An Address

ON

MATERNAL MORTALITY FROM
PUERPERAL SEPSIS.*AN ANALYSIS OF THE FACTORS OF CONTAGION, TRAUMA,
AND AUTO-INFECTION.

BY

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It is unnecessary in these days to emphasize the tragic importance to the community of the large mortality amongst young women which is incurred yearly through child-bearing and childbirth. In these islands the maternal death rate is about 4,000 each year; between 1911 and 1926 inclusive there were 66,421 deaths from these causes in England and Wales. Septic infection is by far the most important single cause. According to the official figures for 1926 the total maternal mortality for England and Wales was 5.14, and the sepsis mortality 1.60, per 1,000 live births.

This loss by death, however, is in some ways not the gravest consequence of child-bearing, for we have come to realize that, for each mother so lost, there are many more whose health is in varying degrees chronically undermined by the damage they have sustained in childbed. Infection and mechanical damage thus acquired are amongst the main causes of the frequent chronic ill health which dates from childbirth. We can assess within accurate limits the loss by death, but of the incidence of persisting invalidism of child-bearing origin we have no exact knowledge. We know with certainty, however, that many of the common gynaecological disorders, the uterine haemorrhages, the leucorrhoeas, the pelvic pains, the displacements, etc., and many general disturbances, debility, indigestion, neurasthenia, rheumatism, etc., are to be traced to the lesions of childbirth. It is probably not an over-statement to say that about 60 per cent. of hospital gynaecology is a legacy from vitiated child-bearing, and of this a very large part falls into the category of infection.

Septic inflammation of the genital canal during labour or in the puerperium may, as a useful basis for analysis, be considered as falling into one or other of three clinical types according as it is caused by (1) contagion, (2) trauma, or (3) auto-infection.

In the case of *contagion* we are dealing with an invasion of the passages by a virulent septic micro-organism, which is introduced from without. Here, as in other infective diseases—for example, scarlet fever, erysipelas, typhoid, or tetanus—our etiological quest is dominated by the consideration of the nature, virulence, and source of the infecting microbe. This type of puerperal infection is seen in its simplest form when it occurs after a normal, easy labour, with an absence of trauma of the maternal passages, and in this category are to be classed the epidemic scourges of the maternity hospitals of a former day.

Puerperal sepsis due to *trauma* is, in its essential etiological features, entirely different from that caused by contagion. Here the factor which dominates the issue is a lacerating or contusing damage of the walls of the birth passages, and the source and nature of the infective organisms become a matter of comparatively secondary importance. By this is meant that it is the trauma of the soft parts that determines the risk of sepsis. The infecting microbes which can determine the infection in such a case are notoriously widespread in nature. They may be considered as being derived exogenously or endogenously; in either case they may conceivably consist of micro-organisms which only cause disease when settling in devitalized tissue. The point of clinical importance, however, is that these somewhat theoretical matters concerning the origin of the microbe are overshadowed by the vital issue of trauma.

The third clinical type of puerperal fever is that caused by *auto-infection*. Here the micro-organisms which set up

the pelvic inflammation are, in some cases, present in the genital canal before labour begins—for example, in an infected cervix. In other cases they reach the genital passages from some distant source by direct spread or by implantation—for example, from the bowel—or they may travel by the blood stream from areas of focal infection in teeth, tonsils, etc. Auto-infection, like contagion, operates in its simplest form when it supervenes in a normal spontaneous birth in which there has been a minimum of interference and trauma.

The essential distinction between these three clinical types of puerperal infection is in no way undermined by the consideration that, in some cases, two of the factors concerned may be operating together. Where, for example, trauma has occurred the fact of contagion or of auto-infection may constitute an important contributory agency. Before we can hope to grapple with the menace of puerperal fever a knowledge of the manner and degree in which these three factors operate is necessary. Until we have this knowledge much of our effort must be largely haphazard. My purpose in this paper is to draw attention to some considerations which, although they claim no finality, point to possible avenues of approach to this intricate problem.

AUTO-INFECTION.

