Operation.—On consultation with Dr. Rendell of this city we discussed the advisability of hysterectomy and other measures, and decided to try the operation recommended by Dr. Pryor, of New York—namely, of opening the posterior cul de sac and the application of antiseptic dressings.¹ At 4 P.M., under chloroform, with Dr. Rendell's assistance, I curetted the uterus, irrigated with salt solution, and then made a broad incision into Douglas's pouch, applied enough 20 per cent. iodoform gauze to completely fill all parts posterior to the uterus, and packed the uterus with the same. On putting the patient back to bed we applied the icebag over the lower abdomen, injected a pint of normal saline solution into the cellular tissue of the back, and kept up the saline injections into the colon every eight hours, using about a quart or more at each injection.

After-Progress.—By midnight the temperature was 104.4°; the pulse was still 128, but fuller; the urine was very plentiful, and gave the reaction for iodine by the chloroform-nitric-acid test. On January 2nd the temperature had fallen to 101° F. (the pulse to 108), but rose again by the evening. The patient was feeling and looking better; the headache, however, was still severe, and only relieved by the application of ice to the head. She was given gr. $\frac{1}{4}$ of strychnine every three hours, and 3j of brandy every hour. The dressing was changed each day until the third day, when it was left off. From the accompanying chart it will be seen that while an evening rise occurred each day, there was a gradual fall to the normal, which was reached on the seventh day after operation. Further convalescence was uninterrupted nor has any evil effect followed. All through the summer she worked hard and enjoyed good health.

The happy termination of the case, which at one time looked so hopeless, causes me to put it on record. In the treatment of such cases Dr. Pryor lays great stress upon the preparation of the patient by colon injections of salt solution—beforehand, if possible—and in grave cases by intravenous infusion. This is necessary to secure the rapid elimination of the iodine. "Curettage is performed as much for the purpose of creating a raw surface, which can rapidly absorb iodine, as to remove infected tissue." What effect iodine in the blood has upon septicaemia he does not attempt to explain, but regards its action as something more than the mere sterilisation of the pelvis.

REFERENCE.

¹ American Journal of Obstetrics, vol. xl.

A NOTE ON THE SEPARATION OF THE PLACENTA IN THE THIRD STAGE OF LABOUR.

By J. D. SLIGHT, M.A., M.D., CH.B. EDIN.,
Portobello.

In a recent paper read before the Edinburgh Obstetrical Society,¹ Dr. Berry Hart drew attention to the fact that the wall of the third stage uterus, examined *in situ*, is thinner at the placental site than it is elsewhere above the retraction ring. If I remember correctly, the wall where the placenta is attached is about $\frac{1}{4}$ inch in thickness, while the rest of the wall is four times thicker. This fact was used by Dr. Hart to cast doubt upon the view that separation of the placenta is effected by retraction. I venture to suggest that in it there is to be found the key to the mechanism of the third stage.

The hypothesis which I put forward is that in a "pain" the thin part of the wall (that is, the placental site) is stretched by the thicker part, and the connection between placenta and uterus is so torn through.

Suppose a contraction in the third stage to come on gradually and to increase in force by degrees, so that more and more of the uterine energy comes into action. At first the contents (which completely fill the cavity) are squeezed into smaller bulk. Soon, however, further compression of them demands from the thin part of the uterus greater energy than it is able to supply. But contraction is not yet ineffective; compression of the uterine contents can still be accomplished by the power of the thick part, provided that the thin part does not yield. If, as I submit, this yielding must, and does, take place, then, instead of forcing the contents to occupy still less space, the additional energy of the thick muscular tissue merely stretches the thin.

If the placental site is stretched, the separation of the placenta from it is unavoidable. The placenta is firmly grasped by the encircling uterus, and it is quite unable to move or rearrange its parts in the densely packed cavity, for the stretching brings no relief of pressure, and the periphery of the placenta cannot move with the corresponding part of the uterine wall, since the space into which it would seek to pass is already occupied.

According to this theory, separation will in most cases occur first at the lower pole because of the proximity of the placental site to the thin lower uterine segment. I would suggest that, in the cases in which separation does not begin here, there intervenes a considerable area of thick wall between the lower segment and the placental site.

The placenta having been thus freed is next expelled, and in this process of expulsion the membranes are stripped from the wall of the uterus.

Much might be adduced in support of my contention, but I have thought it best to be satisfied in the first instance with a brief exposition of the theory.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, January 19th, 1901, p. 152. ² Barbour, Edinburgh Medical Journal, September, 1884.

CASE OF ECTOPIC GESTATION: OPERATION: RECOVERY.

By H. H. LLOYD PATCH, L.R.C.P., M.R.C.S., L.S.A.,
Hertford.

