Research from the South

Fetal and neonatal mortality: a matter of care? Report of a survey in Curaçao, Netherlands Antilles

H I J WILDSCHUT, M C B J E TUTEIN NOLTHENIUS-PUYLAERT, V WIEDIJK, P E TREFFERS, J HUBER

Abstract

A detailed clinicopathological analysis of 223 consecutive fetal and neonatal deaths was carried out in Curaçao during 1984 and 1985; this included careful histological examination of 210 infants (94%). The crude death rate was 34.2 per 1000 total births. Malformation was the principal cause of death in 28 cases, antepartum haemorrhage in 19, hypertension in 25, and asphyxia in 35. Death was caused by problems of preterm birth in 68 cases. No specific cause could be found for 34 deaths.

Improvement in the quality of obstetric care might substantially reduce both fetal and neonatal death rates.

Introduction

The aim of this survey was to analyse the causes of all fetal and neonatal deaths that occurred in Curaçao in the years 1984 and 1985 and to identify those conditions associated with the deaths in which a reduction of fetal and neonatal mortality might be obtained.

Curaçao is 444 km², the largest island in the Netherlands Antilles; this federation of islands is an autonomous part of the Netherlands. Curaçao has about 160 000 inhabitants, predominantly West Indians of African descent. The total number of births each year increased slightly in the years preceding this survey, but the crude birth rate remained constant at 19.5 per 1000 inhabitants. Maternity health care services are provided at three hospitals, where most of the deliveries take place. Two clinics provide antenatal and postnatal facilities for women who are identified as at low risk in terms of outcome of pregnancy (primary care), and one clinic provides modern facilities for obstetric as well as neonatal special care (secondary care).

Perinatal morbidity and mortality may sometimes be prevented by improving the health care services,¹ but it is difficult to assign perinatal death to a particular cause because of the multiplicity of contributory factors.^{1,2} This has prompted many workers to

develop their own classification systems.^{3,7} The Aberdeen clinicopathological classification has proved useful in identifying those aspects of fetal and neonatal mortality that can be influenced by alterations in management.⁸ For the purpose of this study we adopted a clinicopathological classification based on the Aberdeen method but that took into account the current attitudes and the diagnostic methods available in Curaçao.

Subjects and methods

The total number of babies born in Curaçao weighing 500 g or more was 6514 during 1984 and 1985. About 98% of the deliveries took place in hospital. All three hospitals that provided maternity services took part in the study. From the scoring system of Paneth *et al*⁹ maternity and neonatal services at two of the hospitals were categorised as giving primary care and the other as giving secondary care.

Between 1 January 1984 and 31 December 1985 all fetal and neonatal deaths in the island were subjected to detailed analysis. Perinatal deaths comprised both fetal and neonatal deaths with a minimum birth weight of 500 g.¹⁰ Infants who died within 28 days of life were included. We wrote to all midwives and physicians concerned with perinatal care and asked them to report every fetal and neonatal death as it occurred to two of us (HIJW and VW). Immediately after notification the circumstances surrounding each death were scrutinised in an attempt to identify factors that might have contributed to the death. Children of women not resident in Curaçao were excluded. Frequent examination of the statutory death certificate registers and the labour ward and mortuary registers ensured that no death was missed.

Permission for necropsy was obtained, and coding was completed by two of us. A full necropsy included routine histological examination of major organs and the placenta. A paediatric pathologist from the Netherlands (JH) reviewed most of the histopathological samples. Characteristic morphological and histological changes associated with fetal or neonatal death were defined according to the criteria proposed by Wigglesworth.¹¹ Infiltration of the membranes by polymorphonuclear leucocytes was regarded as diagnostic of chorioamnionitis.¹² Extensive infarction was defined as necrosis of more than 10% of the placental parenchyma.¹² Specimens for bacterial culture were routinely taken from the skin (preferably the ear) and throat of the infant immediately after death. When pulmonary infection or septicaemia was suspected bacterial cultures were taken from the lungs and blood, respectively. The specimens were inoculated on blood and MacConkey agar plates and incubated at 37°C for 48-72 hours in an aerobic (blood and MacConkey agar) as well as an anaerobic chamber (blood agar). Organisms were identified by standard methods. In each case the three microorganisms yielding the heaviest growth were reported. Cultures from the skin and throat that yielded only a minimal or poor growth of any microorganism (<20 colonies/plate) were reported as negative. The specimens were not cultured for chlamydias, mycoplasmas, or ureaplasmas.

