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HOSPITAL INFECTION OF WAR WOUNDS*

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During the war of 1914-18 hospital infection of war wounds was described by certain bacteriologists and surgeons (Stokes and Tytler, 1918-19; Fleming and Porteus, 1919; and Report, 1919), and since that time a number of outbreaks of surgical hospital infection have been recorded (for references, see McKhann *et al.*, 1938). In the present war Levaditi *et al.* (1939) gave evidence of bacterial infection apparently added at a base hospital in France, and it has been observed in certain British hospitals when, as a consequence of the evacuation from Northern France in early June and of intense aerial warfare in this country, they had to deal direct with the war-wounded.

Hospital infection—infection of the tissues with pathogenic microbes derived from the hospital environment—has long been recognized as a risk to patients. To the surgical patient the risk has been greatly reduced by the practice of antisepsis and asepsis and of the segregation of grossly infected cases. It is less generally recognized, however, that the risk is still serious. During treatment patients are often infected by one another or by the hospital staff, and the hospital staff are themselves sometimes infected by the patients.

Hospital infection in a previously non-infected patient is recognizable clinically only if a distinct symptom-complex directly referable to infection—for example, sore throat, fever, cellulitis, erysipelas, or septicaemia—appears in the course of treatment. It may sometimes be inferred from the delayed healing of a supposedly clean wound or from the failure of a skin graft on apparently healthy granulation tissue. Often infection occurs in those already infected; sepsis is piled upon sepsis, frequently with no obvious change in the patient except perhaps an aggravation of already evident signs. Such acquired infections are recognizable by bacteriological examination. War wounds, many of which are infected by certain species of bacteria at the time of wounding, may acquire a different flora during treatment. For example, the appearance of *Staph. aureus* in a previously clean compound fracture; the sudden appearance of *Strep. pyogenes* in a wound that has yielded nothing but *Staph. aureus* during four consecutive weeks; or the gradual replacement, in a dirty wound, of *Cl. welchii*, *Cl. sporogenes*, and micrococci, by *Bact. coli*, and later by *Ps. pyocyanea* (*B. pyocyaneus*) and *Staph. aureus*.

These occurrences are most reasonably interpreted as hospital infections. However, the sudden appearance of a new microbe in a wound is held by some to be due to the late multiplication of a microbe that has remained dormant since its introduction at the time of wounding. As even optimum methods of culture may not reveal small numbers of bacteria this notion cannot be directly proved or disproved. Its validity depends on the assumption that pathogenic microbes remain dormant in the tissues for a remarkably long time not only in initially infected wounds but in sterile wounds as well. Moreover, to insist that all the bacteria found in a wound at any stage entered it at the time of wounding is to discount the opportunities for infection that arise every time the wound is exposed for treatment or inspection, and to ignore the open reservoir of pathogenic bacteria demonstrable in the patients and staff of a hospital.

Wound Flora

On the other hand, the view that the source of the infection is in the hospital environment is supported by the results of the study of wound flora. Change in flora takes place with remarkable frequency. The microbes most commonly cultivated in the later stages of wound treatment are *Staph. aureus* and *Strep. pyogenes*. There is no general bacteriological evidence, comparable to that advanced, for example, in the case of tetanus, that these two organisms lie dormant in the body tissues for long periods. In fact, in those instances of streptococcal and staphylococcal disease in which the time of infection can be ascertained both organisms are rapidly established in the tissues and demonstrable therein by routine bacteriological methods. Moreover, the hospital origin of these two organisms is made more likely by their known prevalence in the environment of the wounds, for potentially pathogenic staphylococci and streptococci are commonly found in noses and throats, on the skin, and in dust.

The evidence of infection from the hospital environment is particularly strong in the case of haemolytic streptococci. Griffith in 1935 described twenty-seven distinct serological types, which included most of those responsible for infection of the human subject, and the number has since been increased. Surveys of the haemolytic streptococci in the throats of patients on admission to hospital wards show that any one type is seldom found in more than one or two patients. If streptococci appear, for example, in a wound after its handling by a person with

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streptococcal sore throat the inference of hospital infection may be made with greater confidence if the two streptococci are found to be of the same type. The haemolytic streptococci may also be classified in groups, labelled A to K, by the Lancefield-Hare precipitin technique. Most of Griffith's numbered types fall into Group A, and a few into Groups C and G. A haemolytic streptococcus which does not belong to a recognized type may at all events be characterized by its Lancefield group. For epidemiological purposes two streptococci of the same type are assumed to be identical. This assumption, however, cannot be made of two streptococci of the same group, which may include several serological subtypes.

