J. Hyg., Camb. (1971), **69**, 179
Printed in Great Britain

Pseudomonas aeruginosa in a Regional Burns Centre

By F. DEXTER

Regional Tissue Bank, Pinderfields General Hospital, Wakefield, Yorkshire

(*Received* 12 *October* 1970)

SUMMARY

The construction of a Regional Burns Centre in Pinderfields General Hospital, Wakefield, presented an opportunity to study *Pseudomonas aeruginosa* infection in patients with extensive burns. During the first year (Barclay & Dexter, 1968) a system of disinfection and bacteriological control created conditions permitting more detailed studies to be undertaken which resulted in a significant reduction of infection and cross-infection.

MATERIALS AND METHODS

Twelve-hour culture settle plates (Oxoid blood agar base No. 2) were placed in all parts of the burns unit each day with a view to assessing bacterial fall-out from the air for each locality. Colony counts and types of bacteria were studied in association with treatment and nursing procedures, and served as a guide to determine the most effective methods of controlling the bacteria by disinfection. Water supplies, drain systems, sluices, etc., were also examined and disinfection procedures were instituted as required. *Pseudomonas* strains grown from patients and their surroundings were pyocine typed (Darrell & Wahba, 1964; Gillies & Govan, 1966, Govan & Gillies, 1969) to establish identity or differences. The isolation of *Pseudomonas* in pure culture was by using 0·03 % Cetrimide in (Oxoid) blood agar base No. 2 and 0·02 % Centrimide in (Oxoid) Nutrient broth No. 2 (Shooter *et al.* 1966). Other routine medium was also used and disinfectant inactivators were included as required.

During the first year 'Idokyl', an iodophor, was the only disinfectant used, but during the subsequent years 'Tego' (MHG), an ampholytic surface-acting biocide disinfectant, was used. Both disinfectants were applied as aerosol mists (Barclay & Dexter, 1968), followed by a disinfectant surface wash; aerosols were never used as a substitute for washing. By this method bacteria were first precipitated on surfaces which were immediately washed, killing most microbial survivals.

RESULTS

Airborne Pseudomonas aeruginosa

Recovering of *Pseudomonas* from settle plates on the floor of infected patients' cubicles, treatment rooms and traffic zones indicated widespread distribution of this organism via the air as evidenced by pyocine typing results. It was also noted

that settle plates situated on window ledges several feet high consistently yielded about the same number of *Pseudomonas* colonies as settle plates on the floor, and it was considered that the air in treatment rooms might be continuously contaminated with *Pseudomonas*. In attempting to assess the incidence of airborne *Pseudomonas* under more controlled conditions, simple structures were prepared permitting daily 7 hr. settle plates to be placed at intervals of 1 ft. between the floor and ceiling of treatment rooms (Barclay & Dexter, 1968). These were in addition to the routine 12 hr. settle plates on the floor. These structures were called 'gravitation trees' and several were used in the cubicles of infected and uninfected

Table 1. *Extract from records (14-day period only) of colony counts of* Pseudomonas *on cultures on one of the 'gravitation trees' in a cubicle occupied by an infected patient (this patient did not receive topical cream treatment)*

Height above floor (ft.)	Colony counts of *Pseudomonas* on day: 3	4	5	6	7	8	9	10	11	12	13	14	15	16
9	1	1	0	13	2	11	0	1	0	13	0	5	0	8
8	3	3	1	15	3	18	1	0	1	22	1	6	0	8
7	0	5	0	11	0	14	0	18	0	11	1	7	1	11
6	1	1	1	11	0	15	0	15	4	20	0	6	0	5
5	0	1	1	9	0	12	2	19	1	16	0	5	0	2
4	3	2	0	5	0	20	1	17	1	23	1	9	0	5
3	3	3	1	5	2	14	0	19	3	25	1	7	0	12
2	0	7	1	19	0	20	0	13	1	13	1	8	4	5
1	0	6	1	16	1	13	0	15	0	17	0	3	0	7
Floor	0	2	1	15	3	11	0	14	1	17	0	5	0	13

