THESIS for Degree of M.D.(Old Regulations)

presented by

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"THE FEVERS OF THE PUERPERIUM."

I hereby certify that the following Thesis has been composed by myself entirely. I have received no help from any one in its preparation.

(Signed)

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'Twere vain to attempt to set forth with any degree of scientific accuracy the history and aetiology of any known disease. The ordinary definition of disease as "a departure from a state of health" means nothing. However widely this definition is elaborated, our knowledge is no whit increased. The relativity of the terms health and disease prevent accuracy of definition. As to the aetiology of this or that disease, mankind has for ages confused the causes of disease with disease itself, and still is repeating the same error. Even the modern physician speaks of fighting disease as though it were an evil spirit. Sir F. Treves recently said "if it were not for disease in the popular sense, the human race would soon be extinct." The natural processes which the body sets in motion to undo the evil effects of an accident are spoken of as symptoms of such and such a disease. The accident may be a shot from a gun or the entrance of germs into the system, small living organisms commonly though erroneously called microbes, and correctly if cumbrously called micro-organisms, and the so called symptoms which we dread to see are the outcome of nature's gigantic efforts to bring about a cure. Were it not for these much dreaded symptoms the result would be sudden and frequent death.

The probability is that the history of disease is at least as old as the history of man. Matter or micro-organisms in the wrong place, that place being the animal body, is as correct a definition of disease as the one quoted above.

The subject which I propose to discuss is that which I have termed "The Fevers of the Puerperium". The reasons which have led me to take up this subject for my Thesis are two. 1. A nine years' experience of general medical and surgical practice in Lancashire, has convinced me that there is no subject more important to the practitioner, and, what is of more account, to the community amongst which he practises, than the pathological states of the puerperium. Inasmuch as the whole period of pregnancy, labour, and the puerperium, ought to bring with it nothing that is not physiological, he who has witnessed a death from puerperal sepsis of a patient under his care, needs no additional stimulus to search for any means which will help to prevent and bringing about a cure of puerperal sepsis.

2. The advance made in recent years in Bacteriology and Serumtherapy, seemed to point in the direction where the cure, if not the prevention lay. It is to this portion of the subject that special attention will be given.

As stated above the history of this class of diseases is probably as ancient as the history of man. One cherishes the thought that long ago in the dim ages of antiguity Mother Eve and her immediate descendants experienced very little of the evils of pathological puerperia. We are left to surmise with what skill Adam tied the umbilical cord of his firstborn, or what was the nature, antiseptic or aseptic, of the first lying-in couch. Hippocrates, 450 B.C and Galen were acquainted with puerperal feverish disorders, which they attributed to suppression of the lochia. This view long held sway. In 1716 Strother first spoke of "puerperal fever", and until recently this term has been in common use. Now it is giving place to "puerperal sepsis" a term more accurately describing the nature of the disease. Smellie and Denman agreed with older writers that the cause was suppression of the lochia. In France it was generally supposed that milk metastasis was the cause. This view was adopted by many English obstetricians, hence the term "milk fever". Others attributed the diseases to inflammation, again mistaking symptom for cause. Fordyce Barker in his book on The Puerperal Diseases asserted that puerperal fever was an acute specific fever, occuring in lying-in women only.

In 1774 Kirkland made what in those days must have been an extraordinary statement, which nevertheless seems to have made little impression - "It sometimes happens that coagulated blood lodges in the uterus after delivery, and putryfying from access of air, forms an active poison which is in part absorbed and brings on putrid fever". One only wonders why the same writer made no mention of such a thing as retained placenta.

The year 1842 marked an important epoch in the history of puerperal fever, when Oliver Wendell Holmes delivered his remarkable Essay, and published it in the New England Quarterly Journal of Medicine and Surgery for April 1843. In this essay Holmes states as a fact that "puerperal fever is sometimes communiceted from one person to another both directly and indirectly--- no man has the right to doubt it any longer---- ----- the disease known as puerperal fever is so far contagious as to be frequently carried from patient to patient by physicians and nurses!" He says nothing about the method of infection, but simply records clinical evidence and deduces therefrom.

About the same period Semmemweis in Vienna was much concerned about the mortality in the maternity wards of the Infirmary, and of one section in particular. This section he discovered, was attended by students who came straight from dissecting, after

no preparation other than washing the hands in water with ordinary soap. A rule was made that the hands must be rinsed in a solution of chloride of lime after washing. The mortality thenceforth diminished. Almost by accident Semmelweis discovered the real nature of puerperal fever. A friend of his died at this time from the effects of a dissection wound which produced phlebitis, inflammations of serous membranes, and secondary abscesses. Then Semmelweis reflected that the emptied uterus was a typical open wound, and now he had all the evidence he required. Much opposition was paid to his views, and he seeme to have got tired of struggling "with tears and travail" to convince his contemporaries. By a strange turn of fate he too met his death from blood poisoning. He pricked himself while operating on an infant, and sepsis set in. The mere knowledge of this fact proved sufficient to unhinge his reason. He was taken to the hospital where he made his great discovery, and there he died.

It would serve no purpose to detail the history of those epidemics of pue rperal fever which decimated the lying-in hospitals of Paris, London, and Edinburgh in the eighteenth century. In London from 1760 to 1770 in some maternity hospitals nearly all the patients died. At the Edinburgh Infirmary in 1773 "almost

every woman as soon as she was delivered or perhaps about twenty four hours afterwards was seized with the disease and all of them died". Of late years the introduction of antiseptics into maternity hospitals has wonderfully diminished the mortality. Nowadays the presence of a case of acute septic infection at a lying-in hospital is almost unknown. Unfortunately the same cannot be said of private practice. Dr. Bloxam affirms that there is a tendency to increase in England, Scotland and Ireland. Why this should be so has given rise to much controversy. General practitioners have laid the blame on the midwife, especially on her of the "Sairey Gamp" type. It is difficult to obtain exact statistics. In all probability there are more cases than find their way into the Registrar - General's returns.

The late Dr. Milne Murray in his Presidential Address to the Edinburgh Obstetrical Society in 1900 objected to the midwife being saddled with the onus of puerperal mortality, and being sent into the wilderness as a scapegoat. He blamed the practitioners for their "misuse of anaesthesia, and for the ridiculous parody which in many practitioners hands stands for antiseptics". Words coming from the lips of such a teacher as Milne Murray must be treated with respect, but such has not been my experience. One may be permitted to assume that the type of midwife

that a fashionable metropolitan Consultant was acquainted with differed somewhat from the type one meets in Lancashire. Fortunately the new Midwives Act is already effecting an improvement. That a very large proportion of cases of puerperal fever in Lancashire can be directly traced to midwives, and other untrained attendants I unhesitatingly assert. Instances of this will be quoted later.

On the other hand one can recall many an instance of difficult labour, with and without anaesthesia, with instrumental interference, and subsequent exploration of the uterus with the hand, without a single case of sepsis ensuing, whereas other cases in which not even a vaginal examination was made, have developed acute sepsis, and, as in one of my cases, proved fatal. These cases were isolated cases, and not epidemic. This is I think, the experience also of the majority of my fellow practitioners. It is a commonly heard remark that the difficult cases do well, and the easy ones go wrong. If one could explain this fact, one would arrive at a solution of the great problem of treatment.

