The Central African Journal of Medicine

Supplementary Issue to 1992 Volume 38,
1991 University of Zimbabwe Annual Research Day

Vol. 38, No. 7, July 1992

The causes of perinatal mortality in Bulawayo, Zimbabwe

C G A AIKEN

SUMMARY

The causes of all perinatal deaths at Mpilo Maternity Hospital were investigated over a 12-month period, during which there were a total of 466 stillbirths and 379 neonatal deaths, with a perinatal mortality rate of 36,0/1000 births in Bulawayo, Zimbabwe.

The causes of death were in order of importance; congenital syphilis (20,5 pc), birth asphyxia (18,8 pc), unexplained stillbirths (11,8 pc), hyaline membrane disease (11,5 pc) neonatal septicaemia (10,8 pc), congenital malformations (7,7 pc), pregnancy induced hypertension (5,4 pc), placental abruption (4,9 pc), congenital infection (2,2 pc) and other causes (6,4 pc).

Eleven pc of mothers booking in antenatal clinics had positive syphilis serology. Most were successfully treated. But over 400 mothers with early syphilis escaped treatment usually because they booked late or failed to book at all at antenatal clinics (74 pc) and occasionally because they had false negative results or were infected after early booking (27 pc).

They delivered 101 stillbirths, most of whom died prematurely before labour and often had abdominal distension. There were 72 neonatal deaths, most of whom were preterm babies with respiratory distress and often hepatosplenomegaly. One half of the deaths from asphyxia were caused by prolonged obstructed labour and one quarter by prolapsed cord, stuck head in breech delivery and retained second twin.

The incidence of both early and late onset neonatal septicaemia was very high with *Group B Streptococci, Kiebsiella* and *Staphlococcus aureus* the predominant pathogens.

Improved antenatal, intrapartum and neonatal care could substantially reduce the perinatal mortality rate by preventing congenital syphilis and birth asphyxia and by treating hyaline membrane disease and neonatal septicaemia.

INTRODUCTION

Perinatal mortality rates in Central African countries are much greater than rates below 10/1 000 births now being achieved in industrialised countries and are associated with high maternal mortality rates and high rates of handicap in surviving infants. Attempts to reduce perinatal mortality by improvements in antenatal, intrapartum and neonatal care would therefore be most desirable but for this to be effective the mortality rate from causes amenable to such treatment must first be known.

Most perinatal deaths are of low birth weight and in general, countries with high perinatal mortality rates have high rates of low birth weight. The prevalence of low birth weight on Central Africa is 12 to 18 pc, compared with four to seven pc in Europe

Correspndence:
Department of Paediatrics
Wellington Hospital
P.O. Box 7343
Wellington South
New Zealand

and North America. This reflects both high rates of intrauterine growth retardation, caused mainly by poor nutrition and malaria,[1] and high rates of preterm delivery, caused to some extent by syphilis,[2] gonorrhoea[3] and possibly HIV infection,[4] all of which are common in Central Africa.

Untreated early syphilis is a well-known cause of perinatal mortality, which can be eliminated by early antenatal screening, but is still common in Central Africa. In Zambia it was estimated to cause 30 to 40 pc of the stillbirths and 15 pc of the neonatal deaths in 1980.[5,6]

Neonatal tetanus still occurs in home deliveries but with appropriate cord care is rare in hospital and clinic deliveries. However, other bacterial infections remain an important problem.

In the 1970s in Zaria, Nigeria, common obstetric complications including prolonged obstructed labour, uterine rupture, malpresentations, cord prolapse and retained second twin occurred in 12 pc of deliveries and were associated with perinatal mortality rates ranging from 300 to 800/1 000 births.[7] Birth asphyxia caused by these complications accounted for perinatal mortality rates of 15/1 000 birth in Nigeria[8] and Ethiopia,[9] but only 3/1 000 births in South Africa,[9] a tribute to better intrapartum care resulting especially from the use of the partogram.[10]

Hyaline membrane disease is an important cause of neonatal mortality but may be relatively uncommon in Central Africa.[11]

MATERIALS AND METHODS

Over a 12 month period the causes of all perinatal deaths at Mpilo Maternity Hospital in Bulawayo, Zimbabwe, were evaluated and classified using a system designed to identify these preventable or treatable causes of perinatal mortality.

Population and health facilities

Bulawayo is situated at an altitude of 1 350m with an estimated population of 793 000. Malaria rarely occurs in the city but is common in the surrounding lower lying rural areas. Most of the population reside in the high density Western Suburbs, where public obstetric facilities consist of 13 City Council Clinics, staffed by midwives, and Mpilo Maternity Hospital, a teaching hospital, which takes referrals from the clinics and from district hospitals outside the city. Mpilo Maternity Hospital has only one operating theatre and no obstetric monitoring equipment. The neonatal unit has six incubators and no neonatal monitoring equipment. A few babies with medical and surgical conditions are ventilated in the adult intensive care unit of the main hospital.

Perinatal mortality

From 1st September 1989 to 31st August 1990, detailed information was recorded on all stillbirths and neonatal deaths at Mpilo Maternity Hospital. Stillbirths were defined as foetal deaths delivered at or after 28 weeks gestation because it was impossible to analyse the large number of second trimester abortions in the gynaecological wards. Neonatal deaths were defined as babies dying before discharge from hospital and therefore included a few babies dying after 28 days.

Over the 12 month study period there were a total of 10 881 deliveries, 3 272 admissions to the neonatal unit, 466 stillbirths and 379 neonatal deaths. Fifty five pc of the stillbirths were fresh and 45 pc were macerated. Seventy two pc of the neonatal deaths occurred during the first 48 hours, 16 pc between 48 and seven days, nine pc between eight and 28 days and three pc after 28 days. Fifty three percent of stillbirths and 56 pc of neonatal deaths were male.

The birth weights of the stillbirths and neonatal deaths are shown in Figure I.

Forty two pc of stillbirths weighed 2,500 or more whereas 51 pc of neonatal deaths weighed less than 1 500g. As shown in Table I, the mortality rate of admissions to the neonatal unit increased dramatically at birth weights below 1 500g.

Allowing for the duration without foetal movements 27 pc of stillbirths were small for gestational age (SGA) with birth weights below the fifth percentile for gestational age in Zimbabwe,[12] compared with 13 pc of neonatal deaths ($p<0,001$). This indicates that intrauterine growth retardation is more commonly associated with death before than after delivery.

Five percent of deliveries were the products of multiple pregnancies. Their perinatal mortality rate was twice as high as singleton deliveries mainly because of a five times greater mortality rate from

Figure 1: Birth weight distribution of stillbirths and neonatal deaths

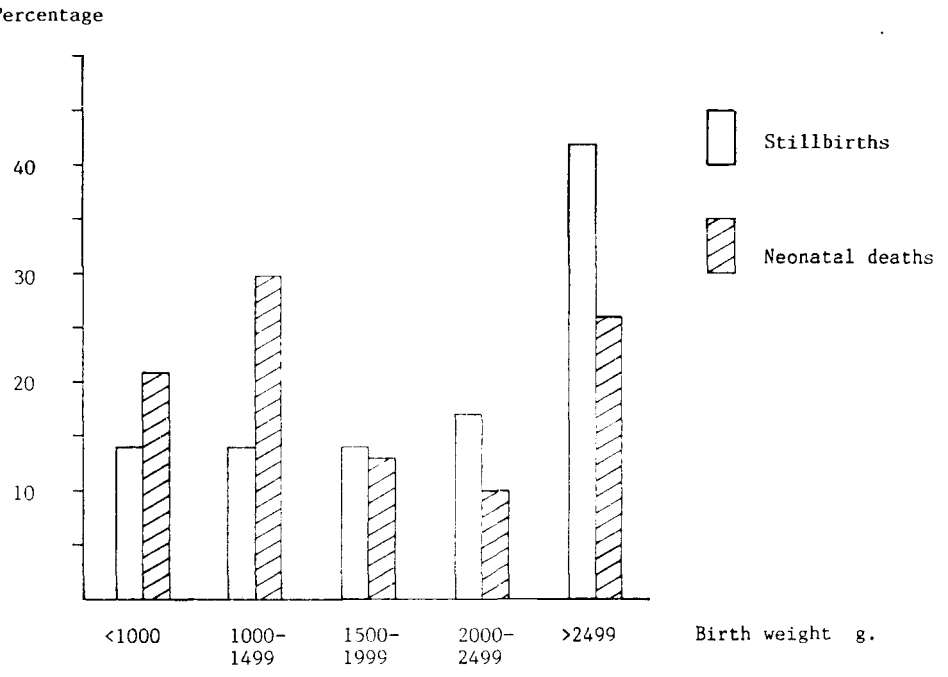


Table I: The mortality rate of admissions to the neonatal unit according to the birth weight

Birth weight g	Neonatal admissions n	pc	Neonatal deaths n	pc	Mortality pc
<1 000	97	(3)	81	(22)	84
1 000–1 249	117	(4)	59	(16)	50
1 250–1 499	130	(4)	46	(13)	35
1 500–1 999	470	(14)	48	(13)	10
2 000–2 499	734	(22)	37	(10)	5
2 499	1 724	(53)	96	(26)	6
TOTAL	3 272		367		

Deaths after 28 days are excluded.

hyaline membrane disease following premature delivery.

