

**An outbreak of
Pseudomonas aeruginosa (*pyocyanea*) infection in a Premature
Baby Unit, with observations on the intestinal carriage
of *Pseudomonas aeruginosa* in the newborn**

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INTRODUCTION

Pseudomonas aeruginosa (*pyocyanea*) infection may occur sporadically, or in outbreaks. It is well recognized that the newborn, and in particular premature babies, are very susceptible to infection with *Ps. aeruginosa* which may vary from symptomless carriage of the organism to a fulminating septicaemia.

This paper describes an outbreak of infection in a Premature Baby Unit, with predominantly intestinal and respiratory signs, which was caused by a single strain of *Ps. aeruginosa*. The outbreak lasted for 16 months because of the persistent carriage of *Ps. aeruginosa* in the faeces of the babies, many of whom were symptom free.

BACTERIOLOGICAL METHODS

Faeces were cultured on 0.03% cetrimide nutrient agar (Lowbury & Collins, 1955) and incubated for 18–24 hr. at 37° C. No enrichment media were used.

In environmental investigations, surfaces of equipment were swabbed with cotton-wool swabs which had been moistened with nutrient broth. The swabs were incubated in tubes of broth for 24 hr. at 37° C. and then subcultured on cetrimide agar and incubated for a further 24 hr. at 37° C. Cetrimide agar settle plates were exposed for 1–2 hr. and then incubated for 24 hr. at 37° C. Liquid samples were diluted 1/10 with nutrient broth and incubated for 72 hr. at 37° C. Liquid disinfectants were also tested by the method described by Kelsey & Maurer (1966).

Ps. aeruginosa was identified by the morphology and colonial appearance, the formation of pigment, the characteristic smell and a positive oxidase reaction (Kovacs, 1956). Non-pigmented forms were not found. If there was doubt about the identification, subculture was made, to enhance pigment formation, on Wahba & Darrell's (1965) modification of Sierra's (1957) medium, which was incubated for 24 hr. at 37° C.

Bacteriophage typing of selected strains was carried out at the Cross-Infection Reference Laboratory of the Public Health Laboratory Service.

THE PREMATURE BABY UNIT

Most of the premature babies were delivered in the labour wards or operating theatre of the Maternity Department, and were transferred to the Premature Baby Unit immediately after birth for an average of 3 weeks. Healthy full-term babies went to the maternity wards with their mothers.

For the first 9 months of the investigation the Premature Baby Unit had accommodation for 14 babies; the premises were very overcrowded and unsatisfactory, and there were no isolation facilities. From the end of January 1966, during the latter part of the outbreak, the Premature Baby Unit was transferred to a newly designed modern ward with accommodation for 24 babies, which was divided into cubicles.

In the early weeks of the outbreak the infants' feeds were prepared in the Premature Baby Unit; later the feeds were prepared and terminally sterilized in a central milk kitchen in a different part of the building.

Before use, the inner and outer surfaces of the premature babies' incubators were cleaned with 2.0% Printol (a clear soluble phenolic fluid); the interior of the incubators was then treated with formaldehyde gas. Bacteriological checks of the incubators showed that the disinfection was effective.

THE OUTBREAK

Between 22 April 1965 and 6 August 1966, 418 babies were admitted to the Premature Baby Unit. The birth weight of nearly all the babies was less than 2500 g. (5½ lb.). *Ps. aeruginosa* was cultured from the faeces of 99 babies; cultures from 62 of these, taken at random at different times during the outbreak, were typed and with one exception they had the phage-typing pattern 7/16/21/44/68/F8/109/119x/352/1214/M4. There were seven deaths due to *Ps. aeruginosa* infection (Table 1). Infections of nose (2), throat (4), eye (2), ear (2), vagina (2), skin (2), due to *Ps. aeruginosa* also occurred during this period.

As a routine, faeces from all babies are examined bacteriologically every week. The unit had been remarkably free from infection during the year before the investigation. Early in April 1965, *Ps. aeruginosa* was isolated more and more often from the babies' faeces (although selective cetrimide medium was not in use at that time). The significance of this finding was not appreciated until later in the month, when infection in three babies became clinically apparent, one baby developing a skin infection, another a nasal infection and the third a throat infection, which were all due to *Ps. aeruginosa*. At that time, it was suspected that the source of this infection was either an aspirator in the labour ward, which was used for the resuscitation of newborn babies and was heavily contaminated with *Ps. aeruginosa*, or a member of the Premature Baby Unit staff who had a chronic otitis externa, due to *Ps. aeruginosa*. However, in both instances, phage typing showed these strains to be different from the epidemic strain, and the original source of the infection was not discovered.

