result of ^{131}I unless they had been off the drugs for at least six months.

The initial control of hyperthyroidism by ^{131}I alone was slower in the half-dose group. After three years the results were similar, two-thirds being euthyroid in each group. Thereafter they were better in the half-dose group. There was one recurrence of thyrotoxicosis in the conventional-dose group and one in the half-dose group.

The incidence of hypothyroidism rose steadily in the conventional-dose group from 8% after one year to 29% after five years. In the half-dose group it was reduced to 4% and 7% at the corresponding intervals.

The advantages of treating thyrotoxicosis with a small dose of ^{131}I , followed when necessary by antithyroid drug therapy, are discussed. Further trials are required to determine the optimal dose of ^{131}I .

We are grateful to the Endowment Fund of the United Sheffield Hospitals for financial support and to Miss Sandra Taylor for valuable technical assistance. We should like to thank our many colleagues in the department who over the years have assisted in the follow-up and aftercare of the patients, and members of the staff of the Medical Physics Department of the Sheffield Regional Hospital Board who collaborated in arranging the ^{131}I therapy.

REFERENCES

- Al-Hindawi, A. Y., and Wilson, G. M. (1965). *Clin. Sci.*, **28**, 555.
Beling, U., and Einhorn, J. (1961). *Acta radiol. (Stockh.)*, **56**, 275.

- Blomfield, G. W., Eckert, H., Fisher, M., Miller, H., Munro, D. S., and Wilson, G. M. (1959). *Brit. med. J.*, **1**, 63.
Crooks, J. (1965). In *Current Topics in Thyroid Research*, edited by C. Cassano, and M. Andreoli, p. 1208. New York and London.
Curran, R. C., Eckert, H., and Wilson, G. M. (1958). *J. Path. Bact.*, **76**, 541.
Dobyns, B. M., Vickery, A. L., Maloof, F., and Chapman, E. M. (1953). *J. clin. Endocr.*, **13**, 548.
Doniach, I. (1958). *Brit. med. Bull.*, **14**, 181.
Dunn, J. T., and Chapman, E. M. (1964). *New Engl. J. Med.*, **271**, 1037.
Eckert, H., Green, M., Kilpatrick, R., and Wilson, G. M. (1960). *Clin. Sci.*, **20**, 87.
Green, M., Fisher, M., Miller, H., and Wilson, G. M. (1961). *Brit. med. J.*, **2**, 210.
— and Wilson, G. M. (1964). *Ibid.*, **1**, 1005.
Greig, W. R. (1965). *J. clin. Endocr.*, **25**, 1411.
— Crooks, J., and Macgregor, A. G. (1966). *Proc. roy. Soc. Med.*, **59**, 599.
Karlson, M. S., Pollock, W. F., and Snyder, W. H. (1964). *Calif. Med.*, **101**, 196.
Kilpatrick, R., Blomfield, G. W., Neal, F. E., and Wilson, G. M. (1957). *Quart. J. Med.*, **26**, 209.
Kogut, M. D., Kaplan, S. A., Collipp, P. J., Tiamsic, T., and Boyle, D. (1965). *New Engl. J. Med.*, **272**, 217.
McGirr, E. M., Thomson, J. A., and Murray, I. P. C. (1964). *Scot. med. J.*, **9**, 505.
Macgregor, A. G. (1963). In *The Thyroid and its Diseases*, edited by A. S. Mason, p. 19. London.
Nofal, M. N., Beierwaltes, W. H., and Patno, M. E. (1966). *J. Amer. med. Ass.*, **197**, 605.
Philp, J. R. (1966). *Postgrad. med. J.*, **42**, 437.
Pochin, E. E. (1960). *Brit. med. J.*, **2**, 1545.
— (1964). *Proc. roy. Soc. Med.*, **57**, 564.
Sheline, G. E., Lindsay, S., McCormack, K. R., and Galante, M. (1962). *J. clin. Endocr.*, **22**, 8.
Smith, R. N., and Wilson, G. M. (1965). In *Current Topics in Thyroid Research*, edited by C. Cassano, and M. Andreoli, p. 1153.
Staffurth, J. S. (1966). *Brit. J. Radiol.*, **39**, 471.

Staphylococcal Sepsis in a New Surgical Ward

PHYLLIS M. ROUNTREE,* D.SC., DIP.BACT.; MARY A. BEARD,† M.SC.

JOHN LOEWENTHAL,‡ M.S., F.R.C.S., F.R.A.C.S., F.A.C.S.; JAMES MAY,§ F.R.A.C.S.; S. B. RENWICK,|| F.R.A.C.S.

