The effect on sepsis rates of closing and cleaning hospital wards

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SYNOPSIS Sepsis rates were studied in five hospital wards before and after closure for cleaning. Each ward was closed because of an outbreak of *Staph. aureus* infection caused by a cloxacillin-resistant strain.

The study shows that sepsis rates, especially sepsis caused by hospital strains of *Staph. aureus*, were greatly reduced in the three-month period following re-opening of the ward, provided that patients infected with such organisms were not readmitted to or allowed to remain in the ward.

In some hospitals it is an accepted practice to close and clean hospital wards when outbreaks of staphylococcal and streptococcal sepsis occur, but little has been published about the effect of this practice on the incidence of sepsis. In the past, wards were not often closed in this hospital when sepsis was prevalent, nor has it been a routine practice to close wards at regular intervals for cleaning. In general, wards were closed only for major alterations and/or redecoration. In this paper the effect on sepsis rates of closing and cleaning wards is described.

Methods

Routine surveillance of all inpatients for sepsis in this hospital was carried out by the Infection Control Sister in cooperation with medical and nursing staff on the wards. Swabs and other samples taken from septic wounds and lesions are submitted to the microbiology laboratory for investigation. All isolations of pathogenic bacteria are recorded and a note is made of those associated with sepsis, together with details of the patient, the site of infection, and the ward. Each isolation from any site, associated with sepsis, caused by a single strain of an organism, is counted as a case of sepsis. Repeated isolations of the same strain from the same site are excluded from the calculations of sepsis rates; only cases of definite sepsis are counted and isolations of potential pathogens colonizing a patient without causing sepsis are also excluded.

Strains of *Staphylococcus aureus* resistant to tetra-

cycline are assumed to be 'hospital strains' (Parker and Hewitt, 1970). In this connexion it is worth emphasizing that, in our experience, multiple antibiotic-resistant strains (including cloxacillin-resistant strains) of *Staph. aureus* have been isolated only from hospital inpatients or from outpatients known to have become infected while in hospital.

Strains of staphylococci which coagulated plasma broth within four hours, on incubation in a water bath at 37°C, were regarded as *Staph. aureus*. Antibiotic sensitivities for *Staph. aureus* were tested on DST agar (Oxoid) at 30°C with Oxoid Multodiscs. Routine testing was carried out with penicillin (1.5 µg), tetracycline (10 µg), erythromycin (10 µg), methicillin (10 µg), fusidic acid (10 µg), lincomycin (2 µg), neomycin (10 µg), and gentamicin (10 µg). After overnight incubation zone sizes were compared with those produced by a strain of Oxford *Staph. aureus* (NCTC 6571) tested under similar conditions. Test strains of *Staph. aureus* which grew to within 1 mm of the methicillin disc under these conditions were considered to be resistant (Hewitt and Parker, 1968). Phage typing of selected isolates was carried out at the Cross Infection Reference Laboratory, Colindale, where further tests for methicillin resistance were also done. The minimum inhibitory concentration (MIC) of methicillin for each resistant strain of *Staph. aureus* was in excess of 12.5 µg/ml. Although methicillin sensitivity is tested in the laboratory, cloxacillin sensitivity is reported to the clinicians, as this is the variety of penicillinase-resistant penicillin most used.

Cleaning of Wards

After the wards in question had been closed to

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Received for publication 10 February 1971.

admissions and emptied of patients, all fabrics were sent for laundering and items issued by the Central Sterile Supply Department were returned for re-processing. Disposable stocks of syringes, blood sample containers, unused swabs, etc, were removed; drug containers were returned to the pharmacy and disinfected.

The cleaning of wards included adjacent offices and side-rooms and involved washing floors, walls, and all other surfaces with hot water containing detergent. Bed frames and furniture were also washed. The cleaning was supervised by the Infection Control Sister and the ward sister concerned.