Table 2 summarizes the positive findings of bacteriological examination of the equipment in the labour wards, maternity operating theatre and Premature Baby

Unit. A total of 210 swabs was taken from different sources, including medicines, hand lotions, soap, distilled water, saline solutions, incubators, babies' feeds and bottles. *Ps. aeruginosa* was isolated on 23 occasions. In spite of the continued faecal

Table 1. *Deaths due to Pseudomonas aeruginosa infection*

Case	Date of birth	Sex	Birth weight (g.)	Age at death (days)	Clinical findings	Bacteriological findings
1	20. v. 65	F, twin	1930	12	Septicaemia	Faeces †Ep Blood culture †Ep <i>Post mortem</i> Meninges †Ep Pericardium *
2	28. vi. 65	M	1680	10	Respiratory distress syndrome, septicaemia	Throat * <i>Post mortem</i> Pericardium * Bronchi * Meninges †Ep
3	11. vii. 65	F	1445	11	Septicaemia	Faeces †Ep <i>Post mortem</i> Meninges †Ep Pericardium * Bronchi *
4	8. i. 66	M	1810	10	Abdominal distension, ileus, laparotomy, gangrene of transverse colon, septicaemia	Blood culture †Ep Mouth * Faeces * Peritoneum *
5	9. iii. 66	M, twin	1560	11	Respiratory distress syndrome, abdominal distension, septicaemia	Faeces †Ep <i>Post mortem</i> Bronchi * Small intestine * Meninges †Ep
6	25. iii. 66	F	1470	50	Respiratory distress syndrome, diarrhoea with blood, septicaemia	Faeces †Ep
7	15. iv. 66	M, twin	1100	13	Respiratory distress syndrome, diarrhoea, septicaemia	Faeces †Ep

† Ep = epidemic strain of *Ps. aeruginosa*.
* = *Ps. aeruginosa* cultured, but not phage typed.

excretion of *Ps. aeruginosa* during the outbreak the epidemic strain was only isolated twice—from a small mop on 12 October 1965, and from a nail brush on 14 October 1965—in the Premature Baby Unit. Both mop and nail brush were immersed in 2.0% 'Printol' when sampled. Settle plates failed to show evidence of airborne infection.

In view of the work of Wilson, Nelson, Phillips & Boak (1961), a special

investigation was made of the tap aerators and the outlet of the wash-basins and the sinks. *Ps. aeruginosa* was not isolated from the aerators or taps, but was present in all 36 of the basin and sink outlets tested. The epidemic strain was not found. Each ward had its own predominant basin and sink outlet strain of *Ps. aeruginosa*. Although we could find no evidence to suggest a relationship between the *Ps. aeruginosa* in the basins and sinks and human infection, we agree with Wilson *et al.* that this is a potential source of infection. There is no effective way of sterilizing these outlets and there is a need, in hospitals, for an outlet that can be easily disinfected and made safe.

Table 2. *Sources of Pseudomonas aeruginosa from the environment (excluding basins and sinks)*

Maternity operating theatre	2. vi. 65	Rubber tubing in sluice	7/21/68/119x/M4
	18. viii. 65	Floor	N/T
	15. x. 65	Nail brush	N/T
	8. iii. 66	Mop	N/T
Labour ward	20. iv. 65	Aspirator	31
	31. v. 65	Aspirator	7/31/73/119x
	12. vi. 65	Aspirator	7/31/73/119x
	13. viii. 65	Bucket	31
	13. viii. 65	Bucket	31
Premature Baby Unit	14. i. 66	Bucket	N/T
	12. x. 65	Mop in 'Printol'	7/16/21/44/68/F8/109/119x/352/1214/M4*
	12. x. 65	Soapy water in bucket	N/T
	12. x. 65	Mops in 'Printol'	7/53+
	14. x. 65	Nail brush in 'Printol'	7/16/21/44/68/F8/109/119x/352/1214/M4*
	14. i. 66	Bowl	N/T
	14. i. 66	Bucket	N/T
	14. i. 66	Mop	N/T
	14. i. 66	Water on floor	68
	14. i. 66	Bucket containing 'Printol'	N/T
Maternity ward	12. x. 65	Aspirator	7/31/73
	12. x. 65	Nail brush	N/T
	14. x. 65	Bucket	31
	17. x. 65	Bottle brush	68+

* = Epidemic strain. N/T = Non-typable.