patients, traffic zones and other treatment rooms in the unit. The results clearly demonstrated that living *Pseudomonas* was randomly distributed in the air of rooms in which infected patients were being treated (Table 1). Persistence of *Pseudomonas* in the air during the more active daily periods and less active nightly periods continued throughout the infective phase of patients' wounds. Continuous shedding of *Pseudomonas* from wounds on bedding resulted in frequent eruptions of this organism into the air when patients moved, or during treatment and bed linen changes. It was found that the presence of *Pseudomonas* in the air originated from infected patients, whereas the remaining bacteria in the air had many other influencing factors. Effective control of bacteria in the air of infected patients' cubicles did not achieve control of *Pseudomonas* in the air, and there appeared to be no relationship between *Pseudomonas* and the remaining bacterial population.

In the Burns Centre positive-pressure filtered air was provided in the dressings-treatment room, and air extraction in the dirty utility room (Barclay and Dexter 1968). Heavy growths of *Pseudomonas* were recovered from settle plates in all the rooms of the dressing suite during treatment of infected patients, and *Pseudomonas*, of the same pyocine type as that carried by the patient receiving treatment, was also recovered from other parts of the burns unit remote from either the patient or the dressing suite. It was established beyond doubt that the positive-pressure ventilation system was responsible for the widespread airborne distribu-

tion of this organism. The positive pressure was eventually replaced by negative pressure with a positive-pressure air-lock at the entrance to the dressings suite. From this time, *Pseudomonas* was effectively contained within the dressings suite during treatment of infected patients.

Surface-borne Pseudomonas aeruginosa

Press-plate cultures (Oxoid blood agar base No. 2) were mainly used to recover *Pseudomonas* from surfaces, and recoveries were made from many parts of the environment, including gowns and overshoes worn by staff. These led to recoveries being often made at considerable distances from confirmed sources. *Pseudomonas* recoveries from surfaces in infected patients' cubicles rapidly diminished after discharge of patients. In moist conditions, however, where growth of the organism was possible, it could become established as resident and create additional sources of infection.

Table 2. *Disinfection and bacteriological control of 73 saline-bath treatments*

Organisms	No. of times the listed organisms were grown		
	Before bath	During bath	After disinfection
Ps. aeruginosa	1	44	2*
Proteus spp.	0	30	3
Staph. aureus	1	25	0
Coliforms	0	15	1
Staph. albus	5	0	1
Enterococci	1	11	0
B. anthracoides	0	1	0
Strep. pyogenes	0	0	0
Strep. viridans	0	0	0
Diphtheroids	0	0	0

* Untrained staff used incorrect disinfection procedure on these two occasions.

When an infected patient was given treatment in the saline bath, of which there was only one in the unit, the inner bath surface was found to become heavily contaminated with *Pseudomonas* and other organisms, and thus became a potential source of infection. This is shown in Table 2, which also shows the effects of disinfection of the bath with 'Tego' (MHG) after treatment of infected patients. A routine system of disinfection and bacteriological control was applied for each bath treatment. The inner bath surface was first wiped with undiluted 'Tego' and washed with water. After saline had been added to the bath, the patient was immersed using a hydraulic hoist, and on completion of treatment the patient was removed and the dirty water drained off. The bath was then two-thirds filled with a 1/100 dilution of Tego into which the hydraulic hoist was immersed for not less than 30 min.; the bath was then washed with water. Swabs for bacteriology were taken from the inner bath surface before the first application of undiluted Tego, after the dirty water had been drained from the bath, and on completion of disinfection.

Resident Pseudomonas aeruginosa.