To my mind puerperal mortality may be described as due

1. To the midwife, or other untrained attendant.
2. To the general surroundings of the patient.
3. To the doctor.

Aetiology:-

Puerperal sepsis is pre-eminently the feverish disease of the puerperium. A few other conditions must however be mentioned. Constipation, if neglected will send up the temperature one, two, or even three degrees. Temperament, too, plays an important part in influencing the temperature of a puerperal woman. A nervous temperament is easily and markedly acted upon by emotion, as evinced by a rapid rise of temperature.

Then there is the vexed question as to whether the secretion taking place in the mammary glands two or three days after labour produces a condition of fever, and to what extent. In 326 of my own cases I have only been able to convince myself that this cause produced a rise of more than one degree in 5 cases. In cases in which catheterisetion is required after l labour, one may meet with the condition of catheter fever. On account of the dread which lying-in women have of this operation the rise in temperature is doubtless increased. These causes and their treatm are sufficiently obvious , their enumeration alone is sufficient.

9. Aetiology:-

The dread condition formerly known as "Puerperal Fever" we now know to be due to the presence of micro-organisms. We make this statement with two reservations:- 1. We have not yet discovered what the poison in infective disease really is. Are the toxins, the toxic albumins though undoubtedly toxic the final cause? We don't know whether the toxins are themselves the poison, or whether they produce poisonous enzymes.

2. There must be some explanation, as yet unoffered op the extreme susceptibility of lying-in women to septic disease. There is no reason why these same organisms should not prove equally troublesome after ordinary operations on the genital organs.

It has been hitherto assumed that these micro-organisms have been introduced into the genital tract by the attendants on hands and instruments. The occurence of "autogenetic" poisoning in puerperal women has been, and still is, generally denied. One needs to be chary of denying anything regarding the causation of septic disease. As mentioned above cases in which no examination has been made, in which strict cleanliness has been observed with regard to clothing and personhave turned out to be septic. These cases hit hard at the "heterogenetic" theory. A case I saw a few weeks ago has an interesting bearing on this subject. A colleague went away for a holiday, and left me in charge of a woman who had been delivered

three days previously. Her temperature on the third day was 102. but as Dr. B. remarked "There can be nothing septic por there was no examination made, the child being born as I entered the house". On the fourth day the temperature rose to 103. with a rigor, pulse 130. I must here state that no one had made an examination. This was verified. On exploration of the uterus a secondary placenta was discovered, the size and shape of a large pear, firmly adherent to the uterine wall. Septic mischief was well established. How did the micro-organisms enter the uterus? This seems to be one more link in the chain of clinical evidence that infective material may be suspended in the atmosphere, and reach the site indirectly. Dr. Armand Routh and Dr. W.S.Playfair refer to four cases occuring amongst butchers' wives who were confined "in an atmosphere permeated with meat". Playfair in his "Science and Practice of Midwifery" lays great stress on sewer gas being a cause of puerperal septicaemia, yet the air of sewers has been proved to contain no bacteria. Playfair in support of his theory suggests that sewer gas alone may not be the cause, but that it may produce a condition favourable to the growth and absorption of pathogenetic germs. One naturally asks the question Why living under such conditions as these two last, does the non parturient woman remain free from septic disease, andthe parturient woman on the other hand is so unfortunate as to develope septicaemia? True the latter has an

open wound, but how do the organisms gain access to it? This naturally leads us to a study of the blood in pregnant and in parturient women.

The Blood during Pregnancy and Parturition.

The blood of pregnant women has been carefully examined, especially cytologically, by such accurate observers as Hubbard, White, Cabot, Henderson, Halla, and Reider. A summary of their results reveals the fact that the leucocytes gradually rise in number from the fourth month of pregnancy. These writers differ as to their figures, but agree as to the general statement. They are also at one in stating that the rise is most marked in primiparae. This is worth noting when one remembers that puerperal sepsis is more common in primiparae than in multiparae. In the puerperium the number of leucocytes falls with great rapidity during the first week, less rapidly during the second, regaining the normal in about 15 days. How it is no easy matter to estimate the clinical significance of leucocytosis, especially in pregnancy. Inflammatory conditions influence it markedly. In health too, there is a considerable variation in different women. In puerperal sepsis, however, instead of the number of leucocytes returning to normal, there is an increase, usually steady, but rapid in the case of a localised abscess. In some

rare cases on the other hand, there is sepsis without leucocytosis.

No theory has as yet been adduced to explain the significance of the leucocytosis in pregnancy.

So far as our knowledge goes at present, the properties of lueucocytes are four:- 1. They have amoeboid movements. 2. They exert a phagocytic action upon bacteria. 3. They multiply in inflammatory conditions. 4. They possess certain secretory qualities. On this last p point our knowledge is limited, indeed almost nil, for speculation is not by any means knowledge.

It seems to me just possible that the leucocytes throw out a secretion in pregnancy, for purposes perhaps of nutrition, which forms a suitable pabulum for micro-organisms of infection after labour has taken place. Again one draws attention to the fact that there is a greater degree of leucocytosis in primiparae, and a greater tendency to septicaemia than in multiparae. This subject needs further investigation. Herein must lie the secret of the extreme liability of lying-in women to septic mischief.

The red corpuscles play a very secondary part. When anaemia is present the haemoglobin is of course reduced, but many pregnant women show no reduction in blood colour index.

Enough has been said to show the difficulty of classifying the causes leading up to puerperal sepsis. Formerly they were classed as "heterogenetic" and "autogenetic". As it is now assumed that the bacterium has to be introduced from without in every case, this definition is driven out of court. My own classification would be as follows:-

1. The condition of the patient herself, at the time of delivery and shortly afterwards. The state of the nervous system is an important factor. One is familiar with cases in which the patient has a presentiment during pregnancy that she will not come through all right. These often turn out to be only too true a forecast.

2. During the puerperium there are certain causes which favour the absorption of septic matter. Such are:- (a) the breaking down tissue of the placental site. (b) the absorbing raw surfaces. (c) the abundant venous and lymphatic supply of the uterus. These causes are all aggravated in pathological labour. A portion of placenta or of the membranes may be retained. The placental site may be more extensive than normal, as in twin births, or in placenta praevia. If the uterus is inert and relaxed after delivery there is a greater liability to septic infection.

3. Cadaveric poison brought by contact with the dead body, and especially its viscera.

4. Septic discharge from an abscess.

5. All putrifying substances such as decomposed blood clot, and urine.

6. Contamination by menstrual fluid. In one of my cases I was able to satisfy mysely that this was probably the cause.

7. Sewage gas, defective drainage etc.

8. Contact with zymotic diseases, especially scarlet fever.

9. The poison of Erysipelas.

Pathological changes:-

These depend on the quantity of the poison received, the quality of the poison, that is its degree of virulence, and thirdly on the condition of the patient. One cannot over-emphasise the fact that if two patients receive an equal quantity of poison, one of whom is in robust health up to this point, and the other is weak and exhausted, the former may develope only very slight symptoms of septicaemia, or none at all, whereas the other rapidly succumbs to the effects of the poison.

