group of similar organisms regularly found in tap outlets and elsewhere in hospital wards, as well as in brook water and soil (Olsen 1969).

*Survival and Multiplication*

Gram-positive cocci survive drying well, persist in dust and on dry surfaces, and are regularly found in the air of occupied rooms, but they do not multiply in the hospital environment. The Gram-negative organisms, on the other hand, suffer an immediate loss in numbers of 90–99% within a few minutes of drying (Lowbury & Fox 1953), though the survivors persist almost as well as do staphylococci. Thus, the number of Gram-negative organisms in the air and dust is usually only a tiny proportion of those present in the 'wet' sources from which they came. All the Gram-negative organisms survive well in moist places and the 'free-living' strains have simple nutritive requirements and a wide temperature-range for growth. They will therefore multiply at room temperature in fluids apparently devoid of organic material. Many of the enterobacter-serratia group and the fluorescent pseudomonads (but not *Ps. aeruginosa*) will also grow at refrigerator temperature (3–5°C). Some pseudomonads can use unlikely materials such as phenolic and quaternary ammonium compounds as sources of carbon and energy for growth.

Many Gram-negative bacteria are said to be exceptionally resistant to disinfectants, but in fact they are almost as easily killed by many 'conventional' disinfectants as are other non-sporing bacteria. Nevertheless, in hospitals these organisms are often found not only to survive but also to multiply in chemically disinfected equipment and in the disinfectants themselves. The disinfectants used in hospital are, unfortunately, often not 'conventional' and are sometimes used to treat objects that are almost impossible to disinfect by any chemical means. Places where disinfectants are ineffective are also usually places in which the Gram-negative survivors can multiply, so the consequences of incomplete destruction are much more serious than they are with Gram-positive organisms.

*Patterns of Infection*

There are three common situations in which sepsis due to Gram-negative bacilli occurs in hospital.

(1) *Common-source outbreaks:* These occur when an organism contaminates a piece of equipment or a fluid and is transferred by this means directly to a number of patients. In nearly every case the organism appears to have multiplied in the environmental reservoir and it is therefore the 'free-living' organisms that are responsible for most of these outbreaks. The organism may be injected into or inhaled by the patient, but in many cases, particularly in newborn infants and very debilitated patients, it appears to be sufficient for the organism to be ingested or deposited on the skin (*see* Bassett 1971).

(2) *Hyperendemic situations:* In certain departments of the hospital there are concentrations of patients who are all highly susceptible to infection at a particular site, e.g. burns wards and urological surgery departments. Here the infected patients provide a rich source of organisms; infection is spread from patient to patient by contact in the strains, especially of *Ps. aeruginosa*, proteus and klebsiella organisms, persist endemically for months or years and may cause high infection-rates (*see* Lowbury 1971).

(3) *Sporadic infections:* Many infections acquired in hospital, particularly with *Ps. aeruginosa*, are apparently sporadic, and those occurring in a particular ward are usually due to a variety of different types of the organism (Darrell & Wahba 1964). There is evidence that many of these are self-infections with organisms from the patient's own gut. Nevertheless, many of these organisms are of types seldom found in the gut of patients outside hospital (*see* Shooter 1971).

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## Common-source Outbreaks

Common-source outbreaks of sepsis due to Gram-negative organisms arise because of the persistence or multiplication of the organisms in items of equipment or in solutions used in hospitals. This paper reviews forty-three such outbreaks, selected from the published reports of the last twenty years to illustrate the many different

