BIRTH ASPHYXIA — PRESENTING THE CASE FOR 'A STITCH IN TIME'

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Objectives. To review the current birth asphyxia and subsequent cerebral palsy (CP) rates at a teaching hospital in a developing country, and to place these rates within the context of the current caesarean section (CS) rate. To determine the number of cases of birth asphyxia that are preventable.

Design. Retrospective, descriptive study.

Setting. Neonatal nursery and intensive care unit, Johannesburg Hospital.

Methods. Maternal and neonatal records were reviewed for 48 babies weighing less than 1 800 g born between 1 January and 31 December 1997 with birth asphyxia. Outcome after discharge was determined from the neonatal follow-up notes until 31 March 1998.

Results. Mortality in the group of birth-asphyxiated babies was 12.5%. The birth asphyxia rate was 6/1 000 live births, and the CP rate in the study group was 1.15/1 000 live births. The CS rate for the group was 29%, with an overall CS rate at the hospital of 20.5%. In 22 cases (46%) the cause of birth asphyxia was considered to have been preventable.

Conclusion. The CP rate is considerably higher than that quoted for developed countries, and a significant number of cases of birth asphyxia in the study were preventable. In the face of the high birth asphyxia and CP rates, the CS rate appears to be inappropriately low. The CS rate should be audited in the context of the birth asphyxia and CP rates.

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Birth asphyxia is an important cause of adverse neonatal outcome, including death and cerebral palsy (CP). Recent literature from developed countries, including the USA, suggests that most cases of birth asphyxia are unavoidable and unrelated to intrapartum management.[1,2] It is also suggested that asphyxia is a relatively uncommon cause of CP in these countries.[3-5] However, birth asphyxia rates appear to be considerably higher in developing countries, such as South Africa, than in developed countries.[6] It is our contention that labour-related problems remain an important cause of birth asphyxia in South Africa, and that asphyxia is a more common cause of CP than is the case in developed countries. In order to evaluate this hypothesis a review was conducted of all asphyxiated babies delivered at Johannesburg Hospital during 1997.

METHODS

A retrospective analysis was conducted on all babies with a diagnosis of asphyxia who were delivered at Johannesburg Hospital from 1 January to 31 December 1997. Babies were included in the sample if the birth weight was > 1 800 g, the 5-minute Apgar score was ≤ 6, or if the birth history and subsequent clinical course strongly suggested asphyxia (i.e. there was no evidence for another cause of the neonatal encephalopathy such as central nervous system malformation or depression from administration of maternal drugs). Data regarding the peripartum and perinatal course of the neonates were obtained from the maternal and neonatal bed letters, and outcome was assessed from the follow-up notes of the babies after discharge (until 31 March 1998). Data collected on the mothers included demographic information, antenatal history, parity, gravidity, and mode and complications of delivery. This information was reviewed by an obstetrician (FG) and a judgement was made as to whether the asphyxia was related to obstetric management or whether it was unavoidable. If there were insufficient data available to make this judgement, the case was termed 'unclassified'. Prolonged first stage of labour was defined as any latent phase over 14 hours' duration for both primi- and multigravid patients. Both primi- and multigravid patients were considered to have a prolonged second stage of labour if this was longer than 1 hour in duration. (Although these definitions are not strictly in accordance with World Health Organisation definitions, they are the ones used in the Johannesburg Hospital labour ward because they are more appropriate from a practical standpoint (F Guidozzi — personal communication).) Neonatal information included demographic data, Apgar score, birth weight, gestational age, need for ventilation, presence of seizures, grade of hypoxic ischaemic encephalopathy (HIE) (Sarnat classification),[7] mortality, and duration of intensive care unit (ICU) and hospital stay. The long-term neonatal outcome was assessed as normal or abnormal according to the presence or absence of microcephaly, hydrocephalus, CP, seizures, developmental delay and blindness.

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RESULTS

During the study period, 7 765 babies were delivered at the Johannesburg Hospital. Sixty-two neonates > 1 800 g with a diagnosis of asphyxia were nursed in the neonatal unit during 1997, but only 48 of these were inborn patients and eligible for audit. Maternal and neonatal data are presented in Tables I and II.

