J. Hyg., Camb. (1966), **64**, 321
Printed in Great Britain

Staphylococcal infection in thoracic surgery: experience in a subdivided ward

By O. M. LIDWELL, SHEILA POLAKOFF, M. PATRICIA JEVONS and M. T. PARKER
Cross-Infection Reference Laboratory, Central Public Health Laboratory, London, N.W. 9

R. A. SHOOTER, VALENTINE I. FRENCH and D. R. DUNKERLEY
Department of Bacteriology, St Bartholomew's Hospital, London, E.C. 1

(*Received* 26 *January* 1966)

INTRODUCTION

Most studies of the epidemiology of staphylococcal infection in British hospitals have been conducted in large open wards containing twenty or more patients. Such wards are now rarely built, as the present tendency is towards subdivision into separate rooms, each containing four patients or less. Among the reasons advanced to support this change is the impression that patients housed in small groups are less likely to suffer from sepsis due to cross-infection than those in large undivided wards.

The new surgical block in St Bartholomew's hospital consists of wards divided into a number of separate rooms. We now report on an investigation in the Department of Thoracic Surgery, which occupies one of these wards, to find out whether this subdivision was effective in preventing the spread of staphylococcal infection among the patients.

Architectural features

The ward under study occupied the third floor of the new block (see Fig. 1). It included a large room (A) containing ten to twelve beds, three four-bedded wards (B1, B2, B3) and four single-bedded cubicles (C1, C2, C3 and C4). One other four-bedded ward (BR) was used as a recovery room. Each room had a door leading to the corridor, but these doors were usually left open. All the rooms had windows which could be opened, and there was no artificial ventilation. The total volume of the ward was 65,000 cu.ft. The rooms occupied by patients had a total volume of about 34,000 cu.ft., or 1100 cu.ft. per patient when the ward was full. [1200 cu.ft./patient in A, 1000 cu.ft./patient in B1–3, and 1400 cu.ft./patient in C1–4.]

A set of twin operating theatres on the 5th floor served the whole block. They were shared by the Departments of Thoracic Surgery, Neurosurgery, and Ear, Nose and Throat Surgery. They had in common a 'clean' and a 'dirty' sterilizing room, which were separated from each other by a bank of double-ended autoclaves. The doors separating the theatres from the sterilizing rooms were kept shut

while not in use. The theatres were ventilated by positive-pressure at a rate of about twenty air changes an hour.

Ventilation of the ward

Ventilation and air movement within the ward were determined entirely by weather conditions and the opening of doors and windows, and varied considerably from time to time. The most consistent feature was a strong rising current of air

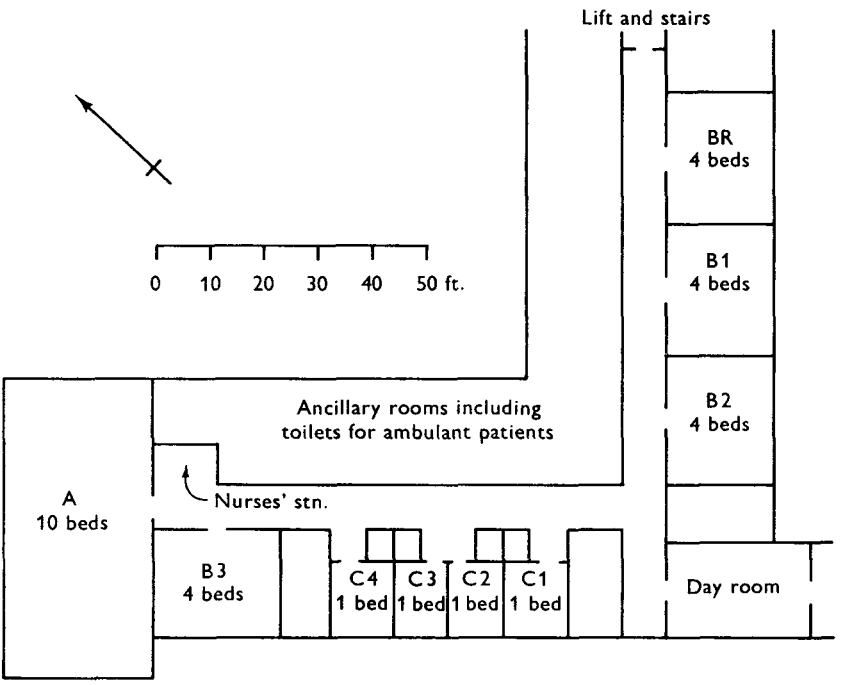

Fig. 1. Plan of the ward

up the stair-well, which entered the ward through the swing-doors at the north-east end of the corridor. The resulting pattern of air movement was explored by smoke tests on twenty-eight occasions spread over the whole of a calendar year. On no single occasion was the air flow between the corridor and the rooms of the ward uniformly in one direction, i.e. from the corridor to all the rooms or vice versa, and on only four occasions was it in the same direction for even as many as two-thirds of the rooms. In all, 33 % of the 252 observations on individual rooms showed air flow from the room into the corridor, 27 % flow in the reverse direction, and the remaining 40 % convectional exchange between room and corridor. The volumes of air involved in these movements were not measured, but a velocity as low as 20 ft. per min. across the upper or lower half of a door opening—and the room doors were open for the greater part of the day—will move as much as 6000 cu.ft. of air per hour.

More detailed quantitative studies were made on three occasions using nitrous oxide as a tracer gas. These confirmed that substantial volumes of air were moving

between the rooms and the corridor. The detailed patterns of air movement were complex. In general, however, the figures indicated an average level of exposure to airborne contamination of the order of one-third of that obtained in a similar experiment carried out in an open ward. This reduction represents the combined effect of restricted mixing in the subdivided ward and of differences in effective ventilation between the two wards. The ventilation of a complex system can be represented only approximately by a single figure, but for this ward on three occasions the average value appeared to be about $4\frac{1}{2}$ air changes per hour, reckoned on the volume of the rooms containing patients and the corridor.

