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Neonatal septicemia due to group B streptococci — Perinatal risk factors and outcome of subsequent pregnancies

Gerd Faxelius[1], Katarina Bremme[2], Karen Kvist-Christensen[3], Poul Christensen[3], and Signe Ringertz[4]

Department of Pediatrics[1], Department of Obstetrics and Gynecology[2] and Department of Clinical Microbiology[4], Karolinska Hospital, Stockholm, Sweden, and Department of Clinical Microbiology[3], University Hospital, Lund, Sweden

1 Introduction

Group B streptococci (GBS) are a major cause of neonatal infection and contribute considerably to perinatal mortality in the Western world [16]. The early onset disease, occurring within a few days of life, is usually acquired by vertical transmission from the mother; whereas, in late onset infection, a delayed acquisition, e. g. nosocomial, has been suggested [1].

Among parturients, 15–35% are colonized with GBS in the urogenital tract [1]. Prematurity and/or prolonged rupture of membranes increase the risk of an infant of a colonized mother to contract infection [6]. Low maternal levels of type-specific IgG to GBS also increase the possibility for septicemia [4].

GBS have also been the predominant cause of early onset septicemia in our neonatal intensive care unit, as previously reported [13]. Since 1975, all cases of GBS-septicemia have been prospectively registered in the department. Since 1981, women who have previously given birth to an infant contracting GBS-septicemia have undergone carfeul surveillance during subsequent pregnancies using a special prevention program [10]. In the present study we have reviewed all cases of early onset GBS-septicemia during the time period of 1975 to 1986 with special regard to perinatal risk factors. The outcome of a subsequent pregnancy in mothers of infants with GBS-septicemia has also been studied in order to evaluate the prevention program.

Curriculum vitae

GERD FAXELIUS, *M. D., was graduated from Uppsala University in 1964 and qualified as a specialist in Pediatrics in 1971. 1969–1970 she had a fellowship in neonatology at Vanderbilt University. Since 1975 she has been on the staff of Department of Pediatrics, Karolinska Hospital and Karolinska Institute, Stockholm, Sweden.*

2 Material and methods

2.1 Definitions

Septicemia was defined by one positive blood culture with growth of GBS combined with clinical signs of infection and early onset septicemia occurred within the first five days of life.

2.2 Infants

All infants with early onset GBS-septicemia born between January 1975 and December 1986 at the Karolinska Hospital are included in the study. These neonates were all admitted to the neonatal intensive care unit (NICU) at the hospital. Thirty-two thousand one hundred and forty-three infants

were born alive in the hospital during the period, and 40 of them contracted early onset GBS-septicemia. Among these infants were 37 singletons, one of a twin pair, and both infants of another twin pair. Late onset GBS-septicemia was diagnosed in two infants born at the hospital and cared for in the NICU during the time of the study. Since April 1982 no infants have been readmitted to the hospital after discharge from maternity wards.

2.3 Mothers

The obstetrical records of the 41 mothers were reviewed retrospectively. Obstetrical data of the 39 mothers to the infants with early onset GBS-septicemia are presented in table I. Among the women, two had twin pregnancies, two had diabetes mellitus and two had mild pregnancy-induced hypertension. Three of the women received cerclage, which in one of whom was due to a twin pregnancy. Labor was induced in six of the patients, in four of whom because of postmaturity.

Previous obstetrical history was also investigated. One of the women had a previous late intrauterine fetal death, another had an earlier history of three miscarriages.

One of the two mothers whose infants contracted late onset GBS-septicemia was delivered by cesarean section, the other had a preterm vaginal delivery.

2.4 The subsequent pregnancy

During the subsequent pregnancy(ies) (one to three) the mothers were referred to a special prenatal clinic. In all, 24 infants were born to 17 of the mothers up to December 1986. One of these infants was born in another hospital. Cultures were obtained from the urethra and cervix once weekly after the 28th week of pregnancy. If colonized with GBS, the mother was given penicillin V orally 1 g twice daily during the remaining part of pregnancy, followed by intravenous bensyl penicillin 3 g every sixth hour during labor until delivery. Furthermore, cardiotocography (CTG) registration was performed weekly as well as cervical palpation.

