BRITISH MEDICAL JOURNAL

LONDON SATURDAY OCTOBER 18 1947

THE AETIOLOGY AND PROPHYLAXIS OF PUERPERAL SEPSIS*

BY

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As this Congress is being held to celebrate the two-hundredth anniversary of the foundation of the Rotunda Hospital it is not, I hope, inappropriate to attempt to correlate the battles in the long war against puerperal sepsis with the history of the hospital. I propose to divide the two hundred years of the hospital's existence into four periods each of half a century.

The first half-century, 1745-95, presents a melancholy succession of epidemics of childbed fever alternating with brief interludes during which the prevalence of the disease was endemic rather than epidemic. In founding a lying-in hospital, Mosse was trying a more dangerous experiment than he realized. Childbed fever existed in the homes of rich and poor alike in the eighteenth century, but it was not then a very prevalent condition. When the attendants were aged wise women, with an amateur rather than a professional status and a strictly limited clientele, the danger of transfer of infection from one woman to another was slight and epidemics were unlikely to occur. Mosse's plan of herding in one building a number of parturient women and entrusting the supervision of their deliveries to professional midwives had inherent in it the seeds of disaster. So long as no case of childbed fever occurred all was well, but a single case had as its almost inevitable sequel an epidemic.

The rise of the man midwife was in itself responsible for an increase in the number of cases of puerperal sepsis. The man midwife undertook the care of a far larger number of labours than did the woman, and to the risk of transferring the seeds of sepsis from uterus to uterus he added the further risks of transferring by his hands to the birth canal of his patient these seeds derived from other fertile nurseries—wounds with their "laudable pus," droplets from the throats of scarlet fever patients, corpses in the anatomy room of the medical school. The wonder is not that puerperal sepsis was constantly present in the Rotunda, as it then was in every other lying-in hospital, but that these institutions did not acquire an even more evil reputation and that they and their staffs escaped from the fury of the mob, incensed at the slaughter of so many innocents.

In the next half-century (1795-1845) two remarkable Masters, Labatt (1814-21) and Collins (1828-33), stand out above the others. They sought, not entirely unsuccessfully, to combat the disease by such limited degree of isolation as was available, by scouring of furniture, by washing and stoving of bedding, by fumigation of wards, by applications

of chloride of lime, and by whitewashing. They anticipated Holmes and Semmelweis in suspecting that puerperal fever was infectious, that infection was conveyed from the woman suffering from puerperal sepsis to the healthy parturient woman, and that the transmitting agent was far too often the hands of the doctor.

The third half-century, 1845-95, started badly. Reactionaries such as Denham (1861-8) and Johnston (1868-75) opposed the new ideas of Labatt and Collins and of Holmes and Semmelweis, but slowly those ideas spread, and when in 1879 Pasteur dramatically demonstrated the cause of childbed fever as "chapelets en grain," the time was ripe and a new era was born. Pasteur inspired Lister, and the third half-century ended nobly in the Rotunda with Atthill (1875-82), Macan (1882-9), and Smyly (1889-96), under whom, within a period of less than twenty years, scepticism and opposition gave place to a whole-hearted acceptance of Listerism, and the antiseptic era reached its peak.

The fourth half-century (1895-1945) brings us almost up to to-day. In no previous half-century had similar discoveries been made in aetiology, therapeutics, and prophylaxis. So numerous were the advances that it is difficult even to catalogue them. At the beginning of this period bacteriologists had largely accepted the streptococcus as the cause of puerperal sepsis, but it was hard to keep one's faith when streptococci were so frequently present without sepsis. With Schottmüller's application of culture on blood agar to the differentiation of *Str. haemolyticus* from *Str. non-haemolyticus* a real advance was made. Now all was well: puerperal fever was due to *Str. haemolyticus*. But doubts again appeared. A haemolytic streptococcus could be cultivated from the lochia of a woman who was certainly not suffering from puerperal sepsis, who was not even morbid. Further subdivision of the haemolytic streptococci was urgently required. Fermentation of carbohydrates was used by many workers, including myself, but without either satisfaction or enlightenment. Then serological methods, for which credit must be accorded to Lancefield (1928, 1933) and Griffith (1926-8), were introduced, and almost at once the dark places were illuminated. We now group the haemolytic streptococci by Lancefield's method and recognize an organism of group A as the *Streptococcus pyogenes*, the main cause of puerperal sepsis. By Griffith's technique we subdivide *Str. pyogenes* into more than a score of types, and so, not rarely, it is possible to trace to its source the streptococcus responsible for a case of sepsis.

