

## **Perinatal asphyxia at Port Moresby General Hospital: a study of incidence, risk factors and outcome**

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### **SUMMARY**

**We investigated the incidence and outcome of perinatal asphyxia (PA) at Port Moresby General Hospital by a retrospective chart review and prospective collection of data, spanning a total of 2.5 years. 125 babies weighing more than 2000 g at birth with a gestation of 34 weeks or more and with no obvious congenital abnormalities were diagnosed to have PA. During the same time period 22,700 liveborn babies were delivered, a PA incidence of 5.5/1000 livebirths. There was a 31% mortality and considerable morbidity. Hospital records for 114 affected babies and 115 controls (the next baby born by normal delivery) were compared. Significant risk factors for PA were: previous stillbirth or neonatal death, fetal heart rate abnormalities, membranes ruptured for more than 12 hours prior to delivery, meconium staining, antepartum haemorrhage, maternal fever, prolonged first and second stages of labour, preterm or post-term delivery and operative delivery. In only 73 affected babies was the 5-minute Apgar score recorded as 6 or less. All 34 of the babies with grade 3 hypoxic ischaemic encephalopathy (HIE) either died (30) or had serious neurological impairment. The treatment of affected babies remains largely supportive and some causes of PA are currently unavoidable. It is, however, widely accepted that some cases of perinatal asphyxia may be prevented by the delivery of high-risk pregnancies in obstetric facilities with appropriate intervention and by good neonatal resuscitation. Sophisticated or expensive equipment is not a necessity.**

### **Introduction**

Perinatal asphyxia (PA) refers to an insult to the fetal or neonatal brain as a result of hypoxia occurring during labour and/or delivery. When abnormal neurological activity such as impaired consciousness, disturbance in muscle tone or convulsions follows, a diagnosis of hypoxic ischaemic encephalopathy (HIE) is applied (1). Severe PA also results in pulmonary hypertension, respiratory failure, the syndrome of inappropriate ADH secretion, metabolic disturbance such as hypoglycaemia and hypocalcaemia, renal impairment (acute tubular necrosis) and necrotizing enterocolitis.

Meconium aspiration syndrome is part of the spectrum of PA. In the most stringent of definitions of PA, evidence of multiorgan dysfunction is required (2). PA is one of the leading causes of perinatal death in both developed and developing countries (3,4). A number of risk factors for PA have been recognized, which should allow for the identification of high-risk mothers and their selection for supervised care and delivery.

Fetal heart monitoring, which can be satisfactorily achieved using regular auscultation through a fetal stethoscope, allows for detection of impending fetal distress so that

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measures such as operative delivery and intensive neonatal resuscitation can be taken to avoid or minimize the duration and severity of hypoxia. Where antenatal services and obstetric care are inadequate and regular monitoring of the fetal heart is not carried out the incidence of PA may be expected to be high.

PA results in a spectrum of severity of HIE, with a corresponding spectrum of outcome. Classifications of HIE based on EEG abnormalities have been proposed (5,6) and continuous EEG monitoring postnatally has been shown to have high predictive value (7). In the absence of EEG, the clinical classification proposed by Sarnat and Sarnat (8) and modified by Fenichel (9) allows for reasonably accurate prediction of long-term outcome for the mild and severe grades. This classification is based on level of consciousness, muscle tone, tendon reflexes, myoclonus, primitive reflexes (including the moro and grasping reflexes), autonomic function and seizures.

The health of the newly born baby is assessed by the Apgar score in which a score of 0-2 is given for each of colour, heart rate, grimace response, activity (muscle tone) and respiration. A score done only at 1 minute does not predict long-term outcome. However, a change of score over time has prognostic value and a score of 3 or less at 20 minutes has been associated with a mortality of 59% and a cerebral palsy rate of 57% (10,11).

Despite recent advances in the understanding of the pathophysiology of PA, there have been no comparable advances in the management of affected newborns.

