CLASSIFICATION AND CAUSES OF PERINATAL MORTALITY*

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PART II. FACTORS IN PREGNANCY AND LABOUR INFLUENCING PERINATAL MORTALITY

Here some of the factors shown in Table V to be of importance in perinatal mortality are discussed from the clinical aspect.

Maternal Age

There was some increase in the perinatal mortality rate when the mother was over 30 years of age and a sharp rise over the age of 35 years both in primiparae and in multiparae (Table VI). This relationship of maternal age to foetal loss is of course well known (cf. Crosse and Mackintosh, 1954).

TABLE VI.—*Perinatal Mortality Rate in Different Maternal Age Groups Based on* 10,028 *Consecutive Births at U.C.H. Between* 1948 *and* 1955

	Maternal Age in Years					
	Less than 20	20–24	25–29	30–34	35–39	Over 40
No. of births	387	2,772	3,622	2,076	972	199
Percentage death rate	1·8	2·7	2·7	3·9	6·3	8·6

Table V shows that babies in the maceration-only, ante-partum asphyxia, and birth trauma groups had a significantly high proportion of mothers over 35 years. In the case of birth trauma both premature and full-time babies were involved. Any other associations of high maternal age apparent in Table V were not statistically significant. It can be seen that high maternal age leads to foetal loss mainly in the ante-partum stage, but that it also increases the possibility of fatal birth trauma. The way in which high maternal age operates remains obscure. Toxaemia is a known association of pregnancy in mothers over 35 years (Eastman, 1950) and analysis of our figures suggested that while this association could account for some of the deaths in the group of ante-partum death with maceration only it could not account for the increased loss of babies from ante-partum asphyxia and birth trauma with high maternal age. However, the figures relating to this analysis were not statistically significant.

Over the age of 35 birth trauma was not related to any particular complication of labour. Most of the common difficulties were encountered. In particular, birth trauma after an apparently normal labour bore no relationship to maternal age.

Toxaemia and Hypertension

We have based our definition of these conditions on that of Eastman (1950)—that is, blood-pressure readings of at least 140 mm. Hg systolic and/or 90 mm. Hg diastolic on two or more occasions not less than 12 hours apart and before the onset of labour.

TABLE V.—*Clinical Associations of Major Causes of Perinatal Mortality in* 337 *Consecutive Necropsies Expressed as the Percentage Incidence*

Cause of Death	Toxaemia and Hypertension	Maternal Age of 35 or More	Ante-partum Haemorrhage	Diabetes	Post-maturity (42 Weeks or More)	Labour of 24 Hours or More	Other Complications of Vaginal Delivery	Caesarean Section	Proportion of Primiparae	Multiple Pregnancy	Total Cases
Ante-partum death with maceration only	44*	30*	15*	6·6	1·6	—	—	—	55	11*	61
Intra-partum asphyxia (premature)	28	22	17	Nil	—	11	45	5·6	66	11	18
Intra-partum asphyxia (full-time)	19	19	5·4	2·7	25*	35*	41*	2·7	65	11*	37
Birth trauma (premature)	16	37*	11	Nil	—	16	53*	Nil	65	32	19
Birth trauma (full-time)	22	28*	Nil	,,	11	39*	72*	5·5	65	11*	18
Ante-partum asphyxia	42*	31*	53*	5·5	2·8	—	—	—	59	14*	36
Pulmonary syndrome of newborn	22	14	20	Nil	—	7	25	28*	53	11	36
Intraventricular haemorrhage	18	36	9	,,	—	12	27	27	45	9	11
Pneumonia	11	16	5·5	,,	16	11	F.T. 25.* Prem. 50	F.T. 36.* Prem. 12	47	Nil	F.T. 11. Prem. 8
All births	17	12	3·3	0·3	11	17	10	4·5	67	2·6	10,028
Premature births	27	21	12	0·0	—	11	26	8	57	20	593
Full-time ,,	16	11	2·7	0·3	12	17	9	4·3	59	1·6	9,435

* Statistically significant.

The incidence of maternal toxaemia and hypertension was significantly raised in the groups of ante-partum death with maceration only and ante-partum asphyxia. Any other apparent associations in Table V were not statistically significant.

Toxaemia and hypertension are a known cause of premature labour, but once born the "toxaemic" premature baby is no more at risk than the premature infant born of a non-toxaemic mother. This is shown by the fact that the incidence of maternal toxaemia and hypertension in premature neonatal deaths was 17%, compared with 27% in all premature live births. Indeed, these figures suggest that the risk is less, possibly because "toxaemic" babies tend to be small for their age and therefore more mature for a given birth weight than other premature infants. It is worth noticing from Table V that toxaemia and hypertension do not appear to be a cause of the mysterious pulmonary syndrome of the newborn, except in so far as they cause premature labour.