The neonates of mothers who were GBS-carriers and/or had been treated with antibiotics during delivery were all admitted to the NICU for surveillance. They were given bensyl penicillin intravenously every twelve hours for 48 to 96 hours. The treatment was discontinued if blood cultures were negative and clinical and laboratory signs of infection were absent.

2.5 Laboratory methods

From each infant cultures were taken from the external auditory duct, umbilicus, nasopharynx and throat on arrival to NICU. When infection was suspected from the clinical picture, cultures were obtained from blood and cerebrospinal fluid (CSF). Urethra and cervical specimens from the mothers were cultured for potentially pathogenic bacteria (*Enterobacteriaceae*, beta-hemolytic streptococci, staphylococci, *Listeria, Hemophilus influenzae*) using routine bacteriological methods [18]. These specimens were also processed using a selective broth medium [3].

Table I. Obstetrical data in mothers to infants with early onset GBS septicemia (n = 39)

	Preterm delivery* (n = 21)		Delivery at term (n = 18)		All (n = 39)	
	No.	%	No.	%	No.	%
Parity						
nulli	12	57	14	78	26	67
multi	9	43	4	22	13	33
Rupture of membranes > 12 hours	9	43	10	56	19	49
Fever	4	19	5	28	9	23
Cesarean section	10	48	2	11	12	31
Vacuum extraction	1	5	9	50	10	26
Age; mean (range)	27.8 (21–44) years		24.8 (19–37) years			

* < 37 weeks gestation

Blood for culture was drawn from a peripheral vein. The blood specimens were inoculated into a set of two culture bottles containing Brain Heart Infusion Agar (Difco, USA) slants with a p-aminobenzoic-acid (PABA) and meat infusion broth with PABA and penicillinase. After 1977 typing of GBS was performed as previously described [8].

Blood specimens from 27 of the 41 mothers whose infants had contracted GBS-septicemia were also available. The specimens were taken by venipuncture as soon as the diagnosis was made in the infant. Antibodies to GBS types I a, I b, II and III were measured with radiolabelled protein A [10]. In brief, the serum to be tested was absorbed with a GBS strain without type-specific antigens. The absorbed sera were mixed with a standardized suspension of the type of GBS to which antibodies were to be measured. Each specimen was tested in duplicate and the difference between a pair of duplicates for a given type was not allowed to exceed 10% of their mean value. The reference intervals for each type presented in the results represents the range obtained with sera from 100 healthy pregnant women.

Results were expressed in percent according to the formula

$$\frac{P - L}{H - L}$$

where H = the highest radioactive count obtained with the 100 reference sera, L = lowest value, and P = the patient's value. C-reactive protein was detected by latex agglutination (RapiTex CRP, Behring, Behringwerke AG, FRG). When positive (> 6 mg/l) the serum was retested by a quantitative immunodiffusion method (LC-Partigen, Behring, Behringwerke AG, FRG). GBS antigen in serum was detected by latex agglutination (Wellcogen Strep B, Wellcome, England). Blood cell and platelet counts were determined using routine methods.

3 Results

3.1 Incidence

The incidence of early onset GBS septicemia during the period was 1.24 per 1000 live births. The annual variation is shown in figure 1, demonstrating a marked top in 1981 in connection with an abrupt increase in the number of deliveries in the obstetrical ward as previously reported [5].

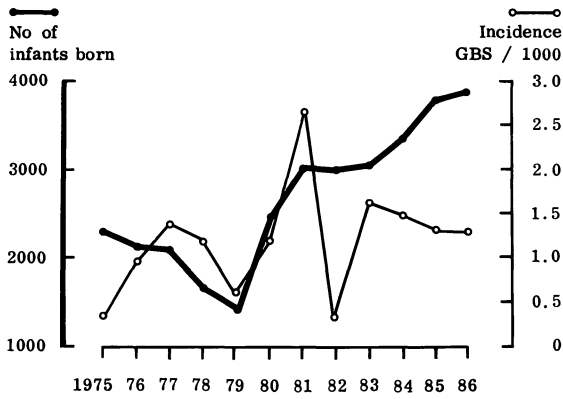

Figure 1. Incidence of early onset group B streptococcal (GBS) septicemia in infants born at the Karolinska Hospital 1975–1986.

