

## Observations on the distribution of *Staphylococcus aureus* in the atmosphere of a surgical ward

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### INTRODUCTION

The presence of *Staphylococcus aureus* in the air and bedding of hospital wards has been reported by many investigators (e.g. Blowers, Mason, Wallace & Walton, 1955; Shooter, Smith, Griffiths, Brown, Williams, Rippon & Jevons, 1958; Colbeck, 1956; Howe, Silva, Marston & Woo, 1961). The significance that such organisms may have in the causation of hospital-acquired staphylococcal sepsis has not, however, been clearly established.

The recovery of *Staph. aureus* in large numbers from woollen blankets (Blowers & Wallace, 1955) has led to the introduction of cotton blankets in surgical wards, since cotton materials can be laundered more often and at higher temperatures than can woollen ones and so freed of staphylococci. Pressley (1958) has challenged the concept that cotton blankets are preferable to woollen ones and has published evidence that the majority of fibres (presumably bacteria-carrying) in ward air are of cellulose rather than protein.

The substitution of cotton for woollen blankets is based on the assumption that the staphylococci recovered from ward bedding may be responsible for sepsis in surgical wounds. Evidence for this view is largely circumstantial and, indeed, direct evidence may be very difficult to obtain. The introduction of cotton blankets has often been accompanied by other measures designed to control wound infection. Gillespie, Alder, Ayliffe, Powell & Wypkema (1961) in a 4-year study found that no one measure was successful in reducing the incidence of wound infection and that a combination of measures dealing with nasal carriage, dust and bedding was required to achieve this. Rountree (1947) found that the use of oiled woollen blankets had no effect on the incidence of staphylococcal sepsis in a surgical ward.

An investigation of the possible routes of infection of surgical wounds in a particular surgical unit (Rountree, Harrington, Loewenthal & Gye, 1960) produced evidence that, apart from self-infection with staphylococci present in the noses of the patients when they were admitted to hospital, infection of the wounds occurred in the ward, rather than in the operating theatres.

Preliminary evidence of the contamination of the ward environment having been obtained, more extensive investigations were undertaken. These included weekly sampling of the ward air and bedding, and of the patients' noses and bacteriological examination of all infected wounds. The object was to obtain information on the contamination of bedding by nasal carriers and by clinically infected persons,

to compare the bacterial load of woollen and cotton blankets and to see what part contaminated bedding might play in the initiation of nasal carriage. During certain periods of the investigations a trial of nasal prophylaxis with a chlorhexidine-neomycin (hibitane) cream was carried out.

#### METHODS

The wards have been described in detail (Rountree *et al.* 1960). Briefly, they comprise two old open wards parallel to each other and sharing common services and staff. During the present study the men's ward had an average of fourteen beds and the women's ward twelve beds.

Air samples were taken once a week in the men's ward for 12 months from November 1960 to October 1961, with the exception of a 6 weeks' break in February and March. The women's ward was sampled weekly for 7 months from April to October 1961.

The samples were taken immediately before, during and after bedmaking (between 1 and 2 p.m.), two methods being used. Settle plates were exposed for 60 min. on bedside lockers 3 ft. above floor level, the same nine sampling positions being used each week. Air samples of 5 cu.ft. were taken by a small model Bourdillon slit sampler, running at a rate of 1 cu.ft. per min. The samples were taken at the ends of the beds adjacent to which the settle plates were exposed. A total volume of 45 cu.ft. of air was usually taken on each occasion. The sampler was mounted on a trolley and the air inlet was 3 ft. 6 in. above floor level. Blankets were sampled weekly in both wards for 12 months, the topmost blanket on each bed being examined. The number of blankets on each bed varied with the weather. The sweep plate method of Williams (Blowers & Wallace, 1955) was used; six sweeps were taken over an area of 12 sq.in. since it was found that a larger number of sweeps often resulted in overcrowded plates impossible to count. The plates used were plastic Petri dishes, 3¼ in. in diameter and with sharp edges. All the sampling was carried out by one person in an attempt to minimize errors due to variations in technique.

The medium used for sampling of both air and blankets was phenol phthalein phosphate agar (P.P.P.) (Barber & Kuper, 1951). A series of settle plates using the mercuric chloride agar medium of Moore (1960) proved unsatisfactory as low staphylococcal counts were obtained compared with the P.P.P. agar. Plates were incubated at 37° C. for 18 hr. before counting. Colonies of *Staph. aureus* were identified by exposure to ammonia vapour, counted and all or a proportion picked off and subcultured.

