

Rotavirus in Newborn Nurseries: Negative Results From Honolulu and the New Hebrides

ANDREW G. DEAN, M.D., M.P.H., DONALD K. BOWDEN, M.B., B.S., M.R.C.P., DAVID EASA, M.D., SORRELL H. WAXMAN, M.D., PAMELA COURTNEY, R.N. and KATHLEEN A. POON, M.D.

• *A survey of 100 newborn infants in 2 newborn nurseries in Honolulu and 36 newborns in a hospital in the New Hebrides failed to reveal rotavirus infection, despite its presence in the surrounding community.*

Rotavirus infection in the neonatal period appears to be endemic in some nurseries in England and Australia, but absent from others in England, Australia, the U.S., and the New Hebrides. The infection is often asymptomatic in neonates. Rotavirus infection in the older infant (6 to 24 months) usually produces gastroenteritis and is common in both industrialized and non-industrialized countries. Whether prior neonatal infection with rotavirus is beneficial or detrimental to the older child is unknown. Longitudinal studies of rotavirus infections of children beginning at birth would be required to answer this question, and to explore the reasons for the uneven distribution of rotavirus among various newborn nurseries.

Rotaviruses are the most common cause of acute gastroenteritis in young children; over 50% of infants have been infected by the age of 2 years.¹ These viruses are prevalent during the winter months in the temperature zones and throughout the year in the tropics.² In children after the neonatal period, the presence of

rotavirus has a high correlation with symptoms—fever, vomiting, diarrhea and dehydration^{3,4}—although adult contacts of cases often have asymptomatic infections.⁵

In neonates, asymptomatic infection of a third or more of individuals appears to be common. In some newborn nurseries, infection seems to be endemic,^{6,7,8,9,10} but studies in other nurseries in the U.S.¹¹ and Britain¹² have not detected rotaviruses.

The present study was performed to ascertain the incidence of rotavirus infections in nurseries in a U.S. city—Honolulu—and in a less developed area—the New Hebrides.

Methods

Stools were collected from 2- to 18-day old neonates in nurseries of 2 different hospitals in Honolulu and from 3- to 5-day old neonates in a nursery in the New Hebrides from January to April, 1978.

The stools were diluted 1:5 with water, shaken with glass beads, centrifuged at 2100 r.p.m., filtered through 0.45 micron pore size filter, and centrifuged again at 35,000 r.p.m. (100,000G) for 90 minutes. The pellet was suspended in a few drops of water, applied to a 300 mesh Formvar-coated grid and stained with 3% phosphotungstic acid. At least 4 squares of each grid were examined at 30,000 magnification with a Zeiss EMU S-2 electron microscope. Some stools were re-examined, using the method of Murphy et al.⁶ and by direct (uncentrifuged) application of the specimens to the grids. All 3 techniques were tried initially on stools containing rotavirus and gave almost identical results.

