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IMMEDIATE OUTCOME OF BABIES WITH LOW APGAR SCORE IN MULAGO HOSPITAL, UGANDA

C. Ondoa-Onama, MBChB, MMed and J.K. Tumwine, MBChB, MMed, FRSH, Department of Paediatrics and Child Health, Makerere University Medical School, P.O. Box 7072, Kampala, Uganda

Request for reprints to: Prof. J.K. Tumwine, Department of Paediatrics and Child Health, Makerere University Medical School, P.O. Box 7072, Kampala, Uganda

# IMMEDIATE OUTCOME OF BABIES WITH LOW APGAR SCORE IN MULAGO HOSPITAL, UGANDA

## C. ONDOA-ONAMA and J.K. TUMWINE

# ABSTRACT

*Background:* Birth asphyxia contributes significantly to perinatal morbidity and mortality especially in resource poor countries. Although the Apgar score has been in use for over 50 years, the prevalence of low Apgar score and attendant risk factors and outcome have not been established in many sub-Saharan countries including Uganda.

*Objective:* To determine the prevalence of low Apgar score and establish immediate outcome and possible risk factors for poor outcome in babies with low Apgar score. *Setting:* Labour wards, operating theatres and special baby care unit, Mulago Teaching and referral Hospital, Uganda.

*Subjects:* Babies delivered in Mulago Hospital between September and October 1999. Those with low Apgar scores, together with an equal number of babies with normal scores matched for sex as controls, were followed up for 48 hours.

*Measurements:* Clinical features, anthropometry, gestational age, oxygen saturation, blood glucose and autopsy of babies who died.

*Main outcome measures:* Clinical improvement, death, complications such as HIE, RDS, aspiration pneumonia, hypoglycaemia, hypothermia, hypotension and hypoxaemia.

*Results:* The prevalence of low Apgar score at one and five minutes was 8.4% and 2.8% respectively. Adverse outcome was seen in 57.3% of cases: death in 12.1% and clinical complications in 45.2%. HIE occurred in 21.8%, hypoxaemia in 12.9%, hypoglycaemia in 16.9% and aspiration pneumonia in 4.8%. Maternal factors significantly associated with low Apgar scores included primiparity, abnormal delivery, age and medical diseases during pregnancy, while birth injuries and cord accidents were the baby factors. Poor outcome was associated with birth injury, hypothermia, hypoglycaemia, hypotension, aspiration pneumonia, hypoxaemia and severe birth asphyxia.

*Conclusion:* Even though the prevalence of low Apgar was only 8.4%, adverse outcomes associated with it were observed in more than half the patients. Therefore there is need to carefully evaluate and monitor babies with low Apgar scores immediately after birth.

## INTRODUCTION

Of the 120 million babies born in the resource poor countries each year, 3.6 million develop birth asphyxia and require resuscitation(1). It is estimated that 25% or 900 000 of the asphyxiated newborns die as a result of the asphyxia. The Apgar score, first introduced by Virginia Apgar(2) in 1952, is a practical method of systematically assessing newborn infants immediately after birth to help identify those requiring resuscitation(3). Perinatal morbidity and mortality can be reduced if high risk infants can be identified and adequately resuscitated. Several authors have established the association between very low Apgar scores and increased perinatal morbidity and mortality(4-5).

The value of Apgar scores in the prediction of future neurological outcomes, however remains in doubt(6) and has not been considered in the current study. Despite the available information, there is only a handful of published studies(4,7) from the developing countries and none from Uganda, to shed light on the magnitude of the problem of low Agar scores. Moreover, the immediate morbidity and mortality of babies with low Apgar scores in many of these countries has not been established. The current study was therefore designed to determine the prevalence and determinants of low Apgar scores, and the immediate outcome (within the first 48 hours) of babies with low Apgar scores in Mulago Hospital. It also sought to establish the risk factors for immediate adverse outcome in these babies.

## MATERIALS AND METHODS

*Study site:* This study was carried out in Mulago Hospital, Uganda's National Referral and Teaching Hospital Kampala, from September to October 1999.

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The patients were recruited from the two labour wards. Mothers without complications are first seen in the Upper Mulago Maternity Centre while those who develop complications are transferred immediately to lower Mulago for further management. The Special Care Baby Unit is adjacent to the Lower Mulago labour ward and admits only patients who develop complications within the first 24 hours of birth. On average the hospital handles about 20 000 deliveries per year of whom 19 000 or so are live births.