*Table 1*
**Outbreaks due to unappreciated risks**

| | *Route and source of infection* | *Organism* | *Patient* | *Illness* | *Authors* |
|---|---|---|---|---|---|
| | *A: Respiratory* | | | | |
| (1) | Suction apparatus | *Ps. aeruginosa* | Newborn | Meningitis, pneumonia | Bassett *et al.* (1965) |
| (2) | Suction apparatus | *Ps. aeruginosa* | Newborn | Respiratory | Rubbo *et al.* (1966) |
| (3) | Oxygen apparatus (Emerson bags) | *Ps. aeruginosa* | Newborn | Septicæmia, pneumonia | Fierer *et al.* (1967) |
| (4) | Ventilator (reservoir nebulizer) | *Serratia marcescens* | Adult | Respiratory, urinary, wound | Cabrera (1969) |
| (5) | Ventilator (humidifier and inspiratory tubing) | *Ps. aeruginosa* | Adult | Pneumonia | Phillips & Spencer (1965), Phillips (1967) |
| (6) | Tracheal catheters (holders contaminated) | *Ps. aeruginosa* | Adult | Pneumonia | Sutter *et al.* (1966) |
| | *B: Intravascular* | | | | |
| (7) | Catheterization (hand lotion) | *Klebsiella pneumoniæ* | Adult | Septicæmia | Morse *et al.* (1967) |
| (8) | Catheterization (tap aerator) | *Pseudomonas* sp. | Adult | Wound, arterial thrombosis | Cross *et al.* (1966) |
| | *C: Wound* | | | | |
| (9) | Orthopædic (plaster bucket, cellulose wadding) | *Ps. aeruginosa* | Adult | Wound sepsis | Sussman & Stevens (1960) |
| (10) | Craniotomy (shaving brush, failed skin disinfection) | *Ps. aeruginosa* | Adult | Meningitis | Ayliffe *et al.* (1965) |
| | *D: ? External* | | | | |
| (11) | ? leaking sink trap | *Flavobacterium meningosepticum* | Newborn | Meningitis | Cabrera & Davis (1961) |
| (12) | ? skin lotion | *Flavobacterium meningosepticum* | Newborn | Meningitis | Plotkin & McKitrick (1966) |
| (13) | ? saline | *Serratia marcescens* | Newborn | Umbilical sepsis | McCormack & Kunin (1966) |
| | *E: Oral* | | | | |
| (14) | Ice machine | *Ps. aeruginosa, Enterobacter cloacæ* | Adult | Pneumonia | Newsom (1968) |

sources and routes of infection that have been identified. The reported outbreaks probably represent only a very small part of the number that occurred. They may be grouped according to the following etiological factors: (1) unappreciated risks; (2) contaminated fluids given intravenously; (3) ineffective bacteriostatic agents used in medications; (4) unsuccessful chemical disinfection of equipment; (5) the spread of organisms by means of contaminated disinfectants.

*Unappreciated Risks*

Table 1 shows those outbreaks that occurred because a need for sterility or disinfection was not recognized.

Section A shows those connected with respiratory apparatus of various sorts. The first two outbreaks listed were of infections in premature babies. In each of these, the particular strain of *Pseudomonas aeruginosa* was traced to a suction device. In the first (Bassett *et al.* 1965), the sucker was so constructed that it could be neither heat-sterilized nor effectively cleaned and, being uncleanable, resisted chemical disinfection. The use of this sucker was abandoned. The suction apparatus in the second outbreak (Rubbo *et al.* 1966) was found suitable for modification and continued use, with a regular disinfection procedure. A report of infection spread in a nursery by suction apparatus was published ten years before the first of these outbreaks (Rubenstein & Fowler 1955), but this did not prevent the continuing manufacture and use of unsuitable equipment.

Fierer *et al.* (1967) traced an outbreak of *Ps. aeruginosa* infection (No. 3 in Table 1) to the Emerson bags on oxygen apparatus. These bags were not disinfected but were washed at a tap which was a source of the pseudomonas; residual water in the bags provided a very suitable environment for the organism.

Intermittent positive pressure ventilators gave rise to two outbreaks; one (No. 4) was due to a machine with a reservoir nebulizer contaminated with *Serratia marcescens* (Cabrera 1969): only removal of the nebulizer served to stop the spread of infection. The second (No. 5) came from a ventilator in which the humidifier and inspiratory tubing were contaminated with *Ps. aeruginosa* (Phillips & Spencer 1965, Phillips 1967). The danger of infection from humidifiers had been indicated some years earlier by McPherson (1958).

In outbreak No. 6 (Sutter *et al.* 1966), catheters used for tracheal suction in debilitated patients were kept in unsterile tap water at the bedside. Most of the containers were contaminated with *Ps. aeruginosa*, and containers were not always returned to the same patients after filling.