(We are greatly indebted to Dr. F. Griffith and Dr. Dora Colebrook of the Medical Research Council's Research Laboratory for Streptococcal Infections for the typing of the large numbers of streptococcal strains isolated in the course of this work. Without this collaboration the tracing of hospital infection would have lost a great deal of its force and precision.)

There is good evidence not only that hospital infection of war wounds occurs and that ample reservoirs of infection exist in the hospital environment, but that lapses in routine prophylaxis whereby pathogenic bacteria might enter a wound are not infrequent in surgical treatment. Sometimes the routine measures adopted are insufficient for prophylaxis against such infection, and sometimes an adequate routine is inadequately observed, especially by partly trained personnel. It is the object of this paper to relate recent surgical experiences which strongly indicate that the usual routine precautions taken were inadequate, and to suggest modifications of the prophylactic routine of wound treatment. Some of the suggestions might profitably be adopted on the strength of our present knowledge. Others are made tentatively: and it is hoped as a result that surgeons and bacteriologists will collaborate in establishing the mechanisms and extent of hospital infection of wounds and in devising methods of reducing them.

Examples of Presumed Hospital Infection of Wounds

Hospital infection may be presumed from the appearance of pathogenic bacteria in wounds previously free from them, particularly if the bacterium appears some time after the infliction of the wound. Our evidence, based primarily on the demonstration of additions to the bacterial flora of wounds, falls under three main headings:

1. *Comparison of Wound Flora in Groups of Wounds at Different Stages of Treatment.*—For example, Stokes and Tytler (1918-19) found a rise in the incidence of haemolytic streptococci from 14.8% in wounds before operation to 34.6% in wounds four to fourteen days after operation; Fleming and Porteus (1919) demonstrated a similar rise from 15% in recently inflicted wounds to more than 90% in wounds at base hospitals in France.

2. *Additions to flora in individual wounds sampled at intervals.*

3. *Additions to Wound Flora, with Evidence of a Spread of Infection among a Group of Wounded Persons.*—The demonstration of one type of bacterium in several wounds may, of course, be fortuitous, and though it offers evidence of hospital infection it does not necessarily imply infection from a common source or spread from wound to wound. If, however, the bacterium is rare or, better still, if its distribution in the hospital is known at the time of the outbreak, the evidence for spread of infection may be very strong. The readily distinguished thirty types of haemolytic streptococci are admirable "indicator" organisms for investigation of spread of infection. They are common secondary infectors, and their reservoirs—wounds themselves and the upper respiratory tracts of patients and personnel—may be kept under constant survey by periodic swabbing. Our strongest evidence of spread of infection comes from wards in which such a survey was maintained.

Comparison of Initial Wound Flora in Different Groups of Wounded

The bacteria present in wounds include both the pathogenic and the traditionally non-pathogenic. An analysis of gunshot, shrapnel, and air-raid casualty wounds gave the distribution of organisms recorded in Table I. Haemo-

TABLE I.—Wound Flora from Swabs taken on Admission to Advanced Base Hospitals in a London Sector

Type of Bacterium	Percentage Occurrence in		
	105 Dunkirk Wounds	49 Air-raid Casualty Wounds	Total Wounds (154)
Streptococci : haemolytic	31.4	14.3	25.3
viridans and non-haem.	18.1	6.1	13.9
Staphylococci : aureus	54.3	55.1	53.3
albus, micrococci, etc.	19.0	40.8	25.4
G.-negative cocci	1.0	2.0	1.3
Diphtheroids	7.6	18.4	10.8
G.-negative bacilli : coliforms ..	24.7	14.3	20.8
Proteus	6.7	6.1	6.3
Ps. pyocyanea	7.6	6.1	7.0
Haemophilus	1.0	0.0	0.6
Spore-bearers : aerobic	14.3	16.3	14.5
Cl. welchii	22.8	4.1	16.4
Cl. sporogenes	10.5	4.1	8.2
Cl. septicum	3.8	0.0	2.5
Other Clostridia ..	4.7	8.2	5.2
Sterile	11.4	4.1	8.8

