

Control of cross-infection in an intensive care unit

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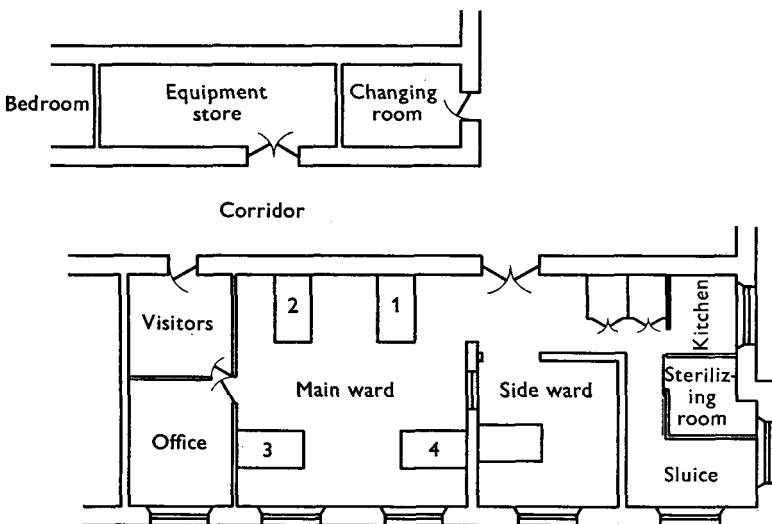
(Received 1 March 1969)

Considerable attention is currently being paid to the problem of controlling cross-infection in intensive care units. Patients requiring endotracheal intubation, tracheostomy or intermittent positive pressure ventilation (I.P.P.V.) are particularly susceptible to infection, often by *Pseudomonas aeruginosa* and organisms of the coliform/*Proteus* group. Many strains of these species are not antibiotic-sensitive, and their accumulation in a unit caring for severely ill patients presents a formidable hazard. This paper describes the routine measures we employ to prevent cross-infection, and records our experience of infections occurring during the first 13 months of our unit's existence.

NATURE OF THE UNIT

The Intensive Therapy Unit cares chiefly for patients in respiratory failure, unconscious patients, and those requiring continuous E.C.G. monitoring for coronary thrombosis and cardiac arrhythmia. The construction of the unit is shown in the figure (the numerals indicate the four beds in the main ward).

Walls and ceilings have smooth, painted surfaces; dust-gathering protuberances are reduced to a minimum, and windows are double-glazed. Air admitted to the



Plan of the Intensive Therapy Unit at the Royal Hospital, Sheffield.

main ward via bacterial filters is maintained at a positive pressure with respect to the corridor; air in the side ward, maintained at a pressure 5 lb./in.² higher than the main ward, finds its outlet through a loaded wall vent into the sluice and thence into the street. Welded-seam vinyl sheet flooring is used throughout the unit.

Most patients requiring I.P.P.V. are nursed on the unit's three Cape ventilators. A Watson 'Barnet' Mk. III ventilator is used for cases requiring a 'square-wave' form of ventilation. Humidification for patients with tracheostomies or endotracheal tubes *in situ* is provided by ultrasonic and cold air nebulizers.

METHODS

Measures designed to prevent cross-infection

After use by a patient, the internal components and exterior of the ventilators are cleaned with 1/200 alcoholic chlorhexidine; sterilization is not routinely attempted. Bacterial filters are not employed in the ventilator circuit, but we attempt to prevent initial contamination of the ventilators by daily autoclaving of the connector tubing. Moistened swabs are used to sample the bacterial flora of the tubing before sterilization. The exhaust of the ventilator, and walls, window-ledge and other surfaces in its vicinity, are sampled during ventilation. Humidifiers are filled with 1/5000 aqueous chlorhexidine (Phillips & Spencer, 1965) which is changed daily and tested for sterility weekly. Those not in use are sealed with sterile rubber bungs. Perspex tracheostomy cages are cleaned with alcoholic chlorhexidine then sterilized by boiling, and sterile tracheostomy sets are provided on Bowie trays. Sterile disposable forceps are used for changing tracheostomy dressings and inserting disposable suction catheters. Wall suction tubing is autoclaved daily in the main hospital autoclave; a small autoclave in the unit is used for bowls and loose instruments. Disposable bed-pans and urinals are macerated in a 'Clinimatic' disposal unit. Ceilings and walls are washed regularly, and bed areas are washed with 1% 'Stericol' (Izal Ltd.) after the discharge of grossly infected patients.

