found tubercle bacilli in small numbers in four cases of strumous disease of joints, in three cases of strumous glands, and in lupus. Schuchardt and Krause were the first to make an elaborate investigation on this subject, and they carefully examined 40 cases of surgical tuberculosis in patients of various ages with and without hereditary taint, the diseases to which they directed their attention affecting bones, joints, sheaths of tendons, skin (including lupus), walls of tuberculous abscesses, lymphatic glands, tuberculosis of tongue, testicle, uterus, and Fallopian tubes. Tubercle bacilli were found in all these parts, but in the great majority of instances they were few in number and only found after a long search. These authors thought that the small number of the organisms was due to the fact that these diseases are very chronic, and they supposed that the bacilli are most numerous in the early stages, although, as a matter of fact, they had no absolute evidence in support of this view. These observations have been repeated by several other observers with varying results. Thus Bouilly always succeeded in finding bacilli, though only after a long search. Mögling examined in all 53 specimens, of which 28 were from joints or bones; and he also obtained positive results, though the bacilli were, as a rule, present only in small numbers, nor were they specially limited to the giant cells, as had been asserted by earlier observers. Kanzler confirmed these statements. Schlegtendal, employing Ehrlich's method, examined the contents of 23 abscesses connected with bones and joints, and obtained 8 positive and 15 negative results, but he did not examine the walls of the abscesses. In all, including abscesses and sinuses unconnected with bone, 17 positive results were obtained with the pus of 40 unopened abscesses, and 9 with the discharge of 60 fistulæ or ulcers.

Müller examined from 30 to 35 cases of tuberculous disease of bones and joints, the specimens being chiefly obtained by excision, and he confirmed Schuchardt and Krause's statement, that with patience it is possible in most cases to find bacilli; in several cases, however, where recovery was taking place he looked for them in vain. His investigations do not add any support to the view that the bacilli are most numerous in the early stage of the disease, but he adds that in many preparations, whether bacilli were present or not, there were peculiar bodies, like drops of oil, which retained the red stain, and which he looks on as probably remnants of bacilli.

I have on repeated occasions examined the bones, synovial membrane, and pus from strumous joints, for tubercle bacilli, but as I have not always kept notes of the cases I cannot give the precise figures. The general result of my examinations has been that after sufficiently careful and prolonged search bacilli could always be found, but that in most cases they were extremely few in number. That the bacilli are present even when difficult to demonstrate by means of the microscope is evident from cultivation experiments, and from the results of inoculation of animals, but it is not easy to explain their apparent small numbers, and various theories have been advanced. As I have already mentioned, some authors suppose that the bacilli are most numerous at the commencement of the disease, and subsequently decrease in numbers. According to others they rapidly pass into the spore stage, and are then no longer recognisable by means of the microscope. My own belief is that we do not as yet possess a method by which we can stain all these bacilli at all stages of growth. I found very early in working with the tubercle bacillus that if we took sections in which large numbers of bacilli were present, and stained them in a simple watery solution of fuchsin, without any aniline oil, carbolic acid, or other similar body, a considerable number took up the stain and a few retained it even after a short immersion in the dilute nitric acid. Thus it was evident that the staining reactions of the bacilli differ even in the same specimen, and this is I think due to differences in the age and stage of growth of the organisms. At a certain age or stage of growth the sheath is probably more easily penetrated by the stain than at another age, and this corresponds with what we know with regard to other organisms. As showing that the external conditions cause differences in the staining reactions, I may recall the fact that the bacilli in smegma stain like tubercle bacilli, but after being cultivated on the ordinary culture media they lose this reaction. Similarly various bacteria,

when grown on solid culture media covered with a layer of butter, show the same reaction as tubercle bacilli in retaining the primary stain in spite of washing in nitric acid.

In sections of tuberculous synovial membrane stained by Ehrlich's method (bacilli red, tissues blue) I have not uncommonly found that while a few red stained bacilli were present there were also some which were blue, but which nevertheless presented the same microscopical appearance as the tubercle bacilli, and bore the same relations to the giant and epithelioid cells, and I have no doubt whatever that these are in reality tubercle bacilli in a different stage of development. The first case where this idea occurred to me was that of an undoubted tuberculous ulcer of the tongue, in which I failed to find any red stained bacilli, but, on the contrary, considerable numbers of faintly stained blue organisms resembling tubercle bacilli both in situation and in microscopical characters. Since that time I have paid great attention to this matter, and have seldom failed, in well stained sections from tuberculous joints, to find blue stained bacilli often in larger numbers than those stained red. I believe, therefore, that the apparently small number of tubercle bacilli in these local tuberculosis is often mainly due to the fact that they are growing slowly and with difficulty, and that their staining reactions differ at different periods of their existence. We require further research as to the methods of staining before we can conclude because we fail to find them by the ordinary methods of staining that they are therefore either absent or in the spore stage.