Brit. med. J., 1967, **1**, 132-137

The building of a new professorial block at Royal Prince Alfred Hospital (Loewenthal, 1962) has afforded the opportunity to study the effect of environmental factors on the incidence of surgical sepsis in patients undergoing a variety of general surgical operations. The new block was occupied at the end of December 1962, and the 1,337 patients admitted to its surgical ward up to the end of April 1965 form the subject of the present report.

Prior to this the patients of the professorial surgical unit were housed in an old open ward where there were high infection rates (Rountree, Harrington, Loewenthal, and Gye, 1960) and where there was widespread contamination of the air and bedding with *Staphylococcus aureus* (Rountree and Beard, 1962). Measures introduced into that ward in an attempt to reduce the infection rate included the sealing of all suitable wounds with plastic seals, the use of individual dressing-trays, the substitution of cotton for woollen blankets, and the institution of nasal prophylaxis with a cream containing neomycin and chlorhexidine (Rountree, Loewenthal, Tedder, and Gye, 1962). None of these measures was more than partially successful, and in 1962 14% of 326 operation wounds

were infected, the majority of these infections occurring in the ward.

Design of the Ward

The plan of the new ward was given by Loewenthal (1962). It is an adaptation of the Nuffield Ward and contains beds for 30 patients. Twenty-four beds are in six open bays containing four beds each; there are four single-bed rooms and one room with two beds. Each open bay has its own water-closet and wash-basin. There is an air-conditioned treatment-room in a central position in the ward.

Methods of Investigation

Swabs were taken from all surgical wounds which showed evidence of sepsis in the form of pus or discharge. Any other lesions, such as ulcers or pustules, present when the patient was admitted or which developed later were also swabbed. A nasal swab was taken from each patient on the day of admission or as soon as possible after this; thereafter each patient was swabbed once a week. At irregular intervals nasal swabs were taken from the nursing and medical staff.

From January 1963 to November 1964 environmental contamination was measured once a week. Settle plates were used rather than the slit sampler, since the architecture of the ward did not lend itself to the use of the slit sampler. These plates

* Bacteriologist, Royal Prince Alfred Hospital, Sydney.
† Senior Research Officer (National Health and Medical Research Council), Sydney.
‡ Professor of Surgery, University of Sydney.
§ Surgical Registrar (1962-3), Professorial Unit, Royal Prince Alfred Hospital, Sydney.
|| Surgical Registrar (1964-5), Professorial Unit, Royal Prince Alfred Hospital, Sydney.

TABLE I.—Incidence of Wound Infection in Male and Female Patients from January 1963 to April 1965

	1963			1964			1965			Total		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
No. of patients	316	216	532	364	269	633	95	77	172	775	562	1,337
No operation	85	56	141	87	82	169	17	22	39	189	160	349
Operation	231	160	391	277	187	464	78	55	133	586	402	988
No sepsis	193	139	332	239	166	405	67	49	116	499	354	853
Sepsis	38	21	59	38	21	59	11	6	17	87	48	135
<i>Staph. aureus</i> sepsis ..	24 (10%)	13 (8%)	37 (9%)	29 (10%)	12 (7%)	41 (9%)	7 (9%)	5 (9%)	12 (9%)	60 (10%)	30 (7%)	90 (9%)
Minimal infection (no swabs)	8	2	10	1	2	3	—	—	—	9	4	13
Other than <i>Staph.</i> ..	6	6	12	8	7	15	4	1	5	18	14	32

were exposed for one hour each week at the same positions in the ward, the majority on bedside lockers, in the middle of the morning at a time of maximum activity. Contact plates (Rubbo and Dixon, 1960) were taken from one set of curtains in each bay. Contact plates were also taken from one blanket or counterpane on every occupied bed.

All cultures of *Staph. aureus* isolated from lesions and noses were tested for antibiotic sensitivity and were phage-typed. When only a few colonies grew on the settle or contact plates all were typed, but when large numbers were present only a proportion were examined. The typing phages used were the international set of Blair and Williams (1961), with the addition in January 1964 of the three experimental phages—B5, D, and 77Ad (Jevons and Parker, 1964).

Sepsis Rates

Wounds

Of the 1,337 patients 349 had no operation. Of the 988 who were operated on the wounds of 853 healed without any signs of sepsis. A total of 90 patients (9%) had wounds infected with *Staph. aureus*. Table I shows that the incidence of infection in the years 1963, 1964, and 1965 did not vary greatly. There was more sepsis in the men (10.2%) than in the women (7.4%). This difference between the sexes has been noted consistently throughout our studies. Thirty-two patients had sepsis due to organisms other than staphylococci; these were chiefly *Escherichia coli*, *Proteus*, and *Pseudomonas aeruginosa*. In the remaining 13 infection was minimal and no swabs were taken.