METHODS

Patients

The ward contained both male and female patients. The large room (A) was nearly always occupied by male patients. The allocation of the four-bedded wards (B 1–3) and the single-bedded cubicles (C 1–4) to male and female patients was made according to the number of patients of either sex and their surgical requirements. After operation, most of the patients spent some time in the recovery room (BR). They usually stayed there for less than 24 hr., but a few requiring intensive therapy were there for several days.

Wound dressings. Dressings were removed on the third post-operative day. Wounds showing no clinical signs of infection were sprayed with Nobecutane (Evans Medical Supplies) and left uncovered. Stitches were removed on the eighth day, and Nobecutane was again applied.

Determination of sepsis. A wound was considered to be septic only when visible pus was present.

Treatment with antibiotics. Nearly all patients subjected to thoracotomy received injections of benzyl penicillin (1 million units 8-hourly) for 4 days, followed by procaine penicillin (600,000 units 12-hourly) for 3 days. Patients for whom artificial circulation was used were given benzyl penicillin (1 million units 6-hourly) and streptomycin (0·5 g. 12-hourly) by injection for 7 days; some of them were also given penicillin V orally for a further month.

In all, 506 of 714 patients (71%) and 490 of 618 patients who underwent a surgical operation (80%) received an antibiotic while in hospital. Patients who were operated upon and received no antibiotic were in general subjected to minor procedures, or to ones in which there was no cutting of the tissues. Indeed, only six patients who had a major cutting operation did not receive an antibiotic at some time. The percentages of patients receiving individual antibiotics were as follows: penicillin 63, streptomycin 14, tetracycline 7, chloramphenicol 4, methicillin or cloxacillin 3, ampicillin 2, any other antibiotic less than 1.

Staff

The staff consisted of two consultant surgeons, one or two registrars and two house-surgeons. In addition to the sister in charge, the day staff included six staff-nurses and nine nurses in training. At night there were one staff-nurse and six

student nurses in the ward. About fifteen other professional and technical persons (anaesthetists, respiratory physiologists, physiotherapists, etc.) worked intermittently in the ward.

Bacteriological examinations

Nasal swabs were taken from patients on admission and thereafter weekly on a fixed day. A further swab was taken on the day of each operation. Wounds were swabbed at the first dressing, and again when the stitches were removed. Further swabs were taken if necessary. If there was clinical evidence of sepsis elsewhere, the appropriate specimens were sent to the laboratory. Coagulase-positive staphylococci (*Staphylococcus aureus*) were tested for sensitivity to antibiotics and phage-typed. Nasal swabs were also taken weekly from all members of the surgical, nursing, and other staff of the ward.

Air-sampling

Air-sampling was carried out on 28 days, at approximately fortnightly intervals. On each day, three slit-samplers were run simultaneously for two successive periods of 1 hr., in the course of each of which 108 cu. ft. (3·06 cu.m.) of air were sampled by each machine on a phenolphthalein-phosphate serum-agar plate. The first sampler was always in the 10-bedded ward (A). The second was placed for the first hour alternately in one of the 4-bedded rooms (B 1 or B 2), and for the second hour was moved into the other. The third was placed for the first hour alternately in C 1 or C 2 and was moved for the second hour from C 1 to C 3 or from C 2 to C 4.

Colonies resembling *Staph. aureus* were subcultured and tested for coagulase production. Up to ten coagulase-positive cultures from each plate were phage-typed and tested for resistance to penicillin and tetracycline. When more than ten colonies of *Staph. aureus* were present on a plate, strains were assumed to be distributed in the sample in the same ratio as in the ten colonies that had been phage-typed.

Method of analysis

We tried to determine the source of the *Staph. aureus* strains which caused septic infection or colonization, and to relate the risk of either event to the position in the ward of both donor and recipient, and to the presence of the infecting organism in the air. Strains of *Staph. aureus* isolated from lesions, from nasal swabs, and from the air were therefore matched, both as regards their phage-typing patterns and their sensitivity to penicillin and tetracycline. In determining the source of a staphylococcus which was acquired by a patient, or was isolated from the air, we considered only those persons present in the ward at the time who were judged to be carrying an indistinguishable organism.

A major difficulty was that often two or more patients were carrying indistinguishable strains of *Staph. aureus* at the relevant time. In an earlier investigation (Shooter *et al.* 1963) we accepted that, when there were two or more alternative sources for an organism, the nearest should be accepted as the true source. This introduced an element of bias into our results. This time we based our analysis on unambiguously located sources, that is to say, organisms acquired by a patient,

or organisms found in the air, which were carried by one or more members of the staff, or by a single patient-source (see (*f*) below). An exception we made was that an organism carried by a patient at one body-site was always considered to be the source if he or she subsequently acquired it elsewhere.

Because we cultured the nose and lesion of patients, and the nose of members of the staff, only at intervals, we had to adopt certain conventions, rather like those used by Williams *et al.* (1962), about the carriage-state and location in the ward of the patients on each intervening day of the investigation. They are summarized as follows.

(*a*) When two weekly nasal swabs gave different results, a change in carriage-state was deemed to have taken place on the day following the mid-point of the intervening period; but if the number of days between two swabs was odd, the day after the middle day was the one on which the change was considered to have occurred. A few patients were not swabbed on admission, but had a positive first swab. They were considered positive for 3 days before this.

(*b*) The interval between successive nasal swabs of patients occasionally exceeded 7 days. If an organism was present in one swab and absent from the other, its carriage was assumed for half the intervening period, or for a total of 7 days, whichever was the shorter. A few patients were omitted on the swabbing day just before their discharge from hospital. When the last swab to be taken was positive, carriage was assumed up to the day of discharge only if there had been two previous positives for the same strain.