The method by which the poison spreads is comparable to that which takes place in say a cut finger in the post-mortem room. There may be merely an inflammation of the cellular tissue and lymphatics,

or there may be a rapidly fatal result from acute septicaemia, or a result similar but less speedy from chronic septicaemia with pneumonia, arthritis etc. or again the veins in the vicinity of the wound may undergo thrombosis, the thrombi may break down producing infarcts, abscesses, empyaema and death.

The placental site has only to be substituted for the cut finger in the dissecting room. There is this vast difference in the two cases however. The puerperal case has greater absorbing powers, and a vast peritoneal surface close at hand.

A convenient method of classifying the pathological changes of puerperal septicaemia is that which has relation to the various sites where inflammation occurs.

(a) Inflammation of the vagina and vulva. In normal labour there is a certain amount of vaginal oedema, and loss of tone in the tissues. Add to this a torn vagina or perineum or both and one obtains a fertile source of infection should the parts become infected through uncleanliness on the part of the nurse. The smallest tear if neglected may lead to fatal results. It is not only the mucous membrane which is involved. The underlying tissue becomes infected and resembles erysipelas, only it is internal.

(b) Inflammation of the uterus and Fallopian tubes. The result of this is a septic endometritis,

with extensive uterine discharge. This in its turn may cause a septic metritis, or a septic salpingitis, or both. This condition most frequently arises from retained placenta or membranes, or from injury to uterus during labour.

(c) Inflammation spreading to the cellular tissue around the cervix. The acute inflammation and oedema run along the lines of cell tissue to the broad ligaments and lateral fornices. It may extend upwards into the iliac fossa, and into the peri-nephritic tissue. Occasionally it extends to the sciatic notch and large vessels, and results in phlegmesia. When there is much cellulitis the neighbouring lymphatics thrombose. The cellular tissue around the ovary may become affected , then the ovary itself. " A left sided cervical tear often is succeeded by inflammation spreading along the utero-sacral or utero-vesical ligaments"
(Milne Murray, Lectures)

(d) Peritonitis either[1] an extension of inflammation from the uterus ~~or tu~~ through the tubes. It may be confined to the pelvic peritoneum or it may become general.[2] On the other hand it may be produced directly through injury to the peritoneum during labour.

(e) Infection through the circulation produces two varieties of septiceamia:-

1. Septicaemia Venosa from the absorption

of septic thrombi into the circulation through the uterine sinuses and veins. This is the most rapidly fatal type of puerperal septicaemia, death resulting in less than 48 hours in many cases. In less rapid cases secondary foci are formed in lungs, liver, spleen, kidneys. This latter condition is known as Pyaemia.

2. Septicaemia Lymphatica starts in the cellular tissue, spreads through the lymphatics, may spread to the leg causing the condition known as Phlegmesia Alba Dolens, or may result in secondary infection of the peritoneum, pleura, joints etc.

(f) Lastly the condition of Sapraemia must be mentioned. This is caused by septic mischief taking place, usually in the uterus, from whence poisonous products are absorbed and transmitted all over the body, producing a low feverish condition. The usual cause of this condition is retained placenta.

Signs and Symptoms of Puerperal Sepsis:-

Having enumerated the pathological changes caused by sepsis in the puerperium, the natural sequence is found in the various signs and symptoms which are manifestation of such changes. There are five main sets of symptoms, according as the pathology varies. These are the symptoms of 1. Sapraemia. 2.Septicaemia. 3.Pyaemia. 4. Pelvic Cellulitis. 5. Peritonitis.

Sapraemia.

Derived from the Greek σαπρός meaning putrid Sapraemia denotes a state of septic intoxication the result of the absorption into the blood not of microorganisms, but of their toxic products. These products are alkaloidal, and are comparable to other forms of alkaloid poisoning such as Strychnine. Their action is consequent immediately on their entering the circulation. The effect produced is in proportion to the amount absorbed. If the poison is early eliminated form the body the symptoms abate, and providing no more is introduced they disappear.

In the puerperium symptoms of Sapraemia may develope 24 hours after ~~labour~~ childbirth, or at any period from this to the end of the third week. More usually symptoms manifest themselves during the first week. The onset of the condition is usually marked by a rigor, or if not this, then by a feeling of chilliness, and a rise of temperature:- 102, 104 or higher.

The heart's action is weakened, the pulse is rapid, soft, and easily compressed. The tongue is dry and covered with a brown fur. Appetite is lost. There is often headache, vomiting and diarrhaea. The urine is diminished in quantity, with a large increase in urates. There is often delirium, especially at night. The lochia is usually though not always foetid. Sometimes the discharge is retained. In fatal cases

cases the disease lasts a few days. As death approaches the skin becomes cold and clammy, the pulse fluttering, there is low, muttering delirium, and there is incontinence of urine and feces. Finally come stupor, coma and death.

Septicaemia.

This is a form of septic poisoning the result of pyogenic bacteria entering the blood stream, and being carried to the viscera, where further changes are produced. At the same time toxins are being produced at the primary seat of infection. The smallest breach of surface is sufficient to admit of the organisms gaining an entrance. The onset is marked by a chill or rigor, while the temperature ranges from 103. to 106. The temperature , however is very variable, being rapid, feeble & easily compressed.
The pulse is a more reliable guide. The tongue is dry and coated. There is marked thirst, vomiting and diarrhoea. The amount of urine is diminished, and the proportion of urates increased. If the disease progresses the pulse becomes increasingly feeble, the breathing shallow, the skin cold and clammy. In many cases jaundice is present.

It is very difficult to distinguish between septiaemia and sapraemia. The one may result from the other and frequently does. Also the same conditions favour

favour the one which favour the other. Their early symptoms are the same. In the early stages ,however, the amount of prostration produced by septicaemia is infinitely greater than that produced by sapraemia.

The prognosis is always grave, and depends on our ability to thoroughly disinfect at the primary seat of infection, and what is also of paramount importance, on the condition of the patient herself, her ability to withstand a long siege.

Pyaemia.

This is a form of poisoning brought about by the entrance into the circulation of pus containing microorganisms. As a consequence ~~we get~~ there ensue secondary foci in different parts of the body. In the puerperium the pus usually results from uterine phlebitis. Septic emboli form in a thrombosed vein. These breaking down are carried into the blood stream. They may lodge in the small vessels of the lungs, liver, kidneys, spleen and synovial membranes especially the peritoneum. These are the commonest situations where pyaemic abscesses form. Occasionally joints are affected. Any tissue may be affected.