Prematurity apart, the risk of toxaemia and hypertension to the life of the baby appears to operate only in the ante-partum period.

Ante-partum Haemorrhage

For this we have taken Browne and Browne's (1955) definition of haemorrhage from the genital tract occurring before the onset of labour and after the 28th week of pregnancy. The incidence of ante-partum haemorrhage was significantly raised in the groups of ante-partum death

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with maceration only and ante-partum asphyxia. It is of interest that there was an apparently raised incidence of ante-partum haemorrhage in the pulmonary syndrome of the newborn, although the increase was not statistically significant, possibly owing to the relatively small numbers involved.

The nature of the bleeding showed a marked difference in the two groups. In the nine cases of ante-partum haemorrhage associated with foetal maceration only, eight had a small loss, usually extending over a period of days or weeks, the other being a case of type I placenta praevia.

On the other hand, in the 19 cases of ante-partum haemorrhage resulting in foetal ante-partum asphyxia, 15 were severe accidental haemorrhages, one followed an external version, one followed a fall, and only two consisted of slight repeated bleeding. Because of this difference the occurrence of ante-partum haemorrhage in both the maceration-only and the ante-partum asphyxia group does not diminish the validity of their separation into two pathological categories.

Post-maturity

A baby has been considered post-mature when pregnancy has been prolonged 14 days or more beyond the expected date of delivery. The increased risk to the baby of post-maturity is well known (*British Medical Journal*, 1955). Table V shows that in our cases the only significant risk to the baby consisted of an increased liability to intra-partum asphyxia.

The increased rate of fall of oxygen saturation of umbilical-vein blood after the 40th week of pregnancy (Walker, 1954) led us to examine the effect of other factors likely to increase placental insufficiency in post-mature pregnancies. We regard the chief factors to be toxaemia and hypertension and a labour lasting more than 24 hours.

Without these complications, the risk to the baby from post-maturity was no greater than in all non-premature deliveries (Table VII). When, however, the post-mature pregnancy was associated with a labour over 24 hours, a significantly greater risk to the baby was apparent, while with maternal toxaemia and hypertension the increased risk was of borderline significance ($0.1>P>0.05$).

TABLE VII.—*Effect of Toxaemia and Hypertension and Labour Lasting More than 24 Hours in Post-maturity. Ante-partum Deaths are Excluded from All Groups*

	Post-mature Pregnancies Only		Full-time and Post-mature Pregnancies	
	No. of Births	Percentage Perinatal Mortality Rate	No. of Births	Percentage Perinatal Mortality Rate
Cases with toxaemia and hypertension	217	3·1	1,543	1·4
Cases with a labour lasting more than 24 hours ..	237	4·2	1,626	1·8
Cases with neither toxaemia and hypertension nor a labour over 24 hours ..	745	0·9	6,266	0·9

Labour over 24 Hours

Table V shows, as might be expected, a high incidence of labours lasting more than 24 hours in the "full-time" intra-partum asphyxia and birth trauma groups but not in the corresponding premature groups. It is obviously important in considering length of labour in cases of intra-partum foetal death to calculate duration of labour up to the death of the baby and not to its birth. Similarly in assessing the effect of obstetric manœuvres, manipulations performed after the death of the foetus should be excluded. It is obvious that, post-maturity apart, prolonged labour will extend the period of foetal hypoxia that normally occurs and predispose the infant to intra-partum asphyxia. The association of labour over 24 hours with fatal birth trauma may be due to excessive moulding of the head, especially when disproportion delays delivery, or to injuries occurring during efforts made to save the baby's life.

It is of interest that there was no apparent association between prolonged labour and foetal pneumonia.

Complications of Vaginal Delivery

Under this heading are included forceps delivery, breech delivery, breech extraction, other malpresentations, impacted shoulders, and disproportion, all of which may prove traumatic to the baby. Prolapse of the cord and intra-partum haemorrhage, which are apt to interfere with the supply of oxygen to the foetus, are also included.