*Table 2*
**Outbreaks due to contaminated intravenous fluids**

| | *Fluid* | *Organism* | *Illness* | *Authors* |
|---|---|---|---|---|
| 15) | Blood | *Enterobacter* sp. | Transfusion reaction (endotoxic shock) | McEntegart (1956) |
| (16) | Human albumin | *Pseudomonas* sp. | Transfusion reaction (endotoxic shock) | Dykes (1962) |
| (17) | Glucose Ringer (muscle relaxant) | *Klebsiella pneumoniæ*, *Enterobacter cloacæ* | Transfusion reaction (endotoxic shock) | Sack (1970) |
| (18) | Glucose saline | *Serratia marcescens* | Meningitis, wound infection | Rabinowitz & Schiffrin (1952) |
| (19) | Various drugs, in intravenous anæsthesia | *Flavobacterium meningosepticum* | Transient bacteriæmia | Olsen (1967) |

*Table 3*
**Outbreaks due to ineffective bacteriostatic agents**

| | *Preparation* | *Agent* | *Organism* | *Illness* | *Authors* |
|---|---|---|---|---|---|
| (20) | Eye irrigation fluid | Methyl- and propyl-hydroxybenzoates (0·06%) | *Ps. aeruginosa* | Panophthalmitis, hypopyon | Ayliffe *et al.* (1966) |
| (21) | Lignocaine jelly (endotracheal tubes) | Methyl-hydroxybenzoate (0·02%) | *Ps. aeruginosa* | Respiratory | Phillips (1966) |
| (22) | Bronchodilator (in nebulizer) | Chlorbutanol (0·5%) | *Klebsiella pneumoniæ* | Pneumonia | Mertz *et al.* (1967) |
| (23) | Steroid cream | Chlorocresol (0·1%) | *Ps. aeruginosa* | Skin lesions | Noble & Savin (1966) |

In section B of Table 1 there are two outbreaks in which the organisms were introduced during intravascular catheterization. In No. 7 (Morse *et al.* 1967), *Klebsiella pneumoniæ*, identified as serotype 18, was recovered from 5 patients with septicæmia, and from a handcream dispenser. This led to a study of commercially-prepared handcreams and lotions (Morse & Schonbeck 1968) in which unopened bottles were found contaminated with pseudomonas, klebsiella, serratia and other Gram-negative rods, with counts reaching 1 million per ml. Outbreak No. 8 (Cross *et al.* 1966) resulted from the introduction of a pseudomonad into cardiac catheterization wounds. The organism evidently originated in a tap aerator, and so probably reached the patients on the hands of the staff.

In section C, there are two outbreaks in which infection was introduced into wounds. In No. 9, described by Sussman & Stevens (1960), removal of the infected plaster bucket did not stop the outbreak, but the removal of the second source by autoclaving the supplies of cellulose wadding was successful. Pseudomonads are normally thought of as contaminants of wet places, but clearly they may also occur in less typical sites. In outbreak No. 10 (Ayliffe *et al.* 1965) the shaving brush used pre-operatively appeared as the common source of infection and subsequent skin disinfection failed to remove the contamination. Two neurosurgical cases developed post-operative meningitis and 4 others developed wound infection.

In the two outbreaks of infection with *Flavobacterium meningosepticum*, in section D of Table 1, the organisms were not known to have been introduced or injected into the affected patients and it would appear that, in the newborn infant, external contact provided sufficient opportunity for the organism to colonize and cause disease. Asymptomatic nasal colonization was twice as common as overt disease in the outbreak described by Cabrera & Davis (1961) (No. 11). Organisms from a leaking sink trap contaminated some cleaning materials used in a premature baby unit and infections ceased after this source was removed. In the outbreak described by Plotkin and McKitrick (1966) (No. 12), contaminated saline was used to clean the babies' eyes. When this flavobacterium caused disease, it was usually fatal: 10 out of 14 of those infected from the leaking sink trap died.

Outbreak No. 13 consisted of umbilical infections due to *Serratia marcescens* (McCormack & Kunin 1966). Isolations of the organism were made from suction tubing and from plastic bottles containing saline. The latter were more directly relevant to the infections that occurred and the outbreak stopped when both sources had been eliminated.