3.2 Obstetrical data in the early onset cases (table I)

Twelve (31%) of the 39 mothers were delivered by cesarean section compared to an over-all incidence of 14% ($p < 0.01$; Chi Square Test, one tail with Yates correction). The indications for cesarean section were preterm labor and breech position (n = 5), slow progress of labor (n = 3), fetal asphyxia (n = 3) and placenta previa (n = 1). Remarkably, 10 (26%) of the women were delivered by vacuum extraction, compared to a usual frequency of 12% ($p < 0.01$; Chi Square Test, one tail with Yates correction). Fever (> 38 °C) was registered in nine (23%) of the mothers before, during or after delivery. Eight (21%) of the women, five of whom were delivered by vacuum extraction and three by cesarean section, had clinical evidence of endometritis during the puerperium. Three women who were delivered by cesarean section contracted a wound infection.

3.3 Clinical and laboratory findings in the infants with GBS septicemia (table II)

Early onset septicemia: Twenty-two (55%) of the 40 infants with early onset septicemia were preterm. Nineteen (48%) were born after prolonged rupture of membranes defined as > 12 hours between the rupture of membranes and delivery. The corresponding figures of prematurity and prolonged rupture of membranes for all deliveries in the hospital were 7.2% and 12%, respectively.

Table II. Neonatal characteristics and events

	All n = 40	Preterm infants		Term infants	
		Survivors n = 15	Deaths n = 7	Survivors n = 16	Deaths n = 2
Gestational age (weeks); mean (range)	35.2 (25–42)	31.7 (26–36)	30.6 (25–34)	39.7 (37–42)	41
Birth-weight (g); mean (range)	2589 (700–4480)	1986 (910–3360)	1490 (700–2100)	3577 (2660–4480)	3265
No. of infants < 1500 g (%)	10 (25)	7 (46.7)	3 (42.8)		
Rupture of membranes > 12 hours (%)	19 (47.5)	8 (53.3)	1 (14.3)	8 (50)	2 (100)
1 min Apgar score < 7 (%)	12 (30)	5 (33.5)	5 (71.4)	2 (12.5)	0
Respiratory distress (%)	31 (79)	11 (73)	7 (100)	11 (69)	2 (100)
Boys/girls (ratio)	22/18 (1.22)	7/8 (0.88)	3/4 (0.75)	11/5 (2.2)	1/1

Taken together, prematurity and/or prolonged rupture of membranes were present in 33 (83%) of the infants and in all with a fatal outcome.

Twenty-six (67%) of the infants had onset of symptoms within six hours after birth. Pulmonary involvement with respiratory distress and radiological changes corresponding to pneumonia or hyaline membrane disease (HMD) were seen in 31 (79%). Twelve (30%) of the infants had a fulminant course with shock.

One of the preterm infants had evidence of persistent fetal circulation. Whereas all CSF-specimens were culture negative, GBS was recovered from one infant at day 8 of life in specimens from the hip joint. This infant, treated with penicillin since the second day of life, showed osteomyelitis clinically and radiologically.

A total leukocyte count of $< 5 \times 10^9$/L was seen in six of the 35 (17%) infants, whereas a neutrophil count of $< 1 \times 10^9$/L was seen in three of 34 (9%). Five of 35 (14%) infants had platelet counts of $< 100 \times 10^9$/L. Initial C-reactive protein above 15 mg/L was found in 14 of 27 (52%) infected infants in whom this test was performed. GBS-antigen in serum was detected in five of the nine tested (55%) infants.

The fatality rate was 22%. Seven (32%) of the preterm infants died versus two (11%) of the term infants (N.S. $p > 0.05$).

Late onset septicemia: Both mothers were GBS-carriers and were treated with antibiotics during delivery due to fever. Both infants were diagnosed as having GBS-pneumonia (without septicemia) during the neonatal period. One infant born at term was readmitted at six weeks of age with signs of meningitis and a CSF positive for GBS. The other infant born at 26 weeks gestation with a birth-weight of 907 g has an episode with GBS-septicemia at six weeks of age.

