Investigations of Staphylococcal Infection Acquired in Great Britain's Hospitals

R. E. O. WILLIAMS, M.D.

In his brief account of the work on staphylococcal infection in British hospitals during the past 10 years, Dr. Williams offers the following observations:

"Practically all the staphylococci responsible for epidemics are resistant to penicillin and most are resistant to other antibiotics as well. It is possible that by now selection by antibiotic treatment has increased the proportion of virulent strains as well as of antibiotic resistant strains, but I do not think there is good evidence that the resistant strains of today are fundamentally more virulent than the sensitive strains of yesterday. If the use (or misuse) of antibiotics has increased the incidence of staphylococcal cross-infection, this is more likely to be due to our reliance on drugs instead of asepsis for preventing infection rather than to any malign effect of the antibiotics in selecting especially virulent staphylococci. If there were no cross-infection, the development of drug resistance would have a relatively limited importance."

IT IS convenient to start a review of British investigations on the hospital spread of staphylococcal infection by recalling the work of Devenish and Miles (*1*). They studied a series of postoperative infections in a surgical unit and were able to trace them quite clearly to the introduction of staphylococci, at the time of operation, by one surgeon who was a healthy nasal and skin carrier. This study was important not only because it showed the potential danger of a healthy carrier of staphylococci but also because it was the stimulus for a great deal of basic work on the frequency with which the cocci were carried by normal adults (*2*, *3*).

There was in Britain a great deal of interest in hospital infection during the war years. At that time the hemolytic streptococcus was regarded as the important cross-infecting organism, and there were at first only sidelong glances at the staphylococcus. The streptococcus offered a simpler problem than the staphylococcus has proved to be, even without considering antibiotics, because its rarity in fresh wounds and relative rarity in the respiratory tract made it immediately clear that cross-infection, not self-infection, must be involved. The work during the war was important in showing that streptococcal cross-infection of surgical wounds could be controlled by closing channels of contact infection, by no-touch dressing techniques, and by the maintenance of an aseptic routine in the wards as rigorous in its way as those already regarded as standard in the operating theater (*4*).

For the protection of most wounds from streptococci, attention to contact infection seemed to suffice. But with burns this was not enough, and Colebrook (*5*), in pioneer work at Birmingham in 1950, supplemented the no-touch dressing technique with chemoprophy-

Dr. Williams, director of the Streptococcus, Staphylococcus, and Air Hygiene Laboratory, Public Health Laboratory Service, London, England, delivered this paper at the National Conference on Hospital-Acquired Staphylococcal Diseases held in Atlanta, Ga., on September 15, 1958. *He wishes to acknowledge the benefit derived from numerous discussions with colleagues, especially Drs. R. Blowers, R. A. Shooter, O. M. Lidwell, and M. Patricia Jevons.*

laxis and the use of an air-conditioned room for performing the dressings.

At about the same time Miles and I were investigating infections in small industrial wounds (*6*). Sepsis was usually due to staphylococcal infection, and the staphylococci were very commonly those carried on the patient's skin before the infliction of the wound. We thought that in these small wounds streptococci indicated hospital infection, staphylococci self-infection.

Any idea that this distinction would prove generally true was soon shattered when it was observed that penicillin-resistant staphylococci were becoming increasingly common in surgical wards, and that the resistant strains differed in phage type from the sensitive strains previously infecting the patients and probably did not arise as variants of them (*7*). The fact that staphylococcal cross-infection in the wards became evident when the staphylococci had acquired the mark of drug resistance has led some to think that cross-infection is a new phenomenon, characteristic of the drug-fast strains. I do not think there is any reason to believe this, although reliable statistics of incidence for pre-antibiotic days, as for the present, are difficult to find and to interpret.

Prevalence of Hospital Infection

Determination of the prevalence of hospital infection is the first question that faces us today. Generally, the incidence of sepsis cannot be satisfactorily discovered from a review of ordinary hospital case records. Several hospitals have therefore devised special sepsis records. The surgical unit at St. Bartholomew's Hospital in London keeps a "wound book" in the operating room (*8*). In this book all operations are recorded and it is noted whether the wounds are healing satisfactorily or developing sepsis.