All isolates suspected of being *Staph. aureus* were tested for coagulase by the slide method of Cadness-Graves, Williams, Harper & Miles (1943). The coagulase positive strains were phage typed using the basic set of international phages (Blair & Williams, 1961) namely: 29, 52, 52A, 79, 80; 3A, 3B, 3C, 55, 71; 6, 7, 42E, 47, 53, 54, 75, 77; 42D; 81; 187 and three phages of local value, 31B, 47D and 315.

A nasal swab was taken from each patient as soon after admission as possible. Thereafter all patients were swabbed once each week. All strains of *Staph. aureus* isolated were phage typed.

## RESULTS

*Types of Staphylococcus aureus isolated*

More than 3600 isolations of *Staph. aureus* were phage typed. The phage group distributions found are shown in Table 1, which gives the figures for the men's and women's wards separately. The figures for blankets and noses in the women's ward are for 12 months and those for the settle plates and air samples are for the 7 months from April to October 1961.

Table 1. *Phage group distribution of strains of Staphylococcus aureus isolated in two adjoining wards*

(The strains were isolated from November 1960 to October 1961, with the exception of the air sample and settle plate strains in the women's ward which were isolated from April 1961 to October 1961.)

Phage groups	Men's ward							
	Settle plates		Air samples		Blankets		Patients' noses	
	No.	%	No.	%	No.	%	No.	%
I	34	4.8	20	4.0	44	4.7	35	9.8
II	15	2.1	5	1.0	23	2.4	21	5.9
III 47	498	72.2	377	75.6	599	64.3	146	40.9
Others	101	14.6	74	14.8	173	18.5	82	23
IV	2	—	—	—	—	—	—	—
M	6	—	3	—	4	—	8	—
N.C.	—	—	—	—	—	—	2	—
N.T.	33	4.8	20	4.0	90	9.7	63	17.6
Total typed	698	—	499	—	933	—	357	—
Total isolated	1718	—	1121	—	4240	—	—	—
	Women's ward							
I	8	4.6	3	—	39	5.8	33	12.3
II	7	4.1	4	—	23	3.4	22	8.2
III 47	90	52	50	51.5	395	58.4	96	35.8
Others	50	28.9	30	30.9	119	17.6	44	16.4
IV	—	—	—	—	2	—	12	—
M	—	—	—	—	13	—	2	—
N.C.	—	—	—	—	—	—	2	—
N.T.	18	10.4	10	—	84	12.5	57	21.3
Total typed	173	—	97	—	675	—	268	—
Total isolated	228	—	111	—	1,865	—	—	—

The distributions for the air and blankets are not strictly comparable with those from the nasal swabs. Unless there were colonial differences, only one colony from each positive nasal swab was typed, and many swabs yielded hundreds of colonies. A much larger proportion of all the staphylococci present on the air and blanket plates was typed.

In both wards the predominant strain was resistant to penicillin, streptomycin, tetracycline and chloramphenicol and often also to erythromycin and novobiocin. It was lysed only by phage 47 at  $1000 \times$  R.T.D.

When broth cultures of this strain were inoculated on to mercury plates they grew profusely; however, the counts of this strain on mercury agar settle plates were always significantly lower than on P.P.P. settle plates.

There was a close correspondence between the phage-group distributions of the strains isolated from the air samples, settle plates and blankets in both wards, which indicated a common origin of these staphylococci.

There was less correspondence with respect to the strains from the patients' noses, where a higher proportion of strains belonged to phage group I or were not typable. Many of such strains were isolated on only one occasion from the swabs taken on admission before the patients received naseptin; there would therefore be little opportunity for such strains to be incorporated into the flora of the ward atmosphere.

Table 2. *Phage types of Staphylococcus aureus causing clinical infection from November 1960 to October 1961 (including wounds, sputum and skin infections)*

Phage types	Men's ward.	Women's ward.	Total
	No. of patients	No. of patients	
47 at $1000 \times$ R.T.D.	24 (75 %)	6 (37 %)	30
Other Group III strains	3	3	6
Other types	1	3	4
Not typable	4	4	8
Total	32	16	48

During the 12 months, there were 48 cases of staphylococcal sepsis in the two wards. Table 2 gives the phage type distribution of the strains responsible. In the men's ward 24 out of 32 infections (75 %) were due to phage Type 47 staphylococci; these included 3 cases of staphylococcal pneumonia. In the women's ward, there were 16 infections and only 6 were caused by the Type 47 strains. No other strain, with one exception, was isolated from more than one patient; the exception was two patients infected with phage Type 52/52A/80/81 between whom there was no apparent connection.

The prevalence of one strain in the air, blankets and patients' noses was therefore associated with its occurrence as the chief cause of sepsis in the ward.