Half of the mothers were below 25 years old, as shown in Table II, which also gives the stillbirth and neonatal mortality rate for each age group.The stillbirth rate was weakly associated with maternal age, the highest rates being found between 25 and 39 years. The neonatal mortality rate was strongly associated with maternal age, the highest rates being found surprisingly below 25 years and the lowest rate above 39 years.

The mothers of 78 pc of the stillbirths had received some antenatal care compared with only 59 pc of the neonatal deaths. Seventeen pc of neonatal admissions had no antenatal care. Their mortality rate was three-and-half times greater than those receiving antenatal care, largely because they were more often of very low birth weight. Sixty-four pc of neonatal deaths below 1 500g had unbooked mothers compared with only 18 pc of those weighing 1 500g or more (p<0,001). This simply reflects the common habit in Zimbabwe to book late, often in the third

Table II: Maternal age of deliveries, stillbirths and neonatal deaths

Maternal age years	Deliveries n	Deliveries pc	Stillbirths n	Stillbirths Rate /1 000	Neonatal deaths n	Neonatal deaths Rate /1 000	Perinatal deaths n	Perinatal deaths Rate /1 000
<20	2348	22.2	74	31.5	76	32.4	150	63.9
20–24	2 991	28.2	128	42.8	103	34.4	231	77.2
25–29	2 120	20.0	106	50.0	54	25.5	160	75.5
30–34	1 628	15.4	77	47.3	32	19.7	109	67.0
35–39	1 077	10.2	53	49.2	21	19.5	74	68.7
>39	425	4.0	16	37.6	6	14.1	22	51.8
Total	10 589	100.0	454	42.9	292	27.6	746	70.5
X^2				11.43		17.12		7.07
p				<0.05		<0,005		NS

The mother's age was counted once only in multiple pregnancies and in a few cases was not known. No allowance for the age at death was made in calculating neonatal and perinatal mortality rates.

trimester; very premature babies being born before their mothers had planned to attend an antenatal clinic. There is no evidence that antenatal care, per se, prevents preterm delivery or intrauterine growth retardation.[1]

During the survey 8 834 deliveries, 18 stillbirths and three neonatal deaths were recorded at the Bulawayo City Council Clinics. This very low perinatal mortality rate was achieved by transferring all complicated cases to Mpilo Maternity Hospital. A further 1 918 home deliveries including 35 stillbirths were recorded. Combining these figures and excluding transfers from outside Bulawayo, which accounted for 16 pc of the stillbirths and eight pc of the neonatal deaths at Mpilo Maternity Hospital, gives a perinatal mortality rate (stillbirths plus neonatal deaths in the first seven days) of 36,0/1 000 births for mothers in Bulawayo. These mothers account for 80 pc of the deliveries in Bulawayo.

Causes of stillbirths and neonatal deaths

The causes of the stillbirths and neonatal deaths were determined from the mothers' history and examination antenatally and intrapartum, from examination of the babies before and after death and from the results of investigations, principally for infection.

Autopsies were performed on 90 pc of neonatal deaths weighing 1 000g or more but on only 21 pc weighing less than 1 000g and were seldom performed on stillbirths. Lung histology was examined with haematoxylin and eosin staining in all cases with respiratory distress.

Babies with respiratory distress were given penicillin and gentamicin after first obtaining blood for culture and syphilis serology using the rapid plasma reagin (RPR) and *Treponema pallidum* haemagglutination (TPHA) tests. These investigations were also performed after death on blood obtained from the heart, unless an obvious cause of death was apparent and RPR and TPHA tests were obtained from the mothers of all unexplained stillbirths.

From the clinical and pathological findings and the results of investigations, the stillbirths were classified according to the foetal or maternal condition resulting in death as shown in Table III. The neonatal deaths were classified according to the neonatal condition resulting in death as shown in Table IV.

The causes of death were considered in the order given in the Tables and only the first established diagnosis was recorded.

The Tables also give the birth weight distributions and the proportions and the proportions of low birth weight (<2 500g), preterm (<37 weeks) and SGA (<5th percentile) infants with each diagnosis, as well as the proportions of fresh or macerated stillbirths and the age of death in neonatal deaths.

Table III: Causes of stillbirths

				Birth weight g							
	n	Fresh n	Mac n	<1 000 n	1 000 – 1 499 n	1 500 –1999 n	2 000 –2 499 n	>2 499 n	LBW pc	PRE pc	SGA pc
Congenital Malformations	34	25	9	3	7	3	8	13	62	62	35
Placental abruption	41	41	0	6	5	5	11	14	66	76	15
Pregnancy induced hypertension	46	17	29	14	10	12	4	6	87	83	54
Birth asphyxia	111	111	0	2	3	8	13	85	23	21	9
Congenital syphilis	101	25	76	22	19	15	25	20	80	83	31
Other causes	33	18	15	0	4	6	4	19	42	27	27
Unexplained Stillbirths	100	20	80	18	15	16	13	38	62	52	33
TOTAL	466	257	209	65	63	65	78	195	58	55	27

***Mac** = Macerated*
***LBW** = Low birth weight*
***PRE** = Preterm*
***SGA** = Small for gestational age*

Table IV: Causes of neonatal deaths

		Age hours		Birth weight g							
	n	<48 n	>48 n	<1 000 n	1 000 –1 499 n	1 500 –1 999 n	2 000 –2 499 n	>2 499 n	LBW pc	PRE pc	SGA pc
Congenital malformations	31	18	13	0	3	4	7	17	45	42	32
Birth asphyxia	48	44	4	0	1	1	7	39	19	17	8
Congenital syphilis	72	50	22	14	26	15	5	12	83	86	12
Early onset septicaemia	39	39	0	5	14	5	6	9	77	77	8
Late onset septicaemia	42	0	42	12	21	7	2	0	100	98	14
Meconium aspiration syndrome	12	12	0	0	0	0	1	11	8	0	17
Hyaline membrane disease	97	93	4	44	36	11	5	1	99	100	4
Other causes	38	17	21	6	11	7	6	8	79	58	32
TOTAL	379	273	106	81	112	50	39	97	74	72	13

***LBW** = Low birth weight*
***PRE** = Preterm*
***SGA** = Small for gestational age*

Table V: Serious congenital malformations

		SB	NND	LIVE	TOTAL
Central Nervous	Anencephaly	10	0	0	10
	Spina bifida	6	3	8	17
	Hydrocephaly	3	0	2	5
Cardiovascular		0	6	3	9
Alimentary	Cleft lip and/or palate	1	0	5	6
	Oesophageal atresia	0	1	1	2
	Duodenal atresia	0	1	1	2
	Jejunal atresia	0	1	0	1
	Anorectal atresia	0	0	3	3
	Diaphragmatic hernia	0	5	0	5
	Gastroschisis	1	0	0	1
Multiple		9	5	0	14
Chromosome	Down's syndrome	0	4	12	16
Syndromes	Edwards' syndrome	0	3	0	3
	Patau's syndrome	0	1	0	1
	Thanatophoric dysplasia	1	1	0	2
Teratoma	Sacrococcygeal	2	0	0	2
Conjoined Twins		1	0	0	1
TOTAL		34	31	35	100

SB — Stillbirths
NND — Neonatal deaths
LIVE — Surviving infants discharged from the neonatal unit
Notes giving details of the cardiovascular malformations, multiple malformations and other congenital malformations not included in the Table are available on request.

