lung for radiographic purposes, but I believe it is the only efficient means we possess for the treatment of chronic cases of tuberculous pyopneumothorax. As the radiograms show, it is possible in these cases, by replacing the gas and fluid by oxygen, gradually to draw out the lung without producing any symptoms of distress or discomfort. The process must be repeated on three, four, or even five occasions at intervals of two or three weeks, leaving on each occasion an increasingly high negative pressure. If the lung is completely fixed by adhesions, it cannot be made to re-expand, but it will be found that the tendency for the fluid (at first pus, and then serum) to reaccumulate will rapidly diminish—a state of affairs which is greatly appreciated by the sufferer. One of my patients with a pyopneumothorax, who had been unable to work for five months owing to the discomfort and shortness of breath, occasioned apparently by the pus (which was sterile), has been at work for over a year since I treated him by this method. Although he has only one lung, he says he is inconvenienced by shortness of breath only when he has to climb a long ladder.

Two cases I would like to recount in greater detail.

CASE I.

A man, aged 24, developed, in January, 1913, acute pneumothorax, and was admitted to University College Hospital, under Sir John Rose Bradford, with a hydropneumothorax. The lung was collapsed against the mediastinum and the fluid reached the level of the angle of the scapula. At the beginning and at the end of June I withdrew the fluid by oxygen replacement, withdrawing on the first occasion 3,060 c.cm. and on the second 1,260 c.cm. When I saw the patient again in August there was some fluid but no gas in his chest. The upper part of the lung was expanding. I withdrew on this occasion by oxygen replacement 1,860 c.cm. of fluid, and an x ray taken immediately after showed that practically all the fluid had been removed, and that the lung had expanded still further. The patient returned to full work in October, and I was unable to see him until three weeks ago. The skiagram now shows complete expansion of the greater part of the lung, marked thickening of the pleura, and possibly a little fluid at the base.

CASE II.

A man, aged 39, seven years ago had an attack of pneumonia on the right side. After the attack had subsided, he was "tapped," according to his account, and pus was withdrawn from the right pleural cavity. He was again "tapped" on four subsequent occasions during the next two years, pus being obtained each time. When admitted to University College Hospital under the care of Dr. Bolton in August of last year, the right side of his chest was full of fluid. He was wasted, and suffered from breathlessness and general debility, but there was no cyanosis or clubbing of the fingers. He was aspirated on August 30th and 2,280 c.cm. of fluid drawn off. This fluid was thick and yellow, in appearance resembling pus. It was found to consist of masses of epithelial cells, pus cells, and lymphocytes, and contained two lipoids. It was, however, sterile. The following day this radiogram was taken (Fig. 2). On September 1st I removed by oxygen replacement 2,800 c.cm. of a fluid similar in character. The variations in pressure during the replacement are shown in the chart. At the end the needle became blocked by a solid plug, and I was in consequence unable to withdraw more fluid. This plug, I discovered later by radiographic examination, came from a mass of practically solid debris at the base of the cavity. On September 4th I withdrew 485 c.cm. of fluid; the negative pressure at the end was equal to 29 mm. of mercury, which was tolerated without pain. At the end of the month I withdrew a further 1,950 c.cm. of fluid, again by oxygen replacement, the negative pressure at the end being equal to 28 mm. of mercury. The radiogram taken after this showed that the lung was beginning to expand (Fig. 3). On October 6th, by the same method, 1,485 c.cm. of fluid were drawn off, and a negative pressure left in the pleura equal to 36 mm. of mercury. The fluid, which on the previous occasion had been somewhat less opaque, was now much more definitely serous in character, and the proportion of cells was strikingly less. On November 30th, after 1,455 c.cm. of fluid had been aspirated by oxygen replacement, the negative pressure left was equal to 40 mm. of mercury. The fluid had the appearance of an ordinary serous effusion, and the expansion of the lung was considerably increased. On January 4th of this year, after again an interval of five weeks, I could withdraw only 950 c.cm. of fluid; the intrapleural pressure was at the beginning 17, and at the end 42 mm. of mercury. The radiogram (Fig. 4) shows a further increase in the expansion of the lung, and it can be seen, in addition, that the high negative pressure in the pleural cavity is causing some displacement of the mediastinum. That this displacement of the mediastinum is not injurious is shown by the fact that the patient was able to return to work four months ago, and has continued without intermission since then. Further, Dr. Lewis, who has kindly taken electro-cardiographic tracings, tells me that there is no evidence of any impairment in the heart's action. By February 16th less than 600 c.cm. of fluid had reaccumulated,

and I again withdrew this by oxygen replacement. On this occasion I obtained a negative pressure as high as 54 mm. of mercury without producing pain, and left him with a pressure of -50. At the end the lung was drawn out to the lateral wall of the chest, and could be felt with the point of the needle. The improvement in the man's general health is most gratifying. He has lost his breathlessness, has put on flesh, and says he now feels perfectly strong and well.