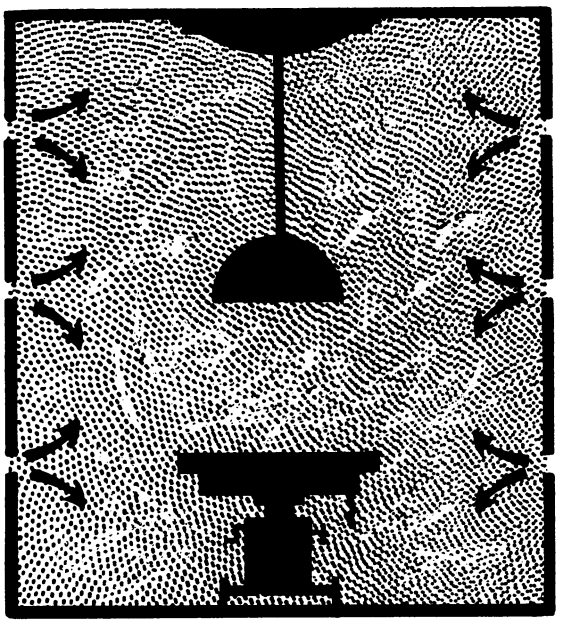
Elsewhere similar records have been kept in the wards (*9*). Many hospitals have systems for reporting cases of infection to the laboratory or to an infection control officer, usually the hospital's medical bacteriologist, whose function it is to keep a special watch on all aspects of infection throughout the hospital. It is our feeling that systems that simply require notification of cases of infection are less likely to give complete records than those that demand recording of the outcome, whether infection develops or not. Most hospitals now have an infection control committee, which includes representatives of all the important departments within the hospital, and in many cases also includes the medical officer of health of the district in which the hospital is situated.

The development of these systems is relatively new, and little information has yet been published on the prevalence of staphylococcal infection. For this reason the Public Health Laboratory Service in England and Wales has instituted a survey of postoperative infection in selected hospitals in different parts of the country. All clean operations performed by one surgical team in each hospital will be observed for 1 year, and the mode by which wounds heal will be assessed jointly by the surgeon and the bacteriologist. Preliminary results from three hospitals, for example, show from 5 to 9 percent of clean surgical wounds developing staphylococcal infection with clinical sepsis (B. Moore and R. J. Henderson in personal communications). The rates for some of the other hospitals are mostly about 5 percent.

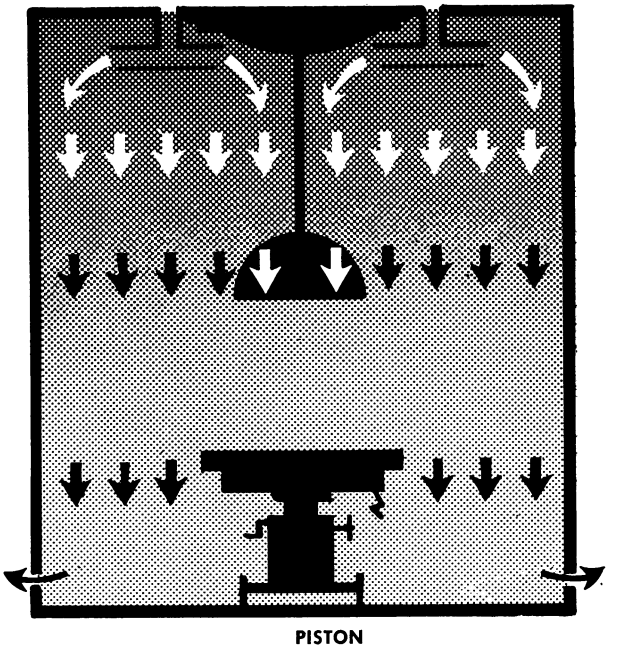
A number of surveys have also been made of the incidence, apart from epidemics, of skin sepsis in newborn babies. We made one such study in a maternity hospital 2 years ago and in a 6-month period found that 15 percent of the babies developed some staphylococcal lesion (conjuctivitis or skin sepsis) before discharge. Dr. M. H. Hughes, in an unpublished survey in a hospital in South Wales, found an incidence of 14 percent, but lower rates have been observed by others. The incidence of breast abscess is very difficult to determine because the disease so often develops after the patient's discharge from the hospital (even when this is at the 10th to 12th day as it is in Britain) and is treated by a different physician. Hughes found an incidence of 1 percent, and others have reported very similar rates (B. Wilkinson in a personal communication, 1958).

It seems that, even apart from recognized epidemics, 5 percent or more of clean operation wounds and 10–15 percent of newborn babies develop septic lesions due to staphylococci.

Ventilation in an Operating Room



CONVENTIONAL (turbulence)



PISTON

These figures are high enough, but to them must be added the quota from epidemics in which the attack rates may be much higher. The only measure we have of the frequency of such epidemics is the number of incidents from which staphylococci are sent for type identification. In 1957, the Staphylococcus Reference Laboratory at Colindale, London, received specimens from 30 outbreaks with 10 or more patients affected. The laboratory serves an area with a population of 13½ million, and it is sent material only from a proportion, perhaps a small proportion, of the epidemics that occur.

Nor do wound infections and infant sepsis exhaust the list of staphylococcal infections that are acquired in hospitals. Pneumonia is certainly not uncommon: we observed 5 cases of pneumonia in the course of a surgical ward epidemic having 17 cases of wound infection and an attack rate of about 2 percent in the same ward in a subsequent 8-month period free from epidemics (*10*, *11*). Urinary infections and skin lesions among adult patients are also seen.

