The risk of bacteriuria from a single catheterization of the bladder is conservatively quoted at 3-4%. With an indwelling catheter the incidence of bacteriuria is directly related to the length of catherization and it varies from 5 to 10% per day of catheterization. This is in direct contrast to the 1% risk that non-catheterized patients will acquire bacteriuria during hospitalization. Though the majority of this is asymptomatic bacteriuria, it may progress to symptomatic urinary tract infection in 10-30% of patients. Bladder irrigation, antiseptic solutions in the drainage bag, and uretheral meatus cleaning have shown little benefit [7].

In a study by Summitt et al [8] on 100 women undergoing routine vaginal hysterectomy, randomly assigned to have an indwelling Foley's catheter for 24 hours following surgery or no catheter the incidence of febrile morbidity was markedly different and statistically significant between the two groups viz., 24.5% in the catheter group vs only 8% in the non-catheterised group, despite equal demographics and use of prophylactic antibiotics in both the groups.

In another prospective randomized controlled trial by Dunn et al [9], 250 women who underwent hysterectomy and who did not require bladder suspension or strict fluid replacement, the indwelling catheter was removed either immediately or on the first day after the operation. The findings of this study suggest that the immediate removal of the catheter at the completion of an uncomplicated surgical procedure was not associated with any adverse outcome like recatheterization, urinary tract infection, and febrile episodes.

Further, more patients in the immediate post-surgery catheter removal group reported less pain, which was associated with the use of an indwelling catheter. The pain was either no different than that after the operation or was less than that in the group in which the catheter was maintained for 24 hours [9].

Summitt et al [8] did a randamized prospective trial in women underging hysterctomy – 100 with post-operative indwelling bladder catheter drainage and 100 without – and found a significant difference in the incidence of post-operative urine culture between the two groups at both 48 hours and 2 weeks after surgery. At 48 hours the incidence of positive urine culture was 48% in the indwelling catheter group as compared to 16.3% in the group without indwelling catherter. They have stopped using post-operative catheter drainage of the bladder after vaginal hysterectomy.

Short term catheter drainage for 24 hours after simple vaginal or abdominal hysterectomy is still a common practice, whether for patients convenience, for monitoring of urinary output, or for anticipated patient comfort. But it appears to be unnecessary. We must encourage the practice of no indwelling bladder catheter drainage after uncomplicated vaginal or abdominal hysterectomy. Will this information be considered sufficient to convince some to abandon or at least modify a tenet of gynecologic surgery [2] ? Abandoning indwelling catheterization will result in no technical operative problems, low post-operative catheterization rates, low incidence of infection, potential cost savings, less patient discomfort, and early patient ambulation.

Patients' and nurses' education is extremely important, as is working experience, which permits better predictability as to how quickly to intervene with distress catheterization. Richardson et al [6] conclud that the use of urethral catheterization after routine abdominal or vaginal hysterectomy defies a rational explanation and that gynecological surgeons have an emotional attachment to the catheter. It is time to abandon routine use of indwelling catheter after simple hysterectomy.

References

1. Cardosi RJ, Cardosi RP, Captain MC. Infectious urinary morbidity with bladder catheterization after hysterectomy. *Am J Obstet Gynaecol 2003;189:380-4.*
2. Dixon RE. Effect of infection on hospital care. *Ann Intern Med 1978;89:749-53.*
3. Givens CD, Wenzel RP. Catheter associated urinary tract infections in surgical patients: a controlled study on the excess morbidity. *J Urol: 1980;124:646-8.*
4. Martinez DV, Civette JM, Anderson K et al. Bacteriuria in the catheterized surgical care patient.*Criti care Med 1986;14:188-91*
5. Bartzen PJ, Hafferty FW. Pelvic laparotomy without an indwelling catheter. *Am J Obstet Gynaecol 1987;156:1426-32.*
6. Richardson AC, Lyon JB, Graham E E et al. Abdominal hysterectomy - selationship between morbidity and surgical technic. Am J Obstet Gynecol 1973;116:953-61.
7. Warren JW. Catheter associated urinary tract infection. *Infect Dis Clin North Am 1997;11:609-22.*
8. Summitt RL, Stovall TG, Bran DF. Prospective comparison of indwelling bladder catheter drainage versus no catheter after vaginal hysterectomy. *AM J Obstet Gynecol 1994:70:1815-9.*
9. Dunn TS, Judith S, Dave F. Are indwelling catheter necessary for 24 hours after hysterectomy? *Am J Obstet Gynecol 2003;189:435-7.*

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REVIEW ARTICLE

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Obstetrician and the newborn

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Introduction

General pediatricians as well as obstetricians besides neonatologists, are often involved in the care of neonate and receive calls for newborns that somehow do not seem to be doing well. Often such calls are trivial and do not need anything more than reassurance to a parent. However there is a fine line of distinction between an apparently healthy neonate with innocuous manifestation and a one with much more severe condition needing urgent attention.