Table I. Maternal data (*N* = 48)

Age (yrs) (mean (SD))	24.7	(4.6)
Parity (mean (SD))	0.84	(1.04)
Gravidity (mean (SD))	2	(1.06)
Booked	37	(77%)
Pre-eclampsia	3	(6%)
PROM	7	(14.5%)
Meconium-stained liquor	13	(27%)
Fetal distress	13	(27%)
APH	3	(6%)
Delivery		
Vaginal	26	(54%)
CS	14	(29%)
Vacuum	7	(15%)
Forceps	1	(2%)

PROM = prolonged rupture of membranes; APH = antepartum haemorrhage.

Table II. Neonatal data (*N* = 48)

Birth weight (g) (mean (SD))	3 061	(658)
Gestational age (wks) (mean (SD))	38.9	(2.2)
Hospital stay (d) (mean (SD))	12.3	(7.5)
Apgar score at 1 min	2.4	(1.4)
Apgar score at 5 min	4.7	(1.8)
HIE		
Grade 1	7	(14.5%)
Grade 2	16	(33%)
Grade 3	10	(21%)
Seizures	24	(50%)
Ventilated	19	(40%)
Cranial sonar		
Not done	29	(60%)
Normal	10	(21%)
Oedema	2	(4%)
IVH 1 and 2	3	(6%)
IVH 3 and 4	3	(6%)
PVE/minor abnormality	1	(2%)
Mortality	6	(12.5%)
Follow-up	28	(67%)
Outcome		
Normal	15	(32%)
Abnormal	13	(27%)

IVH = intraventricular haemorrhage; PVE = periventricular echodensity.

Seventy per cent of the pregnancies in this audit had an obstetric complication, and 70% of the babies were delivered vaginally. No epidural anaesthetic was administered to any mother in the study group. The caesarean section (CS) rate of the group was 29% and the assisted delivery rate 15%. The overall CS and assisted delivery rates at Johannesburg Hospital during the study period were 20.5% and 2.3%, respectively.

Twenty-two of the cases (46%) were considered to be preventable causes of birth asphyxia (see Table III).

A further 3 cases were complicated by delay in delivery as a consequence of being transferred from an outlying centre without CS facilities. Eleven of the deliveries were unclassifiable, and the remaining 12 were considered to be unavoidable causes of birth asphyxia (such as abruptio placentae). The 25 deliveries with potentially avoidable causes of birth asphyxia accounted for 83% (5/6) of neonatal deaths and 40% (4/10) of CP cases. Seven babies in this subgroup (28%) were lost to follow-up.

The birth asphyxia rate was 6/1 000 live births (1/169 babies delivered) and the CP rate was 1.15/1 000 births. These rates have been compared with those in other centres in Table IV.

At follow-up, 13/48 (27%) babies were found to have some neurological abnormality. Of these, 3 patients had evidence of mild motor developmental delay and the remaining 10 had evidence of CP. Of the severely affected babies, 5/10 had additional problems of epilepsy, microcephaly, hydrocephalus, and brain atrophy with *ex vacuo* dilatation of the lateral ventricles. These data are presented in Table V.

Five patients had culture-proven congenital sepsis, a further 1 had suspected meningitis (high polymorphonuclear cell count in cerebrospinal fluid, but culture negative), and another 3 patients were born to mothers treated for chorioamnionitis. Of these 9 patients, 7 were classified as HIE grade 1 and 5/7 were normal at follow-up (2/7 lost to follow-up). The remaining 2 patients were classified as having HIE grade 2 and both patients were lost to follow-up.

DISCUSSION

Much that has been written recently in developed countries about the unavoidability of neonatal encephalopathy questions the causative role of birth asphyxia in this condition.[4] It is clear from the results of the above audit that in a developing country such as South Africa, a significant proportion of cases (46%) of birth asphyxia are avoidable. Both the birth asphyxia and CP rates in this review are higher than those reported from other South African centres and the CP rate is approximately three times that in the USA.[2] The CP rate for this audit may in fact be higher, considering that 36% of the babies were lost to follow-up (46% of babies with HIE grade 2 were lost to follow-up). On the other hand, Johannesburg Hospital is a referral hospital, and the normal deliveries taking place at Alexandra Clinic

Table III. Details of delivery events and outcomes in potentially preventable birth asphyxia