Postoperative staphylococcal diarrhea has been observed in a number of hospitals, both in sporadic cases and in epidemics (*12*), but the incidence is too low to obtain a general attack rate.

In some epidemics, particularly those due to staphylococci of phage type 80/81, skin infections among the staff are very common (*13*).

Air Hygiene in the Operating Room

The institution of the "wound book" for recording the incidence of postoperative sepsis led Shooter and his colleagues to an elegant study on air hygiene in the operating room (*8*). Over a period of 8 months, 9 percent of clean surgical wounds developed postoperative sepsis, and the staphylococci from these patients fell into many different phage types. The operating room was at the top of the hospital and its ventilation was such that air was sucked into the room from the adjoining corridors and ultimately from the wards. It seemed likely that this air stream carried staphylococci from the wards into the operating room, where they could enter the wounds. This idea was confirmed when adjustment of the ventilation to provide a positive pressure of air within the operating room was followed immediately by a decrease of from 9 to 1 percent in the incidence of sepsis apparently due to theater infection. There was also a striking reduction in the number of bacteria found in the air of the operating room.

Blowers and his colleagues (*14*) had earlier studied an operating room in a thoracic surgi-

cal unit in which suction ventilation led to the contamination of air in the operating room with ward staphylococci. As a consequence of these studies and of the still earlier work of Bourdillon and Colebrook (*15*), it is now accepted that when operating rooms are built within the hospital, they need to be ventilated under positive pressure to exclude contaminated air issuing from the hospital. The rate at which the air within the room is changed must also be great enough to insure that any contamination liberated at one operation is cleared before the next operation is commenced. This usually implies a rate of 10–20 air changes per hour. Blowers has been investigating the best ways of getting the required rate of air changes with the least expenditure of energy, and he has shown that it is advantageous to bring the air in at the ceiling in such a way that it tends to descend through the room in a piston fashion, rather than by inducing turbulent mixing (see chart). This piston ventilation can be achieved by proper design of the air inlets and, in some circumstances, by using the incoming air as the principal source of heating for the room. The change from turbulent mixing to downward displacement may have another advantage. With a really steady piston effect it may be possible to protect the sterile area to some extent from contamination dispersed by the staff even within the operating room.

In several reports, Blowers has stressed how the number and activity of the staff in the operating room affect the bacterial count of the air. He showed, in one instance, that training the staff to avoid all unnecessary movement reduced the count as much as improving the ventilation did (*16*, *17*), though the source of contamination in the two cases is different. Blowers' observations and the recent work of Hare and his colleagues (*18*, *19*) on the dispersal of staphylococci from contaminated clothing of nasal and skin (especially perineal) carriers of staphylococci stress the importance of a rule that all persons working in the operating room should change all their clothing (which is not by any means general in Britain) or should wear some special protective suit as suggested by Duguid and Wallace (*20*).

The operating room air can be contaminated by ward staphylococci not only by air streams from the ward but also by blankets and the like used to cover the patient on his journey to the operation. It is best that the patient be covered with sterilized material sent to the ward from the operating room. If he has to be transferred from the operation table directly to his bed, this should not be done within the operating room itself.

Carriers in the Operating Room

Airborne infection in the operating room is probably not uncommon, but probably it is not often responsible for epidemics of sepsis. Several epidemics of operating-room sepsis have been traced to staff members who were dangerous staphylococcal carriers. In some instances the carrier had a septic lesion as did the surgeon described by McDonald and Timbury (*21*); in others the carrier was apparently quite healthy (*1*, *22*). Knowing how widespread the staphylococci are on the skin of anyone with a septic lesion and knowing the frequency of accidental glove-puncture during surgery, it is not surprising that a surgeon with a boil infects the patients on whom he operates. The healthy carriers present a much greater problem, because we know that some 50 to 70 percent of all hospital staff carry staphylococci in the nose and 20–40 percent carry them on the skin. Why then do they not cause epidemics more often? It may be that some carriers are qualitatively different from others: the surgeon described by Devenish and Miles (*1*) seems to have been a particularly profuse carrier on the skin of his forearm. Or it may be that the staphylococcus has to be virulent: the carrier described recently (*10*) certainly had a staphylococcus of more than average virulence, as judged from its behavior in the ward.

Carriers in the Wards

The peculiar ability of some individuals to spread their staphylococci has also been recognized as responsible for the spread of infection in wards. In maternity departments there have been several outbreaks of infection (*23*, *24*) in which a single nurse who was a carrier of the epidemic type of staphylococcus seemed to be responsible for many cases of infection, and Jellard (*25*) has recently referred briefly to one

such nurse who started outbreaks in several wards in which she worked.