In section E, which shows an example of infection by the oral route, only one outbreak appears (No. 14). In this outbreak, the way in which a machine providing ice for tracheostomy patients

to suck was plumbed allowed organisms from the drain to contaminate the ice (Newsom 1968).

*Contaminated Fluids Given Intravenously*

This group of outbreaks (Table 2) is small because a contaminated bottle of fluid for intravenous administration is unlikely to affect more than one patient. McEntegart (1956) described 2 fatal reactions to contaminated blood; both bottles contained the same organism. This was attributed to contamination of the plastic bottle caps, which were supplied in unsterile preserving fluid. Dykes (1962) described 2 fatal reactions to reconstituted human albumin. He found fourteen bottles contaminated with a pseudomonad which multiplied at refrigerator temperature to reach counts of over 1 million organisms per ml after seven days storage. Sack (1970) reported 5 non-fatal cases of endotoxic shock following intravenous anæsthesia. Only patients who had received a freshly prepared solution of succinylcholine chloride were affected. It seems clear that the diluent was contaminated before the drug was added. The suggested route of entry for the bacteria was a minute crack, such as occasionally occurs in bottles, which can admit organisms, particularly while the bottle is cooling. Rapid cooling or warming of bottles in unsterile water tends to increase this risk.

The repeated use over a period of time of an initially sterile solution may also lead to trouble and is generally undesirable. The outbreaks described by Rabinowitz & Schiffrin (1952) and by Olsen (1967) arose from this practice, when solutions were contaminated and then re-used. The faulty injection technique described by Olsen allowed sequential contamination of vials to occur.

*Ineffective Bacteriostatic Agents Used in Medications*

The incorporation of bacteriostatic agents in fluids that are to be re-used over a period of time has been associated with numerous outbreaks of infection, particularly of the eyes. An early report of a common-source ourtbeak of *Ps. aeruginosa* infections in eyes was that of Garretson & Cosgrove (1927). Since then there have been numerous references to similar events. Theodore & Feinstein (1952) wrote that almost every pseudomonas infection encountered was due to contamination of eye solutions. Outbreak No. 20 in Table 3 (Ayliffe *et al.* 1966) was of particular importance because its impact caused the revision of the section on eye preparations in the British Pharmaceutical Codex (Supplement 1966), by which the hydroxybenzoates were withdrawn, and convinced many people that single-dose dispensing of eye drops was desirable. In the 1966 B P C supplement the alternative antibacterial agents listed include benzalkonium chloride 0·01 % and chlorhexidine 0·01 %; neither of these would seem an ideal agent against pseudomonads. Outbreak No. 21 was also due to the failure of a hydroxybenzoate to prevent the growth of *Ps. aeruginosa* (Phillips 1966).

The presence of *Klebsiella pneumoniæ* in a bronchodilator solution containing chlorbutanol was associated with the deaths of 5 patients on intermittent positive pressure ventilation (No. 22, Mertz *et al.* 1967). In the laboratory, the organisms did not survive exposure to 0·5 % chlorbutanol for 48 hours, but laboratory cultures do not necessarily show organisms in their most resistant state.

The contamination of a steroid cream (No. 23, Noble & Savin 1966) was attributed to the partitioning of the chlorocresol between the oily and aqueous phases of the cream, which appeared to leave only a tenth of the intended concentration in the aqueous phase.

*Unsuccessful Chemical Disinfection of Equipment*

The sources listed in Table 4 are very similar to those in Table 1, the difference being that in these outbreaks the need for disinfection had been appreciated by the users of the equipment but the means of disinfection failed.

The failures to eliminate *Ps. aeruginosa* by means of chlorxylenol (No. 24, Rogers 1960) and chlorhexidine (No. 25, Tinne *et al.* 1967; No. 29, Rogers 1960) might be attributed to the choice of disinfectants. However, more potent agents failed to disinfect the ventilators concerned in outbreaks Nos. 26, 27 and 28 (Cartwright & Hargrave 1970, Grieble *et al.* 1970, Ringrose *et al.* 1968) and in outbreak No. 25 the failure of chlorhexidine was followed by the unsuccessful use of ethylene oxide. The problem is evidently one of ensuring contact between disinfectant and organism; formalin failed to disinfect in outbreak No. 26 because of the accumulation of scale in the humidifier.