*Patients/study population:* The study population included babies born in Mulago Hospital during September and October 1999.

*Inclusion and exclusion criteria:* Babies delivered in Mulago Hospital during the study period with Apgar score of 6 and below and whose mothers consented to participate in the study were recruited as cases, while babies with Apgar scores 7-10 were the controls.

Babies with gross congenital malformations, still births or whose mothers died soon after delivery were excluded.

*Ethical issues*: Permission to carry out the study was obtained from the departments of Paediatrics and Child Health, Obstetrics and Gynaecology, the Makerere faculty of Medicine research committee, Mulago Hospital Ethics committee and the Uganda National Council for Science and Technology. Informed consent was obtained from the mothers after explaining to them the objectives of the study, and all information collected on each individual was held in confidence. Babies with low Apgar scores were resuscitated and treated accordingly, while mothers of babies who died or had severe morbidity were counselled.

Sample size consideration: In order to estimate the prevalence of low Apgar score a minimum sample size of 1452 babies was calculated using a formula developed by Kish(8). This was based on an estimated prevalence of low Apgar score of 6% in Mulago Hospital in April 1999, allowing for a 1% error and 95% confidence interval. In the investigation of the possible risk factors for low Apgar score the formula for case control studies was used to estimate the minimum sample size for cases and controls.

The calculation used the proportion of the exposure (primiparity) among mothers of babies with low Apgar scores as a risk factor. This proportion was taken as 0.6 from results of a similar study by Nathoo, *et al* (7). With a power of 80%, odds ratio worth detecting at 2.05 and 95% confidence interval, a sample size of 124 cases and 124 controls was calculated using the formula by Fleiss(9).

To estimate the sample size for the follow up and outcome of babies with low Apgar score, the method for calculation of sample size for cohort studies was used(9).

With an 80% power, 95% confidence intervals and relative risk worth detecting at 5.4, the sample size of the exposed population (babies with low Apgar score) and of the controls (babies with Apgar scores 7-10) was 122. This was based on death as the outcome measure. Death was estimated to occur in 10.8% of the babies with low Apgar score and in only 2% of the controls based on two previous cohort studies(3,10).

Sampling procedure: A systematic sampling procedure was used in which the respective labour wards and operating

theatres formed the sampling frame. Babies were systematically sampled from these places according to the average number of deliveries per day. Hence 420 were recruited from Upper Mulago, 827 from Lower Mulago, and 205 from the two theatres.

Data collection and clinical assessment: Data was collected on socio-demographic characteristics of the mothers and on their obstetric and gynaecological history. Measurements of the babies included physical examination, anthropometry, pulse oxymetry, blood glucose levels and autopsy examination on those who died.

Apgar scoring was carried out by trained research assistants who were qualified midwives but who were not routinely employed to carry out the deliveries. Instead the deliveries were carried out by the attending midwives on duty and who were not part of the study team. The babies were assessed as soon as they were born at one, five and twenty minutes using the Apgar scoring method which consisted of the five physical signs of heart rate, respiratory effort, reflex irritability, muscle tone and colour(2,3,11). Babies needing resuscitation received it.

*Clinical examination:* Examination of the babies was done by one of us (C O-O) within six hours of delivery and at 24 and 48 hours of age. The evaluation included the general examination (such as temperature, assessment for pallour, cyanosis) and anthropometry. In the respiratory system, signs of respiratory distress and changes in the pattern of breathing were sought. Presence or absence of rales, reduced or increased breath sounds, irregular or no spontaneous breathing were noted. Signs and stages of hypoxic ischaemic encephalopathy (HIE) as proposed by Sarnat and Sarnat(12) were assessed. Gestational age assessment using Ballard's method(13) was done 24 hours after birth.

*Measurements: Weight:* These were measured using a Beam scale manufactured by CMS weighing equipment limited, London, UK. Babies were weighed nude to the nearest 0.1 gm. The scale was checked with a standard weight at the beginning of each day and adjusted accordingly

*Length:* This were measured using a stadiometer constructed locally according to a model provided by Appropriate Technology Action Group, London. Supine measurements were performed and recorded to the nearest 0.1 centimeter.