In our own studies in a surgical ward at St. Bartholomew's Hospital (*10*, *11*), we thought that we could distinguish particular patients who were actually or potentially dangerous. For example, it seemed very likely that a sharp epidemic in 1956 was started by a patient with a staphylococcal pneumonia; he was probably the source of several ward infections and he may well have infected the member of the surgical team who became a carrier and certainly infected many patients at operation.

In later studies, we recognized a number of patients who were especially apt to disperse their staphylococci. In some cases there seemed to be a good reason for this aptitude, such as urinary infection with incontinence or pneumonia with a tracheotomy. But the individual who contaminated air to the greatest degree was a healthy carrier.

We were so impressed by the ability of some infected patients to disperse their staphylococci that we rigorously isolated in a separate room any patient infected by what appeared to be a dangerous staphylococcus, which we provisionally took to be one resistant to tetracycline. The physical isolation was, of course, supplemented by a very strict isolation-nursing technique, great care being taken that no material that had been near the patient was used in the ward without sterilization. On 6 separate occasions in 2 years of this regimen we have been able to isolate all patients with septic lesions as well as healthy carriers known to be infected with supposedly dangerous types. Only once did infection spread to another patient, probably because of a breakdown in the isolation. On one occasion when we isolated the septic patients but had insufficient space to isolate all the carriers, two secondary cases occurred; the strain was of phage type 80, which is known to be very communicable.

The isolation nursing of infected surgical patients has in general been curiously neglected; yet, when we see the extent to which infected patients can contaminate the ward, it surely seems ridiculous to nurse them among patients with susceptible wounds that have to be redressed daily. One of the most valuable contributions that hospital administrators can make to the solution of cross-infection may well be the provision of really adequate isolation facilities in all wards, sufficiently equipped to simplify the routine of isolation nursing.

Spread of Infection in Maternity Wards

Although infection is commonly introduced into a maternity ward by a nurse who is a carrier, it seems clear that subsequent spread is often from one baby to another. In seeking the route of this spread, some have stressed the umbilical stump as a reservoir. It recently became fashionable to leave the stump uncovered, and it was easy to show that in these circumstances the stump became contaminated with staphylococci as or more quickly than the nose. Jellard (*26*) found that if the umbilical stump was painted daily with an antiseptic dye, the staphylococcus seemed to spread less readily through the nursery. Her studies, however, were confined to the rate at which newborn babies acquired bacteria. Gillespie (*27*) has had a similar experience with the use of a hexachlorophene dusting powder for the umbilicus. Cook, Parrish, and Shooter (*28*) studied a variety of nursing techniques for their effect on the rate of nasal colonization. Although they could not reduce the rate greatly by any method, they found the best to be the reservation of individual clean gowns for the nurse's use when handling each baby, coupled with the application of antiseptic dye to the umbilical stump. We have recently found that air disinfection with ultraviolet irradiation had no effect on the nasal colonization rate, but that daily bathing with soap containing hexachlorophene might be of some use. Forfar and MacCabe (*29*) could detect no effect on the incidence of minor sepsis when the nurses in one of two nurseries gave up wearing special gowns and masks.

Environmental Contamination

To the bacteriologist, the ease with which staphylococci can be isolated from ward dust and from bedding and curtains is inescapable. It seems inconceivable that such heavily contaminated material should not be a reservoir for the spread of infection to patients. This thought has prompted many attempts to eliminate the contamination. The wool blanket

offers the greatest problem, for dust and fluff are readily dispersed from its surface, and ordinarily it is laundered at too low a temperature to kill staphylococci. Indeed washing is so harmful to blankets that it has commonly been practiced as rarely as possible.

As a legacy from earlier work on streptococcal infection we had the method of oil impregnation of wool blankets to prevent dispersal of the bacteria, without killing them (*30*). Later Blowers and Wallace (*31*), following earlier work by Rountree and by Barnard, devised a simple method for disinfecting wool blankets during laundering, using a nonionic detergent for washing and a cationic detergent for disinfection. This process, which effectively kills staphylococci—though not *Pseudomonas pyocyanea* or *Mycobacterium tuberculosis*—and which does little harm to the blankets, has been quite widely adopted. Other methods are also being investigated by which wool blankets can be disinfected safely.

But clearly a blanket that could withstand the laundry temperatures used for linen and cotton material would have great advantages. Blowers, Potter, and Wallace (*32*) tested three materials: loose woven cotton, thick toweling, and Terylene, all of which can be boiled. Both cellular cotton and the toweling make good blankets and can be recommended.

It is therefore perfectly possible to provide patients with sterilized bedding, but it is still not known just how much good we may expect to do by this. The effect of sterilized bedding on the air contamination in the wards has been tested somewhat, but there is very little work yet on the incidence of sepsis. Nevertheless, the general view seems to be that it is undesirable to harbor in the ward a large pool of staphylococci on blankets and curtains and that their regular disinfection is likely to reduce the incidence of sepsis.

