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An outbreak of post-operative sepsis due to a staphyloccoccal disperser

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SUMMARY

A staphylococcal disperser employed as a theatre technician appeared to have been the source of 11 cases of wound sepsis over a period of about 3 years. He was primarily a nasal carrier and after attempts to eradicate *Staphylococcus aureus* from his nose failed, his skin dispersal was controlled by daily washing with 4% chlorhexidine detergent ('Hibiscrub') and he was allowed to resume his theatre duties under careful bacteriological surveillance. Over the following 2 years 173 dispersal tests showed a mean dispersal of 1·7 c.f.u. per 2800 l air compared with a mean of 152 c.f.u. per 2800 l air in the month immediately preceding treatment and 55 c.f.u. per 2800 l in the period after cessation of treatment. One case of wound sepsis was attributed to the technician during the 2 years in which he received skin disinfection treatment.

INTRODUCTION

Outbreaks of post-operative staphylococcal wound sepsis caused by operating personnel have been well documented (Williams *et al.* 1966) and the organisms responsible have usually been traced to staff with infected skin lesions, or to symptomless carriers who were profuse aerial dispersers of staphylococci. It is generally accepted that staff with infected skin lesions should be excluded from theatre duty and, during outbreaks, it would seem wise to exclude symptomless carriers of the staphylococcal phage types responsible.

A difficult problem may arise if a staphylococcal disperser responsible for an outbreak is an essential member of the surgical team and previous accounts of outbreaks have provided little useful guidance for dealing with this situation. This report describes an outbreak caused by such a disperser, and shows how aerial dispersal was controlled and monitored permitting a resumption of duty by the disperser.

MATERIALS AND METHODS

Aerial dispersal of staphylococci

Aerial dispersal of *Staphylococcus aureus*-carrying particles was investigated in a test-chamber using a Casella slit-sampler at a flow rate of 700 l/min whilst the subject exercised for 2 min in a standard manner (Mitchell & Gamble, 1974). The

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subject was tested in standard theatre clothing immediately after an operating session of about 4 h. Milk salt agar (Oxoid blood agar base No. 2, containing 7·5% salt and 33% milk) was used as a selective medium for *Staph. aureus*. Plates were counted after 48 h incubation, and results expressed as colony forming units (c.f.u.) per 2800 l (100 ft^3) of air.

Sampling of theatre clothes

Two sweep plates were used to sample clothing worn in theatre, one for the upper part of the body, and one for the lower part; each plate was swept up and down five times over the fabric.

Skin swabs

A swab moistened in quarter strength Ringer's solution was rubbed over a 5 cm area of skin and plated on horse blood agar and milk salt agar. A chlorhexidine neutralizer (3% Tween 80) was not incorporated, as preliminary tests had shown that its addition to nutrient agar base had no effect on bacterial counts. Skin swabs were taken from the wrists, axillae, scalp, back chest and perineum. Hair was examined by applying a salt agar plate directly to the head.

Nasal swabs

In addition to conventional nasal swabs, a quantitative estimate of nasal staphylococci was made. Both anterior nares were sampled in a standard manner using an alginate swab, which was inserted in a Stomacher bag containing 10 ml Calgon Ringer's Solution (Oxoid), and dissolved by agitation in the Stomacher 80 (A. J. Steward, London SE1). Twenty 0·02 ml drops of this solution, and of 1/10 and 1/100 dilutions, were plated on blood agar and salt milk agar. Total and *Staph. aureus* counts were made after 48 h incubation at 37 °C, and results expressed as c.f.u.'s per swab.

Identification of Staph. aureus

Colonies of presumptive *Staph. aureus* were tested for coagulase production by the slide technique and phage typed using the international set of phages (Blair & Williams, 1961).