An estimate was made of the place where the wound had been infected (Table II). Autogenous infection was presumed when the phage type of the infecting staphylococcus, or the antibiotic sensitivity of a non-typable strain, was identical with that isolated from a lesion or the nose before operation. In 1963 38% of the infections were of this kind, in 1964 22%, and in 1965 25%. Only seven infections were thought to have occurred in the operating-theatres, and it is possible that some of these were autogenous and caused by staphylococci present on sites other than the patient's nose. The proportion of infections that occurred in the ward increased from 49% in 1963 to 61% in 1964. This increase, which affected the men patients chiefly, was due to the introduction of a neomycin-resistant hospital strain early in the year (Rountree and Beard, 1965). In the old ward, during 1959–61, 65% of the infections were presumed to have occurred there.

The incidence of staphylococcal sepsis according to the anatomical site of the wounds is shown in Table III. (Several patients had more than one operation.) The highest incidence

TABLE II.—Probable Place of Staphylococcal Infection of Surgical Wounds

Place of Infection	1963			1964			1965			Total		
	M	F	Total	M	F	Total	M	F	Total	M	F	Total
Theatre	2	1	3	1	3	4	—	—	—	3	4	7
Ward	10	8	18	19	6	25	6	3	9	35	17	52
Autogenous ..	10	4	14	7	2	9	1	2	3	18	8	26
Uncertain ..	2	—	2	2	1	3	—	—	—	4	1	5

was in thoracic wounds (17%) and in the limbs (12% in the upper and 14% in the lower).

The incidence of infection in certain types of operations was also examined (Table IV). No staphylococcal sepsis was seen in 121 appendicectomies. In contrast with this, the highest rate was in 144 vascular operations, 26% of which were infected,

TABLE III.—Sites of Operations Infected with *Staph. aureus*

Site of Operation	No. of Operations	No. Infected	With <i>Staph. aureus</i>	
			No.	%
Head	23	—	—	—
Neck	50	5	5	10
Thorax	29	7	5	17
Abdomen	585	64	43	7
Upper limb	43	5	5	12
Lower	234	42	33	14
Skin only	29	—	—	—
Total	993	123	91	—

TABLE IV.—Incidence of Staphylococcal Sepsis in a Variety of Surgical Procedures

Type of Operation	No. of Operations	No. Infected	With <i>Staph. aureus</i>	
			No.	%
Appendicectomy ..	121	3	—	—
Cholecystectomy ..	36	4	3	8
Hernia repair ..	51	3	2	4
Sympathectomy ..	61	2	1	1.6
Splenectomy ..	20	2	—	—
Vascular operations ..	144	37	28	20
Varicose vein operations	60	3	3	5
Total	493	54	38	—

chiefly with *Staph. aureus*. Although these operations comprised only 14.5% of all operations, infection in them accounted for 30.8% of the total sepsis. The high incidence of infection in these patients is associated with hypertension and peripheral ischaemia (May, Chalmers, Loewenthal, and Rountree, 1966). They were almost invariably associated with long incisions extending from the xiphisternum to pubis, or transversely across the whole of the anterior abdominal wall, or in the lower limb from groin to knee.

Sepsis other than Wounds

Staphylococci were isolated from a number of sites other than operation wounds, including ulcers, bedsores, and burns (Table V). Many of these patients were admitted with infection, but the burned patients in particular were infected with "hospital" strains and were potent sources of infection for

TABLE V.—Numbers of Patients with *Staph. aureus* in Sites Other than Surgical Wounds

	1963	1964	1965	Total
Abscess	2	1	1	4
Furuncle	1	2	—	3
Sinus	1	4	1	6
Ulcers and bedsores	7	12	—	20
Burns	1	7	—	8
Malignancies ..	4	1	1	6
Conjunctivitis ..	—	1	—	1
Sputum	3	1	2	6
Total	19	29	6	54

others. Although there were six isolations of staphylococci from sputum, only two patients were thought to have staphylococcal pneumonia.

Phage Types of *Staph. aureus* in Lesions

A total of 152 strains were identified in the surgical wounds and other lesions, more than one strain being found in eight patients. Table VI gives their phage-type distribution.