The incidence of all complications was significantly raised in the "full-time" intra-partum asphyxia, "full-time" and "premature" birth trauma, and "full-time" pneumonia groups (Table V). There was a suggestion that these complications were also associated with the "premature" intra-partum asphyxia and "premature" pneumonia groups, but the figures were not statistically significant. Obviously, complicated labour may lead to birth trauma, but its association with intra-partum asphyxia seems at first sight to throw doubt on the need to separate these two pathological groups. However, in most cases of intra-partum asphyxia in which complications of vaginal delivery occurred they were not of the potentially traumatic variety but those apt to interfere with the foetal oxygen supply. This distinction is set out in Table VIII, in which the figures for full-time babies are significant ($P < 0.001$), but not those for prematures.

TABLE VIII.—*Incidence of Potentially Traumatic and Non-traumatic Complications of Vaginal Delivery in Babies Dying of Birth Trauma and Intra-partum Asphyxia*

	Full-time				Premature			
	Birth Trauma		Intra-partum Asphyxia		Birth Trauma		Intra-partum Asphyxia	
	No.	%	No.	%	No.	%	No.	%
Potentially traumatic complications	14	78	7*	19·5	10	50	6	33
Non-traumatic complications	4	22	29	80·5	10	50	12	67
Total ..	18	100	36	100	20	100	18	100

* Two of these seven cases had a secondary degree of birth trauma.

Therefore, to separate the intra-partum asphyxia and birth trauma groups, rather than merge them into one category of "intra-partum stress," may enable the obstetrician to decide which particular event in labour was responsible for the death of the baby.

In the premature babies the relatively high incidence of non-traumatic delivery in the birth trauma group is explicable by the widely held view that the delicate premature structures are easily damaged by the normal stresses of labour.

Of the six cases of potentially traumatic delivery in the "premature" intra-partum asphyxia group, four had a complication apt to produce asphyxia, one was an uncomplicated breech delivery, and in one foetal distress occurred. Further, in four of the cases doubt exists whether the foetus was still alive when the potentially traumatic delivery occurred. One baby had a slight degree of birth trauma insufficient in itself to account for death.

In all four cases of pneumonia in full-time babies associated with complications of vaginal delivery, these were of the potentially traumatic variety. It is clear that manipulations might result in the introduction of infection. It is likely, however, that these manipulations were associated with pneumonia merely because they were often performed for foetal distress, which is in itself an association of pneumonia. The apparently raised incidence of complicated labour in premature babies with pneumonia was not statistically significant.

Caesarean Section

Table V shows the incidence of caesarean section to be raised in the pulmonary syndrome of the newborn, intra-ventricular haemorrhage, and "full-time" pneumonia

groups. The increase is statistically significant in the case of pulmonary syndrome and "full-time" pneumonia and of borderline significance in intraventricular haemorrhage. All babies in this last category born by caesarean section had, in addition to their cerebral lesion, evidence of the pulmonary syndrome of the newborn.

The pneumonia group is characterized by three features: (1) the association with caesarean section is confined to the full-time babies; (2) the operation was performed for foetal distress in labour in three out of four cases, and in the fourth case an obvious source of infection was present; and (3) the membranes had ruptured before delivery in all three cases showing foetal distress. These circumstances suggest that pneumonia is particularly apt to follow foetal distress occurring at term after the membranes have ruptured and that the association of pneumonia with caesarean section is therefore merely coincidental.

Babies dying of the pulmonary syndrome and born by caesarean section differed from the pneumonia group in that 8 out of 10 were premature. The incidence of caesarean section was raised in the pulmonary syndrome in both full-time and premature babies. Another difference between this group and the pneumonia category is that in all 8 cases of premature pulmonary syndrome the operation was performed before the onset of labour when the membranes were intact. The indications for operation were equally divided between ante-partum haemorrhage, and toxaemia and hypertension.

The association of caesarean section with the pulmonary syndrome of the newborn could be due to the operation itself, especially in relation to prematurity, or to the complications for which the operation was performed (although apparently not toxaemia and hypertension) or to both combined. In any case the precise way in which these factors could operate remains obscure.

Multiple Pregnancy

Table V shows the incidence of multiple pregnancy to be raised in all ante-partum deaths, in "full-time" intra-partum asphyxia and birth trauma, and in premature babies dying of birth trauma, although this last figure is not statistically significant. The presence of multiple pregnancy does not increase the risk of premature babies dying of asphyxia during labour or in the neonatal period from any cause.

The additional hazard of multiple pregnancy in labour is well known. Its influence in ante-partum deaths is not so clear, although the association of toxaemia with multiple pregnancy (Eastman, 1950; Browne and Browne, 1955), may play a part. The mothers of all cases in the ante-partum asphyxia group had toxaemia and hypertension. Of the seven babies in the group of ante-partum death with maceration only one was born of a toxaemic mother, another set of twins was associated with maternal hereditary thrombasthenia, and the remainder were born after normal pregnancies.