RESULTS

Over a period of several weeks at the end of 1971, seven patients developed staphylococcal wound sepsis due to phage type 77/84/85/88+ which was penicillin resistant, but sensitive to tetracycline, erythromycin and cloxacillin, and in seven other patients wounds were colonized with this organism without evidence of sepsis. The patients were in five different wards, and sepsis was apparent at first wound dressing, suggesting that infection occurred in the theatre. Nasal swabs from the five different surgical teams involved revealed only one source of the epidemic strain, a theatre technician who had been present at all of the operations, and who was found to have had a boil on his forearm for about 2 weeks. A swab from the boil yielded the epidemic staphylococcus.

The technician was relieved from duty, and 2% hexachlorophane nasal cream

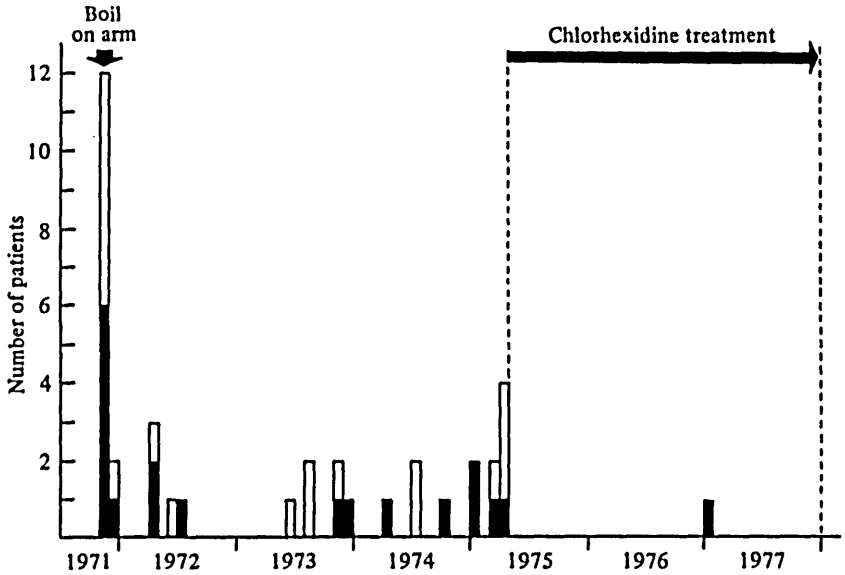

Fig. 1. The number of patients with infected (solid columns) or colonized (open columns) wounds during the period 1971–77.

and daily hexachlorophane baths were prescribed. Within 2 weeks the boil had healed, nasal swabs were clear, and he returned to duty. During the next 3 years no further cases were identified by routine bacteriological surveillance of all surgical wounds, and phage typing of all staphylococci isolated. However, during a study of aerial contamination in theatres in March and April 1975, several strains of types 77/88, 85+ and 75/85/88+ were isolated from air samples and from two patients with wound infection at that time. The technician, who was still employed in the theatre, had no septic lesions but nasal swabs and sweep plates of his theatre clothing yielded *Staph. aureus*. The staphylococci from the technician, the infected patients, and the air samples, were indistinguishable when phage typed together (77/84/85/88 if penicillin sensitive, and 77/85/88 if penicillin resistant).

A review of phage types of strains isolated from wounds over the previous $3\frac{1}{2}$ years revealed 18 strains similar to 77/84/85/88, differing only in minor reactions. Eleven of these strains were still available, and when they were typed as a batch with the original 1971 strain and the more recent strains all were found to be indistinguishable. It was considered that the other seven strains which were not available were probably similar, and that over the $3\frac{1}{2}$ years since the original outbreak, 11 cases of wound sepsis, and 12 of wound colonization, had probably been due to the technician's strain of *Staph. aureus* (Fig. 1).