TABLE VI.—Phage-type Distribution of 152 Strains of *Staph. aureus* Isolated from Surgical Wounds and Other Lesions

Phage Groups	1963	1964	1965	Total No.
Group I { 80/81	7	5	—	12 (7.9%)
{ Other	4	5	2	11
Group II { 53 at 1,000 × R.T.D. ..	1	2	—	3
Group III { B5/77Ad	13	17	3	33 (21.7%)
{ Other	—	9	—	9
Group IV	17	14	5	36
Miscellaneous	1	—	1	2
Not classifiable	—	1	—	1
Not typable	1	1	—	2
	15	20	8	43 (28%)

The chief hospital strain was always resistant to penicillin, streptomycin, and tetracycline and sometimes to chloramphenicol or erythromycin; it was lysed only by phage 53 at 1,000 × R.T.D., though it showed inhibition with other phages of group III, including phage 47. This strain has been previously reported as type 47 at 1,000 × R.T.D. (Rountree and Beard, 1962). In 1960–61 this strain accounted for 62% of all infections in the old ward. In the new ward only 33 (22%) were caused by it. Of these 33 patients eight had the strain in their nose or in lesions when admitted, either from other parts of the hospital or as readmissions, having been in the old ward earlier. Another hospital strain which was neomycin-resistant and of type B5/77Ad caused nine infections after its introduction in 1964. Type 80/81 strains were found in 12 patients, in some of them as hospital infection but in others as a result of self-infection. These strains were usually resistant only to penicillin, and in this hospital do not now behave as endemic hospital strains.

Nasal Carriage of *Staph. aureus*

In the old ward nasal prophylaxis with a cream containing neomycin and chlorhexidine (Naseptin) had been introduced for all patients in 1961. It was continued after the move to the new ward but was stopped at the end of August 1964 after the neomycin-resistant type B5/77Ad strain had been isolated from 8.9% of the nasal swabs taken in July and August.

The nasal carrier rates in patients, based on the weekly swabs, are given in Table VII, together with those for the 15

TABLE VII.—Weekly Nasal Swabs of Patients from October 1961 to April 1965, Showing Incidence of *Staph. aureus*: 1961–2 in Old Ward; from January 1963 in New Ward

Period	Males		Females		Total			
	No.	% Positive	No.	% Positive	No.	% Positive	% with Multiple Resistant Strains	% with Neomycin-resistant Strains
Oct. 61–Dec. 62 (Naseptin) ..	1,000	30	936	27	1,936	29	12	—
1963: Jan.–Dec. (Naseptin) ..	1,018	21	737	18	1,755	20	1.7	—
1964: Jan.–Mar. (Naseptin) ..	237	22	176	17	413	20	3.6	1.5
Apr.–June (Naseptin) ..	227	29	188	25	415	27	10.6	5.8
July–Aug. (Naseptin) ..	173	32	118	15	291	25	14	8.9
Sept.–Dec. (No Naseptin)	263	26	185	37	448	31	5.6	1.2
1965: Jan.–Apr. (No Naseptin)	277	29	198	40	475	34	6	—

months before the move. They can be examined to determine the effect of the new environment on the carrier rate of hospital strains, both during the use of Naseptin and without it.

In 1963 the move to the new ward had a profound effect on the carrier rate of the hospital strain, which was reduced to 1.7% compared with 12% in the preceding period. Though 13 infections were caused by these strains, they were found in only 30 nasal swabs from 19 patients. During 1963 there were acquisitions of new strains in 58 (3.3%) of 1,755 swabs, but only 12 (2.3%) out of the 532 patients admitted acquired the hospital strain. The overall carrier rate during 1963 was 20% compared with 29% in the old ward.

This satisfactory situation did not continue in 1964. In Table VII the figures for this year are divided into four periods to illustrate the increase in carriage of hospital strains, which was due to both the type 53 and the type B5/77Ad strains and reached a percentage of 14% in July and August.

With the cessation of Naseptin prophylaxis the selective action of neomycin on the nasal flora disappeared, as did the neomycin-resistant strains, none being found in the first four months of 1965.

The figures for 1965 can be compared with those in the old ward before Naseptin was used. During a period of six months in 1961 nasal swabs from patients acting as controls in trial of Naseptin prophylaxis showed 46% of positive swabs with 24% yielding hospital strains. The comparable figures in 1965 are 34% and 6%.

We concluded that the layout of the new ward substantially reduced the transfer of staphylococci to the noses of patients. Though the introduction of hospital strains could not be prevented, widespread colonization with them occurred only when they were adapted to survive in the presence of a powerful selective agent such as neomycin. Even under such conditions the carrier rates did not reach those found in the old ward.

Environmental Contamination

Air

Over the period of 23 months from January 1963 to November 1964 1,811 settle plates were exposed. These gave a mean total bacterial count of 26.3 colonies per plate per hour. The mean total count per month fluctuated widely, the lowest count being 15.3 in September 1964 and the highest 42.2 in July 1963. No correlation could be seen between the season and the mean monthly count.

