# **Risk Factors for Hypoxic-Ischemic Encephalopathy in** Asphyxiated Newborn Infants

Sitthivuddhi Futrakul MD, MSc\*, Pramote Praisuwanna MD\*, Pimolrat Thaitumyanon MD\*

\* Neonatal Unit, Department of Pediatrics, King Chulalongkorn Memorial Hospital, Chulalongkorn University

*Objective:* To determine the risk factors for hypoxic-ischemic encephalopathy (HIE) in asphyxiated newborn infants.

*Material and Method:* A retrospective study of 17,706 newborns, who were admitted to the Neonatal Unit of King Chulalongkorn Memorial Hospital, from July 1999 till the end of December 2000. 84 infants with perinatal asphyxia were enrolled in the present study. All of the possible risk factors that might have contributed to asphyxia were identified and recorded. HIE was diagnosed based on the Modified Sarnat-Sarnat Score for the diagnosis of neonatal encephalopathy. The clinical data of the HIE group were compared with those of the HIE negative group. The categorical data were analyzed for statistical significance (p < 0.05) by Chi-square test or Fisher exact test, or Student t-test. The odds ratio and 95% CI were calculated for those with statistical significance. Stepwise multiple logistic regression analysis used to determine the independent factors that may predispose an infant to HIE.

**Results:** Inappropriate antenatal care (OR 9.4; 95%CI: 2.6-35.4), post-term gestation (OR 7.4; 95%CI: 1.4-34.8), vacuum extraction (OR 5.4; 95%CI: 1.1-26.8), male (OR 4.8; 95%CI: 1.3-19.1), prolapsed cord (p = 0.01) and 1 and 5-minute Apgar scores, (p < 0.0001) were significant risk factors for HIE. However, by multiple regression analysis, only a 5-minute Apgar score was significantly associated with HIE (p = 0.001). **Conclusion:** Sophisticated or expensive equipment is not necessary for the treatment of HIE patient. HIE depends mainly on adequate and effective supportive strategy. The delivery of high risk pregnancies, under obstetric facilities and with appropriate intervention and with good neonatal resuscitation, may prevent the perinatal asphyxia and thereby minimize the occurring of HIE.

Keywords: Risk factors, Perinatal asphyxia, Hypoxic-ischemic encephalopathy

J Med Assoc Thai 2006; 89 (3): 322-8 Full text. e-Journal: http://www.medassocthai.org/journal

Perinatal asphyxia, one of the most devastating neurological processes is characterized by different degrees of hypoxia-ischemia during labor and delivery, with the outcome depending on the severity of the underlying neuronal damage. The reported incidence of hypoxic-ischemic encephalopathy (HIE) in full-term infants is 2.9-9 per 1000 deliveries<sup>(1,2)</sup>.

In spite of major advances with sophisticated monitoring technology and knowledge of fetal and neonatal pathology, perinatal asphyxia or more appropriately, HIE, remains a serious condition, causing significant mortality and long-term morbidity. The syndrome leaves significant handicaps in 100% of survivors of severe HIE and in 30% of survivors of moderate HIE<sup>(3)</sup>. Although the understanding of pathophysiology after perinatal asphyxia has been more extensively discussed, there has been little change in the outcome of acute perinatal asphyxia. One reason is that we do not know specifically the crucial risk factors that would assist to intervene both prenatally and postnatally to prevent HIE, which may follow intrapartum asphyxia and ultimately results in death or cerebral palsy. Nor do we know how to identify the neonates with asphyxia who are the greatest risk of developing encephalopathy<sup>(4)</sup>.

Correspondence to : Futrakul S, Department of Pediatrics, Faculty of Medicine, King Chulalongkorn Memorial Hospital, Chulalongkorn University, Rama IV Rd, Pathumwan, Bangkok 10330, Thailand. Phone: 0-2256-4708, 0-5211-0201, E-mail: sitvudfu1@yahoo.com