In therapeutics, vaccines and antisera had their day; but only a few enthusiasts had faith in them, and both are now

*A paper read before the International Congress of Obstetricians and Gynaecologists, held in Dublin, July 7-11.

discarded. In 1935 came Domagk's "prontosil," to be followed hot-foot by the polyonymous sulphonamides. In 1929 Fleming discovered and described most of the properties of penicillin, but it was only after ten years that the greatness of his discovery was realized. It required the vision and persuasiveness of Florey and a world war to make available the first, and still the greatest, of the antibiotics.

In the field of prevention progress was steady if not spectacular. The application of Lister's antiseptic methods to midwifery ended for all time the major epidemics of puerperal fever characteristic of the practice of the Rotunda, as of other lying-in hospitals, in the eighteenth and the early nineteenth century. The combination of antiseptics and asepsis prevents the transfer of infection from patient to patient by the hands or instruments of doctor or nurse. But sporadic cases still occur and, much more rarely, small epidemics. Once the infectivity of puerperal sepsis had been realized the transfer of the causative organism from the uterus of one woman to that of another could be stopped relatively easily. Comparatively little effort yielded a large return, but puerperal sepsis did not cease to exist.

The uterus is a hollow viscus in relatively free communication with the exterior at a situation on the surface of the body where bacteria of various types abound. After childbirth it contains in its interior blood clot, an excellent nutrient for bacteria, and this is in contact, at the placental site, with underlying tissue temporarily deprived of the protective covering of epithelium. The uterus itself and the surrounding parts have recently suffered various insults incidental to the process of childbirth. The fire is set: only a spark is required to start a conflagration. Others who follow me will deal with the nature of the conflagration and how it may be extinguished. My concern is with the spark which may kindle the fire, its nature and source, and how it may be kept at a safe distance.

The most important cause of puerperal sepsis to-day is *Str. pyogenes*. There is little doubt that the same organism was responsible for the epidemics of the eighteenth and nineteenth centuries, but the sources have changed. Then the main source was the uterus of the septic patient. Other sources were septic wounds, sore throats, erysipelas, and impetigo. Transmission was accomplished, all too easily, by the unwashed hands of the doctor. Now conditions are entirely different. There are far fewer cases of puerperal sepsis to act as sources of infection, the obstetrician's hands are much less often contaminated with *Str. pyogenes* from other infections, and the barriers of washing, antiseptic lotions, and rubber gloves are interposed to prevent the transfer of the organism to the genital passages of the woman at the time of childbirth. When a group of cases occurs in a ward at intervals of a few days there is a presumption that the infection has been transferred from one to the other—not usually, it should be said, owing to a lack of cleanliness of the obstetrician's hands but to some less obvious break in the essential barrier between patient and patient. The ideal method of avoiding case-to-case transfer of infection is to provide a separate room for each woman admitted. The next best is prompt isolation of any woman on the first suspicion of sepsis: morbidity demands isolation. But there is a period, possibly only of hours, before sepsis suggests its presence by morbidity, and during this period *Str. pyogenes* is multiplying in and being discharged from the affected uterus. We cannot make a diagnosis of sepsis early enough to avoid all risk of spread, so we employ additional defences. These are isolation, as complete as local conditions permit, and the making over to each woman admitted of a set of all essentials of toilet and

hygiene. The dangers of the communal bed-pan are lessened by disinfection: they are still further reduced if a bed-pan is reserved for each woman and if it is disinfected.