Radiological investigation was carried out only if indicated. Samples of maternal blood were taken for serological examination (for toxoplasma, cytomegalovirus, treponema, and rubella). Cytogenetic, viral, and biochemical investigations were not performed.

All fetal and neonatal deaths were assigned to one of the seven categories in our classification derived from the Aberdeen clinicopathological clas-

Academisch Medisch Centrum, University of Amsterdam, 1105 AZ Amsterdam, The Netherlands

H I J WILDSCHUT, MD, obstetrician
P E TREFFERS, MD, professor of obstetrics

Landslaboratorium, Curaçao

M C B J E TUTEIN NOLTHENIUS-PUYLAERT, MD, pathologist

Geneeskundige Gezondheids Dienst, Curaçao

V WIEDIJK, MD, physician

State University of Utrecht, The Netherlands

J HUBER, MD, professor of paediatric pathology

Correspondence to: Dr Wildschut.

sification.⁸ So that the classification was used consistently allocation to the seven categories followed strict rules.

Malformation—Any malformation that was incompatible with life took precedence over all other pathological abnormalities.

Antepartum haemorrhage—This category comprised all cases in which death was obviously the result of antepartum haemorrhage (for instance abruptio placentae and placenta previa). It took precedence over all the others except lethal malformation. Deeply asphyxiated infants born after clinically suspected abruptio placentae were included in this group.

Hypertension—Death was attributed to hypertension when a minimal diastolic blood pressure of 100 mm Hg was recorded ante partum on at least two occasions six hours apart. Essential hypertension and hypertension secondary to chronic nephritis were included in this category.

Asphyxia—This group comprised all cases in which death was attributed to fetal distress that had resulted in a non-macerated stillbirth or a neonate in poor condition at birth. Deaths due to birth trauma and prolapse of the

optimal care would probably not have prevented the adverse outcome. The following criteria for unavoidable death were used: gestational age less than 28 weeks; lethal malformation; death ascribed to abruptio placentae; death in spite of appropriate treatment in the miscellaneous category; and unexplained death with histological evidence of acute stress. Death allocated to one of the other categories was considered potentially avoidable, indicating that it could possibly have been prevented by an improvement in the quality of care.

Results

During the two years 223 fetal and neonatal deaths occurred, including 18 twins; 210 (94%) full necropsies were performed. We also examined 176 placentas (79%). Preterm delivery (less than 37 completed weeks) was a contributory factor in 147 (66%) cases. In singletons the incidence of

TABLE I—Distribution of fetal and neonatal deaths by gestational age

Clinicopathological classification	Gestational age (weeks)					Total deaths	Death rate/1000 births
	≤27	28-32	33-36	37-41	≥42		
Malformation	2	5	10	10	1	28	4.3
Antepartum haemorrhage	3	10	4	2		19	2.9
Hypertension	2	8	6	9		25	3.8
Asphyxia		1	2	25	7	35	5.4
Problems of preterm birth	53	14	1			68	10.4
Miscellaneous		3	5	6		14	2.2
Death of unknown origin	1	10	7	13	3	34	5.2
Total (%)	61 (27)	51 (23)	35 (16)	65 (29)	11 (5)	223 (100)	34.2

TABLE II—Distribution of fetal and neonatal deaths by birth weight

Clinicopathological classification	Birth weight (g)						Total deaths
	500-999	1000-1499	1500-1999	2000-2499	2500-3999	≥4000	
Malformation	4	2	5	5	12		28
Antepartum haemorrhage	4	1	12	1	1		19
Hypertension	8	5	5	4	3		25
Asphyxia		1		3	26	5	35
Problems of preterm birth	50	14	3	1			68
Miscellaneous	1	1	1	3	7	1	14
Death of unknown origin	4	6	5	4	15		34
Total (%)	71 (32)	30 (14)	31 (14)	21 (9)	64 (29)	6 (3)	223 (100)

umbilical cord were also included. Infants of less than 28 weeks' gestation were excluded.