(*c*) Staff swabbing was necessarily more intermittent, because of temporary transfers to other wards and periods of leave. Carriage was therefore assumed for not more than 13 days on either side of a positive swab, and a change in carrier-state was taken to have occurred midway between two relevant swabs.

(*d*) A swab or other specimen from a lesion was taken as a central point, with carriage of 3 days on either side, unless it was preceded or followed by another swab positive for the same organism, when carriage was assumed for up to 14 days between positive swabs.

(*e*) When determining the source of an acquisition, the earliest date of appearance of the organism at any site was the one considered.

(*f*) A single patient-source was usually the only patient carrying the particular strain at the relevant time. On two occasions, however, when two patients in the same subdivision of the ward were carrying indistinguishable strains they were regarded as a single source.

(*g*) A source for a particular infection was considered to be unambiguously located only when neither donor nor recipient had changed their position in the ward since the last swabbing day. Thus a change in bed position caused ambiguity for the six days lying between successive swabbing days.

RESULTS

Incidence of sepsis and wound-colonization

We observed 714 patients in the 20 months between October 1962 and July 1964, and 618 of them underwent 690 surgical operations. In calculating the incidence of post-operative sepsis we excluded 190 operations in which the tissues were not cut, and a further 38 where the patient died within 3 days of operation or where for some reason no wound swab was taken. Wound sepsis followed 11 of the remaining 462 cutting operations, and *Staph. aureus* was isolated from 6 of the swabs, giving a wound-sepsis rate of 2·4 and a staphylococcal wound-sepsis rate of 1·3 per 100 operations. There was one death directly attributable to staphylococcal wound sepsis. *Staph. aureus* was isolated from a further 15 wounds in which there was no clinical evidence of sepsis. Thus, the total wound-colonization rate was 21 in 462 operations, or 4·5 per 100 operations (Table 1).

Table 1. *Staphyloccocal wound sepsis and wound colonization, excluding tracheostomy wounds*

(In parenthesis: rate per 100 operations. Cardiac (*a*): operations with artificial circulation and profound hypothermia. Cardiac (*b*): without hypothermia.)

	Number performed		Staphylococcal wound sepsis		Total staphylococcal colonization*	
	M	F	M	F	M	F
Operations						
Cardiac (*a*)	53	32	2 (3·8)	0	6 (11·3)	3 (9·4)
Cardiac (*b*)	42	132	1 (2·4)	0	1 (2·4)	2 (1·5)
Pulmonary	103	33	2 (1·9)	0	6 (5·8)	0
Other cutting operations	36	31	1 (2·8)	0	3 (8·3)	0
Total cutting operations	234	228	6 (2·6)	0	16 (6·8)	5 (2·2)
	462		6 (1·3)		21 (4·5)	

* Staphylococcal wound sepsis plus colonization of wounds.

This table shows also that staphylococcal wound sepsis was found only among male patients, in whom the incidence was 2·6 per 100 operations. Total colonization of wounds with *Staph. aureus* was also more frequent in male than in female patients (respectively 6·8 and 2·2 per 100 operations).

Tracheostomy wounds were not included in these totals, because the clinical significance of the presence of *Staph. aureus* in them was difficult to assess. The organisms present were usually those also isolated from the sputum.

Staphylococcal sepsis occurred at sites other than the operation wound in 20 of the 714 patients (2·8%), and was more common in males (17 out of 393: 4·3%) than in females (3 out of 321: 0·9%). It was seen only in those who had had an operation. In all, six of these patients were seriously ill, and four suffered from empyema, but no death was directly attributable to any of these infections.

Post-operative wound sepsis was, therefore, not common in the Thoracic

https://doi.org/10.1017/S0022172400040602 Published online by Cambridge University Press

Surgery unit, despite the severity of many of the operations performed. Indeed, sepsis rates were lower than those seen in general surgical wards in the same hospital (Williams *et al.* 1962; Shooter *et al.* 1963), which were of the order of 6 % for total wound sepsis, 4–5 % for staphylococcal wound sepsis, and 4 % for other staphylococcal sepsis.

Staphylococcus aureus *in the ward air*

We examined the staphylococci isolated from the ward air for evidence that the design of the ward prevented or hindered the dispersal of staphylococci by the airborne route. The average number of staphyloccoci in the air on the 28 days of sampling was only 35 *Staph. aureus* per 1000 cu.ft. (28·3 m.^{3}), several times lower than the values we had observed earlier in open surgical wards in St Bartholomew's Hospital (Noble, 1962). Numerous measurements in three wards in the years 1956–61 gave average counts of 220, 180 and 180 per 1000 cu.ft. respectively. In 1961, one of these wards was divided by a partition (Shooter *et al.* 1963), and the following year the counts were rather lower, with means of 70 and 80 per 1000 cu.ft. on the two sides of the ward.

There were considerable variations in the count from day to day, and from one room to another in the thoracic surgery ward, but there were few 'broadcasts' of staphylococci resulting in very high counts. Indeed 90 % of all counts were less than 80/1000 cu.ft. The corresponding 90-percentile figure for the open wards (Noble, 1962) was 600/1000 cu.ft. One really high count (1150/1000 cu.ft.)—twice as great as the next highest—was obtained in a cubicle occupied by a moribund patient.

Table 2. *Recovery of airborne* Staphylococcus aureus *in relation to presumed source*

(Colonies per 1000 cu. ft. air attributable to a single source-carrier. (No air-samples were collected in rooms B3 and BR.))