### **Material and Method**

A retrospective study consisting of 17,706 deliveries from July 1999 until the end of December 2000 was performed in the Neonatal Unit, Department of Pediatrics, King Chulalongkorn Memorial Hospital. 111 newborn infants who were delivered with low Apgar scores were diagnosed as perinatal asphyxia. The criterion for diagnosis of birth asphyxia in the present study was having an Apgar score of < 6 at 1 minute. Those newborn infants with stillbirth, gestational age < 36 weeks, congenital anomaly, and chromosomal abnormalities were excluded from the study. 84 newborn infants (47 males and 37 females) were enrolled in the present study. All of the possible factors that placed infants at high risk for asphyxia were identified and recorded by the neonatal resuscitation team. The clinical records were reviewed to collect data related to maternal, antenatal and neonatal course and outcome. HIE was diagnosed based on the Modified Sarnat-Sarnat Score for the diagnosis of neonatal encephalopathy. The possible factors of the HIE group were compared with those of the HIE negative group. The data were analyzed by SPSS programs and the Confidence Interval Analysis Program for statistical significance (p < 0.05). The number of patients with HIE was used to calculate the incidence. Mean and standard deviation of demographic data and possible factors were compared between the HIE group and the HIE negative group using Pearson Chi-square test or Fisher exact test and Student t-test where appropriate. p < 0.05 was considered to be statistically significant. The odds ratio and 95%CI were calculated for those with statistical significance. Comparison between groups was carried out using non-parametric Mann-Whitney U test. Stepwise multiple logistic regression analysis using the maximum likelihood method was used to determine the independent factors that may predispose an infant to HIE.

#### Results

Among 17,706 live birth infants, 111 newborn infants had Apgar scores < 6 at 1 minute. The incidence of perinatal asphyxia was 6.3/1000 live births. Among 84 eligible infants (47 males and 37 females), 22 newborn infants (26.2 %), 1.2/1000 live births, manifested clinical signs of HIE according to the Modified Sarnat-Sarnat Score for the diagnosis of neonatal encephalopathy as shown in demographic data of the study population. Mean gestational age was  $39.4 \pm 1.9$ weeks. Mean birth weight was  $2964.2 \pm 503.4$  grams, (Table 1).

Regarding the relationship of maternal factors between HIE and HIE negative group (Table 2), inappropriate antenatal care (ANC) was found more common in the HIE group (OR 9.4; 95% CI: 2.6-35.4).

In the comparison of intrapartum complication and delivery process between both groups (Table 3), vacuum extraction was significantly associated with HIE (OR 5.4; 95% CI: 1.1-26.8). Statistical significance of prolapsed cord was observed only in HIE group (p =0.01) however, OR and 95% CI could not be calculated.

Comparing the neonatal factors (Table 4), postterm was significantly observed to be more prevalent in the HIE group (OR 7.4; 95% CI: 1.4-34.8). Male gender was more commonly found (OR 4.8; 95% CI: 1.3-19.1). A greater magnitude of Apgar scores difference was

 Table 1. Demographic data of asphyxiated newborn infants

Clinical data	N = 84
Male (%) Female (%) Gestational age (wk) $(X \pm SD)$ Birth weight (gm) $(X \pm SD)$ HIE:NON-HIE	$48 (57.1)  36 (42.9)  39.4 \pm 1.9  2,964.2 \pm 503.4  22:62$

Factors	HIE N = 22	HIE (-) N = 62	p-value	OR	95%CI
Maternal age	$29.5 \pm 7.0$	$28.8 \pm 6.7$	0.6	-	-
Eldery mother	6	8	0.22	2.5	0.6-9.7
Teenage mother	1	6	0.40	2.2	0.2-4.2
Primigravida	8	46	0.03	0.2	0.6-6.1
Inappropriate ANC	12	7	< 0.001	9.4	2.6-35.4
Maternal disease	3	4	0.26	2.3	0.4-13.8
Complication during pregnancy	6	8	0.22	2.5	0.6-9.7