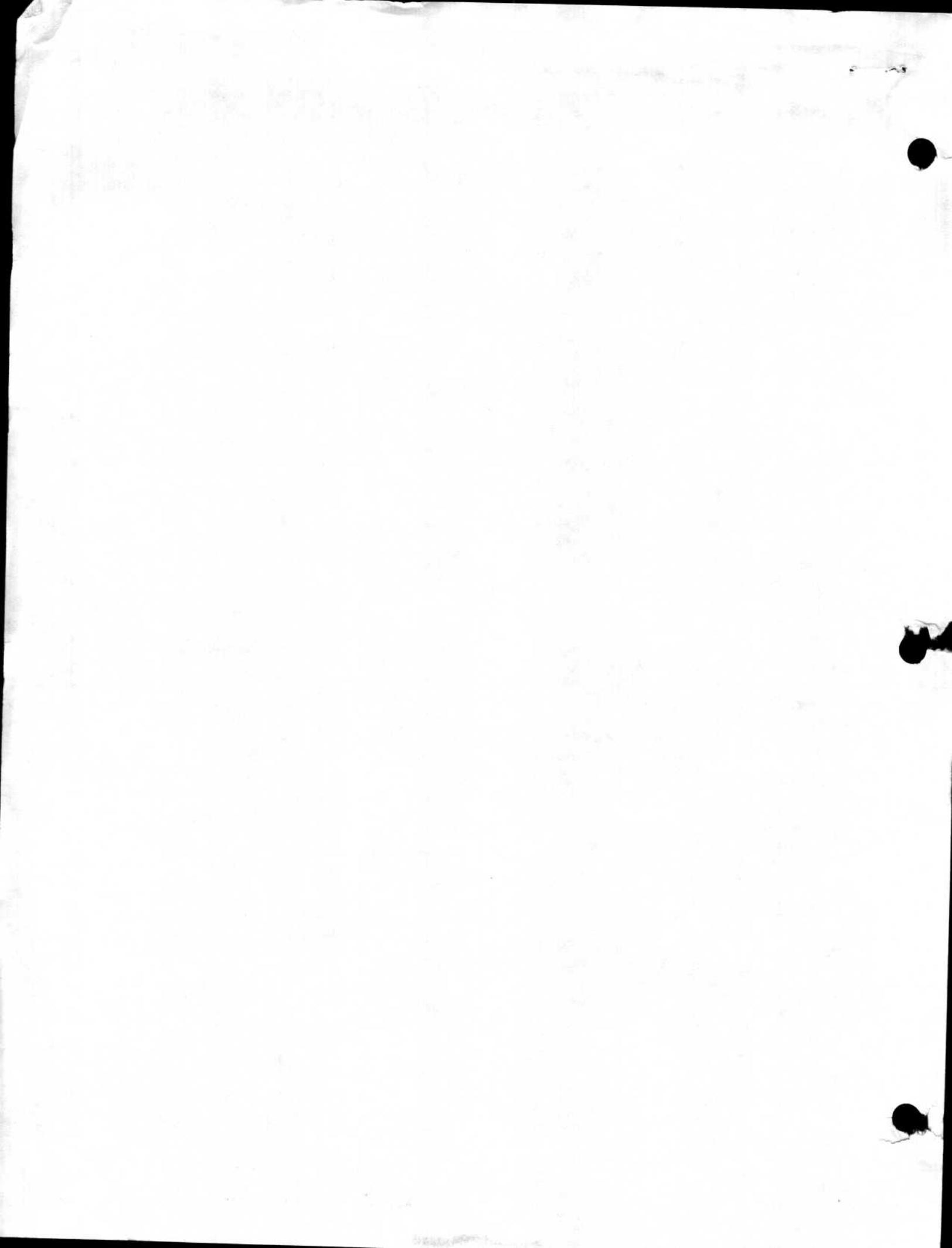
From the Department of Pediatrics, University of Hawaii Medical School (Drs. Easa and Waxman); Kapiolani-Children's Medical Center (Drs. Easa, Waxman, and Ms. Courtney); Kaiser Medical Center (Dr. Poon); Pacific Center for Geographic Disease Research (Dr. Dean) Honolulu, Hawaii; and the British Medical Service, Port Vila Hospital (Dr. Bowden) New Hebrides.

Reprint requests to Dr. Dean, who is now the State Epidemiologist, Minnesota Department of Health, 717 Delaware Street S.E., Minneapolis, Minnesota 55440.

This article was presented in part at the International Conference on Infant Nutrition and Diarrhoeal Disease, November 1979, at Kuala Lumpur, Malaysia.

This work was partially supported by contract NIAID #NO1-A102228 of the National Institutes of Health.

Accepted for publication February, 1980.



Results

Stools of 100 neonates from Honolulu and 36 neonates from the New Hebrides were examined. No rotavirus was found. During this same period, rotavirus was found in 8 of 34 older children with acute viral gastroenteritis in Honolulu and 10 of 16 children with gastroenteritis in the New Hebrides by the same techniques.

Discussion

There have been few studies of rotavirus infection in neonates in the United States. Our data and the data of Steinhoff and Gerber¹¹ indicate that rotavirus infection is not a "normal" occurrence in nurseries in upstate New York, Honolulu and the New Hebrides. Other studies⁶⁻¹⁰ indicate that rotavirus infection and gastroenteritis are a common finding in nurseries in England and Australia. In England, when nurseries in one hospital were monitored over a 1 year span, rotavirus infection rates varied from about 15 to 50%, depending on the season.⁷ Murphy et al. in Australia⁶ studied 6 different neonatal units, 5 of which had a continuing infection rate of 39 to 65%. Rotavirus was absent from the remaining nursery, but possible reasons for this were not given.

Differences in viral detection techniques or in age of the infants do not appear to explain our negative results. The stools of the Honolulu and the New Hebrides neonates were re-examined using the technique of Murphy et al. Several techniques were equally effective in detecting rotavirus in older children. The Honolulu neonates were 2 to 18 days of age (average 3.6 days) when their stools were examined. Murphy et al.⁶ found that by 2 days of age, their neonates had an infection rate of 37% and that most of the infected neonates were excreting rotavirus by 3 days of age. A prospective study⁸ from Australia showed that neonates had the onset of rotavirus excretion from 2 to 13 days of age.