THE following case shows what can be done in the cottage of a labouring man, provided cleanliness and nursing can be obtained.

History.

E. S., aged 26, who had always been strong, was married in 1893; her first child was born in May, 1894. She had three children subsequently, and had never had any miscarriage. About the middle of September, 1900, I treated her for an attack of pelvic peritonitis, and at that time there was an offensive bloodstained vaginal discharge.

Condition on Examination.

On November 26th, 1900, she was taken ill with acute pain in the stomach, more severe over the pubic region; this was accompanied by sickness and profuse menstruation. On December 4th I saw her, and found the following condition: Face pale and acutely drawn, as though the patient was in much pain; eyes sunken; pulse rapid and small, 136 to the minute; respirations quickened; temperature 102.8° F.; abdominal muscles contracted and rigid, much pain caused by palpation; percussion note was dull over the abdomen below the umbilicus, and absolute dullness over a portion extending from pubes below to within an inch and a half below the umbilicus, above, and about 3½ inches in breadth; below this dull area a tumour could be felt. The vomit had a stercoraceous odour and was most offensive; and there was retention of urine necessitating the use of the catheter. On vaginal examination the uterus was found lower than normal and somewhat fixed; the cervix was not enlarged to any extent and there was a bulging inwards of the posterior and left lateral walls of the vagina above, and obliteration of the left fornix. The vaginal discharge was very offensive and blood-stained. Having in view ectopic gestation I decided to operate.

Operation.

On December 6th under chloroform I made an incision in the median line of the abdomen 3 inches in length, the lower extremity ending one inch above the pubes. On the general peritoneal cavity being opened a tumour the size of a foetal head at full term presented itself, extending from the pelvis to within an inch of the umbilicus. The abdominal opening I extended upwards 1 inch. The tumour was covered in patches with recent lymph; its wall was composed of peritoneum, a quarter of an inch thick, and the tumour was found to be fluctuating. Its upper two-thirds was fairly free, but the lower portion was matted by adhesions, so that it was impossible to form a pedicle. The uterus was felt on the right side adherent to the lower portion of the tumour, its body being slightly enlarged. I then opened the tumour, and found inside the thickened wall a thin membrane which bulged into the wound when pressure was made on the tumour. I incised the membrane, being careful to protect the general peritoneal cavity. The contents were composed of fluid resembling liquor amnii, and when the tumour was relaxed I could feel a small body floating in the remaining portion of fluid. I then tried to separate

the membrane from the thickened walls, but it was in parts intimately adherent. I decided to connect the opening in the tumour to the abdominal wall opening by sutures, and this was done after thoroughly sponging out the peritoneal cavity, my idea being to allow the contents of the tumour to be passed through the abdominal opening.

The small body felt at the operation was expelled on the twenty-fourth day after operation; it turned out to be a small ovum about the size of a broad bean. Little by little the membranes were expelled through the wound, and as portions were protruded, being offensive, I cut them away, but the major piece was discharged in a mass on the twenty-fifth day after operation. The temperature was 102.8° F. on the third day as the discharge became offensive, but, by using plenty of antiseptics, it gradually came down, and disappeared entirely when the membranes were expelled. The patient made a good recovery.

In conclusion, I have to thank Dr. Boucher, of Hertford, for kindly administering the anæsthetic, and our district nurse, who acted as my assistant at the operation and has helped materially since.

THE LETTSOMIAN LECTURES

ON

DISEASES AND DISORDERS OF THE HEART AND ARTERIES IN MIDDLE AND ADVANCED LIFE.*

Delivered before the Medical Society of London.

By J. MITCHELL BRUCE, M.A., LL.D., M.D., F.R.C.P.,
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LECTURE III.

PROGNOSIS AND TREATMENT.

ETIOLOGY AS A FACTOR IN PROGNOSIS.

Now, prognosis and treatment, to be rational and useful, have to be based on as full and as correct a diagnosis as knowledge permits. The present disposition is to fall short of this—to rest content with an incomplete diagnosis. We say that the patient's "heart is dilated," that he has "arterial degeneration," that there is "fatty degeneration." But you will remember that we have found that cardiac dilatation may be present in every kind of cardio-vascular degeneration; that the arteries are naturally enlarged and thickened after middle life; and that we refused to call these changes morbid. Clearly, therefore, a purely anatomical diagnosis of this sort is insufficient. If you are asked what the prognosis is of fatty degeneration of the heart, you answer that you must first be told whether syphilitic or gouty disease of the coronary arteries, or strain, or alcoholism, or phosphorus poisoning, or anæmia is the cause of it. When you are planning the treatment of dilatation of the heart, you first determine whether the dilatation is a result of the stretching of a sound heart by overfilling during muscular effort, or of the insufficient emptying of failing chambers with degenerated and feeble walls. Obviously what we ought to determine in these instances and in every instance is the origin of the disease. The ultimate diagnosis to be reached for practical purposes is the etiological diagnosis.