The importance attaching to this mode of infection becomes apparent when we note that within recent years many observers have attempted by its means to explain infection occurring in those spontaneous cases which have not been examined at any stage of labour. Further, it is clear that, if operating in an appreciable ratio, it would go far to explain the persisting high death rate from sepsis despite the application of the aseptic principle. Victor Bonney and others have urged its importance with considerable force and plausibility, and its possible significance is referred to in the recently published report of the Committee appointed by the British Medical Association (*Supplement to the British Medical Journal*, April 28th, 1928, Appendix iv, p. 165). Undoubted examples of auto-genous infection can be cited—for instance, puerperal fever arising during scarlet fever, erysipelas, etc.

A consideration of the available data, however, suggests that as a factor in the death rate autogenous invasion probably plays a minor part. In a subsequent part of this paper I shall refer to some facts which emerge from a study of the practice of the maternity hospitals during the pre-Listerian days, and shall educe evidence that in these old lying-in institutions puerperal fever was a negligible cause of death during those periods when contagion was obviously in abeyance. Equally suggestive evidence may be found in an analysis of the records of many extensive maternity practices at the present day. The extern practice of the Edinburgh Maternity Hospital shows a consecutive series of about 5,000 spontaneous births with 2 deaths from sepsis; the extern department of the Birmingham General Hospital records 888 cases with an absence of sepsis mortality; whilst there is the record of 47,503 deliveries, both normal and abnormal, in the unselected practice of the East End Maternity Hospital in London with 5 deaths from sepsis, or 1 in 9,500. The practice of the Queen Victoria's Jubilee Institute midwives is likewise instructive. During 1927 there were 53,502 deliveries with 6 deaths from sepsis in normal spontaneous births, or 1 in 8,900 of the total. If we include all the deaths in this record of 53,502 cases which can directly or indirectly be attributed to sepsis, and including normal and abnormal cases, we get a maximum figure less than 0.5 per 1,000. To the production of this figure all causes have contributed.

The fact which seems to emerge clearly from these data is that the very small sepsis rate in these large and unselected bodies of women is an argument against self-infection operating alone being an appreciable component of the sepsis death rate in the rest of the community. It is especially important that our ideas on this question be clarified, because much of the uncertainty and confusion in the minds of those who are anxiously seeking a remedy for the present state of affairs springs from the difficulty in assessing the true significance of this factor.

Much recent bacteriological research has been directed to the elucidation of these questions, more especially in an

* Communicated to the Edinburgh Obstetrical Society, May 9th, 1928.

attempt to relate the vaginal and cervical flora with the incidence of puerperal sepsis. As a result of this work we must now concede a comparatively frequent occurrence of streptococci in the genital passages of pregnant women, but, although it is established that puerperal infection is commonly caused by strains of streptococci, there is no evidence that the streptococci frequently present in the genital canal can play any part in initiating this infection. The fact of their frequent presence, combined with the rarity of puerperal sepsis in the absence of other factors which have a determining role, such as trauma, implies that this streptococcal flora possesses practically negligible primary virulent properties. Another finding which bears directly on this question is that, although severe puerperal infection is usually caused by a haemolytic streptococcus, the type present so frequently in the genital canal is of the non-haemolytic variety (Bigger and FitzGibbon,¹ Lockhart,² Colebrook³). Recently Burt-White and Armstrong⁴ record the finding of streptococci of various kinds in the cervix of 40 per cent. of 55 pregnant women examined. In one only did the strain present the characters of *Streptococcus pyogenes*, the common cause of puerperal fever. In 15 of these cases, including the one harbouring *S. pyogenes*, the women were followed through their puerperium, and in none did symptoms arise.

We thus see that the consensus of bacteriological study is against the view that the bacteria of the genital canal play any primary part in puerperal sepsis. These observations are entirely in keeping with, and at the same time corroborate, the clinical observation of large series of deliveries to which I have directed attention. Whilst along such lines the inference would seem to be well established, we must, however, not entirely shut our minds to the possibility that the normally innocuous denizens of the genital canal may, under certain conditions, assume pathogenic properties—for example, after trauma and devitalization of tissue.

The data, when so marshalled, would seem to warrant as a reasonably safe conclusion the view that auto-infection as a primary factor plays a quite unimportant part in the death rate from sepsis, and that in our search for essential causes we must address ourselves to the remaining factors of contagion and trauma.