Recovery in not so rare as used to be supposed. When death occurs it is usually towards the end of the second week. The disease may manifest itself

itself any time after the first week after childbirth. The onset is heralded by a severe rigor with a temperature ranging from 103. to 106. Soon the temperature falls and there is profuse perspiration. ~~Now the temperature falls, in some cases almost reaching normal.~~ In a few days there is another rigor with rise of temperature, then remission and so on. The intervals between the rigors diminish as the disease progresses. A significant fact from the diagnostic point of view is that the pulse is always rapid even though the temperature may approach the normal. Headache, vomiting and diarrhoea are present. If the kidney be infected the urine shows albumin and blood casts. If the liver be infected there is jaundice; If the lungs haemoptysis and pneumonia may ensue; The breath has the characteristic smell of pyaemia namely that of new-mown hay. Emaciation and prostration are alike profound. The pulse is small and feeble the tongue is dry and furred.

Pelvic Cellulitis.

This is an inflammatory affection of the pelvic tissue due to microorganisms, usually the streptococcus pyogenes. The seat of inoculation is usually some distance from the cellulitis, the organisms travelling via the lymphatics. The inflammatory process is often

often very severe and very rapid. This is readily understood when one remembers how readily owing to its open construction cellular tissue lends itself to inflammation. Further the pathogenic process is aided by the free lymphatic circulation, as well as by the virulence ou the organisms.

If the cellulitis be diffuse the onset of the symptoms is a marked one. A rigor and high temperature are constant initial symptoms in severe cases. The pulse is rapid and feeble. The tongue is dry and furred. The face is flushed. There is frequently delirium. There is considerable pain in the pelvis. On examination the broad ligaments are found to be swollen. The swelling spreads into the groin, down the thighs, or back through the sacro-sciatic notch, travelling along the lines of least resistance. Death occurs in about two weeks.

These severe forms of pelvic cellulitis are happily rare. More commonly there is a certain amount of pelvic pain during the second week after delivery, of which unless there is much fever but little notice is taken, or which may yield to hot fomentations or poultices, but which if neglected gives further trouble. The patient gets weaker instead of stronger, and the temperature keeps between 100. and 102. Then on examining a swelling is found in the broad ligament commonly unilateral. This travels upwards between the fascia and the peritoneum forming a swelling parallel with

with Poupart's ligament. In cases which are early recognised and treated, the disease is usually arrested here by absorption taking place. This takes place in from two to three weeks, the pain and swelling gradually disappearing. In unfavourable cases suppuration takes place, the swelling in the groin increasing and pointing. Free opening and draining is now the treatment. In very rare cases the pus travels by a different route, namely along the psoas muscle between it and the brim of the pelvis. Owing to the psoas being put out of action the thigh which is bent up at an angle with the pelvis and cannot be straightened.

In these cases of suppuration recovery takes place in from two to three months usually.

Peritonitis.

This may be due to an extension of uterine inflammation through the Fallopian tubes, or spreading through the cellular connective tissue of the pelvis and the wall of the uterus. Less frequently it is the result of direct injury of the peritoneum.

Other causes of peritonitis are gonorrhoea, (through upward extension), a localised inflammation of a tube, or an ovarian cyst injured in delivery.

No matter which of these causes has given rise to the condition there is always pain and tenderness, fever and prostration. In the cases of local inflammation

inflammation the pain and tenderness are both localised and the prostration is not so severe as in general peritonitis. Furthermore these cases are often cured by the surgeon. On the other hand general peritonitis proves fatal in two or three days, the abdomen becoming increasingly tender and tympanitic, the pulse thready and the breathing feeble. There is the typical attitude of peritonitis, the patient lying on her back with knees drawn up and face pinched. Death takes place from exhaustion.

The above classification, though it seems by most obstetricians to be the only feasible one, is nevertheless most unsatisfactory. We ask in vain "Where do the putrid cases end , and the septic cases begin?" To increase the difficulty the two forms are often found together, that is to say the clinical features of both forms are often present in the same case. The difficulty arises when we come to the question of treatment, (for after all classification is of little practical importance) for the treatment of the two forms is quite different. For the putrid form curetting is the treatment, while in the septic form it might prove positively harmful. In other words if the infection is limited to the uterine wall curetting can only convey the infection into deeper tissues.

If one allows that the best curette is the finger, then curetting and douching could not appear to do

much harm.

Bacteriology.

Bacteria are minute vegetative organisms, also called microorganisms or microbes. They are composed of protoplasm finely granulated, and surrounded by a thin gelatinous capsule. Most bacteria multiply by simple fission, each cell dividing into two cells. This procees is a very rapid one millions of cells being produced in twenty four hours. Some varieties of bacteia increase by spore fomcation .

Bacteria are divided into two classes according to their shape. %The globular cocci. 2. The rod-like bacilli. All cocci (micrococci) are minute round bodies. They multiply by fission. If after dividing the cells remain in pairs they are called diplococci. If they form irregular grape-like bunches they are termed staphylococci. Most pus-forming organisms are staphylococci. When longs chains of cocci are formed they are called streptococci. These are met with in puerperal disorders as well as in erysipelas.

The enormous rapidity with which these bacteria proliferate has an all-important bearing on those septic states of which they are primarily the cause. In other words the incubation stage of these puerperal fevers is probably shorter than that of any other fever. This has to be borne in mind when one comes to consider the treatment by Serumtherapy. Such if not early administered is absolutely useless.

There are othe r conditions besides this rapid proliferation which must be taken into consideration when discussing the life history of bacteria. Their food is obtained froc the animal body whose proteids and carbohydrates they disintegrate into their primal elements. Moisture is a necessary condition for their growth. Alkalinity favours it, as also does the presence of Oxygen. Some bacteria, however, can thrive without Oxygen, though better with it; such are called "facultative anaerobes". Some cannot live if oxygen be present. These are called anaerobic.

The surest method of killing bacteria and their spores is by heat - boiling for twenty minutes-. The chief chemical agents inimical to bacteria are the biniodide and perchloride of mercury, and carbolic acid. The ter m saprophyte is given to the bacterium which only grows in dead matter, in contradistinction to that of parasite, the bacterium invading living matter.

A fact of practical interest whilst discussing discussing Bacteriology of the puerperal septic states is that if a tissue be devitalised, through laceration for instance, its ability to resist bacterial invasion is largely diminished. Further, a natural corollory of the preceeding statement, any lowering of the general vitality of an individual predisposes to bacterial invasion and assists bacterial

growth.

The rapid proliferation of bacteria, and the degree of the individual's general and tissue vitality are the two factors on which depend the virulence of the bacterium. One makes this statement with a certain reservation. Certain races are peculiarly liable to certain diseases. There is no reason, however, to suppose that the organisms of puerperal sepsis are predisposed in favour of any particular race. The attendant ills of increasing civilisation would seem to impair a people's vitality. One cannot help contrasting the easy labour of the North American Indian's sqaw, with that of the pinched anaemic Lancashire factory " hand. In the wigwam of the one, the only weapon present was her chief's tomahawk. In the cottage of the other it is only too often the axis-traction forceps.

How do bacteria produce their injurious effects?