Is this possible? Does our knowledge of the nature, characters, and course of these cardio-vascular affections enable us to say after investigating a case what the kind of the pathological change is that constitutes the disease, or in what respect the physiological mechanisms are disordered? Can the cause of senile degeneration of the heart and arteries be determined in each instance? How is the practitioner to proceed to do so? What method might be followed with advantage in making a complete diagnosis of heart disease in older subjects?

A man of 60 consults us about his heart. He says that it has caused him a good deal of anxiety lately. More specifically he describes a sense of oppression behind the sternum as often as he exerts himself, and palpitation with consciousness of irregular cardiac action when he goes to bed. We inquire for other familiar cardiac symptoms, such as pain,

angina, fluttering, faintness, giddiness, and a sense of impending death. We find that one or more are present occasionally, and that they have increased in number and degree during the last few months or years. Perhaps cough, nocturnal orthopnoea, and dropsy may be beginning to give trouble.

The next part of the inquiry relates to the patient's previous history from childhood upwards. Which of the acute diseases has he had? Acute rheumatism, chorea, scarlet fever, typhoid, diphtheria, and influenza must be mentioned individually, and in women the nature of any puerperal disease from which they may have suffered. Gout, irregular gout, gravel, eczema, sick headache, asthma, must be inquired after with the same minuteness, and so must syphilis. We next hear an account of any accident which the patient may have met with, such as a blow or fall from a horse or a carriage. This brings us naturally to question him about his occupation and modes of relaxation and amusements—whether active or sedentary, regular or irregular, their characters otherwise, and their direct effects, including strain. More difficult to elicit is a correct account of the patient's habits in respect of food, stimulants and tobacco, and his manner of life generally. As I said in my first lecture, this is an inquiry which the family practitioner has an opportunity to carry out much more successfully than the hospital physician or consultant. The family practitioner has known for years of his cardiac patient's work and worries—it may be of his large eating, of his secret drinking, of the history of syphilis in earlier years. It is always well also to inquire after a family history of gout, rheumatism, and heart disease. A list of questions like this sounds far more formidable than it is in reality. A few minutes suffice to arrive at the truth. We already have a pretty fair notion what we have to deal with—whether strain, gout, syphilis, tobacco, an old rheumatic lesion, or a combination of two or more of these.

THE INTERPRETATION OF MURMURS.

We next proceed to physical examination, beginning with the pulse and arteries, and passing on to the heart and associated structures. The characters of the precordial impulse—particularly the seat of the apex beat and the strength of the impulse—are closely—I might almost say laboriously—investigated. We must never yield to the temptation to disregard weakness or absence of the impulse. Like many other negative signs it is apt to be overlooked. Then the precordial dullness is mapped out by means of light percussion. Finally, auscultation reveals to us the presence or absence of murmurs and the characters of the sounds, in the standing and recumbent postures, and, if necessary, after a little exertion. The relative loudness of the first and second sounds over the different parts of the precordia is particularly worthy of note.

Now let us suppose that we have found a mitral systolic murmur. We ask ourselves whether it is structural or whether it is functional, that is due to relaxation and dilatation of the ventricular walls. If structural, with which (if any) of the diseases elicited in the man's previous history would it correspond? Most probably with gout or glycosuria. Thus we attempt to connect the lesion with its cause, and the cause with its effects, and have reached the ultimate diagnosis. So with other valvular murmurs; for example, aortic diastolic murmur proves to be related to syphilis. If there be no murmur audible, we naturally think of dilatation with failure, or of enlargement from strain, from Bright's disease, from arterial sclerosis, from emphysema, from an insufficient or impure blood supply in the coronary arteries, from disordered innervation, or from some rarer cause, such as adherent pericardium; and, again, with these associations in our minds we review once more the patient's history, and generally succeed in our diagnosis.

Here let me recount the significance of the principal signs and symptoms which I detailed to you in my last lecture, considered in the reverse order on this occasion, some of which are of real value in differentiating the causes of cardio-vascular degeneration. To begin with negative facts: a mitral presystolic murmur is never significant of a senile lesion. Secondly, when we meet with an aortic diastolic murmur, whether alone or along with an aortic systolic murmur, we may safely conclude that we have to deal with something more than atheroma produced by regular or irregular gout and associated metabolic disturbance, cardio-vascular disease

* A few passages are omitted from the text of the lecture as delivered.