THE CROONIAN LECTURES

ON
THE PULSE.*Delivered at the Royal College of Physicians of London, March, 1887.*

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LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—My first lecture was occupied by certain general considerations with regard to the pulse, after which I began to describe the more important variations in frequency and abnormalities of rhythm. The next to be considered is the infrequent pulse.

Infrequent Pulse.—It has already been remarked that the effect upon the pulse of nearly all departures from a normal state of health is to increase its frequency, and the diseases are extremely few which reduce the pulse-rate in any marked degree. High tension in the aorta—induced experimentally in animals or man by pressure upon the abdominal aorta, or upon a number of the larger arteries—slows the pulse, and the high tension of renal disease has some effect in the same direction, but less than might have been anticipated. In jaundice the pulse falls in frequency, and in various affections of the nervous system this effect is observed; but, except under circumstances which will be discussed later, the reduction is not striking. A pulse is really infrequent when the number of beats is below 40 per minute. A pulse of 60 is not uncommon, of less than 60 rare. Persons are met with, however, in the enjoyment of vigorous health with a pulse of less than 40. I have known an athletic young man, tall, well built, and muscular, who distinguished himself at football and other violent games, whose pulse-rate was 36. Some years after I last saw him, I heard of his death while swimming; he was taken with "cramp," so-called, and sank. In this cramp it is the heart which is brought to a standstill by the resistance in the peripheral circulation, due to the combined effects of arterio-capillary contraction from contact with the cold water, and of pressure on the surface from immersion, and it is not unlikely that slow action of the heart would predispose to this result. Infrequency of the pulse to the degree under consideration is sometimes met with in fatty degeneration of the heart, and is enumerated among the indications of this disease, but it is far more commonly absent than present, even in an advanced stage of fatty change in the heart.

The infrequent pulse, however, is so closely associated with the bigeminal pulse, and with a peculiar modification of the rhythm of the heart, in which only every other beat reaches the wrist, that it will be well to consider these.

In the interesting variety of pulse rhythm called the "pulsus bigeminus," the beats come in couples, a strong beat being followed quickly by a somewhat weaker one, after which there is a pause. The second of the two beats may vary greatly in strength, being sometimes almost as distinct as the first, sometimes scarcely perceptible, and the interval between the first and second may vary somewhat in length.

On examining the heart its action is found to correspond; there are two unequal impulses, the second weaker than the first, and usually felt at a different point; and, on auscultation, the sounds associated with the second impulse are less loud, and of a lower pitch. If a murmur is present, usually a mitral systolic, it will be louder and higher pitched with the first, and may be inaudible with the second. I have sometimes heard a mitral murmur with one beat and a tricuspid with the other.

Closely allied to this condition is that in which there are two beats of the heart to one of the pulse, one beat being dropped; not that transitions are common, for, in the absence of valvular disease, the

pulse may number 40 one minute, the heart beating 80 in couples, and the next may be perfectly regular at 80. In the form of valvular disease, however, mitral stenosis, in which the pulsus bigeminus and the dropped beat are most frequently met with, the former may be a stage in the return towards a regular mode of action from the latter; and although (in the absence of valvular disease) a systematic bigeminal pulse does not succeed the dropping of a beat, yet an occasional bigeminal beat occurs when the weaker systole happens to raise the aortic valves, so that there is a clinical similarity which permits of their being considered together. Both, as has just been said, are met with in mitral stenosis, and especially when under treatment by digitalis. I have had several cases in which the pulsus bigeminus, or dropping of one beat, could be induced at will by administering this drug.

Taking the more advanced condition, when there are two beats of the heart to one of the pulse, a careful examination of the heart is most instructive. It is often difficult to resist the impression that the two ventricles are acting alternately. The impulse in the first of the two beats will give the sharp apex tap characteristic of an advanced stage of this form of heart-disease, the second will be felt only, or mainly, over the right ventricle. On auscultation, if there is regurgitation as well as stenosis, the systolic murmur and short sharp first sound will be heard at the apex with the first beat, but will be absent at the second, and the same will be the case with the aortic second sound at the base, it will be distinct with the first, absent at the second; while the sounds of the right side of the heart will be heard alone at the second beat with the tricuspid regurgitant murmur, if one is present, and will be comparatively weak with the first and stronger of the two beats; the pulmonary second sound, however, bearing witness that the right ventricle has contracted. When there is no regurgitation, the first beat will have the loud, short, sharp first sound at the apex and both sounds at the base, the second the low-pitched first sound of the right ventricle and a loud pulmonary second sound, but no aortic second sound. Usually the presystolic murmur will have disappeared before the coupled beats under consideration set in.

In the absence of mitral stenosis the double heart-beat with only one pulse is considered by Tripier, of Lyons, to be of very serious import, and to occur only in connection with epileptiform attacks. To quote his conclusion on this point, he says, "il semble donc qu'il n'y a pas de pouls lent avec épilepsie sans déviation du rythme cardiaque, de même qu'il n'y a pas de déviation du rythme cardiaque avec ralentissement du pouls sans épilepsie, ou sans l'action du digitale, tout au moins d'après les faits dont nous avons connaissance." Such has not been the case in my own experience, but before relating the cases which seem to me to be in opposition to Dr. Tripier's conclusion, I may add one more to the series which he has collected with so much industry and employed with so much ability, illustrating the connection upon which he insists between epileptiform attacks and the dropped beat and bigeminal pulse.

C. W., a carman, aged 47, but looking ten years older, was admitted into St. Mary's Hospital under my care, March 2nd, 1877. He had had rheumatic fever eight years before, and had suffered from occasional attacks of bronchitis. For eighteen months he had complained of palpitation after hard work, and he had had "fits," of which no exact description was obtained. After a recent attack of

bronchitis, from which he had recovered about three weeks, he had had for a fortnight an increasing sense of palpitation of the heart, with pain, which he called "working pains," across the upper part of the abdomen.

On admission he had an anxious look and the skin was clammy, the temperature 97°; the urine had a specific gravity of 1010, but

contained no albumen; the pulse was 51, large and strong, not quite regular either in force or frequency; at times there was an echo of the beat (pulsus bigeminus), see sphygmograms. The heart was of normal size, the apex beat was distinct and often followed at a brief interval by a second beat. At the apex a low-pitched systolic murmur beginning with an accent was heard; it was soon lost beyond the apex, but within it, was audible over the right ventricle and up to the pulmonary area. The aortic second sound was heard over the entire heart. As a rule the heart-beat was single, but from time to time there was a double beat. When he was made to walk rapidly the patient felt faint, but did not stagger, and secondary beats were more numerous. He was in the hospital two months, and improved much in strength, no fits occurring: the pulse however remaining at 28. At one time, when suffering more than usual from "working pains" all over the abdomen, the pulse was 59 and quite regular. His subsequent history is unknown.