Persistence of *Pseudomonas* in a growing state provides new sources of infection and may lead to successive patients being infected. Soap dishes, denture and receiving bowls, water in flower vases, sink cloths, cleaning utensils, sinks and bath overflows and particularly sink traps are typical examples, and when established over a long period the original sources of contamination can no longer be traced. With so many additional sources of infection available it becomes difficult to study the distribution of *Pseudomonas* or determine how patients become infected. When such a source of infection is revealed, the pyocine types of *Pseudomonas* found may be those from past or present patients, from patients in other parts of the hospital (transmission by staff), or from contaminated materials, including hands which have been washed in contaminated sinks. The efficiency of removing *Pseudomonas* from hands by washing after contact with infected patients was tested, and invariably resulted in living *Pseudomonas* being deposited in the sink bowls and sink traps, with the attendant risk of survivals on the hands being transmitted to other patients. Residue of hand washing under these conditions provides a continuous build-up of resident *Pseudomonas* and considerably increases the risk of other patients becoming infected. Considering the number of times each day that hands are washed after contact with infected patients, it is not surprising that infection of severe burn wounds, although undesirable, is accepted as inevitable. It has been established over a 4-year-period that regular disinfection is an effective method of preventing resident *Pseudomonas* from becoming established. Sink traps were dosed on alternate days with 1 fluid oz. of undiluted Tego, which was also used to wipe the inner surface of the sink bowls. Cleansing utensils were completely immersed in a 1/40 dilution of Tego when not in use. Flower vases with water were not permitted in the unit during the first 3 years. Within a few weeks of their introduction during the fourth year *Pseudomonas* was recovered in a growing state from a vase, but not before it was established that dirty water from the vase had been ejected into several sinks in the unit. Further enquiries revealed that this vase had been placed in different patients' cubicles over a period of time. Examination of the contaminated sinks and sink traps did not show any living *Pseudomonas* and further confirmed the effectiveness of disinfection. In all instances, the methods of disinfection were applied after bacteriological examination had proved them to be effective.

Control of cross-infection

Air borne *Pseudomonas* mainly originates in dry infected necrotic tissue broken down into minute *Pseudomonas*-laden particles which are frequently shed into the air. It seemed that if these particles were confined to the wounds of infected patients, airborne contamination would be considerably reduced. Anti-bacterial creams (Aserbine, Gentamycin, Sulfamylon, etc.) were liberally applied to wound areas, which resulted in a dramatic reduction of *Pseudomonas* being shed from the patients into the air (Table 3). It was realized, however, that although the creams were effective as bacterial suppressive agents, living *Pseudomonas* was still present

Table 3. *Extract from records* (14-*day period only*) *of colony counts of* Pseudomonas *on cultures on one of the 'gravitation trees' in a cubicle occupied by an infected patient* (*this patient received topical cream treatment*)

Height above floor (ft.)	Colony counts of *Pseudomonas* on day: 3	4	5	6	7	8	9	10	11	12	13	14	15	16
9	0	0	0	0	0	0	0	0	0	0	0	0	0	0
8	0	0	0	0	0	0	0	0	0	0	0	0	0	0
7	0	0	0	0	0	0	0	0	1	0	0	0	0	0
6	0	0	0	0	0	0	0	0	0	0	0	0	0	0
5	0	1	0	0	0	0	0	0	0	0	0	0	1	0
4	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
Floor	0	1	0	0	0	0	0	0	0	0	0	0	0	0

Table 4. *Pyocine typing* 1966: *chain-like infections with two types of* Pseudomonas aeruginosa *affecting* 23 *patients, suggestive of cross-infection*

