TITLE

EPIDEMIOLOGY OF GROUP B STREPTOCOCCUS (GBS) CARRIAGE IN MOTHERS AND THEIR NEONATES AT KENYATTA NATIONAL HOSPITAL.

BY

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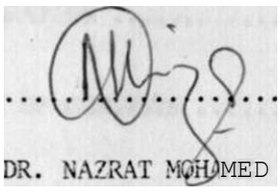
DECLARATION'

I certify that, this dissertation is m> own original work and has not been presented for a degree in any other University.

Signed 

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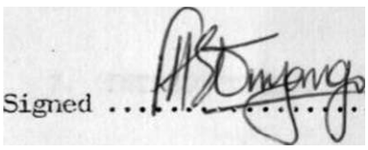
Signed 

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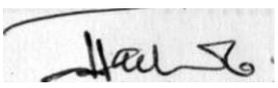
Signed 

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I 1ST 01 TAIilFS

LIST OF ABBREVIATIONS

KNH	Kenyatta National Hospital
GBS	Group B Beta Haemolytic Streptococci
SGA	Small for gestational age
AGA	Appropriate for gestational age
LGA	Large for gestational age
SVD	Spontaneous vertex delivery
C/S	Caeserian section
V/E	Vacuum extraction
g	Grams
SBA	Sheep blood agar
LBW	Low birth weight

Sunmary

Two hundred mothers and their newborn babies were examined for the presence of Group B streptococcus (GBS), at Kenyatta National Hospital (KNH), during a three month period, between May and August 1985-

Ten of the two hundred mothers were found to be positive for GBS, giving a vaginal colonization rate of *5%.* Of the two hundred babies examined, twenty nine" were positive for GBS giving a carriage rate of 14* - 60jS of the positive mothers had babies who were also positive, while only 12.1/S of the GBS negative mothers had positive babies, thus indicating that the chances of a baby acquiring GBS were significantly higher if the mother was vaginally positive for GBS.

There was no association between GBS colonization and maternal age, parity, contraceptive use or obstetrical complications. No association was found between GBS colonization of the babies and the infants sex, gestational age or birth weight. However, when GBS disease was considered, though numbers were small, the low birth weight (LBW) babies were mainly affected.

The attack rate for GBS disease was found to be 6.9 per 100 colonized babies. The death rate was higher in the GBS colonized babies, however the difference was not statistically significant.

GBS was found to be sensitive to penicillin, cloxacillin. ampicillin and erythromycin, but resistant to streptomycin.

Introduction

Groups B streptococcus (GBS), taxonomically known as Streptococcus agalactiae has been causally linked to human disease since 1938, and is now the leading cause of menigitis during the first two months of life in several geographical regions (1, 2, 3)- From 1940 to 1970, though sporadic cases of GBS disease were reported to occur in both parturients and neonates, coliform organisms predominated as the major aetiologic agents of neonatal septicaemia and meningitis (1, 4)• In 1958, Nyhan and Fousek reported the first cases of GBS neonatal meningitis (5) and in 1964 Eickhoff stressed its importance as a human pathogen (1, 6, 7)• Since then GBS has become a frequent agent associated with serious infection among neonates and young infants (3, 4, 8).

Reasons for this upsurge in the incidence of invasive disease due to GBS remain obscure (4). To try and explain this, it has been hypothesised that use of contraceptives may alter vaginal bacterial flora, increasing its likelihood of colonization with GBS. This situation leads to greater colonization of the skin and mucous membrane of neonates in utero or during delivery, resulting in disease in some of them (4, 9). There is no evidence to prove or disprove this hypothesis. However it is unlikely that the upsurge is due to changes in the resistance pattern of these bacteria since GBS is still highly susceptible to most commonly used antibacterial agents (4).

Streptococci were classified into group; by Lancefield in 1933 (10, 11). She characterized two polysaccharide antigens from the GBS. The group specific B polysaccharide common to all strains and the type specific polysaccharide that distinguishes four serotypes la, lb, II and III. These type specific polysaccharides are believed to be capsular and are distinct from each other and from the group specific substance (]0, 11. 12). An additional serotype Ic was defined in 1969 by Wilkinson and Lngon (13).

JVo distinct clinical syndromes, the early onset septicaemic and the late-onset nieningitic types of infection have been defined with GBS infection in infants.

The early-onset type is a severe foi-m of illness with a high mortality of 40 to 80* (1). It usually occurs before 10 days of age with an overwhelming majority (over 90^) having their symptoms during the first 48 hours of life (8, 12). This early onset type of illness is associated with a high incidence of maternal obstetrical complications, especially premature onset of labour and prolonged rupture of membranes (8, 14, 15)- A large majority of infants with early onset infection are of low birth weight (15, 16). The illness is characterized by progressive respiratory distress with cyanosis leading to respiratory failure and peripheral vascular collapse (l). It is clinically and radi©graphically difficult to differentiate prospectively between neonates with earlj' onset GBS pulmonary disease and those with respiratory distress syndrome (16, 17).

Acquisition of GBS by neonates with early onset- infection is either vertical from the maternal genital tract during passage through the birth canal or ascending infection in utero (l, 15). The serotypes of GBS isolated from the neonates with early onset infection are identical to those isolated from the genital tract of their mothers. Colonization of the infants depends on the extent of maternal colonization* the more heavily colonized a mother is the greater the chance of her infant becoming colonised, and at more sites (lS).

Reports of cervicovaginal colonization rates with GBS among parturients vary between *2%* to *35%*, while that of infants age 1 - 36 hours range from *6%* to *35%* (15, 10, 20, 21, 22). Isolation of GBS from urethral cultures of sexual partners of vaginally colonized women occurs frequently, at an approximate rate of *50%* (1, 4- 81 and may therefore serve as an important reservoir of their re—infection or re-colonization. Of neonates born to rr others cf vhom vaginal colonization is detected at delivery, approximately *70%* to *75%* will have GBS isolated from their skin or .tucous r.^libraries during the first 4S hour's of]ife. Though

the attack rate of neonatal infection is low (1 - 7 P^r 1000 live births), the high mortality makes this a serious illness (15).

The **serotype** of **GBS** isolated commonly in the early onset infection is type la (l, 6). However, when early onset infection is complicated with meningitis (as occurs in of cases), greater than 80% of the isolates belong to serotype III (2, 8).

The late-onset type of illness usually occurs after 10 days of age (1, 2). Maternal obstetrical complications are infrequently associated and the patients are usually not as severely ill at the time of diagnosis as those with early onset illness. .It carries a comparatively low mortality rate of approximately 20 - 40%. The exact mode of transmission of the organisms to the infant is not well defined but some studies suggest nosocomial acquisition (19, 20).

Type III GBS are isolated from greater than *95%* of infants with late onset illness, thus suggesting that type III capsular polysaccharide may have a special tropism for the meninges of neonates (8, 10). The clinical features include drowsiness, fever, irritability, tachypnoea and seizures.

