THE INVESTIGATION OF HOSPITAL STAPHYLOCOCCUS OUTBREAKS BY PHAGE TYPING

BY

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which is a major infection has long been a feared
treatment. Today, infection in hospitals due to
staphylococci persists in causing concern to both clinicians and
bacteriologists. There are two reasons for this: the first, the
occurrence of emerge in antibiotic-resistant forms
of staphylococci, multiplying-resistant
in hospitals, particularly in surgical
treatment, secondly the emergence of clone strains of staphylococci.

INTRODUCTION

The first is a brief survey of publication
literature in the field followed by a description of the
results of the use of drugs against Staphylococcus aureus.
Recent advances in knowledge of the microbiology of hospital
staphylococcal infections are then discussed in a review of the
Introduction

The risk of acquiring infection has long been a hazard of hospital treatment. To-day, infection in hospitals due to Staphylococcus aureus is causing concern to both clinicians and bacteriologists. There are two reasons for this: the first, the ability of the organism to emerge in antibiotic-resistant forms; and the second, the tendency of some of these antibiotic-resistant strains to cause epidemics in hospitals, particularly in surgical and maternity units. The purpose of this study was to investigate by means of phage typing, the epidemiology of outbreaks of staphylococcus infection in hospitals. (The term "staphylococcus" as used in this study refers only to coagulase-positive members of the genus.)

This thesis begins with a short history of published studies of hospital infections followed by a description of the development of the technique of phage typing Staphylococcus aureus. Recent advances in knowledge of the epidemiology of hospital staphylococcus infections are then discussed in a review of the literature on the subject. My personal experiences in establishing a phage typing laboratory are related including some
of the difficulties encountered and the way in which they were overcome. Four illustrative outbreaks of hospital staphylococcus infection are described. The 4 outbreaks were chosen for detailed study and description from among the material available to me partly because they were episodes whose significance first became apparent to me because of my vantage point in the phage-typing laboratory. I was able, in these 4 outbreaks, to generate by personal reporting the kind of clinical interest and activity which made it possible to demonstrate how the technique of phage typing could be used to elucidate the epidemiology of the infection and to prevent or control the development of further sepsis. Some interesting features of these outbreaks are discussed together with their significance in relation to the problem as a whole. Finally, certain conclusions are drawn from this work which it is hoped may be of value in preventing future outbreaks of infection.
CHAPTER I

THE HISTORY OF HOSPITAL INFECTIONS

...mention the repetition, spread, accidents, and mortality relating to surgical operations. However, in the early 19th century, Joseph Lister, surgeon to the Liverpool Infirmary, performed a series of operations on ameliorated infected wounds without the loss of a patient or any serious injury.
The History of Hospital Infections

For centuries, suppuration, pyaemia, septicaemia, and death, were expected sequels to surgical operations. Nevertheless, as early as 1782, Edward Alanson, surgeon to the Liverpool Infirmary, performed 35 consecutive amputations on unselected cases in the infirmary without the loss of a patient or any serious wound infection. Since, in these days, amputations were particularly hazardous operations, this series can be regarded as a monumental achievement. Alanson recognised the association of crowded insanitary hospitals and the development of suppuration and advocated the isolation of "all offensive, gangrenous or other putrid sores" and adequate ventilation of the wards. He also recommended that the clothing of patients admitted from infected places and the bedding of any patient with a "putrid" disease "should be baked in an oven constructed for the purpose by which all vermin and infection will be destroyed".

The association between infection and the hospital environment was noted by Oliver Wendell Holmes and Ignaz Semmelweiss in the middle of the nineteenth century. These
workers demonstrated epidemiologically the spread and sources of infection of puerperal fever — then the scourge of maternity wards. It was not until 1867, however, that the first light of understanding of the cause of putrefaction in wounds was shed by Lister. He applied the researches of Pasteur into the nature of fermentation to postulate that putrefaction in wounds might also be due to minute living organisms and advocated the practice of antiseptic surgery using carbolic acid to dress infected wounds (Lister, 1867a). He also suggested that dilute carbolic acid should be used to prevent the access of infection in simple incised wounds at operation (Lister, 1867b). So successful did the antiseptic treatment of wounds prove to be that Lister (1867b) was able to claim that "...... my wards, though in other respects under precisely the same circumstances as before, have completely changed their character; so that during the last nine months not a single instance of pyaemia, hospital gangrene or erysipelas has occurred in them".

The adoption of Lister's methods, and later the aseptic technique evolved by the German workers, Von Bergmann, Neubar, and Schimmelbusch in the years 1880 to 1893, caused a revolutionary
change in hospitals. No longer were surgical wards haunted by the dread of suppuration and hospital gangrene; the era of safe surgery had arrived.

During the twentieth century, however, with the tremendous advances in medical science the problem of hospital infection has again attracted attention. In 1925, for example, Meleney (1935) was disturbed to find that the sepsis rate in clean operation wounds in a hospital in New York was as high as 15%. In maternity units also, some workers described outbreaks of serious infection among the newly born (McGuire, 1903, Benians and Jones, 1929, Poole and Whittle, 1935). The main concern about the problem, however, followed the introduction of penicillin in the therapy of infections. Unlike Streptococcus pyogenes, formerly a serious cause of hospital infection but now invariably amenable to treatment with penicillin, Staph. aureus soon revealed an extraordinary ability to emerge in penicillin-resistant forms. Reports from several workers showed clearly that the proportion of penicillin-resistant staphylococci isolated in hospitals greatly increased after the introduction of the antibiotic into general use and that these strains were well established in many hospitals.
(Barber, 1947; Barber and Rozwadowska-Dowsenko, 1948; Barber, Hayhoe and Whitehead, 1949; Rountree and Thomson, 1949). Later, as other antibiotics were developed and brought into use, hospital strains of staphylococci rapidly emerged which were resistant to some or all of them (Rountree, Barbour and Thomson, 1951; Rountree and Thomson, 1952; Clark, Dalgleish and Gillespie, 1952; Knight and Holzer, 1954). In order to study the epidemiology of infections caused by these antibiotic-resistant staphylococci it was necessary to be able to type them. Two methods of doing this were in use by the late 1940's. The first was the serological method first described by Cowan (1939) who used a slide-agglutination technique with absorbed sera to distinguish three main types of \textit{Staph. aureus}. This method was later extended with the recognition of more types by Christie and Keogh (1940) and Hobbs (1948). Serological typing is still used by some workers — for example, by Brodie and his colleagues in Dundee (Brodie, Kerr and Sommerville, 1956) and Oeding in Norway (Oeding and Williams, 1958). It has been largely superseded, however, in general use by the typing of strains of \textit{Staph. aureus} by their susceptibility to a series of bacteriophages. Oeding and Williams (1958)
have recently published an article in which the bacteriophage and serological methods of typing staphylococci were compared in detail. They found that the 3 major groups of phage types corresponded broadly to the four groups defined among serotypes. A few serotypes were associated with particular phage types but in general there was little correlation between the individual types recognised by the 2 techniques. However, consistent results in the recognition of related strains were obtained with either method. A far greater number of strains can be identified by phage typing but the proportion of untypable strains is higher than with serological typing. There is some variation in the types identified by both methods but this has been assessed more accurately in relation to phage typing. They conclude by stating that although serological typing is technically simpler and more rapid to perform it is less suited to the testing of large numbers of strains than is phage typing.
CHAPTER II

THE DEVELOPMENT OF THE TECHNIQUE FOR

PHAGE TYPING  Staphylococcus aureus
In 1915, Twort described the phenomenon of spontaneous lysis in a culture of a white micrococcus obtained from calf lymph. He showed how the lysis could be transmitted to fresh cultures in filtrates from the original culture. D'Herelle published his observations on the same phenomenon in 1917 followed later by three monographs which reported his detailed work on the subject. D'Herelle suggested the name "bacteriophage" for the lytic agent and held the view that it was a filtrable virus parasitic on bacterial cells. The name of "bacteriophage" for the lytic agent has been widely adopted but is now generally contracted to "phage".

Williams and Timmins (1938) seem to have been the first workers to have used phages to distinguish between different strains of Staph. aureus. They added loopfuls of 4 of the staphylococcal phages isolated by Burnet and Lush (1935) to different broth cultures of staphylococci. They recorded whether cultures showed clearing or lessening of turbidity, and thus distinguished six strains of staphylococci by their different susceptibilities to the phages.
The present method of typing is based on the work of Fisk (1942). He made use of the fact that many strains of staphylococci are lysogenic to isolate phages from them by means of a cross-culture technique. This consisted of spreading a thin streak of one culture on an agar plate and spotting on to it drops of 4 different staphylococcal cultures. If either the basic strain or one of the drop cultures was lysogenic with a phage to which the other was susceptible, small areas of lysis or plaques could be seen in the drop area after incubation. The plaques were picked with needles into 0.5 or 1 ml. of water and propagated by culture with the susceptible strain until confluent lysis was obtained. Fisk’s phage solutions were suspensions of areas of confluent lysis sterilised by the addition of zephiran germicide. The phages were tested on 24 different staphylococcal strains, and 24 phages which exhibited culture-strain specificity were thus identified. Fisk typed a number of staphylococcal strains by spotting drops of the phage solutions on to agar plates spread with the different cultures. After incubation the strains could be distinguished by their different patterns of phage reactions. Fisk noticed that strains from related sources were lysed by the
same phages whereas those from independent sources showed a wide variety of phage reactions. Later, in 1944, Fisk and Mordvin described a further study on the phage typing of staphylococci in which they investigated the epidemiological relationships of a number of strains.

Fisk's work was later extended by Wilson and Atkinson (1945) of the Central Public Health Laboratory, London — now an international centre for staphylococcal phage typing. Wilson and Atkinson modified Fisk's original technique and were able to set up a routine phage typing service for the investigation of epidemics of staphylococcal infection. They isolated phages by cross culture and also obtained new phages by adapting those already isolated to new propagating strains. The phages were propagated by serial passage in fluid medium and the resultant solutions were sterilised by filtration. Great care was taken to ensure the purity of the filtrates and each phage was twice repropagated from a single plaque. Phage filtrates of relatively high titre were obtained and typing was carried out at a critical dilution defined as the highest dilution producing confluent lysis of the propagating strain. In assessing results, Wilson and
Atkinson disregarded any reaction weaker than confluent lysis and were able to differentiate 21 types or subtypes of staphylococci most of which reacted in this way with only one phage filtrate.

Wahl and Lapeyre-Mensignac (1950) suggested another approach to the definition of types. They divided staphylococci into groups by their susceptibility to 5 major phages and further into subgroups by their reactions with 16 minor phages. They regarded reactions of 20 plaques or more as significant.

Further work has shown that neither of these methods of defining types is really satisfactory. That of Wilson and Atkinson had the disadvantage that even identical strains often differ from each other in the strength of their phage reactions and that regarding confluent lysis as the only significant reaction might lead to errors in interpretation: secondly, many strains are typable only with reactions weaker than confluent lysis and would under their scheme be regarded as untypable. The method proposed by Wahl and Lapeyre-Mensignac is rather complex and the division of phages into major and minor phages now seems of doubtful validity, especially in view of the stress which they laid on their major phage 68, which is now known to be a polyvalent phage with a wide
The method of phage typing now widely used throughout the world is that described by Williams and Rippon in 1952, and evolved by them from the work of Wilson and Atkinson and later of Dr. V.D. Allison at the Central Public Health Laboratory, London. Williams and Rippon do not define rigid types or subgroups of staphylococci, although by their method most staphylococci fall into 3 main groups, which correspond broadly to Cowan's (1939) 3 serological groups. They prefer to identify strains by recording a list of the phages with which they give strong reactions. In this way numerous different patterns of phage reactions or "types" can be recognised within each group. Because this method of typing is the one used in this study it is necessary to describe the technique in detail, especially as workers who take it up seldom establish it in their laboratories without a good deal of trouble.

The Technique of Phage Typing of Staphylococcus aureus described by Williams and Rippon (1952).

21 phages are used routinely to type strains of Staph. aureus. They are divided into 3 main groups and one small
subsidiary group. Table I gives a list of the phages and the
groups to which they belong.

In October 1958, phage 70 which is listed in Table I was
omitted from this basic set of typing phages and phage 187 (which
does not belong to any of the 4 phage groups) was introduced into
it.

**Propagation of Phages**

Phages are propagated by growing them with cultures of
the appropriate propagating strain of staphylococcus. There are
two ways of doing this. In the first a fluid medium is used.
The phage is added in suitable dilution to a bottle containing
\( \frac{1}{3} \) of its volume of nutrient broth which has been freshly inoculated
with a \( \frac{1}{1000} \) dilution of a 2-hour broth culture of the propagating
strain. It is usually necessary to add extra calcium to the
culture: this is done by adding a 1% solution (w/v) of calcium
chloride in distilled water to give a final concentration in the
medium of 200 \( \mu \)g. calcium per ml. With some phages a concentration
of 400 \( \mu \)g. calcium per ml. is necessary. The culture is incubated
at 37°C for 6 hours. Frequently it can be seen that the culture
has lysed but sometimes phage is successfully propagated in the
**TABLE I**

**The 21 Typing Phages**

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<th>Group</th>
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<tr>
<td>I</td>
<td>29, 52, 52A, 79, 80</td>
</tr>
<tr>
<td>II</td>
<td>3A, 3B, 3C, 55, 71</td>
</tr>
<tr>
<td>III</td>
<td>6, 7, 42E, 47, 53</td>
</tr>
<tr>
<td></td>
<td>54, 70, 73, 75, 77</td>
</tr>
<tr>
<td>IV</td>
<td>42D</td>
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absence of visible lysis. A preliminary titration is carried out and if the phage titre is satisfactory, the culture is filtered through either a Seitz E.K. filter pad or a Gradocal membrane (average pore diameter 0.7 to 0.9 μm). The filtrate is then stored at 4°C, at which it generally remains stable for about a year — or longer with some filtrates.

The second method of propagation is on solid medium but this has now been superseded by the first method described above. Briefly, it consists of spreading an appropriate dilution of phage over the surface of a nutrient agar plate which has been freshly inoculated with a 4 to 6-hour broth culture of the propagating strain. The plates are incubated overnight at 30°C and in the morning any areas of unlysed growth are cut out with a scalpel. The plate is then frozen at -20°C and thereafter allowed to thaw at room temperature. The agar disintegrates and the resultant fluid, which contains the phage, is pipetted off, titrated, and filtered in the usual way.

**Testing of Phage Filtrates**

After filtration the titre of the phage filtrate is
checked again. Titration is carried out by spotting, from a measured pipette, drops of 0.02 ml. of 10-fold dilutions of the phage filtrate in broth on to the surface of a nutrient agar plate previously flooded with the propagating strain. After overnight incubation at 30°C the degree of lysis in each drop area is recorded. The titre of the filtrate is defined as the highest dilution which just gives confluent lysis on the propagating strain (in practice semiconfluent lysis is used as the end point). This dilution is the one used in typing and is known as the Routine Test Dilution or R.T.D. Phage filtrates must reach a titre of \( \frac{1}{1000} \) or higher before they are satisfactory for use in typing.

Phage filtrates of a satisfactory titre are next tested to ensure that they have undergone neither variation nor contamination. This is done by checking their "lytic spectrum" — that is, the strength and range of their reactions on a series of 22 standard staphylococcal strains, 13 of which are standard propagating strains. These reactions should remain constant for each individual phage, and any deviation from the standard pattern means that the filtrate must be discarded. Fortunately this
rarely happens.

The Typing of Strains of Staphylococci

4-hour broth cultures of the staphyloccocal strains being typed are flooded on to Difco dehydrated nutrient broth agar plates previously dried for 1½ hours in an incubator. As in all phage-typing work, it is essential that the solid media used should contain only 1.0 to 1.2% shred agar. The plates used for typing are marked on the back with a grid of 25 squares and after the culture has been absorbed one drop of each phage filtrate is placed in the appropriate square. Special finely-drawn pipettes are used for this and the drops of phages are spotted on the plates in the order shown in table I. After incubation at 30°C overnight the plates are examined in a good light and all phage reactions are carefully recorded.

Staphylococci are first typed with the phages at their Routine Test Dilutions (R.T.D.) but if no reactions are observed they are retyped with the phage filtrates at 1,000 x R.T.D.

The Recording of Phage-Typing Results

Phage reactions are graded according to the degree of
lysis observed in each drop area. The following symbols are used to record reactions:

- ++ = more than 50 plaques = strong reaction
- + = 20 to 50 plaques = weak reaction
- + = less than 20 plaques = minor reaction

Usually the phage type of a strain of staphylococcus is reported as a list of the phages with which it gives strong or ++ reactions. Sometimes weak or + reactions are also reported although they are qualified by the letter "w". Minor or + reactions are not normally reported except in so far as + following a list of strong reactions indicates the presence of additional minor reactions. When the phages are used at 1,000 x R.T.D. the reactions are recorded as before except that additional symbols are used to draw a distinction between confluent lysis (++++) and strong lysis with secondary growth (+++). Reactions elicited only with the phages at 1,000 x R.T.D. often present considerable difficulty. Unlike the results with the phages at R.T.D., cross reactions between the phages of different groups are common and the pattern of reactions is often very complex. The main
disadvantage, however, is the inhibitory effect of the strong filtrates on some staphylococcal strains. This inhibition may give rise to areas of clearing indistinguishable from confluent lysis or confluent lysis with secondary growth unless the filtrate responsible is titrated on the culture. Unlike true phage reactions, inhibition reactions never give rise to discrete plaques on dilution. In routine practice titration of all possible inhibition reactions is far too laborious and reliance is placed on the quality of secondary growth and other characteristics in attempting to distinguish them. Until recently inhibition reactions were disregarded in assessing typing results but it is now considered that they are significant because they probably represent phage-staphylococcus reactions without phage multiplication.

Interpretation of Phage Typing Results

Most staphylococci of human origin and association, when typed by phages at R.T.D., fall into one of the 3 phage groups I-III (table I) although some cross reactions between phages of groups I and III are not infrequent. It has already been mentioned
that cross reactions are seen far more frequently with the phages at 1000 x R. T. D. Within each phage group, many different patterns of phage reactions are observed although, not surprisingly, some patterns are far more common than others. Many strains of staphylococci react with a single phage but the majority react with more than one. Staphylococci of phage group III often react with 5 or 6 or even more phages.

The main purpose of phage typing is to recognise whether certain strains are identical or not and this is occasionally quite difficult to assess on account of the normal variation observed in typing results. Thus strains from a single source, especially if typed on different occasions, may differ by the loss or gain of a strong reaction or more than one weak reaction. Variations of this sort are more common with staphylococci of phage group III than with those of phage groups I and II. As a general rule, 2 strains of staphylococci are regarded as different if their phage patterns differ by 2 strong reactions; the loss or gain of weak or minor reactions is not usually regarded as significant. In practice strains of phage groups I and II would probably be regarded as different on the basis of less than 2 strong
reactions but hard and fast rules on the interpretation of results cannot be laid down: much depends on experience and knowledge of the frequency distribution of phage types in the environment of an investigation.

CHAPTER XII

CONTROL OF STAPHYLOCOCCAL INFECTION IN HOSPITALS: A REVIEW
CHAPTER III

THE INVESTIGATION OF STAPHYLOCOCCAL INFECTION IN HOSPITALS.

A REVIEW.
The Investigation of Staphylococcal Infection

in Hospitals. A Review.

A recent bibliography compiled by Dr. Dorothy Bocker of the National Library of Medicine, Washington, listed approximately 500 articles on "Staphylococcal Infection" which had been published since 1952. Since there are probably at least as many articles in the literature of the 6 years preceding 1952, it is necessary to be selective rather than comprehensive in reviewing the subject. The following account, therefore, deals mainly with investigations into the epidemiology of staphylococcal infections in which phage typing was used and which have materially increased our understanding of the many complexities of the subject.

Phage typing has shown that outbreaks of staphylococcal infection in hospitals can usually be divided into 3 categories (Anderson and Williams, 1956). In the first category, most or all of the infections are due to one phage type of staphylococcus. Only one or 2 carriers of this strain can be found and it is sometimes possible to demonstrate that one of them is responsible for the spread of infection. In the second type of outbreak also, most of the infections are due to the same phage type of
staphylococcus but in this instance the epidemic strain is found
to be widespread in the hospital or ward environment. In the
third type of outbreak, the infections are due to many different
strains of staphylococci and there is clearly no one common source.
Such outbreaks are usually attributed to a lowering of aseptic
standards and general hygiene.

Most infections in hospital to-day are due to antibiotic-
resistant staphylococci, and phage typing has shown that hospital
endemic strains belong predominantly to phage group III.
Penicillin-resistant staphylococci, when they first appeared,
belonged predominantly to phage group III but are now observed
quite frequently in phage group I — usually when they are
discovered as responsible for outbreaks of infection in maternity
units. To a lesser extent penicillin resistance is now also
appearing in phage group II staphylococci. The majority of
strains which are resistant to more than one antibiotic, however,
are found in phage group III. It is with strains of phage group
III, therefore, that we are concerned in considering the problem
of most hospital infections, although recently a phage group I
strain with some unusual properties (type 80) has been responsible
for some serious outbreaks of infection. Type 80 is commonly resistant to several antibiotics and this distinguishes it from most other staphylococcal strains of phage group I.

Since antibiotic-resistant staphylococci are now ubiquitous in the hospital environment it is not surprising that they are carried in the noses of many of the hospital staff (Rountree and Thomson, 1952; Duncan, Collins, Neelin and Roy, 1957; Hutchison, Green and Grimson, 1957). A vast amount of work has been undertaken in the study of the nasal carriage of staphylococci. The main relevant points which have emerged are summarised below.

Although the staphylococcal nasal carriage rate among the general population is about 50% (Gillespie, Devenish and Cowan, 1939; Rountree, Freeman and Barbour, 1954), the carriage rates among hospital staff and in-patients are usually higher still. Rountree and Barbour (1951) found that the nasal carriage rate among trainee nurses rose from 52.6% to 71.4% within 5 weeks of their entering the hospital wards and Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons (1958) found that 62% of the staff and 60% of patients who had spent 3 weeks or longer in a surgical ward
were nasal carriers. There is a tendency for people to be either persistent carriers or persistent non-carriers (Miles, Williams and Clayton-Cooper, 1944) although a considerable group of people are found who can be classified as temporary carriers and who may be more liable to acquire hospital staphylococci than the others (Hutchison, Green and Grimson, 1957). There is a close association between skin and nasal carriage of staphylococci (Miles, Williams and Clayton-Cooper, 1944) and in the majority of cases the same phage type of staphylococcus is carried on both skin and nose (Williams 1946). An association also exists between nasal and faecal carriage of antibiotic-resistant staphylococci in patients in hospital (Brodie, Kerr and Sommerville 1956).

Hospital staphylococcal infection is particularly troublesome in surgical and maternity units — doubtless because surgical wounds and newborn infants are especially susceptible to infection. A great deal of work has been done to elucidate the epidemiology of staphylococcal infection in both these situations and it is proposed to discuss this more fully in 2 separate sections.
I. Staphylococcal Infection in Surgical Units

Surgical wounds may acquire infection either during operation or, later, in the wards. Some workers have found that the operating theatre is the most frequent source of wound infection — for example, Blowers, Mason, Wallace and Walton (1955), Clarke (1957), Kinmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958). Others have found that the majority of wound infections are due to ward cross infection (Rountree, 1947; Clarke, Dalgleish and Gillespie, 1952; Gillespie, 1957).