In section B of Table 4 the same problems are again illustrated, with cardiac catheters (Schickman *et al.* 1959), heart-lung machines (Keown *et al.* 1957, Linde & Heins 1960) and hæmodialysis equipment (Roques *et al.* 1969) involved in outbreaks of bacteriæmia, septicæmia and endocarditis. The resistance of *Ps. aeruginosa* to benzalkonium chloride contributed to three of the outbreaks but, in No. 33, ethylene oxide was used unsuccessfully and heat sterilization was required

*Table 4*
**Outbreaks due to unsuccessful chemical disinfection**

| | *Route and source of infection* | *Disinfectant* | *Organism* | *Illness* | *Authors* |
|---|---|---|---|---|---|
| | *A: Respiratory* | | | | |
| (24) | Suction apparatus | Chloroxylenol | *Ps. aeruginosa* | Respiratory | Rogers (1960) |
| (25) | Ventilator (tubing and bag) | Chlorhexidine | *Ps. aeruginosa* | Respiratory | Tinne *et al.* (1967) |
| (26) | Ventilator (humidifier) | Formalin | *Ps. aeruginosa* | Not stated | Cartwright & Hargrave (1970) |
| (27) | Ventilator (fine particle humidifier) | (*a*) Phenolic (*b*) 0·25% acetic acid | *Ps. aeruginosa* | Respiratory | Grieble *et al.* (1970) |
| (28) | Ventilator (ultrasonic nebulizer) | Glutaraldehyde | *Serratia marcescens* | Respiratory | Ringrose *et al.* (1968) |
| (29) | Mucus catheters (holders contaminated) | Chlorhexidine | *Ps. aeruginosa* | Respiratory | Rogers (1960) |
| | *B: Intravascular* | | | | |
| (30) | Cardiac catheters and adaptors | Benzalkonium chloride | *Ps. aeruginosa* | Bacteriæmia | Schickman *et al.* (1959) |
| (31) | Extracorporeal circulation (oxygenator on heart-lung machine) | Benzalkonium chloride | *Ps. aeruginosa* | Septicæmia | Keown *et al.* (1957) |
| (32) | Extracorporeal circulation (plastic parts of hæmodialyser) | Benzalkonium chloride | *Ps. aeruginosa* | Septicæmia | Roques *et al.* (1969) |
| (33) | Extracorporeal circulation (heart-lung machine) | Ethylene oxide | *Achromobacter* sp. | Endocarditis | Linde & Heins (1960) |
| | *C: Wound* | | | | |
| (34) | Bigelow's extractor | Chlorhexidine | *Ps. aeruginosa* | Urinary | Moore & Foreman (1966) |
| | *D: ? External* | | | | |
| (35) | Incubators for newborn | Benzalkonium chloride | 'Paracolobactrum aerogenoides' | Meningitis | Rance *et al.* (1962) |
| (36) | Incubators for newborn (sleeves) | Hypochlorite | *Ps. aeruginosa* | Umbilical, eye sepsis | Barrie (1965) |

to eliminate the organism from the heart-lung machine.

The outbreak of post-operative urinary infection (No. 34, Moore & Foreman 1966) involved 39 patients in six months: heat sterilization of the Bigelow's extractor stopped the outbreak.

Outbreak No. 35 (Rance *et al.* 1962) was caused by 'Paracolobactrum aerogenoides'. From the description of the organism given this might have been a nonpigmented serratia. Eleven premature babies died of meningitis or hæmorrhagic encephalitis during a 4-year period. Thorough cleaning and disinfection of the incubators was achieved only when the manufacturers dismantled the machines.

Outbreak No. 36 (Barrie 1965) was a simple example of an effective disinfectant failing to reach parts of a contaminated surface. Routine disinfection of the incubator sleeves *in situ* failed, but removal and immersion of the sleeves in hypochlorite was effective.