The Port Moresby General Hospital (PMGH) is the only tertiary hospital in Papua New Guinea (PNG). It serves as the provincial hospital for the National Capital District (NCD) (population of 300,000) and the referral hospital for Central and part of Gulf Provinces. The Obstetric Division catered for 9324 deliveries in 1997 and PA is a major cause of mortality and morbidity (12). The importance of the maternal, labour, delivery and neonatal risk factors for PA in patients at PMGH is not known. This study was intended to provide accurate information relating to PA and its

outcome at PMGH. The specific aims of the study were to determine the incidence of PA and identify its risk factors. A subsidiary aim was to examine the treatment of affected babies and to assess outcome. It was hoped that the results might enable the introduction of new policies, or the refinement and reinforcement of current management policies, to reduce the incidence of PA.

## Methods

This was a case-control study performed predominantly retrospectively (January 1995 - February 1997) and partly prospectively (March 1997 - June 1997). Labour Ward records and Special Care Nursery (SCN) records were reviewed. Liveborn babies were included as cases of PA on the basis of the following:

- 1 Death in the first 24 hours of life in the absence of obvious congenital abnormality, or
- 2 Abnormal neurological manifestations within the first week of life with hyperirritability, alteration in muscle tone or conscious level, convulsions, or abnormal primitive reflexes

and

Prolonged resuscitation (defined as more than 5 minutes to spontaneous respiration), or a 5-minute Apgar score of 6 or less.

Since prematurity and intrauterine growth restriction are independent risk factors for PA, babies were excluded from the study if they weighed <2000 g or were estimated (by clinical assessment using the Dubowitz score and antenatal ultrasound measurements where available) to be <34 weeks of gestation.

For each case, the next baby recorded in the Labour Ward delivery book with no features of PA, with a weight of 2000 g or more and with a gestation (by dates and/or scan) of >34 weeks was selected as a control.

For each baby in the study, the following information was extracted from the records:

- a) Cases and controls: sex, birthweight, gestational age, mode of delivery, Apgar scores. For cases: resuscitation method,

medication during and after resuscitation, complications, duration of stay in the Special Care Unit and final outcome. The grade of HIE (8,9) was determined from available data in the case records.

- b) Mothers of all cases and controls: age, parity, antenatal clinic attendance, previous obstetric history, antenatal problems, duration of rupture of membranes, presence and degree of meconium staining, antepartum haemorrhage, length of labour, intrapartum observations, accoucheur and medication used in labour.

All information was collected on a pretested information sheet and entered and analyzed using the Epi Info 6 package. Differences between cases and controls were assessed using the Mantel-Haenszel  $\chi^2$  test with Yates' correction or the Fisher exact test where appropriate, and odds ratios (ORs) with 95% confidence intervals (CIs) calculated where relevant.

We calculated that if at least one risk factor was present in 20% of pregnancies a sample

size of 105 cases and the same number of controls would give the study a power of 80% to detect differences at a level of  $p < 0.05$  and an odds ratio of 2.5.

**Results**

A total of 125 babies with perinatal asphyxia were recorded during the 2.5 year study period. During that time 22,700 liveborn babies were delivered. The incidence of PA was therefore 5.5/1000 livebirths. An additional asphyxiated baby was born before arrival and 3 were delivered elsewhere and referred to PMGH, giving a total of 129 in the study.

Case records for 15 of the babies, 9 of whom had died, could not be retrieved and they were therefore excluded from the analysis of risk factors. Information was available for 115 of the controls. Of the 114 cases available for full analysis 31 died; this gives an overall mortality in the 129 cases of 40/129 (31%).

The characteristics of the study population are summarized in Table 1. There were

**TABLE 1**

CHARACTERISTICS OF THE STUDY POPULATION

Neonatal characteristics	Cases	Controls	p value
Number of males	68	66	ns
Duration of gestation:			
Number preterm	10	1	<0.005
Number post-term	7	0	
Birthweight			
Mean	3016	3132	
Range	2000-4750	2000-4470	
Weight for age			
SGA	11	7	0.3
LGA	12	7	0.2
Apgar score 6 or less at 5 minutes	73	-	