The incidence of neurological sequlae following recover}' from meningitis has been reported to be upto *50%* by horn ana Zimmerman et al (23), although other studies report a much lower figure (2).

The immune response to GBS challenge appears to be mediated through non-specific plasma factors including opsonin, phagocytic-ability and presence of type specific agglutinin (24). Kelsius and Zimmerman et al (24) suggest that the rapidly fulminant type la illness is due to the fact that 90 - *95?:* of neonates have none of the factors described above as a first line of immunologic defence. Therefore neonates aspirating GBS la organisms are simply overwhelmed by the infection while those who survive an acute GBS la sepsis probably do so because of passively acquired GBS la antibodies and minimal challenge with the organism (24).

Baker and Kasper (25, 26) showed that many women of child bearing age possess circulating aritibooy to the neutral buffer polysaccharide antigen of type III GBS and that this antibody is transferred to neonates via the placental circulation. Women delivering infants with proved type III GBS disease of either the early or late-onset type rarely have detectable antibody in their sera (25)•

Susceptibility to infection in neonates may therefore be due to lack of passive immunity, immaturity of the immunolgic system or inability of polvmorphoneuclear leucocytes to phago-cytose the bacteria (1, 10, 18, 27).

Reports on the epidemiology of GBS in the developing countries and indeed in Africa are scarce. Longe (28) in his study of neonatal meningitis in Nigerian infants over an eight year period (from 19 74- 1982; noted a conspicuous absence of GBS as an aetiological pathogen. Instead he noted a predominant role of E. coli and Staphylococcus aureus. This observation is in keeping with previous reports from Nigeria on infections in the newborn (29. 3C). Malenga in 1981 (31), in her study of infection in the newborn at Kenyatta National Hospital (KNH) found no GBS among 89 bacterial isolates. Klebsiella and E. coli were the commonest organisms incriminated in the aetiology of neonatal infections. However more recently there has been increasing reports of GBS infection. Onille (32) reported 4 cases of GBS infection in infants at the University College Hospital Ibadan during the period August 1977 to March 1979. He reported both the early and late onset types of illness . In 1982 at KNH, Onyango and Ndinya-Achola et al reported GBS infection in 6 neonates over a six month period (33)• 4 had meningitis, one septicaemia and one with both meningitis and septicaemia. The isolates were identified as type III. Whether this indicates an upsurge of GBS infection and possibly colonization in our environment, or merely the use of more specific laboratory techniques is difficult to comment on because of paucity of epidemiological studies on GBS infection and colonization in our setting.

It. is tJie lack of such information, and the apparent upsurge of the infection tliat prompted the undertaking of this study.

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Aims and Objectives:

1. To determine the incidence of GBS carrieage among parturients at KNH and their neonates.

2. To determine possible associated factors in relation to the colonization and/or infection with GBS.

Materials and Methods

Tlie study was conducted in Nairobi at the Kenyatta National Hospital (KMK). Xliis is the national referral hospital, but also serves the population within Nairobi who are not **necessarily** high risk pregnancies. The study was conducted over a 3 month period from 20th May to 24th August 19S5.

A sample size of 200 mothers and their neonates were randomly selected.

Study areas were visited every day between S.00a.m. and 4•30p.m. In eventualities such as the death of a study neonate the author was contacted within 1 - 2 hours by the members of staff on duty in the study area.

1. Patient Selection:

a) Mothers:

Selection of the mothers was random, by picking on every odd number who was admitted in labour- (i.e. 1, <, 5 etc) to the labour ward. A verbal consent was obtained from the mother for recruitment into the study, after which they assisted in filling in of the proforma (Appendix 9).

b) Neonates:

The neonates of the randomly selected mothers were examined and weighed within 36 hours after delivery. Weighing was uone using the Detecto Infant Scale. Assessment of gestational age at birth was done by calculating from the date of the mothers last monthly period (LMP) and also by the Dubowitz scoring system, which is accurate to + 2 weeks (34).

2. Swabbing:

a) Mothers:

Using a Falcon disposable polyester I'iber-tip.ieCi wood applicator (produced i»v HecLoir Dickinson ia'owaie of California) the lower

vagina was swabbed through 360°, alter parting the labia with gloved lingers. Swabbing was done before delivery, irrespective of whether the membranes were l-uptured or not.

b) Neonates;

Swabbing was done from two sites, the nasopharynx ana umbilicus using the same type of applicator stick as used for the mothers above.

i) Nasopharynx:

After depressing the tongue with a disposable wooden tongue depressor, the swab was then inserted behind the uvula and soft palate into the nasopharynx, avoiding mouth contamination of the swab bud.

ii) Umbilicus:

The swab was swept round the whole base of the umbilicus. All the swabs were immediately streaked on sheep blood agar (SBA) plates in the labour ward and were incubated within one hour of plating. The neonates were foilowed up during their hospital stay and any infant who developed features of sepsis during this period was investigated, where possible, under the following lir.es:-

Cultures were taken from the nasopharynx, umbilicus, blood, urine ana cerebrospinal fluid and the neonate was appropriately treated. In the event of any c;f the study neonates dying during this period, post-mortem heart blood and lung aspiration was done within one to two hours after death and cultured for GBS. after an informed consent was obtained from the parents.

Bacteriology:

The plates were incubated aerobically 37°C and were read at 24 and 45 hours and any plate that had no growth at this time was discarded.

(IBS wore recognized by oor,)hology oi" colonics and their han.Kilytic pattern of beta haemolysis or nun haemolysis, catalase negative, oxidase negative, gram positive cocci. Other characteristics included nannitol fermentation, aesculin negative and bacitracin resistance. Typing was performed using commcercially available antisera (Phaaebact Brocades manufactured by Pharmacia diagnostic AB I'ppsals - Sweden).

Strains that were positively identified as GBS had antibiotic sensitivity carried out on them using Polydisc antibiotic discs (produced by Smith Biolab. Ltd., NewZealand). Counter checking of penicillin in the polydisc was done by using penicillin from another source - Oxoid Ltd. (England).

Semi-quantitative assessment of growth density of GBS was done on the primary plates as follows:- 1+ - a few colonies occupying less than a quarter of the plate; 2+ = colonies covering almost, half of the plate; 3+ = colonies coverijig almost three-quarters of the plate; 4+ - growth almost completely covering the plate.

Statistical Test

Four types of statistical analysis were used. Conditioned test for means, Chi square (X^) test, McNemars test and exact test for the two by two tables.

Results:

A total of 200 mothers and their 203 babies were swabbed. There were 3 sets of twin deliveries. For the purpose of this study, cnly the mothers and their first born babies are considered in cases of multiple deliveries.

(In the 3 set of twin deliveries only one mother and her first twin were positive for GBS, the rest were negative).