Of the 24 twins that died in the intra-partum and post-partum periods, 12 were first-born and 12 second-born. This is at variance with the often expressed view that second twins are more at risk, particularly because of birth trauma and intra-partum asphyxia. In fact, both these pathological groups contained exactly similar numbers of first and second twins.

Diabetes

There were 29 cases of maternal diabetes in our 10,028 deliveries, 6 of whom were delivered by caesarean section at 37 to 40 weeks for other maternal complications. The remainder were allowed to go into spontaneous labour (which occurred in 70% by 39 weeks). There were no neonatal deaths, but seven stillbirths, the foetal loss being 24%. This is similar to the foetal loss of 27% reported by Peel (1955), who favours termination of pregnancy before the end of the 37th week and whose fatal cases included a proportion of deaths from hyaline membrane in the neonatal period.

Of our seven stillbirths, three were examples of ante-partum death with maceration only, two of ante-partum asphyxia, one of congenital malformation, and one of intra-partum asphyxia. This last death was directly attributable to the large size of the baby.

PART III. CLINICAL PICTURES IN BABIES DYING IN THE NEONATAL PERIOD

This section is concerned with the correlation of clinical and post-mortem findings and the differential diagnosis of the common conditions causing death. 221 neonatal deaths were studied. They occurred among 9,250 full-time and 593 premature babies born in the Obstetric Hospital, together with 201 infants, all premature save one, admitted from home or other hospitals after birth.

The main causes of death and their distribution according to the babies' birth weights are shown in Table IX. Over four-fifths of the deaths were accounted for by the pulmonary syndrome (27%), birth trauma (18%), congenital malformations (15%), pneumonia (12%), and intraventricular haemorrhage (10%). Excluding congenital malformations, it was found that the clinical pictures of these four conditions were often similar and consisted essentially of one or more of three symptom-complexes. These were poor condition at birth, respiratory difficulty, and cerebral irritation.

TABLE IX.—*Mortality by Birth-weight Groups, and Cause of Neonatal Deaths, Among Babies Born in U.C.H. Obstetric Hospital and Those Admitted After Birth*, 1948–55

Birth Weight (g.)	Live Births	Deaths		Causes of Death Expressed as Percentage of Total Deaths					
		No.	%	Pulmonary Syndrome	Intra-ventricular Haemorrhage	Pneumonia	Intracranial Birth Trauma	Congenital Mal-formations	Other
Up to 1,000	52	49	94	23	12	18	16	2	29
1,001–1,500	89	46	51	44	15	4	15	9	13
1,501–2,000	203	35	17	43	20	3	14	14	6
2,001–2,500	449	27	6	26	0	15	19	19	21
Over 2,500	9,251	64	0·7	9	2	17	24	28	20
Total ..	10,044	221	2·1	27	10	12	18	15	18

Poor Condition at Birth

More than half the babies showed this feature. The diagnosis was made when the infants were limp and cyanosed, without the usual response to external stimuli; many also showed pallor of the skin. Respiration consisted at first of irregular gasps, and usually their onset was delayed for more than 30 seconds after birth. The clinical course of fatal cases took one of three forms. Some infants died after a period of gasping. In others, irregular gasping gradually or rapidly gave place to normal rhythmical breathing, but after a varying period respiratory difficulty returned. Other babies developed rhythmical breathing, but persistent respiratory difficulty was shown by the grunting character of the respirations. In the last four years of the eight under survey, resuscitation has been attempted by the administration of intragastric oxygen and rocking. Prior to that time oxygen was given by nasal catheter, except in the severest cases, when the intratracheal route was used.

Respiratory Difficulty and Cyanosis

This occurred sooner or later in every case, and was shown at first by unduly rapid respirations. An expiratory grunt was often present. Later there was marked irregularity of respiratory rhythm with apnoeic periods, and, finally, the gasping respirations of severe hypoxia developed.

Cyanosis, either persistent or in attacks, was often present from an early stage. This central cyanosis has to be distinguished from the peripheral type. Cyanosed extremities are common in normal babies during the first few hours of

life, and result from slow peripheral circulation and polycythaemia. The pink colour of the skin of the trunk and normal respiratory movements should prevent confusion with central cyanosis. However, if doubt persists, one limb may be warmed and the skin will then become pink if the cyanosis is peripheral in origin.