Case No.	Delivery events	Delivery	Follow-up
1	Diabetic mother, previous IUD. Shoulder dystocia, failed vacuum	Forceps	CP, hydrocephalus
2	Retained 2nd twin, failed vacuum	CS	Died
3	Prolonged 2nd stage	NVD	CP, epilepsy
4	Fetal distress, MSL, prolonged 2nd stage	NVD	Lost to follow-up
5	Fetal distress, MSL	NVD	Normal
6	Decreased fetal movements 2 days before delivery, non-reactive NST	CS	Lost to follow-up
7	POG2. Previous IUD. Breech diagnosed in labour	VD	Died
8	ROM > 24 hrs. Prolonged 1st stage, fetal distress	Vacuum	Normal
9	Fetal distress, failed vacuum	CS	CP
10	Pre-eclampsia, deflexed head at vaginal exam, prolonged 2nd stage	NVD	Normal
11	Prolonged 2nd stage	Vacuum	Died
12	Prolonged 2nd stage (95 min)	Vacuum	Died
13	Prolonged 2nd stage (70 min), shoulder dystocia	Vacuum	Lost to follow-up
14	Maternal trauma with severe anaemia, fetal distress, 2-day delay to delivery	CS	Lost to follow-up
15	Poor maternal weight gain, referred for NST 1 week before delivery, not followed up	NVD	Normal
16	Prolonged 1st stage, MSL, face presentation. Two-hour delay to CS	CS	CP, microcephaly
17	ROM 28 h. Fetal distress	NVD	Lost to follow-up
18	P3G4. Previous SB and CS. ROM 12 days	NVD	Normal
19	Prolonged 1st stage, fetal distress	NVD	Lost to follow-up
20	APH, fetal distress at 8 cm dilatation	NVD	CP
21	Prolonged 2nd stage (120 min). Head impacted in vagina	CS	Normal
22	Chorioamnionitis, fetal distress	NVD	Normal

* Fetal distress is defined as the presence of late decelerations on cardiotocograph monitoring in labour.
IUD = intra-uterine death; MSL = meconium-stained liquor; NST = non-stress test; NVD = normal vaginal delivery; ROM = rupture of membranes; SB = stillbirth.

(approximately 3 000 per year) and elsewhere have not been included. Even if the CP rate were halved by these normal deliveries, the figure would still be several times higher than the figure quoted for developed countries.

It is known that neonates born with congenital sepsis have lower Apgar scores than normal, and some authors have suggested that intra-uterine exposure to maternal infection is associated with an increased incidence of CP.[13] The results from this audit do not support this hypothesis, and the contribution of culture-proven sepsis to the CP rate is not significant.

In their article Freeman and Nelson[5] propose four criteria that should be met before assuming that birth asphyxia is the cause of CP.

Firstly, there should be evidence of prolonged intrapartum asphyxia. The patients with CP in this audit all had an Apgar score ≤ 5 at 5 minutes, but unfortunately 10-minute Apgar scores and cord pH and base deficit results were not available.

Secondly, there should be signs of moderate or severe HIE in the neonatal period. Of the 10 CP children in this review, 7 were classified as HIE grade 2 and 2 as HIE grade 3; 1 was unclassified in the notes. In addition, 9 infants had seizures in the neonatal period, all had feeding problems because of a poor suck reflex, and none was discharged before 8 days of age, indicating significant neurological involvement.

Thirdly, the child's current neurological condition should be one that intrapartum asphyxia would explain. All the children we have labelled as abnormal have CP, a condition well described as resulting from birth asphyxia.

Finally, the CP child should have had a work-up to exclude other conditions that may account for the neurological signs. Although the work-up of CP children in general in developing countries is not as detailed as in developed countries, 8 of the 10 CP patients had had at least one cranial sonar, excluding a structural brain abnormality.

When reviewing the above results, the limitations of a retrospective analysis must be borne in mind. It is possible that some patients were missed and even that some cases of neonatal encephalopathy may have been due to structural brain abnormalities not apparent at birth. In addition, misclassifications of the grade of HIE probably occurred in some cases, as evidenced by one baby classified as HIE grade 3 but found to be neurologically normal at follow-up. Sixty-two per cent of the babies in the audit did not have a cranial ultrasound, but of the 10/16 babies with HIE grade 2 who had this investigation, none had a major structural brain abnormality. It is therefore unlikely that any such cases were truly missed.

The high birth asphyxia and CP rates must also be viewed in the context of an increasing number of deliveries taking place at Johannesburg Hospital. Where < 2 000 deliveries took place at the hospital in 1989, approximately four times that number occurred in 1997. The increased number of deliveries excludes referrals from Alexandra Clinic that were previously managed at Baragwanath Hospital. This increase in the number of deliveries and referrals has not been accompanied by an increase in obstetric and paediatric facilities or staff.