A well-marked example of infrequent pulse with double heart-beat, but without epileptiform attacks or serious effects on the health, came under my notice in November, 1883, in a tall, strong, broad-chested man, aged 52 years, who had been engaged in exciting political agitation, and had suffered a severe domestic loss. He was complaining chiefly of continuous dreaming, and of depression and loss of energy, with heaviness and confusion of the head. Having found a pulse of 40, I wished to examine the chest, but he said this was quite unnecessary, since, whatever else might be amiss, the heart and lungs at any rate were all right, and he declared that he could walk up hill as well as ever, and even run. There was no impulse or apex beat, but on auscultation a normal first and second sound were heard all over the heart; followed, rather sooner than usual, by a first sound without a second, a weak pulmonary second sound, however, being audible when listened for with care. The first of the two beats only gave a pulse at the wrist, the second indeed not raising the aortic valves.

All the symptoms disappeared; but two years later, after another period of excitement and anxiety, he again consulted me, and with similar complaints he had a return of the peculiar action of the heart. The pulse was 34, quite regular. There was a diffused double cardiac

impulse without defined apex beat, one of them strong, giving a pulse, followed quickly by a weaker one which sent no wave to the wrist. The sounds were much as they have been described before, but the first sound of the second weaker beat was almost inaudible at the apex and distinct over the right ventricle. This patient again recovered, and is still making much noise in the world.

In the same year, 1881, I saw a medical man of about 55, who, when rather overtaxed by work, had run some distance on being called to an urgent case. This was followed by strange faint feelings, and an uneasy sensation about the heart. He was not otherwise out of health. He had discovered that his pulse was 40 instead of 70, and on examination this was found to be due to the heart-beating in couples—a strong beat with normal sounds which gave a pulse, followed quickly by a weaker beat with sounds which appeared to belong to the right ventricle only. This had been going on for ten days before I saw him, and continued for ten or fourteen days longer, coming and going towards the end of this time. Rest and change restored the equilibrium of the circulation, and this gentleman is in active practice at the present moment.

In November, 1883, I saw a gentleman, aged 53, who had enjoyed good health until the previous June, when he had an attack of congestion of the lungs, and mitral regurgitation was found to exist. He looked well, but complained of shortness of breath on exertion. The pulse was large, rather tense, perfectly regular at 72 beats a minute, except that from time to time the rate was 36, which was found to be due to the fact that, while this continued, the beats of the heart were in couples, one only of which reached the wrist. The apex-beat was well-defined and forcible, and near the normal situation. A long, smooth, high-pitched musical murmur was heard at

and to the left of the apex, and in the back; another murmur of lower pitch was audible along the right edge of the upper part of the sternum up to the sterno-clavicular articulation; the aortic sound was accentuated, the pulmonary masked by over-lapping lung. When the coupled beats were present no second sound followed the later of the two, so that the sounds ran 1 2 1—1 2 1; both the mitral and the aortic murmur were louder with the first beat than when the heart's action was regular, and absent, or scarcely audible, with the second; this second beat, however, gave a loud first sound, so that it was not abortive from mere weakness, but the absence of the murmurs and of the second sound was probably due to an imperfect diastole. A week later, after rest in bed, the physical signs were unchanged, except that, when a series of coupled beats occurred, which was as frequent as before, the second of the two beats was weaker and more precipitate.

This patient, after a Mediterranean trip, again came under my care in February, 1884, and was then observed to have Cheyne-Stokes respiration, which, however, did not prevent him from going to the City, until thrombosis of the deep tibial veins came on. He died from the effects of thrombosis in the right innominate vein. The peculiar heart-rhythm was never heard again; but the case is the more interesting from the occurrence of this peculiarity in the action of the heart, and later, of Cheyne-Stokes respiration, which Tripter considers to belong to the same order of phenomena.

The double heart-beat for each beat of the pulse was present again in the case of a lady seen in October, 1885. She had worked very hard in her father's parish, which involved much walking up-hill, from April to June, when she began to look ill, and was breathless—which, however, she did not notice herself. She then suddenly broke down, and had been resting up to the time when she consulted me. She looked well, and all the functions were natural, but she felt as if she could sleep continually. The pulse was infrequent, and two beats of the heart, one like an echo of the other, were felt and heard for every pulsation at the wrist. The aortic, as well as the pulmonary second sound, seemed to be audible in the second weaker beat, although no evidence of this systole having raised the aortic valves

is recognizable in the tracing. Under the excitement produced by the application of the sphygmograph the rhythm gradually became normal, not suddenly as is usual.

Another case has recently come under my notice in a gentleman, aged 63, who consulted me on account of faintness and flatulency. He had been nervous about his heart for thirty years or more, and had been afraid of exercise. He had neither over-work nor anxiety; but some months before he consulted me he had had a fall from his horse and had broken his collar-bone, since which he had not ridden. The pulse was at first 108, and regular, but from agitation, or from the effort of taking off his coat, it fell to 54, the number of heart-beats being exactly double, a strong and weak beat being coupled together with sounds belonging predominantly to the left and right ventricle respectively. This continued to the end of the interview, and a tracing of it is here given. I have learnt subsequently from

his regular medical attendant that this rhythm has been observed from time to time for years. In none of these cases had epileptiform or syncopal attacks been known to occur.

Still more recently I have seen, with Mr. Stanley Smith, an example, the only one I have met with, except, as I have said before, in mitral stenosis, in which the bigeminal pulse had followed the dropped beat. An old lady, aged 77, had been to Olympia on February 11th. She was ailing on the 12th and 13th, and prostrate on the 14th, complain-

ing of oppression in the chest and respiratory distress. She was found to have a pulse of only 35 to 40, instead of 76, and the double heart-beat. She improved under stimulant treatment, and when I saw her on the 17th had a pulse of 60 in coupled beats, which were about equal in strength and volume, and followed each other rather quickly, the pause being long enough for two more beats at the same rate. The rhythm was that of common time in music, 1, 2, 3, 4—1 and 2 being beats and 3 and 4 silent. No cardiac impulse could be felt, but the beats, as followed by means of the sounds, had the same rhythm as the pulse, and there was a systolic mitral murmur with each. A doubtful abortive systole seemed to be audible at times.

A single instance of the pulsus trigeminus may be given. The patient, a needlewoman, aged 41, employed in St. Mary's Hospital, had been in better circumstances before taking this situation, and after fifteen months in it was set to do laundry work. Lifting and carrying heavy baskets caused blood to well up into her mouth, but in spite of this and of feeling weak and languid, she persisted without complaining for a fortnight, when she was sent into the ward on September 14th, 1885. She looked languid, and had from time to time sharp pain in the cardiac region. The pulse was trigeminal, with two distinct and similar beats, and then an intermission, the third beat, however, being very faintly indicated by the sphygmograph. On auscultation of the heart, which was of normal size, a triple rhythm was observed; the first beat gave perfectly normal sounds, the second a good first sound but a reduplicated second sound at the base, the third beat had no second sound at all and the first was rather precipitate. A cardiogram, taken as well as the sphygmogram by my clerk, Mr. S. A. Tidey, shows the rhythm of the heart. As the

patient gained strength the cycle was extended to four and five beats while in bed, but reverted to the trigeminal type when she began to walk about, and it had this character when she returned to her duties as needlewoman.