Limitation of vaginal examination, a strict ritual of hand and glove preparation, the wearing of gowns, and disinfection by steam of all instruments eliminate relatively easily the major risks of introducing streptococci into the birth canal.

At one time it seemed possible that puerperal sepsis would become extinct, but the curve showing its prevalence, which declined sharply as a result of the application of antiseptic and aseptic precautions to midwifery, failed to reach the zero line. There is a hard core of puerperal sepsis, and only by the greatest exertions can this hard core be reduced. An explanation of the sporadic case of puerperal sepsis which for a time appeared adequate was that the source of infection was endogenous—that is, that the *Str. pyogenes* was present in the vagina before the onset of labour and that from that situation it spread to the then highly vulnerable uterus after delivery. Some twenty-five years ago this seemed reasonably probable. Support for the theory was given by the finding of haemolytic streptococci in the flora of the vagina, both before and after labour, in from 3 to 6% of parturitions. But when our fumbling attempts to group haemolytic streptococci on the basis of fermentative capacities were replaced by surer serological methods and *Str. pyogenes* was accurately delimited, it was found that the haemolytic streptococci of the vagina were never, or practically never, of group A. The journey of that organism to the uterus is a longer one than from the adjacent vagina.

The proximity of the anus to the vulva suggested the possibility of infection of the uterus being derived from the faeces. This actually occurs, but not in the case of *Str. pyogenes*. This organism never gives rise to the carrier state in the intestine, and only very infrequently does it occur there as a pathogen.

Throat Carriers

We now know that the main reservoir of *Str. pyogenes* is the human throat. There it flourishes as a pathogen, producing scarlet fever or simple sore throat, and, still more commonly, there it lurks as a saprophyte on a mucous membrane which appears slightly, if at all, abnormal. The rate of throat carriers of *Str. pyogenes* varies greatly with time, population, place, and season, but a conservative estimate is that some 5% of a group of people harbour *Str. pyogenes* in their throats. Having traced the chief organism of puerperal and, indeed, of all types of sepsis to the not obviously abnormal throat, little imagination is required to picture its transfer to the uterus. Every time we cough or sneeze, shout or speak loudly, we spray out from our mouths and noses thousands of small droplets of saliva and mucus. If the sprayer is a throat carrier of streptococci these organisms leave his body in enormous numbers in the droplets. Good manners demand that, when coughing or sneezing, the hand or handkerchief be held before the face, and so the hand is contaminated directly from the droplets or indirectly from the handkerchief or the pocket into which it is put. It may be further contaminated by contact with objects on which droplets have fallen.

The hands of the doctor are, as we have seen, usually adequately prevented from becoming the transmitting agent of the streptococcus; but there are other hands, and especially there are the hands of the woman herself. If she is a throat carrier of *Str. pyogenes* her hands may very easily transfer the organism to her vulva; but even if she is not a carrier her hands may transfer the organism, picked up from the air, from bedding or clothing, or from some

other source. The other hands to be considered are those of the nurse. When carrying out treatment involving contact with the genital tract they should be, and presumably are, just as carefully prepared as are those of the doctor; but the nurse has many duties—dressing and washing the patient, making her bed, handling her bed-pan, and adjusting the vulval pad—and some of these involve either direct contact with the vulval region or contact with something which later makes such direct contact.

The hand has been dealt with first because it is the main vector of streptococci on their journey from the mouth and nose to the uterus, but transfer may occur in other ways. Few doctors or nurses sneeze upon the vulvae of their patients, and so direct transfer by droplets from throat to genital tract is rare, but direct transfer from throat to throat does occur. A carrier, whether patient, doctor, nurse, or visitor, may introduce *Str. pyogenes* into a ward, where it may establish itself in the throats of several inmates, each of whom constitutes a fresh focus of infection. Sooner or later droplets fall to the ground, the bed, or other surfaces, where they dry and enrich the dust with their streptococci. *Str. pyogenes* is usually considered to be a rather delicate organism, but when dried in dust it can survive for a period to be measured in weeks rather than days.

