J. Hyg., Camb. (1963), **61**, 95
Printed in Great Britain

A study of post-operative wound infection in a provincial general hospital

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(*Received* 16 *November* 1962)

INTRODUCTION

Between 1957 and 1959, staphylococcal infection in patients and nursing staff became an increasingly serious problem at the Torbay Hospital, Torquay. A preliminary survey in 1957 showed that staphylococci of the 80 phage group were widely disseminated in the main male surgical ward of the hospital, causing wound sepsis in patients and a high incidence of styes, boils and carbuncles in the nursing staff. During the ensuing year, this high incidence of infection persisted throughout the hospital, and initial efforts to deal with the problem were no more successful than those recorded from another provincial hospital about this time by Williams, Talbot & Maughan (1959). Absenteeism in the nursing staff because of skin sepsis raised serious staffing problems, the incidence of sepsis in the nursing staff being of the same order as that recorded at the London Hospital in 1958–59 by Davies (1960).

Early in 1959, as one of a number of measures to combat infection, a whole-time Infection Control Sister was appointed at the hospital (Gardner, Stamp, Bowgen & Moore, 1962). One of her earliest tasks was to organize, as part of a more comprehensive survey by a Public Health Laboratory Service (P.H.L.S.) committee (Public Health Laboratory Service, 1960), a clinical and bacteriological study of all surgical patients undergoing certain categories of operative procedure in the hospital during the first week of April, 1959. Although this limited study was all that the P.H.L.S. committee required, a more extended survey seemed desirable as a basis for infection control. Accordingly, the methods used in the initial survey were applied to a study of post-operative infection in surgical patients admitted to the Torbay Hospital in three alternate approximately quarterly periods between 1 April 1959 and the end of June 1960. The decision to make the survey an intermittent one was taken, partly to limit the necessary laboratory, clinical and clerical labour, and partly so that the Infection Control Sister could devote her energies to special problems during the intervening months.

During the period covered by the three surveys, no change occurred in the consultant surgical personnel of the hospital, in the range of operations performed, nor indeed in the general level of wound sepsis. The results of the three surveys have therefore been combined for analysis below, except where otherwise stated.

The 559 patients studied were admitted under one or other of three general surgeons, a gynaecologist or an orthopaedic surgeon. The great majority were operated on by the consultant surgeon concerned, but some patients were operated on by a surgical registrar and a few by a house surgeon.

MATERIAL AND METHODS

Selection of patients

As in the earlier P.H.L.S. study, the scope of the present surveys embraced all patients whose operation necessitated an incision through healthy skin, except those operated on primarily because of local sepsis, e.g. to incise an abscess or drain an empyema. Operations on the lower urinary tract, rectum and anus and on accidental wounds were excluded. All emergency abdominal operations were included, even though some were found to entail drainage for sepsis, e.g. an appendix abscess.

Routine investigation and records

Each patient had a nasal swab taken on the day of admission to hospital. When possible, this swab was taken before the patient reached the ward, because, particularly during the first quarterly period, a number were found to harbour staphylococci of type 80 or related types in the nose on their arrival in hospital. The original intention was that, when a week or more elapsed before the operation, the nose should be swabbed again on the day of operation. Because of acute bed shortage, however, patients were for the most part admitted to the hospital on the day before operation, and, of those included in the present study, some 80 % were operated on within 48 hr. of admission, and 95 % in less than a week. An immediate pre-operative swab was thus taken from very few patients, except during the second survey, when 106 patients were swabbed both on admission to the hospital and in the operating theatre, to see whether many of those who were not carrying staphylococci in the nose on admission became established carriers during the brief pre-operative period.

All wounds were inspected by the Infection Control Sister at the time of the first dressing, and wound swabs were taken for bacteriological examination. Dressings were, of course, not disturbed merely to permit swabbing. Where evidence of sepsis was observed, the Control of Infection Officer (A.M.N.G.) inspected the wound with the sister. Further wound swabs were collected weekly, or more often if necessary. When the patient was discharged to an outlying hospital, the sister kept in touch with the hospital matron and arranged further swabbing, or herself visited the hospital if this was thought desirable. Many patients had a final check when they paid their last visit to the out-patient clinics.

During the first month of the first survey, in April 1959, the clinical course of wound sepsis in a number of patients strongly suggested a theatre infection. Nasal swabs were therefore taken during the last week of April from all members of the theatre staff, both medical and nursing. More recently, a standard policy of swabbing theatre personnel once every 2 months has been adopted, but unfor-

tunately no further swabbing was done during this study until March 1960. Apart from the initial stages of the survey, therefore, no adequate information was available on the possible association of members of the theatre staff with wound sepsis in patients, unless they themselves were sent off duty with septic infections and thus came under closer scrutiny.

Bacteriological and clinical details on all patients were entered in a special book, and the details were in due course transferred to 6 × 4 in. Cope.-Chat. punch cards for analysis.

All swabs were sent by overnight post to the Public Health Laboratory at Exeter, as the hospital laboratory was too busy to cope with the amount of work required. When urgent bacteriological investigation was necessary in the interests of individual patients, replicate swabs were sent down to the hospital laboratory for immediate culture, and a few routine swabs collected at week-ends from patients admitted as emergencies were similarly examined at the hospital.

Laboratory methods

Nasal and wound swabs were cultured for *Staphylococcus aureus* and other pathogens by direct plating on blood agar and by broth enrichment and subculture. Wound swabs were in addition plated on to an electrolyte-deficient medium (Sandys, 1960) that inhibited the swarming of *Proteus* strains, while permitting good growth of staphylococci and of coliform organisms.

All coagulase-positive staphylococcal strains were phage-typed by the methods of Williams & Rippon (1952). The battery of phages available at the time did not include phage 81. Most of the strains described in this report as belonging to type 80 would probably now be designated 80/81.