Bacteriological and immunological findings: The GBS-strains from 30 of the infants with early onset disease were available for serotyping: 14 (47%) belonged to type III, 6 (20%) each to types Ia and Ib, and 4 (13%) to type II. The strains from seven of the nine fatal cases were serotyped: 4 belonged to Ia, and 3 to III. The late onset cases were caused by serotype II (meningitis) and Ib (not present in CSF). Among the 27 mothers from whom sera were available, 24 (89%) were very low in serum levels of IgG antibodies against the infecting type of GBS (figure 2) compared to mothers colonized with GBS but giving birth to healthy infants. Three mothers had normal levels. Among the latter three, one was delivered prematurely in week 29 (patient No. 1; figure 2), and another with forceps after 34 hours of ruptured membranes (patient No. 2; figure 2). The third was delivered at term without any characteristic abnormalities except for diabetes mellitus (patient No. 3; figure 2).

Outcome of new pregnancies in mothers to infants with GBS-septicemia: In all, 17 mothers went through 24 new pregnancies. In 11 pregnancies,

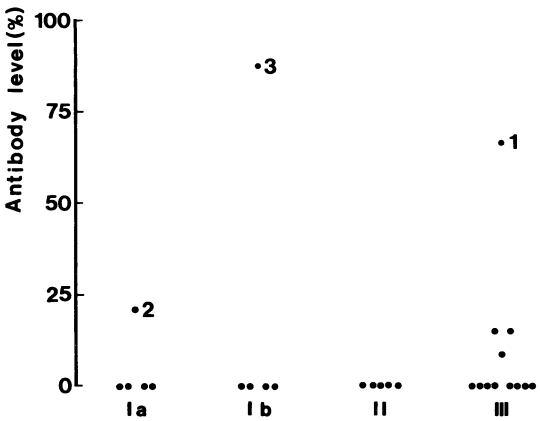

Figure 2. Determination of antibodies against surface antigens of group B streptococci in sera from mothers of infants contracting neonatal septicemia. Each serum was tested against the infecting type. Results were expressed according to the formula

$$\frac{P - L}{H - L} \times 100\%$$

where H = the highest radioactive count obtained with 100 sera from normal women, L = the lowest value, and P = the patient's value. I a denotes antibodies against type I a group B streptococci etc. 1, 2 and 3 refer to patients commented in the results.

the mothers were colonized with GBS. Seven received penicillin orally starting at week 28 and three from later in pregnancy, when a colonization was detected. Cultures from the urogenital tract obtained during oral penicillin treatment revealed occasional GBS in three, coliforms in two, *Klebsiella* in two and combined *Klebsiella* and *Escherichia coli* in one patient.

Four of the infants were born preterm (table III). One of these born at another hospital at 25 weeks' gestation with a birth weight of 700 g died from GBS-septicemia; neither the infant nor the mother received antibiotics.

Three infants presented with pulmonary maladaptation (PMA) syndrome. In the remaining infants born after prophylaxis the neonatal course was uneventful.

One of the infants of mothers without antibiotic treatment was colonized with GBS at one site at birth, whereas, four (40%) of the infants in the prophylaxis group were colonized with gram-negative rods at one or more sites. Cultures revealed coliforms in one, *E. coli* in two and combined *Klebsiella* and *E. coli* in one infant.

Table III. Obstetric and infant data in 24 pregnancies of 17 mothers with a previously born infant contracting GBS-septicemia

	No.	%
Pregnancies (n = 24)		
Preterm delivery	4	17
Rupture of membranes > 12 hours	2	18
Cesarean section	3	13
Vacuum extraction	1	4
GBS colonization	11	46
Infants (n = 24)		
Gestation < 37 weeks	4	17
Birth weight < 1500 g	1	4
1 min Apgar score < 7	3	13
Pulmonary maladaptation	3	13
GBS colonization	1	4
GBS septicemia	1	4
Mortality	1	4

Time between pregnancies varied between 15 and 50 months (mean 2 years)

Sera were available from 12 women giving birth to 15 "new" infants, and sera were again obtained during the "new" pregnancy from 11 women. Nine of the 11 showed unchanged serum levels during the "new" pregnancy. Two patients with low antibody levels at the pregnancy resulting in an infant with GBS septicemia showed normal levels the following pregnancy. Both these were delivered at term in the "new" pregnancy but prematurely in the previous.

4 Discussion

The present Swedish panorama of GBS-septicemia differed from that reported from the Southern part of USA. Late onset GBS-septicemia constituted only 5% (2/42) of the registered infections whereas dominance of the late onset form was reported from Alabama [21], Dallas [15] and Houston [2]. Furthermore, our two late onset infections were caused by serotypes I b and II, in contrast to the total dominance of type III reported from the USA [2]. The over-all type distribution was more consistent with the situation in Denmark [17], Holland [7] and Chicago [14].