The recovery of *Staph. aureus* on the settle plates likewise fluctuated; the peak months of *Staph. aureus* isolations usually, but not invariably, corresponded with those of high total counts (see Chart). A total of 1,501 colonies of *Staph. aureus* were identified; they were found on 21.5% of the plates. The mean count per positive plate was 3.8.

Mean monthly counts of total bacteria and of *Staph. aureus* on settle plates exposed each week in the ward.

These values for *Staph. aureus* may be compared with those found in the open ward (Rountree and Beard, 1962), where 57% of 646 plates were positive, the mean count per positive plate being 4.1. We interpret this as meaning that, while there was much less staphylococcal contamination in the new ward, when it did occur the load was approximately the same.

Bedding

From January to November 1963 both cellular cotton and woollen blankets were in use. After November only cotton was used. At the times of sampling about 20% of the beds had no blankets on them; their cotton counterpanes were therefore sampled.

Table VIII compares the results obtained from these three kinds of bedding with those from cotton blankets in the old ward in 1960-1. It should be noted, however, that the counts per plate are not strictly comparable between the two wards, as sweep plates were used in the old ward and the more convenient contact method in the new one. We compared the results obtained on 15 sets of plates, both methods on cotton blankets in the new ward being used. Single contacts gave a mean count of 37.4 compared with 53.2 for sweeps. Even when this adjustment is made to the counts from cotton blankets in the new ward, it is apparent that the mean total count is less than half of that obtained in the old ward.

TABLE VIII.—Comparison of Bacterial Loads on Textiles in Old and New Wards (Old Ward, Sweep Plates; New Ward, Contact Plates)

	Old Ward	New Ward			
	Cotton Blankets	Blankets		Counterpanes	Curtains
		Wool	Cotton		
No. sampled	1,005	497	1,507	514	613
Mean total bacterial count/plate	130	75	33	114	27
With <i>Staph. aureus</i>	608	90	257	184	129
%	60.5	18.1	17	35.4	21
Mean <i>Staph. aureus</i> /plate	5.1	1.5	1.1	6.2	0.7

Both the numbers of blankets from which *Staph. aureus* was isolated and the mean *Staph. aureus* count were much lower in the new ward.

Counterpanes had not been examined previously. They were found to be much more heavily contaminated than were the blankets. Approximately twice as many counterpanes as blankets yielded *Staph. aureus*, and in much larger numbers.

Curtains

The curtains in the bays are made of a cotton material with a rough woven surface. They are arranged so that they can be pulled around the beds when privacy is required. The possibility therefore exists that they may be contaminated by the nurses' hands or by droplets from the patients' respiratory tracts and that these contaminants may be liberated when the curtains are moved later on. They were infrequently laundered.

Sampling of 613 curtains yielded *Staph. aureus* on 21% of occasions. The average yield per positive plate was 3.43 colonies. The mean total bacterial count was 27 per plate. These counts corresponded closely with those found on the settle plates.

Phage Types of Staphylococci in the Ward Environment and Patients' Noses

Table IX gives the phage-type distribution of the staphylococci in the patients' noses and of colonies grown from the curtains, bedding, and air.

There was good correlation between the strains found in the bedding and in the air, but some minor differences were found between these results and those on the curtains and in the patients' noses. For example, there were fewer non-typable strains and more hospital strains on the curtains than in the air and bedding.

TABLE IX.—Phage Group Distribution of Strains from Bedding and Patients' Noses

	Blankets and Counterpanes		Curtains		Settle Plates		Patients' Noses	
	No.	%	No.	%	No.	%	No.	%
Group I { 80/81 ..	92	7.4	16	6.7	62	9.8	27	3.3
Others ..	45	3.6	29	12.2	13	2.0	69	8.4
Group II { ..	39	3.1	4	1.7	16	2.5	40	4.9
Group III { "Hospital strains" ..	216	17.4	60	25.3	110	17.3	184	22.4
Others ..	474	38.2	69	29.0	213	33.7	170	20.7
Group IV { ..	11	0.9	1	0.4	18	2.8	6	0.8
Misc. and non-classifiable ..	18	1.5	2	0.8	3	0.4	27	3.3
Not typable ..	348	27.9	57	23.9	199	31.5	298	36.2
Total ..	1,243		238		634		821	

One interesting point was that there were more type 80/81 isolations from all parts of the ward than from the patients' noses; the proportion of type 80/81 strains corresponded to that found in the lesions. Possible explanations of this difference are that the type 80/81 strains are dispersed in larger numbers from those carrying or infected by them or that they persist for longer once they have been shed from their hosts. There is a small amount of evidence, described below, that the second explanation is likely.