If the side ward contains an infected patient, the patient in bed 1 is potentially at greater risk of infection than the other patients when the door of the side ward is opened; bed 1 is therefore now used only when no other is available.

Infections occurring among the staff are investigated, and affected personnel suspended from duty until bacteriological cure is achieved.

Bacteriological methods

Sputum, urine and swabs from tracheostomies, other wounds and infective lesions are examined twice weekly throughout the patient's stay. Conventional methods are employed, except that culture in 10% CO₂ is preferred to aerobic culture for all specimens except urine. Swabs from equipment are cultured on blood agar, incubated aerobically for 4 days. Humidifier fluid is neutralized with Lecithin/Lubrol W broth and incubated at 37° C. for 4 days; the control organism is *Staphylococcus aureus*. Sensitivity tests to eleven antibiotics are performed on 'Oxoid' sensitivity-test agar plates flooded with a broth culture. Sulphonamide

sensitivity tests are performed on antagonistic-factor-free agar containing varying concentrations of sulphonamide; a light inoculum is employed. All strains of *Staph. aureus* are phage-typed (Blair & Williams, 1961).

Each potentially pathogenic species isolated from a specimen was considered to represent one 'infection' (though the term 'infection' is somewhat unsatisfactory, since colonization by potential pathogens did not always affect the patients' clinical course). Some patients had infections with the same species in more than one site; each site was considered a separate 'infection' only if the organisms belonged to demonstrably different strains. In Table 3 such infections are classified by the site in which they first appeared. Repeated isolation of the same strain from one site constituted one infection.

RESULTS

Clinical results

There were 207 admissions, representing 196 patients (nine being admitted on two occasions and one on three occasions). The patients fall into three groups: (I) Those requiring I.P.P.V.; (II) Patients, other than those in group I, requiring endotracheal intubation or tracheostomy; (III) Patients admitted for continuous monitoring. The numbers of patients in each of these three groups, with the numbers admitted from Casualty (i.e., from outside a hospital environment), or from the wards or other hospitals, are shown in Table 1; this also shows the incidence of infection, on admission and acquired in the unit, for each of these groups. It is seen that there is a considerable difference between the incidence of infection on admission in patients admitted from Casualty and that in patients

Table 1. *The incidence of infection among patients admitted from the Casualty Department, or from the wards or other hospitals, in relation to the type of treatment given*

	Total patients		Patients admitted from Casualty			Patients admitted from wards or other hospitals			
	No.	Died	Infected on admission		Infection acquired	No.	Infected on admission		Infection acquired
			No.				No.		
I	85	44	34	1	10 (29)*	51	15 (29)	24 (47)	
II	27	6	12	0	3 (25)	15	5 (33)	6 (40)	
III	95	13	48	0	1	47	4 (8)	9 (19)	
Total	207	63	94	1	14 (15)	113	24 (21)	39 (34)	

Group I. Patients requiring I.P.P.V. Group II. Patients other than those in group I, requiring endotracheal intubation or tracheostomy. Group III. Patients admitted for continuous monitoring. *Figures in parentheses indicate percentages.

transferred from the wards or from other hospitals. Those transferred from a hospital environment also show a greater tendency to acquire infection during their stay in the unit; this difference is not real, because 17 of the 34 patients in group I who were admitted from Casualty were cases of drug overdose, and were basically healthy before admission. Furthermore, many patients in group I admitted from the wards already had respiratory embarrassment or other

Table 2. Influence of I.P.P.V. on acquisition of infection

Diagnosis	Patients requiring I.P.P.V.				Patients requiring tracheostomy or endotracheal tube, but not I.P.P.V.				
	Total No.	No. with E.T.T.	No. with T.	No. with infection On admission	Total no.	No. with E.T.T.	No. with T.	No. with infection On admission	Total
Drug overdose	18	0	0	1	8	8	0	0	0
Intracranial haemorrhage, etc.	12	0	0	0	4	3	1	0	3
Respiratory inadequacy and post-op. complications	19	13	6	7	7	4	3	4	2
Totals	49	43	6	8	19	15	4	1	6
									(47.4%)
									(48.7%)

E.T.T. = Endotracheal tube. T. = Tracheostomy.

conditions which rendered them liable to infection. In groups II and III the proportion of patients admitted on account of primary lung disease or major trauma was much lower than in group I.