*Temperature:* All temperatures were obtained by the rectal route using a YSI tele - thermometer manufactured by Yellow Springs Instrument Co., Inc. Ohio, USA in conjunction with Simpson Electric., Elgin II, 60120, USA. The instrument was calibrated before taking measurements on each baby.

*Pulse oxymetry:* This was performed at first examination, and at 24 and 48 hours using the Ohmeda Biox 3700 pulse oxymeter (Ohmeda, Boulder Co., 803011, a division of BOC Group Inc; BOC Health care, USA). Oxygen saturation below 92% was considered hypoxaemia. Blood glucose estimation was done using a Surestep Brand glucose meter (Lifescan, Johnson and Johnson Company USA) and SurestepPro test strips, which employ a dry agent technology, based on the glucose oxidase method. The heel

was cleaned with antiseptic solution and dried before obtaining the sample by puncturing the site with a sterile lancet. Blood glucose was estimated on first examination, at 24 and 48 hours after birth.

Autopsy studies. This was done following the Lettulle method as recommended by Ludwig(14).

*Management and follow up of cases:* All babies with low Apgar scores were resuscitated and admitted to the Special Care Unit for further management. All babies (cases and controls) were examined on the first day and then followed up at 24 and 48 hours after birth. Daily monitoring included general and systemic examination, pulse oxymetry, and blood glucose estimation.

*Main outcome measures:* The main outcome measures were clinical improvement, complications or death. Clinical improvement included normal temperature, cardiovascular and respiratory systems and no signs of encephalopathy. Clinical complications were defined using the following: respiratory distress, aspiration pneumonia, apnoea, periodic or disorganised breathing.

In the cardiovascular system the presence of shock or hypotension (diastolic blood pressure < 40mm Hg), hypertension (diastolic blood pressure > 60mm Hg), tachycardia (heart rate > 160/minute), and bradycardia (heart rate < 100/minute) were considered. Neurologic complications included signs of hypoxic ischaemic encephalopathy. These range from mild (hyper alertness/ irritability, slight feeding difficulties, minor disturbance of muscle tone, presence of primitive reflexes such as Moro, sucking and tonic neck) mydriasis, tachycardia, slight bronchial and salivary secretions and recovery by 48 hours) to moderate and severe.

Moderate neurological signs included lethargy, poor feeding, hypotonia, seizures, spontaneous respiration, occasional apnoea, bradycardia, profuse bronchial and salivary secretions increased diarrhoea, miosis, suppressed primitive reflexes and no recovery by 48 hours.

Severe signs included stupor/coma, flaccidity, absent primitive reflexes, seizures, failure to maintain adequate ventilation, often unequal, midposition pupils with poor light reflex.

Data management and statistical analysis: Data was analysed using EPIINFO and BMDP software. Data was summarised using frequency tables, means and standard deviations. Possible risk factors for low Apgar score and poor outcome were analysed as follows: for categorical risk factors, contingency tables were used and the strength of association was measured using the Chi-squared test statistic and its associated p-value. For continuous risk factors the students t-test was used to compare the mean value of the risk factors in the study and control babies. To assess the interactions and joint effects of the risk factors, logistic regression analysis was used. The risk factors considered included maternal disease during pregnancy, booking status, maternal age, parity, obstetric history, duration of labour, mode of delivery, cord accidents, birth injuries, birth weight and gestational age.

*Quality control:* The questionnaire was pre-tested before the actual data correction and the research midwives were

trained to fill the questionnaire and carried out the Apgar scoring of the babies accordingly.

## RESULTS

From lst September to 31st October 1999, a total of 1479 newborn babies were studied and of these 769 (52%) were males and 710 (48%) were females. The majority (92.2%) had normal birth weight with only 7.8% having low birth weight (<2.5kg). The mean birth weight was 3.08 kg (SD 0.48).

*Prevalence of low Apgar score:* Of the 1479 babies studied, 124 (8.5%) had low Apgar score (6) at one minute. Only 14 or 2.8% of the 1478 babies had low Apgar score at 5 minutes. By 5 minutes one child had died of severe asphyxia (Apgar score 3). A smaller percentage (1%) of babies had low Apgar score at 20 minutes. The prevalence of severe birth asphyxia (Apgar score 3) at one minute was 1.6% while at 5 and 20 minutes was 0.5% respectively.

*Risk factors for low Apgar score*: The babies with low Apgar score and controls matched for sex had similar characteristics (Table 1). The gestational ages and birth weight of the cases and controls were not statistically different.