Description of the population studied:

Mothers:

Table I (a) Mothers age:

Age in years	Number of mothers	%
< 20	42	21
20 - 29	138	69
30 - 39	18	9
'>40	2	1
n	200	100

The age range of the mothers was 16 to 44 years. 90$ of them were below 30 years of age. 13 of the 42 mothers (31%) below 20 years were in the age group 16 to 17 years, while 69% wore 18 to 19 years old. There were two mothers over 40 years, one was 41 years and the other 44 years old.

Table Hb > P^riCv:

Parity	Number of mothers	~JC
0	71	35
1 - 2	70	35
3 - 5	36	18
>5	23	12
n	200	100

Majority of the mothers (70/5) were para 2 and less. Only 12« were over para 5- Of these. 43? were para 6, 30% para 7, 17% para 8 and 9% para 9-

Marital status:

156 (7 o f the mothers were married and 44 (22%) v%ere single. Included among the single mothers was one who was separated from her .spouse and another who was widowed.

93-5^ of the mothers had attended antenatal clinic at least once before delivery.

Description of the neonates studied:

Table 2 (a) Gestational Age:

Gestational Age in weeks	Number of babies	b
24 - <38	33	16.5
fcjS - £42	161	80.5
>42	6	3
n	200	100

117 '58.520 of the babies were males and 33(41.55?) were fcrales, giving a iiiale to female ratio of 1.4:1. i6 - 5;" of the babies wore preterms with the smallest having been born at 24 weeks of gestation. Most of the premature babies (b?.%) were between gestational age of 34 to 37 weeks.

Table 2 (b) Weight:

Weight in grams	Number of Babies	%b
< 2500	31	15-5
^2500	169	84-5
n	200	100

The weight range was between *500g* to 4500g. The low birth weight babies comprised 15-5% of the total. Of these, 24 *(77%)* were small for gestational age (SGA).

GBS Isolation Patterns:

200 mothers were swabbed. 10 were found to be positive for GBS, giving a vaginal carriage rate of $%• 6 of the.se positive mothers had babies who were also positive, while 4 had babies who were negative. Of the- 190 mothers who were negative, 23 (12%) had babies who were positive.

Of the 200 infants swabbed, 29 were positive for GBS. giving an infant carriage rate of 14'55.

Table Relationship bx. :.wei-n GBS colonization

in the rootner*-. and babi»rs:

		Mothers		
		Positive for GBS	Negative for G3S	n
Babies	Positive for GBS		23	29
	Negative for GBS	4	167	171
	n	10	190	200

X^2 = 13.80 T-^3.72 P = 0.0008 Positive association.

605? of the positive mothers had positive babies, while *12.1%* of the negative mothers had positive babies. On the other hand *21%* of the positive babies had mothers who were also positive, compared to 2.3$ of negative babies who had positive mothers. This indicates that the chances of a baby being colonizcd arc significantly higher if the baby is born to a colonized mother, as compared to the chances of a baby born to a negative mother.

198 (*99%*) of the babies were swabbed within 24 hours of delivery, while all the babies (100£) who were GBS positive were swabbed within the first 24 hours of life.

Two sites, the throat and umbilicus were swabbed in each infant and the isolation rates from each of the two sites is shown in table 4 below.

Tahle 4: GBS Isolation rates at I he nrnhiliens and throat:

		Umbilicus		
		Positive for GBS	Negative for GSS	n
Throat	Positive for GBS	7	8	15
	Negative for GBS	14	171	185
	n	21	179	200

X^2 =18.60 T=4-31 P= 0.0002 Positive correlation

Table 4 shows that the probability of both sites being positive are significantly higher' than of one site being positive alone. Though isolation of GBS was much higher from the umbilicus (72!?) than from the throat (51>7$)j the difference was not statistically significant (P = 0.27).

Four (66.7$) of the 6 positive babies whose mothers were also positive had GBS isolated from both sites while *in 2 (23-3%)* the organism was isolated from the umbilicus only. On the ether hand, only 2 (13$) of the 23 positive babies whose mothers were GSS negative had the organism isolated from both sites, while in 12(52.2^) the organism wds isolated from the umbilicus and in 8(34.8$) from the throat.

Using a semiquantitative methed for estimation of growth density of GBS on sheep blood agar plates, there was an apparent trend of the heavily colonized mother having colonized babies. However when put to statistical

test this was not found to be significant (Appendix i).

Relationship between maternal characteristics and GBS colonization:

Table 5' Association between maternal age and coloni/.at'ion:

	Mothers				Babies			
Age in years	No. of Mothers *ve for GBS	No. of Mothers -ve for GBS	Total No. of Mothers	% +ve for G3S	No. of +ve Babies	No. of -ve Babies	Total No. of Babies	% +ve for GBS
<20	2	40	42	4.8	4	38	42	9 o
20-29	6	132	13S	4-3	20	11S	138	14o
30-39	2	16	18	12.5	4	14	18	22
} 40	0	2	2	0	1	1	2	50
•	10	190	200	5	29	171	200	U o

X^2 =1.18 P>0.25 X" = 2.60 P = 0-25

The age range was 16 to 44 years with a mean of 24-6 years. Hie mean age of the positive mothers was 25.6 years and that for negative mothers was 24.5 years. The mean age of mothers with positive babies \%as 26.1 years wiiile that for negative babies was 24.4- Though the trend appears that GBS isolation increased with maternal age, the difference was not statistically significant.

Table (>: Association between Marital status and colonization:

6 (a) Single mothers:

		Single Mothers		
		Positive for GBS	Negative for GBS	n
Babies	Positive for GBS	1	3	4
	Negative for GBS	2	38	40
	n	3	41	44

P = 0.252

The number of single positive mothers was too small, however there was no correlation between colonization of single mothers and colonization of their babies.

6 (b) Married mothers:

		Married Mothers		
		Positive for GBS	Negative for GBS	n
	Positive for GBS	5	20	25
Babies	Negative for GBS	2	129	131
	n	7	149	156

X^2 = 10.65 T = 3.26 P = 0.001

There was a positive correlation between the positive married mothers and positive babies, suggesting that the chances of a baby being colonized when a married mother is colonized is statistically significant.

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Table 7: C.BS colc.ni/at ion in relation to Parity:

	Mothers				Babies			
Parity	No. of Mothers +ve	No. of Mothers -ve	Total No. of Mothers	% -ive for GBS	No. of +ve Babies	No. of -vc Babies	Total No. of Babies	% +ve for GBS
0	4	67	71	5.6	8	63	71	11
1-2	4	66	70	5-7	12	58	70	17
3-5	0	36	36	0	5	3r	36	13.s
	2	21	23	2	4	19	23	17.4
n	10	190	200	5	29	171	200	14.5
	P > 0.50				P = 0.50			

Colonization rates appear higher in the mothers with parity 2 and below as compared to those para 3 and above. Hov.ever the difference was not statistically significant, indicating no association between colonization of the babies and the mothers parity.

