment.—Treatment for hypertrophy as such has always appeared to me to be out of place. The functional vigour and energy of the overgrown and overstrong heart could no doubt be reduced by various means—low diet, enforced rest, and such drugs as aconite—but unless it is clear that the hypertrophy has gone beyond the requirements of the condition which has given rise to it, the advantages of this procedure would be more than doubtful. Even in the attempt to relieve the incidents of hypertrophy, palpitation, throbbing sensations in the chest or head, irritable or irregular action of the heart, præcordial oppression, the employment of direct cardiac depressants is rarely of service, and is at times attended with danger. In aortic stenosis, for example, I have known aconite, given with a view of quieting tumultuous action of the heart, so far reduce the contractile energy of the left ventricle that it was no longer able to cope with the obstruction, and death quickly followed from cardiac asthenia, the pulse becoming imperceptible, the extremities livid, and the surface of the body cold and damp. This would be less likely to occur where the cause of the hypertrophy was obstruction in the peripheral circulation, as the arteries and capillaries are relaxed by such agents as depress the action of the heart.

To speak of the treatment of the causes of hypertrophy would be out of place on this occasion, and would take up much time. All that we have really to consider, then, is not the treatment of hypertrophy or its causes, but treatment suggested by the hypertrophy. We shall recognise, for example, the necessity of diminishing the volume and improving the quality of the blood by appropriate diet and hygiene, and, if necessary, by tonics. We shall recognise, also, the desirability of diminishing the resistance to the onward movement of the blood in the arterio-capillary network by care in diet again, by aperients, and by eliminants of various kinds. In some cases the resistance in the peripheral circulation may be further lessened with advantage by the physiological relaxants of the arterioles and capillaries such as nitroglycerine and the nitrites. By these means the work thrown upon the heart is reduced, and, if necessary, the heart may be strengthened by such remedies as strychnine and digitalis.

All these measures are specially required when the hypertrophy is no longer quite equal to the task which it had originally been developed to perform, and the reduplication and other modifications of the sounds are present. It is now that the incidents of hypertrophy, palpitation, and the like, are most commonly complained of, and they will be best alleviated or removed by the measures just sketched out, by relieving the heart of work on the one hand, and by helping it on the other.

Dilatation.—The word dilatation requires no explanation as applied to the cavities of the heart. Individual cavities may be dilated, as for example the left auricle in mitral stenosis or regurgitation, the right ventricle in pulmonary emphysema or mitral disease, but when spoken of as a form of heart disease, dilatation usually means dilatation of the left ventricle, mostly with, but sometimes without, dilatation of the right ventricle. Together with the expansion of the cavity of the ventricle there may be more or less thickening of its walls representing an attempt at hypertrophy, compensatory of the dilatation itself, or of the difficulty in the circulation which has led to it. Not infrequently the hypertrophy has preceded the dilatation. Or the walls of the heart may have an approximately normal thickness, which will really imply a certain degree of hypertrophy, or they may be distinctly thinned. The parietes may have a normal colour, consistence, and structure, or they may be pale, flabby, and degenerated, or dense and tough from fibroid substitution. But even more important than the anatomical condition is the physiological or functional condition, the special characteristic of which is that the ventricle does not complete its systole, but only expels a portion of its contents.

Dilatation of the heart may be contrasted with hypertrophy rather than compared with it. Hypertrophy is compensatory and an evidence of vigour in the heart; dilatation is, for the most part, a confession of failure on the part of the heart muscle and an aggravation of other causes of interference with the circulation which may be in operation. In hypertrophy the augmented mass and increased strength of the muscular walls enable the ventricles to complete their contraction in the face of difficulty. The result of dilatation is that the ventricles habitually fail to expel the whole of their contents. Very frequently it is only a very small proportion of the blood which is projected into the great arteries. In well marked cases the chambers of the heart are always full, and little blood being received and expelled there is a stagnation in the auricles and ventricles which may allow of the deposition of fibrin among the fleshy columns and pectinate muscles. It has seemed to me that the imperfect emptying of the ventricles has not always been fully realised as the special feature of dilatation, but it will be seen that if a dilated ventricle launched the whole of its contents into the arterial circulation, the amount being much larger, the rate of movement of blood would be greatly accelerated, whereas the con trary is the case.

The dilatation attending aortic regurgitation belongs to a different category, and, instead of aggravating the difficulties of the circulation, it is a part of the compensatory arrangement. It is clear that if a certain proportion of the blood projected into the aorta at each systole returns to the ventricle during diastole, it is an advantage, and indeed a necessity, that the capacity of the ventricle should be increased,

so that, in spite of the reflux, a normal amount of blood may remain in the arteries and be distributed to the tissues. The dilatation, however, in aortic regurgitation is accompanied by hypertrophy which enables the ventricle to contract perfectly, so that the characteristic of dilatation—the partial and imperfect emptying of the ventricle—is not present.

It is probable, again, that the dilatation of the left ventricle met with in mitral regurgitation may have a similar compensatory effect. It would seem to be an advantage, since some of the blood regurgitates into the auricle, that the ventricle should contain sufficient to allow for this, and yet discharge a due amount into the aorta. In dilatation consecutive to mitral regurgitation, moreover, there is a certain degree of hypertrophy, and the systole is carried through. The dilatation of mitral regurgitation stands, then, on a totally different footing from primary dilatation.

The mode of production of dilatation of the heart is highly complex; it is, I think, usually understood to be the result of a gradual yielding of the walls of the ventricles, either from their own inherent weakness or from undue resistance to the onward course of the blood. It is often supposed also, when dilatation exists, that it is an established and more or less unvarying or progressive condition. Both these ideas require modification.

All violent or protracted exertion is attended with temporary dilatation of the heart, which may go so far, even in strong and healthy persons, as to give rise to temporary murmurs. Loud murmurs so produced I have heard at the apex, over the tricuspid area and over the pulmonary area, in individuals who had no evidence of cardiac weakness at the time, and who developed no valvular or other cardiac disease for many years afterwards. A personal friend always had a loud systolic pulmonary murmur after hunting, which sometimes, when there had been a severe run, lasted two or three days. It is, again, not very uncommon for young and strong men to return from climbing in Switzerland with more or less dilatation of the heart, which may persist for weeks. This is usually when little exercise has been taken during the year, and considerable ascents or very long walking excursions have been made without sufficient preliminary training. Boat races no doubt give rise to temporary dilatation, but no instance has come under my own observation. I have, however, met with it as a result of training for races. It might, perhaps, be better to speak of distension of the cavities of the heart in these instances, rather than of dilatation.

But the circumstances which are capable of producing a temporary distension of the ventricles in a sound and vigorous state of the organ will be competent to give rise to dilatation when it is weak and flabby, and other conditions are present which tend to dilatation, and it is more likely that the weakly ventricles give way from time to time under stress and fail to recover perfectly, than that the yielding is gradual and continuous. In contracted granular kidney with persistent and often extreme arterial tension, dilatation at an early period is comparatively rare, which would scarcely be the case were continuous resistance to the emptying of the ventricle the most efficient cause.

In violent exercise the pulse in becoming extremely frequent also becomes extremely soft and short, the arterioles and capillaries are relaxed in order to facilitate the rapid movement of the blood which is necessary to supply fuel and

oxygen to the muscles during exertion and the arterial tension is low. The resistance to be overcome by the ventricles is thus reduced to a minimum, which diminishes the liability to over-distension.

In the production of dilatation a common and important, if not a constant, factor is habitual high arterial tension. A second factor, less constant perhaps but more important, is inherent weakness in the ventricular walls. Sometimes one will predominate, sometimes the other. With these proximate causes, to use an old fashioned term, will co-operate very varied influences—exertion, excitement, chills, imprudence in eating and drinking, constipation, augmenting the stress on the heart; anæmia, anxiety and depressing emotions diminishing its power of resistance.

Acute dilatation of the heart is more common than is generally supposed.

In a very large proportion of the cases admitted into hospital suffering from symptoms due to cardiac dilatation, there has been an acute aggravation of the affection from work or exposure, and under treatment a considerable diminution in the size and capacity of the heart is commonly observed. But cases occur in which there is ground for supposing that dilatation has been induced at once in a heart not previously affected. The following are examples selected as illustrations.

A gentleman at the age of 70, in vigorous health and capable of any ordinary amount of exercise, overtook a labourer pushing a heavily laden wheelbarrow uphill, who had to stop and rest every few yards. Proud of his strength, he told the man to stand aside, and himself wheeled the barrow for some distance at a good pace. He lost his breath and found that he did not recover it as he expected, but that he continued to pant and to be conscious of violent action of the heart, accompanied by a sense of oppression in the chest. He got home with difficulty, the least exertion was attended with shortness of breath, and he could not rest at night. After a few days he sent for his medical man, when the physical signs of dilatation of the heart were found to be present. Mitral insufficiency was quickly established and when I saw him considerable dropsy existed. He died shortly afterwards.

A gentleman, aged about 55, remarkably strong and active, who was said to have had a slight attack of pleurisy shortly before, ran quickly up a long flight of stairs in the City. On arriving at the top he was found gasping for breath, unable to speak and scarcely able to stand. He was soon sufficiently recovered to be sent home, and a few days later he was brought by his medical attendant to my consulting room, chiefly in order that I might aid in enforcing the rest and care which were considered necessary. The patient admitted that he was weak and soon out of breath, but declared that he was quite equal to business. The pulse was irregular in force and frequency, the apex beat of the heart diffuse, devoid of the impulse or thrust, and displaced downwards to the sixth space and outwards beyond the nipple line, and the other physical signs of a considerable degree of dilatation were present. With great reluctance a certain amount of rest and treatment was submitted to, and the heart and pulse became stronger and steadier, and the apex beat came in towards its normal situation about an inch.

Two months later the patient ran down from a committee room in the House of Commons for a bag of papers which he

had forgotten, and back again. An attack of the same kind as that just described came on, and this time lasted longer. The evidences of dilatation of the left ventricle were more marked, and œdema of the ankles soon came on. The patient was kept in his room, and as far as practicable at rest in bed or on a couch. Improvement was again obtained but much more slowly than before, and as my visits were the chief restraint upon him, after a brief attendance at short intervals, further consultation was put off indefinitely.

Within a few days the exertion of going downstairs and a serious imprudence in diet brought on a return of symptoms, and the œdema of the lower extremities was shortly complicated by thrombosis of the deep femoral vein of the left side, which extended along the iliac vein to the vena cava and down the right iliac and femoral veins. A tedious illness followed, which finally proved fatal by extension of the thrombosis to the renal veins, the heart itself appearing to improve somewhat.

Another case was that of a boy who was admitted into St. Mary's Hospital with extreme dilatation of both ventricles. He had had no previous illness, but had been underfed. Most of my hearers, in driving about London, will be familiar with the appearance of lads in the scanty attire of the cinder-path, who are making use of the streets as a training ground for races, usually in the dusk of the evening, after working hours; perhaps, also, this time is chosen to avoid police interference. I have myself often been astonished at the pace and endurance of these athletes-under-difficulties. The patient had been training in this way, and had persevered in spite of shortness of breath, till severe symptoms set in. He died soon after admission, and the ventricles were found to be greatly dilated.