Sensitivity tests against penicillin, streptomycin, tetracycline and chloramphenicol were done on most of the wound strains of *Staph. aureus*, on epidemiological rather than clinical grounds, and, during the second and third surveys, staphylococci were also examined for mercury sensitivity (Moore, 1960).

RESULTS

General findings

Of the 559 surgical patients investigated in the course of the three quarterly surveys, 71 (12·7 %) developed clinical evidence of post-operative wound sepsis and 51 (9·1 %) definite suppuration. The number of patients with proved staphylococcal sepsis was 48 (8·6 %).

That the incidence of wound infection remained fairly uniform throughout can be seen from Table 1, in which the findings of the three surveys are shown separately. Although no statistically significant change occurred during the period of the study in the incidence of total wound sepsis, wound suppuration or staphylococcal sepsis, the figures suggest a slowly diminishing incidence of staphylococcal sepsis, but this fall was more than offset by the increase in wound sepsis due to coliform organisms. Examination of individual patients' records shows that the increase in coliform sepsis can be largely accounted for by an 'epidemic' of

appendix abscesses that occurred during the third survey and cannot be cited as another instance of the increasing importance of hospital infection due to Gram-negative organisms.

In all, 17 patients (3%) died in hospital during the post-operative period. Although 5 of these had septic wounds, the sepsis did not appear to be related to the cause of death.

Table 1. *Incidence of post-operative wound sepsis in the three Torbay Hospital surveys*

	1 Apr.–June 1959	2 Oct.–Dec. 1959	3 Apr.–June 1960
Total no. of patients	191	218	150
No. of septic wounds	21 (11)	28 (12·8)	22 (14·7)
No. of suppurating wounds	17 (8·9)	20 (9·2)	14 (9·3)
No. of wounds with sepsis due to:			
Staph. aureus	18 (9·4)	19 (8·7)	11 (7·3)
Coliforms	1 (0·5)	5 (2·3)	10 (6·7)

Figures in parentheses are percentages.

Analysis of sepsis due to staphylococci

Of the 48 wounds in the survey that became clinically septic and yielded staphylococci on culture, 30 were infected with organisms of the 80 group, mainly type 80 or type 52/52A/80, but including two strains of type 29/52/80. Fourteen of the 80 group infections occurred during the first survey, 9 in the second, and 7 in the third.

During the second survey, 4 apparently unrelated patients who developed wound sepsis, in November 1959, were all infected with a mercury- and antibiotic-resistant type 6/7/47/53/54 strain.

Preliminary analysis of the punch card records brought the quite unexpected finding that post-operative wound sepsis had occurred far less frequently in patients who were staphylococcal nasal carriers on admission to hospital than in non-carriers. This finding seemed so much at variance with the importance ascribed to the nasal carrier state in relation to hospital sepsis (Weinstein, 1959; McNeill, Porter & Green, 1961; Williams *et al.* 1962) that the relationship of staphylococcal nasal carriage to wound infection in the present study has been critically analysed below. An attempt has also been made to assess the incidence of self-infection, and of wound infection directly ascribable to infection by a member of the theatre staff.

Relationship of nasal carriage to wound sepsis

Of the 559 patients investigated, 538 had nasal swabs taken on admission to hospital. The incidence of subsequent staphylococcal sepsis according to the initial carrier state is shown in Table 2. Of the 153 patients who were carriers on admission, 9 (5·9%) developed post-operative staphylococcal sepsis, while the corresponding

number among the 385 who were initially non-carriers was 36 (9·4 %). The 9 septic patients in the carrier group included, as discussed below, at least 3 whose wound infection was very probably due to the staphylococcus they carried on admission. If these are excluded, the figures for the incidence of sepsis due to staphylococci acquired in hospital become 6/153 (3·9 %) for carriers and 36/385 (9·4 %) for non-carriers.

Table 2. *Incidence of staphylococcal sepsis in patients who were nasal carriers on admission to hospital and in non-carriers*

	Carriers	Non-carriers
Total no.	153	385
No. of wounds with staphylococcal sepsis	9 (5·9 %)	36 (9·4 %)
Septic wounds with staphylococci acquired in hospital	6 (3·9 %)	36 (9·4 %)

Table 3. *Incidence of staphylococcal sepsis according to sex and carrier state on admission to hospital*

	Total no.	Wounds with staphylococcus sepsis
Males		
Carriers	46	3 (6·5 %)
Non-carriers	159	16 (10·1 %)
Females		
Carriers	108	6 (5·6 %)
Non-carriers	225	20 (8·9 %)

The unexpected excess of wound sepsis in non-carriers raised the question whether there might not be some material difference between the carrier and the non-carrier group that made them not truly comparable. One such difference was in the sex distribution in both groups. The staphylococcal carrier rate on admission to hospital was found to be somewhat lower in males than in females, and thus the proportion of males in the carrier group (30·6 %) was lower than among the non-carriers (42 %). The incidence of sepsis according to sex and carrier state was therefore calculated and is shown in Table 3.

The table shows a remarkable similarity between the sexes in the relative excess of sepsis in non-carriers. The difference in sepsis incidence can thus not be explained in terms of differences in sex distribution in the carrier and non-carrier groups.

Another possibility was that the nature of the operations undergone by the carrier and non-carrier groups happened to differ significantly, so as to make the non-carriers more susceptible to wound sepsis, not by virtue of their non-carrier state, but because the operations done on them were more conducive to subsequent wound sepsis than those done on the carrier group. This again was excluded as an adequate explanation of the differences found. The percentage composition of the carrier and non-carrier groups in respect of 14 major categories of operative procedure is shown diagrammatically in Fig. 1. The only major difference between the

two groups was in the number of hernia operations, which totalled 47 (13 %) in the non-carrier group and only 9 (5·9 %) in the carrier group. As, however, the incidence of sepsis after hernia was far lower than for the series as a whole, this difference between the two groups would tend to under-estimate the difference in sepsis rates rather than exaggerate it.