Notes

Cardiovascular:

NND — 1 Hypoplastic left heart syndrome
1 Transposition of great arteries
1 Total anomalous pulmonary venous drainage
1 Cardiomyopathy
2 Heart failure with cyanosis and murmur but no autopsy

LIVE — 2 Ventricular septal defect
1 Pulmonary stenosis

Multiple:

SB — 1 Hydrocephaly, anophalmia, cleft lip and palate, anorectal atresia
1 Microcephaly, prominent eyes, absent nose, sacral hairy patch
1 Hydroencephaly, proboscis above partially fused eyes
1 Cleft lip and palate, distended abdomen, syndactyly
1 Omphalocele, anopthalmia, club feet, polydactyly, renal cystic dysplasia
1 Omphalocele, hydrocephalus, short limbs
1 Hydronephrosis caused by enlarged cystic uterus, polydactyly
1 Absent femora, 11 ribs, preauricular skin tags
1 Cystic hygroma, polydactyly, contractures

NND — 1 Hydroencephaly, prominent eyes, central cleft lip and palate
1 Anorectal atresia, exomphalos, hydronephrosis, undescended testis, VSD, short limbs, polydactyly, thin skin with streaks of pigmentation
1 Polycystic kidneys, VSD, genu recurvantum
1 Pierre-Robin syndrome, absent arms, atresia of external audatory meati
1 Hypotonia, wide sutures, parietal bossing, micropthalmia, absent nails, rocker bottom feet, single palmar creases, undescended testis

Congenital malformations not included in the Table:

LIVE — 3 Microcephaly
1 Encephalocele
1 Anopthalmia
1 Cataract
1 Coanal stenosis
2 Exomphalos
1 Congenital adrenal hyperplasia
8 Club feet
4 Genu recurvatum
1 Arthrogryposis
2 Radial/thumb defects
1 Hemihypertrophy
2 Beckwith-Wiedemann syndrome
2 Collodion skin

Table VI: Causes of birth asphyxia

	SB	NND	HIE	TOTAL		PARITY		DELIVERY					
	n	n	n	n	pc	P	M	NVD	BR	C/S	VAC	SYM	DES
Obstructed labour					49,5								
Prolonged 1st stage	18	12	6	36		26	10	12	1	19	2	0	2
Prolonged 1st + 2nd stage	11	8	3	22		15	7	9	0	3	9	1	0
Prolonged 2nd stage	10	6	11	27		15	12	14	0	5	7	0	1
Ruptured uterus	11	0	0	11		0	11	0	0	11	0	0	0
Stuck head in breech	6	5	3	14	7,2	4	10	0	14	0	0	0	0
Proplapsed cord	25	1	0	26	13,4	8	18	12	2	8	4	0	0
Retained 2nd twin	8	2	1	11	5.7	2	9	7	0	4	0	0	0
Other causes					11,9								
Shoulder dystocia	2	0	1	3		0	3	3	0	0	0	0	0
Placental abruption	0	3	1	4		0	4	3	0	1	0	0	0
Cord knot	2	0	0	2		1	1	2	0	0	0	0	0
Aminiocentesis	0	1	0	1		0	1	0	0	1	0	0	0
Post term >42 weeks	4	3	0	7		1	6	6	0	1	0	0	0
SGA <5th centile	4	1	1	6		3	3	6	0	0	0	0	0
Unexplained	10	6	8	24	12,4	9	15	21	3	0	0	0	0
TOTAL	111	48	35	194	100,1	84	110	95	20	53	22	1	3

SB — Stillbirths
NND — Neonatal deaths
HIE — Surviving infants with moderate hypoxic ischaemic encephalopathy
P — Primigravidae
M — Multigravidae
NVD — Normal vaginal delivery
BR — Breech delivery
C/S — Caesarian section delivery
VAC — Vacuum extraction
SYM — Symphisiotomy with vacuum extraction
DES — Destructive procedure

Definitions and Notes
Prolonged first stage was defined as slow rate of cervical dilation during the active phase such that the rate plotted on a partogram crosses the four hour action line, which is a line drawn four hours after the cervix reaches three cm dilation with a slope of one cm/hour. The first stage lasted for more than 24 hours in 33 pc of cases. Fifty two cases were caused by cephalopelvic disproportion with marked caput and moulding, one was an occipito-posterior presentation, one was a face presentation and four were transverse lies.
Prolonged second stage was defined as a second stage lasting more than one hour. The second stage lasted more than two hours in 55 pc of cases. Forty seven cases were caused by cephalopelvic disproportion and two were occipito-posterior presentations.
Five of the 11 cases of ruptured uterus occurred through a previous Caesarian section scar.
Stuck head in breech delivery was defined as a more than 10 minute delay in delivering the head in a breech delivery. Retained second twin was defined as retention of the second twin for more than one hour.

Stillbirths 1. Congenital malformations: Thirty four stillbirths had serious congenital malformations, summarised in Table V. Central nervous system malformations, either alone or in combination with other abnormalities, accounted for 68 pc.

2. Placental abruption: Forty one fresh stillbirths followed severe placental abruption, diagnosed clinically. Twenty eight of their mothers were given blood and fresh frozen plasma for shock and prolonged clotting times and 10 had associated hypertension. During the survey only 10 live babies were delivered by Caesarian section following placental abruption, four of whom subsequently died.

3. Pregnancy induced hypertension: Forty six stillbirths were attributed to severe pregnancy induced hypertension. All their mothers had proteinuria, 29 had blood pressures of 160–250/120–160mm Hg, 17 had blood pressures of 140–190/100–110mm Hg and five developed eclampsia. Pregnancy induced hypertension is common, being found in 9,4 pc of admissions, but most mothers have no proteiunuria with low risk to the foetus. During the survey 64 live babies were delivered by Cesarean section because of severe hypertension with proteinuria or eclampsia, seven of whom subsequently died.

4. Birth asphyxia: This was considered to be the cause if death occurred during labour, excluding cases with no foetal heart on admission in labour. One hundred and eleven cases were identified. As shown in Table VI, prolonged obstructed labour, ruptured uterus, prolapsed cord, stuck head in breech delivery and shoulder dystocia caused 91 (82 pc) of the cases; definitions of these complications being given in Table VI.

Table VII: Syphilis serology results in stillbirths, neonatal deaths and surviving infants discharged from the neonatal unit

	n	No result pc	RPR+ treated pc	RPR- pc	RPR- TPHA- pc	RPR+ TPHA- pc	RPR- TPHA+ pc	RPR+ TPHA+ pc
SB and NND								
Malformation	65	4,6	1,5	27,7	47,7	4,6	6,2	7,7
Abruption	41	19,5	4,9	14,6	46,3	2,4	7,3	4,9
PIH	46	17,4	0,0	13,0	56,5	4,3	4,3	4,3
Asphyxia	159	7,5	3,1	21,4	52,2	7,5	4,4	3,8
Survivors	2 893	5,3	6,6	67,1	14,3	3,4	0,5	2,7
Remaining SB	234	2,1	0,9	6,4	43,2	3,0	1,7	42,7
Remaining NND	300	1,7	0,7	8,3	59,7	4,3	2,3	23,0

***RPR** = Rapid plasma reagin test (Reditest, Biokit)*
***TPHA** = Treponema pallidium haemagglutination test (Wellcosyph HA, Wellcome)*
***SB** = Stillbirths*
***NND** = Neonatal deaths*
***PIH** = Pregnancy induced hypertension*
The proportion of RPR+ and TPHA+ infants were similar in perinatal deaths with malformations, abruption/PIH and asphyxia and in surviving infants (X2 7,35 df 3 p>0,1). Compared with these infants, the proportion of RPR+ and TPHA+ infants was significantly greater in the remaining stillbirths (X2 623,33 p<0,001) and remaining neonatal deaths (X2 236,25 p<0,001).

5. Congenital syphilis: The mothers of 100 of the remaining 234 (43 pc) stillbirths were TPHA and RPR positive but untreated; a much higher proportion than three to eight pc found in death from the above conditions and surviving infants, as shown in Table VII. This indicates that syphilis was very likely to be the cause of death in these 100 stillbirths. One further stillbirth, whose mother had an untreated primary chancre and was TPHA positive but RPR negative, was ascribed to syphilis, giving a total of 101 cases.