Droplets and Dust

The dangers of droplets and dust can be and should be reduced by the wearing of adequate masks and by the application of dust-controlling oils to floors and bedding. Only exceptionally should it be necessary to resort to disinfection of the air in a ward by ultra-violet radiation or, more simply and economically, by disinfecting mists. Valuable as these measures are, they come into operation at too late a stage. They seek to limit the diffusion of *Str. pyogenes*, but our aim should be to exclude that organism completely from our wards.

Each person regularly visiting a ward in a maternity hospital should have a throat swab examined for *Str. pyogenes* every week. If gentian violet blood agar is used it is possible, within 24 hours, to isolate streptococci from a throat and to identify those which are haemolytic even when very few are present. All found harbouring these organisms should be put off duty for 24 hours while the haemolytic streptococci are being grouped. If they are of group A—that is, if they are *Str. pyogenes*—the off-duty period should continue until in three consecutive daily examinations the throat is found to be free from that organism. If the streptococcus does not belong to group A the carrier presents but a slight danger and may be returned to duty.

This control should be applied to all members of the staff, whether doctor, nurse, or maid, whose duty takes them into a ward used for women before, during, or after labour. I fully realize how unpopular this would be and how liable it might be to disorganize the usual routine of a hospital; but a carrier of *Str. pyogenes* is such a menace that, in my opinion, eradication of puerperal sepsis will not be possible without it. Since there is an almost universal tendency to regard oneself as indispensable, efforts may be made to obtain a falsely negative result by various subterfuges, which must be rigorously guarded against. The duration of the carrier period may be greatly reduced by local application of a sulphonamide or penicillin. I do not advocate these substances for the prevention of the development of the carrier state.

Casual visitors, who should be reduced to a minimum, present a smaller problem, but still a problem. They should wear masks and gowns and should not be allowed to approach the patient too closely. I scarcely dare to advo-

cate that, during the visit of the husband or mother of the patient, a glass screen should be interposed between her visitor and herself and that conversation should be conducted by microphone, but it would be a rash man who would say that such precautions will not ultimately be instituted.

What of the throat of the patient, which we have so far neglected? A *Str. pyogenes* of the same type has so often been found in the throat and septic uterus of a patient as to suggest that in such cases the uterus became infected from the throat. The alternatives that the throat was infected from the uterus or that both were infected from another person cannot, however, be overlooked.

Str. pyogenes in the throat of a woman at childbirth constitutes a danger to her own uterus and also to the uteri of other patients. That danger may be guarded against, in part, by bacteriological examination of throat swabs, but an obstetrical hospital is often an emergency hospital in that the patient is not always, or not even usually, admitted sufficiently long before labour to permit the bacteriologist to report whether she is a carrier of *Str. pyogenes* or not before delivery. The history of the Rotunda could furnish details of delivery occurring at every stage of the patient's journey from her home to the labour ward. The ideal arrangement is, as I have already stated, a separate room or cubicle for every patient. The second best is isolation, as complete as possible, from other patients of all new admissions until they have been found not to be carriers of *Str. pyogenes*. A patient found to be a throat carrier of *Str. pyogenes* should be isolated completely from all other patients and provided with her own nurses for as long as she is a carrier. Local treatment of her throat should be promptly instituted, and if she is still a carrier at the onset of labour her uterus should be safeguarded by systemic administration of penicillin.

The only other precaution to be considered is the general prophylactic use of chemotherapy. Sulphonamides have been used to some extent for this purpose, but the results have not been encouraging. Penicillin has not, so far as I am aware, been extensively reported upon as a prophylactic. I certainly would not advocate its routine use, but, if a case of puerperal sepsis should occur in a hospital, I believe that every woman admitted should receive systemic penicillin from the onset of labour until five days after delivery. I do not believe that the introduction of anti-septic substances into the vagina before or after delivery should be required for the prevention of puerperal sepsis due to *Str. pyogenes*. The advisability of such prophylactic methods as a guard against other organisms will be considered later.

