PULSE OXIMETRY AS A SCREENING TOOL TO DETECT HYPOXIA ASSOCIATED WITH EARLY ONSET NEONATAL SEPSIS IN ASYMPTOMATIC NEWBORNS

By

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Dissertation submitted to

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN

PAEDIATRICS

Under the guidance of

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LIST OF ABBREVIATIONS

LBW- Low Birth Weight
MOD- Mode Of Delivery
LSCS- Lower Segment Caesarean Section
NVD- Normal Vaginal Delivery
PROM- Premature Rupture Of Membrane
HTN- Hypertension
MSLA- Meconium Stained Liquor Amnii
UTI- Urinary Tract Infection
RT UL- Right Upper Limb
RT LL- Right Lower Limb
LT UL- Left Upper Limb
LT LL- Left Lower Limb
CFT- Capillary Filling Time
AGA- Appropriate For Gestational Age
SGA- Small For Gestational Age
PCV- Packed Cell Volume
ANC- Absolute Neutrophil count
N- Neutrophils
L- Lymphocytes
PLT- Platelet count
CXR- Chest Xray
IT Ratio- Immature To Total Neutrophil count
EONS- Early Onset Neonatal Sepsis
PO- Pulse oximeter

ABSTRACT

Background

Neonatal sepsis is a clinical syndrome characterized by signs and symptoms of infection with or without accompanying bacteraemia in the first month of life. The incidence of neonatal sepsis in India is as high as 30 per 1000 live births, and the diagnosis of EOS is often missed due to non specific or absent sign and symptoms.

Hypoxaemia, a reduction of the oxygen concentration in arterial blood, is strongly associated with sepsis and is seen in one- third of septic neonates. Pulse oximeter measures oxygen saturation and is a simple, reliable and accurate method for detecting hypoxaemia.

Objective

i.)To assess the feasibility of using of pulse oximetery as a screening tool to detect Hypoxia associated with early onset neonatal sepsis in asymptomatic newborns and

ii.) To exclude cardiac cause of hypoxia.

Methodology

Hospital based Prospective Observational study was performed. All asymptomatic newborns born after 35 weeks gestational age were screened on two occasions using pulse oximetry. Newborns with oxygen saturation below predefined thresholds(reading below 90%) within 6 hours of life or the repeat readings remained between (90%-94%)within 24 hours of life) were defined as test positive and all these babies underwent a septic screen which included complete blood count, c- reactive protein, blood cultures and chest x ray. Echocardiography was also done in test positive cases to rule out cardiac cause of hypoxia. Test negative newborns were followed up before discharge to ascertain if they were diagnosed to have sepsis.

Results:

A total of 282 eligible newborns were screened. Five (5/282, 1.8%) newborns tested positive and all 5 were diagnosed with probable early onset sepsis. All test negative newborns were followed up till discharge and none had evidence of early onset neonatal sepsis at follow up.

Conclusion:

Early Onset Neonatal Sepsis is one of the major causes of morbidity and mortality among the neonates. Prevalence of EONS in our centre is around **1.8%**. Blood Culture is the gold standard for diagnosis of Early Onset Neonatal Sepsis. Our study has demonstrated that pulse oximetry can be used as a screen for early onset neonatal sepsis in asymptomatic neonates. None of the test negative neonates had Early onset sepsis. Our study shows that using pulse oximetry for screening for sepsis is feasible with 100 % sensitivity. It requires minimal training of the hospital staff and on an average about 5 minutes to complete the process of screening. This study may therefore help formulating guidelines on using pulse oximetry to rule out sepsis and firm basis for future research.

Keywords: Pulse oximetry; neonate/newborn; early-onset neonatal sepsis; screening

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INTRODUCTION

Neonatal sepsis is a clinical condition that is typified by indications and symptoms of infection with or without the associated bacteraemia in the first month of life. [1,2]

It is identified when the characteristics of sepsis are associated with the proliferation of bacteria from one or several locations. [3] The systemic infections of the neonate such as septicaemia, meningitis, arthritis, pneumonia, osteomyelitis and urinary tract infection are included in it.

About 30 lakh neonates die each year within 7 days of birth, the world over. [3,4] Of these, 99% of deaths are in the developing countries. Neonatal sepsis and pneumonia are responsible for a fourth of the total neonatal deaths [4]. The incidence of neonatal sepsis ranges from 1 to 5 cases per 1000 live births.

If the onset of sepsis is within 72 hours of life it is termed as Early-onset neonatal sepsis. It is caused by organisms such as E.coli, Klebsiella and Enterobacter species which may be present in the maternal genital tract or the delivery room and maternity operation theatre, [2].

Majority of neonates with early-onset sepsis manifest as respiratory distress due to intrauterine pneumonia. As the signs and symptoms of sepsis are non specific or are absent it goes undiagnosed on several occasions. Very often, newborns are discharged from the hospital within 24 hours of birth, leading to a delay in the recognition of early-onset neonatal sepsis.[4] Detection of sepsis at an early stage will help to decrease the associated morbidity and mortality.

The decrease in the partial pressure of oxygen in the blood or hypoxaemia is related with sepsis and is a vital risk factor for mortality. It's diagnosis is often missed due to erratic physical signs, difficult clinical identification of cyanosis and lack of diagnostic tools. Pulse oximetry offers a straight forward, dependable and accurate method for detecting

hypoxaemia. Pulse oximetry is used as a screening tool for congenital heart defects in newborns in developed countries [5]. Frequently, neonatal sepsis is identified as a secondary condition at the time of checking for congenital heart defects. The use of pulse oximetry makes it possible to detect hypoxaemia at an early stage and start oxygen therapy, thereby lowering the neonatal and childhood mortality.[6,7] Regular use of pulse oximetry in all asymptomatic newborns can be an important method to decrease neonatal mortality from early-onset neonatal sepsis.

OBJECTIVE OF STUDY

i) To assess the feasibility of using pulse oximetry as a screening tool to detect hypoxia associated with early-onset neonatal sepsis in asymptomatic newborns and

ii) To exclude cardiac cause for hypoxia.

REVIEW OF LITERATURE

The term neonatal sepsis or *sepsis neonatorum* is used to describe any bacterial infection in the blood that is documented by positive blood culture in the first month of life [8]. It may be categorized as early-onset (day of life 0-3) or late-onset (day of life four or later) based on the postnatal age at onset. Of newborns with early-onset sepsis, 85% present within 24 hours of life (median age of onset 6 hours), 5% present between 24-48 hours of life, and a fraction of newborns present within 48-72 hours of life. Premature neonates have a faster onset[9].

Early-onset sepsis is acquired by vertical transmission from the mother from the labour room or operation theatre. Infection can occur via haematogenous, transplacental spread from an infected mother or, more commonly, through ascending infection from the cervix. Many organisms that colonize the mother's genitourinary (GU) tract. These may be acquired by the newborn as it passes through the colonized genitourinary tract at delivery [9]. It usually has a fulminant onset with multisystem involvement and has a higher case-fatality rate than late-onset sepsis [8].

Incidence and mortality

The incidence of neonatal sepsis ranges from 1 to 5 cases per 1000 live births. Before the antibiotic era, neonatal sepsis was largely fatal. Mortality rates decreased significantly after the introduction of antimicrobial agents and with technological improvements in neonatal care. Over the past two decades, the case-fatality rate has declined to approximately 5% to 10% [8].

Microbiology

The bacteria responsible for neonatal sepsis have changed over time. In the United States, gram-positive cocci, including group A *Streptococcus,* were the common pathogens in the pre-antibiotic era. Later, when the use of antimicrobial agents became commonplace, gram-negative enteric bacilli became the most common causative pathogens. In the 1950s and early 1960s, *Staphylococcus aureus* and *Escherichia coli* were the main neonatal pathogens. In the late 1960s, group B *Streptococcus* (GBS) surfaced as a perinatal pathogen, and till date, it continues to be a vital causative bacteria in newborn infections, as is *E. coli*. In the investigation conducted for early-onset sepsis as part of the Active Bacterial Core surveillance in the U.S. over a multistate area during 1998-2000 (248 184 births), it was found that GBS was the contributing bacteria in 41 % cases, *Escherichia coli* in 17% cases, *Viridans streptococci* in 16 % cases, *Enterococcus* species and *Staphylococcus aureus in 4% cases each,* Group D *Streptococcus* in 3 % of cases and *Pseudomonas* species in 2 % of cases, other gram-negative enteric bacilli in 4 % of cases and others in 9 % of cases . A rise in the incidence of *E. coli* and a decrease in GBS has been observed over the last decade. [10]

Other pathogens, including CONS, *Candida* species, and *S. aureus,* are more commonly causative agents in late-onset infection. Community-acquired methicillin-resistant *S. aureus* (MRSA) strains have appeared as a substantial cause of sepsis in neonates hospitalized in the neonatal intensive care unit since birth. Going by history, there is likelihood of the causative agents of neonatal sepsis to change further. Hence, continued monitoring is necessary. [10]

Transmission

Early-onset neonatal sepsis is transmitted in the following ways:

a. Vertically

1. By ascending amniotic fluid infection: Ascending intra-amniotic infection followed by aspiration of infected amniotic fluid can lead to systemic neonatal infection, especially in the face of prolonged rupture of membranes (ROM). On an average, 1% to 4% of newborns born to mothers with intra-amniotic infection develop systemic infection [8].
2. By delivery through an infected or colonized birth canal: Neonatal infection can also be caught during vaginal delivery from bacteria colonizing maternal lower genital tract. [8]

b. Non-vertically

1. Inadequate handwashing by the nursing personnel can enhance the transmission of microorganisms from an infected to an uninfected infant or from the hands of colonized staff to the neonate [8].
2. The use of instrumentation, such as endotracheal tubes, nasogastric feeding tubes, umbilical catheters, central venous catheters, and urinary catheters, dramatically enhances the risk of neonatal infection [8].

Risk Factors

a. Maternal risk factors

Maternal factors can affect the progression of systemic bacterial infection in the neonate. Phares et al. [11] have reported that blacks have a higher overall incidence of neonatal GBS infections than other racial groups, though the reasons are unclear. Maternal factors, such as malnutrition and sexually transmitted diseases, can also raise the chances of infection. Maternal colonization with GBS is a well-established risk factor for neonatal sepsis.

Colonisation during the third trimester in an uncomplicated pregnancy carries roughly a 1% risk of infection if the acquisition by the newborn is not prevented by intrapartum antibiotic prophylaxis; this risk is increased if colonization is due to prematurity, maternal fever, or prolonged ROM. Asymptomatic bacteriuria has been linked to premature birth. Colonization with genital mycoplasmas has been linked to low birth weight. [8]

b. Peripartum risk factors

Some peripartum factors responsible for an enhanced risk of neonatal infection include untreated or incompletely treated maternal focal infections (including urinary tract, vaginal, or cervical infections) and systemic infections, such as maternal septicemia or maternal fever without a focus. Uncomplicated ROM lasting more than 24 hours involves a 1% risk of neonatal sepsis above the baseline rate of 0.1% to 0.5%. The peril of infection increases four-fold if prolonged ROM coexists with chorioamnionitis. Prematurity and low birth weight are associated with an increased incidence of sepsis. [8]

c. Neonatal risk factors

Although no significant gender difference has been documented for infections acquired in utero, it was observed in the 1960s that male sex had a higher incidence of neonatal sepsis than female sex infants, probably associated with X-linked immunoregulatory genes. Metabolic disorders can predispose to infection. [8]

d. Other risk factors

It has been suggested that bottle-feeding can predispose to infection as the prepared formulas lack several important biologic factors found in colostrum, such as bacterial agglutinins and iron-binding proteins, which have a local gastrointestinal protective effect

against gram-negative enteric bacilli. Breast milk also contains components which help in immunologic defence such as immunoglobulins, macrophages, and lymphocytes [8].

Diagnosis

Symptoms and signs

The signs and symptoms of neonatal sepsis are frequently nonspecific. The body temperature of an infant with sepsis may either be elevated, depressed, or normal. Respiratory signs, including cyanosis or apnoea, are observed by most infants, though not all. Other non-specific signs such as feeding difficulties or lethargy may be subtle or insidious. A high degree of suspicion is needed to pinpoint and evaluate at-risk infants. [8]

Clinical manifestations

Although febrile infants are given medical attention, fever is not a finding specific for infection. Several non-infectious processes such as dehydration, drug withdrawal, and extensive hematomas can result in fever [8].

Palazzi et al. [12] studied the relation between fever and presentation of neonatal sepsis and concluded the following:

- Fever is uncommon in full-term infants. If there is an occurrence of elevated temperature only once, it is not likely to be associated with systemic infection.
- Fever that lasts for 1 hour or more is most likely related to infection.
- Fever without other signs of infection is not very common.
- A single reading of temperature should not be taken alone as an indication of infection, but a series of temperatures along with newborn's clinical status should be considered. [12]

Septic infants can present with neurologic findings such as seizures and full fontanelle even in the absence of meningitis. Gastrointestinal symptoms include hepatomegaly,

abdominal distention, vomiting, diarrhoea, guaiac-positive stools, and jaundice.

Besides jaundice, other cutaneous manifestations are uncommon in neonatal sepsis. Focal infections like cellulitis, impetigo, soft tissue abscesses, omphalitis, conjunctivitis, otitis media, meningitis, and osteomyelitis can develop prior to, or accompany neonatal sepsis. The presence of certain focal infections is suggestive of the causative agent, such as streptococci in case of cellulitis, staphylococci in case of abscesses, and *Pseudomonas aeruginosa* in case of necrotic skin lesions. Complications of neonatal sepsis include metastatic foci of infection, disseminated intravascular coagulation, congestive heart failure, and shock [8].

Differential diagnosis

Because the signs and symptoms of neonatal sepsis are nonspecific, non-infectious etiologies should be considered in the differential diagnosis. Sepsis with or without pneumonia can show as respiratory distress, transient tachypnea of the newborn, and meconium aspiration. Central nervous system (CNS) symptoms can be caused by sepsis and meningitis, and by intracranial haemorrhage, drug withdrawal, and inborn errors of metabolism. Intestinal obstruction, gastric perforation, and necrotizing enterocolitis can manifest with some of the gastrointestinal signs and symptoms that are seen with sepsis. Some nonbacterial infections, such as disseminated herpes simplex virus (HSV) infection, can be indistinguishable from bacterial sepsis and must be considered in the differential diagnosis. [8]

Microbiologic tests

Cultures and buffy coat examination are the two microbiological tests available for the detection of sepsis, each having their limitations. They are as given below:

1. Cultures

A definitive diagnosis of neonatal sepsis can be made only with a positive blood culture. Blood, urine, and CSF should be obtained from all infants suspected to have sepsis. The ideal number of blood cultures and the volume of blood per culture have not been established for neonates. Obtaining more than one blood culture can help distinguish blood culture contaminants from true pathogens. A minimum of 0.5 mL of blood per bottle is recommended. Several manual and automated methods are available for detecting growth in the blood culture medium. Many new automated radiometric techniques can detect growth 8 hours after collection, and almost always within 24 to 48 hours after collection.

The yield from a urine culture is low in early-onset sepsis and very often signals spread to the bladder in the setting of bacteraemia. CSF should be obtained before antibiotic administration and sent for blood cell count, differential, and chemistry determinations, and for Gram stain and culture. Although debatable, some authorities believe that lumbar puncture may be postponed or excluded from the evaluation of an infant with suspected early-onset disease manifested by pneumonia. Meningitis is seen along with sepsis in roughly 10% of infants with early-onset disease and more often with late-onset disease. Meningitis cannot be diagnosed or excluded solely based on symptoms, and blood cultures can be sterile in 10% to 15% of infants with early-onset meningitis and in one-third of infants of VLBW with late-onset meningitis. Other cultures should be obtained, as indicated by clinical findings. Cultures of tracheal aspirates should be obtained in intubated newborns with a clinical picture which points to pneumonia, or when the characteristics and volume of the secretions are altered significantly and are consistent with a pneumonic process. However, an extensive number of cultures of tracheal secretions can often be difficult to interpret. Aspirates or biopsy specimens of skin and soft tissue lesions can be sent for stains and cultures. If a bone or joint infection is suspected, evaluation of aspirated material is vital to establish the diagnosis and

determining the susceptibility of the infecting pathogen. Stool cultures assist in the diagnosis of neonatal septicaemia caused by enteric pathogens such as *Shigella, Salmonella,* and *Campylobacter,* but most often the bacteria recovered from stool cultures reflect gastrointestinal colonization, rather than infection. Cultures of gastric aspirates obtained on the first day of life signal amniotic fluid infection and do not forecast the development of neonatal infection. [8]

2. Buffy Coat Examination

Leukocyte smears made from the buffy coat layer of centrifuged, anticoagulated blood can be stained with Gram stain and methylene blue or with acridine orange, then examined microscopically for intracellular bacteria. A positive buffy coat smear endorses the diagnosis of sepsis and identifies the morphologic and Gram stain characteristics of the organism, but does not pinpoint the infectious agent or include or exclude other foci of infection. Buffy coat examination is infrequently used since the introduction of automated blood culture monitoring systems. [8]

Antigen detection assays

Immunoassays that detect bacterial cell wall or capsule carbohydrate antigens in body fluids are an add-on to diagnosis. Several studies have shown that antigen tests are an inadequate substitute for correctly performed bacterial cultures in the diagnosis of neonatal sepsis. With the available radiometric blood culture technology, rapid antigen testing is now infrequently required or indicated. Besides, these tests can provide misleading results. The only specimens recommended for testing with these devices are serum and CSF. A positive result should be taken to indicate the presence of antigen and not the presence of viable organisms. [8]

OTHER LABORATORY TESTS

1. Leukocyte Counts

Various characteristics of the leukocyte count have been examined for their value in predicting the diagnosis of sepsis. Leucocytosis and leukopenia, defined as more than 20,000/ mm^3 (leucocytosis) and less than 5000/mm^3 (leukopenia), have proven insensitive and nonspecific. A single leukocyte count obtained shortly after birth is not sufficiently sensitive for diagnosing sepsis. [8]

The total neutrophil count has been examined for its value in predicting the presence of infection. Neutropenia, especially if it occurs in the first hours of life and is associated with respiratory distress, has a strong association with early-onset GBS sepsis. Nevertheless, many non-infectious processes such as maternal hypertension, stressful labour, asphyxia, meconium aspiration syndrome, prolonged crying etc. are also linked either with neutropenia or with neutrophilia. Many infants with documented sepsis have normal total neutrophil counts at the time of the initial evaluation. Hence total neutrophil count is not a very specific predictor of sepsis. [8]

The absolute total immature neutrophil count has also been extensively studied. All newborns, but especially premature infants, have a relatively large number of immature neutrophils in the first few days of life. Infected neonates can have an increase above the upper limits of normal in immature cells released from the bone marrow in response to infection, but this response is inconsistent and sometimes delayed, and is an insensitive marker for the early diagnosis of infection. It is unusual, however, for uninfected infants to have an elevated absolute total immature neutrophil count above the reference ranges; if such a finding is present, further evaluation for occult infection should be considered. [8]

The I: T ratio (immature neutrophils: total neutrophils) has been investigated as an early predictor of sepsis. The maximal I: T ratio in uninfected neonates is 0.16 in the first 24 hours, decreasing to 0.12 by 60 hours. The upper limit of normal I: T ratio for neonates of 32 weeks' gestation or less is slightly higher, at 0.2. The test has an excellent negative predictive value; that is, there is a high likelihood that infection is absent if the I: T ratio is normal. Most infected neonates have an elevated I: T ratio some time during the infection, so repeatedly normal I: T ratios can be reassuring. The value of this test is restricted because many non-infectious processes, including prolonged induction with oxytocin, labour, and even prolonged crying, are associated with increased I: T ratios. [8]

2. Determination of Acute-Phase Reactants

The acute-phase response is a response of the body to infection or trauma clinically revealed by malaise, anorexia, fever, leucocytosis, negative nitrogen balance, and hepatic production of acute-phase proteins. Acute-phase reactants (APRs) are proteins produced by hepatocytes in response to inflammation. The inflammation can be secondary to infection, trauma, or other processes of cellular destruction. There are many different APRs, including C- Reactive Protein, fibrinogen, α1-acid glycoprotein, α1-antitrypsin, and elastase α1-proteinase inhibitor. These APRs have different plasma half-lives and different incremental responses to inflammation. The method for the detection of APRs has improved with the development of rapid, automated, quantitative specific immunoassays. Numerous studies have evaluated APRs as early indicators of neonatal septicaemia; an elevated APR does not differentiate between inflammation caused due to infectious and non-infectious origins. [8]

3. Erythrocyte Sedimentation Rate

The erythrocyte sedimentation rate (ESR) is not a direct measure of an APR, but instead reflects changes in many serum protein APRs. A micro-ESR has been developed for use in infants. An approximation of the maximal normal rate in the first two weeks of life can be

obtained by adding 3 to the age of the newborn in days. Beyond two weeks of life, the maximal rate varies between 10 and 20 mm/h. Owing to inter-laboratory variation, each laboratory must develop its reference range. The ESR is limited in that other factors unrelated to inflammation (e.g., anaemia, hyperglobulinemia)can affect the rate. Micro-ESR values vary inversely with the hemaetocrit but are affected little, if at all, by birth weight or gestational age. Slightly elevated micro-ESR values can occur with superficial infections and with non-infectious processes, including asphyxia, aspiration pneumonia, and respiratory distress syndrome. Markedly elevated values in the absence of infection are unusual but have been observed with Coombs-positive haemolytic disease and physiologic hyperbilirubinemia. The long delay after onset of the inflammatory process before the peak ESR is reached and its long half-life renders it of limited usefulness in monitoring the progress of bacterial infections in neonates. [8]

4. Determination of Fibrinogen

Plasma fibrinogen concentrations are known to increase in association with infection, although some factors can result in a low fibrinogen level despite severe infection, including disseminated intravascular coagulation, exchange transfusion, and respiratory distress syndrome. Measurement of fibrinogen concentrations is not useful in the early diagnosis of infection because there is considerable overlap in values between infected and healthy infants. [8]

5. Determination of Fibronectin

Fibronectin is a multifunctional, high-molecular-weight glycoprotein produced primarily by the liver and endothelial cells, and widely distributed in the body, including in plasma and body fluids, on cell surfaces, and in the extracellular matrix. Fibronectin is involved in haemostasis, vascular integrity, and wound healing. It is vital in embryogenesis,

directing cell migration, proliferation, and differentiation. Fibronectin aids in the immune response by boosting macrophage and neutrophil phagocytosis and acting as a nonspecific opsonin for the reticuloendothelial system. The plasma fibronectin concentration varies with age. In healthy neonates, it is approximately half that found in adults, whereas healthy premature infants have approximately one-third of the amount in normal adults. After birth, the plasma concentration gradually increases, reaching adult values by two months of age. Fibronectin is decreased in neonates with infection and in neonates with asphyxia, respiratory distress syndrome, and bronchopulmonary dysplasia. [8]

6. Cytokines

Cytokines such as IL-1β, IL-6, IL-8, TNFα, and others are endogenous mediators of the immune response to inflammation. There is evidence that measuring cytokine concentrations can be helpful in the early diagnosis of neonatal sepsis, but the study design employed and the method for each assay can affect the reported performance. According to Edwards [8], Girardin and colleagues have reported that assay performance is excellent. However, he goes on to report that Buck and coworkers have not got very promising results in their evaluation of the use of IL-6 and CRP measurements in the diagnosis of early-onset sepsis. Many investigators have evaluated colony-stimulating factors in the neonatal period. Data are conflicting but suggest that concentrations of granulocyte colony-stimulating factors vary with gestational age and are influenced by the mode of delivery, nutritional status, maternal hypertension, maternal glucocorticoid therapy, and infection. A peak in granulocyte colony-stimulating factor concentration was observed approximately 7 hours after birth in healthy newborns, with a corresponding increase in the total neutrophil count 7 to 12 hours after birth. Infected newborns had a much higher peak concentration at 7 hours than uninfected infants. [8]

7. Screening Panels

Excluding cultures, none of the previously mentioned tests, when used alone, is sensitive or specific enough to diagnose or exclude neonatal sepsis reliably. Hence several investigators have suggested the use of a combination of several different tests for diagnosing sepsis.