Most of these mothers were asymptomatic, presumably with early latent syphilis, but a few features of secondary syphilis. Eighty pc of these stillbirths were of low birth weight and 31 pc were SGA. Seventy five percent were macerated, usually markedly so, 52 pc had abdominal distension and five pc had hydrops, all well recognized features of congenital syphilis.[13] Sixty pc of these mothers had no RPR test antenatally, 28 pc were documented as RPR negative on booking and 12 pc were RPR positive but untreated or had begun treatment less than two weeks before delivery.

6. Other causes. a) Infection. Thirteen fresh stillbirths were thought to be due to infections other than syphilis. Seven died after prolonged rupture of membranes for more than 48 hours with features of amnionitis. Four died with maternal fever thought to be due to amnionitis in two and malaria in two cases. In one blood culture from the heart grew *E. coli*. One stillbirth was SGA, weighing 1 660g at 38 weeks, with hepatosplenomegaly and was HIV-1 positive.

b) Post maturity. In eight stillbirths foetal movements stopped before the onset of labour at 42 to 44 weeks gestation, confirmed by examination, presumably because of declining placental function after 42 weeks.

c) Placenta praevia. Preterm stillbirths followed antepartum haemorrhage from placenta confirmed at Caesarian section in two and by utrasound in one case.

d) Diabetes. Three macerated stillbirths were associated with poorly controlled or undiagnosed diabetes, which also contributed to the two macrosomic stillbirths dying from asphyxia caused by shoulder dytocia.

e) Hydrops. Two stillbirths were hydropic without evidence of syphilis or rhesus iso-immunization. Rhesus disease caused no stillbirths or neonatal deaths during the survey and affected only two surviving infants.

f) Shock. Two mothers became shocked with loss of foetal movements caused by severe diarrhoea and

Table VIII: Lung histology in neonatal deaths with respiratory distress

		Hyaline membrane disease		Congenital syphilis		Early neonatal septicaemia		Congenital pneumonia		Meconium aspiration syndrome	
Number		46		42		26		5		11	
Birth weight	g. Mean (SD)	1 387	(382)	1 786	(824)	1 804	(805)	2 628	(347)	3 062	(401)
Gestational age	wk. Mean (SD)	29,8	(2,1)	31,8	(4,0)	32,1	(4,6)	37,0	(3,5)	40,3	(1,2)
Age at death	hr. Mean (SD)	13,2	(13,8)	23,4	(23,9)	17,6	(15,3)	23,0	(18,0)	17,4	(12,1)
Atelectasis	Generalised (pc)	100		86		54		0		18	
	Focal (pc)	0		10		8		0		55	
Hyaline membranes	Many (pc)	76		24		27		0		18	
	Few (pc)	2		5		23		0		0	
Excess inflammatory cells (pc)		0		29		15		0		36	
Pneumonia	(pc)	0		2		31		100		36	
Intralveolar haemorrhage pc		17		21		23		20		18	
Alveolar oedema	(pc)	0		2		0		0		0	
Meconium aspiration (pc)		0		5		0		0		100	

vomiting in one and by a large haematemesis in the other.

g) Cord knot. Two macerated stillbirths had tight cord knots.

7. Unexplained: The remaining 100 stillbirths were unexplained. Sixty two percent were of low birth weight and 33 pc were SGA, suggesting that some may have been caused by placental insufficiency or intrauterine infections. Ten percent were fresh stillbirths weighing more than 2 500g with no foetal heart head on admission in established labour, perhaps because of asphyxia occurring prior to admission. One set of twins could have died from twin-twin transfusion. Kleihauer tests were not performed to detect foeto-maternal haemorrhage.

Neonatal deaths: 1. Congenital malformations: Thirty one neonatal deaths had serious congenital malformations. As shown in Table V, eight had chromosomal abnormalities, diagnosed clinically, eight had alimentary malformations and six had cardiovascular malformations.

2. Birth asphyxia: This was considered to be the cause in 48 infants, who died with signs of hypoxic ischaemic encephalopathy,[14] after delay in establishing spontaneous respiration. Eight never breathed despite resuscitation, 16 took longer than 30 minutes to breath, 32 had signs of severe encephalopathy and eight had signs initially of moderate encephalopathy with convulsions,but deteriorated and died from apnoea. As shown in Table VI, prolonged obstructed labour, stuck head in breech delivery, retained second twin and prolonged cord caused 34 (71 pc) of the cases. Table VI also gives the causes of asphyxia in 35 infants with moderate encephalopathy, who survived during the study.

3. Congenital syphilis: Sixty one of the remaining 300 (23 pc) neonatal deaths were TPHA and RPR positive without treatment in pregnancy. This is a significantly greater proportion than three to eight pc found in deaths from the above conditions and survivors, as shown in Table VII. Like stillbirths in congenital syphilis, 83 pc were of low birth weight but only 12 pc were SGA. Sixty two (90 pc) developed respiratory distress soon after birth, 32 had hepatomegaly, 22 had splenomegaly, one had hydrops and one had vesiculobullous skin lesions.

Fifty one deteriorated despite penicillin and died within 72 hours, lung histology being obtained in 42 cases. As shown in Table VIII, 86 pc had severe generalised atelectasis but unlike hyaline membrane only 29 pc had hyaline membrane (despite surviving longer) and 31 pc had an obvious acute inflammatory response.

No significant difference in lung histology was found between those with and without hepatosplenomegaly, which is not a feature of hyaline membrane disease. A further difference is that infants dying from respiratory distress with syphilis had significantly greater birth weights than those dying from hyaline membrane disease ($p<0,001$). Seven of these early deaths had positive blood cultures. The remaining 18 infants died after 72 hours with

Table IX: The proportion of infants in each birth weight category whose mothers had untreated early syphilis in pregnancy

Birth weight g	<1 000	1 000-1 499	1 500-1 999	2 000-2 499	>2 499
Total number of infants	162	310	535	812	1 919
Number tested	156	302	530	772	1 783
Number with syphilis	38	57	54	54	67
Proportion with syphilis (pc)	24,4	18,9	10,2	7,0	3,8
Stillbirths	23/61	21/56	16/60	27/73	26/180
Neonatal deaths	14/79	26/111	17/50	5/39	13/95
Surviving infants with signs	1/16	8/135	15/420	10/660	12/1 508
Surviving infants without signs	0/16	2/135	6/420	12/660	16/1 508

A mother was considered to have untreated early syphilis if she was TPHA +ve and either RPR +ve or delivered an infant with signs of congenital syphilis. Total number of infants is the sum of stillbirths, neonatal deaths and surviving infants discharged from the neonatal unit. In the second part of the Table, the figures give the number of infants with syphilis/number tested.

Table X: The perinatal mortality rate from untreated early syphilis in pregnancy

	BULAWAYO				PHILADELPHIA[15]			
	Untreated syphilis		No syphilis		Untreated syphilis		No syphilis	
	n	rate/1 000	n	rate/1 000	n	rate1 000/	n	rate/1 000
Deliveries	426		20 639		220		10 323	
Survivors	264		20 001		135		9 827	
Stillbirths	96	225	350	17	55	250	270	26
Neonatal deaths	66	155	288	14	30	136	226	22
Perinatal deaths	162	380	638	31	85	386	496	48

septicaemia (10), culture negative meningitis (two), conjugated hyperbilirubinaemia (five), a swollen hip with metaphyseal erosions (one) and heart failure from a patent ductus arteriosus (one).

Of the seven neonatal deaths who were TPHA positive but RPR negative and untreated, three died from respiratory distress with either hepatomegaly, atelectasis without hyaline membranes or congenital pneumonia. These three deaths were ascribed to congenital syphilis giving a total of 72 cases. Fifty eight percent of their mothers had no RPR test antenataly, 25 pc were documented as RPR negative on booking and 17 pc were RPR positive but had not begun treatment before delivery.

During the survey the RPR status of 95 pc of the neonatal admissions was known. TPHA tests were performed on untreated RPR positive babies and on babies with respiratory distress or signs of congenital syphilis. Seventy nine surviving infants were TPHA and RPR positive without completed treatment in pregnancy, giving a case fatality rate for neonatal admissions with untreated syphilis in pregnancy of 47 pc.