1. Sources of Infection in Operating Theatres

Since nasal carriage of staphylococci is so common in the adult population it follows that many of those present in theatre during operations will harbour staphylococci in their noses. Nevertheless it is clear that not all nasal carriers are necessarily dangerous. Hare and Thomas (1956) and Hare and Ridley (1958) have shown that although in most carriers staphylococci are not readily dislodged from the nose, contamination of carriers' skin and clothing is frequent, and these sites may be important sources of infection. They also discovered that some carriers disseminate
large numbers of staphylococci into the surrounding atmosphere on movement — even when they wash their hands. This dissemination was not prevented by their changing into sterile clothing. There is no recorded outbreak of infection traced to a nasal carrier who was also shown to be a disseminator, but nasal carriers have been found to be responsible for some outbreaks. Devenish and Miles (1939) described an outbreak in which the source of infection was a surgeon who was a nasal and skin carrier of staphylococci. They showed how the organisms could be transmitted to the wounds through glove punctures and considered that permeable gown sleeves were another possible route for the spread of infection. A surgeon who was a nasal and skin carrier was implicated in another outbreak of wound infection described by Shooter, Griffiths, Cook and Williams (1957). The staphylococcus carried by the surgeon was the same phage type as those isolated from most of the infected wounds. A similar incident was also described by Blowers, Mason, Wallace and Walton (1955). In 2 other outbreaks of theatre-acquired infection the sources of the infection were traced to theatre nurses who were nasal carriers of the epidemic strain. Penikett, Knox and Liddell (1958) found that the
staphylococcus responsible for the sepsis was spread to the
patients via "Terylene" sutures which had been contaminated by the
nurse who arranged them. In the second outbreak Sompalinsky,
Hermann, Oeding and Rippon (1957) showed that post-operative
infections due to the epidemic strain ceased when the 2 assistant
nurses who carried it were instructed in a stricter aseptic regime
including the wearing of double masks and sterile gloves.

Although Lister 1867 (a) regarded air as a dangerous
source of surgical infection, this view lost favour during the
early part of the twentieth century. Interest in the air as a
source of infection was revived, however, by the work of Bourdillon
and Colebrook (1946). Using a slit sampler to measure the
bacterial content of the air in dressing rooms for burns and major
wounds, they found that large numbers of pathogenic organisms,
including Staph. aureus, were liberated into the air during the
changing of the dressings of large infected wounds. The degree
of bacterial contamination of the air increased when the blankets
and clothing of the patients were disturbed. They also showed
that a system of forced ventilation supplying highly filtered air
at about 10 changes per hour prevented an accumulation of airborne
bacteria and presented evidence that this played an important part in reducing to a low level the incidence of added infection among burned patients. They recommended that operating theatres should be ventilated by a positive pressure or plenum system of ventilation in which warm filtered air enters the theatre at a higher rate than that at which it is extracted. A positive pressure is thus created which prevents air contaminated with dust from the hospital corridors from being sucked into the theatre. A "piston" effect is created by arranging the input in the ceiling and the extract at floor level. In 1955, Blowers, Mason, Wallace and Walton described a serious outbreak of theatre-acquired wound infection in a thoracic surgery unit. One phage type of staphylococcus, the predominant cause of the infections, was widespread in the hospital environment. The unit was closed and reopened after the institution of numerous measures aimed at the prevention and control of infection. The authors attributed the resultant fall in the incidence of infection largely to improved ventilation in the operating theatre, which caused a dramatic reduction in the general bacterial content of the air with the virtual elimination of Staph. aureus from the air sampled. They
regarded the isolation of infected patients, regular sterilisation of hospital bedding, and the exclusion of blankets from the theatre as other important factors in the reduction of the incidence of sepsis.

A second outbreak of post-operative wound infection attributed to inefficient ventilation in the operating theatre was described by Shooter, Taylor, Ellis and Ross (1956). In this instance the infections were due to many different phage types of staphylococci and high bacterial counts in the theatre air were associated with a sepsis rate in clean wounds of 9%. When the ventilation was changed to produce a positive pressure in the theatre with a cross draft over the operating table, the sepsis rate fell to 1%. Kinmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958), on the other hand, found that the introduction of a cross draft over the operating table in a theatre already ventilated by a plenum system reduced the bacterial content of the theatre air but failed to reduce the rate of post-operative wound sepsis.

One difficulty in unreservedly accepting contaminated air as an important source of wound infection is that none of these workers has been able to demonstrate a high level of staphylococcus-
carrying particles in the air. The wounds are not likely, therefore, to be contaminated by more than a very few staphylococci from the air during their exposure at operation. There is, of course, the possibility that each airborne particle carrying staphylococci (which would be recorded as a single colony on a slit sampling plate) might carry large numbers of the organisms, but this seems, on the whole, to be unlikely. A plenum ventilation system also prevents the accumulation of bacterial aerosols which are rapidly cleared when they are set up by the movement of those present in theatre. This may well be the most important function of this sort of controlled ventilation system.

The possibility that staphylococci from the patient's skin might contaminate the wound at operation has been considered by some workers to be an important factor in wound infection. Harrison and Cruickshank (1952) showed by phage typing staphylococci from the skin and from the infected wounds that 4 of 16 staphylococcal wound infections were due to skin-autogenous infection. They attributed a reduction in the incidence of post-operative sepsis to changing the preoperative skin disinfectant from acriflavine to tincture of iodine. Blowers (1957) described
similar investigations in a series of patients with wound infections. In half the patients the same phage type of staphylococcus was obtained by repeated swabbing of the skin preoperatively as that subsequently isolated from the wound infections. Kinmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958) found that acquisition of hospital staphylococci by patients in the wards before operation was an important cause of infection apparently acquired in the theatre.

2. Sources of Infection in Surgical Wards

There are numerous possible sources of infection in most surgical wards. These include: blankets (Rountree, 1947; Blowers, Mason, Wallace and Walton, 1955); the dust and air of the wards (Clarke, Dalgleish and Gillespie, 1952; Shooter, Griffiths, Cook and Williams, 1957); the noses of the medical and nursing staff (Rountree and Thomson, 1949 and 1952); and infected lesions and the noses of the patients (Clarke, Dalgleish and Gillespie, 1952; Barber and Dutton, 1958; Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons, 1958). Unfortunately, it has proved to be very difficult to demonstrate the sources and routes of
spread of staphylococci in ward cross infections. Nevertheless many new facts have come to light in recent years and the epidemiology of ward cross infection has been to some extent, although not entirely, elucidated.

Rountree, in 1947, observing the high bacterial content of ward blankets, investigated the effect of treating them with the disinfectant Fixamol C and oil. She found that although the numbers of bacteria in the blankets and released into the air during bed-making were much reduced, there was no effect on the cross-infection rate in wounds. The presence of some non-pathogenic bacteria as well as *Streptococcus pyogenes* and *Staph. aureus* was used as evidence of cross infection. (Here the word "infection" is clearly used in the sense of transferred bacterial contamination as distinct from clinical sepsis). Phage typing showed that one episode of staphylococcal wound cross infection, involving 8 patients in both the control ward and that in which the treated blankets were in use, was probably due to 2 nurses who were nasal carriers of the epidemic strain. Both nurses were responsible for the dressing of wounds. It is interesting that the strain responsible for this small outbreak could not be
discovered in the ward dust. Similar findings with regard to the effect on nasal and wound cross-infection rates with penicillin-resistant staphylococci were reported by Clarke, Dalgleish, Parry and Gillespie (1954). In this instance oiling of the ward floor, bed clothes, and screen covers had no effect on nasal and wound cross-infection rates although a considerable reduction was observed in the numbers of bacteria released into the air during bed making. These workers suggested that dust from bedclothes played at most a minor part in the transfer of antibiotic-resistant staphylococci to patients in the ward.

Nevertheless there is no doubt that ward blankets are often heavily contaminated with hospital staphylococci (Blowers, Mason, Wallace and Walton, 1955) and considerable research has been done into methods of sterilising them. Disinfection of woollen blankets with quaternary ammonium compounds has been recommended (Blowers and Wallace, 1955) and more recently the use of blankets made of towelling, cotton weave or "Terylene", which can be boiled without damage, has been advocated (Blowers, Potter and Wallace 1957). Schwabacher, Salsbury and Fincham (1958) found that provision of woollen blankets, freshly washed with a quaternary
ammonium compound, for each patient caused a reduction in the total bacterial counts on settling plates and especially in the numbers of an epidemic staphylococcal strain. They found also that the use of cotton blankets caused a greater reduction in total bacterial counts than that obtained by using "Terylene" blankets. They claimed that no cross infection was observed during the trial of cotton blankets, but since the experiment was carried out in a side room containing only 3 beds, this finding is of doubtful significance. They also stated that the use of freshly washed and disinfected woollen blankets was associated with a reduction in cross infection but no figures were given in support of this.

It is clear, then, that although blankets and the dust and air of surgical wards are often contaminated with hospital staphylococci, proof that they are often responsible for the cross infection of patients is still lacking. An interesting point has recently been raised by the finding of Pressley (1958) that airborne dust (previously considered to be contaminated largely from blanket fluff) consisted essentially of cellulose and not woollen fibres, in wards where woollen blankets were used. The dust was found to contain \textit{Staph. aureus} which presumably was
acquired from a source other than the blankets. However, Pressley's use of a "Terylene" filter for collecting the dust has been criticised by Watson (1959) on the grounds that the "Terylene" would repel any particle with the same electrostatic charge. Watson states that it can be inferred from the results of Pressley's experiments that wool particles have a charge similar to that of the "Terylene" filter.

Much the same difficulties are found in assessing the role of nasal carriers on the staff in the spread of infection in surgical wards. The possibility is very difficult to rule out that nasal carriage of staphylococci merely reflects rather than causes the spread of staphylococci. A close correspondence between strains of staphylococci isolated from patients' lesions and from the noses of the staff was found by Rountree and Thomson (1949 and 1952). They mention, however, that this would be found whether the nasal carriers were reservoirs of infecting strains or had merely acquired the strains from the same sources as the patients. Nevertheless an instance has already been quoted in which Rountree (1947) found evidence that 2 nurse nasal carriers were responsible for infecting 8 patients whose wounds they had dressed. Gould
and Allan (1954) showed that there was a sharp fall in the number of hospital staphylococcus infections when nasal carriage of staphylococci among the staff was reduced by intra-nasal treatment with 1% "Terramycin" cream. The incidence of infection rose when the nasal carriage rate again rose after treatment was stopped. This finding, if corroborated, would be striking evidence for the importance of nasal carriers among the staff as a source of infection. Nevertheless it is interesting that in this instance the staphylococci isolated from blankets, dust, fomites and the operating theatre were not the same phage types as those which were epidemic in hospital infections, so that these sites could, in this instance, be ruled out as possible sources of infection.

It has long been recognised that septic lesions are a most important source of infection and Bourdillon and Colebrook (1946) have shown that large numbers of bacteria are liberated into the air when the dressing of a large infected wound is changed. It is also a well accepted fact, at least among bacteriologists, that infected patients should not be nursed in the same wards as patients with "clean" wounds. Unhappily, few general hospitals have isolation facilities nowadays and most surgical wards contain
at least one or two patients who are suffering from infections of one kind or another. Barber and Dutton (1958) have recently described an outbreak of severe infection in a surgical ward in which the epidemic strain was introduced by a patient who was admitted while suffering from superficial septic lesions due to it. In this instance, the epidemic strain was one widely recognised as being of unusual infectivity and this fact may have been partly responsible for the severity and extent of the outbreak. It may have been equally important in this episode that the cross-infecting organisms were derived from active lesions.

It has already been mentioned that the nose and skin of patients often become colonised with hospital staphylococci in the wards and that this may be responsible for the development of wound infection apparently acquired during operation (Kimmouth, Hare, Tracy, Thomas, Marsh and Jantet, 1958). Doubtless a patient's nose and skin may also be colonised in the ward after operation and give rise to autogenous infection with a hospital organism; in this case, of course, the colonisation of the patients would in the first place be due to ward cross infection and the fact that the wound is infected primarily from the patient's skin.
rather than from a source in the hospital is to some extent irrelevant.

An article recently published has helped greatly to clarify the confused state of our knowledge of the mechanism of ward cross infection. This describes an investigation into the spread of staphylococci in a surgical ward undertaken on its reopening and continued over a period of 8 months (Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons, 1958). The principal findings were as follows. On 9 occasions "broadcasts" of certain types of staphylococci into the air and dust of the ward were observed. Seven different phage types of staphylococci were associated with the 9 "broadcasts" and the 3 types which appeared in the air in the greatest numbers did not give rise to any secondary sepsis although they colonised the noses of several patients and members of the staff. Three strains of phage group III, all of which became broadcast, gave rise to 16 of the 24 ward infections observed. There can be little doubt that these strains were more infective than the many other types observed in the ward from time to time, which regardless of their ubiquity gave rise either to no sepsis or to sporadic infections only. It was not
always possible to be certain of the sources from which these broadcasts of staphylococci emanated, but several were attributed to patients with infected lesions. These lesions were of a kind which were particularly liable to disperse — for example, from an infected tracheotomy wound, a urinary infection in an incontinent patient, and a wound infection requiring frequent dressing. In 5 "broadcasts" the strain was first isolated from the nose of a patient or nurse.

The following conclusions may be drawn from the results of this investigation. Staphylococcal contamination of the air is not a reliable index of the risk of ward cross infection and the air itself is probably not a common source of infection. One of the most important factors in ward infection is the infectivity or the ability to produce disease of any particular staphylococcal strain which has access to the patients. Patients with infected lesions, especially those which cannot be kept covered, are important sources from which staphylococci may be dispersed. Nasal carriers may often be responsible for introducing a strain into a ward without necessarily being the immediate cause of its dispersal or "broadcast".
II. Staphylococcal Infection in Maternity Units

Infection due to antibiotic-resistant staphylococci is endemic among the babies in most maternity units to-day. The infections are usually mild superficial lesions — conjunctivitis, for example, or skin pustules, but sometimes the incidence of sepsis builds up to a high level (Barber, Hayhoe and Whitehead, 1949) with occasionally serious and even fatal infections (Beaven and Burry, 1956). Many investigations have been carried out to elucidate the epidemiology of neonatal sepsis, but the picture remains complex and confusing. Numerous reservoirs of staphylococci and possible routes of spread have been demonstrated, but it has proved as difficult to assess the relative importance of these in maternity units as in surgical wards. The following account describes some of the recent advances in our knowledge of the subject, and attempts to assess the situation with regard to staphylococcal epidemiology in neonatal nurseries as it is understood to-day.

In some hospitals babies are nursed beside their mothers in the post-natal wards, but in the majority the babies stay in
communal nurseries and it is in these that staphylococcal infection has become a particular problem. It has been shown that newborn babies rapidly become colonised with hospital staphylococci, and that by the first week or two of life nearly all carry the organism in their noses (Cunliffe, 1949; Wallmark and Melin, 1954; Cook, Parrish and Shooter, 1958). The majority also harbour staphylococci on their skin (Wysham, Mulhern, Navarre, LaVeck, Kennan and Giedt, 1957) and on their umbilicus (Forfar, Balf, Elias-Jones and Edmunds, 1953; Jellard, 1957). It is clearly important to know from what sources and by what means these staphylococci spread to the infants, and to determine the factors which may influence the development of infection.

Possible sources or reservoirs of staphylococci abound in maternity units. Firstly, the mothers may transmit their staphylococci to their babies during breast feeding. Barber, Wilson, Rippon and Williams (1953) have shown that many of the mothers acquire hospital staphylococci in their noses during their stay in hospital. Nevertheless these workers, and also Parker and Kennedy (1949) and Rountree and Barbour (1950), found that the infants only rarely acquired staphylococci from their mothers.
There is some evidence, indeed, from recent work that the infants transmit staphylococci to their mothers (Cook, Parrish and Shooter, 1958; Munro and Markham, 1958).

The second possible reservoir of staphylococci is the noses of the nurses. Several workers have noted that the strains of staphylococci which cause infections in the babies are also present in the noses of many of the nurses (Allison and Hobbs, 1947; Barber, Hayhoe and Whitehead, 1949; Rountree and Barbour, 1950). Allison and Hobbs (1947), Barber, Wilson, Rippon and Williams (1953), and Barber and Burston (1955) have suggested that babies commonly acquire staphylococci from their nurses; and the observation by Rountree, Heseltine, Rheuben and Shearmann (1956) that the neonatal infection rate in a nursery fell simultaneously with the introduction of treatment of staff nasal carriers with intra-nasal antibiotic cream, seems to support this view. On the other hand Parker and Kennedy (1949) and Hutchison and Bowman (1957) found that cases of infection appeared when no nasal carrier of the epidemic strain could be demonstrated among the nurses. Forfar and McCabe (1958) investigated the effect of a strict gowning and masking regime on the staphylococcal
infection and carriage rates in a nursery with a high incidence of sepsis. They observed no reduction in the infant infection and colonisation rates, and concluded that direct transfer of staphylococci from the nose and mouth of attendants in the nursery is not important in infant infection. It should be pointed out, however, that in view of the findings of Hare and Thomas (1956), if any nurse nasal carrier in this experiment had been a disseminator of staphylococci, then strict gowning and masking could not be considered sufficient precautions to prevent the spread of the organisms. The third possible reservoir of staphylococci is the other infants in the nursery and many workers now believe that most infection is the result of infant-to-infant transmission — for example, Parker and Kennedy (1949), Hutchison and Bowman (1957), Wysham, Mulhern, Navarre, LaVeck, Kemen and Giedt (1957). Wentworth, Miller and Wentworth (1958) recently postulated an interesting view. They found evidence that although infant-to-infant transmission was responsible for epidemic spread of the infecting staphylococcus, the organisms were maintained in inter-epidemic periods by chronic nasal carriers on the staff who caused sporadic disease among the infants by direct contact.
Since the majority of babies are extensively colonised with staphylococci after the first few days of life they are obviously reservoirs from which the others may become infected — particularly in epidemic periods when the incidence of infection with a strain is high. The reappearance of an epidemic strain might well be due to the occasional transference of the strain to an infant from a chronic nurse nasal carrier.

There are several different routes by which staphylococci may be spread from infant to infant. Firstly, the nurses may transmit the organisms via their hands or gowns and, secondly, staphylococci may spread via contaminated air, dust or bedding. Staphylococci can occasionally be isolated from the hands or gowns of the nurses (Barber, Wilson, Rippon and Williams, 1953). The work of Cook, Parrish and Shooter (1958) suggests that although this may be the route of spread in some cases it is not responsible for the colonisation of most babies. These workers found that the use by the nurses of chlorhexidine hand cream, and of individual gowns for each infant, caused a small but significant reduction in the staphylococcus nasal carriage rate of the babies. The majority of infants continued to acquire staphylococci in their
noses, however, and they suggest that transmission in these cases must have followed a route other than direct contact from the nurses — possibly the air. Jellard (1957), showed that the infant umbilicus could be a prolific source of staphylococci, and Gillespie, Simpson and Tozer (1958) have confirmed the importance of this source of staphylococci by their observation that the application of dusting powder containing hexachlorophene to the umbilicus and surrounding skin greatly reduced staphylococcal colonisation and cross infection. Jellard also found evidence that babies' noses were colonised via the air. She noted that treatment of the umbilicus by triple dye caused a reduction in skin and nasal carriage of staphylococci and a fall in the staphylococcal contamination of the air suggesting that the organisms may spread from infant umbilicus via the air to another infant's nose. Rountree and Barbour (1950), Wallace and Duguid (1952), and Wysham, Mulhern, Navarre, LaVeck, Kennan and Giedt (1957) have shown that staphylococci are often present in the air of nurseries. Nevertheless the role of the air in the transmission of staphylococci in infant nurseries requires further elucidation. It would be interesting if work could be undertaken to assess if
improvement in nursery ventilation is followed by a reduction in the infant colonisation and sepsis rates.

Much the same difficulties complicate assessment of the significance of dust and bedding in the spread of staphylococci in nurseries. Dust has been shown to be often heavily contaminated with staphylococci (Rountree and Barbour, 1950; Hutchison and Bowman, 1957) but these workers also observed this in the absence of any significant sepsis. Barber, Wilson, Rippon and Williams (1953) on the other hand were unable to isolate any staphylococci from the dust of a nursery where strains of staphylococci were demonstrated to be spreading in the nursery environment. That blankets in neonatal nurseries are often heavily contaminated with staphylococci has been shown by Wysham, Mulhern, Navarre, LaVeck, Kennan and Giedt (1957) and Cook, Parrish and Shooter (1958). Cook and her colleagues found that sterile blankets often became contaminated before the babies acquired their nasal strains but pointed out that although blankets might be responsible for colonising the babies' noses they were not the primary source of infection. The possibility always remains that the contamination of dust and bedding may simply reflect the spread
of staphylococci in the environment: the role of contaminated dust and bedding in the genesis of infection is still obscure.

Two papers have been published which throw light on some of the problems of staphylococcal epidemiology in maternity units. Both demonstrate the spread of staphylococci in nurseries in the absence of severe sepsis. The first, that of Barber, Wilson Rippon and Williams (1953), described how a strain, previously shown to be capable of causing outbreaks of infant infection (Barber, Hayhoe and Whitehead, 1949) could spread extensively in the nursery without causing more than a sporadic case of mild sepsis. The strain was the predominant staphylococcus in the noses of both infants and nurses but was not isolated from bedding or dust. In the second paper, Hutchison and Bowman (1957) observed the spread of staphylococci in a nursery during 3 months, and their findings correspond in many respects to those of Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons (1958) who carried out a similar investigation in a surgical ward. Briefly, Hutchison and Bowman found that different relatively predominant strains of staphylococci waxed and waned from week to week with undercurrents of minor strains appearing sporadically. A high
rate of colonisation by a strain could be found in the absence of infection due to it, although on the whole the incidence of infection by a strain was parallel to its carriage rate in infants' noses. Strains sometimes colonised the infants' noses extensively although absent from those of the nurses, and it was concluded from this that, in infant colonisation and infection, staphylococci were usually transmitted from baby to baby and not from nurse to baby. The dust of the nursery was found to be heavily contaminated with staphylococci and this was due almost entirely to dissemination from infants who acted as the culture medium.

It is clear from the foregoing account that the mechanism of infection in maternity units is still not entirely understood. Why, for instance, are staphylococci sometimes widespread in a nursery in the absence of significant sepsis? The answer probably lies in the differing degrees of infectivity or the ability to produce disease of various staphylococcal strains. Barber and Burston (1955), noting the absence of serious infections from a nursery until a nurse came on duty with a boil on her face, suggested that strains derived from an active lesion might be more virulent than those from healthy nasal carriers. Beaven and
Burry (1956) found an increase in virulence in epidemic staphylococci which resulted in the development of severe and sometimes fatal infections. They found that cross infection and aerial contamination was promoted by overcrowding and postulated that the more rapid passage of organisms which resulted might have lead to an increase in virulence. There remains a third possibility — namely, that some strains of staphylococci may be inherently more infective than others. In the past 3 years events have come to light which confirm that this is so. The one strain now recognised in countries all over the world as being of unusual infectivity and especially liable to give rise to epidemics in hospitals is phage type 80. This unusual strain will now be discussed in more detail, as it deserves.