lytic streptococci, *Staph. aureus*, and micrococci, coliform bacilli, and spore-bearers are commonest. The percentages take no account of the relative predominance of the various organisms in wounds with a mixed flora, but diphtheroids, aerobic spore-bearers, *Cl. sporogenes*, and *Cl. septicum* were present only in small numbers. The lower frequency of haemolytic streptococci, coliform bacilli, and bacteria of the gas-gangrene group in air-raid casualty wounds is striking. It may be due to the greater time-lag in admission to hospital of the military casualties and to the more immediate treatment of the air-raid casualties in casualty hospitals. The date of wounding is not known for a number of the military casualties recorded in Table I, but the average time-lag was certainly greater than that in the air-raid casualties. The analysis in Table II strongly

TABLE II.—Percentage Frequency of Bacteria in First Swab of Wounds, arranged According to Lapse of Time between Infection of Wound and Swabbing

Type of Bacterium	Lapse of Days between Wounding and Swabbing		
	1-3	4-12	13-40
Streptococci : haemolytic	8.7	18.5	20.0
viridans and non-haem.	15.3	5.5	6.7
Staphylococci : aureus	39.3	50.0	86.5
albus, micrococci, etc.	65.5	35.2	6.7
G.-negative cocci	2.2	0.0	0.0
Diphtheroids	15.3	11.1	33.4
G.-negative bacilli : coliforms ..	28.3	13.0	26.7
Proteus	4.3	1.8	0.0
Ps. pyocyanea	0.0	7.4	20.0
Haemophilus	2.2	0.0	0.0
Spore-bearers : aerobic	47.8	14.8	6.7
Cl. welchii	34.7	11.1	0.0
Cl. sporogenes	6.5	0.0	0.0
Cl. septicum	2.2	0.0	0.0
Other Clostridia ..	8.7	7.4	0.0
Sterile	8.7	11.1	0.0
No. of wounds in each group ..	46	54	15

suggests that *Strep. pyogenes*, *Staph. aureus*, and *Ps. pyocyanea* were commonly added to wounds in the course of treatment before admission to hospital. The percentage frequency of various bacterial species in admission swabs of thirty-seven soldiers and seventy-eight air-raid casualties

is entered in three columns, according to the lapse of time between wounding and swabbing. The 115 wounds are selected only in that none was enclosed in plaster, and the time of wounding in each case was accurately known. The air-raid wounds had mostly been treated by excision, suture, and in some cases by chemotherapy. The military wounds had been treated only at field ambulances and advanced casualty clearing stations. In the early swabs *Staph. albus*, and micrococci, aerobic spore-bearers, *Staph. aureus*, *Cl. welchii*, and coliform bacilli predominate. In the later swabs the frequency of all but the coliform bacilli and *Staph. aureus* declines sharply. Those of low frequency in the early swabs also decline, excepting *Strep. pyogenes*, diphtheroids, and *Ps. pyocyanea*. Even if the percentage values in the "13-40 days" column, derived from fifteen wounds only, are set aside as inconclusive the figures suggest that *Strep. pyogenes*, *Staph. aureus*, and *Ps. pyocyanea* are added to wounds some time after their infliction.

Wound Flora Apparently Acquired in Hospital

(a) Twenty representative wounds in troops, mainly gunshot and shrapnel-inflicted in Belgium and France, were followed bacteriologically by means of weekly swabs from the time of operation at a London sector hospital to the time of healing. One wound was initially sterile. The rest were infected at the time of admission—8 with *Staph. aureus*, 7 with anaerobes, 3 with *Strep. pyogenes*, 3 with micrococci, 2 with coliform bacilli, and 1 each with an aerobic spore-bearer and *Ps. pyocyanea*. Seventeen of the 20 became infected with additional bacteria, and commonly with more than one additional species. The frequency with which the additional organisms appeared in the 17 cases was: *Staph. aureus* 9, *Strep. pyogenes* 8, aerobic spore-bearers 3, *Ps. pyocyanea*, non-haemolytic streptococci, micrococci, and diphtheroids 2 each, and anaerobic spore-bearers and coliform bacilli once each. As noted above, the two most frequent of these secondary invaders, the streptococci and staphylococci, are organisms commonly carried in the human upper respiratory tract. The additional bacteria appeared between three and twenty-seven days after the initial swabbing, and in 7 cases the appearance was accompanied by local and constitutional signs of infection. In these 7 cases the additional organism was *Strep. pyogenes* 3, *Staph. aureus* 3, and *Ps. pyocyanea* 1.