With over 28 million births every year in India, the burden of primary evaluation of a neonate often lies at the doorstep of the obstetrician, who is the fetal care giver most of the times. Neonatal care providers need to be aware of danger signs of a sick newborn such as poor sucking, rapid breathing at rates greater than 60 breaths/minute, difficult breathing, baby cold to touch, lethargy, uprolling of eyeballs, jaundice, abdominal distention, and cyanosis.

The obstetrician of yesteryear was the primary physician to the neonate. As pediatrics developed as a speciality in our country this onus shifted to the pediatrician, and now it lies on the neonatologist if he is available. With neonatology now a definite subspecialty, the obstetrician has slowly handed over the responsibility of perinatal care to this younger and often well-trained super specialist. In the process the eagle eyed obstetrician of the past is now a silent spectator to newer high-tech neonatal diagnosis and procedures.

Yet, the importance of updating and re-educating oneself and also of participating in the management of neonatal care must be nurtured by every obstetrician, as so often the parents seek his advice and guidance in every critical care management issue. Finding appropriate guidance and answers on his own can be difficult for the obstetrician since neonatal literature is often prosed in complex jargon and written for the benefit of graduating neonatologists.

Obstetricians are often presented with a situation that requires tertiary level care, not available in most delivery centers and hence the newborn needs to be transported away from the mother. This is often a frightening and alarming scenario for parents. The practice of in-utero transfer has not yet gained momentum in India, although in larger metropolises this is now catching up. In this brief review we have tried to focus on a few important day-to-day issues, which concern the welfare of the neonate soon after birth. Circumstances often render it mandatory for the obstetrician to shoulder the responsibility of handling these issues.

Cardiopulmonary status

Birthing is a traumatic transport of the fetus from a warm, cozy, intrauterine environment to a cold, often callous and cruel world. This transitional period from fetus to neonate involves a number of complex physiological readaptative mechanisms and the recognition of problems during this period of neonatal life requires astute clinical acumen and experience.

The breathing pattern of the newborn is governed by both his respiratory drive, which is central, and by his ability to reabsorb fetal lung fluid within the first few hours after birth. Transient tachypnea of newborn (TTN) occurs when this fluid absorption mechanism is delayed and is indeed the commonest cause of respiratory distress in an otherwise normal, unasphyxiated neonate born at term [1].

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Breathing at birth is sometimes shallow and occasionally irregular, with pauses of up to 10 seconds. Pauses of greater than 20 seconds (apnea), particularly if associated with a drop in the heart rate, result in hypoventilation and cyanosis, and herald an underlying major problem. Apneic neonates with ventilatory insufficiency and dropping heart rate need positive pressure ventilation. Whereas in the past it was considered essential to use 10% oxygen for resuscitation, a large body of evidence has now documented that ventilation with room air is equally effective [2,3].

Similarly, transitional circulatory changes occur due to a shift from a predominantly high resistance pulmonary vascular circuit to a predominantly low pulmonary pressure circuit as a result of expansion of alveoli and establishing respiration. Respiratory distress syndrome (RDS), meconium aspiration syndrome (MAS) and pneumonia i.e. parenchymal lung disease, precipitate pulmonary vasoconstriction whereby postnatal elevation of pulmonary vascular resistance results in the continuation of a fetal like circulation with right to left shunting resulting in cyanosis. Peripheral cyanosis is often seen in neonates with exposure to cold, yet peripheral cyanosis may be an early sign of sepsis and hypoglycemia, and must not be ignored unless more serious conditions are ruled out. Almost all neonates with cardiopulmonary defects are not only tachypneic and cyanosed soon after birth but are unable to feed, as the process of feeding requires intact and clear airway and an uncompromised hemodynamic circulation.