**Phage Type 80 — A Staphylococcal Strain of Unusual Infectivity**

In 1955 Rountree and Freeman described an epidemic due to a new staphylococcal strain which reacted only with a newly isolated phage numbered 80. This strain was first observed in 1954 in a hospital in Sydney, where it caused an epidemic of
unusually severe skin lesions in both patients and staff. The rate of nasal carriage of the strain was disproportionately low, and this, together with the fact that the infections which it caused among the staff appeared to be superimposed on the normal stratum of autogenous infection, suggested that it possessed enhanced virulence. The unusual severity of the lesions supported this view. After its discovery in Sydney it was identified as the cause of numerous epidemics throughout Australia and one in New Zealand. It has since been reported in outbreaks of infection in many other parts of the world — for example, in the United Kingdom (Duthie, 1957; Gillespie and Alder, 1957), the United States of America, where it is known as type 42B/52/81 with or without 80, 44A, and 47C (Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright, 1958; Ravenholt, Wright and Mulhern, 1957; Shaffer, Sylvester, Baldwin and Rhiens, 1957), Canada (Bynoe, Elder and Comtois, 1956), New Zealand (Munro and Markham, 1958), and Uganda (Hennessey and Miles, 1958).

In most outbreaks, type 80 has shown a clear predominance as a cause of sepsis; and infections due to it are often unusually severe (Gillespie and Alder, 1957; Bass, Stinebring, Willard and
Felton, 1958) although Munro and Markham (1958) found that this is not always so. In maternity units, type 80 has been the cause of widespread infection among both babies and mothers — and in some instances fatal infections in the babies have been described (Fekety, Buchbinder, Shaffer, Goldberg, Price and Pyle, 1958; Cooper and Keller, 1958). In surgical units it has caused wound infections and septic skin lesions in both patients and staff (Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright, 1958; Duthie, 1957; Barber and Dutton, 1958). The ability of the strain to colonise the nose seems to have differed from outbreak to outbreak. Thus Duncan, Collins, Neelin and Roy (1957) and Duthie (1957) found that it rapidly became widespread in the noses of hospital staff, whereas others found that its prevalence in the nose was low compared with its prevalence in infections (Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright, 1958). However, in a recently published review of epidemic staphylococci, Williams (1959) reported that type 80 commonly generates a high rate of carriage, and often a high incidence of skin lesions also among the hospital staff. He describes type 80 as "... a strain that has more recorded epidemics
to its credit than any other type; whose epidemics are, in our experience, twice as extensive as the average of all other types."

Recently some American workers investigated the incidence of sepsis in babies and mothers sent home from hospitals where type 80 was epidemic (Ravenholt, Wright and Mulhern, 1957; Wysham, Mulhern, Navarre, LaVeck, Kennan and Giedt, 1957; Fekety, Buchbinder, Shaffer, Goldberg, Price and Pyle, 1958). This revealed that a considerable amount of infection develops in babies and mothers only after their discharge from hospital. Wentworth, Miller and Wentworth (1958) and Cooper and Keller (1958) showed that type 80 might spread to the families of infants and mothers recently discharged from a hospital. In this way the strain might become, in the community at large, a cause of a kind of infection which was mostly resistant to several antibiotics.

Rountree and Beard (1958) published an excellent description of the effects of type 80 on the population both in and outside hospitals in Australia during the 3 years since it first made its appearance there. The strain was responsible for 70 of 86 major outbreaks of staphylococcal infection in maternity hospitals and was the major cause of puerperal breast abscess in
mothers and of serious staphylococcal disease in both infants and older children. It was the predominant cause of infection among patients and staff in a large general hospital and there was evidence that it had also spread in the community at large. A marked increase in the number of cases of generalised staphylococcal infection admitted to a general hospital was almost entirely due to this strain and it was an important cause of fulminating staphylococcal pneumonia following Asian influenza. Rountree and Beard regard the strain as having high infectivity rather than high virulence because they found that although it appeared to cause more severe disease, the mortality rate in cases of generalised infection due to it was not significantly higher than in those due to other strains. They considered that the extensive spread of this phage-group I organism in the community was due both to its penicillin resistance and to its ability to invade the healthy skin — a property which is not a characteristic of the antibiotic-resistant strains of phage group III which normally predominate in hospital wards.

It is clear that this particular strain of staphylococcus is causing considerable concern to both clinician and bacteriologist.
It also offers the bacteriologist a challenge. Type 80, because of its unusual communicability and ability to produce disease, provides a unique opportunity to investigate the epidemiology of hospital staphylococcal infection and to apply the knowledge acquired in these studies for the control of its further spread.
CHAPTER IV

PERSONAL EXPERIENCE OF PHAGE TYPING

a) Establishing a Phage-typing Laboratory
b) Local Distribution of Staphylococcal Strains
c) An Outbreak of Staphylococcal Infection in the Western Infirmary, Glasgow
d) An Outbreak of Staphylococcal Infection in Law Hospital, Lanarkshire
e) An Outbreak of Staphylococcal Infection in the Southern General Hospital, Glasgow
f) An Outbreak of Staphylococcal Infection in the Royal Maternity Hospital, Glasgow
PERSONAL EXPERIENCE OF PHAGE-TYPING

a) Establishing a Phage-typing Laboratory

The technique of phage typing staphylococci has been described in detail in Chapter II. Dr. R.E.O. Williams and Dr. Joan E. Rippon, who evolved the method now widely used, have taught the technique to other workers who have wanted to use it when studying staphylococcal epidemiology. When it was decided that a laboratory undertaking the phage typing of staphylococci should be established in the Western Infirmary, Mr. D.B. Colquhoun, the technician who has assisted in this work, and I each spent a week at the Staphylococcus Reference Laboratory in the Central Public Health Laboratory, Colindale, London, learning the technique from Dr. Williams and Dr. Rippon.

The technique is complex and numerous difficulties confront those embarking on the work for the first time, not all of which are apparent while one is working in the central laboratory at Colindale. In this laboratory one year's work was required to establish typing on a reliable basis.

The first few weeks of the work were spent in assembling
all the necessary equipment. Stocks of measured pipettes and finely drawn typing pipettes were prepared, petri dishes were marked, and the special medium for typing ("Difco" dehydrated nutrient broth agar) was obtained and made up. When the laboratory was stocked with all the necessary equipment the phage filtrates brought from London were examined and titrated. This, unfortunately, revealed that the majority were no longer at a satisfactory titre. Since we, like most laboratories (excepting some in the Public Health Laboratory Service in England) had to propagate the phages ourselves, we started attempting to raise the filtrates to satisfactory titres. This proved to be unexpectedly difficult. For phage propagation we had been advised to use "a good nutrient broth," but it soon became apparent that the nutrient broth satisfactory for routine bacteriological work in the Western Infirmary was unsuitable for propagating phage. The results were most erratic; with a very few phages, satisfactory titres were achieved after only one or two attempts at propagation, but with the majority there was little or no phage multiplication even after repeated attempts. The use of nutrient agar as a solid medium for propagation gave no better results. Finally, after
unsuccessful trials of various media, we obtained the recipe for the nutrient broth used for phage propagation in the central laboratory at Colindale. The basic difference between this medium and the one used routinely in the Western Infirmary was that the Colindale medium was made from fresh ox heart or muscle instead of from horse heart. We made the medium ourselves so that certain necessary precautions could be better observed. These included the following points. The meat used must be fat free and obtained fresh from the slaughterhouse; it is then infused overnight with copper-free triple-distilled water. In propagation, heating is kept to a minimum and care is taken in adjusting the pH so that no alkali is added after the precipitation and removal by filtration of the phosphates in the medium. The broth should be made in a container free from traces of contaminating copper and a covered stainless steel pan was recommended for this. After all these precautions were carefully observed the nutrient broth so prepared proved entirely successful. We were warned that occasionally a batch might inexplicably give poor results in propagation, but so far our medium has always proved satisfactory. The majority of phages now reach a titre of
1 in 10,000 or higher on first propagation although a few of the more difficult phages rarely rise above a titre of 1 in 1,000 and may need more than one propagation to achieve this. Propagations have now become an entirely routine procedure and we have never been worried about our stocks of phages since using the medium prepared exactly as described above.

Another difficulty became apparent after we had overcome the problem of phage propagation when Mr. D.B. Colquhoun discovered that horse heart nutrient broth agar gave lower readings of phage titrations than did the typing medium ("Difco" dehydrated nutrient broth agar). It was also found that when the "Difco" medium was underlaid with peptone water agar — and this is recommended by the central laboratory, Colindale, in the interests of economy — better results were obtained than with the "Difco" medium alone. Mr. Colquhoun carried out further experiments which showed that titrations on peptone-water agar itself gave higher readings than on any other medium. It was clear that the same medium must be used for both titrations and typing, since any variation would mean that the phages used in typing were not at the correct routine test dilution (R.T.D.) on the typing medium. It was therefore
decided to use peptone water agar for both titrations and typing and this has proved completely successful over a period of nearly 3\(\frac{1}{2}\) years. One possible objection to the use of this medium is that some strains of staphylococci might require a richer medium for adequate growth on the typing plates. In practice, this has been a very rare event and only 6 strains have failed to grow on the medium.

The phage filtrates have proved to be remarkably stable and most retain their titre over many months in storage at 4°C. Although many phages require repropagation in about 8-12 months, some have remained at a good titre for over 18 months. One constant problem is the prevention of contamination of stock phage solutions. These are stored as broth filtrates without any preservative, and thus may be contaminated with moulds, and sometimes bacteria, from time to time. Moulds may be removed by filtration but we prefer to discard any filtrate which is found to contain one. The danger of contamination has been reduced to a minimum by very careful handling of the filtrates and by storing them in a series of small screw-capped bottles instead of one large one. Titrations and dilutions for typing are made from one
bottle only, and if, as a result of repeated opening, it becomes contaminated one of the other bottles may still be used. The diluted filtrates used in typing are also kept in 5 ml. screw-capped bottles, and although they are opened nearly every day, they have proved remarkably free from contamination. Due to the volume of work now undertaken, fresh dilutions are made up about every 2 to 3 months. The routine test dilutions are tested by spotting each phage on its propagating strain approximately every 3 weeks, to ensure that the phage still produces semi-confluent lysis.

The lytic spectra of the phages have also been very constant. Variations have occasionally been observed elsewhere; in America, for example, type 80 was lysed by additional phages which had undergone mutation (Blair and Carr, 1958). The only phage which we have found to vary is 42D which is one of the least useful phages in the basic set and rarely lyses human staphylococcal strains by itself. Our filtrates of 42D have shown a persistent tendency to lyse some of the group III strains in the lytic spectrum. Even after a successful propagation from fresh stocks of phage and propagating strain, 42D has again shown this change on repropagation after an interval of time. The explanation for this
change is unknown but 42D has been found by workers at the central laboratory at Colindale to show an unusual tendency to variation (Dr. Rippon — personal communication).

The method of recording phage reactions has been slightly modified from that used in the central laboratory which is described on page 18. Confluent lysis, whether obtained with phage at R.T.D. or 1,000 x R.T.D., is designated as +++ but the degree of secondary growth at 1,000 x R.T.D. is estimated and expressed as the denominator of a fraction. In this way reactions with maximal secondary growth are recorded as \( \frac{+++}{4} \) and those with minimal secondary growth as \( \frac{+++}{1} \). Many of the reactions with secondary growth are probably inhibition reactions but as these are now regarded as significant phage reactions this does not affect the interpretation of results. This system of recording the amount of secondary growth is exceedingly useful as it permits one to visualise in retrospect the appearance of the original reaction. Semi-confluent lysis is recorded as ++(+); all other reactions are designated by the method used in the central laboratory. In June 1958 we introduced 2 new phages into the routine typing set. These were designated "B" and "C"
and were isolated by Mr. Colquhoun from lysogenic cultures of strains which failed to react with any of the basic phages. Phages B and C have proved exceedingly valuable in identifying some important locally-distributed strains which would otherwise be untypable. Both these phages were sent down to the central laboratory in London for further testing with the result that phage C has now been incorporated in one of the extra "pool" of phages used there.

When all the difficulties had been encountered and overcome, the phage typing technique very quickly became a routine procedure. Considerable experience is necessary, however, in the reading of typing plates and particularly in the interpretation of results. This has gradually been built up over the months and in the 3 years since we began regular typing, we have phage typed more than 9,000 strains of staphylococci. A considerable proportion of these have been sent in from several hospitals in the west of Scotland and, in fact, the laboratory in the Western Infirmary has now become a local staphylococcal reference laboratory for those who care to call upon it for this service.
In October, 1957, an opportunity arose to test our phage typing methods, when the central laboratory at Colindale sent to all those undertaking phage typing, 33 staphylococcal strains for a comparative phage typing test. The strains were typed in the usual way and a report was returned to the central laboratory at Colindale of the phage types of the strains with full details of every phage reaction obtained with each. Twenty-nine phage typing laboratories took part in the test; 10 used phage filtrates supplied from the central laboratory and 19 propagated their own phages, although these had originally been obtained from the laboratory at Colindale. The typing in the Western Infirmary was carried out with peptone-water agar but was repeated using the recommended "Difco" medium for comparison. The results, however, were in very close agreement and the few differences which were noted were minor ones only. Since it was the medium in routine use, the results on peptone-water agar were submitted for assessment in the test.
The Results of the Test

The analysis of the results obtained in the test was most interesting. The typing results of a laboratory were assessed by comparing the phage reactions obtained with each of the 33 test strains to a "modal" pattern. "Modal" patterns were defined as a list of the phage reactions obtained by the greatest number of laboratories. The typing results of each test strain were graded into 5 categories according to the following convention:

**Category 1.** Identical with "modal result".

**Category 2.** Minor differences from "modal" pattern with 1 or more phages, which did not alter the report on the strain.

**Category 3.** Minor differences with 1 or more phages which did result in alteration of the report.

**Category 4.** One strong (++) difference with or without additional minor differences.

**Category 5.** Two strong (++) differences with or without additional minor differences.
This can be interpreted as meaning that strains in category 5 would be regarded as significantly different from a strain of the "modal" pattern. Those in categories 3 and 4 although showing considerable variation do not necessarily differ significantly from the "modal result", whereas strains in categories 1 and 2 would definitely be considered the same as strains of the "modal" pattern.

The overall result of the survey enabled the following conclusions to be drawn.

1. The results from laboratories using phage prepared in the central laboratory showed less variation than those from laboratories propagating their own phages.

2. The results with strains of groups I and II were more homogeneous than those with strains of group III or group I/III.

3. Taken as a whole the results could be regarded as very satisfactory and confirmed the usefulness of the technique.

The results obtained in the Western Infirmary showed that our category grading was placed about the middle of the gradings of laboratories propagating their own phages. Table II
# Table II

Category Grading of Strains Typed in the Western Infirmary for the Comparative Phage-typing Test, 1957

<table>
<thead>
<tr>
<th>Number of Strains Typed</th>
<th>35*</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Strains in Category 1</td>
<td>9</td>
</tr>
<tr>
<td>No. of Strains in Category 2</td>
<td>4</td>
</tr>
<tr>
<td>No. of Strains in Category 3</td>
<td>9</td>
</tr>
<tr>
<td>No. of Strains in Category 4</td>
<td>9</td>
</tr>
<tr>
<td>No. of Strains in Category 5</td>
<td>4</td>
</tr>
<tr>
<td>No. of Strains in Categories 1 and 2</td>
<td>13</td>
</tr>
<tr>
<td>Average Category Grading</td>
<td>2.86</td>
</tr>
</tbody>
</table>

*2 strains were typed at both R.T.D. and 1,000 x R.T.D. and the results have been assessed separately.
shows our grading results in detail.

Table II shows that although there were 18 strains in Categories 3 and 4, only 4 strains were in Category 5 which represents a significant deviation from the "modal" pattern. 13 strains showed no variation or only very minor differences from the "modal" result.

The individual performances of the phages used by the various laboratories were also assessed by recording the number of strong (++) reactions they gained or lost compared to the "modal" reactions. The overall results showed that the group III phages of most laboratories showed considerable variation compared to those of group I (with the exception of phage 79). Group II phages showed very little differences from "modal" results.

The results obtained in the Western Infirmary are shown in Table III. The results of this test of individual phage performances show that although the majority of phages showed no tendency to lose or gain strong reactions, phages 79, 7, 70, 73 and 75 were over-reactive and phage 47 was under-reactive. All these phages had correct lytic spectra and it was interesting that 4,2D, which had shown a distinct tendency to vary in its lytic
TABLE III

Individual Performances of Phages Used by the Western Infirmary Laboratory in the Comparative Phage-typing Test, 1957

<table>
<thead>
<tr>
<th>Phage</th>
<th>No. of ++ reactions which should have been negative</th>
<th>No. of negative reactions which should have been ++</th>
<th>No. of strains whose modal phage reaction was negative</th>
<th>No. of strains whose modal phage reaction was ++</th>
</tr>
</thead>
<tbody>
<tr>
<td>29</td>
<td>0</td>
<td>0</td>
<td>27</td>
<td>6</td>
</tr>
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<td>25</td>
<td>8</td>
</tr>
<tr>
<td>52A</td>
<td>1</td>
<td>0</td>
<td>24</td>
<td>8</td>
</tr>
<tr>
<td>79</td>
<td>3</td>
<td>0</td>
<td>29</td>
<td>3</td>
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<td>80</td>
<td>0</td>
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<td>20</td>
<td>10</td>
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<td>3A</td>
<td>0</td>
<td>0</td>
<td>31</td>
<td>1</td>
</tr>
<tr>
<td>3B</td>
<td>0</td>
<td>0</td>
<td>30</td>
<td>2</td>
</tr>
<tr>
<td>3C</td>
<td>0</td>
<td>0</td>
<td>29</td>
<td>4</td>
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<tr>
<td>55</td>
<td>0</td>
<td>0</td>
<td>27</td>
<td>5</td>
</tr>
<tr>
<td>74</td>
<td>0</td>
<td>0</td>
<td>29</td>
<td>4</td>
</tr>
<tr>
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<td>73</td>
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<td>75</td>
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<td>0</td>
<td>27</td>
<td>5</td>
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<td>77</td>
<td>0</td>
<td>0</td>
<td>30</td>
<td>3</td>
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<td>42D</td>
<td>0</td>
<td>0</td>
<td>32</td>
<td>0</td>
</tr>
</tbody>
</table>
spectrum, gave no extra reactions.

The comparative phage typing test was most reassuring. Only 4 strains had shown a typing result which differed significantly from the "modal" pattern and the majority of phages had no tendency to gain or lose strong "modal" reactions.

Although our results showed much more variation than those from the laboratories using phages prepared in the central laboratory, when compared with those from the other laboratories propagating their own phages, they were most satisfactory.
b) Local Distribution of Staphylococcal Strains

In July 1958 and in June 1959 the percentage frequency distribution was calculated of the phage types of staphylococci isolated from septic lesions and examined in the Western Infirmary during the 2 years from 1st June, 1957 to 31st May, 1958 and from 1st June, 1958 to 31st May, 1959. 8,231 strains of staphylococci were phage typed in these 2 years but only 3,356 have been included in the surveys — the remainder being omitted either because they were not isolated from lesions or because their origin was uncertain. Staphylococcal strains from hospital patients in surgical and general wards and in maternity units were compared with those from outpatients.

Several hospitals in the west of Scotland submit staphylococci to the Western Infirmary for phage typing and many more were doing so during the second survey than in the previous year. The second survey, therefore, includes nearly twice as many strains as the first. Staphylococci from surgical and general wards (the majority of which were in fact from surgical wards) came from 3 hospitals where routine phage typing was carried out on all staphylococci isolated (the Western Infirmary,
Glasgow, Law Hospital, Lanarkshire, and the Vale of Leven Hospital, Alexandria), and from 8 hospitals from which batches of selected strains were examined from time to time (Glasgow Royal Infirmary, The Royal Hospital for Sick Children, Southern General Hospital, Belvidere Hospital, Robroyston Hospital, and Ruchill Hospital, in Glasgow, the Ayrshire Area Laboratory, and Gateside Hospital, Greenock). Most of the strains isolated in maternity units, on the other hand, came from the Glasgow Royal Maternity Hospital and its associated hospitals, the Ross Hospital, Paisley, and Redlands Hospital, Glasgow. However the Bellshill Maternity Hospital, Thornhill Maternity Hospital, Johnstone, and the Rankine Memorial Hospital, Greenock, also submitted strains from time to time and these, together with strains isolated from cases of puerperal breast abscess and infant infection after discharge from hospital, have contributed to this part of the surveys. In general, therefore, the staphylococci from surgical and general wards have been derived from a wider variety of sources than those from maternity units.

The results of the 2 surveys are shown in tables IV and V. The staphylococci assessed have been divided into phage
<table>
<thead>
<tr>
<th>Group</th>
<th>Phage pattern</th>
<th>Septic lesions in hospital patients</th>
<th>Hospital outpatients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Surgical &amp; general</td>
<td>Maternity</td>
</tr>
<tr>
<td>I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29, 29</td>
<td>3.2</td>
<td>7.5</td>
<td>(3.6)</td>
</tr>
<tr>
<td>29/52</td>
<td>0.7</td>
<td>0.5</td>
<td>-</td>
</tr>
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<td>52, 52</td>
<td>2</td>
<td>1.8</td>
<td>(1.3)</td>
</tr>
<tr>
<td>52/52A, 52/52A</td>
<td>0.2</td>
<td>-</td>
<td>(0.4)</td>
</tr>
<tr>
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<td>3.6</td>
<td>4.9</td>
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<td>(0.9)</td>
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<td>4.4</td>
<td>13.4</td>
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<td>10.5</td>
<td>4.5</td>
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<td>3.1</td>
<td>4.9</td>
</tr>
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<td>4.9</td>
<td>5.4</td>
</tr>
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<td>32.3</td>
</tr>
<tr>
<td>II</td>
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<tr>
<td>71</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>36/55/71</td>
<td>(0.7)</td>
<td>3.1</td>
<td>4.5</td>
</tr>
<tr>
<td>36/71</td>
<td></td>
<td>(0.3)</td>
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</tr>
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<td>7.7</td>
<td>17.4</td>
</tr>
<tr>
<td>Total for Group</td>
<td>6.6</td>
<td>12.9</td>
<td>30.8</td>
</tr>
<tr>
<td>III</td>
<td></td>
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<td></td>
</tr>
<tr>
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<td>0.3</td>
<td>-</td>
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<td>0.5</td>
<td>-</td>
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<td>1.8</td>
<td>-</td>
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<tr>
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<td>(0.3)</td>
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<td>6.3</td>
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<td>7.1</td>
</tr>
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<td>Total strains</td>
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<td>224</td>
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<td>Group</td>
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<td>Septic lesions in hospital patients</td>
<td>Hospital outpatients</td>
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<td>---------------</td>
<td>-----------------------------------</td>
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<td>I</td>
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<td>4.2 1.4 1.3</td>
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<td>1.6 (1.3)</td>
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<td>1.6 (1.3)</td>
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</tr>
<tr>
<td></td>
<td>Other Group I</td>
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<td>6.9 (1.3)</td>
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<td></td>
<td>Total for Group</td>
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<td>1.3 (1.3)</td>
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<td>related patterns</td>
<td>5.2 (1.3)</td>
<td>1.9 (1.3)</td>
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<tr>
<td></td>
<td>7/47/54/75/77, 75/77, ..., 75, ..., 77</td>
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<td>1.1 (1.3)</td>
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<tr>
<td></td>
<td>related patterns</td>
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<td>2.3 (1.3)</td>
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<tr>
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<td>Other Group III</td>
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<tr>
<td></td>
<td>Total for Group</td>
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<td>22.1 (1.3)</td>
</tr>
<tr>
<td>IV</td>
<td>Total</td>
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<td>(0.8) (1.3)</td>
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<tr>
<td></td>
<td>187</td>
<td>(0.3) (0.4)</td>
<td>(0.4) (1.3)</td>
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<tr>
<td></td>
<td>B</td>
<td>(0.4) (0.4)</td>
<td>(0.2) (1.3)</td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>2.5 (1.3)</td>
<td>2.9 (1.3)</td>
</tr>
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<td>4.6 (1.3)</td>
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<td>Mixed groups</td>
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<td>8.2 (1.3)</td>
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<td>Untypable strains</td>
<td>14.0 (1.3)</td>
<td>13.5 (1.3)</td>
</tr>
<tr>
<td></td>
<td>Total strains</td>
<td>898 (1.3)</td>
<td>832 (1.3)</td>
</tr>
</tbody>
</table>
which had caused a small outbreak of infection. It is necessary, therefore, to know the epidemiological background of the hospitals where infections were under investigation to assess fully the significance of tables IV and V.