Table 8: Association between contraceptive use and colonization:

	Mothers				Babies			
	No. of +ve Mothers	No. of -ve Mothers	Total No. of MoU-rrs	% +ve for GBS	No. of +ve Babies	No. of -ve Babies	Total No. of Babies	% +ve for GBS
Contra-ceptive users	4	51	55	7.3	10	45	55	lS.J
non Cont ra-ccptive users	6	139	145	4-1	19	126	u s	13.1
n	10	190	200	^	29	171	200	14-5
		P = 0.467			P = 0.50			

27-5." of all the mother*-, had u*ed contrace.pt iv«— within the last 3 years prior to the present delivery. The nx>st popular form of contraceptive was the pill which had been used by 35(63.6£) of the mothers. This was followed by the intrauterine contraceptive device (I.U.C.D.) which was used by 17 (30.9%) of the mothers. The less common methods used were condoms (3-6^) and foaming tablets (1.850.

Though it **appears** from table 8 **that** colonization rates were higher in the mothers who used contraceptives, when put to statistical test, the difference was not significant.

Table 9: Duration of last coitus to delivery in relation to colonization:

	Mothers				Babies			
Last coitus before delivery	No. of +ve Mothers	No. of -ve Mothers	Total No. of Mothers	% +ve for GBS	No. of +ve Babies	No. of -ve Bab;es	Total No. of Babies	% -rVC for GBS
< 1 week	3	27	30	10	A	26	30	13-3
1-4 weeks	1	31	32	3	4	28	32	12.5
>4 weeks	6	132	133	4-3	21	117	138	15.2
n	10	190	20C	5	29	171	200	14.5
	P = 0.15				P = 0.30			

Higher colonization rates were found in mothers who had sexual activity within one week prior to delivery as compared to those who did not. The difference however, was not statistically significant. Such an association was not found in the negative mothers whose babies were positive.

Table 10: Obstetrical problems (lin ing tiie antenatal jx'i iod in relation to colonization:

	Mothers				Babies			
	No. of +ve Mothers	No. of _ve Mothers	Total No. of Mothers	% +ve for GBS	No. of +ve Babies	No. of -ve Baoies	Total No. of Babies	% +ve for GBS
No Obstet. Problem	6	146	152	3.9	19	133	152	12.5
Obstet. Problem Present	4	44	48	8.3	10	38	48	20.8
n	10	190	200	5	29	171	200	14o

X = 0.70 P = 0.40 X = 2.04 P = 0.15

The obstetrical problems in the 4 positive mothers consisted of vaginal discharge in 2 mothers, drainage of liquor in one and vaginal bleeding in the other.

The 10 mothers who had obstetrical problems and had positive babies, had vaginal discharge in 6, vaginal bleeding in 2. drainage of liquor in one and one with a Shirodkar stitch.

The colonization rates appear higher in the mothers with obstetrical problems and also in the babies whose mothers had obstetrical problems. When put to test, however, the difference was not statistically significant.

'I.iblr Jl: A-ssik i.it ion Ix't-wccu <iin .il ion of mpl nrc of nkmbranos to delivery and colonization:

	Mothers				Babies			
Time before delivery in hours	No. of +ve Mothers	No. of -ve Mother-s	Total No. of Mothers	% -t-ve for GBS	No. of -tve Babies	No. of -ve Babies	Total No. of Babies	% +ve for GBS
<6	1	74	75	1.3	6	69	75	8
6-12	6	62	68	8.8	15	53	68	22
13-18	0	30	30	0	2	28	30	7
>15	. 3	24	27	11	6	21	27	22.
n	10	190	200	5	29	171	200	14.5

(test on means) X =2.04 P=0.15 X'=1.69 P=0.20

In 173 (86.57°) of the mothers, membranes were ruptured less than 18 hours before delivery.

Two (2C'fr>) of the positive mothers and 10 {5•3$) of the negative mothers had their membranes ruptured more than 24 hour's before delivery Though a higher colonization rate was found in mothers who had prolonged rupture of membranes (greater than 24 hours), the difference was not statistically significant. There was no association between colonization of the infants and duration of rupture of membranes.

Duration of second stage of labour in relation to colonization:

lSl (90.5^) of all the deliveries had the second stage of labour lasting less than 30 minutes. All the mothers who were positive for G3S also had the second stage lasting less than 30 minutes.

Only **2** of the 23 positive babies whose mothers were negative had delivery associated with second stage of labour lasting between 30 to 60 minutes.

Table 12: Relationship between mode of delivery •md colonization:

	Mothers				Babies			
Mode of delivery	No. of +ve Mothers	So. of -ve Mothers	Total No. of Mothers	% +ve for GBS	No. of +ve Babies	No. of -ve Babies	Total No. of Babies	% +ve for GBS
SVD	5	128	133	3-8	15	118	133	11.2
C/S •	4	52	56	7-1	11	45	56	19.t
V/E	l	10	11	9.1	3	8	11	27-2
n	10	190	200	5	29	171	200	14.5

SVD Vs the rest (C/S - V/E) X=1.35 P=0.50

SVD Vs the rest (C/S + V/E) X =3.74 T =1.93 P=0.088

There was no association between mode of delivery and colonization of the mothers. However, there war. a difference, though not statistically significant (P-0.088). in the colonization rates between babies delivered by spontaneous vertex delivery (SVD) and those delivered by caesarian section (C/S) and vacuum extraction (V/E). The 5 positive mothers who delivered SVD, 3 (60$) nad babies who were colonized. 2 (50^) of the 4 positive mothers delivered by caeserian section had babies who were colonized.

Eleven positive babies were delivered by C/S. Ten of them (90i?) had their re^mfcranes ruptured before delivery. The eleventh one was delivered as an elective C/S to a negative mother with intact membranes. However, this mother had a vaginal discharge during the third trimester.

Ilio |v>sitivc mother who was delivered by vacuum extraction had a baby who was also colonized.

Relationship between Infant characteristic and GiiS co11>ni/ation:

Table 13: Gestational age in relation to colonization:

Gestational age i.n weeks	Babies Positive	Babies Negative	Total No. of babies	% +ve for GBS
24- 458	5	28	33	15
^38- 42	23	138	101	14.3
>42	1	5	6	17
n	29	171	200	14-5

P> 0.50

17.2$ of th3 positive babies and 16.4% of the negative babies were preterms.

There was no association between gestational age and colonization rates.

Infants' weight:

31 babies (15-52) were below 2500g. 3 (IC%) of these babies were positive for GBS. Of the 169 (S4.) babies above 2500g, 26 (15-3$) were positive, thus indicating no association between trie infant's weight and colonization. 24 babies (12%) were small for gestational age f'SGA), 169 (84-5/?) were appropriate for gestational a<*e (AGA)

oiid 7 (3 < 5," > vvvr-e largo for gestational age (IGA). QIS positivity rates in these 3 categories were 4 1 5 . H % and 14.3a! respectively. There was no statistcal difference between these 3 groups. (Appendix 2).