#### *The bacterial load in the ward air*

The methods of sampling were designed to measure the bacterial load in the ward air at times of maximum activity, when the bacteria released by the movement of bedclothes were circulating in the ward atmosphere.

Representative counts obtained in the two wards during the same week are shown in Table 3. In the men's ward, there was a high level of staphylococcal contamination, with a mean count of 1.7 colonies of *Staph. aureus* per cu.ft. and a mean count of 1.7 colonies settling per sq.ft./min. The cocci were spread through-

out the ward, being recovered from all but one of the air samples and all but one of the settle plates. The total number of other bacteria-carrying particles ranged from 6 to 27 per cu.ft. air, with a mean of 13·5 and the number settling per plate/hr. varied from 60 to 200, with a mean count per sq.ft./min. of 27·3. On this occasion, then, 12% of the organisms recovered by the slit sampler were *Staph. aureus*, and of those on the settle plates 6% were *Staph. aureus*. By contrast, in the women's ward, although the total bacteria-carrying particles were not significantly lower than in the men's ward, there was an almost complete absence of *Staph. aureus*.

Table 3. *Counts obtained with air samples and settle plates in two wards on two days of the same week*

Bed No.	Air samples		Settle plates	
	Total count/ 5 cu.ft.	Total <i>Staph. aureus</i> count/5 cu.ft.	Total count/ 60 min.	Total <i>Staph.</i> <i>aureus</i> count/ 60 min.
Men's Ward				
1	42	5	91	0
2	35	12	200	5
3	30	9	160	16
4	84	9	91	7
6	72	9	124	8
7	39	0	105	8
9	88	6	76	4
11	136	18	78	12
12	81	10	60	2
	Mean/cu.ft. 13·5	1·7	Mean/ sq.ft./min. 27·3	1·7
Women's ward				
1	57	1	73	0
2	39	0	113	1
3	60	0	160	0
4	78	1	90	0
6	62	1	64	0
7	58	0	61	0
9	73	0	68	0
11	83	0	95	0
12	72	0	150	0
	Mean/cu.ft. 12·9	0·06	Mean/ sq.ft./min. 24·5	0·02

The weekly counts in each ward were used to calculate the mean counts per month. Fig. 1 shows the monthly averages for the men's ward during the 12 months and for the women's ward for 7 months. In the men's ward there was considerable fluctuation in the air sample counts but less fluctuation in the settle plate counts for total bacteria-carrying particles.

There was no relationship between the total count and the *Staph. aureus* count. In November there was a high level of staphylococci but their numbers fell in December, possibly coincident with the use of 'naseptin' prophylaxis, and then remained at a low level until May. In May there was a sudden increase in the

counts in the male ward which remained at a high level for the remaining months, with the settle plates showing a peak in August. This increase in May coincided with the advent of cold weather, an increased number of blankets on each bed and difficulties with laundry supplies, but there were no cases of sepsis in the ward at that time. In June, a patient with two infected wounds and a sputum laden with

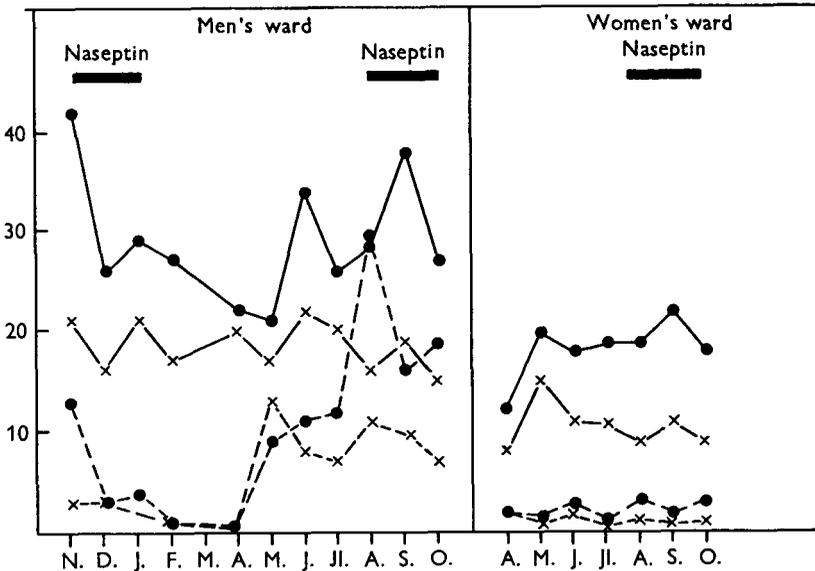


Fig. 1. Bacterial counts obtained in the men's ward, November 1960 to October 1961 and in the women's ward, April to October 1961. Monthly averages of weekly counts. ● - - - ●, *Staph. aureus* settling/10 sq.ft./min.; × - - - ×, *Staph. aureus*/10 cu.ft.; ● — ●, total bacterial count settling/sq.ft./min.; × — ×, total bacterial count/cu.ft.