All staff and patients from the wards were screened for nasal carriage of epidemic strains of *Staph. aureus*; carriers were treated with 2% hexachlorophane soap and nasal cream (Noone, Griffiths, and Taylor, 1970). When appropriate, patients were isolated and were not readmitted to cleaned wards until shown to be free from infection or carriage (with the exception of ward 4—see below).

Outbreaks of Sepsis

Each of the five wards studied was closed for cleaning following an outbreak of sepsis, usually caused by a cloxacillin-resistant strain of *Staph. aureus*. Four of the wards were surgical and the fifth was a gastroenterological ward containing both medical and surgical patients.

None of the wards studied had isolation facilities apart from one single-bed cubicle and a two-bed side-room. Apart from ward 2 there were no separate rooms for dressing wounds which, therefore, had to be dressed on the main ward.

WARD 1

This is a gynaecological ward. The outbreak of sepsis began with an old lady suffering from carcinoma of the vulva. Following an operation for removal of the carcinoma her wound became infected with a cloxacillin-resistant *Staph. aureus* (phage type RTD 29/42E/47/54/83/84/81): this organism was resistant also to benzyl penicillin, tetracycline, and erythromycin. This patient remained in a side-room of the ward until she died: at necropsy *Staph. aureus* of the same phage type and with the same resistance pattern to that described was recovered from heart blood and from a spleen swab. Six more patients on the ward became infected with the same organism within the next 10 days, urinary tracts and surgical wounds being chiefly involved. Two nurses were shown to be nasal carriers of this strain, while a further isolation was made from the uniform of another nurse. In view

of continuing sepsis the ward was closed for cleaning and redecoration.

WARD 2

This is a neurosurgical ward. The ward was closed when there was an outbreak of *Staph. aureus* sepsis caused by a strain resistant to cloxacillin and to benzylpenicillin, tetracycline, erythromycin, lincomycin, and chloramphenicol. This strain (phage type RTD 53/75/77/85) was first isolated from a patient who developed a scalp wound infection with a heavy purulent discharge following intracranial surgery for trauma. Further isolations of strains of the same phage type and antibiotic resistance pattern were made from other patients in the ward, including one from the upper respiratory tract of a young man with an open fracture of the skull and cerebrospinal fluid rhinorrhoea, and one from the cerebrospinal fluid of a patient who developed meningoencephalitis following an emergency ventricular tap which was carried out on the ward for raised intracranial pressure. The same organism was isolated at necropsy from this patient's brain.

WARD 3

This is a gastroenterological ward for acutely ill female medical and surgical patients. The outbreak of sepsis which led to closure of this ward was caused by *Staph. aureus* (phage type RTD 42E/47/53/54/75/81) resistant to cloxacillin, benzylpenicillin, and tetracycline, but sensitive to erythromycin. Of three patients who developed bronchopneumonia caused by this strain, two died.

WARD 4

This is an acute general surgical ward for female patients. Before closure there was a steady background of sepsis caused by a variety of organisms infecting wounds and respiratory and urinary tracts. There were four cases of sepsis caused by *Staph. aureus* (phage type RTD 29/6/42E/47/53/54/83A/84/81) resistant to cloxacillin, benzylpenicillin, tetracycline, and erythromycin which resulted in closure of the ward.

WARD 5

This is an acute general surgical ward for male patients. Before closure of the ward there were five cases of sepsis caused by *Staph. aureus* (phage type RTD 29/53/83A/84) resistant to cloxacillin, benzylpenicillin, tetracycline, and erythromycin.

In the six months following closure and cleaning of the ward there were three cases of sepsis caused by *Staph. aureus* resistant to cloxacillin, benzylpenicillin, tetracycline, erythromycin, lincomycin, and chloramphenicol (phage type RTD 53/75/77/85).