It is very surprising with what comfort the patient tolerates the high negative intrapleural pressure, and suffers apparently no inconvenience from it (Fig. 8). The only precaution that I have taken with him on the last four occasions has been to keep him in bed during the day on which the replacement is done. He gets up the next morning and returns to his home at Cambridge, resuming work the following day.

I believe that there are still other intrapleural conditions which can be treated by this simple process, and I hope in the future to be able to show that it is a satisfactory substitute for the open operation for certain types of acute empyema, especially the post-pneumonic in children.

THE NEED FOR RESEARCH IN ANTE-NATAL PATHOLOGY.*

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OBSTETRICIANS have recently been considering whether more cannot be done to lessen the enormous loss of life which goes on during gestation.

If I am right in my estimate¹ that there are about four times as many abortions as stillbirths (that is, 2.2 stillbirths and 8.8 abortions to 100 live births), it means that in England and Wales about 100,000 fertilized ova die annually before they are born, and that about as many infants die in their nine months of intrauterine life as during the survivors' first year of life. These estimates are much below those given by Priestley,² who said "there is one abortion to every three or four full term deliveries, and that from three to six out of every ten women abort at least once during their married life." Can this Section suggest how statistics regarding abortion can be obtained from a large series of average married women, so that the relation of abortions to live births can be definitely known? Even then there would be numerous unrecorded, because unrecognized, very early abortions.

We know very little of the causes of this terrible mortality. I have tabulated them roughly in the address to which I have already referred, but if my colleagues have been as lax as I have been in my past efforts to ascertain the causes of abortions or stillbirths, they will realize how many opportunities have been missed in not having the expelled products of conception systematically and scientifically examined with a view to discover the cause of death. Theoretically at all events, with the methods now at our disposal, we ought to be able to determine the cause of an intrauterine death as correctly as of an extrauterine death. Probably we are all agreed that a few of the abortions and premature births are due to primary uterine contractions, the fetus being normal. Maternal and perhaps paternal malnutrition may be a large etiological factor both as regards early abortions and births of sickly children. I understand that Dr. Darwall Smith has been investigating the subject of malnutrition, and will give us his conclusions. All the other spontaneous premature deliveries are due ultimately to some pathological condition of the fetus and its membranes, or of the maternal or fetal placenta.

DIFFERENCE BETWEEN EMBRYONIC AND FETAL PATHOLOGY.

Ante-natal disease and death must be considered in its early embryonic stages, as well as in its later or fetal stages. Ballantyne³ points out the great differences between the results of pathological activity in the embryo and in the fetus, and he shows that whilst in the fetus diseases are more or less of the same nature as the maladies of post-natal life, "in the embryo the morbid

* Read at a meeting of the Obstetrical and Gynaecological Section of the Royal Society of Medicine on April 2nd, 1914.

conditions are extraordinarily dissimilar from anything else in the whole field of pathology." He shows that this remarkable difference in the pathology of the embryo and of the fetus depends largely upon the initial and equally important difference between the physiology of the embryo and of the fetus. In the embryo the main function is organogenesis, the formation of tissues and organs, whilst in the fetus the functions of these organs are gradually developed, and these functions, with certain variations, are continued and perfected in the post-natal period of life.

Maternal diseases affecting the embryo during the period of organogenesis cause teratological results, tending mainly to structural anomalies or monstrosities, whilst the same diseases affecting the functionally active fetus would be pathological, causing disordered function, on lines approaching more and more to functional disease in post-natal life. In both stages the life of the embryo or fetus may be lost. In the study of ante-natal pathology we have to find out how paternal and maternal disease, such as syphilis and tubercle, affects the fertilized ovum in its embryonic and in its fetal stages, and it is evident, therefore, that whilst ante-natal pathology should be studied as an important unit with its own characteristic processes, yet it can only be successfully dealt with by pathologists who are familiar with post-natal pathology in all its variations.

THE TOXAEMIAS OF PREGNANCY.

We know very little concerning the intrauterine mortality due to the toxæmias of pregnancy, especially in obscure cases where autotoxaemia is merely causing renal inadequacy with little or no albuminuria, but with a greatly reduced output of uræa. These unrecognized cases are not infrequent causes of fetal death in the last week of pregnancy. Research again will prove whether in cases of maternal albuminuria, eclampsia, or acute yellow atrophy of the liver, the fetal organs participate in the pathological changes found in the mother in these diseases. In such serious and often fatal maternal toxic diseases our every effort is concentrated upon the mother, and the pathology of the fetus, which is often dead, is liable to be disregarded.