We have made several studies of the bacterial content of the air of a surgical ward (*11*). We found a basal level of about 0.1 particle containing *Staphylococcus aureus* per cubic foot. Much higher counts (0.5 to 5.0 particles per cubic foot) were found, even during relatively quiet periods in the ward, when one of the patients was an active disperser, but such high counts were not constantly associated with the occurrence of cases of sepsis due to spread within the ward, although it is true that a peak occurred whenever spread took place.

Treatment of the Carrier State

The inanimate hospital is an enormous reservoir of staphylococci derived from infected patients and from carriers, but staphylococci do not multiply in dust or on bedding and it seems more rational to attempt to control dispersal by an attack on the breeding places rather than on the resting places. Gould (*33, 34*) has made a series of studies of the antibiotics and disinfectants applied to the nose to eliminate the carrier state. He finds that applying a cream containing, for example, 0.5 percent neomycin and 1 percent chlorhexidine (Hibitane) for 7 to 14 days rids most carriers of detectable nasal staphylococci for a period of a few weeks. This method used by several workers has often been successful for treating nurses who have become carriers during an epidemic (*35*), but some carriers seem to be quite resistant to this form of treatment, even though their bacteria are sensitive to the antibiotic in vitro. Gould and Allan (*36*), working on the assumption that hospital infection with staphylococci was derived from carriers among the staff, treated all the staphylococcal nasal carriers on the staff of a small hospital with a tetracycline cream for 1 week and found that the incidence of hospital infection decreased strikingly during the period following this treatment. On the other hand, Gillespie (*27*) has proceeded on the assumption that the patients are (or become) nasal carriers and infect their own wounds. He therefore applied an antibiotic cream to the patients' noses from the time of their admission to the ward and throughout their stay, and the frequency with which staphylococci were isolated from open wounds fell from about 15 to about 3 percent. The incidence of clinical sepsis was not reported.

Clearly, these two concepts need further study. In our first year's work in the surgical ward at St. Bartholomew's, Dr. Shooter and I could find no evidence that the wound infections were derived from either the patients' or the staff's noses; infection seemed to be from one infected patient to another. On the other hand, during the past year we have studied 15

patients who developed postoperative sepsis, and 7 of them were nasal or skin carriers of staphylococci before the operation. One might think that when we reach the stage at which we can attribute so much of the postoperative infection to the patients' own preoperative staphylococci we may be nearing our goal. But we need to beware, for patients who are in hospitals for any time before an operation often become nasal carriers of the hospital staphylococcus (*37*) and this hospital strain may be the one that infects the wound.

The records of the staphylococci sent to us at Colindale for typing reveal an interesting fact on the frequency of carriers among the staff. In 4 years we typed material from staff and patients in 94 separate epidemics. Altogether about 18 percent of the staff carried the type of staphylococcus that was locally epidemic, but there was a striking difference between epidemics due to staphylococci of phage group 1, with some 25 percent of the staff who were carriers, and those of other phage groups, with 3–13 percent carriers (see also *38*). The notorious type 80 (or 80/81) did not differ in this respect from other types in phage group 1. Apart from type 80, which occurs in all sorts of hospitals, phage group 1 strains are not often epidemic outside maternity hospitals. The results suggest that widespread nasal carriage of the epidemic strains among hospital staffs is not common enough to justify general nasal disinfection as a routine and that it is more likely to be relevant in maternity hospital outbreaks than in outbreaks in surgical wards, unless the infecting strain is type 80.

Chemoprophylaxis and chemotherapy of the wounds have been widely used, but too rarely precisely assessed; and only in the management of burns has a serious attempt been made to integrate antibiotic treatment with the work on the prevention of cross-infection (*39*). There is a great need for more detailed work to discover just what can be prevented by prophylaxis, and to what extent treatment can, at least, prevent an infected wound from being a dangerous source of cross-infection.

Epidemic Types of Staphylococcus aureus

It used to be thought that all strains of coagulase-positive staphylococci were similar in virulence, but this idea can certainly be held no longer. On the other hand, the recent worldwide spread of one type known as 80, or 80/81 (or 52/44A/42C/47C, and the like), has led some to think that it is only this strain that is important and strains of all other types can be neglected. This idea is likewise quite untenable.

Our studies in the surgical wards emphasized the different capabilities of different strains of staphylococci. From all sites in the ward, in an 8-month survey, some 186 different strains of *Staphylococcus aureus* were isolated, but only 13 of these caused disease, and only 3 caused disease in more than 1 patient. Some strains seemed to have remarkably little virulence. One was present in the air of the ward, often in large numbers, for 6 months, but never caused a secondary case of sepsis and only colonized 3 patients' noses. Another was present in the ward for 8 months without causing any secondary cases of sepsis, although it colonized the noses of 57 persons. In contrast, the strain present in the same ward in the previous year caused 34 cases of wound sepsis and other disease in a 2-month period. It was widespread in the air and dust, but it colonized only two noses.

