PERINATAL MORTALITY IN THE BANTU

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A postmortem study was undertaken at Baragwanath Hospital to determine the relative importance of cerebral birth injury and pulmonary pathology in the perinatal period. This hospital serves a large Bantu community in which there is malnutrition, ignorance and inadequate antenatal care. Owing to the inherent shape of the Bantu pelvis there are a large number of cases of cephalo-pelvic disproportion which are rarely seen in more highly-developed communities.[1] As a result there are a large number of stillbirths, premature deliveries and neonatal deaths. These factors should theoretically produce a high incidence of cerebral birth injury.

MATERIAL AND METHODS

During a period of 86 days, autopsies were performed on 238 (95·2%) of 250 children who were born dead or who died during the first four weeks of life. During the same period there were 2,359 deliveries of which 517 were premature babies (birthweight less than 2,500 G). In 186 deliveries surgical intervention was required and consisted of caesarean section in 97 cases, forceps delivery in 66, symphysiotomy in 18 and foetal destruction in 5 cases.

RESULTS

Intracranial Haemorrhage

This condition was found in 24 cases, 15 of whom were premature babies (Table I). In only 13 of the 24 cases was cerebral haemorrhage the sole cause of death. There were a further 4 cases in whom cerebral haemorrhage was also associated with pulmonary pathology.

There was no difference between premature and full-term babies in the incidence of cerebral pathology.

TABLE I. INTRACRANIAL HAEMORRHAGE (24 CASES)

No.	*Weight in G*	*Subarachnoid*	*Subdural*	*Intra-ventricular and/or intracerebral*	*Pulmonary pathological change*	*Cause of death*
1	700	—	—	+++	—	Cerebral
2	1,000	—	+++	—	—	Cerebral
3	1,000	+		+++	—	Cerebral
4	1,000	—	+++	—	—	Cerebral
5	1,100	—	—	+	Pneumonia	Pulmonary
6	1,200	—	—	++	—	Cerebral
7	1,200	—	—	+	Pneumonia	Pulmonary
8	1,250	+	+++	—	Pneumonia	Cerebral + pulmonary
9	1,500	—	+++	—	—	Cerebral
10	1,500	+++	—	—	Pulmonary haemorrhage	Cerebral + pulmonary
11	1,800	—	—	+++	—	Cerebral
12	1,850	—	—	+	Pneumonia	Pulmonary
13	2,250	—	+	—	Pneumonia	Pulmonary
14	2,300	—	+++	—	—	Cerebral
15	2,300	+	—	—	Pneumonia	Pulmonary
16	2,750	+	—	—	Pneumonia	Pulmonary
17	2,750	++	+++	++	—	Cerebral
18	2,800	++	—	+++	Pneumonia	Cerebral + pulmonary
19	2,900		Traumatized brain			Cerebral
20	3,150	+++	—	+++	Pulmonary haemorrhage	Pulmonary + cerebral
21	3,200	—	—	+++	Pulmonary haemorrhage	Cerebral
22	3,550	—	—	++	—	Cerebral
23	3,700	—	++	—	—	Cerebral
24	5,310	—	+	—	Pneumonia	Pulmonary

Hyaline Membrane Disease

This disease accounted for 33 of the deaths (Table II). The duration of life in the 33 cases varied from 2 to 36 hours, with 25 (76%) of the babies dying within the first 10 hours of life (Fig. 1). The birthweights of these children ranged from 650 G to 2,400 G with 19 babies weighing between 1,000 and 2,000 G. Adverse factors noted in the histories were: birth before arrival in hospital in 15, hypothermia in 9 and ante-partum haemorrhage in 6 instances.