Careful examination of the organs and tissues of a fetus expelled during the severe types of chorea gravidarum may help us to determine the causation of that complication—as to whether, for instance, it is toxæmic in origin.

GENERAL DISEASES OF THE MOTHER.

As regards some of the more general causes of ante-natal disease and death, I would ask a few questions. Why do cases of small-pox and measles so often cause abortion? Why is scarlatina relatively so rare in pregnancy? Enteric fever is said to cause abortion in about 50 per cent. of women attacked during pregnancy, and it is stated that the typhoid bacillus is often found in the fetus. Is the fetal death in these cases due to fetal infection or to toxæmia?

What is the etiology of pyelonephrosis gravidarum? Does it affect the fetus *in utero*, or is it solely maternal and local?

Women who abort during acute pneumonia almost invariably die. Is the fetus expelled by uterine contractions caused by the eccholic action of the deficiently oxygenated blood, or does the fetus die for want of oxygen, or from hyperpyrexia, or from toxæmia, or from infection by the pneumococcus, which has been found in a few cases?

Is the severe diabetes of pregnancy as described by Matthews Duncan,⁴ Whitridge Williams,⁵ and others, due to an autotoxaemia, affecting especially the pancreas or one of the ductless glands?

What are the sources of maternal autotoxaemia? It is probably mainly placental in origin, but our knowledge of the functions of the placenta is still very imperfect. We know that ferments are produced by the trophoblastic layers of the chorionic epithelium, and that these exert a destructive action upon the surrounding maternal tissues, with a result that toxins and compensatory maternal antitoxins are formed. Whether the autotoxaemias are the result of excess of toxins or of antitoxins is still uncertain.

Abderhalden⁶ has demonstrated these ferments after the sixth week of gestation, and Dr. Herbert Williamson⁷ considers that their presence provides us with a

reliable serum diagnosis of pregnancy. This reaction is not, however, reliable in pregnant women who are syphilitic, for it may then be negative.

SYPHILIS.

Bacteriology has led to the discovery of the specific germs of so many of the maternal diseases which cause fetal death that our task now is much easier, and this is especially true as regards syphilis since the discovery of *Spirochaeta pallida* by Schaudinn in 1905, of the diagnosis of syphilis by Wassermann's reaction in May, 1906, and of its treatment by salvarsan by Ehrlich in 1909. Then there is the still more recent (1912) discovery by McDonagh, not yet generally adopted, of spores which are the result of the multiple division of the fertilized female cells, and which, after entering a connective tissue cell, develop there into both sexual and asexual organisms. In McDonagh's⁸ view the cause of syphilis is the spores, whilst the *Spirochaeta pallida*, which is developed ultimately from the male gametocyte of the sexual organism, is responsible for the symptoms. These views, if proved to be correct, will help to explain many of the difficulties which the discovery of the *Spirochaeta pallida* created, such as the possibility of paternal infection of the ovum; the explanation of Colles's law; the negative Wassermann reaction in some mothers of syphilitic children; the recurrence of syphilitic phenomena after apparent cure, the spirochaete being destroyed, but the spores persisting; and the involvement of the nerve centres, where the spirochaete is not always found.

Evidences of Syphilis in the Stillborn Fetus.

Eden⁹ states briefly that these are "a bullous eruption (pemphigus) on palms and soles, gummata in liver and spleen, hyperplasia of the cartilaginous element along the line of junction of the shafts of the long bones and the epiphysis." Elsewhere we are told that the *Spirochaeta pallida* is found in the fetal lungs in 87 per cent., pancreas 80 per cent., skin 66 per cent., suprarenals 64 per cent., spleen 62 per cent., liver 59 per cent., kidneys 54 per cent., but it is easiest to find it in the liver and spleen.

Placental Syphilis.

In my student days we were taught that a syphilitic placenta was small and shrivelled. Now we know that, relatively to the fetus, syphilitic placentae are large and heavy, bearing the proportion of 1 to 3 instead of the normal 1 to 5.5, according to Blacker, who also states that the syphilitic placenta is pale, soft, friable, and mottled with yellow patches, whilst the cord may be oedematous and the liquor amnii excessive.

The villi are enlarged, and the intervillous spaces often obliterated. Periarteritis and endarteritis are present, leading to degeneration of the stroma. Sometimes the syncytial cells proliferate. Blacker says the *Spirochaeta pallida* can be demonstrated readily in the villi, but is rarely found in the maternal decidua.

