Group B streptococcal serotypes correlated with maternal parity and carrier sites

We were interested in the observation of Islam¹ that primigravidae and second gravidae were almost exclusively colonised by group B streptococci (GBS) of types I, R, X and non-typable isolates with a conspicuous absence of type III strains. In a study based at the Royal Berkshire Hospital, Reading and undertaken between July 1979 and February 1980 we found a much more even distribution of serotypes (Table 1).

This study will be reported fully elsewhere, but, in brief, low vaginal and anal swabs were taken from 30 mothers whose babies had been found to be colonised by GBS (group I) and from a control group of 50 mothers whose babies were not colonised by this organism (group II). After being transferred to the laboratory in a selective transport medium, the swabs were cultured on an enrichment medium and then subcultured on Columbia blood agar supplemented with defibrinated horse blood (10% vol/vol), nalidixic acid 15 mg/l and neomycin 30 mg/l. The isolates were serologically typed using modified Lancefield acid-extracted antigens, serotyped by precipitation tests in gel and phage-typed.² The mothers and babies had further upper respiratory, anal and low vaginal swabs taken at home at monthly intervals for the next 12 weeks.

Twenty-eight (93%) mothers from group I and 12 (24%) mothers from group II were found to carry GBS at some time during the study period. In contrast to Islam's findings we did not find that any serotype favoured any particular site (Table 2).

Anal, urethral and upper respiratory swabs were also taken from fathers on one occasion during the follow-up period. Fourteen of 28 (50%) of fathers of babies who had been colonised by GBS at birth (group I) and 11 of 45 (25%) of fathers of uncolonised babies (group II) were GBS carriers. In all cases, the strains isolated from the father's urethra were indistinguishable both serologically and by phage type from their consort's isolates. One husband carried a serotype III in his nose in addition to the "family strain" isolated from the anal swab and carried by the rest of the family (serotype Ia). Two other fathers had a strain in the anus which was different to their family strain.

In common with Sanderson *et al*³ reporting in the same January issue of this Journal, we found that the anorectal

Table 1 Group B streptococcal distributions correlated with parity

Parity	n	Serotype distribution*				
		I	II	III	NT	R or X
Para 1	17	7	4	5	1	0
Para 2	16	6	7 ^a	2	1	1
Para 3	5	2	1	2	1 ^b	0
Para 4	1	0	0	0	1	0

*Serotype I comprises Ia, Ib, Ic; II comprises II, IIR, II/Ic; III comprises III, IIIR.

^a = one subject colonised by type Ib and IIR

^b = one subject colonised by type III and NT

NT = non-typable.

Table 2 Site of culture compared with serotype in 40 women (see text) during 12 weeks post-partum

Site	Serotype distribution*					Total
	I	II	III	NT	R or X	
Low vaginal swab	32 (22%)	26 (18%)	17 (12%)	8 (5%)	2	146
Anus	32 (22%)	22 (15%)	20 (14%)	6 (4%)	2	143
Throat	4	2	4	0	0	108

*Serotype I comprises Ia, Ib, Ic; II comprises II, IIR, II/Ic; III comprises III and IIIR.

NT = non-typable.

region was the commonest site of colonisation in babies, mothers and fathers. We feel that the high urethral carriage among fathers of babies who became colonised by GBS may have important epidemiological implications, but we are unable to confirm the effect of parity.

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Role of the Schwartzmann reaction in necrotising enterocolitis

In the paper by Gray and her colleagues,¹ comments on the role of the Schwartzmann reaction in necrotising enterocolitis are

made, together with the assertion that there was "never any clinical evidence to support" the theory that this reaction may underlie enterocolitis. In the paper by Fraser and myself² we comment on a child whose colostomy was observed during a circulatory collapse which was followed by the clinical picture of necrotising enterocolitis and where changes suggesting a Schwartzmann reaction were found. This, in fact, lead to our subsequent experiments reproducing colitis of this type by the Schwartzmann reaction in the rabbit.

The study by the Aberdeen group is particularly valuable in that immunofluorescence studies have been carried out. Studies of this type will enable the pathogenesis of the syndrome to be determined and any pathologist seeing this lesion would be advised to freeze some of the tissue from the affected gut.

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