Table 2. Comparison of maternal factor between HIE group and HIE (-) group

Factors	HIE N = 22	HIE (-) N = 62	p-value	OR	95%CI
Fetal distress	8	15	0.41	1.8	0.5-5.7
Thick meconium stained amniotic fluid	9	26	0.86	0.9	0.3-0.86
Abnormal position	5	5	0.14	3.3	0.7-15.6
Preeclampsia	3	7	0.51	1.2	0.1-6.2
Prolonged 2 <sup>nd</sup> stage	3	5	0.34	1.8	0.6-9.9
Breech presentation	2	8	0.48	0.67	0.01-3.9
PROM > 12 hrs	2	3	0.39	1.96	0.02-16
Prolapse cord	3	0	0.01	-	-
Abruptio placenta	2	1	0.16	6.1	0.1-249
Twins	2	2	0.27	3.0	0.05-178
Chorioamnionitis	1	1	0.45	2.9	0.01-7.6
Oligohydramnios	0	3	0.78	0.9	0.7-11.1
Placenta previa	3	7	0.51	1.2	0.1-6.2
Medication during labour:					
oxytocin	7	11	0.28	2.2	0.6-7.5
narcotics	7	24	0.75	0.7	0.2-23.1
Type of anesthesia					
epidural block	1	0	0.26	-	-
spinal block	5	28	0.11	0.3	0.1-1.2
General anesthesia	6	10	0.4	1.9	0.5-7.1
Mode of deliveries:					
Normal labour	3	12	0.40	0.6	0.3-2.9
Forceps extraction	5	9	0.57	1.7	0.4-6.8
Vacuum extraction	6	4	0.02*	5.4	1.1-26.8*
Cesarean section	8	34	0.17	0.4	0.1-1.3
Breech assisting	0	3	0.54	-	-

Table 3. Comparison of intrapartum complication, delivery process, type of anesthesia and mode of delivery between HIE group and HIE (-) group

\* p < 0.05

	Table 4. Comparison of neonatal factor between HIE group and HIE (-) group	
--	--	--

Factors	HIE N = 22	HIE (-) N = 62	p-value	OR	95%CI	
Gender M:F	18:4	30:32	0.005*	4.8	1.3-19.1*	
Gestational age	$39.6 \pm 2.1$	$39.2 \pm 1.8$	0.83	-	-	
Pre-term	2	6	0.65	0.9	0.6-5.8	
Post-term	6	3	0.008*	7.4	1.4-34.8*	
Birth weight	$2700 \pm 637.5$	2714 <u>+</u> 770.9	0.43	-	-	
LGA	1	3	0.72	0.9	0.7-11.1	
SGA	3	2	0.10	4.7	0.6-54.4	
Apgar scores						
1 min	$2.2 \pm 1.2$	3.9 <u>+</u> 1.2	< 0.0001*	-	-	
5 min	$5.2 \pm 2.2$	7.5 <u>+</u> 1.5	< 0.0001*	-	-	

\* p < 0.05

noted in the HIE group. A significant difference of 1minute Apgar score in the HIE and HIE (-) group was observed  $2.2 \pm 1.2$  and  $3.9 \pm 1.2$  respectively; p < 0.0001. Likewise the 5-minute Apgar score was expressed at  $5.2 \pm 2.2$  and  $7.5 \pm 1.5$  respectively; p < 0.0001. However, HIE was significantly correlated with only a 5-minute Apgar score (p < 0.001).

For live birth infants at  $\geq$  36 weeks of gestation, the neonatal mortality rate of both HIE and perinatal asphyxia was 750 per 1000 for infants with 5-minute Apgar score of 0 to 3. The risk of neonatal death in the HIE group with 5-minute Apgar score of 0 to 3 was significantly observed (OR 51; 95% CI: 1.7-1058), compared with 5-minute Apgar score of more than 3. Likewise, the perinatal asphyxia group also found statistical significance of the risk of neonatal death with 5-minute Apgar score of 0 to 3 (OR 117; 95 %CI 6.0-786), (Table 5).

### Discussion

In the present study, the authors used an Apgar score of less than 6 to define asphyxia in infants with 36 weeks gestation or greater. The incidence rate of birth asphyxia was 6.3/1000 live born infants. Apparently because of varying definitions the true incidence of asphyxia is unknown. The incidence of birth asphyxia depends largely on the definition used to diagnose the condition as well as the gestational age of the infant<sup>(5-8)</sup>. The incidence of birth asphyxia ranges from 0.3/1000 in a Swedish study <sup>(6)</sup> to 36.6/1000 live birth infants in an Indian study<sup>(8)</sup>.

Although the Apgar score has been criticized because it does not accurately identify or predict subsequent neurodevelopmental outcome of newborn infants, and many considered it to be an obsolete measure of the condition of neonates, few would deny that its application at one and five minutes of age accomplishes Dr. Apgar's goal of focusing attention on the condition of the infant immediately after birth<sup>(9-11)</sup>. Apgar score is still the most feasible and practical to perform in the delivery system<sup>(12)</sup>. Therefore, until a more useful index for assessing neonates is developed, the 5-minute Apgar score, is still a valid and a rapid index for assessing the effectiveness of resuscitative efforts and the vitality of the infant<sup>(13)</sup>.