It has been suggested that late onset GBS-septicemia is due to nosocomial spread of GBS [18]. It is tempting to speculate that the difference between the present results and those reported from

the Southern USA can be explained by differences in hospital hygienic procedures, but could also be explained by differences in colonization rate. In a Swedish study the rate of infant carriage of GBS at discharge from the maternity unit was 13% [11] whereas a Texas study revealed 65% colonization [20]. Late onset meningitis might be a disease which is partially preventable by hygienic measures. This view was further corroborated by the finding that the two mothers giving birth to infants acquiring late onset GBS-septicemia both received antibiotics during the delivery. Since both were GBS-carriers, the treatment may have interrupted a vertical transmission of GBS during delivery while a transmission from mother to infant then could have occurred later. In other words, the two late onset infections may represent "converted" early onset infections.

The attack rate of GBS-septicemia has been reported to 0.02 – 0.6 per 1,000 live births in Europe [19]. The present result of 1.24 per 1,000 live births cannot be explained by admittance of diseased infants from other clinics since only babies born at the hospital were included.

An encouraging finding was that as many as 83% of the early onset GBS infections – and all fatal infections – belonged to one or both of the two risk groups prematurity and prolonged rupture of the fetal membranes. Combined with screening for maternal carriage of GBS during pregnancy, selective prophylaxis during delivery with antibiotics to these risk groups can prevent early onset septicemia [12]. A simple screening method suitable for use at antenatal clinics was recently described [9]. Although 4 – 8 hour methods for GBS screening are available [22] screening at the obstetrical ward cannot be recommended because it will delay the treatment 4 – 8 hours. A high proportion of the infants (67%) showed symptoms within six hours after birth.

The finding that mothers with preterm delivery also showed low levels of IgG antibodies against the infecting type has not been reported previously to our knowledge. This raised the question whether low levels of IgG to GBS combined with urogenital carriage of GBS may induce premature delivery. In support of this view, an association between GBS-carriage and prematurity was recently reported [20]. As many as 55% of the infants were born before pregnancy week 37 among the 40 early onset infected babies whereas only two of 10 receiving penicillin prophylaxis in the "new" pregnancy were delivered prematurely. In this context it was of interest that penicillin treatment recently was shown to prevent premature rupture of membranes in GBS carriers (A. C. Thomsen, personal communication).

The effect of the prevention program designed for "new" pregnancies was difficult to ascertain from the present study. Remarkably one mother gave birth to a "new" baby in pregnancy week 25 acquiring GBS-septicemia.

In conclusion, the present panorama of GBS-sepsis closely resembled that reported from Chicago [6]. The majority of our GBS-sepsis cases appear to be preventable by combined screening during pregnancy and selective antibiotic prophylaxis.

Summary

All cases of early onset group B streptococcal (GBS) septicemia in infants born at Karolinska Hospital 1975 – 1986 were reviewed. GBS-septicemia was diagnosed in 40 infants within the first five days of life. The incidence was 1.24 per 1000 births. Fifty-five percent of the infants were preterm and 48% were born more than or equal to 12 hours after rupture of membranes. Prematurity and/or prolonged rupture of membranes were present in 83% of all neonates with fatal outcome. Case fatality was 22%. Deliveries by both cesarean section (31%) and vacuum extraction (26%) were increased in the mothers when compared to an overall incidence of 14 and 12% ($p < 0.01$). Twenty-four (89%) of 27 mothers had low type specific IgG antibodies against the infecting GBS-serotype. Late onset GBS-septicemia was diagnosed in only two infants during the period.
Seventeen mothers went through 24 subsequent pregnancies. In 11 of those the mothers were colonized with GBS and 10 received penicillin prophylaxis during pregnancy and/or delivery. None of the infants born after prophylaxis were colonized with GBS. Two were born prematurely and all had an uneventful course; whereas one infant delivered at 26 weeks gestation of a colonized untreated mother died of GBS-septicemia. Screening of parturients at risk and selective antibiotic prophylaxis may help to prevent early onset GBS-septicemia.