Tables IV and V show, firstly, that the group distribution of staphylococci varies among the 3 different kinds of patients. Thus phage group III strains predominated in surgical and general wards and phage group I strains predominated in maternity units. The higher incidence of phage group I strains in surgical and general wards in table V was almost entirely due to the extraordinary predominance of the highly epidemic phage group I strain — type 80. If allowance is made for this unusual strain, phage group III strains emerge clearly as the most frequent cause of infection in these wards. In outpatients, strains of phage group I and II were responsible for most of the infections — of which phage group III strains caused only a small proportion.

Secondly, tables IV and V show that phage types as well as phage groups varied in their distribution among different kinds of patient. The overall epidemiological picture of phage types
was of a pool of hospital strains which varied in the frequency
with which they gave rise to disease. These strains could be
classified broadly as epidemic, endemic or sporadic.

Strains were regarded as epidemic when they became a
predominant cause of infection within one unit or hospital. One
major epidemic strain produced extensive epidemics and there were
several minor epidemic strains also which caused small scale
outbreaks of infection. The endemic strains formed the larger
part of the hospital staphylococcus pool. Although some from
time to time emerged as minor epidemic strains the majority did
not predominate but simply caused infection regularly but with
varying frequency. There was a tendency for an individual strain
to vary in its frequency distribution in the 2 kinds of hospital
patient. Sporadic strains had no real epidemiological
significance but appeared at random as a cause of occasional
infection. When the epidemiological background of a unit or
hospital was known, sporadic strains could be distinguished from
endemic strains which were relatively uncommon.

Type 80 was the only example of a major epidemic strain
and showed a remarkable ability to spread and rapidly to become
the major cause of infection in any kind of hospital unit. Its incidence in maternity units was lower in 1958-1959 (table V) than in the previous year and this was due to the type-80 epidemic in the Royal Maternity Hospital during the first survey. In the following year type 80 caused epidemics in several other maternity units in the area and although a far greater number of specimens were submitted for phage typing from the Royal Maternity Hospital and its associated hospitals, type 80 was so predominant in the infections originating in these other units that it outnumbered all other strains as a cause of maternity hospital infection.

Several minor epidemic strains were observed during the 2 years of the surveys which although they did not cause widespread infection, emerged as the predominant infecting strain in a unit or in a hospital for varying periods of time. Examples of minor epidemic strains in surgical and general wards were type 6/47/53/54 in the surgical wards of Glasgow Royal Infirmary, type 6/47/75 followed later by type 47/54/77 (1,000 x R.T.D.) in the gynaecological wards of the Western Infirmary. Type 53 was the predominant strain in infections in Ballochmyle Hospital, Ayrshire, for a period during the first survey and this accounts
for its higher incidence in table IV than in table V. In the same way the higher percentage frequency of type 77 in table V is due to its emergence as a minor epidemic strain in the surgical wards of the Western Infirmary in the last 2 months of the survey. During the period of the first survey, the Royal Maternity Hospital suffered an epidemic due to type 80, which necessitated its closure and reopening after disinfection. Before the epidemic specimens had not been taken routinely from every infection there so that not all of the infecting strains were available for phage typing and for several weeks after the epidemic the infecting strains belonged to a wide variety of phage types and it appeared that no staphylococcal pool had yet become established. Later, maternity hospital endemic staphylococci emerged once more but showed little tendency to become predominant and very few became minor epidemic strains. Two things may have contributed to this: firstly the stricter policy for the prevention of sepsis which was introduced after the epidemic and secondly, the nursing of the infants beside their mothers (instead of in a crowded infant nursery) in the Ross Hospital, Paisley and, more recently in 2 of the 3 units
in the Royal Maternity Hospital. One interesting endemic strain, however, became a minor epidemic strain during the second survey. This was type B/C which in a period of 6 months caused most of the infant infections in the A unit of the Royal Maternity Hospital. This minor epidemic was of particular interest because A unit was the only unit in the hospital where the infants still spent much of their time in a communal nursery.

Tables IV and V show that most of the common endemic staphylococci had a similar frequency distribution in both surveys. Endemic strains with a high incidence in surgical and general units were types 6/47/53/54 and 77, and their related patterns, and types 52A/79 and 52/52A/80. Similarly, endemic strains in maternity units were types 52A/79, 52/52A/80, 29 and the related patterns of types 6/7/47/53/54/75 and 77. Other common maternity hospital strains were type 29/7 and type 29/52/79/80 which, although not listed as individual phage types in tables IV and V, are included in "mixed groups" and "other group I" respectively. A few strains appeared to cause disease readily in any kind of patient — for example, types 52/52A/80, 52A/79 and the related patterns of type 77. Type 52/52A/80,
however, is an unusual strain which merits further discussion.

Rountree and Beard (1958) noted a close epidemiological association between type 52/52A/80 and type 80 and this association has been observed in the type-80 epidemics investigated here. Type 52/52A/80 does not seem to possess the same epidemic properties as type 80 but it has appeared as a frequent cause of infection during and following epidemics due to type 80. There is evidence that in the laboratory type 80 can be transformed into type 52/52A/80 by the action of phage isolated from lysogenic cultures of type 52/52A/80 (Asheshov and Rippon, 1959; Rountree, 1959) and it is possible that this also happens in the field.

Although any coagulase-positive staphylococcus must be regarded as capable of producing disease the results of the surveys shown in tables IV and V suggest that different strains vary in their ability to do so. Hospital strains, moreover, show a tendency to flourish more readily in either surgical and general wards or in maternity units. This difference is more marked if the staphylococci are considered in relation to the phage groups to which they belong and has been commented upon by Williams (1959) who states that it is more marked when only
strains which have caused outbreaks of infection are considered. It is, of course, the epidemic strains which are of particular interest and importance in the investigation of outbreaks of hospital infection and several of these strains have been described above. Many of these epidemic strains gave rise to small scale outbreaks of infection which occasioned no real concern to the clinicians concerned but a few caused serious and sometimes extensive outbreaks. Of these outbreaks, four which were of unusual interest and were investigated fully will now be described in detail.

As the outbreaks in which they became known.

**Clinical Record**

A course of postoperative sepsis was instituted in the incidence and course of infection in the general ward concerned. The epidemic was in fact very low, not only when within 3 days four patients developed
An Outbreak of Staphylococcal Postoperative Wound Infection in the Western Infirmary, Glasgow

(Published, jointly, in The Lancet, 1957, ii, 863)

This outbreak of postoperative sepsis occurred in a surgical unit where Dr. Sheila McDonald and I were carrying out a survey of wound infection. The infections in the outbreak were serious and 2 patients died. Nevertheless the events which this investigation revealed might have passed without notice if details of every wound infection had not been recorded and if all staphylococci isolated in the unit had not been submitted for phage typing. To emphasise that it may be difficult to detect epidemics of wound infection and to find their source, the events are described in the sequence in which they became known.

Clinical Record

The survey of postoperative sepsis was instituted to determine the incidence and sources of infection in the general surgical unit concerned. The sepsis-rate was in fact very low, but it rose suddenly when within 8 days four patients developed postoperative infections. Case 1 had a superficial wound
infection which was present at the time of first dressing on the 7th postoperative day. Cases 2, 3, and 4 had deep wound abscesses which discharged on the 11th, 8th, and 15th postoperative days respectively. These three wounds had appeared satisfactory at the time of first dressing. The pus examined from these four infected cases yielded a pure growth of coagulase-positive staphylococci with a notably greyish-white pigmentation. This cultural character was important, because it led to the discovery of a hitherto unrecognised case in the outbreak (case 5).

A colleague who had examined postmortem specimens from case 5 (of fatal peritonitis) commented on the unusually white appearance of the coagulase-positive staphylococci isolated from the peritoneal pus. It was then discovered that the patient had recently had a gastroenterostomy in the unit whose outbreak we were investigating.

The postoperative course had been complicated by abdominal distension, vomiting, and pyrexia. On the 6th postoperative day the patient collapsed with peripheral circulatory failure and a foul-smelling fatty fluid was discharged from his wound. Despite administration of intravenous fluids and
erythromycin, he died later on the same day. At necropsy a collection of pus and clear yellow fluid was found in the peritoneal sac in the deep aspect of the surgical incision. There were also signs of generalised fibrinous peritonitis. There was no leakage from the anastomosis. Coagulase-positive staphylococci were obtained in pure culture from the peritoneal pus and isolated from the spleen and contents of jejunum and rectum. Histological sections showed clumps of gram-positive cocci in the exudate on the peritoneal surface of the intestine.

At this point it was realised that another patient (case 6) from the same unit had died a fortnight earlier in similar circumstances.

This patient developed severe abdominal distension after partial gastrectomy. His wound began to discharge, and coagulase-positive staphylococci, *Escherichia coli*, and enterococci were isolated from the pus. In spite of treatment his condition deteriorated; the possibility of a leak from the anastomosis was suspected, so he underwent a second operation. No leak was found, but there were signs of plastic peritonitis in the stages of resolution. No specimens were taken for bacteriological
examination. He died a few hours later, and necropsy confirmed the findings recorded at the second operation. Fortunately the staphylococci cultured from the wound of the first operation were available for further investigation.

In each case the site and character of the infection suggested that it had been acquired at operation, and review of the operating theatre lists showed that all the six infected patients had been operated on by surgeon A. In operations undertaken by surgeon A during a period of 15 days (table VI) only two of his eight patients escaped staphylococcal wound infection. Two cases with which he assisted during this period also, remained free from infection. It then came to light that surgeon A had been operating during this time with a small boil on his forearm. This lesion caused little pain or discomfort, and no systemic disturbance. It measured about $\frac{1}{2}$ in. in diameter, and was on the ulnar aspect of his right forearm above the glove but where the gown sleeve might become wet. While the boil was discharging, the surgeon had taken the precaution of covering it with a waterproof dressing. When seen 3 weeks later the lesion was crusted over and almost, though not completely, healed.

From a broth-
TABLE VI

Sequence of events in an outbreak of postoperative wound sepsis. The numbers assigned to the cases reveal the order in which they become known.

<table>
<thead>
<tr>
<th>Day no.</th>
<th>State of bail on Surgeon A's arm</th>
<th>Surgeon A's operating-lists</th>
<th>Postoperative course</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>First noticed by surgeon</td>
<td>None</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>Partial gastrectomy</td>
<td>Wound abscess; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cholecystectomy</td>
<td>Uneventful</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prostatectomy</td>
<td>Wound infection; coliforms</td>
</tr>
<tr>
<td>4</td>
<td>Discharged</td>
<td>Case 6</td>
<td>Patient died with peritonitis after operation</td>
</tr>
<tr>
<td>9</td>
<td>-</td>
<td>Second operation</td>
<td>Deep wound abscess; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td>10</td>
<td>-</td>
<td>Partial gastrectomy</td>
<td>Superficial wound infection; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td></td>
<td>Case 1</td>
<td>Polya - Billroth conversion</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>Case 4</td>
<td>Cholecystectomy</td>
<td>Deep wound abscess; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td>15</td>
<td>-</td>
<td>Gastroenterostomy</td>
<td>Patient died on 6th postoperative day with peritonitis; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td></td>
<td>Case 3</td>
<td>Polya - Billroth conversion</td>
<td>Deep wound abscess; Staph. aureus type 52A/79</td>
</tr>
<tr>
<td>18</td>
<td>-</td>
<td>Laporotomy</td>
<td>Uneventful</td>
</tr>
<tr>
<td>24</td>
<td>First swab taken from almost healed lesion; Staph. aureus type 52A/79 (scanty growth)</td>
<td>Partial gastrectomy</td>
<td>Uneventful</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cholecystectomy</td>
<td>Uneventful</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Removal of thyroid cyst</td>
<td>Uneventful</td>
</tr>
</tbody>
</table>
soaked swab rubbed over the area, a scanty growth of coagulase-positive staphylococci was obtained. A second swab taken 6 days afterwards yielded a very scanty growth of the organism but subsequent swabs were negative. Routine nasal swabs taken from surgeon A a month before the outbreak and twice during the investigation did not yield coagulase-positive staphylococci.

The point of interest is that the presence of the boil and its relation to the outbreak were discovered only because the recently instituted wound-infection survey revealed a sudden change in the previously low sepsis rate, and the phage typing disclosed an epidemic type of staphylococcus whose origin was investigated with a good deal of persistence.

The staphylococci isolated from each of the six patients and from surgeon A's boil all had the characteristic greyish-white colonial appearance already mentioned and were found to be the same phage type — namely, 52A/79. This strain had not been isolated from any wound in the unit in the previous 6 months.

Nasal swabs from surgeons and theatre nurses showed that seven of eleven carried coagulase-positive staphylococci. Phage typing showed these strains to be different from each other and from the
epidemic strain with one exception — namely, that one surgeon carried in his nose a strain of type 52A/79 which gave a well-marked golden pigment. This surgeon had no contact with any of the infected cases. While infected cases were still in the wards, one of the house-surgeons developed a boil on his neck. Several weeks later, when the incident was apparently ended, two patients developed superficial wound infections after first dressings. Type 52A/79 with greyish-white pigmentation was isolated from these infections. These three incidents, which were probably due to ward cross-infection, are the only evidence of subsequent spread of the epidemic strain in the unit. Three and a half months after the first operation the patient of case 1 developed a deep stitch abscess requiring operative treatment and, a year later, suffered a recrudescence of the same infection. This developed into a chronic wound sinus which was still discharging type 52A/79 in the pus 2 years after the first operation. It is clear that this patient, who had been regarded as the only case of superficial wound sepsis in the outbreak, also had a deep wound abscess. The patient of case 2 was readmitted for a repair of an incisional hernia 4 months after the original operation.
A wound abscess developed after this second operation, which was not carried out by surgeon A. A growth of staphylococci of type 52A/79 of greyish-white pigmentation was cultured from both of these lesions.

Discussion

In the present outbreak unusually severe infections developed in six patients after operations performed by a surgeon who had a boil on his forearm. Three patients had deep abscesses, necessitating a long stay in hospital, and two died. In one of the two fatal cases, the cause of death was staphylococcal peritonitis. The other patient died in similar circumstances, and though the evidence was incomplete, there can be little doubt that staphylococcal infection contributed to his death. It is interesting that this patient underwent operation before the boil discharged, and that the other patients became infected though the lesion was covered with an occlusive dressing.

The situation of the boil suggested that the route of infection may have been by direct spread of the organism through the surgeon's gown sleeves. Gown sleeves often become wet above
the margin of the gloves — particularly during abdominal operations. The possibility of inoculation through glove punctures cannot of course be ruled out since it is probable that the surgeon's hands were also contaminated with staphylococci. Nevertheless the first hypothesis seems more likely to be the correct one in this instance because only 2 of surgeon A's 8 cases escaped infection during a 15 day period.

The danger of a surgeon operating while suffering from a septic lesion was emphasised in The Medical Research Council Memorandum no. 6 (1941). No reports, however, could be found in the literature of any outbreaks with such an origin.

Coburn (1944), studying the epidemiology of Streptococcus pyogenes, came to the conclusion that when this organism was derived from subacute infections it might be more communicable than when it was derived from a healthy mucous membrane. Barber and Burston (1955) suggested that this might be true for coagulase-positive staphylococci. Investigating staphylococcal cross-infection in a maternity unit, they noted that, despite a high nasal-carriage-rate among patients and staff, infections were few and trivial until a nurse came on duty with a boil on her face.
Within a few days, one baby had acute mastitis — a much more serious infection than any seen previously during the study — and another had conjunctivitis. Gould (1957) found that the incidence of superficial staphylococcal infection is higher among members of hospital staff than among the general population. There is little doubt that surgeons occasionally operate while suffering from septic lesions, and outbreaks of this sort are probably more common than is realised. Because of the mildness of the associated systemic illness and also, perhaps, because the introduction of antibiotic therapy has given rise to a certain sense of complacency, few surgeons with skin infections are willing to forgo operating. This outbreak emphasises the extreme danger of this. Not only are lesions dangerous sources of infection while they are actually discharging, but they may give rise to infections before discharge and for many days thereafter.
d) **An Outbreak of Staphylococcal Wound Infection**

**in Law Hospital, Lanarkshire**

*(In press, jointly, Lancet)*

In May 1958 in the surgical unit of Law Hospital, there was an epidemic of postoperative wound infection which illustrated two important features: the first, the danger of a surgeon operating while suffering from septic lesions, and the second, the extent to which the unusually infective *Staphylococcus aureus*, phage type 80, may spread among the patients when it is introduced into a surgical unit.

**The Surgical Unit**

The epidemic began in a surgical unit of 6 wards containing 180 beds in all. The unit is staffed by 6 surgeons who carry out general surgical work including urology. The wards are single-storey buildings connected by long airy corridors which have windows and doors opening on to the surrounding hospital grounds. A newly built operating theatre serves the surgical as well as the gynaecological unit in the hospital. The theatre is well designed and equipped and is
ventilated by a positive pressure ventilation system.

The Start of the Epidemic

Before the epidemic wound sepsis in the unit was infrequent, but not recorded. No one of the 6 surgeons appeared to have a significantly higher incidence than any other. In 1958, six weeks before the epidemic, routine phage typing of all staphylococci isolated from infected surgical wounds had been instituted. This revealed that several different strains of staphylococci, most of which belonged to phage group III, were responsible for the infections. At that time there was no evidence of an epidemic strain.

During May 1958, however, one surgeon, Mr. B, noticed a sudden rise in his sepsis rate in that 9 of 51 or 18% of his cases developed wound infections. Since most of the infections were deep wound abscesses it was thought that the infections must have been acquired at operation. At this point the surgeon stated that since the end of April he had been suffering from a series of boils affecting both axillae and that he had operated upon all the infected cases during the course of these infections.
Phage typing showed that all the infected cases were due to type 80, and this same type was isolated from one of surgeon B's axillary boils in May. There was therefore little doubt that surgeon B had infected these cases from his axillary boils during operation.

Shortly after this outbreak it was realised that there had been a smaller but similar outbreak of wound infection among surgeon B's cases in March, when 5 of 45 of his patients developed wound infection. At the end of February surgeon B had gone off duty with a septic finger. The infected patients had been operated upon when he returned to work when the lesion was scabbed over. Although no phage typing was being done in March 1958, the staphylococci from these infections and from surgeon B's finger had the antibiotic sensitivity pattern of the epidemic type 80 strain (i.e. resistant to penicillin and the tetracyclines, sensitive to chloramphenicol and erythromycin, and variable in sensitivity to streptomycin.) Later, in June, type 80 was isolated from one of the patients operated on in March who had a chronic discharging wound. This evidence therefore suggested that the March outbreak was also caused by type 80, in this instance
derived from surgeon B's scabbed-over septic finger and that his axillary boils in May were due to the same staphylococcal type as this earlier infection. It is interesting that in the interval between episodes of sepsis, when he was in all probability still harbouring the organism, surgeon B did not transmit it to his patients.

**Secondary Spread of type 80 Infection in the Surgical Unit**

Shortly after the first small outbreak of infection in March phage typing was begun. This showed that there had not been any secondary spread of type 80 in the surgical wards at this time. A widespread epidemic of wound sepsis, however, followed the second appearance of the strain in surgeon B's cases in May and a further 47 cases of wound infection due to type 80 appeared during the succeeding 7 months. The first two secondary cases of wound infection were found in May after the outbreak in surgeon B's cases. They were apparently due to cross-infection since they followed operations performed by different surgeons. Similarly, in the secondary epidemic, wound infections were associated with all 6 surgeons in the unit. Although the infections appeared
among patients in each of the surgical wards, two wards were more severely affected than the other four.

Table VII records the monthly incidence of wound infections due to type 80 and the proportion of the surgical wounds at risk which became infected.

It may be seen that after the introduction of type 80 in surgeon B's cases in May the sepsis rate of the unit in June and July was 5% and 4% respectively. In August, when various measures had been instituted in an attempt to reduce the number of infections, it fell to a low level and remained low during the next 4 months (from September to December). No type 80 infections appeared in January, 1959. The course of the epidemic in the surgical unit therefore extended over a period of 8 months and although the number of infected cases in each month was not large the morbidity among these patients was considerable. The figure shows the monthly incidence of all staphylococcal wound infections in relation to the incidence of those due to type 80 and of those due to other strains of staphylococci. In the first 4 months type 80 was clearly predominant as a cause of wound infection; but as the epidemic
TABLE VII

Number of Operations per Month followed by Wound Infection due to Type 80

<table>
<thead>
<tr>
<th>Month</th>
<th>Total no. of surgical operations</th>
<th>No. of operations followed by type 80 infection</th>
<th>% of wounds at risk which became infected with type 80</th>
</tr>
</thead>
<tbody>
<tr>
<td>May</td>
<td>335</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td>June</td>
<td>301</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>July</td>
<td>245</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>August</td>
<td>262</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>September</td>
<td>306</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>October</td>
<td>301</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td>November</td>
<td>276</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>December</td>
<td>301</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

* The percentage of operation wounds in May becoming infected with type 80 was not calculated because all except 2 of the infections appeared in cases operated upon by surgeon B.
Figure. Monthly incidence of wound infections due to type 80 in relation to those due to other staphylococcal strains.
waned in the later months wound infections due to other strains of Staph. aureus began to appear more frequently.