Cardiopulmonary difficulty soon after birth may be due to any of the following critical conditions – bilateral choanal atresia, RDS, MAS, congenital heart defect, congenital diaphragmatic hernia (CDH) and esophageal atresia. All the above conditions can be easily diagnosed and should be essentially recognized by the obstetrician. An erect x-ray film, with a 5 or 6 F nasogastric tube (NG) passed into the stomach is often all that is needed to clinch the diagnosis without the expertise of a neonatologist. So also the administration of 100% oxygen by hood at 5-6 L/ minute which clears or reduces cyanosis is evidence of a respiratory pathology causing poor alveolar capillary exchange. Choanal atresia albeit rare, and almost invariably undetectable on fetal ultrasonography (USG), is a life-threatening emergency and can easily be diagnosed by inability to pass a NG tube in to the nostril. Fortunately most CDHs are now diagnosed on fetal USG, enabling immediate postnatal transfer and early surgical correction. The outcome of neonates with CDH has improved dramatically since the availability of fetal USG for diagnosis.

The delivery room and the operation theater temperature are often too low for the comfort and well being of a neonate, particularly those born low weight or compromised. Most temperature settings are suited for the comfort of delivery room personnel. We wish to emphatically remind the obstetricians that nothing can be more damaging than hypothermia to a fetus accustomed to a warm cozy intrauterine environment. It is not an uncommon sight to see newborns left uncovered and unattended in bassinets, for significant lengths of time till the obstetrician completes the delivery procedures. This practice is detrimental to the welfare of a newborn that already has to cope with so many transitional physiologic changes. The ensuing hypothermia exacerbates hypoxia and acidosis, and neonates are most susceptible to hypothermia in the first 12-24 hours of life. This can be particularly more damaging to a preterm, low birth weight (LBW), or growth retarded new born who has poor mechanism of temperature maintenance due to inadequate brown fat content [4]. All delivery rooms and nurseries must be equipped with a source of heat, preferably a radiant warmer to maintain an ambient temperature of approximately 28 degree Celsius. In our experience more than 80% of the preterm, LBW, and sick neonates are hypothermic on admission to neonatal care units. It is, hence, very important to examine all neonates in a thermal neutral environment, when assessing their cardiopulmonary status. Currently all neonates with cardiopulmonary disorders are monitored by pulse oxymetry which is a non-invasive and reliable method for monitoring oxygen saturation [5].

Hypoxia

The immediate assessment of the newborn is traditionally done by using the apgar score, which has come under criticism because of its inability to accurately predict perinatal asphyxia and long-term neurological defects. First described by Dr Virginia Apgar in 1952, it is an objective tool that measures 5 signs of physiological adaptatoin. These measures are heart rate, respiratory effort, color, muscle tone and reflex response to stimulus. Many informed mothers today would like to know the apgar score on delivery itself [6]. Asphyxia is defined as hypoxia, hypercarbia and metabolic acidosis [7,8]. All fetuses experience some degree of asphyxia due to uterine contractions, however only when this is significant there are clinical evidences of hypoxic ischemic encephalopathy (HIE). A diagnosis of perinatal asphyxia must be made with great degree of caution, as there is no real accurate clinical definition of asphyxia and one is likely to unfairly incriminate an obstetrician colleague.

The ischemia and ensuing reperfusion, produce neuronal

injury resulting in HIE as a result of cerebral edema, infarction, and/or bleed. Currently long standing recommendation for use of 100% oxygen in neontal resuscitation stands challenged, as there is compelling evidence to suggest that the explosive release of free oxygen radicals during resuscitation with 100% oxygen can worsen brain injury. This has prompted clinicians to standardize resuscitation with room air [2,9] .

Five percent of the babies at birth do not establish respiration. Initial stabilization includes drying and placing the neonate under the radiant warmer and cleaning the airway of apneic or gasping neonates. Routine airway suction is not required in vigorously crying babies and in fact it may induce apnea [10]. Presence of meconium stained amniotic fluid requires oral suction at the delivery of the head and tracheal suction is to be done only in depressed neonates [11]. Although cardiopulmonary resuccitation in the delivery room is a life saving procedure ethical issues arises when the baby is extremely LBW or preterm and has severe birth asphyxia or lethal congenital malformation.