Patient	Admitted	Pyocine types of *Pseudomonas aeruginosa*						
E.L.	Dec. 65	—	F	—	—	—	—	—
C.L.	Jan. 66	—	—	O3	—	—	—	—
D.E.	Feb. 66	—	—	—	B1C	—	—	—
I.B.	Feb. 66	B1A	—	—	—	—	—	—
N.E.	Feb. 66	—	—	—	B1C	—	—	—
S.M.	Mar. 66	—	—	—	B1C	—	—	—
A.F.	Mar. 66	—	—	—	B1C	—	—	—
W.I.	Apr. 66	—	—	—	—	B1B	—	—
G.O.	Apr. 66	—	—	—	B1C	—	—	—
S.I.	Apr. 66	—	—	—	B1C	—	—	—
B.E.	Apr. 66	—	—	—	B1C	—	—	—
M.I.	Apr. 66	—	—	—	—	B1B	—	—
W.R.	May 66	—	—	—	B1C	—	—	—
H.O.	June 66	—	—	—	—	B1B	—	—
W.I.A.	June 66	—	—	—	—	B1B	—	—
S.H.	July 66	—	—	—	—	B1B	—	—
C.R.	Aug. 66	—	—	—	—	B1B	—	—
W.I.	Sept. 66	—	—	—	—	—	N5	—
R.I.	Sept. 66	—	—	—	—	B1B	—	—
H.E.	Sept. 66	—	—	—	—	B1B	—	—
W.E.	Sept. 66	—	—	O3	—	—	—	—
W.H.	Oct. 66	—	—	—	—	B1B	—	—
H.O.L.	Oct. 66	—	—	—	—	B1B	—	—
B.A.	Nov. 66	—	—	—	—	B1B	—	—
S.E.	Nov. 66	—	—	—	—	B1B	—	—
L.O.	Nov. 66	—	—	—	—	B1B	—	—
H.E.	Nov. 66	—	—	—	—	—	—	NT
B.U.	Nov. 66	—	—	—	—	B1B	—	—
S.H.	Dec. 66	—	—	—	—	B1B	—	—

https://doi.org/10.1017/S0022172400021410 Published online by Cambridge University Press

on infected wounds, and could be transmitted to other patients by direct contact.

Out of a total of 71 patients who were admitted into the unit during 1966, the wounds of 29 (41 %) were colonized with Pseudomonas, and 23 of these produced infections of two distinct pyocine types, which indicated that they were cross-infected by direct contact (Table 4).

Table 5. *Pyocine typing* 1967*: random scatter of pyocine types resulting from cross-infection counter measures*

Patient	Admitted	Pyocine types of *Pseudomonas aeruginosa*						
G.E.	Jan. 67	—	—	—	—	—	Q22	—
H.U.	Jan. 67	—	—	—	—	—	—	O25
W.E.	Jan. 67	—	—	—	—	—	—	O25
B.R.	Feb. 67	—	—	—	—	—	—	O25
M.I.	Feb. 67	—	—	—	B1B	—	—	—
F.I.	Feb. 67	—	—	—	—	—	Q22	—
C.L.	Feb. 67	—	—	—	—	NT	—	—
H.O.	Apr. 67	—	—	—	B1B	—	—	—
S.I.	May 67	—	P30	—	—	—	—	—
H.U.	May 67	—	—	—	—	NT	—	—
H.U.	June 67	B1A	—	—	—	—	—	—
A.P.	July 67	—	—	—	—	NT	—	—
C.O.	Aug. 67	—	—	—	—	NT	—	—
W.I.	Aug. 67	B1A	—	—	—	—	—	—
A.C.	Sept. 67	—	—	F	—	—	—	—
A.P.	Oct. 67	—	—	—	—	NT	—	—
T.H.	Nov. 67	—	—	—	—	NT	—	—
H.A.	Nov. 67	B1A	—	—	—	—	—	—
W.A.	Dec. 67	—	—	—	B1B	—	—	—
O.L.	Dec. 67	—	—	—	B1B	—	—	—