Recovery from air. Room	Location of source carrier. Room: A	B1, B2	B3, BR	C1, C2, C3, C4	Staff	No known source*
A	**5·25**	0·23	0·23	1·68	0·24	1·74
B: B1, B2	0·80	0·68	0·11	1·68	0·07	3·29
B: Same room	—	**4·73**	—	—	—	—
C: C1, C2, C3, C4	0·62	2·59	<0·11	3·36	0·18	2·82
C: Same room	—	—	—	**75·2 (21·9†)**	—	—

* Colonies per single strain, source carriers being unidentifiable

† Excluding one exceptionally high count.

Figures in bold are recoveries in the same room as the presumed source.

Dissemination of staphylococci from known sources

Staphylococci isolated from the air were allotted when possible to known carriers in the ward. A total of 630 isolations had been made; 410 of them (65 %) could be attributed to a single carrier-source, 69 (11 %) appeared to have no source in the ward, and 151 (24 %) were strains for which there was more than one carrier-source.

https://doi.org/10.1017/S0022172400040602 Published online by Cambridge University Press

Our main analysis was confined to those for which there appeared to be a single source.

Table 2 shows the recovery of airborne organisms in the same room as the source, and in other rooms, expressed as colonies recovered from 1000 cu.ft. of air. A single carrier-source produced a much higher count in the room in which he or she was placed than in other rooms. The difference was of the order of tenfold, whether the patient's room was a large one or a small one. Dissemination by members of the staff was detected at a rather low level throughout the ward.

Table 3. *Recovery of airborne* Staphylococcus aureus *in relation to antibiotic resistance*

(Colonies per 1000 cu. ft. air attributable to a single source carrier. S = sensitive to penicillin and tetracycline. P = resistant only to penicillin. T = resistant to penicillin and tetracycline; or to tetracycline alone.)

Location of source carrier	Antibiotic resistance of strains isolated			All organisms
	S	P	T	
Patients: same room	18·2 (4·1*)	4·4	33·3	15·2 (7·5*)
Patients: other rooms	0·75	0·4	2·42	0·84
Staff	0·04	0·23	<0·40	0·16
No known source	2·50	2·32	7·20	2·62

* Excluding one exceptionally high count.

Table 4. *Average number of nasal carriers of* Staphylococcus aureus *to whom a patient was exposed (a) in the same room, (b) in other rooms, and (c) among the staff*

Exposure to	Antibiotic resistance of carried strain			All carriers	All persons
	S	P	T		
Patients: same room	0·57	0·52	0·32	1·42	4·6
Patients: other rooms	2·92	2·78	1·66	7·35	24·4
Staff	2·54	9·71	1·14	13·39	34·4

The last column of Table 2 shows that strains for which no source could be identified appeared in larger numbers in sections B and C of the ward than in section A. This supports the view that a proportion of them were organisms reaching the ward in the air from the stair-well.

When we examined the antibiotic resistance of the airborne organisms emanating from known sources (Table 3) we found that the dispersal of multiple-resistant *Staph. aureus* by carriers among the patients was several times as profuse as the dispersal of sensitive organisms or of those resistant only to penicillin.

Although a patient in a room was more heavily exposed to a source of airborne infection in that room than to one in another room, the number of potential sources in the other rooms exceeded those in the same room (Table 4). We therefore attempted to compare the total amount of exposure of the average patient to

sources in the same and in different rooms. The relative contributions of patients in the same and in other rooms varied in the different rooms according to the number of patients they contained. Since, however, the number of cases of sepsis and of nasal acquisition observed in the investigation was not very large, it was necessary to combine them into a few groups in order to obtain reasonable numbers in each. From the average carrier-rate of patients in each room and the figures for airborne dispersal given in Table 2 we calculated the effective exposure to airborne organisms derived from all patients in the same and in different rooms, from the staff, and from unknown sources, further subdividing this according to the antibiotic resistance of the organisms (Table 5). Total exposure to potential patient-sources in other rooms (6·3/1000 cu.ft.) was about two-thirds of the total exposure to sources within the room (9·8/1000 cu.ft.). For resistant strains, however, sources in other rooms contributed only about one-half as much as sources within the same room, while for sensitive strains they contributed twice as much. This would imply either that sensitive strains were disseminated more readily from room to room or, more probably, that the carriers of resistant strains were not randomly distributed through the rooms of the ward but tended to be grouped in the same room.

Table 5. *Effective exposure to airborne staphylococci. Colonies of* Staphylococcus aureus *per* 1000 *cu.ft. air*

	Antibiotic resistance of strain			
Source of organisms	S	P	T	All organisms
Patients: same room	1·1	1·8	6·9	9·8
Patients: other rooms	2·0	1·1	3·2	6·3
Staff	0·1	0·9	$<0·1$	1·0
No known source	3·9	4·0	0·8	8·7
Total	7·1	7·8	10·9	25·8

Nasal carriage of Staphylococcus aureus

Unexpected changes were found in the nasal carriage of *Staph. aureus* by patients during successive weeks of their stay in hospital (Fig. 2). In the first 2 weeks there was a reduction in the total carriage-rate from 38 % to 25 %. This was accounted for almost entirely by a loss of sensitive organisms. This loss continued, though at a rather slower rate, for the rest of the stay of patients in the ward, so that between three-quarters and four-fifths of all sensitive organisms had disappeared by the end of the 6th week. A substantial number of patients carried tetracycline-resistant organisms on admission, but the rise from 6 % to a maximum of 9 % while in hospital was a relatively small one.

These findings contrasted sharply with our earlier experiences in open surgical wards (Williams *et al.* 1962) in which the total carrier-rate increased with the duration of stay in hospital, and the percentage of patients carrying staphylococci resistant both to penicillin and to tetracycline increased at least sevenfold in 6 weeks (Fig. 3C–E). The findings in the current investigation (Fig. 3A) exhibit in rather more extreme form the situation we observed among patients nursed in cubicles communicating with the open air, many of whom were also receiving

antibiotics (Parker, John, Emond & Machacek, 1965; see also Fig. 3B). Here, the total carrier rate was substantially unchanged, many sensitive staphylococci were lost, and the increase in multiple-resistant organisms was trivial.