Problems of preterm birth—This category included cases in which death was the result of immaturity of one or more organs and their functions (for instance, hyaline membrane disease, massive pulmonary haemorrhage, bronchiopulmonary dysplasia, superimposed pneumonia and atelectasis, or intraventricular haemorrhage). Infants of less than 28 weeks' gestation with disorders causing death during labour or in the neonatal period other than those mentioned above are allocated to this category.

Miscellaneous—This category included specific causes of death not already covered (for instance, haemolytic disease or cases in which the immediate neonatal condition was satisfactory but overwhelming infection supervened later).

Death of unknown origin—Deaths placed in this category were unexplained and could not be allocated to any of the preceding groups. Pregnancy and labour had usually been normal.

Necropsy findings were tabulated individually and pathological findings were not mutually exclusive. Pathological abnormalities were tabulated against clinicopathological changes. We looked for histological evidence of stress reactions in the adrenal glands of stillborn infants¹³ and in the thymus glands of neonates¹¹ and refined three patterns: acute stress (adrenals—type I; thymus—normal or starry sky pattern); subacute stress (adrenals—type II; thymus—cortex progressively depleted of lymphocytes, with medulla retaining its cellularity); and chronic stress (adrenals—type III; thymus—advanced depletion of lymphocytes). Stress patterns were tabulated against the clinicopathological group.

From the obstetric point of view death was considered unavoidable when

preterm delivery was 85.2 per 1000 total births. Tables I and II show the distribution of fetal and neonatal deaths by gestational age and birth weight, respectively, in each category of the clinicopathological classification.

Table III gives details of the times of death of the infants studied. There were 116 (52%) fetal deaths, and of 85 infants who died in the early neonatal period (0-6 days), 41 weighed less than 1000 g at birth (48%).

Malformation accounted for 28 deaths; eight were due to lesions affecting the central nervous system and four to congenital heart disease. Five infants, including a conjoined twin, died of multiple congenital abnormalities. The remaining 11 deaths were due to structural defects of various kinds; one was probably due to congenital rubella virus infection. Congenital toxoplasmosis and cytomegalovirus infection were never diagnosed. The death rate from malformations was 4.3 per 1000 total births (table I).

Antepartum haemorrhage—Premature detachment of the placenta was recorded in all 19 deaths due to antepartum haemorrhage. This resulted in 14 stillbirths and five neonatal deaths (table III). Concomitant hypertension, defined as a minimal diastolic blood pressure of 100 mm Hg, was identified in three mothers; one suffered an eclamptic seizure during labour.

Hypertension was associated with 25 fetal and neonatal deaths. Most (17) occurred ante partum (table III). Four mothers had eclamptic seizures ante partum.

Asphyxia accounted for 35 deaths, most occurring in mature infants (table I). Twenty five infants died during labour and 10 in the first week of life (table III). Two deaths in this group were due to prolapse of the umbilical cord. No infant had pathological evidence of a tentorial tear. The death rate from asphyxia was 5.4 per 1000 total births (table I).

Problems of preterm birth were responsible for 68 deaths. Fifty three infants

were born before 28 weeks' gestation. In 28 of the 68 deaths histopathological examination of the lungs showed polymorphonuclear leucocytes in varying numbers (table IV). Nine twins died, including four pairs.

Miscellaneous—Fourteen deaths were listed as miscellaneous. Five were due to generalised infection, including herpes (one case). Four deaths were due to fetal hydrops and one to kernicterus. One fetus died as the result of maternal shock caused by fatal amniotic fluid embolism. One death was associated with a large haematoma of the umbilical cord. The two remaining deaths were due to serious pulmonary disease.

Death of unknown origin—No specific cause of death was found in 34 cases, the outcome in 32 being a macerated stillbirth.

category problems of preterm birth. In addition, examination of the placentas showed 19 specimens with extensive infarction (table IV). Twelve were associated with clinical hypertension and four with unexplained death.