As reported by Edwards [8], Krediet and colleagues measured daily CRP values and I: T ratios in all newborns admitted to the nursery—185 patients during the first four days of life and 107 infants after the fourth day of life. A sepsis workup, including cultures, complete blood count, and radiologic studies, was performed as clinically indicated. For early-onset disease, the positive predictive value of either test alone was 18% to 23%, and the negative predictive value was 95% to 98%. When the tests were used together, the positive predictive value increased to 32%, and the negative predictive value decreased to 95%. These researchers concluded that a screening panel comprising CRP and the I: T ratio had limited value. [8]

Tegtmeyer and coworkers [13] assessed a screening panel in 74 infected neonates. Their panel comprised CRP, I: T ratio, granulocyte count, and $E\alpha_1$-PI. Elastase is a highly active proteinase, and a major constituent of either azurophilic or primary granules of neutrophilic granulocytes, released during the inflammatory response, Complexed with its major inhibitor $alpha_1$−proteinase inhibitor ($E\alpha_1$−Pl), it is an indicator of granulocyte activation during bacterial infection. Elevated Eα−PI concentration in tracheobronchial aspirates is a helpful indicator of local inflammation before it is detectable by a systemic inflammatory response. The sensitivity of CRP and I: T ratio test done alone ranged from 36% to 70% except for $E\alpha_1$-PI, which ranged from 87% (early-onset sepsis) to 100% (late-onset disease). When all the tests were used together, the sensitivity increased to 100%. Because the authors assessed only

infected infants, they were unable to determine the specificity or predictive accuracy of their screening panel.

As reported by Edwards, [8] Philip and coworkers evaluated the predictive accuracy of a three-part screen ($E\alpha_1$-PI, I: T ratio, and CRP) in more than 300 infected and uninfected infants admitted to the neonatal intensive care unit. When used alone, $E\alpha_1$-PI was more sensitive than the other two tests for diagnosing sepsis but had a lower positive predictive value. When the three tests were used as a panel, the sensitivity was 23%, the specificity was 99.7%, the positive predictive value was 87.6%, and the negative predictive value was 99.7%. The use of screening panels does not significantly improve the positive predictive accuracy; however, the predictive accuracy of a negative panel often increases to 98% to 100%, and a panel with this accuracy of performance in excluding disease can provide useful information[8].

Pulse Oximetry

All the methods stated above have their limitations, and none of the available laboratory tests can be considered as an ideal marker. However, there is evidence to link sepsis with an early onset of hypoxia (reduction of the oxygen concentration in arterial blood [14] . Microcirculatory dysfunction plays a vital role in the pathogenesis of sepsis and septic shock [15]. It is known that cytopathic hypoxia occurs in the mitochondria within cells when sepsis occurs [16]. Besides, functional shunting in the microcirculation and in the mitochondria, which lead to the deficit of oxygen extraction is also observed in sepsis and septic shock [15,17]. Hypoxia results in apparent cyanosis; however, mild degrees of hypoxia cannot be detected by clinical observation, even by experienced clinicians. The difficulty is exacerbated in infants with pigmented skin [18]. Hypoxia can be diagnosed by measuring haemoglobin oxygen

saturation either by invasive blood sampling or by pulse oximetry (PO) [19]. Pulse oximetry was developed as a non-invasive method to determine arterial oxygen saturation (SpO2) and has been widely used in intensive care, operating theatres and emergency units for over 30 years. The ability to detect the different absorption spectra oxygenated and deoxygenated haemoglobin allow pulse oximeters to measure the amount of oxygen-saturated haemoglobin in the capillaries of an extremity, such as a finger or an ear lobe in an adult, or a hand or foot in a baby. Pulse oximetry thus allows the detection of hypoxaemia that would not necessarily produce visible cyanosis; the technique correlates well with arterial blood gas measurements56 and does not require calibration [18].

The fetus is hypoxic in utero, with oxygen saturations of 30–60%. The events that follow delivery – clamping of the umbilical cord, initial inflation of the lungs allowing pulmonary gas exchange and an increase in pulmonary circulation – result in a rapid rise in the arterial oxygen tension. The mean pre-ductal (right hand) and postductal (foot) saturations rise to 73% and 67%, respectively, within the first 2 minutes after birth, and to 92% and 89% by 10 minutes. The difference between right hand and lower limb saturations reflects right-to-left shunting across the ductus arteriosus, and this generally diminishes with time in most infants, with both pre- and postductal saturations reaching 95% by 1 hour of age. Saturations are generally stable in the first 24 hours with a mean of 98% (similar to values obtained from older neonates); however, periods of desaturation may occur, and more extensive studies have shown that many healthy newborns (up to 5%) may have episodes of saturation of < 95% within the first 24 hours [18].

Based on these findings, studies were conducted to evaluate the possibility to use pulse oximetry in detecting hypoxaemia associated with congenital heart disease (CHD) and

other conditions in seemingly healthy newborns. Several papers have been published that have used pulse oximetry as a screen for CHDs in this group. [20,21,22,5]

History of Pulse Oximetry

Takuo Aoyagi, a biomedical engineer, working for the Shimadzu Corporation in Kyoto, Japan, invented pulse oximetry in the early 1970s. While studying methods of measuring cardiac output, he discovered the spectrophotometric measurement principles of pulse oximetry by serendipity. As Pulse oximetry (PO) offered continuous monitoring of the patient's physiological status, its use spread rapidly and extensively [23]. Pulse oximetry technology was extensively introduced in the United States in the early 1980s. The initial application of pulse oximetry was in perioperative care, but it soon expanded into neonatal, paediatric, and adult intensive care units (ICUs). By 1989 there were 29 manufacturers producing 45 different models of oximeter [24].

Measurement Principles

Pulse oximetry determines arterial oxygen saturation by measuring the absorption of light (of 2 wavelengths, approximately 660 nm and 940 nm) in human tissue beds emitted by the sensor attached to the patient's body. As light transverses human tissue, it is absorbed in varied proportions by tissue, blood vessels, fluids, bone, skin, arterial blood and venous blood including various types of haemoglobin. The absorption of light is altered as the quantity of blood in the tissue bed is altered and as the proportional amounts of oxygenated and deoxygenated haemoglobin change.

Oxygenated haemoglobin permits red light to pass through, but absorbs infrared light, while deoxygenated haemoglobin permits infrared light to pass through and absorbs more of the red light. Capturing the differences in light absorption permits estimation of heart rate and

arterial oxygen saturation. For accurate results, the oximeter must discriminate between the background (or constant) absorption and the pulsatile changes in absorption due to the changing blood volume with each heartbeat. The background absorption is altered when there is a change in the position or shape of the tissues through which the light passes, which can result in false readings.

A traditional pulse oximeter measures the ratio of the absorption of 2 wavelengths of light, discriminates the changes that it assumes are due to pulsatile changes and oxygenation changes, takes the mean of the readings over a short period, and then looks up the resulting absorption ratio in a table or calibration curve of corresponding arterial saturation. These calibration tables/ curves are developed from experimentations with volunteers, by comparing simultaneous light absorption readings and blood oxygen values measured via co-oximetry [23]. Pulse oximeter readings are used clinically as a substitute for arterial oxygen saturation and have had a considerable influence on how oxygenation is managed. Continuous monitoring of pulse oximetry is now a de facto standard of care for nearly all infants and children receiving mechanical ventilation or intensive care, and its use is rising in the non-ICU population [23].

Further development in the conventional transmission pulse oximetry is reflectance oximetry. In conventional transmission PO (TPO), a red/infrared light emitter and photo-detector are kept on opposite sides of a narrow tissue segment (e.g. digit, earlobe); the tissue partly absorbs transmitted light, and the detector measures the remaining transmission. To address the distinctive challenges of performing PO in young infants, a less commonly used approach, called reflectance PO (RPO), offers likely advantages. Although PO technology is the same in both TPO and RPO, in RPO single-surface probes are used. Also, the light emitter and detector are next to one another so that the probe can be kept on a flat, more centrally-located skin surface such as an infant's chest. Instead of measuring transmitted residual light, the RPO sensor detects the residual light reflected from the pulsating artery [25].

A significant drawback of conventional Pulse oximetry was its unreliability during patient motion and low perfusion. Conventional pulse oximetry works under the assumption that by looking at only the pulse and normalizing the pulsating signal over the non-pulsating signal, oxygen saturation (SpO2) can be measured without calibration. This assumption has one major flaw–it assumes the only pulsating element is arterial blood. Unfortunately for conventional pulse oximetry, venous blood moves every time the patient moves or breathes. This causes conventional pulse oximeters to display false low or high SpO2 and pulse rates–resulting in false alarms as high as 90% in ICUs and recovery rooms. New technology by the name of Signal Extraction Technology(SET) developed by Masimo Corporation, employs advanced signal processing techniques and assumes that both the arterial and venous blood can move and uses parallel signal processing to separate the arterial signal from sources of noise (including the venous signal) to measure SpO2 and pulse rate accurately, even during motion. In several studies, the signal processing of Masimo SET® consistently resulted in significantly fewer false alarms and true alarm detection [26]. The current study also uses Masimo SET® pulse oximeter.

Figure 1: Masimo SET® pulse oximeter.

Limitations of Oximetry

Pulse oximetry has several well-known limitations, including the effects of ambient light, dyshaemoglobinemia, skin pigmentation, low peripheral perfusion states, and motion artefact. These affect precision, bias, the applicability of the instrument, and clinician confidence in the readings [23].

Ambient light can affect oximeter functioning, but this problem can be overcome by merely wrapping the oximeter probe in opaque material. Skin pigmentation also affects pulse oximeter performance. As skin pigmentation darkens, oximeter performance declines. This could be because the experimental calibration data was derived from predominantly white volunteers [23].

Dyshemoglobinemia also gives erratic pulse oximetry readings, because pulse oximeters are unable to distinguish between oxygenated haemoglobin and the various dysfunctional haemoglobins, such as methaemoglobin and carboxyhaemoglobin, which are unable to bind with and carry oxygen [23].

The limitations of motion artefact and low peripheral perfusion states are taken care of in the newer models of Pulse oximeters.

Studies using Pulse oximetry

Literature review reveals three large scale studies and several smaller ones that used pulse oximetry screening primarily to detect congenital heart diseases.

In a large population-based prospective multicentre study of postductal (foot) arterial oxygen saturation (SpO_2) in apparently healthy newborns in Sweden, about 50,000 subjects were screened. Of the infants screened, 324 (0.6%) failed the test. Of these, 43 (13%) had CHDs (27 critical), and 134 (41%) had pulmonary diseases or other disorders, including neonatal sepsis [27].

A prospective Swedish study in around 40,000 participants in 5 maternity units between July 2004 and March 2007 studied the ability of pulse oximetry to correctly identify duct dependent congenital heart disease. The "false" positive rate for oximetry screening for CHD was 69/39 821 (0.17%). Of the 45% (31/69) of the "false positive" babies detected by pulse oximetry had other significant heart malformation, lung problem, or infection. In terms of benefit derived by early detection of babies with pathology other than duct dependent heart disease, 12% required cardiac surgery, 29% required further follow-up, and that neonatal intensive care was required in ≥5 days for 26% and <5 days in 13%. The study concluded that early detection of sepsis, lung pathology and congenital heart disease requiring surgery is of definite benefit to the baby (all required neonatal intensive care) [28].

In a study in 6 maternity units of United Kingdom, consisting of about 20,000 asymptomatic newborns at ≥ 35 weeks' gestation age, pulse oximeter readings were taken prior to discharge from hospital, and the results of this index test were compared with a composite reference standard (echocardiography, clinical follow-up and follow-up through interrogation of clinical databases). There were 169 false positives while screening for critical CHD, of which 40 cases were of respiratory or infective illness requiring medical intervention (18). This goes to show that pulse oximetry can be used to detect neonatal sepsis which would otherwise have gone undetected at an early stage.

Screening of the newborn for critical congenital heart disease (CCHD) with the help of pulse oximetry is an effective and life-saving strategy in developed countries. While most studies have reported false-positive results during CCHD screening, they have not elaborated on the detected disease types. Jawin et al. [29] studied the effectiveness and outcomes of pulse oximetry newborn screening for non-cardiac hypoxemic diseases such as neonatal sepsis, respiratory diseases, and CCHD in a middle-income country like Malaysia. Fifteen of 5247 term newborns had positive screening results. The median age at screening was 20 h. Thirteen

newborns (0.24%) had significant non-cardiac diseases: sepsis (n = 2) and respiratory diseases (n = 11) that required hospitalization and treatment. The remaining two newborns with normal antenatal ultrasonograms had a positive screening test and confirmed to have CCHD. Another 18 newborns with negative screening test were later admitted for treatment of sepsis (n = 16) and pneumonia (n = 2). All newborns were treated and alive at the end of the study. The sensitivity and specificity of pulse oximetry screening for non-cardiac diseases were 42% and 99.9% respectively, and 100% and 99.7% for CCHD, respectively [29].

In 1995, Maneker et al. [30] made a prospective comparison of blinded, clinical evaluation by physicians with subsequent pulse oximetry readings to determine whether routine pulse oximetry in a paediatric emergency department could be helpful to identify patients with a low oxygen saturation that was unexpected based on clinical evaluation. A convenience sample of 368 patients presenting to the paediatric Emergency Department with respiratory illnesses was considered for the study. The history, physical examination, paediatric ED management, and therapy were recorded. Based on the clinical assessment, the physician was questioned if the patient had a low Sao2(<92%). Room air pulse oximetry was then obtained, with subsequent treatment and management plans recorded. The clinical assessment had a sensitivity of 33%, a specificity of 86%, a negative predictive value of 35%f or detecting children with low oxygen saturation. Unexpected low oxygen saturation usually led to a change in patient management or disposition. The U.S. study concluded that clinical evaluation in a paediatric ED did not screen adequately for the detection of hypoxaemia and should be supplemented by routine pulse oximetry in all patient with respiratory findings.

King et al. [4] undertook a study to evaluate the likelihood of using pulse oximetry as a screening tool to detect hypoxia associated with early-onset sepsis in asymptomatic newborns in Tanzania between January and March 2013. All eligible asymptomatic newborns of

gestational age more than 33 weeks born during the study period were screened twice using pulse oximetry. Newborns with oxygen saturations less than the predefined thresholds were considered test positive. The researchers documented the pulse oximetry measurements, time taken to obtain the readings and the acceptability of pulse oximetry use to mothers and healthcare personnel. The rates of hypoxaemia by pulse oximetry and clinical diagnosis of sepsis in asymptomatic newborns were compared. A total of 316 asymptomatic newborns were evaluated, of which eighteen (5.7%) were grouped as test positive. Sepsis was diagnosed on the basis of clinical exam in 41 newborns (13%). These included eight newborns who tested positive with pulse oximetry screening. A majority of mothers (n=50) and healthcare professionals (n=18) were satisfied with screening. Hence the study concluded that it was practicable to use pulse oximetry as a screening tool to detect early-onset sepsis in a low-income setting. The test was acceptable to mothers and healthcare professionals. Nevertheless, to determine the accuracy of the test measurements in identifying sepsis, further studies were recommended by the authors.

A similar prospective cohort study was done by Swamy et al. [31] in two hospitals in Bangalore, India, between April and June 2013. All asymptomatic newborns born after 35 weeks gestational age were screened twice using pulse oximetry. Newborns who had oxygen saturations below predefined thresholds (reading below 90% or the repeat readings remained between 90 to 94%) were deemed to be test positive, and all these babies underwent a septic screen which included full blood count, C-reactive protein, blood cultures and chest x-ray. Test negative newborns were followed up in the outpatient department to ascertain if they were diagnosed to have sepsis in the first week. A total of 213 eligible newborns were screened. Two (2/213, 0.93%) newborns tested positive, and both were diagnosed with early-onset sepsis. All test- negative newborns were followed up, and none had evidence of early-

onset sepsis at follow up. The study showed that it was feasible to use pulse oximetry to screen for early-onset sepsis in asymptomatic newborns.

METHODOLOGY

Material and methods :

Source of data:

All asymptomatic newborns(more than 35 weeks period of gestation) admitted to Shri B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA, KARNATAKA who meet the inclusion criteria for a period of one and half year.

Types and Duration Type of study:

It is a prospective observational study done for a period of 18 months from November 2018 till May 2020.

Inclusion criteria:

All asymptomatic neonates more than 35 weeks of gestation whoever were delivered (both LSCS and NVD) at Shri B.M. Patil Medical College and Hospital, Vijayapura and whose parents gave the informed consent will be eligible for study.

Exclusion criteria:

Newborns with symptoms and signs of sepsis prior to screening and those admitted to neonatal unit were excluded.

Method:

The primary outcome was detection of hypoxemia secondary to EONS in asymptomatic newborns. A Masimo radical-7 pulse oximeter with a reusable probe M-LNCS Neo was used to measure functional oxygen saturations. The probe was secured using disposable tape to both the right hand and Both foot until a consistent reading

was obtained. The test was performed on the postnatal wards within 6 hours of delivery, 24 hours of delivery and again 1-2 hours before discharge . The process of checking saturations on an average, took less than 5 minutes. If the oxygen saturations in all the four limbs were more than 90% at 6 hours of life and repeat readings of saturation above 94% within 24 hours of life were considered normoxaemic(test negative).

All test negative infants were followed up till the time of discharge to identify those who developed infections. If the saturations were less than 90% in first 6 hours of life or the repeat readings 90% to 94% within 24 hours of life were considered as test positive(hypoxaemia) , and all the test positive newborns underwent a clinical assessment and septic work up with full blood count, C-reactive protein, IT ratio, blood cultures, chest X-ray and 2 D Echo. Any infant who had a serum CRP more than 10mg/dl, I/T Ratio>0.2 or a positive blood cultures or radiological signs of pneumonia on the chest X-ray were considered to have early onset neonatal sepsis.

DATA ANALYSIS

Sample size calculation:

Using expected incidence of sepsis as 30%, expected sensitivity as 99% and expected specificity 85%, and desired precision as 5%, the minimum sample size was 282.

This sample size has given the precision of 5% or less for both sensitivity and specificity.

By using the form ula:

$$n = \frac{z^2 p(1-p)}{d^2},$$

where

Z= z statistic at 5% level of significance

d = margin of error

p= anticipated prevalence rate

Statistical analysis

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation (SD) was used. For categorical data, the number and percentage were used in the data summaries and data were analyzed by Chi square test for association, comparison of means using t test, ANOVA and diagrammatic presentation.

Ethical clearance:

Institutional ethical clearance was undertaken for the study

RESULTS

Distribution of Cases according to Mother's Age

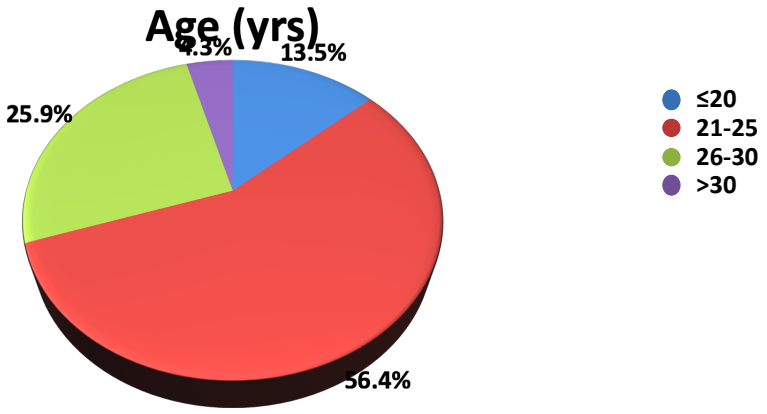
A majority of babies (56.4%) had mothers who were in the age group 21 years to 25 years, followed by 25.9 % babies whose mothers were in the age group of 26 years to 30 years. 13.5% of babies had mothers who were less than 20 years of age, and the mothers of 4.3 % of babies were aged more than 30 years.

Table 1: Distribution of Cases according to Mother's Age

Age (yrs)	N= 282	Percent(%)
≤20	38	13.5
21-25	159	56.4
26-30	73	25.9
>30	12	4.3
Total	282	100

Descriptive Statistics	Min	Max	Mean	SD
Age (yrs)	18	42	24.3	3.7

Figure 2: Distribution of Cases according to Mother's Age



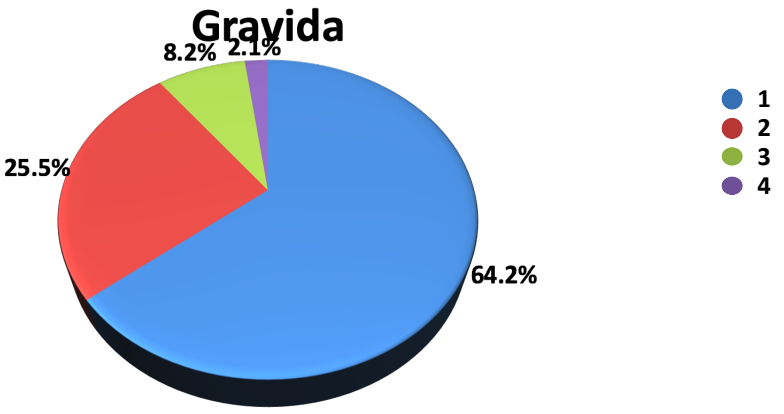
Distribution of Cases according to Mother's Gravida

Out of 282 cases, Primigravida mothers were predominant (64.2%), followed by Gravida 2 mothers (25.5%), followed by gravida 3(8.2%), followed by gravida 4(2.1%).