It seems to me that the cases cited invalidate Tripiér's conclusion that the peculiar modification of the cardiac rhythm, in which every other beat is dropped, is always associated with and due to epilepsy, and usually of very serious import; nor can I accept the other conclusion, that a slow pulse with epileptiform attacks is always due to dropping of alternate beats. The decision is of importance, since upon it turns the question of whether, as Tripiér believes, it is the epilepsy which affects the cardiac rhythm, or whether, as I think, it is the slowing of the circulation which causes the convulsions. Before entering upon any discussion on the subject, however, I may bring evidence to show that there may be a slow pulse with syncope or epileptiform attacks which is not due to abortive heart-beats. I have myself seen a case of fatty degeneration of the heart attended with syncope attacks, which soon after proved fatal suddenly, in which the pulse was infrequent without any interposed feeble heart-beats being detected on careful examination. The patient, however, was seen only once in consultation. The notes of a hospital case, with numerous tracings, which I think would have corroborated this, are unfortunately missing.

In the *Lancet* for 1885, cases of slow pulse with epileptic seizures are related by Mr. F. St. George Mivart and Dr. A. T. Gibbings (p. 288). Mr. Mivart's patient was a man, aged 61, who had been subject to rapid and violent palpitation of the heart. His first fit was six years before he came under Mr. Mivart's care, and this and most subsequent ones came on while he was stooping. His pulse-rate was 24, and there is no record either of interposed weak beats or of abortive systoles. In Dr. Gibbings's case the patient, aged 65, had for some time had intermittent pulse of about 60. After an attack of bronchitis, he suffered from dyspnoea and faintness, and the pulse was found to be

only 44. Still later, he became subject to frequent epileptiform attacks, and the pulse had now fallen to 22, but was full and regular, the heart beats corresponding in time. It is noteworthy that the epileptiform attacks would come on when the patient sat up. The pulse continued to fall, and it reached 12 or 13, the attacks being now so frequent that he went out of one into another; the urine also became albuminous. With a subsequent return of the pulse to 31, the albumen disappeared, and he was so much better as to be able to do a little work. While apparently better, he fainted at stool, and died. Nothing abnormal was found either in the heart or in the nervous centres.

This patient was seen by Sir Andrew Clark and the late Dr. Moxon, and it is distinctly stated that the heart-sounds were normal. We may conclude, therefore, that there were no abortive beats. It is true that it is not expressly stated that they were absent, but in a case of such interest they could not have been overlooked.

The infrequent pulse of fatty heart may fairly be attributed to the structural degeneration of the organ; but an infrequent pulse, when the heart is sound, and especially if it is due to the occurrence of alternate weak beats, can scarcely be explained in any other way than by the interposition of the nervous system. This does not bring us much nearer a true comprehension of the phenomenon, and, as I have already stated, I do not accept the conclusion at which Tripiér has arrived that it is an effect of epilepsy. On the contrary, I look upon convulsive attacks, when they occur in connection with an infrequent pulse, as a result of cerebral anæmia, produced exactly in the same way as the convulsions after great hæmorrhage. In Mr. Mivart's case the convulsions came on when the patient stooped, in Dr. Gibbings's when he sat up, changes of attitude being sufficient, in the extremely feeble state of the circulation, to determine such an arrest of the blood-supply to the brain as would give rise to an attack. The late Dr. Moxon, it will be remembered, went so far as to suggest that the initial event in common epilepsy was a stoppage of the heart.

Another question is suggested by some of the cases of dropped heart-beat. In almost all, while the aortic second sound of the weaker beat which fails to reach the wrist is absent, the pulmonary sound is audible. In many the impulse and first sound of the right side of the heart are more distinct than those of the left, and occasionally there is such a difference in the situation of the impulse and in the character of the sounds as to suggest that the two sides of the heart are beating alternately; there may even be a mitral murmur with one beat, a tricuspid with the other. The extreme case is met with only in mitral stenosis under digitalis. Now while it must be taken as demonstrated that there is no such occurrence as an alternating action of the two ventricles, it is certain, from numerous observations, that in the beat which does not raise the aortic valves, the systole of the right ventricle is often forcible, and the pulmonary second sound is usually distinct. This last sign means, of course, that blood has been thrown into the pulmonary artery and not into the aorta, and as the amount of blood which passes through the right and left heart in a given time must be equal so long as the equilibrium between the pulmonary and systemic circulation is maintained, the propulsion of blood from the right ventricle only must be supplementary. In mitral stenosis it is easy to understand that such an extra contraction of the right ventricle might be useful in maintaining the pressure in the pulmonary circulation which is necessary in order to force the blood through the constricted orifice; or, regarding it in another aspect, the abortive contraction of the left ventricle is due to its being inadequately filled during one diastole so that a second is needed. No such necessity, however, is apparent when both valves and walls of the heart are normal.

Intermittent and Irregular Pulse.—With regard to the intermittent and irregular pulse I have nothing to say which will not be familiar to all my hearers. Intermission, occasional or habitual, of the pulse is compatible with health and vigour up to extreme old age, and, in the absence of symptoms, may practically be disregarded. The intermission of the pulse is associated with a hurried and imperfect beat of the heart, of which the first sound is sometimes sharp and following immediately upon the second of the preceding normal beat, at other times it is weak. A weak pulmonary second sound is usually audible. An intermittent pulse may, however, be among the signs of fatty degeneration of the heart, but it will not stand alone. In case of doubt the patient should be made to walk briskly for a minute or two, when the really weak heart will falter, while, if the heart is healthy, the intermission will usually disappear. It will often disappear also during the administration of chloroform.

Marked irregularity of pulse is most frequently met with in mitral regurgitations, next in dilatation of one or both ventricles, but it occurs independently of any disease of the heart. The fact that it is so commonly associated with incompetence of the mitral valve, while it

is rarely observed in other forms of valvular disease, except when the heart is failing, seems to indicate a mechanical rather than a nervous origin of the disturbed rhythm in mitral regurgitation, and it appears to me to be related with the varying pressure upon the heart in respiration. When the mitral valve is incompetent, and the blood can pass back into the auricle as well as forwards into the aorta, the proportion which is driven in one or other direction will be determined by the comparative degree of resistance. Now the dilated left auricle will be supported by a certain amount of pressure during expiration, which will tend to resist distension by the regurgitant blood, but at the end of expiration the conditions are suddenly reversed, and it will be exposed to the negative pressure or suction action which fills the chest with air, so that the reflux is favoured. The sum total of the resistance to the ventricular systole is thus diminished at this moment, and the systole is consequently very rapid, at the same time that less of the blood goes forward into the aorta. At the end of inspiration opposite forces come into play, and the repetition every two or three beats of these disturbing influences quite accounts for the irregularity of the heart's action. In an early stage the irregularity can be seen to occur coincidentally with the end of inspiration and expiration. It is the incompetence of the mitral valve which exposes the ventricle to the respiratory pressure variations, but when, from any affection of the lungs, such as bronchitis and emphysema, the difference between the pressure on inspiration and expiration is greatly augmented, we find the action of the heart disturbed in like manner, and careful watching will show that the chief irregularities coincide with the reversal of the action of the chest.