The technician was again relieved of theatre duties and given a nasal cream containing 0·1% chlorhexidine and 0·5% neomycin ('Naseptin'). This was discontinued when hypersensitivity and acute sinusitis developed for which he was treated with erythromycin for 2 weeks. Nasal swabs during treatment showed a gradual diminution in the number of *Staph. aureus* colonies, but 4 days after stopping the erythromycin, a nasal swab again yielded a heavy pure growth, skin swabs were positive, and he was dispersing 38 c.f.u. of *Staph. aureus* per 2800 l air. A 3-week course of cloxacillin was then given, and whilst on treatment nasal swabs

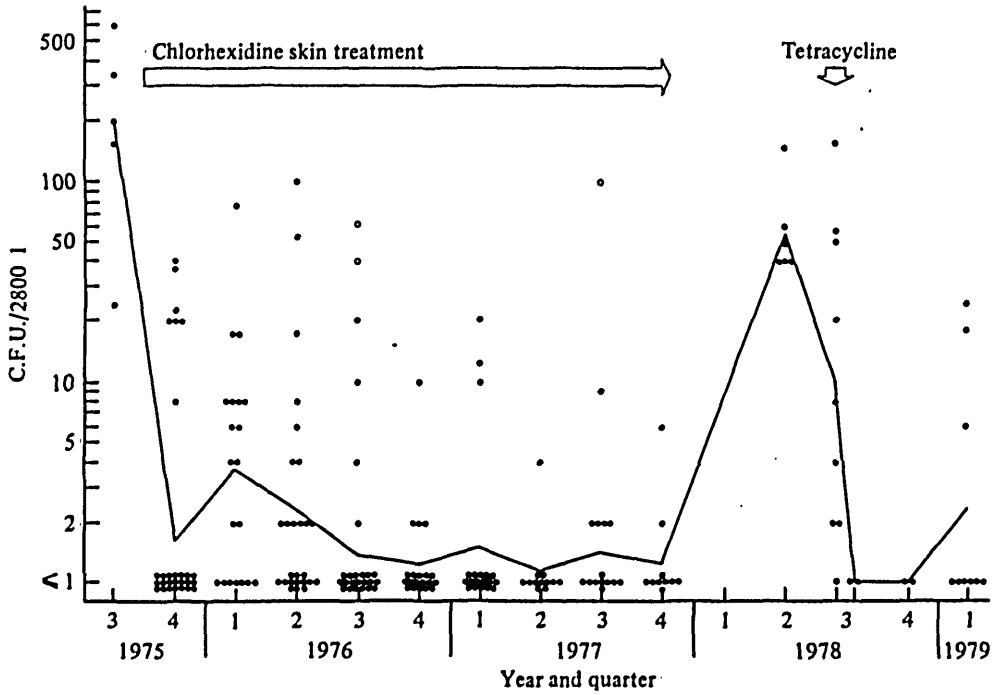

Fig. 2. Effect of treatment on staphylococcal dispersal counts.

yielded only a scanty growth of *Staph. aureus*, and skin swab and dispersal tests were negative. Three weeks after treatment he was again dispersing large numbers of *Staph. aureus*; in six tests over 4 weeks dispersal ranged from 24 to 600 c.f.u. (mean 152 c.f.u.) per 2800 l (Fig. 2).

Since treatment of nasal carriage had been unsuccessful, an attempt was made to reduce dispersal by skin disinfection. Daily providone iodine baths ('Steribath') were ineffective; after a week's treatment the dispersal count was 340 c.f.u. per 2800 l. Daily bathing and hairwashing with 4% chlorhexidine detergent ('Hibiscrub') was next prescribed, and when this was regularly carried out, dispersal was greatly reduced and skin swabs became negative, but nasal carriage continued. During the first 3 months of this treatment, dispersal counts in 28 tests ranged from < 1 to 40 c.f.u. (mean 2·2) per 2800 l, and in only six tests did it exceed 10 c.f.u. (Fig. 2). In view of the key role of this technician in the surgical team and the difficulty of replacing him, he was allowed to resume duty under bacteriological supervision.