Table 2: Distribution of Cases according to Mother's Gravida

Gravida	N= 282	Percent
1	181	64.2
2	72	25.5
3	23	8.2
4	6	2.1
Total	282	100

Figure 3: Distribution of Cases according to Gravida



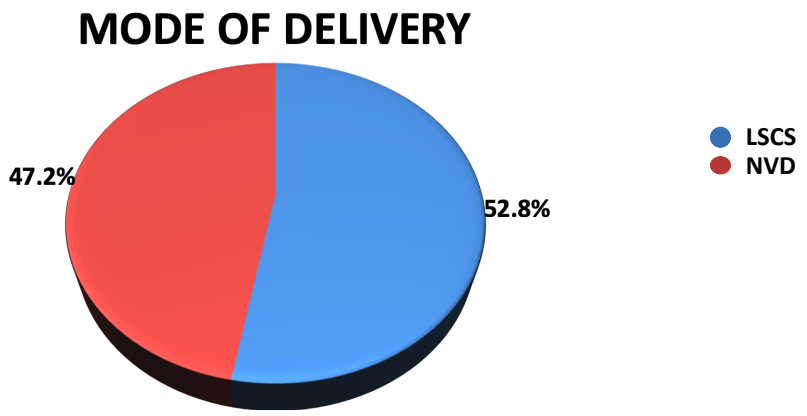
Distribution of Cases according to Mode Of Delivery

52.8 % of neonates were delivered by Caesarean section and 47.2% by normal vaginal delivery.

Table 3: Distribution of Cases according to Mode Of Delivery

MOD	N = 282	Percent
LSCS	149	52.8
NVD	133	47.2
Total	282	100

Figure 4: Distribution of Cases according to Mode of Delivery



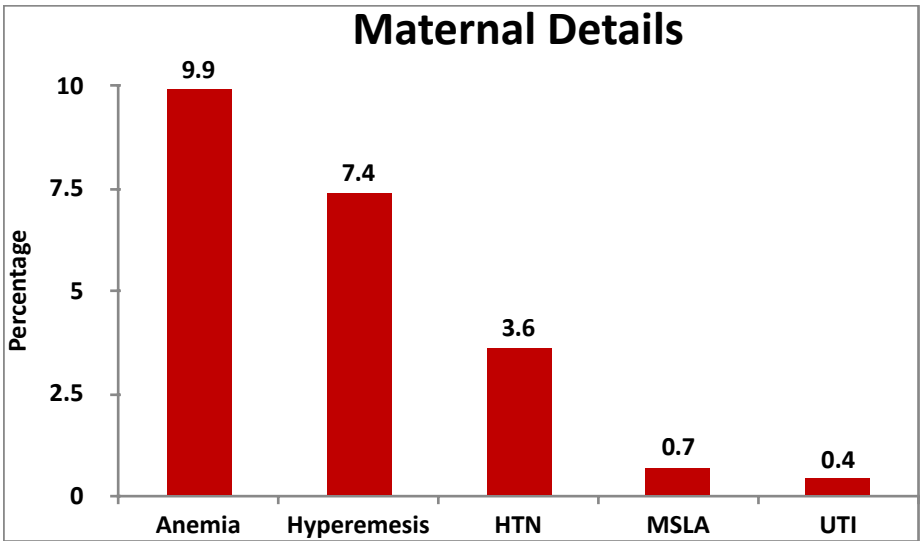
Distribution of Cases according to Maternal Details

Out of 282 cases 28(9.9 %) of neonates had anaemic mothers. Out of 282 cases 21(7.4 %) of neonates had mothers who experienced hyperemesis. 3.6 % of babies had hypertensive mothers, and 0.7 % of babies experienced MSLA during birth. Urinary tract infections were present in 0.4 % of mothers. None of the mothers had a fever with rash, oligohyadraminous or polyhydraminous and fever with rash.

Table 4: Distribution of Cases according to Maternal Details

Maternal Details	N	Percent
Anaemia	28	9.9
Hyperemesis	21	7.4
HTN	10	3.6
MSLA	2	0.7
UTI	1	0.4
Fever with rash	0	0
Oligo/Poly	0	0
GDM	0	0
Heart Disease	0	0

Figure 5: Distribution of Cases according to Maternal Details



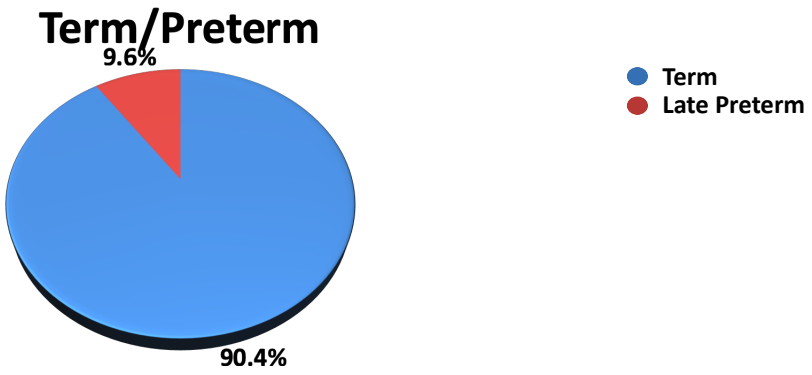
Distribution of Cases according to Term/Late Preterm

90.4 % of babies were Term babies (>37weeks), and 9.6 % were late preterm babies (35-37) weeks.

Table 5: Distribution of Cases according to Term/Late Preterm

Term/Preterm	N	Percent
Term (>37weeks)	255	90.4
Late Preterm (35-37 weeks)	27	9.6
Total	282	100

Figure 6: Distribution of Cases according to Term/Late Preterm



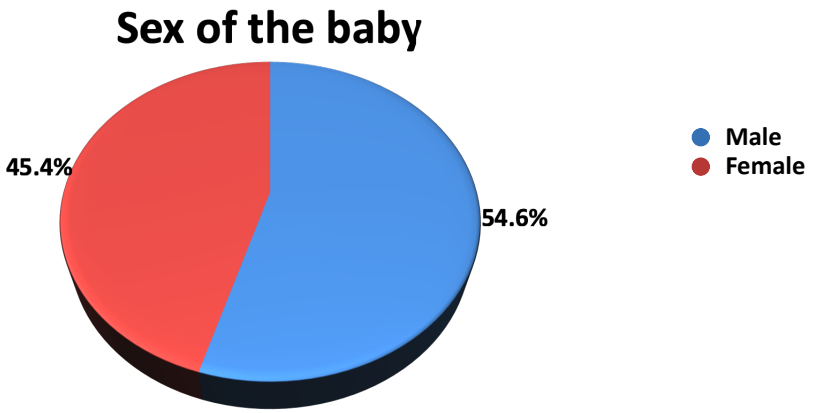
Distribution of Cases according to Sex of the baby

Out of 282 cases 54.6% of babies were male, and 45.4% were female. The male: female ratio was 1: 0.83.

Table 6: Distribution of Cases according to Sex of the baby

Sex of the baby	N	Percent
Male	154	54.6
Female	128	45.4
Total	282	100

Figure 7: Distribution of Cases according to Sex of the baby



Distribution of Cases according to Birth Weight

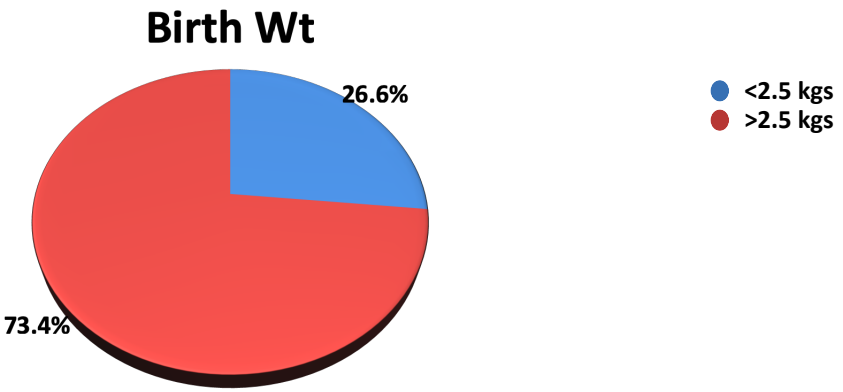
A majority of babies (73.4%) had birth weight > 2.5 Kg. 26.6 % of babies had birth weight < 2.5 Kg. The mean birth weight of the babies was 2.7 kg ± 0.4 Kg, with the minimum being 1.8 Kg and maximum being 3.8 Kgs.

Table 7: Distribution of Cases according to Birth Weight

Birth Wt. (kgs)	N	Percent
< 2.5 kgs	75	26.6
>2.5 kgs	207	73.4
Total	282	100

Descriptive Statistics	Min	Max	Mean	SD
Birth Wt. (kgs)	1.8	3.8	2.7	0.4

Figure 8: Distribution of Cases according to Birth Weight



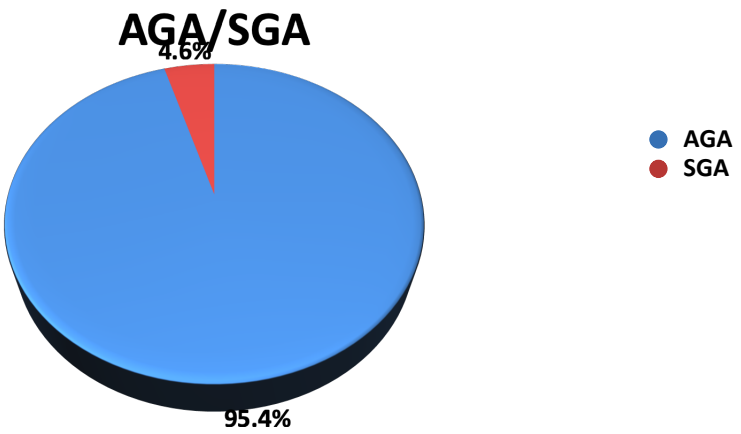
Distribution of Cases according to AGA/SGA

Out of 282 cases 95.4 % of babies were appropriate for gestational age, and 4.6% were small for gestational age.

Table 8: Distribution of Cases according to AGA/SGA

AGA/SGA	N=282	Percent
AGA	269	95.4
SGA	13	4.6
Total	282	100

Figure 9: Distribution of Cases according to AGA/SGA



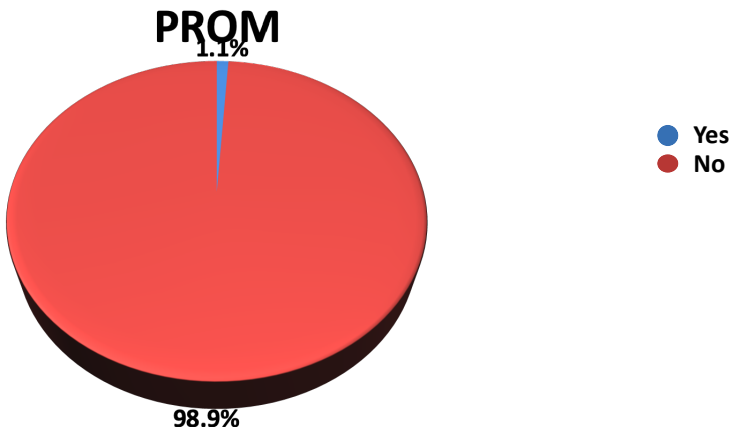
Distribution of Cases according to PROM

Out of 282 cases 1.1% of cases showed premature rupture of membrane.

Table 9: Distribution of Cases according to PROM

PROM	N=282	Percent
Yes	3	1.1
No	279	98.9
Total	282	100

Figure 10: Distribution of Cases according to PROM



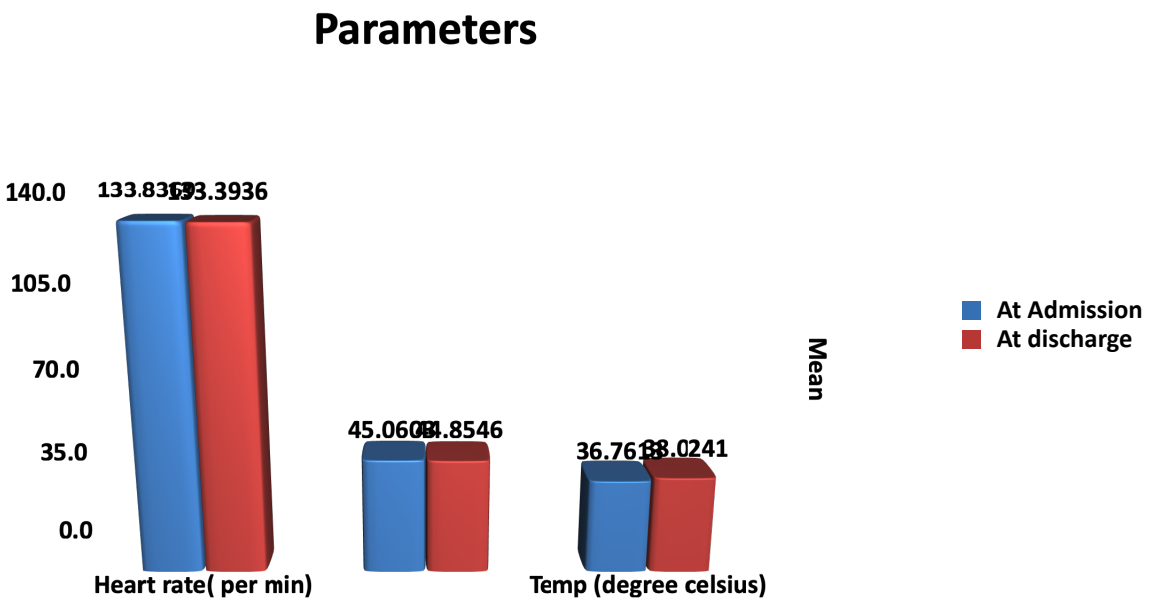
Change in hemodynamic Parameters according to time

This table shows change in heart rate, respiratory rate and body temperature of babies at admission and at the time of discharge.

Table 10: Change in hemodynamic Parameters according to time

Parameters	At Admission		At the time of discharge		p value
	Mean	SD	Mean	SD	
Heart rate(per min)	133.8	9.2	133.4	8.2	0.534
Resp rate(per min)	45.1	4.7	44.9	3.9	0.568
Temp (degree celsius)	36.8	0.3	38.0	19.0	0.267

Figure 11: Change in hemodynamic Parameters according to time



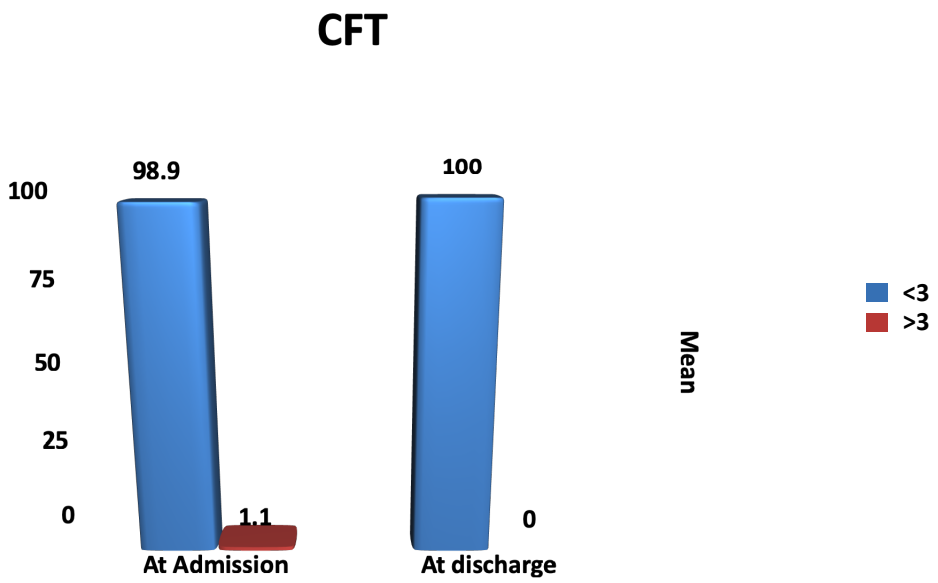
Distribution of Cases according to CFT

Out of 282 cases 3 babies had a CFT > 3 seconds at admission into the study. At the time of discharge, all 282 babies had CFT< 3 seconds.

Table 11: Distribution of Cases according to CFT

CFT(sec)	At Admission		At the time of discharge	
	N	Percent	N	Percent
<3	279	98.9	282	100
>3	3	1.1	0	0
Total	282	100	282	100

Figure 12: Distribution of Cases according to CFT



Distribution of SPO2

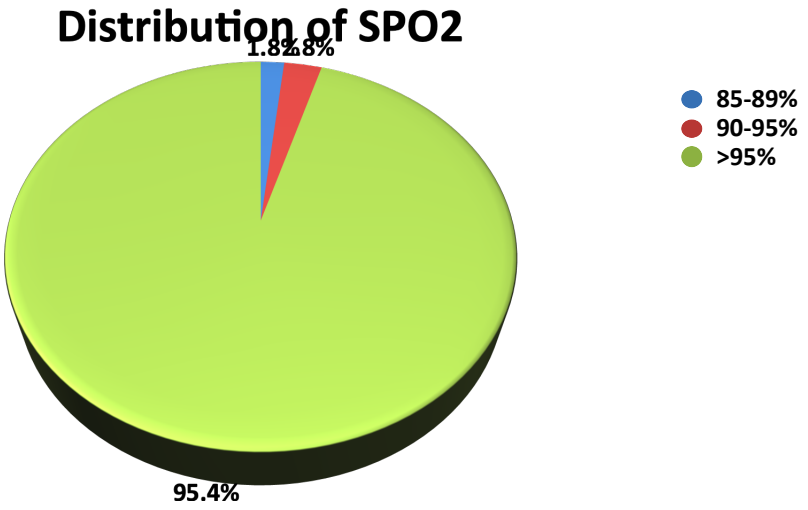
1.8 % of babies (n=5) had oxygen saturation between 85-89 %. We considered this as sepsis- positive population. 2.8% of babies had oxygen saturation between 90- 95 % and 95.4 % of babies had oxygen saturation > 95%. The mean oxygen saturation was 97.5 % ± 1.2 % with the minimum value being 85 % and maximum being 100 % (Table 13 and Figure 14).

Table 12: Distribution of SPO2

SPO2(%)	N	Percent
85-89%	5	1.8
90-95%	8	2.8
>95%	269	95.4
Total	282	100

Descriptive Statistics	Min	Max	Mean	SD
SPO2 (%)	85	100	97.5	1.2

Figure 13: Distribution of SPO2



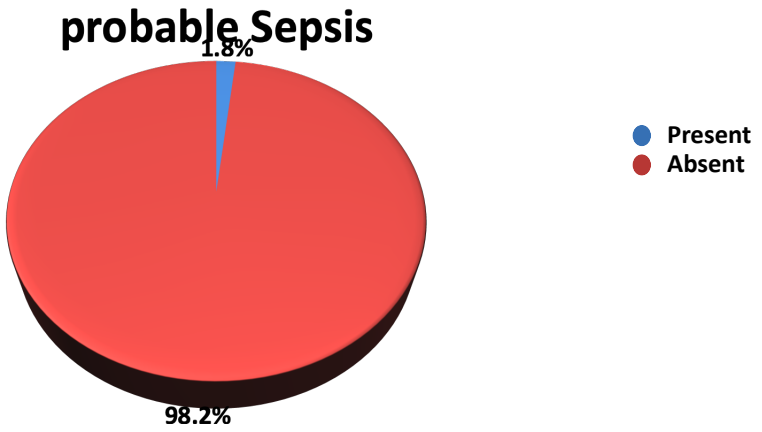
Distribution of Cases according to probable Sepsis

Out of 282 cases 1.8 % of babies (n=5) had probable sepsis, and in 98.2% of babies, there was no Sepsis.

Table 13: Distribution of Cases according to probable Sepsis

Sepsis	N	Percent
Probable sepsis	5	1.8
Absent	277	98.2
Total	282	100

Figure 14: Distribution of Cases according to probable Sepsis



Lab parameters of probable sepsis cases

This table shows the lab parameters of probable sepsis cases. Though the blood culture was sterile in all the cases of probable sepsis, the CRP value was raised (>10 mg/dl) in all the 5 cases of probable sepsis. All the 5 cases had a total WBC count > 20000 cells/ mm^3. I/T ratio > 0.2 gives a clue about early-onset sepsis in newborns in the first 24 hours. In this study, all the 5 cases of probable sepsis had an IT ratio of >0.2.

Table 14: Lab parameters of probable sepsis cases

CASES	CRP(mg/l)	TOTALCOUNT (cells/cmm)	I: T RATIO	ANC($mm^{3)}$	BLOOD C/S
CASE I	44	24600	0.18	2800	STERILE
CASE II	31	28060	0.28	3200	STERILE
CASE III	23	22530	0.25	2400	STERILE
CASE IV	28	20210	0.16	2200	STERILE
CASE V	11	25990	0.24	2600	STERILE

Investigations Of the probable sepsis cases

The chest X-ray was normal in 2/5 cases of probable sepsis, and it was suggestive of congenital pneumonia in 3/5 cases of probable sepsis. Out of the 5 cases one baby had Patent Foramen Ovale (PFO) as revealed by 2D Echo .The blood culture was sterile for all the 5 cases of probable sepsis.

Table 15: Investigations Of the probable sepsis cases

CASES	CHEST XRAY	2D ECHO
CASE I	Normal	Normal study
CASE II	Congenital pneumonia	Normal study
CASE III	Normal	PFO
CASE IV	Congenital pneumonia	Normal study
CASE V	Congenital pneumonia	Normal study

Descriptive Statistics of probable Sepsis Cases

The mean value of the WBC count in the 5 cases of probable sepsis was 24278 cells/ mm^3 ± 3039.0 cells/ mm^3, with the minimum value being 20210 cells/ mm^3 and maximum

value being 24278 cells/ mm^3.In the present study, the mean value of NLR for the 5 cases of probable sepsis was 3.4 ± 2.2, with the range being 1 to 6The mean value of haemoglobin for the 5 cases of probable sepsis was 17.0 gm % ± 1.7 gm %, with the range being 16 gm % to 19 gm %.The mean platelet count was 2.9 lakhs/ mm^3 ± 0.7 lakhs/ mm^3, with the range being 2-4 lakhs/ mm^3 for the 5 cases of probable sepsis. The mean packed cell volume was reported to be 47.3 % ± 5.9 %, with the minimum value being 42 % and the maximum value being 57%. The mean CRP values for the 5 cases of probable sepsis was 27.4 mg/l ± 12.0 mg/l, with the range being 11 to 44 mg/l.

Table 16: Descriptive Statistics of probable Sepsis Cases

Descriptive Statistics	**Min**	**Max**	**Mean**	**SD**
TC(cells/cmm)	20210	28060	24278.0	3039.0
N/L (%)	1	6	3.4	2.2
HB(gm%)	16	19	17.0	1.7
PLT(lakhs/cmm)	2	4	2.9	0.7
PCV(%)	42	57	47.3	5.9
CRP(mg/l)	11	44	27.4	12.0

Distribution of Other Parameters of probable Sepsis Cases

Out of 5 Probable sepsis cases 3 cases had features suggestive of congenital pneumonia.