Table 4. *Distribution of colonies of Staphylococcus aureus on settle plates exposed in the men's and women's wards*

Ward	Period	No. of plates	No. of plates with <i>Staph. aureus</i>	%	Total no. of <i>Staph. aureus</i>	Mean <i>Staph. aureus</i> /positive plate
Men's	Nov.—Apr.	155	86	56	238	2.3
	May—Oct.	231	176	76	1480	8.4
Women's	Apr.—Oct.	260	106	41	173	1.6

Type 47 staphylococci was in the ward for 3 weeks and was considered to be the probable source of showers of these organisms during that month. In August, further showers were found, particularly on the settle plates and appeared to be associated with an ambulant patient who had two infected skin graft sites, a conjunctivitis and heavy nasal colonization from all of which Type 47 staphylococci were isolated.

The rise in the contamination of the ward air with Type 47 strains was followed

in June by an increased incidence of wound infection with this particular phage type. From November to April there were 6 infections with this strain and one in May while in the next 5 months 14 infections occurred.

In contrast to the men's ward, the total counts in the women's ward were less liable to fluctuation and were in general at a lower level. *Staph. aureus* was not found in large numbers on any occasion, the maximum number being 19 in 45 cu.ft. on one day, and these were of a phage type not frequently isolated. Staphylococcal sepsis due to the Type 47 strain occurred in only 6 patients during the 12 months' investigation, the cases being distributed throughout the year.

The results of the settle plate counts of *Staph. aureus* can also be expressed as the proportion of exposed plates growing *Staph. aureus* and the mean count per positive plate. Table 4 shows that 41% of plates exposed in the women's ward were positive, with a mean count of 1.6 per plate. In the men's ward, during the period when the counts were relatively low, 56% of plates were positive, with a mean count of 2.3 per plate, while during the period of higher counts 76% of the plates were positive, with a mean count per plate of 8.4.

#### *Size of the bacteria-carrying particles in the ward air*

The counts obtained with the slit sampler and the settle plates were used to calculate the mean size of the particles on which the bacteria were carried. The method used, which gives a rough approximation of the size, was that of Bourdillon, Lidwell & Lovelock (1948), use being made of their nomograms.

The Petri ratio (i.e. the number of particles in 1 cu.ft. of air divided by the number of particles settling on to a Petri dish  $3\frac{1}{4}$  in. in diameter in 1 min.) was calculated for 168 pairs of plates. The counts for 15 sampling days in the men's ward and 6 sampling days in the women's ward were used for calculations of the particle size of the total count. For *Staph. aureus* a smaller number of pairs of counts were suitable for calculation since no samples in the women's ward contained a sufficient number of these colonies and on many days in the men's ward the numbers were too low to allow accurate calculations.

The results for bacteria-carrying particles in the men's ward (Table 5) showed a range from 13 to  $25\mu$ , with a mean of  $18\mu$ . The variation was due chiefly to variability in the air samples rather than in the settle plates.

The women's ward showed a narrower range of variation from 17 to  $21\mu$ .

There was no essential difference in the mean size of the particles carrying *Staph. aureus* which ranged from 14 to  $32\mu$  with a mean of  $20\mu$  (Table 6).

Considering that these calculations give only a rough approximation of the particle diameters and are influenced by a number of variables such as the strength of convection currents, resuspension of sedimenting particles and the opening and closing of doors and windows, the variability found was small. The calculations indicate that the bacteria found in the ward air during periods of maximum activity are carried on particles of small size.

Table 5. *Mean particle size of bacteria-carrying particles in ward air calculated from the Petri ratio with nomograms of Bourdillon, Lidwell & Lovelock, (1948)*