Perinatal hypoxic-ischemic encephalopathy (HIE) occurs as a result of hypoxic and/or ischemic insults during antepartum and intrapartum periods. Induced by an interruption in placental blood flow and gas exchange perinatal HIE extends into a recovery period after resuscitiation. The present study found the incidence of HIE was 1.2/1000 live births. The syndrome leaves significant handicaps in 100% of survivors of severe HIE and 30% of survivors of moderate HIE. The present study also assessed the influence of possible perinatal risk factors in the etiology of birth asphyxia and the relative importance of these factors in determining the risk of asphyxiated infants to develop HIE.

Associated factors of HIE include maternal factors, intrapartum complication, delivery process and neonatal factors. The present study found that the improper antenatal care (ANC) was a significant factor associated with HIE. The relatively high figure could be explained by the fact that 22.6 % of asphyxiated infants' mothers did not have regular antenatal care; and many of them attended the hospital late with signs of fetal distress already present. This result is similar to that obtained in the Jordan study<sup>(14)</sup>. Regarding primigravida, there was no significant association

**Table 5.** Demonstrate the risk of neonatal death with 1-minute and 5-minute Apgar score of 0 to 3 and Apgarscore more than 3 in HIE group and perinatal asphyxia group

HIE N = 22	Apgar 0-3	Apgar >3	p-value	OR	95%CI	mortality rate
1-minute Apgar mortality	4/17	0/5	0.32	-	- 1.7-1058*	235/1000
5-minute Apgar mortality	3/4	1/18	0.009*	51		750/1000
Perinatal asphyxia N = 84	Apgar 0-3	Apgar >3	p-value	OR	95%CI	mortality rate
1-minute Apgar mortality	4/33	1/51	0.07	6.9	0.7-93.8	121/1000
5-minute Apgar mortality	3/4	2/80	0.0004*	117	6.0-786*	750/1000

\* p < 0.05

between HIE and primigravida which was different from the Macdonald study,<sup>(15)</sup> that shows primigravidity carried a high risk for an asphyxiated infant.

With respect to the intrapartum complication, maternal preeclmapsia with HELLP syndrome and umbilical cord prolapse have been shown to be a risk for asphyxia<sup>(16)</sup>. The present study revealed that prolapsed cord and abruption placenta was frequently observed in the HIE group but did not reach the statistic significance due to the small number of cases. A significantly higher proportions of HIE group than HIE (-) group were delivered by vacuum extraction. This delivery method might play an important role to prolong asphyxia or increased hypoxic-ischemic injury. In the present study, no effect of anesthetic type was significantly demonstrated.

In the neonatal factors, there was a statistically significant relationship between HIE and male gender. This concurs with other studies<sup>(17-19)</sup> which mentioned that the male gender is highly vulnerable to any threatening factors such as increasing the risk of sepsis, bronchial hyperesponsiveness, atopy, and mortality of RDS etc. In addition, the present study revealed gestational age particularly post-term gestation significantly associated with HIE; this might be related to the uteroplacental insufficiency. There were no significant relationship between HIE and birth weight. The small for gestational age (SGA) infants showed a slightly increased risk (OR 4.7) but this difference was not statistically significant. However, Aired<sup>(20)</sup> reported that infants with intrauterine growth retardation play a significant role in occurrence of severe asphyxia. The small sample size in the present study contributed to the observed negative correlation of SGA and increasing risk of HIE.

Recent studies have been focused on the usefulness of the Apgar score for the immediate assessment of neonates. It has been suggested the Apgar score is antiquated and that its predictive value has been considerably weakened as a sensitivity marker for asphyxia<sup>(21,22)</sup>. Furthermore, the value of the Apgar score for the evaluation of very premature infants has been questioned because assigning of the Apgar score may be affected by the maturity of the preterm infants. Therefore, the observation by Nelson<sup>(23)</sup> showed the majority of children with cerebral palsy did not have a low Apgar score.

However, the present study observed that the 1 and 5-minute Apgar scores were significantly lower in HIE infants than asphyxiated infants. Furthermore, a 5-minute Apgar score of 3 or less in infants of more than 35 weeks had a high risk of neonatal death in both group. The present study supports the previous report by Casey<sup>(13)</sup>, that a 5-minute Apgar score of 3 or less was indicative of a severely compromised infant who was unable to respond to resuscitative efforts and prone to the risk of neonatal death. The risk in this infant was 8 times greater than of term infants with umbilical-artery pH values of 7.0 or less<sup>(11)</sup>.

