

Fetal and Early Neonatal Mortality

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SUMMARY

A format setting out and comparing perinatal deaths is presented. It is not new, but has not previously been used in South Africa. Gestational age and birthweight are considered, and group predictions for mortality (and indirectly for morbidity) are made, and can be used for comparison.

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The importance of a satisfactory outcome of each wanted pregnancy cannot be overemphasised. This is particularly so where family planning is advocated. Despite recent advances in perinatal care, high perinatal mortality (PNM) rates are still experienced in many hospitals.¹ The PNM rate represents the tip of an iceberg, and accounts for the deaths or disasters. This rate is easily measured. However, it does not include the much larger group of near-deaths, which constitutes the perinatal morbidity rate, which accounts for most subsequent mental and motor retardation.

The purpose of this article is to present a practical approach to perinatal deaths, incorporating recent recommendations of the World Health Organisation (WHO).² Although this study is applicable to any population group, Cape Coloured births have been used to illustrate the methodology, and to show where the major perinatal problems lie.

CLINICAL MATERIAL AND METHODS

During 1972 and 1973, the Peninsula Maternity Services delivered 13 344 infants of Coloured mothers. Details of life at birth (as evidenced by breathing, beating of the heart, pulsation of the umbilical cord, or definite movement of voluntary muscles),² and the subsequent outcome were coded and stored on computer tapes. Infants were weighed and the gestational age was recorded. The latter was based on the antenatal notes for stillbirths and on the Dubowitz scoring system³ for live births. A stillbirth (SB) was diagnosed when there was no evidence of life at, or immediately after, birth.² Early neonatal deaths (NND) included the deaths of liveborn infants during the first 7 days. Perinatal deaths included SBs and early NNDs, and are expressed as percentages of the total number of births.

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Throughout, mortality is expressed as a percentage or rate/1 000. Smoothed mortality rates are also superimposed on the intra-uterine growth curves for Coloureds whose babies were delivered by the Peninsula Maternity Services during 1972.⁴ Infants below the 10th percentile were grouped as being small for gestational age (SGA), those between the 10th and 90th as being appropriate for gestational age (AGA), and those above the 90th percentile as being large for gestational age (LGA). In accordance with WHO recommendations,² preterm infants were defined as those born at less than 37 completed weeks. Term infants include those from 37 to less than 42 completed weeks (259 to 293 days). Post-term refers to 42 completed weeks, or more. Infants who weigh less than 2 500 g at birth, but whose gestational age is not known, should simply be classified as being of a low birthweight.²

The WHO has recommended that local statistics should include all fetuses and infants delivered who weighed 500 g or more (corresponding to 22 weeks or more). For purposes of international comparison, only those weighing 1 000 g or more at birth should be included and the statistics should be designated 'Standard'.

RESULTS

Of the 13 344 deliveries, 309 were SBs (rate 23.2/1 000) and 160 were early NNDs (rate 12.0/1 000), as shown in Table I. The birthweights of 16 early NNDs were unrecorded, and 144 were left for analysis.

The lowest PNM occurred at 37-41 weeks and with birthweights between 3 500-4 000 g. PNM rate increased in both preterm and post-term groups, and infants over 4 000 g were at greater risk. The incidence of preterm deliveries was 9.4%, but this was associated with a PNM rate of 254/1 000. The majority of infants were born at term (89.7%) and only 0.9% were post-term. The PNM rates for term and post-term infants were low in comparison with the preterm deliveries, but they nevertheless accounted for a larger number of preventable deaths over-all.

Fig. 1 graphically demonstrates the mortality rates on a logarithmic scale. The logarithmic scale is used to incorporate the widely differing figures on one graph. As expected, the mortality fell with gestational age. Early in the third trimester, the NND rate exceeded the SB rate. At 31 weeks there was a dramatic drop in the NND rate (from 470 to 99/1 000) and a reversal of the NND/SB ratio. The NND rate thereafter remained at about a third of the corresponding SB rate. Once the fetus had reached 37 weeks, the risk of neonatal death was very small. The SB rate, however, increased with prolongation of pregnancy to 41 weeks or more.