Infant's sex:

GBS colonization rate was *J4'4%* in males and 14.1$ in females, indicating no sex predilection.

Table 14: Association between congenital malformations ana colonization:

	No. of Positive Babies	No. of Negative Babies	Total No. of babies	% -rve for GBS
Congenital defect present	3	8	11	27.3
No Congenital defect	20	163	189	13.7
n	29	171	200	14.5

$X^2 = 0.64$ P=Q.202

Overall, the rate of congenital nalformations was 5. Sf" Although there were higher colonization rates among babies with congenita] defects than in those with 110 defects, the difference was not statistically significant.

See appendix 3 for details of congenital malformations.

Table 1 Rclat ion.-J.iip hotwmi Infant Complications and colonization:

	No. of Positive Babies	No. of Negative Babies	Total No. of Babies	% +ve for GBS
Complications present	6	32	38	15-7
No Complications	23	139	162	14-2
n	29	171	200	14.5

P>0.50

There was no statistical difference in the incidence of complications between the colonized and r.on colonized babies.

6 positive babies developed complications. 3 of whom died *(50%)*. 5 of the 32 negative babies *(15-6%)* with complications died. See details of the complications in Appendix 4«

It was observed that the more sites colonized, the greater the incidence of complications (Appendix 5)• A greater percentage of complications (28.5$) arose in babies whose GBS growth on sheep blood agar was heavy (i.e. 3+ and above) as compared to those whose growth was light (13.35b) i.e. 2-r and below. This difference was however not statistically significant (Appendix 6).

Iatalitits:

"1.tiilc 16; Death t-at os in the (t>h>ni zed and ;ion colonized infants:

	Total No. of Babies	No. of Deaths	% Death rate
GBS colonized Babies	29	o	10.3
Non colonized Babies	171	5	2.9
n	200	8	4

X^2 = 1.89 P = 0.093

Though the death rate among the GBS colonized babies was much higher than in the non-colonized group, the difference was not statistically significant (P=0.093)•

.All the three deaths in the positive babies occurred in those heavily colonized (3+ and 4-r), see Appendix 7- Two of those who died had GBS isolated from the umbilicus, throat, postmortem hear-t blood and lung aspirates. The third baby had .isolates from the umbilicus only. Therefore of the 3 deaths in the colonized babies two can be directly attributable to GBS infection. This gave an estimate of GBS disease attack rate of 2 out of 29 colonized babies (6.89/0. The exact attack rate could not be calculated from this study, as follow-up of all the colonized babies was not done and also for technical reasons it was not possible to thoroughly investigate all the GBS positive babies who developed complications. The above disease attack rate could therefore be an underestimate.

(see details of the dead babies in Appendix 8)

Table 17: Antibiotic sensitivity pattern of (IBS to sorr.c- of the corrt.ionly used antibiot jcs in the neonates:

Antioiotic	No. sensitive (% sensitive)	No. resistant {% resistant)	Total
Penicillin (single disc)	14 (93-3$)	1 *(6.7%)*	15
Penicillin (multidisc)	2 (5-7%)	33 (94-3%)	35
Erthromycin	35 (10052)	0 *(0%)*	35
Cloxacillin	28 (80%)	7 (20%)	35
Ampicillin	31 (88.6%)	4 (11.4%)	35
Streptomycin	0 (0%)	35.(10020	35

There were 39 isolates of G3S in total. Only 35 were tested against the above antibiotics, because 4 cultures were contaminated with proteus and it was difficult to set up purity plates from these contaminated cultures.

Polj'disc antibiotics were used to test for sensitivity. It was only possible to counter check with penicillin single disc. From local experience it appears that penicillin loses potency much faster than the other antibiotics in polydiscs (35) and this could explain the marked difference noted in table 17 between the two penicillins.

It was apparent that erythromycin, penicillin G, arapicillir. and cloxacillin were antibiotics that **G83** strains were sensitive to> while 100?! resistance was found to streptomycin.

Discussion

The finding of 5% GBS carriage rate in the mothers and 14-5% in the infants in this study falls within the range of GBS colonization reported in North America and Europe (l, o, 7, 12, 15, 18, 22. 36). but not in concordance with what was found in Nigeria (37), where a higher colonization rate (19.5$) was found in the mothers, with a lower rate (8.9*) in the neonates.

There is a large variation in carriage rate in women in labour ranging from **4.6%** for parturients in Colorado (l) to a high rate of 35% in Houston, Texas (3o) and a similar range has been obtained for acquisition of GBS by the neonates (18, 12, 37, 38, 39).

The factors that have been propounded to account for these differences include varying geographical locations, intrinsic differences in study population, use of selective enrichment media, the number of speci>nensj collected from each individual and the number of sites cultured (8, 12, 37. 47)• Some of the colonization rates reported from different studies are shown below.

	Geographical region	colonization of mothers (%)	colonization of babies (%)
Franciosi et al (1973)	Colorado	4-6	1.2
Baker & Barret (1973)	Texas	29.8	20.2
Ferrieri et al (1975)	MLnnesotta	5.6	37
Pass et al (1978)	Alabama	–	12.5
Amona et al (1978)	Minnesotta	8	3-4
Hilderbrand & Schreiner (1980)	N. America	3-40	1-20
Dawoau et al (19S0)	Nigeria	19.5	8.9
Mirza (19S5)	Kenya	5	14.5

Description ol' the study population:

There were a total of 1600 deliveries during the study period. The 200 patients studied represented 12.5$ of the total deliveries. Compar ing the structure of the study population with the Nairobi birth survey results (39, 40), there was concordance in most of the variables. For example, 695? of the mothers in the study population were between 20 - 29 years, 70/6 of them were of parity 2 and less, 76$ were married and 93 • 5% of them had attended antenatal clinic at least once. Corresponding figures given in the Nairobi birtJh survey are 64-7$, 77%, 84-4/? and 96.4$. The population of the babies studied was also fairly representative of the population at KNH as indicated in previous studies. (41, 42).

GBS Isolation patterns:

60% of the uacthers who were GBS positive had babies who were also colonized. There was also an apparent , though not signficant, trend to greater colonization of the babies in mothers who were heavily colonized.

Several workers have shoivn that if the mother is culture positive for GBS the chances of her baby being colonized are 29 to 70% (1, 6, 7, 8, 12, 22, 43)- The finding of this study of 60% therefor-e lies within this range. Dawodu et al in Nigeria found a aich lower correlation, with only 29% of babies bom to colonized mothers also being colonized.