Rotavirus was found in 25% of older children with gastroenteritis tested in Honolulu and in 60% of children with gastroenteritis tested in the New Hebrides during the period of this study. It does not appear that the absence of virus from the nurseries simply reflected conditions in the surrounding community. In 2 other negative studies^{1,12} rotavirus infection was also present in both the hospital and the community during the

period studied.

Varying nursery techniques might be responsible for the presence of endemic rotavirus. Bishop et al.⁹ found diarrhea to be common in nursery babies but unusual in those "rooming in" with their mothers. The Rochester study involved mostly infants in intensive and special care nurseries, in which special washing and growing techniques were observed. The Honolulu and New Hebrides nurseries contained mainly full-term newborns. One of the Honolulu hospitals practiced cohort isolation, with the infants regularly sent to their mothers for feeding. In the other hospital, rooming-in was encouraged with visiting limited to family members. In both hospitals, nursery personnel spent time in both the nurseries and in the maternity wings but were not used in surgical or acute care wings.

The immunological protection of breast milk may be a possible reason for a low incidence of neonatal gastroenteritis due to rotavirus infection. In one report,¹⁰ rotavirus excretion was less frequent in breast-fed babies. Symptoms were present in only 8% of those infected, but were more frequent in the bottle-fed group. In our study, 50% of the Honolulu neonates and almost all those in the New Hebrides were breast fed. The absence of rotavirus did not allow a comparison of rates in breast- and bottle-fed infants. In Honolulu, only disposable, commercially-prepared nipples and bottles containing standardized formula or water were used. In the New Hebrides, reusable bottles and nipples were used for water and formulas that were prepared in a central area.

Presumably an asymptomatic infection with rotavirus in the neonatal period protects a child against subsequent rotavirus disease of the same serotype, although second infection with a different serotype has been described.¹³ The reasons why infection is asymptomatic in a large proportion of neonates may offer valuable clues for vaccine development. The contributions of maternal antibody titer, differences among rotavirus strains, the dose and route of infection, and the response of intestinal mucosal cells at different ages remain to be defined. Long-term studies of newborns with and without neonatal infection, and of nurseries with and without endemic rotavirus would provide much information about this important human pathogen.

REFERENCES

1. Blacklow NR, Echeverria P, and Smith DH: Serological studies with reovirus-like enteritis agent. *Infection and Immunity* 15:63 (June) 1976.
2. Hieber JP, Shelton S, Nelson JD, et al: Comparison of Human Rotavirus Disease in Tropical and Temperate Settings. *Am J Dis Child* 132:853, 1978.
3. Rodriguez WJ, Kim HW, Arrobbio JO, et al: Clinical Features of Acute Gastroenteritis Associated With Human Reovirus-Like Agent in Infants and Young Children. *J Pediatr* 91:188, 1977.
4. Tallet S, MacKenzie C, Middleton P, et al: Clinical laboratory and epidemiological features of a viral gastroenteritis in infants and children. *Pediatr* 60:217, 1977.
5. Kim HW, Brandt CD, Kapikian AZ, et al: Human reovirus-like agent infection. *JAMA* 238:404, 1977.
6. Murphy AM, Albrey MB, Crewe EB: Rotavirus infection of neonates. *Lancet* 2:1149, 1977.
7. Chrystie IL, Totterdell BM, Banatvala JE: Asymptomatic endemic rotavirus infection in the newborn. *Lancet* 1:1176, 1978.
8. Cameron DJS, Bishop RF, Veenstra AA, et al: Pattern of shedding of two noncultivable viruses in stools of newborn babies. *J Med Virol* 2:7, 1978.
9. Bishop RF, Cameron DJS, Veenstra AA, et al: Diarrhea and rotavirus infection associated with differing regimens for post-natal care of newborn babies. *J Clin Microbiol* 9:525, 1979.
10. Totterdell BM, Chrystie IL, Banatvala JE: Rotavirus infections in a maternity unit. *Arch Dis Child* 51:924, 1976.
11. Steinhoff MC, Gerber MA: Rotavirus infection of neonates. *Lancet* 1:775, 1978.
12. Appleton H, Buckley M, Robertson MH, et al: A search for faecal viruses in new-born and other infants. *J Hyg Camb* (1978) 81:279.
13. Fonteyne J, Zissis G, and Lambert JP: Recurrent rotavirus gastroenteritis. *Lancet* 1:983, 1978.