TABLE I. PERINATAL MORTALITY BY BIRTHWEIGHT AND GESTATION FOR CAPE COLOURED INFANTS

Birthweight (g)	Pre-term			Term 37 - 41 wks	Post-term ≥ 42 wks	Total
	≤ 27 wks	28 - 31 wks	32 - 36 wks			
4 000			0/1	10/446 (2%)	0/5	10/452 (2%)
3 500			3/9 (33%)	13/1 757 (0,7%)	1/15 (7%)	17/1 781 (1%)
3 000		1/2	2/14 (14%)	37/4 500 (0,8%)	1/48 (2%)	41/4 564 (0,9%)
2 500		1/2	16/84 (19%)	32/4 080 (0,8%)	1/29 (3%)	50/4 195 (1%)
2 000		3/7 (43%)	37/450 (8%)	25/1 076 (2%)	0/12	65/1 545 (4%)
1 500	1/1 (100%)	13/26 (50%)	53/363 (15%)	14/98 (14%)		81/488 (17%)
1 000	13/13 (100%)	64/112 (57%)	35/87 (40%)	0/4		112/216 (52%)
500	43/44 (98%)	25/32 (78%)	9/11 (82%)			77/87 (89%)
Total	57/58 (98%)	107/181 (59%)	155/1 019 (15%)	131/11 961 (1%)	3/109 (3%)	453/13 328 (3,4%)

TABLE II. MAIN CLINICAL CAUSES OF NEONATAL DEATHS

Cause	Weeks of gestation					Total
	20 - 27	28 - 31	32 - 36	37 - 41	42 - 43	
Anoxia	0	4	3	7	0	14
Immaturity	16	40	9	0	0	65
Respiratory distress	1	9	16	7	2	35
Infection	0	1	4	4	0	9
Metabolic causes	0	0	1	0	0	1
Birth trauma	0	0	0	7	0	7
Congenital malformations	0	2	2	7	0	11
Blood disorders	0	1	0	3	0	4
Unknown	4	1	3	6	0	14
Total	21	58	38	41	2	160

The main clinical causes of neonatal death are given in Table II. Immaturity included those preterm infants with recurrent apnoeic attacks which were not due to any demonstrable cause and formed the largest group. It was followed by the respiratory distress category. The main cause of death was coded for each infant, since many conditions obviously co-existed, e.g. preterm delivery and hyaline membrane disease. Infection accounted for a high incidence of morbidity, but was a relatively uncommon primary cause of death. Among term infants malformations, anoxia, trauma, and respiratory distress accounted for the majority of deaths. Only 2 post-term infants died, both from meconium aspiration. The causes of intra-uterine deaths were unfortunately not classified and coded.

The combined effect of gestation and intra-uterine growth is shown in Figs 2 and 3. The growth curves depict percentiles for birthweight at different gestations. Such intra-uterine curves have become standard references for the growth of the fetus, and are akin to the growth curves for older children. In general, fetuses and neonates

between the 50th - 90th percentiles had the lowest mortality rates. The fetus above the 90th or below the 10th percentile had a markedly increased risk of death (Fig. 2).

Liveborn infants (Fig. 3) who were SGA had an increased mortality, but LGA infants seemed to do as well as the AGA group. The advisability of using the 10th percentile (as opposed to the 3rd percentile) as the cut-off for SGA infants, is clearly demonstrated in that more infants at risk were thus included. Note that the group between the 10th and the estimated 25th percentiles also had a higher PNM.

DISCUSSION

The correct use of gestational age has markedly altered the approach to perinatal problems,^{6,8} presenting a far more sensitive and accurate guide to the outcome of the fetus and the newborn than weight alone. It also allows for perinatal morbidity and death to be classified according to intra-uterine growth. The trends for Coloureds are