Ward	Date	Settle plates			Air samples			Petri ratio	Mean particle size ( $\mu$ )
		No.	Total count	Mean count/plate/min.	No. of cu.ft.	Total count	Mean count/cu.ft.		
Men's	7. xii. 60	8	899	1.8	40	506	12.6	7	21
	14. xii. 60	9	696	1.3	45	262	5.8	4	25
	4. i. 61	10	460	0.7	50	317	6.3	9	18
	7. ii. 61	6	382	1.6	30	384	12.8	8	19
	13. vi. 61	8	1138	2.37	40	1518	37.9	16	13
	21. vi. 61	9	1138	2.1	45	844	18.7	9	18
	27. vi. 61	8	1013	2.1	40	644	16.1	8	19
	11. vii. 61	8	859	1.7	40	585	14.6	8	18
	18. vii. 61	7	542	1.3	35	459	13.1	10	17
	25. vii. 61	9	1289	2.4	45	1439	32	13	15
	22. viii. 61	8	585	1.2	40	472	11.8	10	17
	31. viii. 61	7	742	1.8	35	651	19	11	17
	7. ix. 61	9	923	1.7	45	529	11.7	7	21
	14. ix. 61	8	954	2.0	40	691	17.2	9	18
	21. ix. 61	6	804	2.2	30	761	25.3	11.5	16
	Women's	29. vi. 61	9	1086	2.0	45	787	17.5	9
6. vii. 61		9	559	1.0	45	448	9.9	10	17
20. vii. 61		6	348	0.9	30	200	6.6	7	21
3. viii. 61		7	526	1.2	35	302	8.6	7	21
5. ix. 61		9	874	1.6	45	582	12.9	8	19
12. xi. 61		8	574	1.2	40	319	7.9	7	21

Table 6. *Mean particle size of particles carrying Staphylococcus aureus in men's ward*

Date	Settle plates			Air samples			Petri ratio	Mean particle size ( $\mu$ )
	No.	Total count	Mean count/plate/min.	No. of cu. ft.	Total count	Mean count/cu.ft.		
14. xii. 60	9	11	0.02	45	10	0.2	10	17
13. vi. 61	8	85	0.18	40	64	1.6	9	18
21. vi. 61	9	32	0.06	45	20	0.4	6.6	21
27. vi. 61	8	29	0.06	40	32	0.8	13	15
11. vii. 61	8	39	0.08	40	42	1.0	12.5	16
18. vii. 61	7	40	0.09	35	15	0.4	4.4	25
25. vii. 61	9	56	0.10	45	14	0.3	3	32
22. viii. 61	8	55	0.11	40	34	0.8	7	21
31. viii. 61	7	265	0.63	35	145	4.2	6	22
7. ix. 61	9	62	0.11	45	78	1.7	15	14
14. ix. 61	8	49	0.10	40	42	1.0	10	17
21. ix. 61	6	32	0.09	30	25	0.8	9	18
Total	96	755	—	480	521	—	—	—
Mean	—	—	—	—	—	—	—	20

*The blankets*

The blankets used in the wards were of wool and of cotton. There was a greater number of cotton blankets which were introduced early in 1960 and were of the 'Osman' cellular type. Some woollen blankets were left in use so that they could be compared with the cotton ones with regard to bacterial load when used under the same conditions. Woollen blankets are not laundered after each patient. It was planned to send cotton blankets to the laundry after each patient's discharge but, in actual practice, this was not always done.

*Bacterial counts on sweep plates*

In Table 7 are given the mean counts obtained from the examination of 1005 blankets. The mean total counts per sweep plate were significantly higher in the men's ward than in the women's. The mean numbers of bacteria-carrying particles released by sweeping cotton blankets were higher than those obtained from the woollen ones but the differences were not of a high order of magnitude.

Table 7. *Total bacterial counts obtained by sweep plates and proportion of blankets yielding Staphylococcus aureus*

Ward	Type of blanket	No. of blankets	Mean total count/sweep	Blankets with <i>Staph. aureus</i>		Mean <i>Staph. aureus</i> /blanket sweep
				No.	%	
Men's	Wool	107	144.8	71	66	6.8
	Cotton	409	158.9	263	64	6.9
Women's	Wool	78	96.9	47	60	4.5
	Cotton	411	112.8	227	55	3.0

*Staph. aureus* was isolated from 65% of all blankets in the men's ward and from 56% in the women's ward. The mean *Staph. aureus* count/sweep was calculated from the total number of blankets examined since it was considered that this figure gave an index of the overall degree of blanket contamination.

Calculation of the mean *Staph. aureus* count using only those blankets from which the staphylococci were isolated, increased the mean count to approximately 10/sweep in the men's ward and 6/sweep in the women's ward.

There was, however, a wide variation in the yield of *Staph. aureus*/sweep, exceptionally high counts/sweep being obtained on a number of occasions in the men's ward; 35 blankets yielded counts ranging from 15 to approximately 800 per sweep. Eighteen of the patients occupying the beds on the day these high counts were obtained were nasal carriers of the strain present on the blankets but only three had clinical staphylococcal infection.