Examination of the chest in cases with respiratory difficulty showed varying degrees of inspiratory recession of the lower ribs and sternum, impaired percussion note over the lungs, diminished air entry, and sometimes inspiratory rales.

Cerebral Irritation

The major sign in this group was convulsions, often associated with other indications of cerebral irritation. Less commonly, signs of cerebral irritation occurred without convulsions. These signs included excessive wakefulness, contrasting with the normal baby, who sleeps when not feeding, restlessness, irritability when disturbed, shrill high-pitched cry, tense anterior fontanelle, and alterations of muscle tone, usually spasticity of the limbs or trunk muscles, but sometimes hypotonia.

Convulsions were 17 times more common in premature infants, with an incidence of 4.2% compared with 0.25% in full-time babies. Although sometimes merely a terminal phenomenon, they often occurred 24 hours or more before death.

In only 24% of fatal cases of cerebral irritation was post-mortem evidence of intracranial trauma found, while in 63% the only abnormal findings were in the lungs. In most of the latter group the cerebral signs are probably attributable to the effects of hypoxia and carbon dioxide retention on the brain and cerebral vessels. Among the infants who develop a tense fontanelle are some in whom right-sided cardiac failure, which is accompanied by an increase of cerebrospinal fluid pressure (Friedfeld and Fishberg, 1934), seems to be an important factor. These infants have enlarged liver, and in non-fatal cases of cerebral irritation we have sometimes observed a diminution of liver size and a lessening of fontanelle tension to precede improvement in the babies' condition.

Pulmonary Syndrome of the Newborn

The pulmonary syndrome accounted for 27% of neonatal deaths. Approximately 10% of the babies had a birth weight of over 2,500 g., and these formed 10% of all deaths in full-time infants. Pulmonary syndrome showed its highest incidence as a cause of death (43%) in babies weighing from 1,000 to 2,000 g. at birth. The condition was particularly common after caesarean section and caused 45% of neonatal deaths following this method of delivery.

One-half of the babies were in poor condition at birth, and thereafter showed persistent respiratory difficulty. The condition of the remainder usually deteriorated within 12 hours of birth; indeed, only 3 of the 53 premature infants—two with extensive pulmonary haemorrhage and one with hyaline membrane—first developed symptoms over the age of 12 hours. From the corresponding figure for pneumonia, intraventricular haemorrhage, and intracranial birth trauma, which are given below, it will be seen that pulmonary syndrome is the least likely diagnosis of the four in a baby who first falls ill after 12 hours of life. A sign in favour of a diagnosis of pulmonary syndrome was the blood-stained frothy mucus often produced by infants in whom the main lesion was pulmonary haemorrhage or oedema.

The incidence of cerebral signs was noteworthy. Of the 59 cases, 20 (34%) developed convulsions and a further six cerebral irritation without convulsions. This illustrated the difficulty in differential diagnosis of pulmonary syndrome from intracranial birth trauma. Cerebral irritation in the former condition was more likely to occur over the age of 12 hours, whereas in birth trauma the opposite was true; but this fact is of little value in diagnosis of the individual case.

Donald and Steiner (1953) have described a sequence of radiological changes in the lungs which indicate the presence of a hyaline membrane. However, in our series, such membranes were found in 42% of babies with intraventricular haemorrhage and in 44% of premature infants with intracranial birth trauma. Therefore the presence of characteristic radiological changes of hyaline membrane does not exclude the presence of other pathology. In our opinion the main value of radiology is twofold. Firstly, in babies who recover from respiratory difficulty, it may indicate whether or not a hyaline membrane was present. Secondly, it will exclude the presence of congenital malformations, such as diaphragmatic hernia, which is mentioned below, and also of a pneumothorax.

Deaths occurred after an illness lasting from a few hours up to nearly three days; only one baby was alive at the age of 3 days, and she died 12 hours later.

Intraventricular Haemorrhage

This condition accounted for approximately 10% of the neonatal deaths, but was confined almost entirely to babies weighing less than 2,000 g. at birth. A similarity between the clinical picture of this condition and that of the pulmonary syndrome was to be expected, because 62% of primary deaths from intraventricular haemorrhage showed evidence of the pulmonary syndrome as well. However, it might be thought that all cases of intraventricular haemorrhage would show cerebral signs, especially a tense anterior fontanelle, but in our experience such signs have been no more frequent than in the pulmonary syndrome occurring alone.