We have not yet learned the secret of what the poison really is in infection. When bacteria settle in a tissue they break it up into food stuffs for themselves, and many chemical products are evolved thereby. Amongst these are albumoses, peptones, carbonic acid gas and sulphuretted hydrogen. All such evolved products which possess poisonous properties are termed toxins. The name ptomaine is given to

to the nitrogenous alkaloid which is tde product of putrifaction. Bacteriologists assert that the ptomaines play only a sefondary part. The albumoses are admitted to possess toxic properties, but whether they are the final toxic cause or not, is not yet determined. Some assert that the toxins themselves produce a poisonous enzyme. ~~Here speculation would lead one to suppose that both the microbes and their toxins are poisonous, and this has been in the~~

The fermentative action is the most striking property of the bacteria, but in addition there is probably a secretory and and an excretory action.

" The aim of bacterial research must be to establish the specificity of the bacterial products, to prove that the toxins and the microbes are alike specific." In three diseases this proof has been established viz. diphtheria, tetanus and anthrax. The same must be true for infective disease, its specific symptoms due to specific poisons, and the accompanying symptoms due to the other products of fermentation .

Immunity is the freedom from infection by certain diseases. Some persons enjoy an absolute immunity, others having had one attack of an acute disease are no more infected by it and thus have obtained an acquired immunity from that disease.

To take the case of small-pox. It was realised centuries ago that one attack of this disease was very seldom followed by a second, even though the individual were exposed to severe infection. As a result inoculation(from mild cases) was practised, in England in 1721, whither the practice was introduced from Turkey. The objection to this inoculation was that there was no assurance that the resulting attack would be a mild one. In ~~DNOE~~ 1796 Jenner by vaccinating was the first to bring about successful immunisation. How the introduction into the system of this Vaccinia or modified form of small-pox really acted in preventing small-pox itself was of course unknown, inasmuch as the nature of infection was unknown. Bacteria were as yet undiscovered, though it was supposed that infectious diseases were spread by the transmission of living particles from one person to another. It was reasonable to assume that only a very minute particle of infectious matter was transmitted to the healthy individual, and it follows that the infectious matter must multiply within the body. The next advance was made by Pasteur who discovered that the yeast plant is the producer of fermentation. Close resemblances were observed between fermentation and infective disease. It was then asserted by Henle in ~~DSAR~~ 1840 that vegetable organisms would probably be found to be the cause of infectious diseases. This assumption was proved to

be correct in the case of anthrax, whose bacillus was ~~disco~~ discovered in 1850 by Davaine. In 1863 Davaine showed that anthrax could be produced by means of the bacillus.

When Koch later discovered that by using solid media for the growth of organisms, pure cultures could thereby be obtained, the science of Bacteriology was firmly established.

Now, Septicaemia is one of the many diseaseswhich have been proved to be caused by bacilli.

Many infective diseases run a natural course, and then terminate, or die out spontaneously. There have been various theories propounded to explain this, notably the Exhaustion Theory of Pasteur, and the Retention Theory of Chauveau. The first is based on the fact that a plant cannot live for more than a limited period on a limited quantity of soil. It was found however that micro-organisms could be cultivated "on the juices derived from an animal which had just recovered from the very disease which was due to their activity, a and which, therefore, all available nourishment for them should theoretically have been exhausted". Bassanguet

The Retention Theory is founded on the suggestion that the products of the bacteria by their poisonous action prevent the growth and development of the bacteria themselves. Unfortunately for this theory no such accumulation of excretory products takes place in living animals, on the contrary they are rapidly passed out of the system.

Metchnikoff explained , or thought he did, resistance to disease by his theory of Phagocytosis. This theory has since been shewn to be only partly true. At all inflammatory sites leucocytes collect, and doubtless they act as a defensive force, but if they absorb the bacteria they may only spread the disease by carrying them into the circulation.

Phagocytosis has been replaced by the Humoral Theory, which is now accepted as the true cause of resistance to disease.One cannot enter into this theory at length in a thesis such as this. It has been expounded at great length by Ehrlich, who has named his views the "side-chain hypothesis". The essence of this theory is however that it is the fluids and not the cellular elements which are the chief factors in resisting bacteria. "It has been shown that the serum of the blood, even when all formed elements such as the corpuscles had been removed, still exerted in many instances an inhibitory action on the growth of micro-organisms. (Nuttall,Buchner.) There must therefore be present in the plasma some substance of a protective nature upon which immunity depends. To these hypothetical substances Bluchner gives the name alexines."

Serumtherapy dates from the year 1890 when Behring and Kitasato showed the possibility of rendering animals immune to tetanus. They injected animals with the toxins of tetanus, at first in infinitesimal

doses, afterwards increased. Animals treated in this way came to endure without ill effect doses so large as to kill animals not so immuned, that is animals of the same species. Experiments were then made with the serum of an animal so immunised. Such serum was mixed with an equivalent amount of the poison and injected into a non-immune animal, with no ill effect. The immune serum itself was injected into a non-immune animal, and thus made the animal resistant to the poison. Furthermore an animal was inoculated with the bacilli of tetanus and shortly afterwards a dose of immune serum was injected into the animal. Tetanus did not ensue.

The next disease in which immunisation by anti-toxins was established was diphtheria. This was the first disease in connection with which the term antitoxin was used. Antitoxin is therefore the name given to the substance in the serum, as yet unknown, which has the property of neutralising the toxine. In diphtheria serimtherapy has been of immense value. With regard to many other diseases it is still on its trial, and their name is almost legion, ranging from hay fever to foot and mouth disease. As yet , unfortunately, puerperal septicaemia is one of those conditions in which its value is still sub judice.

There are two kinds of serum:-

1. Anti-bactericidal or anti-bacterial
2. Anti-toxic.

To prepare an antitoxic serum the toxin is first obtained and injected into animals. To prepare an antibactericidal serum, the animals are injected with the bacteria themselves, and a serum is produced which is inimical to the bacteria. To this class belongs antistreptococcic serum with which we are concerned in treating puerperal septicaemia. Assuming for a moment that the streptococcus is the chief organism in septic conditions of the puerperium, then if we inject an animal with streptococci until it is very resistant to these germs, then add a little of its blood serum to a culture of streptococci, the organisms will be found disintegrated. No quantity of this serum however, will neutralise a lethal dose of the poison of the streptococci.

It follows on the other hand that an antitoxic serum cannot prevent organismal growth.

There are other important facts in relation to antibacterial serum which must be referred to in this place.

1. It must be used fresh, as its activity diminishes with age.

2. Some species of bacteria are made up of a great many different varieties. There are, for instance a great many varieties of streptococci, and these different varieties react differently towards any particular combination of varieties in any particular serum. This explains why such varied results are

Horodaber Amer. Jred 1903 p. 877 chemicals 18 different species

are recorded in treatment. In order to increase the chances of success, as many varieties as possible of streptococci are obtained with which to immunise the animal, and the resulting serum is termed "polyvalent".