CONTAGION.

Contagion is known to play a part in puerperal sepsis at the present day, as witness pelvic infection following contact with the virus of scarlet fever, or the epidemic deaths which occasionally occur in maternity homes and hospitals. To study the features of this type of infection satisfactorily, however, we have to turn to the records of the pre-Listerian lying-in hospitals. Epidemic waves of the disease were, as is well known, the scourge of these institutions. For example, "in the large hospital at Vienna, out of 21,120 women delivered from 1840 to 1846, 2,260 died, or about 1 in every 10 mothers delivered perished, chiefly from puerperal fever."⁵ In 1774 at the Hôtel-Dieu, Paris, every patient attacked died—altogether about half the patients confined in the hospital.⁶

The striking lesson for us is that, in these old hospitals, puerperal fever would seem to have been so predominantly a matter of contagion that other factors were largely negligible. I have been strongly impressed with this somewhat remarkable fact during a recent study of the records of the old Edinburgh Maternity Hospital. From 1823 to 1844, out of 3,906 women delivered in this hospital, 75 died from all causes. The records are not complete enough for an accurate differential analysis of the causes of death for this whole period, but in one of his striking papers on puerperal fever Sir James Young Simpson⁷ gives accurate data for the period from 1823 to 1837, when there were 47 deaths among 2,890 women delivered; 36 out of these 47 deaths were, according to Simpson, due to puerperal fever. When we turn to the records we find that the distribution of these deaths is instructive. We discover that 30 occurred in three years, when obviously contagion was rife. On the other hand, during the four years 1826 to 1829 inclusive there were 821 successive births without a death. We may deduce from this that during this period

of fifteen years, when contagion was eliminated, there was little or no mortality from puerperal fever.

The records of other hospitals exhibit the same facts. Thus Collins, the master of the Dublin Lying-in Hospital, reported a series of 16,654 births during the seven years dating from November, 1826, with 88 deaths from puerperal fever, all occurring during the first three years. He says that "for the four remaining years of my mastership we did not lose a single patient from this disease,"⁷ which implies 8,000 or more successive cases without a death from puerperal fever.

In retrospect it cannot but impress us as remarkable that, despite the dangers lurking within the walls of these older hospitals, and the consequent wholesale sacrifice of women during the eighteenth and nineteenth centuries, no widespread attempt to disestablish the system is apparent. Every now and then, it is true, a warning voice is raised in reputable medical circles.* Furthermore, we know that the women midwives engaged in a bitter contest with the men midwives on this and other questions.

The fact that contagion was the dominating cause of puerperal fever in those days is brought out also by a study of the records of the outdoor practice of the hospitals. In this connexion the records of the Edinburgh Maternity Hospital from 1826 onwards, which I have recently had an opportunity of studying, are instructive. At that period the indoor department was limited to destitute cases, and the arrangement by which abnormal cases were removed to hospital did not come into operation till well beyond the time which these records cover. The outdoor practice may, therefore, be taken as representing the average type of midwifery of the class of women concerned. This was carried out by the students attached for service to the hospital and by the midwives, who, as we know, at this period had little or no scientific training.

Between 1826 and 1857 there were 15,144 successive deliveries with 61 deaths—that is, a rate of 4 in 1,000. The cause of death is given in 44, and in 19 it is stated as puerperal fever—that is, about 43 per cent. If we assume that this figure applies throughout the series the sepsis deaths would represent under 2 per 1,000 deliveries. I have shown that, in the indoor department, there were considerable periods without any mortality from sepsis, clearly due to the chance absence of contagion. The same is true of the outdoor practice. Thus there is a record of 3,288 successive births during the nine years from 1839 to 1847 with no sepsis deaths. The elimination of this element brings the total death rate during this period down to 5 in 3,288, or about 1.5 per 1,000.