Irregularity of pulse of nervous origin is illustrated by the disturbance of the cardiac rhythm in dyspepsia and by tobacco; but it may be present in an extreme degree independently of any recognisable influence, and may be habitual. There need be no affection of the general health or impairment of vigour or endurance. In one of the worst cases I have ever seen, the patient, who was for some time under my observation, was, long after the age of 60, in the habit of addressing public meetings. The trace exhibited was taken recently

upon a gentleman, now aged 70, who consulted me twenty years ago on account of irregular action of the heart, and has had it ever since. I am unable to fix any prognostic value on irregularity of the pulse as such, and when it occurs in connection with dilatation of the heart, the prognosis is determined by other considerations.

I now proceed to the consideration of variations of tension, and I shall first speak briefly of low tension.

Tension in the pulse, as has been already stated in the first lecture, depends primarily on the freedom of the flow through the capillary network; and it is low when the capillaries and arterioles are relaxed, allowing the blood to pass readily. As the capacity of the arterial system increases towards the periphery, the branches at each division being collectively larger than the trunk from which they spring, and as the capillary channels are larger still, it is conceivable that the blood might escape as fast as it could be injected by the heart. The actual realisation of this possibility is incompatible with life, as the functional activity of the nerve-centres is dependent upon a continuous flow of blood through them, and under the circumstances supposed it would be intermittent. The resistance, however, in the arterioles and capillaries may be so slight that the blood has still a pulsatile movement when it reaches the veins. Such venous pulsation in the veins of the back of the hand can often be demonstrated in aortic regurgitation, but here it is not so much the diminished resistance in the capillaries as the exaggeration of the pulsatile variations of pressure in the arteries which brings it about. In order to see pulsation in the veins, however, the capillaries must be relaxed by putting the hand in hot water, or there must be pyrexial relaxation of the peripheral vessels, which often renders it very evident. Occasionally venous pulsation is met with when there is no heart-disease or any other cause than abolition of the normal resistance in the capillaries. I witnessed it in a gentleman who was gradually sinking from the effects of alcohol without any of the usual alcoholic disease of liver and kidneys. He took less and less food, and came to live on alcohol, and finally had slight pyrexia and occasional attacks of

hæmoptysis. The arteries were large, thin, and soft; and for some time pulsation in the veins of the dorsum of the hand could be rendered visible at any time by dropping the wrist so as to allow the veins to fill.

The characteristic mark of low tension in the pulse is the complete subsidence of all blood-pressure in the artery between the beats. It therefore allows itself to be flattened under the finger in the intervals of the pulsation, and this causes the impact of the pulsation to appear sudden. Further, as we have seen, it must be short. Variations are observed according to the size of the artery and the action of the heart. The artery must be relaxed. When the artery is large and the heart is acting forcibly, we shall have a large, short, strong pulse—the full and bounding pulse of old writers; and dicrotism will usually be conspicuous. When, with a large relaxed artery, the heart is weak, the pulse will be large, short, and weak, still with dicrotism. We cannot have a contracted artery and powerful heart's action with low tension. A feebly acting heart and small artery—the artery being small simply from want of distending force—will give the small, short, weak pulse described as thready; and the absence of a distinct wave of pressure gives it an undulatory character.

One of the most constant effects of pyrexia is to relax the arterioles, and the pulse characteristic of all forms of fever is one of low tension. The pulses above given are, in effect, if also frequent, the pulse of pyrexia in different forms and stages. On these, consequently, I do not dwell.

A low-tension pulse in the absence of pyrexia, unless it is a physiological peculiarity of the individual (and it may be remarked in passing that a pulse of a very low tension may run in families), is indicative of debility. It may go so far as to suggest the existence of fatty degeneration of the heart, except that the pulse of cardiac weakness is usually frequent, while that of fatty decay is more commonly under the average rate. The heart-sounds also are weak and short. The symptoms associated with a pulse of low tension are extremely varied, and they are, for the most part, not the result of the weak pulse, but concomitant effects of a common cause. Many of them are equally common when the pulse-tension is high, and the question is not what symptoms arise out of low or high pressure in the arteries, but, given certain symptoms, what is the state of arterial tension, since this will be an important guide in the treatment. When the tension is low, purgatives are badly borne, and mercurial aperients give rise to great depression. Sometimes, indeed, the subjects of low arterial tension feel much better when the bowels are constipated, and exhausted after each action.

There are no doubt cases in which the chief cause of deficient pressure in the arteries is want of functional vigour in the heart, and it would be of service to be able to distinguish such cases from those in which the primary cause is absence or resistance in the arteries and capillaries, but this is not easy. A low state of arterial tension is not in itself a source of injury to the heart or vessels. It has an important bearing on affections of the nervous system; but questions relating to this are reserved for the third lecture.

High Tension.—I have already said so much elsewhere on the subject of high tension in the pulse, that I shall here condense my remarks into much smaller compass than would otherwise be demanded by the relative importance of the subject. The cause is obstruction to the free flow of blood through the minute arterioles and capillaries to the veins. There is some difference of opinion whether the exact seat of this obstruction is in the arterioles or capillaries. It is probably not in all cases the same. In high arterial tension of purely nervous origin, as, for example, in hysteria, in cerebral and spinal disease, in rigor and the cold stage of fever, there can scarcely be any question that the cause is tightening up of the minute arterioles; but in tension of blood origin, as in renal disease, gout, and allied conditions, it seems to me that the primary obstruction is in the capillaries, contraction of the arterioles being no doubt associated with this, but secondary to it, either by way of continuity, or as a defensive counter-action against the distending internal pressure set up by the obstacle beyond.

I may briefly enumerate some of the reasons which have led me to this conclusion. The most important are derived from an examination of the minute arterioles in the brain, after death resulting from contracted granular disease of the kidney. When there have been uræmic convulsions, capillary hæmorrhages are almost always present in various parts of the cortex, so much so that our late distinguished colleague, Dr. Mahomed, attributed the convulsions to capillary rupture. If the capillaries were protected by contraction of the arterioles, such rupture could scarcely occur. Again, these arterioles are themselves at certain points dilated into miliary aneurysms, and liable to rupture, not where they are given off from larger

branches, but just where they break up into the capillary network, which shows the obstruction to have been beyond, and not in, the arterioles, while not only are the perivascular spaces round them large, as if from distension, but a ring of the surrounding brain substance is pale, as if from pressure. Another reason is that, although the medium sized arteries, such as the radial, and the small arteries, such as the digital, are usually contracted and small in high tension; this is not always the case, and it seems improbable that, in vessels anatomically continuous and physiologically correlated, there should be contraction in the minute arterioles without a corresponding condition of the arteries leading to them. In further support of this view, I may cite the important experiments of Drs. Ringer and Sainsbury on the action of the digitalis group of remedies, related in vol. lxvii of the *Medico-Chirurgical Transactions*, 1884. It is there shown that when all reflex contraction of the arterioles is rendered impossible by destruction of the brain and spinal cord, digitalis and other substances having similar properties give rise to marked diminution of the flow through the arterio-capillary channels. The action is thus proved to be direct upon the contractile structures of the capillaries and arteries, and not indirect through the vaso-motor nerves.