HIE can be evaluated clinically as well as with the help of EEG, and neuroimaging (USG, CT/MRI) the brain. In general it would be safe to say that a neonate who is neurologically intact at the end of a week and has a normal EEG at one week is unlikely to have sustained permanent brain injury and is expected to have a normal neurodevelopment [9] . The entire exercise of the above evaluation is to institute early neuroprotective measures. Various agents or measures including phenobarbitone, calcium channel blockers, magnesium sulphate, allopurinol, mannitol and even selective head cooling have been tried in an attempt to reverse the adverse effect of cerebral injury [12,13]

Congenital anomalies

After assessment of the cardio-pulmonary status, the first duty of the attending physician is to make a search for congenital anomalies. These may be obvious (overt) but more often than not, not quite so. Malformation may be trivial or life threatening. With the advent of fetal USG fortunately most renal and urinary tract anomalies, CDHs, upper gastro-intestinal obstructions, many congenital heart defects, and neural tube defects are already suspected and diagnosed before delivery. There remain a few malformations, which are likely to be overlooked unless a conscious search is made for the same. A cleft palate without a cleft lip is often overlooked and so are congenital anorectal anomalies. Congenital anomalies are important causes of neonatal mortality. Although prenatal diagnosis of severe birth defects has led to termination of the affected pregannncy before viability many infants are born with major malformations. Although it is beyond the scope of this review to detail all congenital anomalies, it should suffice to say that the few anomalies listed above must be ruled out in every infant.

One anomaly that needs special mention is ambiguous genitalia. The first question ever posed to the obstetrician and pediatrician on delivery is the sex of the infant. There can be nothing more disastrous than informing the parents that the gender of the neonate is indeterminate. Although this is not a major life-threatening defect, except in cases of salt losing congenital adrenal hyperplasia (CAH), the adverse psychological impact on parents and family is unimaginable. The gender of the infant must be correctly established at the earliest and appropriate counseling offered to the family.

Early neonatal infection

Bacterial sepsis is a serious problem in the neonatal period. After residing in a relatively sterile environment throughout fetal life, the newborn who has a poorly developed humoral and cellular immune system is often challenged with bacterial invasion at or soon after birth. Intra-amniotic infection and premature rupture of membranes causing maternal bacterimia are important causes of early neonatal sepsis and are responsible for increase in perinatal mortality rate (PNMR) [16]. Factors predisposing to chorioamnionitis include repeated unclean vaginal examination and prolonged rupture of membranes (>12-18 hours). The diagnosis of chorioamnionitis is made in the presence of maternal fever, fetal tachycardia, uterine tenderness, foul smalling amniotic fluid, and maternal leucocytosis. Such vertically transmitted infections manifest early in newborn period. The management of such maternal infection consists of the administration of intrapartum antibiotics and early delivery of the fetus. The standard recommended antibiotics include a combination of ampicillin and gentamycin, since the pharmacokinetics including placental transfer of above antibiotics have been extensively studied in pregnancy [17].

Untreated neonatal sepsis is life threatening and therefore its early identification and treatment becomes essential. The symptoms of early sepsis are few and even in established septicemia these are so nonspecific and vague that it is often not possible to identify whether a sick newborn is bacterially infected or has any other life threatening disorder like intracranial hemorrhage (ICH), hypoglycemia, inborn errors of metabolism (IEM) etc.

The overuse and misuse of antibiotics during the perinatal period has lead to the emergence of resistant bacterial strains, which are sensitive only to the newer, later

generation, and more expensive antibiotics. The practice of routine use of antibiotics in a neonate after a clean vaginal or cesarean delivery is to be discouraged. However the outcome of infected newborn is so poor that it is essential that all those delivered following chorioamnionitis must receive prompt and adequate antibiotics. Fortunately the incidence of neonatal tetanus and omphalitis has decreased significantly in our country due to near universal maternal immunization against tetanus and the practice of good hygienic cord care practices.

The anemic newborn

Anemia in the new born can be caused by blood loss, decreased red cell production, and increased red cell destruction. Cord hemoglobin (Hb) in term infants approximates 16.5 g/dL and anemia in a term neonate is defined as Hb less than 13.5 g/dL although clinically recognized anemia is usually indicative of Hb less 10 g/dL. Severe anemia at birth presents with pallor, heart failure, and shock. From a practical stand point there are only two important and common causes of anemia during this period and they are, hemolytic anemia due to Rh incompatibility and anemia due to fetal and neonatal hemorrhage.

