THE PROBLEM OF PUERPERAL SEPSIS

AND ITS SOLUTION.

Thesis submitted for the

M.D. Glasgow

by

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Introduction.

There is little doubt that primitive or prehistoric woman went off by herself as the animals still do, to bear her young. Having crouched down in a semi-sitting position, she would then void her young as she would her faeces and having cleansed herself in the nearest running water, she would return to the tribe. Modern woman calls in her mother, sisters, friends and attendants. The social customs necessitate much kissing and handling by the various people. The microbe of sepsis resides unsleeping in the noses and throats of 80% of all people. It is in the air and on the skin, seeking like its host its own propagation and determined to lose no opportunity of becoming infective. It is small but in its compass lies a death that is sometimes so swift that the strongest person may be swept out of existence in a few hours.

As civilisation so called progressed, the parturient woman sought sympathy and help in her childbearing. So long as only sympathy was extended no harm was done, but with the coming of help came infection, and with infection, death. Mankind being

prone to confuse "propter hoc" and "post hoc" argued that this Death by Infection was simply the result of too little help and increased therefore their efforts at relief until in 1921 in the age group 15–45, 784 women died in Scotland from puerperal conditions. In the same year, 102 women and 627 men died from accidents so that by far the puerperal risks outrun those ranked as dangerous occupations!

There is a picture, "Dining with Caesar Borgia" in which Caesar Borgia is seen with red skull cap on his head and his sister Lucrezzia standing behind him. They are both looking at a guest who has been asked to take wine with them. The look of fear and agony on the guest's face as he lifts the priceless glass to his lips while the basilisk Borgia's eyes watch him, constitutes the "motif" of the picture. Similarly I have always been shaken by the thought that beneath all the flowers and trappings that surround the social side of childbirth, lay in wait the enemy who would make a mock of all our joy and render vain our utmost efforts surgically.

What an enemy this universal, lethal and unsleeping infection is! It kills over 70% of its victims. All our efforts at cure are unavailing.

If the patient recovers, it is only by her own miraculous staying power. How necessary and invaluable then must be Prevention. It is our only hope.

Recent investigation has established the fact that this infection is brought to the patient by her helpers! That a woman should have to bear alone the pangs of childbirth appeals to our chivalry, that she should die an avoidable death, nay that such avoidable death should be of our actual bringing, is a reproach to our profession. It must not any longer be. Apart altogether from the shame and horror of the thing to us as a profession the loss; and the quality of the loss; to all concerned is a very serious matter. Let us just analyse what it means. The Mother, after all her hopes and pain and fears, loses her life and is forever cut off from that fruition which is her highest function.

The child, if it dies, loses its rightful portion. It is cheated of its inheritance. The Husband and Father is robbed of the love and care and services of his partner. If left with the child he has a problem that is only solved at best, badly. He has to choose among the evils of a housekeeper, stepmother or relative.

The doctor and nurse suffer loss of prestige and income and the doctor definitely is shaken in that "pride of skill" which is the core of his professional being. There is a definite shock and disintegration of his fibre by a puerperal septicaemia case in his practice. The neighbours tell of the case and possible pregnancies pass him by. The State loses both a valuable citizen and the potential life increase the marriage had led it to expect. We therefore must admit that in all medicine there is no subject more important than the securing of a safe puerperium.

Surgery is important in that a person who would otherwise be maimed or dying can be restored to health by it. If he dies at the hand of the surgeon the people say, "well, he would have died anyway."

But in midwifery things are vastly otherwise. Here is a normal act turned into a tragedy. Something must be to blame for this and all look for a scapegoat. The specialist blames the general practitioner. The general practitioner the nurse, and the nurse the patient. The patient dies! The Public Health official who does no midwifery is probably the most omniscient and olympian of all our medical brothers on these tragedies. Meanwhile

the streptococcus secures a death rate among our parturient women that compels the Registrar General to say that "childbearing is a dangerous occupation."

Since graduation in 1908 these thoughts have been ever present with me, so much so, that after twenty years busy midwifery work, I never go to a maternity case without feeling the tremor of stage fright. I determined to take notes of all my maternity cases and to try, by carefulness in action, to lessen the incidence of morbidity. I tried to learn from the cases that went wrong and to improve as I went along. This is my excuse for putting down the fruits of a twenty year's fairly strenuous midwifery life as a General Practitioner. I believe that there is no better attendant for a woman in maternity than her own doctor, that there is no better place for her confinement than her own home and I hope in what follows, to demonstrate that while Institutions have their place and are admirable in many ways, it is a mistake to press for universal Institutionalism in Maternity.

What are the desiderata?

They are briefly to guide the mother from the time of conception to labour - Ante-Natal Care. To

conduct her labour so that she suffers a minimum of pain and risk. That we deliver her undamaged of an undamaged living child and that she be as well and strong during and after the natural process of labour as she was before she was pregnant. Can this be secured? In my opinion, yes, in practically all normal cases. The abnormal cases, discovered by careful ante-natal care are the fit subjects for Institutional experts. I am indebted to Professor Mitchell of the Royal (Dick) Veterinary College, Edinburgh,(1) for a kind answer to my query as to the incidence of puerperal septicaemia in animals. He writes, "I have no hesitation in saying that puerperal infections are quite common in domesticated animals. Just as in human beings a small proportion die, a large number recover completely and a number pass into chronic endometritis, etc. The rapid death of cows for instance following calving is in many cases a true puerperal septicaemia." In a further letter to me in answer to a query as to the concomitant handling in labour, he writes: "Occasionally cases of puerperal infection in cows die from an acute septicaemia. These have calved normally without any handling or interference. Many cases of difficult

parturition which have even been handled by laymen without any precautions against sepsis, never develop any fever."

"In bitches - cases which go septic are those which have been handled. It is very rare to get a case which has not been handled, developing fever."

The above is interesting, coming as it does from so high an authority as it coincides very closely with the experience of the medical profession in human beings. Asked as to the incidence etc., in wild animals, he stated he had no knowledge of these. It is, however, a matter of common knowledge that animals which have their young in holes in the ground, the wolf, badger and rabbit for instance, take advantage of a sunny day to carry out their bedding and tease it in the sunshine immediately before they bear their young. This undoubtedly is an attempt at asepsis.

The painstaking care of the ant, bee and wasp regarding all that concerns their embryo conditions is another case in point.

It is the <u>elusive fact</u> of sepsis occurring without any interference that is the lion in the path of the perfect puerperium.

Two gardeners working among rose bushes pricked their thumbs with a rose thorn. This happened with the same rose bush at the same hour, ten in the morning. One of these men was dead that night with malignant septicaemia and the other was dancing at a rustic soiree with his sweetheart. A woman was confined in a pig sty and a filthy untended one at that. She lay there all night among the muck and was found next morning by the farmer and taken into his house. Mother and child had an uneventful time. Another lady, a well known athlete, married a wealthy man and in her confinement was attended by all that money could command - specialists, nurses, etc., etc. She died in five days with puerperal septicaemia. This is what makes our blood turn to water in our veins. We know that the pregnant woman's vagina contains streptococci and other possibly lethal germs. A wavering testimony by bacteriologists classes these endogenous streptococci as really domestic pets and harmless while the same microbes on the attendant's hands are lethal in the highest degree. I shall enter into this more fully in its own place, meantime I only wish to stress its mental effect on the harassed general practitioner who has to labour under

the Argus eyes of the specialist and Public Health Official.

But even if we could eliminate by care the proportion of so called "exogenous cases" the result would be an enormous fall in the incidence of puerperal septicaemia. To show the magnitude of the condition, I quote from the Registrar General's report for Scotland for 1929 (2).

Year 1929.

Total Births - 92,880.

Total Deaths in Pregnancy.	Deaths in Puerperium.	Deaths from Sepsis.
638	390	221

Age Groups in Septic Deaths.

Age.	Deaths.
15 - 25	50
25 - 35	110
35 - 45	59
45 -	2

There can thus be no question of its enormous importance to the State. It is, however, of as great importance to the General Practitioner.

Every doctor as he is launched from Alma Mater into General Practice, finds that if he is to build

a practice securely on the belief and trust of his community, he must secure the confidence of the female portion of that community. There is no method more likely to do this than the ability to pilot the women folk through a safe and successful maternity case. It lies at the root of all successful family practice. Any disaster here is irreparable. If successful, the mother, confident and pleased, brings her children and her husband to him. He is the good and beloved physician. In time, as the children grow up and enter into their inheritance of wedlock, they seek the services they have proved in the past and so the good work goes on. I wish to emphasise this atmosphere of warm personal regard that exists between a well proved medical attendant and the patients he has known and treated for many years. In my opinion, it is inestimably the best atmosphere for a woman in her maternal trials. In an Institution, especially a Public Institution, she enters an atmosphere where she ceases to be warmly and personally regarded as an important entity she becomes a "case". Her nurses, her doctors are all strange and however kind, able and gifted, they may be, nothing can get over the fact that they are strangers to

whom she is a "case". She, therefore, has more anxiety and fear and loneliness in an Institution than in her own home. There are other drawbacks to Institutional treatment of midwifery cases that I will emphasise in their own place. This - the Psychological one - is very real and very important.

Anything then, that upsets this delightful atmosphere of mutual regard and trust reacts very badly on the General Practitioner's local outlook. The women whisper together and if the least tangible thing can be made out against the doctor or nurse - and even if nothing can be so demonstrated - there is a serious set-back to the practice of those concerned. The public look on a death in private practice as being due probably to the doctor's hand whereas in an Institution, it is the hand of God. It is all the more regrettable, therefore, that the leaders in the Profession safely entrenched behind the walls of their Institutions should have lent themselves to the "fama" that puerperal sepsis is the product of faulty general practitioner and nursing work, and that the cure for the position is universal Institutionalism for Maternity cases.

The situation is all the more Gilbertian in that

the facts of sepsis are all against them. The sepsis rate is highest in Institutions and lowest amongst Nurses' cases.

I append a table for Glasgow in 1925 taken from a special Report sent me by Mr.Jones of the Corporation, which shows this effectively. (3)

PUERPERAL SEPSIS.

Cases and Deaths occurring in Glasgow during the year 1925.

	Births	Total Cases	Cases per 1,000 Births	Deaths	Deaths per 1,000 Births
Midwives Cases - in 20 Assisted by Doctors.	11,949	90	7.5	14	1.2
Maternity - Outdoor.	3,840	43	11.2	8	2.1
Maternity - Indoor	2,897	49	16.9	26	9.0
Oakbank	138	2	-	2	-
Barnhill	35	2	-	1	-
Southern General Hospital	82	1	-	1	-
Beyond Boundaries, per Glasgow Institutions.	-	4	-	1	-
No one (Abortions)	-	19	-	2	-
Doctors' Cases (In 14 assisted by Midwives)	7,414	90	12.1	17	2.3
Institutions and Others with no cases.	569	-	-	-	-
	26,924	300	11.1	72	2.7

In three further Notifications a changed diagnosis was received:-

(1) Acute Tubercular Peritonitis, with T.B. Salpingitis of recent origin. (Result of P.M.)

(2) Threatened Abortion.

(3) Aseptic Abortion.

270 of above Births are not included in Glasgow 1925 Total, as they were indoor Maternity Cases, with Home Address beyond the Boundary.

Public Health Department,
GLASGOW: 18th February, 1926.

On the other hand, it is only fair to show how good an Institution can be so for comparison I append the figures for the E. End Lying-in-Home, London, given in the Report of the Medical Officer of that Institution. (4) This Institution is run by a General Practitioner who has, of course, thereby won special knowledge and skill.

During the year the following cases have been treated:- <u>In the Home</u>.

Mothers delivered	1,087
Infants born alive	1,066
Infants stillborn	30
Sets of twins	7
Sets of triplets	1

<u>Out-Patients</u>.

Mothers delivered	1,092
Infants born alive	1,084
Infants stillborn	23
Sets of twins	13
Sets of triplets	1
Midwives' Visits.	18,803
Cases visited by Doctors	122

The work of the Ante-natal Department has been much facilitated by the improvements in the consulting room, and the value of this work to the mothers can hardly be exaggerated. Our figures show that the nearer we can approach to a physiological pregnancy, the fewer are the risks and danger run by the mother and child during delivery. We have thus been able to avoid all necessity for Caesarian section, we have had only one case of Eclampsia and that not a severe one, while the number of instrumental deliveries has been very small and the number of deaths of children due to instrumental delivery, has, I am pleased to say, been reduced to a minimum.

The Post-natal Department too, has fully justified its institution. Many little troubles, both of mother and child have been seen to and put right, which, if left, might have caused severe disease.

One mother after a very difficult confinement, developed Pelvic Cellulitis and was sent to the London Hospital for operation, and one, who was admitted on account of illness, became seriously ill after delivery and died from Septicaemia.

Safer midwifery - the vanishing of sepsis therein - will not accrue from a falling out among ourselves. The specialist must come in as a help to the less expert general practitioner and he again must support and not blame the nurse. By all means discountenance slip shod work but let us recognise the subtle and widespread nature of this infection. Let us admit it can slip beneath our guard. At least it is only fair to the general practitioner that he should have the same shelter as our institutions have. The problem of puerperal sepsis will not be solved in an atmosphere of blame. It has been urged - and rightly urged - that the graduate has too little training in Midwifery. One prominent authority (5) suggests that before a graduate be capped he should put in a year unpaid as an intern in a Maternity Hospital. This is very good counsel provided that the general practitioner, then adequately trained, has the maternity work of the nation in his hands. If it is to be all Institutional then there is no object in so equipping the general practitioner.

I have written here at length on this aspect in the endeavour to show the importance of the fact

of puerperal sepsis to the general practitioner. It is no doubt important also to the specialist. The figures show that he sees more sepsis in midwifery than any other worker in the profession. I am concerned, however, with the approach to puerperal sepsis from the general practitioner's standpoint only and will not and am not qualified to deal with the specialist's outlook.

Dr. Arnold W. W. Lea (6) gives a list of the mortality and morbidity tables of Maternity Hospitals in different countries. The lowest percentage of febrile cases was held by Vienna (Schauta) with 6%. The highest by Copenhagen (Meyer) with 46.9%. Rotunda (Dublin) Tweedy 7.35%. Queen Charlotte's Hospital, London, 21.6%. These figures were for the years between 1893-1909. He was under the impression that the sepsis rate in general practice was much higher than that of lying-in Hospitals, but confesses he has no statistics to go on.

Dr. Jellett (7) states that, "there has been no improvement in the death rate among women in childbirth in the past twenty years."

In this brief introduction to the subject of puerperal sepsis I have endeavoured to show:-

i. How terrible an event it is socially and economically.

ii. The immense importance of successful and safe midwifery and to define such.

iii. How widespread it is in Nature and the danger there of interference.

iv. Its relative importance to the individual, her relatives, the State, the general practitioner and the specialist.

v. The importance of the general practitioner being the attendant.

vi. The spheres of general practitioner and specialist and the necessity of team work in the securing of good results.

vii. The size of the problem as proved by statistics.

viii. The fact that we are not making the progress we should in abolishing sepsis in midwifery.

Historical Outline.

As far as the records go back in Medicine, they show an acquaintance with and knowledge of puerperal sepsis. Hippocrates (8) (460-377 B.C.) describes the disease. He believed it was due to lochial suppression. This lochial flow instead of coming out of the body turned inwards and carried its poison through all the tissues. He therefore advocated free purgation as the cure of this disease. He records his belief, however, that it was almost uniformly fatal. There is, of course, no record of its relative prevalence then. It is interesting in view of the modern belief in insuction as a cause of puerperal sepsis that Ambrose Pare in 1575 believed puerperal sepsis was caused by cold air entering the womb at childbirth. Jellett (9) in 1929 advised the crouching position in delivery and not the left lateral position in order to prevent insuction. Strother (10) used the term "puerperal fever" as an entity for the first time. The actual infection, was however, not generally understood. This was ascribed to various factors ranging from an act of God to bad drains. Dr. Thos Kirkland (11) dwelt on the importance and frequency of Peritonitis occurring

in these cases. He was convinced that it was a specific contagion and advocated raising the patient in bed in order to allow free drainage of the uterine contents.