12% of the mothers who were vaginally negative for G3S had babies who were colonized. This agrees with the findings of Franciosi et al (1) and .Anthony et al (44). Some of the possible reasons for the finding of culture positive babies born t. o vaginally negative mothers include:-

1. "False negative" cultures, since only one site was cultured in the mothers. It has been observed elsewhere (36) that with single-vaginal culture ,

false negative rates of SO? to 60? occur with use of non selective agar plates or broth media. In their two studies, Franciosi ct al (1) found that some of the positive babies had negative mothers and they attributed this to their culture methods and commented that these mothers would probably be found positive if more sites were cultured using several methods.

The vagina may also not be the most suitable site for recovery of GBS, since recent studies suggest that the lower gastrointestinal tract may be the primary site for asymptomatic GBS colonization in women and that isolation of this organism from vaginal cultures represent contamination from this site (12, 36, 45).

2. Antiseptic lotion^ that may have been used by the obstetrician before swabbing for culturing was done could have rendered the lower vagina relatively " sterile" but unaffecting the upper vagina and cervix.

3- Nosocomial acquisition either from the mothers or health personnel. Although all the babies who were GBS positive were swabbed within 24 hours of delivery, with 41% of them being swabbed witiiin 12 hours of life, it has been shown that the newborn infant is exposed to bacteria from the mothers vaginal and intestinal microflora during birth and then from the external environment. Rotimi and Duerdin (46) found that colonization occurs rapidly from 6 hours of life and bacteria mainly streptococci viridans and staphylococcal can be cultured within 24 hours of life. Nosocomial infection rates of upto 40« have been reported elsewhere (36). GBS carriage rate in health personnel in our environment is not known but several reports (l. 7, 8, 36) indicate that the carriage rate in health personnel, pregnant mothers and non pregnant mothers is almost the same. Therefore the probability

of nosocomial acquisition in the GBS positive babies with negative mothers cannot be ruled out., and in this study it was not possible to conclusively deduce the source of GBS in these 23 babies.

Maternal characteristics:

Age and Parity:

There was no relationship between maternal age and parity with GBS colonization. Though the mean age of the mothers who were positive was slightly higher than those negative for GBS, the difference was not statistically significant. These findings differ from those of Gerard et al (22) and Dawodu et al (37) who found that a significant proportion of trie colonized mothers were primigravida, but. agrees with those of Kaj-mond and Deiter (7) who found no statistical significant difference in the age or parity of the women in the culture positive and culture negative groups.

Marital status:

There was a positive association between GBS positive mothers and delivery of babies who were also positive. No such association was found with the single mothers. This could partly be explained by the fact that there were more married mothers (76/;) than single mothers. Presumably the other possibility is that a large proportion of the married mothers had consistent sexual partners., some of whom could have been GBS positive. It cannot, however, be deduced likewise that the single mothers had more than one sexual partner or that the frequency of sexual relationship was any different from that of the married mothers. It was not possible to derive such information from the mothers as these are rather confidential and sensitive issues.

However, it has been shown that about 50^ of sexual partners of GBS positive women are also positive (ft. 12, 36) and that GBS is now recognized as one of several sexually transmissible agents (.36).

Conl-racf'pt.ivp use:

Hicrc was no difference in the G'iS carriage in mothers wno itsed contracept ives and those who did not. There was also no statistically significant difference in GBS acquisition oetween the baoics whose mothers tised contraceptives and those whose mothers did not. Contraceptives, therefore did not seem to play a role in GBS carriage, though one of the hypothesis put l'or-ward to explain the apparent upsurge of." GBS in North America is the maternal use of contraceptives (4)»

Obstetrical Complications:

GBS colonization was neither related to duration of last coitus to delivery nor to any maternal obstetrical complications during the antenatal period or delivery.

Three (10.3^) of the GBS positive babies and ten *iS-2%)* of GBS negative babies had mothers whose membranes had ruptured more than 24 hours before delivery. There was no association between duration of rupture of membranes and GBS colonization. Membrane rupture for more thrn 24 hours has been strongly implicated as a predisposing factor to GBS colonization of the infant (1, 6, 12, 22, 38, 43), while other studies have not shown such an association (7, 37). Baker and Barrett (15) found that although maternal complications aid not influence the prevalence of asymptomatic colonization with GBS, prolonged rupture of membranes was significantly associated with proved early onset infection. In this study, among the two babies with proved GBS infection, one had a mother whose membranes had ruptured 28 hours before delivery-. However, since all the babies who developed GBS infection in this study could not be detected, the association between GBS disease and ruptur-e of membranes could not be established.

Mode of Delivery:

66.5% of all the deliveries in the study were spontaneous vertex delivery (SVD). 28% were caeserian sections (C/S) and 5o£ vacuum extractions (V/E). This represents a higher rate of C/S and V/E as compared to the 19S4 KNH figures of S3.4/' SVD, 14-4$

C/S and *2.2%* V/E (47).

There was a higher incidence of GBS acquisition in the infants born by C/S and V/E as compared to those born SVD, though not statistically significant at a p value of 0.088. Although studies have pointed to the fact that maternal obstetrical complications may lead to increased GBS colonization, none has indicated that C/S and V/E were predisposing factors.

Eleven (37-9%) of GBS positive babies were born by C/S. One *(9%)* was born as an elective C/S with intact membranes. This finding is not unusual. Eickhoff (6) described a baby bom by elective C/S with intact membranes who was acutely septic. This emphasizes the possibility of ascending infection with apparently intact membranes, resulting in colonization and/or infection of the babies.

In this study, no association was found between duration of second stage of labour and GBS colonization.

Infant Characteristics:

There was no predilection of GBS colonization to the infants' sex.

A marginal increase in colonization with increasing birth weight was observed (15-3% of those over 2500g were positive compared to *10%* in those under 2500g). This difference was however not statistically significant.

GBS infection was proven to have occurred in two cases. Both were preterms and both died. Premature and SGA infants have been found to have a higher risk of developing neonatal GBS sepsis and meningitis (12, 22, 38). Baker arid Barret (15) found that birth weight, and sex did not, influence the prevalence of asymptomatic colonization of GBS but that low birth weight (LBW) significantly increased the chances of earjv onset infection. Gerard (22) found a trend, though not

statistically .significant, to an increase of preterm delivery or LHW infants born to ('.US positive mothers. Whether or not infection contributes directly to preterm delivery, the degree of immaturity influences gravity of infection and obviously lessens the chances of survival (43)'

Isolation Sites:

Two sites, the umbilicus and throat were swabbed in the inf ants.

If the umbilicus alone was swabbed, then G5S carriage rate would have been 10.5% and if the throat alone was swabbed, the carriage rat e would have been 7' 5%> close to what was found in Nigeria by Dawodu (37) of 8.9% where only the external auditory canal was swabbed. Though in this study, GBS recover}- was higher from the umbilicus than the throat, the difference was not statistically significant. A strong correlation was however found in the isolation of the organism from the umbilicus and throat, thus stressing what has been found by other workers that the more sites swabbed the greater the positivity rate (8, 12, 36, 38, 48). Baker and Barrett (15) found that the sj'iigle most frequent site of colonization was the umbilicus, and the role of the umbilicus as the initial colonization site and subsequent endogenous source of serious systemic infection in individual infants has been acknowledged (12. 31, 34, 46). However, Ferrieri et al (48) found the external ear canal was a favourable site for detecting neonatal colonization with GBS with 94% recovery rate. It is apparent therefore that the throat and umbilical cultures cannot be expected to detect all infants who are colonized (46).