Again, there was the possibility that, for some reason or other, the wounds of non-carriers had been exposed to a heavier load of staphylococcal contamination

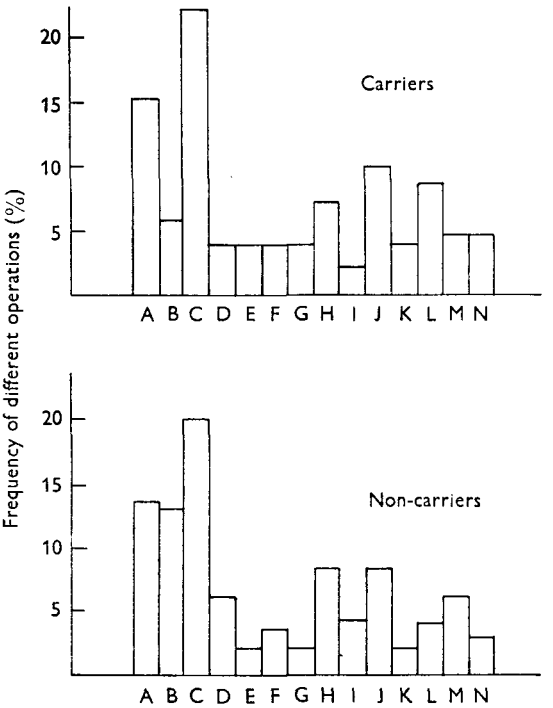

Fig. 1. Relative frequency of different operations on carriers and non-carriers. A, gynaecological; B, hernia; C, appendix; H, gallbladder; J, gut resection; L,bone-pinning.

than those of carriers. This was impossible to exclude with any confidence, but some evidence against it was available. Many wounds in both groups were infected with staphylococci although not clinically septic. The combined figures of clinical and subclinical infection with staphylococci might be expected to reflect in some measure the relative exposure to staphylococci of wounds in the two groups. The actual figures were closely similar for both groups, namely 38/153 (24·8 %) for the carrier group, and 87/385 (22·6 %) for the non-carriers. For what this evidence is worth, therefore, it suggests no major difference in exposure to staphylococcal contamination between the two groups.

Finally, the comparability of the carrier and non-carrier groups was tested in respect of age distribution. This was of some importance because susceptibility to wound sepsis has been shown to increase with age, irrespective of other factors (Lidwell, 1961), while the staphylococcal nasal carrier rate diminishes with advanc-

ing age (Williams, 1947). One might, therefore, observe the increased sepsis rate in non-carriers through inclusion in the non-carrier group of a relatively higher proportion of more susceptible older people.

The decline in the staphylococcal carrier rate with advancing age was confirmed in the present survey, e.g. 35 % of those under 40 were staphylococcal nasal carriers on admission to hospital, as compared to 25 % of those over 40. The age-specific staphylococcal wound sepsis rate still showed an excess, however, in each major age group among those who were non-carriers on admission to hospital. The sepsis incidence in those over and under 40, for instance, in relation to carrier state is shown in Table 4.

Table 4. *The incidence of staphylococcal wound sepsis in younger and older patients according to nasal carrier state*

Age	Carriers	Non-carriers
Under 40	2/57 (3·5 %)	6/105 (5·7 %)
Over 40	7/92 (7·6 %)	30/269 (11·2 %)

Ages of patients were determined in retrospect from hospital records. The total of 523 patients in this table represents those who were swabbed on admission to hospital and whose ages had been entered in the case notes.

Table 5. *Incidence of staphylococcal sepsis according to nasal carrier state on admission to hospital and presence or absence of post-operative drain*

	Wounds with drain		Wounds without drain	
	Total no.	No. with staphylococcal sepsis	Total no.	No. with staphylococcal sepsis
Carriers	39	3 (7·7 %)	114	6 (5·3 %)
Non-carriers	102	21 (20·6 %)	273	15 (5·5 %)

No information available on presence or absence of drain in 10 non-carriers.

The higher incidence of staphylococcal sepsis in those over 40 is of course not a simple age effect, as in general the major operative procedures were carried out on older patients.

The foregoing analysis would seem to indicate that the excess of staphylococcal wound sepsis in those who were non-carriers on admission to hospital cannot be explained in terms of differences between the carrier and non-carrier groups in age, sex, operative procedure undergone or exposure to staphylococcal contamination.

A study of the individual patients' records provided a useful pointer to the kinds of operation that contributed most to the excess of sepsis in non-carriers. It was clear that, as reported by previous workers (e.g. Gillespie, Alder, Ayliffe, Bradbeer & Wypkema, 1959; Public Health Laboratory Service, 1960), wounds requiring post-operative drainage had shown a higher incidence of sepsis than wounds not drained. The incidence of sepsis in drained and non-drained wounds was therefore calculated for carriers and non-carriers. First of all, the proportions of wounds requiring drainage were very similar for the two groups, namely 39/153 (25·5 %)

and 102/375 (27·2 %) for carriers and non-carriers respectively. No information was available as to whether drains had been inserted after operations on 10 non-carriers; hence the total of 375 instead of 385. Analysis of the incidence of staphylococcal sepsis in terms of the staphylococcal carrier state and the presence or absence of a drain is shown in Table 5. This shows that the excess of sepsis in the non-carrier group was concentrated in those patients whose wounds required a post-operative drain. If the closely similar sepsis figures in the other three categories of the table are pooled, the difference between these groups and that of 'drained non-carriers' is highly significant (χ^2 with Yates correction = 21·73; $P < 0{\cdot}001$).