(b) Thirty air-raid wounds, mainly of the head, were similarly investigated. One was sterile, the remainder were infected chiefly with *Staph. aureus* and micrococci. Three were infected with *Strep. pyogenes* and 2 with *Cl. welchii* (without signs of gas gangrene). Additional bacteria appeared in 10, between three and eighteen days after the initial swabbing; again the bacteria added to a single wound often belonged to more than one species. Their frequency was micrococci 5, *Strep. pyogenes* 4, diphtheroids and coliforms 3 each, *Staph. aureus* 2, aerobic spore-bearers and *Strep. viridans* 1 each.

(c) Wounds treated by the closed-plaster method.—The flora at successive plaster changes was observed in 24 cases (15 at one hospital, 9 at another). All were initially infected. Three were swabbed at six, 2 at four, 6 at three, and 13 at two plaster changes. Of these 24 wounds, 16 showed additional flora. The number of new species acquired was in general greater the longer the plasters were observed. Six wounds acquired one new species, 4 acquired two, 1 acquired three, and 3 acquired four. For example, in one of the three wounds observed at six plaster changes, originally infected with *Staph. aureus*, *Ps. pyocyanea* appeared after thirteen days and *Proteus* and a Group C streptococcus after twenty-six days. Another was originally infected with *Staph. aureus* and a Type 27 streptococcus. After twenty-eight days the Type 27 streptococcus had gone and *Proteus* and a Type 1 streptococcus appeared. After thirty-three days *Bact. coli* and *Cl. welchii* appeared. The gradual addition of five and six new species was observed in two patients. The added organisms and their frequencies were: diphtheroids 8; *Strep. pyogenes* 6; *Proteus vulgaris* and *Ps. pyocyanea* 5; coliforms, *Cl. welchii*, and *Staph. albus* 3; enterococci, *Staph. aureus*, and aerobic spore-bearers 2; and *Strep. viridans* 1. *Strep.*

pyogenes, whether initially present or added, usually persisted in the wounds beneath plaster, even though the other organisms died out. It was sometimes obtained in pure culture from a nearly healed wound. It disappeared from only 3 of 26 plaster wounds so infected.

The results under (a), (b), and (c) are representative of a much larger series of observations as yet incomplete. They show that of seventy-four wounds subjected to serial swabbing during their treatment in hospital forty-three (58%) apparently acquired new infecting bacteria. The new species appeared on the average about ten days after the first swabbing in hospital, and on the average about fifteen days after the infection of the wound.

Acquired Wound Flora with Evidence of a Spread of Additional Infection

(a) INFECTION BY AN ATYPICAL PROTEUS

In one ward an unusual atypical *Proteus* appeared in three plasters on July 27 and August 5, 1940. The patients concerned had on several occasions in the preceding three months been removed to the theatre for plaster changes, and in view of the fact that most of the plasters in this ward were removed in the ward itself it is probably significant that all these three plasters were changed on the same day—June 6, 1940.

(b) INFECTION BY UNUSUAL TYPES OF HAEMOLYTIC STREPTOCOCCI

Type B. 3264 was obtained on October 1, 1940, from an abscess in the patient AJ. Three days later JB, a patient five yards across the gangway from AJ, yielded the same streptococcus from a slowly healing purulent wound of the leg that was being treated by exposure to the air of the ward. It was also obtained ten days later from a wound in the same ward treated in a closed plaster.

Type 28/11 (i.e., agglutinating with sera of both Type 28 and Type 11) was isolated from an abscess of the cheek in patient B. O'R, in the next bed, was moved two days later to another ward, where, after an interval of two days, she developed an acute Type 28/11 tonsillitis.