This strain was similar to that which caused the outbreak of sepsis on ward 2. Two of the patients on ward 5 who became infected with this organism were operated on in the same theatre as the infected patients from ward 2 within two weeks of one another. Four other patients, each from a different ward (and not included in this study), and each under the care of a different surgeon or physician, also became infected with a similar strain of *Staph. aureus*. The only connexion between these patients was that they underwent operation in the same theatre within a period of a few weeks of one another. Carriers of the relevant strain were not found among theatre, medical, nursing or surgical staff, although some members of staff had left the hospital before investigations were made.

Results

The results are summarized in Tables I and II.

ANTIMICROBIAL THERAPY

Thirty-four patients on the five wards studied had infections with cloxacillin-resistant strains of *Staph. aureus*. Twenty-eight were already receiving antimicrobial therapy at the time they became infected.

Twenty-one were receiving some form of penicillin therapy: nine ampicillin; four ampicillin and cloxacillin; one cloxacillin alone; four benzylpenicillin and streptomycin; three benzylpenicillin and sulphadiazine; five patients were receiving tetracycline; one patient trimethoprim-sulphamethoxazole and one patient streptomycin, isoniazid and para-amino salicylate for a tuberculous breast abscess which became secondarily infected with a cloxacillin-resistant *Staph. aureus*. Each patient receiving cloxacillin, either alone or in combination with ampicillin, was being given 500 mg four times in 24 hours.

The treatment the patients received, once infection with cloxacillin-resistant *Staph. aureus* was confirmed, varied considerably. Three patients were in a terminal stage of carcinomatosis and received no antimicrobial treatment; one patient died before antimicrobial therapy could be changed to an appropriate course. Nineteen patients received no systemic antimicrobial therapy. In general inappropriate systemic antimicrobial agents were withdrawn where they were already being administered and local treatment with 2% hexachlorophane cream or wound powder was undertaken. None of these patients died.

Ward	Period of Time in Relation to Closure	Total Isolations of <i>Staph. aureus</i> Associated with Sepsis	Total Isolations of Gram-negative Organisms Associated with Sepsis	Total Isolations of Other Organisms Associated with Sepsis	Total No. of Organisms Isolated Associated with Sepsis	Total No. of Admissions	Sepsis Rate (%)
1	{ 3 months before	14	7	4	25	184	14
	{ 3 months after	4	6	10	20	170	12
2	{ 3 months before	9	25	3	37	148	25
	{ 3 months after	1	1	0	2	170	1
3	{ 3 months before	7	9	4	20	132	15
	{ 3 months after	1	4	2	7	125	6
4	{ 6 months before	18	44	12	74	268	28
	{ 6 months after	16	19	4	39	291	13
5	{ 6 months before	18	44	8	70	296	24
	{ 6 months after	15	29	5	49	292	17

Table I Time of infection related to closure

Ward	Period of Time in Relation to Closure	Total Number	Associated with Sepsis	Fully Sensitive or Resistant to Penicillin Only	Resistant to Two or More Antibiotics	Cloxacillin Resistant	Total No. of Admissions	<i>Staph. aureus</i> Sepsis Rate (%)
1	{ 3 months before	17	14	5	12	11	184	8
	{ 3 months after	4	4	4	0	0	170	2
2	{ 3 months before	12	9	4	8	6	148	6
	{ 3 months after	1	1	1	0	0	170	0.6
3	{ 3 months before	8	7	4	4	4	132	5
	{ 3 months after	1	1	1	0	0	125	0.8
4	{ 6 months before	22	18	7	15	7	268	7
	{ 6 months after	17	16	13	4	1	291	5
5	{ 6 months before	19	18	13	6	5	296	6
	{ 6 months after	21	15	9	12	6	292	5

Table II Isolations of *Staph. aureus*

Eleven patients received systemic antimicrobial therapy as in each case the infection was considered to be serious.