The figures I have just quoted, embracing as they do a fairly extensive record of what may be taken as the average midwifery in Scotland about a hundred years ago, naturally challenge comparison with the practice of the art at the present time. Before any legitimate comparison is possible, however, it must be recognized that the above figures refer to deliveries and abortions, and do not include deaths from such complications of pregnancy as hyperemesis and ectopic gestation, which appear in the ordinary maternal mortality returns of the present day, and which constitute about 10 per cent. of the deaths in the Registrar-General's Report for Scotland. The births in Edinburgh during the years 1924 to 1926 numbered 24,173, and the maternal death rate was 5.6 per 1,000. By deducting 10 per cent. we get an amended rate of 5.1 per 1,000, as against the comparable rate of 4 per 1,000 in the case of 15,144 deliveries in the outdoor practice of the Edinburgh Maternity Hospital about a century ago.

The limited figures just quoted support the belief that during the past hundred years there has been no depreciation in maternal mortality, and this is in accord with the national statistics. For Scotland the decennial mean figures from 1855 to 1914 are 4.9, 5.1, 5.2, 5.3, 4.6, and 5.6 per

* Thus, in discussing the second annual report of the Edinburgh Maternity Hospital, December 19th, 1845, a contributor to the *Monthly Journal of Medical Science* (vol. vi, 1846, p. 269) points out that the death rate in the hospital was 1 in 25 women delivered, whereas the death rate in the outdoor practice of the hospital was 1 in 331. He then remarks that "the question, therefore, naturally arises whether it would not be better to improve the comforts of the poor women at home during their confinement, rather than advise them to enter the hospital. . . . At best, they are of questionable propriety, as they are so apt to become foci of deadly disease."

1,000 births, and the mean for the period 1915 to 1922 is 6.2. Likewise the proportion of deaths from sepsis during this period has shown little change—namely, 1.7, 1.8, 2.0, 2.5, 1.9, 1.7, 1.7.

To realize the significance of these findings we must visualize the manner in which childbirth was managed in the pre-Listerian days and how this differed from our modern methods. The first fact of which we must remind ourselves is that our predecessors had no knowledge of aseptic or antiseptic procedure. It is abundantly clear that it is to this we must attribute in the main the ravages of puerperal contagion, for they examined freely during labour, and, in cases of retained placenta, they had no hesitation in introducing the hand into the uterus. Abdominal expression of the placenta by the Credé method did not, in point of fact, come into use till about 1867. The other important fact regarding the practice of those days is that it was based firmly upon the teaching of Harvey, Smellie, Hunter, Denman, and others, who insisted that child-bearing was a physiological process, which must be left to Nature, and which was apt to be vitiated by interference on the part of the accoucheur. This was to be stringently withheld until Nature's effort had unmistakably failed. Instruments of any sort were the last refuge. Simpson, for example, in the Edinburgh Maternity Hospital, used the forceps in 1 in 472 labours, and other hospitals had a similar record. In studying these old records and noting what to us seems almost the extremity of patience with which the tedious cases were managed, one is tempted to wonder how far the factor of exhaustion and delay may have contributed to the frequency of infection. This, however, does not seem to have been a factor of any moment, for Collins, in discussing this very point, reports that in 71 out of his 88 deaths from puerperal fever the labour was completed within twelve hours.

These old records have a further bearing on the subject-matter of our discussion, for, as we know, there was little regard paid to the danger of repeated vaginal examination. Despite this, and the obvious and common contamination of the genital passages which must have then occurred, there was a negligible mortality from sepsis except where virulent contagion was present. This suggests that the microbes introduced by such contamination had little infectiveness in the case of labour conducted with a maximum regard to physiological needs.

The records of these pre-Listerian times would seem to suggest that, although contagion was a common cause of death, trauma was a factor which operated comparatively rarely, and this we must attribute to the principles which then underlay the practice of the art. I have shown that so strongly was this the case that at times the results obtained by our forefathers were such as compare favourably with the best practice of the present day.

It would clearly be to our advantage could we analyse the factors that stand behind the present high rate of sepsis as satisfactorily as is possible in these older records. Unfortunately in modern times the subject is hedged around with difficulties and uncertainties that make the study baffling in its complexity.

Contagion as a Factor in the Present Sepsis Death Rate.