Most of the infections in the epidemic were wound infections but type 80 also gave rise to other forms of septic lesion in 11 patients. Of 4 patients who had wound infections, 3 later suffered from boils, and one developed an abscess of the thigh. One patient whose wound was not infected also suffered from boils. Four patients had infections of the urinary tract: three became infected after being catheterised and the fourth, who developed the urinary infection pre-operatively, later suffered from wound sepsis due to the same organism. Type 80 was the cause of fatal parotitis and suppurative otitis media in one elderly patient and was isolated from the stools of another who had undergone prostatectomy and developed severe postoperative diarrhoea. Apart from surgeon B only one member of the hospital staff is known to have become infected with type 80 during the epidemic period. This was a nurse in the outpatient department who developed a septic finger.

Nine surgical patients who had infections due to type 80 died, but in 5 death was mainly due to other causes. Although
type 80 materially contributed to the deaths of the remaining 4 patients their general condition had previously been poor and it is probable that the supervention of any infections would have hastened death.

Infections due to type 52/52A/80

The staphylococcal strain type 52/52A/80 has been observed to be closely related to type 80 (Rountree and Beard 1958). During the present epidemic this strain was isolated from 4 surgical patients — from three who had wound infections and a fourth who had parotitis. A nurse working in one of the surgical wards also developed a septic finger due to the strain.

Type 80 in the Hospital Environment

In August, nasal swabs were taken from the entire staff of the surgical wards. This included surgeons, anaesthetists, nurses, orderlies and domestics. A total of 114 nasal swabs were collected and 7 nurses were found to be carrying the epidemic strain. Six of the nurses worked in the surgical wards but the seventh was a theatre sister. Further inquiries however showed that she could not have been responsible for the spread of the
epidemic strain because she had assisted at only 4 of the operations which were followed by sepsis. The rather low carriage rate (6%) of type 80 in the nursing staff in spite of ward contamination may have been due to the rotation of the nurses among other units. Three nurses were also found to carry the related strain type 52/52A/80 in their noses. Samples were taken from blankets and floor dust of the 2 wards which were most affected by the epidemic and yielded strains of type 80.

The Source of the Epidemic Wound Infections

The most striking feature of the type 80 wound infections in the secondary epidemic was that the majority were deep wound abscesses and therefore appeared to have been acquired during operation rather than in the wards postoperatively. There was no demonstrable source of infection in the operating theatre. Moreover, it was also used for gynaecological cases, among whom there had been no type 80 infections; therefore it was unlikely that the theatre itself, or any of the nursing staff there, could be responsible for the spread of type 80.

The possibility remained that the patients were
themselves carrying the epidemic strain from the wards into the theatre. In the present epidemic no investigations were undertaken to estimate the staphylococcal skin and nasal carriage in patients before operation and there was therefore no direct evidence that this was a common route of infection. Nevertheless there was evidence which suggested that this was the probable explanation for the theatre-acquired infections which were an important feature of the secondary epidemic. Firstly, type 80 was known to have contaminated the ward environment and as all the wards contained cases of sepsis due to it there were obvious sources of infection within them; secondly, the wards with most cases of sepsis continued to show more new infections than the others; and thirdly, no source of infection could be found within the operating theatre. We learnt by chance of another way in which ward contamination could be brought into theatre from the wards. It was discovered that clean linen was returned to the wards in the same basket as that which had delivered soiled linen to the laundry. A swab was taken from one of these baskets and yielded a type 80 staphylococcus. Patients were sent to theatre dressed in socks, and in a "clean" gown (from
the soiled linen basket) which during operations was folded up under the patients' arms. The gown, with its possible contamination, would therefore be placed only a few inches from the wound during abdominal operations. The arrangements for delivery of clean laundry were altered as soon as this state of affairs was discovered and now linen is sent to and from the laundry in washable canvas hampers.

Measures taken for Control of the Epidemic

The first step was to rid the affected surgeon of his recurrent infections. This proved to be extremely difficult. As he was a nasal carrier he began intra-nasal treatment with neomycin/bacitracin cream and was advised to wash with hexachlorophene soap both in hospital and at home. After a short course of the intra-nasal antibiotic, his nasal swabs became negative and he resumed operating. His boils, however, recurred while he was away on holiday and on returning he was once again unable to resume operating because of a boil on his finger due to type 80. Further treatment with the intra-nasal antibiotic cream and the antiseptic soap was then carried out:
in addition his clothing was washed with a quaternary ammonium disinfectant and all outer garments were dry cleaned (it was hoped that the steam pressing would destroy any staphylococci contaminating them). Despite these measures type 80 was isolated from surgeon B’s hands three weeks later although his boils were then healed. Had one recurrence not coincided with his holiday he would have been absent from duty for 3 months out of 6 with recurrent boils. This history illustrates how difficult it is to be certain of curing recurrent sepsis in any individual and also the enormous inconvenience which may be caused to a surgeon under these circumstances.

When it was realised that type 80 was causing a general epidemic a very strict aseptic technique was begun in the wards to avoid cross-infection during dressing rounds. All nasal carriers of type 80 among the nursing staff of the surgical unit were sent off duty and treated with neomycin/bacitracin cream. Later, at the end of August, the surgeons were asked to use a hand rinse of chlorhexidene in alcohol just before donning surgical gloves. The efficacy of the hand rinse was demonstrated on one occasion when we asked surgeon B to scrub up and don
surgical gloves for 2 hours — once with and once without the chlorhexidine hand rinse. Type 80 was isolated from his hands and the inside of his gloves when he omitted the hand rinse but no Staph. aureus was isolated when it was used.

These measures were followed by a sharp fall in the incidence of type-80 infections during and after August.

Discussion

This epidemic illustrates again the high incidence of postoperative sepsis which may result when a surgeon operates while suffering from a septic lesion. In the outbreak previously described a surgeon who operated while suffering from a boil on his forearm caused a small outbreak of severe wound infection involving 6 patients. In that instance the epidemic strain was type 52A/79 and no widespread epidemic followed in the surgical unit concerned. In the present outbreak the infecting strain was type 80, which, after its appearance in the surgeon's own cases, became the predominant cause of postoperative sepsis in the unit. There was evidence that a similar smaller outbreak, probably also due to type 80, had appeared 2 months earlier than
the major outbreak yet in this earlier outbreak there was no sign of secondary spread of the organism. It is noteworthy that surgeon B did not infect his cases when he was carrying the epidemic strain but was not suffering from sepsis due to it. This provides further suggestive evidence of the possible increase of danger from organisms derived from active lesions as distinct from those carried on the healthy skin or nasopharynx.

The site of the surgeon's lesions in the present outbreak was interesting. Septic lesions on hands or forearms are an obvious risk, but the danger of spread from a lesion in the axilla may not be so apparent. It is well known that the skin surrounding discharging lesions is heavily contaminated with the infecting organism. Staphylococci from the axillae may spread down the arm inside the sleeves of surgical gowns or everyday clothing in sweat, air currents or by other means to contaminate the forearm and hands. Spread from there to the patients' wounds may have been through glove punctures or wet gown sleeves or possibly by dissemination into the air. Colebrook and Ross (1947) found that *Streptococcus pyogenes* from a scabbed lesion on a surgeon's arm was disseminated into the air and this may
represent an important route for spread of organisms from a staphylococcal lesion also.

The history of recurrent sepsis in this particular surgeon emphasises the unsatisfactory state of treatment of this condition and also the inconvenience to both the surgeon and the hospital deprived of his services during the episodes of sepsis. Nevertheless it is clear that the danger of infecting wounds at operation is so great that no surgeon should ever operate while suffering from sepsis of hands or arms. Even in the absence of direct evidence boils anywhere on the body must be regarded as a surgical hazard of unknown magnitude and a surgeon who is suffering from this form of sepsis should first ascertain that his hands are not contaminated before operating and make use of hand rinses such as have been described.

Type 80 has been described as a strain which causes more extensive epidemics than any other staphylococcus and which often produces a high incidence of skin lesions (Williams 1959). In the present epidemic, however, the incidence of skin sepsis was relatively low and most of the infections were wound infections. Robertson (1958) has recommended that separate
containers be provided for clean and soiled linen and in the present epidemic the isolation of type 80 from a ward laundry basket, which was used for the transport of both clean and soiled linen, emphasises the importance of this. This obviously dangerous and indefensible practice was being carried out at a time when special precautions were being taken to avoid the further spread of infection and illustrates the necessity of supervising carefully all details of ward hygiene.
e) Two Outbreaks of Staphylococcal Infection in the Southern General Hospital, Glasgow

This report illustrates the difficulty in tracing the epidemiology of staphylococcal infection in retrospect and without full access to all the relevant information. Nevertheless there were some interesting features about the infections in this hospital which were revealed by phage typing and the other bacteriological investigations which were carried out.

In October 1957 I was asked to help in an investigation into postoperative wound infection in the surgical unit of the hospital. The senior surgeon in the unit, surgeon C, was particularly worried because a number of patients who had undergone cardiac and thyroidectomy operations had developed postoperative infection and because 2 patients had died of severe staphylococcal infection after cardiac operations. It was noticed that sepsis appeared more commonly after operations performed in the newly built and theoretically plenum-ventilated operating theatre than in those carried out in the old theatre which had no controlled system of ventilation.

Fortunately all strains of Staphylococcus aureus
isolated in the hospital in the preceding year had been retained and I suggested that all those isolated from infections in the surgical unit be submitted for phage typing. Phage typing was also carried out on strains isolated from new infections as they appeared.

Other investigations were carried out — for example:

(1) Nasal swabs were taken on several occasions from all surgeons and theatre nursing staff.

(2) Skin and glove swabs were taken from some of the surgeons.

(3) Cultures were made from the inner surface of the masks of the senior surgeon and theatre sister.

(4) Numerous sites in the theatre were sampled for contamination with *Staph. aureus*.

(5) Some tests were performed to estimate the bacterial content of the theatre air.

The last 2 investigations were carried out in the new theatre where, in fact, most of the operations were performed.
Results

It soon became apparent that two epidemic strains of staphylococci were responsible for most of the surgical infections. These strains were (1) Type 3Bw/55vw/71 (Normally very weak phage reactions are not reported: however the minor reaction of this strain with phage 55 was so constant and so characteristic, that I decided to include it in the phage type report) (2) Type 80.

The first strain, which belongs to phage group II, had appeared sporadically but persistently in wound infections in the preceding 8 months. The second strain had caused a few sporadic infections during the preceding 7 months but had shown a dramatic rise in incidence towards the end of 1957. Unlike the first strain it appeared in infections in patients throughout the hospital.

I. Epidemiology of Infections due to Type 3Bw/55vw/71

Clinical Record

Phage group II staphylococci are a rare cause of wound infection and type 3Bw/55vw/71 is itself a very uncommon group II strain. It was therefore surprising to find this strain
appearing so frequently in surgical infections. It had been
discovered, however, that surgeon C was a persistent nasal carrier
of this particular staphylococcus and it seemed likely that he was
in some way transferring his nasal strain to cases during
operations.

Table VIII shows the monthly incidence of cases from
which type 3Bw/55vw/71 was isolated and also the surgeons who
operated upon them. 2 patients died of staphylococcal infection
following cardiac operations and in each, 2 strains of staphylococci
were responsible for the infections.

Case 1 was that of a patient who died in October 1956
after valvotomy and from whom 2 strains of staphylococci were
isolated. One was a group III strain — the other a group II
strain which at the time of typing in 1956 differed in some of
its phage reactions from the very characteristic and constant
reactions of the strain which was epidemic in 1957. It is
impossible to say whether this 1957 strain was a variant of the
original 1956 one or whether it was a different strain altogether.
The significant point is that, in 1956, surgeon C carried in his
nose a staphylococcus identical with that which was isolated from
## Table VIII

### Monthly Incidence of Infections due to Type 3Bw/55vw/71

<table>
<thead>
<tr>
<th>Month</th>
<th>Case no.</th>
<th>Operation</th>
<th>Surgeon</th>
<th>Sites from which Type 3Bw/55vw/71 was isolated</th>
</tr>
</thead>
<tbody>
<tr>
<td>October 1956</td>
<td>1</td>
<td>Valvotomy</td>
<td>C</td>
<td>Type 3B/3C/55/71 - blood culture (life) heart blood, spleen (post mortem). Also isolated = type 6/4/75 sputum, pleural fluid (life) pleural fluid, pericardium and lungs (post mortem)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>February 1957</td>
<td>2</td>
<td>Oesophagectomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td>April 1957</td>
<td>3</td>
<td>Thyroidectomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>Thyroidectomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td>May 1957</td>
<td>5</td>
<td>Valvotomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>Appendixectomy</td>
<td>D</td>
<td>Wound infection</td>
</tr>
<tr>
<td>June 1957</td>
<td>7</td>
<td>Unknown</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td>August 1957</td>
<td>8</td>
<td>Nephrectomy</td>
<td>E</td>
<td>Wound infection</td>
</tr>
<tr>
<td>September 1957</td>
<td>9</td>
<td>Valvotomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td>October 1957</td>
<td>10</td>
<td>Thyroidectomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>Operation for relief of coarctation of aorta</td>
<td>C</td>
<td>Wound infection (life) blood culture, pericardium, pleural cavity, spleen and lungs (post mortem). Also isolated = type 53w/77+ from blood culture, pleural fluid (life), pericardium and pleural cavity (post mortem)</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>Valvotomy</td>
<td>C</td>
<td>Blood culture, nasal and throat swabs. Also isolated = type 71+ (1000 R.T.D.) from throat swab, blood culture, wound infection.</td>
</tr>
<tr>
<td>November 1957</td>
<td>13</td>
<td>Repair diaphragmatic hernia</td>
<td>C</td>
<td>Pleural cavity; later, type-80 also isolated.</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>Splenectomy</td>
<td>C</td>
<td>Wound infection</td>
</tr>
</tbody>
</table>
this patient. Two strains of staphylococci were isolated from each of 2 other patients who had undergone cardiac operations (cases 11 and 12). In case 12 the second strain was similar to but not identical with type 3Bw/55vw/71, and may have been a rather phage-resistant variant of it. In case 11, like case 1, the second staphylococcus isolated belonged to phage group III, but there is no evidence to indicate what the sources of these group III organisms were.

Table IX records the staphylococci isolated from surgeon C's nasal swabs and also from cultures of the inner surface of his mask and gloves. Numerous attempts were made to isolate staphylococci from surgeon C's gloves and skin, but the organism was found only on one occasion in his gloves.

Tables VIII and IX show that the majority (12 of 14) of the infections due to this unusual strain were in cases operated upon by surgeon C and that he carried this strain in his nose over a long period of time. Although he acquired the second epidemic strain, type 80, in his nose in November 1957, he continued to harbour type 3Bw/55vw/71 also — at least until 12th December 1957. No other member of the medical or nursing staff carried
**TABLE IX**

Staphylococcal Strains isolated from Surgeon C's Nose, Mask and Gloves

<table>
<thead>
<tr>
<th>Date</th>
<th>Site</th>
<th>Strain isolated</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.12.56</td>
<td>nose</td>
<td>type 3B/3C/55/71</td>
</tr>
<tr>
<td>29.4.57</td>
<td>nose</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>24.10.57</td>
<td>nose</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>31.10.57</td>
<td>nose</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>4.11.57</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td>11.11.57</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td>14.11.57</td>
<td>nose</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>18.11.57</td>
<td>inner mask</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>28.11.57</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td>9.12.57</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td></td>
<td>inner mask</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>12.12.57</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td></td>
<td>inner mask</td>
<td>type 3Bw/55vw/71</td>
</tr>
<tr>
<td>16.12.57</td>
<td>gloves</td>
<td>type 80</td>
</tr>
<tr>
<td>9.1.58</td>
<td>nose</td>
<td>type 80</td>
</tr>
<tr>
<td></td>
<td>inner mask</td>
<td>type 42E/53/75 (1,000 x R.T.D.)</td>
</tr>
</tbody>
</table>
this strain except for one house surgeon from whom it was isolated on one occasion only in October 1957.

Discussion

There are two principal routes by which this surgeon's nasal strain may have been communicated to wounds at operation.

1. It could have been transmitted from the skin of the hands via glove punctures.

2. It could have spread by the air from mask, skin or clothing.

It is not known which of these routes was the more important. The sporadic nature of the infections suggests that the organisms passed only intermittently from surgeon C to the wound. Glove punctures are common and in most theatres about 30% of gloves have to be discarded after use for this reason. Despite repeated tests of surgeon C's skin and gloves his nasal strain was isolated from his gloves on only one occasion, in December 1957, when he was carrying type 80 in his nose. This shows, however, that the skin was occasionally contaminated with release of the organisms into the inside of the gloves. This is
in keeping with the sporadic nature of infections and with the fact that the infections tended to follow lengthy operations when glove punctures might be more frequent.

It is striking that positive cultures were obtained on several occasions from the inner surface of the mask. The masks used in the theatre did not have an impermeable layer of cellophane inserted between their folds, and it is therefore quite probable that contamination of the outer mask surface took place. Reference has already been made to the fact that there was an impression that infection was particularly troublesome in operations carried out in the new theatre. This theatre is modern in design. In theory it has a positive pressure system of controlled ventilation. Unfortunately this did not function as intended as a plenum ventilation system — because of the presence of a large hatch in one of the walls and because of inadequate thrust of the inlet fans — and air was drawn into the theatre through the doors. Air counts estimated on a slit sampler showed that the bacterial content of theatre air was higher than the desirable level but did not show a dangerous degree of bacterial contamination. The louvres of the input
vents in the ceiling, however, directed air straight down on to the wound from above the surgeon's head and it is possible that in this way staphylococcus-carrying particles could have been dislodged from the surface of the surgeon's mask and carried into the wound.

In the 2 cases not operated upon by surgeon C (cases 6 and 8), there was no evidence to indicate the source of the infection and in the absence of known contact with either surgeon C or a patient infected with the organism, this remains unknown.

II. Epidemiology of Infections due to Type 80

Clinical Record

During the 3 months from 1st November 1957 to 1st February 1958, there was an epidemic of infections due to type 80 (which in this instance was resistant to penicillin and streptomycin but sensitive to the other usual antibiotics) in the surgical unit. Type 80 had first appeared several months previously as a cause of sporadic surgical infection but it was not until November 1957 that it became the predominant cause of infection. Many of the patients developed septic lesions other
than wound infections; these included boils, carbuncles, infected ulcers, pustules, two cases of pneumonia and one of abscess. The monthly incidence of these lesions and of wound infections is shown in table X.

Table X shows the sudden increase of type 80 infections in November 1957. During the following 3 months there were 20 cases of wound infections and 15 cases of other septic lesions. The wound infections were not confined to the cases of any one surgeon or to patients in any one ward. One male ward, however, was more severely affected than the others. During the epidemic period type 80 showed a clear predominance as the infecting strain in the unit and was responsible for 62.5% of all wound infections.

Sepsis due to type 80 was not confined to the surgical unit, but affected patients throughout the hospital. Information is incomplete about these infections in other parts of the hospital but it is known that at least 12 patients developed septic lesions due to type 80.

Several members of the medical and nursing staff also developed infections caused by the epidemic strain. These
### TABLE X

**Monthly Incidence of Infections**

due to Type 80 in Surgical Patients

<table>
<thead>
<tr>
<th>Date</th>
<th>Wound Infections</th>
<th>Other Septic Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1957</td>
<td></td>
<td></td>
</tr>
<tr>
<td>March</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>April</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>May</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>June</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>July</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>August</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>September</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>October</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>November</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>December</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>1958</td>
<td></td>
<td></td>
</tr>
<tr>
<td>January</td>
<td>9</td>
<td>5</td>
</tr>
</tbody>
</table>

(2 in patients with infected wounds due to type 80)

Total = 26

Total = 21
included two surgeons, who had boils on the finger and wrist respectively, and a surgical ward nurse who had a boil of the forearm. Two nurses in other departments suffered from septic fingers and the operating theatre orderly, who had been discovered to be a skin and nasal carrier of type 80 in October 1957, later developed recurrent boils of the face and neck. The epidemic strain also colonised the noses of several members of staff and patients. A close watch had been kept on staphylococcal nasal carriage in the theatre staff from the beginning of the investigation. This showed that the theatre orderly and surgeon C were the only persistent nasal carriers of type 80. Surgeon C first acquired type 80 in November and continued to carry it during the following 2 months. Three other members of the theatre staff were temporary carriers; these were the theatre sister and one of the surgeons who, on different occasions, each had a single nasal swab positive for type 80, and another surgeon from whose gloves the strain was once isolated although he was never a nasal carrier. Nasal carriage among the staff and patients in surgical wards was never investigated on a large scale. However nasal swabs taken in February from nurses and patients in the
most affected ward showed that 3 of 14 nurses and 5 of 41 patients carried the epidemic strain.

**Discussion**

It was not possible in this outbreak to obtain enough clinical details about the wound infections to determine whether they had been acquired in the theatre or ward. Nevertheless it was obvious from the number of patients developing septic lesions other than wound infection that ward cross-infection was on an extensive scale. That some ward nurses and patients acquired the strain in their noses is further proof that type 80 was spreading in the wards. Patients with discharging lesions are likely to be more dangerous sources of infection than nasal carriers, and, since there were no isolation facilities for infected patients, this doubtless contributed to further spread of the epidemic strain in the environment of the wards. Many of the wound infections may have been acquired in this way — either through contamination during wound dressing or by colonisation of the patient prior to operation.

The members of the staff who developed septic lesions
due to type 80, with one exception, went off duty until the lesions had healed and were therefore unlikely to have been responsible for any of the infections. The exception was the theatre orderly who was a skin and nasal carrier of type 80 and who was discovered in December to be suffering from recurrent boils of the face and neck. It was realised that he might be a dangerous source of infection in the operating theatre. His duties included the loading and unloading of dressing drums from the steriliser and he could have contaminated the contents of the drum when handling them to close the vents after sterilisation. There was no evidence that type 80 had been spread widely in the environment of the operating theatre. Swabs taken from numerous sites in the theatre, and settling plates exposed during operations, showed that staphylococcal contamination was minimal; and type 80 was isolated from a sample of theatre dust on one occasion only.

It is unlikely in the type-80 outbreak that surgeon C was an important source of infection. Although he may possibly have transmitted the epidemic strain to a few of his patients type-80 wound infection frequently followed operations performed by the other surgeons on the unit.
In this outbreak there were several possible sources of infection in both wards and theatre. Despite the considerable amount of ward cross-infection it was impossible to detect the major source or sources of infection and to trace the spread of type 80 in the unit concerned. This was partly because a great deal of the work was carried out in the absence of much relevant and necessary information — particularly with regard to conditions in the surgical unit. In this case the phage typing disclosed possible patterns of the spread of infection; but the full elucidation of the possibilities remains, as always, a matter requiring diligent inquiry by those in contact with the epidemic. Without this activity, phage typing of the many staphylococci submitted is tantalisingly suggestive but infuriatingly inconclusive. Such was the fate of this particular effort.
f) An Outbreak of Staphylococcal Infection in The Glasgow Royal Maternity Hospital

(Published, jointly, in The Lancet, 1958, ii, 1081)

This outbreak was due to type 80 and was characterised by a sudden and dramatic increase in the severity of infections among the babies. With the co-operation of Dr. T.S. Wilson of the Public Health Department, Glasgow, a follow-up survey was conducted at the homes to which the babies and mothers were discharged. This showed the extent of infection in the babies which was undetected at the time of their discharge from hospital.