Dr. Gordon of Aberdeen (12) was the first to draw attention to the fact that the disease was carried from one patient to another by doctor or handywoman or other attendant. He says, "I had evident proofs of its infective nature - which operated more speedily than any other infection with which I am acquainted." Puerperal sepsis was now recognised to be a specific infection affecting pregnant women and the necessity of isolation, plentiful fresh air and cleanliness was insisted on by the Profession. Thus Dr. Charles White (13) who practiced in Manchester in the latter half of the eighteenth century was of opinion that foul air and bad surroundings, filthy bedding, lochial retention and constipation were the causes of puerperal sepsis. He gives a picture of the conduct of labour at that time. "As soon as she is delivered if she is a person in affluent circumstances, she is covered up close in bed with additional clothes. The curtains are drawn round the bed and pinned together, every crevice

in windows and doors is stopped close, even the key-hole, the windows are guarded not only with shutters and curtains but even with blankets the more effectually to exclude the fresh air, and the good woman is not supposed to put her arm or even her hand out of bed, for fear of catching cold. She is constantly supplied out of the spout of a teapot with large quantities of warm liquors to keep up perspiration and sweat, and her whole diet consists of them. She is confined to a horizontal position for many days together whereby both the stools and lochia are prevented from having a free exit. This happens not only from the posture of the patient but also from the great relaxation brought on by the warm liquors and the heat of the bed and the room, which prevent the overdistended abdominal muscles from speedily recovering their tone, whereby they are rendered unable to expel the contents of the abdomen, which, lodging in the intestines many days, become quite putrid." Again he says, "Most, if not all, of these disorders which are usually supposed to be peculiarly incident to the puerperal state are either the effects of mismanagement by the accoucheur or nurses or else arise from the patient's own imprudence. They may always

except in lying-in hospitals - be avoided."

He recommended practically all the measures practised today such as absolute cleanliness of patient and attendants, cool airy room, quiet and restful atmosphere, light diet and gentle aperients. When a patient developed fever, he advocated rigorous isolation. He also ordered disinfection of the patient's room, etc. If these were the conditions of the puerperim in the best circles what must have been the state of those less blessed with the world's goods! For this we must go to Dickens,(14) who drew for all time the portrait of the midwife of the period in the famous Mrs Gamp. Dirty, ignorant and debauched she was too often the precursor of the undertaker who held her in high regard accordingly.

In 1843, Oliver Wendell Holmes, (15) the famous author of "The Autocrat of the Breakfast Table" etc., published an essay on the contagious character of puerperal fever. He maintained therein and led much evidence to show that the disease was frequently carried demonstrably even from patient to patient by doctor, nurse or attendant. These views attracted little notice at the time and what there was of it was hostile.

In the early 19th century, the contagious nature of the disease was well known but accurate knowledge thereof was due to Semmelweiss (16) who practised in Vienna in 1846. He was appointed in that year to the lying-in Hospital there, under Johann Klein. This hospital had two divisions, one attended by students, the other by nurses. In the first, out of 4010 deliveries, there was a mortality of 359 women - 11.4%. In the second out of 3,754 deliveries 105 died or 2.7%. This to him was very striking and he pondered over the facts and tried to discover the cause. One of his colleagues died following a wound sustained at a post mortem on one of the puerperal cases. The appearance of the corpse was similar to that of the septic cases in the puerperium. He came to the conclusion therefore, that the cause was identical in the two classes of cases. The students were in the habit of going direct from the dissecting rooms to the lying-in cases with only the scantiest of cleansing of hands etc. He insisted on both nurses and students thoroughly cleansing the hands with chloride of lime water, with the result that the mortality fell to 1.27%. Not content with this, he carried out experiments on rabbits. He inoculated

them with lochia from septic cases and found that they contracted a disease in all points the same as puerperal fever. He maintained to the end of his life that the essential cause of puerperal fever was the introduction at labour into the vagina of decomposing animal organic material by the attendants' hands. He held that infection could be prevented except in a small proportion of cases of auto infection by careful cleansing of the surroundings of the lying-in woman. Towels, sponges, instruments, hands, etc., were all to be sterilised and the infected separated from the healthy. His views were met by a storm of ridicule. Drs. Skoda and Rokitansky supported these views but Klein took the opposite views and drove Semmelweiss from Vienna. In 1851 the methods of Semmelweiss formed the subject of an address by Arneth to the Academy of Medicine, Paris. Professor Simpson of Edinburgh in the same year, published a communication embodying the suggestions of Semmelweiss which he adopted (17). He used a solution of potassium Cyanide as a hand disinfectant.

Tarnier (18) advocated the isolation of all puerperal women as the most efficient step in the prevention of puerperal fever. He was the first to

use the term "Puerperal Septicaemia". Pasteur (19) in his classical experiments on the fermentation of milk in 1860-3 proved that the changes of fermentation were due to the presence of micro organisms in the air. In 1879 in observation at the Hotel Dieu on lochial discharge, he found the streptococci in lochia and throughout the tissues of the body of those who died of puerperal sepsis. These were applied to Surgery in general by Lister (20) who endeavoured to produce an atmosphere inimical to microbes in the atmosphere of his operating room.

Professor Matthews Duncan (21) in 1870 published his work on childbirth and Maternity Hospitals. At that time there was no registration of Births and Deaths worthy of the name. Nevertheless he succeeded in collecting statistics of both private and institutional cases and his review of these led him to the conclusion that the puerperal mortality in England was not less than 1 in 120 of all women within four weeks of delivery. In 1865 Meyerhofer identified the streptococcus as the causal agent. It will naturally be thought that with these facts as to puerperal sepsis being established the incidence would be greatly lessened. Such, however, has not been the case. We find that in 1879 the incidence per 100

births in England and Wales was 1.6% (22). (registrar General's report) Dame Janet Campbell (23) finds in 1900 "that deaths from puerperal sepsis accounted for "2.18 per 1,000 births, while in 1922, the proportion was 1.46. Throughout the intervening years, it has varied from 2.6 to 1.3 per 1,000 births. This shows that though it is known that the general death rate has been reduced by a third and the infant mortality halved since the beginning of the century yet the Maternity Mortality is little lower than it was 20 years ago." This report by Dame Janet Campbell attracted and still attracts a great deal of notice. The general trend of it seemed to suggest that the general practitioner was more or less of a danger to the maternity case and there arose a strong tendency to force institutional confinement on the patient. We find that at a meeting of the Section of Epidemiology of the Royal Society of Medicine, Dr. Evelyn D. Brown (24) read a paper on, "The relation of puerperal septicaemia to other infective diseases including a reference to the propriety or otherwise of admitting Maternity cases into hospitals." Her analysis of statistics over a number of years led her to conclude that there was no association be-

tween the rise and fall of Scarlatina and puerperal septicaemia. There was no parallelism between the incidence of puerperal septicaemia and other infective fevers with the possible exception of Measles where there was a significant parallelism.

Dr. John S. Fairbairn (25) published an article on "Maternal Mortality in Midwifery Service of the Queen Victoria's Jubilee Institute." He says, "Throughout the years for which figures are available the mortality in midwifes' cases is under half that of general rate for England and Wales. The consistently lower mortality rate may be accepted as demonstrating the value of the services of a well trained corps of midwives....and as bearing out the lower maternal mortality of those European countries such as Holland, Italy and the Scandinavian natives which have had for generations a well organised midwifery service." On page 50 he says, "It may be worth while to take as an example of what might be arrived at throughout the country the figures of an institution which has succeeded better than any other in reaching the highest standard of obstetric practice, the East End Mothers' Lying-in Home. The ordinary work is carried on by midwives. The ante-natal

supervision, and rendering of medical aid when called for by the nurses, are in the hands of a local practitioner. Its results are obtained amongst its own patients; it neither sends elsewhere its complicated midwifery nor takes difficult midwifery from outside its own patients. Maternal mortality is 0.67 per 1000 deliveries, i.e., it has one death in 1,500 deliveries instead of 1 in 250, which is the general rate of the country.....If the results shown by the Q.V.L.I. midwives with the help of local practitioners obtained throughout the country maternal mortality could be more than halved."

Dr. Florence E. Barrett (26) in a most exhaus - tive article begins by postulating that "certain facts as to how puerperal septicaemia arises are well known. (1) There are undoubted cases of auto infection - from a pre-existing focus in the patient herself which is roused to renewed activity immediately after delivery when the puerperal woman passes from a condition of comparative immunity to one of special susceptibility. (2) Introduction of pathogenic germs by accoucheur. (3) Through lacerations followed by careless nursing, etc., she goes into minute details as to the conduct of the puerperium and

concludes by emphasising that she has been dealing with normal cases as operative ones are best in an Institution.

In 1928, Dr. Parlane Kinloch (27) established the fact that the streptococcus Haemolyticus was the chief microbe in the causation of puerperal septicaemia. He also proved that it was carried in the nose and throat of doctor, nurse, visitor or patient. He came to the conclusion that it was a droplet infection conveyed by hands or instruments etc., and in a table showed that the death rate from puerperal septicaemia per 1,000 maternity cases was 2.8 in the practice of midwives, 6.9 in that of doctors and 14.9 in in-patient institutional practice. He goes on to say, "The most striking feature of the Table is the extraordinarily high mortality especially from sepsis, among institutional cases, even when these are corrected so as to comprise only such cases as have had no previous interference and have been admitted to hospital without any interference by doctor or midwife." These articles gave pause to the clamour for universal Institutional midwifery. It had been thought that owing to various causes such as the pressure of relatives and patient on the general

practitioner to "do something" forceps abuse was a prime cause of puerperal septicaemia. He showed, however, that "there were no deaths among midwife cases in which forceps were used, and that the evidence suggested that so far as the midwife cases were concerned the application of forceps had no prejudicial effect."

Finally in January, 1391, Dr. J. Smith (28) of the City Laboratory, Aberdeen, conclusively proved that the streptococcus Haemolyticus was the causative factor in practically all puerperal septicaemia and that it was carried in the nose or throat or ears of patient or attendants. On page 6 of that report he says, "it may be concluded that most workers believe that puerperal fever is mainly caused by the Streptococcus Haemolyticus and that undoubtedly the majority of deaths are due to that organism."

I have endeavoured in the above to show the historical position as regards puerperal sepsis. How from Hippocrates down to the present day, light as to the cause of puerperal sepsis has gradually filtered in. It has been the work of countless searchers, many of them unknown; but the result has been that today we are in the possession of the

secret as to the actual cause and manner of infection. It only follows that we apply this knowledge to ensure prevention; for once the patient is infected the chance of doing much to prevent a fatal issue is a slender one. It is great gain that we are out of the wood of bad drains, mephitic state of the atmosphere, and all the other comfortable phrases we once used to cloak our ignorance and soothe our consciences.

Bacteriology.

Puerperal Sepsis is a septicaemia. Septicaemia literally means "putrefying blood" or a condition in which putrefying matter has been introduced into the blood. (29)

Professor Muir (30) says, "when bacteria gain a foothold and multiply in the blood, the term septicaemia is applied." He goes on to demonstrate varying characteristics of different bacteria in the blood. He says it is only rarely that bacteria in the blood can be seen microscopically but they can be got on culture. He shows that the microbes are more abundant in the capillaries of the internal organs than in the peripheral circulation. He says that in some classes of virulent streptococcal infection, e.g., meningococcus septicaemia, the microbes multiply actively in the blood and rapidly cause a fatal result. He considers this class of case the gravest of all. The mere presence of bacteria in the blood does not necessarily mean a fatal issue. It can readily be understood that where we have bacteria thus in the blood they may settle in greater or smaller numbers in some favourable place in the body, some place that offers an inviting

surface or pabulum for their activities. When this happens we get abscess formation and the name given to this is pyaemia.

Muir states (30a) that abscesses form thus in two different ways. "They may settle in the capillary walls of various organs where they grow and produce minute abscesses. This is typically exemplified in staphylococcal infection, e.g., Osteomyelitis. Certain organs are specially prone to this, viz., heart, kidneys, lungs. On the other hand organisms may be disseminated by portions of infected thrombus which become mechanically arrested and thus produce suppurating infections. It may thus be seen that after infection has occurred it may (1) remain localised, (2) be carried by the blood stream and produce like inflammatory lesions in other parts of the body, (3) may multiply in the blood and become thus a true septicaemia."

Again Muir and Ritchie (31) state, "It is important to draw a distinction between the mere presence of organisms in the blood - bacteraemia - and their active multiplication in the blood - septicaemia. The former condition represents merely an overflow of bacteria from the tissues into the

blood. But there is no progressive multiplication in such cases and we may say in bacteraemia that organisms would soon disappear if the source of supply were removed."

In addition to these two variations of infection there is a third called sapraemia or toxaemia. Even if an infection remains local, the bacteria simply multiplying in situ, they are capable of serious disturbances. Locally they have a digestive action on the tissues. Thus Muir and Ritchie (32) divide the local action into two (a) a change of a neurotic character and (b) a reparative change on the part of the body cells of a defensive nature. The cells are guided towards the scene of action by a force called "chemio-taxis" and in acute inflammation the white corpuscles of the blood especially the polymorphonuclear type are the main cells involved in the struggle with the invader. Observation has shown that the mother cell of these polymorphs - the neutrophile myelocyte-increase in the bone marrow in infections that call for a long continued struggle. The connective tissue reaction is really of two kinds (a) increase of functional activities - phagocytosis, secretion, etc., and (b) increased cell growth and

division. It is not yet known whether secretion or exudate, is defensive or a confession of destruction. (33). The local lesion may be very oedematous, may be haemorrhagic or may have abundant exudate. There are always present the cardinal four characteristics of inflammation. "Calor, Dolor, Rubor, Tumor."

Though the bacteria are and remain local, they elaborate substances called toxins. These toxins are of two kinds; exotoxin which the bacteria exudes or forms from its media, and endo-toxin which remains inside its body. Antitoxin can be prepared by injecting suitable and graduated doses of exotoxin into animals. The body cells of these animals acquire the power of elaborating antitoxin and after this process has taken place blood is taken from these animals, suitably acted on in the laboratory and put up as antitoxin.

So far this has not been found possible with endotoxins and there is much doubt as to whether what is got in the culture tube represents what is elaborated in the body or lesions of the bacterium. (34).

The effects of the toxins on the body in Sapraemia or Toxaemia are shown by fever, rapid pulse, rapid breathing, delirium, sweating and collapse.

Many organisms have been isolated from the lochia and uterus of puerperal fever patients. The commonest are Streptococcus Pyogenes. This is an organism that grows in chains of cocci or round bodies, each coccus being about 1 mu in diameter. The chains vary in length depending on their environment. It is Gram Positive. It grows readily on nutrient agar at 37°C. The addition of blood or serum enhances growth therefore blood agar or serum agar is used as a routine medium for growth of streptococci. The appearance of the growth on agar is that of a collection of small circular semi-translucent discs which show a great tendency to separate. The separate colonies remain small, rarely exceeding 1mu in diameter. On blood agar the colonies are surrounded by a clear zone of laking or haemolysis due to haemolysin produced by the organism.

In Peptone bouillon a stab culture shows about the second day a thin line which in its subsequent growth becomes a row of minute rounded colonies of a whitish colour. In milk an acid reaction but no clotting is produced.

It ferments Glucose, Lactose, Saccharose and Salecin. It produces no fermentation of Inulin so

differing from the pneumococcus.

Varieties of Streptococci. At one time streptococcus pyogenes and streptococcus erysipelatus were supposed to be two distinct species. It is now thought that they are really the same. It is all a matter of varying virulence (36). Streptococci have been classified according to length of chain thus streptococcus longus - long chain, and streptococcus Brevis, short chain, varieties. St. Conglomeratus so called from its forming in bouillon minute granules in very long chains. As a rule the long chained variety is pathogenic and virulent in the human body whilst the short variety is non virulent. There are, however, exceptions to this rule. Taking all the observed facts into consideration, pathogenicity and morphology cannot be relied on as a basis of classification. (37).

It is therefore necessary that other means should be employed and these are fermentation and haemolytic tests, Andrewes & Horder (38) by using, (1) Milk clotting, (2) fermentation with acid formation of Saccharose, Lactose, Raffinose, Inulin, Salicin, Coniferin and Mannite have differentiated six species of which five occur in man.

1. Streptococcus Mitis, a short chained variety which occur chiefly in saliva and faeces as a saprophyte. It ferments saccharose and lactose. Produces an acid reaction in milk but no clotting.

2. Streptococcus Pyogenes, which is the most pathogenic.

3. Streptococcus Salivarius which corresponds to the streptococcus Brevis of the mouth. It ferments saccharose, lactose and raffinose and rarely inulin. It clots milk.

4. Streptococcus Anginosus corresponds with Streptococcus Conglomeratus. Ferments saccharose and lactose and sometimes raffinose. Is actually haemolytic. Clots milk and does not grow on gelatine at 20°C.

5. Streptococcus Faecalis from the intestine where it swarms, is short chained and has great fermentative powers. It forms sulphuretted hydrogen and does not haemolyse.

6. Streptococcus Equinus. Common in horse dung and the air of towns. Ferments saccharose and forms no acid in milk.

To all these types there are, however, variants and this also extends to their fermentative powers.