## *Organisms Spread by Disinfectants*

A disinfectant may not only fail to destroy bacteria on equipment but may also be the vehicle in which organisms are spread to the patient. Table 5 summarizes outbreaks in which the evidence suggests that the infecting organisms multiplied in the disinfectants.

In three of these outbreaks cotton swabs were kept in benzalkonium chloride and the patients suffered from septicæmia following intravenous procedures. The adsorption of benzalkonium chloride to cotton fibres was shown by Kundsin & Walter (1957) but the practice of keeping swabs in this disinfectant continued, as outbreaks Nos. 37, 38 and 39 clearly show (Plotkin & Austrian 1958, Lee & Fialkow 1961, Malizia *et al.* 1960). Benzalkonium chloride is in any event a very poor disinfectant for use against pseudomonads. In outbreak No. 39 the enterobacter was also isolated from a stock bottle of the disinfectant yet, in the

*Table 5*
**Outbreaks due to organisms spread by disinfectants**

| | *Disinfectant* | *Organism* | *Illness* | *Authors* |
|---|---|---|---|---|
| (37) | Benzalkonium chloride 1 in 1,000 ● | *Pseudomonas* sp. | Septicaemia | Plotkin & Austrian (1958) |
| (38) | Benzalkonium chloride 1 in 1,000 ● | 'Pseudomonas-achromobacter' | Septicæmia | Lee & Fialkow (1961) |
| (39) | Benzalkonium chloride 1 in 750 ● | *Enterobacter cloacæ* | Septicæmia | Malizia *et al.* (1960) |
| (40) | Printol 1 in 100 and/or chlorhexidine 1 in 5,000 | *Pseudomonas* sp. | Wound sepsis | Cragg & Andrews (1969) |
| (41) | Chlorhexidine 1 in 5,000 | *Ps. cepacia* | Urinary sepsis | Mitchell & Hayward (1966) |
| (42) | Chlorhexidine + cetrimide (Savlon H. C. 1 in 30) | *Ps. cepacia* | Wound sepsis | Bassett *et al.* (1970) |
| (43) | Detergicide (quaternary) | *Ps. cepacia* | Urinary sepsis | Hardy *et al.* (1970) |

● In contact with cotton swabs

laboratory experiment, failed to survive in that solution unless cotton swabs were present.

The survival of a pseudomonad in a phenolic disinfectant (No. 40, Cragg & Andrews 1969) illustrates that such disinfectants may have a rather narrow and unpredictable range of activity against particular strains of Gram-negative bacteria.

*Ps. cepacia* (otherwise *Ps. multivorans*, *Ps. kingii* or E.O.-1) is highly resistant both to certain disinfectants and to most antibiotics. The contaminated chlorhexidine in outbreak No. 41 (Mitchell & Hayward 1966) caused a series of post-operative urinary infections when it was used to disinfect a bladder-irrigation reservoir. The inclusion of a contaminated quaternary-ammonium compound in a commercially-prepared catheter kit caused the outbreak (No. 43) reported by Hardy *et al.* (1970). Counts of up to 100,000 organisms per ml were found in samples of the disinfectant-detergent solution in the kits (Mackel 1970).

In outbreak No. 42, *Ps. cepacia* multiplied in 1 in 30 Savlon Hospital Concentrate – the highest recommended concentration – and led to a series of post-operative wound infections (Bassett *et al.* 1970). The minimum inhibitory concentration of Savlon in broth for a laboratory culture of the organism was 1 in 320, but after adaptation to Savlon the organism would grow in a 1 in 30 dilution of Savlon in distilled water, but not in the same dilution in tap water. The pseudomonad was traced back to the water supply of the hospital and surrounding district. Savlon was subsequently issued only in small volume containers as an autoclaved solution. No further infection occurred.

*Discussion*

From the difficulties that are encountered in the disinfection of hospital equipment it is evident that bacteriological advice is needed at the design stage of such equipment. Three basic principles are offered here:

(1) Only when it is essential to use heat-sensitive materials for non-disposable items should the idea of chemical disinfection be considered.
(2) Reliance on chemical disinfection is impossible if the surfaces to be disinfected are not regular or are inaccessible for cleaning.
(3) The more elaborate the procedure necessary to disinfect a machine, the less suitable the machine is for use in a busy and understaffed hospital.