Up to the present I have dealt with puerperal sepsis as if it were a specific disease like scarlet fever, due always to a haemolytic streptococcus of group A. I have done so because the epidemics of the past and the majority of cases of what I may call classical puerperal sepsis of to-day are due to this organism. But other bacteria must also be considered.

Let us deal first with those organisms so commonly found in the vagina ante partum as to constitute its normal bacterial flora. The most numerous of these are lactobacilli and anaerobic streptococci. It is probable that these came originally from the faeces, from which they may again be reintroduced from time to time, for they readily adapt themselves to the conditions present in the vagina. The lactobacilli may at once be exonerated. So far from being dangerous, by their powers of glycolysis leading to acid production they are highly beneficial commensals. The anaerobic streptococci are much less innocent and, indeed, present a special problem which will be considered later.

The richest bacterial flora of the body is found in the faeces, and the proximity of the anus to the vulva makes it obvious that the complete exclusion of faecal bacteria from the birth canal would be quite impossible. Coliform bacilli, so characteristic of the faeces, are but rarely found in the vagina ante partum. Their numbers increase somewhat post partum, but it seems clear that they are merely saprophytic in the lochia and are very rarely, if ever, the primary cause of puerperal sepsis, although by their presence they may have some enhancing effect on the virulence of other organisms.

Disease, and indeed disease of severe and often fatal type, may be produced by *Cl. welchii* and *Cl. tetani*. These organisms may be derived from the faeces, but, since their highly resistant spores have been found in cotton-wool and in sanitary pads prepared from it, an obvious alternative source of infection is available. Danger from this source must be guarded against by thoroughly efficient heat sterilization of cotton-wool used in connexion with midwifery. These clostridia have slight invasive power and present little danger provided there is no retention of lochia, no necrosis of tissue, and no extensive multiplication of other bacteria which might render conditions suitable for their development.

The faeces may be the source of many other bacteria sometimes to be found in the vagina ante partum and, in still larger numbers, post partum, but even in the post-partum uterus they have little if any pathogenic power—they are mere saprophytes.

It is curious that *Staphylococcus pyogenes*, such an important cause of sepsis elsewhere, is so rarely incriminated in puerperal sepsis. Cases of undoubted infection produced by it are recorded, but much less frequently than its ubiquity would suggest. Midwifery practice cannot ignore this organism, on account of its frequent association with sepsis of perineal lacerations and its marked tendency to cause thrombosis. Its reservoir is the nose, in which, as also on the skin and particularly the skin of the hands, the carrier condition is established. The wearing of adequate masks and of rubber gloves, and preparation of the hands, are probably adequate safeguards against it. *Staph. albus*, so slightly pathogenic as to be regarded as a saprophyte, may be found in the lochia but is not important.

Haemolytic streptococci, other than *Str. pyogenes*, which gave rise to so much confusion before the introduction of Lancefield's serological grouping, have quite often been isolated from the lochia of women who show no evidence of puerperal sepsis. Their pathogenic powers are much more feeble than those of group A streptococci, and in the uterus they usually behave as saprophytes. Occasionally, however, one or other gives rise to serious and even fatal sepsis. Streptococci of these groups show a marked tendency to spread from the uterus to the blood stream, causing fatal endocarditis. Streptococci of groups C and G, like those of group A, are unable to survive long in the vagina, and so in order to cause puerperal sepsis they must be introduced shortly before, during, or soon after delivery. The usual source of both is the human throat, for the organisms are harboured there in the carrier state or as causes of mild tonsillitis. Direct infection from the horse, in which group C organisms cause strangles, is improbable. Streptococci of groups B and D are occasionally found in the vagina, where they can apparently persist as saprophytes, and so have an easy route to the uterus after delivery. Direct infection of the uterus post partum by milk of cows suffering from bovine mastitis, which is caused by group B streptococci, is not a likely accident. Group D streptococci are commonly present in the faeces, from which methods of transfer to the uterus are obvious.