I must, at the risk of tedious repetition, again describe the high-tension pulse. Its distinguishing characteristic is that the artery is full between the beats. So full is it that it can be rolled under the finger as if it were a tendon, and followed like a solid cord some distance up the forearm, and, when the tension is well marked, no pulsation may be felt while this is being done, unless decided pressure is made by the fingers. Above the wrist, when the artery rests upon the muscles of the forearm and not on the radius, it may be difficult to detect pulsation. Not unfrequently, especially when the skin is thin, the artery can be seen to form a distinct projection, but no pulsation is visible in it, unless it is thrown into a curve, when this will be seen to be accentuated at each beat. The artery is usually small from contraction of its muscular coats, unless it has been dilated by protracted distension, when it may have much beyond the average diameter.

When pressure is applied in order to bring out the character of the beat, it is found that this does not strike the finger sharply but rather lifts it powerfully; it persists for an appreciable time, and then gradually subsides with little or no dirotic echo. It is the long pulse, "pulsus tardus." The size of the vessel will make some difference in respect of all these points; the distinctive features are best marked when it is small; when it is large, both the incidence and the subsidence of the pressure will seem to be more abrupt, especially if the artery is thoroughly flattened by the fingers. With moderate compression, the beat feels less sudden, and has the gradual rise, sustained duration, and slow decline which is characteristic of high tension.

Another character of the high-tension pulse comes out when its strength or force is tested by attempting to arrest the beat. While the fingers rest lightly on the vessel, little pulsation is felt; but, as pressure is employed, the more forcible does it seem to be; and, when the artery is greatly contracted, it is really difficult to distinguish the wave.

While the prime cause of high tension in the pulse is obstruction in the peripheral circulation, the actual pressure in the arteries is maintained by the heart. Usually the increased difficulty in driving on the blood is met and counteracted by increase of the contractile energy of the heart; but it is clearly possible that, while there is obstruction in the arterioles and capillaries, the heart may not rise to the occasion. We shall then have, in feeling the pulse, an artery full between the beats and capable of being rolled under the finger, while at the same time it is compressible. This is, in fact, very common, and we require a term which may be applied to such a condition. I have been in the habit of speaking of it as virtual tension. Now, as the artery allows itself to be flattened, the pulse wave which lifts it into the cylindrical form appears to be sudden, and the pressure, after lasting a certain time, seems to drop suddenly. The artery may be small or large, and the pulse varies accordingly.

We meet with the conditions which give rise to virtual tension at the two extremes of kidney disease. At the outset of acute renal dropsy, while there is obstruction in the arterioles and capillaries, the heart is rendered weak by the pyrexia and low diet, and there is more or less tendency to it throughout the attack. Again, as the heart becomes worn out in the course of chronic granular disease of the kidney, it ceases to be capable of maintaining the high tension which is characteristic of this disease, and virtual tension takes its place. It is at this period that reduplication of the first sound is so well marked. The same course of events is observed after protracted high tension from whatever cause; and as this is the origin of dilated left ventricle, the

pulse of virtual tension, in its best marked form, comes to be the characteristic pulse of dilatation of the heart. At the same time that it is expanding the left ventricle, the continued obstruction in the arterioles and capillaries is overcoming the contractile power of the medium-sized arteries, the lumen of which becomes enlarged, while their coats are thickened.

The causes of high tension are now so generally recognised that I shall do little more than enumerate them. They are as follows: 1. Heredity. This is illustrated not only by the every-day experience that paralysis and heart-disease run in families irrespective of the habits and mode of life of different members, which may differ greatly, but by the fact that high pulse-tension and functional derangements arising out of it may be present in childhood and all through life in every member and in successive generations of many families. 2. Kidney-disease of all kinds, except such as is attended with suppuration, but the most marked tension is met with in the contracted granular kidney. 3. Gout and allied conditions, including almost the entire range of affections attributed by Murchison to functional derangement of the liver. 4. Lead-poisoning, with or without renal disease or gout. 5. Anæmia.

Here I may make a few remarks. It is not easy to understand how anæmia can give rise to high tension in the pulse. One would have expected the watery blood to pass readily through the capillaries, and the *vis a tergo* applied by the heart to be deficient, but it is a matter of daily observation that the artery is full between the beats, and that the pulse, if more abrupt than in renal disease, is long. It has been conjectured that the defective oxygen-carrying power of the corpuscles may cause oxidation to be imperfect and so lead to the formation of substances which are not readily eliminated, and which provoke resistance in the arterioles and capillaries, but the instability of the tension is suggestive of the intervention of nervous influences, and there are other peculiarities which lend support to the hypothesis that the arterial contraction and powerful heart-action may be vaso-motor phenomena. However this may be, there are rare and exceptional cases of anæmia with extremely low tension, and it has been an object of attention with me to make out whether any constant difference of another kind attends this difference in the state of the circulation. In particular it seemed probable that there might be some influence on the production of hæmic murmurs which might throw light on their causation, especially from the point of view of Marey's theory, which refers all cardiac murmurs to low pressure beyond the point at which the vibration is set up. Up to the present, however, I have been unable to recognise any distinction between anæmia with high tension and anæmia with low tension, except the difference in the circulatory conditions.

To return to the enumeration of causes of high tension, we have, in addition to those already named: 6. Pregnancy. 7. Constipation. 8. Plethora—an overlaid state of the entire vascular system. 9. Chronic bronchitis and emphysema.

With regard to the effects of high tension, again, I shall be almost equally brief, for the reason that I have little to add to what I have before written and said on the subject. The effects upon the heart are easily understood, the high blood-pressure in the arterial system calls for increased contractile energy on the part of the ventricle, in order that it may overcome the pressure and carry its contents into the aorta. If the resistance with which the heart has to contend is augmented gradually it will respond by hypertrophy; if, however, the increase in the resistance is sudden, the heart will be unable to complete its systole and expel the whole of its contained blood. Dilatation is then the result, and it will take place all the more readily if the nutrition of the heart is interfered with. Dilatation of the left ventricle may result from high arterial tension at almost any period of life, and is met with under a great variety of circumstances. It is very common in anæmic girls, and is frequently attended with mitral regurgitation, which is curable, as has been well shown by Dr. George Balfour. Some contributory exciting cause will be traceable. I have known it to be due in several instances to hunting, in others to lawn-tennis, in others to much walking up hill. In one case recovery only took place when the family of the patient left a house in an elevated situation which made every walk terminate in a climb. While curable if taken in time, the dilatation of anæmia often establishes permanent heart-disease. Acute dilatation, which is of far more frequent occurrence than has been supposed, has for its predisposing cause peripheral resistance in the circulation and high pressure in the arteries. It may take place in the course of acute disease or as a sequel, if the pyrexia has not neutralised the habitual tension by relaxing the arterioles. It may again be produced either by too violent or by too protracted exertion, and this at almost any time of life at which high tension is common. The gradual dilatation which occurs

with varying degrees of antecedent hypertrophy in chronic gout or renal disease, or as a result of chronic resistance to the circulation, from whatever cause, need only be mentioned. Valvular changes, mitral and aortic, and the dilatation of the aorta, are also so familiarly referred to high tension that they require no further notice, and the same may be said of cerebral hæmorrhage.