*Relationship between Staphylococcus aureus present in blankets and patients' noses*

Data were available for 960 blankets, the occupants of which had had their noses swabbed 1 or 2 days previously.

No significant differences were found between the results of the male and female patients and the results have been combined.

Table 8 shows the relationship between the nasal carrier state of the patients and the presence or absence of *Staph. aureus* on their blankets. The results for cotton and woollen blankets are given separately but there was no difference between them.

There were 352 positive nasal swabs and from 168 (48%) of the blankets occupied by these patients, the same staphylococcus was isolated. Ninety-one blankets yielded a strain different from that found in the nose and there were 93 blankets from which the staphylococci present in the nose of the occupant were not isolated.

Table 8. *Relationship of the nasal carrier state in patients to the staphylococci on their blankets*

Category	Woollen blankets. No.	Cotton blankets. No.	Total	
			No.	%
Nose positive. Blanket positive				
Same strain	34	134	168	17.5
Different strain	13	78	91	9.5
Nose positive. Blanket negative	16	77	93	9.6
Nose negative. Blanket positive	56	248	304	31.7
Nose negative. Blanket negative	52	252	304	31.7
Total	171	789	960	—

It seemed, therefore, that less than half the nasal carriers were contaminating their blankets. Whether this was a correct estimate of the proportion of 'shedders' is however open to question, since staphylococci were recovered from 304 blankets whose occupants were not carriers. Reference to Table 1 shows that there was a greater proportion of Type 47 strains isolated from the air samples, settle plates and blankets than was present in the noses of the carriers. It is possible therefore that the source of some of the Type 47 strains isolated from the bedding of patients carrying this strain was not necessarily the nose of the occupant. That 32% of the blankets were positive in the absence of nasal carriage of the patient in that bed was a reflection of the widespread seeding of the wards with *Staph. aureus*.

#### *Acquisition of strains from blankets*

There were 33 patients who acquired staphylococci in their noses of the same type as had been found previously on their blankets; 26 of these people acquired the widespread Type 47 strain and there is therefore no unequivocal evidence that the bedding was the direct source of their nasal colonization. Five patients were colonized by strains of various distinctive phage types that had been isolated from their bedding the previous week and two patients acquired non-typable strains of antibiotic sensitivities identical with non-typable strains isolated previously from their bedding. In these cases there was therefore good evidence that the source of their strains had been their bedding, particularly as the strains in question had not been recovered from the air samples.

#### *Source of the bedding strains*

Apart from the patients in which there was correspondence between the staphylococci in their noses and on their bedding, the question arises as to the source of the staphylococci found on blankets from beds whose occupants were not nasal

carriers. Some may have been contaminated by carriage on parts of the body not examined but a more probable source was nasal carriage by a previous occupant of the bed who had contaminated the blankets. Instructions had been issued that all cotton blankets were to be sent to the laundry after the patient's discharge but sometimes this was not done; the woollen blankets were washed infrequently. On one occasion the next occupant of a bed whose blankets had not been changed acquired a distinctive strain that had been carried by the previous occupant and with which the bedding was heavily contaminated.

On several occasions widespread dissemination of Type 47 staphylococci took place, showers of organisms apparently being deposited on all the beds in the men's ward. As already mentioned, two of these occasions were associated with the presence in the ward of patients heavily infected with this strain. On another occasion when all the beds were heavily contaminated the walls and light fittings in the ward had been brushed down on the previous day, prior to painting.

Table 9. *Relationships of Staphylococcus aureus present in infected surgical wounds, noses of the patients and their blankets*

Category	No. of infections	No. of infections with Type 47
1. <i>Staph. aureus</i> in blanket prior to nose and wound	9	8
2. <i>Staph. aureus</i> in blanket prior to wound, nose negative	7	5
3. <i>Staph. aureus</i> in blanket and nose prior to wound	7	7
4. <i>Staph. aureus</i> in wound prior to blanket and nose	1	1
5. <i>Staph. aureus</i> in wound prior to blanket, nose negative	5	3
6. <i>Staph. aureus</i> in wound, blanket negative	2	1
7. No correspondence between wound, blanket and nose	5	—
Total	36	25

*Significance of the blanket strains in the causation of sepsis*

Of the 48 patients with staphylococcal sepsis during the investigation, full data on the staphylococci in their noses, blankets and wounds and the times of isolation of the positive cultures were available for 36.