I have made an analysis of the phage types of staphylococci from septic lesions sent to Colindale in the 4 years 1954–57. Altogether we had 1,131 independent strains (counting all the strains from any one epidemic as one strain), 638 from surgical units and 493 from maternity units. A great many different types could be recognized, but some 20 types or groups of closely related types were each represented by 10 or more strains. Among the 1,131 independent strains, 178 were, in the particular time and place, epidemic strains, and 69 percent of these were found in no more than 7 types. Type 80 had the highest proportion of epidemic strains. This type was equally common in maternity and surgical units; but other common epidemic types were 52A/79 and 71 in maternity units and 75/77, 47/53/75/77, and 7/47/53/54/75 in surgical units.

In Britain, as elsewhere, type 80 has spread rapidly in the last few years; at Colindale we had specimens from 5 or 6 epidemics in each of the 3 years 1954–56, and from 21 epidemics in

1957. Thirty percent of all the strains that we had from septic lesions in 1957 proved to be type 80, although this figure may be somewhat inflated by the general interest shown in this particular type. But even now, quite extensive epidemics due to other phage types are being seen.

Practically all the staphylococci responsible for epidemics are resistant to penicillin and most are resistant to other antibiotics as well. It is possible that by now selection by antibiotic treatment has increased the proportion of virulent strains as well as of antibiotic resistant strains, but I do not think there is good evidence that the resistant strains of today are fundamentally more virulent than the sensitive strains of yesterday. If the use (or misuse) of antibiotics has increased the incidence of staphylococcal cross-infection, this is more likely to be due to our reliance on drugs instead of asepsis for preventing infection, rather than to any malign effect of the antibiotics in selecting especially virulent staphylococci. If there were no cross-infection, the development of drug resistance would have a relatively limited importance.

Conclusion

Although some progress seems to have been made in understanding what happens in hospitals, it is clear that much remains to be done. In the laboratory, especially, factors related to virulence need to be explored and in the wards all the numerous measures that are advised for the prevention of cross-infection need to be more precisely tested. For it is certain that if we prescribe too many rules their observance will be neglected.

In my opinion there is no one way in which staphylococci spread in a hospital, and there is no one prophylactic method by which spread can be prevented. The routes of infection are numerous and probably often devious, and the precautions needed are many and often complex.

Staphylococcal hospital infection is an infectious disease, with this subtlety, that while most staphylococci are, when given the best opportunity, able to produce septic lesions, relatively few of them seem able to produce epidemics. Probably all hospitals have their endemic level of staphylococcal infections, while some, from time to time, suffer epidemics. Our preventive measures have therefore to minimize the endemic level, to prevent the emergence of epidemics, and to terminate epidemics when they occur.

It seems probable that an epidemic is usually started when an especially virulent staphylococcus is introduced into a hospital by someone who is able to disperse it readily. If we are prepared to try, it should not be too difficult to recognize people who are likely to disperse the bacteria, but we have at present no satisfactory measure of the virulence of staphylococci other than the retrospective record of what a particular strain has achieved. There is some correlation of epidemicity with phage type, but we know of many introductions of well-known virulent types into hospitals with no subsequent spread. Perhaps this means that they were not being dispersed sufficiently, or very likely there may be variations of virulence within the type. The combination of two factors, virulence and dispersal must be present; but we must hope that these two, although necessary, are not sufficient. Our preventive measures ought to be such that we can limit the spread of virulent strains, even from the profuse dispersers.

When an epidemic has started, the best approach to control it is to attempt the elimination of the epidemic strain from the hospital. This means, first, definition of the type of staphylococcus that is causing infection in the patients, and, second, a search for carriers of this type in the hospital personnel or patients. Sometimes such a search will reveal the one dangerous carrier whose exclusion terminates the epidemic. Often the search will reveal several carriers. Even though it may be possible to judge from epidemiological analysis that one is important in an epidemic of any severity, it is worth treating or excluding all of them. These searches, with the examination of fomites and the typing of all staphylococci, will often exceed the facilities of the hospital laboratory. Outside agencies, such as public health laboratories, should be equipped and ready to help in these investigations, and their help should be readily sought by the hospitals.

The prevention of outbreaks would be greatly

helped by a laboratory test of virulence so that the potential initiators of epidemics could be isolated. Otherwise, it seems that our only hope is to regard all persons, whether surgical patients, babies, or staff, with staphylococcal lesions as having virulent strains, and to treat them in strict isolation as one would cases of infectious disease. It would also be wise, when possible, to isolate known carriers of strains resistant to many antibiotics. The hospital also needs to have a recording system sensitive enough to recognize quickly any increase in the prevalence of infection and someone with clinical authority to scrutinize the records and initiate the investigations that may be able to stem an epidemic.