One word may be permitted on the injurious effects of peripheral resistance in aortic or mitral regurgitation. In both these valvular lesions high tension, or rather the tendency to this condition, is disguised by the effects of the regurgitation, and is liable to be overlooked. Actual tension, as manifested by sustained fullness of the artery between the beats, is indeed impossible in aortic regurgitation, since the fulcrum is wanting, and it is not easily recognised in mitral regurgitation. The subjects of such lesions, however, may be gouty or may have renal disease, and the peripheral resistance incident to these affections will not be suspended. The injurious tendency of this resistance will be manifest at once. The greater the force required to drive the blood through the capillaries, the greater will be the force with which it will be driven back through a leak in the mitral valve, and the more powerful the dilating effect on the left ventricle when the protection of the aortic valve is lost. The prognostic bearing of such an influence in the early stages of these valvular diseases makes it important that it should be recognised, especially in the young, when treatment may do much to avert the evil consequences. In aortic insufficiency an idea may be obtained by careful examination of the pulse when the hand is well below the level of the shoulder and of the heart; it should not hang down, as it is then difficult to distinguish between the persistent fullness due to the weight of the column of blood and that due to the obstruction in the capillaries. In mitral regurgitation it will be sufficient to bear the question in mind and to examine the pulse in different positions of the hand. Information may also be obtained from an examination of the pulse of other members of the family.

In connection with the subject of renal disease and gout and high pulse-tension, I must find an opportunity for calling attention to the fact that cases are met with in which the tension is altogether absent, and this not because the heart is weak or failing, but because there is even less than the normal resistance in the arterioles and capillaries. The prognosis when such is the case is extremely unfavourable. It is in connection with the chronic, large, white kidney that we most commonly see a weak, soft, low-tension pulse with albuminuria. The amount of albumen is often very great; it varies from day to day, often without traceable cause, but often, again, it is increased by exertion or mental depression, or constipation, and it is usually greater at night than in the morning. Any development of tension in the pulse is attended with diminution of the albumen. The patient may not have the least appearance of kidney-disease, and dropsy may be entirely absent. Tension, again, may fail to supervene in acute albuminuria, and, when such is the case, a fatal result is, according to my experience, always to be expected. I have come, indeed, to take the development of high arterial tension as one of the chief guides in prognosticating the course and issue of this affection; if it makes its appearance early, and becomes well marked, the prognosis is favourable; there appears to be an increased liability to uræmic convulsions, but the patients bear the treatment required by this and other complications, and usually recover. If the tension is slowly developed, and especially if it is fitful and inconstant, the recovery is slow and uncertain, while, as has been said before, if the pulse remains soft, short, and weak throughout, the prognosis is bad. Dropsy is usually considerable, and there is often an unusual amount of blood in the urine, as well as casts.

In contracted granular disease of the kidney, absence of tension is very rare, and I have no record of a well marked instance in which the diagnosis has been verified by *post-mortem* examination. In the following case I had no doubt as to the nature of the case during life. The patient, aged about 55, was under my observation from time to time, from June, 1880, up to his death in September, 1881. He had suffered from gout and eczema, and, when I first saw him, albumen had just been discovered in his urine, which had a specific gravity of 1011. He had always lived freely, and could not be prevented from taking a considerable amount of whisky. His eczema was removed and his gout relieved, and he usually looked and felt well; the quantity of urine was not far from normal, and the specific gravity usually varied from 1017 to 1021, while a mere trace of albumen was present; and it was only the knowledge that he had albuminuria which induced him to present himself. He was well nourished, had a good colour, and a largish, soft, short pulse; there was no evidence of degeneration in the walls of the vessels, nor any change in the heart.

When the shooting began in September, 1881, he went out as

usual, but was more easily tired, and took extra whisky to enable him to go on. On the 14th and 15th he said queer things at dinner. On the 15th, Mr. Martin, of Crawley, saw him, and found him languid and sleepy. After a jalap powder he was better, but in the night of the 17th he was seized with violent convulsions. I saw him with Mr. Martin at 1 p.m. on the 18th, when he was lying on his left side, the left side of his face boring into the pillow, his eyes directed violently to the left, the left arm bent across the chest, with the hand up at the right shoulder, pulling violently at the night-shirt, which was torn. He was smacking his lips loudly. These movements came on in paroxysms, but with such short intervals as to be almost continuous. The left leg was very little affected. Sensation was very greatly impaired in both arm and leg, and might be said to be lost. He was conscious and intelligent, and in the intervals between the paroxysms could direct his eyes to the right, and could give either hand. The convulsions continued, except when he slept, and in the course of the 19th deglutition failed, the breathing becoming deep and loud, and death by coma supervened.

I have entered into these particulars to show that the convulsions were not of the ordinary uræmic character. They were clearly due to cortical irritation, but this is not the place to discuss the character of the lesion, and no *post-mortem* examination could be obtained.

The case is quoted to illustrate the position that absence of arterial tension, where such tension is a part of the disease, is not a favourable sign. The patient had not the look of kidney-disease; he was well nourished, and had a good colour; the heart gave no evidence of disease, and the arteries were soft and flexible; the high tension which predisposes to rupture of cerebral arteries was absent; there was nothing, in fact, suggestive of impending danger.

Functional Effects.—Without recognisable organic change in the heart or arteries, or where the subsequent history shows that such changes as were present were not of themselves the cause, high tension may give rise to the most severe angina pectoris. High tension, again, may produce effects in the way of breathlessness and sudden powerlessness which simulate the effects of the most advanced heart-disease.

Intermittent functional albuminuria, when not traceable to imperfect assimilation of food, is, so far as it has come under my observation, associated with high pulse-tension, and is most readily amenable to treatment which reduces arterial pressure; and the cases of this affection described by Dr. Clement Dukes, of Rugby, and shown in his admirable papers on the subject to be common among boys and adolescents, appear to belong to the same class as mine. When this liability to the appearance of albumen in the urine on exertion or slight exposure is met with in children, I have always found a strongly neurotic family history as well as hereditary high pulse-tension. When it does not come on till the approach of adolescence, there may be no family neurosis, but I have never failed to recognise tension, and to trace its effects in other members of the family.

Treatment.—Although high arterial tension is fraught with so many evil contingencies, it is not to be looked upon as *hostis humani generis*, and attacked whenever found. I have already stated, and exemplified by cases, that, supposing renal disease or gout to exist, the prognosis is unfavourable if the pulse-tension is not high. This is equivalent to a declaration that under such circumstances the high pressure in the circulation serves some useful end. We do not, therefore, treat tension as such, unless it is actually causing suffering, or doing present or prospective harm. For the most part, indeed, the object of our treatment is to remove the condition on which unduly high tension depends.