Incidence of wound sepsis in carriers according to phage group of staphylococcus carried on admission to hospital

McNeill *et al.* (1961), and Williams *et al.* (1962) reported higher wound sepsis rates in patients who acquired staphylococci in the nose after admission to hospital or who changed the type of staphylococcus carried while in hospital, than in those who remained non-carriers or continued to harbour their own initially carried strain throughout.

Whether acquisition of staphylococci in the nose while in hospital predisposes to or determines wound sepsis, or merely reflects the contaminated environment to which the patient has been exposed, will be discussed later. In the context of the present study, however, the phage group distribution of staphylococci carried in the noses of patients on admission to the Torbay Hospital is of some interest for two reasons. First, the interval between the patient's admission to hospital and the operation was usually so brief that the likelihood of pre-operative acquisition of staphylococci in the nose and of sufficient multiplication of the acquired strains to establish a dangerous carrier state would seem remote. Certainly, none of the 106 patients in the second Torbay Survey, who were swabbed both on admission to hospital and in the operating theatre, showed any evidence of such a precocious development of a profuse nasal carrier state in the brief interval before operation. To this extent, therefore, the influence of acquired nasal staphylococci on the incidence of sepsis must be far more tenuous in a hospital such as the Torbay Hospital than it might be in a teaching hospital, where according to, e.g. Table III of Williams *et al.* (1962), the average stay of surgical patients in the hospital before operation was probably considerably longer than at Torquay.

In another respect, however, the Torquay material provides an interesting counterpart to the surveys of McNeill *et al.* (1961) and of Williams *et al.* (1962). Presumably because of the high incidence of infections due to the 80 group at Torquay from 1957 onwards, 38 (7·1 %) of the 538 patients who were swabbed carried a staphylococcus of the 80 group in the nose on admission to hospital. In this group of 38 patients, 5 (13·2 %) developed post-operative staphylococcal wound sepsis as compared to a sepsis rate of only 2·6 % in patients who carried other phage types of staphylococcus in the nose when admitted to hospital. Thus, patients who had acquired staphylococci of the epidemic 80 group in the nose before admission to hospital suffered an incidence of staphylococcal sepsis of an order

very similar to that experienced by those patients in the St Bartholomew's Hospital surveys who acquired a staphylococcus in the nose while in hospital. Even with this small group of 5 patients, however, in only 1, as discussed below, could the post-operative sepsis be confidently ascribed to the strain carried on admission to hospital.

These findings on the relationship between the staphylococcal nasal carrier state and the incidence of wound sepsis are discussed below in the context of other work on this subject. First, however, the incidence of wound sepsis due to self-infection with staphylococci carried on admission to hospital, and of wound infection associated with the operating theatre, is assessed in the following sections.

Self-infection

As shown in Table 2, 9 of the 153 patients who were staphylococcal nasal carriers on admission to hospital developed post-operative staphylococcal wound sepsis. In one of these nine patients, the sepsis was almost certainly the result of self-infection. The patient in question yielded a nasal staphylococcus of phage type 187 on admission to hospital on 22 May 1959 and had a total hysterectomy on the following morning. The wound discharged a large amount of pus when the sutures were removed 9 days later, and staphylococci of the same phage type were isolated. The only other occasion during this survey when an organism of this type was isolated from any patient was a month later, when a type 187 staphylococcus was isolated from a clinically healthy wound.

A second female patient, also admitted for hysterectomy, in December 1959, was found to have a staphylococcus of an unusual group III pattern in her nose on admission, and her wound showed a slight sero-purulent discharge due to organisms of the same phage pattern 8 days later.

As mentioned above, 5 patients who carried staphylococci of the 80 group in the nose on admission to hospital developed post-operative staphylococcal sepsis, but in only 1 could self-infection be deemed highly probable, and this man had just been an in-patient in a medical ward of the hospital. Of the remaining 4, two carried type 80 staphylococci in the nose on admission and their wounds yielded staphylococci of the closely related type 52/52A/80. Though these may have been instances of self-infection, staphylococci of type 52/52A/80 were so widely distributed in the hospital at the time that other sources of infection, whether in the theatre or in the wards, could not be excluded. Finally, one patient whose nose yielded type 52/52A/80 staphylococci developed wound sepsis due to a type 80 strain, and another who also carried type 52/52A/80 in her nose developed a type 52 wound infection. Technically, either might have been due to self-infection but a doubt remains.

Infection associated with the operating theatre

Gillespie, Alder, Ayliffe, Powell & Wypkema (1961) were of the opinion that, although the nasal carriage of staphylococci by the nursing staff was not of epidemiological significance in the wards, this was not so of carriers working in the theatre. The first Torbay Hospital survey, from April to June 1959, furnished

clear evidence in support of this view. The high incidence of septic infections in the nursing staff during 1958 has already been mentioned. A high staphylococcal carrier rate in the theatre staff was therefore to be expected, but the theatre personnel had not been recently swabbed when the survey started. By the last week of April, however, a number of patients had developed staphylococcal wound sepsis under circumstances that pointed strongly to an operating theatre infection, and all the surgical and nursing personnel of the theatre were accordingly swabbed during that week. Correlation of the swabbing results with the incidence of wound sepsis showed that two appendicectomy patients with severe type 52/52A/80 wound sepsis had both been operated on by a house surgeon who was found to be a heavy nasal carrier of staphylococci of this type, and who also had an external otitis infected with this organism. The same house surgeon had assisted at another operation on a third patient, whose nephrectomy wound became septic and again yielded staphylococci of type 52/52A/80. Two further cases of severe wound sepsis, operated on within 3 days of each other during the 2nd week of April, had staphylococci of type 29/52/80 in their wounds, a relatively stable phage pattern in our experience and associated with two of the most severe cases of wound sepsis seen in the course of another survey, in Exeter, in 1958. The junior theatre sister who took part in the operation on one of the Torquay cases proved to be a nasal carrier of type 29/52/80 staphylococci. She had been on sick leave, suffering from boils, on two occasions during the previous year. She may well have infected the 2 patients in question.