3. To go back to the theory of immunisation- for immunity to take place an unknown substance existing in the body of one animal must unite with a second substance supplied by another animal, in order to ensure the destruction of the bacterium in question. If these two bodies do not fit one another no curative result follows. Hitherto antistreptococcic serum has been prepared from horses. It is just possible that a more "homologous" serum, that is one prepared from an animal more closely allied to man such as the ape would have a stronger curative action. It is improbable that the blood serum is identical in any two species of animals.

It is a very difficult matter to standardise an antibactericidal serum. The serum is diluted in two strengths, e.g. B : 100 and 1 : 1000. "Two guinea pigs are taken, one of which receives 1cc. of the first dilution along with a loopful of a virulent culture of streptococci, while the other receives the same quantity of the second dilution with the loopful of the bacteria. Within forty minutes search is made in the peritoneal cavities of the animals ts see whether the organisms therein are flourishing - multiplying and moving actively about - or whether they are in process of disintegration. If the smaller dose of serum has failed to

to kill them, while the larger one has done so, further experiments are necessary to determine the exact quantity of serum which just suffices for the purpose; if the lower dose has proved sufficient, then smaller quantities still are tried, and so on." Bosanquet

(Serums, Vaccines and Toxines. page 48

The bacteria of Puerperal Sepsis:-

Drs. Foulerton and Bonney in an article on "An Investigation into the Causation of Puerperal Infections" published in the Transactions of the Obstetrical Society of London for 1904 enumerate the varieties op bacteria found in puerperal septic conditions as follows:-

1. Streptococci.
2. Micrococcus pneumoniae.
3. Staphylococcus pyogenes aureus.
4. Staphylococcus pyogenes albus.
5. A "diphtheroid" bacillus.
6. An unnamed diplococcus (Gram).
7. Micrococcus gonorrhoea.
8. Anaerobic Bacteria.
9. Bacillus coli communis.
10. Bacillus diphtheriae.

One other bacillus has been mentiosed as a cause of primary puerperal infection, viz. B. typhosus, by Dobbin in the American Journal of Obstetrics for 1898.

A very careful analysis is given by Foulerton and Bonney of 54 casesof primary puerperal infection. In 15 of theoe cases the uterus was sterile of bacteria. In 3 of these 15 cases no bacteria were found in the generative tract at all. In the remaining 12 cases there was laceration, and ten of these were primiparae. Of the 39 cases in which bacteria were present in the uteous 14 were fatal. Of these 14 fatal cases 10 shewed streptococci in utero. These results agree with those of other writers who attach most importance to the streptococcus as the chief organism of puerperal sepsis. It is worth noting that only one of these 14 fatal cases shewed the prseence of Staphylococci in utero, and in this case the B.coli communis was present. One other noteworthy fact is the extreme fatality in those cases in which the B. Coli Com. is found along with streptococci, viz. 83%.

The micrococcus gonorrhoeae deserves special mention. Many old practitioners are convinced that gonorrhoea is a fertile sourle of septic infection in the puerperium. One of these speaking from thirty years experience, informs me that he has always made a practice of interviewing the husband of the woman suffering from puerperal fever and "in a great many cases" to use his own words, did he satisfy himself that his patient had gonorrhoea during pregnancy. This certainly favours the autogenetic theory, only one

one must remember that purulent ophthalmia in the infant does not by any means imply that the mother must of necessity be suffering from puerperal sepsis. In none of my cases of purulent ophthalmia neonatorum was the mother suffering from sepsis. Further gonorrhoea in the female is so common that if one is to believe that it is a cause of puerperal sepsis, one would expect to meet with even a much greater percentage of cases of sepsis than one does.

Several cases ha e been recorded of "puerperal diphtheria". Haultain published a case in the Trans. of Edin. Med. Soc. Vol.22. 1897. in which a typical pure culture of the Klebs-Loeffler bacillus was obtained from the interior of the cervical canal during the puerperium. Anti-diphtheritic serum was administered and the patient recovered. One must beware , however, in assuming that a false membrane on a mucous surface means diphtheria. A similar membrane results from streptococcic infection, as well as from other bacteria. One is familiar with the "membrane" that forms on the tonsils after tonsillotomy. Puerperal diphtheria is certainly rare.

Laceration .

In 2 of Foulerton and Bonney's cases there was more or less laceration. All these were mild cases, and none exhibited the streptococcus.

Treatment.

In the present uncertain state of our bacteriological knowledge, the only safe method is to treat all cases of puerperal infection as though they were of streptococcic origin. At the first onset of symptoms a careful examination should be made of the whole genital tract from below upwards. Cleanse thoroughly any lacerations. If a sutured perineum is suppurating remove the stitches so as to afford free drainage.

Next a specimen of uterine secretion should be obtained for microscopic examination and culture. The best apparatus consists of a double tube, an inner and an outer. The outer is covered with a thin diaphragm. The tube is first sterilised, then the outer tube is passed within the cervix, and the inner tube pushed through the diaphragm then having received the secretion it is withdrawn into the outer tube, and the whole apparatus is removed. If, now, saprophytes only are found, the case is most probably sapraemia, and the treatment is repeated washing out of the uterus, every eight hours until there is a complete subsidence of the symptoms, and afterwards twice a day for two days.

If septic organisms are present the case is probably septicaemia.

In those cases in which douching proves of no benefit the uterus should be explored with the finger and any retained placenta or membrane removed, followed by thorough douching, which must be frequently repeated.

The first douche should be sf Carbolic Acid 1 in 60, the subsequent ones of sterilised water. For exploration of the uterus with the finger it is advisable to give an anaesthetic. In my opinion the best curette, and the only curette that ought to be used in most cases is the finger, for reasons stated above.

If improvement is not observed within an hour or two of adopting this treatment , no time should be lost in injecting antistreptococcic serum. Commence with 20cc. In ten hours inject a further quantity of 10cc. If the result is unfavourable inject a further 10cc. in another ten hours. If the result is favourable leave the third injection until twenty four hours after the second.

I have used antistreptococcus serum in twenty one cases, my own and those of my colleagues. In eight of these cases I feel convinced that recovery was due to the use of the serum. Unfortunately I was not able to get a bacteriological examination save in eight cases. In each of these cases it was the uterine secretion that was examined. Four of these contained streptococci, and all were severe cases. In two of these four streptococcic cases a marked favourable reaction took place within nine hours, with no relapse. The other two cases were unaffected by the serum and terminated fatally. Four of my bacteriologically examined cases contained staphylococci. In each of these cases was there more or less laceration. One of these I will describe in

in detail.

Case 1. Mrs.O. aged 24, primipara. Occipito-posterior presentation, difficult forceps case. Perineum ruptured and sutured. On the third day temp.101., pulse 110. perineal wound suppurating, removed stitches and cleansed wound with strong carbolic. On fourth day temp.104., pulse 130,uterus douched with no result. On 5th. day pulse 140, temp.105½., delirium, some abdominal pain. Injected 10cc. B.W.& Co's Serum. In eight hours the temp. fell to 101½., and the pulse to 120. A second injection of 10cc. was administered twelve hours after the first. I saw her ten hours afterwards when her temperature was 99. and pulse 102. Recovery was uninterrupted, with no further treatment save dieting and the administration of Iron. This case was remarkable for the rapidity with which the symptoms subsided. No local treatment was adopted after the first injection. There must have been streptococci present in this case, though only staphylococci were obtained.