Unlike the infants that died,only 67 pc of the survivors were of low birth weight and nine were SGA. Thirty six had no signs of congenital syphilis, 30 had respiratory distress, 16 had hepatomegally, 13 had splenomegaly, 22 developed hyperbilirubinaemia with elevated conjugated bilirubin levels in nine, six developed early anaemia, three had vesiculobullous skin lesions, three nasal discharges, four had scepticaemia and one was microcephalic. Three surviving infants who were TPHA positive and RPR negative had signs of congenital syphilis; one had respiratory distress, hepatosplenomegaly and jaundice and two had conjugated hyperbiliru-

Table XI: Bacteria causing early and late onset neonatal septicaemia

	DIED		SURVIVED		
	Early	Late	Early	Late	Mortality pc
Streptococcus group B	11	4	19	4	39
group D	4	1	3	3	45
group A	1	1	1	1	
Pneumonia	0	0	1	0	
Staphylococcus aureus	3	4	4	9	35
epidermidis	1	4	?*	?*	
Klebsiella	10	13#	2	2	85
Escherichia coli	3	2	3	1	56
Coliforms	0	5	1	1	
Proteus	0	2	0	0	
Pseudomonas	0	1	0	0	
Salmonella	0	0	0	1	
Haemophilus	1	0	0	0	
Bacillus	0	1	0	0	
TOTAL	34	38	34	22	

** Staph. epidermidis was grow in 32 blood cultures (26 <48 hours and 6 >48 hours) most of which were thought to be contaminants.*
 # Three blood cultures growing Klebsiella grew in addition Strep. group B, Strep. group D or E. coli.

binaemia and anaemia. None of the 198 neonatal admissions, who were RPR positive and had completed a course of penicillin before delivery, had signs of congenital syphilis.

As shown in Table IX, the proportion of tested infants with untreated syphilis in pregnancy increased from four pc in infants weighing 2 500g or more to 24 pc in those weighing less than 1 000g, in keeping with other evidence that syphilis causes preterm deliveries.[15] The high incidence of septicaemia in these babies appears to be due to their prematurity because the proportion of under 1 500g babies with septicaemia was similar in those with syphilis (18 pc) and those without syphilis (21 pc).

This survey should have detected almost all mothers with untreated syphilis delivering stillbirths, neonatal deaths and surviving preterm or sick infants. What is not known is the number of infected mothers delivering healthy term babies. This can be estimated from the data given in Table IX, which shows that 16 out of 1 508 surviving infants weighing 2 500g or more with no signs of congenital syphilis had mothers with untreated syphilis. Using this proportion, 182 of the 17 185 healthy infants not admitted to the neonatal unit could be expected to have mothers with untreated syphilis. By combining these figures, as shown in Table X, the perinatal mortality rate from untreated syphilis was estimated to be 380/1 000 births, very similar to the rate found in Philadelphia in the 1940's,[15] with about 60 pc of the deaths being stillbirths and 40 pc neonatal deaths.

4. Neonatal septicaemia: Eighty-one neonatal deaths were caused by septicaemia and were divided into two groups according to their age at deaths. a) Early onset neonatal septicaemia. Thirty-nine died in the first 48 hours with respiratory distresss. Thirty-four had positive blood cultures, with bacteria responsible shown in Table XI. Lung histology, obtained from 26, showed severe generalised atelectasis in 55 pc, hyaline membranes in 50 pc and an obvious acute inflammatory response in 46 pc (Table VIII). Five had negative blood cultures but lung histology was typical of congenital pneumonia. Forty-nine percent of the whole group weighed less than 1 500g.

b) Late onset neonatal specticaemia. Forty-two died after the first 48 hours from a rapidly progressive illness presenting with lethargy, vomiting or apnoeic attacks, but seldom with respiratory distress, and sometimes accompanied by jaundice, pulmonary or gastrointestinal haemorrhage. Thirty-eight had positive blood cultures, with the bacteria responsible shown in Table XI. Four had negative blood cultures but typical features of septicaemia.

Table XII: Neonatal deaths thought to be caused by congenital infections other than syphilis and early septicaemia

No	Age day	BW g	GA wk	Clinical features	Pathological features	HIV-1
1	10	1 980	32	RD Anaemia Jaundice	Congenital tuberculosis	-ve
2	16	1 380	29	RD Skin vesicles	Massive hepatic necrosis	-ve
3	50	1 800	37	SGA HS Jaundice	Neonatal hepatitis syndrome	-ve
4	3	1 460	40	SGA RD H/S Talipes	Hepatitis Histiocytic pneumonia	-ve
5	1	1 600	34	SGA RD H/S Pet	Focal atelectasis Horseshoe kidney	-ve
6	2	1 740	36	SGA RD H/S Pet	No autopsy	nil
7	13	2 200	38	SGA RD H/S Pet Jaundice	Hepatitis Septicaemia	+ve
8	2	1 190	39	SGA RD H/S Pet Micropth	No autopsy	+ve
9	5	1 540	35	SGA RD H/S Jaundice	Congenital pneumonia	+ve
10	1	2 160	38	SGA RD H+ Fits	Congenital pneumonia	+ve
11	2	1 640	37	SGA RD	Generalised atelectasis Meconium	+ve
12	1	1 380	33	SGA RD Anaemia	No autopsy	+ve
13	1	820	27	RD H+	No autopsy	+ve
14	5	3 000	41	RD Fits	Histiocytic pneumonia	+ve
15	1	3 800	40	RD Fits	No autopsy	+ve
16	135	1 100	28	H/S FTT	Multiple septiple septicaemia	+ve
17	78	1 120	28	CLD H/S FTT	Multiple septicaemia	+ve
18	31	2 260	40	SGA Cleft VSD FTT	Multiple septicaemia	+ve

BW — Birth weight; GA — Gestational age; SGA — Small for gestational age; RD — Respiratory distress; H/S — Hepato-splenomegaly; H+ — Hepatomegaly; Pet — Petechia; FTT — Failure to thrive; CLD — Chronic lung disease; Micropth — Micropthalmia; Cleft — Cleft palate; VSD — Ventricular septal defect

Unlike early onset septicaemia, 79 pc weighed less than 1 500g.

As shown in Table XI, 34 babies with early onset septicaemia survived with antibiotic therapy. The mortality rate varied from less than 40 pc for *Group B Streptococci* and *Staph. aureus* to 85 pc for *Klebsiella*. As expected the spectrum of bacteria responsible for early onset septicaemia (59 pc *Streptococci*, 28 pc gram negative enterobacteria and 12 pc *Staphylococci*) differed significantly from late onset septicaemia (47 pc gram negative enterobacteria, 28 pc *Staphylococci* and 23 pc *Streptococci*).

Early onset septicaemia was most commonly caused by *Group B Streptococci* acquired from the mother before delivery. Prolonged rupture of membranes for more than 24 hours occurred in 120 neonatal admissions of whom 14 (12 pc) developed early onset septicaemia. *Klebsiella* and *Staph. aureus* were most commonly responsible for late onset septicaemia and, typical of nosocomial infections, they were resistant to multiple antibiotics but were sensitive to ceftrioxone and cloxacillin respectively.

5. Meconium aspiration syndrome: Twelve neonatal deaths were caused by meconium aspiration syndrome. They were all 39 to 43 weeks gestation with meconium stained liquor, birth asphyxia and respiratory distress leading to death in the first 48 hours. Lung histology showed meconium in the airways with focal or diffuse atelectasis and often an acute inflammatory response (Table VIII).

Twenty eight infants, one of whom was ventilated, recovered from meconium aspiration syndrome, with respiratory distress lasting more than 48 hours following meconium aspiration and negative syphilis serology and blood cultures. This gave a case fatality rate of 30 pc. Term babies with syphilis or early onset septicaemia often closely resembled meconium aspiration syndrome clinically.

6. Hyaline membrane disease: Ninety seven neonatal deaths were attributed to hyaline membrane disease. They were all preterm infants with respiratory distress leading to death in the first 72 hours and negative syphilis serology and blood cultures. Forty five were less than 28 weeks gestation. Lung histology, obtained in 83 pc of those weighing 1 000g or more but seldom in under 1 000g infants,

Table XIII: Causes of perinatal mortality at Mpilo Maternity Hospital with the estimated perinatal mortality rate in Bulawayo.