A certain degree of dilatation of the left ventricle usually occurs at the onset of acute renal disease, under the combined influence of the resistance in the peripheral circulation and of the enfeeblement of the heart. In other acute diseases this may happen. I have twice seen considerable dilatation of the heart in mumps attended with collapse and cerebral disturbance; in one case it was fugitive; in the other, seen with Dr. George Coates, it lasted some time and recurred afterwards. In both the arteries were tightened up, giving rise to high tension and great resistance to the onflow of the blood. Whether the severe collapse and delirium which sometimes supervene as the acute symptoms are subsiding in mumps are always associated with dilatation of the heart it is not in my power to say.

A common cause of dilatation of the heart is anxiety. Nothing is more certain than the influence of prolonged mental depression upon the heart, and the sensation of aching, oppression, and weight which attends grief and anxiety, and which was considered to point to the heart as the seat of emotion generally, is indicative of an injurious effect upon this organ. It is almost literally true that people die of a broken heart. The combination of overwork, excitement, worry, and trouble often met with in City life, especially on the Stock Exchange or in mercantile or financial circles during a commercial or financial crisis, brings us many cases of cardiac dilatation among men, and it is needless to say that domestic anxieties—grief on account of children who have died or given trouble—have the same effect among women.

Among the special causes of dilatation of the heart—acting, no doubt, on pre-existing tendencies—which have come under my notice are injudicious hydropathic treatment, the so-called Banting method of reducing obesity, and inhalation of the fumes of Himrod's powder for the relief of asthma.

In one case a gentleman suffering from dyspeptic symptoms, probably due to cardiac weakness and consequent sluggish circulation in the abdominal viscera, underwent a routine treatment by baths, wet cold packs, and compresses, under which attacks of dyspnœa came on and œdema set in. He had previously been under the care of competent observers, who had not found any serious degree of dilatation. The dropsy advanced in spite of treatment, and when I saw the patient it was considerable, and the physical signs gave evidence of extreme dilatation and thinning of the left ventricle. Death occurred suddenly during a paroxysm of dyspnœa.

Another patient came straight to my consulting room from a hydropathic institution, where he had undergone vigorous treatment for digestive and liver derangements attributed to long residence in India. He was very anæmic, and breathless; the heart was greatly dilated and its action irregular, and there was incipient œdema. Fortunately he recovered.

One of the most extreme cases of dilatation I have ever met with was in the case of a lady who had undergone an amateur course of Banting treatment for obesity. She had lost some of her fat, but had become extremely breathless, so that to walk across a room or put on an article of clothing with the assistance of her maid caused her to pant and gasp for breath in a very painful way. The pulse was weak, small, and very irregular; no impulse or apex beat could be felt, and the size of the heart, as mapped out according to the deep dulness, was incredibly large, had not the results of percussion been confirmed by the sounds being audible over the entire area of dulness, and, later, by a feeble apex beat in the seventh space near the mid-axillary line. Apparently habitual high arterial tension had been exaggerated by an exclusively nitrogenous diet, and, under it, the ventricle had given way.

It may, perhaps, be well to add that in the treatment of obesity by beef steak and copious draughts of hot water there has not been, according to my experience, increase of arterial tension, but the reverse.

The following case is the most serious of several attributable to Himrod's powder:—

In December, 1883, I saw with Dr. Andrews, of Hampstead, a gentleman, aged about 36, apparently of sturdy constitution, who, after an attack of bronchitis, suffered from several nocturnal attacks of asthmatic dyspnœa. A month at Hastings had apparently restored his health; but, after a fortnight of work in London, his nocturnal asthma and shortness of breath were as bad as ever. For the asthmatic attacks he had inhaled immoderately the fumes of Himrod's powder, obtaining relief for the time, but with disastrous after-effects. The whole house was reeking with the odour of the fumes at the time of our consultation. Dr. Andrews had witnessed the rapid development of the condition of the heart which existed. The area of cardiac dulness was greatly increased; no apex beat was recognisable; there was a doubtful systolic murmur (mitral) in the region of the apex, a loud systolic murmur (tricuspid) near the ensiform cartilage, and a faint

systolic aortic murmur. Tricuspid incompetence was further distinctly manifested by great enlargement of the liver, and by marked jugular pulsation. There was nothing in the patient's habits, or mode of life, or previous history which was at all calculated to give rise to dilatation of the heart, and I had the less hesitation in attributing it to the solanaceous fumes from having seen similar effects in other cases. In about a fortnight, after free purgation by calomel and the administration of digitalis, the patient was much better, free from asthma and able to walk upstairs, while the liver had gone down to its usual size. The heart remained very large, gave no defined apex beat and only a diffuse general impulse, while a high pitched mitral murmur, a louder tricuspid murmur of low pitch, and a faint aortic murmur, all systolic, were audible. The action of the heart was curious; now and then there was a sudden bump against the palm of the hand placed over the right ventricle, and it was found that the beats of the heart were in pairs, only the first of which was accompanied by a systolic apex murmur, the second having a loud first and second sound, but scarcely reaching the pulse. Within both beats the loud tricuspid murmur was present. The improvement continued, so that the patient resumed his duties before the end of January, and attended regularly to business through the month of February. In March came another relapse which led to dropsy, and the patient died early in April. Fortunately an examination of the body was permitted, notes of which are as follows:—

There was much œdema of the legs and right arm, but no fluid in the abdomen. The heart was enormous, measuring six inches and a half from base to apex, and sixteen and half in circumference. It was flabby, lying flat, pale, fatty looking, not lacerable. The right auricle was greatly dilated and very thin, the wall being translucent at one part; the appendix was filled up by solid clot and stained black by blood. The tricuspid valve took four fingers easily; there was no roughness. The right ventricle was enormously dilated, and would almost have held a duck's egg; the walls were thin, soft, and flabby, the valve stained black; the flaps were thin and the cords delicate. The pulmonary artery and valves were normal. The left auricle was not at all dilated, the appendix contrasting with the right, and compared with the other cavities curiously small. The mitral orifice would admit two fingers. The left ventricle, like the right, was enormously dilated, and looked as if it would hold a fist; the walls were thin, mottled, and flabby, but not lacerable; the papillary muscles were small, the mitral valve and cords quite normal, and remarkably free from thickening. The aorta was very small and the valves normal.

Physical Signs.—The diagnosis of dilatation of the heart is comparatively easy to anyone who has had a fair amount of experience, and is prepared to exercise sufficient care in making the requisite physical examination. Certain precautions are necessary which will be pointed out later. When, however, the dimensions of the heart have been made out, and an estimate has been made of the relative degree of dilatation and hypertrophy, and of the comparative condition of the two ventricles, a small step only has been taken towards the attainment of the knowledge which is necessary in order to forecast the course and issue of the case, and to direct the treatment. This may be illustrated by a reference to experience which must have been met with by most of the

physicians here present. A patient is seen on account of dropsy and other severe symptoms due to dilatation of the heart. Under treatment he gradually recovers, and is, apparently, much in the same state of health as before. After a shorter or longer time—months in some cases, years in others—a relapse of the symptoms indicative of cardiac failure takes place. The general conditions may not apparently be worse, there may be no marked difference in the physical signs, or in the state of the pulse; but the patient's chances of recovery are not the same. Besides the fact that he is older by so much, a change has taken place in the muscular structure of the heart which is not appreciable by means of physical signs. There is not the same reserve of force or nutritional vigour, and the response to treatment is not the same. If a second attack does not prove fatal, a third or fourth will, and the briefer the interval, the more serious is the significance of the relapse, unless it is accounted for by some sufficient cause. But the defective vital energy, which in one case becomes manifest only after one or more serious derangements of the balance of the circulation with dropsy and other symptoms, may in another be present from the first. There may be hereditary tendency to weakness of the heart, or the heart may be worn out by an unhealthy mode of life, or by protracted emotional strain; or the patient's tissues generally may lose their nutritional vigour early. Facts of this class are not revealed by physical signs; some of them may be known to the family medical man, but otherwise they are ascertained only by persevering and careful inquiry. It is obvious, then, that many other considerations enter into a true and thorough diagnosis of dilatation of the heart besides its anatomical condition.

With this introduction, the physical signs which reveal dilatation may be described together with the pulse.

The pulse in advanced dilatation of the heart is usually irregular in rhythm and unequal in force of beat, and is sudden, short, unsustained, and usually easily compressed; the artery at the wrist may be large or small. It will be specially large when there has been antecedent high tension which has dilated the arteries. The significance of a pulse of this character is not absolute, as all these characters may be present when there is no recognisable change in the walls, cavities, or valves of the heart, apparently from disordered nervous influence only. The pulse, again, need not be irregular in advanced dilatation of the heart so long as the patient is in repose and the breathing is tranquil and easy. The regularity, however, is easily disturbed by exertion or effort, or by bronchitis, or merely by deep breathing. In moderate and slight dilatation the pulse may be regular, but, while irregularity is the rule, there is no such constant relation between the degrees of regularity or irregularity of the pulse and the amount of dilatation of the heart as to make one diagnostic of the other.

In the examination of the heart, inspection and particularly palpation will be of the greatest importance. The visual examination will be directed to the situation and character of the apex beat and to the impulse of the right ventricle. Retraction or bulging of intercostal spaces, elsewhere than at the visible apex beat, will be noted. The apex will be displaced outwards and downwards, and it may be visible over a large area. Very commonly it cannot be seen at all, and the point of maximum visible impulse does not necessarily be-

long to the true apex. The right ventricle impulse will be diffuse, if recognisable at all, and as a rule it is inconspicuous.

Palpation in most cases of dilatation furnishes information which contributes more to precision of idea as to the actual state of the heart than any other branch of physical exploration. The right hand should be applied closely over the entire cardiac region, the palm over the right ventricle, the fingers, spread out and close together alternately, over the apex region. Distinct impulse over the right ventricle, while it indicates more or less obstruction in the pulmonary circulation, indicates also some degree of vigour in this ventricle available for compensatory work. A mere vibration has a converse significance. The first object of attention, however, will be the identification of the point of maximum impulse in the apex region, and a careful estimation of the area over which the apex beat extends, and of its force and character; whether, for example, it is a mere concussion of the chest wall or a more or less distinct thrust at any point. Further exploration will be made by pressing the fingers into the intercostal spaces around and beyond the point of maximum impulse, and it must be borne in mind that this impulse may not be the real apex beat, but the impulse of the right ventricle. Sometimes impulse is detected much above the normal situation, in the fourth space perhaps as far outwards as the anterior axillary fold or behind it, or it may be concealed by the female mamma. It may be the left edge of the right ventricle resting on the interventricular septum which is here felt, or a part of the rounded apex of the left ventricle. According as the apex is capable of giving a distinct thrust or communicates only a diffuse shock, and, according as the beat is well defined and steady, or vaguely felt over a considerable area, will be the estimate of the degree of dilatation and of the thickness or thinness of the heart wall. Not infrequently neither impulse nor apex beat can be detected, or the impulse is so vague that it cannot be localised; unless this is due to overlapping lung, it indicates great weakness of the muscular walls of the heart.