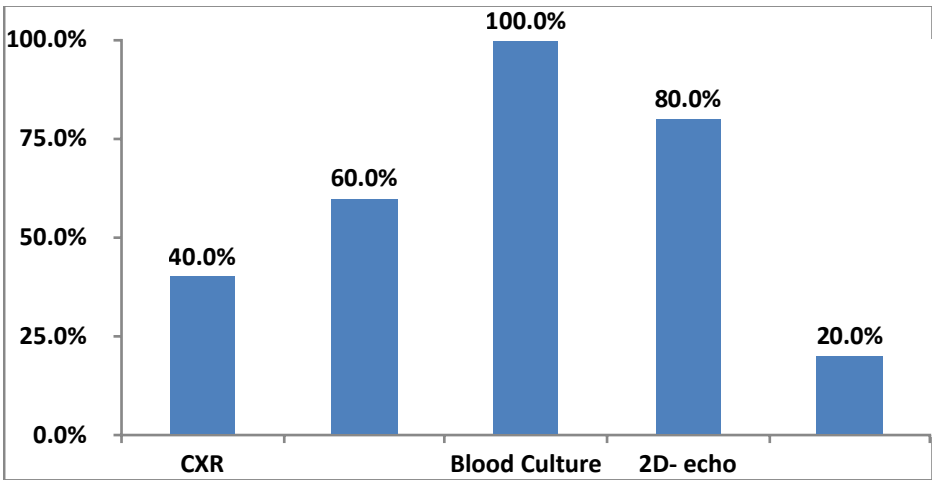
Out of 5 probable sepsis cases 1 had PFO and other 4 cases 2D ECHO was normal.

Blood culture was sterile in all the 5 probablse sepsis cases.

Table 17: Distribution of Other Parameters of Sepsis Cases

Parameters		**N**	**Percent**
CXR	Normal	2	40.0%
	Pneumonia	3	60.0%
Blood Culture	Sterile	5	100.0%
2D- echo	Normal	4	80.0%
	PFO	1	20.0%
Total		5	100.0%

Figure 15: Distribution of Other Parameters of probable Sepsis Cases



Mean Saturation within 6 hours between probable Sepsis & Non-Sepsis Cases

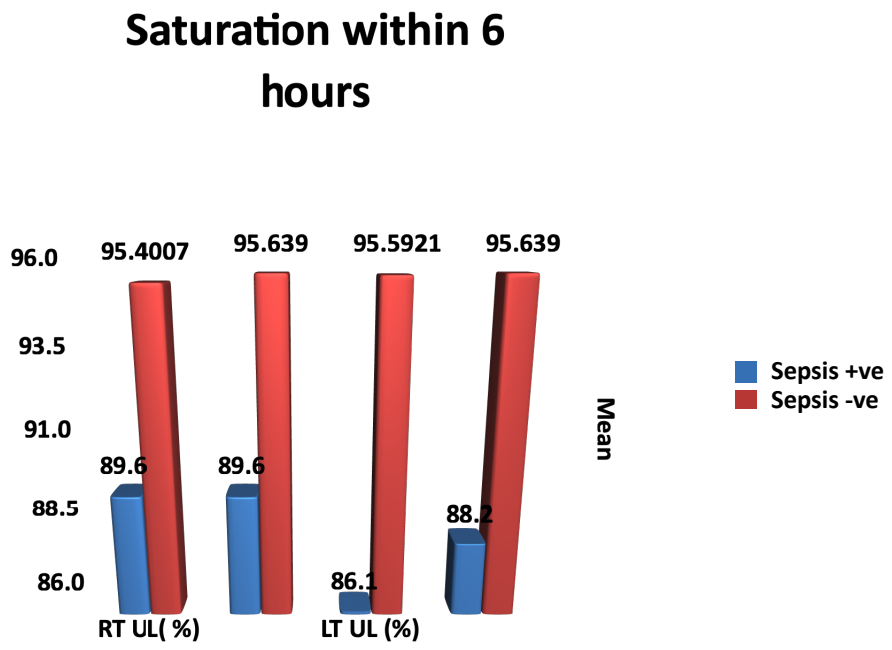
The mean oxygen saturation level in the 5 cases of probable sepsis was significantly lower than the mean oxygen saturation level of the cases who had no sepsis (89.2 % ± 0.8 % vs 97.5 % ± 1.2 %, $p<0.001$).

Table 18: Mean Saturation within 6 hours between probable Sepsis & Non-Sepsis Cases

Saturation within 6 hours	Probable Sepsis		Sepsis -ve		p value
	Mean	SD	Mean	SD	
RT UL(%)	89.6	1.8	95.4	1.3	<0.001*
RT LL(%)	89.6	1.8	95.6	1.4	<0.001*
LT UL (%)	86.1	2.1	95.6	1.4	<0.001*
LT LL(%)	88.2	2.0	95.6	1.5	<0.001*

Note: * significant at 5% level of significance (p<0.05)

Figure 16: Mean Saturation within 6 hours between probable Sepsis & Non-Sepsis Cases



Mean Saturation within 24 hours between probable Sepsis & Non-Sepsis Cases

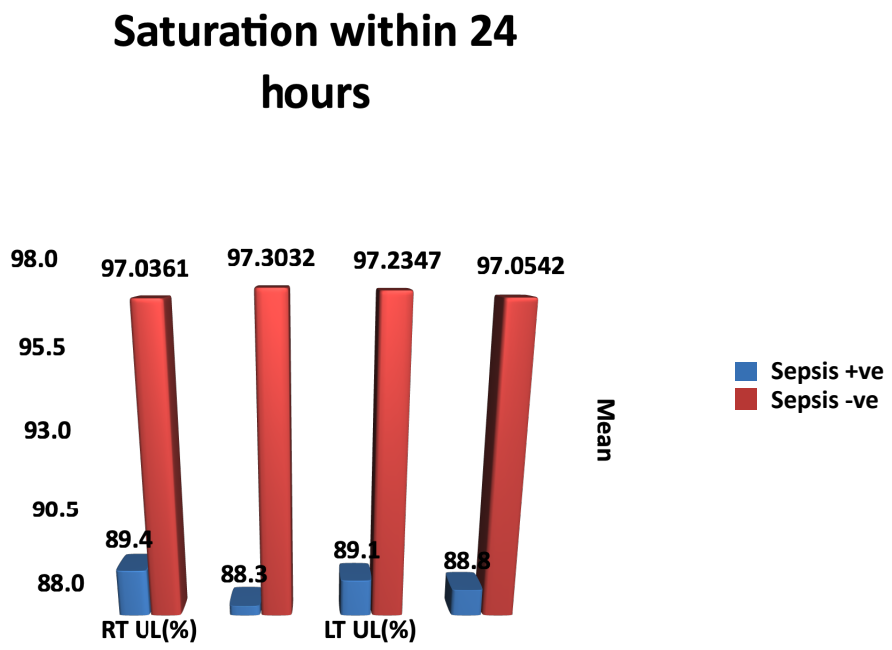
Even at 24 hours, the SPO2(%) from all the four limbs in cases who had probable sepsis were significantly lower than the corresponding values in non-sepsis cases (p<0.001 for all four limbs).

Table 19: Mean Saturation within 24 hours between probable Sepsis & Non-Sepsis Cases

Saturation within 24 hours	Probable Sepsis		Sepsis -ve		p value
	Mean	SD	Mean	SD	
RT UL(%)	89.4	0.9	97.0	1.1	<0.001*
RT LL(%)	88.3	0.8	97.3	1.2	<0.001*
LT UL(%)	89.1	0.5	97.2	1.2	<0.001*
LT LL(%)	88.8	0.4	97.1	1.2	<0.001*

Note: * significant at 5% level of significance (p<0.05)

Figure 17: Mean Saturation within 24 hours between probable Sepsis & Non-Sepsis Cases



SPO2 between probable Sepsis & Non-Sepsis Cases

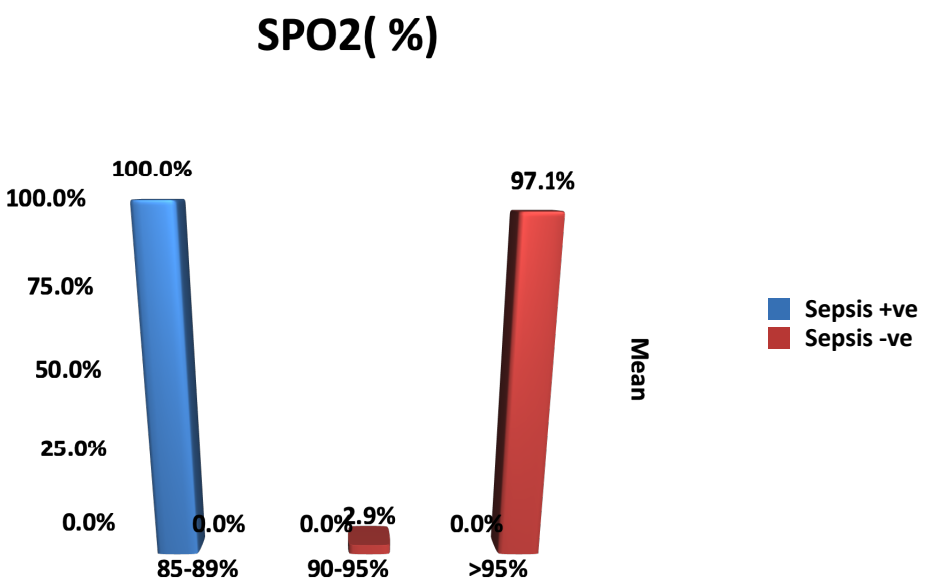
The SPO2(%) from all the four limbs in cases who had probable sepsis were significantly lower than the corresponding values in non-sepsis cases (p<0.001 for all four limbs) at admission in the study.

Table 20: SPO2 between probable Sepsis & Non-Sepsis Cases

SPO2(%)	Probable Sepsis		Sepsis -ve		p value
	N	%	N	%	
85-89%	5	100.0%	0	0.0%	<0.001*
90-95%	0	0.0%	8	2.9%	
>95%	0	0.0%	269	97.1%	
Total	5	100.0%	277	100.0%	

Note: * significant at 5% level of significance (p<0.05)

Figure 18: SPO2 between probable Sepsis & Non-Sepsis Cases



Heart rate according to time

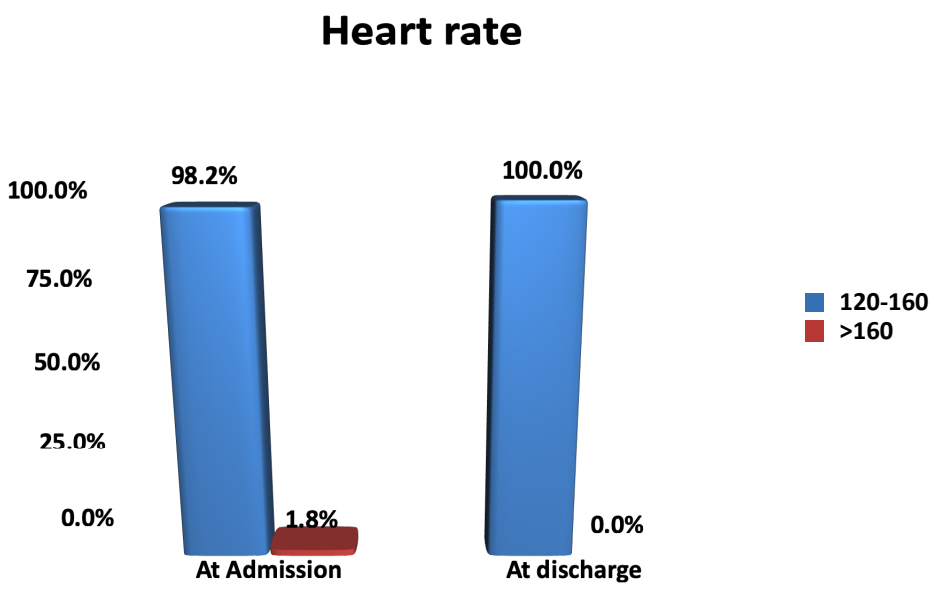
1.8 % (n=5) of babies had tachycardia at admission, which was ultimately resolved at the time of discharge (p=0.025).

Table 21: Heart rate according to time

Heart rate(per min)	At Admission		At the time of discharge		p value
	N	Percent	N	Percent	
120-160	277	98.2%	282	100.0%	0.025*
>160	5	1.8%	0	0.0%	
Total	282	100.0%	282	100.0%	

Note: * significant at 5% level of significance (p<0.05)

Figure 19: Heart rate according to time



Respiratory rate according to time

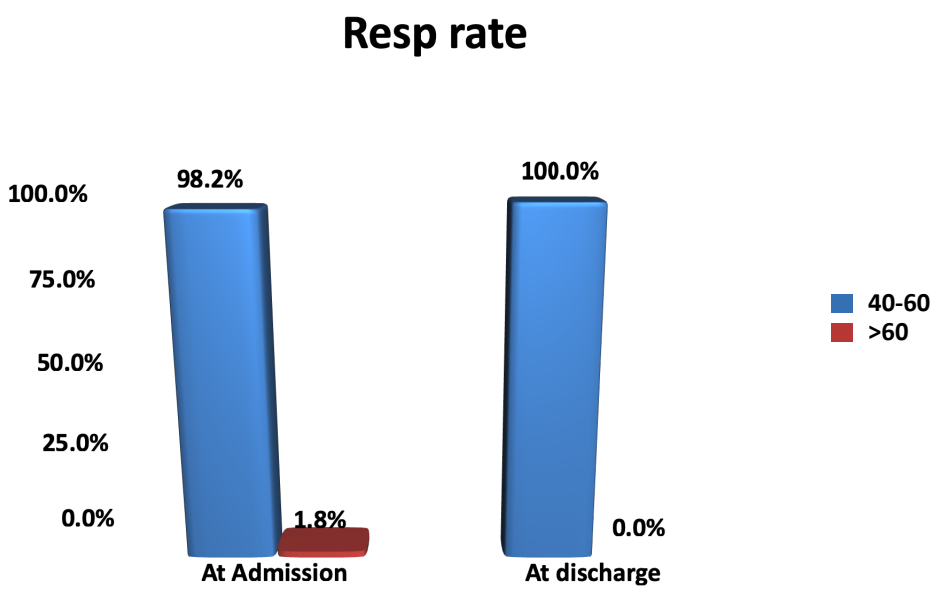
1.8% (n=5) babies had a respiratory rate > 60 per minute at admission, which was entirely resolved at the time of discharge (p=.025).

Table 22: Respiratory rate according to time

Resp rate(per min)	At Admission		At the time of discharge		p value
	N	Percent	N	Percent	
40-60	277	98.2%	282	100.0%	0.025*
>60	5	1.8%	0	0.0%	
Total	282	100.0%	282	100.0%	

Note: * significant at 5% level of significance (p<0.05)

Figure 20: Respiratory rate according to time



Temperature according to time

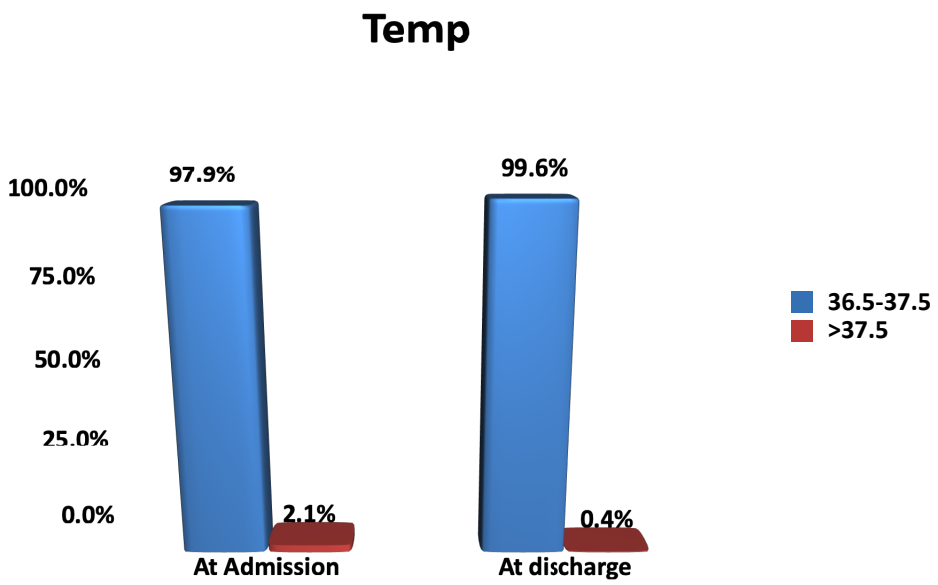
2.1% of babies (n=6) had a body temperature > 37.5°C at admission in the study. At the time of discharge, 0.4% (n=1) of babies had body temperature > 37.5 °C.

Table 23: Temperature according to time

Temp (degree celsius)	At Admission		At the time of discharge		p value
	N	Percent	N	Percent	
36.5-37.5	276	97.9%	281	99.6%	0.047*
>37.5	6	2.1%	1	0.4%	
Total	282	100.0%	282	100.0%	

Note: * significant at 5% level of significance (p<0.05)

Figure 21: Temperature according to time



Mean HR, RR, Temp and SPO2 between probable Sepsis & Non-Sepsis Cases

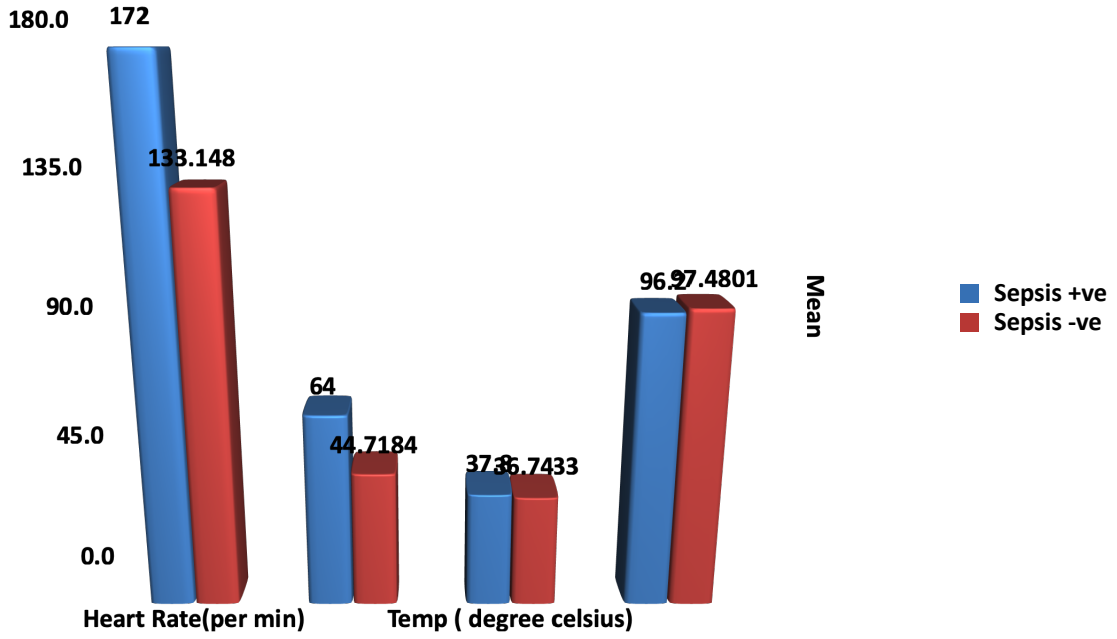
In the 5 cases who had probable sepsis, the mean haemodynamic parameters of heart rate,respiratory rate and body temperature at admission were significantly higher as compared to cases who had no sepsis (172.0 ± 7.6 vs 133.1 ± 7.6, 64.0 ± 1.9 vs 44.7 ± 4.0 and 37.8 °C vs 36.7°C respectively. The p-value was <0.001 for all the three parameters.

Table 24: Mean HR, RR, Temp and SPO2 between probable Sepsis & Non-Sepsis Cases

Parameters	Probable Sepsis		Sepsis -ve		p value
	Mean	SD	Mean	SD	
Heart Rate(per min) at 6hr	172.0	7.6	133.1	7.6	<0.001*
Resp rate(per min) at 6hr	64.0	1.9	44.7	4.0	<0.001*
Temp (degree celsius) at 6hr	37.8	0.2	36.7	0.2	<0.001*
SPO2(%)	89.2	0.8	97.5	1.2	<0.001*

Note: * significant at 5% level of significance (p<0.05)

Figure 22: Mean HR, RR, Temp and SPO2 between probable Sepsis & Non-Sepsis Cases



DISCUSSION

Very often, newborns are discharged from the hospital within 24 hours of birth, leading to missed diagnosis of early-onset neonatal sepsis. Sepsis leads to hypoxaemia. One third of newborns who are diagnosed to be hypoxaemic by are subsequently diagnosed with sepsis [32]. Hypoxaemia is often undiagnosed due to unreliable physical signs, difficult clinical detection of cyanosis and lack of diagnostic tools. Pulse oximetry is a straight forward, reliable and accurate method for detecting hypoxaemia. We undertook a study to assess the feasibility of using pulse oximetry as a screening tool to detect hypoxia associated with early-onset neonatal sepsis. A total of 282 babies were enrolled in the study. We recorded the maternal and neonatal parameters of the babies, along with the study parameters.

A study by Wong N et al [33]. has identified low maternal age as a risk factor for neonatal septicaemia. This association may result from a young, primiparous mother having more prolonged labour, which has itself been linked to an increased risk of neonatal septicaemia. Besides, the more traumatic and protracted deliveries experienced by primiparous mothers may promote the acquisition of organisms from the birth canal. [33] However, a study by Salem et al. [34] has reported an advanced maternal age to be a risk factor for early-onset sepsis. A study by Soman et al. [35] reported that the relationship of maternal age with early-onset neonatal sepsis (OR = 2.00, p = 0.01 if 20 years and OR = 1.74, p = 0.05 If >30) parallels the overall risk of many pregnancy-related complications in these age groups.

In our study, a majority of babies (56.4%) had mothers who were in the age group 21 years to 25 years, followed by 25.9 % babies whose mothers were in the age group of 26 years to 30 years. 13.5 % of babies had mothers who were less than 20 years of age, and the mothers of 4.3 % of babies were aged more than 30 years .

High gravidity is a risk factor for early neonatal sepsis. [34] Primigravida mothers were predominant in the present study (64.2%), followed by Gravida 2 mothers (25.5%).

A study by Adatara et al. [36] has reported caesarean section delivery as a variable that was statistically associated with the risk of developing neonatal sepsis. Though newborns delivered through CS are not exposed to vaginal and faecal bacteria, but they often experience prolonged hospital stay and late initiation of breastfeeding. Late initiation of breastfeeding after CS may deny the neonate the protective effect of colostrum against different pathogenic microbes that have harmful effects to the survival of the newborn baby and its ability to provide immunity for the neonate. [36] However, a few other studies have found the mode of delivery not to be statistically related to neonatal sepsis. [37] In the present study, 52.8 % of neonates were delivered by Caesarean section and 47.2% by normal vaginal delivery.

In the present study, we documented cases according to maternal comorbidity of anaemia, hyperemesis, hypertension, Meconium stained liquor amnii, Urinary tract infection, fever with rash, oligohydramonous/polyhydroaminous, Gestational DM and heart disease.

Anaemia is one of the most prevalent nutritional deficiency problems affecting pregnant women and is associated with an increased incidence of both maternal and foetal morbidity and mortality. [38] Adam et al. [39] reported that maternal anaemia was a risk factor for fetal anaemia(OR=2.1, 95% CI=1.4 – 3.1; $p< 0.001$). [39] In our study, 9.9 % of neonates had anaemic mothers.