Schottmuller (39) used growths on blood agar as a means of differentiation. His medium was two parts human blood and five parts melted agar. He distinguished.

1. Streptococcus Longus or Erysipelatus which formed grey colonies with marked haemolysis.

2. Streptococcus mitior or viridans, a short chained green coloured variety with little or no haemolysis.

3. Streptococcus Mucosus Encapsulatus which produced capsules in slimy colonies. A combination of fermentative and haemolytic tests would, of course, greatly strengthen ones powers of classification of streptococci and Dr. Gordon (40) summarises the characteristics of the three chief streptococci thus:-

Streptococcus	Haemolysis	Raffinose	Mannite.
Pyogenes.	+	-	-
Salivarius.	-	+	-
Faecalis.	-	-	+

Attempts have been made to classify streptococci serologically, mainly by agglutination with specific antisera. These seem to show that streptococci are a heterogeneous group (41). From this it is reasonable to say that the streptococcus group is variable as to

form and as to behaviour and it is difficult to be sure that one group does not pass into another or that a form which may be non virulent today may not be exceedingly virulent tomorrow. Everything really depends on the environmental conditions.

Staphylococcus Albus. This is a spherical coccus .9 mu. in diameter. Grows readily in all media at room temperature. Is aerobic as a rule but can also grow under anaerobic conditions. It stains readily with all basic aniline dyes and is Gram positive. The growth is intensely white in colour.

Staphylococcus Pyogenes Aureus. Is similar in character to Staphylococcus Pyogenes Albus but the growth is yellow in colour.

Bacillus Coli Communis. Grows in bouillon or agar. Is 2.4 mu. long and .5 mu. broad. There may be longer forms up to 10 mu. or shorter coccus-like forms are sometimes seen. Motility is variable. It stains with Carbol-fuchsin and is Gram negative. It has numerous flagellae springing from all round the organism. (42). It has the power of curdling milk with acid formation.

When there is a mixed infection with other pyogenic organisms, cultures for diagnostic purposes

should be made on ordinary nutrient agar. One thus gets a satisfactory growth of the other bacteria present as well as Bacillus Coli. (43).

Bacillus Aerogenus Encapsulatus. This is the bacillus of gas gangrene so prominent during the Great War. It is 4-6 mu. long and is of varying thickness. Its ends are rounded. Some forms are filamentous, some short and square ended some almost like cocci. They stain readily with the basic dyes and are Gram positive. In the body tissues it forms a distinct broad capsule though this may be absent in media. Spores are produced in fluid media. These are sometimes at the centre, sometimes almost at the end of the bacterium. It flourishes on most media but is a strict anaerobe.

Bacillus Pyocyaneus. This organism is a small rod 1.5 - 3 mu. long and less than .5 mu. thick. It is very motile with a terminal flagellum. It does not form spores. It stains readily with basic dyes but is Gram negative. It grows readily on all media at room temperature and forms a green pigment therein. It liquifies gelatine. It has a distinct pathogenic action in certain animals. Thus a small dose injected into a rabbit causes a local suppuration. A larger

dose causes a septicaemia. Intravenous injection causes septicaemia if the dose be large but sometimes only a chronic wasting with albuminuria occurs. (45).

Pneumococcus. This is a small oval coccus about 1 mu. in longest diameter. They are arranged in pairs and sometimes in chains of four to ten. Their free ends are lancet shaped. They are surrounded with a capsule. The organism is Gram positive. It is difficult to cultivate outside the body from the sputum, the best medium being blood agar. Their appearance in culture is very similar to streptococcus pyogenes. In dried sputum or blood, it retains both vitality and virulence for a considerable time. (46).

Sometimes the Pneumococcus grows in chains and this fact and the fact that streptococci sometimes develop capsules, has led some observers to consider the actual relationship of these two groups.

Pneumococci do not exhibit haemolysis while streptococci do. They are soluble in bile. Streptococci are not. Pneumococci ferment inulin. Streptococci do not. Certain workers, however, by treating pneumococci with optogrine, have succeeded in transforming them into streptococcus viridans and back again into pneumococci. (47). The above shows the close resemblance between these two groups.

In the above, I have endeavoured to define exactly the meaning of septicaemia, bacteraemia, pyaemia and sapraemia or toxaemia. The actual pathological processes have been described and the results shown.

The causal microbes have been named and their morphology and habits described.

The general belief that the most serious infection is caused by streptococci and the streptococcus haemolyticus specially has been stated. It has been also sought to emphasise the heterogeneus character of streptococci and that there is a very nebulous line of demarcation between the streptococci themselves as species and that there is little to mark them off as a different species from the pneumococcus. This has a bearing on the clinical work which follows.

Etiology.

"Puerperal Infection is the general term applied to all infective conditions which arise from the entrance of organisms into the wounds in the generative tract in connection with labour or the puerperium. It is essentially wound poisoning or wound infection and is strictly comparable to surgical wound fever. (48).

It has long been known that the streptococcus pyogenes was responsible for the severe and grave forms of puerperal fever and the researches among others of Drs. Fitzgibbon and Bigger of the Rotunda Hospital, (49), (50), Dr. Parlane Kinloch (51) and Dr. J. Smith of Aberdeen (52), have established that the streptococcus haemolyticus is the predominant - in fact almost sole-factor in the causation of puerperal septicaemia. Drs. Fitzgibbon and Bigger draw the following conclusions from their investigations.

1. That acute puerperal infection is almost invariably due to streptococci most commonly haemolytic.

2. That when haemolytic streptococci are obtained from any part, the prognosis is most serious and probably the infection is always exogenous in

origin.

3. That non haemolytic streptococci from the uterus is serious and from the blood most serious.

4. That non haemolytic streptococci from the uterus, the blood or both is probably often of an extra genital origin.

5. That staphylococcus aureus, bacillus coli and gonococci are likely to produce local infections but do not tend to become general.

They also found:-

1. Streptococci from the vagina before and after delivery in 68% of cases. They may be regarded therefore as among the normal flora of the part.

2. Out of 108 vaginal swabs, haemolytic streptococci were found only in two, and streptococcus pyogenes never.

3. The predominant non haemolytic streptococci were streptoccus faecalis and streptococcus mitis.

4. Non haemolytic streptococci were found in 20% of post partum uteri.

They further conclude that "the commonest form of puerperal sepsis that caused by streptoccus haemoliticus (and particularly streptococcus pyogenes) is due to exogenous infection. Non haemolytic strepto-

cocci do occasionally cause puerperal sepsis. They are present in the vagina of most pregnant women and are found fairly frequently in the uterus post partum. Normally they are saprophytes but are opportunists as regards pathogenicity. Puerperal sepsis due to a non haemolytic streptococcus is endogenous in origin. The cause is the bacterium plus some unknown factor - local or general lowering of the patient's resistance or exalted virulence of the streptococcus or both."

Dr. Kinloch (51) in his report page 30, states, "it has been shown conclusively that the streptococcus haemolyticus plays by far the most important role in the causation of puerperal sepsis." On page 27 of the report, he states that "in his series of cases infection due to streptococcus haemolyticus was the cause of all deaths except one. His researches also proved that "even in cases of local infection only the streptococcus haemolyticus can be obtained in pure culture from the uterus in the majority of cases."

He found it impossible to give any differentiation into special types of the streptococcus haemolyticus from scarlatinal, puerperal or pyogenic sources. He found, page 28, that "the streptococcus haemolyticus cannot by any known method be grouped

into varieties causing puerperal fever, scarlatina, erysipelas or pyogenic infections."

Dr. Smith (52) in January of this year, issued a report and in a summary on pages 40-41, he states inter alia, "out of 18 cases, 15 were caused by streptococcus haemolyticus alone. In 13 of these infection was extrinsic in origin, 11 of these being four attendants. In only 1 case was the infection autogenous - a septic focus in the hand of the patient. In 2 cases of septic abortion due to streptococcus haemolyticus one was extrinsic and the other intrinsic in origin. In infections due to bacillus coli, it was found that these originated in bowel or bladder."

From above facts, it is established then that the cause of puerperal sepsis is the streptococcus haemolyticus and that it is practically always exogenous. There remains the unknown factors that determine invasion - The Predisposing Factprs, as they have been called. Anything that affects the general health must weaken the patient's resistance to infection. It is a matter of common knowledge for instance that a gardener, ostensibly a strong and healthy man, may be pricked by a rose thorn from

which death by septicaemia ensues in a few hours' time. Was this due to enormous exaltation of the microbes virulence or weakness in the body defences of the victim? We have no means of knowing. We know only that it happens. The remarkable thing in regard to streptoccal infections is that one attack if recovered from, does not fortify against another, thus Park & Petruschky (53) tried a most interesting experiment. They inoculated a man suffering from malignant disease with streptococcus erysipelatous. He developed an attack which lasted ten days. On its subsidence, he was re-inoculated. He developed a new attack which ran the same course over the same area. This was repeated ten times with the same results. From the experience clinically of twenty three years, it has not seemed to me that the debilitated were more prone to streptococcal infection than the well nourished. It is notorious that such diseases as erysipelas, pneumonia and scarlatina are highly recurrent. One of my cases developed erysipelas of the left side of the face fourteen times in three years. She eventually had her tonsils and adenoids removed and thereafter never had an attack. We therefore must look to every possible

source of ill health or nidus of infection in the maternal woman. Among conditions which may lead to infection one must deal with unhealthy teeth, tonsils, nose or ears. Septic tonsils and adenoids should be dealt with surgically and carious teeth extracted. It is wrong to stop and preserve these as this simply converts an open infection into a confined one. It is very instructive to examine X-ray plates of the mouth in cases of filled teeth etc., especially if the tooth has been killed by nerve extraction. Even though there be no symptoms of infection, the X-ray plate shows that two out of three of these teeth are active foci of infection. The only remedy is extraction and this should be done early for obvious reasons. It prevents further absorption of toxin and it obviates shock to the women in the later stages of pregnancy. The late Dr. Robert Jardine (54) says, "to lessen shock an anaesthetic should be used even in small operations or extraction of teeth."

Overwork is a patent cause of under conditions in many puerperal cases. This obtains both in primipara and multipara. Economic conditions often necessitate the wife carrying on at her work after

marriage; when she returns at night, her home devours more of her energies and thus her strength is over-taxed. With this goes often - underfeeding, as often she is the sole wage earner of the household and too often the little food there is, is devoured mainly by the idle husband. It is a popular belief that a man must have his food and a woman needs little. This underfeeding leads to anaemia and poor resistance to disease of any kind. Pregnancy seems, however, to brace the women up in some way and it is only after the child is born that weakness develops. I am convinced also that excessive tea drinking leads to poor uterine muscle tone. I have been able again and again to diagnose excess in tea from the poor pains in labour and the poor uterine tone after delivery. Tea in excess is a uterine poison.

Alcoholism is supposed to be a cause of predisposition to infection in maternity cases. It has not, however, been my experience. I must bear testimony to the sobriety - the teetotalism of any women I have confined. Where it is present, however, it certainly has a detrimental effect. It is not, however, so bad in its effects as tea. Actual drunkeness of course, endangers the pregnancy in that violent

and inchoate action, falls, etc., are likely to produce abortion and serious haemorrhage. It is well known, of course that alcoholics do not stand septic infection well.

Dr. Kinloch (55) says regarding alcoholism:- "the health visitors made a special note of evidence of definite intemperance among expectant mothers, and found such evidence in a porportion of 1 - 93 as against an experience of 1 - 126 among women who died from puerperal conditions, so that alcoholism cannot be said to have played any important part as a cause of death."

Diabetes is well known to predispose to septic infection so much so that it is routine in carbuncle, etc., to test the urine for sugar. The only maternity case in my own experience was one of hyper-pituitary glycosuria. She went to full term without a setback, except that she became very emaciated. She developed hydramnios and at term gave birth to my only case of sclerema, the child (10 lbs weight) being like a stucco statue in texture. Great pressure on its body or limbs produced no impression and the child died from asphyxia owing to its inability to expand the chest under the iron restraint of the flesh.

The very eyelids were like stone and would not open. The mother made an uninterrupted recovery and the glycosuria disappeared no sugar being present in the urine in six months and in the blood in nine months from birth.

Tuberculosis is an unusual but potent cause of malaise in child bearing. Once again the body seems to call on reserves in pregnancy complicated by tubercle and the patient seems to improve in health. In consumption cases, however, after child birth, they usually rapidly deteriorate and as a rule die within the year of tuberculosis.

Bandelier and Roepke (56) in a treatise on tuberculosis entitled, "A clinical system of Tuberculosis" says, "there is now a consensus of opinion that pregnancy frequently makes manifest a latent tuberculosis and aggravates an already existing disease. This is not only true for phthisis but also for surgical tuberculosis and lupus, while urogenital tuberculosis usually remains uninfluenced. The grade and form of the disease are naturally of importance. Early cases, torpid, fibrous forms tending to encapsulisation and contraction pass through pregnancy much better than severe, dangerous

open, diffuse, ulcerating, cavity forming, and advanced cases. Even in cases of the first category it happens often enough that after a good progress at first with stationary physical signs an acute exacerbation occurs and that a single pregnancy irresistibly annihilates the best results of prolonged sanatorium treatment. A relapse may happen without warning during the second half of pregnancy." They advocate procuring abortion in all such cases.

Insanitary surroundings, have been blamed for the development of sepsis in child birth. I am very sceptical of the importance of this as a cause of sepsis. The fact that it is notorious that an uneventful puerperium is almost the rule in conditions and surroundings of the worst description leads one to hesitate in classifying this as a determining cause of sepsis. Personally I have greater fear of sepsis in a confinement in the ordinary nursing home than in the average poor working class household. Dr. Parlane Kinloch (57) says quite definitely, "the analysis of the records of maternity deaths in Aberdeen over a period of ten years as obtained by means of a special system of inquiry, has not revealed any definite relationship between environmental condition and puerperal mortality."

Again (58) he states, "regarding the cleanness of house as good, medium and bad, the health visitors found 95.4% good, 4.3% medium, and .2% bad. The corresponding puerperal death rates were 92.8%, 6%, .8%, so that the figures reveal no definite relationship between lack of cleanliness in the house and puerperal mortality."

Regarding size of house, he says, p. 10.

Deaths per 1,000 cases.	1 Room.	2 Rooms.	3 Rooms.	4 or more Rooms.
Sepsis.	1.7	1.8	1.8	2.3

So that actually the sepsis rate is higher in the larger houses.

I am sure the explanation of this apparent contradiction of all known laws of infection lies in some personal or social factor which spells a greater likelihood of carrier infection. A more searching enquiry into each case would reveal the reason. Of course all this is not to say that unclean or insanitary conditions should be tolerated. The utmost care should be exercised that every step is taken to render the puerperal conditions as sanitary as is possible. Absolute cleanliness, plenty of fresh air and drains above suspicion, should be a sine qua non.

To show how obscene conditions may be and yet a

normal puerperium be secured, I append notes of three cases.

1. I delivered a rag-picker of crossed twins on the floor of a one-roomed slum on a bed composed of filthy rags. The antiseptic was a small pudding-dishful of lukewarm lysol solution. I had to administer the chloroform as well as deliver the twins.

2. I delivered a Polish woman, a primipara, on a bed in a single apartment and could not understand the stench of the room until three days later; I discovered that the front of the bed which was in a recess, was composed of wire-netting behind which six hens and four ducks were kept. To all appearance they had not been cleaned out for many weeks. Our inability to understand each other, owing to neither of us knowing the other's language, accounts for my tardiness in discovering the cause of the stench. The roof of the coop was actually the mattress on which the woman was lying.

3. I was called to assist a colleague who had been wrestling ineffectually with a case for four hours. He had been giving an anaesthetic and attempting delivery unaided. The woman, a syphilitic, inhabited a single apartment in which there was nothing

but a recess-bed and a baking bowl. This bowl was three-quarters full of cold lysol solution in which for the past three and a half hours my colleague had been at intervals cleansing himself. The woman had been under chloroform for three hours. Her skin ingrained with dirt was still darker from paralysis of the cutaneous vessels. The case was one of impacted brow and the child was dead. As my colleague had been attempting delivery with forceps for the past three hours odd without result, I proceeded at once to break up the head of the child. We had no instrument with which to do this, but on searching round, we discovered on the windowsill outside, a pair of rusty scissors that had been used obviously for gutting fish. After scrubbing them in the lysol solution craniotomy was successfully carried through, and on the collapse of the head, no further difficulty was experienced. As there was no fire in the house, and no means of making any, and the woman was in a collapsed condition and had insufficient bedclothes, we left our overcoats in place of blankets. She got up on the 7th day, pawned the overcoats, got drunk on the proceeds, and was arrested by the police for being drunk and disorderly in the streets of the city.