Of general preventive measures, the aseptic handling of patients should receive the greatest stress. Every staff member's approach to the patient must be informed by a realization of the infectiousness of staphylococcal infection. Everything taken from the infected patient must be sterilized, and all material used for the treatment of all patients must not only be sterilized but must be kept sterile right up to the time that it is used. The provision of proper facilities and equipment is an essential contribution from the administrators; the rapid and detailed recognition of the infecting bacteria is the duty of the laboratory; but there are no gadgets and no drugs or vapors that can relieve the people who handle the patients of their perennial responsibility for handling them aseptically.

REFERENCES

(*1*) Devenish, E. A., and Miles, A. A.: Control of *Staphylococcus aureus* in an operating theatre. Lancet 236: 1088–1094, May 13, 1939.

(*2*) Gillespie, E. H., Devenish, E. A., and Cowan, S. T.: Pathogenic staphylococci. Their incidence in the nose and on the skin. Lancet 237: 870–873, Oct. 21, 1939.

(*3*) Williams, R. E. O.: Skin and nose carriage of bacteriophage types of *Staphylococcus aureus*. J. Path. & Bact. 58: 259(1946).

(*4*) Williams, R. E. O., Clayton-Cooper, B., Howat, T. W., and Miles, A. A.: The control of hospital infection of wounds. Brit. J. Surg. 32: 425 (1944–1945).

(*5*) Colebrook, L.: A new approach to the treatment of burns and scalds. London, Fine Technical Publications, 1950, 174 pp.

(*6*) Williams, R. E. O., and Miles, A. A.: Infection and sepsis in industrial wounds of the hand. A bacteriological study of aetiology and prophylaxis. Medical Research Council, Great Britain, Spec. Rep. Ser. No. 266. London, His Majesty's Stationery Office, 1949.

(*7*) Barber, M.: Coagulase-positive staphylococci resistant to penicillin. J. Path. & Bact. 59: 373–384, July 1947.

(*8*) Shooter, R. A., Taylor, G. W., Ellis, G., and Ross, J. P.: Post-operative wound infection. Surg. Gynec. & Obstet. 103: 257–262, September 1956.

(*9*) Jeffery, J. S., and Sklaroff, S. A.: Incidence of wound infection. Lancet 272: 365–368, Feb. 15, 1958.

(*10*) Shooter, R. A., Griffiths, J. D., Cook, J., and Williams, R. E. O.: Outbreak of staphylococcal infection in a surgical ward. Brit. Med. J. No. 5016: 433–436, Feb. 23, 1957.

(*11*) Shooter, R. A., Smith, M. A., Griffiths, J. D., Brown, M. E. A., Williams, R. E. O., Rippon, J. E., and Jevons, M. P.: Spread of staphylococci in a surgical ward. Brit. Med. J. No. 5071: 607–613, Mar. 15, 1958.

(*12*) Cook, J., Elliott, C., Elliott-Smith, A., Frisby, B. R., and Gardner, M. M. N.: Staphylococcal diarrhoea. With an account of two outbreaks in the same hospital. Brit. Med. J. No. 5018: 542–547, Mar. 9, 1957.

(*13*) Duthie, E. S.: Generalized staphylococcal epidemics in a hospital group. *In* Hospital coccal infections. Symposium of the Association for Clinical Pathology and Medical Research Council. London, 1957.

(*14*) Blowers, R., Mason, G. A., Wallace, K. R., and Walton, M.: Control of wound infection in a thoracic surgery unit. Lancet 269: 786–794, Oct. 15, 1955.

(*15*) Bourdillon, R. B., and Colebrook, L.: Air hygiene in dressing rooms for burns or major wounds. Lancet 250: 561, Apr. 20, 1946; 601, Apr. 27, 1946.

(*16*) Blowers, R.: Theatre hygiene and ventilation. *In* Hospital coccal infections. Symposium of the Association for Clinical Pathology and Medical Research Council. London, 1957.

(*17*) Blowers, R.: Wound infection in operation theatres. *In* Medical annual. Bristol, John Wright & Sons, Ltd., 1958, pp. 21–32.

(*18*) Hare, R., and Thomas, C. G. A.: The transmission of *Staphylococcus aureus*. Brit. Med. J. No. 4997: 840–844, Oct. 13, 1956.

(*19*) Hare, R., and Ridley, M.: Further studies on the transmission of *Staphylococcus aureus*. Brit. Med. J. No. 5062: 69–73, Jan. 11, 1958.

(*20*) Duguid, J. P., and Wallace, A. T.: Air infection with dust liberated from clothing. Lancet 255: 845–849, Nov. 27, 1948.