### Table 1

Birth weight and gestational age of babies with low Apgar scores and their matched controls

	Cases	Control	p-value
Sex			
Male	76	76	-
Female	48	48	_
Mean Weight (kg).(SD)	2.93 (0.62)	3.06(0.55)	0.081
*Gestational age in			
weeks mean (SD)	39.6(1.92)	39.3(2.40)	0.278
<37 weeks	17	9	0.973
37 weeks	107	115	_
SGA	24	20	_
AGA	85	93	0.1138
LGA	5	11	_

\*10 babies died before they had their gestational age assessed

The background socio - demographic characteristics of the mothers of the study babies and their controls were not statistically different (Table 2).

Maternal risk factors for low Apgar score included primiparity and assisted delivery. Booking status, age of the mother, antepartum haemorrhage, early rupture of membranes and duration of labour were not significant risk factors for low Apgar score (Table 3).

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# Table 2

Socio-demographic characteristics of mothers of babies with low Apgar scores and their controls

Mothers	Cases (%)	Controls (%)	P-value
Mean age ± SD	21.9 ± 5.6	$23.1 \pm 4.8$	0.068
Age range in years	15-37	15-39	
Marital status			
Married	103 (83.7)	103 (83.1)	
Single	14 (14.6)	20 (16.1)	0.777
Widow	1 (0.8)	1 (0.8)	
Formal education status			
Nil	5 (4)	6 (6.4)	
Primary	70 (56.5)	67 (54.0)	
Secondary	42 (33.9)	45 (36.3)	
Post secondary	7 (5.6)	6 (4.8)	0.953
Occupation			
Housewife	72 (66.1)	65 (60.2)	
Business	15 (13.8)	18 (16.7)	
Professional	12 (11.0)	16 (14.8)	
Peasant	2 (1.8)	6 (5.6)	
Student	3 (2.8)	1 (0.9)	
Other	5 (4.6)	2 (1.9)	
Parity			
1	68 (54.8)	47 (37.9)	
2	22 (17.7)	38 (30.6)	
3	14 (11.3)	10 (8.1)	0.142
4	6 (4.8)	12 (9.7)	
5+	14 (11.3)	17 (13.7)	

## Table 3

	Maternal risk factors for low Apgar scores				
Risk factor	Cases	Controls	Odds ratio	95% CI	P-value
Parity					
Primiparity	68	47	1.99	1.16-3.43	0.008*
Mode of delivery					
Spontaneous vaginal	63	79	1.70	0.99-2.93	0.039*
Booking status					
Not Booked	11	8	0.70	0.24-1.99	0.463
Booked	112	116			
Antepartum haemorrhage					
Yes	6	7	0.86	0.24-2.98	0.787
No	117	117			
Early rupture of membranes					
Yes	38	36	1.08	0.6-1.95	0.780
No	84	86			
Mean duration of	52(12.4)	30.2 (37.4)	-	-	0.21
second stage in minutes (SD)					
Mean duration of labour	12.71 (6.06)	10.75 (5.17)	-	-	0.056
in hours (SD)					
High blood pressure					
Yes	155	10	1.57	0.63-3.98	0.292
No	109	114			
Poor obstetric history					
Yes	17	19	0,88	0.41-1.90	0.719
No	107	105			
Other medical disease					
Yes	72	61	1.34	0.78-2.32	0.255
No	51	58			

\* p-value significant Cl = Confidence Interval

However logistic regression analysis, which was done using the BMDP stepwise regression programme, found low Apgar score to be significantly associated with birth injury, cord accidents, mode of delivery, maternal age and a history of medical diseases during pregnancy (Table 4).

#### Table 4

Coefficients and p-values of risk factors for low Apgar score in the logistic regression analysis

Variable	Coefficient	Odds ratio	95% CI	P- value
Birth injury	1.44	4.23	1.41-12.6	0.012
Cord accident	1.46	4.31	1.02-18.2	0.049
Maternal age	-0.06	0.94	0.89-1.00	0.034
Medical disease				
in pregnancy	0.62	1.86	0.98-3.56	0.05
Abnormal delivery*	-0.050	0.61	0.33-1.10	0.033
Constant	1.41	4.10	0.90-18.8	0.02

CI = Confidence interval

\* Delivery other than by spontaneous vertex delivery

After controlling for birth injury, cord accident, medical diseases and mode of delivery, maternal age was found to be a significant risk factor for low Apgar score (p=0.0344).