Examination of faeces

Because of the failure to find a source or reservoir of infection in the environment of the ward or operating theatre, nose and throat swabs and specimens of faeces were obtained from the staff, and the faeces of all babies, both full term and premature, were examined. *Ps. aeruginosa* was not isolated from the staff.

Premature babies

Figure 1 shows the results of a weekly survey of the faeces of the babies in the Premature Baby Unit. Most of those babies who acquired *Ps. aeruginosa* did so during the first 2 weeks of life.

In order to determine the duration of faecal excretion of *Ps. aeruginosa*, 10 premature babies were followed up after they had left hospital, and samples of their faeces were examined at weekly intervals. When, for administrative reasons, this investigation was stopped, five of the babies were still excreting *Ps. aeruginosa* in their faeces 2 months after they were first found to be faecal carriers.

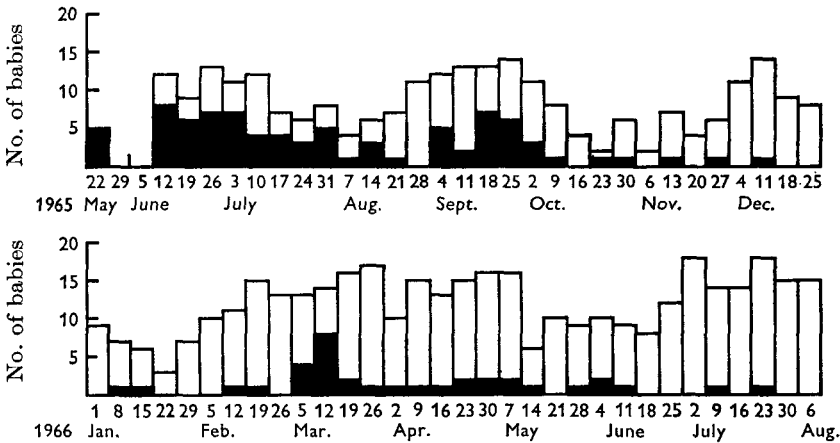


Fig. 1. Weekly isolations of *Pseudomonas aeruginosa* from faeces of babies in the Premature Baby Unit. ■ = *Ps. aeruginosa* isolated from faeces; □ = *Ps. aeruginosa* not isolated from faeces

Table 3. Monthly examination of faeces of full-term babies in maternity wards

	1965							1966			
	June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	April
<i>Ps. aeruginosa</i> absent	33	33	38	23	39	15	33	34	—	31	18
<i>Ps. aeruginosa</i> present	34	4	11	0	0	1	1	0	—	1	0
Epidemic strain of <i>Ps. aeruginosa</i>	20/21	4/4	5/5	—	—	1/1	1/1	—	—	1/1	—

Thirty-three strains of *Ps. aeruginosa* were typed; of these, 32 were the epidemic strain.

Full-term babies

Table 3 shows that, on 2 June 1965, 34/67 normal full-term babies in the Maternity Department wards were excreting *Ps. aeruginosa* in their faeces, many of them in large numbers. Twenty-one strains from these babies were phage typed; 20 belonged to the epidemic strain. All these babies were well and had no symptoms of infection, except one baby who had mild diarrhoea. Monthly bacteriological examination showed a reduction in the faecal carrier rate of these babies which may have been due to improvement in hygiene after the recognition of the infection.

Reservoir of infection and means of spread

Because *Ps. aeruginosa* was not found in the environment, we considered that the babies' faeces were the main reservoir that maintained the infection, for the faeces and nappies were always a prolific source of the organism. The infection was probably spread from baby to baby by contact, by way of the nurses' hands.

DISCUSSION

Many outbreaks of neonatal *Ps. aeruginosa* infection, mainly affecting premature babies, have been reported from the Continent and from the U.S.A., but there have been comparatively few reports from this country. The subject has been reviewed by Alison (1961) and by Paul & Marget (1964).