Table VI shows the type of stress diagnosed histologically for fetal and neonatal deaths by clinicopathological classification. Because of severe maceration histological sampling of the adrenals yielded no specific diagnosis in two infants, and in one infant staining failed due to technical error. Deaths ascribed to the categories malformation, antepartum haemorrhage, and problems of preterm birth were often associated with the changes of acute stress, whereas those ascribed to hypertension and death of unknown origin were often preceded by chronic stress.

TABLE III—Time of fetal and neonatal deaths

Clinicopathological classification	Stillbirths		Neonatal deaths		Total deaths
	Antepartum	Intrapartum	Days 0-6	Days 7-27	
Malformation	4	2	19	3	28
Antepartum haemorrhage	13	1	5		19
Hypertension	14	3	4	4	25
Asphyxia		25	10		35
Problems of preterm birth		17	43	8	68
Miscellaneous	5		3	6	14
Death of unknown origin	32		1	1	34
Total (%)	68 (30)	48 (22)	85 (38)	22 (10)	223 (100)

TABLE IV—Necropsy findings in 210 fetal and neonatal deaths

	Clinicopathological classification*							Total No
	1	2	3	4	5	6	7	
No of deaths	28	19	25	35	68	14	34	223
No of necropsies (%)	28 (100)	18 (95)	24 (96)	31 (89)	65 (96)	12 (86)	32 (94)	210 (94)
Necropsy findings:								
Congenital abnormality	28		4	1			3	36
Hydrops	2				2	3		7
Polymorphonuclear leucocytes in lungs	5	1	4	10	28	2		50
Hyaline membrane disease		2	4		17			23
Alveolar haemorrhage	1		3	3	2	1		10
Intraventricular and subependymal haemorrhage		2	3	1	20			26
Bronchopulmonary dysplasia					7			7
Lung hypoplasia	19		1			1		21
Infarction of the placenta	1	1	12	1			4	19
Chorioamnionitis	4	4	5	7	33		5	58

*1=Malformation; 2=antepartum haemorrhage; 3=hypertension; 4=asphyxia; 5=problems of preterm birth; 6=miscellaneous; 7=death of unknown origin.

TABLE V—Bacteriological findings in 210 cases of fetal and neonatal deaths in which necropsy was performed

	Infants with polymorphonuclear leucocytes in lungs (n=50)			Infants without polymorphonuclear leucocytes in lungs (n=160)	
	Lungs	Skin	Throat	Skin	Throat
No of cultures	31	39	34	114	107
No of cultures positive (%)	23 (74)	25 (64)	24 (71)	65 (57)	71 (66)
No of isolates of:					
<i>Pseudomonas</i> spp	8	2	4	5	0
<i>E. coli</i>	7	8	6	30	36
<i>Klebsiella</i> spp	7	3	2	11	14
β Haemolytic streptococcus group B	2	8	4	13	4
Others	14	24	20	43	49

Table IV shows the association between specific pathological findings at necropsy and the seven categories of the clinicopathological classification. Polymorphonuclear leucocytes in pulmonary alveoli or interstitium, or both, was the commonest histological finding (50 cases), and bacterial cultures from the lungs were available from 31 of these 50 infants (table V). Bacteria were recovered from 23 of the 31 swabs. Microorganisms most commonly isolated were *Pseudomonas* and *Klebsiella* spp, and *Escherichia coli*. The proportions of positive bacterial isolates from the skin and throat did not differ significantly between infants with histological evidence of polymorphonuclear leucocytes in the lungs and those without (table V).

Table IV shows the incidence of chorioamnionitis in each clinicopathological category. This condition was most often diagnosed in the

Discussion

In Curaçao the combined fetal and neonatal death rate was 34.2/1000 total births in 1984-5. The death rate from malformations was 4.3/1000 total births. In England and Wales the reported death rate from congenital malformations was 3.5/1000 births in 1978.¹⁴ Most of the malformations did not seem to be caused by any identifiable teratogen, so improvement in death rates by primary prevention or early detection of congenital malformations is probably limited in this population. The incidence of deaths from antepartum haemorrhage (all due to abruptio placentae) was

2.9/1000 births. This rate is much higher than the reported 1.0/1000 births in England and Wales.¹⁴ Because of the insidious and acute nature of this disease death attributed to it is regarded as unavoidable.