How far contagion or trauma, or both combined, are responsible for the death rate from sepsis at the present time we have no accurate means of discovering. If we address ourselves first to contagion we have, however, certain data which are worthy of analysis. In this connexion we have to remember that virulent contagion notoriously may act where there is a minimum of interference, and the invasion of the genital canal may even occur when there has been no vaginal examination at all, the microbes being then conveyed to the vaginal opening on the fingers or dressings of the medical attendant or nurse, or in some other way. In pre-Listerian practice the high death rate was dependent upon such infection of the normal case, and the extent and severity of the danger were reflected accurately in the ratio of such cases involved. In any large practice the criterion of the normal case provides a sufficient index of the degree in which this factor is operating. It is clear that the omission of the abnormal and instrumental case from such an analysis does not vitiate the

result *qua* simple contagion. If the sepsis rate in such cases be increased because of the major interference employed it is legitimate to attribute such increased risk to the interference and the possible trauma incurred.

We have, at the outset, to remark that the massive sepsis of the pre-Listerian days, when surgical contagion repeatedly overwhelmed the surgical and maternity wards and often spread into extra-hospital practice, is no longer with us, and it would seem certain that the elimination of this virulent and spreading contagion must necessarily have led to a lessening of this contact factor in modern practice. Despite this, however, we know that contagion does still operate—as, for example, where the scarlatinal virus is transmitted to a parturient woman, or where two or more women are clearly infected from one common source. The exact extent to which infection so conveyed is responsible for modern sepsis is, however, more difficult to define than in the case of the older records. Whilst this is so, we are not altogether without evidence. The figures I have already quoted in connexion with auto-infection are again of service here. In the outdoor practice of the Edinburgh Maternity Hospital, for example, during four years there have been about 5,000 normal deliveries with 2 deaths. During the year 1927 there were 53,502 deliveries in the practice of the midwives of the Queen Victoria's Jubilee Institute in England and Wales with 6 deaths from sepsis in normal births—that is, 1 in 8,900 total deliveries, or about 0.1 in 1,000. This total figure refers, of course, to all cases, normal and abnormal. If we exclude the forceps cases we get a figure approximately equal to 1 in 8,000 or 0.12 in 1,000. This indicates the rarity of contagion in the practice of midwives, and when we remember that in England and Wales over 50 per cent., and in Scotland over 30 per cent. of the maternity service of the community is in their hands, we have ample reason for the belief that, in this proportion of the country's service at least, contagion is of comparatively minor importance. Then we have a record of 47,503 successive cases in the outdoor and indoor practice of the East End Maternity Hospital, with a total of 5 deaths from sepsis, or 1 in 9,500 cases. It is, of course, not necessary to conclude that all the sepsis deaths in these records are due to contagion. The figures are quoted with the object of indicating the maximum possible mortality from this cause when this is operating alone.

I have given these data not because they demonstrate necessarily any superiority in the practice of the institutions concerned over that obtaining throughout the rest of the community in respect of this matter of contagion, but for the reason that they constitute the only large series of figures available. It is not easy to conceive any reason why this small ratio of contagion should be appreciably different in the rest of the community not covered by these precise records. In other words, such an analysis as I have given leaves in one's mind the impression that contact infection, while it does admittedly still occasionally operate to maintain the incidence of fatal puerperal sepsis, is probably a factor of comparatively minor value, and that to explain the persistently high rate in modern practice we have to direct our attention to other factors.

Contagion in Present-day Hospitals.

To the enduring benefit of labouring women we have to acknowledge with ungrudging gratitude that the era inaugurated by Lister has brought assurance into obstetric practice, and in no way is this more strikingly manifest than in the enhanced safety of the modern maternity hospital and in the surgical procedures on behalf of the mother and child on which we now embark with confidence. At the same time the magnificence of this great new instrument is apt to blind us to the fact that even in the hospital system of our own times there are still perpetuated in some degree the risks with which our forefathers battled in vain. There is still intrinsic in it the danger incidental to the assembling of numbers of parturient women within four walls. The reality of this risk is at once apparent when we recall that, whilst the main practice of such hospitals is concerned with normal childbirth, it is also largely engaged in serving, and in attracting to itself, often from a wide district, the abnormal, which is often synonymous with the

frankly or potentially infected case. This is especially true of the large central hospitals, where the fact of numbers naturally multiplies the risks. A study of this problem has long convinced me that these risks are by no means negligible, for, even when the technique of the hospital is good, the risk of the leakage from infected to clean case can never be completely blocked, more especially at times when the hospital practice is working at high pressure.