Hyperemesis is often associated with nutritional deficiencies, maternal weight loss, and fluid and electrolyte imbalances, concerns about possible adverse perinatal outcomes are raised. In our study, 7.4 % of neonates had mothers who experienced hyperemesis.

In our study, 3.6 % of babies had hypertensive mothers, and 0.7 % of babies experienced MSLA during birth. Urinary tract infections were present in 0.4 % of mothers. None of the mothers had a fever with rash, oligohydramonous/polyhydraminous, Gestational DM and heart disease.

The strongest predictor of early-onset sepsis (EOS) risk within the overall birth population is low gestational age (GA). Preterm infants are at significantly higher risk of EOS compared to term infants; the magnitude of the difference varies with gestational age. Extremely low gestation is associated with poorly-developed innate immune responses and deficiency of maternally-derived, passively-acquired, pathogen-specific antibody. When analyzed by gestational age within a VLBW cohort, a gradient of increasing risk is observed as gestation decreases from 28 to 22 weeks [40].

In the present study, 90.4 % of babies were Term babies (>37weeks), and 9.6 % were late preterm babies (35-37 weeks). In a study in Tanzania, King et al. [4] reported 30.1 % of babies were born at 39-40 weeks of gestation and in 29.1% of babies accurate data on the gestational age of babies was not available but it was clinically evident that they were born at term. 13.6 % of babies had gestation age 37-38 weeks, 8.9% of babies had gestation age 35-36 weeks, and 2.2% of babies had gestation age 34 weeks.

A study by Soman et al. [35] reported an increased risk of neonatal sepsis for males (odds ratio (OR) = 1.75, p = 0.012). In the present study, 54.6% of babies were male, and 45.4% were female. The male: female ratio was 1: 0.83 . A similar study by King et al. [4] in Tanzania reported 53.2 % male babies and 46.8 % female babies. A study in Bangalore by Swamy et al. [31] reported M: F ratio as 109: 104. In all the three studies, male babies were slightly predominant.

The incidence of early-onset sepsis is nearly ten times higher in infants with VLBW (< 2000 g) as compared to normal birth weight. In the present study, a majority of babies (73.4%) had birth weight > 2.5 Kg. 26.6 % of babies had birth weight < 2.5 Kg. The mean birth weight of the babies was 2.7 kg ± 0.4 Kg, with the minimum being 1.8 Kg and maximum being 3.8 Kg. Soman et al. [35] reported a large risk of neonatal sepsis associated with low birth weight (OR = 99.1, p <0.001 if <1,500 g and OR = 5.17, p < 0.001 if 1,500−2,500 g).

Low GA and low birth weight (BW) are often used interchangeably and are highly interactive, but the increased risk of neonatal EOS is more strongly associated with low GA than with BW [40]. In our study, 95.4 % of babies were appropriate for gestational age, and 4.6% were small for gestational age.

The fetal membranes form a barrier to ascending maternal genital tract bacteria. Invasive infection rarely occurs through intact membranes during prolonged labours. Premature rupture of the membrane(PROM) occurs before the onset of labour, and preterm ROM is defined as rupture before 37 weeks gestation. [40] ROM provides an opportunity for ascending colonization of placental and fetal tissues, and the consequences of that colonization are different depending on the colonizing organism and level of immune function available at different gestational ages. Among very preterm infants, ROM may be a consequence of the ongoing infectious process that results in preterm delivery. [40] In the present study, 1.1% of cases showed premature rupture of membrane.

This study shows the change in heart rate, respiratory rate and body temperature of babies at admission into the study and at the time of discharge. 1.8 % (n=5) of babies had tachycardia at admission, which was ultimately resolved at the time of discharge (p=0.025)

The capillary fill time (CFT) is a parameter of shock. CFT tends to be prolonged in patients with sepsis. [41] In the present study, three babies had a CFT > 3 seconds at admission into the study. At the time of discharge, all 282 babies had CFT< 3 seconds.

Pulse oximetry provides the means for continuous non-invasive monitoring of blood oxygenation by measuring % oxygen saturation (SpO_2). In the present study, 1.8 % of babies (n=5) had oxygen saturation between 85-89 %. We considered this as sepsis- positive population. 2.8% of babies had oxygen saturation between 90- 95 % and 95.4 % of babies had oxygen saturation > 95%. The mean oxygen saturation was 97.5 % ± 1.2 % with the minimum value being 85 % and maximum being 100 % .

Our study shows the lab parameters of probable sepsis cases. Though the blood culture was sterile in all the cases of probable sepsis, the CRP value was raised (> 10 mg/dl) in all the 5 cases of probable sepsis. All the 5 cases had a total WBC count > 20000 cells/ mm^3. I/T ratio > 0.2 gives a clue about early-onset sepsis in newborns in the first 24 hours. In the present study, all the 5 cases of probable sepsis had an IT ratio of >0.2 . Absolute neutrophil count is a neutrophil percentage multiplied of the total leukocytes in the blood. The decrease of the ANC values correlated with the degree of sepsis.

The chest X-ray was normal in 2/5 cases of probable sepsis, and it was suggestive of congenital pneumonia in 3/5 cases of probable sepsis . Of the all 5 cases who had probable sepsis, one baby had Patent Foramen Ovale (PFO) as revealed by 2D ECHO.

The mean values and range of hemogram parameters of the 5 cases of probable sepsis are shown in our study. Total count is a nonspecific predictive parameter of sepsis as WBC count may either rise (>20,000 cells/mm3) or fall (< 5,000 cells/mm3) in sepsis. Many septic patients exist between these two extremes, with a normal WBC. Such patients often develop

leucocytosis in a delayed fashion. In the present study, the mean value of the WBC count in the 5 cases of probable sepsis was 24278 cells/ mm3 ± 3039.0 cells/ mm3, with the minimum value being 20210 cells/ mm3 and maximum value being 24278 cells/ mm3 .

The Neutrophil Lymphocyte Ratio (NLR) is simply the ratio of neutrophils/ lymphocytes. Physiologic stress as in sepsis generally increases the number of neutrophils and decreases the number of lymphocytes so that it will drive up the NLR. Sepsis also stimulates lymphocyte apoptosis so that septic shock may cause particularly dramatic elevation of NLR, compared to other forms of physiologic stress. A normal NLR is about 1–3. [42] In the present study, the mean value of NLR for the 5 cases of probable sepsis was 3.4 ± 2.2, with the range being 1 to 6 .

There is an acute reduction in haemoglobin levels in the setting of sepsis, due to reduced production of red blood cells induced by the systemic inflammatory response, as well as increased destruction of red cells due to haemolysis and bleeding. [43] In the present study, the mean value of haemoglobin for the 5 cases of probable sepsis was 17.0 gm % ± 1.7 gm %, with the range being 16 gm % to 19 gm % . [44]

The normal platelet count in the healthy newborn is ≥ 1.5 lakhs/ mm^3. Thrombocytopenia (platelet count < 1 lakh/ mm^3) may be a presenting sign of neonatal sepsis. [42] In the present study, the mean platelet count was 2.9 lakhs/ mm^3 ± 0.7 lakhs/ mm^3, with the range being 2-4 lakhs/ mm^3 for the 5 cases of probable sepsis.

In our study, the mean packed cell volume was reported to be 47.3 % ± 5.9 %, with the minimum value being 42 % and the maximum value being 57 % .

The chest X-ray was normal in 2/5 cases of probable sepsis, and it was suggestive of congenital pneumonia in 3/5 cases of probable sepsis. Of the all 5 cases who had probale sepsis, one baby had Patent Foramen Ovale (PFO) as revealed by 2D Echo .

The blood culture was sterile for all the 5 cases of probable sepsis.

The 2 D echo was normal in 4 cases of probable sepsis and was suggestive of PFO in 1 case, thereby attributing the hypoxia in the probable cases to sepsis rather than any heart disease .

The SPO2 readings from all the four limbs in cases who had probable sepsis were significantly lower than the corresponding values in non-sepsis cases ($p<0.001$ for all four limbs) at admission in the study.

Similarly, even at 24 hours, the SPO2 readings from all the four limbs in cases who had probable sepsis were significantly lower than the corresponding values in non-sepsis cases

The mean oxygen saturation level in the 5 cases of probable sepsis was significantly lower than the mean oxygen saturation level of the cases who had no sepsis (89.2 % ± 0.8 % vs 97.5 % ± 1.2 %, $p<0.001$). The significantly low mean oxygen saturation level obtained in pulse oximeter readings in cases of probable sepsis corroborated with the significantly high values of haemodynamic parameters in the cases, thereby demonstrating an excellent predictive value of Pulse Oximeter (PO) readings(. $p<0.001$ for all four limbs).

1.8 % (n=5) of babies had tachycardia at admission, which was ultimately resolved at the time of discharge ($p=0.025$).

1.8% (n=5) babies had a respiratory rate > 60 per minute at admission, which was entirely resolved at the time of discharge ($p=.025$).

2.1% of babies (n=6) had a body temperature > 37.5°C at admission in the study. At the time of discharge, 0.4% (n=1) of babies had body temperature > 37.5 °C.

CONCLUSION

Early Onset Neonatal Sepsis is one of the major causes of morbidity and mortality among the neonates. Prevalence of EONS in our centre is around 1.8**%**. Blood Culture is the gold standard for diagnosis of Early Onset Neonatal Sepsis. Our study has demonstrated that pulse oximetry can be used as a screen for early onset neonatal sepsis in asymptomatic neonates. None of the test negative neonates had Early onset sepsis. Our study shows that using pulse oximetry for screening for sepsis is feasible with 100 % sensitivity. It requires minimal training of the hospital staff and on an average about 5 minutes to complete the process of screening. This study may therefore help formulating guidelines on using pulse oximetry to rule out sepsis and firm basis for future research.

Limitation of the Study:

The main limitation of our study is limited validation of all the test negative neonates with the confirmatory blood test for the sepsis.

SUMMARY

This prospective observational study was conducted in Shri B M Patil medical college hospital and research centre, vijayapura during November 2018 –May 2020. During this study period 282 cases of asymptomatic newborns born after 35 weeks of gestational age were screened on two occasions using pulse oximetry (Saturation was checked in all four limbs). Newborns with oxygen saturations below 90% within 6 hours of life and SPO2 90-94% within 24 hours of life were defined as test positive. All the test positive babies underwent a septic screen which included full blood count, C reactive protein,I/T Ratio, blood cultures, chest x- ray and 2 D Echo. The test negative newborns were examined before discharge to ascertain that they have not developed sepsis .

In our study we made the following observations:

- The prevalance of early-onset neonatal sepsis was about 1.8 % in our Centre.
- The mean age of the mothers was 24.3 years ± 3.7 years, with the minimum age being 18 years and maximum age being 42 years. Primigravida mothers were predominant in the study (64.2%), followed by Gravida 2 mothers (25.5%).
- 52.8% of neonates were delivered by Caesarean section and 47.2 % by normal vaginal delivery.
- Anaemia was the most frequent presentation in mothers, followed by hyperemesis in 7.4% mothers, hypertension in 3.6 % mothers, Meconium Stained Liquor Amnii in 0.7 % mothers and UTI in 0.4 % mothers.
- 90.4 % neonates were delivered at full term (> 37weeks) and 9.6% were late preterm deliveries.

- Out of 282 neonates enrolled, 54.6% babies were male and 45.4% were female. The male: female ratio was 1: 0.83.
- A majority of neonates (73.4%) had birth weight > 2.5 Kg. 26.6 % babies had birth weight < 2.5 Kg. The mean birth weight of the babies was 2.7 kg ± 0.4 Kg, with the minimum being 1.8 kg and maximum being 3.8 Kg.
- 95.4 % neonates were appropriate for gestational age and 4.6% were small for gestational age.
- 1.1% of neonates had a premature rupture of membrane.
- 1.8 % of (n=5) of neonates had tachycardia at the time of admission, which was completely resolved at the time of discharge with significant P value. (p = 0.025).
- 1.8 % of neonates had a respiratory rate > 60 per minute at admission, which was completely resolved at the time of discharge (p=.025).
- 2.1 % of neonates had a body temperature > 37.5°C at the time of admission, which significantly reduced to 0.4 % babies with a body temperature > 37.5°C at the time of discharge (p = 0.047).
- At admission 3 neonates had a CFT > 3 seconds. At the time of discharge, all 282 babies had CFT< 3 seconds.
- At admission, 95.4 % neonates had Oxygen saturation > 95 %, 2.8 % neonates had Oxygen saturation between 90-95 % while 1.8 % of neonates had Oxygen saturation between 85-89 %.
- 1.8 % neonates (n=5) were found to have probable sepsis based on Pulse oximetry measurements.
- The vitals of the neonates having probable sepsis were significantly higher that the non-sepsis babies (p<0.001).

- The oxygen saturation of the neonates having probable sepsis was significantly lower than the non- sepsis neonates ($p<0.001$).
- The mean oxygen saturation measurements obtained from all the four limbs at 6 hours and 24 hours in the probable sepsis neonates was significantly lower than those obtained from the sepsis-negative neonates ($p<0.001$).
- The 5 babies who had probable sepsis based on Pulse oximetry measurements were suspected to have early onset sepsis clinically as evidenced by increased total count, CRP and NLR levels, IT ratio.
- The chest X ray of 2 neonates were normal while that of 3 babies was suggestive of congenital pneumonia.
- The blood culture of all the 5 neonates was found to be negative and hence we designate them as probable sepsis cases and not sepsis positive cases.
- The 2 D Echo of 4 neonates out of 5 was normal thereby excluding the cardiac cause for hypoxia. One neonate was found to have PFO.

Thus, our study was able to detect all the cases of early- onset neonatal sepsis using hypoxia as measured by blood oxygen saturation level with a pulse oximeter. Our study shows that using pulse oximetry for screening for sepsis is feasible with 100 % sensitivity.

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103

IEC/NO: 286/2018
17-11-2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : Pulse oximetry as a screening tool to detect hypoxia associated with early onset neonatal sepsis in asymtomatic newborns.

Name of P.G. Student : Dr Silky Singh.
Department of Paediatrics

Name of Guide/Co-investigator: Dr.M.M.Patil, Professor of Paediatrics.

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
B.L.D.E. (Deemed to be University)
Shri B.M. Patil
Medical College Hospital & Research Centre
Vijayapur-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

ANNEXURE II

PROFORMA

B.L.D.E.(DU)'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

Department of Paediatrics

PULSE OXIMETRY AS A SCREENING TOOL TO DETECT HYPOXIA ASSOCIATED WITH EARLY ONSET NEONATAL SEPSIS IN ASYMPTOMATIC NEWBORN.

S.NO

PROFORMA

Name - **DOB-**

Age- **TOB-**

Sex -

Address -

IP No -

ANTENATAL REGISTERATION -

GESTATIONAL AGE-

MODE OF DELIVERY(Normal vaginal/caesarean/foreceps/vaccum) :

PROM-

APGAR SCORE-

MATERNAL OBSTETRIC HISTORY-

Age- **Gravida- 1/2/3/4/>5**

Fever with rash- **Hyperemesis-**

Anemia- **UTI-**

HTN- **Oligohydraminous/polyhydraminous-**

Vaginal bleeding- **Heart disease-**

Gestational DM-

Meconium stained liquor amnii-

GENERAL PHYSICAL EXAMINATION-

BIRTH WEIGHT-

HR- RR- CFT- TEMP-

SYSTEMIC EXAMINATION-

CVS- RESPIRATORY SYSTEM-

PER ABDOMEN- CNS-

SATURATION IN ALL FOUR LIMBS-

WITHIN 6 HOURS OF LIFE WITHIN 72 HOURS OF LIFE

RT UL- RT LL- RT UL- RT LL-

LT UL- LT LL- LT UL- LT LL-

SEPSIS SCREENING:

CBC: CRP- I/T ratio- ANC(mm^3)-

TC- N- L- M- E- B- PCV- HB- PLT COUNT- MCV- MCHC- RDW-

CHEST X RAY- CSF ANALYSIS-

BLOOD CULTURE-

2D ECHO-

AT THE TIME OF DISCHARGE-

HR- RR- CFT- TEMP- SPO2-

CRY/ACTIVITY/TONE-

PROVISIONAL DIAGNOSIS-

FINAL DIAGNOSIS-

Signature of the candidate-

ANNEXURE III

CONSENT FORM

BLDEA's Shri B.M.PATIL Medical College, Hospital & Research Centre,

Vijayapur, Karnataka -586103.

TITLE OF THE PROJECT : "PULSE OXIMETRY AS A SCREENING TOOL TO DETECT HYPOXIA ASSOCIATED EARLY ONSET NEONATAL SEPSIS IN ASYMPTOMATIC NEWBORNS**".**

GUIDE: DR.M M PATIL, MD

PROFESSOR,

DEPARTMENT OF PEDIATRICS

PG STUDENT:DR. SILKY SINGH

PURPOSE OF RESEARCH:

_____I have been informed that the present study will help to assess the feasibility of using of pulse oximetry as a screening tool to identify early-onset neonatal sepsis in asymptomatic newborns.

PROCEDURE:

_____I understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a final work up of the procedure and its outcome is planned.

RISK AND DISCOMFORTS:

_____I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

_____I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the treatment.

CONFIDENTIALITY:

_____I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file. If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

_____I understand that I may ask more questions about the study at any time; Dr. SILKY SINGH, at the department of paediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

_____I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. SILKY SINGH may terminate my participation in the study after he/she has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to my child resulting directly from child's participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the child. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

DR. SILKY SINGH Date

(Investigator)

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr. SILKY SINGH is doing a study on pulse oximetry as ascreening tool to detect hypoxia associated with early onset neonatal sepsis in asymptomatic newborns in Shri B. M. Patil Medical College Hospital, Vijayapur, Karnataka. Dr. Silky Singh has explained to us the purpose of research and the study procedure. We are willing to allow our child to get treated in Shri B.M. Patil Medical College Hospital, Vijayapur. We have been explained about the study, benefits and possible discomforts in detail in our native language and we understand the same. We are aware that child will get best treatment ,and no compensation like financial benefits will be given if our child's condition deteriorates and any un happens, and we will not sue anyone regarding this. Therefore we agree to give our full consent for child's participation as a subject in this research project.

_____ _____

(**Parents / Guardian**) **Date**

_____ _____

(**Witness to signature**) **Date**

ANNEXURE IV

KEY TO MASTERCHART

F	Female
M	Male
Gest Age	Gestational Age
MOD	Mode of delivery
PROM	Premature rupture of membrane
N	No
Nn	Neonate
B W	Birth weight
HR	Heart rate
RR	Respiratory rate
TEMP	Temperature
CVS	Cardiovascular system
PA	Per abdomen
CNS	Central nervous system
RS	Respiratory system
WNL	within normal limit
GD	Good
RT	Right
LT	Left
UL	Upper limb
LL	Lower Limb
TC	Total count
NEUT	Neutrophil
LYMP	Lymphocyte
HB	Hemoglobin
PLT	Platelet
PCV	Packed cell volume
CXR	Chest xray

ANC	Absolute neutrophil count
I/T Ratio	Immature to neutrophil count
CRP	C reactive protein
2 D ECHO	2 D Echocardiography
T	Term
A	AGA
LPT	Late preterm
OLIGO	Oligohydraminous
POLY	Polyhydraminous
GDM	Gestational diabetes mellitus
MSLA	Meconium stained liquor amnii
UTI	Urinary tract infection
SPO2	oxygen saturation
C/A/T	Cry activity tone
EONS	Early onset neonatal sepsis
LBW	Low birth weight
SGA	Small for gestational age
AGA	Appropriate for gestational age