It is worthy of note, however, that Dr. Claud Buchanan Kerr (59), draws attention to the influence of bad surroundings in the propagation of erysipelas. He says, "It was not infrequent in surgical beds in the Edinburgh Royal Infirmary for erysipelas to be associated with one or more beds. There was one bed in a surgical ward in which almost every operation case developed the disease and the surgeon was reduced to using this bed for simple fracture cases. In a similar instance elsewhere, it was found that erysipelas appeared in a ward where a drain outside the window was left uncovered."

Abnormal Labour has undoubtedly a very great influence on the hazard of puerperal infection. There is no fact more important than this among the predisposing causes of puerperal sepsis. The causes of abnormal labour are really roughly divisible into two. (1) Faults in the host and (2) Faults in the Passenger. Among common faults in the mother are (1) Syphilis. This tends to produce abortion at all stages of gestation. The abortion is usually accompanied with much bleeding and nervous shcok. The mental attitude of fear and disappointment is marked. The patient's blood stream is already so poisoned

that it cannot keep life in her progeny and the result is an ideal concatenation of circumstances for the streptococcus haemolyticus to acquire a footing. These cases if infected, do very badly.

Dr. Arnold Lea (60) says, "the existence of any chronic disease such as syphilis.....increases the tendency to infection.....by impairing the bacteriolytic and phagocytic power of the blood and tissues, thus diminishing the intensity of the local reaction and facilitating generalization of infection."

Premature Rupture of Membranes. This usually results in a long slow labour. The vagina as we have seen normally contains streptococci and saprophytes, etc., whose conduct is to say the least, questionable. The cervix slowly dilates and little streams of liquor amnii flow per os. The way is thus opened for penetration by vaginal bacteria. The long continuance of the labour gives them ample time and the exhaustion and anxiety of the patient supplies the final atmosphere for infection to take place. Dr. Lea (61) says, "it has been noted that the amniotic fluid, which is normally acid, soon becomes strongly alkaline (following rupture) and thus affords a favourable medium for the growth of bacteria, especially if considerable quantities of amniotic fluid are retained

in the uterus. Demelin found that if rupture occurred 24 to 48 hours before delivery, the morbidity was definitely increased; if two to five days before delivery, infection occurred in 11% of cases."

Induction of Premature Labour.(62). Sommer found fever present in 34% of cases. The risk is specially great in pelvic contractions necessitating induction and is to be attributed to slow course of labour, premature rupture of membranes, and repeated examinations and manipulations often needed in those cases."

I cannot but think that probably some of those inductions were either done too late or in too greatly contracted pelves. I have had no trouble with the few inductions I have done.

As regards delay in labour or rather protracted labour, Dr. Lea quotes Stotz as observing that "up to twenty-four hours there is no definite rise in the chances of infection. After this time, however, liability to infection is definitely increased. Prolongation of the expulsive stage up to three or four hours provided there is no obstruction is quite inocuous." (63).

Obstructed Labour. In these cases in addition to delay there is the tendency in fact the certainty of undue bruising and laceration of tissues. This reduces their visability and the actual lesions give pabulum to any microbes that are present. There is therefore a high morbidity rate placed by some observers from 15% to 30% Lea. (64). Malpresentations and pelvic deformities act in the same way. The best way to meet these difficulties is good antenatal care with X-ray examinations if need be and consequent correction of malpresentation or surgical intervention in suitable malformed pelves. Caesarean section is the real solution in many cases.

Jellett (65), however, in discussing Caesarean Section summarises his position thus:

1. Caesarean Section under most favourable conditions has a mortality of nearly 2% and may be followed by peritoneal adhesions and subsequent rupture of the scar.

2. Under unfavourable conditions mortality is 10-50% and the after risks to survivors is proportionately greater.

3. The only way to avoid Caesarean section is careful antenatal diagnosis and the only way to reduce

mortality when they are necessary, is hysterectomy.

4. Unless there is good reason for thinking that the uterine incision has healed satisfactorily, it may be unwise to allow a patient to deliver herself at subsequent pregnancies.

5. The treatment of eclampsia by caesarean section has a mortality of 16-34%. Conservative treatment has a much lower mortality.

6. The treatment of placenta praevia by caesarean section has a mortality of 11-20%. Obstetrical treatment is followed by a much lower mortality.

Appended is a table showing results of caesarean section and other operations in three American Hospitals and the Rotunda, Dublin.

Hospital.	Numbers of Deliveries.	C.S.	Proportion of others.	C.S.	% Mortality. Eclampsia.	Total
Jefferson 1921-1924	1453	1.63	-	-	-	2.3
Boston 1924	1133	1.12	1 in 2.4	7.6	5.4	2.2
Bellevue 1922	4286	1.97	-	7	4.8	2.5
Rotunda 1889 - 1922	47,412	1 in 366	1 in 14.5	-	12.5	0.49

From above it would appear that caesarean section

has not the relative importance as a remedial measure some observers think. The above table emphasises what good ante-natal care can do. Much again would depend on whether a viable child was desired. The above table would have been more valuable had it included the foetal death rate. One fact, however, emerges and that is that caesarean section should not be lightly embarked on.

Placenta Praevia. This has been quoted as a predisposing cause of infection. I regard those cases as suitable for Institutional delivery especially where the placenta is central or where it obtrudes seriously on the cervical region. It has never been other than successful with me but I regard it as too serious a condition to deal with in ordinary general practice conditions. It is to me a rare condition and should be discovered by careful ante-natal examination and appropriate measures taken.

Retention of Lochia, is also quoted as a cause. It has never happened in my experience.

Retention of blood clot. This also would naturally afford an ideal pabulum for the growth of bacteria and in addition the elastic pressure of a clot of any dimensions would interfere with the proper contraction and subsequent involution of the uterus.

Retention of Membranes would bring into play the same forces. My practice is to give time to the separation or expulsion of placenta. The careful watching of the pulse and observation as to excessive discharge from the vagina following delivery of the child are the important steps here. Gentle control of the fundus and the avoidance of anything of the Crede's manipulation results, in practically all cases, in the uterus emptying itself thoroughly. Forcible expulsion of the placenta in my opinion is nothing less than an assault on the patient. I know of no measure more calculated to produce shock than this. After the contents of the uterus are expelled, gentle kneading and rubbing of the uterus for five to ten minutes plus the injection into the buttock of 10 units of Allen & Hanbury's pituitrin obviates any massive clot formation in the uterus. After twelve hours, the patient is raised to the semi-sitting position which secures efficient drainage and prevents ante-flexion or retro flexion.

Retention of Placenta. This is rare in my experience provided one has patience to wait and try the above gentle measures. In midwifery, nothing succeeds so well as gentleness. If, however, an

hour has passed and there is no sign of the placenta coming away, there is no good end served in waiting further. If a gentle Crede measure is ineffectual, then the hand in sterile gloves must be inserted into the uterus, while the other hand controls the fundus. If the fundus is not so controlled, the operation is really giving repeated blows direct to the solar plexus. Nothing more calculated to kill the patient by shock can be conceived. It must always be remembered that the manoeuvre is that most calculated to produce sepsis, and haemorrhage or both.

Dame Janet Campbell (66) says, "the removal of an adherent placenta is regarded as one of the most hazardous obstetric operations, because the operator's hand comes directly in contact with the open blood-vessels and the raw wound of the placental site. Even in hospital it is an operation not to be lightly undertaken. Except when rapid treatment is needed owing to haemorrhage, the uterus should not be hurriedly explored for adherent or retained placenta or membranes and never without full antiseptic precaution."

Wounds of Parts.

During labour, there is often more or less bruising and laceration. If there be any abnormality

in the child or any contraction in the pelvis of the mother, or both, then such bruising and laceration is accentuated. It is a matter of observation and experience, however, that labour is rarely unattended with some tear of the cervix, or perineum, or both. This takes place even in the most normal of cases without any interference by attendants. Bruising is also practically a normal happening. From the standpoint of infection a bruise is the same as a wound. Both offer a ready ingress to infection. A ragged bruised wound offers a greater attraction to infection than a clean regular cut. It is more difficult to deal with the repair of the former and there is always more exudate and less life in the tissues. There is therefore less resistance to sepsis.

On the other hand, I cannot recall a case in which streptococcal infection supervened on a local staphylococcal infection. I am sure that a staphylococcal infection in some way acts as a preventive of the more serious condition. Not only is this a fact in midwifery cases but it holds true of all wounds. Exceptionally in tuberculous sinuses, however, there is certainly a tendency to the development of streptococcal infection. Dr. Buchanan Kerr (67) is emphatic

as to above. He says, "clean operation wounds, rather than those already septic are most likely to be infected with streptococci. When I first took charge of the Edinburgh City Hospital, I was much struck by the fact that though many patients with dirty suppurating wounds, but not suffering from erysipelas were admitted to the wards, none of them ever contracted the disease, though the conditions were all in favour of a spread of infection, the precautions taken against it being extremely primitive. This was very different to my previous experience as a house surgeon in a general hospital, where, in spite of much more attention to antisepsis, I had seen erysipelas spread to patients who had been recently operated on, and whose wounds bid fair to heal by first intention. The presence then of other micro-organisms in a wound may to some extent be a protection against infection."

Depending on the site of wound, infection locally may cause Vulvitis. This is an infected wound of the perineum or vulva. The parts swell up and are red and angry looking. There is a profuse discharge and abscesses may form in the tissues of the vulva and especially in the glands of Bartholin. As a rule

the microbes are staphylococcal and gonococcal. Dr. Lea (68) places the percentage of gonococcal infection in these at 50.

Vaginitis is either an extension of the inflammation from the vulva or it originates in a bruise or wound of the vagina itself. The parts are swollen and painful and inflamed and in many cases an actual impediment to the lochial flow is established. It is best treated very conservatively, careful antiseptic lavage and raising the patient's body answer well.

Metritis is a still further extension of the inflammation upwards till it involves the uterine wall. It may go on to abscess formation and discharge of pus. The organisms are as a rule mainly staphylococcal. Posture is the safest method in dealing with this.

Pelvic cellulitis with or without abscess formation may also occur. It occurs in two forms, diffuse or localised. It is due to the spread of organisms from wounds in region of the cervical vagina or the cervix itself per the lymphatics into the pelvic cellular tissue. The diffuse acute type is as a rule streptococcal and is therefore serious. It is accom-

panied by severe general symptoms and is usually fatal from generalised septicaemia. There may be very few local signs if the infection is very lethal but there is usually swelling in the hypogastric or iliac regions. Pus forms in the broad ligaments and may reach the diaphragm producing pleurisy or pneumonia. In the acute localised type, the patient may be quite well for six or seven days or even longer and then the temperature goes up with or without a rigor. The pulse which has never been quite right - oftenest in the region of 90 or 100 goes up to 120 or 140 with a temperature of 103° - 104°F. Constipation is a marked feature and defaecation is painful. There may be frequent micturition or retention of urine. In favourable cases the temperature comes down usually by lysis in about a week but it is well to be on the watch for abscess formation. Rectal examination should be the rule and by it one can detect swellings to either side of the uterus and running out almost to the pelvis. The abscess may rupture or absorb. In the former case it often ruptures into the rectum. Absorption takes a variable time, sometimes as much as 16 weeks, depending on its nature and quantity. Following resolution there may be

uterine displacements and fixations depending on the locus of the abscess.

Salpingitis is usually a result of extension of pelvic cellulitis in which the organisms have not been streptococcal. It is found on examination as an elongated boggy tender swelling running from one or other of the two uterine cornua outwards. It is a fertile source of sterility as it is usual for it to form adhesions. Dr. Lea (69) says, "Salpingitis in the later days of the puerperium is almost always gonorrhoeal."

Phlegmasia Alba Dolens is a condition in which there is phlebitis and thrombosis of the veins of the leg plus an infection of the lymphatics. It is most common in the left leg and this is almost certainly due to the fact that the left side of the cervix is that most usually lacerated, this laceration being almost certainly again due to confinement in the left lateral position. It really is an extension of inflammation in the broad ligament. The affected limb swells very greatly sometimes enormously and becomes so tense that it will not pit on pressure. It has a peculiarly transparent appearance owing no doubt to obstructed lymph. The swelling may extend on to

the abdomen. It may appear in the second week of the puerperium or as late as six or eight weeks after delivery.

Dr. Robert Jardine (70) quotes Dr.Tyler Smith as saying, "I believe infection plays a very important part in the production of the disease. I look on a woman attacked with Phlegmasia Alba Dolens as having made a fortunate escape from the greater dangers of profuse phlebitis or puerperal fever."

The above are the most common local septic complications of the puerperium. In addition, however, there are general infections of which the most common are toxaemia and septicaemia and pyaemia.

Toxaemia. When organisms remain local and their poison or exotoxins gain access to the blood the condition known as toxaemia is in evidence. It is shown by quickened pulse, elevation of temperature, a sensation of chilliness, it may even cause a rigor though such rigor is not repeated. There is headache, sleeplessness, sweating and it may be slight wandering of the mind though this last is rare and is not of good omen. At no time is it possible to grow organisms from the blood. The pulse though fast is generally proportionately slower than the temperature warrants

and the slower the pulse relatively, the better the prognosis. In mild cases, the temperature rarely exceeds 101°F. and the pulse 90. In severer cases, the temperature may be 103°F or 104°F. and the pulse 100 to 110. In benign cases, the condition subsides in two to seven days but in grave cases, a fatal result may supervene in that time. A progressive hastening of the pulse is of evil augury. In severe cases which end fatally, the clinical condition is similar to septicaemia. The mind is usually clear to the end. Clots and detritus generally are thought to be a factor in the production of this condition as affording pabulum for the bacterial growth, and it is in these cases that douching seems to be of service though personally I am most averse to anything of the kind. I believe more in the efficacy of the raised posture following labour and the securing of no detritus or clots in the organs or passages by proper measures at birth. This, plus general supporting treatment, I find of most service in these cases.

Puerperal Septicaemia. This grave condition means that the bacteria themselves have entered into the blood and lymphatics and are there multiplying. They can be grown directly from the blood in culture

tubes. It is seldom or never possible, however, to see them by microscope in a direct film. When as we have seen, a strong man can be killed in twelve hours from one prick of a rose thorn, it is not difficult to realise the enormous gravity and enormous facility for infection of this nature in case of childbed. When one considers all the facts relative to the nature of the streptococcus, its wide prevalence in nature and the lesions of the female tract which are inevitable in even the most normal of cases and when in addition, we know that final asepsis in the genital tract is an unattainable ideal one wonders that puerperal septicaemia is not infinitely more prevalent than unfortunately it actually is.

The streptococcus then gains access either through wounds or abrasions in the genital tract from the perineum or directly to the uterine placental site. There may be no local reaction whatever and this is always a bad augury. The streptococci are carried in the blood and lymph streams throughout every organ of the body. There is in severe cases no body cell that has not its accompanying microbes. In the most severe and lethal cases the patient shortly after delivery becomes unconscious

with
sighing respiration and hypostatic staining of the body, rapidly increasing failure of the pulse, subnormal temperature, and death. It is possible for all this to happen in a few hours after birth. These cases were at one time looked on as post partum shock but it has been found that post mortem while there were no signs of reaction to infection in any organ, yet there was no organ from which streptococci could not be cultured. Thus Fitzgibbon and Bigger (71) say, "In several of these cases (puerperal sepsis cases) collapse temperature with very rapid or imperceptible pulse developing shortly after delivery and persisting to death, was the only indication of infection. In such cases the peritoneum will invariable be found infected without any reaction or symptom." Professor Crichton of Capetown reported that, having seen an observation on this subject in the last Rotunda report he had a blood culture made just before death in a case of unaccountable shock following delivery by craniotomy and found pure streptococcus haemolyticus. The fact has been established in the Rotunda Hospital, not only in maternity cases that have been tested, but also in gynaecological cases dying of apparent post operative shock."

In their conclusions, they state, "that 'labour shock' is never a justifiable diagnosis of the case of deaths after protracted labour until infection has been excluded by a cultural examination of a swab from the peritoneal cavity. These cases are frequently the most rapid and virulent type of sepsis."

The most usual bacterium in these cases, is the streptococcus pyogenes or streptococcus haemolyticus. Other organisms have been observed, viz., bacillus coli, gonococcus, staphylococcus, bacillus pyocyaneus and the bacillus aerogenes capsulatus. Apart from the streptococcus haemolyticus and bacillus coli, all the rest are very rare. Moreover, where an infection takes place of a staphylococcal or gonorrhoeal nature it is rare for streptococcal infection to supervene. Thus the same law seems to be obeyed here as obtains in ordinary wound cases as mentioned above (67), viz., that streptococci only attack virgin wounds, i.e., wounds not already infected by other organisms.