However, none of the observed differences was large, and it is apparent that the staphylococci in the ward air, textiles, patients' noses, and lesions were similar. The same situation was found in the old ward, where, however, there was a great preponderance of group III hospital strains.

Spread of Certain Strains in the Ward

Some weeks before the ward was opened an official opening ceremony took place outside it. After the ceremony some hundreds of people walked through the ward. Twenty settle plates were exposed on this occasion but no colonies of *Staph. aureus* were grown from them.

As soon as patients were admitted staphylococci were isolated from the ward. They were found in small numbers in January and February and were mainly non-typable strains or the hospital strain, typing with phage 53 at 1,000 × R.T.D. Among the first patients were two who had been known nasal carriers of the hospital strain, and one had his wound infected with this organism four days after operation. Within the next three weeks three further patients were admitted carrying the strain, and one of these had a skin graft infected with it. It was isolated from the air and bedding in January and February, but then disappeared until May. Thereafter, though present in reduced numbers compared with the old ward, it was never absent for more than a few weeks at a time from bedding, air, lesions, or noses, being continually reintroduced by patients.

Type 80/81 strains were often introduced into the ward during 1963. The first isolation was made from a bed in the male end of the ward on 15 January and from the curtains and in the air of the same bay on 5 February. No patient carrying the strain had been found at this time, but a nurse was a nasal carrier on 6 February and may have been the source of this contamination. This nurse may also have infected a young girl who was at the other end of the ward and who had had a nephrectomy on 23 January. Her wound discharged pus on 31 January and she was moved to a single room. The type 80/81 cocci were found on her bed on 5 February and were still present on 5 March, though her wound had healed by then and she was not a nasal carrier.

Probable source of subsequent recoveries of type 80/81 strains are difficult to determine, since nine carriers of the strain were admitted in the weeks between 11 February and 22 April. Two of them admitted on 22 February and 1 March occupied beds 22 and 24 in the same four-bed bay, and, though they received Naseptin and were not nasal carriers after the first swabbing, beds 23 and 24 were found to be contaminated on 5 March. On 18 March the patient in the adjacent bed 21, who had been admitted on 6 March and operated on on that date, was found to have acquired the strain at the weekly nasal swabbing, and on 20 March her wound was infected with the strain. The events in this bay suggested that spread to the patient had occurred via the bedding and air. On 1 April the patient in bed 25 was found to have acquired the strain in her nose.

A possible example of the persistence of the type 80/81 cocci in the ward environment and their ability to infect subsequently another patient occurred later in the year. On 13 May a nasal carrier was admitted to the two-bedded room at the end of the ward and was still carrying on 21 May, when he was transferred to another ward. He had been too ill with head injuries to have Naseptin prophylaxis. Four weeks later, on 24 June, and again on 1 July, the bedding in this room was found to be contaminated. A patient admitted to the same room on 5 August, who had not at any time been found to be a nasal carrier, sustained heavy infection of a discharging sinus of the thigh on 15 August. During her stay in the room the bedding, air, and curtains were heavily contaminated, and type 80/81 cocci were still present on the curtains in large numbers three days after she had been discharged. The curtains were then laundered and there were no further isolations of the strain from this room.

The spread of the neomycin-resistant type B5/77Ad strain in this ward in 1964 has already been described (Rountree and Beard, 1965). The source of the episode of heaviest contamination was a burned patient, who, though in a single room, released very large numbers of the organisms from his burns. At this time these type B5/77Ad cocci were found at 18 sites in the ward, and were particularly heavy at the men's end. After Naseptin prophylaxis was abandoned and the infected patients and carriers were discharged this strain disappeared.

Discussion

The infection rate of surgical wounds with staphylococci was significantly reduced in the new ward. This applied particularly to infection with "hospital" strains. Along with this reduction in actual infection there was also a reduction in morbidity. During the first 18 months in the new ward the mean number of extra days spent in the ward by infected patients was 11.3 compared with 17.2 days in the old ward (May *et al.*, 1966).

Side by side with the reduction in sepsis rates there was also a reduction in the contamination of the ward environment with staphylococci. Though a different method of sampling textiles was used and adjustments must be made to the counts obtained by the two methods, the overall results showed a fall in the proportion of blankets contaminated with staphylococci, and this was particularly so with the hospital strains. The staphylococcal content of the air and the phage-type distribution of these staphylococci also showed a reduction of the proportion of plates yielding *Staph. aureus* from 57 to 21.5% and a reduction of hospital strains from 72.2 to 17.3%.