By deep percussion the outline of the heart can be mapped out. It is more rounded in the apex region than normal, and the area of dulness is greatly extended to the left. When no impulse of any kind can be felt we may have to depend entirely on percussion for information as to the size of the heart. I have found continuous deep dulness outwards as far as the mid-axillary line and downwards to the seventh space, shown to be cardiac by the intensity of the sounds at the extreme limits, and, when the heart had gained strength, by an apex beat recognisable by palpation at the farthest point.

The characteristic modification of the sounds of the heart produced by dilatation is that the left ventricle first sound becomes short; usually it is also louder than normal. Probably from this change in the character of the left ventricle first sound it is almost always audible in the aortic area, contrasting in this with hypertrophy. It is audible also to the left of the apex beat. When no impulse or apex beat can be felt, the sounds, and especially the first, must be made use of to ascertain how far to the left the left border and apex of the heart have been carried by the dilatation, and in what degree the enlargement is due to dilatation or hypertrophy. Percussion, of course, maps out the deep dulness, and shows

approximately the limit of the heart and its extension to the left, but the point of maximum intensity of the sounds and the area over which they are audible will corroborate or correct the idea formed as to the size of the heart from percussion, and percussion yields no information whatever as to the kind of enlargement, but leaves it to be supplied by the character of the sounds on auscultation.

Not uncommonly dilatation of the left ventricle is accompanied by and gives rise to a systolic apex murmur, obviously due to mitral incompetence. This is induced by imperfect accomplishment of the constriction of the orifice, which is part of the normal contraction of the ventricle, and which cooperates with the curtains of the valves in preventing regurgitation into the auricle. Now mitral valvular disease is attended with precisely the same combination of conditions—incompetence of the valve and dilatation of the ventricle—and of physical signs—a systolic murmur and displacement of the apex beat with increased cardiac dulness. The prognosis is very different in the two cases, and it is therefore extremely important to distinguish between them. This cannot be done with any degree of confidence by means of physical signs alone, and the discussion of the diagnosis must be reserved, but it may be said that the presence of the left ventricle first sound in primary dilatation and its absence in primary valve lesion will sometimes be of great service as a factor.

There is nothing in dilatation of the cavities of the heart to affect specially and directly the second sound, but, in proportion as this condition of the ventricles impairs the propulsive energy of the systole the second sound will be enfeebled and the aortic second sound is usually weak as compared with the left ventricle first sound. The relative loudness of the sounds therefore enters into the considerations from which may be calculated the efficiency of the ventricle.

Very important information is often supplied by observation of the intervals between the first and second and the second and first sounds, the short and long pauses respectively. When, with dilatation of the left ventricle there is resistance in the arterioles and capillaries, which is very commonly the case, the interval between the first and second sound may be prolonged, and as this marks the duration of the systole it shows that the ventricle is endeavouring to cope with the difficulty and to complete its contraction. The short or systolic pause may be so prolonged as to equal the diastolic, or long pause, and the sounds thus become equidistant, the first also having become short and sharp, the only difference between the sounds is one of emphasis or of pitch, and it is often difficult to say which is which. The sounds may be compared when the heart is acting slowly to the ticking of a clock, when rapidly to the puffing of a distant locomotive.

On the other hand, the first and second sounds may be approximated, and as we cannot suppose that a dilated and enfeebled ventricle completes its systole in a shorter time than normal the only possible explanation is that it is quickly brought up short by the resistance in the arterial system and expels but a small proportion of its contents. This abbreviation of the systolic pause is therefore a serious indication of failure of the ventricles, and when carried to an extreme, so that the second sound follows the first immediately and almost seems to overtake, it is significant of immediate danger.

In two successive attacks of symptoms due to dilatation of

the heart when the degree of dilatation appears to be exactly the same, a difference in the length of the systolic pause is sometimes the chief, if not the only, point which makes a difference in the prognosis. The first and second sounds are spaced in the first attack, which is survived and approximated in the second, which proves fatal.

Dilatation: Symptoms: Treatment.

The symptoms which attend the earlier stages of dilatation are extremely varied and very vague. An imperfect and fluctuating supply of blood to the brain will give rise to impairment of bodily and mental energy, and to irresolution and vacillation of purpose; the memory is liable to fail, especially in regard of recent events, and the power of sustained attention and consecutive thought is diminished. The frame of mind will often be despondent, and the temper may be irritable. There may be attacks of giddiness or faintness; in one case the patient, a vigorous old gentleman, aged 78, fell down from time to time as if he had been shot, with momentary loss of consciousness, getting up again at once apparently none the worse; sleep is usually disturbed, and sometimes almost absent, and, whether the nights are good or bad, there may be sopor at any period of the day, the patient dropping off to sleep even over his morning newspaper. For the same reason, the irregular and imperfect blood supply and the back pressure in the veins, digestion, and the action of the liver will be deranged. The appetite is usually bad, and food is followed by discomfort and by flatulent distension, which latter again reacts on the heart, and gives rise to oppression, or palpitation, or irregular action. The bowels are usually torpid. The urine is usually deficient in amount and high coloured, and there is often an habitual deposit of urates. Turbidity of the urine day after day, whatever the food and drink, or weather, or mode of life, should direct attention to the state of the circulation.

There is no one, and scarcely any combination, of the symptoms enumerated, however, which may not occur independently of weakness and dilatation of the heart, especially in states of system attended with high tension; and it would be waste of time to attempt to disentangle those directly due to the state of the heart from those which are merely accidentally associated with it.

The special symptom which calls attention to the heart as the probable starting point of a number of ailments is breathlessness on slight exertion, but even this may be produced by other causes—by anæmia, for example; after middle age, pernicious anæmia may often give rise to extreme breathlessness, which may excite a suspicion of heart disease. High arterial tension alone, which has not yet given rise to dilatation, simple debility, very sedentary habits, may also cause great shortness of breath. These facts are mentioned in order to warn against a too ready inference that heart disease exists simply because the breath is short in speaking. An interesting fact in connection with breathlessness due to dilatation of the heart is that it is often relieved by exercise of the voice. I have met with numerous instances in which a clergyman has climbed into the pulpit with the utmost difficulty, and has not only preached a sermon comfortably, but has been all the better for it. A sense of breathlessness

coming on during repose, and inciting the patient to make frequent deep inspirations, is usually a symptom of nervous depression, and has no necessary relation to heart disease. For the most part, as the disease advances, symptoms arise which are indicative of back pressure in the systemic veins, a gradually advancing œdema, and the like.

Prognosis.—But the problems which come before us in the prognosis of dilatation of the heart will best be elucidated by discussing them as they present themselves at different stages of the disease, and we may first take the fully developed effects. The patient is dropsical, œdema invading the thighs, loins, and abdominal parietes, as well as the legs, and there may be fluid in the abdomen and perhaps in one or both pleural cavities. The feet and legs will be cold and pale, or purple and livid, especially if hanging down; the hands also will be cold, and are often crimson or purplish, and the nails of a deep or dusky, instead of a bright pink. A white patch on the hand, produced by pressure, is very slowly invaded by returning colour. The sufferer is probably unable to lie down in bed, and is propped up by pillows, or he must have his legs down, and therefore spends days and night seated in his chair. Remarkable exceptions, however, are met with in this respect, some sufferers, while extremely ill, being able to lie down without distress. The face is pale, and perhaps puffy, especially under the eyes, with injected capillaries over the cheeks, and wears an expression of distress, and the eyes are watery. The lips are pale or bluish; the breathing is more or less laboured, even in repose, and the sufferer constantly supplements his reflex respiration by voluntary deep breaths. When he speaks it is in fragmentary sentences, and with evident effort and aggravation of the respiratory distress. The least movement brings on shortness of breath, which is often painful, even to witness. The pulse is frequent, irregular, and probably greatly deficient in tension, but not always. The urine will be scanty, of a deep colour, and probably high specific gravity; it most commonly throws down a copious deposit of brickdust lithates, and it may contain albumen. The appetite will be very bad, and there may be nausea; the tongue probably furred, the bowels constipated or irregular. One of the most distressing symptoms is sleeplessness, and when, after hours of weary shifting of position, the sufferer is overcome by fatigue and drops off, he has painful dreams, and wakes suddenly in affright and suffocation for lack of voluntary help to his respiration. The longest and best sleep will be obtained while sitting in a chair, and sometimes by day rather than in the night.

Pursuing the examination, the jugulars will be found full, but not, as a rule, greatly distended or pulsating forcibly. The liver will be enlarged, coming down sometimes as low as the umbilicus, and extending across the epigastrium into the left hypochondrium. It will often be jogged by the right ventricle, but does not usually exhibit true expansile pulsation. There may be fluid in the peritoneal or pleural cavities, more commonly not, and there may be physical signs of œdema and congestion of the lungs, or the percussion note may be good and the entry of air free and unattended with adventitious sounds of any kind to the very base of both lungs. By means of the physical signs of dilatation, as already described, we know that this is the condition underlying the symptoms of embarrassment of the circulation. We are called upon to answer the question, Has the patient a

chance of recovering from the condition described, or will he die?

The first element in the judgment to be formed will be the urgency of the symptoms, and special importance will attach to two of the series—the nausea, and loss of appetite and the sleeplessness—from the effects which they have on the patient's strength. Frequent vomiting of food is of very grave import, not only because the patient does not get the benefit of the nourishment, but because it shows that the stasis in the abdominal circulation has reached a point which interferes seriously with the digestive secretions. Attacks of faintness and of extreme exhaustion, or of severe dyspnœa, are also very serious.

An observation is here necessary. It is not always when the dropsy is excessive that the condition of the patient is worst. From the late appearance or entire absence of dropsy in fatty degeneration of the heart, in aortic disease, and mitral stenosis, it would appear that a certain degree of pressure in the arterial system is required to co-operate with the back pressure in the venous system for the full development of dropsical effusion; and when the œdema remains moderate in amount, while other symptoms—such as breathlessness, faintness, and muscular weakness—the latter especially, indicate great cardiac inefficiency, it may be because the left ventricle propels the blood with very little force. Degeneration may be a factor in the case. A frequent, short, unsustained pulse and heart beat, without much œdema, may thus indicate a more grave condition than extreme dropsy. Long-continued excessive frequency of the pulse is an unfavourable sign.

The urgency of the symptoms being about the same, a larger degree of dilatation (as indicated by a more extended area of dulness, displacement, and especially by greater diffuseness and weakness of the apex beat, and impulse and greater shortness of the first and weakness of the second sound) will add gravity to the prognosis, and it will be borne in mind that approximation of the first and second sounds is always a very serious indication. Favourable points in the physical examination will be a good right ventricle impulse, and it has appeared to me that there is a greater margin for treatment when the liver is considerably enlarged.

