soaking for two minutes in a strong antiseptic, such as a 2 per cent. solution of lysol or dettol or iodine, or a 0.2 per cent. solution of biniodide of mercury in spirit. (This statement is not intended to apply to spore-bearing bacteria, although in most cases these also would be completely eliminated.)

These results seem to me to promise a substantially greater margin of safety than heretofore, since the hands of the doctor and midwife (and also the vulva) treated with dettol or iodine will be provided with a lasting chemical barrier against infection. By tightening up our protective network in this way, and by minimizing the opportunities for infection from the respiratory tract—either of the mother herself or a “contact”—I believe we may legitimately hope to save a large proportion of the tragic deaths following normal labour, many also of those following abnormal labour, and an immense amount of serious illness.

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DIAGNOSIS OF PUERPERAL SEPSIS *

BY

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The actual diagnosis of puerperal sepsis presents very little difficulty, since it is exceptional for sepsis to be unaccompanied by pyrexia. It is of course possible for pyrexia occurring in the puerperium to be due to some cause other than sepsis, particularly to chest complications, but in most of these the diagnosis is easy and obvious. There is, however, one group of infection that frequently occurs which is not strictly genital sepsis—I refer to pyelitis—but which is so closely allied to genital infection that I am going to include it in my contribution to this discussion. My remarks are based, as Dr. Colebrook's have been, on the collected records of nearly three years' work at Queen Charlotte's Isolation Block, and our experience and a critical examination of our records lead us to suggest that the different types of sepsis should be classified as follows:

- (a) Local sepsis in the genital tract.
- (b) Spread of infection to the blood stream—septicaemia.
- (c) Spread of infection to the peritoneal cavity—peritonitis.
- (d) Infection spreading into clots in thrombosed veins.
- (e) Direct extension along the Fallopian tubes or lymphatic spread to the cellular tissue at the base of the broad ligament—salpingitis and pelvic cellulitis.
- (f) Infection of the urinary tract, usually with *Bacillus coli*.

It is my aim to-day to point out the very great difficulties in arriving at a correct differential diagnosis of these conditions. These difficulties have often been demonstrated by post-mortem findings totally at variance

with what was expected. It may surprise many people in this room to hear that on several occasions we have found at post-mortem examination as much as three pints of pus in the general peritoneal cavity, in a case which, during life, gave rise to no suspicions whatsoever of peritonitis. It has been even more common to find septic thrombosed veins in cases in which no suspicion of their existence had arisen.

LOCAL SEPSIS IN THE GENITAL TRACT

In this group we include not only the infection of the placental site, but the infection of severe lacerations of the cervix, vagina, and perineum. Presumably all cases start as local infection, and in some, due of course to the resistance of the patient, or perhaps to the low virulence of the infecting organism, the infection does not spread further. These cases are generally characterized by moderate pyrexia—up to 102° F.—with a pulse rate raised but usually lower than 120. Frequently there is a copious offensive discharge. Constitutional signs include headache, constipation, and, occasionally, inability to empty the bladder. Examination usually reveals a dirty tongue. Abdominally the uterus is larger than would be expected at this stage of the puerperium. It is not tender either on palpation or manipulation, and on vaginal examination is mobile. There is no infiltration in the fornices, and the cervix is patulous. When there is an infected laceration of the perineum all these signs are usually present, and in addition there is oedema of the vulva. The temperature often oscillates between fairly wide margins, and a swinging temperature is no particular indication of a blood stream infection. I have described this group at some length as it is the basis particularly of Groups (b), (c), and (d).

SEPTICAEMIA

Precisely the same signs and symptoms are present in septicaemia. In some cases rigors occur occasionally, but by no means always, and the diagnosis of septicaemia is only made on obtaining growth of organisms in a blood culture. In many cases we have taken repeated blood cultures, and the variation in the number of organisms per cubic centimetre day by day has, on many occasions, been most striking, although it has been very rare for us to find a negative blood culture occurring on one day's examination in a case which before and after has given a positive result. Sometimes the obtaining of a reliable blood culture presents definite difficulties. At Queen Charlotte's we are lucky in that our laboratories are in close contact with the wards, and whenever a blood culture is required the pathologist studies the temperature chart of the patient with a view to catching her when her temperature is rising: we have found that that is definitely the time to take a sample of blood for culture. Secondly, as a routine we always culture the blood aerobically and anaerobically, and we have found that the anaerobic infection is comparatively common. The importance of diagnosing septicaemia is largely one of prognosis. The mortality in cases infected with *Streptococcus haemolyticus* is, in our hands, well over 80 per cent., whereas if the infection of the blood stream is an anaerobic streptococcus the mortality has been 20 per cent. We have so far had very few cases of any other organisms in the blood stream, so I am unable to give a percentage mortality, but it is interesting to note that in only two patients have we found staphylococcus, and both died. We are quite convinced that the diagnosis of septicaemia needs the closest co-operation between clinical and laboratory staffs, and that it can only be efficiently carried out in an institution where all facilities are available.

* Read in opening a discussion in the Section of Obstetrics and Gynaecology at the Annual Meeting of the British Medical Association, Dublin, 1933.