There was no significant difference between the mean birth weight of the babies with low Apgar scores  $(2.93 \pm 0.62 \text{ kg})$  and the controls  $(3.06 \pm 0.55 \text{ kg})$ . Similarly the gestational age of the cases  $(39.6\pm1.9 \text{ weeks})$  was not statistically different from that of the controls  $(39.3\pm2.4 \text{ weeks})$ .

Immediate (48 hours) outcome of babies with low Apgar score: Mortality and morbidity were the factors considered as immediate outcome in the babies. Of the 124 babies with low Apgar score, 15 (12.1%) died, while no death occurred among the control group. Of the 109 survivors, 56 (45.2%) had adverse clinical conditions such as hypoxic ischaemic encephalopathy, aspiration pneumonia, hypoxaemia, hypoglycaemia and others (Table 5).

Within the first 48 hours, 15 (12.1%) of the babies with low Apgar score had died The clinical conditions in these babies ranged from hypoxaemic ischaemic encephalopathy, hypothermia to the respiratory distress syndrome (Table 6).

		hours of age			
Clinical condition	Cases (n=109)	Control (n=124)	Relative Risk(RR)	95% CI	P-value
Persistent tachypnoea	39	26	1.44	1.1-1.9	0.012
Changes in breathing patterns*	35	11	1.92	1.5-2.4	< 0.001
Hypoxaemia	16	1	2.19	1.8-2.7	< 0.001
Hypoglycaemia	21	12	1.45	1.1-2.0	< 0.037
Hypoxic	27	2	2.32	1.9-2.8	< 0.001
Ishaemic encephalopathy Aspiration pneumonia	7	0	-	-	-

Table 5

Clinical conditions of the babies with low Apgar score and the controls, at 48

Cl =Confidence Interval

\* Changes in breathing patterns include apnoea, periodic, and disorganised breathing

#### Table 6

Clinical conditions of the 15 babies who died with low Apgar scores

Clinical condition	No.(%)
Hypoxic ishaemic encephalopathy	14 (93.3)
Hypothermia	14 (93.3)
Hypoxaemia	12 (80.0)
Hypotension	10 (66.7)
Respiratory distress syndrome	9 (60.0)
Change in breathing pattern (apnoea,	
periodic breathing)	8 (53.3)
Aspiration pneumonia	5 (33.3)
Hypoglycaemia	4 (26.6)
Bradycardia	4 (26.6)

Post mortem examination was done on 9 out of the 15 who died

Two babies had no anatomical or histological changes; two had hyaline membrane disease, while the rest had amniotic fluid embolism, meconium aspiration in one, subdural haemorrhage in one, toxic injury in one and haemosiderin in the lungs in one.

Factors associated with poor outcome of the babies with low Apgar score: Unfavourable outcome was considered to be clinical complication or death, both of which occurred in babies with low Apgar scores. Factors associated with dying in the first 48 hours after birth are shown in Table 7.

## Table 7

Factor	Died	Lived	Odds ratio	95% CI	P-value
Birth injury*					
Yes	7	12	6.56	1.73-25.3	0.001
No	8	90			
Sex					
Female	10	38	3.74	1.06-13.84	0.018
Male	5	71			
Hypothermia*					
Yes	14	72	7.00	0.98-304.1	0.037
No	1	36			
Hypoglycaemia					
Yes	12	49	4.90	1.17-23.58	0.037
No	3	60	7		
Hypotension					
Yes	10	23	7.48	2.04-30.15	0.001
No	5	86			
Hypoxaemia					
Yes	11	39	4.94	1.33-22.4	0.005
No	4	70			
Tachypnoea					
Yes	4	73	5.58	1.48-22.8	0.003
Irregular respiration					
Yes	2	1	16.62	0.78-988.7	0.038
No	13	108			
Aspiration pneumonia					
Yes	7	96	8.44	2.26-32.27	0.001
No	8	13			
Severe birth asphyxia					
Yes	9	14	10.18	2.76-39.01	0.0001
Poor obstetric history					
Yes	5	12	4.04	0.99-16.24	0.018
No	10	97			
Booking status					
Booked	11	101	0.29	0.06-1.64	0.112
Not booked	3	8			

Factors associated with poor outcome in the babies

\*Some babies did not have clinical examination recorded, hence the totals are less than 124

## DISCUSSION

The main objective of this study was to determine the prevalence of low Apgar score and to establish the immediate outcome and possible risk factors for low Apgar score in babies born in Mulago, Uganda's main teaching and referral hospital. We also sought to establish factors associated with poor outcome in babies with low Apgar score.