Among the measures for the reduction of tension, one of the most potent is bleeding. This is usually practised only in extreme cases, and when it is employed it is as a remedy for the high tension itself, or for some of its more serious and dangerous effects, such as convulsions. When cerebral hæmorrhage has occurred it is too late to bleed, but were high tension in the pulse more closely studied, and were less attention paid to the public prejudice against venesection, it would be anticipated and prevented in hundreds of cases every year. Physiological experiment seems to throw doubt on the production of a rapid diminution of pressure in the arteries by blood-letting, but there can be no question as to the effect in the mind of anyone who has practised venesection in a case of uræmic convulsions. The results are more than can be easily attributed simply to the withdrawal of so much blood. In acute nephritis, when bleeding has been necessary because of convulsions, I have more than once seen unaccountably rapid disappearance of the albumen from the urine, and complete recovery. The statement made by trustworthy observers, when blood-letting was common, that at times patients could not be got under the influence of mercury until they had been bled, and that then sali-

vation was induced at once, is evidence of some profound change in the system; possibly a more rapid absorption, resulting from the diminution of intra-vascular pressure, may enter into the explanation, and the taking up into the blood of fluid effused into the cerebral meninges or ventricles.

The spring- and fall-bleeding of our forefathers must have been a great relief to many of those upon whom it was practised, and in some I have no doubt it prolonged life. I have had under my care from time to time an old lady, the only clue to whose age is the fact that her father, who was an historical character, died in 1803, who had been bled fifty times, first for puerperal convulsions and for their prevention afterwards. The punctures at the bend of the elbow had been so numerous that there was no room for more, and the later bleeding had been done from a vein in the foot. I do not think the old lady would have been living now but for the bleeding. When I last saw her, about two years since, she was managing the family of a deceased daughter. All her children are dead, the last, a daughter, who was the only one I saw, died from chronic Bright's disease, her grandchildren by another daughter, who died in childhood, are the subjects of high tension.

Apart from bleeding, tension can be lowered by reducing either the force of the heart or the resistance in the periphery. The former method can be called for only in some emergency, such as threatened cerebral apoplexy, and, if employed, will lay the heart open to dilatation, unless the obstruction with which it is contending is also removed. The latter is the chief indication to be followed, and it can be met in two ways, either by drugs which relax the minute arterioles, such as amyl-nitrite, nitro-glycerine, and the nitrites, or by modifying the blood-condition which provokes the obstruction in the arterio-capillary circulation. The remedies which relax the arterioles are invaluable where either there is active arterial spasm, which is of itself a cause of pain and danger, as in angina pectoris, or when the contraction of the arterioles, without being abnormal in degree, is contributing to overpower the heart. They may thus be of service in high tension of renal origin, and are said to have contributed to the cure of diseases of the kidneys. I have administered nitro-glycerine repeatedly in different forms of renal disease, but without any other effect than temporary relief of some of the symptoms, and improvement in the patient's appearance from the admission of more blood to the pallid surface. I am quite prepared to believe, however, that continued diversion of blood from the diseased organ, which will be one effect of the general relaxation of the arteries, may favour their return to a normal condition.

The blood-condition which gives rise to tension, I have already stated to be, in my opinion, the presence in it of some form of impurity, and, for the most part, nitrogenised waste. The treatment demanded, then, is simply elimination. When the cause of this is disease of the kidneys or liver, the primary object of the treatment will be its cure, but, side by side with this, and often indeed forming a part of it, will be required measures for the removal of the matters which are retained in the blood in consequence of the suspension of the functions of these organs.

Any or all of the eliminating organs and surfaces may be made use of. In certain cases free perspiration is useful. While, however, the altered distribution of the blood which is brought about by the means taken to induce perspiration, such as the Turkish bath, and its determination to the surface of the body is often of service, the cutaneous excretion is chiefly water, and no great amount of organic matter is carried away, except in kidney disease, when an efflorescence of urea may be found on the surface after a copious perspiration. According to my experience, elimination by the skin is rather an accessory than a primary means of keeping down tension.

The kidneys are capable of carrying off a much larger amount of nitrogenised waste than the skin, and diuretics may in this way contribute to the relief of tension. But many diuretic remedies give rise to tension, and apparently produce their effects by raising the blood-pressure. The elimination desired for the purpose we are now considering is best obtained by the diuretics which promote tissue-change and oxidation—by copious draughts of pure water night and morning, which will wash out the tissues and carry off any soluble matters, and by potash and lithia, which are solvents of uric acid. It is not altogether a matter of indifference what salts of these alkalies are used; the citrates can be taken in larger doses, and they will render the urine alkaline quite as easily as the carbonates, if this is desired, but the carbonates appear to have more metabolic influence, and liquor potassæ has a still greater effect of this kind.

The elimination which has the greatest effect on arterial tension is that from the liver and bowels; and of the aperients which are available for the production of purgation, the most efficacious for this par-

ticular purpose are such as contain mercury. This is a conclusion based entirely on experience and observation. Whether as regards relief of symptoms associated with high tension, or as regards the effect on the pulse, a moderate and, indeed, scarcely recognisable aperient action by a pill containing mercury in some form or other is much more efficacious than free purgation by other means, and pills of this kind can be taken once or twice a week for years in suitable cases with advantage.

The pulse in heart-disease has been so thoroughly studied, and is so frequently and fully described, that I shall treat it very briefly, and I only refer to it at all in order to insist on the necessity of taking it into account in both the diagnosis and prognosis of affections of the heart. It is not every systolic murmur at the right second space that indicates obstruction of the aortic orifice, and if we must admit that a systolic apex murmur is proof of some mitral reflux, the murmur tells us absolutely nothing of its amount. The pulse must be brought in to interpret the murmur and fix its significance.

Aortic Stenosis.—The effect of narrowing of the aortic orifice is obviously to intercept the pressure of the ventricular systole and retard the current, and, as the blood cannot gain in pressure or speed, the pulse must be gradual and long. But, besides this, the entire arterial system seems to contract down upon the diminished blood-stream, and the pulse is also small. One does not see altogether why, with normal resistance in front, the arteries should not be kept at the usual size by the pressure from the heart, because this pressure takes effect more gradually, but the small diameter of the radial and other arteries is a constant phenomenon, and the pulse of aortic stenosis is, when well marked, one of the most peculiar met with: small, full between the beats, and destitute of anything like impact.

Aortic Regurgitation.—The pulse of aortic regurgitation is too well known to require any description. I would only call attention to the element of size in the arteries as important, to the necessity of ascertaining carefully whether the column of blood is at all sustained in the radial when the arm is held up, or whether the blood drops out of the vessel immediately the ventricular pressure is withdrawn, and to the extraordinary influence of pyrexia in exaggerating the collapsing character of the pulse. The slightest departure from regularity in the collapsing pulse is ominous; when this takes place, it is due at first to an occasional faltering beat, and it rarely reaches any considerable degree.

Mitral Regurgitation.—The irregular pulse of mitral incompetence has already been alluded to. It is by no means constantly met with when a mitral regurgitant murmur is audible; and while it indicates, when present, decided impairment of the functional efficiency of the valves, with secondary effects arising out of this, I am not prepared to assert that irregularity of pulse is an absolutely necessary consequence of even large regurgitation, nor does it always indicate a dangerous failure of compensation.