When the symptoms and physical signs have been well weighed, the first question to be asked in view of prognosis is whether there has been any adequate exciting cause of the symptoms. If there has been recognisable overexertion or excitement, or grave anxiety—any mental or bodily strain—or if there has been a chill, giving rise to bronchitis or other affection of the lungs, or deranging seriously the liver and digestive organs, it may be hoped by means of rest and treatment to undo the ill-effects and restore the balance. If, on the other hand, the symptoms have crept on gradually without traceable cause of the kind mentioned, and especially when due care has been exercised, and there has been no habitual error of regimen or neglect of bowels, the probabilities are that the symptoms are the outcome of conditions which cannot be reversed—of radical inherent weakness of the heart.

The previous mode of life, active or sedentary, careful or imprudent, and especially the habits with regard to alcoholic stimulants, will have a very important bearing on the probable issue of the attack, as will also the general soundness

and the absence or existence of disease of any other important organs, especially of the kidneys and liver. The patient's cheerfulness, hopefulness, and courage under his sufferings, or his despondency, will make powerfully for or against him, not only as direct influences, but because the state of mind is often an index of the state of the system.

Another inquiry of great prognostic weight will be as to the patient's family history. It is through this that we obtain an idea of his vital tenacity and of the trustworthiness of his tissues, and sometimes of the special liabilities of his heart. There are few tendencies which run more strongly in families than those whieh are manifested in the heart and vascular system, whether to high arterial tension with the effects on the vessels and heart, which follow from this, or to dilatation and weakness, or to degeneration, and a prognosis, otherwise not unfavourable, might have to be instantly revised on learning that the father and an uncle or two, or a brother, had died at about the patient's age from heart disease. Finally the response to treatment will speedily afford an indication of the utmost value, and this, the treatment, may be described at once.

In the treatment of advanced symptoms due to dilatation of the heart, we have to deal at the same time with defective propulsive power on the part of the ventricle, and damming back of blood in the venous system—the former being the primary and principal difficulty—and the indications are to relieve the ventricles of work and give them strength, and at the same time to deplete the venous engorgement.

This last object must, indeed, be taken first. We must undo the consequences of the original departure from the normal condition backwards and reach causes through effect; the venous stasis and the distension of the right side of the heart being the ultimate effects.

Venesection, the most effectual means of relieving the right side of the heart, is very rarely applicable in dilatation. It is conceivable that under some pulmonary complications resulting from chill, the engorgement of the right ventricle and auricle might be such as to make blood-letting the less of two dangers; but, the initial fault being weakness of one or both ventricles, we cannot trust the heart to adjust itself to a rapid change of any kind, and the right ventricle may not be in a condition to take advantage of the relief afforded it. Usually, however, the venous engorgement is developed slowly, and the indications for venesection do not arise.

The application of six or eight leeches over the liver is safer, and it will usually effect all that can be done by direct abstraction of blood. The indication for this local bleeding is enlargement of the liver and, when this is considerable, it rarely fails to afford striking relief. Very frequently, for example, the patient, who has previously been tortured by sleeplessness, will sleep at once and sometimes for a night or two afterwards. The reason for selecting the hepatic region for the application of the leeches is simply that pain and tenderness felt there are relieved; it is not supposed that blood is drawn from the liver, or that the same amount abstracted elsewhere would not be equally efficacious. The leeches will be followed by a hot poultice, which, besides encouraging the bleeding, will bring blood to the surface. When the liver is not enlarged, and especially if the right ventricle impulse and sounds are weak, there is no advantage to be gained by leeching.

Concurrently with the application of leeches a purgative will be given, which will deplete the portal system at the same time that the leeching depletes the systemic veins. Afterwards this will be the principal means of keeping down the venous engorgement. In a large majority of cases, indeed, we have to depend entirely upon purgatives for this purpose, as abstraction of blood by any method is inadmissible. It is not a matter of indifference what purgatives are employed. The object of a purgative is not simply to carry off as much fluid as possible and so drain the tissues. This may be the case, perhaps, in ascites from cirrhosis of the liver; but in heart disease of any kind, and especially in dilatation, much more is to be gained by rectifying the balance of the circulation, when the kidneys will resume their function and remove the excess of liquid.

Purgatives, then, may be made to contribute to this; and before considering them further we must refer to the first object of treatment, the relief of the heart from work and the increase of its vigour. Of these we can be much more sure of the first than of the second; we can more easily and certainly diminish the resistance in the arterioles and capillaries than we can lend strength and efficiency to the action of the heart, and, without removing the obstruction in the peripheral circulation, it might only do harm to incite the heart—weakened as its structures are—to greater effort to overcome it. Now, mercurial purgatives have this effect of diminishing arterio-capillary resistance and of lowering arterial tension, and therefore of relieving the heart. The hypothesis by which, as it seems to me, this observed fact is best explained, is that mercury influences the liver chemistry and promotes the elimination of impurities which, when retained in the blood, give rise to resistance in the capillaries. But, whatever the explanation, the fact that the arterial tension is notably lowered by mercurial aperients is one which is confirmed by daily experience. It is remarkable how frequently the statement recurs in works on heart disease that other remedies often fail to act until a dose of calomel or other mercurial preparation has been given.

Mercurial purgatives, then, have the double effect of depleting the portal system, which relieves the enlargement of the liver and the distension of the right side of the heart, and of diminishing the resistance in the peripheral circulation, and so relieving the left ventricle of stress. Very commonly the best of testimony as to the beneficial character of the result is given by refreshing sleep. The disadvantage, if such it be, that less fluid is carried off than by hydragogue cathartics is often compensated by an increased flow of urine; and elaterium, gamboge, pulv. jalap. co., and the like, when repeated, give rise to great exhaustion.

Calomel, then, or blue pill or grey powder, should be given in doses of from 1 to 5 grains, according to the urgency of the case, with colocynth and hyoscyamus or rhubarb, followed by some mild saline. After one or more full doses at the outset, a moderate dose may be given every second or third night. The acid tartrate of potash will often co-operate beneficially, both by its action on the bowels and on the kidneys.

The heart being relieved of work may be urged to more vigorous contraction by digitalis, strophanthus, spartein, squills, caffeine, convallaria, apocynum, the special heart tonics, with which strychnine may usually be combined with advantage. In a case of extreme suffering, digitalis may be

given with ammonia, ether, and nux vomica; in a more chronic stage, with iron and perhaps strychnine. Sometimes an effect can be obtained by giving citrate of caffeine in a pill, at the same time with digitalis in a mixture, when singly neither seems to be efficacious. Squills, again, may be given with digitalis, as in the well-known pill with mercury, or in some liquid combination. Next to digitalis stands strophanthus, which is a most valuable alternative when digitalis seems to produce sickness, as is sometimes the case, or when it fails to exercise a favourable influence on the heart. Sulphate of spartein I have seen to be of great service when digitalis and strophanthus appeared to have exhausted their influence. Of convallaria I have little to say. Apocynum has, in one or two cases, seemed to carry off dropsy in a remarkable way, but one patient died suddenly when apparently just well.

It is not necessary to go into greater detail with regard to these remedies. Throughout a case of the kind the medical man has to fight, so to speak, with both hands, and continuous watchfulness will be necessary to meet the vicissitudes which occur, and many changes in method may be required, while the same principles are held in view.

The prognosis, as has been said, will be greatly influenced by the response to treatment. This will be energetic, especially in the matter of purgatives, in proportion as the symptoms are urgent, and if no favourable effect is produced, the prospect of recovery is very poor. Very commonly improvement takes place up to a certain point, and then progress seems to come to an end. This is a trying stage both to the patient and to the medical man. Change of remedies and new combinations must be tried, both in regard of the aperient and of the tonic, not frequently and capriciously, but with careful study of the results and due allowance of time for obtaining them. Sometimes it does good to suspend all medicines for a few days and start afresh. Under these circumstances, again, it may be of the greatest service to drain the fluid from the legs, even when the extent of the œdema is not such as actually to call for it. The good result of removing an ascitic accumulation, should this be present, may be still more striking. Even a moderate amount of effusion in the pleural cavity, such as we should not think of dealing with under ordinary circumstances, should be aspirated. A straw may turn the balance either way, and a very slight obstacle may prevent the heart from regaining control over the circulation. It is not always desirable to postpone the removal of fluid till the particular conjuncture described arrives; it may be an urgent necessity at a very early period. Usually, however, it is prudent to give remedies a chance before resorting to puncture or paracentesis. As regards the method of drainage to be employed, Southey's tubes are, in my opinion, much the best, whether for œdema or ascites, but particularly in case of ascites.

The feeding of the patient through the long course of treatment will be a task of extreme difficulty. We have to contend with nausea and distaste for food amounting to disgust; sometimes the sufferer positively cannot swallow anything requiring mastication. The object to be held in view is to keep down the volume of the blood while maintaining its quality. A small amount of solid or semisolid food should be taken about every three hours; when the patient is not too ill to take his meals at the accustomed times it is a great en-

couragement to him to be allowed to do so, and he may then eat what he can of fish, fowl, tender meat, game, milk puddings. When the amount is small the regular meals may be supplemented by intermediate nourishment, such as a beaten-up egg, a little milk, or perhaps a small cup of strong soup or beef-tea, or a little beef or chicken jelly, or meat extract. Soups and jellies have the disadvantage of containing little proteid and much liquid, but they are stimulants to the flagging heart. Potted meat sandwiches are a great resource, and the pulp of raw beefsteak can be given in this form, disguised by cooked meat or concentrated gravy. A German physician of my acquaintance tells me that he frequently feeds his patients, suffering from cardiac dropsy of the kind under consideration, entirely on raw ham, and has great success.

The amount of fluid must be restricted as far as possible, especially that taken with food. Stimulants are usually imperatively necessary, but should be kept within limits known to and defined by the medical man. The patient is under a great temptation to resort to them for the relief of faintness, exhaustion, and nausea. Cream of tartar drink may be taken to quench thirst between meals, and in some cases a copious draught of hot water once or twice a day will run through the system rapidly and wash out the organs and tissues without augmenting permanently the volume of the blood or adding to the dropsy. When this is tried it must be ascertained definitely that the amount of urine is correspondingly increased.

A question which arises in almost every severe case is whether the patient must be urged to remain in bed or allowed to get up. Bed is undoubtedly the best place for him at first, during what may be called the crisis of the attack, for many reasons; the rest and warmth protect the heart from the strain of exertion and changes of temperature. On the other hand, the dyspnœa is usually worse in the recumbent posture, even with the shoulders raised, and may be intolerable unless the legs are allowed to hang down; not infrequently it is simply impossible for the patient to remain in bed. A suitable chair, therefore, is always necessary, with support for the elbows, shoulders, and head, which can be taken advantage of in turn in the frequent changes of position to which the patient has recourse to ease his breathing or elude discomfort, and the quickness and ingenuity of the medical man or nurse in devising expedients may greatly alleviate his sufferings. A bed table or other form of support, upon which the patient may rest his arms or elbows and head when leaning forwards while sitting in his chair, will often be very useful. A patient will frequently sleep better in this position than in any other. Perhaps the most common state of things is that the patient is up during the day, and tries to spend more or less of the night in bed. When he cannot at once bear to go to bed at night, he may undress and sit in his chair wrapped in blankets near the bed for a time, when he will often, after a nap or two, be able to lie down and sleep.