Cause	No. of deaths	pc	Perinatal mortality rate /1 000 births
Congenital syphilis	173	20,5	7,4
Birth asphyxia	159	18,8	6,8
Unexplained stillbirths	100	11,8	4,2
Hyaline membrane disease	97	11,5	4,1
Neonatal septicaemia	91	10,8	3,9
Congenital malformations	65	7,7	2,8
Pregnancy induced hypertension	46	5,4	2,0
Placental abruption	41	4,9	1,7
Congenital infection	19	2,2	0,8
Other causes	54	6,4	2,3
TOTAL	845	100,0	36,0

The estimated perinatal mortality rate in Bulawayo excludes transfers from outside the city. Neonatal septicaemia includes 10 stillbirths with amnionitis or positive blood cultures.

showed severe generalised atelectasis in all, with hyaline membranes in 78 pc and no acute inflammatory response (Table VIII).

Sixty two infants over 1 000g, four of whom were ventilated, recovered from hyaline membrane disease with respiratory distress lasting more than 48 hours and negative syphilis serology and blood cultures giving a case fatality rate in infants above 1 000g of 47 pc.

7. Other causes: a) Congenital infections. Eighteen neonatal deaths were thought to be due to congenital infections other than syphilis and early onset septicaemia, the diagnosis being on clinical and laboratory features, as shown for each case in Table XII.

Case one had tuberculosis with miliary foci of caseous necrosis in the lung and liver containing acid fast bacilli and few inflammatory cells. Case two probably had *Herpes simplex* infection with a vesicular skin rash, micro-abscesses in the lung and massive hepatic necrosis. Cases 16, 17 and 18 had AIDS with failure to thrive, multiple episodes of septicaemia and positive HIV serology. Nine of the remaining 13 cases were HIV positive, a much higher proportion than in neonatal deaths from other causes, suggesting that HIV infection was in some way responsible for these deaths.[16]

b) Birth trauma. Nine babies died from birth trauma producing subdural haemorrhage in three, a large subarachnoid haemorrhage in one, subgaleal haemorrhage in two (two infants survived), ruptured livers in two very low birth weight infants and pulmonary haemorrhage in a bruised retained second twin.

c) Extreme prematurity. Five babies weighed less than 1 000 g. One died from severe hypothermia. Heart failure from a patent ductus arteriosus caused death in one, air embolus from a syringe pump in one and apnoea possibly from prematurely in two, although blood cultures were unfortunately not taken to exclude septicaemia, a much commoner cause of death.

d) Kernicterus. Two babies weighing 1 650g and 1 120g died from kernicterus, with serum bilirubin levels of 795umol/L and 44umol/L, caused by ABO incompatibility and severe bruising respectively. During survey only one exchange transfusion was performed.

e) Miscellaneous. Four babies died one each from perforation of the terminal ileum, milk aspiration following over feeding, dehydration from diarrhoea and vomiting, and post haemorrhagic hydrocephalus.

DISCUSSION

The causes of perinatal mortality at Mpilo Maternity Hospital and the estimated perinatal mortality rate in Bulawayo from each cause are summarised in Table XIII. The major causes were in order of importance congenital syphilis, birth asphyxia, hyaline membrane disease, neonatal septicaemia, congenital

malformations, pregnancy induced hypertension and placental abruption, which together accounted for 80 pc of the deaths.

1. Congenial syphilis: Congenital syphilis accounted for 20 pc of the deaths and produced a perinatal mortality rate of 7,4/1 000 births. During the survey, 2 243 out of 20 309 (11 pc) mothers booking in antenatal clinics in Bulawayo were RPR positive, similar to rates of five to 15 pc found elsewhere in Central Africa.[2] These mothers and their partners should have been treated with three doses of benzathine penicillin 2,4 megaunits at weekly intervals. Provided treatment was started more than three weeks before delivery, the outcome for the baby was good with no signs of congenital syphilis at birth.

However more than 400 mothers with early syphilis escaped treatment, usually because they failed to book or booked late in the third trimester and the outcome in their babies was very poor. Around 23 pc had stillbirths and 15 had neonatal deaths, very similar to the outcome found in mothers with untreated early syphilis in Philadelphia during the 1940's.[15] Nearly half their babies were of low birth weight and one sixth of the survivors had signs of congenital syphilis in the newborn period. Fifty pc of deaths were delivered at 24 to 31 weeks, 37 pc at 32 to 37 weeks and 13 pc at 38 to 42 weeks gestation.

This indicates that early booking preferably before 20 weeks is essential if perinatal deaths from syphilis are to be prevented. Treatment at around 20 weeks appears to produce the best results,[15] reinfection becoming a problem with earlier treatment. Unfortunately mothers in Zimbabwe tend to book late, often in the third trimester, too late to prevent many of the perinatal deaths.

Many reasons are given why mothers book late or fail to book at all and studies to demonstrate what factors are important would be helpful. Undoubtedly ignorance of why early booking is important is a major factor and a vigorous health education program explaining these reasons and persuading mothers to book at the latest when foetal movements are first felt is essential.

The RPR test is very cheap but not ideal as a screening test for syphilis in pregnancy. Between 0,3 and 0,6 pc of antenatal women have false positive RPR results.[17-19] More worrying is that in this survey the mothers of 27 pc of the perinatal deaths from syphilis were recorded as RPR negative on antenatal booking. This could be because they were infected after booking early.[6,20] This is likely in 42 pc who booked more than 60 days before delivery, but is unlikely in 29 pc who booked 30 to 60 days before delivery and very unlikely in 29 pc who booked less than 30 days before delivery. These women either had false negative RPR results perhaps because of the prozone phenomenon[21] or the wrong result was recorded in error.

One stillbirth, three neonatal deaths and three surviving infants with evidence of congenital syphilis were RPR negative but TPHA positive. The TPHA test appears to have a very low false negative rate, and in one study all antenatal women with early active syphilis, indicated by being FTA-ABS IgG and IgGM positive, were TPHA positive but 19 pc were RPR negative.[17]

Most of the neonatal deaths from syphilis were preterm appropriate for gestational age infants with respiratory distress and uniform atelectasis on lung histology, resembling hyaline membrane disease, but differing in generally being larger babies, often with hepatosplenomegaly but seldom with hyaline membranes. Previous reports of congenital syphilis in the newborn describe respiratory distress in less than a quarter of cases[22-24] or fail to mention it at all.[25-27] The diagnosis depended on finding positive serology together with two or more typical features of congenital syphilis.[20] Such features do not include respiratory distress and many of the deaths attributed to syphilis in this survey did not have or survive long enough to develop two or more typical features.

There is little doubt though that syphilis caused these deaths either by direct infection or by causing preterm delivery. Infection can only be confirmed by identifying the organism either by dark field microscopy in the few cases with skin lesions or by histological examination using special stains not available for this survey.

Using histological confirmation the perinatal mortality rate from congenital syphilis was found to be 5,0/1 000 births in Ethiopia and 3,2/1 000 births in South Africa,[9] probably underestimates of the true rates, which may have been closer to the rate of 7,4/1 000 births in this survey.

2. Birth asphyxia: Birth asphyxia accounted for 19 pc of the deaths and produced a perinatal mortality rate of 6,8/1 000 births in Bulawayo or 1,4/1 000 births at Mpilo Maternity Hospital. This rate is

similar to rates of about 15/1 000 births in teaching hospitals in Nigeria[8] and Ethiopia,[8] but is much higher than a rate of 3/1 000 births reported from South Africa.[9]

Most of the cases weighed 2 500g or more and were caused by obstetric complications that could have been prevented or treated successfully without expensive monitoring equipment. Improvements in obstetric care, particularly the active management of labour using the partogram, could result in a considerable reduction in the mortality rate. Birth asphyxia is also an important cause of handicap in infants that survive hypoxic ischaemic encephalopathy; at least 3,2/1 000 deliveries at Mpilo Maternity Hospital recovered from moderate encephalopathy, one third of whom were likely to be handicapped.[28]

One half of the cases were caused by prolonged obstructed labour with delay in the active phase of the first stage of labour crossing the four hour action line on a partogram and/or with delay in the second stage lasting more than one hour and often more than two hours. This was almost always the result of cephalo-pelvic disproportion, with marked caput and moulding. It was more common in primiparous women, especially prolonged first stage, a few of whom were given oxytocin which precipitated foetal distress. Half of the babies were delivered operatively in an attempt to save their lives, failure to do so usually being due to delay in performing the procedure.