These data have been divided into seven categories and are given in Table 9. This shows that in 23 patients, the causative staphylococci were isolated from their blankets prior to signs of sepsis in their wounds. Seven of these patients never had positive nasal swabs but in the remainder the nose was positive at some time during the course of the infection. However the interpretation of these findings is difficult since 20 of these patients were infected with the Type 47 strain and it is

therefore possible that the infection was acquired in some other way than from the bedding at the time that their wounds were dressed. It should be noted, however, that each patient had an individual dressing tray and that patients with sepsis, although they remained in the ward, were placed last on the dressing list. It was thought unlikely, therefore, that infection was being transferred directly from patient to patient during the dressing procedure. In three patients the infecting organisms were of unusual phage types and there is reasonably good evidence that their blankets could have been the source of their infection.

#### DISCUSSION

The wards examined in this study differed from those described by Shooter *et al.* (1958) and by Burke & Corrigan (1961). Both these groups of workers found no one predominant phage type of *Staph. aureus* in the ward air or patients and little evidence of spread of strains from one patient to another. In our wards, one antibiotic-resistant strain has been the predominant strain for at least two and a half years and no other strain introduced by a clinical infection or a nasal carrier has spread to more than one other patient. This predominance of one strain renders difficult any assessment of the relative importance as a cause of sepsis of the staphylococci isolated from the air and from the patients' beds. The findings are however suggestive that in some at least of the patients the route of infection may have been via the bedclothes to the wound and in three patients with unusual phage types the evidence for this is a little better.

The investigations showed clearly that there was a correlation between low staphylococcal counts in the air and bedclothes and low sepsis rates in the patients in the men's ward during the first part of the year, and in the women's ward. The contamination in the men's ward increased suddenly at the end of May in the absence of clinically infected patients and was thought to be due to the contamination of bedclothes by nasal carriers. It was followed in June by clinical infections including a case of staphylococcal pneumonia. Thereafter high levels of sepsis and of staphylococcal contamination of the air and bedding persisted for the remainder of the period of observation.

Our findings support the idea that a cycle can be set up in which an 'epidemic' strain passes from the nose of one patient to his bedding and surroundings and thence to the nose, an open wound or the lungs of another patient.

Data to be published elsewhere indicate that certain strains of *Staph. aureus*, distinguished by their frequent implication in hospital epidemics, can persist for long periods of time on various types of textiles and so provide a source from which a new cycle of infection may be started.

Our results indicate that only a proportion of nasal carriers are shedders of their organisms. Hare & Cooke (1961) made similar observations but found that the only patients in their series who heavily contaminated their environment were those with skin lesions, pneumonia or enterocolitis. For this reason they felt that blankets played little part in the spread of staphylococcal infection. Some nasal carriers that we examined did contaminate their bedding heavily and we consider that the role of blankets in spreading infection cannot be dismissed.

The staphylococci deposited on bedding by the patient will be protected by mucus from the nasal cavities, by mucus from the pharynx or bronchi or by protein exudate and disintegrating leucocytes in pus. On drying, covered with this protective coating, the organisms may be dispersed from textiles in small particles not necessarily associated with textile fibres.

Rubbo, Pressley, Stratford & Dixon (1960) in a study of the possible vehicles of transmission of airborne bacteria in hospital wards found no significant difference between the numbers of 'staphylococci' collected at various heights from floor level to 9 ft., while the amounts of fibre collected fell markedly above the height of 3 ft. From this they concluded that airborne infection in wards is caused by the dispersion of pathogens travelling as free organisms or in association with microscopic 'fibre nuclei'. Since their method of collection and counting did not distinguish *Staph. aureus* from all 'staphylococci' growing on 8% sodium chloride agar it is not certain that their conclusions apply to *Staph. aureus* which might have a different height distribution if it were carried on larger particles than the other organisms.

Sampling of the ward air by settle plates and the slit sampler concurrently provided data from which the approximate mean size of the bacteria-carrying particles was calculated. The data, based on the collection of samples at a height of 3 ft. 6 in., indicated that the majority of both *Staph. aureus* and saprophytic bacteria were carried on particles of a mean size of 18–20  $\mu$ . Lidwell, Noble & Dolphin (1959) using a size-grading sampler obtained more accurate estimates of particle diameters which indicated that 75% of *Staph. aureus* in ward air were carried on particles up to 18  $\mu$  in size.

These estimates of size do not preclude the carriage of staphylococci on large fibres but suggest that the majority of the cocci are on very small particles of dust, debris or minute textile fibres sheared from the bedding.

The counts of total bacteria bore no relationship to the *Staph. aureus* counts which was to be anticipated if the initial contamination of the air and bedding is an event depending on the staphylococci status of the ward inhabitants.