The Hospital

The epidemic broke out in a maternity hospital with 120 beds divided into 3 separate units. Babies normally stay beside their mother's beds in the post-natal wards but are taken to the nurseries, which are attached to each unit, for bathing or changing or if they are noisy.

The Sick Nursery

The hospital has a sick nursery which is run by paediatricians, and to which ill or premature babies from all 3
units are admitted. Both before and during the epidemic there appeared to be more infections in the sick nursery than in the babies being nursed in the units. This impression may have been due, to some extent at any rate, to the routine bacteriological investigations which are carried out on even the mildest infections in the sick nursery. For this reason, and also because of the better case records kept there, the infections in the sick nursery form the substance of the account of the epidemic in the hospital.

**Staphylococcal Infections before the Epidemic**

Mild infections due to *Staphylococcus aureus* were common in the sick nursery before the epidemic. It was fortunate that because the staphylococci isolated in the 5 months before the outbreak had been phage typed, the strains which had been causing these infections were known. In practice, only about 85% of these strains were actually submitted for typing but failure to submit all strains was due to familiar human errors of one kind or another and no selection was involved.

Phage typing showed that a variety of types had caused
these infections and although some strains were isolated from more than one infection none was really predominant. Type 80 was not isolated in the hospital during this period.

The Epidemic

Type 80 was first isolated on 19th November 1957 from a submandibular abscess in a baby transferred to the sick nursery from one of the units. Thereafter until the hospital closed 6 weeks later, on the 4th January 1958, type 80 caused 14 of the 18 infections appearing in the sick nursery. During the epidemic period the incidence of all staphylococcal infections in the babies was 11%; the figure for infection due to type 80 was 8%.

Severity of Infections due to type 80

The most noteworthy feature of the epidemic was the high case incidence of serious infections. In table XI the infections during the epidemic period have been classified according to severity.

"Minor" infections include skin pustules, conjunctivitis, inflamed umbilicus, and mouth sores. "Serious" infections include abscesses, pneumonia and 1 case of osteitis. Nine of
## TABLE XI

Clinical Assessment of Infections in Sick Nursery During the Epidemic Period (November 19, 1957, to January 4, 1958)

<table>
<thead>
<tr>
<th>Minor</th>
<th>Serious</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 80</td>
<td>Type 80</td>
</tr>
<tr>
<td>Other strains</td>
<td>Other strains</td>
</tr>
<tr>
<td>5</td>
<td>9 (5 fatal)</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
</tr>
</tbody>
</table>

*2 babies are included who had minor infections due to type 80 and subsequently developed serious infections, from which the organisms were not available for typing.*
the 18 infections in the epidemic were serious infections; and all the serious infections were due to type 80. This high incidence of serious infection is especially significant since there had been very few such cases in the 5 months preceding the epidemic. Five of the 9 babies who had serious infections died.

Deaths associated with the Epidemic

Eight deaths in all were associated with the epidemic. Complete bacteriological investigations were not possible in every case. The first 6 cases had been nursed in the sick nursery; cases 7 and 8 were nursed in the units.

Case 1. A male child who had severe cerebral birth injury died on the 7th day of life, and type 80 was isolated from a blood-culture taken just after death. Postmortem examination showed a tentorial tear, cerebral haemorrhage, enteritis, and bronchopneumonia.

Case 2. A male child with cerebral contusion died on the second day. Postmortem examination revealed tentorial bruising and right lower lobe pneumonia. Type 80 was isolated from a post-mortem specimen of lung.
Case 3. A premature female baby had a skin pustule on the 4th day from which type 80 was isolated. A scapular abscess developed but she made an apparent recovery and was discharged home on the 10th day. She died in another hospital 7 weeks after birth from bronchopneumonia; type 80 was isolated from the lungs at postmortem examination.

Case 4. A male baby developed a skin pustule due to type 80 on the 6th day and subsequently paronychia and osteitis. He died on the 11th day; permission for postmortem examination was refused.

Case 5. A premature female child developed conjunctivitis due to type 80 on the 5th day. She was transferred to another hospital on the 16th day and died on the 18th day. At postmortem examination death appeared to be due to enteritis and pneumonia. Type 80 was isolated in pure culture from the pleural fluid.

Case 6. A premature female baby developed septic spots on the 6th day from which Staph. aureus was isolated but not phage typed. She died on the 7th day and postmortem examination showed right upper lobe pneumonia. No bacteriological cultures were made but
histological sections of lung showed clusters of gram-positive cocci.

**Case 7.** A female child developed a skin pustule due to type 80 on the 9th day. She was transferred to another hospital on the 12th day with an axillary abscess due to type 80. After an apparent recovery she was discharged home but died 11 weeks after birth in a different hospital. Postmortem examination showed bronchopneumonia and type 80 was isolated from the lungs.

**Case 8.** A female baby suffered from septic spots on the skin but was discharged from hospital, apparently well, on the 8th day. She died a week later in another hospital from multiple abscesses and bronchopneumonia from which *Staph. aureus* type 29/7 (1,000 x R.T.D.) was isolated. This strain had caused a few sporadic infections in the hospital before the epidemic and was therefore probably acquired in the hospital.

**Nasal Carriage of Staph. aureus**

**Staff.**

When the hospital was closed, nasal swabs were taken from 475 members of the medical, nursing, administrative and
domestic staffs. Thirty eight per cent were found to carry **Staph. aureus** in their noses but only 0.6% (3 persons) harboured the epidemic strain. Of these 3, one was a maid who did not work in the wards and 2 were nurses. Both nurses had suffered from boils during the epidemic but had gone off duty when the boils appeared. It was clear from their duties that they were unlikely to have been responsible for the general spread of the epidemic. It was interesting to find that many nurses were nasal carriers of the strains of staphylococci which had caused infections before the epidemic started.

**Babies.**

Unfortunately nasal carriage of **Staph. aureus** in the babies was not investigated on a large scale. In the 5th week of the epidemic nasal swabs were taken from 16 healthy babies in the units. **Staph. aureus** was isolated from the nose of 13 babies and 3 were found to carry type 80. In relation to the clear predominance of the epidemic strain in infant infections, this was rather a low rate of nasal carriage in the infants.
Staphylococcal Contamination in Wards and Nurseries

When the hospital was closed numerous sites were sampled for Staph. aureus: these included dust from floors, curtains, screens, blankets, furniture, and equipment. Staph. aureus was isolated from many of the sites tested but type 80 was found once only — in a communal blanket used during changes of infants' clothing in one of the unit nurseries. Several of the strains which had caused sporadic infections before the epidemic were isolated from sites in the nurseries. The failure to isolate the epidemic strain, which had been so clearly predominant in infections in the sick nursery, was unexpected.

Infections after Discharge Home

Babies.

While the hospital was closed, notifications were made by health visitors of the public health department of infections developing shortly after discharge from the hospital in babies born in the last two weeks of the epidemic period. In this way, it was discovered that 21 of 99 babies had developed infections at home. Nearly all these infected babies had been nursed in
the units in the hospital. With the exception of 1 case of breast abscess all the infections were mild. Eighteen of these babies were visited at home about one month after their discharge from hospital and swabs were taken from their nose, umbilicus and lesion, and from the mother's nose. Swabs were taken from the lesions of a further 2 babies although nasal and umbilical swabs were not obtained.

The strains of staphylococci which were isolated from lesions in this investigation are shown in table XII. Sixteen lesions yielded cultures of Staph. aureus but only 4 were due to type 80. This is in striking contrast to the distinct predominance of type 80 in infections in the hospital, but is in accord with the type 80 nasal-carriage rate found in infants in the hospital. Strains other than type 80 which were isolated from these lesions belonged to a variety of phage types; but 10 of the 12 strains were penicillin-resistant indicating that they, too, were probably acquired in hospital.

Nasal Carriage of Staph. aureus in Babies and Mothers

One month after discharge.

The results from the nasal swabs taken from babies and
### TABLE XII

**Staphylococci Isolated from Infections in Babies**

**One Month After Discharge from Hospital**

<table>
<thead>
<tr>
<th>No. of lesions swabbed</th>
<th>No. of lesions from which Staph. aureus was isolated</th>
<th>Other strains</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Type 80</td>
</tr>
<tr>
<td>20</td>
<td>16</td>
<td>4</td>
</tr>
</tbody>
</table>
mothers in the survey are shown in table XIII. Seventeen of the 18 babies and 10 of the 16 mothers from whom nasal swabs were obtained were staphylococcal nasal carriers. Four babies harboured type 80; these were the same 4 babies who had infections due to type 80. Three mothers carried type 80 and in each case their baby was infected with the same strain.

Four months after discharge

Four months after the epidemic all babies were visited who were known either to have been infected with or to have been nasal carriers of the epidemic strain. In this second survey nasal swabs were again taken from mothers and babies and any history of infection in either was noted. The results are shown in table XIV.

The number of staphylococcal nasal carriers among both babies and mothers was much lower than in the previous survey one month after discharge. Thus 7 of 16 of the babies and 6 of 16 of the mothers were staphylococcal nasal carriers. Two babies and 3 mothers harboured type 80. Six babies and 4 mothers had a history of infection which clinically would be regarded as
TABLE XIII

Nasal Carriage of *Staph. aureus* in Infected Babies and their Mothers one Month after Discharge from Hospital

<table>
<thead>
<tr>
<th>No. visited</th>
<th>No. of staphylococcal nasal carriers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
</tr>
<tr>
<td><strong>Babies:</strong></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>17</td>
</tr>
<tr>
<td><strong>Mothers:</strong></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>10</td>
</tr>
</tbody>
</table>
TABLE XIV

Nasal Carriage of Staph. aureus in Babies Colonised or Infected by Type 80 and their Mothers Four Months after Discharge from Hospital

<table>
<thead>
<tr>
<th>No. visited</th>
<th>No. of staphylococcal nasal carriers</th>
<th>No. with history of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Type 80</td>
</tr>
<tr>
<td><strong>Babies:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td><strong>Mothers:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>
staphylococcal but no bacteriological investigations had been carried out on the lesions.

**Mothers**

No systematic attempt was made to investigate infection in the mothers. 5 mothers were discovered by chance to have developed infections due to the epidemic strain, type 80. In 3 the infections were breast abscesses; in one impetigo and abscess of the hand; and in the 5th recurrent boils of the skin of the chest wall. This last case was discovered in the survey of infant infection carried out one month after discharge. In this survey another mother was found to have similar lesions due to a different strain of Staph. aureus which was also the cause of the baby's lesion.

The 4 mothers with a history of infection in the survey 4 months after discharge included the mother mentioned above who had boils of the skin of the chest wall due to type 80. She still carried type 80 in her nose and in fact the boils had persisted on her chest and neck until within 2 weeks of the later survey. The other infections discovered in this survey were
2 cases of breast abscesses, and one case of stye of the eye. No bacteriological investigations had been carried out in these cases.

Re-opening of the Hospital

On the 4th January 1958, the hospital was closed to new admissions and by 10th January all patients had been transferred from it. The whole building was then thoroughly disinfected; all wards, duty rooms, labour wards and operating theatres were sprayed with 10% liquid formaldehyde and after a few days were washed down with water to give a second fumigation. All the equipment in operating theatres and labour wards had been treated with carbolic-acid solution before the formaldehyde fumigation. The blankets, bed linen, clothing, curtains, and screens, were sterilised with steam under pressure. After 18 days the hospital re-opened (22nd January). No infections due to type 80 appeared in the hospital during the six months following its re-opening. Staphylococcal infections have been fairly common but they have almost all been mild; staphylococci of many phage types have been isolated from them — no one strain being predominant.

The position with regard to infection in the hospital, therefore,
is virtually the same as it was before the epidemic.

**Discussion**

Outbreaks of staphylococcal infection in maternity hospitals due to *Staph. aureus* of phage-type 80 have now been reported from many different parts of the world. Some workers — for example, Rountree and Freeman (1955) and Gillespie and Alder (1957) — noted that the infections caused by type 80 were unusually severe and that its rate of colonisation of the noses of hospital staff was low; they therefore regarded the strain as unusually virulent. Munro and Markham (1958) on the other hand, described an outbreak in which minor infections were common and severe complications rare and in which the strain did not therefore appear to be unusually virulent. In the outbreak described the epidemic type 80 strain corresponded closely to the more virulent type of organism described by Rountree and Thomson and Gillespie and Alder. Clinically the epidemic was characterised by a sudden and alarming rise in the number of serious infections among the babies in the hospital concerned. Phage typing showed that the strain had not been present in the
hospital before the epidemic and that its appearance in infant infections coincided with this sudden increase in serious infections. There was no gradual build-up of infection due to the strain; it appeared quite suddenly as the predominant cause of infant infection and remained predominant until the hospital closed six weeks later. It was therefore clear that in this epidemic type 80 was of higher infectivity than other staphylococcal strains. Despite the fact that it caused 78% of the infections in the sick nursery (table XI) the rate of nasal carriage in the staff and of colonisation of the environment of wards and nurseries was extremely low. The latter was a most unexpected finding since it has been shown that as soon as infants were colonised or had open sepsis the infecting strains could be found in the ward dust (Hutchison and Bowman, 1957).

It was most interesting that some of the strains of staphylococci which caused the mild infections before the epidemic were isolated from many sites in the hospital, as well as from the noses of many nurses. It is, of course, quite possible that the property of enhanced infectivity in a strain is not necessarily associated with the ability to colonise widely. In this
epidemic the less infective staphylococci appeared to have greater powers of colonisation than the epidemic strain.

No vigorous attempt was made in the nurseries to control the infections. It is doubtful if this could have been effective in view of the absence of satisfactory isolation facilities at the time of the epidemic; this was due to overcrowding which was unusually great because the building was being altered. When a strain of staphylococcus becomes established as a major cause of dangerous infection without colonising widely, it seems unlikely that it could be eliminated by the measures usually recommended which are mostly aimed at preventing widespread colonisation. Although the closure of the hospital and its subsequent disinfection were naturally unwelcome measures they were successful in eliminating the epidemic strain from the hospital.

In the surveys on infants and mothers after discharge an attempt was made to assess how far type 80 had spread outside the hospital. Despite considerable effort, it is probable that only a part of the total picture was detected. The nasal swabs taken from infants after discharge showed a decline in the staphylococcal nasal-carriage rate from 94% at one month (table XIII)
to 44% at 4 months (table XIV). In the survey 4 months after discharge only 2 babies and 3 mothers still carried type 80, although infants were selected who were known to have been involved in the epidemic. There was therefore no evidence that type 80 persisted longer in the nose than any other strain, and the danger of its spreading widely in the general population as a result of the epidemic appeared to be small.

It was interesting to find that type 80 did not show such a clear predominance in infections at home as it had done within the hospital. This, of course, may simply be a reflection of the strain's low powers of colonisation because its prevalence in these infections, which was 25% (table XII), corresponds closely to its prevalence in the infant staphylococcal nasal carriers in hospital (23%). But an unusually infective staphylococcus would have been expected to cause disease more frequently than normally pathogenic staphylococci, and why this did not happen in babies discharged from hospital is not known. The incidental discovery that penicillin-resistant strains other than type 80 (which was resistant to penicillin and the tetracyclines) were causing infections in infants at home, suggests
that even in the absence of the epidemic strain, hospital-acquired staphylococci would still be giving rise to a considerable amount of infection. This raises two important points. Firstly, penicillin should never be used in these cases until the antibiotic sensitivity of the infecting organism has been determined. Secondly, these infants and their mothers may introduce antibiotic-resistant staphylococci into the general population.

It is clear that follow-up surveys are essential to determine the true extent of staphylococcal infection acquired in maternity hospitals and this has been emphasised recently by American workers (Ravenholt, Wright and Mulhern 1957, and Wysham, Mulhern, Navarre, La Veck, Kennan and Giedt 1957). Unfortunately it is very difficult to carry out follow-up surveys in this country. In the outbreak described by Ravenholt and his colleagues, inquiries were made by telephone about the subsequent progress of babies and mothers — a method which would, of course, be quite impractical in the United Kingdom. Wysham and his colleagues examined babies and mothers when they attended postnatal clinics. This might be a practical method of follow-up
in this country, particularly if inquiries could also be made at child-welfare clinics. As things are, it is difficult for staff in maternity hospitals to be aware of the extent to which staphylococci acquired in hospital cause disease in babies and mothers after discharge. The surveys reported here show the possible extent of this kind of infection. It was also discovered that 3 infants died of staphylococcal disease after they had been discharged home apparently well.

It is very doubtful if these cases would have been traced to the hospital if, with the co-operation of other bacteriologists in the area, an especial effort had not been made to discover cases of infection in these babies. Because it is clearly important that maternity hospital staff have these cases brought to their notice it is suggested that serious staphylococcal infection appearing in infants after discharge be reported to the local health authority and to the hospital where they were born.
A DISCUSSION

Outbreaks of staphylococcal infection in hospitals today. It is essential to understand...

CHAPTER V

Outbreaks of staphylococcal infection in hospital.

A DISCUSSION.
Outbreaks of Staphylococcal Infection in Hospitals

A Discussion

Staphylococcal infection is acknowledged as a major problem in many hospitals to-day. It is especially troublesome in surgical and maternity units — doubtless because patients with surgical wounds and newborn infants are more liable to develop infection than other patients. Two factors have largely been responsible for renewed interest in the centuries-old problem of hospital infection: first, the emergence of antibiotic-resistant strains of staphylococci and second, the appearance of epidemics of infection due to the organism in hospital wards.

This thesis describes the investigation of hospital staphylococcal outbreaks by phage-typing. The phage-typing technique has been described in detail. As a method of typing staphylococci it is laborious and time-consuming but once the initial difficulties have been overcome it is invaluable for investigating staphylococcal epidemiology. Numerous different types of staphylococci can be identified by phage-typing and although variation is found, this has been carefully analysed
(Williams and Rippon, 1952) and with experience, the significance of the results can be assessed without great difficulty. Tables IV and V show the percentage frequency of phage types isolated from infections in different groups of patients and phage typed at the Western Infirmary during the 2 years from June 1957 to June 1958 and from June 1958 to June 1959. The results demonstrate another way in which experience is of great value in this work. The epidemiological significance of individual types can be more easily assessed if the hospitals, units, lesions and so on, with which they are commonly associated, are also known. The importance of being familiar with the types causing infection in a unit prior to the appearance of an epidemic can hardly be over-emphasised. With information of this sort, an epidemic can be recognised at an early stage and investigations can be undertaken which may elucidate the source and mode of spread of infection before the epidemic strain has become firmly established in the unit concerned. It is in any event extremely difficult to investigate epidemiology in retrospect. It may be unreasonable to expect every hospital to phage type all staphylococci isolated
in its precincts, but a case can be made out for making facilities of this sort available to all or most maternity hospitals. In the Royal Maternity Hospital, Glasgow, and its associated hospitals, the Ross Hospital, Paisley, and Redlands Hospital, Glasgow, routine phage typing is undertaken by the laboratory in the Western Infirmary. There is good evidence that, since the 1957-58 outbreak due to type 80, this service has helped to prevent the development of further outbreaks. In this respect, the value of personal discussions between those doing phage typing and those dealing with the investigations in the hospitals must be emphasised. In the four outbreaks described in this thesis this sort of co-operation helped greatly to ensure that appropriate epidemiological investigations and measures for controlling the infections were instituted. Of these four outbreaks, three involved surgical patients and the fourth took place in a maternity hospital. This discussion deals with the principal features of these four outbreaks and considers them in relation to the general problem of outbreaks of infection in surgical and in maternity units.
I. The Epidemiology of Outbreaks of Staphylococcal Infection in Surgical Units

The epidemiology of infections due to Staphylococcus aureus in surgical units was studied by investigating individual outbreaks of infection as they became known in various hospitals. The principal features of the outbreaks described here were as follows:

1. The Outbreak in the Western Infirmary, Glasgow.

The source of this outbreak, which was due to type 52A/79, was a boil on a surgeon's forearm. He was not a nasal carrier of the epidemic strain and the site of the lesion and the nature of the infections (all of which were deep wound abscesses) indicated that the route of infection was direct contact with the wound tissues through the wet sleeves of his gown. There was an extremely high incidence of infection (6 among 8) in the cases operated upon by the surgeon while he was suffering from the boil and two of the infected patients died. Although the epidemic strain did not subsequently become a predominant cause of infection in the unit there were three incidents of ward cross-
infection due to it.

2. The Outbreak in Law Hospital, Lanarkshire.

The first 9 cases of infection in this epidemic were due to a surgeon who operated while suffering from boils in the axillae which were due to type 80. There was some evidence that 2 months previously he had infected some of his cases from a scabbed over lesion on his finger and that this incident, also, was due to type 80. The route of infection from the axillary boils may have been from contamination of the skin of his hands and arms through glove punctures or wet gown sleeves but could possibly have been by dissemination of the organisms into the air. Although the surgeon was a nasal carrier of type 80 he did not infect his cases except when he himself was suffering from active sepsis. Subsequent to this, type 80 became epidemic as a widespread cause of wound infection in the unit. Most of the infections appeared to have been acquired during operation. It seemed likely that contamination in the wards of either the patients' skin or the gowns in which they were dressed for operation, was the source of these infections. A possible route
by which the gowns could become contaminated was discovered when 
type 80 was isolated from a linen basket used for transporting 
both clean and soiled linen to and from the laundry.

3. **The Outbreak in the Southern General Hospital, Glasgow.**

Two epidemic strains were responsible for the majority of infections in the surgical unit of this hospital. The source of the first strain, type 3Bw/55vw/71 was the nose of a surgeon. The route of infection was probably either through glove punctures from the skin of his hands or from dissemination of organisms from his mask into the air. The infections due to this strain had appeared sporadically over several months and showed a marked tendency to follow long and serious operations. The second epidemic strain was type 80 and nearly half the infections it caused were septic lesions other than wound infections and were clearly due to ward cross-infection. Due to lack of necessary information it was impossible to determine if the wound infections had been acquired in the theatre or in the wards. There were sources of infection in both, because the theatre orderly was suffering from recurrent furunculosis of
the face and neck, and septic cases were being nursed in the wards among the other surgical patients several of whom — as well as some of the nurses — were nasal carriers of type 80.

Staphylococcal wound infections appear in most if not all surgical units. Often the incidence of infection is low and this sporadic sepsis does not as a rule give rise to concern unless in individual cases when it may seriously delay recovery or even endanger the patient's life. Sometimes however the incidence of infection rises steeply and instead of being sporadic becomes epidemic. It is this epidemic infection which, to-day, is causing concern to both surgeons and bacteriologists. The outbreaks which have been described illustrate some of the ways in which this can happen. The infections in the Southern General Hospital which were due to type 3Bw/55vw/71 cannot be correctly called epidemic because they appeared only sporadically and over a considerable period of time. Nevertheless they gave rise to concern in several patients, and two died. The other outbreaks were associated with a relatively high incidence of infection and in each, one phage type of staphylococcus was responsible for most of the infections. Outbreaks have been
reported, for example by Shooter, Taylor, Ellis and Ross (1956), in which many different phage types had caused the infections but most of the epidemics known to this laboratory have been associated with one predominant phage type.