Weber¹⁰ states, however, that in most cases in which the mother has presented a true Wassermann reaction he has found spirochaetes in the maternal part of the placenta. Trinchese¹¹ states that spirochaetes can usually be found in the centre of the villi of the placenta if present in the fetal organs, but are found also in the intervillous maternal spaces, and in the epithelium of the villi, showing that they can pass from the maternal to the fetal blood stream and vice versa. If it could be shown that, in those cases in which an apparently healthy mother bears syphilitic children and yet presents a negative Wassermann reaction, spirochaetes are only found in the fetal part of the placenta, it would explain partly what is at present a difficulty.

Infection of Fetus.

Fetal syphilis may be due to direct paternal infection of the ovum at the date of fertilization, or the early fertilized ovum may be infected by the paternal semen whilst it is in the Fallopian tube or in the uterus. More usually the fetus is infected by the mother, either by the infection of the ovum before conception whilst in the Graafian follicle, or in its passage along a syphilitic Fallopian tube, or during its first attachment to a uterus in the mucosa of which *S. pallida* exists; but, as a rule, maternal infection of the fetus is transplacental.

Some observers have thought that syphilis does not affect the uterus, but Dr. Louise McIlroy¹² and Mr. Beckwith Whitehouse¹³ have made valuable researches into the question of the possibility of syphilis being one of the causes of chronic metritis and fibrosis uteri, and their papers open up the important point whether *S. pallida* may not after all be found in the uterine tissues, and if so, able to infect a freshly implanted ovum.

Probably the first published approach to a routine examination of stillbirths in this country after the discovery of the *S. pallida* was made by Drs. Herbert Williamson and Eardley Holland,¹⁴ who read a valuable paper in April, 1908, at this Section on "A case of intrauterine death of the fetus occurring in six consecutive pregnancies, with observations upon the importance of the examination of the fetal tissues for the presence of the *Spirochaeta pallida*." In this case neither husband nor wife afforded any history or clinical evidence of syphilitic infection, and in their absence specific treatment was not adopted till her sixth confinement, in September, 1906, when search was made for the *S. pallida* in the macerated fetus by Dr. Eardley Holland, then resident medical officer at Queen Charlotte's Hospital. He found the organism freely distributed in the connective tissue of the liver and spleen, but not in the umbilical cord. The placenta had been destroyed. In May, 1907, the woman was again pregnant, and was put under treatment by mercury and potassium iodide. She was delivered of living twins on November 1st after induction of labour. Unfortunately these were born too prematurely owing to a miscalculation of the date, and both children died, but no evidence of syphilis was found in either. The Wassermann reaction was not then available, or Dr. Williamson might have discovered that the mother or father, or both were infected. Dr. Eardley Holland, as is only fitting as a potential successor to Sir W. Priestley at King's College, is now again examining a series of macerated stillbirths. He tells me that he found *S. pallida* in six out of the first seven and in twelve out of the next eighteen macerated fetuses examined by him. As he and Dr. Ridge are making investigations, not only of the fetus but of every placenta, and into the question of the paternal and maternal infection in every case, we shall look forward to valuable contributions from him, as well as from other members of our Section.

The Effect of Syphilis in causing Abortions and Stillbirth.

Much difference of opinion on the subject exists. Blacker¹⁵ expresses the view held by most observers, that "syphilis is the commonest cause of repeated abortions and miscarriages"; but we only have clinical evidence of this, and some recent pathologists disagree, because the spirochaete is not found in early abortions. Thus Eardley Holland quotes Frantz Weber,¹⁶ who has investigated a series of 300 abortions in which no clinical evidence of maternal syphilis was forthcoming. Weber does not believe that abortions are often due to syphilis. He tried the Wassermann reaction in 67 of these cases, and in 35, where abortion had occurred before the sixteenth week of pregnancy, reaction was uniformly negative, and no spirochaetes were found in the embryo; in 32 cases, between the sixteenth and twenty-eighth week, 12 gave a positive reaction, and in 9 of these spirochaetes were found in the embryo. On the other hand, Weber found spirochaetes in the organs of 84 per cent. of macerated fetuses born in the later months of pregnancy.

Fournier in 1878 pointed out that the coexistence of anomalies of structure, and more especially of malformations and a syphilitic parentage, cannot be regarded as accidental. Among such malformations he mentioned hare-lip, extroversion of the bladder, spina bifida, and imperforate anus, and he considers that some of the dystrophies of the embryo, such as infantilism, cranio-facial malformations, and dental anomalies, are peculiar to syphilis, but the whole subject wants working at with the new aids to diagnosis of parental syphilis. Ballantyne³ considers that, in addition to syphilis, tubercle and alcohol, and perhaps sepsis and the enteric poison, may occasionally produce dystrophic effects. Maternal malnutrition and overwork may be other causes of fetal dystrophies. The Registrar-General draws attention to the exceptionally high fetal mortality arising from "developmental defects"

in women who work at industrial trades in the later months of pregnancy.