Rh hemolytic disease which was the commonest cause of anemia in the past is now so rare, that many of the newer pediatricians are yet to see a hydropic fetus born due to Rh incompatibility. The credit for this near eradication is entirely due to excellent antenatal surveillance of potentially Rh affected fetuses, evaluation on Rh antibody titres during pregnancy, universal post-delivery use of Rh anti-globulin, and reduction in family size.

Anemia due to Rh disease is associated with hepatosplenomegaly, rapid rise in indirect serum bilirubin, a strongly positive direct coombs test, and high reticulocyte count [18]. Hemolytic disease caused by ABO incompatibility is milder and seldom, if ever, presents as severe early anemia.

Anemia due to blood loss may be either obvious such as in bleeding from the cord or hidden i.e. feto-maternal or feto-fetal hemorrhage. Acute feto-maternal bleeds, particularly if large, are likely to leave the neonate in depleted blood volume as well as decreased oxygen carrying capacity and may be hazardous. If the bleeding is chronic the neonate, although anemic, is able to compensate for blood loss and is usually not distressed.

The diagnosis of feto-maternal hemorrhage is suspected in any anemic newborn without hepatosplenomegaly or without other laboratory evidence of hemolysis [19]. Large feto-maternal bleeds that leave a neonate volume depleted are diagnosed by the detection of fetal cells in maternal circulation by the Kleihauer-Betke test and are best treated by emergency blood transfusion of maternal to the newborn to maintain Hb levels of >10g/dL. Feto-fetal bleeds i.e. twin to twin transfusion syndrome which can occur in monozygous twins can also be occasionally observed but often does not need intervention. The recipient twin is volume overloaded and polycythemic and donor twin is volume depleted and anemic. In life threatening situations a volume of 10 to 15 mL/kg blood can be removed from recipient twin and transfused directly to the anemic twin.

Hemorrhage due to causes other than those mentioned above are extremely rare at birth, although deficiency of various coagulation factors and like fibrinogen, and factor VIII and XIII is responsible for neonatal bleeds, these usually occur after first few days of life. The commonest defect in the coagulation cascade, which can cause hemorrhage soon after birth, is classic hemorrhagic disease of newborn, which is easily prevented by routine administration of 1mg vitamin K at birth [20].

Breast feeding

Fortunately the old adage *'breast is best'* is in vogue again. Lactation failure was the terminology coined and enforced by industries interested in promotion of infant milk formula. It is our confirmed opinion that if a woman can defecate and urinate she must also be able to lactate. From the late 60's and up to early 80's breast feeding (BF) practices had dwindled to the extent that many women, particularly of the upper socio-economic strata have forgotton the virtue of breast milk and breast-feeding. All this is now reversed because of the concerted efforts of *baby friendly initiative*. It must be the priority of every perinatal caregiver to promote BF. The virtues of breast milk and feeding have been reiterated so often that they do not require mention here. However it required a herculean effort from the Unicef, WHO and other breast-feeding promoting agencies to bring *'sanity'* back in to place. It is now up to us to maintain such sanity by ensuring a positive attitude towards continuing breast-feeding. Without effective education and advice women are apt to receive conflicting advice often based on hearsay.

The 10 steps to successful breast-feeding provided the base line for effecting such a change. These policies must be stringently followed in every birthing center. In brief these steps include maintaining a written breast feeding

policy, training all perinatal personnel and educating every pregnant woman about the benefits of breast feeding, helping young, inexperienced mothers on how to breast feed, rooming in newborn with the mother, and absolute avoidance of infant foods, prelactaeal feeds, pacifiers etc. It must be the aim of every birthing center to get baby friendly accreditation. The implementaton of the above policies required a change in direciton as we continue to transform our earlier practices. Perinatologists must provide information on breast-feeding, encourage skin-to-skin contact of mother and neonate, and initiate breast feed *within one hour* of birth. Mothers who are separated from the newborn must pump out breast milk at least 4-5 times a day [14]. Breast-feeding should be encouraged on demand and a policy of *ban the bottle* adopted. Offering bottle-feeds leads to nipple confusion and a preferential trend on the part of the infant to suck from an easier accessible source i.e. bottle, leading to decrease in the production of breast milk [15].

Breastfeeding however is avoided under exceptional cirucmstances as in mothers who are taking medication such as radioisotopes, anti-metabolies, cancer therapy and anticoagulants, and in neonates with galactosemia. Besides these uncommon situation breastfeeding should be universally promoted under all circumstances.