(c) WARD INFECTIONS BY TYPED HAEMOLYTIC STREPTOCOCCI WHOSE DISTRIBUTION WAS KNOWN

Strep. pyogenes was recovered from the wounds of 11 men in two interconnecting thirty-three-bed wards containing part of a Dunkirk convoy. In 8 the infection was apparently acquired in the hospital. There was one group of 5 infected with a Type 27 streptococcus. The weekly swabbing results were as follows:

J.A. Bed 29, Ward 8. Wound Swabs

2/6/40:	<i>Strep. faecalis</i> , <i>Staph. aureus</i>	
10/6/40:	" "	<i>Strep. 27</i> , aerobic spore-bearer
24/6/40:	" "	"
1/7/40:	" "	"
8/7/40:	" "	"

J.S. Bed 32, Ward 8. Wound Swabs

2/6/40:	<i>Cl. sporogenes</i> , micrococci, <i>Strep. Group C</i> , <i>Ps. pyocyanea</i> , <i>Staph. aureus</i> , <i>Bact. coli</i>	
10/6/40:	" "	<i>Strep. 27</i>
24/6/40:	" "	"
1/7/40:	" "	"
8/7/40:	" "	"
15/7/40:	" "	"
25/7/40:	" "	"

DI. Bed 34, Ward 8. Wound Swabs

2/6/40:	<i>Bact. coli</i> , <i>Ps. pyocyanea</i>	
5/6/40:	<i>Bact. alkaligenes</i> , <i>Bact. aerogenes</i>	
12/6/40:	<i>Bact. aerogenes</i> , <i>Ps. pyocyanea</i> , <i>Strep. 27</i>	
25/6/40:	" "	"
1/7/40:	" "	"
8/7/40:	" "	"
15/7/40:	" "	"
22/7/40:	" "	"

BB. Bed 11, Ward 8. Wound Swabs

(The first swab was taken three weeks after admission. No direct evidence of a hospital infection.)

26/6/40 : *Strep.* 27
1/7/40 : "
8/7/40 : "
15/7/40 : "

JM. Bed 66, Ward 9. Wound Swabs

10/6/40 : Non-haemolytic strep., *Staph. aureus*
26/6/40 : *Strep.* 27
1/7/40 : " *Ps. pyocyanea*
4/7/40 : " "
7/7/40 : " "
15/7/40 : " "
22/7/40 : " "

YB. Bed 9, Ward 8. Wound Swabs

5/6/40 : *Strep.* 4, *Staph. aureus*, diphtheroid
24/6/40 : " " "
1/7/40 : " " " diphtheroid
8/7/40 : " " "

WT. Bed 8, Ward 8. Wound Swabs

1/6/40 : *Cl. sporogenes*, *Cl. welchii*
5/6/40 : " " *Staph. aureus*
12/6/40 : " " " *Strep.* 4
24/6/40 : " " "

FM. Bed 4, Ward 8. Throat Swabs

18/6/40 : No haemolytic streptococci
25/6/40 : *Strep.* 4
2/7/40 : No haemolytic streptococci

The beds of the first three patients were close together, and the fourth only 24 feet from Bed 29, diagonally across the ward. The bed proximity associated with an outbreak of *Strep. pyogenes* infection is reminiscent of outbreaks of streptococcal infections investigated in oto-laryngeal wards by Okell and Elliott (1936) and in children's wards by Wright (1940). At the time of the outbreak no other Type 27 streptococcus was isolated from the wounds in the two wards. Nor, with three exceptions, was it found in the throats of the nurses or men from June 18 to September 3. The three exceptions were the patient BB himself, whose throat harboured Type 27 on July 2, the patient OJ, and Nurse W.

In both these groups the suggestion of hospital infection is strong, though the presumptive source could not be traced.

Another patient on June 1 had his arm amputated for a compound fracture wound containing *Cl. welchii* and *Bact. coli*. On June 18 a routine throat swab yielded a Type 11 streptococcus. On June 24 the amputation stump suppurated and the pus contained *Staph. aureus* and *Strep.* 11. The *Strep.* 11 had disappeared from the throat plates on July 7 and from the wound plates on July 8, but a blister on the foot treated by the patient with a penknife suppurated, and on July 15 yielded *Staph. aureus* and *Strep.* 11. Here in the absence of any *Strep.* 11 in the rest of the wards we may postulate an infection transferred

FIG. 1.—A Ward Outbreak of Streptococcal Infection. Eight patients with streptococcal wounds are entered in order of their date of admission. Seven were air-raid casualties, and x marks the date of wounding; TF underwent a "clean" operation; x marks the date of operation. The continuous line marks the date of admission to the date of discharge, death, or transfer to other wards. A square above the line represents a wound swab; below the line, a throat swab. A white square: no haemolytic streptococci isolated (swab not necessarily sterile). A black square: a haemolytic streptococcus isolated; a number refers to its Griffith type, a letter to its Lancefield group, U (untypable) = Lancefield A, not any known Griffith type. On the "TH" line are recorded any positive throat cultures at the date indicated in nursing, theatre, and surgical staffs and in the remainder of the patients in the ward.