Such organic results of infection of the early embryo, especially if associated with structural anomalies of the fetal membranes, would often produce early abortions, in which, apart from dystrophic changes in the embryo, there might be no other evidence of disease.

Early abortions are, perhaps, the result of paternal infection of the ovum. Cases have been described by Howard Kelly¹⁶ and Ballantyne²¹ where the apparently healthy wife of a syphilitic father has had frequent abortions, but, after antisyphilitic treatment of the father alone, the subsequent pregnancies have gone on to the full term. Leroux¹⁷ believes firmly in paternal syphilis, but thinks it should be divided into two classes: (1) Patent paternal syphilis with latent maternal conceptional syphilis; (2) patent paternal syphilis without maternal syphilis. Paternal syphilis, he says, gives rise to more abortions; maternal syphilis or mixed transmission gives rise to more macerated infants.

Clinical evidence in favour of abortions being caused by syphilis is very strong, abortion often alternating with stillbirths or macerated fetuses, and treatment of the mother with mercury is known to result in the birth of healthy children. Why spirochaetes cannot often be found in such abortions requires explanation.

Maternal syphilis, whether derived direct from the father or by way of the infected ovum, is, of course, more important to the nation, as she is the child-bearer. Hochsinger¹⁸ has been able since 1869 to keep under observation 134 women who showed no signs of syphilis, but had given birth to syphilitic children on 569 occasions; 253 of these children were born dead—that is, 44.4 per cent.—and 316 were born alive. Of those born alive, 263 were syphilitic, and only 53 were healthy. Of the 263 syphilitics, 55 died before the fourth year. These are appalling figures.

Colles's Law.

The vexed question of the significance, the interpretation, and even the truth of Colles's law must be settled, for treatment depends upon it. All that Colles originally stated was that a woman who gives birth to a syphilitic child does not get infected by that child, whilst the child can infect another woman—for example, chance on nipple. It is still uncertain whether this fact means that the mother is herself infected and so cannot be reinfected, or whether the ovum is paternally infected, and the mother is rendered immune by the nine months' interchange of fetal antibodies during pregnancy. The further fact that the Wassermann reaction is often negative in these mothers during the pregnancy, but may become positive a month or so afterwards, requires careful consideration. The negative Wassermann reaction is not due to the altered blood state in pregnancy, for if a woman is herself infected before or after conception the Wassermann reaction is positive during the pregnancy, whether the clinical evidences are well-marked or very slight.

There are at least four theories which may explain the fact that mothers of syphilitic children are not infected by their children and yet are themselves apparently free from the disease. The first two theories presuppose paternal infection of the ovum.

1. *Founded on McDonagh's Discovery of "Spores."*—These spores, which are very minute and capable of being conveyed in a spermatozoon, are to a great extent indestructible by syncytial ferments or by salvarsan.⁸ The spores pass up with the semen along the Fallopian tubes, and find themselves in both the fetal and maternal portions of the early embryo—that is, in the villi and the intervillous spaces. The spores in the villi of the chorion or among the embryonic cells develop into spirochaetes, and may or may not destroy the embryo. If it survive, it becomes a congenital syphilitic, with a positive Wassermann reaction at or soon after birth. The spores in the decidua and the maternal portion of the early placenta do not develop into spirochaetes during the pregnancy owing to the presence of syncytial ferments and their resulting toxins, which circulate in the maternal blood, but not in the fetal. The "spores" are not destroyed because their chemical structure, unlike that of the spirochaete, is not seriously affected by the toxins present. This delay in development in the maternal tissues during pregnancy may explain the negative maternal Wassermann reaction

during that period. The development of *S. pallida* goes on shortly after labour and the mother then presents a positive reaction. This is one variety of conceptional syphilis.

2. *The Mother Rendered Immune*.—The ovum is fertilized and infected on the same date. The mother is rendered immune by antibodies formed in the fetus and by cells of the fetal syncytium thrown off into the maternal circulation during pregnancy.

3. *Latent Syphilis*.—The mother has been previously infected, but the disease is latent.

4. *Asymptomatic Syphilis*.—The mother is infected, but clinical evidences are insignificant or absent.

ANTE-NATAL TUBERCULOSIS.

