the years 1918-24. This may be more apparent than real. The figures from 1912 to 1917 may well undervalue the actual numbers, for the system of isolation was during the earlier part of that period an innovation, whilst school medical inspection was less rigorous during the war than

CHART II.—Average monthly incidence of tinea capitis during the period 1912-1930.

afterwards. Owing to the possibility of a naturally occurring periodicity in the case incidence (apart from the effect of isolation and supervision) it cannot be concluded that the decreasing incidence during the years 1925-30 indicates the slow elimination of the disease as a result of

CHART III.—Average incidence of tinea capitis per five days during the period 1912-1930. (One small square=five days.)

preventive measures. The explanation may rest in a lower virulence of the infecting fungi, or in a combination of both these factors. As far as the available figures show, the disease is exactly comparable to scarlatina or diphtheria as regards its occurrence over a period of years.

The monthly and five-day incidence charts (Charts II and III) show clearly that the isolation of cases as they occur has a beneficial influence on the case incidence or the number of contact infections during the school terms. This holds good in spite of the fact that most of the children are highly infectious for at least a few days before they are isolated, and when the lesions are developing to recognizable dimensions. The highest figures of incidence are reached at the commencement of the school terms—that is, following a period during which fresh cases probably escape detection for a longer time than is the case in term time, when the children are subjected to routine medical inspection. The magnitude of the rise in case incidence after a vacation seems to vary directly with the length of the vacation. It is recognized that in a given community of children infection may be acquired from outside sources during a vacation as a result of temporary changes of abode, or of temporary changes in the distribution of the general population. Infections acquired in such a manner cannot, however, account for more than a small increase in case incidence in any one school population, and the increase noted can only be

attributed to the natural spread of the disease in the school locality as a result of the absence of any controlling measures to deal with new cases during the vacation period. This fact raises the question whether infection is more readily acquired in home surroundings than at school. The curve of incidence can be to a great extent accounted for by the former hypothesis.

The average number of cases recognized during the first five days of September over a period of eighteen years was 8.5—the highest incidence of the whole year. Each of these cases had a greater or less opportunity to infect others before they themselves were recognized and isolated. If school contact offers ample opportunity for several children becoming infected from one child when there is a limited period during which infection can occur, then an abrupt rise in incidence might be expected during the second, third, or even fourth five-day period of September. This, however, does not occur, and there is a steady diminution in case incidence during the remainder of the term. It suggests that tinea capitis spreads slowly, and that a sudden rise in case incidence does not take place when infection is first introduced into a non-infected population. The endemicity is due to a slow spread of infection, and

to the occurrence of infected cases which are overlooked for a considerable period. The disease is not characterized by sudden sharp epidemics. While variations in the virulence of the infecting fungus may account for the fall in case incidence during the past six years, it cannot account for the variations in the monthly and five-day incidence, calculated from figures taken over a period of eighteen years.

That cases of tinea capitis already under treatment do not act as potent sources of infection is shown by the decrease in case incidence during school terms. This occurs despite the fact that the children are only segregated during school hours.

On account of the epidemiological characters of the disease, it is doubtful whether any system capable of being put into operation under existing conditions, where supervision throughout the entire year is impossible, would be able to eliminate the disease completely from a large school population.

PUERPERAL SEPSIS AND PROPHYLACTIC VAGINAL ANTISEPSIS

BY

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It has always seemed to me that the ordinary conduct of a simple forceps delivery was a contradiction of all the rules and axioms of surgical procedure. It is well recognized that the vagina contains organisms in every case. Recent work has shown that in the apparently normal woman staphylococci are present in about 60 per cent., streptococci in 42 per cent., and the deadly *S. haemolyticus* in 2.7 per cent., while other organisms, such as *Bacillus coli*, are also present. In the simple forceps case the operator sterilizes the skin of the patient's perineum, his own hands, his gloves and instruments, and then straightway passes them through the bacteria-laden mucus of the vagina into the uterus—over a surface and through a medium that no general surgeon would tolerate for one moment. It is true that the vagina may be scrubbed out with ether soap, but this is

impossible when the head occupies most of the pelvic cavity. I am safe in saying that most forceps operations are conducted without any treatment of the vagina whatever. Yet we expect these cases to remain afebrile!