We may now come to the prognosis and treatment of dilatation of the heart at a period when it has not given rise to the serious consequences just described, and an enormous degree of dilatation may be arrived at before such effects are developed.

As regards physical signs, the most important evidence

will not be as to the actual size of the heart, but as to the strength remaining in its walls. A greatly dilated heart which is capable of giving a recognisable apex beat and fair impulse of right or left ventricle is more to be trusted than one which has apparently undergone less change in its dimensions, but the movements of which can scarcely be felt at all. There is always a possibility that degeneration may enter into the causation of the symptoms. It is better, again, that the sounds should be strong than weak, spaced than approximated, and it is important that the second sound of both ventricles should be well marked. Irregularity of action is, according to my experience, of less consequence than frequency, and I have learnt not to attach serious importance to the regular alternation of a strong and weak beat, so long as there is no great frequency with it.

The pulse is of importance chiefly as an indication of the vigour with which the blood is propelled into the arterial system. Dilatation of the heart presupposes high arterial tension at the time of its production, and it is a favourable sign that a certain degree of this tension persists. When we find a short, small, low tension pulse, unless this is the result of treatment, it means that the heart has not sufficient strength to maintain the pressure in the arteries.

The probability of improvement or of prolonged immunity from ulterior consequences will next depend on the general condition of the patient, on the soundness and nutritional vigour of his tissues and organs, and especially of his blood-vessels; if he is weak and anæmic, on the cause and character of the impairment of health—whether it is inherent or accidental, whether, in effect, it is remediable or not. When the state of the heart and of the general health is attributable to alcoholic or other excesses, including the abuse of tobacco, to habits or mode of life or external conditions adverse to health—whether or not the patient is willing and able to renounce his self-indulgence, and relinquish his vices, or alter his ways and surroundings. Considerations of this kind are of the utmost importance, but there is not time to enlarge upon them here.

When we come to consider the treatment of cardiac dilatation in this stage, the first question to be asked is, What object may we venture to set before ourselves? What assurance can we give our patients? Can we ever reduce existing dilatation? Can we even arrest the tendency to increase, and avert or postpone the evil consequences? To no question can we return a more confident answer. Every day we see dilatation reduced in some degree in favourable cases, and, at the worst, much can be done to prevent the development of its ill effects. It has already been stated that in the acute aggravation of dilatation which gives rise to symptoms, the apex and extreme limit of dulness may be seen to return towards the normal position day by day, and the well named "curable mitral regurgitation" of Dr. George Balfour is a form of dilatation incident to anæmia.

The means to be taken are in the first place the removal of any recognised cause in the habits, mode of life, and surroundings so far as this is practicable. Next, to improve the general health by favourable hygienic influences and by suitable remedies. The amount of exercise to be taken will be a most important question, but no rule can be laid down applicable to all cases. Speaking generally, exercise is good, and whatever amount of walking can be done without breath-

lessness or exhaustion, and especially with enjoyment, that will be safe and beneficial, and, provided due prudence is exercised, walking up hill need not be forbidden. It is injurious as well as cruel to insist on an amount of exercise which distresses the patient. The exercise should be regular and daily, not spasmodic with intervals of inaction. A walk may be taken day by day with advantage which, taken only once a week, would be injurious. The exercise, again, should not come immediately after food, and it is better taken early in the day than in the afternoon or evening. A walk into the City, for instance, might do good, when a walk back after the day's work would do harm.

In some cases the treatment may have to be begun by actual rest in bed for two or three weeks, perhaps with massage, and the patient may have to resume exercise with just the same caution as food is resumed after hæmatemesis from gastric ulcer.

The question will now come before us whether we should recommend the systematic graduated walking up hill at high altitudes known as the "Oertel" treatment for heart disease. In my judgment the idea is good, inasmuch as many people will submit to and carry out strict regulations, to which a certain amount of mysterious virtue is attached, who will not obey common sense instructions. I doubt very much whether any good will be done in fatty degeneration of the heart. Fatty overloading of the heart in connection with obesity, and some cases of early dilatation will be benefited, and an enormous number of imaginary cases of heart disease will be cured; the class of patients who come to us saying they have heart disease will be attracted by the treatment. A few patients will be killed.

But it is not always possible either to modify the patient's life in such a way as to put an end to the conditions which tend to produce and aggravate dilatation of the heart, or to secure for him all the desirable hygienic influences. It would be useless, for example, to recommend a city clerk to relinquish the sedentary occupation which is his only possible livelihood, and little less vain to order a lady who has never walked in her life to take regular exercise. We can, however, do much to keep down the arterio-capillary resistance or high arterial tension, which plays such a fatal part in the production of dilatation of the heart; we can regulate the diet and warn against habits and occasions which are injurious, and, by securing efficient and continuous elimination from the liver and bowels, we can often make just that difference which is required for the safety and comfort of the patient. This can usually be effected by an aperient pill, containing a small dose of one or other of the appropriate mercurial preparations, taken twice a week, and we must not be afraid to order patients, whose hearts have given way under protracted arterial tension, to go on with such a pill for years. Tonics of various kinds may be given with advantage from time to time with acids or alkalies. It is not often that digitalis or remedies of the same class will be required continuously, though they may render service occasionally.

Fatty Degeneration.

No form of heart disease is regarded with so much apprehension as fatty degeneration. More than any other, it carries with it the danger of sudden death and the liability to angina

pectoris, and, although happily it is not very common, it would be a most important acquisition to be able to make the diagnosis with certainty at an early period.

Its causation, which must be considered as it bears upon the prognosis, is in some cases clear, that is, when there is disease of the coronary arteries or obstruction of these vessels by any other means. The heart—perpetually at work—cannot afford to be mulcted of its full supply of blood. When, from any cause, this is defective the wear and tear of the muscular fibres, which must go on, is not repaired and their structure breaks down. Whether the *débris* actually forms the fatty granules and globules which are found within the sarcolemma or the fatty particles are substituted for the atrophied sarcous elements is not, perhaps, a settled question. The important point is that the primary change is atrophy of the muscle substance, the invasion of the fibres by fatty matter being secondary to this and consequent upon it.

Disease of the coronary arteries being thus a cause of fatty degeneration of the heart, the existence of conditions which may lead to the implication of the coronary arteries or their orifices in morbid processes will warrant a suspicion that cardiac weakness, which may be recognised, is the result of degeneration. For example, an aortic murmur coming on after middle age may not indicate serious valvular lesion, but, as it is probably the result of atheromatous changes in the valves or arterial walls in close proximity to the orifices of the coronary arteries, there is reason to apprehend that the disease may cause obstruction here or may have extended to the vessels themselves, and progressive weakness of the heart, were this to supervene, would be attributable to degenerative change in its walls. Acute aortitis is recognised mainly by a train of effects on the heart, produced in the same way, for example, by blocking of the mouths of the coronary arteries.

But there may be fatty degeneration of the heart when the coronary arteries are healthy. It is usually present—sometimes in a very advanced degree—in pernicious anæmia, and granular degeneration, which is an acute form of the disease, is a constant effect of severe typhoid fever and of fatal phosphorus poisoning. Cases occur from time to time in which a patient convalescing from typhoid dies suddenly on sitting up in bed. Here, again, in fatal anæmia and enteric fever the process must be the resultant of a balance on the wrong side, as between the catabolic and anabolic operations, disintegration and repair, but it is now the quality of the blood which is at fault, not the supply, and in typhoid fever there is also the injurious effect on the nutritional processes of long-continued high temperature. It is not to be wondered at that in pernicious anæmia and fever the heart suffers more than the voluntary muscles, since these are at rest and there is no functional wear and tear, whereas in the heart this is continuous and excessive. From what takes place in typhoid fever again, it is seen how rapid degeneration may be.

Diabetes, alcoholic excess—tippling rather than drunkenness—a sedentary mode of life, may conduce to fatty degeneration of the heart, probably through deterioration of the blood, or it may be secondary to myocarditis. Cases are met with for which no explanation can be found, and we are almost compelled to assume that there may be a defective assimilative action in the muscle cells of the heart, or possibly some unrecognised blood condition.

There is very little that is characteristic in the symptoms. In a large proportion of cases the subject of this affection has had no ailment which has led him to consult a medical man, when he is overtaken by sudden death during exertion or excitement, or the administration of chloroform, or after a full meal. Or the excitement or exertion may be passed through safely, and death follow some hours later, next day even. Rupture of the heart is one mode of termination, and this may take place on very slight provocation. Sometimes the patient has been engaged in his usual avocation up to the moment of its occurrence. In one case which came under my observation, an old gentleman of quiet retired habits, with nothing beyond the weakness incident to age, was heard to knock at the wall against which his bed was placed, and was found dead, the bedclothes scarcely being disturbed. A neat slit was found in the left ventricle near the apex close to and parallel with the septum.

The bearing of such occurrences on prognosis is direct and simple. No doubt in many cases of sudden death there have been warnings which the patient has ignored or has not spoken of. These will sometimes be acknowledged in the course of examination when they have not been mentioned.

When the course of the disease has been sufficiently chronic to permit of the recognition of symptoms, which in my experience is chiefly when the degeneration is secondary to change in the coronary arteries or to old-standing hypertrophy, with or without dilatation, they will be such as are produced by a slackening circulation, and they are not so different from those attending dilatation as to permit of any distinction being drawn between the two conditions in an early stage without physical examination. There may, perhaps, be greater fluctuations in dilatation, though even in degeneration there may be great temporary improvement under care and treatment. In advanced stages characteristic differences make their appearance. The symptoms of advanced dilatation have already been described; those attending degeneration are evidences of heart failure of another kind. A noteworthy point is that well-marked dropsy is rare, and probably never occurs in uncomplicated degeneration. The significance of this is that the special effect of the disease is defective pressure in the venous system; and it is to this are due the syncopal, apoplectic, and epileptiform attacks, which, together with angina pectoris, are the most characteristic later effects of fatty degeneration.

The syncopal attacks vary greatly in intensity. So far as they have come under my observation, they have been marked rather by duration than intensity, and have rarely been so complete as to be attended with absence of consciousness; they have usually been accompanied by prolonged coldness of the extremities and of the surface. I have not met with instances of sudden and complete loss of consciousness and immediate recovery as in dilatation. These syncopal attacks are very significant, and are often premonitory of fatal syncope.