Mitral Stenosis.—In mitral stenosis the pulse is regular, small, and full between the beats. The contracted state of the arteries is probably due to the arterial system accommodating itself to the restricted output of the ventricle. The regularity of the pulse in this form of valvular affection has been called in question, but in the absence of associated mitral regurgitation, and of considerable secondary regurgitation at the tricuspid, it is maintained up to a very late stage of the disease. When, from pulmonary complications or failing adjustment, regularity is lost, the first deviation is in strength of pulse, not in rhythm, then beats fail to reach the wrist, and the pulse is irregular, while the heart is acting regularly. Finally, the irregularity may defy description, and there is apparently no correspondence between the pulse and the heart, and, when both are felt simultaneously, they might belong to a different person.

The pulse of hypertrophy of the heart is that of its cause, and no more need be said of it.

The pulse of dilatation has already been described under the head of virtual tension. A number of sphygmograms from cases of dilatation of the left ventricle are given, but they convey a very inadequate idea of the characters of the pulse as recognised by the finger. Scarcely even in aortic regurgitation can the state of the heart be foretold from the pulse more definitely than in dilatation. The first trace is from a

case in which acute dilatation with hæmoptysis had occurred from over-exertion; a mitral systolic murmur remained; the artery was not large.

In the case from which the second was taken there were breathlessness, difficulty of lying down, and enlargement of the liver, all of which were relieved by treatment. The patient whose pulse is represented by the third, was liable to stagger and even fall, on rising from a semi-recumbent position, with momentary loss of consciousness, and, on slight exertion, he would suddenly lose all power. The

artery was always full between the beats, notwithstanding the appearance of the trace. He improved under treatment, could walk eight miles, was more cheerful, and could lie on his side; the pulse gave

the second trace. Later, over-exertion aggravated the dilatation, and thrombosis of the left middle cerebral artery occurred, the case ending fatally.

THE LUMLEIAN LECTURES

ON

THE PATHOLOGY OF INTRA-UTERINE DEATH.

*Delivered at the Royal College of Physicians of London,
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LECTURE II.—DISEASES OF FETAL APPENDAGES.

I PROCEED now to speak of the causes of intra-uterine death which are associated with faulty conditions of the foetal envelopes and foetal appendages, and I shall take those first which properly belong to the earlier part of pregnancy.

In the earlier stages of its development, the ovum is in shape like an egg, and consists of the central embryo, with its surrounding envelopes. The outer one, or decidua, which lies next to the uterine wall, is the earliest in its formation, for it appears in the uterine cavity before the descent of the fecundated ovule from the Fallopian tube. It is now well known to be the product of the uterus itself, and to consist of the mucous membrane lining the interior of the womb, thickened and modified in such a way as to fulfil the necessary requirements of a foetal envelope. A membrane in all respects like this, both in external appearance and minute structure, is sometimes expelled from the unimpregnated uterus at the catamenial periods in cases of dysmenorrhœa. It is then produced by an over-activity of formative elements, which occurs as the result of some reflex irritation, probably in the ovary, and which simulates the commencement of pregnancy. The expulsion of such membranes, therefore, does not always mean that an abortion has taken place, for they may be formed and extruded

without impregnation; but, in a considerable number of cases in which these membranes are thrown off by married women, they are the result of conception. This is inferred from their frequent recurrence so long as sexual relations are continued, and their cessation when coitus is intermitted. Besides, the catamenial period is often missed once, at least, before expulsion, in women who at other times are perfectly regular. When entire, these membranes generally appear as membranous sacs, of triangular form, corresponding to the shape of the uterine cavity, and the walls of the sac are thick or thin according to the amount of organised material of which they are composed. The outer surface is commonly rough, and, if the preparation is placed in water, shreds of flocculi float out in the fluid. When laid open, it is a common thing to find the cavity quite empty. The inner surface is smooth, and marked everywhere with the apertures of hypertrophic glandular follicles, but there is no embryo. The ovule has either missed getting into the decidua cavity, or it has been endowed with so small an amount of vitality that it has undergone solution before it could plant itself in the decidua tissue. The reflex stimulus which has come from the ovary or tube, in whichever locality fecundation took place, has been enough to evoke the formation of the decidua in the womb; but the ovule being wanting, development is arrested, no decidua reflexa is formed, and the useless decidua vera is eventually thrown off as a foreign body. Here the fault in procreating power, as distinguished from generative power, may be either with the male or female; and, if the woman is otherwise healthy, it is quite as likely to be some defect in the fertilising fluid as faulty ovulation on the part of the female. These deciduæ of early pregnancy are in many cases found covered with coagulated blood, and the cavity may be filled with clot, soft or semi-fluid, if recently deposited there—firm and partially decolourised, if some time has elapsed since its extravasation. But these membranes are not always expelled in their entirety. More frequently they are thrown off in detached portions or shreds at variable intervals, and mixed with the fluid or coagulated blood which escapes at the same time. It is no uncommon occurrence for a patient, or even the medical man, to infer that the supposition of pregnancy has been erroneous because no substantial mass has been expelled—the discharges being taken for an over-profuse and perhaps deferred period. Eigenbrodt and Hegar,¹ in a memoir "On Apoplectic Destruction of the Decidua," found that in some cases the presence of the elements of the decidua could only be ascertained by the aid of the microscope. This accords with my own observation.

Akin to this is another defect of development, which has been described by Matthews Duncan² and copied into the various textbooks. This consists, according to the author named, in a defective development of the decidua round the ovum. The egg then comes in contact only with a limited surface of the mucous membranes which corresponds to the serotina, and as it is not supported, as usual, by growth of the decidua round it, it becomes, as it were, pediculated, and is easily detached from its limited adhesion. This is probably one of many faults of development occurring in early gestation. In one specimen I examined, which had been expelled at an abortion, the ovule had evidently failed to get inside the decidua cavity, and began its development on its outer rough surface, attaching itself, as it does in extra-uterine pregnancy, to the surrounding structures. The surrounding structures were partly the outer surface of the decidua, and partly the wall of the uterus, and for three-fourths of the circumference of the ovum the clear transparent chorion was quite uncovered, its villi there being either atrophied or never developed. The decidua seems in some instances to be detached from the uterine wall, and to lie so loosely in the uterine cavity, more especially if atrophied or imperfectly developed, that one can imagine the ovule as it enters the uterus from the Fallopian tube dropping between the decidua and the uterine wall.

We get some sort of idea, now that our knowledge is more precise concerning the formation of the decidua, and of the behaviour of the ovule when it enters the uterine cavity, of the way in which placenta prævia and other variations in the site of the placenta are produced. Ordinarily the thickened decidua fills pretty fully the cavity of the uterus, and the ovule is received into the cushion so formed, and is sustained in the upper part of the uterus. When, however, the decidua is less luxuriant in growth, its cavity will be larger, and the fertilised ovule may then drop down to the lowest part of its cavity, and become implanted there. Dr. Robert Lee³ has recorded the appearances of a very early placenta prævia, which bears out this idea, and such an anatomical fault favours the occurrence of abortion, although we know by experience that those cases may go on to the later periods of pregnancy.

¹ *Monatsschr. für Geburtsh.*, Bd. xx, 1868.

² *Researches in Obstet.*

³ *Theory and Practice of Midwifery.*