*Prevalence of low Apgar score:* This at one minute was 8.4% while at five and at twenty minutes it was 2.8% and 1% respectively. The prevalence of one minute Apgar score in the current study was much higher than in previous studies(3,15). Only 1.6% had an Apgar score of 0-3, a finding close to that of Ikonen(15) (1.2%) but deviating greatly from that of Apgar(3) (7.4%) and Drage(16) (6.7%). The differences

could be due to individual observer variation given the subjective nature of the Apgar scoring system. In most previous studies the scoring system was made by experienced persons, but only a few had individuals whose primary responsibility was that of observing and scoring the newborn(3,16). In the current study, however, all the research assistants were trained in carrying out the Apgar score and were not part of the routine staff in the delivery room. It is possible that in the previous studies the scoring was made by midwives who were involved in the delivery of the babies, and this has been documented to affect the score(2,16).

*Risk factors associated with low Apgar score:* Among the maternal factors, primiparity and the mode of delivery other than spontaneous vertex delivery, were found to be significantly associated with low Apgar scores (p-values 0.008 and 0.039). In the current study 68 (55%) of the 124 mothers whose babies had low Apgar scores were primiparous while only 37% of the control group were primiparous. These are similar to findings in Nathoo's study in Harare Zimbabwe(7), in which 60% of the mothers of the babies with low Apgar score were primiparous. This is not surprising given the fact many authors have demonstrated primiparity to be a risk factor in low Apgar score babies(16,17). In fact teenage(18) and eldery primiparae(19) often have prolonged labour, perform poorly during delivery and are likely to have cephalo-pelvic disproportion necessitating Caesarian section.

The mode of delivery, other than spontaneous vertex delivery, was also a risk factor for low Apgar scores. It is possible that factors contributing to the abnormal delivery such as fetal distress, cord accidents, cephalo-pelvic disproportion and ante-partum haemorrhage are risk factors for low Apgar score themselves. Most abnormal modes of delivery such as forceps, vacuum extraction or Caesarian section are often a result of onset of prolonged labour and onset of fetal distress(7,17).

Even though the difference between the mean ages of the mothers of babies with low Apgar score (21±5.6 years) and the mothers of control babies (23±4.8 years) were not statistically significant (p = 0.068), stepwise regression analysis revealed maternal age to be a significant risk factor (p = 0.003). However the regression coefficient was only -0.0612 which means that for each decrease in maternal age by one year, the risk of getting a baby with low Apgar score increases by one. Teenage mothers are more likely to have cephalo-pelvic disproportion(20), and be delivered by Caesarian section. They also perform poorly in labour and delivery(19) and are more likely to have abnormal modes of delivery. The negative effect of maternal age on birth and neonatal outcome have also been observed in mothers older than 35 years(20).

The current study has found a history of medical disease in pregnancy to be a significant risk factor for low Apgar score. This is consistent with findings of other studies(7,17) and is not surprising given the fact that some of these diseases such as diabetes mellitus, malaria and anaemia are themselves risk factors for low Apgar score(7,17,20). Several other factors such as prolonged labour, antepartum haemorrhage and pregnancy induced hypertension, were surprisingly, not found to be significant risk factors for low Apgar score even though other workers found them significant.

This could be due to the fact that most of the mothers delivered by Caesarian section in the current study did not have their second stage determined. Secondly the mothers with antepartum haemorrhage were attended to urgently hence reducing the chance of their babies getting asphyxia. The reasons why pregnancy induced hypertension was not significantly associated with low Apgar score are not clear.

Factors in the babies found to be significantly

associated with low Apgar score included birth injuries and cord accidents (p = 0.002 and 0.002 respectively) consistent with other studies which also found cord accidents (prolapse, entanglement, cord presentation, tight cord around the neck) to be significant risk factors(21,22). Cord accidents often lead to reduced blood flow from the placenta to the fetus causing impaired gaseous exchange and the babies are usually born asphyxiated(1).