OJ. Bed 51, Ward 9. Throat Swabs

18/6/40 : No haemolytic streptococci
25/6/40 : One colony *Strep.* 27
2/7/40 : No haemolytic streptococci
9/7/40 : A few colonies *Strep.* 27

OJ appears to have been a carrier of small amounts of Type 27, and may have been the source of all five Type 27 wound infections, including the case in Bed 66 of the interconnecting Ward 9. The five men had one other feature in common—namely, that they went to the operation theatre on the same day, June 2. However, the same may be said of nearly half the occupants of the sixty-six beds in the two wards, and there is no evidence that the men were operated on in one batch. It is noteworthy that in three instances *Strep.* 27 was accompanied by *Ps. pyocyanea*; the two may have been transmitted together. It is possible that Nurse W acquired a *Strep.* 27 during this outbreak in Ward 9. She had a sore throat from July 29 to August 5, 1940, and on August 1 throat swabs yielded abundant *Strep.* 27.

Strep. 4 was found in two wounds and one throat, as follows:

from one site to another in the same patient.

In Fig. 1 are recorded the relevant features of another *Strep. pyogenes* outbreak. In this case the complete investigation of patients and of surgical and nursing staff was comparatively easy, owing to the small size of the ward and the maintenance of approximately the same nursing staff over the ten-weeks period concerned. The ward was a large room containing twelve male and eleven female beds, separated by a 12-ft. partition not reaching to the ceiling. All twenty-three beds were handled by one nursing unit. The patients were either air-raid casualties or "clean" cases in hospital for therapeutic operations. The streptococci spread mainly among the air-raid wounds. Of the eight patients infected with haemolytic streptococci only SD and TF yielded the organism at the first swab. BW's wounds yielded a Group G at the third and a Group C at the fourth sampling. The Group C presumably came from the patient's skin, which on admission yielded a Group C strain. TF's *Strep.* 11 appeared at the first dressing after a "clean" operation. No Type 11 was found in the ward or theatre staff. TF himself carried a Group G in the throat. The remaining five patients apparently acquired their streptococci in the ward. A

Type 28, a Group A streptococcus not belonging to any known type, and a Type 14 were implicated.

Type 28.—EI and FM had this streptococcus for a short while. FM was at the time severely infected with *Staph. aureus*, *Bact. coli*, *Strep. viridans*, and an organism resembling *Proteus melanovogenes* (Miles and Halnan, 1937), and the Type 28 had no observable effect.

In EI (and in FJ, see below) the appearance of the streptococcus sharply retarded the healing of an otherwise healthy wound containing only a little *Staph. aureus*.

Group A (U).—The untypable Group A infected FJ, and was responsible for the second of HT's hospital infections. There was no evidence of ill effect in HT. These two untypable A's are being investigated. If they prove to be serologically identical we might presume a cross-infection between HT and FJ, whose beds were only a few feet apart, for there were no other untypable A's in wounds or throats at the time.

Type 14.—SD first yielded Type 14 from a slowly healing wounded ear and from the throat. Subsequently, in the absence of any other demonstrable Type 14 in the ward, it appeared in HT, in FM as a second streptococcal infection, and in RM. SD was in the next bed to FM, and RM six feet away on the opposite side of the room. HT was the other side of the partition. Type 14 was not found again in SD's throat, but this patient was later infected by it, under the scab of a light wound that had healed over four weeks previously; the patient had been picking at the scab. FM's wound progressed very slowly, and required three operations in all. There is little doubt that the Type 14 infection was responsible for the delay and difficulties. RM's wound, initially infected with a few micrococci, healed slowly, but, considering the severity and situation of the wound and the presence of infected bone fragments, the delay could not unequivocally be attributed to the Type 14. Nevertheless the streptococcus constituted an unwelcome threat in an already serious situation.