From the point of view of the contribution of bacteria to the ward atmosphere, the present observations do not support the superiority of wool over cotton or *vice versa*. Both kinds of textile were quickly contaminated with *Staph. aureus* and yielded similar numbers of total bacteria when examined by the sweep plate method. The problem of providing clean blankets appears therefore to hinge on the frequency of washing rather than the type of textile used.

#### SUMMARY

The environment of two contiguous surgical wards was examined over a period of twelve months by means of a slit sampler, settle plates and blanket sweep plates. At the same time, nasal swabs were taken each week from the patients and all cases of sepsis examined bacteriologically.

Phage typing of more than 3600 isolations of *Staph. aureus* showed that there was one predominant strain in the air, bedding, patients' noses and infected wounds.

There was no relationship between the total number of bacteria in the ward air and the numbers of *Staph. aureus*.

The recovery of large numbers of *Staph. aureus* from the air at certain periods was associated with a high contamination rate in the blankets and with an increased incidence of staphylococcal sepsis.

Not all nasal carriers of *Staph. aureus* contaminated their bedding. There was evidence that some patients became nasal carriers of strains of staphylococci previously isolated from their bedding.

Some evidence was obtained that blankets may play a role in the transmission of staphylococci from patient to patient.

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#### REFERENCES

- BARBER, M. & KUPER, S. W. A. (1951). Identification of *Staphylococcus pyogenes* by the phosphatase reaction. *J. Path. Bact.* **63**, 65.
- BLAIR, J. E. & WILLIAMS, R. E. O. (1961). Phage typing of staphylococci. *Bull. World Hlth Org.* **24**, 771.
- BLOWERS, R. & WALLACE, K. R. (1955). The sterilization of blankets with cetyl trimethylamine bromide. *Lancet*, i, 1250.
- BLOWERS, R., MASON, G. A., WALLACE, K. R. & WALTON, M. (1955). Control of wound infection in a thoracic surgery unit. *Lancet*, ii, 786.
- BOURDILLON, T. B., LIDWELL, O. M. & LOVELOCK, J. E. (1948). Studies in air hygiene. *Spec. Rep. Ser. med. Res. Coun., Lond.*, no. **262**, p. 347.
- BURKE, J. F. & CORRIGAN, E. A. (1961). Staphylococcal epidemiology on a surgical ward. *New Engl. J. Med.* **264**, 321.
- CADNESS-GRAVES, B., WILLIAMS, R., HARPER, G. J. & MILES, A. A. (1943). Slide test for coagulase-positive staphylococci. *Lancet*, i, 736.
- COLBECK, J. C. (1956). Studies in hospital infections. *Canad. Serv. Med. J.* **12**, 563.
- GILLESPIE, W. A., ALDER, V. G., AYLIFFE, G. A. J., POWELL, D. E. B. & WYPKEMA, W. (1961). Control of staphylococcal cross-infection in surgical wards. *Lancet*, i, 1299.
- HARE, R. & COOKE, E. M. (1961). Self contamination of patients with staphylococcal infection. *Brit. med. J.* **2**, 333.
- HOWE, C. W., SILVA, T. F., MARSTON, A. T. & WOO, D. D. B. (1961). Staphylococcal contamination of mattresses and blankets on a surgical ward under non-epidemic conditions. *New. Engl. J. Med.* **264**, 625.
- LIDWELL, O. M., NOBLE, W. C. & DOLPHIN, G. W. (1959). The use of radiation to estimate the numbers of micro-organisms in air-borne particles. *J. Hyg., Camb.*, **57**, 299.
- MOORE, B. (1960). A new screen test and selective medium for the rapid detection of epidemic strains of *Staph. aureus*. *Lancet*, ii, 453.
- PRESSLEY, T. A. (1958). The fibre composition of hospital dust. *Lancet*, ii, 712.
- ROUNTREE, P. M. (1947). Cross-infection of wounds in a surgical ward during a trial of the use of oiled blankets. *Med. J. Aust.* i, 427.
- ROUNTREE, P. M., HARRINGTON, M., LOEWENTHAL, J. & GYE, R. (1960). Staphylococcal wound infection in a surgical unit. *Lancet*, ii, 1.
- RUBBO, S. D., PRESSLEY, T. A., STRATFORD, B. C. & DIXSON, S. (1960). Vehicles of transmission of airborne bacteria in hospital wards. *Lancet*, ii, 397.
- SHOOTER, R. A., SMITH, M. A., GRIFFITHS, J. D., BROWN, M. A. E., WILLIAMS, R. E. O., RIPPON, J. E. & JEVONS, M. P. (1958). Spread of staphylococci in a surgical ward. *Brit. med. J.* i, 607.