PERITONITIS

The classical symptoms and signs of peritonitis are entirely absent in the unlocalized general peritonitis of acute puerperal sepsis. I do not think this extremely important variety of puerperal sepsis is generally recognized. Oldfield and Pyrah recently read an account of fifty-six cases of peritonitis associated with puerperal sepsis, but their cases were totally different from those I am about to describe. During the first fifteen months of our work at Queen Charlotte's Isolation Block we had thirty deaths due to general peritonitis. In all these cases sepsis had occurred early in the puerperium, as the following table will show:

	Number of Days after Delivery							Undiagnosed
	1st	2nd	3rd	4th	5th	6th	Later than the 6th (one case as late as the 28th day)	
No. of cases	1	2	1	5	6	3	10	2

Two cases undiagnosed were found to have peritonitis on post-mortem examination, and the onset of infection in both cases was about the fourth day of the puerperium. In 60 per cent. peritonitis developed before the sixth day of the puerperium. For the last eighteen months we have endeavoured to diagnose peritonitis at its onset, and we find that an entirely new picture is presented from what have been considered the classical signs and symptoms of peritonitis. First of all we usually have a typical picture of local infection of the genital tract as already described, except that in many cases the tongue is not markedly furred, but may appear fairly clean and moist. The general expression of the patient sometimes gives a hint; she is anxious-looking, whereas in septicaemia it is not uncommon to find the patient cheerful. Abdominal examination usually reveals only a very few of a large variety of possible symptoms. Pain is rare; it occurs in, roughly, less than one-third of the patients. Abdominal tenderness may be elicited, not on pressure but rather on releasing pressure, and it is frequently noticed that if a patient complains of no pain or tenderness on comparatively deep palpation she winces visibly when the examining hand is lifted off the abdomen. The abdomen often moves well on respiration. On other occasions pain is elicited on moving the uterus from side to side of the abdomen over the promontory of the sacrum. I particularly remember one case admitted to the Isolation Block three days after delivery and some six hours after the onset of pyrexia. The only abdominal sign I found was this definite pain on moving the uterus. I opened the abdomen the same day, and found free pus in the peritoneal cavity which grew haemolytic streptococci. The patient recovered.

Rigidity has been present in only a very small percentage—about 12 per cent.; in the rest there has been no rigidity or guarding of the abdominal muscles. Vomiting has occurred in about the same percentage; it is by no means a common feature. More often the patient has diarrhoea; in fact, we look upon diarrhoea that occurs after two or three days' pyrexia as conveying the strongest suspicion of early peritonitis. By far the commonest sign is distension of the abdomen, and a markedly tympanitic note, with no other symptoms, is almost sufficient to make a diagnosis of early peritonitis. Distensions and tympanitis have been present in over 70 per cent. of our cases of early peritonitis. By the time free fluid could be demonstrated on clinical examination the case was beyond hope.

To complete my picture of peritonitis I must progress on to treatment, since the treatment of early

peritonitis is very definitely early drainage; and we have adopted the principle that the patient is desperately ill, and the smallest possible operative procedure must be undertaken. I make an incision 1 in. to 1½ in. long, just above the level of the fundus. Through this I insert a special perforated tube, guided by two fingers into Douglas's pouch, and aspirate any fluid which may be present, usually only a few cubic centimetres. This fluid has always been examined in direct films, and cultured anaerobically and aerobically. On opening the abdomen in this manner in a case of early peritonitis in which free pus has been found containing haemolytic streptococci it is most striking to observe the peritoneum of the abdominal wall and covering the intestines and omentum. It is almost always clear, smooth, and not injected, while there is free purulent fluid, with no signs of any attempt to shut off the pelvis by adhesions. I think this is one of the most important features of early general peritonitis in the puerperium, since it demonstrates the complete absence of any reaction on the part of the peritoneum. There is not even hyperaemia of the intestines, although purulent fluid containing virulent living organisms is free in the peritoneal cavity.

INFECTED THROMBOSED VEINS

Septic thrombosis of the ovarian veins is even more difficult to diagnose clinically than peritonitis. At post-mortem examination we have found septic clots in the veins of women who had a blood infection with an anaerobic streptococcus, and more frequently in those cases in which two different organisms were grown from blood. Usually these cases have repeated rigors, with a markedly swinging temperature, and often the number of organisms in the blood varies greatly from day to day.

SALPINGITIS AND PELVIC CELLULITIS

The diagnosis of these conditions is usually fairly easy, as a very definite tender mass is felt on one or other side of the lower abdomen; it is even more easily detected on pelvic examination. The temperature in these cases is usually steady—between 101° and 102°. Pain is often complained of on the affected side, and in our experience these cases are best left with local treatment only, in the hope that the acute inflammation will subside.

URINARY INFECTIONS

Infections of the urinary tract are most commonly characterized by a very sudden and unexpected rise in temperature—to as high as 104° F.—frequently accompanied by a rigor. This high temperature often falls, initially at any rate, just as rapidly to normal, only to be followed after a short interval by a further sudden rise and rigor in the course of twenty-four hours or so. Pyelitis during the puerperium rarely gives rise to pain, frequency, or dysuria; in fact, the diagnosis is often made on the temperature chart in the absence of any further findings. The diagnosis is, of course, confirmed by bacteriological examination, and to get a reliable report it is necessary that a culture should be made within half an hour of withdrawing the urine; it is extremely difficult to obtain a catheter specimen entirely free from contaminations, and contaminating organisms multiply with amazing rapidity in the urine. At Queen Charlotte's we like to make our cultures from the urine at the bedside immediately after withdrawal.

I cannot claim that this contribution has done very much to make a differential diagnosis of the various types of puerperal sepsis any clearer, but it points out the great difficulties that are present, and the extreme need for all laboratory assistance in endeavouring to save these disastrous cases.