Equipments needed at a birthing center

The following equipments must be available in working order at every birthing centre -

1. Resuscitation bag and mask.
2. Oxygen hood, and source
3. Mucus extractor
4. Infant laryngoscope with straight and curved blades
5. Endotracheal tubes of size 2,2.5 and 3.3
6. Suction machine
7. Suction catheters No. 8F and 10F
8. Nasogastric tubes of 5F and 6F
9. Low temperature reading thermometer
10. Infant radiant warmer
11. Phototherapy
12. Pulse oxymeter
13. Emergency medications - atropine, adrenaline, calcium gluconate, sodium bicarbonate.

References

1. Rigatto H. Control of breathing in fetal life and onset and control of breathing in the neonate. In: Polin RA, Fox WW (eds.) *Fetal and neonatal physiology. 2nd edn.* Philadelphia, WB Saunders. *1998;1118-29.*
2. Ramji S, Ahuja S, Thrupuram S et al. Resuscitation of asphyxia in newborn with room air or 100% oxygen. *Pediatrics Resuscitation. 1993;34:809-12.*
3. Ola DS. The role of oxygen in neonatal resuscitation. *Clin Perinatal 2004;31:431-43.*
4. Kimberlec C, Michael EF, John PC. Temperature control. In: Cloherty JP, Ann RS (eds.). *Manual of neonatal care. 4th edn.* Philadolphia. Lippincott and Raven. *1998:139-42.*
5. Reich JD, Miller S, Brogdon B et al. The use of pulse oxymetry to detect congenital heart disease. *J Pediatric 2003;142:268-72.*
6. Papile LA. The Apgar score in the 21st century. *N Engl J Med 2001;344:519-20.*
7. Low JA. Intrapartum fetal asphyxia – definition, diagnosis, and classification. *Am J Ostet Gynecol 1997;100:1004-14.*
8. Steven RL, Uptala GD. Low Apgar scores and definition of birth asphyxia. *Pediatr Clin N Am 2004;737-45.*
9. Volpe JJ. *Neurology of newborn 9th edn*. Philadelphia: WB Saunders. *2001:331-94.*
10. Bloom R. Delivery room resuscitation of the newborn. In: Fanaroff AA, Martin RJ (eds). *Neonatal, Perinatal Medicine. 7th edn.* St. Louis : Mosby. *2002:416-39.*
11. Locus P, Yeoman SE, Croshy U. Efficacy of bulb versus Delee suction at delivery complicated by meconium stained amniotic fluid. *Am J Obstet Gynecol 1989;161:1106-10.*
12. Van BF, Shadid M, Moison RM et al. Effect of allopurinol on post-asphyxial free radical formation cerebral hemodynamic and electrical brain activity. *Pediatrics 1998;101:185-93.*
13. Battin MR, Dezoete JA, Gonn TR et al. Neurodevelopment out come of infants treated with head cooling and mild hypothermia after perinatal asphyxia. *Pediatrics 2001;107:480-4.*
14. Barbara LP, Anne M. The baby-friednly way: the best breast feeding start. *Pediatr Clin N Am 2004;761-83.*
15. William CH. The feeding of infants and children In: Behraman RE, Kliegman RM, Jenson HB (eds). *Nelson Textbook of Pediatrics. 17th edn*. Philadelphia. WB Saunders. 2003: 157-67.
16. Gibbs RS, Duff P. Progress in pathogenesis and management of clinical intarpartum infection. *Am J Obstet Gynecol 1991;164:1317-26.*
17. Sperling RS, Ramamurthy RS, Gibbs RS. A comparison of intrapartum vs immediate postpartum treatment of intraamniotic infection. *Obstet Gynecol 1987;70:861-5.*
18. Dacie J. *The Hereditary hemolytic anemias Part I.* Edinburg. Churchill Livingstone 1985.
19. Barbara JS, Robert MK. Blood Disorder In: Behrmen RE, Kliegman RM, Jenson HB (eds). *Nelson's textbook of pediatrics 17 th edn.* Philadelphina. WB Saunders.*2003;599-607.*
20. Sutor AH, von Kries R, Cornelissen EA et al. Vitamin K deficiency bleeding in infancy. Thromb Haemost 1999;81:456-61.