Table 2 is an attempt to assess the influence of I.P.P.V. on the acquisition of infection. For the three categories of patient considered (all of whom had been subjected to tracheostomy or endotracheal intubation), the incidence of infection was approximately the same whether I.P.P.V. had been used or not.

Table 3. *Bacteria responsible for infections*

Causative organism	No. of infections	Site of infection			
		Sputum	Tracheostomy	Wound swab	Urine
Coliforms	39	18	6	5	10
<i>Ps. aeruginosa</i>	21	11	7	2	1
<i>Proteus</i> spp.	9	5	1	1	2
<i>H. influenzae</i>	6	5	1	0	0
<i>Strep. pneumoniae</i>	3	3	0	0	0
<i>Staph. aureus</i>	21	10	4	7	0
<i>Strep. faecalis</i>	4	0	1	3	0
<i>Strep. pyogenes</i>	3	0	0	3	0
<i>Candida</i> spp.	14	3	11	0	0
<i>Clostridium welchii</i>	1	0	0	1	0
Totals	121	55	31	22	13

Bacteriological results

The bacteria responsible for the infections diagnosed during life are classified in Table 3. In addition, two infections were diagnosed bacteriologically at autopsy (one case of aspergillosis of the lung, and one of septicaemia caused by a Group A β -haemolytic streptococcus).

We use the term 'coliform' to include all lactose-fermenting Gram-negative bacilli. Precise biochemical identification and serological typing were not considered feasible as a routine, so the antibiotic sensitivity pattern was used as the epidemiological 'marker' for these strains. Twenty different patterns were detected of which four accounted for 19 of the 39 isolations. Most of the remaining types were isolated only once during the period, and types isolated more than once were widely spaced in their occurrence as a rule. On two occasions the same sensitivity-type was isolated almost simultaneously from two patients. In each case the strains involved were obtained from the patients' sputum immediately after their transfer from other wards; there was no evidence to suggest that cross-infection had occurred within the unit.

Table 3 shows that the incidence of infection with *Ps. aeruginosa* was comparatively low. Infections with this species were distributed evenly throughout the period. Precise information on the possibility of cross-infection is lacking, since pyocine typing was introduced only towards the end of this period, but one episode of probable cross-infection is considered below. Typing is now carried out on all strains, and it is our preliminary impression that little cross-infection occurs.

Six phage-types of *Staph. aureus* were encountered, and six strains were untypable. There was no evidence of cross-infection between patients.

Bacteriological tests on equipment

All samples of humidifier fluid were sterile, adequate neutralization of the chlorhexidine being demonstrated by profuse growth of the control organism.

Ventilator tubing was sampled on 68 occasions. The tracheal end and tracheostomy cage or Y-piece invariably yielded bacteria identical with those in the patient's sputum; organisms were isolated on six occasions from the distal end of the expiratory tubing, but never from any point further on in the expiratory circuit, and rarely from the inspiratory tubing. Three cases are of particular interest:

Patient A, a case of terminal true emphysema, was nursed on the same ventilator for 4 weeks, during which *Ps. aeruginosa*, *Staph. aureus* and a coliform organism were repeatedly isolated from sputum, tracheostomy wound and the proximal expiratory tubing, and on four occasions from the distal end of the expiratory tubing. These organisms were never isolated from the exhaust of the machine or from surfaces in its vicinity. After A's death, the ventilator was dismantled as far as possible. The bellows were found to be dry and clean internally; swabs taken from the interior of the bellows, and from various points in the internal circuit, proved sterile on culture.

Patient B was admitted in coma 3 days before A's death, into bed 2 (A was in bed 3). I.P.P.V. was begun with a ventilator which had not been used recently, but stored in a clean, dry condition. *Ps. aeruginosa* was soon isolated from the sputum and proximal and distal ends of both inspiratory and expiratory tubing, but not from the exhaust or the humidifier fluid. Patient B died 1 week after admission; bacteriological tests on the ventilator after his death yielded entirely negative findings.

Patient C, in acute renal failure, was then being nursed in the side ward on the ventilator just vacated by A, and her sputum became infected by *Ps. aeruginosa* of the same pyocine type as that isolated from B. After C's death, the internal parts of her ventilator were again found to be sterile. The strain of *Ps. aeruginosa* isolated from A was unfortunately not typed; it seems likely that A was the source of the infection in B and C, but it is impossible to conclude that the ventilators were concerned in its transmission.