There are 3 main factors which may contribute to the development of epidemic infection. These are, firstly, the susceptibility of the hosts, secondly, the sources and routes of infection and thirdly the infectivity of the epidemic organism. It is proposed to discuss the four illustrative epidemics which have been described in relation to each of these factors.

1. The Susceptibility of the Hosts.

Surgical wounds are highly susceptible to infection. During an operation the body tissues inevitably undergo varying degrees of trauma and haematomata or areas of relative ischaemia may be left in the depth of the wound. All this can, of course, be minimised with expert surgical technique but it is nevertheless likely that any wound constitutes a readily target for the development of infection. McDermott (1956) has suggested that, because of advances in what surgeons now undertake, the hospital
population to-day is less able to combat infection than formerly and that the present increase in hospital staphylococcal disease is due to "... this increased congregation of more susceptible hosts". He mentions various surgical treatments which may lower the body's defences to infection including such operations as total gastrectomy and partial pulmonary resection.

In the outbreaks which are described here, there was, with one exception, no evidence that the patients involved had undergone unusually severe operations or had been subjected to any other treatment which might render them more susceptible to infection. The exception was the outbreak in the Southern General Hospital due to type 3Bw/55vw/71 in which there was a marked tendency for infection to follow cardiac and other long and serious operations. There was evidence, however, that the surgeon who was a nasal carrier of the infecting staphylococcus might have transmitted this strain to his cases through glove punctures from contamination of his hands or via wrongly directed air currents due to the faulty ventilation of the theatre.

Gloves would be more liable to be punctured in the course of long and difficult operations and it is possible that the
association of infection with unusually severe operations may have been at least partly due to the increased opportunity offered for the transmission of infection in these cases. Probably increased susceptibility of the hosts and increased opportunity for infection both contributed to the development of infection in this outbreak. Apart from this, the patients involved in the other outbreaks had undergone the usual type of operation carried out in general surgical units and there was no evidence that they were less able to combat infection than any other patient with a surgical wound. It may be concluded, therefore, that there was, on the whole, no evidence that undue susceptibility of the hospital population to infection had been a major factor contributing to the development of these four epidemics.

2. The Sources and Routes of Infection

If they were not unusually susceptible to infection, were the patients affected by these epidemics exposed to particularly dangerous sources or routes of infection? Surgical wounds may become infected in the operating theatre or post-operatively in the wards. They are probably less susceptible to
infection in the wards than during operation, because the healing process, or the primary union, of a clean incised wound takes place very rapidly. Thus fibroblasts are active by the end of 12 hours and the wound edges are firmly sewn together by the 4th day (Boyd, 1947). During this period most surgical wounds would be firmly covered by an occlusive dressing unless they contained drainage tubes. It might be assumed from this that it is important to ensure only that the operating theatre is free from sources of staphylococci; but there are two reasons why conditions in the wards are of great importance also. Firstly, although theatre-acquired infections may be more common, many wounds undoubtedly become infected in the wards post-operatively. In the epidemics described here, although most of the infections were acquired at operation, in each instance some infections were due to postoperative contamination in the wards. In the Southern General Hospital, ward cross infection giving rise to various forms of septic lesion, was on a considerable scale. Secondly, the ward may be the source of infection although the wounds actually become contaminated at operation. The wards were almost certainly the source of many of the deep wound infections
which appeared in the later stages of the widespread outbreak in the surgical unit in Law Hospital.

Nevertheless, it is probable that a dangerous source of staphylococci will be more dangerous if it has access to wounds during operation. The principal sources of staphylococci in an operating theatre are the surgeons and nurses, the patients, and possibly the air. Of these sources, surgeons are probably the most important; their hands are in direct contact with the wound and organisms disseminated from their skin and clothing will be in close proximity to it. Devenish and Miles (1939) have shown how staphylococci from a surgeon's hands may be transmitted through glove punctures, and the frequency of these makes this an important and dangerous route of infection. This outbreak, together with that reported by Shooter, Griffiths, Cook and Williams (1957), shows that surgeons who are healthy nasal and skin carriers may infect a high proportion of their cases at operation. Although nurses in an operating theatre are not in close contact with the wound they may contaminate equipment used during the operation; and Penikett, Knox and Liddell (1958) have reported an outbreak due to a nurse who contaminated "Terylene" sutures with her nasal
Blowers (1957) and Kimmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958) have found evidence that infections are often due to staphylococci present on the patients' skin, and Kimmouth and his co-workers noted that these organisms were usually hospital strains which had been acquired in the wards prior to operation. Theatre air contaminated by dust from the hospital corridors due to faulty ventilation has been incriminated as a source of infection, and outbreaks of infection have been attributed to this by Blowers, Mason, Wallace and Walton (1955) and by Shooter, Taylor, Ellis and Ross (1956).

Two of the epidemics described here were due to surgeons operating while suffering from septic lesions and both were associated with a high incidence of infection among the cases at risk. It therefore appears that septic lesions on surgeons' hands or arms may be an exceptionally dangerous source of wound infection. There are two possible reasons for this. Firstly, staphylococci may be both more numerous and more infective when they are derived from an active lesion rather than from a healthy carrier, and secondly, direct planting of these organisms from the surgeons' hands or forearms into the exposed wound tissues may be
an exceptionally dangerous route of infection. It was first suggested by Barber and Burston (1955), who were investigating staphylococcal epidemiology in a maternity unit, that staphylococci from an active lesion were more dangerous than those from a healthy nasopharynx. The 2 epidemics described here lend further support to this view. Both the surgeons infected an extremely high proportion of their cases while they were suffering from boils; moreover, the surgeon in the second outbreak did not infect his cases when he had no sepsis himself, although he continued to carry the strain in his nose. In this instance there was a marked association of sepsis in the surgeon with the development of infection in his cases. It is interesting to compare these 2 outbreaks with the third which was due to a surgeon who was a nasal carrier (and an intermittent skin carrier) but who did not suffer from sepsis. The infections were sporadic and had appeared over a long period of time and they showed a definite tendency to follow long and serious operations when the patient's resistance to infection may have been lowered. This suggests that while a surgeon who is a healthy carrier may occasionally infect his patients, he may be less likely to cause
a serious outbreak of wound infection than a surgeon who is suffering from active sepsis. However, serious outbreaks, which were due to surgeons who were healthy carriers, have been reported by Devenish and Miles (1939) and by Shooter, Griffiths, Cook and Williams (1957) although in the outbreak described by Devenish and Miles the surgeon had suffered from boils some months previously. The type of carrier who can cause outbreaks of this sort is probably rare and dangerous only when the staphylococci he carries have access to wounds during operation through the dangerous route of inoculation by direct contact. Nevertheless the possibility of a healthy carrier being a dangerous source of infection must be borne in mind. Even in the outbreak described here, although infections were only sporadic, the consequences in some of the patients were extremely serious.

It is obvious that the vast majority of nasal carriers among surgeons and members of the theatre staff are not sources of infection to others. In the course of the investigations recorded here many surgeons and nurses were proved to be nasal carriers of staphylococci which never caused infections in patients. Hare and Ridley (1958) estimated that about three-
fifths of a small group of nasal carriers had sufficient numbers of staphylococci on their skin and clothing to make them capable of acting as staphylococcal donors. If these results are correct and generally applicable, clearly the staphylococci disseminated from healthy carriers seldom give rise to infection. This might be regarded as further evidence that most of the staphylococci from the healthy nasopharynx are of relatively low infectivity but further work is necessary on the dispersal of staphylococci from carriers under operating-theatre conditions before it can be assumed that such a high proportion of nasal carriers actually disseminate organisms in theatre without causing infection in their patients.

In the epidemic which followed the appearance of type 80 in the surgical unit in Law Hospital it seemed probable that staphylococci acquired in the wards were being transmitted to the wounds during operation from either the patients' skin or their gowns. There was little direct evidence for this beyond the finding that the gowns in which the patients were clad had been taken from a linen basket contaminated with the epidemic strain and the fact that there was no possible source of infection in the
operating theatre which could be demonstrated except the patients
themselves. Kinmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958)
found that many patients became skin and nasal carriers of
hospital staphylococci in the wards prior to operation. Since it
is unlikely that any method of pre-operative skin preparation can
render the skin sterile, (Lancet, 1958) this may be an important
mode of infection in an epidemic where one strain has become
widespread and predominant in infections in the wards. In the
surgical wards in Law Hospital there were numerous sources in the
wards from which type 80 could have been acquired. The most
important were probably the patients infected with type 80 who
were being nursed alongside those admitted for operations in the
usual way but the epidemic strain was also found in samples from
ward dust and blankets.

The role of the theatre air as a source of staphylococci
has aroused much interest in recent years. It is obviously
desirable that the air in operating theatres should be
bacteriologically as clean as possible and that the system of
ventilation should be able to remove rapidly any bacteria
liberated into the air. It is a little difficult to be convinced
the very few staphylococcus-carrying particles which it has been possible to demonstrate on a slit sampler in badly ventilated theatres can be regarded as a dangerous source of infection.

Nevertheless, Shooter, Taylor, Ellis and Ross (1956) have described an outbreak which provides at least suggestive evidence that bad ventilation might sometimes be responsible for a high incidence of wound infection.

The only outbreak described here in which the theatre ventilation system may possibly have played a part was that due to the surgeon who was a nasal carrier. Staphylococci were regularly present on his mask and the vertical down-draught from the wrongly-directed input vents above his head may have transmitted the organisms into the wounds. If this were indeed so, the theatre air in this outbreak could be regarded as a route for conveying infection rather than the source of it.

It is often relatively easy to demonstrate the source and to deduce the probable route of infection acquired in the operating theatre. The investigation of the epidemiology of staphylococcal cross infection in the wards, on the other hand, is fraught with difficulties. This is largely due to the
profusion of possible sources of staphylococci in most surgical wards — particularly under epidemic conditions — and the virtual impossibility of demonstrating the routes by which the organisms spread. Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright (1958) describing an epidemic of ward cross infection in America due to type 42B/52/81 (which is similar to the strain known as type 80 in this country) considered that the most important means of spread of infection was by direct contact with either staff or patients suffering from active lesions or heavily contaminated hospital materials. They found a low incidence of nasal carriage of the epidemic strain among the staff (4.1%) and noted that outbreaks of infection in some wards had been preceded by a cutaneous infection in a member of the ward staff. They concluded that nasal carriers played an insignificant role in the spread of the epidemic strain and that infected hospital personnel were the most important source of infection. The danger of members of staff carrying out ward duties while suffering from active sepsis is apparent but recently an outbreak has been described which emphasises that patients also may be a dangerous source of infection if they are
nursed in an open ward while suffering from superficial skin
sepsis. Barber and Dutton (1958) observed a serious outbreak of
infection in a surgical ward which was traced to the admission of
a patient who had a septic skin condition due to type 80. The
epidemic strain was isolated from blankets, bed curtains, the ward
air and also from the ward sister's nose. They point out that
the carriage of the strain by the ward sister was probably a
reflection of the spread of the strain rather than its cause, and
suggest that superficial skin sepsis is a more dangerous source
of hospital cross infection than are nasal carriers, whose role
may be relatively unimportant. The study carried out by
Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons
(1958) on the spread of staphylococci in a surgical ward showed
that in several of the staphylococcal "broadcasts" which they
observed the strains responsible were first introduced into the
ward by a nasal carrier among the patients or nurses. Most
often, however, the probable sources from which the staphylococci
became widely dispersed were infected lesions — particularly
when these were of a nature which would render adequate cover by
dressings difficult. Although nasal carriers may occasionally
transmit their strain to others and may thus be responsible for sporadic infection, active lesions are probably the main sources of spread of epidemic infection. There is little doubt that contaminated dust, blankets, bed screens, and other articles of ward equipment may act as secondary reservoirs in the spread of infection but the staphylococci which contaminate them must be maintained from other sources in the ward. They could therefore perhaps be better described as routes rather than the sources of infection.

None of the hospitals whose epidemics are described in this thesis had isolation facilities for infected surgical patients. This meant that most of the surgical wards contained several patients with septic lesions being nursed with the other surgical patients admitted for operations in the usual way. In an epidemic this, of course, results in the congregation of dangerous sources of the same staphylococcal strain among those who, because of their surgical wounds, are especially liable to develop staphylococcal infection. This was particularly noticeable in the widespread epidemics due to type 80 in Law Hospital and in the Southern General Hospital where infections continued to appear more
frequently in some wards than others. These more severely affected wards each contained several patients infected with the epidemic strain. In general, when a strain became established as the predominant cause of infection in a ward, one or two of the nurses were found to be nasal carriers and it could often be demonstrated in the ward environment. It is, of course, almost impossible under these conditions to demonstrate the actual source which is responsible for the infections but this suggests that these nurse nasal carriers simply reflected the spread of the epidemic strain without necessarily being the cause of it. In some of the surgical wards in Law Hospital none of the nurses were nasal carriers, and it was clear that in these wards the epidemic was being sustained by infected patients who acted as sources of infection to the others.

Apart from the surgeon in the Southern General Hospital whose strain caused sporadic infections in his cases there was no evidence that nasal carriers played any part in the spread of the epidemics described. Active lesions were found to be the most important sources of staphylococcal infection and were especially dangerous when organisms from them had access to wounds by direct
contact at operation. Contamination with staphylococci acquired in the wards pre-operatively from infected cases was probably responsible for many of the wound infections which had apparently become infected during operation.

3. The Infectivity of the Epidemic Organism

The third factor which may influence the development of epidemic staphylococcal infection is the infectivity of the staphylococci themselves. It is now known that individual strains differ considerably in their ability to produce disease and the results of the present study further confirm this. In making possible the identification of different strains of staphylococci phage typing has been particularly valuable in this respect.

Tables IV and V give details of the percentage frequency of phage types isolated from infections in surgical and general wards. Most of these infections were in patients in surgical wards. It can be seen that type 80 was by far the most important cause of infection: this was mainly due to its marked tendency to become rapidly the predominant cause of infection in any unit into which it became introduced. This ability to cause
widespread infection is well seen in the outbreaks in Law Hospital and the Southern General Hospital. Type 80 has, in fact, been the main problem in staphylococcal infections investigated by this department over the past 2 years. Williams (1959) in a review of epidemic staphylococci points out that epidemics due to type 80 have been reported from many countries all over the world and that at present we are witnessing its pandemic spread. He also states that type 80 is "... a strain that has more recorded epidemics to its credit than any other type; whose epidemics are, in our experience, twice as extensive as the average of all other types". Most of the epidemics reported in recent literature have troubled maternity units but extensive outbreaks involving the patients in surgical wards have been described by Duthie (1957) and Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright (1958). These workers noted a tendency for type 80 to cause cutaneous lesions among patients and members of staff but whereas Duthie observed a high nasal carriage rate, Caswell and his co-workers found that this was relatively low. In the epidemics described here, a high incidence of septic lesions was observed in the Southern General Hospital where many patients and some
members of staff developed this kind of infection, but in Law
Hospital infections other than wound infections were relatively
rare. If a staphylococcal strain produces an outbreak of
superficial skin infections in a hospital, it is evident that it
causes disease more readily than most other staphylococci and can
probably be regarded as being unusually infective. However,
even in Law Hospital where this tendency was not marked, type 80
behaved as a dangerous organism with a particular propensity for
epidemic spread. Although Williams (1959) has observed that
epidemics with type 80 commonly generate a high carriage rate
this has not been a characteristic of outbreaks investigated
here.

Several workers have reported that type 80 tends to
cause more severe disease than is usually seen with most strains
of staphylococci and it has therefore been regarded as unusually
virulent. This seems to have been most apparent in outbreaks
in maternity units (Rountree and Freeman, 1955; Gillespie and
Alder, 1957; Bass, Stinebring, Willard and Felton, 1958) and may
be due to the greater susceptibility of newborn infants.
Although Duthie (1957) describes the surgical infections as
"severe" it is not clear whether he regards wound infection per se as a severe form of sepsis or the wound infections in this particular epidemic as being unusually severe of their kind. Caswell and his co-workers state that type 80 was the cause of the severe infections in their hospital but give no convincing clinical facts to support this statement. In the outbreaks reported in this thesis there was no real evidence that the individual lesions were unusually severe. Four patients died in the outbreak in Law Hospital, and in each, infection with type 80 played a part; but the patients' general condition was poor and, on the whole, the organism did not appear to cause more serious disease than any other staphylococcus although it did seem to cause disease more readily. Rountree and Beard (1958) have noted that although type 80 caused an increased amount of severe disease in the 3 years since it first appeared in Australia, the case mortality rate in infections due to it was no higher than with other staphylococcal strains. They prefer to call type 80 highly infective rather than highly virulent. This description is therefore more appropriate to the organism in the epidemics described here than "highly virulent".
Type 80 is a particularly dangerous cause of epidemic staphylococcal infection but it must be emphasised that other strains can, and frequently do, give rise to outbreaks of infection. Staphylococcal strains of phage group III have been the cause of several outbreaks in surgical wards (Blowers, Mason, Wallace and Walton, 1955; Gillespie, 1957; Shooter, Griffiths, Cook and Williams, 1957; Williams, 1959). Tables IV and V show that phage group III strains caused many infections in the surgical and general wards from which strains were submitted to the Western Infirmary for phage typing. Although they have not given rise to any epidemics as widespread as those due to type 80 these strains have been a major cause of wound infection in surgical units. Shooter, Smith, Griffiths, Brown, Williams, Rippon and Jevons (1958) found that phage group III strains formed a high proportion of the staphylococci present in the environment of surgical wards and caused, from time to time, small scale outbreaks of infection. This picture is probably typical of most surgical wards unless they are experiencing an epidemic due to a strain of increased infectivity. Shooter and his co-workers also found evidence that staphylococcal strains
differed greatly in their ability to cause disease. Although 186 different phage types were isolated from the ward under investigation only 13 gave rise to disease and only 3 of these caused infections in more than one patient. Probably any staphylococcus which is coagulase-positive is capable of producing disease but it seems certain from these observations that different strains vary greatly in their ability to do so. Many different grades of infectivity may exist among staphylococci, ranging from the highly infective organisms — of which type 80 is an extreme example — through some of the more dangerous phage group III strains and others which appear less frequently in infections, to the strains which very rarely cause disease. This gradation in degrees of infectivity among the staphylococci responsible for infections in surgical and general wards can be seen in tables IV and V and has been discussed in Chapter IV (b). It is unlikely that any staphylococcus can be regarded as of little or no importance as a cause of infection and incapable of producing an outbreak of sepsis. Type 52A/79 is not a common cause of epidemic infection in surgical units yet when it had access to surgical wounds from a dangerous source in which its natural
infectivity may have been increased (in this instance an active lesion on a surgeon's forearm) it produced an outbreak of serious wound infection. The other epidemic strain — type 3BW/55vw/71 — which was responsible for a number of infections in the Southern General Hospital, had been hitherto virtually unknown as a cause of hospital infection. But when it became transmitted to wounds during operation from a healthy carrier who was a surgeon, it gave rise to serious infection in several patients and undoubtedly contributed to the death of 2 of them.

It can be concluded that some strains of staphylococci have a higher infectivity or ability to produce disease than others and so are more likely to produce epidemics of hospital infection. Type 80 is an extreme example of this kind of organism and the outbreaks described here give evidence of its unusual abilities to spread and to give rise to disease more readily than other strains. Other strains may, however, have their natural infectivity enhanced by being derived from an active lesion (which therefore constitutes an exceptionally dangerous source of infection). Staphylococci which very rarely cause infection in the normal course of events may nevertheless do
so when they are transmitted to a surgical wound at operation, although there is no evidence that their infectivity is in any way increased. Possibly the dangerous direct route of inoculation is partly responsible for this.

No staphylococcal strain, given exceptional means and opportunity, can be ignored as a possible cause of outbreaks of surgical infection although some strains have inherent properties of infectivity which make them more dangerous as potential causes of epidemic infection.

The Control of Outbreaks of Staphylococcal Infection in Surgical Units

It is probably unrealistic to attempt to abolish staphylococcal infection from surgical wards. Since at least half of both patients and members of the staff carry staphylococci in their noses, a few sporadic infections from time to time would not be surprising. However the incidence of infection should and probably could be kept to a very low level — 1% or less in clean surgical wounds. Most hospital staphylococci are resistant to at least one and usually to several antibiotics,
and it is clear, therefore, that the excessive use of these drugs, far from providing an answer to the problem, will merely result in the appearance of more and more resistant strains. Planned control of or even a thoughtful self-discipline in their use might help to prevent the frequent emergence of strains resistant to several antibiotics. Of itself, this would render serious infections more amenable to therapy, but alone it would be unlikely to prevent outbreaks of infection. The control of infection must ultimately depend on the elimination of sources and the blocking of routes of infection.

The elimination of dangerous sources of infection is the most urgent problem in surgical units. Strict observance of the aseptic technique in the operating theatre and in the dressing of surgical wounds in the wards is vital. Unfortunately there is reason to believe that with the advent of antibiotic therapy, and loose thinking on the subject, aseptic routine is not carried out so rigidly as before. In the epidemics described here 2 surgeons were discovered to have operated while suffering from boils of the arms. This, of course, is a gross breach of the aseptic technique; yet it came twice to notice in
a relatively short period of time — and a time, moreover, when wound infections were under investigation in the units concerned. The suspicion is inescapable that this serious lapse is probably more often allowed than is ever realised or discovered. Nurses, also, sometimes continue to carry out theatre and ward duties with septic lesions of their hands, arms and face — perhaps without being aware of the dangers to which they expose their patients. Errors of this sort should never be allowed. Members of the hospital staff should always and at once be relieved of duties in operating theatre or wards if they are suffering from active sepsis, and they ought not to return until the lesion is completely healed.

Although any source of infection is probably more dangerous if it has access to wounds during operation, measures are also necessary to rid the wards of the more important sources of infection. If staphylococcal infection is spreading in a ward, it is obviously essential to isolate those suffering from the disease from those who are susceptible to it. Isolation is an accepted principle in the prevention of infectious disease and it ought therefore to be applied to staphylococcal
disease in hospital. Few hospital authorities or clinicians have faced up to this: most recognise that staphylococcal infection is a problem in many surgical wards but few have made any provision for the isolation of septic cases. Until infected cases can be segregated from the other patients, staphylococcal infection is bound to continue. Lack of isolation facilities is particularly serious when an epidemic breaks out and is undoubtedly an important way of keeping the infecting strain active as a major cause of infection. If these dangerous sources of infection were denied access to the operating theatre or wards, most outbreaks of infection would probably be prevented or soon cut short.