According to most British authorities, ante-natal tuberculosis is almost non-existent—that is to say, they do not believe that a tuberculous father's spermatozoon ever directly infects the ovum at the time of fertilization (Dr. W. C. Wilkinson in a private letter refers to Gaertner's elaborate experiments upon animals as strongly supporting this view). They say, further, that the mother practically never infects the fertilized ovum because the tubercle bacilli very rarely circulate in her blood. Placental tuberculosis also is said to be very rare for the same reason, and even if present, the tubercle is in the intervillous space and does not penetrate through the syncytial cells of the villi themselves. Research is required to decide these points.

On the other hand, I would urge that paternal infection of the ovum is surely not only possible but probable in cases, for instance, where the genito-urinary organs of the male parent are infected, as in some early cases of tuberculosis of the bladder or vesiculae seminales. Whitridge Williams states that experiments upon rabbits and guinea-pigs seem to show that the male parent can thus infect the ovum.

Maternal infection of the fertilized ovum is difficult to prove, for very few children of tuberculous parents have clinical evidence of tubercle at birth. It has, however, been demonstrated that tubercle bacilli are sometimes seen in the interior of ova which are still within the Graafian follicle. Ballantyne¹⁹ gives several instances of fetal tuberculosis in which tubercle bacilli were found, but says that such cases are rare. Cases are also reported in which no tuberculous lesions have been present, yet tubercle bacilli were found, and inoculation of animals with the fetal blood or tissues from these cases has led to the development of tubercle.

It is difficult to explain why tubercle is so rarely found in the fetus. I have already alluded to some explanations. Tuberculosis of the maternal generative organs would not be likely to infect the fertilized ovum, for it would probably cause sterility, so that direct infection of the ovum or attached placenta by contact with tuberculous tissue would not occur. Supposing, too, that tubercle bacilli reached the maternal parts of the placenta, it is quite possible that the syncytial ferments and the toxins locally produced would so affect the bacilli as to prevent them from penetrating into the villi themselves.

Another possible explanation of the rarity of tuberculous lesions in the fetus may be that the infection of the early embryo may, as in syphilis, show itself by malformations and anomalies leading to early or unrecognized abortions. Whether such early abortions are often due to tubercle is a very difficult problem. It is apparently true that tubercle bacilli are not found in the early embryo, but the same is true as regards *S. pallida* in the abortions in syphilitic parents, and yet the spirochaetes abound in the stillborn fetus a few months later. Dr. Jane Walker, in a private letter, says: "Tubercle bacilli do not produce abortion in my experience." Dr. W. C. Wilkinson says much the same, and points out that tuberculous cows rarely abort, but he states that placental tuberculosis, though rare, may cause such lesions in the placenta as to lead to abortion.

V. Hanot²⁰ has described various dystrophies which may have been due to ante-natal tuberculosis, and had apparently taken the place of a true tuberculous lesion.

There is another interesting point of resemblance in the children of tuberculous and syphilitic parents, in that, according to Dr. Wilkinson and Dr. Eric Pritchard, the children of tuberculous mothers within the first few weeks of life do not react to tuberculin, but a month or so afterwards the tests may be positive. This is difficult to

explain except on the ground of acquired immunity during intrauterine life, and reduced immunity afterwards. Whilst the fetus was *in utero* it would be immune to tubercle, owing to its receiving antituberculous toxins from the mother, which she would be developing beyond her own requirements, owing to her improved general health during her pregnancy. After birth the child loses this constant supply of antitoxin and offers less resistance to tubercle bacilli already in its tissues or due to fresh infection, and swollen glands and other evidence of early tubercle would develop. In this connexion I would allude to experiments referred to by Ballantyne, in which tubercle bacilli were introduced into the hen's egg, and it was found that they remained latent, setting up tuberculosis in the chick only after it was hatched out.

If the structural abnormalities of the embryo derived from syphilitic and tuberculous parents respectively were noted, it might be possible to discover some abnormalities which were common to both diseases, and possibly other dystrophies peculiar to or characteristic of each infection.

These remarks tend to show how divergent views are and how little we are sure of as regards ante-natal pathology. I hope, too, that the discussion will stimulate obstetrical pathologists towards research, which will not only be interesting, as all pioneer work is, but will yield results which will soon be apparent in preventing disease and saving ante-natal life.

HOW TO PROVIDE FACILITIES FOR RESEARCH.

Every general hospital and lying-in hospital in our larger towns should be provided with ante-natal research laboratories. In addition, wherever practicable, laboratories should be formed in every county and county borough under the supervision of the medical officer of health, where abortions and stillborn fetuses could be collected, and either examined there or sent on in sealed jars (labelled and with history appended) to the nearest ante-natal laboratory.