SGA = small for gestational age  
LGA = large for gestational age

**TABLE 2**

## PERIPARTUM FACTORS ASSOCIATED WITH PERINATAL ASPHXIA

Peripartum factors	Cases (N=114)	Controls (N=115)	p value	OR (95% CI)
Fetal heart rate abnormalities	24	1	0.005	30.4 (7.1-183.41)
Duration of rupture of membranes				
12-23 hours	20	12	<0.005	3.96 (1.84-8.66)
≥24 hours	16	0		
Meconium staining				
Nil	36	90	<0.005	0.13 (0.07-0.24)
3+	44	1		
APH	5	0	0.03	Undefined
PET	5	7	ns	0.71 (0.19-2.69)
Maternal fever	9	0	0.002	Undefined
Duration of labour				
1st stage >24 hours	24	6	<0.005	4.84 (1.81-15.04)
2nd stage:				
<10 minutes	16	53	<0.005	0.24 (0.12-0.48)
10-29 minutes	38	54	0.42	0.77 (0.42-1.39)
30-59 minutes	20	5	<0.005	5.97 (2.04-21.1)
60+ minutes	19	3	<0.005	9.5 (2.63-51.38)
Mode of delivery				
NVD	54	102	<0.005	0.11 (0.05-0.24)
Assisted breech	6	3		
Vacuum extraction	24	7		
Forceps	3	3		
LUSCS	25	0		
Symphysiotomy	2	0		

APH = antepartum haemorrhage

PET = preeclamptic toxemia

NVD = normal vaginal delivery

LUSCS = lower uterine segment caesarian section

ns = not statistically significant

significantly more mildly preterm and post-term babies among the cases than the controls. Birthweights of the two groups were similar. More cases than controls were either small for dates or large for dates, but these differences did not reach statistical significance. An Apgar

score of 6 or less was documented in 73 of the cases.

The peripartum factors are shown in Table 2. There were significant differences between cases and controls in fetal heart rate

**TABLE 3**

**MATERNAL RISK FACTORS ASSOCIATED WITH PERINATAL ASPHYXIA**

<b>Maternal risk factors</b>	<b>Cases (N=114)</b>	<b>Controls (N=115)</b>	<b>p value</b>
Age: <20 years	23	17	0.28
>40 years	2	0	
Parity: 0	55	49	0.39
≥5	5	8	0.40
Obstetric history:			
Previous miscarriage	5	6	
Previous stillbirth	4	0	0.003
Previous neonatal death	4	0	
ANC attendance:			
Booked	94	110	0.118
Referred	6	2	
Unbooked	5	0	
Mean number ANC visits	6.3	6.4	
Mean duration of gestation at first visit (weeks)	25	25	
Haemoglobin level:			
8-9.9 g/dl	18/90	24/98	0.11
<8 g/dl	9/90	4/98	0.095
VDRL positive	2	3	

ANC = Antenatal Clinic

VDRL = Venereal Disease Research Laboratories test for syphilis

abnormalities, duration of rupture of membranes, meconium staining, antepartum haemorrhage, maternal fever, duration of first and second stage of labour and operative delivery.

The maternal risk factors often associated with PA are summarized in Table 3. The mean age and the parity of the case and control mothers were similar. A previous history of stillbirth or neonatal death was strongly associated with PA. There were more referred and unbooked case mothers than controls, but the number of antenatal visits and the gestation at the time of booking were the same in the two groups. More of the case mothers were

severely anaemic, but the difference did not reach statistical significance.

Details of the resuscitation and management of the affected babies are shown in Table 4. 82 babies (72%) were documented to have received intermittent positive pressure respiration by one of three techniques. 38 (33%) received bicarbonate, 23 (20%) received dextrose and 29 (25%) received naloxone.

During postnatal management anti-convulsants were used in more than 40% of babies and antibiotics were given to 92%. Intravenous fluids were given to 77% of the babies and continued for a mean of 2.4 days.