The measures suggested to reduce the dangers of infection by group A streptococci apply to exogenous infection by other haemolytic streptococci foreign to the vagina (C and G) and should prove adequate for the purpose.

Endogenous Infection

We are now left with the difficult problem of endogenous infection of the post-partum uterus by organisms of the vagina and particularly by anaerobic streptococci and by aerobic streptococci of groups B and D. Again I wish to emphasize the frequency with which these organisms are found in the uteri of women whose puerperium causes their obstetricians not a moment's anxiety. They are not commonly the cause of puerperal sepsis, and in the uterus they usually behave as saprophytes. Only in a small proportion of cases in which they are present in the uterus do they give rise to morbidity, and in only a small proportion of such cases do they actually cause sepsis. Of the two, the anaerobic streptococci are of much greater importance. They are, in fact, second only to *Str. pyogenes* as causative organisms of puerperal sepsis. They are rarely present in the lochia in pure culture in cases of sepsis, but are usually accompanied by other organisms, such as *Staph. albus* or diphtheroid bacilli, which, alone, are devoid of pathogenic power. It may be that the combination of one or more saprophytes with anaerobic streptococci endows the latter with abnormal pathogenicity. While the streptococci are endogenous the other bacteria are exogenous, and thus all the precautions which are effective in excluding bacteria from the genital tract should be of value in reducing sepsis due to endogenous anaerobic streptococci.

Prophylaxis by the application of antiseptic substances to the vagina has been fairly extensively, but not very critically, tried. I am somewhat prejudiced against this method, but some American experiences seem to show that it is of value, and it certainly appears to be safe. If any such application is to be made it should, in my opinion, be made before the onset of labour rather than after. In place of mercurochrome, which has been used for the purpose, I would advise a combination of penicillin, a sulphonamide, and proflavine. The first two are suggested because some of the organisms against which protection is sought are sensitive to one and some to the other. Proflavine is added to deal with bacteria resistant to both. The combination is unlikely to damage tissue and so open fresh portals to infection, but there is a danger that by inhibiting the growth of lactobacilli it may deprive the birth canal of a valuable means of protection.

I do not wish it to be thought that I am a strong advocate of this procedure, and I suggest its use only because we have no other method of protection against endogenous infection. If it is to be used at all, it should be so used as to demonstrate beyond doubt whether it is of value or not. This requires a large series of treated cases with an equal number of strictly parallel controls so as to yield statistically significant figures.

I have attempted to lay before you the essentials of the bacteriology of puerperal sepsis, and I have ventured to make recommendations which might, I believe, reduce still further the present satisfactorily low level of the disease. I propose to conclude with a warning. The continuation and extension of the precautions which have been so successful will in time make puerperal sepsis such a rare condition that obstetricians even of considerable experience will never have seen a case and will tend to regard it as a disease as extinct as cholera. It will then be very hard to maintain, day by day, month by month, and year by year, all the elaborate, time-consuming, and irritating precautions which we now know to be essential and which have given

us the victory, but which to the young obstetrician will appear but meaningless ritual. Then there will be a tendency, which it will be hard to resist, to relax some of the precautions, but so long as *Str. pyogenes* continues to exist in the throats of the people such relaxation will inevitably be followed by the reappearance of puerperal sepsis, the history of which is reflected in the history of the Rotunda Hospital.

PENICILLIN AND THE TREATMENT OF INFECTED HANDS IN OUT-PATIENTS

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Penicillin is widely used in the treatment of the infected hand. It has been employed as a cream or powder for local application, and by direct injection into the theca in cases of suppurative tenosynovitis. In this review of cases treated at the Westminster Hospital it is our aim to assess the value of a method of administering systemic penicillin which can be used on out-patients. Only two of the patients treated with penicillin were admitted to hospital. This was from necessity rather than choice in the first instance, but later, when supplies of penicillin became available, out-patient treatment was deliberately adopted to save blocking hard-pressed bed space.

