THE LUMLEIAN LECTURES ON 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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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 tendencies 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 murmurous, 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 sensa-

tions 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 contrary 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 dyspnoea came on and oedema 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 dyspnoea.

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 oedema. 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 dullness, was incredibly large, had not the results of percussion

been confirmed by the sounds being audible over the entire area of dullness, 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 dyspnoea. 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 dullness 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 oedema 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 belong 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 co-operates 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, 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.

GOULSTONIAN LECTURES ON LEAD POISONING IN ITS ACUTE AND CHRONIC MANIFESTATIONS.

Delivered at the Royal College of Physicians of London.

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[With Coloured Lithographic Plate.]

LECTURE II.

Analysis, Symptoms, and Physical Signs of Plumbism.—Anæmia: Blue Lines on Gums: Lines Caused by other Substances.—Colic: Nature and Cause of: Pulse during Attacks of Colic and in Intervals.—Difference in Radial Pulses.—Difference in Pupils.—Disordered Menstruation.—Liability of Female Lead Workers to Miscarry.—Forms of Lead Paralysis.—Is the Lesion Peripheral or Central?—Sensory Disturbances.—Lead Poisoning and Optic Neuritis: Two Forms met with.—Transient Amblyopia.—Loss of Eyesight for Twenty-one Weeks: Recovery.—Plumbism and Insanity.

Of all the symptoms and physical signs of lead poisoning, the one physical sign above all others which indicates that the system is becoming impregnated with lead and is suffering in consequence, is the development of anæmia, which becomes gradually confirmed, and is spoken of as saturnine cachexia. Almost from the first day of exposure to lead, hæmatisis is interfered with. I have never found in any lead workers the normal number of red blood cells. The average range varies from 2,500,000 to 4,000,000 in one centimetre. There is no marked increase in the number of white cells,

but the colouring matter falls as low as 45 or 50 per cent. of the normal. Not only does the face assume the appearance of anæmia, but the features become rounded and expressionless. The first effect of lead upon the system, in my opinion, is to disturb the functions concerned in the making of blood, and closely following upon this comes interference with nutrition. The bones have, in some of my cases, contained lead, and to this fact the anæmia may be partially attributed. Raimondi observed atrophy and degeneration of the bone marrow in lead poisoning.

The physical sign of plumbism, however, which is most looked for and generally met with is the presence of a blue or slate-coloured line on the gums, noticed by Burton, but previously described by Tanquerel. When present it is a valuable sign, but it may be absent; it generally is so when the teeth and gums are perfectly sound and the mouth is kept cleansed. And yet there may be colic and no blue line, or a girl may die from acute encephalopathy, with no blue line on the gums characteristic of the poison present from first to last. There are two blue lines on the gums that must be distinguished from each other: one is a delicate line just at the margin of the gums and teeth, but not involving the gum; it is a deposit in the interstices between the teeth, and is seen in workers who have been exposed to white-lead dust for a few hours. This is removed by rinsing the mouth and cleansing the teeth; it is simply a deposit of lead sulphide on, and not in, the gum. The other and characteristic line (see coloured lithograph, Fig. iv) is bluish-black, and occurs also at the margin of the gum, where it is not in complete apposition with the teeth. There the gum is frequently ulcerated and irregular in outline. The teeth are discoloured, and are covered with a brownish, soft, pulpy fur; the odour of the breath is disagreeable, and the patient complains of a metallic taste in the mouth. Where the teeth are absent there is no blue line. I have seen this line develop the day after a large dose of acetate of lead had been taken; the line persisted for a few days, and then disappeared. Out of several scores of cases examined by me, I find when the blue line is absent in one gum that it is more frequently found in the lower than the upper; if present in both gums, that it is usually more deeply developed in the lower. The deepest tint is observed underneath the outer incisors and canines. The fact of the line being absent where the teeth are perfectly white and the gums healthy suggests as a necessity for the development of this physical sign that there must be a broken surface of the gum; this alone, rather than bad teeth, although as the two are generally associated it is difficult to assign to each its proper share. That the gum, however, contributes most to this is shown by the fact that in girls who have been most exposed to lead dust for a few hours or a day or two, and in whom no blue line has been observable, I have on several occasions found the gums swollen and red, but not painful, as if they were the seat of irritation. It is in gums such as these that we subsequently meet with the blue line. An interesting point about blue line in the gums is that I have never found it in animals poisoned by lead. The blue line, however, though noticed most frequently, and as being most highly developed along the outer margin of the gum, is also seen running along the inside towards the molar teeth. The blue line may be present for months, and yet no other evidence of lead poisoning may be present save that of saturnine cachexia, general malnutrition, occasional constipation, and slight colic. It is difficult to say positively how long the blue line of plumbism will remain after removal of the individual from the influence of lead; in some of my own cases it has disappeared very quickly—in one within eight days—but usually from two to three weeks in its minor forms, and three months or longer in the severer cases. This is the limit which I expressed at a meeting of the Royal Medical and Chirurgical Society two years ago, in answer to a question of Dr. Stephen Mackenzie, who, with Gowers, regarded a year as the period during which the line may be present. Since that date I have carefully watched the disappearance of this line, and can confidently say that within three months all traces of it may have disappeared, whilst ulceration might still remain.

In addition to the blue line on the gum there are occasionally to be noticed discoloured patches of mucous membrane (see coloured lithograph, Fig. v), varying in size from a split pea to a shilling, inside the cheek—most frequently met with oppo-