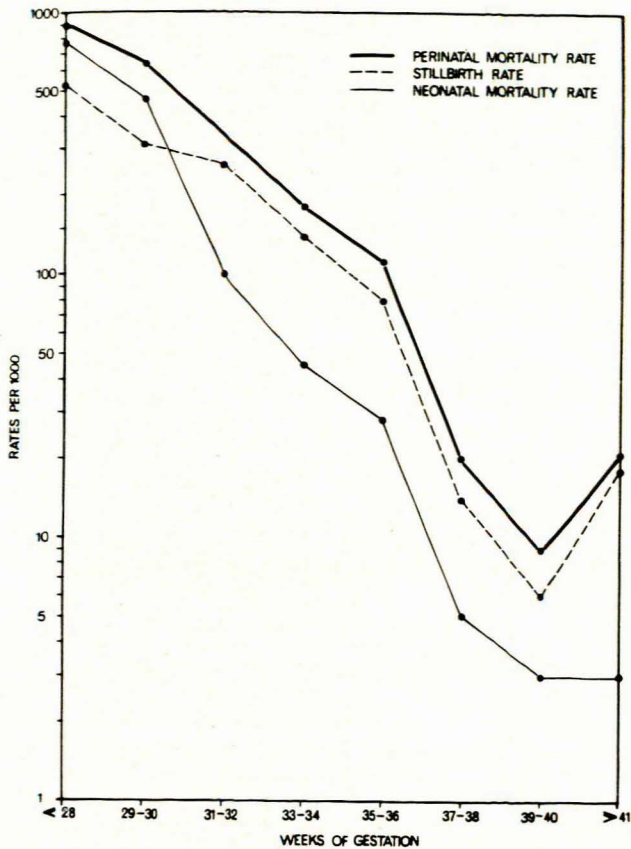


Fig. 1. Fetal and early neonatal mortality rates plotted against gestational age.

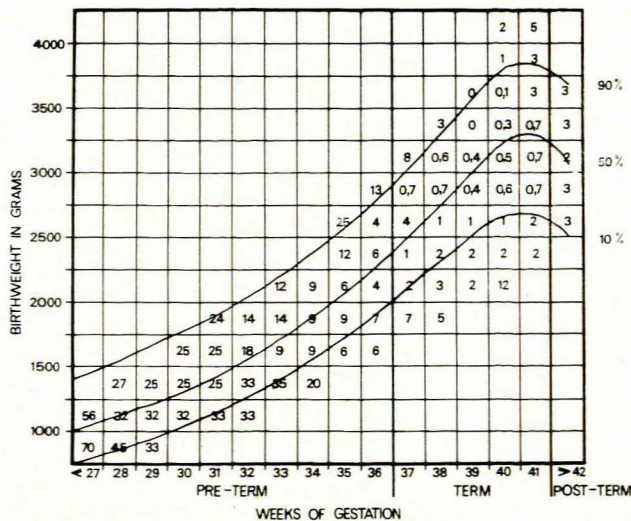


Fig. 2. Percentage stillbirths according to intra-uterine growth.

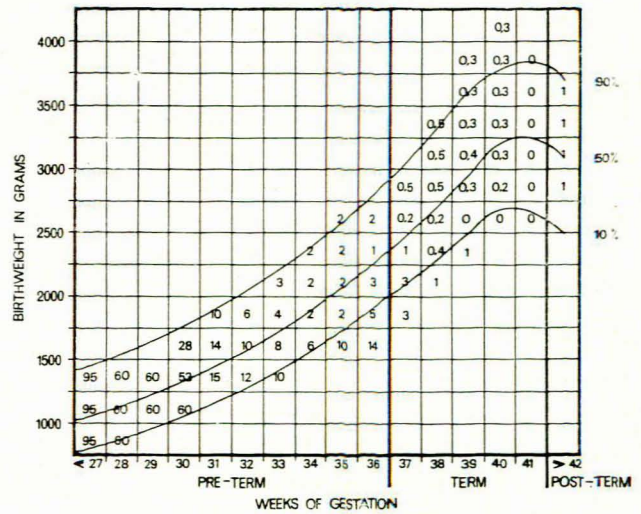


Fig. 3. Percentage early neonatal deaths according to intra-uterine growth.

The value of the above system, apart from reaffirming international concepts,² lies in its practical application. The gestational age provides an approximation of what to expect, should maternal or fetal disease necessitate intervention. At 31 - 32 weeks, the risk associated with extra-uterine life decreases. A closer look at the fetal deaths revealed that 85% of SBs occurred before the onset of labour,⁴ which emphasises the need for antenatal care. The increased risks for the fetus who is SGA or LGA is well documented, and is again emphasised.