TABLE II. HYALINE MEMBRANE DISEASE (33 CASES)

Weight in G	*BBA*	*Hypo-thermia*	*APH*	*Life in hours*	*Caesarean sections*	*Diabetes*
620	—	—	—	10	—	—
650	—	—	+	2½	—	—
730	+	—	—	7½	—	—
820	—	—	—	6	—	—
870	+	+	—	4	—	—
930	+	+	—	4	—	—
960	—	—	—	2	—	—
960	—	+	—	4	—	—
1,020	—	—	—	18	—	—
1,075	+	—	—	14	—	—
1,075	—	—	—	6	—	—
1,130	+	—	—	5½	—	—
1,160	+	+	—	18	—	—
1,360	+	—	—	12	—	—
1,385	—	—	—	2	—	—
1,440	+	+	—	12	—	—
1,470	+	+	—	4	—	—
1,530	—	—	+	11	—	—
1,530	—	—	+	4	—	—
1,580	—	—	—	10	—	—
1,610	—	—	—	13	—	—
1,640	+	—	—	?	—	—
1,680	—	—	—	7	—	—
1,830	+	+	—	9	—	—
1,870	+	—	—	3½	—	—
1,880	+	+	—	36	—	—
1,980	—	—	—	14	—	—
2,090	+	—	—	4½	—	—
2,200	+	+	+	4	—	—
2,200	—	—	—	8	—	—
2,200	—	—	—	8	—	—
2,260	—	—	+	12	—	—
2,400	—	—	+	4	+	—

BBA = Birth before arrival in hospital
APH = Ante-partum haemorrhage

TABLE III. INTRAPULMONARY HAEMORRHAGE (67 CASES)

No. of cases		Antepartum haemorrhage	Obstructed labour with caesarean section	Cord abnormalities	Hypothermia	Toxaemia	No material cause	Duration of life in hours		
								0–12	13–72	73
Stillbirth	40	21	3	10	0	0	6	—	—	—
Live birth										
Full-term	9	3	5	—	0	1	0	3	5	0
Premature	18	0	0	0	13	0	19	1	14	4

Intrapulmonary Haemorrhage

This condition accounted for 67 perinatal deaths of which only 27 babies were alive at birth (Table III). Of the 40 stillbirths, a maternal history of antepartum haemorrhage was obtained in 21, in 10 delivery had been complicated by a prolapsed umbilical cord, and in 3 there had been obstructed labour with caesarean section. Uncomplicated delivery had occurred in only 6 of these cases.

Of the 27 babies born alive, 5 were born by caesarean section because of pelvic obstruction, in 3 antepartum haemorrhage had occurred and in one case the mother was suffering from toxaemia of pregnancy.

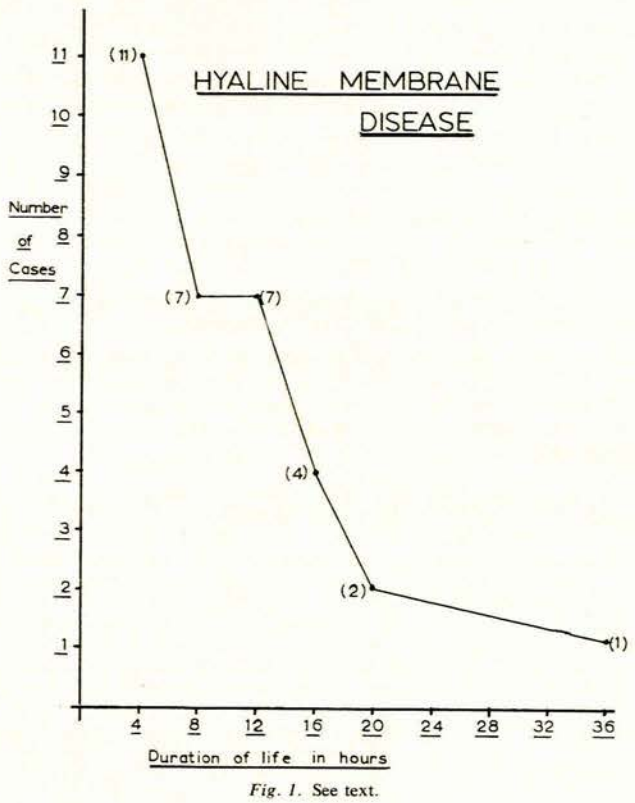

Fig. 1. See text.