Number of Patients Treated	Antimicrobial Therapy
2	fusidic acid + cloxacillin (500 mg qds)
2	fusidic acid + lincomycin
1	fusidic acid alone
1	fusidic acid + trimethoprim/sulphamethoxazole
2 (1 died)	fusidic acid + gentamicin
1	chloramphenicol alone
1 (died)	chloramphenicol + novobiocin
1	trimethoprim/sulphamethoxazole alone

Apart from the six patients who died, the rest recovered though several needed extra surgery, such as drainage of pus, wound toiletry, etc, and, apart from one patient who died, all these patients remained in hospital for at least two weeks. Twenty-two patients remained in hospital for at least one month, and seven remained hospitalized for over three months with chronic wound infection, two of them being young patients who had undergone elective surgical operations.

Discussion

WARD 1

Following cleaning, the *Staph. aureus* sepsis rate fell from 8% to 2% (Table II). Moreover, in the three months after cleaning all *Staph. aureus* infections were caused by antibiotic-sensitive strains very different from the strain causing the epidemic before closure and of no consistent phage type, suggesting possible self-infection with the patient's own commensal strain.

The amount of infection caused by Gram-negative and other organisms remained much the same in the two periods, which is not surprising, as most of this sepsis was of the genital and urinary tract, and was probably caused by organisms derived from the patient's own vaginal, perineal, or bowel flora (Grüneberg, 1969).

WARD 2

As may be seen from Table I, there was a very marked reduction in sepsis rates from 25% to 1% following cleaning of the ward. *Staph. aureus* sepsis rate fell from 6% to 0.6% (Table II). Moreover, infections caused by 'hospital strains' of *Staph. aureus*, ie, multiple-resistant strains including those resistant to cloxacillin, were eliminated. Gram-negative infections were also greatly reduced.

WARD 3

The total sepsis rate fell from 15% to 6% following

closure and cleaning of the ward (Table I). The *Staph. aureus* sepsis rate fell from 5 to 0.8% (Table II) with the elimination of infections caused by hospital strains of *Staph. aureus* (including cloxacillin-resistant strains). Gram-negative infections also were more than halved.

WARD 4

Total sepsis rate fell from 28 to 13% after closure and cleaning of the ward (Table I). The *Staph. aureus* sepsis rate was reduced from 7 to 5% (Table II), although the sepsis rate attributable to multiple antibiotic-resistant, ie, 'hospital', strains fell from 4 to 1%. The number of infections caused by cloxacillin-resistant strains fell from seven to one in the two periods. The effects of cleaning would probably have been more striking had not at least two patients with 'resistant' *Staph. aureus* infections (including one with a cloxacillin-resistant strain) been readmitted to the re-opened ward before their infections had been eliminated.

WARD 5

The total sepsis rate fell from 24% in the six months before closure to 17% in the six months after closure (Table I), but the *Staph. aureus* sepsis rate fell only from 6 to 5% (Table II), although in the first three months following closure the rate had fallen from 6 to 4%. The rate of sepsis caused by multiple-antibiotic-resistant strains of *Staph. aureus*, ie, 'hospital strains', increased slightly from 1.7 to 2.4%, while the number of isolations of cloxacillin-resistant *Staph. aureus* increased from five to six. It is significant that the cloxacillin-resistant strains of *Staph. aureus* isolated before cleaning were all of similar phage type and that this was a phage type completely different from that of the strains isolated after re-opening the ward. Although cleaning the ward appeared to eliminate infection by one strain of cloxacillin-resistant *Staph. aureus*, another patient became infected with a cloxacillin-resistant *Staph. aureus* of a different phage type. As previously mentioned, this patient may have been infected in the operating theatre. Subsequently he was nursed on ward 5 for several months, during which time other patients on the ward became infected.

Conclusions

This study clearly shows that, after closing and cleaning wards in the manner described, outbreaks of sepsis caused by *Staph. aureus* stop. Although the wards all catered for the same categories of patients upon re-opening as they had done before closure, for several months after re-opening wards sepsis rates were reduced, especially sepsis caused by 'hospital

strains' of *Staph. aureus*. The results on wards 4 and 5 were not as good as on the other three wards. In one instance two infected patients were readmitted before their infections had been eradicated. There was also failure to isolate effectively newly admitted patients who were, or who became, infected. Effective isolation of infected patients in the wards included in this study was extremely difficult through lack of suitable facilities.