The risk factors for liveborn infants should be used to identify those who need continued postnatal observation and special care. The figures do not necessarily apply to individual cases, but do provide a very good guide. The predictions should be of use to the medical or nursing attendant in providing the parents with a reasonable prognosis. Congenital malformations form a larger group in other series. The Coloureds seem to have a lower incidence of malformations.⁷ The over-all NND rate is 52% of the SB rate. Although this ratio should ideally be 1 : 1 (as is found in developed nations), SBs seem to predominate in most countries.^{8,9} Equality is only approached in those countries with the highest socio-economic status.^{8,9} In Cape Town it probably reflects poorer antenatal attendance and care, and lower socio-economic status. The small number of Coloured infants born post-term also suggests earlier delivery in this population group.

The perinatal charts are also useful when deciding how best to improve the present situation, and how to use the format for comparison with other centres. In the first instance, it is obvious that the fetus who is too small or too large, or who remains *in utero* too long, is a 'high risk' fetus. Assessment of intra-uterine growth and gestational age is therefore of critical importance. Preterm delivery (especially before 31 weeks) and the undergrown infant pose problems when dealing with the liveborn. It would be interesting to see if these findings are true for other population groups in South Africa, and whether they will improve in the future.

clearly demonstrated and show many similarities to other studies.^{5,6} We wish to point out that due to small numbers, smoothed mortalities were used in several categories.

The PNM charts reflect not only the deaths, but, in many instances, the infants who are at higher risk of brain damage. Although this is a generalisation, it would be true of infants who survive perinatal anoxia such as fetal distress, asphyxia neonatorum and recurrent apnoeic attacks. One of the aims of perinatal medicine should be to prevent such tragedies wherever possible.

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Osteonecrosis Following Renal Transplantation

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SUMMARY

Subarticular osteonecrosis has been described with increasing frequency after renal transplantation and the use of immunosuppressive corticosteroids. Six cases have been encountered among 170 patients in the Johannesburg renal transplantation programme. The commonly affected sites are the femoral head, the femoral condyles, the humeral head, the talus and the capitulum.

The radiological and morbid anatomical features of this complication are described, alternative theories of pathogenesis are considered and an approach to treatment is outlined.

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Osteonecrosis of the femoral head has been described in a variety of conditions: after fractures and dislocations of the hip,¹ in caisson disease,² in Gaucher's disease,³ after ionising radiation⁴ and in patients receiving corticosteroid therapy.⁵⁻¹⁰ The same condition has been described in patients after renal transplantation¹¹⁻¹⁵ and is generally attributed to the large doses of corticosteroids used for immunosuppression. The frequency of this complication varies considerably from centre to centre; indeed, the precise incidence cannot be determined unless repeated radiographic examination is carried out on every patient after renal transplantation, and even then it would depend upon the length of follow-up. Thus, it has been seriously suggested that all such patients will ultimately develop

osteonecrosis if they survive long enough, a consideration which, though at present theoretical, may hold grave implications for the future.

Between 1968, when the first renal transplant was carried out in Johannesburg, and 1972, only 1 case of femoral head necrosis was diagnosed among 100 patients. During the past 2 years another 5 cases were encountered in 70 patients.

The significant features of this condition are described in this article.

PATIENTS

Case 1

A 30-year-old White man was the first patient among those in the Johannesburg renal transplant series to develop this complication. In 1971, 32 weeks after renal transplantation he presented with a 10-12-week history of pain in the right hip. Radiographic examination showed the characteristic features of subchondral necrosis and distortion of the right femoral head (Fig. 1). The patient had by then received over 1000 mg of corticosteroids, mainly during the first month after operation. Total hip replacement was carried out and the patient returned to full activity.

Six months later, a similar sequence of events started in the opposite hip and replacement arthroplasty was ultimately necessary. In retrospect, it was apparent that the early radiographic changes of femoral head necrosis were already present soon after the first hip operation.

Again, after resuming normal activity, further symptoms manifested themselves. Firstly the right knee and then the left knee became painful, and radiographs showed osteonecrosis of the femoral condyles (Fig. 2). Pain was evidently

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