For the next 2 years the technician continued his daily chlorhexidine treatment and remained on duty; he was seen at least weekly, when swabs were taken and *Staph. aureus* dispersal estimated. During this time, 173 dispersal tests showed an average dispersal rate for the whole period of only 1·7 c.f.u. per 2800 l, and in 118 (68%) no dispersal was detected at all. On three occasions, when chlorhexidine bathing had been omitted on the previous day, high dispersal rates of 40, 60 and 100 c.f.u./2800 l were found (Fig. 2) and it seemed likely that the high rates detected on other occasions may have coincided with lapses in the bathing regi-

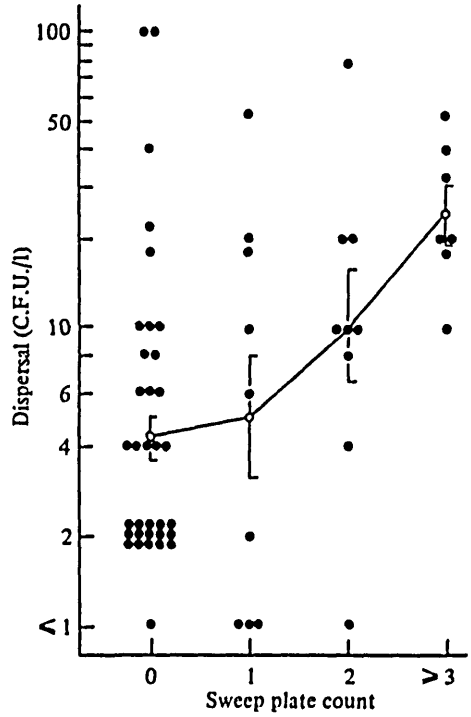

Fig. 3. The relation between staphylococcal skin dispersal counts and sweep plate counts.

men. During these 2 years all surgical wounds were examined bacteriologically and all strains of *Staph. aureus* isolated were phage typed together with the technician's strains; only one infection could be atttributed to the technician (Fig. 1).

Colony counts of *Staph. aureus* on sweep plates of theatre clothing were clearly correlated with the results of dispersal counts (Fig. 3), but these studies were only done while the technician was using skin disinfectant when sweep plate counts were always low, ranging from 1 to 6 colonies. A sweep plate count of three or more colonies was found on only seven occasions, but on each of these, the dispersal count was 10 or more c.f.u./2800 l.

The presence of *Staph. aureus* on skin or hair was also more frequent when dispersal counts were high (Table 1) but the correlation was weaker than that between theatre clothing sweep plate counts and dispersal. When dispersal counts exceeded 10 c.f.u. per 2800 l, *Staph. aureus* was isolated from skin or hair in 68% (17/25) of tests, and in 18% (19/108) when dispersal was <1 c.f.u. per 2800 l. Hair was more frequently positive than other sites, but no particular site showed noticeably better correlation with dispersal counts than others.

Quantitative studies of the flora of the anterior nares between March 1976 and September 1977 showed that the total organisms per swab varied little, ranging from $1{\cdot}5 \times 10^6$ to 3×10^6 c.f.u. By contrast, the number of *Staph. aureaus* varied from 25 c.f.u. to $2{\cdot}8 \times 10^6$ c.f.u. per swab, but these variations showed no correlation with dispersal counts. On only 3 of the 100 test days was *Staph. aureus* not isolated.

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Table 1. *Relation between staphylococcal dispersal and skin carriage of* Staph. aureus *whilst the technician was bathing with chlorhexidine*

Staph. aureus		Sites positive for *Staph. aureus* (%)				
C.f.u. per 2800 l air	No. of counts	Hair	Axilla, chest and back	Wrists	Perineum	Any site (%) positive
<1	108	15 (14)	3 (3)	5 (5)	2 (2)	19 (18)
1–9	28	16 (57)	3 (11)	3 (11)	0 (0)	18 (64)
⩾ 10	25	15 (60)	8 (24)	3 (12)	4 (16)	17 (68)
Total	161	46 (29)	14 (9)	11 (7)	6 (4)	64 (40)

The technician stopped the chlorhexidine skin treatment in January 1978 when he ceased working in the theatre to take up another post in the hospital. Dispersal counts again rose to high levels, the majority exceeding 10 c.f.u. per 2800 l (Fig. 2). These raised counts were associated with heavy staphylococcal colonization of the skin of one axilla, and on several occasions with heavy colonization of his hair. During August 1978, skin swabs were negative, and dispersal counts were low on several occasions, but he remained a nasal carrier.