It has been stated here that most nasal carriers are unimportant as sources of epidemic infection. They cannot of course be ignored, as there is no doubt that some of them infect patients from time to time, and occasionally on an extensive scale (Devenish and Miles, 1939; Shooter, Griffiths, Cook and Williams, 1957). In an epidemic, carriers of the epidemic strain should be relieved of duty and an attempt must be made to abolish the strain from their noses. Although it is sometimes
difficult to do this, the results obtained by Gould and Allan (1954) and Gould (1957) suggest that the intra-nasal application of antibiotic cream may be effective — at least as a temporary measure. The member of staff who is a persistent carrier of an epidemic strain, and who does not respond to treatment, presents a particularly difficult problem. It is probably reasonable to allow such people to resume duties unless there is definite evidence that they are infecting patients. Apart from a small outbreak described by Rountree in 1947, there is little published or other evidence that nasal carriers have caused widespread infection in a ward but it is certain that they have occasionally done so in the operating theatre. The outbreaks described by Devenish and Miles and Shooter and his co-workers suggest that infection of this sort might be prevented if surgical gloves could be made of some material less prone to punctures than that at present in use, and if surgeons wore impermeable oversleeves over their ordinary gowns. The institution of hand rinses in antiseptics, such as chlorhexidene in alcohol, before donning gloves might also help to prevent staphylococci from being transmitted to the wound from the surgeon's hands in this way.
Recently, several measures have been recommended to prevent staphylococci from reaching the patients' wounds. These include the provision of wound dressing rooms separate from the wards, a plenum ventilation system in theatre supplying filtered air, the sterilisation of woollen blankets by quaternary ammonium compound disinfectants, (or by boiling blankets made of cotton weave, towelling or "Terylene") and the oiling of floors, damp dusting, vacuum sweeping and adequate ventilation of surgical wards. It is most desirable, of course, that surgical units should have facilities of this kind but as considerable expenditure would be involved if they were introduced on a large scale, this is unlikely to happen in the near future. The observations of Clarke, Dalgleish, Parry and Gillespie (1954) that oiling of the floor, bed clothes and screen covers had no effect on the nasal and wound cross infection rates, and of Kinmouth, Hare, Tracy, Thomas, Marsh and Jantet (1958) that positive-pressure ventilation in a theatre with a cross draught over the table failed to reduce post-operative sepsis, indicate that by themselves these measures will not prevent outbreaks of infection. There is at present a tendency to overlook the point
that these sites are only secondary reservoirs of staphylococci which must be maintained by other sources. The original sources from which staphylococci in the hospital environment must be derived are infected lesions and carriers. I believe that the principal and most dangerous sources of infection are active lesions and that if these were rigidly excluded from theatre and wards, the problem of hospital infection in surgical units would largely be solved. Clearly, if outbreaks of surgical infection are to be entirely prevented, extra precautions are necessary to ensure that carriers do not infect wounds during operations — and measures which might achieve this have been described — but the approach which is most likely to yield dramatic results in the first instance, is the isolation of all active lesions due to staphylococci from wards and theatre. If these dangerous sources of staphylococci are ignored, measures directed solely at controlling spread from the secondary reservoirs are unlikely to succeed. The application to surgical units of the first principle of the control of infectious disease — namely the isolation of those with the disease from those susceptible to it — would probably prevent the most highly infective
staphylococcus from establishing itself as a cause of epidemic infection.

II. The Epidemiology of Outbreaks of Staphylococcal Infection in Maternity Units

Staphylococcal infection among the babies in maternity units is probably even more troublesome than in surgical units. Mild infections are common in the nurseries of most maternity hospitals, but from time to time outbreaks of infection have disastrous consequences. The outbreak of this sort in the Glasgow Royal Maternity Hospital was investigated more completely than is often possible because phage-typing facilities were available and were being systematically used for some months before the epidemic began. The epidemic has already been described in detail in this thesis and it is proposed to discuss the results of the investigations in relation to the general problem of outbreaks of staphylococcal infection in maternity hospitals.

The main features of the epidemic were as follows:

As in most maternity hospitals, mild staphylococcal
infections among the babies were not uncommon before the epidemic. They had been due to many different phage types of staphylococci, however, none of which was really predominant. The epidemic began when type 80 suddenly appeared as the predominant infecting organism; this was associated with a sharp increase in the number of serious infections which led to the hospital being closed 6 weeks after the first appearance of the epidemic strain. Investigations carried out just before and just after the closure of the hospital showed that the nasal carriage rate of type 80 among a small group of babies was low compared to its incidence in infections and that, although staphylococci were present in numerous sites throughout the hospital, contamination of the hospital environment with type 80 was minimal. The epidemic strain also exhibited very low powers of colonisation in the noses of members of the staff. However, many of the strains isolated from sporadic infections before the epidemic were found to have colonised widely. Inquiries at the homes of some of the babies and mothers revealed a number of infections due to staphylococci acquired in the hospital which had appeared after their discharge home.
Type 80 showed no tendency to persist unduly in the noses of the babies and mothers. Three infants died of staphylococcal infection after discharge home from hospital: in 2 cases the infecting strain was type 80, in the third it was a hospital staphylococcus of a different phage type. Infection with type 80 probably contributed to the deaths of 4 other infants.

In the discussion of the epidemiology of outbreaks of staphylococcal infection in surgical units, three factors were considered as possibly responsible for the development of epidemic infection. These were, firstly, the susceptibility of the hosts, secondly the sources and routes of infection, and thirdly the infectivity of the infecting organism. It is proposed to discuss the maternity hospital epidemic in relation to each of these factors.

1. **The Susceptibility of the Hosts**

Newborn babies are particularly susceptible to staphylococcal infection, which usually takes the form of such minor lesions as skin pustules, conjunctivitis, and inflamed umbilicus but may sometimes be more serious disease — for
example: abscess, osteitis, and pneumonia. The infant's body cannot of course be protected like a surgical wound by a firm occlusive dressing, and infection may appear anywhere upon it, although sites subjected to the minor trauma of rubbing or chafing are more often affected (Gillespie, Simpson and Tozer, 1958).

Newborn infants are, in any case, probably less able than adults to combat bacterial invasion perhaps because they have not yet developed an adequate immunological defence mechanism.

In this epidemic the sick and premature infants in the sick nursery appeared to be more affected than the healthy babies in the units. This was probably partly due to the inadequacy of the records concerning the latter and to the practice carried out in the sick nursery, but not in the units, of taking routine bacteriological specimens from even mild infections. The infants in the sick nursery may have been more susceptible to epidemic infection because their physical condition made them less able to combat infection than the healthy babies, and also because the crowding of infants together into one nursery provides a fertile soil for the spread of infection. Infants in the units are nursed beside their mothers in the lying-in
wards although they normally spend some time each day in the unit nurseries for bathing and changing or if they are noisy. These facts may explain why the infants in the sick nursery appear to be more affected by the epidemic but they do not alone explain why the epidemic broke out. Under normal conditions there are always ill and premature babies in the sick nursery and for several weeks preceding the epidemic there had been some overcrowding there. The infants at this time were certainly exposed to staphylococci yet infections were mild and sporadic. It can be concluded, therefore, that although the hosts may have been particularly susceptible to staphylococcal infection, another factor was primarily responsible for determining the outbreak of epidemic infection.

2. **The Sources and Routes of Infection**

The investigation of the sources of infection in infant nurseries is probably even more difficult than in a surgical ward. Staphylococci abound in the environment of most infant nurseries: they contaminate the air, dust, blankets, and articles of equipment, and they are often present in the
noses of the nursing staff. It has been shown that, in addition to this, nearly all infants born in hospital become colonised within a few days of birth and harbour the organisms in their nose, on their umbilicus and on other areas of the skin surface (Gillespie, Simpson and Tozer, 1958). With this profusion of possible sources of staphylococci it is extremely difficult to determine the actual source or sources which are the most important in the spread of infection. A considerable amount of work has been carried out to elucidate this and the following conclusions may be drawn from it. Nurse nasal carriers are often responsible for the introduction of a staphylococcal strain into a nursery (Jellard, 1957) but subsequent spread is usually from baby to baby (Parker and Kennedy, 1949; Hutchison and Bowman, 1957). The most important source of staphylococci in an infant who has become colonised is probably the umbilicus (Jellard, 1957; Gillespie, Simpson and Tozer, 1958) where staphylococci may multiply as in an open wound and which may therefore be a prolific source from which infection is readily disseminated into the air. The air may be an important route by which the organisms spread from infant to infant (Jellard, 1957)
although contact via the nurses' hands is probably responsible in a small number of cases (Cook, Parrish and Shooter, 1958).
Blankets and bedding, however, appear to play little part in the spread of staphylococci among the infants (Gillespie, Simpson and Tozer, 1958).

In the epidemic described here type 80 appeared for the first time in an infection in a baby in one of the units. The infant was later transferred to the sick nursery. Thereafter type 80 became the predominant cause of infection throughout the hospital. It was quite clear that the epidemic was spreading from infant to infant and that nasal carriers among the nurses could not be held responsible for the infections. Nor had there been any extensive contamination of the hospital environment which might also have contributed to the spread of infection. The predominance of type 80 as a cause of infection would result, of course, in the congregation of sources of this one strain among the infants in the nurseries: in addition the epidemic strain was known to have colonised about one third of the healthy babies who must therefore also be regarded as possible sources of infection. There is evidence that in surgical wards the most
important sources of staphylococci are active lesions rather than healthy carriers. This conclusion may not apply to maternity wards to the same extent. There is, of course, a possible reason for this in that the neonatal umbilicus, which is probably one of the most dangerous sources of staphylococci in a nursery, can reasonably be regarded as an open wound where staphylococci are able to multiply without necessarily causing overt signs of infection. Newborn infants whose umbilical stumps become colonised may therefore be compared to adult patients with infected surgical wounds. In this epidemic although some of the infections were superficial — for example: skin pustules, conjunctivitis, and inflamed umbilicus, from all of which organisms may readily be disseminated, many were of a more serious nature and took the form of abscesses. Abscesses, however, can be described as "closed" lesions (in contrast to the former more superficial infections which constitute "open" lesions) and are unlikely to be sources of infection unless they are actually discharging. In practice, infants with infections of this kind were usually transferred to another hospital for further treatment before this happened and so were
unlikely to have acted as sources of infection to the other infants. The babies who suffered from pneumonia on the other hand may well have been responsible for disseminating the organisms into the air and thus for spreading the infection to others.

There are numerous routes, of course, by which staphylococci may spread from infant to infant. These include the air, the nurses' hands or gowns as they handle the infants, bed linen, or the communal equipment used in the nurseries for bathing and changing. It was impossible to demonstrate which of these routes might have been the most important in the spread of the epidemic but the fact that type 80 was isolated from only 1 sheet, although numerous samples were taken from linen, bedding, dust and equipment generally, suggests that the epidemic spread principally by some other route. There was in fact no evidence that this epidemic was due to spread from a particularly dangerous source of infection. It seemed probable that the dominant factor was the high infectivity of the epidemic staphylococcus.
3. The Infectivity of the Epidemic Organism

The epidemic is a striking illustration of how dangerous an unusually infective staphylococcus may be when it appears among the highly susceptible population of a maternity unit. The epidemic strain was type 80, whose special properties have already been discussed in relation to infection in surgical wards. It has been responsible for numerous outbreaks of infection in maternity units in recent years (Williams, 1959) and in many instances the severity of the infections has been a prominent feature (Rountree and Freeman, 1955; Gillespie and Alder, 1957; Bass, Stinebring, Willard and Felton, 1958). Type 80 is quite clearly a strain which is particularly liable to cause extensive epidemics of infection and may often be responsible for unusually severe disease but some reports suggest that it does not always have these characteristics. Thus Munro and Markham (1958) described an outbreak in which nearly all the infections were mild, and Gillespie, Simpson and Tozer (1958) described a nursery in which type 80 was present as a minor cause of mild infection. The outbreak described here, however, shows the tendency of type 80 to produce unusually severe disease. Williams (1959) has
stated that staphylococcal strains of phage group I are particularly prone to colonise the noses of the hospital staff and to spread widely in the environment and that type 80 commonly generates a high rate of nasal carriage among the members of the hospital staff. The extremely low rate of nasal carriage among the staff and colonisation of the hospital environment generally was one of the most remarkable features, however, of this epidemic. The explanation for this is unknown. It may have been a special property inherent in the particular type 80 strain which became epidemic in this hospital, or it may have been that the shortness of the epidemic period (6 weeks) allowed insufficient time for the strain to become established in the unit. This latter explanation is unlikely because the epidemic strain had been established as the predominant cause of infection for the 6 weeks preceding the closure of the hospital. The more probable reason for the low rate of colonisation is that it was due to some individual characteristic of the strain and this has been observed previously in outbreaks described by Rountree and Freeman (1955) and Gillespie and Alder (1957). It was interesting that both the nurses who carried type 80 in their
noses had suffered from attacks of boils. Duthie (1957) noticed a marked tendency for nasal carriers of type 80 to develop superficial septic lesions and this, of course, affords further proof of the strain's ability to produce disease more readily than other staphylococci.

Tables IV and V show that type 80 was the most important cause of infection in the maternity units from which strains were submitted to the Western Infirmary for phage typing. This is largely due to its appearance in some of these maternity units as a cause of epidemic infection. Other strains of staphylococci can also be responsible for outbreaks of infection in maternity units and although type 80 is probably more dangerous than most other strains it must not be regarded as the only staphylococcus which need occasion concern. Extensive outbreaks due to other phage types have been described by Barber, Hayhoe and Whitehead (1949) and Colbeck (1949). Tables IV and V show that, unlike what is found in surgical units in which phage group III strains are the most common cause of infection, phage group I strains predominate in maternity hospitals. It is probable that phage group I strains constitute a high proportion of the staphylococci
which are normally present in most maternity hospitals and some of which may, from time to time, cause small scale outbreaks of infection. The picture of a few relatively predominant strains waxing and waning from week to week with undercurrents of minor strains was revealed by the investigations of Hutchison and Bowman (1957) into staphylococcal epidemiology in a maternity unit.

In the epidemic described in this thesis, some staphylococci which had colonised extensively had caused very few infections in the preceding months and it was clear that they were of relatively low infectivity. Others appeared to be slightly more infective because they had caused a higher proportion of infections than the former strains. It was plain, in fact, that as in surgical units individual strains of staphylococci differed among themselves considerably in their ability to produce disease and that this was not necessarily related to their ability to colonise.

It is interesting that phage group I strains should flourish more readily in maternity units than in surgical wards. This has been noted and discussed by Williams (1959) in a recent review of epidemic staphylococci. On the whole, it is unlikely
that different strains have been selected in the 2 types of unit as a result of the use of different antibiotics, for as far as can be ascertained, the same antibiotics are commonly used in both. The reason may lie in some as yet unknown property which enables some strains to cause infection more readily among the infants in maternity units than among the adult patients in surgical wards. The difference in the types of staphylococci which frequently cause infection in maternity units on the one hand and surgical wards on the other hand is not particularly clear cut and several strains are fairly common in both. Nevertheless the difference exists and the possible reasons for it afford interesting speculation.

One strain which seems capable of causing disease in either kind of unit is, of course, type 80. Although more outbreaks have been reported from maternity units (Williams, 1959) it can spread just as extensively in surgical units (Caswell, Schreck, Burnett, Carrington, Learner, Steel, Tyson and Wright, 1958). There is little doubt that the newborn infant is particularly susceptible to infection by staphylococci and that an unusually infective organism, such as type 80, more often results in serious and fatal infection when it becomes epidemic.
in a maternity hospital. In this outbreak there was no evidence that a particularly dangerous source played any part in spreading infection and the special properties of type 80 appeared to be the main factor in determining the development of the epidemic.

The Control of Outbreaks of Staphylococcal Infection in Maternity Units

The control of infection among the infants in maternity units presents a far more difficult problem than that in surgical units. Conditions in most maternity hospitals to-day make control of infection virtually impossible to achieve. Probably the single most important factor responsible for the frequency of staphylococcal infections among the newborn is the crowding together of the infants in small nurseries or beside their mothers in an open ward (when they usually still spend a considerable amount of time in a communal nursery). The newborn infant is particularly susceptible to infection with staphylococci and as most become colonised by the organism within a few days of birth it is not surprising that infection is so common in maternity hospitals. The investigations reported here on infection in
babies shortly after their discharge home from hospital, indicate that the true incidence of hospital-acquired staphylococcal disease is much higher than is realised from a study only of the infections observed inside the hospital. The very high incidence of infection which was revealed by these investigations cannot be regarded with complacency. Nor can the fact that some of the infections may be serious and even fatal be ignored. Three babies were discovered to have died from staphylococcal sepsis after their discharge from hospital: in 2 cases the infecting strain was type 80 but the third child died of infection with a strain which had caused sporadic disease in the hospital but had never appeared to be particularly infective.

It is difficult to see how infants can be protected from the staphylococci which are ubiquitous in maternity hospitals except by the provision of single rooms where they can be nursed alongside and by their mothers. Unfortunately few hospitals have this sort of accommodation at present and until it is available on a large scale the incidence of staphylococcal infection is likely to remain high.

Attempts to prevent infants from becoming colonised by
staphylococci are almost certainly doomed to failure when they are being nursed in the conditions pertaining in most hospitals to-day. Various measures have been recommended in an attempt to reduce cross infection and colonisation in infant nurseries. These consist for the most part of either the application of antiseptics to what are considered to be the principal sources of infection or precautions in the handling of infants to avoid the transfer of organisms from one to the other. A strict aseptic technique is of course desirable but is difficult to carry out when a large number of infants require frequent attention from a few nurses. The work by Cook, Parrish and Shooter (1958) and Forfar and McCabe (1958) suggests that most infants become colonised by a route other than via the nurses' hands. The most realistic approach would appear to be to prevent the organisms from reaching and disseminating from the most important sources of infection. The reasons for believing that the umbilical cord may be an important source of staphylococci have been discussed and good results have been reported from the application of antiseptics to it in an effort to reduce cross infection and colonisation. Jellard (1957) used triple dye for this and noted a reduction in skin and nasal
carriage rates and Gillespie, Simpson and Tozer (1958) reported similar results with a fall in the incidence of sepsis after dusting the cord with powder containing hexachlorophene. The study by Gillespie and his co-workers however makes it clear that this alone will not prevent more than half the infants from becoming colonised by about the 6th day of life. Measures like this may prevent a considerable amount of infection and should probably be instituted in most nurseries but it must be admitted that they are unlikely to have much effect on an epidemic due to a highly infective organism of the sort described here. When a strain like type 80 becomes introduced and established as a major cause of infection then more stringent precautions are necessary. If prompt action were taken to isolate all infected cases and carriers of the organism at the outset it might be possible to break the circle of infection. This of course can be done only when phage typing facilities are immediately available. The severity of the infections described here indicates that action of this sort ought to be contemplated if an organism known to be highly infective makes its appearance. It may be that not all epidemics due to type 80 are attended by such serious consequences.
as this one, but the risk does not seem to be worth taking and it should be emphasised that it is often extremely difficult to ascertain the number and severity of infections unless inquiries are extended beyond the hospital where the infants were born. Unless prompt action — as outlined above — is taken, it may become necessary to close the hospital. This is always a most unwelcome measure but is preferable to risking infant deaths from infection.

Under present conditions it seems likely that staphylococcal infection will remain a problem in maternity units. Until single rooms can be provided for each baby and mother the dangerous circle of infection from infant to infant will continue, and outbreaks of infection will appear from time to time when a more than usually infective organism becomes established in the nurseries. We are to-day facing the question whether the benefits of confinement in hospital are outweighed by the greatly increased risk of serious infection.

Strenuous efforts should and must be made to ensure that our hospitals do not constitute a danger to the patients, particularly those in maternity units and in surgical wards.
Now that the staphylococcus seems likely to remain unconquered by any new antibiotic which may be introduced it becomes imperative that we take measures to protect patients from infection with this organism. This study suggests that more could be done to prevent hospital staphylococcal infection than is being done at present. Staphylococcal disease in hospital is an infectious disease: much harm has arisen from the failure to regard it and treat it as such.
Summary

All of these typing staphylococci surveys has been
conducted in detail together with some of the difficulties
which we encountered in the setting up of a phase typing
laboratory.

2. Differences in the percentage frequency distribution of
phage types isolated from infections in surgical and general
wards in maternity units, and in outpatients are discussed.

3. Phage typing was used to SUMMARY rate in some cases the

incidence of infection or staphylococcal infection in 3
surgical wards and in a maternity hospital.

4. In the surgical wards, most cases of staphylococcal
infection had been acquired in the operating theatre, but in one outbreak
there was some evidence that contamination with staphylococci
on the hands prior to operation was probably responsible for

a series of cases.
Summary

1. The method of phage typing *Staphylococcus aureus* has been described in detail together with some of the difficulties which are encountered in the setting up of a phage typing laboratory.

2. Differences in the percentage frequency distribution of phage types isolated from infections in surgical and general wards, in maternity units, and in outpatients are discussed.

3. Phage typing was used to investigate in some detail the epidemiology of outbreaks of staphylococcal infection in 3 surgical units and in a maternity hospital.

4. In the surgical units, most cases of wound infection had been acquired in the operating theatre, but in one outbreak there was some evidence that contamination with staphylococci in the wards prior to operation was probably responsible for a number of these.

5. The development of outbreaks of infection was found to be determined either by the infectivity of the infecting organism, by the wounds being exposed to an unusually
dangerous source of organisms in which their natural
infectivity is increased, or by the organisms having access
to the wounds by a particularly dangerous route of
inoculation.

6. Type 80 is a staphylococcal strain of exceptional infectivity.

It showed a marked tendency to establish itself rapidly as a
predominant cause of widespread infections. In one outbreak
it caused frequent septic lesions other than wound infections.
It did not cause unusually severe infections in the surgical
units.

7. Active lesions were the most dangerous sources of
staphylococci, and there was evidence that organisms derived
from them were more infective than those from healthy
carriers. The presence of infected patients in the wards
was considered to have been an important factor in the
spread of the epidemics.

8. Wounds are particularly susceptible to infection during
operation and direct contact with organisms from a surgeon's
hands is probably the most dangerous route of infection.

9. Most outbreaks of wound infection could be prevented if all
those suffering from active lesions, whether patients or members of staff, were isolated from the operating theatre and wards.

10. In the epidemic in the maternity hospital, the main sources of infection were the infants themselves and the infection spread from infant to infant.

11. Newborn infants, particularly if they are ill or premature, are unusually susceptible to staphylococcal infection, but the epidemic was primarily determined not by this, but by the appearance of the highly infective type 80 among them.

12. Type 80 in the maternity hospital showed a marked ability to cause severe and sometimes fatal disease and to spread rapidly as the predominant infecting strain. The high case incidence of severe disease was probably due to the greater susceptibility of neonatal infants to staphylococcal infection as compared to adults. It had low powers of colonisation — in the noses of the nurses and in the hospital environment generally.

13. A considerable amount of infection due to staphylococci acquired in the maternity hospital was discovered only
after the infants had been discharged home.

14. Conditions in most maternity hospitals, where babies are congregated into small nurseries or open wards, are held to be largely responsible for the high incidence of staphylococcal disease there and for the appearance of outbreaks of infection from time to time.

15. The spread of staphylococci from infant to infant and the consequent danger of the outbreak of serious infection could probably be prevented if babies were nursed beside their mothers in separate rooms.

16. Hospital staphylococcal outbreaks could be prevented by using the methods by which we successfully control other infectious diseases.
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