Apart from these 5 patients, whose wounds were very probably infected directly by theatre personnel at a time when the hospital staff showed a high nasal carrier rate of epidemic staphylococci, several other patients who developed post-operative wound sepsis were clinically theatre infections with deep suppuration and constitutional symptoms. The precise mode of infection could not be established, however, partly because the theatre staff had not been swabbed at the relevant time, and partly because most of these infections were with organisms known to be widely disseminated in the wards, and therefore possibly brought to the theatre on the person of the patient concerned.

Since August 1960 all members of the theatre staff—medical, nursing and technical—have been swabbed routinely every 2 months. Very few carriers of epidemic staphylococci have been found, and only 3 in the past 18 months. The incidence of post-operative sepsis, and of skin infections in the nursing staff, have also fallen considerably during the past 2 years.

DISCUSSION

In three British papers published in recent years (Williams, Jevons *et al.* 1959; McNeill *et al.* 1961; Williams *et al.* 1962), the acquisition of nasal staphylococci of hospital types by patients admitted to surgical wards has been singled out as an important determinant of post-operative wound infection. An alternative view, as expressed, for example, by Rountree, Harrington, Loewenthal & Gye (1960), would deem it equally likely that, where patients acquire ward strains of

staphylococcus at about the same time in their noses as in their wounds, both nose and wound have been infected from some other source.

The distinction is of practical moment in deciding what preventive measures are most likely to reduce the incidence of post-operative sepsis. If the nose is the main source of wound staphylococci, suppression of the nasal carrier state should materially lower the incidence of wound sepsis. Gillespie (1958), reviewing the earlier findings of the Bristol investigators, noted that, where 'nasal prophylaxis' was applied in two surgical wards, as a sole precaution, there had been a marked fall in the nasal carrier rate but a far smaller effect on the incidence of wound sepsis. He suggested that, to control cross-infection in surgical patients, it might be necessary to deal with both nasal carriers and the infected open wound, while at the same time blocking some of the more obvious routes of ward cross-infection, such as contaminated blankets or baths. More recently, the controlled trials of Henderson & Williams (1961) and of Stokes & Milne (1962) confirmed the ineffectiveness of nasally applied antibiotics in reducing the incidence of wound sepsis.

The findings of the Torbay Hospital survey, as discussed in the foregoing pages, and of Bassett *et al.* (1963), are in sharp contrast to those of the St Bartholomew's Hospital group (Williams *et al.* 1962). In the Torbay Hospital study, the presence of a staphylococcus in the nose on admission to hospital was almost a guarantee of freedom from wound sepsis rather than a predisposing cause, and Bassett and his colleagues also report a lower incidence of wound sepsis in nasal carriers than in non-carriers. Can these conflicting findings be reconciled on closer examination?

One source of confusion has been the use of the term 'nasal carrier' in a number of quite different senses. Thus, in discussing the source of infection of surgical wounds, Williams, Blowers, Garrod & Shooter (1960) cite the papers of Weinstein (1959), Williams *et al.* (1959), and Colbeck, Robertson, Sutherland & Hadley (1959) as evidence that nasal carriers of *Staph. aureus* suffer wound infection more often than non-carriers, and the investigations of the Public Health Laboratory Service (1960) and of Bassett *et al.* (then in preparation) as having yielded different results. Of these papers, those of Colbeck *et al.* (1959) and of the Public Health Laboratory Service (1960) refer to the staphylococcal nasal carrier state of patients on admission to general hospitals for surgery. Knowledge of staphylococcal carriage at this point of time permits later assessment of whether or not the hospital bears responsibility for cross-infection of a patient's wound with the bacterial flora of other patients, or whether it has simply failed to exclude the patient's own flora from the depths of his wound. For Williams *et al.* (1959), on the other hand, the nasal carrier group includes not only those who harbour staphylococci in the nose on admission to hospital, but also those who acquire nasal staphylococci while in hospital. Weinstein (1959) uses the term in yet another sense. His patients were not swabbed at all on admission to the thoracic surgical unit concerned, but, on average, about 4 months later, when they were about to be operated on. For him, therefore, the nasal carrier state reflects a 4 months' interaction of the patient with an environment contaminated with hospital staphylococci, particularly of the 80/81 group. Some confusion is inevitable until a great deal more has been learned about the nature of the nasal carrier state. Meanwhile, the use of the term merely

reflects the successful isolation of a staphylococcus from a nasal swab at a particular time, and some caution is required in correlating such success with the variables of hospital infection.