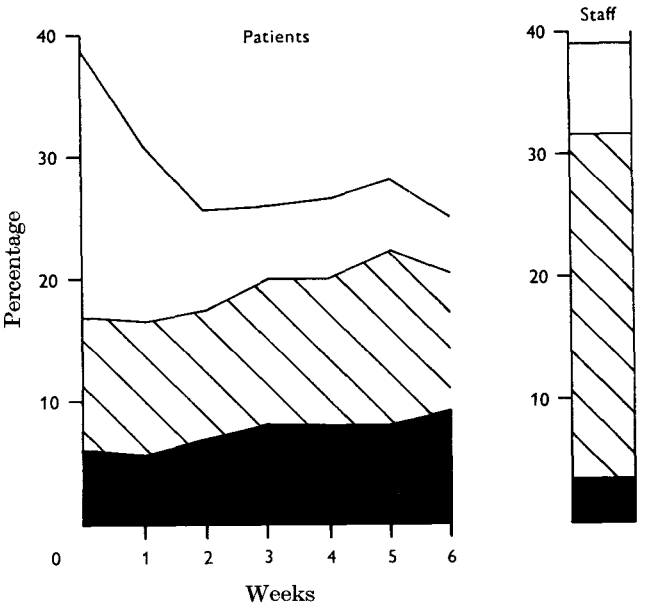

Fig. 2. Nasal carriage of *Staph. aureus* by patients during each week of stay in the thoracic surgery ward. Average rate of nasal carriage by members of the staff. □, Sensitive to penicillin and tetracycline; ▨, resistant to penicillin only; ■, resistant to tetracycline.

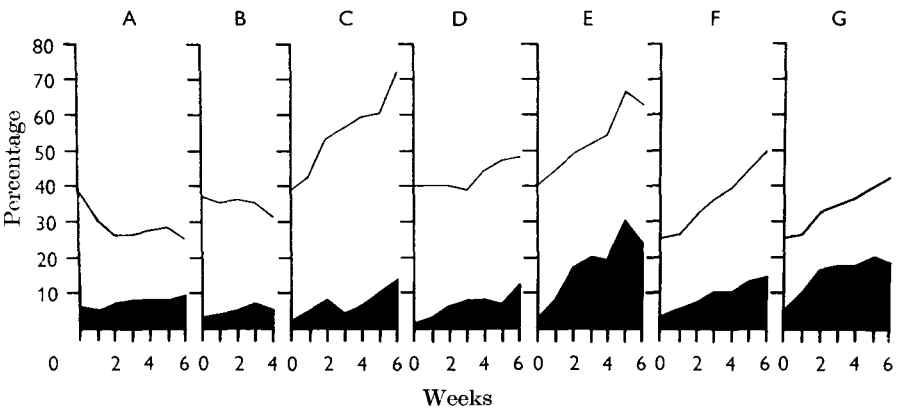

Fig. 3. Nasal carriage of *Staph. aureus* by patients in various hospital wards. Percentage positive in each week of stay in the ward. Upper line: all strains of *Staph. aureus*. Lower line: tetracycline-resistant *Staph. aureus*. [A, B, F, and G: all tetracycline-resistant strains; C, D, and E: all strains resistant to penicillin and tetracycline.] A, This investigation; B, patients in cubicles (Parker *et al.* 1965); C, D, E, three open surgical wards (Williams *et al.* 1962). F, medical wards: patients not receiving antibiotics; G, Medical wards: patients receiving antibiotics. (F, G. P.H.L.S. Cross-Infection Committee (unpublished results).)

It appeared, therefore, that a large proportion of the sensitive staphylococci present on admission to the thoracic surgery ward were being removed or suppressed by antibiotic treatment, and were not being replaced by multiple-resistant organisms, as so frequently happens in other wards. This was remarkable in view of the very high rate of administration of antibiotics, and the often-repeated observation that multiple-resistant staphylococci are more rapidly acquired by those who receive antibiotics than by those who do not (Fig. 3F, G.)

Apparent acquisition of new strains of Staphylococcus aureus

Ninety-six strains of *Staph. aureus* not found on admission were subsequently isolated from the nose swabs of patients. The rate of apparent acquisition in 2790 patient-weeks of exposure was therefore 3·44 per 100 patient-weeks, a figure little more than half of that seen in patients isolated in cubicles (Parker *et al.* 1965). There is, however, reason to believe that a considerable proportion of 'apparent' acquisition is an artifact due to the intermittent isolation of organisms present before admission; most of this spurious acquisition is of sensitive organisms, or organisms resistant only to penicillin, and is first detected in the swabbings immediately following the admission swab. A high rate of antibiotic administration would tend to reduce this spurious element in acquisition, and lower the total rate of apparent acquisition. The rate of apparent acquisition of multiple-resistant staphylococci, which are less often found on admission, is therefore a better index of the exposure to infection in hospital. In the present investigation, this figure was 1·33 per 100 patient-weeks, which corresponded closely with the figure of 1·40/100 patient-weeks for patients in cubicles. Comparable figures for the apparent acquisition of multiple-resistant organisms in open wards range from 2 to 12 per 100 patient-weeks (Parker *et al.* 1965).