Thus Dr. Smith (72) states, "out of 196 cases, uterine culture showed streptococcus haemolyticus alone in 120 cases. Thus 76% of cases showed infection by streptococcus haemolyticus alone. The

blood changes that take place in puerperal sepsis are in very severe or fatal cases a leucopenia. That is to say there is an absence or reduction in the white corpuscles. In those that re-act a polymorphoneuclear leucocytosis takes place. In mild cases, the leucocytes may amount to 10,000 - 12,000 per C.M.M. In severe cases, this may rise to 40,000 - 60,000 per C.M.M. There is in addition an increase in the blood plasma which serves the purpose of diluting toxin. Clinically the condition is ushered in usually by a rigor on the second or third day after the birth. The sooner the rigor, the graver the outlook. The temperature rises to 104°. The pulse is rapid often in the region of 140-160. The pulse is a more valuable prognostic guide than the temperature. A fall in the pulse rate is of good omen. The rigors may be repeated and the temperature swings up and down between subnormal 97°F or 96.5°F and 105°F. Patient's tongue becomes dry in centre and moist at edges. In some cases a scarlatinal rash appears. There is profuse sweating, marked diarrhoea or constipation, and steadily increasing weakness. The mind as a rule is clear to the end though delirium is fairly common. Headache and sleeplessness are marked features as a rule. Early in the case the

lochia and milk dry up.

The urine is scanty and often contains albumen. The patient is emphatic that she feels well and persists in this mental attitude to the end, as a rule. The better they declare themselves to be, the graver is the case.

Dr. Jardine (73) says, "the pulse is a better index of the patient's condition than the temperature. In some cases the temperature may remain low even subnormal and yet the pulse be very high. Such a case is likely to prove fatal. You occasionally see a case where a woman is from her appearance manifestly ill yet maintains she is quite well. Such a case generally ends fatally." Distension of the abdomen is variable. Sometimes there is great, even enormous, distension, in others there is none at all. Pain on palpation is also very variable. There is in my experience neither pain nor rigidity on abdominal palpation. The patient as the case goes on develops increasing pallor with slight icterus or creamy lemon appearance. This is the result of the red corpuscles being lysed by the streptococci and is of grave, in fact, fatal omen. In some cases, mania develops of a very violent type but this is rare. The patient

gradually sinks and dies in from a week to twelve weeks' illness.

Pyaemia. This is a localised form of infection in a generalised septicaemia. Some of the organisms have settled in a suitable place and there go on to abscess formation. This sometimes also arises from a septic thrombus becoming detached and passing as an embolus to a vessel of narrower calibre. Or again, vegetations may form in the heart valves and particles becoming detached from them act as septic emboli.

The first indication is usually a rigor and rise of temperature with great perspiration. This happens at irregular intervals and at the end of roughly a week, abscesses may make their appearance. These may be few or many, small or large, purulent or watery or sanious, foul smelling or odourless. The patient's general condition rapidly deteriorates. The heart is often dilated early in the disease and the pulse is rapid and feeble. The commonest sites of abscess formation are in their order, joints, superficial cellular tissue, lung, brain, kidneys. Wherever possible abscesses should be fully incised whenever diagnosed. Free drainage should be secured. This is especially the case in joint mischief. Often the

case seems to improve on abscess formation, as if this was a turning point, and frequently on incision and drainage of the abscess, recovery begins. Severe types of this condition with high remittent fever, many rigors and increasingly rapid pulse may die within a week or ten days of delivery. Dilatation of the heart is a common feature. In those cases which, though acutely ill for many weeks, eventually recover, it is common to find marked subinvolution of the uterus. This is one of the most unfailing signs of sepsis having been present in the puerperium.

The mortality from this condition is said to be "about 60%." (Lea).

Having discussed the predisposing causes of infection and shortly described the septic conditions that follow such infection, it is pertinent to ask how infection is usually brought about. To be candid there has been a great deal of unwillingness to face facts and a manifest desire to shift blame from one attendant to another attendant or to the patient herself. Ignoring the fact that the sepsis rate in Institutions is the highest of all; the masters of the art, the specialists, are agitating in both the lay and medical press for universal Institutional

midwifery as the one step needful to secure asepsis in the puerperium. The general practitioner tends to blame the nurse for any untoward febrile calamities, the nurse, who, so far, shows the best statistics of all, whispers to the patient her suspicions of the docotr and to the doctor her suspicions of the patient. Into these cross currents comes the public health official who is intent on seeing that officialdom shall continue to expand and grow like the Upas tree. He therefore is all for Institutionalism and Officialdom. The experience of the War gave an immense impetus to this regimentation idea. The lay public is split into sections all of them alarmed and suspicious. They make certain of Safety First. No sepsis will occur if there be no pregnancy. So the birth rate drops alarmingly. Under these circumstances, it is refreshing to calmly study the facts of scientific research in puerperal sepsis. We have seen that the streptococcus haemolyticus for all practical purposes the microbe of puerperal sepsis. Where does it come from to the puerperal woman? Though there has been an enormous amount of bacteriological research on this subject, there is a division of opinion among the experts. Dr. Bonney (75) is of opinion the infection is endogenous mostly. The source being faecal.

Dr. Doris Gordon (76) agrees with Dr. Bonney.

Fitzgibbon and Bigger (77) think the infection is exogenous.

Drs. Armstrong and Burt White (78) suggest that the minor degrees of puerperal infection are due to endogenous infection with bacteria of low virulence.

Bacteriological investigation of the female genital tract during pregnancy have been carried out and reported on by among others, Fitzgibbon and Bigger (79), Kinloch Smith and Stephen (80) and Drs. Harris and Smith (81). These find that though various streptococci were found the streptococcus haemolyticus was not frequent. Drs. Kanter & Pilot (82) were the first to suggest that droplet infection from the nose and throat of attendants was the cause of puerperal infection.

Finally Dr. Smith (83) proved in a series of cases that droplet infection from nose and throat of attendants was the cause of puerperal fever in almost all cases. In short, it was practically always exogenous. Dr. T.J.Mackie and Dr. G.S. Maclachlan (84) dealt with the serological characteristics of strains of haemolytic streptococci found in erysipelas, puerperal fever and tonsillitis and scarlet

fever. They found that serologically they were similar. Dr. J. Smith (85) showed that the toxins from various diseases can be neutralised by antitoxin prepared for the toxin of scarlatinal strains, or by antitoxins prepared for the toxins of puerperal strains or by antitoxins prepared for the toxins of erysipelas strains. Dr. Smith (86) says, "the incidence of streptococcus haemolyticus in the normal throat has been found to range from 10-60 per cent. or higher, and this location must be considered the normal habitat of this organism.....it has become apparent therefore that normal persons are frequently carriers of streptococcus haemolyticus capable of producing on occasions various disease processes by contagion, it being understood that the term "contagion"embraces all channels by which droplet or spray infection is spread." (87). Dr. Smith (88) by serological tests reached the conclusion that "in no less than 11 cases of puerperal fever the infecting strains had their origin in the throat or nose of doctor, nurse or student in attendance."

In a bacteriological examination of the cervix and throats of pregnant women in the last week of pregnancy I found that though streptococci and many

other bacteria were common both in vagina and throat, I was not successful in obtaining either streptococcus Pyogenes or streptococcus haemolyticus. Under the terms of the National Health Insurance Act expectant mothers are allowed money during the last month of pregnancy. It is as well to note also that they are entitled to and take advantage of antenatal care from their Panel Doctor. They must all, however, attend during the final month. I took advantage of this to swab twelve cases each week during that terminal month. I swabbed both throat and cervix and had the hospitality of the Royal Infirmary Bacteriological Department put at my disposal and the advantage of the counsel and help of Dr. J.A.G.Burton. The mode of collection of swabs was as follows. I put on a mask and sterile rubber gloves. A small size Ferguson's speculum was in boiling antiseptic solution. I had at my side serum agar culture tubes. The patient's throat was swabbed and the appropriate culture inoculated. It was marked with an identification letter of the alphabet and a capital T signifying 'throat'. The patient then knelt in the knee elbow position and her external genitals were thoroughly cleansed with spirit and liquid antiseptic soap. The speculum

was then taken from the solution and inserted gently into the vagina. A platinum loop mounted on a glass rod was then introduced down this speculum, great care being taken not to touch, the sides and by the light of an electric forehead lamp the cervix was gently entered and a loopful of secretion obtained; withdrawn with the same care not to touch the speculum as had been taken on entry, and immediately inserted in the culture tube marked "V". I give the results in tabular form of the last week only. The other results were practically similar.

Table of Cultural Results from Throats and Cervices in last week of Gestation.

Index Letter.	Throat.	Cervix.
A	Abundant streptococci and Saprophytes.	Mainly Diphtheroids.
B	Streptococci, Staphylococcus and Saprophytes.	Staph. Albus and Saprophytes.
C	Abundant streptococci and Saprophytes.	A few streptococci and Saprophytes.
D	Scanty Streptococci and Saprophytes.	No growth.
E	Streptococci and Saprophytes.	Abundant streptococci & Diphtheroids.
F	Diphtheroids and Saprophytes.	Streptococci and Diphtheroids.
G	Saprophytes.	Saprophytes.
H	Abundant streptococci.	Saprophytes.
I	Streptococci and Diphtheroids.	Saprophytes.
J	Diphtheroids and Saprophytes.	Saprophytes and Diphtheroids.
K	Streptococci.	Streptococci, Diphtheroids and Saprophytes.
L	Streptococci.	Streptococci and Diphtheroids.

The cases containing Streptococci were then picked out and plated on Blood Agar but neither streptococcus haemolyticus nor streptococcus pyogenes

was found.

We thus see that over 50% of the throats and over 40% of the cervices harboured streptococci within a few days of labour. These cases again have all to report back to me at the end of a month after birth of the baby to receive further certificates for funds.

On enquiring, I found that some had been confined in the Maternity Hospital, some by midwives and some by midwives plus doctor. Four of the twelve had had instrumental delivery one of these being "F" in the seven who had streptococci and diphtheroids in her cervix. All had had an uneventful puerperium. The above agrees with most of the bacterial research done on the parturient vagina. From this, it would appear that the streptococci in the cervix of a woman at labour are as a rule innocuous, but Dr. Smith (89) records that "in two cases of septic abortion due to streptococcus haemolyticus was endogenous." Fitzgibbon and Bigger (90) say, "streptococci haemolytici were found twice out of 108 swabs of the pre-labour vagina." I must regard these facts as strongly supporting the importance and likelihood of endogenous infection. Even if this is the usual proportion and only 1 of the 2 became lethal to the

patient then that would give almost 5 per 1000 or 1 - 200 as endogenous in origin. It is interesting to turn to the experience of other workers in their endeavours to find the conditions that rule infection here.

Dame Janet Campbell (32) says, "the most dangerous area in the generative tract is the recently denuded placental site." Lacerations of cervix, vagina, perineum and vulva are also potential sites of infection." In a footnote she adds, "An aggregation of patient's certainly increases the risks of infection." In a note under Maternity Hospitals she states that she visited a hospital in August and examined 128 charts and found no evidence of puerperal fever but that later from January to June, 1923, 528 patients were delivered in this hospital 9 cases of puerperal fever were notified and 113 patients had a temperature of over 100.4 = 21%. She states that the routine followed seemed excellent and she cannot account for the increase of morbidity. Dr. Lea (92) states that "attention was called to the influence of climate on the incidence of puerperal fever and erysipelas. Charts were prepared by Dr. Galabin for England and Wales and also for London.

These showed that the frequency of these diseases varied inversely with the rainfall. There was an increase of both in dry weather. It was further shown that puerperal infection increased in winter. The figures for London, New York, Manchester and Hamburg established this clearly. It is therefore possible that climatic factors were at the root of the rise in infectivity stated by Dr. J. Campbell. Dr. Smith (93) mentions the case of a woman dying of puerperal sepsis who a fortnight previously had been wounded by a splinter of wood entering her finger. There had been lymphangitis and pus formation. Swabs from her uterus and finger and the throat of the doctor in attendance all revealed streptococcus haemolyticus but the serological reactions indicated the identity of the uterine and finger streptococci. It would have been very interesting if all three had been identical. I am suspicious of our ability to so nicely discriminate in such a protean infection as that caused by streptococci. It is not indicated here whether the swabber and examiners generally were all masked, etc., against the possibility of infecting the culture. If not, then the whole research is vitiated.

Dr. Jardine (94) gives many instances of this type of blood infection occurring both before and after birth. They are very fatal. Among his cases he quotes (1) a finger infection, (2) Old Retro Renal Infection, (3) Old Retro Caecal Abscess, (4) Old double Pyo Salpinx. All were fatal. It is in my experience quite possible for an old streptococcal focus to be dormant for years and then to awaken to fatal effect following childbirth. I am of opinion that the streptococci and pneumococci are essentially similar organisms both in morphology and pathogenicity. I believe they are the real factor in lung disintegration following child birth in Phthisis patients.

I might quote here a case which I sent to a distinguished gynaecological specialist in the Western Infirmary, Glasgow. The woman, six para - suffered from cystocele and rectocele. She was operated on in the usual way with finished precautions as regards sepsis, etc., masks, gloves and all the technique of a first class operating theatre. In two days she had a rigor, developed septicaemia and died in six days. This illustrates the nature of the foe we have to deal with. Of course, in this case, the patient was under the handicap of clean surgical

incision. Had she had a good staphylococcal infection of the vagina no septicaemia would have developed according to the law stated in another section, q.v.

I have written at some length here for I wish to emphasise that it is not so easy to get rid of puerperal sepsis as many of us imagine. I also wish to emphasise my scepticism of the innocent nature of a streptococcus that is already in situ a day or two before the confinement. What is it there for? There is no such thing in nature as an idle microbe. The mere fact of its presence shows that it has an object in view.

The post mortem appearances of those dead of puerperal sepsis are very striking. The body does not stiffen much, that is to say rigor mortis sets in early, it is not very complete and soon lessens. Of course, this means that decomposition sets in early. Long before death hypostatic staining is in evidence and this deepens after death. The blood is black and remains fluid. There are petechiae on the membranes especially the pericardium and pleurae and meninges. Often the valves of the heart show vegetations which are soft and crumbling and easily broken up. The heart itself is usually dilated and

the muscle soft and diffluent. Cloudy swelling of kidneys, spleen and liver is usual. Locally the genitals may show nothing out of the usual as in severe "shock" cases or they may present the last appearance of foul digested degeneration. The uterus may show nothing unusual in the same way or it may be large, flabby and soft like butter and contain anything up to almost all the placenta.

Symptomatology.

These cases all run remarkably true to type. Having had an experience of one, you may be said to have seen all. They may be divided, however, into two classes. (1) The mild - which recover and (2) the serious which die. Very seldom do you get cases between. Usually after a few days, the issue is only too clear.

Mild cases. Here the patient, after two to five days develops a temperature of about 101°F. This is usually remittent, being down in the morning to normal or subnormal and up at night. It is wise, therefore, to make your second day and third day and fifth day visit at night. The pulse is to me the most important feature. If it does not rise above 110 to the minute and keeps its "tone" the issue will

be favourable. It is the first sign to show improvement. Even without any febrile temperature an acceleration in the pulse to even 90 per minute should make one look for the reason till he finds it. There will be one. The respirations are increased in varying proportion from 36 to 50. The normal respiration pulse ratio is 24 - 72. If the increase observes this proportion, the case will do well. If it does not, the issue will be obscure to fatal. A rigor in my opinion is a serious feature. If repeated, it is extremely serious in proportion to the repetition. It is generally an initial symptom. If it is not the case is serious.

Fitzgibbon & Bigger (95) say, "of the 57 cases recorded, 24 had a sudden onset in 6 with rigor, 4 had a sudden rise, 2 with rigor." Of three cases, they quote in detail, two had rigor and both were fatal. In my own series all who had rigor died. I therefore regard rigors as a very fatal symptom. A rigor means a fresh blood infection.

There is usually profuse sweating and the lochia becomes evil smelling and dries up. The mammary secretion also fails. These are all serious symptoms. Therefore in mild cases there is as a rule no rigor

or an initial one only and the lochia though smelly, remains flowing. The milk also remains. There is often headache usually severe. There is slight abdominal distension but no pain on palpation. There is no delirium and sleep is not much affected. The patient complains of being unwell and is anxious about her condition. This is a very good feature. The condition improves in about a week or ten days when temperature and pulse return to normal and the patient recovers.