In 11 multiparous women prolonged obstructed labour resulted in uterine rupture, half of them through a previous Caesarian section scar. One quarter of the cases were caused by prolapsed cord, stuck head in breech delivery and retained second twin, and these were more common in multiparous women.

Two thirds of the cases with prolapsed cord were referred to hospital after the event, usually with no cord pulsation. Eight were delivered by Caesarian section too late to save the baby. Cord prolapse, being an acute event, rarely resulted in potentially handicapped survivors, whereas prolonged labour, producing chronic partial asphyxia, commonly caused handicap. One eighth of the cases were caused by post maturity, intrauterine growth retardation, shoulder dystocia and other unusual causes, and the remaining one eighth were unexplained.

In most cases there was no problem in diagnosing these complications or, with the exception of prolapsed cords, transferring the mothers from clinics to the hospital. Delay in transfer did occur in a few cases sent from outlying district hospitals. The major problem however lay in failure to take appropriate action at Mpilo Maternity Hospital sufficiently early to save the baby. This usually resulted from the shortage of experienced doctors or the inability to perform an emergency Caesarian section because the theatre was already in use. Expansion of the delivery and theatre facilities is planned.

Saving these babies need not necessarily produce a dramatic increase in the Caesarian section rate, which was 16 pc during this survey, because many cases are already delivered by Caesarian sections, which are delayed too long to save the babies. Oxytocin is advocated in the management of prolonged first stage in primiparous women but careful monitoring for foetal distress is needed and may not always be possible without monitoring equipment.

A particular problem was mothers needing elective repeat Cesarean sections, who often tried to avoid them by attempting to deliver at home, sometimes with the aid of traditional medicines to stimulate contractions, thus greatly increasing the risk of ruptured uterus and birth asphyxia. Improved resuscitation of asphyxiated babies is needed but by itself is unlikely to reduce the mortality rate.

3. Hyaline membrane disease: Hyaline membrane disease accounted for 11 pc of the deaths and produced a perinatal mortality rate of 4,1/1 000 births. The incidence was 7,9/1 000 live births in Bulawayo and 15,6/1 000 live births at Mpilo Central Hospital. This is similar to rates in Zambia, South Africa and industrialised countries,[11] with a case fatality rate of 60 pc.

Nearly half of the deaths weighed less than 1 000g, some of whom could have been salvaged with sophisticated and expensive intensive care, whereas most of the cases weighing more than 1 000g could have been successfully ventilated without the need for expensive monitoring equipment or intravenous feeding. Over the last two years, using one available neonatal ventilator, 19 babies over 1 000g with hyaline membrane disease were ventilated, 14 of whom survived. Expansion of the facilities to ventilate babies and a trial of surfactant replacement therapy are planned.

4. Neonatal septicaemia: Neonatal septicaemia accounted for 11 pc of the deaths and produced a perinatal mortality rate of 3,9/1 000 births. The incidence was 6,6/1 000 live births in Bulawayo and 13,2/1 000 live births at Mpilo Central Hospital. These were much higher than rates of 2–3/1 000 live births in Scandinavia with case fatality rate of 59 pc also much higher than 10–30 pc in Scandinavia.[29,30] Half of the cases had early onset septicaemia most commonly due to group *B streptococci* and half had late onset nosocomial septicaemia most commonly due to *Klebsiella* and *Staph. aureus*. The spectrum of pathogens was similar to that in Finland,[30] except for a higher percentage with *Klebsiella* as in earlier studies in Europe and USA,[32] and a low pc with *Staph. epidermidis*, presumably because vascular catheters were seldom used.[32] Unlike Saudi Arabia and Nigeria,[31] *Salmonella* septicaemia was rare, with only four cases all of whom survived over the last two years.

The high incidence of gram negative septicaemia occurred despite all babies being on their mothers' milk, indicating that breast milk does not provide protection from these infections in Africa.[33]

The overcrowded and understaffed working conditions at Mpilo Maternity Hospital may contribute to the high incidence of late onset septicaemia, although so far no significant reduction has been achieved by intensive efforts to encourage clean hands, feeding utensils, incubators and aseptic procedures with sterile equipment.

Early treatment with penicillin and gentamicin for respiratory distress or ceftrioxone and cloxacillin for suspected late onset septicaemia has reduced the mortality rate from septicaemia from 59 pc ($p<0,05$) in the eight months following this survey. One important factor accounting for the high incidence of neonatal septicaemia is maternal HIV infection, 40 pc of the deaths being HIV positive.[16]

5. Congenital malformations: Congenital malformations accounted for eight pc of the deaths and produced a perinatal mortality rate of 2,8/1 000 births, compared with 2,7/1 000 births in USA[9] and 4,3/1 000 births in Scotland.[34] As expected neural tube defects were the most common, accounting for 32 pc of serious malformations, with an incidence in Bulawayo of 1,2/1 000 deliveries, compared with 3,8/1 000 deliveries in England and Wales in 1974.[35]

Chromosomal abnormalities accounted for 20 pc.Two thirds of mothers delivering babies with Down's syndrome were over 35 years and already had four or more children. Alimentary malformations accounted for 20 pc, most being amenable to surgical correction; eight out of 11 babies with oesophageal atresia were successfully treated in the last two years.

6. Pregnancy induced hypertension and placental abruption: Pregnancy induced hypertension and placental abruption together accounted for 10 pc of the deaths and produced perinatal mortality rates of 2,0 and 1,7/1 000 births respectively, compared with 1,4/1 000 births for either condition in Scotland.[34]

The mortality rate from pregnancy induced hypertension could be reduced by early antenatal care and premature delivery of those with severe hypertension and proteinuria, with a good outcome at birth weights above 1 250g because hyaline membrane disease is uncommon.

In contrast the outcome from placental abruption is difficult to improve because emergency Caesarian section delivery often results in babies with severe asphyxia or hyaline membrane disease.

This study demonstrates that a substantial reduction in perinatal mortality in Bulawayo could be achieved by improvements in antenatal, intrapartum and neonatal care aimed at preventing congenital syphilis and birth asphyxia and treating hyaline membrane disease and neonatal septicaemia. However these improvements must go hand in hand with family planning measures if the babies that survive are not to be destined to a life of poverty.[36]

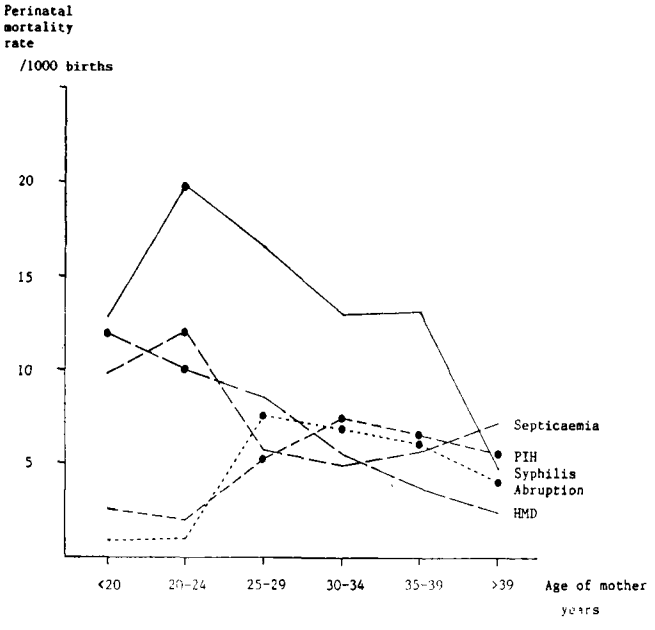
Health education should be directed particularly at teenagers and young women in their early 20's, who at present account for half of the deliveries.

Surprisingly the perinatal mortality rate and particularly the neonatal mortality rate was highest in the 20–24 year group. This was because the mortality rates from congenital septicaemia were highest in this age group, as shown in Figure 2.