							Maternal Details																Saturation within 6 hours				Saturation within 24 hours				Sepsis Screening										At The time of discharge						
Sr. no.	IP No.	Age / sex	Gest. Age	MOD	PROM	APGAR Score (/ 10)	Age (yrs)	Gravida	Fever with rash	Hyperemesis	Anemia	HTN	UTI	Oligo / Poly	GDM	HeartDs	MSLA	BW (kgs)	HR (bpm)	RR (per min)	CFT (sec)	Temp (degree celsius)	CVS	RS	PA	CNS	RTUL(%)	RTLL (%)	LTUL(%)	LTLL (%)	RTUL(%)	RTLL (%)	LTUL (%)	LTLL (%)	TC(cells/ cmm)	Neut/ Lymp(%)	HB(gm%)	PLT(lakhs/ cmm)	pcv(%)	CXR	ANC(mm)	I/T Ratio)	CRP(mg/l)	Blood Culture	2D-echo	HR(bpm)	Resp rate(per min)	CFT(sec)	Temp(degree celsius)	SPO2 (%)	C/A/T	Final Diagnosis
1	38898	1/F	38+4W	LSCS	N	7, 9	28	2	N	Y	N	N	N	N	N	N	N	2.6	130	42	<3s	36.5	WNL	WNL	WNL	GD	97	96	96	96	96	97	96	96											142	42	<3	36.5	98	GD	T/A/F/N
2	38926	1/M	36+5W	LSCS	N	7, 9	25	1	N	N	N	N	N	N	N	N	N	2.8	140	48	<3	36.6	WNL	WNL	WNL	GD	97	97	96	98	98	96	98	97											142	46	<3	36.6	100	GD	T/A/M/N
3	38954	1/M	40W	LSCS	N	7,9	26	1	N	N	Y	N	N	N	N	N	N	2.7	138	42	<3	36.5	WNL	WNL	WNL	GD	98	98	96	97	98	96	96	96											148	46	<3	36.6	99	GD	T/A/M/N
4	39100	1/F	38W	LSCS	N	7,9	22	1	N	Y	Y	N	N	N	N	N	N	2.5	130	40	<3	36.6	WNL	WNL	WNL	GD	96	96	96	96	96	97	97	97											132	42	<3	36.5	100	GD	T/A/F/N
5	39103	1/M	41W	LSCS	N	7,9	24	2	N	N	Y	N	N	N	N	N	Y	2.9	140	42	<3	36.6	WNL	WNL	WNL	GD	94	95	94	94	96	96	96	96											130	44	<3	36.7	100	GD	T/A/M/N
6	39203	1/F	39W	NVD	N	7 ,9	28	3	N	Y	Y	N	N	N	N	N	N	2.6	142	38	<3	36.6	WNL	WNL	WNL	GD	95	95	94	95	96	96	96	97											130	42	<3	36.5	96	GD	T/A/F/N
7	39201	1/M	39W	NVD	N	7, 9	22	1	N	N	N	N	N	N	N	N	N	2.6	140	42	<3	36.8	WNL	WNL	WNL	GD	94	93	93	93	96	96	96	96											148	42	<3	36.6	99	GD	T/A/M/N
8	39270	1/M	36+5 W	NVD	N	7, 9	24	1	N	N	N	N	N	N	N	N	N	3	140	42	<3	36.7	WNL	WNL	WNL	GD	94	93	94	92	96	96	96	96											140	44	<3	36.6	98	GD	LPT/A/M/N
9	39216	1/F	39W	NVD	N	7,9	26	3	N	N	Y	N	N	N	N	N	N	2.9	130	44	<3	36.8	WNL	WNL	WNL	GD	95	95	96	95	96	96	98	96											120	42	<3	36.5	97	GD	T/A/F/N
10	39256	1/M	39+2W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.8	128	44	<3	36.6	WNL	WNL	WNL	GD	94	94	93	94	97	97	98	97											130	44	<3	36.5	97	GD	T/A/M/N
11	39271	1/F	39 W	NVD	N	7,9	29	2	N	N	N	N	N	N	N	N	N	2.8	142	40	<3	36.7	WNL	WNL	WNL	GD	96	97	95	95	98	98	98	99											140	40	<3	36.6	100	GD	T/A/F/N
12	39310	1/M	39+2 W	NVD	N	7, 9	22	1	N	Y	Y	N	N	N	N	N	N	2.5	132	42	<3	36.8	WNL	WNL	WNL	GD	96	96	96	96	97	96	96	96											132	44	<3	36.7	96	GD	T/A/M/N
13	39326	1/M	38W	NVD	N	7,9	22	2	N	N	N	N	N	N	N	N	N	2.5	130	50	<3	37	WNL	WNL	WNL	GD	96	95	97	96	96	96	98	96											140	48	<3	36.4	97	GD	T/A/M/N
14	39039	1/F	38+2W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.7	132	42	<3	36.7	WNL	WNL	WNL	GD	95	94	94	94	96	97	96	96											142	44	<3	37	96	GD	T/A/M/N
15	39356	1/M	39W	LSCS	N	7,9	29	2	N	N	N	N	N	N	N	N	N	2.9	132	44	<3	36.6	WNL	WNL	WNL	GD	96	96	97	98	98	96	98	99											130	44	<3	37.2	96	GD	T/A/M/N
16	39356	1/F	39 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.8	142	44	<3	36.5	WNL	WNL	WNL	GD	94	95	95	93	96	97	96	98											128	46	<3	36.6	96	GD	T/A/F/N
17	39601	1/M	40 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.8	144	45	<3	36.6	WNL	WNL	WNL	GD	94	93	94	95	96	98	96	99											130	42	<3	36.5	96	GD	T/A/M/N
18	41866	1/F	39+6 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	3.2	148	44	<3	36.6	WNL	WNL	WNL	GD	95	96	95	97	97	96	96	96											120	42	<3	36.6	96	GD	T/A/F/N
19	6510	1/M	40+3 W	NVD	N	7,9	23	1	N	N	N	N	N	N	N	N	N	3.2	146	42	<3	36.8	WNL	WNL	WNL	GD	93	94	94	95	96	96	96	96											144	40	<3	36.5	99	GD	T/A/M/N
20	6733	1/M	38 +2 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.2	140	42	<3	36.6	WNL	WNL	WNL	GD	96	98	96	99	99	96	96	96											130	42	<3	36.8	96	GD	T/A/M/N/LBW
21	6724	1/M	37 +6W	LSCS	N	7, 9	25	1	N	N	N	N	N	N	N	N	N	2.4	128	46	<3	36.7	WNL	WNL	WNL	GD	94	96	96	96	99	98	98	98											130	42	<3	36.5	99	GD	T/A/F/N/LBW
22	6736	1/F	37 W	NVD	N	7,8	30	2	N	N	N	N	N	N	N	N	N	2.1	144	44	<3	36.7	WNL	WNL	WNL	GD	93	95	95	94	96	96	96	96											142	44	<3	37.2	96	GD	T/A/F/N/LBW
23	6817	1/F	39 W	LSCS	N	7,9	23	1	N	N	N	N	N	N	N	N	N	3.4	120	48	<3	36.8	WNL	WNL	WNL	GD	95	95	95	94	97	97	97	96											140	42	<3	36.6	97	GD	T/A/F/N
24	6784	1/F	36 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.4	122	46	<3	36.6	WNL	WNL	WNL	GD	96	97	96	98	98	98	98	99											130	40	<3	36.6	99	GD	LPT/A/F/N/ LBW
25	6809	1/M	41 +3 W	LSCS	N	7, 9	26	2	N	N	N	N	N	N	N	N	N	2.8	140	42	<3	36.6	WNL	WNL	WNL	GD	96	95	95	95	10(	10(	99	10											120	44	<3	36.7	98	GD	T/A/M/N
26	6816	1/M	36+4 W	LSCS	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.8	132	40	<3	37	WNL	WNL	WNL	GD	96	94	94	94	99	99	98	99											130	42	<3	36.6	98	GD	LPT/A/M/N
27	6826	1/F	35 +6 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.8	140	44	<3	36.8	WNL	WNL	WNL	GD	97	96	96	96	10(	10(	99	10											140	42	<3	36.8	98	GD	LPT/A/F/N/
28	6831	1/M	39 W	LSCS	N	7, 9	20	1	N	N	N	N	N	N	N	N	N	3.3	142	42	<3	37	WNL	WNL	WNL	GD	95	94	95	94	98	96	96	96											128	44	<3	37.2	99	GD	T/A/M/N
29	7002	1/F	40+1 W	LSCS	N	7, 9	20	1	N	N	N	N	N	N	N	N	N	2.5	130	40	<3	37.2	WNL	WNL	WNL	GD	94	94	94	95	98	97	97	97											130	48	<3	37	99	GD	T/A/F/N
30	7227	1/M	40 W	LSCS	N	7,9	20	1	N	N	N	N	N	N	N	N	N	3	140	46	<3	37	WNL	WNL	WNL	GD	95	93	93	94	98	10(	98	97											128	40	<3	36.8	96	GD	T/A/M/N
31	7539	1/F	41 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	3	130	42	<3	36.6	WNL	WNL	WNL	GD	94	94	96	94	96	96	96	96											130	44	<3	37.2	97	GD	T/A/M/N
32	7759	1/F	39 W	LSCS	N	7, 9	23	1	N	N	N	N	N	N	N	N	N	2.9	128	44	<3	37.2	WNL	WNL	WNL	GD	97	97	96	97	99	97	97	97											122	50	<3	37.3	98	GD	T/A/M/N
33	8236	1/M	40 W	LSCS	N	7, 9	28	3	N	N	N	N	N	N	N	N	N	2.8	124	55	<3	36.6	WNL	WNL	WNL	GD	95	95	95	95	96	97	98	96											138	55	<3	36.8	99	GD	T/A/M/N

34	8369	1 / M	39+5 W	NVD	N	7, 9	25	1	N	N	N	N	N	N	N	N	N	2.4	130	44	<3	36.8	WNL	WNL	WNL	GD	95	97	95	96	97	97	97	97												132	44	<3	36.7	100	GD	T/A/M/N/LBW
35	8412	1/ M	39+4 W	NVD	N	7, 9	20	1	N	Y	Y	N	N	N	N	N	N	2.1	133	42	<3	36.7	WNL	WNL	WNL	GD	93	93	93	94	97	97	97	96												130	42	<3	36.6	97	GD	T/A/M/N/LBW
36	8628	1/ F	39+ 2 W	LSCS	N	7, 9	21	1	N	N	N	N	N	N	N	N	Y	3	120	44	<3	36.8	WNL	WNL	WNL	GD	95	95	95	94	10	10	100	98												120	40	<3	37.2	97	GD	T/A/F/N
37	8671	1/M	40 W	NVD	N	7, 9	18	1	N	Y	Y	N	N	N	N	N	N	2.4	140	42	<3	37	WNL	WNL	WNL	GD	97	97	97	96	10	10	100	99												130	42	<3	37	100	GD	T/A/M/N
38	8921	1/M	38+6 W	VANV	Y	7, 9	19	1	N	Y	N	N	N	N	N	N	N	2.9	130	42	<3	36.8	WNL	WNL	WNL	GD	96	96	96	95	97	97	97	97												140	50	<3	37.2	98	GD	T/A/M/N
39	9285	1/F	39 W	LSCS	N	7, 9	22	1	N	N	Y	N	N	N	N	N	N	3.2	134	56	<3	36.6	WNL	WNL	WNL	GD	94	93	93	93	96	97	96	96												130	40	<3	37.5	100	GD	T/A/M/N
40	9512	1/ F	39 W	LSCS	N	7, 9	20	1	N	N	N	Y	N	N	N	N	N	3.1	120	54	<3	36.7	WNL	WNL	WNL	GD	95	94	94	94	98	98	98	97												128	40	<3	36.6	97	GD	T/A/F/N
41	9836	1/F	41 W	LSCS	N	7, 9	21	1	N	Y	Y	Y	N	N	N	N	N	2.1	130	46	<3	37	WNL	WNL	WNL	GD	92	93	93	93	96	98	96	96												140	42	<3	36.7	99	GD	T/S/F/N/LBW
42	10092	1/M	39+5 W	NVD	N	7, 9	20	1	N	N	Y	N	N	N	N	N	N	2.5	140	42	<3	36.7	WNL	WNL	WNL	GD	96	96	95	96	97	98	96	96												130	40	<3	36.6	100	GD	T/A/M/N
43	10132	1/F	40 W	NVD	N	7, 9	29	2	N	N	N	N	N	N	N	N	N	3.2	128	42	<3	36.6	WNL	WNL	WNL	GD	92	94	94	96	96	97	98	97												128	44	<3	36.7	97	GD	T/A/F/N
44	10240	1/M	39+3 W	NVD	N	7, 9	24	1	N	N	N	N	N	N	N	N	N	2.7	142	46	<3	36.6	WNL	WNL	WNL	GD	95	95	96	97	98	98	98	99												140	42	<3	36.8	99	GD	T/A/M/N
45	10516	1/M	36 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.2	120	42	<3	37.2	WNL	WNL	WNL	GD	95	95	94	95	98	97	97	97												134	44	<3	37.4	98	GD	LPT/A/M/N/LBW
46	10730	1/M	38+6 W	LSCS	N	7,9	26	2	N	N	N	N	N	N	N	N	N	3.2	138	55	<3	36.7	WNL	WNL	WNL	GD	96	96	94	95	96	97	96	96												120	44	<3	36.6	99	GD	T/A/M/N
47	11195	1/M	38 W	LSCS	N	7, 9	24	2	N	Y	N	N	N	N	N	N	N	2.8	124	40	<3	36.5	WNL	WNL	WNL	GD	97	96	95	96	96	98	97	98												140	42	<3	36.7	97	GD	T/A/M/N
48	11437	1/F	38 W	LSCS	N	7,8	30	1	N	N	N	N	N	N	N	N	N	2.3	130	42	<3	36.7	WNL	WNL	WNL	GD	96	95	95	94	97	98	98	96												130	40	<3	36.7	98	GD	T/A/F/N/LBW
49	11657	1/F	37+ 2 W	LSCS	N	7,9	28	3	N	N	N	N	N	N	N	N	N	3.5	122	55	<3	37.5	WNL	WNL	WNL	GD	95	94	94	94	96	97	97	99												120	44	<3	36.8	99	GD	T/A/F/N
50	11623	1/F	41 W	LSCS	N	7, 9	30	1	N	Y	Y	N	N	N	N	N	N	2	122	44	<3	36.5	WNL	WNL	WNL	GD	93	95	94	95	98	96	98	98												124	42	<3	36.8	98	GD	T/A/F/N/LBW
51	12237	1/F	38 W	NVD	N	7, 9	25	1	N	N	N	N	Y	N	N	N	N	1.9	140	44	<3	36.8	WNL	WNL	WNL	GD	96	95	95	95	96	97	97	97												124	40	<3	36.9	96	GD	T/S/F/N/LBW
52	12313	1/F	41 W	LSCS	N	7, 9	25	1	N	N	N	N	N	N	N	N	N	3.6	140	44	<3	36.8	WNL	WNL	WNL	GD	95	96	94	94	97	98	97	97												130	44	<3	36.6	96	GD	T/A/F/N
53	12463	1/F	35 W	NVD	N	7, 9	21	1	N	N	Y	N	N	N	N	N	N	2.3	130	44	<3	36.6	WNL	WNL	WNL	GD	93	94	94	94	98	98	96	98												140	46	<3	36.7	96	GD	LPT/A/F/N/LBW
54	12470	1/M	38+2 W	NVD	N	7, 9	30	1	N	N	N	N	N	N	N	N	N	1.9	140	48	<3	37	WNL	WNL	WNL	GD	96	96	96	97	96	97	96	96												144	50	<3	37.4	97	GD	T/S/M/N/LBW
55	12618	1/F	36 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.1	140	42	<3	36.6	WNL	WNL	WNL	GD	94	96	94	95	96	96	97	96												130	44	<3	36.4	96	GD	LPT/A/F/N/LBW
56	11834	1/M	38 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.3	130	42	<3	36.4	WNL	WNL	WNL	GD	92	92	94	92	96	96	98	96												122	50	<3	36.7	99	GD	T/A/M/N/LBW
57	12719	1/F	35 +4 W	LSCS	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.2	140	44	<3	36.7	WNL	WNL	WNL	GD	96	98	96	99	96	97	97	97												138	52	<3	36.7	96	GD	Late preterm/ A/F/N/LBW
58	12984	1/F	39+3 W	NVD	N	7,9	34	3	N	N	N	N	N	N	N	N	N	2.5	134	45	<3	36.8	WNL	WNL	WNL	GD	98	96	97	98	96	98	96	96												122	42	<3	36.6	98	GD	T/A/F/N
59	13078	1/M	35+ 5W	NVD	N	7,9	26	3	N	N	N	N	N	N	N	N	N	2.3	130	44	<3	36.8	WNL	WNL	WNL	GD	94	94	94	96	97	98	97	97												134	44	<3	36.7	97	GD	LPT/A/M/N/LBW
60	13296	1/ F	40 W	LSCS	N	7,9	24	1	N	Y	Y	Y	N	N	N	N	N	2.4	140	52	<3	36.6	WNL	WNL	WNL	GD	96	96	96	95	96	97	99	97												122	42	<3	36.7	96	GD	T/A/F/N/LBW
61	13473	1/F	39 W	NVD	N	7,9	22	2	N	N	N	N	N	N	N	N	N	2.6	130	46	<3	36.9	WNL	WNL	WNL	GD	92	93	93	95	96	96	96	96												144	44	<3	36.6	98	GD	T/A/F/N
62	10132	1/F	40 W	LSCS	N	7,9	29	1	N	N	N	N	N	N	N	N	N	3.2	160	62	<3	37.6	WNL	WNL	WNL	GD	89	89	89	88	94	95	95	94	24600	57/33	18	2.6	42.4	NOR	2800	0.18	44	ST	NOR	130	44	<3	36.6	97	GD	T/A/F/N/EONS
63	13595	1/M	40+4 W	LSCS	N	7,9	20	1	N	N	N	Y	N	N	N	N	N	2.5	122	45	<3	36.8	WNL	WNL	WNL	GD	92	92	93	92	96	98	96	96												132	55	<3	37.2	98	GD	T/A/M/N
64	13637	1/F	38 W	LSCS	N	7,9	32	2	N	Y	Y	N	N	N	N	N	N	2.3	138	44	<3	36.7	WNL	WNL	WNL	GD	97	97	97	97	96	97	100	10												130	44	<3	36.8	98	GD	T/A/F/N/LBW
65	13666	1/F	38 W	NVD	N	7,9	21	1	N	Y	Y	N	N	N	N	N	N	2.8	140	42	<3	36.8	WNL	WNL	WNL	GD	95	95	95	96	96	97	96	96												140	45	<3	36.8	100	GD	T/A/F/N
66	13718	1/F	40 W	NVD	N	7, 9	21	2	N	N	N	N	N	N	N	N	N	2.7	130	44	<3	36.6	WNL	WNL	WNL	GD	94	94	95	94	96	96	96	96												132	40	<3	37.2	98	GD	T/A/F/N
67	13850	1/M	38+2 W	LSCS	N	7,9	30	2	N	N	Y	N	N	N	N	N	N	2.8	124	56	<3	36.5	WNL	WNL	WNL	GD	93	93	94	93	97	10	97	96												122	42	<3	36.8	96	GD	T/A/M/N
68	13411	1/M	39+1 W	NVD	N	7,9	23	1	N	Y	Y	N	N	N	N	N	N	3	138	44	<3	36.8	WNL	WNL	WNL	GD	94	94	95	94	96	96	97	99												120	42	<3	36.6	97	GD	T/A/M/N
69	12004	1/M	39 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.7	144	45	<3	36.6	WNL	WNL	WNL	GD	95	95	94	95	97	97	96	97												130	44	<3	36.6	96	GD	T/A/M/N
70	14174	1/M	39 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.5	134	54	<3	36.7	WNL	WNL	WNL	GD	94	94	93	94	98	96	98	97												132	46	<3	37.5	97	GD	T/A/M/N
71	14262	1/M	40 W	NVD	N	7, 9	24	1	N	N	N	N	N	N	N	N	N	2.8	124	54	<3	36.6	WNL	WNL	WNL	GD	95	96	96	98	98	98	100	97												130	46	<3	37	96	GD	T/A/M/N
72	14374	1/F	39+4 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.5	140	46	<3	36.8	WNL	WNL	WNL	GD	95	95	97	96	98	98	99	10												132	44	<3	36.6	97	GD	T/A/F/N
73	14520	1/M	39 W	LSCS	N	7, 9	22	1	N	N	N	Y	N	N	N	N	N	2.6	144	42	<3	36.6	WNL	WNL	WNL	GD	93	94	94	94	97	97	97	96												128	44	<3	37.2	98	GD	T/A/M/N
74	14754	1/F	39+1 W	NVD	N	7,9	22	1	N	Y	Y	N	N	N	N	N	N	2.8	130	46	<3	36.8	WNL	WNL	WNL	GD	96	96	96	96	10	10	98	99												120	50	<3	36.6	98	GD	T/A/F/N
75	14859	1/M	39+2 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.3	128	44	<3	37.2	WNL	WNL	WNL	GD	95	95	94	95	98	98	97	98												138	46	<3	37.2	98	GD	T/A/M/N/LBW
76	15048	1/M	42 W	LSCS	N	7, 9	21	2	N	N	N	Y	N	N	N	N	N	3	128	42	<3	36.5	WNL	WNL	WNL	GD	94	95	95	95	97	96	96	96												128	40	<3	37	100	GD	T/A/M/N