In severe cases, all these features are intensified. Temperature 104 to 105. Pulse 140-2,000. Profuse sweating, absence of milk and lochia. Great abdominal distension but this is very variable. With a high pulse and temperature, absence of distension in abdomen is serious. I am of opinion that abdominal distension means retained products in theuterus. The mind is as a rule clear and the patient is contented even happy about herself. She protests how well she feels. This is almost always a fatal sign. Dilatation of the heart occurs early and hypostatic staining is common and invariably a fatal sign.

There is sometimes delirium which is of a happy type, singing and laughing being prominent. Often

there is violent mania and restraint of the patient is imperative. This precedes the end which is usually sudden and in coma. Death may occur in a few hours or in many weeks, but the issue is none the less certain.

I would venture here to give some details of the cases in my practice of over twenty years into which sepsis entered. My total cases are 1902 in number. Of these 416 were suburban cases, 57 were country cases and 1429 Glasgow cases. 1073 cases were delivered by me before I wore rubber gloves as routine. I have never worn a mask. The remaining 829 cases are all Glasgow cases. They were all delivered with sterile rubber gloves. It is in this last class all my puerperal septicaemia has occurred. In the series of 416 suburban cases, my forceps ratio was almost 80%. The reason quite frankly was to save time. It was an overworked Colliery and Club assistantship and from the time and economic standpoints they were a necessity. I never lost a single case here and never had anything but a normal puerperium. I never did, however, apply forceps before the cervix was fully dilated and the head was in the pelvic basin, i.e., well past the brim. In not one case had I any bad

effects whatsoever. No rectal or bladder fistula were perpetrated. There were only three foetal deaths. The country cases were practically never forceps cases. I, in fact, usually found them born on arrival. There were no foetal deaths in this series the only mishap being the loss of the sight of one child's right eye through gonorrhoeal ophthalmia. This was due to the absence of hospital or district nursing facilities and poor midwifery nursing attendance. The case was three miles from my house and though I went night and morning to lavage the eyes for five weeks, I was able to save only one. I much regret this happening. The mother was an ignorant cot wife who had not led a very virtuous life. She was hopeless as a nurse so far as the eyes were concerned.

Pyelitis has occurred in three cases only in my series. I know that this is so remarkable as to be doubted. But as I have all along taken monthly samples of urine up to the eighth month and weekly samples thereafter and as the symptoms are so painful not to say grave to the patient, I cannot have been ignorant of its presence. It is easy to diagnose. The patient complains of acute pain - generally in the right - in the kidney region behind. There is

intense pain on palpation there. Depending on the degree of infection the patient is more or less acutely ill. There may be high fever of a remittent type as high as 103° - 5°F. and pulse of 140.

Vomiting is common and may be intense and urgent. There is frequency of micturition and the urine contains albumen, pus and sometimes blood. It generally appears from the fifth to the eighth month and in my opinion the increasing size, weight and pressure of the uterus has much to do with the condition in the way of predisposing to infection. Pyelographic studies which I was privileged to see at the Maternity Hospital, Glasgow, a year ago, showed a great dilatation and sometimes actual kinking at the lower end of the ureter. All my cases supervened suddenly on what had been perfectly healthy urine cases. This suggests that actual infection is the cause of the condition. Given stasis and a dilated or kinked ureter suitable conditions for infection at once arise. All my cases were Bacillus Coli infections. The urine has a peculiar opalescent appearance and a strong fishy odour. In no case was enlargement of the kidney found. The condition was treated in each case with

starvation diet, rest in bed and copious draughts of cold water. Drachm doses of Potassium Citrite were given every six hours and all recovered within a month and had a normal puerperium. It would seem, however, that this happy result is not always obtained for Dr. Jardine (96) states that "In 23 out of 53 cases, spontaneous premature labour occurred and 7 out of 60 children were stillborn or died soon after birth". He also states, "The first factor in the causation is dilatation of the ureter from pressure at the brim of the pelvis. The right ureter is much more liable to pressure during pregnancy both from its anatomical position and also from the fact that the ureter generally deviates and rotates towards the right side. The process is an infective one and is generally through the blood stream but in some cases, especially in those occurring in the puerperium, it may travel up the ureter from the bladder".

The urine is generally of low specific gravity in my cases .1008 - .1012 and acid in reaction. In my experience movable kidney is generally confined to the right kidney. Professor Wilson (97) says, "It is several times as often on the right side as

on the left". On examination after the puerperium this was so in all my cases. This may therefore be a contributing factor. It is therefore a point I particularly note in the antenatal period and advise copious fluid and citrate of Potash in all cases of movable kidney during gestation. This may explain the small numbers in my series who contracted Pyelitis.

Breast Cases. I have had 39 cases of Breast Abscess of which I give a short resume. I am of opinion that the best treatment for breast abscess is to put away the milk and to incise freely the inflamed area. Any other treatment I find to result in a more or less large accumulation of pus with consequent pain and exhaustion to the patient and the possible ruin of the breast as a milk organ.

Inflamed breast usually occurs between the third and fifth days following delivery. Incision is usually made on the fifth to the seventh day from delivery depending on the day when inflammation begins. It, therefore, follows that a discharging would is present in the first fortnight of the puerperium which continues to discharge more or less pus for about ten days following incision. In not

one of these cases did generalised septicaemia occur. In fact, any little disturbance of temperature and pulse, which never exceeded 99.5°F. and 84-100 respectively, at once subsided to normal following incision. The infective organism in each case was Staphylococcus Pyogenes Aureus.

Appendicitis. This patient developed a temperature of 103°F. with pain in the lower abdomen on the third day following delivery. At first I thought I had to do with sepsis in connection with the delivery which had been instrumental, but on more carefully considering the clinical signs I became convinced that the case was one of appendicitis. The patient was duly operated on and a purulent unruptured appendix removed. An uneventful recovery took place. The organism in this case was the Bacillus Coli Communis.

Salpingitis. In this case the diagnosis was made by a gynaecologist. The patient, who had a year previously suffered from right-sided salpingitis, developed on the third day after delivery a temperature of 103°F. with pain in the right lower abdomen and purulent discharge from the vagina. Recovery was complete in fourteen days from the date of

delivery. Here there was a case of discharging pus per uterus in which the placental site must have been raw and large and yet no generalised infection took place. The infection was, however, a Staphylococcus Pyogenes Aureus and this is generally strictly local in action. Had it been Streptococcal the patient would, in all probability, have died.

Vulvar Abscess. In this case I found on going to deliver the patient that an abscess as large as a tangerine orange had formed in the right labia majora. No forceps were applied. The patient was a multipara and the labour easy and rapid. The abscess ruptured on the passing of the head and the pus bathed the child's face and head. This was at once washed down by cupfuls of warm lysol solution which I had had prepared against this happening. This lavage was kept up and the patient propped in a semi-sitting position till the abscess drained and healing took place ten days later. At no time were the temperature and pulse other than normal. The infective organisms in this case were the Bacillus Coli Communis and Staphylococcus Pyogenes Aureus.

The above are all cases where a blood infection

should have been likely and yet it did not occur. The microbes were other than streptococci in all cases. Did they protect?

I now pass to my puerperal septicaemia cases indicating briefly their outlines.

1. Primipara. aet. 26. Attendant handywoman. Breech presentation. Perineal rupture requiring two stitches. Temperature 103°F. on the second day with rigor, pulse 140. Died in 16 days. Here there was a good deal of manipulation. The child weighed 8 lbs. and was delivered without injury of any kind. The handwoman was attending other cases, which, she said, were going on all right.

2. Multipara. aet. 36. Attendant handywoman. No forceps. Baby born before my arrival. No rupture of the perineum. Rigor and temperature of 102.5°F. on the third day with appearance of a scarlatinal rash. Pulse 140. Died in 12 days. Five weeks previously her son, aged 7. had been removed to hospital with scarlet fever and was still detained in the institution owing to ear discharge at the time of the confinement. The clothes on the bed and on the woman's person had been taken from a

chest which had escaped disinfection by the Public Health Authorities.

3. Multipara. Attendant handywoman. No forceps. Full term child, putrid. Temperature 102.5°F. Pulse 120 on the second day. No rigors. Recovered in six weeks after a serious illness.

4. Primipara. Aet. 36. Attendant trained nurse. Labour lasted 26 hours. Forceps with head in vagina. Perineal rupture requiring two stitches. Temperature 104°F. on the second day with rigor. Pulse 180. Died in 5 days. In this case, going to the confinement, I was called in passing to see a child who was ill. There was nothing definite to be made out in the child's case except that it had a high temperature. From this call I went on and delivered this woman. On returning in the evening to visit the child I found it had developed scarlet fever. It is noteworthy, however, that this was the man's third wife, his former wives having both died of puerperal septicaemia.

5. Primipara. Aet. 23 years. Trained nurse attendant. No forceps. No rupture of perineum. Labour lasted 18 hours. Temperature

103°F. on the third day with rigor. Pulse 160. Died in six weeks.

6. Multipara. Aet. 40 years. Trained nurse attendant. No forceps. No rupture of perineum. Temperature 104°F. on the second day with rigors. Pulse 180. Died seven days. This case had complete prolapse of the vagina. The baby was born out of the summit of a cone whose apex was the os uteri and the sides the prolapsed vaginal wall. I swathed the parts in sterile lint wrung out of warm lysol solution and on the birth of the placenta restored the vagina to its natural position. On calling on the second day, I found the nurse with her right stocking down bathing an erysipelas of her own leg.

7. Primapara. Aet 36. Very highly strung patient. Confined in a nursing home. Forceps were applied when the head was on the perineum after a labour of 16 hours. No rupture of the perineum. Temperature 102°F. on the fourth day with marked delirium. Pulse 120. No rigor. Complete recovery in 8 weeks after a severe illness. On the third day this patient was visited by a neighbour who lived on the same stair and she herself became No. 8.

8. Primipara. Aet. 36 years. Trained nurse attendant. Confined in her own home a year after the birth of No. 7. No forceps. No rupture of the perineum. Prolonged labour lasting 2½ days. Temperature 102°F. Pulse 120 on the fourth day. No rigors. No delirium. Recovery in 8 weeks after a severe illness. She was visited on the third day by No. 7. It is interesting to compare the similarity of these cases as to incidence, severity and duration.

9. Primipara. Aet. 22 years. Handywoman attendant. No forceps. No rupture of the perineum. Labour lasted 12 hours. Temperature 103°F. on the second day with rigor. Pulse 180. Died 17 days. This was the man's second wife, his former having also died of puerperal septicaemia.

10. Primipara. Aet. 22 years. District nurse attendant. Labour lasted 3 days. Forceps applied when head was on the perineum. No rupture. Temperature 104°F. in 12 hours. Pulse 140. No rigor. Recovery in six weeks after a severe illness. This nurse was in attendance on Mrs. D. (a patient of Dr. F.) who was confined on the seventh March, and who sickened of scarlet fever on the

eleventh March. The infection was probably brought by a boy aged 4. who was dismissed from Belvidere Hospital on 28th February, after being under treatment for scarlet fever. This boy infected his brother who sickened on the 7th March of scarlet fever. The nurse was taken off duty and sent on holiday for a week. On her return she took over attendance on Mrs. M., a patient of Dr. X, confined on the 17th March. This woman sickened of scarlet fever on the 26th March. My patient was delivered on March 25th. The nurse then attended a third case, Mrs. N. the patient of yet another doctor in my neighbourhood. This woman was in the fourteenth day of her puerperium which had been up to then normal. This woman developed a temperature of 102°F and Pulse of 120. She exhibited a true scarlatina.

To sum up then, we have here four cases attended by the same nurse. The first contracts scarlatina from her boy. Nurse is then taken into quarantine. She returns to duty with throat unswabbed and on the same day attends three cases, one at labour, one in the ninth day of an uneventful puerperium and one on the fourteenth day of an uneventful puerperium. All these cases have differ-

ent medical attendants. Each develops infection. The first two both contract puerperal septicaemia. My case recovered, No. 2 died, and No. 3 develops scarlatina and recovers. The nurse's throat is swabbed and found to contain Streptococcus Haemolyticus. I think these facts of enormous interest. It establishes the fact of exogenous infection. It proves that the same Streptococcus can cause puerperal fever and scarlatina. To my mind what constituted the different manifestation in the third case was the length of time (14 days) that had elapsed since birth. The woman's blood stream was not so patent or vulnerable to infection. I consider this case quite unique in interest and importance. Of these ten cases of my own, then, the infection was clearly exogenous and the source known in No. 2, 3, 4, 6, 9 and 10. In Nos. 1, 5, 7 and 8 the source was unknown. No clear opinion can be formed. Any of the attendants may have been carriers. As regards myself as the carrier, I may say that it is exceedingly rare for me to have less than two or three maternity cases going at one time. Often I have six in varying stages of convalescence. It stands to reason then, that were I the carrier I

would infect more than one case in the series. Look at the potentialities of the nurse mentioned above! Yet it is a fact that I have never had puerperal sepsis at other than long intervals and they have always been solitary cases. This would seem to exclude me as the infective agent. It is also worthy of note that every one of the above cases which developed rigors, died. I have dwelt on Fitzgibbon & Bigger's findings qua rigor previously. It is also worth noting that of these ten, seven were primipara and three multipara. Of the primipara four died and three recovered. Of the multipara, two died and one recovered.

Drs. Kinloch, Smith and Stephen, show in tabular form, the comparative danger from sepsis of the various parities:-

	Parity.												
Cause of Death	1	2	3	4	5	6	7	8	9	10	11	over 11	Total.
Sepsis	19	9	9	7	5	1	-	2	2	-	-	1	55

The relative preponderance of primiparous sepsis and mortality is thus again accentuated as it was first in the monograph by Professor J. Matthews Duncan (21) in 1870. While giving rightly due weight to the facts there adduced of longer labour,

more tendency to inevitable lacerations, bruisings and trauma being vital factors in this greater morbidity. I think the mere fact that the primipara is as it were for the first time in the firing line and therefore is for the first time exposed to the risk of becoming a casualty, accounts to a great extent for this higher incidence. The chances are, she then succumbs and never therefore has a chance of again becoming infected. Furthermore, in my experience it is rare for a woman who has once had puerperal sepsis to have another child. She is either afterwards sterile or she shirks the risk and adopts birth control methods. She therefore will not figure as a multipara. I am unaware of any statistics that show a woman as having figured twice in puerperal sepsis. But here I am open to correction.

To sum up. These six regrettable deaths are my total contribution to the death rate in childbirth in twenty odd years. Apart from puerperal sepsis, I have not lost a single woman in childbed. In addition, I have had only four cases of postpartum haemorrhage only one of which was serious. In no case of this kind was the subsequent puerperium

other than normal. Subtracting again the cases where the infection was clearly carried by others, we find that only four are left unaccounted for. Of these two died. Therefore even supposing I was the carrier only two deaths out of almost 2,000 cases can be laid at my door. This is roughly 1 per 1,000 cases which compares favourably with any statistics I know of either institutional or extra-institutional with the exception of the East End Lying-in Home, London, whose record is almost miraculous. Remark that this fatality rate is also my total fatality rate and the position is even better. I cannot pass, however, from this part without putting on record the sorrow and anxiety those cases gave me.

I am of opinion that the above record is not at all singular but is being equalled if not excelled by the average conscientious general practitioner today.

Notification.

Since September, 1929, in Scotland, puerperal sepsis has been notifiable. Any case within 21 days of birth or abortion exhibiting a temperature of 100.4°F. or over for 24 consecutive hours, must

be notified. The case is then removed to an infectious diseases hospital. This shows the great pull the Public Health Officials can exert. That the maternity leaders in our profession should have consented to any such arrangement is astonishing. It means that almost all cases of puerperal temperature are in fact removed to a place which is already housing erysipelas and scarlatina. There, if anywhere, is the place for the Streptococci Haemolytici to be gathered together. What spacing is required to prevent one patient infecting another with Streptococcus Haemolyticus is entirely unknown. I have shown on great authority elsewhere, the fact that when a drain cover outside the hospital altogether was left off, Streptococcal infection broke out in the wards. From my own observation I know that the puerperal sepsis cases in such a hospital as Belvidere are so close together that they can join hands. Through these wards come a regiment of doctors, nurses, maids and visitors. The first three have been mingling freely with like attendants from scarlet and erysipelas wards, and in short there cannot be devised a more suitable atmosphere for the unsleeping Streptococcus Haemolyticus to flourish.

The only chance that the slight case has of not becoming serious under these conditions lies in the happy fact that these slight cases are almost all staphylococcal in character and this prevents a streptococcal infection developing as has before been shown (67).