Sources of nasal acquisition

Table 6 summarizes our attempts to find sources for the 96 apparent acquisitions of *Staph. aureus*. One or more possible sources were identified for 49 of them (51 %), but there were a number of uncertainties. Three acquisitions could be attributed unambiguously to patients in the same room, and 12 to patients in other rooms, but nine further acquisitions were from patients of uncertain location, either because donor or recipient had moved recently, or because two or more patients were carrying identical organisms. Nine acquisitions could have been either from patients or from members of the staff and 16 were definitely attributable to the staff. We attempted to clarify this by distributing the acquisitions where the source or its location were uncertain according to the observed distribution of acquisition from sources with unambiguous locations. The picture that emerges is that 3–4 times as many new strains were acquired from patients in other rooms—and from members of the staff—as from patients in the same room.

There was a preponderance of antibiotic-sensitive strains among the 47 for which there was no known source. This, and the fact that most of the apparent acquisitions of sensitive organisms occurred early in the stay in hospital, is in conformity with the view that many of them were spurious. To estimate what

proportion of those with no known source were 'true' acquisitions we divided them into two parts; (*a*) a fraction with a distribution of antibiotic sensitivity similar to that for acquisitions from known sources, and (*b*) a fraction with a distribution of antibiotic sensitivities similar to that in the patients' admission swabs (Table 6, lines 7 and 8). These fractions might be expected to correspond respectively to 'true' acquisitions from undetected sources in the hospital, and to spurious acquisitions. The result suggested that 24 of the 47 'acquisitions' with no known source—including 14 of the 16 penicillin-sensitive organisms—were spurious.

Table 6. *Source of nasal acquisitions of* Staphylococcus aureus

((1) The figures in parentheses were obtained by distributing those acquisitions where the source or its location were uncertain (lines 3 and 4) according to the observed distribution of acquisition from sources whose location was unambiguous. (2) Acquisition from no known source has been divided into two fractions with distribution of antibiotic sensitivities similar to (*a*) that for acquisition from known sources and (*b*) that found in admission-swabs (see text).)

Source	Antibiotic resistance of acquired strain			Total acquired
	S	P	T	
1. Patients in same room	0 (0·6)	0 (0)	3 (5·2)	3 (5·8)
2. Patients in other rooms	0 (0·6)	4 (6·5)	8 (13·8)	12 (20·9)
3. Patients, location uncertain	1	0	8	9
4. Patients or staff	1	8	0	9
5. Staff	3 (3·7)	9 (14·5)	4 (4·0)	16 (22·2)
6. No known source	16	17	14	47
7. No known source (*a*)	2	10	11	23
8. No known source (*b*)	14	7	3	24
Total	21	38	37	96

Table 7. *Rates of acquisition of* Staphylococcus aureus *from single sources*

(Rate per 1000 patient-weeks exposure per source. The figures in parentheses are derived from an analysis confined to the 31 acquisitions where source and recipient were unambiguously situated. The remaining figures were obtained after redistribution of the acquisitions from known sources where the location was ambiguous (see Table 6). Figures in square brackets are based on insignificant numbers of acquisitions (< 1).)

Source	Antibiotic resistance of acquired strains			All acquisitions
	S	P	T	
1. Patients in same room	[0·35]	[0·38]	5·9	1·48 (1·93)
2. Patients in other rooms	[0·07]	0·84	3·1	1·04 (0·97)
3. Staff	0·47	0·54	1·2	0·58 (0·70)
4. All known sources	0·27 (0·32)	0·58 (0·70)	2·69 (2·54)	0·79 (0·82)

Rates of acquisition from single carriers

Table 7 shows the nasal acquisition rates per 1000 patient-weeks' exposure per known source. The figures in the body of the table were derived from all the acquisitions for which there was a source, after redistribution of those that were ambiguously located. Those in parentheses were obtained from an analysis

https://doi.org/10.1017/S0022172400040602 Published online by Cambridge University Press

confined to acquisitions where source and recipient were unambiguously located throughout the relevant period. The numbers are small, but serve to confirm the results of the less complicated analysis. They suggest that acquisition from a single carrier occurs about twice as often when he is in the same room as when he is in another room. This is about the same as the difference previously observed between adjacent and remote beds in an open ward (Williams *et al* 1962). Acquisitions from a carrier on the staff occurred at about half the rate of acquisitions from carriers in other rooms. This may reflect the fact that members of the staff spend only part of the day in the ward.

There was a numerical preponderance of tetracycline-resistant staphylococci among those acquired from other patients, and of organisms resistant only to penicillin among those acquired from the staff (Table 6). The rate of acquisition (Table 7) of tetracycline-resistant organisms per patient-source was fairly high (5·9/1000 patient weeks in the same room), but the rate for penicillin-resistant organisms per staff-source (0·54/1000 patient-weeks) was less than the average for all acquisitions from single sources. This suggests that patients with tetracycline-resistant organisms were relatively infectious, and that carriers of penicillin-resistant organisms on the staff, although numerous, were not individually very dangerous.

Exposure to airborne staphylococci and risk of nasal acquisition

The distribution of sources of nasal acquisition (Table 6) is closer to the distribution of the number of carriers (Table 4) than to the amount of exposure to airborne organisms derived from them (Table 5). This is compatible with a non-linear relationship between dose and risk of infection such as would result from differences between patients in their susceptibility to colonization or between staphylococcal strains in their transmissibility. Variations in the dose of organisms would then produce proportionately smaller changes in the risk of infection (Lidwell, 1963). Figure 4 shows the relationship between the risk of nasal acquisition and the exposure to airborne organisms for all strains and for tetracycline-resistant strains. The results are consistent with the risk varying as the one-fifth power of the exposure. For this population, an average inhaled dose of 10 airborne staphylococcal particles of a single strain corresponded to a probable risk of nasal acquisition of about 1 in 800 or 1 in 300 for tetracycline-resistant staphylococci.