In connection with this matter it is interesting to note that I have found the same difficulty in finding tubercle bacilli in the synovial membrane of animals in which I have set up typical tuberculous joint disease by the injection of pure cultivations of tubercle bacilli, although they were easily demonstrated in the tubercles in the internal organs.

To sum up, we may take it that only few bacilli can as a rule be demonstrated in tuberculous joints and abscesses, but that they can usually be found in cases where the disease is progressing if the search be sufficiently careful and prolonged; and further, that our present methods of staining do not permit us to draw conclusions as to the numbers present.

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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LECTURE III.—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 sarcolemmal 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 note-

worthy 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 apoplecticform 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 apoplecticform 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 apoplecticform 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 abnormal 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 inadequate 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 turbulence 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 de-

tected, 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 feces. 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 accom-

panying 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 dyspnoea, so that his misery was insupportable, 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 different. 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 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 striae 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 striae.

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.

ON THE IMPORTANCE OF GONORRHOEA AS A CAUSE OF INFLAMMATION OF THE PELVIC ORGANS.¹

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WHEN, in 1872, Dr. Noeggerath for the first time published his views on this question under the title *Latent Gonorrhoea in the Female Sex*, so obviously exaggerated were some of his statements that, as he himself subsequently admitted, they "were not received very favourably by the medical press." Four years later, namely, in June, 1876, he returned to the subject by reading a paper before the American Gynecological Society, entitled, "Latent Gonorrhoea, especially with regard to its Influence on Fertility in Women." This was apparently a reproduction of his original views, and the same exaggeration of statement characterised it. It will be convenient to give here his conclusions as the formulated expression of his views. They are as follows:—

1. "Gonorrhoea in the male as well as in the female persists for life in certain sections of the organs of generation, notwithstanding its apparent cure in a great many instances.

2. "There is a form of gonorrhoea which may be called latent gonorrhoea in the male as well as in the female.

3. "Latent gonorrhoea in the male, as well as in the female, may infect a healthy person either with acute gonorrhoea or gleet.

4. "Latent gonorrhoea in the female, either the consequence of an acute gonorrhoeal invasion or not, if it pass from the latent into the apparent condition, manifests itself as acute, chronic, recurrent perimetritis or ovaritis, or as catarrh of certain sections of the genital organs.

5. "Latent gonorrhoea, in becoming apparent in the male, does so by attacks of gleet or epididymitis.

6. "About 90 per cent. of sterile women are married to husbands who have suffered from gonorrhoea either previous to or during married life."

These conclusions were at the time of the reading of the paper severely criticised by men who represented city and country practice. One gentleman used the naïve but pertinent argument that "he had ascertained in conversation with twenty different physicians who acknowledged having had gonorrhoea in early life, that in no single case had any such symptoms as had been referred to been developed in their wives, and all had had large families of children."

Before the publication of this second paper, the late Dr. Angus Macdonald reported some cases in support of Noeggerath's views. Regarding these I will only say that while Sinclair quotes them with full acceptance, Sanger refers to them as instances of wrong diagnosis.

After the detailed criticism of Noeggerath's second paper by Chadwick, the first to enter the field was the late Dr. Thorburn, of Manchester, who took up the challenge in a paper read before this Association in Manchester in August of the following year, on "Latent Gonorrhoea as an Impediment to Marriage." In contradiction to the conclusions arrived at by Noeggerath, Dr. Thorburn "appealed to the statistics of 81 private families carefully collected by himself. He showed that there had been 33 per cent. of male gonorrhoeic infections previous to marriage, 26 in all; and, taking all the cases of abortion, sterility, uterine and pelvic inflammations and living births that had occurred in these 81 families, he showed conclusively that there had been the merest fractional difference in their proportion between the previously and not previously infected classes. As regards inflammatory pelvic affections, the balance was fractional in favour of the non-gonorrhoeic."

Hitherto opinions on the subject of gonorrhoea were based on clinical investigation, but with Neisser's publication of his observations on the gonococcus (in 1879), which had been discovered ten years previously by Hallier, the question entered upon a new phase; the literature of the subject soon assumed large proportions, and the microscope usurped the place of clinical observation. The fortune of war fluctuated between

¹ Read in the Section of Obstetric Medicine and Gynecology at the Annual Meeting of the British Medical Association held at Birmingham July, 1890.