Nevertheless this environment is not the proper one for these cases. This is a special type of infection. It should never have been allowed out of the midwifery atmosphere. After all, those who - according to the experts - are responsible for the condition, should not be relieved of the consequences. The B.M.A. standard of infectivity necessitating notification ropes in many cases that are really not puerperal sepsis at all and therefore are suffering it may be a lethal injury in being removed to an atmosphere of concentrated infection. These mild cases as a rule recover with no other treatment than posture. This, however, in their own home atmosphere or where they are in absolute isolation from other cases. Dr. Janet Campbell (99) states:- "An isolation block in a maternity hospital is the best place for treatment because the mother and child

remain under a medical and nursing staff who are specialists in midwifery".

In all these cases of Puerperal Sepsis I think that the one really serious clinical sign is a persistently rapid pulse. In the fulminating serious type the prognosis is hopeless.

Prevention.

Overshadowing all other means of dealing with puerperal sepsis is prophylaxis. It offers the one sure hope of ameliorating the present intolerable position. We start in puerperal cases with one enormous advantage - we have time to prepare. For at least six months, we have leisure to mature our plans. We have an opportunity of knowing our difficulties. This is a boon of the first magnitude. To be forewarned should be to be forearmed. The pregnant woman should be the object of careful and unremitting antenatal attention. It is here that team work should show its richest fruits. The woman comes to her doctor and states her condition. She must then be examined thoroughly. Her teeth should be examined, and an X-ray photograph of the roots being an absolute essential. Any faulty teeth must be extracted. The tonsils, nose and

ears should be inspected and swabbed and any septic focus there, dealt with. The blood pressure should be taken and the retinae examined. Any blood pressure over 140 m.m. should be narrowly watched and if accompanied by albuminuria, however slight, the patient should be passed on to an institution.

Drs. O.L.V. de Wesselow and J.M. Wyatt (100) say:- "The exact upper limit of the normal systolic blood pressure in pregnancy has been fixed at different levels by different authors, some of whom have asserted that any systolic pressure above 125 m.m. Hg. is to be regarded as pathological in the pregnant woman. We are not disposed to regard any systolic pressure below 140 m.m. Hg. as definitely abnormal.

In an interesting table they show the relation between high blood pressure and eclampsia thus:-

130 - 140	1 -32	Developed toxic symptoms.
140 - 150	1 -11	Do.
150 - 160	1 - 3	Do.
160 - 180	1 - 2	Do.
180 up.	all	Do.

Dr. John Hewitt (101) gives a table showing the relation of high blood pressure to gravity of case

in eclampsia:-

Cases of	Average Syst.B.P.	Average No.of Fits.
Deep Coma.	186-4 m.m. Hg.	16.0
- Coma.	167.0 do.	13.5
No Coma.	150.5 do.	5.6

The heart and lungs should be carefully examined. Cardiac disease especially of the nature of mitral stenosis and aortic regurgitation (the latter is rare) should be sent to an institution on the slightest suspicion of rales at the bases of the lungs or if the slightest vertigo or faintness occurs. Similarly the appearance of a short resultless cough without any clinical signs in the lungs suggests a cardiac failure in valvular disease cases. These are all institution cases. The liver and spleen should be carefully examined. The urine should be tested for albumin, sugar and its specific gravity and reaction taken. It should be centrifuged once a month in the last three months of pregnancy and the sediment studied under the microscope for bacteria and if need be cultures should be taken. The first specimen taken at the beginning of pregnancy and the last three specimens at least in the series should be catheter specimens.

The abdomen should be examined, particular attention being paid to possible herniae. Mobility or otherwise of the kidneys should be noted and if present, treated by copious fluids and potassium citrate. The pelvis and genital passages should be carefully examined, the former for size and form and the latter for infection. A conjugate measurement of $3\frac{1}{2}$" to $3\frac{3}{4}$" should be sent for consultation with a view to induction later should head of foetus and pelvic brim space be disproportionate. Only time can tell. Any lesser conjugate than $3\frac{1}{2}$" should be sent to institutions. Any pelvic deformed cases should likewise go to experts. Cystic or pathological ovary and disease of the tubes should be carefully examined for and, if found, sent for institutional opinion and, or, procedure. Uterine fibroids if found should likewise go to experts. Uterine displacements must be corrected. The nature of any vaginal or cervical discharges should be ascertained by culture and microscopy. Venereal disease should be treated. Constipation should be enquired for and dealt with by dietary not drugs. Any fissure or wounds in the anal region should be dealt with. Piles should be

injected. Septic diseases of the skin should be dealt with and careful cultural examinations made for streptococci, staphylococcal infections are not so important.

Fitzgibbon & Bigger (102) say:- "Prophylactic doses of sera were given in 16 cases, five of whom died. No conclusion can be drawn from these facts but sera do no harm". This is very poor praise indeed !! I do not believe myself that Antisera are of any use in streptococcal infections either as prophylactic or curative agents. I have never seen a single one benefit from them. This ante-natal case should extend of course to the observation of the unborn child. We must know how it lies in the womb. If it is healthy or not. If it is alive or not. Its relative size to the mother's pelvis, etc. The trained hand can do all this. It is possible in fact for the skilled finger to feel the foetal heart beat of the unborn child. The correction of faulty positions is a matter of simple mechanics. The general bloodstream should be examined, Wassermann tested and sugar threshold found. The presence of any primary anaemia should be ascertained and if present treated.

Finally, if in doubt, an X-ray photograph of the position, relative size, actual presentation, etc., should be taken in the final month. Any abnormality revealed should be dealt with. I am confident that were such a system of antenatal care in action today, avoidable puerperal sepsis would no longer be present with us. I am one of those who think that long before the woman has taken her first labour pain, our work as obstetricians should be almost over. Interventions when the crisis is on us means an overlook of some sort. There remain, however, two exceptions to this. One cannot foresee a short cord - that is a cord either artificially shortened by being twined round the child's neck, etc., or a cord so short in itself as to prevent cranial descent. The second condition is prolapse of the cord. No antenatal care can deal with these. Apart from these, I know of no other condition except the haemorrhages against which we should not have prepared.

This all means, of course, a greatly improved obstetrical service as regards training and education. This applies to all concerned. Nurses, general practitioner and specialists. The Public

Health Official may remain of course critical and olympian. There his sphere ends. It is the duty of the rest of us to cease from mutual recriminations and by our good work give no room for the activities of either the streptococcus haemolyticus or the Public Health Official. It should be our ideal to deprive both of these of the necessary pabulum. Now if some get their way all midwifery will be in institutions. Waiving for the moment the fact that this condemns the pregnant woman to a risk almost seven times as great as she faces now at the hands of the midwife - the figures given by Kinloch (103) are:- Institutes 14.9, general practitioner 6.9, nurse 2.8. The question that faces the profession is, where are you going to get your general practitioners and specialists of the future should midwifery be institutionalised? No man can live on air. So far as I know there is at present no institution in Britain or Ireland which exists for maternity cases, but is either on a pauper or quasi pauper basis. I quote a newspaper report dealing with the most prominent local effort to put obstetrics on a paying basis in an institution. This hospital deals with over three hundred maternity cases a year. The report

shows its financial state (104).

"Sheriff-Principal A.O.M. Mackenzie, Glasgow, has upheld the appeal of Redlands Hospital for Women, Glasgow, against the imposition on them of municipal assessments for the current year as owners and occupiers of property at 9 and 17 Lancaster Crescent, Glasgow.

In his judgment Sheriff-Principal Mackenzie points out that the hospital is managed by a voluntary committee of prominent ladies in Glasgow and district. It has for its special object the treatment of women by members of their own sex, and is intended for women of limited means who cannot be treated in their own homes or afford the expenses of a nursing home, but to whom the publicity of a general infirmary would be a real hardship. The hospital consisted of two separate buildings, and was not self-supporting. It appeared from the accounts for the year ended November 30. 1930, that the income from patients in both parts of the institution amounted to £5,163, whilst the total expenditure amounted to £8,528. After crediting the public charitable contributions of £1,084 and income from other charitable sources there was a

deficiency in ordinary income for the year of £1,465.

Not Self Supporting.

"It appeared from evidence led before him in support of the appeal that if all the medical and surgical patients treated in the hospital paid the highest weekly charge of £4.4.0. the medical and surgical branch would be just self-supporting, and that if all the maternity cases paid at the highest rate charge, £6.6.0. per week, the amount obtained would meet all the expenses of that part of the institution, but the admitted figures to which he had referred demonstrated that the contributions made did not in fact nearly meet the necessary annual expenditure.

The Glasgow Police Act of 1866 provided with regard to the municipal assessments which it authorised that the corporation should not impose any assessment in respect of any building which was solely occupied for purposes of religion or public charity. The contention of the appellants was that Redlands Hospital was occupied solely for the purpose of public charity, and that they were entitled to exemption from municipal assessments on

that account. The argument for the corporation was that the provision in the Act only applied to buildings occupied for the purpose of giving public services free of charge, and did not extend to institutions where the recipients of the benefits conferred contributed towards their cost.

A Considerable Deficiency.

"After quoting the case of Chalmers Hospital versus the Magistrates of Edinburgh, Sheriff-Principal Mackenzie states that in his opinion it makes no difference that certain of the patients treated in both of the branches of the institution make contributions sufficient to meet the cost of their maintenance, for all the funds received from the patients are applied to the expenses of the trust, and after they have been so applied there is a considerable deficiency, which has to be made up by donations and charitable subscriptions. His opinion was that the appeal must be sustained".

There are no plums inside and if there be none outside who will be found quixotic enough to undergo an intensified training to specialise in a pauper hospital? One should not lift a finger lightly against the present day arrangement lest he

sweep away, it may be, the basis of all practice. I contend that the better trained nurse, the better trained general practitioner and the better trained specialist, each working in his or her special sphere - a band of brothers - is the ideal to pursue here.

This postulates as a necessity what I have all along contended and have sought to prove by actual figures, viz. The proper place for a normal midwifery case is her own home with her own nurse and doctor. The proper place for abnormalities is the institution under a specialist. Socially, economically and scientifically, this is the best solution of puerperal asepsis.

This being granted, I think that all maternity nurses should have a more prolonged training and a wider field of activities as obtained in Holland and the Scandinavian countries generally. Jellett (105) says:- "In Holland and the Scandinavian countries the midwife has at her command a body of medical practitioners who are far more highly trained in midwifery than are the practitioners of the British Empire. Further, these again have behind them a considerable number of highly trained specialists".

Dame Janet Campbell (106) says:- "The medical

student does not yet always receive instruction and practice in accordance with the recommendations of the General Medical Council while opportunities for post graduate experience of Midwifery are most inadequate. It is suggested that the whole question of the training and supply of midwives is in need of re-consideration, particularly from the point of view of (a) securing the best facilities and teaching for the pupil who intends to practice as a midwife after gaining her certificate, and (b) of making the profession of midwife attractive to the well educated woman".

"The maternity service of the country as a whole needs strengthening and improving. A complete maternity service which secures to every woman such assistance as is needed to ensure her a safe pregnancy, a well conducted labour, careful nursing and postnatal supervision cannot be provided without wise and far seeing organisation... Such a service might well be based on a scheme for improved domiciliary midwifery in which normal deliveries and all maternity nursing would be performed by midwives with the active support of the patient's own doctor who would be responsible for

the antenatal care, the conduct of normal labour, the treatment of any complication arising in pregnancy or childbirth, the oversight of the lying-in period and the care of the infant. Supplementary facilities - ante- and postnatal clinics for advice and consultation, specialists to act as consultants to the general practitioner, skilled nursing, and maternity beds for such women as require them would be provided by the local authority in order that the standard of actual obstetric practice may be raised". She then goes on to press for an effort to organise and educate public opinion and emphasises the fact "that 'Time, Money, Effort and Goodwill' are all needed in the solution of this problem".

Passing then from antenatal care to labour itself, what are the preventive measures that can simply be taken in the average case? What, in short, is the most effective and cheapest? I speak of course from the general practitioner's standpoint.

First, a word as to personal asepsis of the average general practitioner.

Dame Janet Campbell in her Report on Maternal Mortality remarks that it is an advantage that the

nurse is not likely to be constantly septic as the doctor is. "The nurse is not, like the doctor, constantly liable to contamination of the hands with septic material and the need for gloves is correspondingly lessened".

I would remark that the general practitioner carries with him sterile rubber gloves and does not touch any septic or morbid material without them. He is therefore no more likely to carry sepsis than any other person. I would also remark that the nurse should wear sterile gloves at the confinement and during the puerperium when attending the patient. The room should be bare or have linoleum on the floor. There should be no rugs or carpets or hangings of any kind. The bed should be in a good light and just off the line between door and window. I fear draughts. The bed should be a single one and have a firm non-sagging mattress. It should have on it, beneath the patient, a clean sheet, a large rubber sheet extending from the patient's shoulders to her heels, in length, and overhanging both sides of the bed in width. On this again, should be placed a sterile sheet, folded in four and extending to cover the rubber sheet.

The patient should be dressed in a sterile cotton gown and have on a sterile skirt. The room should be thoroughly washed down with Lysol and water and the floor scrubbed with Carbolic soap. The bed itself should be taken to pieces, thoroughly scrubbed in antiseptic, dried and put together again. The woman immediately prior to labour commencing should have a soap and water enema. After it has acted she should be scrubbed down under a spray or scrubbed standing in a bath or tub. No water that has touched her body should be used again. A lying down bath is a danger to which the parturient woman should not be exposed. The contaminated water, bacteria laden from her body, is apt to find ingress to the vagina. After bathing, her hair is pleated, wrapped round her head and a sterile cap put on. The woman is warned on no account to touch herself below the level of the waist. The body from the waist to the knees is thoroughly scrubbed with Lysol and warm water. It is then dried by sterile towels and surgical spirit rubbed on by the sterile gloved hand of the attendant. A sterile pad of voluminous character is then applied and a pair of rubber knickers donned. I do not shave the parts.

It is a dangerous proceeding as any slight cut or abrasion or even the epilation of one hair is the chance for the Streptococcus Haemolyticus. The hair is a natural protective here and its after care presents no difficulty. I have never therefore interfered with the natural hair. The woman is allowed up and sits by the fireside chatting with the attendant. When her pains come on she goes to her knees at the chair she sits on, in the attitude of prayer and stays there till the pain ceases. Any further voidance of urine or faeces is done into a sterile chamber and following it, the pad is changed and the parts again washed and spirit washed. A particular point is that on no account is a swab ever to be passed from the anus to the vagina. The movement is always from vagina backwards. This is cardinal in importance. So labour goes on. Light nourishment may be taken. Tea and biscuits or bread and butter, beef tea, soups, etc., but no heavy meal is allowed. As the pains increase in violence, I give 1/6 grain of Hypodermic Morphine Hydrochlor and repeat this when the head is well engaged in the pelvic brim. That is all the narcotic I ever use. In no case is vaginal

or rectal examination needed or done. All information one needs can be got from the supra pubic region. One can estimate the progress of the descent of the head with finished accuracy. The examining hand even here must wear sterile gloves. There are only two conditions where this form of examination is inadequate. (1) Short cord. (2) Cord prolapse. A per vaginal examination would not disclose the first and is not needed for the second. The prolapsed cord discloses itsown presence. Antenatal care has doneaway with the necessity for any per vaginal examination in labour. I will not go further into the matter of "short cord" than to say that it is a rare but serious complication. It may be diagnosed by a feeling of traction over the site of placental attachment in the uterus and most particularly by a rise in pulse rate and a failure of the head to descend and engage in the pelvis.

In replacing prolapsed cord an anaesthetic of a slight character is invaluable as an aid. Chloroform is best as it lessens uterine contractions. The patient being lightly anaesthetised her buttocks are raised and she is turned on her face. This

tends to allow the head to withdraw from the pelvic brim. The sterile gloved hand is then inserted under strict aseptic precautions and the cord well replaced. It is no use unless the cord is put up well over the child's head. It should be pushed over the face and tucked beneath the chin. The patient is then lowered to the bed, and put upon her back and allowed out of the anaesthetic. Cord prolapse, however, always means bad engagement or malpresentation and antenatal care should have dealt with this. While the hand is in the vagina replacing the cord, the malpresentation should be examined for and corrected.