Fifty eight deaths (27%) at or after 28 weeks' gestation were due to hypertension and asphyxia. Most of these could have been prevented by better fetal surveillance and consequent management.

Most of the deaths associated with problems of preterm birth occurred before 28 weeks' gestation and were therefore considered to be unavoidable. The aetiology of spontaneous preterm labour is not clearly understood.¹⁵ Infection may be a predisposing factor,¹⁶⁻¹⁸ but it may be difficult to ascertain whether infection was the cause or effect of the preterm labour and subsequent perinatal death.¹⁹ The extent to which the presence of pathogenic or potentially pathogenic bacteria in the birth canal contributed to perinatal infection was not investigated. Only the skin and throat of the infants were cultured

have prevented a considerable number of these unexplained deaths.

Many fetal and neonatal deaths are caused by circumstances surrounding the birth. Perinatal mortality is conventionally regarded as a performance indicator for maternal and neonatal health care services; there is, however, no quick solution to the problem of providing the best health care service.^{22,23} Apart from the quality of care, perinatal mortality is influenced by many other factors, including the social and biological characteristics of the mother, the frequency of preterm deliveries, the birth weight of the baby, and the state of health of the population as a whole.²

Many of the deaths were potentially avoidable. We recognise that attribution of deaths to "avoidable" and "unavoidable" groups has its limitations.^{24,25} Quantifying avoidability of death is difficult and subjective and should be done only in terms of the available skill and equipment. Consequently, in countries with tertiary levels of care criteria for avoidability should be different from those where such

TABLE VI—Types of stress diagnosed histologically and causes of death

Clinicopathological classification	Antenatal death			Neonatal death			
	Acute stress	Subacute stress	Chronic stress	Acute stress	Subacute stress	Chronic stress	Not diagnosed*
Malformation	4		2	17	3	2	
Antepartum haemorrhage	13			4		1	1
Hypertension	1	6	10	3	1	3	1
Asphyxia	12	9	2	4	3	1	4
Problems of preterm birth	14		2	32	3	13	4
Miscellaneous	3		2	5	1	1	2
Death of unknown origin	7	8	14	1			4
Total (%)	54 (24)	23 (10)	32 (14)	66 (30)	11 (5)	21 (9)	16 (7)

*Includes 13 that did not proceed to necropsy and three in which staining failed.

routinely. Microorganisms thus obtained may include pathogens but in themselves do not establish the presence of infection. In our series, therefore, study of bacterial isolates was not informative.

Chorioamnionitis was diagnosed in 33 of the 53 available placentas of infants who died of problems of preterm birth. Histological evidence of a mild degree of chorioamnionitis, however, must be interpreted with great care because it may be a normal feature in labour at any gestational age and does not necessarily imply intrauterine infection.¹⁹

Intrauterine infection is often associated with congenital pneumonia. The diagnosis of congenital pneumonia does not depend solely on the presence of polymorphonuclear leucocytes in the lungs because they may be inhaled from the amniotic fluid in the absence of true bacterial infection.²⁰ Lung tissue was cultured only if clinically indicated. Swabs from the lungs of 23 of 31 infants with histological evidence of polymorphonuclear leucocytes (74%) yielded growths of various Gram negative bacilli. Because of biased selection we were unable to determine the exact rate of bacterial isolation in infants without polymorphonuclear leucocytes in their lungs. In a previous study bacterial cultures from the lung were reported to be positive in 182 of 424 infants who did not have congenital pneumonia at necropsy (43%).²¹ Hence the precise role of ascending infection both in starting preterm labour and in perinatal death in this population remains unknown.

Deaths in the category miscellaneous could not usually be accounted for by the quality of care provided for the population and were therefore usually considered unavoidable.

Thirty four deaths of unknown origin were recorded. These were particularly tragic because the fetuses were normally formed and the pregnancies were apparently normal. Histological changes in the adrenal glands of 23 of these infants, however, showed type III patterns that may have been related to chronic underperfusion of the placenta.¹³ It is impossible to say that these infants were small for gestational age because we do not know the frequency distribution of birth weight in the population. Nevertheless, improved antenatal care with emphasis on detecting retardation of fetal growth might

high standards of care do not exist. Within the limits of the secondary care available in Curaçao the application of our simple clinicopathological classification system was helpful in identifying the shortcomings of the maternity services with regard to the outcome of pregnancy. A substantial reduction of perinatal mortality may well be effected by better fetal surveillance, improved clinical judgment, and better management of fetal distress. The present study clearly shows that it is possible to carry out a population based inquiry into fetal and neonatal mortality in a region that is socially and economically disadvantaged.