It is clear from the outbreaks reviewed that the publication of a report on a common-source outbreak has limited value in preventing the repetition of the same mistake in other places. Bacteriologists perform some service in the detection of the sources of outbreaks, and in reporting them, but they can perform far greater service by searching for potential sources of infection in their hospitals and eliminating them before an outbreak can occur. Much good advice has been published, for example, on the disinfection of ventilators (Fisher & Kyi Kyi 1969) and on the in-use testing of disinfectants (Kelsey & Maurer 1966). The need now is to ensure that existing knowledge is applied to all medical, nursing and domestic procedures in hospital.

How much good would be done by the prevention of common-source outbreaks? In the outbreaks reviewed, 587 patients were needlessly infected, and there were 66 deaths, many directly attributable to the infection. Even in the absence of a recognized outbreak, dramatic reductions in Gram-negative infections have followed the elimination of external sources of infection (Lowbury 1951, Kresky 1964).

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## Control of Infection with Gram-negative Bacteria in Patients at Special Risk

*Pseudomonas aeruginosa* has been described as a 'bacterial hyena' which attacks the debilitated but leaves the healthy individual alone. Severe infection with these organisms, as one might expect, is largely confined to hospitals, The increased proportion of severe hospital infections with these and with some other Gram-negative bacilli reported in recent years (e.g. Finland 1960, Barrett *et al.* 1968) can be seen as evidence not only of our deficiencies in controlling such infections but also, perhaps, of an increased proportion of 'special risk' patients in hospitals.

'Special risk' patients are those who are more likely than most to develop clinical infection caused not only by recognized pathogens but also by bacteria, fungi or other microorganisms which are harmless or relatively harmless towards healthy subjects. Patients with leukæmia, hypogammaglobulinæmia and uncontrolled diabetes are lacking in general resistance, as are patients undergoing treatment with cytotoxic drugs, immunosuppressants and steroids. In addition to the lack of resistance to invasion, some high-risk patients provide greater opportunities for contamination and colonization; for example, any traumatic or operation wound exposes potentially susceptible tissues, but when the meninges or the chambers of the eye or the endocardium or the urinary tract are exposed, the risks of clinical infection are enhanced because of the exceptionally poor antimicrobial resistance in these tissues; extensive burns are particularly susceptible to contamination and the presence of a layer of moist slough allows heavy colonization by bacteria, in particular by *Ps. aeruginosa*, *Proteus* spp. and other Gram-negative bacilli (Colebrook *et al.* 1960). I shall illustrate my discussion of this subject by examples from the treatment of burns which my colleagues and I have studied for many years in Birmingham.

### *Sources and Modes of Transfer*

*Ps. aeruginosa* has, in our experience, been one of the less common bacteria of normal fæces; most of the patients whose burns subsequently acquired *Ps. aeruginosa* have not shown the presence of these organisms in rectal swabs or fæcal specimens, when these were taken on admission. On the other hand, *Ps. aeruginosa* of the same phage types as those acquired by the burns have usually been present beforehand in the burns of other patients in the same ward, but were usually not present in the other ward of the Burns Unit (Davis *et al.* 1969); from which we infer that *Ps. aeruginosa* in burned patients is usually acquired by cross-infection rather than by self-infection with fæcal organisms.

The transfer of *Ps. aeruginosa* seems to occur mainly by contact, and especially by manual contact. The evidence supporting this view is as follows: (1) *Ps. aeruginosa* is commonly found on the hands of nurses working in the Burns Unit (Lowbury & Fox 1954); and (2) a controlled trial, which is still in progress, shows successful control of *Ps. aeruginosa* by nursing patients in a plastic isolator which protects them against manual contamination (because of the obligatory use of gloves in handling the patient), but not against airborne or fomites-transmitted contamination; by contrast, the isolation of patients behind air curtains, which greatly reduce the access of airborne bacteria, has given no protection to burns against *Ps. aeruginosa* (Lowbury, Babb & Ford 1971). However, airborne transfer of *Ps. aeruginosa* can occur, e.g. from heavily contaminated dress-