When the membranes rupture the woman is once more changed and sterilised as before and put to bed. The rubber trousers are removed and she is made to squat on the bed in the defaecating position during pains. This in my opinion, is the original and natural position. It is easiest for the woman and ensures drainage and prevents insuction. It also does away with laceration of left side of cervix which is, in my opinion, a lesion of faulty position during labour. The the birth of the head the woman sinks her buttocks down to within six

inches of the bed and the child is received into the sterile gloved hands of the attendant. The child is removed and its eyes, nose, mouth and ears cleansed with Boric lotion and weak Protargol solution. There is never any need to offer violent manipulations to the child. If it lives, it is in spite of these exertions. The uterus is gently controlled and rubbed and in time it expels the placenta which is received into a large sterile plate which is placed beneath the vagina immediately the child is removed. The uterus is gently massaged for ten minutes thereafter; a second sterile plate receiving the discharges. The woman's pulse showing a slow and steady beat, she is left crouching in the knee elbow posture over the plate till baby is washed and dressed, etc. The nurse then wearing sterile gloves washes her down with moist (but not wet) sterile antiseptic swabs. There must not be any flow of fluid on the patient's skin. She is then dried with antiseptic towels and spirit washed and a sterile pad applied. All is then removed from her body except the gown which has been tucked up round her shoulders and all from the bed from the rubber mat upwards. A new and similar

outfit is applied in their place and the woman is left to sit on a circular cushion shaped like a large corn plaster, with her body raised to a half right angle. This secures drainage and prevents insuction. Care must be taken that all tears and lacerations be known and dealt with. For this purpose before binding the patient up she is turned round in the knee elbow posture and the perineum examined for tears. A good electric head lamp is an essential here. Such lacerations should be carefully sutured with silk worm gut. A cervical laceration should not be searched for at this stage and if found should not be dealt with. It is best done later when there is no placental site to become infected. Dr. Jellett (107) advises this procedure. The placenta and membranes should be examined under water for missing pieces. Small pieces amissing are best disregarded as they are not worth the risk of intra-uterine search. Careful, faithful and gentle massage has never failed with me in securing the evacuation of such debris. Douching or douching plus curettage is a dangerous proceeding and one I have never done. Even those who recommend this step are careful to emphasise

its gravity. Thus, Dr. Jardine of Glasgow (108) says:- "An intra-uterine douche should be looked on in the light of an operation which must be done with the utmost care".

Dr. Eustace Thorpe (109) says:- "Douches after birth should in all cases be supervised by a competent person".

I am firmly of belief that the danger of introducing sepsis is in these cases so great that douching and curetting should have no place after birth in modern obstetrics. Posture and gentle massage will secure perfect results in almost every case. Every maternity case should have an ample supply of sterile dressings and in Glasgow the Public Health Authority will supply such to suitable persons.

Puerperium.

Twelve hours after delivery, the patient is raised to the sitting position, the buttocks being raised, however, from the bed on a circular air cushion suitably aseptic. This allows free drainage of lochia and prevents insuction. She is encouraged if there be no perineal tear to move the limbs and body freely. This prevents venous stasis

which is the precursor of thrombosis and embolus. I have never had a single case of embolism. On the sixth day, she is allowed up to a chair for short intervals. The breasts are carefully swabbed with antiseptic and spirit, and finished with clear cold water and dried with a sterile towel. Boracic Lint is put over each nipple and removed if wet with milk and the process repeated. They are washed before and after feeding as is the baby's mouth, with weak Boric Acid solution. Since adopting this regime, I have had no cases of breast abscess. The patient gets up for increasing intervals at the tenth day; and at the fourteenth day, I look on the case as needing only occasional visits till two months after labour. An examination from time to time for misplacements and their correction completes the case.

Above is the description of a normal case. These constitute roughly 80% - 95% of all cases. The remaining 5% will require forceps delivery. My practice is to wait till the os is fully dilated and no progress has been made by the head for over an hour. The instruments are boiled and taken directly out of the boiling disinfectant by myself

with sterile gloved hands and so applied. I would like to write in letters of gold the following advice in forceps delivery. Imitate Nature but just add the little more. Be slow, be steady in your pull. Be gentle. Release the lock between pulls. I am convinced that most of the damage done by forceps is in not putting them on at the right time and not using them properly when on. One should feel that the forceps was just your extended hand and one's brains should be in the patient's pelvis along with them, visualising what is happening, appreciating the different strains on different structures. The more sensitive your forceps become to you, the better your work will be. Forceps application is an art and a joy when correctly done.

I have stated my beliefs regarding institutions. These are fit places for abnormal cases and that is their proper and only place. The danger of sepsis arising and its serious dimensions and rapid spread in these institutions is a matter of common knowledge. Dr. Jellett (110) says:- "The short - comings of maternity hospitals are so obvious and their connection with maternal mortality so clear that public recognition of the fact is eventually

certain".

Drs. Kinloch, Smith and Stephen (111) say:- "Once a carrier of a virulent strain of streptococcus is introduced into a crowded maternity ward the velocity of infection is such that the most scrupulous asepsis will fail to prevent the spread of infection".

To show what an institution is capable of doing in the way of puerperal sepsis propagation, I append tables by Dame Janet Campbell, (112) of a British institution run by specialists.

"Another example is afforded by a maternity hospital which is a well-planned, modern building, the wards being arranged in separate blocks. It has a specialist medical staff. The incidence of puerperal pyrexia has been higher than is satisfactory at this hospital. The following table shows the number of cases which occurred between 1923 and 1926:-

Date	Total Cases	Total cases of pyrexia. (Over 100.4° for 24 hrs.)	Cases of "considerable pyrexia" (Over 101° for more than 24 hrs' duration)	Notified case of P.F.	Deaths in the series of febrile cases.
1923 Jan.1st-May 31st.	590	113=21.4 per cent of total cases.	103=91 per cent of febrile cases	10	8
1924 Jan.1st-June 30th	568	49=8.6 per cent	37=75.5 per cent of febrile cases.	4+1 Erysipelas.	1
1924 July 1st-Dec.31st.	590	53=8.9 per cent	45-85 per cent of febrile cases.	2+9 S.F.	3
1925 Jan.1st-June 30th	633	52=9.2 per cent	32-61.5 per cent of febrile cases.	1	0
1925 July 1st-Dec.31st.	669	49-7.3 per cent	35=71.4 per cent of febrile cases.	None	1
1926 Jan.1st-June 30th	711	96=13.5 per cent	83=86.45 per cent of febrile cases.	10	2

The above speaks for itself.

Remember that primaeval woman went - as the beasts still do - apart and in solitariness begat her child. Let us learn to imitate her and be wise. There is no bed spacing in an institution so wide as to form a barrier to streptococcus haemolyticus.

Segregation and not aggregation is a cardinal factor in puerperal sepsis diminution.

Treatment.

If in spite or all these precautions infection should supervene, treatment really resolves itself into good nursing. A four hourly chart should be kept and there should be at least two nurses in attendance. The nursing should be four hours on and four hours off as the duties are exacting. The Patient should be kept in a well lighted, sunny, well aired room. She should have an air bed or waterbed. The room should be kept cool, about 56°F. to 60°F.

Eustace Thorpe (113) douches and swabs with Iodine at least once. He does this under an anaesthetic. This, however, is only in cases or less than five days duration. After this, he thinks it is of no use.

In another portion of his work Thorpe (114) says:- "Vaginal discharges of all kinds should be treated by douches".

Fitzgibbon and Bigger (115) say:- "Doughing is of use only in certain mild cases and is always a danger". They are emphatic in their hostility to curettage or any attempt to remove material from the Uterus even with the finger.

My attitude to douching, and, or, douching and curettage is that if proper handling at labour has failed to prevent sepsis then the only and best procedure to adopt is that of Posture and supporting treatment, dieting, sponging, etc. I am convinced douching does no good and curettage is an assault. To take the analogous case of tonsillitis. A septic tonsillitis is not treated by gargles because no amount of gargling has any effect on the septic infection in the tonsils. Here the patient is able to douche her throat a dozen times a day with an antiseptic, yet it is of no avail. How can one then expect better results in the female genitalia? Moreover, if one douches a mild case this is generally mild because the infection is localised and more or less external. We thus run the risk of carrying in infection by douching and transforming a local into a general infection.

The Nurse should note all rigors, faintnesses, vomiting, bladder or bowel pains, etc. on the chart. All discharges should be carefully scrutinised. Copious fluid should be given. Cold water in these cases or, even better, cold water with a

dash of good whisky in it is very beneficial. It is considered routine to give temperature reducers such as Quinine, Aspirin, etc. I have never seen them do any good and both have unpleasant consequences. Quinine causes deafness and blindness and this is very alarming to the patient. It has no effect on the infection. Aspirin causes gastric pain and catarrh of a very intractable type. It also has no effect on the Infection. Vaccines and Sera have never done any good in my experience.

Both Eustace Thorpe and Fitzgibbon and Bigger agree that they are of little use.

Repeated examinations should be made for Pericarditis, Pneumonia and Peritonitis. In fact inflammatory extension can appear in any organ or tissue. Abscess formation must be met with, incision and free drainage. I have great faith in simple general measures. Cold water is the sheet-anchor; sponge and sponge and sponge again. Encourage drinking of cold water. Fill the Rectum with cold water. Wash the mouth repeatedly with cold water.

In 1925 (116) the British Congress of

Obstetrics and Gynaecology had under consideration Puerperal Septicaemia. In their note on Treatment, they say:- "We had hoped to make some useful deductions on this subject but have been unable to do so". There were some sixty different methods of treatment or combination of different methods but the streptococcus was uninfluenced markedly by any of these!

Again, (117) the North of England Committee's Report says of Treatment:- "In investigating the results of treatment, the Committee have been faced with the difficulty that a variety of different methods have been employed in almost every case and it feels unable to express an opinion on the efficacy of any particular method".

Dr. J. Whitridge ~~Wilson~~ (118) speaking on Puerperal Sepsis to the Congress of Obstetrics and Gynaecology said:- "All the information went to show that they were practically helpless in the treatment of severe infections. When women had general Streptococcal Peritonitis, they were likely to die, whatever one did. Pyaemia if left alone, 2/3rds died, if treated properly 1/2 died, on the other hand, less severe cases, if left

alone, might recover; prognosis depending on the Patient's resistance and the organism's virulence.

H.N. Green and E. Mellanby (119) advocate diet rich in Vitamins A and D as a preventive of Puerperal Infection. Their experiments on rats led them to think that a large Vitamin A content in diet prevented the infection of the rat in the puerperium with puerperal sepsis!

Cameron and Thomson (120) applied this principle to puerperal cases in Bellshill Hospital. They advise their patients to partake freely of a diet consisting of fresh vegetables, carrots, cheese and liver.

In addition, they administer prophylactic injections of anti-streptococci puerperal serum. "50 to 70 c.cm. are given during labour or a few days prior to labour if trouble is anticipated. Procrastination deprives the patient of an opportunity to escape the sequels of infection".

The only inconveniences they remark were the appearance of a rash and the not infrequent onset on the eighth or tenth day of the puerperium of a haemorrhage. These phenomena occurred in only 15% of the cases and never gave cause for any anxiety.

In addition they give radiostoleum or adexolin to the patients for its Vitamin A and D content.

I append a table which they give which shows their results:-

Date.	No. of Cases.		Puerperal Pyrexia According to B.M.A. Standard.	
			Number.	Percentage.
1925.	Confinements 517)			
	Post-natals 26)	665	50	7.5
	Abortions 122)			
	Of the 517 confinements the number normal was	476	18	3.8
1926.	Confinements 696)			
	Post-natals 26)	848	55	6.5
	Abortions 126)			
	Of the 696 confinements the number normal was	380	11	3.0
1927.	Confinements 726)			
	Post-natals 47)	898	45	5.0
	Abortions 125)			
	Of the 726 confinements the number normal was	432	Not known	
1928.	Confinements 707)			
	Post-natals 32)	864	41	4.7
	Abortions 125)			
	Of the 707 confinements the number normal was	432	10	2.3

Date.	No. of Cases.		Puerperal Pyrexia According to B.M.A. Standard.	
			Number.	Percentage.
1929.	Confinements 543) Post-natals. 21) 703 Abortions 139)		20	2.8
	Of the 543 confinements the number normal was	279	Nil.	Nil.
1930.	Confinements 700) Post-natals 40) 900 Abortions 160)		19	2.1
	Of the 700 confinements the number normal was	308		

In 1925 and 1926 no serum was used, and in 1927 and 1928 a limited supply only. In 1929 and 1930 serum was used in all complicated cases, and in normal cases with perineal lacerations.

This shows a very great improvement indeed in the results following serum injections. It must, however, be remembered that the East End Lying-In Home in London can show a rate of .6 per 1,000 births without any such measures being taken. The authors make special mention of the fact that the treatment is costly.

I give this outline here as it opens up a very large and interesting field so far largely unexplored, on the relation of diet and its vitamin content to the susceptibility to Puerperal Sepsis. The results so far seem promising.

Looking back over this writing, I feel I must draw attention in conclusion to the immense importance of painstaking Ante-natal Care in the crusade against Puerperal Sepsis.

I have thought over my own Septicaemia cases for instance, and asked myself where ante-natal care fell short and what would have been the sepsis rate if perfect ante-natal care had been in evidence.

In the first case, the presentation being a breech in a primipara necessitated handling. Had this been corrected then the labour would have been normal and therefore there would have been no handling and probably no sepsis. Result one death less.

No. 2. raises the question of Scarlatinal contact. Efficient Ante-natal care here would have insisted on the confinement being furth of her own house and with fresh sterile clothes, etc.,

and so probably another life would have been saved.

No. 3. Putrid foetus case. Ante-natal care here would have discovered the death before putridity developed. May even have discovered by blood test the syphilitic state of the blood and treatment might have saved the life of the child and the illness of the mother.

No. 4. Here the swabbing of the attendants-to-be, relatives, etc., would have shown if the husband was a haemolytic carrier and measures could have been taken accordingly. If on the other hand my contact with the unknown scarlet fever case was the infecting source, no ante-natal care could have foreseen that, so this case is doubtful.

No. 5. Ante-natal care would probably have not made any difference here.

No. 6. Ante-natal Care to be efficient must take into account the haemolytic possibilities of relatives, attendants and Patient. Had this been done here and the erysipelas of the Nurse's leg known, this woman could have been saved.

No. 7. No. 8. Above remarks on No. 6. may apply here but on the whole Ante-natal care would

have had no effect here.

No. 9. Swabbing of husband's throat, etc. would have disclosed Streptococcus Haemolyticus and preventive masks would have saved this case probably.

No. 10. The Nurse's throat should have been swabbed before resuming duty. This would have saved the sequelae as stated above.

We therefore reach the reasoned conclusion that exact Ante-natal Care in my own cases would have possibly saved the lives of four mothers and one child, and prevented the illness of two mothers. In addition, it is just possible that it might have saved still another life (No. 4. of my series) depending on who was the cause.

This means that instead of ten cases of Puerperal Septicaemia, I would have had three only, one of which was fatal.

That is to say that Ante-natal Care could have enabled me to confine in their homes under all social circumstances almost 2,000 women with a total casualty list of three and only one death. In short, Maternity instead of being a dangerous occupation should really be an entirely safe proceeding. It can be made so.

"SURSUM CORDA"

Summary.

I hold the following proved.

Puerperal Sepsis is widespread in Nature both in lower animals and mankind.

It obeys the same laws in both animals and man.

Its incidence is so great that child-bearing is classed as a dangerous occupation.

It is of prime importance to the State, Medical Profession and Individual.

It has been known since the earliest times.

It has shown little or no diminution in incidence or fatality in the last twenty years.

Its cause is now almost exactly established.

The infection is due to Streptococcus Haemolyticus the great proportion being exogenous.

Streptococci are characteristically heterogeneous in character and Erysipelas, Scarlatina, and Puerperal Septicaemia infections are so closely allied as to be indistinguishable constantly bacteriologically and serologically.

There exists an "Unknown Factor" that determines invasion.

The more rigid our asepsis the lower is our rate of infection.

Prevention is the only means at our disposal for lessening the mortality rates in puerperal sepsis.

Scarlatina and Puerperal Septicaemia were demonstrated clinically to be like infections.

The ideal place for normal confinements is the woman's home and the ideal attendants are the General Practitioner for Ante-natal Care and general supervision and if need be, intervention at actual labour on the call of the Nurse.

That abnormal cases should be confined by Specialists in Institutions.

That the worst place possible for removal of cases showing notifiable temperature is a general Fever Hospital.

That each should be isolated in a separate block of a Maternity Hospital under experts.

That the average Scottish General Practitioner can show statistics equal to anything in Europe either Institutional or other.

That puerperal Sepsis will not yield unless to team work of a high degree of excellence.

That an atmosphere of blame is fatal to progress.

That proper Ante-natal Care should reduce puerperal sepsis to vanishing point.

That Segregation not Aggregation is the corner stone in Prevention and Treatment of Puerperal Sepsis.

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