In support of these contentions I may quote the figures of one well-known hospital, for which I am indebted to the registrar. During four years, in 4,278 normal spontaneous deliveries there were 72 cases of puerperal fever with 5 deaths. In yet another well-known hospital during one year there were reported 11 instances of puerperal infection in spontaneous births with 3 deaths in cases in which the birth is described as quite normal. The report of another large hospital shows that, out of 27 deaths from puerperal infection in one year, there were 4 deaths in women in whom the birth was spontaneous and uncomplicated. These hospitals are all actively engaged in abnormal midwifery in addition to catering predominantly for the normal birth, and there can, I think, be no doubt that in the majority of the instances cited the infection is transmitted by contagion from abnormal to normal case. But the added risk to the normal by no means exhausts the dangers of a hospital, for it is obvious that the difficult and abnormal case which has suffered trauma is itself especially liable to succumb to contagion. Similar instances of hospital contagion are related in the Royal Society of Medicine Report on Puerperal Sepsis⁸ and in Dame Janet Campbell's *Protection of Motherhood* (1927, p. 18).

The problem is thus seen to be a complicated one, and, although I do not on this occasion intend to enter into a full discussion of the means required to block this risk of leakage, I would remark that an imperative need of every hospital and home is a complete separation at every stage of the normal and the suspect case. The admission, the examining, the labour rooms, and the wards of the two sets of cases must be rigidly isolated from one another, and the same rigid separation must apply to the nursing. The risk of patient-to-patient transmission is further lessened by the adoption of the small ward principle, especially for the abnormal delivery. In many instances we know that maternity hospital buildings are inadequate, and, in their present state, are unable to meet the demands which a rigid application of the modern ritual of prevention implies.

I feel that, although the consideration of such questions introduces matters of some delicacy, it is urgent that they be reviewed in the frankest possible manner at this time, because, as we all know, there is a strong advocacy in some quarters of an increase in maternity hospital accommodation as one means towards remedying puerperal mortality and morbidity (Janet Campbell, Munro Kerr, Johnstone, and others). Whilst finding myself in sympathy with this attitude, I must admit that I consider any such widespread experiment as having elements of danger, except it be carried out with the most stringent regard to the safeguards I have mentioned. The exact proportion of the total sepsis death rate which this hospital contagion implies we have no means of determining. That, however, we may safely consider it as comparatively small is indicated by the meagre share of the total births which occur in hospital. The maternity hospital beds officially known by the Ministry of Health to exist in England and Wales number only 2,290 in 149 institutions (Janet Campbell, *Protection of Motherhood*, 1927, p. 64).

TRAUMA.

I have already stated that the factors behind the persisting sepsis rate are complex. One example of this suggests itself to the mind. It is often urged that we have lost to parturient women the advantages of a century's progress because we have vitiated child-bearing by a too great recourse to instrumental delivery. It is true that interference with the natural process, by instrumental or other means, was in the old days employed at the most in only one out of several hundred cases, and that, even under the best conditions nowadays, such physiological child-bearing is only rarely found. Even in the case of the practice of the Queen Victoria's Jubilee midwives, who, it may be argued, deal with the most normal class of case, the forceps

deliveries number about 6 per cent. of the total, and in the East End Maternity Hospital, where physiological child-birth is rigidly aimed at, the instrumental rate is over 2 per cent. Over the rest of the community we may safely conclude this rate is still higher.

Faced by this indisputable evidence it is not surprising that many have urged that trauma is at the back of modern sepsis. It cannot be denied that there are cases—and every member of the staff of a maternity hospital can quote instances—in which unjustified interference has led to disaster. On the other hand, to argue on the basis of such individual cases—which usually spring from lack of experience and well-intentioned though over-anxious zeal—that trauma inflicted by malpraxis is the essential cause of the persisting death rate is to lose sight of other factors which may have a wide-reaching significance.