In summary, despite advances in perinatal care over the past three decades, the incidence of HIE attributed to severe birth asphyxia has not changed much. The present study revealed that various risk factors might potentially affect HIE in asphyxiated newborn infants. All of the significant risk factors include inappropriate ANC, vacuum extraction, prolapsed cord, post-term gestation, and male gender as well as 1 and 5-minute Apgar scores. One might anticipate that a 5-minute Apgar score of 3 or less is highly indicative of severe perinatal asphyxia and a strongly predictive risk for HIE and the highest risk of neonatal death. Obstetricians and Pediatricians should be highly concerned for those high-risk conditions and other possible factors in order to minimize birth asphyxia.

#### References

- Brown JK, Purvis RJ, Forfar JO, Cockburn F. Neurological aspects of perinatal asphyxia. Dev Med Child Neurol 1974; 16: 567-80.
- Finer NN, Robertson CM, Richards RT, Pinnell LE, Peters KL. Hypoxic-ischemic encephalopathy in term neonates: perinatal factors and outcome. J Pediatr 1981; 98: 112-7.
- Volpe JJ. Hypoxic-ischemic encephalopathy: clinical aspects. In: Volpe JJ, editor. Neurology of the newborn. 3<sup>rd</sup> ed. Philadelphia: WB Saunders; 1995: 314-69.
- 4. Perlman JM. Markers of asphyxia and neonatal brain injury. N Engl J Med 1999; 341: 364-5.
- Day P, Lancaster P, Huang J. Perinatal statisic series. No. 6. Australia's mothers and babies 1995. Sydney: National Perinatal Statistics Unit (NPSU). Australian Institute of Health and Welfare; 1997.
- Thorberg E, Thiringer K, Odeback A, Milsom I. Birth asphyxia: incidence, clinical course and outcome in a Swedish population. Acta Paediatr 1995; 84:927-32.
- Hall DR, Smith M, Smith J. Maternal factors contributing to asphyxia neonatorum. J Trop Pediatr 1996; 42: 192-5.
- 8. Chandra S, Ramji S, Thirupuram S. Perinatal

asphyxia: multivariate analysis of risk factors in hospital births. Indian Pediatr 1997; 34: 206-12.

- Manganaro R, Mami C, Gemelli M. The validity of the Apgar scores in the assessment of asphyxia at birth. Eur J Obstet Gynecol Reprod Biol 1994; 54: 99-102.
- American Academy of Pediatrics. Use and abuse of the Apgar score. Committee on Fetus and Newborn, American Academy of Pediatrics, and Committee on Obstetric Practice, American College of Obstetricians and Gynecologists.Pediatrics 1996; 98: 141-2.
- 11. Papile LA. The Apgar score in the 21st century. N Engl J Med 2001; 344: 519-20.
- Kolatat T, Vanprapar N, Thitadilok W. Perinatal asphyxia: multivariate analysis of risk factors. J Med Assoc Thai 2000; 83: 1039-44.
- Casey BM, McIntire DD, Leveno KJ. The continuing value of the Apgar score for the assessment of newborn infants. N Engl J Med 2001; 344; 467-71.
- Khreisat WH, Habahbeh Z. Risk factors of birth asphyxia: a study at Prince Ali Ben Al-Hussein Hospital, Jordan. Pak J Med Sci 2005; 21: 30-4.
- MacDonald HM, Mulligan JC, Allen AC, Taylor PM. Neonatal asphyxia. I. Relationship of obstetric and neonatal complications to neonatal mortality in 38405 consecutive deliveries. J Pediatr 1980; 96: 898-902.