Sources of staphylococci causing sepsis and wound colonization

Forty-two strains of *Staph. aureus* were isolated from wounds or from staphylococcal lesions elsewhere. Table 8 shows their presumed sources. Twenty of them (48 %) were isolated from the patient on admission to hospital and must therefore be considered self-infections; nine of them, however, were due to *Staph. aureus* strains that were resistant both to penicillin and to tetracycline, and can probably be attributed to a previous hospital admission. Eight (19 %) were from unknown sources. This number is high, but included a sudden 'burst' of four 83A infections which could not be traced, and were possibly due to failure to detect a person carrying the organism at a site other than the nose.

The remaining 14 infections were undoubtedly acquired from sources in the ward. The six that were probably acquired from other patients were, however, too few to allow any conclusions to be drawn about the relative importance of sources of infection in the same or in different rooms. This illustrates the difficulty of investigating clinical sepsis when its incidence is low.

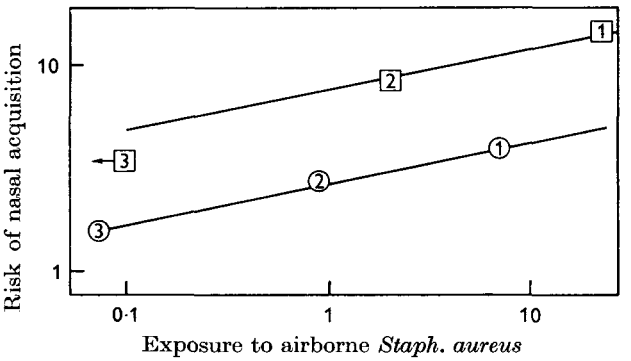

Fig. 4. Relation between the risk of nasal acquisition and the exposure to airborne *Staph. aureus*. Logarithmic scales for both co-ordinates. Risk of nasal acquisition: acquisitions per potential source (carrier) per 2790 patient weeks. Exposure to airborne *Staph. aureus*: colony count per carrier per 1000 cubic foot of air sampled. Lines drawn with a slope of 0·20. ○, All strains; □, tetracycline-resistant strains. Location of carriers: 1, patients in the same room; 2, patients in other rooms; 3, staff.

Table 8. *Presumed source and antibiotic resistance of* Staphylococcus aureus *strains which caused wound sepsis, wound colonization and other septic lesions*

	Presumed source					
	Self	Another patient or patients	Staff	Staff or patient	Not known	Total
Infection						
Wound sepsis	3	1	1	0	1	6
Wound colonization	7	2	1	3	2	15
Other septic lesions*	10	3	3	0	5	21
Sensitivity of infecting organism						
S	2	0	0	0	0	2
P	9	0	2	3	1	15
T	9	6	3	0	7	25
Total	20	6	5	3	8	42

* Two organisms from one lesion.

DISCUSSION

The incidence of staphylococcal sepsis was low in this ward, but it proved unexpectedly difficult to decide why this was so. There are great differences in the reported incidence of post-operative sepsis following operations on the chest, but

https://doi.org/10.1017/S0022172400040602 Published online by Cambridge University Press

there seems no reason to suppose that the risk of infection in this branch of surgery is inherently small. Thus, while Hansen & Eriksen (1964) recorded a total sepsis-rate of less than 2% following thoracic operations over a 10-year period, others have reported the incidence of staphylococcal sepsis to be as high as 8–11% (Blowers, Mason, Wallace & Walton, 1955; Report, 1960; Laurell & Lindbom, 1961; Bassett *et al.* 1963; Lindbom, 1964). Many factors, including selection of patients, operative techniques, the use of antibiotics, ward procedures and the methods of recording sepsis may have contributed to these differences. In addition, high rates of sepsis have sometimes been associated with particular sources of infection in the operating theatre (Blowers *et al.* 1955; Bassett *et al.* 1963).

We found that staphylococcal sepsis and wound colonization were both more frequent in male than in female patients. The males were usually nursed in the 10-bedded ward and the females in smaller units, but the males were on the whole older than the females, underwent different operations, and received more antibiotics. We are unable, therefore, to draw any conclusions from this observation.

Not only was the total incidence of sepsis low in our investigation, but nasal colonization, including the acquisition of endemic strains of hospital staphylococci, was infrequent, and the numbers of staphylococci present in the air of the ward were much lower than those we have previously observed in other wards in the hospital.

While the natural ventilation rate of the ward was probably greater than usual and the relatively spacious construction resulted in a low patient density, these differences were not sufficiently great to provide in themselves a convincing explanation of the low air counts.

The administration of antibiotics will usually reduce the level of carriage of sensitive strains even when it does not eliminate them from the nose of the patients concerned. When this occurs the amount of dispersal of these strains into the environment is also reduced (Bøe & Solberg, 1965). Usually where there has been extensive prophylactic use of antibiotics the removal or suppression of the sensitive strains has been accompanied by a widespread acquisition of resistant 'hospital' strains. In this ward we saw a massive removal of the sensitive staphylococcal population following antibiotic treatment but very little replacement by resistant strains, although potential sources of infection with these were present in the ward for most of the time. Since many patients had been in hospital previously a substantial percentage were admitted carrying such strains.

The low air counts would seem then to be mainly due to the suppression, by antibiotics, of widespread dispersion by carriers of sensitive strains unaccompanied by the appearance of carriers of resistant strains. The rate of dispersal of their staphylococci by carriers of resistant strains was, on the average, about three times as great as that of carriers of sensitive strains (Table 3).

In looking for reasons to explain the low rate of spread of staphylococci within this ward the subdivided construction needs to be considered first. Subdivision could reduce the spread of micro-organisms in two ways.

It might influence nursing procedures in such a way as to reduce the risk of

contact-transfer from patients in one room to patients in another, e.g. by increasing the probability of the nurses washing or performing some other duty between attending to patients in different rooms. There was however more acquisition of staphylococci from patient-carriers situated in other rooms than from those in the same room. It does not, therefore, seem likely that changes of this kind in nursing procedure, if they occurred, were such as to influence the spread of staphylococci.