One of these factors concerns the possible change in the fitness of women for child-bearing and childbirth as compared with their sisters a century ago. During this period there have been gross changes in the mode of life and feeding of the community. Moreover, we have to consider the possible and special bearing of rickets. It is not improbable that the increased protection of child life, which constitutes one of the most remarkable features of modern social hygiene, may have increased the proportion of those with skeletal defect who survive into adult life. As an obstetrician practising amongst the same class of women as those with whom Sir James Young Simpson had to deal nearly a century ago, I am convinced that the need for intervention on mechanical grounds is now greater than it was then, when forceps were employed only in 1 in every 472 cases.

Whilst it is thus undoubtedly arguable that traumatization has played in recent times an increasingly important part in puerperal sepsis—and the data analysed earlier in this paper would seem by a process of exclusion to lead the evidence incriminatingly in this direction—it is at the same time not improbable that at the back of it there are some structural factors not wholly within our control, and therefore not wholly preventable by us as obstetricians. Whilst making these somewhat sombre admissions we have at the same time good cause for hope, for, as I have exemplified by the instances I have quoted, there are many indications pointing towards a better and possible provision for the parturient woman if we are prepared to take advantage of them. The practical conclusion to which my studies have led me is that, whilst auto-infection can be largely displaced from the platform to which it has been raised by some, and whilst contagion likewise probably plays only a secondary part, to trauma from interference it would seem we have to ascribe the chief agency in the maintenance of sepsis, although we are not warranted in assuming that the main burden of responsibility is to be placed on the medical attendant.

The fact that, with a well-organized maternity service, a greatly diminished mortality rate is obtainable points clearly the way to progress, and suggests that the immediate problem for the community is the provision of better machinery for bringing to the help of labouring women the knowledge we possess, rather than the pursuit of a mark-time policy which awaits the outcome of further research.

In support of this contention I would instance the experience of the Queen Victoria's Jubilee Institute, which last year had the splendid record of 53,000 deliveries with a mortality of 72, or 1.3 per 1,000. It is to be noted that these figures refer to the combined practice of midwife and doctor, for medical practitioners were called in to the aid of the midwife in 25 per cent. of the cases. It is a very successful and encouraging example of team work. The midwife's task is only made possible when it is united to the knowledge that she has the doctor behind her in case of need, whilst, on the other hand, the fact that the routine management of the practice is in the hands of the midwife makes for the possibility of a more physiological attendance of the normal case than is to be expected from the practitioner, with whom midwifery is often a disturbing item in the course of a busy day interspersed with urgent calls, and from whom, moreover, regardless of every other consideration, there is too often demanded a speedy termination of labour. It may be argued that the small death rate

in this large practice is dependent upon the fact that the midwives' cases are to some extent selected. This is not denied, and that this selection is not inconsiderable is suggested by the very low death rate in the series from puerperal convulsions and albuminuria—from 0.1 to 0.2 per 1,000, as contrasted with a death rate over the rest of the community of from 0.6 to 0.8 per 1,000. Making every allowance, however, for these facts the record is full of encouragement, and points to the advantages to be obtained from a wider extension of the midwife-doctor system.

The signal advantages of such combination are even more convincingly demonstrated by such an institution as the East End Maternity Hospital in London, where the bulk of the cases are conducted by midwives, and abnormal cases are treated by medical practitioners on the honorary staff. This institution conducts over 2,000 cases yearly, about half being in the hospital. There is a well-organized ante-natal system, and the forceps rate is under 3 per cent. Despite the fact that the practice is amongst the very poor, and that it is practically unselected, it has a mortality rate standing at a little over 1 per 1,000 cases. For four years, in a total of about 9,000 cases, it sank to the extremely low level of 0.67 per 1,000. Sepsis is practically eliminated from this practice, and there can be little doubt that this is due mainly to the excellent administration, the careful supervision, and the low instrumental rate.

It is to be specially observed that the admirable results obtained in the case of these extensive practices flow entirely from the method and machinery employed. There is installed by comparatively simple means a system by which—and this is especially noteworthy in such an institution as the East End Maternity Hospital—the physiological management of labour is encouraged, the abnormal case is seen betimes, and difficulty and danger are thereby anticipated and prevented. The record of this institution and its almost complete freedom from sepsis is one of the most cogent arguments in support of the contention that the dominating factor behind sepsis in ordinary midwifery is trauma. So remarkably successful has this hospital been in its effort to expel from its doors not only sepsis, but also the other complications of labour and the puerperium—for example, eclampsia—that we might at first sight feel ourselves compelled to attribute the result to some unduly favourable circumstance in the material of their practice. Against this, however, it may be urged that the official records show that the death rates of the communities in which this hospital carries on its work are several times greater than that exhibited by the indoor and outdoor practice of this institution.