In September 1978 he was given a 5-day course of tetracycline for a septic finger, and it was interesting that, on seven occasions over the next 3 months his *Staph. aureus* dispersal was < 1 c.f.u. per 2800 l, and hair, skin and nasal swabs were negative. By December 1978 *Staph. aureus* had reappeared in his nasal swabs, and in dispersal tests (Fig. 2) and remained until the end of February 1979 when the investigation was terminated.

DISCUSSION

This incident illustrates the difficulty of recognizing an outbreak when infections occur sporadically, and the experience gained in subsequent investigation and treatment of the disperser may be useful to others faced with a similar problem involving an essential member of the surgical team.

After the initial outbreak, it was only in retrospect that it was realized that the technician had been the source of further sporadic cases of wound infection over the next 3 years. During this time tests were not done, but he was presumably dispersing *Staph. aureus*. Tests before and after chlorhexidine treatment demonstrated that without treatment he was a very profuse disperser. Hill, Howell & Blowers (1974) have shown that about 10% of young males are profuse dispersers of *Staph. aureus* (dispersing > 10 c.f.u. per 2800 l) and it is surprising that theatre sepsis attributable to them is not more frequently reported. Our experience however illustrates the difficulty of recognizing such infections when they occur sporadically. Batch variations in the strength of minor phage typing reactions can be misleading and identical strains may appear to differ if they are tested in different batches.

The technician carried the same strain of *Staph. aureus* for the 6 years he was under surveillance. He had several antibiotics, but the only one with more than a

very transient effect was tetracycline which suppressed nasal carriage for 3 months. During this time *Staph. aureus* was not isolated in dispersal tests, from his skin or hair, suggesting that his carriage was primarily nasal. Ayliffe, Babb & Collins (1974) also found that healthy nasal carriers who are not perineal carriers can be *Staph. aureus* dispersers shedding > 10 c.f.u. per 1800 l air, and that such people tend to carry more staphylococci on their skin and hair than non-dispersers.

White (1961) has suggested that heavy nasal carriers are more likely to disperse staphylococci than light carriers. We found that, while the technician was having chlorhexidene treatment, the total aerobic bacterial count on nasal swabs was remarkably constant, but the *Staph. aureus* count fluctuated widely, and showed no correlation with the degree of dispersal. However, dispersal was much increased by omission of treatment for a day, and irregularities in the disinfection regimen may have been responsible for the lack of correlation between degrees of dispersal and nasal carriage.

The choice of chlorhexidine skin treatment was based on the work of Lowbury & Lilly (1973), who showed that in addition to its immediate effect on the skin flora, it had a persistent residual effect against bacteria subsequently deposited on the skin. The technician was instructed to wash his whole body surface with the disinfectant and the only problem encountered was some drying of the hair towards the end of the second year of treatment, when the frequency of hair washing was reduced.

The efficacy of chlorhexidine treatment was evident from the reduction in aerial dispersal of *Staph. aureus* by the technician, and from the reduction in the number of infected patients during its use. We have had similar success with this treatment on a house surgeon who was a profuse disperser and heavy perineal carrier of *Staph. aureus*. Monitoring the efficacy of treatment was most satisfactorily achieved by regular dispersal tests using a test chamber. The correlation between dispersal counts and the presence of *Staph. aureus* on sweep plates of theatre clothing suggest that the latter may provide a simpler monitoring procedure but without data on sweep plate counts before the institution of skin disinfection the feasibility of this suggestion could not be assessed.

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