The apoplectiform seizures are very remarkable, and in the absence of history and without examination they are not distinguishable from the apoplectic condition resulting from cerebral hæmorrhage. The patient is unconscious; the respiration, if he is allowed to lie flat on his back, may be stertorous—though stertor, after the teaching of Dr. Bowles, ought to be eliminated from the symptomatology of apoplexy

—and there may be hemiplegia, though this will be fugitive. Cheyne-Stokes breathing, which was first observed in connection with fatty degeneration of the heart, has not been present in the few cases which I have actually seen in the apoplectiform state, while I have met with it in a very large number of cases of uræmic coma and in connection with serious consequences of high arterial tension. On examination of the pulse and heart, however, it will be clear that there cannot have been sufficient pressure in the arteries to rupture even the most degenerate vessel, and, on the other hand, thrombosis or embolism is not competent to produce unconsciousness of the character and duration of these attacks. According to my experience, the patient is never quite the same after an apoplectiform attack; he is feebler in mind and body, and sometimes increasingly liable to syncopal attacks.

The epileptiform attacks are not often violent, but resemble *petit mal* rather than a typical epileptic fit; while, however, the convulsion may not be severe, there is profound unconsciousness—not like epileptic coma, but of a syncopal character—and the pulse may be extremely infrequent, sometimes less than 20 in the minute. In my judgment, the heart failure manifested by the slow pulse and the consequent arrest of the cerebral circulation are the cause of the fits, and it is not the epileptiform attack that affects the action of the heart.

By the time any of these forms of attack occur the diagnosis of fatty heart is usually sufficiently clear; but I have had under observation a case of very slow pulse with *petit mal*, in which the strength and volume of the pulse and the degree of impulse of which the heart was capable precluded the idea of advanced degeneration.

Is there anything characteristic in the appearance of a patient suffering from fatty degeneration of the heart? A greasy state of the skin with a sallow pallor of the face has been described, but nothing of the kind is present in a large majority of the cases. Many of the subjects of the disease retain the look of health for a long time, and even up to the moment when the heart ceases to beat. The cause may be local, and, even if a tendency to general deterioration of the tissues is present, the change mostly advances so much more rapidly in the heart than elsewhere, that there is no time for it to become conspicuous in the skin. The picture appears to have been drawn from cases of a universal chronic degeneration of vessels and heart.

Pulse and Physical Signs.—The most constant and significant feature of the pulse is that it is short and unsustained. The size of the artery at the wrist and the condition of its walls may vary greatly. When the arterial coats are healthy they are apt to feel extremely thin. The pulse rate may be regular and about normal, or extremely irregular both in force and time, and it may be frequent or slow. A very slow pulse with extreme low tension is most characteristic, but then it is the most rare.

The physical signs may be described as negative. Unless degeneration has attacked a heart already enlarged the size will be normal. If the fatty change is at all advanced impulse can neither be seen nor felt, or, if perceptible, it is only as a faint vibration. A heart in this condition is incapable either of giving a distinct push or of maintaining continuous pressure in the arteries. The sounds are weak, sometimes so

weak as to be almost inaudible; but except that the first is short, there is nothing abormal about them; the intervals, again, are usually normal. The very absence of murmur, or of conspicuous modification of the sounds or intervals, and of disturbance of the relation between the two sides of the heart, or of increase of dimensions, when symptoms of serious slackening of the circulation are present, and especially when there have been anginal, or syncopal, or apoplectic attacks, adds gravity to the case.

But a weak, short, unsustained pulse is common as a constitutional peculiarity, or may at any period of life be simply a result of general debility, and impulse and apex beat may be entirely absent, and the sounds may be short and weak. In young people there is no danger of such weakness being taken to be indicative of degeneration of the heart, but it may arouse anxiety after middle age, especially if there is also irregularity.

It is important to be able to distinguish between functional weakness of this kind and weakness arising from organic disease. Usually this is accomplished by making the patient walk briskly. A few steps will often be sufficient. If the heart is sound it rises to the occasion. The pulse, and beat, and sounds are all more distinct, and strong, and regular, whereas the fatty heart "goes to pieces," and the pulse becomes irregular and shorter than ever, or may even disappear.

Until the disease is far advanced the diagnosis of fatty degeneration of the heart is not easy, and is scarcely to be made without more than one opportunity of examination. When the diagnosis has once been made the prognosis, for the most part, can contemplate only one result; a fatal termination is merely a question of time and circumstance. Excluding cases in which death has been sudden without warning, the shortest period in my experience over which characteristic symptoms have extended, together with recognised physical signs, has been about six weeks; several patients have survived the diagnosis two years before justifying it by dying suddenly. But circumstance as well as time enters into the question; a slight effort, or a fall, a little hurry or excitement, too hearty a meal, an attack of flatulent indigestion or constipation, a chill, may hurry on the fatal termination and, on the other hand, judicious care may postpone it till the heart is completely worn out and comes to a standstill.

The question must be asked, Is fatty degeneration of the heart ever cured or arrested? If the granular disintegration which is produced by typhoid fever is to be included under the term, the answer must undoubtedly be Yes. The heart may ultimately regain structural soundness and functional vigour when during the fever the first sound has been completely lost and the impulse has been scarcely perceptible; and when degeneration has been the result of other forms of blood poisoning or deterioration it ought to be possible, and now and then to occur, that recovery of the heart should follow a return to a healthy state of the blood.

More than ten years ago I came to the conclusion that a gentleman still living, aged at that time about 55, was suffering from fatty degeneration of the heart. Spare in habit, strictly moderate in eating and drinking, regular in taking exercise, and a great pedestrian, he rapidly lost strength without recognisable cause, became breathless on very slight exertion, so that he could scarcely walk slowly one hundred

yards without actually stopping, either to get his breath, or on account of anginoid pain. On one occasion at least while sitting in his chair he became suddenly pale and unconscious, his head fell on his chest, and the jaw dropped. With this change in his health, the pulse and heart were extremely weak. He would never relinquish exercise, but continued to walk, however slowly and at whatever cost of pain and distress every day, exercising great self-command and measuring his strength very exactly. Little by little he gained ground, and he is now in fair health, but capable of very little in the way of work. It should be added that never at any time were his intellectual faculties at all affected.

This case may have been an instance of arrest and partial recovery.

There is not much to be said on the subject of treatment. Life should be made as easy as possible for the sufferer. He should have as much sun and fresh air as his strength and the weather will permit, but on no account must he be exposed to severe cold. Such exercise as he is capable of is good for him, but he should never incur angina or extreme breathlessness. His food should be simple and his meals strictly regular, and the bowels should be kept well open. The rectification of functional derangements will be of service and tonics may do good.

Disease of the Right Ventricle.

Little has been said about the right ventricle in the discussion of the different structural diseases of the heart for the reason that affections of this ventricle have been reserved for special consideration.

It has seemed to me that our ideas of the effects of disease of the right ventricle have been too much based upon a study of the symptoms which attend affections of this ventricle secondary to disease of the left ventricle or its valves, which therefore are really attributable to the original disease.

Undoubtedly the supervention of dilatation of the right ventricle and of reflux through the tricuspid orifice allows back pressure to be brought to bear upon the veins, but this only intensifies pre-existing symptoms and makes no change in their character.

We are perhaps justified in assuming that the venous back pressure, to which insufficiency of the tricuspid valve will give rise, will in some degree produce the same results, whatever the state of the left side of the heart, and whether or not the tricuspid regurgitation has been caused by obstruction in the pulmonary circulation; but the conditions are fundamentally different when the tricuspid reflux is primary. It is not impossible, for example, that a sound and strong left ventricle may come to the aid of the right ventricle, just as the right ventricle so constantly comes to the aid of the left, notwithstanding the great length of the systemic as compared with the pulmonic circuit and the weak blood pressure in the systemic veins. The pressure which will cause the blood to spurt for two or three feet in venesection might carry a current through the capillaries of the lungs aided by the respiratory movements and the valves of the pulmonary artery.

It has, moreover, seemed to me that weakness of the right ventricle—the left ventricle being in a normal condition—in some cases has given rise to symptoms due rather to inade-

quate supply of blood to the left side of the heart than to damming back of blood in the veins. I have, for example, met with several instances of primary tricuspid regurgitation, either as a constant condition or coming on under very slight provocation. When any effect of this has been traceable it has not been breathlessness on exertion, but tendency to syncope. Perhaps this is what we ought to expect, since the occurrence of tricuspid regurgitation in the breathlessness of violent exertion has been regarded as a safety valve action, since also shortness of breath results from mitral regurgitation. A difference of symptoms ought to attend mitral and tricuspid insufficiency, one giving rise to turgescence and high pressure in the pulmonary circulation, the other to deficient supply of blood and low pressure. If mitral disease produces pulmonary symptoms, tricuspid disease may well produce systemic symptoms.

In a few cases which have come under my notice, in which the right ventricle has appeared to be predominantly or almost exclusively affected by asthenia or degeneration, the effects have been similar to those of tricuspid regurgitation.

A master in a public school, who had been accustomed to vigorous exercise, received a severe blow on the chest. He had for some time great pain in the cardiac region, and when he walked he soon felt faint. When I saw him some time after the injury he had had slight but distinct syncopal attacks. On examination, no valvular disease was present, and the heart was of normal size. There was a fair apex push in the normal situation, and the left ventricle first sound and the aortic second sound were normal in character. No right ventricle impulse, however, could be detected, and the first sound and pulmonary second sound were very weak.

What had happened exactly cannot be stated, but from the contrast between the action and sounds of the two sides of the heart, it seemed as if the right ventricle had been in some way injured, and that its contractile energy was impaired.

The patient regained the power of taking exercise, and with this the sounds of the right ventricle became normal.

A patient, aged 72, who had never had a day's illness in his life, consulted me in 1879 complaining of failing vigour, giddiness on running to catch a bus, tendency to fall asleep in the day. He was constipated; the pulse, which was not more than 60, was sometimes tense, sometimes soft. The heart sounds generally were weak, the aortic second accentuated. In April, 1881, symptoms, which had previously been relieved, returned, and it was now found that over all parts of the right ventricle, and even over the pulmonary area, there was absolute silence. No impulse or apex beat could be detected, but at the apex the sounds were normal; the aortic second sound was accentuated. The pulse was 72, a little irregular, but fair in force and length. A month later he was much better. The pulse was 60, fair in strength and length; the left ventricle sounds were good, the right ventricle sounds faintly audible. From this time neither the right ventricle first nor the pulmonary second sound were ever at any time audible. The pulse varied considerably both in frequency and in tension, but it was usually well sustained, indicating considerable vigour of the left ventricle, and the left ventricle sounds were good. He had from time to time severe fainting attacks, which sometimes threatened to prove fatal. He was always worse when the bowels were not kept freely open. In

June, 1883, the pulse is described as large, full, and tense; the aortic second sound was accentuated at the apex and in the right second space; there were no right ventricle sounds. Towards the end of 1884, when not under my care, the bowels were allowed to get confined, and he fell into a condition of stupor, with incontinence of urine and fæces. He recovered from this condition after free purgation, and was again able to go about, though his mental faculties were impaired and he was childish; but in November, 1885, thrombosis of the left middle cerebral artery took place, giving rise to hemiplegia and aphasia, of which he died.