Alternatively, the subdivision might isolate the patients from each other by hindering the movement of airborne organisms from one part of the ward to another. Smoke and tracer gas studies, however, together with air sampling for staphylococci, showed that in fact there was still substantial interchange of air between the several parts of the ward. Although in some wind and weather conditions the upward current of air in the stair-well of the building, which entered the ward passage through the swing doors at the end of the corridor, swept through this and passed out through the several rooms in a regular manner, there was usually substantial exchange of air between these rooms and the corridor, caused by thermal differences. As in a previous investigation where a ward was divided into two parts by a partition with sliding doors (Shooter *et al.* 1963), it became clear that division of a ward unaccompanied by ventilation to control the direction of air movement does little to prevent the spread of staphylococci from room to room by the aerial route. In fact patients in one room were exposed to organisms from sources in other rooms almost as much as if these had been sources at the far end of a large open ward. We are left therefore without any simple explanation of the undoubted good results obtained. The best that we can offer is the suggestion that the combination of subdivision, spaciousness and above average ventilation reduced the risk of spread below a threshold level so that the generous administration of antibiotics was not accompanied by widespread colonization with resistant strains.

The widespread prophylactic use of antibiotics is undoubtedly hazardous, although it may be justified in a few situations, of which major thoracic surgery is widely held to be one (Eriksen & Hansen, 1964). If it is practised the patients should be segregated from each other as strictly as possible and no antibiotic should be given to a patient already colonized with an organism resistant to it. This second condition was not always observed in our studies, which may have contributed to the fact that our carriers of multiple-resistant strains disseminated more heavily than did the carriers of sensitive organisms; many were undoubtedly receiving antibiotics to which their nasal strains were resistant. This would have been likely to have increased profuseness of carriage and the extent of dispersion (Ehrenkranz, 1964).

SUMMARY

We studied the incidence of staphylococcal infection in a thoracic surgery ward which consisted of a number of separate rooms, and inquired whether the subdivision of the ward was responsible for the unusually low sepsis-rate.

The airborne dissemination of *Staphylococcus aureus* from one room to another appeared to be little less than that in an open ward; but the total number of *Staph. aureus* in the air was very low.

Most of the patients received prophylactic antibiotics. The nasal carrier-rate of *Staph. aureus* by patients fell greatly during their stay in the ward. There was a progressive disappearance of sensitive organisms and little acquisition of multiple-resistant organisms.

When there are urgent clinical grounds for the lavish use of antibiotics, the dangers appear to be reduced by effective segregation of the patients from each other.

We are grateful to our surgical colleagues, Mr O. S. Tubbs and Mr I. M. Hill, for permission to study their patients, and to the Treasurer and Governors of St Bartholomew's Hospital for financial support. We wish also to thank the Sister and nursing staff of the ward for their co-operation, and Miss Anne Brooke, Miss Madeleine John, B.Sc., Mrs Janet Robertson, B.Sc., and Miss Veronica Williams, B.Sc., for assistance in our laboratories.

REFERENCES

Bassett, H. F. M., Ferguson, W. G., Hoffman, E., Walton, M., Blowers, R. & Conn, C. A. (1963). Sources of staphylococcal infection in surgical wound sepsis. *J. Hyg., Camb.*, **61**, 83.

Blowers, R., Mason, G. A., Wallace, K. R. & Walton, M. (1955). Control of wound infection in a thoracic surgery unit. *Lancet* ii, 786.

Bøe, A. J. & Solberg, C. A. (1965). Behandling av nasale stafylokokkbaerere med fucidin. *Tidsskr. norske Laegeforen.* **85**, 527.

Ehrenkranz, N. J. (1964). Person-to-person transmission of *Staphylococcus aureus*; quantitative characterization of nasal carriers spreading infection. *New Engl. J. Med.* **271**, 225.

Eriksen, K. R. & Hansen, J. L. (1964). Prophylactic use of antibiotics in surgery of the lung. *Acta chir. scand.* **128**, 651.

Hansen, J. L. & Eriksen, K. R. (1964). Postoperative infection in thoracic surgery. *Acta chir. scand.* **128**, 698.

Laurell, G. & Lindbom, G. (1961). Wound infections due to *Staphylococcus pyogenes* in a thoracic surgical unit. *Acta chir. scand.* **121**, 165.

Lidwell, O. M. (1963). Methods of investigation and analysis of results. In *Infection in Hospitals*, p. 43. Ed. R. E. O. Williams and R. A. Shooter, Oxford: Blackwell Scientific Publications.

Lindbom, G. (1964). Studies of the epidemiology of staphylococcal infections. II. Staphylococcal infections in a thoracic surgery unit. *Acta chir. scand.* **128**, 421.

Noble, W. C. (1962). The dispersal of staphylococci in hospital wards. *J. clin. Path.* **15**, 552.

Parker, M. T., John, M., Emond, R. T. D. & Machacek, K. A. (1965). Acquisition of *Staphylococcus aureus* by patients in cubicles. *Br. med. J.* i, 1101.

Report (1960). Incidence of surgical wound infection in England and Wales. *Lancet* ii, 659.

Shooter, R. A., Thom, B. T., Dunkerley, D. R., Taylor, G. W., Parker, M. T., John, M. & Richards, I. D. G. (1963). Pre-operative segregation of patients in a surgical ward. *Br. med. J.* ii, 1567.

Williams, R. E. O., Noble, W. C., Jevons, M. P., Lidwell, O. M., Shooter, R. A., White, R. G., Thom, B. T. & Taylor, G. W. (1962). Isolation for the control of staphylococcal infection in surgical wards. *Br. med. J.* ii, 275.