- Prabulos AM, Philipson EH. Umbilical cord prolapse. Is the time from diagnosis to delivery critical? J Reprod Med 1998; 43: 129-32.
- 17. Hubacek JA, Stuber F, Frohlich D, Book M, Wategrove S, Ritter M, et al. Gene variants of the bactericidal/permeability increasing protein and lipopolysaccharide binding protein in sepsis patients: gender-specific genetic predisposition to sepsis. Crit Care Med 2001; 29: 557-61.
- 18. Futrakul S, Deerojanawong J, Prapphal N. Risk factors of bronchial hyperresponsiveness in children with wheezing associated respiratory infection. Pediatr Pulmonolo 2005; 40: 81-7.
- Harms K, Herting E, Kron M, Schill M, Schiffmann H. Importance of pre-and perinatal risk factors in respiratory distress syndrome of premature infants. A logical regression analysis of 1100 cases. Z Geburtshilfe Neonatol 1997; 201: 258-62.
- 20. Airede AI. Birth asphyxia and hypoxic-ischemic encephalopathy: incidence and severity. Ann Trop Paediatr J 1991; 11: 331-5.
- 21. Is the Apgar score outmoded? [editorial]. Lancet 1989; 1: 591-2.
- Marrin M, Paes BA. Birth asphyxia: does the Apgar score have diagnostic value? Obstet Gynecol 1988; 72: 120-3.
- Nelson KB, Ellenberg JH. Apgar scores as predictors of chronic neurologic disability. Pediatrics 1981;68: 36-44.

## ปัจจัยเสี่ยงของความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดในทารกที่มีภาวะ ขาดออกซิเจนระยะปริกำเนิด

## สิทธิวุฒิ ฟูตระกูล, ปราโมทย์ ไพรสุวรรณา, พิมลรัตน์ ไทยธรรมยานนท์

**วัตถุประสงค**์: เพื่อศึกษาปัจจัยเสี่ยงของความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดในทารกที่มี ภาวะขาดออกซิเจนระยะปริกำเนิด

**วัสดุและวิธีการ**: การศึกษาวิจัยเชิงย้อนหลังในทารกทั้งหมด 17,706 คน คลอดที่โรงพยาบาลจุฬาลงกรณ์ในช่วง ระหว่างวันที่ 1 กรกฎาคม พ.ศ. 2542 ถึง 31 ธันวาคม พ.ศ. 2543 พบว่ามีทารกจำนวน 84 คนที่มีภาวะขาดออกซิเจน ระยะปริกำเนิด การวินิจฉัยความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดใช้เกณฑ์ของ Modified Sarnat-Sarnat Score ปัจจัยเสี่ยงที่ตรวจพบในมารดาและทารกจะถูกบันทึกและนำมาวิเคราะห์โดยเปรียบเทียบ ระหว่างกลุ่มที่มีความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดกับกลุ่มที่ไม่มีอาการ และนำมา วิเคราะห์ทางสถิติ โดยวิธี univariate และ stepwise multiple logistic regression

**ผลการศึกษา**: การฝากครรภ์ไม่เหมาะสม (OR 9.4; 95%CI: 2.6-35.4), อายุครรภ์เกินกำหนด (OR 7.4; 95%CI: 1.4-34.8), การคลอดโดยเครื่องดูดสุญญากาศ (OR 5.4; 95%CI: 1.1-26.8), เพศชาย (OR 4.8; 95%CI: 1.3-19.1), สายสะดือย<sup>•</sup>อย (p = 0.01) และคะแนนแอปการ์ที่ 1 นาทีและ 5 นาที (p < 0.0001) เป็นปัจจัยเสี่ยงของความผิดปกติ ของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดในทารกที่มีภาวะขาดออกซิเจนระยะปริกำเนิด อย<sup>'</sup>างไรก็ตาม การวิเคราะห์โดยวิธี stepwise multiple logistic regression พบว<sup>'</sup>าคะแนนแอปการ์ที่ 5 นาทีเท่านั้น ที่สัมพันธ์กับ ความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจนและขาดเลือดมีน้องมีน้องมี (p = 0.001)

**สรุป**: การใช้เครื่องมือที่มีราคาแพงหรือล้ำสมัยไม่ได้เป็นสิ่งจำเป็นสำหรับก<sup>-</sup>รดูแลรักษาทารกที่มีความผิดปกติของ สมองเนื่องจากสมองขาดออกซิเจนและขาดเลือด แต่การเฝ้าระวังในรายที่มารดามีภาวะตั้งครรภ์เสี่ยง การหลีกเลี่ยง ปัจจัยเสี่ยง การรักษาที่เหมาะสมและทันท่วงที และทีมกูชีพทารกที่มีประสิทธิภาพในการช่วยเหลือทารกที่ประสบภาวะ ขาดออกซิเจนระยะปริกำเนิด จะช่วยลดอุบัติการณ์ความผิดปกติของสมองเนื่องจากสมองขาดออกซิเจน และขาด เลือดได้