77	15317	1/M	39 +3 W	LSCS	N	7,9	28	1	N	N	N	N	N	N	N	N	N	3.2	140	45	<3	36.6	WNL	WNL	WNL	GD	95	95	95	94	98	98	97	97												130	44	<3	36.5	98	GD	T/A/M/N
78	15355	1/M	39+ 2 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.5	138	42	<3	36.6	WNL	WNL	WNL	GD	95	95	94	95	97	97	97	96												120	42	<3	36.6	97	GD	T/A/M/N
79	15488	1/F	35+1 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	1.9	138	50	<3	37	WNL	WNL	WNL	GD	95	95	97	95	96	96	98	96												130	45	<3	37	96	GD	LPT/A/F/N/LBW
80	15991	1/M	41+3 W	LSCS	N	7,9	27	3	N	N	N	N	N	N	N	N	N	2.9	130	42	<3	36.6	WNL	WNL	WNL	GD	93	93	93	94	99	98	100	99												120	40	<3	36.5	98	GD	T/A/M/N
81	15857	1/M	40+4 W	NVD	N	7,9	23	2	N	N	N	N	N	N	N	N	N	2.5	140	40	<3	36.6	WNL	WNL	WNL	GD	95	97	95	96	98	97	99	97												130	48	<3	36.8	100	GD	T/A/M/N
82	16083	1/F	40+2 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	3.1	130	42	<3	36.6	WNL	WNL	WNL	GD	95	97	95	94	99	96	97	96												120	42	<3	36.8	97	GD	T/A/F/N
83	16343	1/M	39+6 W	NVD	N	7,9	27	1	N	N	N	N	N	N	N	N	N	2.4	140	40	<3	36.8	WNL	WNL	WNL	GD	95	95	95	96	98	98	98	97												130	40	<3	36.8	97	GD	T/A/M/N/LBW
84	16384	1/M	38+2 W	LSCS	N	7,9	24	1	N	Y	Y	Y	N	N	N	N	N	3.2	142	40	<3	37.2	WNL	WNL	WNL	GD	96	98	96	96	98	98	98	10												120	54	<3	37.2	96	GD	T/A/M/N
85	16596	1/F	39 +1 W	NVD	N	7,9	26	1	N	N	N	N	N	N	N	N	N	3	130	44	<3	37	WNL	WNL	WNL	GD	93	93	94	93	96	96	97	96												120	44	<3	36.5	98	GD	T/A/F/N
86	16759	1/M	39+ 6 W	NVD	N	7, 9	23	2	N	N	N	N	N	N	N	N	N	3.2	138	40	<3	37.2	WNL	WNL	WNL	GD	96	97	96	97	96	98	96	96												120	42	<3	37	97	GD	T/A/M/N
87	16761	1/M	39 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.7	140	42	<3	36.3	WNL	WNL	WNL	GD	95	96	95	95	96	99	96	97												150	54	<3	37.5	98	GD	T/A/M/N
88	16857	1/F	40 W	LSCS	N	7,9	23	1	N	N	Y	N	N	N	N	N	N	2.6	142	55	<3	36.8	WNL	WNL	WNL	GD	96	96	96	97	97	97	99	96												130	42	<3	36.8	98	GD	T/A/F/N
89	16896	1/M	39 W	LSCS	N	7,9	30	2	N	N	N	N	N	N	N	N	N	2.8	142	40	<3	37.1	WNL	WNL	WNL	GD	96	97	96	96	97	97	96	96												130	44	<3	36.6	99	GD	T/A/M/N
90	17051	1/F	40 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.6	130	54	<3	36.6	WNL	WNL	WNL	GD	98	98	100	97	97	97	99	97												130	42	<3	36.7	96	GD	T/A/M/N
91	17183	1/F	40 W	NVD	N	7, 9	26	2	N	N	N	N	N	N	N	N	N	3.4	140	44	<3	37.2	WNL	WNL	WNL	GD	95	95	95	94	96	100	98	96												130	40	<3	36.8	97	GD	T/A/F/N
92	17318	1/M	38 W	LSCS	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.8	138	52	<3	36.5	WNL	WNL	WNL	GD	96	96	97	96	98	100	98	98												132	40	<3	36.6	96	GD	T/A/M/N
93	17446	1/F	41+2 W	LSCS	N	7,9	26	3	N	N	Y	N	N	N	N	N	N	2.9	150	52	<3	36.8	WNL	WNL	WNL	GD	97	97	97	96	97	100	97	97												138	46	<3	36.4	98	GD	T/A/F/N
94	17514	1/M	39 W	NVD	N	7,9	19	1	N	N	N	N	N	N	N	N	N	2.7	130	40	<3	36.6	WNL	WNL	WNL	GD	95	96	95	97	97	97	99	96												140	40	<3	36.6	98	GD	T/A/M/N
95	17614	1/F	38+4 W	LSCS	N	7,9	20	2	N	N	N	N	N	N	N	N	N	2.5	140	44	<3	36.5	WNL	WNL	WNL	GD	96	96	96	97	98	99	100	97												130	40	<3	36.6	97	GD	T/A/F/N
96	17779	1/M	40+3 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.4	140	45	<3	37	WNL	WNL	WNL	GD	95	96	96	96	97	98	97	97												128	40	<3	37.3	96	GD	T/SGA/M/N/ LBW
97	17997	1/M	40 W	LSCS	N	7,9	23	1	N	N	Y	N	N	N	N	N	N	2.8	130	44	<3	36.6	WNL	WNL	WNL	GD	97	98	98	97	98	98	98	99												130	40	<3	36.5	98	GD	T/A/M/N
98	18038	1/M	38+6 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	3.2	140	42	<3	36.6	WNL	WNL	WNL	GD	95	95	96	95	99	96	96	96												120	42	<3	36.8	99	GD	T/A/M/N
99	18045	1/F	39+ 6 W	NVD	N	7, 9	21	1	N	N	N	N	N	N	N	N	N	1.9	140	50	<3	36.5	WNL	WNL	WNL	GD	95	95	96	94	98	98	97	99												140	44	<3	36.6	98	GD	T/SGA/F/N/LBW
100	18280	1/F	37 + 1 W	NVD	N	7,9	21	1	N	Y	Y	N	N	N	N	N	N	1.8	130	44	<3	36.5	WNL	WNL	WNL	GD	93	94	94	94	98	97	97	99												144	55	<3	37	97	GD	T/SGA/F/N/ LBW
101	18473	1/M	40 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2	138	42	<3	36.6	WNL	WNL	WNL	GD	94	94	95	96	97	97	97	98												148	44	<3	36.6	99	GD	Term/SGA/M/ N/LBW
102	18590	DOL	40 W	LSCS	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.9	130	42	<3	36.6	WNL	WNL	WNL	GD	94	95	95	95	97	96	96	96												130	55	<3	36.8	97	GD	T/A/M/N
103	18783	1/M	40 W	NVD	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2.9	130	44	<3	36.6	WNL	WNL	WNL	GD	95	94	94	96	98	96	97	97												120	44	<3	36.7	98	GD	T/A/F/N
104	18850	1/M	40+ 5 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.8	130	40	<3	36.4	WNL	WNL	WNL	GD	93	92	93	93	96	97	96	96												132	52	<3	36.6	100	GD	T/A/M/N
105	18903	1/M	35+4 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2	130	42	<3	36.5	WNL	WNL	WNL	GD	93	92	92	92	98	98	97	98												120	44	<3	36.6	98	GD	Late preterm/ A/M/N/LBW
106	19109	1/M	38 W	NVD	N	7,9	26	1	N	N	N	N	N	N	N	N	N	3	140	42	<3	36.6	WNL	WNL	WNL	GD	94	94	95	94	96	96	98	96												148	42	<3	36.6	99	GD	T/A/M/N
107	19210	1/F	39+5 W	NVD	N	7,9	20	2	N	N	N	N	N	N	N	N	N	3.2	140	44	<3	36.8	WNL	WNL	WNL	GD	97	96	96	96	99	98	98	97												130	46	<3	36.8	98	GD	T/A/F/N
108	19358	1/M	36+5 W	NVD	N	7,9	31	4	N	N	N	N	N	N	N	N	N	3.8	130	40	<3	36.6	WNL	WNL	WNL	GD	96	95	95	95	96	96	96	96												128	42	<3	36.6	99	GD	LPT/A/M/N/LBW
109	19699	1/F	38 W	NVD	N	7,9	35	2	N	N	N	N	N	N	N	N	N	2.3	140	44	<3	36.5	WNL	WNL	WNL	GD	97	97	97	96	96	97	97	98												130	42	<3	36.8	96	GD	T/A/F/N/LBW
110	19813	1/M	39+1 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	3.7	142	48	<3	36.7	WNL	WNL	WNL	GD	94	93	94	92	96	97	96	96												130	44	<3	36.9	96	GD	T/A/M/N
111	19910	1/F	39+5 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	3.5	138	42	<3	36.5	WNL	WNL	WNL	GD	94	95	94	94	96	96	96	96												150	45	<3	36.8	96	GD	T/A/F/N
112	19982	1/M	40 +2 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	1.9	120	40	<3	36.5	WNL	WNL	WNL	GD	97	97	96	97	100	100	100	98												130	44	<3	36.6	97	GD	T/S/F/N/LBW
113	20180	1/F	39+2 W	NVD	N	7, 9	28	4	N	N	N	N	N	N	N	N	N	3.4	140	44	<3	36.6	WNL	WNL	WNL	GD	97	95	95	96	99	98	98	96												144	42	<3	36.6	98	GD	T/A/F/N
114	20435	1/M	38+3 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.3	128	55	<3	36.6	WNL	WNL	WNL	GD	95	94	94	94	97	98	96	96												140	50	<3	36.6	97	GD	T/A/M/N/LBW
115	20762	1/M	38+ 3 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.6	140	40	<3	36.7	WNL	WNL	WNL	GD	99	98	98	98	97	97	97	96												140	44	<3	36.8	98	GD	T/A/M/N
116	20760	1/M	40 W	LSCS	N	7,9	27	2	N	N	N	N	N	N	N	N	N	3.2	130	45	<3	36.6	WNL	WNL	WNL	GD	96	94	95	94	97	96	96	96												130	40	<3	36.6	97	GD	T/A/M/N
117	20803	1/M	40 W	LSCS	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2.8	140	40	<3	36.8	WNL	WNL	WNL	GD	97	95	96	96	98	98	96	98												130	40	<3	36.8	96	GD	T/A/M/N

118	20864	1/M	39+1 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.6	130	42	<3	36.7	WNL	WNL	WNL	GD	94	94	95	94	97	96	96	96												140	44	<3	37.4	97	GD	T/A/M/N
119	20995	1/F	39 W	NVD	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.2	140	40	<3	36.8	WNL	WNL	WNL	GD	97	96	96	96	99	99	99	10												130	42	<3	36.6	99	GD	T/A/F/N
120	21124	1/M	38 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.7	130	52	<3	36.6	WNL	WNL	WNL	GD	97	98	97	96	99	98	98	98												140	46	<3	36.8	98	GD	T/A/M/N
121	21364	1/M	38 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.3	138	42	<3	36.5	WNL	WNL	WNL	GD	97	97	96	96	98	98	98	97												138	42	<3	36.6	97	GD	T/A/M/N/LBW
122	21468	1/M	39+2 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2	130	44	<3	36.8	WNL	WNL	WNL	GD	97	96	96	96	98	98	99	98												120	50	<3	36.7	98	GD	T/A/M/N/LBW
123	21617	1/F	40+2 W	NVD	N	7,9	26	2	N	N	N	N	N	N	N	N	N	2.6	120	40	<3	36.7	WNL	WNL	WNL	GD	95	96	96	96	99	99	99	99												134	42	<3	36.6	98	GD	T/A/F/N
124	21697	1/F	39 W	LSCS	Y	7,9	20	1	N	N	N	N	N	N	N	N	N	2.1	130	44	<3	36.8	WNL	WNL	WNL	GD	94	94	94	96	96	97	97	97												128	45	<3	36.8	96	GD	T/A/F/N/LBW
125	21864	1/M	39+4 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.8	134	44	<3	36.7	WNL	WNL	WNL	GD	96	96	95	96	98	98	97	98												130	44	<3	37	100	GD	T/A/M/N
126	21984	1/M	41+3 W	LSCS	N	7,9	24	1	N	N	N	Y	N	N	N	N	N	2.7	130	44	<3	36.6	WNL	WNL	WNL	GD	95	96	95	95	98	96	97	96												130	46	<3	36.8	98	GD	T/A/M/N
127	21829	1/M	36+6 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.6	130	42	<3	36.8	WNL	WNL	WNL	GD	96	94	94	94	96	96	97	96												140	50	<3	37.3	98	GD	Late Preterm/ A/M/N/
128	22310	1/F	39 W	NVD	Y	7,9	21	1	N	Y	Y	N	N	N	N	N	N	2.9	140	44	<3	36.5	WNL	WNL	WNL	GD	95	94	95	95	96	96	96	98												142	44	<3	37	100	GD	T/A/F/N
129	22646	1/F	39 W	NVD	N	7,9	24	4	N	N	N	N	N	N	N	N	N	3.6	130	45	<3	36.6	WNL	WNL	WNL	GD	96	97	96	96	97	99	97	98												130	55	<3	37.6	98	GD	T/A/F/N
130	22872	1/M	39+2 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.2	144	54	<3	37.6	WNL	WNL	WNL	GD	98	99	98	10	10	99	100	10												150	44	<3	37	99	GD	T/A/M/N
131	22952	1/F	37 W	LSCS	N	7,9	22	3	N	N	N	N	N	N	N	N	N	2.6	130	45	<3	36.6	WNL	WNL	WNL	GD	96	96	96	97	97	97	98	97												130	42	<3	36.8	98	GD	T/A/F/N
132	23215	1/F	38 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.3	144	42	<3	36.7	WNL	WNL	WNL	GD	95	96	95	95	98	98	97	98												134	50	<3	36.7	98	GD	T/A/F/N/LBW
133	23348	1/F	39 W	NVD	N	7,9	26	2	N	N	N	N	N	N	N	N	N	2.7	122	44	<3	36.9	WNL	WNL	WNL	GD	97	95	95	95	98	96	98	98												142	44	<3	36.8	97	GD	T/A/F/N
134	23555	1/M	38 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.5	140	42	<3	36.6	WNL	WNL	WNL	GD	97	95	97	96	98	98	99	10												124	46	<3	36.7	98	GD	T/A/M/N
135	23532	1/F	35 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	1.8	130	52	<3	36.8	WNL	WNL	WNL	GD	95	93	93	94	96	96	97	96												144	52	<3	36.8	100	GD	Late preterm/ A/F/N/LBW
136	23716	1/F	38+5 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.5	132	40	<3	36.6	WNL	WNL	WNL	GD	94	94	94	95	96	97	96	96												130	42	<3	36.7	98	GD	T/A/F/N
137	24088	1/M	40 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	3.6	130	45	<3	36.6	WNL	WNL	WNL	GD	94	93	93	95	96	96	96	97												138	58	<3	36.8	97	GD	T/A/M/N
138	24287	1/M	38+6 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.4	132	46	<3	36.8	WNL	WNL	WNL	GD	94	95	97	95	96	96	99	97												130	44	<3	37	99	GD	T/A/M/N/LBW
139	24448	1/F	38+6 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	3.2	140	44	<3	36.7	WNL	WNL	WNL	GD	94	93	94	95	96	99	99	98												150	54	<3	36.9	98	GD	T/A/F/N
140	24718	1/M	36 W	NVD	N	7,9	23	1	N	N	N	N	N	N	N	N	N	2.7	130	42	<3	36.6	WNL	WNL	WNL	GD	96	96	98	95	98	96	98	98												138	52	<3	36.9	98	GD	T/A/M/N
141	25285	1/F	38+1 W	NVD	N	7,9	22	2	N	N	N	N	N	N	N	N	N	3.2	120	48	<3	37	WNL	WNL	WNL	GD	96	95	95	95	96	97	96	96												120	50	<3	36.8	97	GD	T/A/F/N
142	25293	1/M	38 W	LSCS	N	7,9	20	2	N	N	N	N	N	N	N	N	N	2.9	130	48	<3	37.4	WNL	WNL	WNL	GD	95	95	95	94	97	99	99	97												122	44	<3	36.8	98	GD	T/A/M/N
143	25294	1/F	36+2 W	NVD	N	7,9	22	3	N	N	N	N	N	N	N	N	N	2.4	132	44	<3	37.2	WNL	WNL	WNL	GD	95	96	96	94	98	96	96	96												149	50	<3	37.2	98	GD	LPT/A.F/N/LBW
144	25359	1/F	39+5 W	LSCS	N	7,9	24	1	N	N	N	Y	N	N	N	N	N	2.9	128	50	<3	36.8	WNL	WNL	WNL	GD	97	96	96	96	98	98	98	98												130	44	<3	37.4	100	GD	T/A/F/N
145	25521	1/M	35 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.1	126	40	<3	36.6	WNL	WNL	WNL	GD	94	94	93	94	97	99	96	96												148	44	<3	37.2	98	GD	Late preterm/ A/M/N/LBW
146	24711	1/F	36+5 W	NVD	N	7,9	29	1	N	N	Y	N	N	N	N	N	N	2.9	132	40	<3	36.5	WNL	WNL	WNL	GD	95	95	94	95	98	10	100	97												146	44	<3	37.4	98	GD	LPT/A/F/N
147	25615	1/M	39 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.4	122	42	<3	36.7	WNL	WNL	WNL	GD	96	96	96	95	96	96	96	96												130	40	<3	36.8	98	GD	T/A/M/N/LBW
148	25600	1/M	38 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.9	120	50	<3	36.6	WNL	WNL	WNL	GD	95	97	97	94	98	10	98	98												155	42	<3	37.4	98	GD	T/A/M/N
149	25728	1/M	36+ 6 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	3.2	128	40	<3	36.6	WNL	WNL	WNL	GD	95	94	94	94	98	98	96	98												150	54	<3	37	98	GD	LPT/A/M/N
150	25850	1/F	38+5 W	LSCS	N	7,9	28	3	N	N	N	N	N	N	N	N	N	2.4	130	42	<3	36.6	WNL	WNL	WNL	GD	95	95	95	95	96	98	96	96												134	40	<3	36.8	98	GD	T/A/F/N/LBW
151	25855	1/M	39 W	LSCS	N	7,9	22	1	N	Y	Y	N	N	N	N	N	N	2.7	120	40	<3	36.8	WNL	WNL	WNL	GD	95	96	95	95	96	96	96	96												128	40	<3	37.4	97	GD	T/A/M/N
152	26168	1/F	38+1 W	LSCS	N	7,9	21	2	N	N	N	N	N	N	N	N	N	2.4	132	44	<3	36.4	WNL	WNL	WNL	GD	95	95	95	96	98	98	98	96												130	40	<3	37.2	98	GD	T/A/F/N/LBW
153	26103	1/F	38 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	3	140	44	<3	36.6	WNL	WNL	WNL	GD	94	94	94	95	96	97	97	96												120	42	<3	37.2	97	GD	T/A/F/N
154	25828	1/F	40 +2 W	LSCS	N	7,9	22	2	N	N	N	N	N	N	N	N	N	3.2	140	40	<3	36.5	WNL	WNL	WNL	GD	95	95	96	95	97	97	96	97												140	52	<3	37.5	98	GD	T/A/F/N
155	26176	1/M	40+5 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.2	130	42	<3	36.6	WNL	WNL	WNL	GD	96	98	96	95	98	10	98	96												120	44	<3	37	97	GD	T/A/M/N
156	26189	1/F	40+1 W	LSCS	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.5	120	44	<3	36.7	WNL	WNL	WNL	GD	96	96	96	95	99	98	98	96												140	40	<3	37.2	97	GD	T/A/F/N
157	26260	1/M	38 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.2	140	40	<3	36.8	WNL	WNL	WNL	GD	97	97	96	97	97	10	99	99												130	42	<3	37.4	96	GD	T/A/M/N/LBW
158	26196	1/F	39W	LSCS	N	7,9	27	2	N	N	N	N	N	N	N	N	N	2.8	132	55	<3	36.8	WNL	WNL	WNL	GD	95	97	95	95	98	98	99	96												120	40	<3	37.2	98	GD	T/A/F/N
159	26333	1/F	37+5 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	1.8	120	42	<3	37	WNL	WNL	WNL	GD	96	96	97	96	97	99	97	97												130	44	<3	37	97	GD	T/SGA/F/N/LBW

160	26182	1/F	35 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2	138	42	<3	36.8	WNL	WNL	WNL	GD	97	99	97	96	98	100	98	96												140	44	<3	36.8	99	GD	LPT/A/F/N/LBW
161	26481	1/M	38 W	NVD	N	7,9	26	2	N	N	N	N	N	N	N	N	N	2.4	130	40	<3	36.7	WNL	WNL	WNL	GD	96	98	96	96	97	97	97	96												128	45	<3	37	97	GD	T/A/M/N/LBW
162	26458	1/M	38 W	LSCS	N	7,9	22	2	N	N	N	N	N	N	N	N	N	3.1	130	48	<3	36.8	WNL	WNL	WNL	GD	95	95	95	94	100	100	100	98												120	40	<3	37.2	99	GD	T/A/M/N
163	26446	1/F	37 W	NVD	N	7,9	25	1	N	Y	Y	N	N	N	N	N	N	2.6	120	42	<3	36.5	WNL	WNL	WNL	GD	97	97	97	96	99	100	99	98												130	40	<3	37.4	98	GD	T/A/F/N
164	26483	1/M	40+1 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	3.2	136	40	<3	37	WNL	WNL	WNL	GD	95	94	95	95	96	98	96	96												132	50	<3	37.4	97	GD	T/A/M/N
165	26468	1/M	40 W	LSCS	N	7,9	20	1	N	N	N	N	N	N	N	N	N	3	144	48	<3	36.6	WNL	WNL	WNL	GD	96	98	99	96	100	99	99	97												128	52	<3	36.7	96	GD	T/A/M/N
166	26446	1/F	37 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.6	145	44	<3	36.8	WNL	WNL	WNL	GD	94	93	93	94	96	97	96	96												124	40	<3	36.8	98	GD	T/A/F/N
167	28950	1/M	39+2 W	NVD	N	7,9	35	3	N	N	N	N	N	N	N	N	N	3.2	130	42	<3	36.8	WNL	WNL	WNL	GD	94	95	95	95	96	96	98	96												140	42	<3	36.7	96	GD	T/A/M/N
168	28949	1/F	36+2 W	LSCS	N	7,9	25	1	N	N	N	Y	N	N	N	N	N	2.4	120	50	<3	36.7	WNL	WNL	WNL	GD	94	96	96	94	96	98	97	96												140	48	<3	37.5	96	GD	LPT/A/F/N/LBW
169	29001	1/M	37+1 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.4	130	40	<3	36.5	WNL	WNL	WNL	GD	96	96	96	97	97	97	97	98												150	52	<3	36.6	97	GD	T/A/M/N/LBW
170	29011	1/F	38 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	1.8	128	42	<3	36.8	WNL	WNL	WNL	GD	96	97	96	99	99	98	98	98												124	42	<3	36.7	98	GD	T/S/F/N/LBW
171	29012	1/F	38 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.1	120	40	<3	36.5	WNL	WNL	WNL	GD	95	95	96	95	96	97	96	96												130	44	<3	37.2	97	GD	T/S/F/N/LBW
172	29062	1/F	39 W	NVD	N	7,9	28	2	N	N	N	N	N	N	N	N	N	2.9	155	55	<3	36.7	WNL	WNL	WNL	GD	95	94	94	94	96	98	97	96												138	40	<3	37.3	96	GD	T/A/F/N
173	29128	1/F	40 W	LSCS	N	7,9	35	4	N	N	N	N	N	N	N	N	N	3.2	130	42	<3	37.2	WNL	WNL	WNL	GD	95	94	95	95	98	96	98	96												134	45	<3	36.8	96	GD	T/A/F/N
174	29166	1/F	40 W	LSCS	N	7,9	22	2	N	N	N	N	N	N	N	N	N	2.6	140	46	<3	36.6	WNL	WNL	WNL	GD	96	98	98	97	96	96	96	97												140	42	<3	36.8	97	GD	T/A/F/N
175	29250	1/F	38+1 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.5	125	56	<3	37	WNL	WNL	WNL	GD	95	96	98	95	96	97	96	96												135	45	<3	36.7	100	GD	T/A/F/N
176	29249	1/ M	38 W	LSCS	N	7,9	29	2	N	N	N	N	N	N	N	N	N	2.6	120	40	<3	37.4	WNL	WNL	WNL	GD	92	93	93	93	96	96	97	96												128	40	<3	37.2	98	GD	T/A/M/N
177	29168	1/M	39 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.8	130	44	<3	36.8	WNL	WNL	WNL	GD	96	96	97	96	98	96	96	96												120	42	<3	36.6	97	GD	T/A/M/N
178	29287	1/M	39+3 W	NVD	N	7,9	25	3	N	N	N	N	N	N	N	N	N	2.8	140	48	<3	36.6	WNL	WNL	WNL	GD	95	94	94	94	97	96	96	96												128	50	<3	37.2	96	GD	T/A/M/N
179	29525	1/M	38+6 W	LSCS	N	7,9	22	2	N	N	N	N	N	N	N	N	N	2.4	130	40	<3	37	WNL	WNL	WNL	GD	97	99	99	98	98	98	100	98												138	48	<3	37	100	GD	T/A/M/N/LBW
180	29555	1/F	40 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.8	120	50	<3	36.5	WNL	WNL	WNL	GD	96	96	96	96	97	99	96	96												130	42	<3	36.8	98	GD	T/A/F/N
181	29597	1/F	37 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.1	130	42	<3	36.8	WNL	WNL	WNL	GD	94	96	96	95	96	98	96	96												132	44	<3	37.5	97	GD	T/A/F/N/LBW
182	29605	1/M	36 W	NVD	N	7,9	33	1	N	N	N	N	N	N	N	N	N	2.9	140	44	<3	36.6	WNL	WNL	WNL	GD	96	96	97	96	97	97	97	98												135	46	<3	36.8	96	GD	T/A/M/N
183	29773	1/M	37 W	LSCS	N	7,9	23	1	N	N	N	N	N	N	N	N	N	2.7	130	42	<3	37.2	WNL	WNL	WNL	GD	96	98	96	96	97	97	97	96												144	48	<3	37.2	98	GD	T/A/M/N
184	29802	1/M	38+3 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	3.1	130	42	<3	36.6	WNL	WNL	WNL	GD	97	98	97	96	98	98	99	97												130	46	<3	36.8	97	GD	T/A/M/N
185	29899	DOL	39+5 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.3	140	42	<3	36.7	WNL	WNL	WNL	GD	97	98	96	96	96	99	98	98												146	44	<	36.7	98	GD	T/A/F/N
186	29492	1/F	35 W	NVD	N	7,9	24	3	N	N	N	N	N	N	N	N	N	2.2	144	45	<3	36.6	WNL	WNL	WNL	GD	96	95	95	95	96	97	96	96												140	46	<3	37	96	GD	LPT/A/F/N/LBW
187	29948	1M	39 W	LSCS	N	7,9	23	3	N	N	N	N	N	N	N	N	N	3.2	144	50	<3	36.8	WNL	WNL	WNL	GD	96	97	97	99	96	98	98	97												130	42	<3	37.4	97	GD	T/A/M/N
188	30035	1/M	38 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.7	130	42	<3	36.4	WNL	WNL	WNL	GD	97	97	99	96	96	97	96	96												134	42	<3	37.5	97	GD	T/A/M/N
189	30463	1/M	38 W	NVD	N	7,9	28	1	N	N	N	N	N	N	N	N	N	2.6	140	44	<3	36.8	WNL	WNL	WNL	GD	97	99	98	96	97	97	96	98												130	46	<3	36.7	97	GD	T/A/M/N
190	30583	1/F	35 W	NVD	N	7,9	38	1	N	N	N	N	N	N	N	N	N	2.8	130	44	<3	37	WNL	WNL	WNL	GD	97	97	96	95	97	96	96	96												130	44	<3	37	97	GD	LPT/A/F/N
191	30610	1/M	39 W	LSCS	N	7,9	30	3	N	N	N	N	N	N	N	N	N	2.7	140	45	<3	36.8	WNL	WNL	WNL	GD	96	97	96	96	97	97	96	97												130	44	<3	36.8	97	GD	T/A/M/N
192	30708	1/F	38 W	LSCS	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.5	144	46	<3	37.4	WNL	WNL	WNL	GD	96	97	97	98	97	97	98	97												140	45	<3	36.8	97	GD	T/A/F/N
193	30877	1/M	39 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.1	145	44	<3	37.5	WNL	WNL	WNL	GD	96	96	98	97	98	97	96	97												142	44	<3	36.7	97	GD	T/A/M/N
194	30955	1/M	39+6 W	LSCS	N	7,9	28	1	N	N	N	N	N	N	N	N	N	3.1	144	45	<3	36.8	WNL	WNL	WNL	GD	93	94	94	95	96	96	97	96												130	45	<3	37	97	GD	T/A/M/N
195	31186	1/F	40 +2 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	2.7	130	44	<3	36.7	WNL	WNL	WNL	GD	95	95	94	96	96	98	96	96												140	44	<3	37.2	97	GD	T/A/F/N
196	31369	1/M	38 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.8	140	45	<3	37	WNL	WNL	WNL	GD	98	97	99	99	98	98	99	98												142	50	<3	36.8	99	GD	T/A/M/N
197	31484	1/F	38 W	LSCS	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.5	144	52	<3	36.8	WNL	WNL	WNL	GD	95	96	96	97	98	97	97	99												138	50	<3	37.2	99	GD	T/A/F/N
198	31577	1/F	37 W	LSCS	N	7,9	42	4	N	N	N	N	N	N	N	N	N	3.5	145	46	<3	36.6	WNL	WNL	WNL	GD	95	96	96	97	98	97	98	98												125	50	<3	37	100	GD	T/A/F/N
199	31639	1/M	38+6 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	3.1	140	42	<3	36.6	WNL	WNL	WNL	GD	95	96	95	97	96	96	97	96												140	42	<3	37.4	98	GD	T/A/M/N
200	31847	1/M	40 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.5	144	46	<3	36.8	WNL	WNL	WNL	GD	95	94	94	96	96	96	98	99												144	50	<3	36.8	99	GD	T/A/M/N
201	31879	1/F	38+3 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.9	130	40	<3	36.6	WNL	WNL	WNL	GD	97	97	98	96	96	97	97	99												130	44	<3	37	98	GD	T/A/F/N
202	32030	1/M	40+2 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.1	144	50	<3	36.8	WNL	WNL	WNL	GD	95	96	96	97	96	96	97	96												145	50	<3	37.2	97	GD	T/A/M/N/LBW
203	32308	1/M	40 W	LSCS	N	7,9	27	2	N	N	N	N	N	N	N	N	N	2.7	130	45	<3	36.8	WNL	WNL	WNL	GD	95	95	96	95	96	96	97	97												142	44	<3	37	98	GD	T/A/M/N