**TABLE 4**

## RESUSCITATION AND MANAGEMENT OF AFFECTED BABIES

<b>Procedures/medication</b>	<b>No (%) (N=114)</b>
<b>Resuscitation</b>	
Frog breathing	28 (25)
Bag + mask	16 (14)
Intubation	38 (33)
<b>Medications at resuscitation</b>	
Bicarbonate	38 (33)
Dextrose	23 (20)
Naloxone	29 (25)
Adrenaline	5 (4)
<b>Medications post resuscitation</b>	
Mannitol	7 (6)
Phenobarbitone	47 (41)
Paraldehyde	45 (39)
Diazepam	2 (2)
Phenytoin	3 (3)
Ampicillin/gentamicin	105 (92)
Chloramphenicol	9 (8)
Oxygen	113 (99)
Intravenous fluids	88 (77)
IV fluid duration (days mean)	2.37
Duration of SCN stay (days mean)	6.5
<b>Complications</b>	
Hypoglycaemia	9 (8)
Seizures	49 (43)
Apnoea	4 (4)
<b>Outcome</b>	
Death	31 (27)
Neurological sequelae	10 (9)
Other birth injuries: cephalohaematoma	16 (14)

SCN = Special Care Nursery

Hypoglycaemia occurred in 8% and seizures in 43%. The mean duration of stay in SCN was 6.5 days. Of the 31 babies (27%) who died, 19 (61%) died within 24 hours of delivery, 26 (84%) within 48 hours and 30 (97%) within the first week. 10 (12%) of the 84 babies surviving the first week had neurological damage documented at discharge. 8 were hypertonic and hyperreflexic, 1 had an Erb's palsy and 1 had persistent seizures. 16 babies had cephalohaematomas. None of the control babies died and none were recorded as having neurological abnormality prior to discharge.

The relationships between resuscitation, Apgar score, meconium staining and outcome, respectively, and grade of severity of HIE are shown in Table 5. The degree of meconium staining was not associated with the grade of HIE. All babies with grade 3 HIE either died (88%) or suffered significant neurological damage (12%).

The relationship between Apgar score and outcome is shown in Table 6. 27 of the 31 babies who died had a low 5-minute Apgar score, compared with 46 of the 81 survivors:  $\chi^2=7.79$ ,  $p=0.005$ ; odds ratio and 95% confidence interval 5.14 (1.56-21.77). A low Apgar score was not significantly associated with death after 24 hours.

No babies with grade 1 or 2 HIE died in hospital. 30 of the 31 babies with fatal Grade 3 HIE died in the first week and 1 died at the age of 16 days with a diagnosis of meningitis. All 6 of the babies who were delivered to mothers referred because of high risk died. There was no difference in the mortality rates between babies of booked (24/103) and unbooked (1/5) mothers.

## Discussion

### Incidence

Our study found an incidence of PA of 5.5/1000 livebirths associated with an overall mortality of 31%. Comparison of data from different studies of incidence and outcome of PA is complicated by non-uniformity of inclusion criteria and study populations. We used the same inclusion criteria as Hall et al. in

**TABLE 5**

RESUSCITATION AND OUTCOME BY SEVERITY OF HYPOXIC ISCHAEMIC ENCEPHALOPATHY

	Stage of HIE			Total (114)
	I (N=44)	II (N=36)	III (N=34)	
<b>Resuscitation</b>				
Oxygen and suction	17	9	6	32
Frog breathing	12	12	4	28
Bag and mask	8	4	4	16
Intubation	7	11	20	38
<b>Apgar score (6 or less at 5 minutes)</b>				
Yes	21	23	24	68
No	22	12	10	44
Unknown	1	1	0	2
<b>Outcome</b>				
Death	0	0	31*	31
Neurological sequelae	0	6	4	10
Well	43	30	0	73
<b>Meconium staining</b>				
1 +	5	3	6	14
2 +	5	10	5	20
3 +	17	10	17	44
Clear	17	13	6	36

\* 1 baby with neurological sequelae died at 16 days with a diagnosis of meningitis

HIE = hypoxic ischaemic encephalopathy

**TABLE 6**

OUTCOME IN RELATION TO APGAR SCORE AT 5 MINUTES

Outcome	Apgar ≤6	Apgar ≥7	Total (112*)
Death <24 hours	18	1	19
Death ≥24 hours	9	3	12
<b>Total deaths</b>	27	4	31
Neurological sequelae	9	1	10
No sequelae	37	34	71
<b>Total survivors</b>	46	35	81