No amount of ante-natal care will obviate the introduction of forceps into the tired, exhausted uterus, nor an adherent placenta requiring manual removal. We know that the bacterial flora of the vagina is rich, and yet we go on performing these operations, realizing that we are inevitably infecting the uterus.

Let me present another aspect: what occurs just before "crowning" of the foetal head takes place? The foetal head has been driven down on to the perineum, and is bulging through the vulva; in the intervals between the pains it retreats out of sight into the vagina. There is a to-and-fro motion of the foetal head and the foetal body—down into the vagina to be smeared with bacteria-laden mucus, back into the uterus to deposit its burden of infection there. What occurs just before "crowning" also occurs in a lesser degree throughout the second stage of labour, from the time when the cervix is sufficiently dilated to allow the head to protrude through, and the sac of membranes has ruptured. It follows, therefore, that we must entertain a new conception—namely, *that every woman has her uterus infected from her own vagina during the normal natural course of delivery.*

I am well aware that recent work on cultures from the interior of the uterus after labour shows a great proportion as sterile, but I suggest, first, that no cultural proof is required, and, secondly, that the cultural methods of some of the investigators are not calculated to show up this point.

There is no dispute about the existence of the flora of the vagina; hence, if the sterile foetus is dipped into the vagina, it becomes infected. If the infected foetus is withdrawn into the sterile uterus, the latter is infected. It is axiomatic, and no further proof is required. I believe there is considerable confirmatory evidence of my views in the following facts, known to all—namely, the increased morbidity in cases of: (1) early rupture of the membranes—since this allows the foetus to come into contact with the vaginal walls for a much longer period than when the foetus is enclosed in the intact sac until late on in labour, and is so prevented from touching the actual vaginal walls. (2) Portions of membrane being retained. I do not believe that if the pieces of membrane were really sterile, they would be in the least likely to promote sepsis. The amount of catgut used in haemostasis and suture of a laparotomy wound must in its original unspun form exceed the size of any portions of membrane usually left behind, yet clean laparotomy wounds do not become septic. The correct explanation is that the retained membrane is not sterile, but is infected, as I have shown. (3) Cases of so-called "dry" labour, where there is no liquor amnii left to flush out the sac of membranes after the expulsion of the foetus. (4) When the placental site is in the lower uterine segment—that is, nearer the route of extension upwards from the vagina through the open cervix. There is the least likelihood of a morbid temperature when the placenta separates by Schultz's method. Here the placenta and sac of membranes are turned completely inside out, and no part of the infected foetal surface touches the uterine wall. In Duncan's method of separation the membranes are often torn off from a part of the edge of the placenta; this allows infected material from the interior of the sac to escape into the uterus.

Another unconsidered detail is the movement of the umbilical cord—for example, in premature and ineffectual attempts to express the placenta. The cord in these cases can be seen to come down a couple of inches and recede again. This is not of "no importance." What

surgeon would allow an umbilical cord covered with discharges and vaginal mucus to be dipped into a patient's abdomen? We all know what would happen if it were.

Then again, during the first stage, when the cervix and lower uterine segment are dilating, the membranes cannot stretch equally well, and the vertical split in the membranes is frequently demonstrable when the after-birth is expelled. I mention this to show that a portion of the uterine wall is frequently exposed to the rubbing action of the foetus during "pains," and that organisms are consequently deposited, not simply in the sac of membranes, but on the uterine wall. If we consider these points, then those cases of virulent sepsis in normal labours with no vaginal examinations whatever will not seem so inexplicable. Why should we in these cases try to find a scapegoat in infected teeth, or tonsils, or bowels? People with much dirtier teeth, and just as infected tonsils and bowels, are subjected daily to major operations, as also are patients debilitated from long illnesses, profoundly "shocked," blanched from loss of blood; all these reproduce varying conditions after labour, and yet their clean operation wounds do not become septic. Which of us removes an adherent placenta without misgiving? Yet, I maintain, there is no physical reason why a gloved hand could not be introduced into the uterus without carrying with it any infection, except from the patient's own vagina.