204	32432	1/F	38 +6 W	LSCS	N	7,9	28	1	N	N	N	N	N	N	N	N	N	2.7	130	44	<3	36.6	WNL	WNL	WNL	GD	95	94	94	94	96	96	97	96												140	50	<3	36.8	96	GD	T/A/F/N
205	32459	1/M	38 W	NVD	N	7,9	28	2	N	N	N	N	N	N	N	N	N	2.4	140	42	<3	37	WNL	WNL	WNL	GD	95	97	95	95	97	97	97	98												130	44	<3	37.2	97	GD	T/A/M/N/LBW
206	32650	1/M	38 W	LSCS	N	7,9	28	2	N	N	N	N	N	N	N	N	N	2.8	130	40	<3	37.2	WNL	WNL	WNL	GD	96	96	97	96	96	98	98	97												140	40	<3	37.5	97	GD	T/A/M/N
207	32592	1/M	38+1 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	2.7	140	42	<3	36.6	WNL	WNL	WNL	GD	95	96	95	95	96	97	98	98												138	52	<3	37.4	96	GD	T/A/M/N
208	32834	1/M	39 W	LSCS	N	7,9	24	3	N	N	N	N	N	N	N	N	N	3	120	42	<3	36.5	WNL	WNL	WNL	GD	95	97	97	98	96	96	97	96												140	40	<3	37.5	96	GD	T/A/M/N
209	32955	1/M	39 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.6	130	42	<3	37.2	WNL	WNL	WNL	GD	96	97	96	96	97	98	98	97												120	42	<3	36.7	97	GD	T/A/M/N
210	33106	1/F	40 W	LSCS	N	7,9	21	3	N	N	N	N	N	N	N	N	N	3.5	135	40	<	36.7	WNL	WNL	WNL	GD	95	96	96	97	96	97	96	96												140	44	<3	36.8	97	GD	T/A/M/N
211	33233	1/F	38 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	3.2	130	44	<3	36.6	WNL	WNL	WNL	GD	95	95	94	95	96	96	97	96												130	45	<3	37.4	96	GD	T/A/F/N
212	33357	1/F	37 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	2.1	140	50	<3	36.8	WNL	WNL	WNL	GD	96	97	96	96	97	96	97	97												148	40	<3	36.8	96	GD	T/A/F/N/LBW
213	33454	1/F	35 W	NVD	N	7,9	28	1	N	N	N	N	N	N	N	N	N	2.6	120	44	<3	36.8	WNL	WNL	WNL	GD	96	96	96	97	97	98	98	97												140	44	<3	37.2	98	GD	LPT/A/F/N/LBW
214	33508	1/M	35 W	LSCS	N	7,9	30	3	N	N	N	N	N	N	N	N	N	2.4	130	42	<3	37	WNL	WNL	WNL	GD	98	97	99	99	96	98	96	96												140	42	<3	36.8	97	GD	LPT/A/M/N/LBW
215	33584	1/F	39 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	3	140	44	<3	36.5	WNL	WNL	WNL	GD	93	94	94	94	96	96	97	98												130	42	<3	37	97	GD	T/A/F/N
216	33679	1/M	38 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	3	130	50	<3	36.6	WNL	WNL	WNL	GD	97	96	96	99	97	97	97	96												140	44	<3	37.2	96	GD	T/A/M/N
217	33805	1/F	37+3 W	NVD	N	7,9	30	1	N	N	N	N	N	N	N	N	N	2.2	136	44	<3	36.8	WNL	WNL	WNL	GD	95	97	97	96	97	96	96	96												140	42	<3	36.5	97	GD	T/A/F/N/LBW
218	33910	1/ M	39+3 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.9	135	42	<3	37	WNL	WNL	WNL	GD	96	98	98	97	96	96	96	97												120	40	<3	36.6	96	GD	T/A/M/N
219	34251	1/F	38+ 6 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.5	120	50	<3	36.6	WNL	WNL	WNL	GD	96	97	96	95	96	97	98	96												139	44	<3	37.2	97	GD	T/A/F/N
220	34381	1/ F	39 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.6	130	44	<3	36.6	WNL	WNL	WNL	GD	94	95	95	95	96	96	96	97												140	45	<3	356.7	100	GD	T/A/F/N
221	34794	1/ F	41 W	LSCS	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.5	140	46	<3	36.8	WNL	WNL	WNL	GD	95	96	96	96	97	98	97	97												130	45	<3	37.3	97	GD	T/A/F/N
222	34957	1/F	40 W	LSCS	N	7,9	29	2	N	N	N	N	N	N	N	N	N	2.9	138	52	<3	37.4	WNL	WNL	WNL	GD	96	96	97	96	96	96	96	98												140	44	<3	37	96	GD	T/A/F/N
223	35138	1/M	40+5 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.8	140	46	<3	36.8	WNL	WNL	WNL	GD	96	97	97	98	96	97	96	96												130	44	<3	36.8	96	GD	T/A/M/N
224	35168	1/M	40 W	NVD	N	7,9	21	2	N	N	N	N	N	N	N	N	N	2.5	130	44	<3	36.6	WNL	WNL	WNL	GD	97	97	98	99	96	97	96	96												140	50	<3	37	97	GD	T/A/M/N
225	35228	1/F	39+1 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.1	138	46	<3	37	WNL	WNL	WNL	GD	96	96	97	97	97	96	96	96												130	42	<3	37.2	98	GD	T/A/F/N/LBW
226	35470	1/ M	40 +6 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	2.9	140	44	<3	36.8	WNL	WNL	WNL	GD	95	96	95	95	96	97	97	96												140	44	<3	37.2	96	GD	T/A/M/N
227	35524	1/F	40+1 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.9	130	45	<3	36.5	WNL	WNL	WNL	GD	94	95	95	95	98	96	96	96												144	50	<3	36.8	96	GD	T/A/F/N
228	35666	1/F	37+5 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.2	124	46	<3	36.8	WNL	WNL	WNL	GD	96	96	97	96	97	96	96	96												130	44	<3	36.8	97	GD	T/A/F/N/LBW
229	35810	1/ F	39 W	LSCS	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.7	138	40	<3	36.6	WNL	WNL	WNL	GD	96	96	95	96	96	96	97	96												142	46	<3	36.7	96	GD	T/A/F/N
230	35896	1/F	40 W	LSCS	N	7,9	27	2	N	N	N	N	N	N	N	N	N	2.3	130	44	<3	36.5	WNL	WNL	WNL	GD	95	94	94	94	96	96	96	97												142	46	<3	37.4	96	GD	T/A/F/N/LBW
231	36044	1/F	35 W	NVD	N	7, 9	29	1	N	N	N	N	N	N	N	N	N	2.7	120	48	<3	36.6	WNL	WNL	WNL	GD	93	95	94	94	96	98	99	99												130	42	<3	36.7	97	GD	T/A/F/N
232	36081	1/M	39 W	NVD	N	7,9	30	2	N	N	N	N	N	N	N	N	N	2.9	130	50	<3	36.6	WNL	WNL	WNL	GD	94	95	96	96	96	98	98	97												120	44	<3	36.8	96	GD	T/A/M/N
233	36157	1/M	38 W	NVD	N	7,9	24	2	N	N	N	N	N	N	N	N	N	2.2	140	44	<3	36.8	WNL	WNL	WNL	GD	97	96	97	98	97	98	98	98												130	50	<3	36.5	99	GD	T/A/M/N/LBW
234	36256	1/F	37 W	LSCS	N	7,9	28	1	N	N	N	N	N	N	N	N	N	2.3	130	44	<3	36.7	WNL	WNL	WNL	GD	96	97	96	96	97	98	98	99												140	40	<3	37	98	GD	T/A/F/N/LBW
235	36317	1/M	37+2 W	NVD	N	7,9	20	1	N	N	N	N	N	N	N	N	N	2.3	140	50	<3	36.6	WNL	WNL	WNL	GD	95	95	94	93	96	98	96	97												150	55	<3	36.8	100	GD	T/A/M/N/LBW
236	36583	1/F	40+3 W	LSCS	N	7,9	27	2	N	N	N	N	N	N	N	N	N	2.4	130	46	<3	36.8	WNL	WNL	WNL	GD	96	97	97	99	97	96	96	96												130	42	<3	36.8	97	GD	T/A/F/N/LBW
237	36721	1/M	40 W	NVD	N	7,9	23	1	N	N	N	N	N	N	N	N	N	3.4	120	44	<3	36.5	WNL	WNL	WNL	GD	97	96	96	96	96	96	97	98												140	44	<3	37	98	GD	T/A/M/N
238	36868	1/F	38 W	LSCS	N	7,9	31	1	N	N	N	N	N	N	N	N	N	3	130	40	<3	36.7	WNL	WNL	WNL	GD	96	97	96	96	97	97	97	98												148	50	<3	36.6	97	GD	T/A/F/N
239	36930	1/F	36+2 W	LSCS	N	7,9	20	2	N	N	N	N	N	N	N	N	N	2.4	120	44	<3	36.6	WNL	WNL	WNL	GD	96	95	96	96	96	96	97	96												140	44	<3	36.8	96	GD	LPT/A/F/N/LBW
240	37098	1/M	37 W	LSCS	N	7,9	21	1	N	N	N	N	N	N	N	N	N	3.5	130	42	<3	36.6	WNL	WNL	WNL	GD	97	96	96	96	96	97	98	98												120	50	<3	37.2	96	GD	T/A/M/N
241	37198	1/M	40 W	LSCS	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3.4	140	45	<3	36.7	WNL	WNL	WNL	GD	98	98	99	97	98	98	100	99												140	42	<3	37.4	100	GD	T/A/M/N
242	37422	1/F	39+ 5 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	2.4	130	44	<3	36.7	WNL	WNL	WNL	GD	96	98	96	97	97	97	98	97												120	44	<3	36.8	97	GD	T/A/F/N/LBW
243	37631	1/M	40 W	LSCS	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2.5	120	42	<3	36.8	WNL	WNL	WNL	GD	97	96	98	98	96	96	96	97												130	44	<3	36.5	97	GD	T/A/M/N
244	37734	1/F	37+4 W	NVD	N	7,9	27	2	N	N	N	N	N	N	N	N	N	2.3	140	55	<3	36.8	WNL	WNL	WNL	GD	99	98	98	99	98	99	99	10												144	50	<3	36.8	97	GD	T/A/F/N/LBW
245	37926	1/M	36+6 W	NVD	N	7,9	27	1	N	N	N	N	N	N	N	N	N	3	120	50	<3	37.2	WNL	WNL	WNL	GD	97	96	96	96	97	98	98	98												130	44	<3	37.5	98	GD	LPT/A/M/N
246	37999	1/F	38+6 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.5	130	44	<3	36.8	WNL	WNL	WNL	GD	98	99	99	100	97	98	98	99												120	55	<3	37.2	98	GD	T/A/F/N
247	38250	1/M	40 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.8	120	40	<3	36.8	WNL	WNL	WNL	GD	97	97	96	98	98	98	98	97												130	40	<3	36.8	97	GD	T/A/M/N

248	38315	1/M	40 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.8	150	55	<3	36.5	WNL	WNL	WNL	GD	95	95	96	95	99	10	99	99												120	44	<3	37.2	98	GD	T/A/M/N
249	38387	1/F	38 W	NVD	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2	120	44	<3	37	WNL	WNL	WNL	GD	96	96	97	97	97	98	98	98												130	45	<3	37.4	99	GD	T/S/F/N/LBW
250	38538	1/M	38 W	NVD	N	7,9	29	2	N	N	N	N	N	N	N	N	N	3	130	42	<3	37.5	WNL	WNL	WNL	GD	94	95	95	95	96	96	97	96												140	52	<3	37	97	GD	T/A/M/N
251	38557	1/F	40 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	3	120	50	<3	36.5	WNL	WNL	WNL	GD	95	96	96	98	99	99	98	10												130	44	<3	36.8	97	GD	T/A/F/N
252	40300	1/M	38+4 W	LSCS	N	7,9	23	1	N	N	N	N	N	N	N	N	N	2.2	130	40	<3	36.6	WNL	WNL	WNL	GD	96	96	95	96	97	96	96	96												140	50	<3	36.7	97	GD	T/A/M/N/ LBW
253	40475	1/M	35+1 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2	140	44	<3	36.8	WNL	WNL	WNL	GD	96	97	95	96	97	98	99	99												130	44	<3	37	99	GD	LPT/A/M/N/ LBW
254	41226	1/M	39 W	NVD	N	7,9	21	1	N	N	N	N	N	N	N	N	N	2.3	120	50	<3	36.5	WNL	WNL	WNL	GD	96	96	97	96	96	97	96	96												144	40	<3	36.8	97	GD	T/A/M/N/ LBW
255	41485	1/M	40 W	LSCS	N	7,9	25	2	N	N	N	N	N	N	N	N	N	3.5	122	42	<3	36.8	WNL	WNL	WNL	GD	96	97	96	96	96	96	96	98												120	54	<3	37.4	96	GD	T/A/M/N
256	41710	1/M	38+3 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.5	140	46	<3	37	WNL	WNL	WNL	GD	96	96	96	97	96	98	96	96												130	44	<3	36.8	98	GD	T/A/M/N
257	41759	1/M	39 W	NVD	N	7,9	35	3	N	N	N	N	N	N	N	N	N	3.2	120	44	<3	36.8	WNL	WNL	WNL	GD	94	95	94	94	96	96	97	96												130	44	<3	36.8	96	GD	T/A/M/N
258	41837	1/M	38+ 3 W	NVD	N	7,9	25	3	N	N	N	N	N	N	N	N	N	2.4	130	44	<3	37.2	WNL	WNL	WNL	GD	96	96	98	96	97	98	97	97												140	50	<3	37.4	96	GD	T/A/M/N/ LBW
259	42040	1/M	41 W	LSCS	N	7,9	19	1	N	N	N	N	N	N	N	N	N	3	144	46	<3	36.6	WNL	WNL	WNL	GD	96	97	96	96	96	97	97	97												120	44	<3	36.6	97	GD	T/A/M/N
260	42082	1/F	40 W	LSCS	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2.4	120	44	<3	36.6	WNL	WNL	WNL	GD	96	97	96	96	97	96	96	96												130	50	<3	36.8	96	GD	T/S/F/N/ LBW
261	42446	1/M	40 W	NVD	N	7,9	25	2	N	N	N	N	N	N	N	N	N	2.9	130	48	<3	36.5	WNL	WNL	WNL	GD	96	96	97	96	96	98	98	99												140	46	<3	36.6	97	GD	T/A/M/N
262	42516	1/F	40 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	3	125	44	<3	36.7	WNL	WNL	WNL	GD	96	97	96	96	97	98	98	98												130	44	<3	37	96	GD	T/A/F/N
263	42481	1/M	38+3 W	NVD	N	7,9	19	2	N	N	N	N	N	N	N	N	N	2.6	130	50	<3	37	WNL	WNL	WNL	GD	93	95	96	96	96	96	97	96												140	50	<3	36.6	97	GD	T/A/M/N
264	42457	1/F	39 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.5	130	44	<3	36.6	WNL	WNL	WNL	GD	96	96	97	96	97	97	97	98												120	52	<3	37	97	GD	T/A/F/N
265	42447	1/F	40 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	3	140	55	<3	36.8	WNL	WNL	WNL	GD	96	97	96	96	97	97	98	97												130	44	<3	36.8	97	GD	T/A/F/N
266	42643	1/F	38 W	LSCS	N	7,9	26	1	N	N	N	N	N	N	N	N	N	2.8	130	46	<3	36.6	WNL	WNL	WNL	GD	97	98	99	99	99	99	100	99												140	46	<3	36.7	98	GD	T/A/F/N
267	42617	1/M	39 +1 W	NVD	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.4	120	48	<3	36.5	WNL	WNL	WNL	GD	95	96	96	96	96	97	96	96												130	46	<3	37.2	96	GD	T/A/M/N
268	42622	1/F	38+4 W	LSCS	N	7,9	18	1	N	N	N	N	N	N	N	N	N	2.6	130	44	<3	36.8	WNL	WNL	WNL	GD	97	97	97	96	99	98	98	98												130	44	<3	36.6	97	GD	T/A/F/N
269	42518	1/M	40 W	LSCS	N	7,9	36	2	N	N	N	N	N	N	N	N	N	2.9	125	44	<3	37	WNL	WNL	WNL	GD	96	96	96	97	96	97	97	97												140	50	<3	37.2	97	GD	T/A/M/N
270	42738	1/M	39+2 W	NVD	N	7,9	30	3	N	N	N	N	N	N	N	N	N	3.2	130	50	<3	36.6	WNL	WNL	WNL	GD	96	96	97	96	97	97	98	97												130	44	<3	36.7	97	GD	T/A/M/N
271	42636	1/M	39+6 W	LSCS	N	7,9	26	1	N	N	N	N	N	N	N	N	N	3	120	46	<3	36.9	WNL	WNL	WNL	GD	95	97	96	99	96	97	96	96												140	50	<3	36.8	97	GD	T/A/M/N
272	11100	1/F	40 W	LSCS	N	7,9	22	1	N	Y	N	N	N	N	N	N	N	2.6	170	84	4	37.8	WNL	WNL	WNL	GD	88	89	89	88	93	94	94	93	28060	81.5/12	15	3.0	44.2	PNU	3200	0.3	31	ST	NOR	130	42	<3	36.5	95	GD	T/A/F/N/ EONS
273	11290	1/M	38+6 W	NVD	N	7,9	23	1	N	N	N	N	N	N	N	N	N	2.5	175	80	3	38	WNL	WNL	WNL	GD	87	89	88	89	94	95	94	94	22530	64.2/27	15	3.1	43.5	NOR	2400	0.25	23	ST	PFO	140	45	<3	37	96	GD	T/A/M/N/ EONS
274	41879	1/F	37 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.6	140	44	<3	36.5	WNL	WNL	WNL	GD	95	95	96	95	96	97	97	99												130	44	<3	36.8	95	GD	T/A/F/N
275	41837	1/F	37+4 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.8	144	45	<3	36.5	WNL	WNL	WNL	GD	94	94	95	96	96	97	96	96												140	50	<3	37	96	GD	T/A/F/N
276	41747	1/F	38 W	NVD	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.6	140	46	<3	36.7	WNL	WNL	WNL	GD	95	95	96	97	97	96	96	96												150	44	<3	36.8	97	GD	T/A/F/N
277	41206	1/M	37 W	NVD	N	7,9	26	3	N	N	N	N	N	N	N	N	N	2.9	130	50	<3	36.8	WNL	WNL	WNL	GD	98	98	97	98	99	99	100	99												144	46	<3	36.5	96	GD	T/A/M/N
278	41150	1/M	39 W	NVD	N	7,9	24	1	N	N	N	N	N	N	N	N	N	2.6	140	44	<3	37	WNL	WNL	WNL	GD	96	96	97	96	98	97	97	97												130	50	<3	36.9	96	GD	T/A/M/N
279	17696	1/F	40 W	LSCS	N	7,9	24	1	N	N	N	N	N	N	N	N	N	3	175	84	4	37.6	WNL	WNL	WNL	GD	88	89	89	87	91	93	94	94	20210	77/15	16	3.6	49.6	PNU	2200	0.2	28	ST	NOR	130	44	<3	37.2	96	GD	T/A/F/N/ EONS
280	41047	1/M	37 W	LSCS	N	7,9	22	1	N	N	N	N	N	N	N	N	N	2.9	130	46	<3	36.8	WNL	WNL	WNL	GD	95	96	96	96	98	97	97	97												140	46	<3	36.8	97	GD	T/A/M/N
281	17631	1/F	41 W	LSCS	N	7,9	30	4	N	N	N	N	N	N	N	N	N	2.7	182	82	4	37.8	WNL	WNL	WNL	GD	89	88	88	89	94	94	95	94	25990	52/36	18	1.9	56.6	PNU	2600	0.3	11	ST	NOR	140	50	<3	37.4	97	GD	T/A/F/N/ EONS
282	40923	1/M	35+5	LSCS	N	7,9	25	1	N	N	N	N	N	N	N	N	N	2.9	140	45	<3	36.8	WNL	WNL	WNL	GD	96	97	97	97	96	97	96	96												146	50	<3	36.8	97	GD	T/A/M/N