The entire absence of right ventricle sounds in this case was very remarkable, and I cannot doubt that there was really no action of this ventricle. As it seemed to me impossible that the pulmonary circulation could be maintained without its aid, I formed a very unfavourable prognosis; and when this was belied I watched the case with extreme care, first to make sure that my observation was not at fault, and next in order that I might arrive at some comprehension of the problem presented by the facts. The conclusion appeared to be unavoidable that the left ventricle was carrying on the circulation through the lungs; it was throughout capable of maintaining high tension in the arteries. The amount of blood passing through the pulmonary vessels under these conditions and reaching the left auricle would be easily influenced, and would vary greatly, and the fluctuating supply of blood to the left ventricle would account for the varying character of the pulse.

One of the most serious effects of weakness of the right ventricle is met with in disease of the mitral valve. When the mitral valve from thickening and shrinking of the curtains and tendinous cords becomes inefficient, the right ventricle is for a time the rampart by which the reflux of blood is arrested and a reinforcement to the crippled left ventricle. When we hear a systolic apex murmur telling of mitral regurgitation, the murmur itself gives no trustworthy information as to the amount of blood which is carried back into the auricle. We gather this mainly from the effects upon the right ventricle. The first of these is accentuation of the pulmonary second sound, indicating increased pressure in the pulmonary circulation, and following on this hypertrophy of the right ventricle, by means of which the obstruction to the passage of blood through the lungs is overcome. It is by the augmented strength of the right ventricle that the mitral leakage is neutralised and a working equilibrium established. The greater the regurgitation the greater the amount of hypertrophy required to compensate for it. The change in the right ventricle thus becomes, together with the accompanying dilatation of the left ventricle, a measure of the regurgitation.

When, therefore, we detect a mitral systolic murmur, we at once examine the right ventricle in order to gather from its condition information as to the amount of reflux which is not yielded by the murmur itself. This is specially the case when symptoms of failing compensation have set in; but if the right ventricle is in a state of degeneration or great weakness, these indications fail us altogether; while dropsy and other evidences of serious stasis and back pressure in the venous system are rapidly developed for lack of hypertrophy or compensatory effort on the part of the right ventricle.

Under these circumstances the prognosis is extremely grave. The right ventricle is unable to come to the aid of the left, the mechanism of compensation makes default, and the back pressure bears at once upon the venous system. The fulcrum for some of our most efficacious therapeutic measures is missing. We dare not open a vein however great the respiratory embarrassment and cyanosis; the effect of leeching over the liver is less certainly good, and a dose of calomel is not well borne. Recovery is rare, and twice it has happened in my experience that during apparent convalescence, when an unfavourable prognosis seemed to have been belied, the patient has died suddenly when beginning to walk about.

Similar conditions result from time to time from adhesion of the pericardium. The right ventricle suffers much more than the left from pericarditis. During the attack the muscular fibres immediately subjacent to the serous membrane, which are paralysed by the inflammation, form an appreciable proportion of the thin wall of this ventricle, which is thus weakened and prone to dilate; whereas, although the superficial layer of the muscular fibres of the left ventricle is similarly paralysed, they constitute a relatively unimportant part of the mass of the ventricular muscle. Again, the right ventricle is much more hampered by adhesion of the pericardium than the left, partly because its superficial area is relatively large, but chiefly because of the thinness of its walls; and when the adhesions are general, and especially if there is also adherence of the pericardium to the chest wall and diaphragm, efficient contraction of this ventricle must be impossible. Cases of this kind, then, are not uncommon. There is valvular disease, mitral or aortic. From the size of the heart, the position and character of the apex beat and impulse, the persistence of sounds in spite of the murmurs, there are grounds for concluding that the valvular lesion is not very great, but there is a premature development of symptoms. Under such circumstances, we may often confidently infer adhesion of the pericardium when it cannot be demonstrated by physical signs. The right ventricle may be obviously labouring, and the pulmonary second sound may be less definite and pronounced, which will tend to corroborate the conclusion arrived at.

The right ventricle is undoubtedly sometimes the cause of sudden death, and when the heart is embarrassed or stopped by pressure upwards of the diaphragm by a distended stomach or colon, it must be on the right ventricle that the pressure takes effect. This part of the heart rests upon the diaphragm, and will be directly compressed when it is pushed up. Probably it is the diastole which is mostly interfered with, and it would seem that the proper expansion and filling of the ventricle must be impossible when the pressure upon it is such that the heart is carried up bodily by the diaphragm, especially when the ventricle is dilated and over-distended. A melancholy illustration of this occurred in my experience in the case of an eminent artist. He was suffering from mitral stenosis and regurgitation, and had overthrown the compensation established by hypertrophy of the right ventricle by serious imprudence in the form of over-exertion and exposure undertaken to remedy the effects of overwork. He was suffering in an extreme degree from distension of the right side of the heart, with tricuspid regurgitation, and especially from sleeplessness and dyspnœa, so that his misery was insupport-

able, and life was despaired of. The application of leeches over the liver, which was enormously swollen, and the administration of calomel, at once gave him sleep, and by a repetition of the leeches and regular employment of mercurial aperients, with the usual heart tonics, he so far recovered as to be able to leave his room, and his convalescence seemed to be assured.

One morning after a hearty breakfast in bed, the nurse was about to wash his face and hands as usual, but he impatiently bade her give him the basin, and stand aside. He sat up in bed with the basin between his knees, and, when the time came for washing his face, bent forwards over it. The pressure upwards of a full stomach caused by this movement brought the weak right ventricle to a standstill, and the patient fell back dead.

One cannot help being reminded in relating this incident of the rough and ready, but effectual, way in which a man is brought to who faints after the frightful exertion of a boat race. He is seated on the ground, and his body is bent forcibly forwards, so that his head almost comes to the ground between his knees, or, if he has fallen forwards over his oar, it is done while he is in the boat. The *modus operandi* of the remedy is pressure on the distended right heart, and, when emetics are resorted to in bronchitis, which has gone on to the production of cyanosis, the good effect is due, not only to the emptying of the bronchial tubes, but to unloading of the right auricle and ventricle by compression in the act of vomiting.

Angina Pectoris.

While heart disease generally, of whatever kind, is remarkable for the almost entire freedom from pain—so that, when patients come complaining of pain in the cardiac region it is a presumption against the existence of any serious organic affection of the heart rather than an indication of any such change—there is one form of pain in and around the heart, angina pectoris, which is very definite and constant in its significance of disease and of danger.

In a characteristic attack of angina, there is intense pain in some part of the cardiac region—in the left breast, or behind the sternum, or across the chest, at its upper part usually, but occasionally lower down, with radiation down the left arm. Accompanying the pain is a sense of utter powerlessness and extreme fear and dread. The patient stands still, not daring to move or breathe, and feels as if he were in the act of dying. He will say afterwards that if the pain had lasted another moment he must have died. In no other condition is the physical agony of dying realised in anything like the same degree. The two elements of pain and sense of dying coexist in a true paroxysm of angina, and are almost equally characteristic.

The pain differs in character and situation and in intensity in different cases. Some sufferers will say it is indescribable—nothing in their previous experience suggests even a comparison; others speak of the pain as severe cramp in the heart, or as if the heart were gripped by an iron claw; while pain of a shooting neuralgic character, sometimes intermittent, sometimes persistent, seems to radiate from the chest to the left shoulder, the inner side of the arm, the forearm, and the ring and little fingers. Occasionally there is a sensation as of the wrist being grasped so tightly as to cause pain.

With the pain in the heart there may be pain down both arms or shooting up into the left side of the neck, very rarely in the right arm only. Occasionally the pain may be felt first in the arm and seem to travel up to the chest, or may come in the inner side of the arm as a kind of warning of an attack. Another description of the pain is that it feels as if the sternum were being crushed back to the spine, or, again, as if the whole chest were being held in a vice. In other cases the pain is compared to a bar of iron across the upper part of the chest; in others, again, to a ton weight upon the lower part of the chest. The ramifications of the cardiac plexus and its communications with other nerves make the radiation of pain in all the various directions enumerated comprehensible, and the nerve of Wrisberg has been specially instanced as explaining the pain in the left arm, but no explanation can be given why in one case the pain is felt in one part of the cardiac region, and has some particular character, and takes a given direction down one arm or both or through to the back, while in another case the seat, character, and extension of the pain are quite dif ferent. It is not a pressure effect on the plexus outside the heart, neither heart nor aorta being necessarily enlarged, and extreme fusiform dilatation of the arch of the aorta being common without confirmed pain; and there can be no stretching or mechanical irritation of the ramifications beneath the endocardium at all comparable to that which takes place in acute dilatation of the heart. It seems to me probable that the pain is really central, and that the radiation of irritation giving rise to its extension takes place in the spinal cord.

An interesting point is that at the end of a paroxysm there is usually flatulent eructation from the stomach. The attacks are therefore very commonly attributed to flatulence, and distension of the stomach by food or gases may undoubtedly be, and often actually is, an exciting cause, but more frequently the sensation as of wind on the stomach is only a part of the general commotion, and is due to communicated or sympathetic irritation of the gastric distribution of the vagus, the cardiac branches of which are primarily implicated. The escape of gas from the stomach is often a signal that the paroxysm is over rather than the means of bringing it to an end. Occasionally there is a vehement necessity to pass urine, although the bladder may at the time be empty.

The duration of the attacks is very varied; sometimes it can be reckoned in seconds. Most frequently, perhaps, a paroxysm will last a few minutes, but I have known a patient sit in the same position almost through an entire night, not venturing to make the slightest movement and scarcely seeming to breathe, while the perspiration rolled off his forehead and came through his clothes. According to my experience, it is when the attack comes on in the night, without provocation by exertion or exposure, that it is protracted. When it is started by exertion it generally ceases soon after the exertion is left off.

While it would not be justifiable to say that a patient was the subject of angina pectoris unless he had had one or more paroxysms of intense radiating pain, associated with a sense of immediately impending death, it must be admitted that attacks of true angina occur which fall short of the typical development. For example, when a patient has been taught prudence by one or more bad attacks, he may, by standing

still on the first warning, or by taking remedies, cut short the paroxysm, which will then have been represented only by the initial pain in the breast or arm without the mortal dread. It is possible, therefore, that before any characteristic attack has occurred, pains of a similar kind and intensity, disregarded by the patient or relieved by rubbing the chest or arm, may have the same significance as a fully-developed paroxysm.

Again, a patient who has had had attacks of true angina may cease to suffer pain, but may have attacks of what he calls faintness, in one of which he ultimately dies. These, which have lost their title to the name angina, have an equally serious significance.

The aspect of the patient is one of extreme anxiety or alarm. He is usually pale and often livid round the mouth, but it is said that sometimes the colour does not change. A cold perspiration usually bursts out on the forehead, and may be so copious as to drip off the face. The pulse, in the rare instances in which I have had the opportunity of examining it during a paroxysm, has been irregular, small, and weak. In some cases it has been reported to be very small from contraction or spasm of the arteries. In others, again, it has scarcely been affected at all.