Intrapulmonary haemorrhage is usually a rapidly fatal condition and it should therefore be noted that 4 of the infants died within the first 12 hours of life, 19 between 12 and 72 hours after birth, 2 between the third and seventh days and 2 between the seventh and fourteenth days after birth. The last 4 cases were complicated by meningitis in one instance, cerebral haemorrhage in 2 and bronchopneumonia in 2 cases. In the group alive at birth, 18 were premature infants and 13 of these developed hypothermia. Hypothermia was not found in the 9 full-term children dying from intrapulmonary haemorrhage.

Pneumonia

In 54 cases death was due to pneumonia (Table IV). The maternal history in all these cases was significant. There were 12 instances in which babies were born alive after a normal delivery and these were all premature and asphyxiated at birth. Of the 20 babies born alive after complicated delivery, 14 were full term and 6 were premature, and in this group asphyxia at birth was noted in 18. In this group too, there was obstructive labour in 12 cases, antepartum haemorrhage in 3 cases and premature rupture of membranes in 5 cases. Of 11 stillbirths 7 were premature and 4 were full-term deliveries. In this group there was obstructed labour in 8 cases, antepartum haemorrhage in 2 and premature rupture of membranes in 1.

There were 11 cases of babies born before arrival in hospital of which 10 were premature. All were asphyxiated on admission.

TABLE IV. PNEUMONIA (54 CASES)

	No. of cases	Premature births	Full-term	Obstructed labour	APH	Prematurely ruptured membranes	Asphyxiated at birth
Normal deliveries	12	12	0	0	0	0	12
Complicated deliveries	20	6	14	12	3	5	18
Stillbirths	11	7	4	8	2	1	0
BBA	11	10	1	?	?	?	11

Atelectasis of the Lungs

This was the only positive finding in 33 of the neonatal deaths (Table V). All these children were asphyxiated at birth and 27 of them died within 24 hours of delivery. There were 21 who were prematurely born, of whom 19 weighed less than 2,000 G at birth. The 12 babies born at term had been in obstructed labour in 5 instances, of which 2 were delivered by caesarean section.

TABLE V. ATELECTASIS (33 CASES)

Birth-weight in G	Death within 24 hours	Death after 24 hours	Complicated or obstructed labour	Caesarean section	Asphyxiated at birth
1,000	—	2	—	—	2
1,001–1,500	11	—	—	—	11
1,501–2,000	5	1	—	—	6
2,001–2,500	—	2	—	—	2
2,500	11	1	3	2	12
				Total	33

DISCUSSION

Only 4% of the deaths in this series could be explained on the basis of cerebral haemorrhage despite the high incidence of cephalo-pelvic disproportion, precipitate labour and other obstetrical abnormalities which predispose to cerebral birth trauma. In 90% of the deaths there was gross pulmonary pathology and in the remaining 6% no cause of death was found at autopsy. Severe pulmonary pathology is therefore the most common cause of stillbirths and deaths in the Bantu neonate.

Hyaline membrane disease accounted for 13% of the deaths and it was always associated with prematurity. This corresponds closely with the 11·5% incidence in premature babies reported by Miller and Jennison.[2] Our series shows

no relationship between the mode of birth and hyaline membrane disease. Only 1 of the 97 children born by caesarean section during this study died with the disease. This child was, in addition, a premature infant weighing only 2,400 G. The relationship between caesarean section and hyaline membrane disease is controversial. Cantor *et al.*[3] and Tannenburg[4] assessed the incidence of hyaline membrane disease in neonates dying after caesarean section as high as 70%. Snyder[5,6] came to the conclusion that the disease was a definite hazard in babies born by elective caesarean section before the onset of labour. It is our contention that elective caesarean section often produces a premature baby and that it is the factor of prematurity which predisposes to the development of hyaline membrane.

Hypothermia was present in only 9 of the 33 infants with hyaline membrane disease and its significance in the aetiology of the disease is therefore obscure.

Children in this series received the full amount of blood available from the placenta as it is our practice at this hospital to tie the cord only after pulsation has ceased. Moreover, enquiries revealed that in the children born before the arrival of doctor or midwife, the umbilical cord was usually not clamped or cut until the placenta had already delivered. Landau[7,8] suggested that hyaline membrane might result from shock caused by low circulating volume resulting from either blood loss from the infant or from early tying of the umbilical cord. The infants in our series received the full amount of blood available from the placenta and would not, therefore, have been in the position of developing haematogenic shock.