As a prelude to a wider discussion of the sources of post-operative infection in surgical wounds, some comments are called for on the paper by Williams *et al.* (1959), the first to state a detailed case for ascribing sepsis in surgical patients to nasal staphylococci acquired in hospital. The argument centres essentially on the finding that the incidence of post-operative staphylococcal wound sepsis in the surveys described was 2% in 342 patients who were never nasal carriers of staphylococci and 7·1% in the 380 who carried at some time. The authors reached the conclusion that the nose was often the source of the sepsis, and that the incidence of sepsis might be reduced if acquisition of staphylococci in the nose could be prevented. Alternative interpretations of the main findings were discussed, however, namely (i) that the lesion had infected the nose, or (ii) that both nose and lesion were infected separately from some other source. The first was thought unlikely, because in only very few patients were the staphylococci not found in the nose until after the development of sepsis. This argument is suspect on two counts. First, nasal swabbing of patients was done only once a week, while swabs from septic wounds were presumably taken when the sepsis was observed. In a later paper, Williams *et al.* (1962) describe the assumptions and conventions that had to be adopted in estimating the day of acquisition of a nasal staphylococcus because of this interval between successive swabbings. Their analysis of the priority in time of nasal over wound infection was analysed in terms of days rather than weeks. The conclusions would have been correspondingly more compelling if nasal swabs had been taken daily rather than at weekly intervals. Even, however, if one accepts prior infection of the nose as having occurred, the argument from priority of nasal acquisition is not entirely convincing. If a patient is placed in a heavily contaminated environment, and is in due course operated on, his nose is almost bound to become infected with a staphylococcus present in large numbers in the ward before his wound becomes infected with the same strain, partly because of the respiratory function of the nose which makes it an effective sampler of the ward contamination, partly because of the interval between admission and operation, and also because the wound will presumably normally be protected from ward contamination except during dressings.

Prior infection of the nose is thus compatible with separate infection of both nose and lesion from some other source. This latter possibility was also discussed by Williams and his colleagues, but dismissed on the grounds that 'to explain the differing experience of nasal carriers and non-carriers one would have to postulate some general staphylococcal "diathesis" which made those who became nasal carriers also more likely to become infected in their wound or elsewhere. In this case one would hardly expect the nose and lesion to show the same type of staphylococcus with such frequency.' An alternative argument to that of invoking a staphylococcal diathesis would be simply to regard the acquisition of a nasal staphylococcus as a sensitive indicator of exposure to staphylococci in the ward. Those who acquired a nasal staphylococcus would *ipso facto* be more exposed to

wound contamination. Whether or not one would 'expect nose and lesion to show the same type of staphylococcus with such frequency' requires further scrutiny. If one regards the acquisition of a nasal staphylococcus as an indication of exposure to staphylococcal contamination, phage typing of the nasal staphylococcus permits the more precise inference that the patient has been exposed to contamination with that particular phage type, and to this extent one would expect that his wound might also become contaminated with the same type of staphylococcus. Apart from this, however, reference to the paper in question shows that the close parallelism of staphylococcal phage types in nose and wound was observed only with tetracycline-resistant epidemic strains of staphylococcus. To assess the significance of the acquisition of antibiotic-resistant staphylococci, of the same phage type, in nose and wound at about the same time, would require knowledge of the degree of contamination of the ward at the relevant time with epidemic strains of different phage types. This information is not given in the 1959 paper, but in an earlier paper by the same group and discussing work done in the same series of studies, Shooter *et al.* (1958) make special mention of nine profuse 'staphylococcal broadcasts' of individual phage types of staphylococcus in the ward under investigation. Several of the broadcasts could be attributed to patients, with infected lesions such as a tracheotomy wound or infected urine, that were particularly likely to disperse staphylococci. It seems probable, therefore, that in the foregoing studies, as in others described in the literature, e.g. by Barber & Warren (1962), environmental contamination at any one time tended to be caused by one phage type of epidemic staphylococcus that was widely disseminated in the ward. If this was so, it would again increase the likelihood that the wound and nose of a given patient acquired the same strain of epidemic staphylococcus, independently, from the contaminated environment.

A more serious criticism of these studies of Williams *et al.* (1962) remains to be mentioned. Before ascribing high and low sepsis rates to the presence or absence of a nasal carrier state, it is material to inquire whether the carriers and non-carriers were comparable in other respects, e.g. as regards age or the nature of the operations undergone. The necessary information is not given in the paper in question. In the three male wards studied, however, there were especially large numbers of patients undergoing rectal operations in ward A, vascular operations in ward B and urinary tract operations in ward C. Table III of Williams *et al.* (1962) would seem to point to a lack of comparability in some material respect between those patients who, on the one hand, changed strains or acquired nasal staphylococci while in hospital and had a high sepsis rate, and those in the two low sepsis groups, on the other hand, who were never carriers or who were carriers on admission and kept their own strains. Only 5·5 % of the high sepsis group had a total stay in hospital of 1–10 days, compared to 56·0 % of the non-carriers and 38·5 % of those who carried the initially isolated strain throughout. This difference cannot be accounted for merely by the longer stay in hospital of the septic patients in the high sepsis group, as these comprise no more than about 12 % of the total. It would seem to be explicable only in terms of some quite major difference between the two groups, presumably in the operations undergone. If this was so, the validity

of ascribing the differences in sepsis rates to the nasal carrier state seems questionable on these grounds alone.

Despite these comments, few would dispute the importance of the nasal carrier state in the general context of staphylococcal infection; differences of opinion in this field are largely a matter of varying emphasis. Whether or not a particular patient develops post-operative wound sepsis will depend in the long run on the interplay of two major factors, namely (i) his susceptibility to infection—still largely a qualitative concept—and (ii) the possible access to his wound of a sufficient number of virulent staphyloccoci to give rise to wound sepsis. These two determining elements are discussed in turn in the following paragraphs.

The patient's susceptibility to infection

Not all patients admitted to surgical wards develop post-operative wound infection. Certain factors such as age, the nature and duration of operation and the presence or absence of a drain have long been known to predispose to sepsis, but many such factors are intercorrelated and their independent effects are difficult to assess. Lidwell (1961) applied the method of multiple regression analysis to the records from twelve hospitals collected by the Public Health Laboratory Service (1960). This showed that age, sex, duration of operation, length of incision, and the insertion of a drain were all associated with increased risk of post-operative sepsis. Certain operations appeared to carry a relatively higher basic risk of sepsis than others, and Lidwell showed that this accounted almost entirely for the differences in sepsis rates observed in the various hospitals investigated during the Public Health Laboratory Service study. Clearly, therefore, any comparison between the findings of different surveys of wound infection must be approached with some caution unless the populations concerned can be appropriately matched for such factors as those discussed by Lidwell. Equally, the linking of high or low sepsis rates with other characters such as a nasal carrier state is somewhat hazardous unless the influence of some of the more obvious factors has first been excluded.