The natural corollary of these beliefs is that we should make some attempt to sterilize the vagina as soon as possible in labour, because when the head occupies the pelvic cavity a great portion of the vagina is out of our reach. Even if it were possible to sterilize the vagina we are still confronted with the problem of the cervix. A large percentage of women—multiparae especially—have leucorrhoea. Most of the discharge in leucorrhoea is from an infected cervix, where the organisms are buried in the deep racemose glands. Sometimes, when introducing bougies for inducing labour, I have grasped the lip of a patulous os with a fenestrated sponge forceps instead of a vulsellum, and have remarked the plugs of yellow caseous matter squeezed out of the gland orifices, just as caseous secretion can be squeezed out of tonsil crypts in tonsillectomy. The difficulties should not deter us, however, if we believe the principle correct, and I hope to show that a very definite decrease in morbidity can be attained. I should like to add how illogical it seems for us to take up the attitude that we should not meddle with the vagina during labour, when we consider what we do in gynaecological operations, such as dilatation and curetting, or inductions of labour. Every attempt is made in these to sterilize the vagina by swabbing with such antiseptics as iodine, and I would cite the success of these operations as measured by freedom from serious after-infection. It is extraordinary that one and the same man will take precautions for a dilatation and curetting, or an induction, and will deprecate them for a forceps delivery. If they are essential for the one, they are equally so for the other.

Assuming that all women are infected in the way I have described, it is necessary to explain why the great majority have a more or less afebrile puerperium. I regard the main safeguards as: (1) the natural resistance of the patient herself to infection; (2) the nature of the infection—mainly saprophytes, but sometimes deadly organisms like anaerobic and haemolytic streptococci; (3) the fact that the infection is introduced, not into the actual uterus, but inside the sac of membranes which is shortly going to be expelled; and (4) the rush outwards of liquor amnii—if any remains—after the birth of the child, which washes out infection from above downwards.

Attempts to sterilize the vagina in midwifery have, of course, been made many times previously, but usually

the results have been worse than when no antiseptic was used at all. The work in connexion with the present experiment was undertaken at the Leicester and Leicester-shire Maternity Hospital in Leicester during the past twelve months. At first 10 c.cm. of plain glycerin were instilled into the vagina as soon as possible after the patient's admission. A short series of cases, however, showed "temperatures" to be more common than when nothing whatever was done. Next were tried pessaries of dimol; they also were discarded after being used in fifty cases, for rises of temperature were again more frequent than when nothing was used. The pessaries also took an appreciable time in melting—about one or two hours; after melting a little they became very slippery and frequently were extruded during a "pain."

Next was tried a 2 per cent. solution of mercurochrome in glycerin; 5 c.cm. of this were introduced into the vagina as soon as possible after the admission of the patient, and before any vaginal examination had been made. The technique is very simple and easy. A short length of rubber catheter is inserted as high as possible in the vagina, with due aseptic precautions; the mercurochrome and glycerin is introduced by attaching a 5 c.cm. syringe filled with the solution. A sterile diaper is applied to the vulva, and the patient is kept lying in bed for at least one hour (in those cases not yet advanced in labour). The mercurochrome is repeated each following morning unless delivery is over. No irritant effects whatever were observed, and there is no danger to the infant's eyes; in fact, the Eye Department of the Bristol Infirmary has been using a 2 per cent. mercurochrome for the past four years to the exclusion of other antiseptics (except in gonorrhoeal ophthalmia).