\* No score available for 2 patients

**TABLE 7**

## INTERNATIONAL COMPARISONS OF PERINATAL ASPHYXIA

<b>Author</b>	<b>Country</b>	<b>Population criteria</b>	<b>Incidence per 1000 livebirths</b>	<b>Mortality</b>
Oswyn et al.	PNG	BWt >2 kg >34 weeks	5.5	31%
Hall et al. (13)	South Africa	BWt >2 kg >34 weeks	4.6	9%
Boo and Lye (14)	Malaysia	All gestations	18.7	18%
Airede (15)	Nigeria	Full-term	26.5	18%
MacDonald et al. (16)	USA	All gestations BWt >2 kg >33 weeks	11.6 6.0	NK NK
Finer et al. (1)	Canada	>37 weeks	3.6	7%
Levene et al. (17)	United Kingdom	Full-term	6.0	NK

NK = not known

their study from South Africa (13). Infants born preterm or severely growth retarded were excluded, since both factors are independently associated with PA. The results of several other similar studies are shown in Table 7. The incidence in our study is similar to that in studies in which similar inclusion criteria were used.

### **Risk factors**

#### *Neonatal*

In spite of excluding babies weighing <2000 g, moderate prematurity was still a significant risk factor. Postmaturity was also a risk factor.

#### *Maternal*

Of the maternal parameters studied only a previous stillbirth or neonatal death was significantly associated with PA. However, there were more young (<20 years) and more old (>40 years) mothers among the cases than the controls and more had severe anaemia (Hb <8 g/dl) antenatally. A larger sample size may well have resulted in significance of these

factors, as has been found in other studies.

#### *Antenatal and peripartum*

Antepartum haemorrhage and maternal fever, but not preeclamptic toxemia, were strongly associated with PA.

Signs of fetal distress – fetal heart rate abnormalities and meconium staining of the amniotic fluid – showed a marked association with PA, although the degree of meconium staining did not relate to the degree of HIE.

Prolonged first and second stages of labour were clearly associated with PA whilst a second stage of less than 10 minutes was protective. Since the indications for operative delivery include prolonged labour and fetal distress, it was also not surprising that there was a close association between operative delivery and PA.

#### *Referral*

A total of 8 mothers in the combined groups were referred from health facilities outside the Port Moresby area and 6 babies died - an

extremely disappointing and worrying result. It may be that referral was delayed for one reason or another. A study of the background of all referred pregnancies might indicate ways of improving outcome.

### **Inclusion criteria**

Our use of the inclusion criteria used by several other authors was thought likely to produce reasonably accurate and comparable data. Each of the criteria used alone is imperfect. Death in the first 24 hours following prolonged resuscitation or low Apgar score may, for example, be the result of congenital abnormality incompatible with life. Since autopsies were not performed on the babies who died it is possible that some babies with no external features of abnormality fell into this category. Compared with the results from studies that have not used death in the first 24 hours as an inclusion criterion, results for this study are inevitably biased towards a higher mortality.

The criterion of abnormal neurological manifestations after prolonged resuscitation or low Apgar score would, in the absence of detailed routine neurological examination in all babies following resuscitation, bias the results towards those most obviously and most severely affected. However, in the absence of EEG testing such manifestations are the clinical hallmarks of PA. All the PA cases in the study had abnormal neurological signs.

Only 73 (65%) of the 112 cases for whom an Apgar score was available had a score of 6 or less. This may indicate over scoring by labour ward staff. It is widely accepted that a marked improvement in Apgar score between 1 and 5 minutes is associated with a relatively good prognosis. It is important to consider that the majority of children who develop cerebral palsy are born with a normal Apgar score. In the present study a low 5-minute score was associated with 5 times the risk of death. This may not be surprising in view of our inclusion criterion. On the other hand 4 (13%) of the patients who died had a 5-minute score of 7 or more. Although many babies with grades 2 and 3 HIE had a 5-minute Apgar score of 7 or more, a score of 6 or less was present in 71% of those with grade 3 HIE, 64% of those with

grade 2 and 48% of those with grade 1 (Table 5). An alternative predictive score based on evaluation of consciousness, respiration and Moro and grasp reflex at 30 minutes has recently been proposed (18).