Eighty-five per cent. of all cases reviewed, and all cases receiving penicillin, were seen and treated by us throughout. In the remainder incision was done by casualty officers, but after-treatment was prescribed by us. This ensured uniformity for the purpose of assessment. The cases are considered under two groups: Group I consisted of 168 cases treated without systemic penicillin, and Group II of 17 cases treated with systemic penicillin. Within each group the following types of lesion are differentiated: (1) paronychia and pulp-space infections; (2) tenosynovitis; (3) palmar-space infections.

It is not feasible to give penicillin at four-hourly intervals to out-patients, and it was therefore hoped that a therapeutically adequate serum level might be maintained by two large doses, given morning and evening by intra-muscular injections of aqueous solutions or beeswax-oil suspensions. The aqueous solutions produce high serum penicillin levels of brief duration, whereas the wax-oil suspensions produce a lower but more prolonged serum level and were found clinically to be more efficacious. No untoward effects, such as urticarial reactions or marked pain, were noted.

Diagnosis

(1) Pulp-space infections and paronychia were usually only too obvious, as the average duration of symptoms before the patient arrived at the surgery was six days.

(2) Tenosynovitis was diagnosed by the following criteria: swelling of the whole finger, which was held in a semi-flexed position; great tenderness on pressure over the tendon sheath; and severe pain on attempted passive extension.

(3) Palmar-space infection (middle and thenar spaces) was diagnosed by diffuse tenderness, convexity of the palm overlying the affected space, and gross dorsal oedema.

Treatment

Palmar-space infections were drained by incising into the web, passing a Spencer Wells forceps into the space, and inserting a corrugated rubber drain deep into the space, to be left in place for three days. In the tenosynovitis cases incision of the tendon sheath was not needed. When the inflammation was secondary to superficial infection the latter was drained by through-and-through incisions, with administration of systemic penicillin in Group II cases. When secondary to puncture wounds, penicillin only was used, without incision. Pulp-space infections were treated by bilateral incision with through-and-through drainage. In most cases of paronychia the entire nail was removed, as attempts to save part of it resulted in slower healing with a distorted nail and nail-bed.

The dressing applied at operation was changed after three days, when any drain was removed; the wound was then dressed with 17½% sodium sulphate and left for four days. Subsequent dressings, if required, were done at intervals of a further five to seven days. Soaking in hydrogen peroxide to facilitate removal of hardened dressings was reduced to a minimum. Some wounds were dressed with gentian violet, soda sulphate and acriflavine, "tulle gras," or penicillin cream, but the impression was that the medication made little difference to the rate of healing. All septic hands were kept in a sling at least until the first dressing was done. Adequate analgesics were given on the first two to three days after incision, but, if incision had been adequate, little was needed after twenty-four hours. Most noticeable was the great relief of local pain on starting systemic penicillin. Patients were instructed to keep the dressings dry and undisturbed. This ideal could not be attained by all of them—for example, mothers of young children. Such patients were instructed to remove the wet sodden dressing and replace it with a clean piece of dry lint or gauze with no ointment of any kind. Otherwise all dressings were done separately from the rest of the surgery cases, with sterile dressings by a "clean" nurse. No patients were discharged until they were able to do full work with either no dressings at all or a dressing of such size that it would not interfere with their manual skill or render them liable to accident due to clumsiness because of the bandage—an important consideration, as many of these patients were skilled workers.

Results

An analysis of the 168 cases treated without penicillin is given in Table I. The period of unfitness for work is

TABLE I.—Details of 168 Cases Treated Without Penicillin (Group I)

Lesion	No. of Cases	Av. Duration of Symptoms before First Attendance (Days)	Day of Incision after Treatment Started				Average Time Unfit for Work (Days)
			Same Day	1st	2nd	3rd	
Paronychia and pulp-space infections ..	135	6.1	91 (67.4%)	23 (17%)	10 (7.4%)	11 (8.2%)	21.0
Tenosynovitis	16	5.5	9 (56%)	5 (31%)	2 (13%)	—	17.5
Palmar-space infections	17	4.6	9 (53%)	4 (24%)	3 (17%)	1 (6%)	16.5

Average days unfit for work, 20.3.