Case 2.

Mrs. R. aet.25. primip. Normal labour on Feb.3rd. On the 16th. Feb. I was called in and found her in a state of rigor, temp.104½, pulse 160. ~~5h~~no tenderness over uterus,no headache, but sickness, thirst and sleeplessness. I ordered three 5gr. doses of quinine at intervals of four hours. On the 17th. temp. 102.,pulse 120. sickness and thirst continued. On the ~~D~~8th. as the condition was the same as on the previous day I explored the uterus and removed with a blunt curette some fragments of placenta and membranes,and washed out with weak carbolic lotion. In four hours there was a rigor, temp.103., pulse ~~DHR~~ 120. On the 19th. temp.100%5 pulse 120. Again the uterus was irrigated, and another severe rigor ensued in four hours which lasted 30 mins. A few hours afterwards I injected 10cc. B.W.&Co's serum and eleven hours afterwards the temp. was 98.5. and pulse 108. Eight hours later the temp. was 99.5. On the 21st. the temp. rose to 100.5 when I injected a further 10cc of serum. Eight hours later the temp. was normal and remained so. Streptococli were found in scrapings from the uterus on the 20th. The lochia were offensive on 17th. - 20th. then ceased altogether.

This was a very interesting case for many reasons. In the first place no vaginal examination was made during labour so far as I knew. No doctor or midwife was present at the labour, only a neighbour whose skill was limited to tying the cord. How then did the

streptococci gain an entrance? Secondly this case illustrated the ill effects of curetting, inasmuch as a rigor shortly ensued. Thirdly the effect of the serum was most marked. It was not easy to convince oneself that the same result would have been brought about by nature alone. Anaemia was a marked feature in this case as in most others.

Case 3.

Mrs. B. primip. aet.43. easy normal labour on Jan. 2nd. First symptom was a rigor on Jan. 5th. with temp. 106. No time was wasted ~~in~~ before exploring the uterus, but the results were negative. The temp. was reduced two degrees by Quinine. No pain, no sickness, but increased prostration, and weak thready pulse. On the 6th. 10cc. of serum were injected, and in eight hours the temp. fell from 104. to 100. and the pulse from 140 to 120. The following day a further 10cc. were injected, when the temp. fell to 99. and the pulse to 98. A rapid recovery ensued.

Other similar cases might be detailed, as well as cases in which the serum had no good effect whatever. Thirteen of my twenty one cases were primiparae

many instances have been recorded of success and failure by the serum treatment. It is impossible to form any estimate of the mortality from such statistics. Some apparently hopeless cases recover without any special remedy. One consequently can never be sure whether the cure is due to nature or serum. One has to remember that cases of pyogenic infection differ markedly in severity. In many cases of septicaemia the disease is so virulent and so rapid that it is unreasonable to expect any bactericidal serum to stay its progress. There is not time, in fact in such cases there never has been time for the serum to assert itself.

The Gynaecological Society of America appointed a commission to investigate the value of this serum in puerperal sepsis. The report was published in 1899 in the 60th. vol. of the American Journal of Obstetrics. There were found to have been treated with serum 352 cases with a mortality of 20.74%. Those cases of the disease not treated with serum showed a mortality of 15%. So much for statistics. In other words this new remedy increased the mortality by nearly 6%. It is a well known matter of fact that if the serum does no good it does little harm. The only ill effects which have been known to follow serum injection are certain skin eruptions, mostly erythematous, pains in the joints, usually slight, and albuminuria small in amount and transient.

Summary.

With regard to the serum treatment of puerperal sepsis we can only conclude, that though in the past it has been disappointing, it is still worthy of trial, and especially in those cases in which streptococci are known to be present.

The serum must be used fresh. Antibactericidal serums in particular keep badly. Then again as we have seen in discussing the organisms of puerperal sepsis, streptococci are not the only organisms present in a large proportion of cases, and the staphylococci etc. remain unaffected by the antistreptococcus serum.
In the more chronic cases, if we know the cause to be ~~anti~~streptococci, more than one brand of serum ought to be obtained if possible, so as to obtain as many strains of cocci as possible.

Treatment (continued).

Another method of treatment directed against the bacteria is one tried by Barrows (New York Med. Jour. July 4th. and 11th. 1903.) viz. the intravenous injection of a 1-5000 solution of formaldehyde in sterilised water. It was used after other methods had failed, and, as Barrows claims, successfully, but more evidence is required.

A silver preparation called collargol was intro-

introduced therapeutically in 1897 by Credé. It is soluble in water and in human secretions. In puerperal sepsis it has been used in the form of an ointment 15% (Ung. Crede.) and in intravenous injections 1-200. Dr. Netter(Jour. de Med. 1903, 15, page 14.) has tried it in septicaemia, with good results.

Experimental evidence, however, does not favour the belief that the intravenous injection of antiseptics has any marked therapeutic value. It has been shown that when large doses of formalin solution have been administered, which if retained would have made the blood a solution of 1-1500there was no hindrance to the growth on cultures obtained from the heart's blood post mortem. Shaw (B.M.J. July 1903.)

Unfortunately, as in the case of the antistreptococcus serum the clinical evidence is open to so many fallacies that one cannot pronounce favourably on this treatment. When a drug is discovered sufficient-accomodating to act as a deadly poison to living bacteria in the body, and at the same time to exert no very harmful influence on the cells of the host, then we may expect something from intravenous injection,

Operative measures:-

In acute cases of puerperal sepsis no operative measures are likely to be of any use save removal of the uterus. The risk is naturally very great, and when one considers that the septic infection may have spread

spread beyond this organ, the justifiability of this operation seems very doubtful.

In chronic cases , on the other hand, operative measures often succeed, though they are mostly limited to the opening and draining of abscesses. Pyaemic abscesses in the vicinity of the uterus are by no means easy to diagnose, especially on their first formation. As the case goes on the swelling becomes more defined and distinct. Suppurating tubes or ovaries must be removed. The situation of an abscess will depend of course upon its origin. Vide supra.

In cases of general peritonitis operation ought not to be attempted.

DIET.

This is not by any means the least important element in treatment. The short incubation stage of these fevers entails a strong attack on the part of the bacteria from the very commencement of the disease, and the prostration is marked from the first. To combat this we must administer an abundance of milk food, such as milk and barley water, or soda water, Benger's food, oatmeal or sago gruel, or peptonised milk. These should be administered at frequent intervals. Valentine's meat juice is advisable when there is an abscence of diarrhoea, but must be avoided if there is the slightest tendency to loosenessof the bowels. Alcohol will be necessary in all severe cases. An

An occasional egg flip may be administered, if there is no tendency to sickness; ~~but brandy will be found~~

An occasional small glass of champagne is very grateful to the patient, but brandy will be found the most useful fomm in which to administer alcohol in these cases, in quantity varying from Six to twelve ounces, in the 24 hours according to the feebleness of the pulse.