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Reviewing RAWP

Accounting for cross boundary flows

JOHN BRAZIER

Abstract

RAWP (Resource Allocation Working Party) allows for cross boundary flows by adjusting regional or district health authorities' (DHAs) targets at an average specialty cost. The previous paper in this series examined problems for an inner city district health authority arising from RAWP cross boundary flow adjustment. This paper examines the likely importance of these and other problems for the National Health Service as a whole.

Cross charging has been proposed as an alternative method of funding flows. District health authorities would receive an allocation equivalent to their RAWP target and then all non-emergency flows would be agreed between the authorities where patients live and competing authorities offering treatment at previously negotiated charges based on local estimates of each type of case. The problem of cost estimation is usually cited as a difficulty with this proposed reform, but this paper also discusses other important issues that tend to be neglected.

Introduction

Cross boundary flows exist because the administrative boundaries defining health authority populations often do not correspond to the actual pattern of use of hospital service by patients. These flows arise not only from emergency cases and tertiary referrals but also from patient convenience and established links between general practitioners and individual hospital consultants. RAWP recognised that health authorities should be compensated for the costs incurred in treating these cross boundary flows.¹ There is therefore an adjustment for non-psychiatric inpatient cross boundary flows in health authorities' targets at national specialty costs. The Department of Health and Social Security (DHSS) review of RAWP² is currently considering the adequacy of RAWP methods for accounting for the case mix of cross boundary flows. The paper considers this and other problems with the RAWP methods before considering cross charging as a possible solution.

Problems with current RAWP methods

ADEQUACY OF THE ADJUSTMENT FOR CROSS BOUNDARY FLOWS

Criticisms of the RAWP method of accounting for cross boundary flows have focused on the crudeness of the adjustment to targets. This is at the national average cost for each specialty as estimated using the DHSS specialty cost model.³ It has been argued that flows tend to be more severe and complex than the average. At regional level evidence suggests that inadequacies in the funding of flows are not very significant because flows are quite small, amounting in most regions to only a few per cent of caseload and over 10% in just three out of 14 regional health authorities. A sensitivity analysis of regional targets found that large changes in the specialty costs of flows affected targets by only a few percentage points.⁴

At district level the larger scale of flows suggests that inadequacies in their funding are likely to be more important. Over half the Thames districts have cross boundary flows of over 30% of their caseload, and nearly a quarter have flows of over 50%. But the net flow is considerably less because a district with high flows in tends also to have high flows out. So if flows are more complex than the average then the net effect on costs for each district will probably be neutral. A study of flows between districts in the North Western Regional Health Authority examined the net effect of more complex cross boundary flows on district RAWP targets.⁵ In only one district did the case mix of inpatients, as measured by expected length of stay by disease category, have more than a 1% effect on target. Even where the flows are more complex, this could be because they are tertiary referrals, which are often designated as regional specialties which have their costs protected to varying degrees by regions and are funded closer to their actual costs than the "bread and butter" specialties.

More recent research in the West Midlands Regional Health Authority has examined cross boundary flows of non-regional specialty cases at a teaching hospital in Birmingham.⁶ The research was confined to flows from districts outside Birmingham because it was assumed that flows from the rest of the city were likely to be flows of convenience and hence probably not more complex. They found that such flows had on average a longer length of stay of 15% in general medicine and 20% in general surgery than local cases. The "hotel costs" of the additional stay were likely to be small, but the longer length of stay is a proxy for case complexity, which could also affect treatment costs. However, the impact on the district's RAWP target of allowing for the case complexity would be reduced because

Department of Community Medicine, United Medical and Dental Schools of Guy's and St Thomas's Hospitals, St Thomas's Hospital, London SE1 7EH
J E BRAZIER, MSc, BA, research assistant in health economics