As a preliminary, or perhaps as a permanent, alternative to this extended investigation of abortions and stillbirths, I would suggest that investigation should be carried out in large cities by forming centres of small groups of general hospitals, lying-in hospitals, and Poor Law infirmaries, with the co-operation of old students, so that material for research could thereby be ensured. Funds are, of course, the difficulty, but by concentrating effort in a few centres expense would be minimized. Such a centre must be officered by first an obstetric expert, who is also a pathologist; secondly, a pathological expert, with practical knowledge both of bacteriology and pathological histology; and thirdly, if possible, an expert in physiological and pathological chemistry.

The investigation must not only be pathological, but to increase its usefulness and to round off the knowledge obtained, clinical observation must be added. Thus, if spirochaetes are discovered in a macerated fetus, the case would not be complete unless the history of both parents were inquired into, and the appropriate treatment adopted in view both of the parents' health and that of subsequent children.

In conclusion, I would urge obstetricians to look upon research into ante-natal pathology as only one of the methods which must be advocated in order to deal with statistics, and with the supply of material for research, and for help in the diagnosis, prophylaxis, and treatment of ante-natal diseases, for without such associated methods any large reduction of the ante-natal death-rate is impossible. I allude especially to:

1. *Compulsory registration of stillbirths*, with a medical certificate of the probable cause of death. This certificate should be secret so far as the causes of death are concerned, if desired by the doctor.

2. *Compulsory notification of stillbirths* to the medical officer of health, with a secret certificate of the cause of death. This would make it easy for the parents to be treated if necessary.

3. *Compulsory notification* to the medical officer of health of every abortion of a formed fetus and of every stillbirth within thirty-six hours of birth, or within twelve hours if the mother is not attended by a medical practitioner. The medical officer of health should issue instructions that all products of conception should be sent to his laboratory for examination there or at a city centre.

4. Arrangements by which every poor woman can have medical supervision during pregnancy. Pregnant women who are themselves insured under the National Insurance Act are entitled to a "Pregnancy sickness benefit" if certified to be incapable of work, and also to a "Maternity benefit" after parturition. Such women, therefore, should be encouraged to voluntarily notify their pregnancy to their panel doctor or midwife. If to the latter, the midwife should notify the medical officer of health, and these voluntary notifications would enable the potential mother to be medically supervised by the panel doctor or by the medical officer of health's representative. Medical supervision of pregnant women would be made easy if the administration of maternity benefit and pregnancy sickness benefit were transferred from the approved societies to the local health authorities.

5. As advocated by Dr. Ballantyne in 1891, prematernity wards in large towns, and reserved beds in cottage hospitals in rural districts, should be provided where pregnant women can receive medical treatment.

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PITUITRIN IN LABOUR:

A RECORD OF SIXTY CASES, WITH NOTES ON ITS ADMINISTRATION.

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PITUITRIN, or pituitary infundibular extract, is now becoming generally accepted as a valuable aid to the obstetrician. Its property of inducing strong rhythmical contractions of the uterus, first pointed out by Dale,¹ is so marked that it is surprising that it has not been more generally adopted by the practitioner. In these circumstances, this record of 60 cases may prove a sufficient stimulus to those who have not experienced its merits to induce them to try it in the early future.

It is necessary, however, to observe ordinary caution in its administration, and great care should be taken where there is any obstruction. It is a very valuable aid towards shortening the long and often exhausting first stage of labour, especially in those cases in which the membranes rupture early, and a "dry labour" results.

It has proved in my hands to be a powerful remedy in cases in which shock and collapse have supervened on prolonged chloroform anaesthesia, not infrequently inseparable from a difficult case of contracted pelvis (see Case 6 in the series). In patients of a nervous temperament pituitrin has also a great influence, and the excitement is frequently calmed in an extraordinary manner (see Case 7).

Recovery is almost invariably hastened, and patients who have previously had pituitrin nearly always ask for its repetition. I have never had a case of *post-partum* retention of urine since using this remedy; indeed, in Case 6, in which it might reasonably have been expected, a free and copious emission of urine resulted within one hour of its administration. As a controller of haemorrhage pituitrin ranks very highly in my estimation; in one case (No. 58) it controlled the haemorrhage when a previous injection of *ernutin* had failed. Some writers have recorded *post-partum* haemorrhage as a sequel of pituitrin administration, when delivery of

the child did not take place until an hour or more after its administration. I have observed this on a few occasions, but if a second injection be given this does not as a rule occur. In any case a further dose will almost always control the haemorrhage (see Cases 36, 55, 58). In one case (No. 18) the patient in five previous confinements had had *post-partum* haemorrhage, while on this occasion it was particularly noticeable by its absence.