(*21*) McDonald, S., and Timbury, M. C.: Unusual outbreak of staphylococcal post-operative wound infection. Lancet 273: 863–864, Nov. 2, 1957.

(22) Penikett, E. J. K., Knox, R., and Liddell, J.: An outbreak of post-operative sepsis. Brit. Med. J. No. 5074: 812–814, Apr. 5, 1958.
(23) Knott, F. A., and Blaikley, J. B.: The control of staphylococcus infections in a maternity department. J. Obstet. & Gynaec. Brit. Empire 51: 386–400, October 1944.
(24) Gillespie, W. A., Pope, R. C., and Simpson, K.: Pemphigus neonatorum caused by *Staphylococcus aureus* type 71. Brit. Med. J. No. 5025: 1044–1046, Apr. 27, 1957.
(25) Jellard, J.: Cross-infection with *Staphylococcus aureus* in a maternity unit. *In* Hospital coccal infection. Symposium of the Association for Clinical Pathology and Medical Research Council. London, 1957.
(26) Jellard, J.: Umbilical cord as reservoir of infection in a maternity hospital. Brit. Med. J. No. 5024: 925–928, Apr. 20, 1957.
(27) Gillespie, W. A.: Hospital cross-infection. Med. J. South West 73: 56–68, July 1958.
(28) Cook, J., Parrish, J. A., and Shooter, R. A.: Acquisition of *Staphylococcus aureus* by newborn babies in a hospital maternity department. Brit. Med. J. No. 5062: 74–76, Jan. 11, 1958.
(29) Forfar, J. O., and MacCabe, A. F.: Masking and gowning in nurseries for the newborn infant. Effect on staphylococcal carriage and infection. Brit. Med. J. No. 5062: 76–79, Jan. 11, 1958.
(30) Clarke, S. K. R., Dalgleish, P. G., Parry, E. W., and Gillespie, W. A.: Cross-infection with penicillin-resistant *Staphylococcus aureus*. Effect of oiling floor and bed-clothes in a surgical ward. Lancet 267: 211–215, July 31, 1954.
(31) Blowers, R., and Wallace, K. R.: The sterilization of blankets with cetyl trimethylamine bromide. Lancet 268: 1250–1251, June 18, 1955.
(32) Blowers, R., Potter, J., and Wallace, K. R.: Clean beds. Lancet 272: 629–631, Mar. 23, 1957.
(33) Gould, J. C.: The effect of local antibiotic on nasal carriage of *Staphylococcus pyogenes*. J. Hyg., London 53: 379–385, September 1955.
(34) Gould, J. C.: The control of carriers of *Staphylococcus aureus*. *In* Hospital coccal infections. Symposium of the Association for Clinical Pathology and Medical Research Council. London, 1957.
(35) Gillespie, W. A., and Alder, V. G.: Control of an outbreak of staphylococcal infection in a hospital. Lancet 272: 632–634, Mar. 23, 1957.
(36) Gould, J. C., and Allan, W. S. A.: *Staphylococcus pyogenes* cross-infection. Prevention by treatment of carriers. Lancet 267: 988–989, Nov. 13, 1954.
(37) Clarke, S. K. R.: Nasal carriage of *Staphylococcus aureus*. J. Path. & Bact. 73: 253–259, January 1957.
(38) Alder, V. G., Gillespie, W. A., and Thompson, M. E. M.: Virulence and phage patterns of antibiotic-resistant staphylococci in a hospital. J. Path. & Bact. 70: 503, October 1955.
(39) Lowsbury, E. J. L.: Cross-infection of wounds with antibiotic resistant organisms. Brit. Med. J. No. 4920: 985–990, Apr. 23, 1955.

Sanitary Engineering Courses in Radiation and Water Analysis

A course in sanitary engineering aspects of nuclear energy will be conducted at the Robert A. Taft Sanitary Engineering Center, Public Health Service, in Cincinnati, Ohio, December 1 through 12, 1958. Designed for engineers and scientists in public health, especially those in supervisory posts, the course covers the broad aspects of radiological health.

The program opens with a discussion of radiation fundamentals and instrumentation, then focuses on radiation protection, nuclear reactor operations, and measurement and evaluation of environmental radiation contamination.

During the same 2-week period, the center will give a course in chemical analyses for water quality, for graduate chemists and professional people with extensive background in water supply and water pollution control.

Among subjects covered are: measurement of strength and effect of oxygen-demanding wastes; investigation of toxic industrial wastes, including the determination of toxicity through bioassay; characterization of synthetic organic wastes; water supply problems; and survey and administration, including basic data program.

Applications may be obtained from the Chief, Training Program, Robert A. Taft Sanitary Engineering Center, 4676 Columbia Parkway, Cincinnati 26, Ohio, or from a Public Health Service regional office.