The infection may affect various parts of the body, such as the respiratory tract, skin, eyes and meninges. Kwantes (1960) described a small outbreak in which middle ear infection predominated, with symptomless intestinal carriage of *Ps. aeruginosa*. The intestinal tract is most commonly affected and outbreaks characterized by gastro-intestinal manifestations, such as feeding disturbances, abdominal distension, diarrhoea, intestinal ileus, haemorrhage, mucosal ulceration and intestinal perforation, have been reported (Schaffer & Oppenheimer, 1948; Bielicka & Dzieniszewska, 1953; Brádač, Procházková & Sedlák, 1954; Neter & Weintraub, 1955; Verger & Bentégeat, 1955; Eggers & Wöckel, 1958; v. Gavallér, 1958; Jacobs, 1964; Bassett, Thompson & Page, 1965).

Ps. aeruginosa is not a usual commensal organism of the intestinal tract in infants. McFarlan, Crone & Tee (1949 and personal communication) failed to find it in the faeces of 385 babies from two maternity units; it has been isolated many times from healthy newborn babies in other hospitals, although sometimes it has caused severe illness. In the outbreak described by Bielicka & Dzieniszewska, 70 babies had severe toxic enteritis; these authors and Linde, Koditz & Kittlick (1962) have postulated a secondary infective factor—possibly a virus or unidentified bacterium—which would enable *Ps. aeruginosa* to act as an intestinal pathogen. Different strains of *Ps. aeruginosa* may vary in pathogenicity, which might account for the variation in severity of different outbreaks. In an outbreak quoted by Verger & Bentégeat (1955), eight babies in a premature baby unit had severe diarrhoea and six of them died from the infection. In the present outbreak, although different non-epidemic strains of *Ps. aeruginosa* were isolated from equipment, wash basins and sink outlets, they were never found to colonize the babies' intestines (or to cause any other infection). This might indicate a difference in the ability of different strains to infect the intestine (with or without symptoms) and that the epidemic strain had an affinity for the intestine. The maturity of the baby might also influence the course of the infection. Linde *et al.* (1962) observed that the incidence of *symptomless* intestinal carriage increased with the age of the baby. In our investigations we found that 34/67 (51 %) full-term babies in the maternity unit were excreting *Ps. aeruginosa* in their faeces; with one exception these babies were symptom free. It was only in the Premature Baby Unit that this organism

behaved as a pathogen. An epidemic of infection was occurring in full-term babies, but because of their greater resistance to infection they were not ill.

Why should *Ps. aeruginosa* be so much more pathogenic to premature than to full-term and older babies, and why is infection becoming more frequent? The following suggestions have been made:

(1) Premature babies may be more susceptible because they lack antibodies. Gitlin, Rosen & Michael (1963) showed that newborn babies were deficient in the bactericidin to Gram-negative intestinal bacilli; the transfer of plasma gamma-globulin fractions across the placenta is selective and 19S gamma-globulin, which includes the gram negative bacillus bactericidin, does not cross the placental barrier and is absent from the plasma of the newborn. The experiments of Jones & Lowbury (1965) who protected mice from *Ps. aeruginosa* septicaemia by passive immunisation with human serum containing antibody to *Ps. aeruginosa*, show the ability of humoral antibodies to control the infection.

(2) Pseudomonas infection in children has become progressively more common since antibiotics were introduced about 15 years ago (Asay & Koch, 1960). The increase of infections has been attributed to the use of antibiotics which suppress the antibiotic-sensitive commensal organisms and favour the selection of resistant species such as *Ps. aeruginosa*. In the early stages of the outbreak, many of the premature babies were having prophylactic treatment with penicillin and streptomycin, which may have made them more susceptible to infection.

(3) Hoffman & Finberg (1955) suggested that the high humidity in the babies' incubators favoured the multiplication of *Ps. aeruginosa*.

(4) The incidence of *Ps. aeruginosa* infection may still be rising because of the survival of more premature babies with modern methods of treatment and intensive care.