The investigations of Dr. Leonard Colebrook in this country, Schottmüller in Germany, Schwarz and Dieckman, and Harris and Brown in America, have shown that the anaerobic group of streptococci is responsible for a large amount of uterine sepsis, and that this group is present in the vagina in a considerable number of cases before labour. Their work to a large extent discounts the conclusions at present generally accepted as to the harmless character of vaginal organisms present before labour, and their non-relation to those occurring in the uterus in puerperal sepsis. Cultures grown under anaerobic conditions present an entirely different picture. They show in a very large proportion of cases a definite relationship between the organisms responsible for severe uterine sepsis and those present in the vagina before labour. They provide the bacteriological proof of the necessity of dealing with the vaginal flora. Schottmüller in 1923 recorded 72 fatal cases of puerperal infection following delivery due to anaerobic streptococci from a total of 231 fatal cases.

Harris and Brown use the following mixture in 7 c.cm. doses, repeated every twelve hours if labour is prolonged: mercurochrome crystals, 15 grams; half strength tinct. iodi, 5 c.cm.; glycerin, 500 c.cm. They began to try this antiseptic in September, 1926, and the reduction in mortality from puerperal sepsis is shown in the following table.

	Deliveries	Deaths	Per cent.	
July, 1924-Sept., 1926	1,913	5	0.261	No mercurochrome.
Sept., 1926-Jan., 1930	4,494	4	0.089	Mercurochrome used.

This is the reduction in morbidity:

	Admissions	Infections	Per cent.	
July, 1924-Sept., 1926	2,194	45	2.05	No mercurochrome.
Sept., 1926-Jan., 1930	5,385	67	1.24	Mercurochrome used.

In my own series I used as controls all the cases delivered in hospital during the months of July, August, September, 1930. There were 162 of these, and they were

in no way selected. Mercurochrome in glycerin was given in the next 200 cases, and the results are appended.

	Temp. never above Normal	Above Normal, but not 99°	Temp. 99°, but not above	Between 99° and 100°	100°, but not above	Above 100°, but not Notifiable	Notifiable	Notifiable, Non-uterine Excluded
Controls (162 cases)	21 12.9%	48 29.6%	20 12.3%	39 24.0%	10 6.1%	13 8.0%	11 6.7%	8 4.9%
Mercurochrome (200 cases)	55 27.5%	50 25%	36 18%	23 11.5%	10 5%	13 6.5%	13 6.5%	9 4.5%

From the preceding figures the outstanding facts arise that of the patients who never had a temperature in the puerperium higher than 99° there were 54.4 per cent. among the controls, and 70.5 per cent among those who had mercurochrome, or an increase of 16 per cent. for the better. When we consider the total number of days that each patient had a temperature, the figures are even more striking. In each class the patients receiving mercurochrome had a temperature above normal for about half the length of time of the control patients.

Table showing Total Number of Days above Normal Temperature for Each Class of Case, and the Average Number of Days for Each Patient.

	Above Normal, but not 99°	99°	Above 99°, but not 100°	100°	Above 100°, but not Notifiable	Notifiable	Uterine Notifiables	Total Days, excluding Non-uterine Notifiables
Controls (162 cases)	190	111	236	67	89	106	88	781
Average	3.9	5.5	6.0	6.7	6.84	9.6	11	4.8
Mercurochrome (200 cases)	128	123	95	69	77	156	89	581
Average	2.56	3.4	4.1	6.9	5.9	12	9.88	2.93

There is an amazing reduction in the number of days' total morbidity for a whole year's working of the hospital as the result of this one addition to the technique. Nothing else was changed in the treatment of patients during the time of the experiment, and the nursing personnel remained practically the same as during the time of the control cases. The procedure is simple and safe, requires only a short time to perform, and can easily be carried out single-handed by any midwife with no addition to her equipment other than a 5 c.cm. syringe. I commend the idea to medical officers of health and maternity hospitals. It is far from perfect, but I suggest that if work on these lines were conducted on a national scale, and the results were pooled, something of very great value would undoubtedly be evolved.