It cannot, I believe, be denied that the evidence furnished by an enlightened and critical analysis of all the various forms of maternity effort we have reviewed goes to support the contention of Fairbairn⁹ and others that "a country's maternity service is best built on the foundation of a service of midwives with medical help in difficult cases." Before this ideal can be universally realized much must be done in the way of education of the public and in the reorganization of the present machinery of practice. Doctors can do a great deal in both directions, and there is ample evidence to hand that in these objects they can count on the help of the central and local authorities. The problem in any area can only be satisfactorily dealt with by strenuous local effort.

The high death rate in some areas invites the suggestion that on occasion the subject may become eminently a matter for the active interest and participation of the local body.

CONCLUSIONS.

1. Autogenous infection is a minor primary cause of fatal puerperal sepsis.

2. Contagion is probably of comparatively secondary importance. The well-established risks of contact infection in hospitals call for care in the extension of the hospital system of maternity service.

3. There is evidence that trauma is the most important cause of the death rate from sepsis. This is not entirely a problem involving the medical attendant; it has implications of a wider nature.

4. The immediate need is an improved machinery for maternity practice based on a midwife-doctor combination.

From the standpoint of immediate policy the importance of this overshadows all other considerations—for example, "research"—and there is reason for the hope that by this means alone a lessening of the death rate is possible.

5. Improved education of the public, the midwife, and the student, and the assistance of the central and local authority, are all necessary for the creation and working of a satisfactory machine.

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A Lecture

ON

PUERPERAL SEPSIS.

DELIVERED TO THE BLACKBURN DIVISION OF THE BRITISH MEDICAL ASSOCIATION

BY

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In considering the present state of affairs relating to puerperal sepsis, it would seem that during the last twenty years no progress has been made, in spite of our knowledge of antiseptics and of bacteriological research. In support of this statement I may mention that—

1. No fewer than 3,000 British mothers die every year in childbirth.
2. Of a number of patients examined at a certain welfare centre, 50 were still bleeding at the end of several weeks, of whom 20 came from one hospital.
3. At a similar centre 42 cases returned septic, of whom 28 had been treated by general practitioners and 6 at maternity hospitals.
4. Dr. Harold Miller's investigations at Pittsburg show that of 1,000 women examined as soon as possible after the sixth week following labour no fewer than 70 per cent. showed some degree of erosion of the cervix.
5. Of the fatal cases, 80 per cent. afford a history of operative or manipulative intervention.
6. Inflammation dating from some previous confinement or miscarriage is present in 70 to 80 per cent. of gynaecological cases.

What do we understand by the expression "puerperal sepsis"? In this connexion we may consider the definition of puerperal sepsis enunciated by the Ministry of Health and based on the suggestions of the Royal Society of Medicine—namely, that puerperal sepsis is (a) a fever; (b) a bacterial infection of the raw surfaces left after labour.

Is it a fever? It is true that some cases of puerperal sepsis may be attended by pyrexia, but this is the exception rather than the rule. In support of this it may be submitted that (1) all practitioners meet with many instances of yellow vaginal discharge unassociated with pyrexia; (2) in a large percentage of cases of secondary haemorrhage a high temperature never develops; (3) a patient may die from secondary haemorrhage or gangrene of the uterus without any pyrexia; (4) of 340 patients delivered in a maternity ward in 1926, 83 suffered from uterine sepsis—one-half of these septic cases exhibited pyrexia and the other half secondary haemorrhage.

Is it a bacterial infection of raw surfaces? If so, the inflammatory changes occurring as the result of retention of placental tissues or blood-clot and lack of free drainage are lost sight of, and the significance of these changes in the etiology of uterine disease fails to be recognized.

Immediately after labour or miscarriage a cascade of blood and serum exudes from the raw surfaces for a period