The next point to consider is the source or reservoir of the organism. In other studies various sources have been incriminated such as polluted milk (Hunter & Ensign, 1947); contaminated suction apparatus (Bassett *et al.* 1965; Rubbo, Gardner & Franklin, 1966) and contaminated babies' incubators (Barrie, 1965). Verger & Bentégeat (1955) give evidence that the origin of a severe outbreak of infection in their Centre for Premature Babies was a baby who came from another hospital and developed a severe generalized skin infection due to *Ps. aeruginosa* soon after admission to the Centre. In at least three previous outbreaks, the commonest source of *Ps. aeruginosa* was the babies' faeces. Bielicka & Dzieniszewska found 125/170 (73 %) babies were excreting *Ps. aeruginosa* in the faeces and 98 were symptom-free; in Neter & Weintraub's study 71/143 (50 %) of the babies had *Ps. aeruginosa* in their faeces and most of them were healthy; *Ps. aeruginosa* was cultured from the faeces of 17/21 babies in Verger & Bentégeat's epidemic. The reservoir of infection in the present outbreak was also the babies' faeces, the most probable path of infection being by way of the nurses' hands—the organisms not always being removed by soap and water—to infect another baby during feeding, possibly by contamination of the teats (Florman & Schifrin, 1950; McLeod, 1958; Primavesi, 1964).

There are striking similarities between this outbreak in premature babies and

an epidemic of *Ps. aeruginosa* septicaemia in irradiated mice and rats, described by Wensinck, Van Bekkum & Renaud (1957) in which infection was caused by contamination of drinking devices by faecal excreters of *Ps. aeruginosa*; the epidemic was controlled by elimination of the faecal carriers and sterilization of the drinking bottles.

The type of outbreak described in this paper is difficult to control. The staff should be fully aware of the possible means of spread of the infection and should carry out the every-day routine preventive procedures with meticulous care. This is particularly important because of the unreliable action of liquid disinfectants on *Ps. aeruginosa*. The P.H.L.S. committee report (1965) on the use of liquid disinfectants in hospitals, advises that physical methods (heat) be used whenever possible, because of the uncertain action of these disinfectants. Kelsey & Maurer (1966) showed how inadequate these disinfectants may be in general use; in different hospitals, vegetative organisms were recovered from buckets and jars which contained various phenolic disinfectants. Our findings also expose these drawbacks, which are all the more obvious when pseudomonads—which are more resistant to disinfectants than most vegetative organisms—are present. Brushes and mops should not be used, unless they are disposable or can be sterilized (by heat) after every use. If the staff rely only on disinfectants, they may wrongly assume that materials are sterile.

Ideally, the following measures are recommended to control the spread of infection:

- (1) The babies should be nursed with isolation precautions in separate cubicles.
- (2) Nurses who change nappies should not feed babies.
- (3) Dirty nappies should be changed with extreme care and put directly into disposable polythene bags.
- (4) Disposable gloves should be worn whenever the baby is handled, because it is difficult to ensure that the hands are completely free of *Ps. aeruginosa* after contamination, owing to the resistance of *Ps. aeruginosa* to soap and water and to disinfectants.
- (5) To exclude *Ps. aeruginosa*, regular bacteriological screening tests should be made on the faeces of all premature babies, using a selective culture medium.

These procedures are time-consuming, and shortage of staff may make it difficult for them to be properly carried out.

Because so many of the infected babies are free of symptoms, the results of measures to control infection are not clinically apparent, and it may be difficult to keep the staff enthusiastic about the strict discipline which is necessary. The gravity of the situation, where *Ps. aeruginosa* is spreading silently through a premature baby unit, may not be appreciated until serious infection and deaths occur. 'Il faut redouter le pseudomonas'—pseudomonas must be feared (Alison, 1961)—and everyone should be fully aware of these often hidden but potentially dangerous infections of premature babies in hospitals.

SUMMARY

An outbreak of infection in a Premature Baby Unit due to a single strain of *Pseudomonas aeruginosa* is described. The outbreak lasted for 16 months, during which this organism was isolated from the faeces of 99/418 premature babies, many of them being symptomless carriers. There were seven deaths due to the infection. The source of the epidemic was not found. The main reservoir of infection was the babies' faeces. Though many full-term babies were symptomless carriers of the epidemic strain of *Ps. aeruginosa*, they were not ill.

Measures for control of the outbreak, and the aetiology of neonatal intestinal infection with *Ps. aeruginosa* are discussed.

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