It is difficult to give an opinion on how much the layout of the ward affected the degree of environmental contamination or the passage of staphylococci from patient to patient, whether to their noses or wounds. The epidemic of the type B5/77Ad strain provided evidence that placing a heavily infected patient, such as one with burns, in a single room which was situated in the centre of the ward did not prevent the widespread dispersal of his staphylococci. The single rooms in the unit are not air-conditioned, and air currents from the windows

across the room into the main corridor, especially apparent when doors are opened, are an obvious means of dispersing organisms shed by patients in these rooms.

Improvement in the ventilation of these rooms could be made by the installation in the windows of fans sucking air from the rooms to the outside of the building. Two small cubicles at the end of one bay in the male part of the ward were equipped with such fans and were used for nursing patients admitted with known skin contamination. One male patient known to be a gross disperser of type 80/81 staphylococci was admitted to one of these cubicles on several occasions in 1963 and 1964, and on very few sampling days during his admissions were these staphylococci found outside this cubicle.

Curtains as a potential source of infection have not been studied, so far as we are aware. Our investigations showed that the cotton curtains between the beds in the new ward could be contaminated with the staphylococci present in the ward. Of all the curtains sampled 21% were found to release these organisms on to contact plates, and presumably they would also do so when moved backwards or forwards along their tracks. How often this contamination is due to the hands of nurses and doctors and how often to the patients themselves cannot be established. In other places we have found curtains contaminated with staphylococci of the same phage type as those in the lower respiratory tract of patients with tracheostomy wounds. The laundry facilities available did not permit the frequent changing and washing of these curtains. In any case, their design is such that a ladder must be mounted to remove them from their track. In old-fashioned wards, where patients were screened by movable screens whose cotton covers could be easily removed and washed, this problem did not arise. In the light of our experience we would now regard ward curtains as potential trappers and dispersers of staphylococci.

However, as with other kinds of ward textiles, it is very difficult to incriminate such articles as direct causes of sepsis. One tenable view is that they merely reflect the degree of sepsis in a given place and that other, human, factors are responsible for the transfer of staphylococci to wounds.

Some other general comments may be made on the architecture of the ward as it affected the care of patients and the ease with which the nursing staff carried out their duties.

The siting of the nurses' station was such that not one patient could be observed from it. This is contrary to the principles of hospital design enunciated by Gainsborough and Gainsborough (1964), and in practice it meant that the sister had to spend a fair amount of her time walking around the ward. The room at the end of the ward intended to be her office proved to be too far away from ward activities and has never been used as such.

The treatment room in the centre of the ward was originally intended for the dressing of patients' wounds. Its ventilation was inadequate, and in the hot weather the door into the corridor had to be left open, thus defeating the purpose of the room. Furthermore, the effort and the time required to move patients into the room from their positions in the wards were such that it was found preferable to dress their wounds in their beds, the instruments and trays being supplied by the central sterile supply department. In addition, very ill patients should not be moved any more than is necessary. So, in actual practice, this room was used chiefly for examination of patients attending special outpatient or follow-up clinics.

Difficulties were experienced with the disposal of laundry. No laundry chutes had been built into the building because of the dangers that have been stated to follow the dispersal of staphylococci and infection generally in such chutes (Hurst, Grossman, Ingram, and Lowe, 1958). This meant that because of the vagaries of the laundry service soiled linen removed to the small utility room at the end of the ward might lie there for three days over the week-end before it was removed—surely an undesirable and unsanitary practice.

However, when all these matters are considered it is still the opinion of the surgeons that surgical practice and the care of the patients was greatly superior to that in the old-style ward. Procedures in vascular surgery that had been discontinued because of the high risk of sepsis in the old accommodation were carried out with relatively good results, and there was much less morbidity and bed loss due to sepsis.

Summary

In a new surgical ward built in 1962 1,337 patients were admitted between January 1963 and May 1965, and 988 had operations.

The sepsis rate with *Staphylococcus aureus* in the patients' wounds was 9%, 10% of the men being infected and 7% of the women. This compared favourably with the rate of 14% found in 1962 in an old open ward.

Only 28% of this sepsis was due to multiple antibiotic-resistant "hospital" strains, compared with 56% in the old ward.

There was a reduction in the nasal-carrier rate of hospital strains in patients in the new ward.

There was also a reduction in the amount of staphylococcal contamination of the air and bedding.

Some of the advantages and disadvantages of the design of the new ward as it affected the care of the patients are discussed.

This work was supported in part by a grant from the National Health and Medical Research Council. Our thanks are particularly due to Mrs. Elisabeth Bradshaw for her technical assistance. Miss I. Smyth, ward sister in charge of the old and new ward accommodation, has tolerated with invariable good humour and co-operation the many inconveniences which are an integral part of an investigation of this type.