Although several studies have shown low birth weight (<2.5kg) to be a risk factor for low Apgar score(16,23) it was not significantly associated with low Apgar score in the current study. The small number of low birth weight babies (only 16.1% of the 124 studied and 11.3% of the 124 controls) could explain this. Similarly gestational age was not found to be a statistically significant (p = 0.08) risk factor and this could have been due to the very small numbers of preterm babies (13.7% of the 124 controls).

Immediate (48 hours) outcome of babies with low Apgar score: The main outcome measure in this study was clinical improvement, complications or death. Clinical improvement was determined by normal temperature, cardiovascular system and no signs of hypoxic ischaemic encephalopathy (HIE) and normal oxygen saturation.

Hypoxic ischaemic encephalopathy was present in 27 (24.8%) of the babies with low Apgar score, 13 of whom had moderate HIE and one with severe HIE. This is similar to findings in several other studies(7,20) where the prevalence of HIE in babies with low Apgar score ranged from 24-52%. There were several respiratory complications such as pneumonia, persistent tachypnoea, apnoea and periodic breathing. Previous studies have documented similar findings(17,24). Cardiovascular complications such as hypotension and bradycardia were present as have been observed by other workers(25,26).

The mortality rate among the babies with low Apgar score within the first 44 hours was 12.1% while there were no deaths amongst the controls (with normal Apgar scores). This mortality is comparable to the 10% observed by Lam and Yeung(27), while it is very much lower than what Drage and colleagues(16) found (44%).

Clinical complications present in more than half of the babies who died included hypoxic ischaemic encephalopathy, hypothermia, hypotension, the respiratory distress syndrome and abnormal breathing patterns.

Autopsy was performed in 9 of the 15 babies who died and the findings were divers ranging from two with hyaline membrane disease, amniotic fluid aspiration, meconium aspiration, and subarachnoid haemorrhage. Most deaths occurred within the first 24 hours of life, a finding similar to that of several other researchers(16,25). *Factors associated with poor outcome:* These were either factors in the mothers or in the babies. Poor obstetric history (such as abortion, previous perinatal morbidity and mortality) was significantly associated with increased risk of the baby dying in the first 43 hours after birth. Factors that significantly contributed to low Apgar scores such as pregnancy induced hypertension, anaemia, heart disease, diabetes mellitus, also affected perinatal mortality.

In the babies, the risk factors for poor outcome included birth injury, hypothermia, hypoglycaemia, hypotension, hypoxaemia and severe birth asphyxia. An Apgar score 3 and below at one or five minutes has also been found to be significantly associated with poor outcome in other studies(16,29). Hypothermia was a particularly serious problem with 14 of the 15 babies who died having it.

### CONCLUSIONS

The prevalence of low Apgar score at one minute was 8.5% while at five minutes it was 2.8%. More than half (57.3%) of the babies with low Apgar scores had adverse outcomes: 45.2% had clinical complications while 12.1 % died.

Maternal factors significantly associated with low Apgar score included primiparity, mode of delivery, age and medical diseases during pregnancy, while risk factors in the babies included birth injuries and cord accidents.

Risk factors for poor outcome in babies with low Apgar score included birth injury, hypothermia, hypoglycaemia, hypoxaemia, hypotension, persistent tachypnoea, irregular respiration, aspiration pneumonia and severe birth asphyxia.

The Apgar score is a useful tool for detecting babies needing immediate resuscitation and for predicting immediate outcome. There is need to routinely monitor temperature, blood glucose, blood pressure and oxygen saturation in babies with low Apgar scores.

#### REFERENCES

- 1. WHO *Basic newborn resuscitation:* a practical guide. WHO *Report* 1997; 2-3.
- Apgar, V. A proposal for a new method of evaluation of the newborn infant. *Current Research in Anaesthesia* 1953; 32:260-267.
- Apgar, V. and James, L.S. Further observations on the newborn scoring system. *Amer. J. Dis. Child.* 1962; 104:419-428.
- Paul V.K., Singh M., Sundaram K.R., and Deorari A.K., Correlates of mortality among hospital – born neonates with birth asphyxia. *Natl. Med. J. India.* 1997; 10:54-57.
- Jepson, H.A., Talashek, M.L. and Tichy, A.M. The Apgar score: evaluation, limitations, and scoring guidelines. *Birth*. 1991; 18:83-92.
- American Academy of paediatrics: Use and Abuse of the Apgar Score (RE9621) Policy Statement. *Pediatrics*. 1996; 98:141-142
- 7. Nathoo, K.J., Chimbira, T.H.K. and Mtimavalye, L.A. Mortality and immediate morbidity in term babies with low

Apgar scores (Zimbabwe). Annals of Trop. Paediat. 1990; 10:239-244.