The short life span of our infants with hyaline membrane disease was an unusual finding. In this series 88% of the babies died within the first 16 hours of life. In Snyder's cases[6] the majority (77%) died between 24 and 72 hours after birth, while in the series described by Cantor *et al.*[3] the average duration of life was 26 hours. The cause of early death in our series may be due to the fact that 81% of the babies weighed less than 2,000 G and were therefore smaller and more premature than in the other series described.

Oxygen administration could not have been a cause of hyaline membrane in our cases because it is not the practice in this hospital to nurse any of the children in an oxygen tent or incubator.

In the series of children dying from acute pneumonia the histological examination suggested that the aetiology was bacterial rather than viral. The role of infection in this group is difficult to assess accurately but in the case of the 11 babies who died of pneumonia *in utero* the presence of clear liquor and the absence of evidence of intrauterine infection suggested that the disease was transmitted across the placenta following maternal bacteriaemia, which has been shown to occur in prolonged labour.[9] In fact, in the 11 stillbirths showing evidence of severe pneumonia at autopsy, there was a history of birth difficulty in every case. Of the 32 babies with pneumonia born alive in hospital there was a maternal history of obstructed labour or premature rupture of membranes in 20 instances. In 8 of these cases the foul and infected liquor suggested that the infection was spread directly to the foetus via the liquor. All the remaining 12 babies were premature infants and showed evidence of asphyxia at birth. There is little doubt that prolonged labour from any cause as well as premature rupture of membranes may predispose to either transplacental infection or to an infection acquired before, during, or immediately after birth.

Intrapulmonary Haemorrhage

Infants with intrapulmonary haemorrhage appeared to fall into 3 groups. The first consisted of stillbirths. In many of these cases there was a history of antepartum haemorrhage, prolapsed umbilical cord or strangulation by an umbilical cord. The second group consisted of those full-term babies alive at birth, in whom death occurred between 12 and 72 hours of life. This period corresponds with the period of neonatal hypothrombinaemia and it is significant that there is a high incidence of haemorrhagic disease of the newborn in this community. This is associated with failure of mothers to attend at the antenatal clinic and to receive vitamin K during the antenatal period. Thus the fall of the prothrombin level in the neonate is not prevented.[10] The third group consisted of those babies born alive but who became hypothermic and died with intrapulmonary haemorrhage. All these cases were premature infants and this condition was not seen in the full-term babies. It would appear, therefore, that low body temperatures in premature babies may predispose to pulmonary haemorrhage, especially where there is an increased tendency to the development of haemorrhagic disease of the newborn.[11,12]

In the neonates alive at birth in whom atelectasis was the only positive finding at autopsy, asphyxia was present in all cases during life. Presumably, as a result of intrauterine asphyxia, irreversible anoxic damage to the respiratory centre had taken place and resuscitative measures had therefore failed to produce rhythmical respiratory efforts even in the presence of a beating heart.

SUMMARY

In a perinatal mortality study of 238 Bantu neonates it was found that only 4% of the deaths could be attributed directly to cerebral pathological changes despite the high incidence of cephalo-pelvic disproportion, precipitate labour and other obstetrical abnormalities. Ninety per cent of the deaths resulted from pulmonary abnormality. No relationship was found between hyaline membrane disease and caesarean section, oxygen administration or haematogenic shock but a direct association was found with prematurity in all instances. Intrapulmonary haemorrhage was frequently associated with hypothermia, prematurity and haemorrhagic disease of the newborn. Pneumonia was generally associated with either prolonged labour or premature rupture of membranes. Prolonged labour often caused pneumonia and death *in utero*. All cases of atelectasis were asphyxiated at birth.

This investigation thus confirms the importance of pulmonary pathological changes as a potent cause of perinatal deaths even in a community where cerebral birth trauma might have been expected to have been the predominant aetiological factor.

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