But, whether the Type 14 proved to be deleterious or not, in the absence of any other demonstrable source of the organisms there can be little doubt that it spread through the ward and that the infection was carried from SD's throat or passed from wound to wound. Towards the end of the period the prevalence of Strep. 14 increased. At the routine swabbing on November 14 Strep. 14 appeared in three throats previously free from haemolytic streptococci. There were no signs of infection; three persons—two patients (including RM) and one nurse—thus became clinically undetectable reservoirs of Strep. 14.

(d) THEATRE INFECTION BY TYPABLE HAEMOLYTIC STREPTOCOCCI

In an E.M.S. theatre two patients, Mrs. A and Sergeant B, both clean cases, were operated upon on the same day, in the same theatre, and by the same team. The operations were the first and second on the morning of July 18, 1940, and were left cervical sympathectomy and repair of left brachial plexus respectively. Four days later Sergeant B's incision suppurated, and yielded a Type 11 streptococcus. A shallow ulcer eventually formed, which healed early in September. Five days after operation Mrs. A's incision broke down, and the pus contained Strep. 11. The patient became rapidly worse, and died a few days later from secondary haemorrhage from the subclavian artery. Throat swabs of all the theatre staff present on July 18 showed that one nurse was an abundant carrier of Strep. 11. It seems probable that she was the source of infection because, although there were other cases of Strep. 11 infection in the hospital at the same time, there was no discoverable association between these and the two patients, and no other source of infection was evident. The masks used in the operating theatre were too small to reach well under the chin, and consisted of two thicknesses of gauze with no interposed impervious material. The surgeon himself wore a large and impervious mask.

(The second half of the paper will appear in next week's issue, together with the list of references.)

CEREBROSPINAL MENINGITIS THE USE OF SULPHONAMIDE DERIVATIVES IN PROPHYLAXIS

BY

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The increased prevalence of cerebrospinal meningitis during wartime conditions has long been recognized, and the present war has proved no exception to the general rule.

The use of sulphonamide derivatives in treatment has brought about a marked decrease in the mortality rate, a shortening of the duration of the disease, and a great reduction in the incidence of sequelae. The application of these drugs to the prophylaxis of the disease has not, however, been so successful, and there is still no generally accepted routine procedure for the management of contacts. General measures, such as the swabbing and segregation of contacts, are usually employed, but have proved of doubtful value.

During the outbreak an opportunity was afforded of investigating a number of cases and of studying the effect of intensive sulphonamide therapy on the presence of the meningococcus in the nasopharyngeal mucosa. The results are given in two sections, the first dealing with the cases of cerebrospinal meningitis and the second with problems of prophylaxis.

Technique of the Investigation

Cerebrospinal Fluid.—Samples of cerebrospinal fluid were examined as soon after collection as possible. The deposit after centrifuging for five minutes was inoculated on to blood-agar and boiled-blood-agar plates and Loeffler's serum slopes; films were then made and 5 to 10 c.cm. of glucose broth was added to the remaining deposit. The cultures were incubated at 37° C. and examined daily for seven days. When growth was obtained subcultures were prepared on Loeffler's serum slopes; after twenty-four hours' incubation the subculture was suspended in normal saline, and macroscopic agglutination was carried out at 55° C. with Groups 1 and 2 sera. In a few instances the strains were kindly typed by Dr. W. M. Scott, Types 1 and 3 being identified. The films were stained by Gram's method and also by the technique recommended by Sandiford (1937) for urethral smears from cases of gonorrhoea.

Nasopharyngeal Swabs.—Material was collected from the nasopharynx by means of the West swab and cultured on blood agar. Boiled-blood-agar plates were also used originally, but as they invariably gave results similar to those from the blood-agar plates their use was discontinued. After incubation for twenty-four hours a close examination was made and suspicious colonies were subcultured on to Loeffler's serum slopes. If meningococci were not isolated the original plates were re-examined after incubation for a further twenty-four hours.

Only organisms agglutinating with one or other of the group sera were accepted as meningococci. The characteristic sugar reactions—that is, acid production in glucose and maltose but not in sucrose—were usually but not invariably given by the strains. While so-called inagglutinable strains have been reported by many workers it was considered that criteria other than serological tests were too unreliable for the differentiation of the meningococcus from other *Neisseria*. Wilson (1928), in an investigation of the *Neisseria*, found a considerable amount of