There has been a lot of criticism levelled at high CS rates, particularly in the private sector, both locally and overseas.[14,15]

Table IV. Comparison of asphyxiated neonates in different areas of South Africa

Year	Hospital	*N*	Mortality	Seizures	CP rate (/1 000)	CS rate (%)
1990 - 1991	King Edward VIII[8]	45	15/45 (33%)	31/45 (69%)	0.7	44
1991	Baragwanath[9]	212	55/212 (26%)	20/212 (9%)	0.84	NS
1992 - 1993	Baragwanath[10,11]	201	28/201 (26%)	57/201 (28%)	0.8	27
1989 - 1991	Tygerberg[12]	74	NS	NS	4.6*	53
1997	Johannesburg	48	6/48 (12.5%)	24/48 (50%)	1.15	29

* Represents birth asphyxia rate and not CP rate.
NS = not stated.

Table V. Neonatal outcome related to grade of HIE

HIE	*N*	Died	Follow-up	Handicap
Not stated	15	0	10	1 CP, 1 mild abn.
Grade 1	7	0	4	All normal
Grade 2	16	0	9	7 CP, 1 mild abn.
Grade 3	10	6	4	2 CP, 1 mild abn.
Total	48	6	27	10 CP

Abn. = abnormality.

There is concern about the increased risk of morbidity and mortality to the mother and the significantly increased costs in comparison with a normal vaginal delivery. To date, however, the concomitant birth asphyxia and CP rates have not featured prominently in the argument. In the review the CS rate in this group of seemingly low-risk pregnancies that developed complications during labour appears to be low in the face of unacceptably high rates of birth asphyxia and CP. The CS rate should not be considered in isolation, and birth asphyxia and CP rates should be included in any audit of perinatal care. A slightly higher CS rate may be justified if birth asphyxia can be prevented, particularly as most of the patients who developed complications were originally deemed to be low-risk cases. Although maternal risks are increased with CS it is a safe and easy procedure, with maternal risk of major morbidity in the order of 5%[16] and mortality of < 1%.[17] Of particular concern is the number of assisted vaginal deliveries being attempted by junior members of staff, who are not adhering to the prerequisites for instrument deliveries. It seems that inexperienced operators are attempting this procedure inappropriately, where a timely CS would provide a better outcome.

The whole question of improved obstetric management (not only CS) must be considered. Despite an attempt to regionalise the health services into three levels of care with proper referrals between the different levels, some babies in this survey were asphyxiated due to delay in transfer of mothers from level 1 or 2 to level 3. Another area of concern was the number of mothers with a previous history of stillbirth or intra-uterine death who continued to receive their antenatal care at a primary health care facility until the time of delivery. A further 15 babies with birth asphyxia, not included in the audit, were delivered at peripheral centres and only transferred to the Johannesburg Hospital nursery after the birth. Improved antenatal assessment and monitoring of labour would reduce this number by ensuring earlier transfer of complicated cases to a tertiary care centre for delivery. The need to enlighten staff members and provide primary health care facilities with management strategies is of paramount importance.

Obstetric management within our tertiary care centre is far from ideal. In addition to some of the preventable practices highlighted in Table III, it was notable that no fetal scalp pH monitoring was performed on any of the babies diagnosed as having fetal distress. This simple procedure could have alerted the obstetric staff to the need for an emergency CS in some cases.

An ongoing audit of babies born with birth asphyxia at Johannesburg Hospital indicates that the problems highlighted above are ongoing. A total of 22 asphyxiated neonates were delivered from 1 January to 31 March 1998, of whom 4 (18%) have died. A review of labour ward obstetric protocols is currently underway and it is hoped that some improvement may ensue.

CONCLUSION

This retrospective study of babies with birth asphyxia highlights the differences between patient populations in developed and developing countries. It would appear that many local cases of birth asphyxia are preventable. Our cash-strapped health services can ill afford the burden of asphyxiated neonates and CP children, and every effort should be made to prevent such cases.

This audit also raises the issue that monitoring of maternal care is incomplete unless CS and assisted delivery rates are considered in the context of birth asphyxia and CP rates.

It is hoped that this retrospective study will stimulate prospective evaluation of the important problem of birth asphyxia.

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