Although closing and cleaning wards appears to reduce sepsis rates and end outbreaks of infection, it is not clear how this comes about. There may be more than one reason. There is little doubt that people carrying pathogens are a much more potent source of cross-infection than is the presence of organisms on walls and floors in wards (Williams, Blower, Shooter, and Garrod, 1966). The actual removal of patients from a ward and the detecting, treating, and isolating of carriers of epidemic organisms might well be more important than the actual washing down of a ward. There is no doubt that cleaning a ward has a psychological effect on staff working in it, for it is likely that nursing techniques will be carried out with more care on a clean ward where there is an increased awareness of risks of cross-infection and of sepsis in general.

Not all people who were infected with cloxacillin-resistant strains of *Staph. aureus* became seriously ill, although there were five deaths at least partly attributable to them. Serious sepsis followed elective surgery in two young patients who were otherwise healthy. It is now generally accepted that cloxacillin-resistant strains of *Staph. aureus* are capable of causing sepsis including serious sepsis (Colley, McNicol, and Bracken, 1965; Jensen and Lassen, 1969). The widespread and increasing use in hospitals of penicillins and cephalosporins is associated with an increase in the isolation of cloxacillin-resistant *Staph. aureus* both from septic lesions and carrier sites (Hale and Selkon, 1970; Parker and Hewitt, 1970).

If cloxacillin and related antibiotics are to remain effective for the treatment of staphylococcal sepsis, resistant strains must be controlled in hospitals. Emptying and thoroughly cleaning wards, coupled with the treatment of carriers and the isolation of infected patients as described, can be expected to get rid of such resistant strains from wards until and unless another infected patient or heavy disperser from a carrier site is admitted.

We would like to thank both Dr C. E. D. Taylor and Professor G. Dick for their invaluable help with the preparation of this paper. We would also like to thank Mr M. A. T. Coles and the laboratory staff for their technical work; the nursing and medical staff on the wards involved for their cooperation; and Dr M. T. Parker and the staff of the Cross Infection Reference Laboratory, Colindale, for their help with phage typing of staphylococci. Finally we must thank Mrs P. Hanrahan and Miss C. Esson for all their typing.

References

- Colley, E. W., McNicol, M. W., and Bracken, P. M. (1965). Methicillin-resistant staphylococci in a general hospital. *Lancet*, **1**, 595-597.
- Grüneberg, R. N. (1969). Relationship of infecting urinary organism to the faecal flora in patients with symptomatic urinary infections. *Lancet*, **2**, 766-768.
- Hale, J. H., and Selkon, J. B. (1970). Multiply resistant staphylococci. *Lancet*, **2**, 528-529.
- Hewitt, J. H., and Parker, M. T. (1968). Sensitivity of penicillinase-forming strains of *Staphylococcus aureus* and of their penicillinase-negative variants to cephaloridine, cephalothin, methicillin and benzylpenicillin. *J. clin. Path.*, **21**, 75-84.
- Jensen, K., and Lassen, H. C. A. (1969). Combined treatment with antibacterial chemotherapeutic agents in staphylococcal infections. *Quart. J. Med.*, **NS**, **38**, 91-106.
- Noone, P., Griffiths, R. J., and Taylor, C. E. D. (1970). Hexachlorophane for treating carriers of *Staphylococcus aureus*. *Lancet*, **1**, 1202-1203.
- Parker, M. T., and Hewitt, J. H. (1970). Methicillin resistance in *Staphylococcus aureus*. *Lancet*, **1**, 800-804.
- Williams, R. E. O., Blowers, R., Garrod, L. P., and Shooter, R. A. (1966). *Hospital Infection*, 2nd ed. pp. 231-260. Lloyd-Luke, London.