Great importance attaches to the exciting cause of the paroxysms. In the first instance they are almost always brought on by exertion. The patient while walking perhaps more sharply than usual, or up-hill, or against a wind, is more or less suddenly arrested by pain in the chest, with a feeling as if the heart were about to stop and he to fall down dead. On standing still the pain gradually passes off, and he is able to resume his walk, but only feebly and gently. For a while the attacks only occur when provoked by exertion, but more and more easily as time goes on, and they tend to become more severe. They are more readily induced when a walk is taken, or any imprudent exertion such as stooping, pulling open a drawer, pushing up a window, is made soon after a meal, especially after breakfast. External cold, again, predisposes to an attack, and exercise, which can be taken with impunity in mild weather, brings on a paroxysm if the air is cold and damp. Attacks, again, may be brought on by indigestion or constipation, apparently either through reflex disturbance of the heart, or as a result of pressure from the distended stomach or colon carrying the diaphragm upwards and obstructing mechanically the action of the heart and the expansion of the lungs.

They are also liable to occur during the night, and may be induced in various ways. The contact of cold sheets may have this effect by causing contraction of the peripheral arterioles, and thus throwing increased work on the heart; or the upward pressure of the abdominal viscera, on assuming the horizontal position, may embarrass the heart. Not infrequently an attack comes on after sleep, when the vigour of the circulation has run down.

It is clear that the great exciting cause is a demand for increased effort on the part of the heart to which it is not equal, or, what is equivalent to this, interference with the movements of the heart by a dilated stomach and colon.

The conditions of the heart associated with angina pectoris are varied, but perhaps the most remarkable and significant point in the relations between heart disease and angina is that angina does not attend the chain of events through which stenosis or incompetence of the mitral valve proves fatal, and

is not among the symptoms which arise out of the valve lesion and its effects upon the heart. This fact was duly emphasised by Dr. Walshe, in his classical work on the heart, and no exception to it has occurred in my experience. I have, indeed, known instances in which, after attacks of angina have occurred at intervals for many months, mitral regurgitation has supervened with dilatation of the left ventricle, and concurrently with the establishment of so-called mitral symptoms—pressure in the pulmonary circulation, dilatation of the right side of the heart, and dropsy—the angina has ceased. In these particular circumstances Dr. George Balfour's view, that the giving way of the mitral valve may be an advantage to the sufferer from aortic disease, is justified.

Aortic stenosis may be attended with true angina, as may also, but less frequently, aortic incompetence and a combination of the two conditions of the aortic valve. In association with aortic valvular disease angina may be met with in early adult life, and may continue for many years without proving fatal. The sense of impending death is, however, not fully pronounced in many aortic cases.

Adherent pericardium appears in some cases to be a factor in the liability to anginoid attacks when it coexists with aortic valvular disease, but in my experience it has not given rise to angina when no other lesion was present.

Injury to the root of the aorta has been known to give rise to angina. I have had a case under observation for several years, in which a severe crush of the chest gave rise to a double aortic murmur and to distressing attacks of angina. For a time the attacks came on very frequently, even while the patient was kept in bed, and they continue to occur on very slight provocation, requiring frequent recourse to nitroglycerine, which the patient takes in considerable quantity. There has been scarcely any compensatory hypertrophy and dilatation in this patient, and he has never been able to work.

In aortitis there is usually angina, the attacks at first slight, increasing in intensity and duration, and coming on more frequently as the disease advances. The heart rapidly becomes weaker without notable enlargement, the impulse more feeble, the sounds weak and short. Both the angina and the weakness of the heart point to interference with the coronary circulation, and the orifices of the coronary arteries are found small and contracted by the swelling of the walls of the aorta.

A perfectly characteristic attack of angina has been described to me as having occurred in intermittent fever, and serious weakness of the heart was left behind for some time. Angina, again, has sometimes been an incident of diabetes, possibly from high arterial tension, which is commonly present in this disease late in life. Occasionally, however, a series of severe anginoid attacks, occurring at short intervals, has been followed by rapid heart failure and dropsy, suggesting that the angina was symptomatic of myocarditis.

Attacks of pain in the region of the heart of various kinds, some being true angina, are spoken of as gouty, sometimes, no doubt, in order to disguise the real nature of the paroxysms from a nervous patient to whom the knowledge might be dangerous or fatal.

In a very large proportion of the cases in which angina has proved fatal, the heart has been found, when examined after

death, to be in a more or less advanced stage of fatty degeneration, and in most of these again there has been disease of the coronary arteries, very commonly so far advanced as to have reached the stage of ossification or calcification. Sometimes these vessels can be dissected out from the auricular grooves as rigid calcareous tubes. The fatty change in the walls of the heart may be so far advanced that the fingers sink into its substance on very slight pressure, and that scarcely a trace of muscular fibres can be found on microscopic examination. On the other hand, the degeneration may be comparatively slight, being evident to the naked eye only as yellow striæ or patches in the ventricular walls and in the papillary muscles. The microscope, however, will show fat granules in those parts of the heart which to the eye and touch seem normal, as well as advanced fatty change when degeneration has given rise to yellow striæ.

In some cases the morbid condition found is fibrosis, general or local, apparently from myocarditis. Sometimes a distinct history of an attack of myocarditis is obtainable by questioning the patient. There may, however, be little or no recognisable change in the walls of the heart, especially when the first attack has proved fatal, or death has supervened after only a few paroxysms. It is probable, however, that in such cases something will be discovered on minute examination, perhaps the obliteration of a branch of a coronary artery by endarteritis, or its obstruction by an embolus or thrombus. Something certainly must have happened.

If we now try to bring to a focus the more important conclusions regarding angina pectoris :—First as to the condition of the heart during the attacks. This has been generally supposed to be one of spasm, but there are great difficulties in accepting this view, and probably ideas as to what is meant by spasm of the heart in the anginal paroxysm by those who have employed the term have been diverse and very often vague. If by spasm of the heart is understood tonic contraction or an unrelaxing systole, this is certainly not the condition present. The heart has never been found in this state after death, and in most cases is absolutely incapable of such contraction from the state of its walls. No pulse would be possible were the heart in a spasm of this kind, and the pulse, though small and often irregular, can usually be felt. It has, indeed, in some cases been apparently unaffected by the paroxysm.

But by spasm may be meant an irregular and partial contraction like cramp in voluntary muscles, or a fibrillar contraction, such as is sometimes induced by faradic currents in muscle under experiment. The late Dr. Matthews Duncan, in the last conversation I had the honour to hold with him, suggested that the state of the heart in angina pectoris might be like hour-glass contraction of the uterus. He had probably at that time experienced the pain. Views of this kind cannot be proved to be wrong, but objections might be raised, and, for my part, I have to admit that I have no clear and definite idea of the state of the heart during the paroxysm.

The central fact and essential significance of angina is that stress is put upon the heart, to which, for the moment, it is unequal.

One of the main causes of such stress is persistent resistance in the peripheral circulation, or, in other words, habitual high arterial tension, and we owe to Dr. Lauder Brunton the

knowledge that in many attacks of angina there is an aggravation of habitual high tension by a general contraction of the arterioles. But the habitual state of the arterial circulation may possibly be one of relaxed arterioles and capillaries and low tension, so that the heart has no abnormal resistance to overcome. Here sudden general arterial spasm would put the heart to greater stress than if the habitual tension were high, since the contrast between the work demanded would be greater.

When the paroxysms of angina can be distinctly traced to arterio-capillary resistance, or, when in the case of a patient subject to angina the usual condition of the circulation is one of high tension, the term "angina vasomotoria" may, perhaps, be appropriately employed. It is easy to imagine that stretching of the muscular fibres of the heart in the endeavour to overcome the resistance in the arteries might cause pain. But even when the vasomotor element is most potent, another factor must enter into the causation. Nothing is more common than high arterial tension, and it is met with in an extreme degree and produces fatal results without angina by ruining heart or arteries, or both, in hundreds of cases for one in which angina is present. Acute dilatation of one or both ventricles, again, in which stretching of the muscular fibres is obvious, frequently occurs without angina.

The importance, and even dominance, of this second factor becomes clear when the cases of advanced fatty degeneration are borne in mind, when the fibres must be incapable of producing anything like actual mechanical tension.

It has been assumed that the other element is neuralgic, and in a sense this is true, but not in the sense of a predisposing neurotic tendency. It must be remembered that angina is much more common in the male, which is the least neurotic sex.

Mechanical stretching and neuralgic predisposition being put out of the question, there remains the fact that the existence of the patient is threatened at the moment of the attack by arrest of the heart's action, and were it not for the warning given by the pain and for the cessation of exertion enforced by it, the subject of the particular condition of the heart would die. We must, it seems to me, assume that angina is one of the defensive arrangements by which the adjustment of internal reactions to external conditions is secured.

The prognosis of angina is beset with uncertainty. We can never tell when the next attack will come on, or whether it may not be the last. We are not, however, altogether without guidance, the elements of which will be an estimate of the relative predominance of the two chief factors in the production of the attack—whether inherent weakness of the heart wall, or obstruction in the circulation or other cause of embarrassment of the heart's action.

While the attacks only come on when provoked by exertion or excitement, or by flatulent indigestion (not, of course, taking the patient's word for the last-named cause), the hope may be entertained that by care in avoiding all known occasions they may be postponed indefinitely. The patient, for example, must not take exercise immediately after food, must never hurry or walk against a wind, and even on level ground must adapt his pace to his condition, and if compelled to go uphill must do so very gently and circumspectly. If, further,

there is habitual high tension in the pulse, this is at the same time evidence of obstruction in the arterioles and capillaries, which may be capable of mitigation and of some degree of vigour in the heart. So also will be accentuation of the aortic second sound, and still more any recognisable impulse or apex beat.

Angina, again, in connection with aortic valvular disease, may run a very protracted course. It is when the pulse is soft and the heart is normal in dimensions, with imperceptible impulse and weak sounds—when, in fact, the results of careful examination are negative—that the greatest uncertainty and danger exist. The occurrence of unprovoked attacks and of nocturnal angina will emphasise this conclusion.

Treatment.

The hygiene and general treatment for angina will be such as has already been described in speaking of dilatation and degeneration, which, indeed, mostly underly the angina, and the special precautions against the provocation of attacks have been sufficiently indicated.

Arsenic has long been held to exercise some influence preventive of the paroxysms, and my experience leads me to agree with this view. Phosphorus, which belongs to the same chemical and therapeutical series as arsenic, has a similar influence, which has, in some instances, seemed to be superior.

The great means of cutting short the attacks is that placed in our hands by Dr. Lauder Brunton—nitrite of amyl or nitroglycerine; and a patient subject to angina should never be without one or other of these remedies. They are, no doubt, greatly abused, and many persons, feeling secure of obtaining relief, neglect the important precautions by which the real disease, of which angina is only a symptom, may be held in check; and so, if they escape suffering, hasten on a fatal termination.