Great thirst may be relieved by injecting saline solution into the rectum, as much as it will retain. If the bowel is irritable and rejects the fluid, it may be injected into the loose cellular tissue of the abdomen. ~~or breasts~~.

Of drugs I have only derived benefit from three viz. Tinct. of Aconite, Quinine, and Perchloride of Iron. Quinine in 5gr. doses in an effervescing mixture is useful as an antipyretic. The aconite stimulat es the vagus nerve and steadies the action of the heart. It should be given in small doses - 2m. every three hours. The Iron is of great value when the acute stage is passed, and often in the acute stage if the stomach is not too irritable. It should be given in doses of 15-20 ms. every three hours.

The Prevention of Puerperal Sepsis.

If all that has been written about the causation of puerperal sepsis is correct, it follows that in theory at any rate, it always ought to be prevented. Theoretically there would be no septic conditions developing in the puerperium if the following two conditions were fulfilled during labour:-

1. Complete asepsis of the vulva and the genital tract.
2. Complete asepsis of anything coming into contact with these parts.

One feels bound to assert that it is practically impossible to fulfill these conditions completely. Nevertheless it is the ideal to aim at.

To begin with the parturient woman herself - how is one to ensure asepsis of the genital tract? In cases of a purulent antepartem discharge strong antiseptic ~~douches~~ swabbings, serviceable though they be, can scarcely render the vagina aseptic, and the cervical canal remains as impure as before. In cases of gonorrhoea the infection ~~is~~ may be still higher up. All one can do in these cases is to swab out the vagina with a strong antiseptic lotion, and trust to chance. Our theory has already broken down before the test of practice.

The vulva should be thoroughly cleansed with liquid aetherial soap containing the red iodide of mercury of a strength of 1-1000. At frequent

frequent intervals it should also be washed with an ~~ant~~ antiseptic lotion e.g. perchloride of mercury 1-1000. Needless to say before the vulva is treated in this manner the patient should have the whole body cleansed with soap and water ~~which must~~ and be finally rinsed with running water. So much for the patient herself.

The choice and preparation of the lying-in chamber needs consideration. The room should be well ventilated and well lighted, and of moderate size (in private practice). It should be sufficiently far removed from any possible source of infeation from closets etc. A thorough fumigation with sulphur is advisable. The floor should be well scrubbed with soap and water in which tdere is Carbolic Acid 1-60. Carpets should be removed, also curtains, and all superfluous furniture. Accouchment sheets of which there should be two or three should be sterilised by ~~dry~~ heat, and preferably the linen of the patient.

In practice these rules could scarcely be carried out in 1% of cases. Labours do not always take place when expected, and often enough there is no time for elaborate preparation.

Lastly we have to consider the duties of the medical or other attendant. Theoretically, one ought on being summoned to a labour to bathe the whole body, and, we are often told, put on another suit. This is a useless proceedure unless the second suit happens to be sterilised, which it never is. One

One would naturally change one's clothing if one had recently been in contact with an infectious case, in the hopes that the new suit was less septic than the old. The only rational method is to wear at labours a long sterilised overall.

The obstetric bag is worthy of notice. Edgar and Ballantyne's bag consists of two metal trays containing instruments, sterilised coat, bottles etc. In this way everything can be got ready beforehand. It will be a good thing for womankind when such a bag as this is universally adopted, but ordinary practice does not lend itself to such elaborate proceedure. One has met with obstetric bags which must have been veritable homes for microorganisms. The explanation is simple. The busy practitioner when he returns from a confinement often tired and worn out, lays his bag down in a corner of his consulting-room, and never thinks of it again until his next summons comes. Most bags have removable linings, but how seldom are they removed and washed, much less sterilised.

All instruments used must be boiled.

Disinfection of the hands.

If it is difficult to get the hands surgically pure, (and some say this is impossible), it is still more so to keep them so. To do this one must avoid all contact with anything that is not aseptic. Various methods of hand disinfection have been

adopted. Mr. Leedham-Green of Birmingham has even written a book on the subject. The following plan will I think be found as good as most. In the first place thoroughly wash the hands with soap, water and brush. The so-called antiseptic soaps are of little value. The mechanical scrubbing and washing are the main points in this first stage. Rinse well in running water and then in turpentine. Wash this off with hot water and brush with sterilised brush. Now comes one of the most important points - wash in an 85% alcohol. Finally rinse in sterilised water. The hands should not be dried. It is almost unnecessary to state that rings should be removed, and nails cut short. The soap I invariably use is Duncan and Flockhart's liquid antiseptic soap. A liquid soap is a far better cleanser than a solid.

As an alternative method gloves may be used, ~~either~~ thin rubber for preference. These are easily sterilised by boiling, but interfere slightly with the sense of touch. Unfortunately the life of a pair of thin rubber gloves is a short one, and they need looking after when not in use.

When the patient herself is prepared as stated above, sterilised towels are wrapped around her in the usual method, and now an examination may be considered safe. A golden rule is to make as few examinations as possible, thus diminishing the risk

risk of infection, and avoiding irritating the parts. Dr. D. Berry Hart in The Practitioner for March 1905 p317 states that he arranged his hospital practice for a space of two months on the non-vaginal examination principle without accident. The first case which was examined after this proved to have prolapse of the cord. External or abdominal examination cannot therefore take the place of internal.

The next point is so to manage the labour as to have a minimum of laceration of cervix, vagina, and perineum.

Lastly, don't force the separation of the placenta.

Much that has been written on the prevention of puerperal sepsis may seem superfluous, and possibly is, but it is only by thoroughly understanding how the sepsis is incurred that one realises how necessary it is to take every possible precaution.
The Midwives Act of 1902 has done, and will do more to prevent the mortality in this class of diseases.
I well remember the astonishment of a newly enrolled midwife after the first visit of the Lady Inspector. "She told me I had to get print washing dresses," with a puzzled look of wonderment, and ~~that from~~ 5/- one of the best midwives in the district! Much other goodly and necessary advice do these Lady Inspectors administer to the midwives, which before long is sure to bear good fruit.

What have we gained during recent years in the knowledge of puerperal sepsis? We have ascertained that the primary cause is organismal infection, and that the streptococci are the most powerful organisms. We have discovered the great curative method of Serum-therapy, and we are concluding, rightly or wrongly that because it is absolutely curative in diphtheria it ought to be the same in all infective disease. For my part, while aggeeing with many others that antistreptococcus serum is of benefit in many cases, and perhaps should be given a trial in all serious cases, I cannot conceive, considering the variety of strains of streptocci, and of other organisms associa-with them in septic conditions, of a serum which could possibly be a specific cure for puerperal sepsis.

We have yet to find some condition (or conditions) of which we are at present ignorant, which makes the parturient woman so susceptible to bacterial invasion, apart from the open wound and the proximity of the peritoneum. If one could ascertain the significance of the leucocytosis of pregnancy, we possibly would have a clue to the secret.