Elimination of cross-infection by direct contact was attempted by isolating infected patients to the north wing and uninfected patients to the south wing of the unit. All staff were similarly segregated so that contact between the two groups would not take place during any term of duty. During the subsequent 12-month period, and for the first time, patients with extensive burns passed through their treatment in the unit without any evidence of *Pseudomonas* infection; periods of treatment were from 80 to 120 days. Further confirmation of reduction of cross-infection was found when pyocine types from the year before were compared with those in the year after isolation of the two groups (Table 5). The chain-like pattern of infections during 1966 was completely broken after elimination of direct contact and was replaced by a random scatter of pyocine types. This freedom from cross-infection continued for the next 12 months and the *Pseudomonas* infections were reduced from 41 % in 1966 to 28 % (20 out of 72 admissions) in 1967. During the early part of 1968 the unit became short of staff and strict isolation of the two groups was not possible. Once again a chain pattern of infections involving several patients with a single pyocine type of *Pseudomonas* emerged, which was a clear indication of cross-infection. Only when more staff became available was the chain pattern of infections broken and replaced by a random scatter of pyocine types.

A further reduction of *Pseudomonas* infections was experienced during 1968. Out of a total of 73 patients treated in the unit only 15 (20%) became infected, and this, when considered with other evidence, confirmed that some control of cross-infection by direct contact had been achieved.

DISCUSSION

When patients with *Pseudomonas*-infected wounds are nursed in hospital this organism becomes established and can be recovered from many parts of the environment. Given an environment free from *Pseudomonas* at the outset, the admission of burns patients with infected wounds provides known sources of this organism and permits its environmental distribution to be studied. It seems likely therefore that the question of whether pseudomonas must become a resident or not could be answered. As a result of controlled disinfection, daily bacteriology failed to show resident *Pseudomonas* in any part of the Burns Centre, and after 3 years we concluded that the persistence of this organism was not inevitable. If it should become established it could be got rid of by daily disinfection. These studies have shown that *Pseudomonas* was capable of being airborne for prolonged periods and was being distributed in the air over wide areas from confirmed sources. The spread of *Pseudomonas* from patients' cubicles was controlled by using topical creams to prevent it becoming airborne, but during treatment of patients in the dressings suite and the operating theatre the most effective method of control was achieved by disinfectant aerosol mists followed by a surface-disinfectant wash. Although we undoubtedly experienced cross-infection during the first year, this was considerably reduced by isolation and segregation procedures, and our results seemed to show that persistence of *Pseudomonas* in a Burns Centre is not inevitable, that the main cause of cross-infection is by contact, and that control of cross-infection is possible.

During the four years 1966–9 inclusive the annual numbers of confirmed deaths from *Pseudomonas* septicaemia were 4, 1, 1 and 0 respectively.

I wish to record my profound appreciation to Professor J. W. McLeod for his constant interest and encouragement over the years. My sincere thanks are due to all those who have contributed so much to these studies: Mr T. L. Barclay, Dr J. A. D. Settle, the Sisters and staff of the Regional Burns Centre without whose cooperation this work would not have been possible, and Dr M. T. Parker for his help and advice on pyocine typing. I also have to acknowledge with grateful thanks the valuable assistance I have received from Mr F. Swift, Mrs M. Jordan and Mrs M. Smailes.

REFERENCES

Barclay, T. L. & Dexter, F. (1968). Infection and cross-infection in a new Burns Centre. *British Journal of Surgery* **55**, 197.

Darrell, J. H. & Wahba, A. H. (1964). Pyocine-typing of hospital strains of *Pseudomonas pyocyanea*. *Journal of Clinical Pathology* **17**, 236.

GILLIES, R. R. & GOVAN, J. R. W. (1966). Typing of *Pseudomonas pyocyanea* by pyocine production. *Journal of Pathology and Bacteriology* **91**, 339.

GOVAN, J. R. W. & GILLIES, R. R. (1969). Further studies in the pyocine typing of *Pseudomonas pyocyanea*. *Journal of Medical Microbiology* **2**, 17.

SHOOTER, R. A., WALKER, K. A., WILLIAMS, V. R., HORGAN, G. M., PARKER, M. T., ASHESHOV, E. H. & BULLIMORE, J. F. (1966). Faecal carriage of *Pseudomonas aeruginosa* in hospital patients. *Lancet* ii, 1331.