DISCUSSION

It is difficult to compare the incidence of infection in our unit with that seen in other centres, since there is a striking lack of published information on this point. However, we consider our experience to be unusual in three respects. First, the majority of 'infections' in our unit have been associated with coliform bacilli rather than with *Ps. aeruginosa* or *Staph. aureus*. Secondly, we have not been able to demonstrate much evidence of cross-infection. Finally, in the very few cases where cross-infection may have occurred, we have never been able to incriminate the ventilators in its transmission.

Cross-infection has been a major problem in other units (see Campbell, Reid, Telfer & Fitch, 1967; Tinne, Gordon, Bairn & Mackey, 1967), though its incidence has not been clearly defined. The causative organism in most units has been *Ps. aeruginosa*. Where ventilators and suction equipment have acted as vectors of infection, the published reports either demonstrate grave deficiencies in basic hygiene (e.g., Sutter, Hurst, Grossmann & Calonje, 1966) or give no details of routine preventive measures before the outbreak (Tinne *et al.* 1967).

Various attempts have been made to sterilize ventilators after use, by means of ethylene oxide fumigation (Bishop, Robertson & Williams, 1964) or antibacterial aerosols (Judd *et al.* 1968; Meadows, Richardson, Fish & Williams, 1968; Spencer, Ridley, Eykyn & Achong, 1968). Other writers recommend prevention of contamination by means of bacterial filters; this seems a logical approach, but existing filters are not ideal.

Our frequent changing and autoclaving of connector-tubing prevents multiplication of Gram-negative bacilli, since moisture does not accumulate and dry conditions are unfavourable to these bacteria (Lowbury & Fox, 1953; Pettit & Lowbury, 1968). There is then insufficient accumulation of bacteria in the tubing to make contamination of the ventilator likely. The use of aqueous chlorhexidine for humidification has effectively prevented contamination of the humidifiers from retrograde spread along the inspiratory tubing. We consider that these methods have virtually excluded the possibility of cross-infection occurring via the ventilators in our unit. The main factor predisposing to respiratory infection in our patients appears to be the presence of an endotracheal tube or tracheostomy (especially the latter), and I.P.P.V. does not significantly alter its incidence.

The nature of the basic reservoir of infection is uncertain. Tinne *et al.* (1967), and Rountree & Beard (1968), emphasize the importance of environmental contamination by infected secretions. The patient with an infected tracheostomy is undoubtedly a particularly potent disseminator of organisms; however it is questionable whether their survival in the environment could be sufficient to account for the rather unusual situation found in this unit. The wide variety of coliform strains which we have encountered, and the sporadic nature of the infections, suggest that these infections may be endogenous, the bacteria being derived from the patient's own gut or upper respiratory tract.

It is not quite so easy to explain infections caused by *Ps. aeruginosa*. Most authors have not been able to demonstrate a high faecal carriage rate for this species, though Shooter *et al.* (1966) reported a carrier rate of 24% in patients admitted to a surgical ward. They noted that isolation was particularly frequent in patients with colostomies, and also that the organism was more commonly found in patients who had previously been exposed to various influences inherent in the hospital environment. It is likely that patients admitted to an intensive care unit might show a similarly increased carrier rate. Certainly the infrequency of this species in our unit and our inability in most cases to demonstrate any means of transmission between patients, suggest that the possibility of endogenous infection is worth investigating.

These problems can only be resolved by a more detailed knowledge of patients'

intestinal bacterial flora and the extent to which these bacteria can contaminate the environment. Investigations into these aspects are now in progress, but meanwhile we feel that the measures described in this report can successfully limit the incidence and spread of infection among these highly susceptible patients.

SUMMARY

In a survey undertaken in an intensive care unit, coliform bacilli were found to be responsible for most infections, *Pseudomonas aeruginosa* and *Staphylococcus aureus* being isolated much less frequently. Tracheostomy or endotracheal intubation predisposed to infection, but in our experience intermittent positive pressure ventilation did not significantly affect its incidence. Little cross-infection has occurred, and it has never been possible to incriminate the ventilators in its transmission.

We gratefully acknowledge the assistance we have received from colleagues in the Intensive Therapy Unit and the Department of Bacteriology during the course of this investigation.

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