Following a recent article in the *British Medical Journal* on the relative bactericidal values of different antiseptics, acriflavine in 0.1 per cent. solution in glycerin has been used in the next series of cases. The results have been notably different, as shown in the table below:

1 in 1,000 Acriflavine	Temperature never above Normal	Above Normal, but not 99°	Temperature 99°, but not above	Above 99°, but not 100°	100°, but not above	Above 100°, but not Notifiable	Notifiables	Notifiables, Non-uterine Excluded
300 cases	48	74	58	71	10	30	9	6
Percentage	16.0	24.6	19.3	23.6	3.3	10.0	3.0	2.0

Here we have 180 cases in which the temperature in the puerperium never exceeded 99°, giving a percentage of 60, as against 70.5 with mercurochrome. There is, however, a marked decrease in the notifiable cases of uterine origin, only 6 out of 300, or 2 per cent., as against 4.5 per cent. for mercurochrome. All these cases were slight, the average number of days each patient had a

temperature above normal being only 9.1 from start to finish with acriflavine, and 9.88 with mercurochrome. The complete figures are shown in the following table.

Table showing Total Number of Days above Normal Temperature for Each Class of Case, and the Average Number of Days for Each Patient.

1 in 1,000 Acriflavine	Temperature above normal, but not 99°	99°, but not above	Above 99°, but not 100°	100°, but not above	Above 100°, but not Notifiable	Notifiables	Notifiables, Non-uterine Conditions Excluded	Total Days Non-uterines Excluded
300 cases	230	236	418	69	235	85	55	1,243
Average	3.1	4.0	5.88	6.9	7.8	9.4	9.1	4.1

Taking 550 cases as the normal year's working figure, the following facts emerge. The total number of days in the year when patients had a temperature above normal, excluding notifiables of non-uterine origin, was: no antiseptics, 2,651 days; mercurochrome, 1,597 days; acriflavine, 2,278 days. Mercurochrome thus shows a decrease of 39.7 per cent. in "temperature days," and acriflavine a decrease of 14 per cent.

UNDULANT FEVER IN NORTHERN IRELAND

BY

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It has been stated by Bigger¹ that undulant fever due to *Brucella abortus* is rare in Ireland, only three cases having been reported for the whole country, and he infers from this that the strain of *Br. abortus* infecting Irish cattle is probably of low virulence for man. The following list of cases is published to show that, in Northern Ireland at any rate, the disease is not so rare as might be inferred from this statement.

Within the past two years there have been eight cases of the disease and one of experimental infection. The diagnosis of these cases has been made from the clinical features and from the result of agglutination reactions. In none of them do we possess the absolute proof of the disease which isolation of the *Brucella* would have afforded, but, so far as I am aware, in only one case in the British Isles has this been accomplished.²

The main features of the cases and the result of the agglutination reactions are here shortly summarized.

Case 1.—November 26th, 1929. Male, boot merchant. Headache, nausea, constipation, and pyrexia for three weeks; flatulence, offensive motions; no enlargement of liver or spleen. Widal reaction negative for T.A.B. *Br. abortus* 1 in 500 (+).

Case 2.—February 2nd, 1930. Male, aged 42, farm labourer. History of headache, abdominal pain, diarrhoea, and fever for four weeks; spleen enlarged and tender. Widal reaction negative for T.A.B. and *C. Br. abortus* 1 in 1,250 (+). (Case published in *British Medical Journal*.³)

Case 3.—April 9th, 1930. Male, aged 47, bank official. Ill for six weeks with pyrexia and pulmonary symptoms; abdomen distended; liver enlarged, spleen not palpable. Widal reaction negative for T.A.B. *Br. abortus* 1 in 5,000 (+).

Case 4.—August 5th, 1930. Male, aged 45, shipyard official. History of headache, loss of energy, constipation, and intermittent pyrexia for one month; occasional night sweats; spleen palpable; well-marked relative lymphocytosis (small lymphocytes 58 per cent.). Widal reaction negative for T.A.B. *Br. abortus* 1 in 5,000 (+).

Case 5.—November 4th, 1930. Male, druggist. History of headache, nausea, vomiting, constipation, and typical undulant pyrexia for six weeks; pain and tenderness in right iliac fossa. Widal reaction negative for T.A.B. *Br. abortus* 1 in 250 (+).