### **Resuscitation**

Although it is well recognized that irreparable brain damage may have occurred prior to delivery, there is also no doubt that prompt and efficient neonatal resuscitation will prevent or limit cerebral injury in some asphyxiated babies. 82 (72%) of the cases received positive pressure ventilation either by frog breathing (25%), bag and mask (14%) or intubation (33%) (Table 4). Whilst not everyone is skilled at neonatal intubation, all those having contact with the newborn should be competent at bag and mask ventilation. Correctly performed, this can be almost as efficient for a short time as ventilation via an endotracheal tube. The recent finding that neonatal resuscitation using room air appears to be at least as effective as resuscitation using oxygen is welcome news for those working in hospitals and health centres without a regular oxygen supply (19). Neither ventilation by endotracheal tube nor bag and mask are necessarily dependent on oxygen, whereas frog breathing (closing the baby's mouth and nostrils around a nasopharyngeal oxygen catheter and releasing when the chest expands) requires an oxygen supply. Frog breathing has been taught to nursing staff for many years in Papua New Guinea and anecdotes suggest the technique is effective for brief periods to establish oxygenation and to stimulate respiratory effort. There have been no published trials on the efficacy of frog breathing.

### **Management**

At present the management of HIE is limited to supportive care. This consists of keeping the baby warm and well oxygenated, preventing and treating convulsions, preventing and treating infection and preventing and treating any associated metabolic complications, the most important of which is hypoglycaemia. There are major disturbances in cerebral blood flow and in blood pressure autoregulation in HIE, which

lead to cerebral oedema and raised intracranial pressure (20,21). Judicious fluid restriction is given – generally two-thirds maintenance, though some would apply greater restriction. Other methods of reducing cerebral oedema have been tried. In our series 7 babies received mannitol, though there is no evidence for its efficacy. Dexamethasone, often given for affected babies, is expensive and of no proven benefit. Intractable convulsions were a major problem, occurring in 43%. Hypoglycaemia was documented in 8%.

### Outcome

31 (27%) of the 114 documented patients and 40 (31%) of the original 129 died. 19 of the 31 (61%) died in the first 24 hours. 10 babies had obvious neurological sequelae at the time of discharge. Although no long-term follow-up of the surviving patients has been done, it is likely that more will have sustained permanent neurological damage. Other studies show that almost all of those with grade 3 HIE and about a quarter of those with moderate HIE will either die or be significantly damaged (22). All the babies who died had grade 3 HIE. In contrast, none of the babies with lesser degrees of HIE died. Although the use of early mortality as an inclusion criterion inevitably biases our overall results to a higher mortality, differences between the mortality rate in our study compared with those in other studies using similar inclusion criteria are difficult to explain. The low mortality in our babies with grade 2 HIE suggests that the high mortality overall may not have been the result of poor management of PA, but of the high proportion (30%) of babies with grade 3 HIE, although this is arguable.

### Conclusion

The incidence of PA at PMGH is similar to that found in comparable studies. Many of the established risk factors, particularly those related to obstetric history, are pertinent to our population. Outcome for affected infants was dismal – a 31% mortality and at the most conservative estimate a 9% serious morbidity rate.

The results emphasize the importance of some basic principles in antenatal and perinatal care. Many of the risk factors can be detected

by antenatal history and examination. It is important that women with high-risk pregnancies are detected and referred early enough for supervised care and delivery to an optimal outcome. Signs of fetal distress, antepartum haemorrhage, prolonged rupture of the membranes, prolonged first or second stage of labour and maternal fever during labour are indications of impending or established perinatal asphyxia. The baby should be delivered as soon as possible and a skilled resuscitator, with at least a resuscitation bag and mask in working condition, should be available.

Whilst it is not possible to prevent all cases of PA it is certainly possible, by following standard antenatal and perinatal procedures (23), to prevent many cases using relatively simple equipment and resources.

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