Keywords: Group B streptococcus, neonatal septicemia, perinatal risk factors.

Zusammenfassung

Neonatale Sepsis bei B-Streptokokken-Infektion – perinatale Risikofaktoren und Bedeutung bei nachfolgenden Schwangerschaften

Wir berichten über alle Fälle von früh einsetzender Septikämie durch Streptokokken der Gruppe B (GBS) bei Kindern, die während des Zeitraums 1975–1986 im Karolinska-Hospital geboren wurden. Die Diagnose wurde bei 40 Kindern innerhalb der ersten fünf Lebenstage gestellt, was einer Inzidenz von 1.24 auf 1000 Geburten entspricht. 55% der Kinder waren Frühgeborene und bei 48% lag der Blasensprung 12 oder mehr Stunden zurück. Die Mortalität betrug 22%, davon waren 83% prämatur und/oder nach länger zurückliegendem Blasensprung geboren. Sowohl Sektiones (31%) wie auch Vakuumextraktionen (26%) waren verglichen mit dem Gesamtkollektiv häufiger (14% bzw. 12%; $p < 0.01$). 24 der 27 Mütter (= 89%) hatten niedrige typenspezifische IgG-Antikörpertiter gegen den infizierenden GBS-Serotyp. Eine spät einsetzende GBS-Septikämie wurde in dem oben genannten Zeitraum lediglich in 2 Fällen diagnostiziert.

17 Mütter durchliefen 24 weitere Schwangerschaften. Bei 11 dieser Frauen wurden GBS-Kolonien nachgewiesen und 10 erhielten während der Schwangerschaft und/oder der Entbindung eine Penicillin-Prophylaxe. Bei keinem der Neugeborenen nach Prophylaxe konnten GBS-Kolonien nachgewiesen werden. 2 Kinder waren Frühgeborene mit unauffälligen Verläufen. Ein Kind von einer unbehandelten, infizierten Mutter wurde in der 26. Woche geboren und starb an einer GBS-Septikämie. Wir meinen, daß eine Screening bei Risikopatientinnen und eine selektive antibiotische Prophylaxe bei der Prävention der früh einsetzenden GBS-Septikämie sinnvoll sein könnten.

Schlüsselwörter: Neonatale Sepsis, perinatale Risikofaktoren, Streptokokken der Gruppe B.

Résumé

Septicémies néonatales à strétocoque du groupe B facteurs de risque périnatal et avenir des grossesses ulterieures

On a passé en revue tous les cas de septicémie à stréptocoque du groupe B (GBS) à début précoce chez les enfants nés à l'hopital Karolinska de 1975 à 1986. Chez 40 enfants le diagnostic de septicémie à GBS a été porté au cours des cinq premiers jours de vie. L'incidence est de 1,24 pour 1000 naissances. 55% des enfants étaient des prématurés et 48% sont nés 12 Heures ou plus après la rupture des membranes. Pour 83% des nouveaux-nés ayant eu une évolution fatale, il existait une prématurité et/ou une rupture prolongée des membranes. La mortalité était de 22%. Chez les mères les taux de césariennes (31%) et de ventouses (26%) étaient supérieurs au taux globaux 14% et 12% ($p < 0,01$). Vingt quatre (89%) des 27 mères étaient porteuses d'anticorps IgG peu spécifiques contre les sérotypes des GBS responsables de l'infection. Ce n'estque chez deux enfants seulement au cours de cette période que l'on a diagnostiqué une septicémie à GBS de début tardif.

17 mères ont eu 24 nouvelles grossesses. Il mères étaient porteuses de GBS et 10 ont reçu une pénicillino-prophylaxie au cours de la grossesse et/ou de l'accouchement. Aucun enfant né après prophylaxien a été colonisé par le GBS. 2 sont nés prématuremment et tous ont eu des suites simples, alors qu'un enfant né d'une mère porteuse et non traitéé à 26 semaines de gestation est mort d'une septicémie à GBS. Le dépistage des patientes à risque et une prophylaxie antibiotique sélective pourraient aider à la prévention des septicémies à GBS à début précoce.

Mots-clés: Facteur de risque périnatal, septicémie néonatale, strétocoque du groupe B.

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Gerd Faxelius
Department of Pediatrics
Karolinska Hospital
S-104 01 Stockholm, Sweden