Congenital Malformations:

GBS positivity rates were found to be higher in babies with congenital mai formations as compared to those with no malformations, though the difference was not statistically

significant. One baby with proven GBS infection .»Lso had a severe congenital malformation. Franciosi et nl (l) found that 11.6% of neonates with GBS sepsis in her study had severe congenital malformations. Congenital malformations liave been recognized to be associated with increased risk of development of neonatal sepsis (34)'

Complications in Infants:

Although there was no difference in the complication rates between the GBS colonized and non colonized babies, it was observed that the more sites colonized, the greater the incidence of complications. A greater percentage of complications (28.5%) arose in infants whose GBS growth on SBA was heavy (3+ arid above) as compared to a lower complication rate (13 • 350 in those whose growth was *2-t* and below. Such findings concur with what has been found by other workers (l» 3, 5, 43)'

GBS Disease attack rates:

GBS disease was proven in two of the *29* colonized babies. These two babies had heavy GBS growth from the umbilicus, throat, post mortem heart blood and lui.g aspirates. This gives an attack rate of GBS disease of 6.9 per 100 colonized babies. Though this attack rate is probably an underestimate. since all the colonized babies were not followed up ana some of the positive babies with complications could not be adequately investigated to rule cut GBS disease, it is much higher than the 1-2 per 100 colonized infants quoted by other workers (1. 6, 15, 22, 36). Whether this indicates a more invasive type of GBS in our environment, or an increased susceptibility of the babies to GBS disease from prooably lack of transmissable maternal antibodies is difficult to conclude from this study. Further work is required to look into these possibilities.

Dawodu et al (37) in Nigeria foicid a low incidence of neonat disease despite *p.* high vaginal colonization among pregnant women. They suggested that this was in keeping with earlier findings of

a lower incidence of GBS neonat al sepsis but a higlier vaginal carrier rate among black parturients as compared to their Caucasian counterparts. However, Parker (38) in his review, noted that although the incidence of GBS varied with different geographical regions, the attack rate was independent of this variation.

Fatalities:

There was a remarkable, though not a statistically significant difference in the death rates between the GBS colonized babies (10.3$) and the nor. colonized group (2.9$). All the 3 deaths in the GBS positive babies occurred in those who were heavily colonized (3+ and above). Two of the 3 deaths could be directly attributable to GBS disease. Both these babies were LBW. One was a still birth born to a mother with a bad obstetric history, and the other was a baby who died within 3 hours of delivery. The third baby who died, had GBS isolated from the umbilicus only. Post mortem heart blood arid lung aspirate were negative for- GBS. This could be due to the fact th^t there were actually no organisms present, or, as pointed out by Eickhoff (6) GBS might not have been cultured at post mortem because the baby had received antibiotics.

Antibiotic Sensitivity Pattern:

GBS was found to be sensitive to the commonly used antibiotics: penicillin, erythromycin, cloxacillin and arnpicil]in but resistant to streptomycin. These results illustrate the short-comings of using a nultidisc in which certain antibiotic discs either expire early or are not very sensitive. Unfortunately, due to unavailability of other single antibiotic discs, counterchecking of the multidisc viability for other antibiotics besides penicillin was not possible. As can be seen from the results, the sensitivity pattern between the penicillin in the miiltidisc and in the single disc were completely different.

In this light therefore it is difficult to interpret the sensitivity to streptomycin, but studies done elsewhere also found that penicillin was the most effective agent against GBS followed by erythromycin and ampcillin, while Kanamycin and neomycin were the least active agents (J, 6, 18). It has also been suggested that although GBS is sensitive to both penicillin and ampicillin, the mean inhibitory" concentration (MIC) for these antibiotics is higher than that for group A streptococcus (18, 36).

Conclusions;

1. Q18 carriage rates in mothers at KNU is 5% while that, in neonates is 14-!)%•

2. The more sites swabbed, the greater the GBS recovery rate. Though the umbilicus showed a slightly greater recovery for the organism, the difference was not statistically significant.

3. Tne chances of a baby being colonized by GBS are significaj:- higher if the mother is vaginally positive for GBS as compared to when the mother is vaginally negative.

4- There is an association between married mothers who were positive for GBS and infants positive for GBS. No such association was found for the single mothers.

5- Ihere is no significant association between GBS carriage in the mothers and infants with maternal age, parity, contraceptive use, last coitus before delivery, obstetrical problems in the antenatal period, duration of rupture of membranes to deliver}', gestational age of the baby, birth weight, congenital malformations and complications arising in the infants.

6. There is a remarkable, though not statistically significant difference between GBS carriage rate in babies born by CA-i and those born SVD.

7- Death rate in the GBS colonized babies though much higher than in the non colonized babies, is not statistically significant.

8. The estimate GBS disease attack rate is 6.9 per 100 colonized babies.

9. GBS is sensitive to penicillin, erythromycin, cloxacillin and ampicillin but highly resistant to streptomycin.

Recommendations:

1. A wider based epidemiological study is required to include both the rural and urban areas. GBS carriage in health personnel, pregnant and non pregnant women with their sexual partners needs to be determined using multiple sites inorder to define the magnitude of the problem in our environment.

2. GBS serotypes in our setting need to be determined.

3. Follow-up of colonized babies is required inor der to define the exact GBS disease attack rate locally.

4. **A high index, of suspicion of GBS disease in infants is important especially in our newborn unit, in view of the. relatively high disease attack rate.**

5. Mothers with bad obstetric histories,, should be investigated together with their sexual partners for evidence of GBS infection.

6. Research required to look into the possibility of preventing transmission to the infant by a GBS colonized mother either before or after delivery by chemo— prophylaxis or immunization.

At ki h nv i < •(Ijy ttx't its:

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Appendix

Appendi\ 1 : Density oC maternal col oni/at ion compared to infant, colonization using a semi-quantitative method:

Growth of GBS on sheep blood agar	No. of Mothers +ve for GBS	No. of Infants colonized	% colonization
1+	2	1	50
2+	4	1	25
3+	3 •	3 '	100
4+	1	l	100
n	10	0	60

X~ = 2.24 P = 0.13

ndix 2 : Infants weight : age appi opriatness in relation to GBS colonization:

Wt : Age	Total No. of Babies	+ve Babies	% -tve for CBS	% of Infants with Compli-cations
SGA	24	1	4	4.2
AGA	169	27	15-5	3
LGA	7	1	14-3	0
n	200	29	U.s	3

X^2 = 1.23 P>0.25

Appendix .>: Dctaiis of the congenital defects:

lhe 3 malformations in the GBS positive babies were:-

i) Polydactyly in an infant whose mother was negative.
ii) Downs syndrome born by C/S to a negative mother
iii) Ancncophaly in a premature macerated still birth born to a negative mother.