In the Torbay Hospital study, the curious association of a low sepsis rate with nasal carriage on admission to hospital was observed during scrutiny of the first survey records. Attempts to explain this finding in terms of non-comparability in some other respect of the carrier and non-carrier groups were quite unsuccessful. Thus, one is faced with the problem whether in Torquay at the time in question carriers admitted to hospital for surgery were indeed more resistant to staphylococcal infection than non-carriers admitted at the same time. As already mentioned, the difference in sepsis rates between the two groups apparently concerned patients whose wounds had been drained rather than those with clean-stitched wounds. This finding is largely a matter for record rather than discussion, in the absence of previous data. Curiously, the ratio of the incidence of sepsis in non-carriers and carriers is very similar to that between the proportion, in Rountree & Barbour's study (1951), of non-carrier nurses who picked up a hospital staphylococcus in the nose on entering the wards and of carrier nurses similarly infected. The mechanism of exclusion of hospital strains from the noses of carriers was not explained. The

present findings raise the query whether it might not have an immunological basis rather than a quasi-mechanical one as generally assumed.

A minor finding that might be related to the Torquay results is mentioned in the Public Health Laboratory Service report, namely that, of the 106 patients in the surveys in question who carried nasal staphylococci of phage group II on admission to hospital, only one showed a post-operative wound cross-infection. Possibly a scrutiny of the incidence of cross-infection in nasal carriers of different staphylococcal phage types might throw further light on this subject.

Whatever the explanation of the Torquay findings, clearly staphylococcal nasal carriage on admission to hospital was not, in the series of 559 patients investigated, attended by an increased risk of post-operative wound sepsis, but by a diminished risk. Bassett *et al.* (1963) interpret the findings of their two hospital surveys as indicating that self-infection of wounds was of minor importance, but that cross-infection probably occurred with equal frequency in carriers and non-carriers, the apparent excess of cross-infection in non-carriers being due to the impracticability of demonstrating cross-infection in carriers whose noses and wounds harboured the same phage type of staphylococcus. In the Torquay survey, however, the total incidence of sepsis in those who were staphylococcal nasal carriers on admission to hospital was so low that the existence of an increased resistance to sepsis in this group seems a reasonable inference.

Development of wound sepsis in relation to the infecting dose of staphylococci

The occurrence of post-operative staphylococcal wound sepsis in a given patient is conditioned by the possibility of access to the wound of an adequate number of pathogenic staphylococci. The infecting dose required will presumably vary with the patient's susceptibility, and access of these organisms to the wound will depend on various factors, including the physical breach in the skin barrier caused by the operative incision or by the continuing presence of a drain in the wound during the post-operative period.

Little is known of the initial stages in the establishment of the subcutaneous staphylococcal lesion, but the experimental work of Elek & Conen (1957) suggested that the minimum pus-forming dose for virulent staphylococci by subcutaneous injection was of the order of 2–8 million organisms. As natural infection with the minimum pus-forming dose seemed to them highly improbable, these authors concluded that special circumstances were required to enable the ordinary infecting dose to reach the minimum pus-forming level. One such circumstance was the presence of a foreign body such as a suture, which in their experiments reduced the minimum pus-forming dose several thousandfold. Another was tissue trauma or devitalization, and surgeons stress the importance of this factor. Howe (1956), for instance, maintained that the 'localized suppuration commonly called wound infection is a septic breakdown of devitalized tissue, blood clot or serum'.

Although one might object that the experimental techniques of Elek & Conen must have failed in some way to simulate natural infection, or that the cultures they used were not as virulent as hospital staphylococci—naturally their experimental strains used on human volunteers were not fully antibiotic-resistant, for

instance—nevertheless it is pertinent to consider briefly a few quantitative aspects of some of the accepted modes of wound infection.

The work of Hare and his colleagues, and particularly of Hare & Cooke (1961), showed that the nasal carrier without skin or wound infection usually disseminates remarkably few staphylococci, whereas patients with skin infections contaminate their clothing and environment profusely. Much of the existing literature on wound infection is compatible with the suggestion that the skin may sometimes serve as an enrichment medium for staphylococci, and could well provide wound-infecting doses of the order required in Elek & Conen's experiments, whether from the person of the surgeon with a septic infection or on the patient's susceptible wound area. Williams & Miles (1949), for instance, found that, in patients with industrial wounds of the hand, staphylococcal wound contamination was associated with skin, but not with nasal, carriage of staphylococci. They interpreted this finding as meaning that direct transfer of staphylococci from nose to wound was rare, and that nasal carriage predisposed to wound contamination, only by initiating carriage in one or more discrete areas of skin, which might or might not become the site of wounding. Some remarkable instances of the carriage of *Staph. aureus* on clearly definable areas of apparently normal skin in persons who were not nasal carriers were cited by Thomas (1961), who mapped out the distribution of skin flora using a sticky tape method. Again, the notoriously epidemic group 80 strains are known to cause skin lesions as well as wound infection. Finally, the common view, first expressed by Barber & Burston (1955), that nasal carriers among hospital staff become dangerous to patients only when they develop boils or other skin lesions, might be explained in terms of increasing skin contamination with staphylococci, without postulating an increase in strain virulence.