Case 6.—April 4th, 1931. Male, aged 27, motor mechanic. History of anorexia, abdominal pain, constipation, and pyrexia of three weeks' duration. Abdominal tenderness, right basal congestion, no enlargement of liver or spleen. Widal reaction negative for T.A.B. *Br. abortus* 1 in 5,000 (+).

Temperature fell to normal, and has remained so, after two injections of normal horse serum intramuscularly.

Case 7.—June 19th, 1931. Male, aged 32, farmer. History of headache, profuse sweating, pain in the neck muscles, constipation, and pyrexia for a fortnight. Rigidity of neck, tendon-jerks increased. Cerebro-spinal fluid under pressure, but no increase in cells or globulin. Widal reaction negative for T.A.B. *Br. abortus* 1 in 1,250 (+).

Case 8.—September 16th, 1931. Female, aged 50, clergyman's wife. History of encephalitis seven years ago, with apparent perfect recovery. Headache, malaise, pains in legs, and pyrexia for about one month; spleen not palpable. Blood culture under 10 per cent. CO₂ was sterile. Widal reaction negative for T.A.B. and *C. Br. abortus* 1 in 2,500 (+).

Dr. W. Carson of Belfast, while carrying out some experimental work with *Br. abortus*, infected himself with a culture of the porcine type, and immediately developed undulant fever of a severe nature. In this case the symptoms and fever disappeared after the administration of solganol, 0.1 gram intravenously, on three occasions.

COMMENTARY

In reviewing the salient features of the above cases, one is struck by the frequency with which headache, constipation, and more or less generalized gastro-intestinal disturbance are found in this disease. One feels sure that a large number of these cases occur unrecognized, and pass under the label of "gastritis," "gastric influenza," or the like. The great preponderance of males over females in this series is also noteworthy.

While this list of cases was being compiled, an opportunity was afforded me of re-examining the agglutination reaction of the serum of some of them. The results, although few, are given here, as they may be of some value in interpreting positive reactions in low dilution found in the routine examination of serums.

Case 1.—1 in 25 (—), 1 in 50 (—), 1 in 125 (—), 1 in 250 (—). Twenty-one months after illness.

Case 2.—1 in 25 (C), 1 in 50 (++), 1 in 125 (+), 1 in 250 (—). Twenty months after illness.

Case 6.—1 in 25 (++), 1 in 50 (C), 1 in 125 (C), 1 in 250 (tr.). Six months after illness.

Case 7.—1 in 25 (—), 1 in 50 (++), 1 in 125 (C), 1 in 250 (++), 1 in 500 (tr.). Four months after illness.

In one of these cases (Case 1) no agglutination was given. This was nearly two years after the illness, and the serum during the illness did not agglutinate the organism in a higher dilution than 1 in 500. The other three, at varying times less than two years after illness, gave very definite agglutination in a dilution of 1 in 125 or higher. Case 7 would have been missed if the reaction had not been carried out in higher dilutions than 1 in 25, owing to the presence here of a pro-agglutinoid zone.

In performing these reactions two sets of dilutions were set up. In the first of these the serum as received was used, while in the second the serum was first heated to 56° C. for thirty minutes to eliminate non-specific agglutination. Heating the serum had the effect of lowering the titre slightly in each case, and the figures given above are all for the heated serum. The suspension of *Br. abortus* used was the Oxford standard.

Note.—Since the foregoing was written two further serums have been examined: *Case 3*, 1 in 25 (C), 1 in 50 (+++), 1 in 125 (—), 1 in 250 (—), nineteen months after illness; *Case 8*, 1 in 25 (C), 1 in 50 (C), 1 in 125 (C), 1 in 250 (++), two months after illness. These figures refer to the agglutination performed with the heated serum. The unheated serum in each case gave agglutination in a higher dilution.

My thanks are due to those physicians of Northern Ireland who have permitted me to include their cases in this list, and also to Dr. T. B. H. Haslett, who kindly performed the agglutination reactions.

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