- Kish, L. Survey sampling. John Wiley and sons, New York, 1965; 35-70.
- Fleiss, J.L. Statistical methods for rates and proportions, 2nd edition, John Wiley and sons, New York, 1981: 44-45.
- Quintanilla de la Garza, C., Juarez, C.J.A., Escobar, H.C. and Estrada, G.P. Adolescent primipara. *Ginecol. Obstet. Mex.* 1997; 65:533-537.
- 11. Marlow, N. Do we need an Apgar score? Archives Dis. Child. 1992; 67:765-769.
- Sarnat H.B., and Sarnat M.S., Neonatal encephalopathy following fetal distress. Arch. Neurol. 1976; 33:696-705.
- 13. Ballard, J.L., Kazmaier, K.N. and Driver, M.A. A simplified score for assessment of fetal maturation of newly born infants. *J. Pediat.* 1979; **95:**769-774.
- 14. Ludwig, J. *Current methods of autopsy practice*. W.B. Saunders Philadelphia 1972; **1**:2-3.
- Ikonen, R.S. The Apgar scoring of newborn infants and its relation to neonatal morality. *Annals peadiat. Fenniae*. 1967; 13:111-114.
- Drage, J.S., Kennedy, C. and Schatz, B.K. The Apgar score as an index of neonatal mortality. *Obstet. and Gynae*. 1964; 24:222-230.
- Daga, A., Daga, S.R. and Patole, S.K. Risk assessment in birth asphyxia. J. Trop. Peadiat. 1990; 36:34-39.
- Krokfors, E., Wist, A. and Hirrvensalo, M. Effects of duration of labour, age and parity on the Apgar score of the newborn infant. *Annales Chirugie et gynaecologiae fenniae*. 1963; 52:3-14
- Kumbi, S. and Isehak, A. Obstetric outcome of teenage pregnancy in northern western Ethiopia. *East Afr. Med. J.* 1999; **76:**138-140.
- Nem-Yun, B. and Munn-Sann, L. Factors associated with clinical significant perinatal asphyxia in the Malaysian neonates: a case control study. *J. Trop. Paediat.* 1992; 38:284-289.
- Derham, R.J., Mathews, T.G. and Clarke, T.A. Early seizures indicate quality of perinatal care. *Archives Dis. Child.* 1985; 60:809-813.
- Huge, M.M., Malligan, J.C., Allen, A.C. and Taylor, P.M. Neonatal asphyxia I. Relationship of obstetric and neonatal complications to neonatal mortality in 38405 consecutive deliveries. *Pediatrics*. 1980; **96**:898-902.
- Levy, B.T., Dawson, J.D., Toth, P.P. and Bowdler, N. Predictors of neonatal resuscitation, low Apgar scores and umbilical artery pH among growth restricted neonates. *Obstet. and Gynae.* 1988; **91**:909-916.
- Sacidharan, P. Breathing pattern abnormalities in full term asphyxiated newborn infants. *Archives Dis. Child.* 1992; 67:440-443.
- De Souza, S.W. and Richards, B. Neurological sequelae in newborn babies after perinatal asphyxia. *Archives Dis. Child.* 1978; 53:564-569.
- Weinsberger, B., Anwar, M., Hegyi, T., Hiatt, M., Koons, A. and Paneth, N. Antecedents and neonatal consequences of low Apgar score in preterm newborns: a population study. Arch. Pediatr. Adolesc. Med. 2000; 154:294-300.
- 27. Lam, B.C. and Yeung, C.Y. Perinatal features of birth asphyxia and neurologic outcome. *Acta. Pediatrica-Japan.* 1992; **34:**17-22.
- Ohlsson, A., Shennan, A.T., and Rose, T.H. Review of causes of perinatal mortality in a regional perinatal care centre, 1980-1984. *Amer. J. Obstet. Gynae.* 1987; 157: 443-445.
- 29. Apgar, V. The newborn (Apgar) scoring system. *Pediat. Clinics North Amer.* 1966; **13**:645-650.