A careful quantitative study of staphylococcal skin carriage, both deep and superficial, in an adequate series of surgical patients, and particularly in established nasal carriers of epidemic staphylococci, might well show that some of these patients were operated on through or near localized areas of skin staphylococcal carriage.

How would the foregoing emphasis on skin carriage influence the interpretation of all the relevant evidence on the genesis of wound infection? The acquisition of nasal staphylococci *per se* would be looked on only as evidence of exposure to ward contamination. It would become an indirect determinant of wound infection, in the patient concerned, only where skin carriage followed. The time required for the development of skin carriage might lie in the pre-operative period, as in the Torbay Hospital and Public Health Laboratory Service studies, and also in those of Weinstein (1959) and McNeill *et al.* (1961). All four reported a similar increased incidence of self-infected wound sepsis in persons who were nasal carriers of epidemic strains on admission to hospital or in nasal carriers who had been in hospital for a long time before operation. The development of skin carriage might also follow a long post-operative sojourn in hospital, and explain some instances of late wound sepsis.

To explain wound sepsis in terms of contamination acquired from the environment would appear more difficult. Rountree & Beard (1962) give strong circum-

stantial evidence for the view that some patients acquire both nose and wound infection from ward bedding. Can one envisage the source of an adequate infecting dose of staphylococci in the ordinary ward environment? Two relevant points may be cited here. First, several reports (e.g. Shooter *et al.* 1958; Barber & Warren, 1962; Rountree & Beard, 1962) have indicated that, in surgical wards, very large numbers of epidemic types of staphylococcus may be disseminated from time to time, probably from patients with heavy skin or wound contamination or with staphylococcal lung or urinary tract infection. Rountree & Beard mention unpublished evidence suggesting that these strains can persist for long periods on various types of textile. Increased resistance to drying may therefore explain in part the success of these strains in establishing themselves in hospitals.

Contaminated blankets and sheets have been looked on as a source of staphylococcal broadcasts, during bedmaking, or from friction between blankets and counterpanes (Rubbo, Stratford & Dixson, 1962). The work of Elek & Conen raises a further possibility that has apparently not been investigated, namely that particles of wool or cellulose fluff contaminated with staphylococci gain entry into wounds, at operation or in the ward, and initiate wound sepsis by acting as foreign bodies in the same way as the suture material in Elek & Conen's experiments. One particular observation made during other studies in a surgical ward at Torquay seemed to lend support to some such mechanism. Two apparently unrelated cases of severe wound sepsis occurred in the ward with an interval of several days between them. It then transpired that for various reasons both patients and their beds had been moved round the ward many times—one of them eight times and the other five times. Only once were they in contiguous positions in the ward, namely on the night before the second patient's operation. This seemed to point strongly to massive contamination of the person or clothing of the second patient just before he went to the operating theatre and to direct access of contaminated fluff to his wound.

SUMMARY

1. A survey of post-operative wound infection was done in 1959–60 on 559 surgical patients admitted to a provincial general hospital.

2. Clinical evidence of post-operative wound sepsis was observed in 71 (12·7 %), suppuration in 51 (9·1 %) and staphyloccocal wound sepsis in 48 (8·6 %).

3. Seventeen of the patients died in hospital. Although 5 of these had septic wounds, the sepsis did not appear to have been the cause of death.

4. Contrary to some reported findings, the post-operative wound sepsis rate was considerably lower in patients who were staphylococcal nasal carriers on admission to hospital than in non-carriers. Nine out of 153 carriers (5·9 %) developed wound sepsis and 36 out of 385 non-carriers (9·4 %). When allowance is made for 3 highly probable self-infections, the incidence of wound cross-infection was 3·9 % in carriers and 9·4 % in non-carriers.

5. The excess of sepsis in non-carriers could not be explained in terms of different age or sex distribution in carrier and non-carrier groups, nor by differences in the types of operation undergone or in degree of exposure to staphylococcal contamination.

6. The excess of sepsis in non-carriers was accounted for by the patients whose wounds had drains rather than by clean-stitched wounds.

7. Those carriers who harboured a staphylococcus of the 80/81 group in the nose on admission to hospital had a higher incidence of wound sepsis than carriers of other phage types or staphylococcus.

8. Three probable instances of wound self-infection occurred, and in the early stages of the survey at least 5 wound infections were probably directly caused by two members of the theatre staff carrying staphylococci of the 80/81 group in the nose and with existing skin sepsis or a recent history.

9. The survey findings and a study of the literature suggested: (*a*) that the acquisition of a nasal staphylococcus in hospital was probably, as a rule, evidence of exposure to staphylococcal contamination and not a determinant of wound sepsis, unless the patient also became a skin carrier; (*b*) that a small proportion of patients are self-infected, some are directly infected by theatre personnel, and the wounds of other patients are directly or indirectly contaminated by staphylococci from the ward environment; (*c*) that wool or cotton fluff contaminated with staphyloccoci may cause wound sepsis by falling into open wounds and as foreign bodies induce a significant reduction in the minimum pus-forming dose of staphylococci; (*d*) that before ascribing high or low sepsis rates to factors such as the nasal carrier state, the relevant groups should be shown not to differ materially in respect of other factors known to influence the incidence of wound sepsis.

We gratefully acknowledge the help and encouragement of our surgical colleagues Mr H. P. Guerrier, Mr P. J. W. Monks, Mr J. Macpherson and Mr C. C. Jeffery; and of Dr C. P. Warren, consultant pathologist to the Torbay Hospital, Mrs M. Stamp, Matron, Sister E. M. Cottrell, first Infection Control Sister at the Torbay Hospital, and her successor Mr H. Street.

Acknowledgement is also due to the South Western Regional Hospital Board for its encouragement and for a research grant for technical aid, without which the surveys described would not have been possible.

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