REFERENCES

- Blair, J. E., and Williams, R. E. O. (1961). *Bull. Wld Hlth Org.*, **24**, 771.
 Gainsborough, H., and Gainsborough, J. (1964). *Principles of Hospital Design*. London.
 Hurst, V., Grossman, M., Ingram, F. R., and Lowe, A. E. (1958). *J. Amer. med. Ass.*, **167**, 1223.
 Jevons, M. P., and Parker, M. T. (1964). *J. clin. Path.*, **17**, 243.
 Loewenthal, J. (1962). *Brit. med. J.*, **1**, 1437.
 May, J., Chalmers, J. P., Loewenthal, J., and Rountree, P. M. (1966). *Surg. Gynec. Obstet.*, **122**, 28.
 Rountree, P. M., and Beard, M. A. (1962). *J. Hyg. (Lond.)*, **60**, 387.
 — (1965). *Med. J. Aust.*, **1**, 498.
 — Harrington, M., Loewenthal, J., and Gye, R. (1960). *Lancet*, **2**, 1.
 — Loewenthal, J., Tedder, E., and Gye, R. (1962). *Med. J. Aust.*, **2**, 367.
 Rubbo, S. D., and Dixon, S. (1960). *Lancet*, **2**, 394.

Gastric Mucosa and Susceptibility to Occult Gastrointestinal Bleeding Caused by Aspirin

D. N. CROFT,* D.M., M.R.C.P.; PHILIP H. N. WOOD,† M.B

Brit. med. J., 1967, **1**, 137-141

The relation between taking aspirin and the development of gastrointestinal haemorrhage was first recognized by Douthwaite and Lintott (1938). This association has attracted considerable attention, and the subject has been reviewed by Muir (1963a). The occurrence of overt haemorrhage is not directly related to susceptibility to occult bleeding (Parry and Wood, 1963, 1966), a fact probably responsible for much of the confusion on this subject. The two orders of bleeding are best considered separately, and this paper is concerned primarily with the mechanism of occult aspirin bleeding.

Occult blood loss occurs in the majority of people who take repeated doses of aspirin (Stubbs, 1958; Pierson, Holt, Watson, and Keating, 1961; Wood, Harvey-Smith, and Dixon, 1962). It is unrelated to dyspepsia caused by aspirin, as the same amount of blood is lost when aspirin is taken on an empty stomach as compared with administration after meals (Wood, 1963; Wood and Dixon, 1966), though dyspepsia is much more frequent when the drug is taken on an empty stomach. Furthermore, patients with an active peptic ulcer have been shown not to be unusually susceptible to aspirin-induced occult bleeding (Parry and Wood, 1966), even though they readily develop dyspeptic intolerance of the drug (Muir, 1963b); similarly, Winkelmann and Summerskill (1961) found that aspirin bleeding was unrelated to gastric acid or pepsin secretion.

Gastrosopic observations (Hurst and Lintott, 1939; Weiss, Pitman, and Graham, 1961), examination of the stomach after

surgical excision (Muir and Cossar, 1955, 1961), and fractional gastric analysis (Muir and Cossar, 1961) after the administration of single doses of aspirin have indicated that the stomach is the major site of occult bleeding, and this accords with the finding that delayed release of aspirin, as by enteric coating, is associated with a considerable reduction in the volume of blood lost (Stubbs, Pietersen, and van Heulen, 1962; Wood and Dixon, 1966).

This report presents three series of observations: (1) a description of the lesions observed in the stomach after repeated doses of aspirin; (2) the variation in susceptibility to occult bleeding between different individuals, and the relation between the degree of susceptibility and quantitative estimates of exfoliation of gastric epithelial cells; and (3) an analysis of the features of individuals that are relatively insensitive to aspirin-induced occult bleeding. Taken together these observations enable one to resolve some of the conflicts between previous reports. The data also suggest that lesser degrees of susceptibility to bleeding are sometimes associated with atrophic gastritis, a condition in which there is evidence of an increased turnover of surface epithelial cells (Croft, Pollock, and Coghill, 1966).

Gastrectomy Studies

Previous direct observations of the human gastric mucosa after aspirin has been taken (Muir and Cossar, 1955, 1961; Weiss *et al.*, 1961) have indicated that a single dose of the drug will cause an erosion in up to 20% of the subjects studied. The use of a minimal stimulus, a single dose, and

* St. Thomas's Hospital, London, and West Middlesex Hospital, Isleworth. Now Senior Medical Registrar, St. Thomas's Hospital, London.

† Postgraduate Medical School of London. Now Senior Research Fellow, Manchester University Rheumatism Research Centre, Manchester.