The S congenital malformations in the GBS negative babies with negative mothers included:-

i) 5 babies with musculoskeletal anomalies (2 **Polydactyly,** 2 talipes equino varus and one with multiple skeletal anomalies).
ii) One baby with gross hydrocephalus and spina bifida cystica.
iii) One albino
iv) One with Downs syndrome.

Appendix j : Details of complications in the Infants:

Hie complications in the 6 GBS positive babies included:-

1. Baby T. M. Born as an SGA, first twin to a positive mother. Developed transient respiratory distress which settled in one day.

2. Baby A. W. was a full term baby born SVD. Developed jaundice within 24 hours of birth. The mother was Rhesus positive and was vaginally negative for GBS. The baby improved on phototherapy.

3- Baby E. K. was a full term baby born by vacuum extraction because of prolonged second stage of labour. Developed respiratory distress at birth which settled within one day. The mother was negative for GBS.

4. Baby M. M. was born preterm, 28 weeks gestation to a mother who was heavily colonized with GBS. The baby had

re.spi ratory distress and apnocic attacks. Died three hours after delivery.

5. Bahy M. W. : Term baby delivered SVD to a pr i mi gravida who was negative for GBS. The baby was ill immediately after birth. He had a high pitched cry with crossing of the limbs. He was diagnosed as having query sepsis with brain damage, was put on antibiotics. Died 24 hours after birth. Blood culture done revealed no bacterial growth.

6. Baby H. A. was born as a macerated anencephalic at 24 weeks gestation to GBS negative mother.

The 32 complications in the GBS negative babies included:-

1. 16 babies with respiratory distress (RD). 10(62.5^) were pre-terms. 3 babies with RD died. All 3 were preterms.
2. 4 coivjuctivitis
3. 3 vomiting, cause not known
4. 2 with septic spots
5. 4 birth asphyxia
6. 1 with meconium aspiration -ydrome
7. 2 still births

Appendix 5: Infant complications in relation to the sites where GBS was isolated:

Site	**Total No. of +ve Babies**	**No. of Babies with compli-cations**	**No. of Babies with no compli-cations**	*to* **complications**
Umbilicus	14	2	12	24.3
Throat	8	0 *4*	6	25
Throat + umbilicus	7	0 *JU*	5	28.6
n	**"0**	0	23	20.6

X^ = 0.74

P > = 0.50

SO

Ap]nr]i\ 6 : Cofiel.it ion between growth density of GiSS on sheep blood agar (SBA) and complicat ions in the infants: ***

Density of GBS on SBA	Total No. of Babies	No. of Babies with c.ompl i cat ions	*t* Compli- cations
1+ and 2+	15	2	13.3
3+ and 4+	14	4	28.5
n	29	6	20.7

xj = 0.31

P = 0.29

Appendix 7 : Association between GBS growth density on SBA and fatalities:

GBS growth on SBA	Total No. of Babies	Total No. of Deaths	/0 Deaths
1+ and !•>	15	0	0
3+ and 4+	14	3	21.4
N	29	3	10.3

P = 0.10

Appendix * Description of the babies who died

a) G3S positive babies who aied:

i) Baby K. A. : A macerated, premature anencephaLic baby of gestation 21 weeks and birth weight 500g. Duration of rupture or membranes to delivery was 10 hours. The mother was a para 3 + 0 with a bad obstetric history (2 neonatal deaths and one still birth).

The mother was vaginally negative for GBS. The baby had a heavy growth of GBS from the throat, umbilicus and lung aspirate. The cause of death in this baby was probably due to GBS sepsis though he would still have died from the congenita malformation even if he did not have GBS sepsis. In retrospect, in view of the mother's bad obstetric history, it would be worthwhile to investigate her, together with her spouse for GBS carriage.

ii) Baby M. M. : Died 3 hours after delivery. He was a preterm with a weight of lOOOg and a gestational age of 2S weeks. Duration of rupture of membranes to delivery was 28 hours. Tue mother was commenced on oral ampicillin on admission. She was heavily colonized by GBS and so was the baby's throat and umbilicus. The baby had apnoeic attacks immediately after birth. He was started on parenteral crystalline penicillin and gentamvein immediately after birth. Post mortem 1-ing aspirate and heart blood also had a heavy growth of GBS. The cause of death in this baby was most likely GBS septicaemia.

iii) Baby M. W. : Died within *2d* hours of delivery. The baby was a full term normal delivery to a GBS negative mother. The baby was colonized at the umbilicus only.

He was noted to be ill almost immediately alter birth, with a high pitched cry and crossing of the limbs. Sepsis with brain damage was querried and the baby was started on crystapen and gentanycin. Blood cultures and post mortem lung aspirate done revealed no growth.

CBS negative babies who died:

5 GBS negative babies died. 2 were still births **and** 3 were neonatal deaths.
Post, mortem lung aspirates done in these babies revealed no growth in 3 of the aspirates, one grew gram negative rods and the fifth grew proteus and colifonns.

Appcndix 9

QUESTIONNAIRE

A) PARTURIENTS:

Name:. . . . . 2. IP No

Age:. . . . . 3. Study No_

Marital status:

Single Divorced

Married Widowed

LMP:. . . . . Last coitus before delivery:-

EDD:

Contraceptives used:

No Yes

Pill

I.U.C.D.

Others (specify)

Parity: -

Antenatal Clinic Attendance:

a) Yes. . . . . from gestation. . . . . (months)

b) None

Any gynaecological problem during the antenatal period

a) No. b) Yes

P.V. discharge

P.V. bleeding

PROM

Others (specify)

Any antibiotics used in the last 4 weeks

No. . . . . Yes_

Time from rupture of membranes to delivery

Maternal temp, at delivery

Duration of 2nd stage of labour

a) 30 mins b) 30-60 mins c) 60-90 mins d) **over Q0 mins.**

Assisted delivery

No. . . . . **Yes**

Forceps

Vacuum

Caesarian section

Culture results for GBS

	Positive	**Negative**
Vagina		

B) NEONATE

a) Name of baby

b) Ip No.

c) Study No.

d) Sex

e) Date of birth

f) Age at examination

g) Gestational age according to dates

h) Assessed gestational age

i) Any congenital defects

No. Yes (specify)

j) Weight.....g

AGA SGA LGA

k) Culture for GBS

Positive Negative

a) Throat

b) Umbilicus

1) Complications post delivery

1. Age of onset of complication

2. Nature of complication

3. Any other lab investigations

XR

Culture

4. Treatment given

5. Fate of baby