I have worked out the following technique as the best routine method for the administration of pituitrin. When the os admits one or two fingers, but not before, I inject into the gluteal muscles from 0.75 to 1.0 c.cm. of pituitrin; if the child is not born within fifty minutes give a second injection of from 0.5 to 0.75 c.cm., and repeat if necessary. If *post-partum* haemorrhage supervenes, a further injection will almost always control it. Care should be taken to make sure that the syringe and needle are quite free from alcohol before filling with the pituitrin, as alcohol is said to interfere with its action.

Case 1.—II-para, aged 35. *Post-partum* haemorrhage. Intramuscular injection pituitrin 0.5 c.cm. controlled; after six hours secondary *post-partum* haemorrhage occurred. Intramuscular injection of 1 c.cm. pituitrin given, controlled, and made an excellent convalescence.

Case 2.—I-para, aged 25. On arrival os admitted two fingers. Intramuscular injection pituitrin 0.75 c.cm. Os fully dilated in two hours. A second injection of pituitrin 0.75 c.cm. Child delivered thirty minutes later, stillborn. No haemorrhage. The child's shoulders showed unusual development.

Case 3.—VI-para, aged 40. Os admitted two fingers. Pituitrin 0.75 c.cm. injected. Os fully dilated one hour. A second injection 0.5 c.cm. given. Child delivered ten minutes later. No haemorrhage.

Case 4.—I-para, aged 19. Os size of a shilling. Pituitrin 0.75 c.cm. given. Os fully dilated ninety minutes. Second injection 0.5 c.cm. Child delivered thirty minutes later. No haemorrhage.

Case 5.—III-para, aged 30. Os about half dilated. Primary uterine inertia. Pituitrin 0.75 c.cm. injected. Os fully dilated fifteen minutes later. Delivery followed seven minutes after. No haemorrhage.

Case 6.—I-para, aged 27. Patient had a badly contracted pelvis, and anaesthesia of five hours' duration off and on followed. The patient at the end of it was in a condition of grave collapse, and was very cyanosed, the pulse being almost imperceptible. Pituitrin 1 c.cm. was injected. The pulse rapidly improved. Urine was voided one hour later. She slept for seven hours, after which she sat up in bed, her pulse then being 80. Subsequently recovery was uninterrupted. (This case was seen in consultation with Dr. H. A. Hutt, to whom I am indebted for the details of the convalescence.)

Case 7.—I-para, aged 42. Os admitted two fingers. Pituitrin 1 c.cm. given. After one hour os was about half dilated. A second injection of 0.5 c.cm. was made; the os after sixty-five minutes was fully dilated. A third injection of 0.5 c.cm. was given, delivery following thirty minutes later. *mx ernutin* was injected subsequently as an extra safeguard, but there was no haemorrhage. This patient was of an extremely nervous temperament, but after the pituitrin she was much calmer; and it was as much for this reason as for its other actions that the later injections were given. An excellent recovery followed.

Case 8.—I-para, aged 25. Os about the size of a half-crown. Pituitrin 1 c.cm. injection. Child delivered seventy-five minutes later. No haemorrhage. The patient has exophthalmic goitre. (See also Case 47.)

Case 9.—V-para, aged 32. Os admitted two fingers. Pituitrin 1 c.cm. injected. Delivery followed forty-five minutes later. No haemorrhage.

Case 10.—IV-para, aged 27. Os admitted two fingers. Pituitrin 1 c.cm. Delivery followed in sixty minutes. No haemorrhage.

Case 11.—VI-para, aged 42. Os size of a half-crown. Pituitrin 0.75 c.cm. Delivery followed thirty-five minutes later. No haemorrhage.

Case 12.—I-para, aged 30. A very prolonged chloroform labour. Craniotomy after failure of version. Pituitrin 1 c.cm. given for collapse. Made an excellent recovery.

Case 13.—XII-para, aged 49. Os admitted two fingers. Pituitrin 1 c.cm. Delivery followed in fifteen minutes. The perineum ruptured. At her tenth and eleventh confinements the labours lasted twenty-four hours or more, and the patient was astonished at this speedy termination, more especially as she had already been in labour for seven hours. The long labours were purely the result of weak and inefficient contractions.

Case 14.—III-para, aged 25. Os admitted two fingers. Pituitrin 0.75 c.cm. given. Delivery followed forty-five minutes later. No haemorrhage.

Case 15.—V-para, aged 30. Os about half a crown. Pituitrin 0.75 c.cm. given. Delivery followed in thirty-five minutes. No haemorrhage.

Case 16.—II-para, aged 26. Os about half a crown. Pituitrin 1 c.cm. given. Delivery followed forty-five minutes later. No haemorrhage. About three weeks previous to her confinement the patient thought she was in labour. The os admitted two fingers, and pituitrin 0.75 c.cm. was given, but the pains immediately quietened down and left her.