The perinatal mortality rates from birth asphyxia, unexplained stillbirths and congenital malformations showed no significant variation with maternal age. For each condition the rates marked by spots are significantly greater than those without spots taken together.

As discussed in a separate publication,HIV infection, which predisposes to neonatal septicaemia,

Figure 2: Relation between mothers' age and perinatal mortality rate from ... membrane disease (HMD), neonatal septicaemia, pregnancy induced hypertension ... abruption for deliveries at Mpilo Maternity Hospital.



The perinatal mortality rates from birth asphyxia, unexplained stillbirths and congenital malformations showed no significant variation with maternal age. For each condition the rates marked by spots are significantly greater than those without spots taken together.

is also most frequent in the 20–24 year group.[16] The mortality rate from hyaline membrane disease was greatest under 25 years, perhaps in part because of preterm deliveries caused by other sexually transmitted diseases, such as gonorrhoea.[3] In contrast the mortality rates from pregnancy induced hypertension and placental abruption were greatest over 25 years. The control of sexually transmitted diseases and improvements in family planning for teenagers and young women are urgent health priorities in Central Africa.

ACKNOWLEDGEMENTS

I am most grateful to Dr N T Chaibva, the Medical Superintendent of Mpilo Central Hospital, for permission to publish this survey; to all the staff of the Maternity Hospital, Microbiology and Histopathology Departments for their help, and especially to Sr A M Luza, Sr F M Moyo, Dr J J Valdes Gomez, Dr F J Gonzales de Chaves, Dr B A Java, Dr Z Sibanda, Dr R A Dlodlo, Dr G Gangioli, Mr D A A Verkuyl and Mrs H E Aiken for their invaluable assistance.

REFERENCES

1. Kramer MS. Determinants of low birth weight; methodological assessment and meta-analysis. *Bull WHO* 1987; 65:663–737.
2. Schulz KF, Cates W, O'Mara PR. Pregnancy loss, infant death, and suffering: legacy of syphilis and gonorrhoea in Africa. *Genitorurin Med* 1987; 63:320–25.
3. Elliot B, Brunham RC, Laga M, *et al.* Maternal gonococcal infection as a preventable risk factor for low birth weight. *J Infec Dis* 1990; 161:531–36.
4. Ryder RW, Nsa W, Hassig SE, *et al.* Perinatal transmission of the human immunodeficiency virus type 1 to infants of seropositive women in Zaire. *N Engl J Med* 1989; 320:1637–42.
5. Watts T, Larsen SA, Brown ST. A case-control study of stillbirths occurring at the University Teaching Hospital, Lusaka: serological investigations. *Med J Zambia* 1983; 17:27–29.

6. Bhat G J, Hira SK, Ratman A V, Sehgal D, Chintu C, Mulenga RC. Congenital syphilis in Lusaka. III. Incidence in the neonatal intensive care unit. *E Afr Med J* 1982; 59:374–78.
7. Harrison KA, Lister UG, Rossiter CE, Chong H. Child-bearing, health and social priorities: a survey of 22 774 consecutive hospital births in Zaire, Northern Nigeria. II Perinatal mortality. *Br J Obstet Gynaecol* 1985; (Suppl 5):86–99.
8. Abudu O, Akinkugbe A. Clinical causes and classification of perinatal mortality in Lagos. *Int J Gynaecol Obstet* 1982; 20:443–47.
9. Ross SM, MacPherson T, Wallace J, Khatree MH, Naeye RL, Appelbaum PC. Unsuccessful pregnancies — report on 200 perinatal postmortems. *S Afr Med J* 1978; 53:828–29.
10. Philpott RH. The recognition of cephalopelvic disproportion. *Clinics in Obstet and Gynaecol* 1982; 9:609–24.
11. Coulter JBS. The incidence of respiratory distress syndrome: with particular reference to developing countries. *Trop Geogr Med* 1980; 32:277–85.
12. Munjanja SP, Masona D. Zimbabwe birth weight for gestation standards. *Cent Afr J Med* 1990; 36:144–47.
13. Ingall D, Dobson S, Musher DM. Congenital syphilis. In: Remington JS, Klein JD, eds. Infectious diseases of the fetus and newborn infant. 3rd ed. Philadelphia: WB Saunders, 1988.
14. Fenichel GM. Neonatal neurology. Clinical Neurology and Neurosurgery Monographs. Churchill Livingstone, 1980; 2:79–104.
15. Ingraham NR. The value of penicillin alone in the prevention and treatment of congenital syphilis. *Acta Derm Venereol* 1951; 31 Suppl 24:60–88.
16. Aiken CGA. HIV I infection and perinatal mortality in Zimbabwe. *Arch Dis Child 1992;* In press.
17. Naicker SN, Moodley J, Van Middlelkoop A, Cooper RC. Serological diagnosis of syphilis in pregnancy. *S Afr Med J* 1983; 63:536–37.
18. Gilbert GL. Congenital syphilis — should we worry? *Med J Aust* 1988; 148:162–63.
19. Garland SM, Kelly VN. Is antenatal screening for syphilis worthwhile? *Med J Aust* 1989; 151:368–72.
20. Kaufman RE, Jones OG, Blount JH. Questionnaire survey of reported congenital syphilis: problems in diagnosis, prevention and treatment. *Sex Transm Dis* 1977; 4:135–39.
21. Berkowitz KM, Stampf K, Baxi L, Fox HE. False negative screening tests for syphilis in pregnant women. *N Engl J Med* 1990; 322:270–71.
22. Chawla V, Pandit PB, Nkrumah FK. Congenital syphilis in the newborn. *Arch Dis Child* 1988; 63:1393–94.
23. Ricci JM, Fojaco RM, O'Sullivan MJ. Congenital syphilis: The University of Miami/Jackson Memorial Medical Centre experience. 1986–1988. *Obstet Gynecol* 1989; 74:687–93.
24. Mascola L, Pelosi R, Blount JH, Alexander CE, Cates W. Congenital syphilis revisited. *Am J Dis Child* 1985; 139:575–80.
25. Hira SK, Bhat GJ, Patel JB, *et al.* Early congenital syphilis: clinico-radiological features in 202 patients. *Sex Trans Dis* 1985; 12:177–83.
26. Srinivasan G, Ramamurthy RS, Bharathi A, Voora S, Pildes RS. Congenital syphilis: a diagnostic and therapeutic dilemma. *Pediatr Infect Dis* 1983; 2:436–41.
27. Tan KL. The re-emergence of early congenital syphilis. *Acta Paediatr Scand* 1973; 62:601–7.
28. Levene MI, Sands C, Grindulis H, Moore JR. Comparison of two methods of predicting outcome in perinatal asphyxia. *Lancet* 1986; i:67–8.
29. Bennet R, Eriksson M, Melen B, Zetterstrom R. Changes in the incidence and spectrum of neonatal septicaemia during a fifteen-year period. *Acta Paediatr Scand* 1985; 74:687–90.
30. Vesikari T, Isolauri E, Tuppurainen N, *et al.* Neonatal septicaemia in Finland 1981–85. Predominance of group B streptococcal infections with very early onset. *Acta Paediatr Scand* 1989; 78:44–50.
31. Ohlsson A, Serenius F. Neonatal septicaemia in Riyadh, Saudi Arabia. *Acta Paediatr Scand* 1981; 70:825–29.
32. Ohlsson A, Vearncombe M. Congenital and nosocomial sepsis in infants born in a regional perinatal unit: cause, outcome, and white blood cell response. *Am J Obstet Gynecol* 1987; 156:407–13.

33. Bennet R, Erickson M, Zetterstrom R. Increasing incidence of neonatal septicaemia: causative organism and predisposing risk factors. *Acta Paediatr Scand* 1981; 70:207–10.
34. McIlwaine GM, Howat RCL, Dunn F, MacNaughton MC. The Scottish Perinatal Mortality Survey. *Br Med J* 1979; 2:1103–6.
35. Lorber J. Central nervous system malformations. In: Roberton NRC ed. Textbook of Neonatology. Churchill Livingstone 1986.
36. King M. Health is a sustainable state. *Lancet* 1990; 336:664–67.

ZAR Gomo
Department of Chemical Pathology
University of Zimbabwe
Medical School
P.O.Box A178
Harare