Approximately half the babies were in poor condition at birth, and in 18 out of the 21 the onset of respiratory symptoms was before 12 hours of age. Two-thirds (14 out of 21) died in the first 24 hours of life and it was rare for survival to exceed three days, although one baby lived for 13 days, having occasional cyanotic attacks from the age of 2 days.

It will be clear that there was no distinguishing feature clinically between babies dying from intraventricular haemorrhage and those with the pulmonary syndrome.

Pneumonia

Pneumonia occurred in 12% of deaths, and was seen most often in previable and full-time babies. Three-fifths of the infants were in poor condition at birth, but some appeared to recover after resuscitation, only to develop further respiratory difficulty after an interval of up to several days. Five of 11 full-time and 3 of 16 premature infants (30% of the total cases) were free of respiratory distress in the first 12 hours of life.

It is important to note that cerebral signs were a feature in 9 of the 27 cases, 6 of whom had convulsions.

Over half the babies (15 out of 27) died in the first day, half (8) of these being previable. Seven babies (25%) lived over three days, but five were full-time infants, so that this tendency to longer survival probably does not represent a true difference from cases of pulmonary syndrome occurring mostly in premature babies.

Possible predisposing factors to pneumonia in these infants were investigated. The mothers of five babies suffered from an acute infection during or immediately preceding labour, while in another five cases the membranes had ruptured more than 18 hours before delivery. Three more babies were known to have had foetal distress during delivery, and another 11 were in poor condition at birth. Thus in 24 out of 27 deaths from pneumonia there had been maternal infection, rupture of the membranes for a considerable period before birth, or evidence of foetal hypoxia which might have led to the inhalation of infected material during delivery. After the first five and a half years of the period under survey, we considered that maternal infection, premature rupture of the membranes, foetal distress, and poor condition at birth should be indications for the

administration of prophylactic antibiotics to the baby, even in the absence of clinical signs. Finally, in view of the difficulty of making a clinical diagnosis of pneumonia, all babies who showed respiratory difficulty after birth were treated with antibiotics in addition to any other method used. Penicillin and streptomycin were given by intramuscular injection.

Before following these principles a primary diagnosis of pneumonia was made in 23 out of 154 deaths (15.6%), whereas subsequently the figure has been 4 out of 67 deaths (6%). Further, two of the last four babies with pneumonia died within 15 minutes of birth before antibiotics could be given.

Intracranial Birth Trauma

This was found in 18% of deaths. Among full-time babies the majority (13 out of 15) were in poor condition at birth and eight died in the first hour of life. In contrast, although 19 of the 25 premature infants were in poor condition, only two died within an hour. The common causes of death within an hour of birth were intracranial birth trauma and intra-partum asphyxia. A history of a forceps delivery, especially if a difficult one, breech delivery, or breech extraction was found to be strongly suggestive of intracranial birth trauma.

Among cases surviving over one hour, less than half of the premature babies developed cerebral irritation, but respiratory difficulty was a constant feature and almost always the presenting sign, frequently dating from birth (in three the first symptoms occurred after the age of 12 hours). The clinical picture was therefore indistinguishable from that of the pulmonary syndrome, intraventricular haemorrhage, or pneumonia. The majority of full-time infants surviving more than one hour developed cerebral signs, but again always accompanied by respiratory disturbances. The birth histories of these babies were of much less help in diagnosis, because the delivery had been apparently normal in many cases.

Three-fifths of the 40 babies died in the first day, and only three survived for more than three days.

Differential Diagnosis of the Pulmonary Syndrome, Intraventricular Haemorrhage, Pneumonia, and Intracranial Birth Trauma from other less Common Conditions

It will be clear that it is often impossible to distinguish between birth trauma, the pulmonary syndrome, pneumonia, and intraventricular haemorrhage in premature babies, and between the first three in full-time infants. However, the four conditions may be confused with less common abnormalities, including some congenital malformations which may cause respiratory difficulty in the first three days of life. The differential diagnosis from certain malformations is particularly important. Oesophageal atresia and diaphragmatic hernia, for example, are often amenable to immediate surgical treatment.

Oesophageal atresia should be suspected when an infant brings up an excess of frothy mucus for several hours after birth or when the mother has had hydramnios. Diaphragmatic hernia usually causes marked displacement of the cardiac impulse and absent air entry on the affected side of the chest, but sometimes the condition is revealed only by radiology. Congenital heart disease should be considered when there is central cyanosis out of proportion to the degree of respiratory difficulty, or dyspnoea with rales in the lungs and enlargement of the liver.

In another small group of babies poor condition at birth is due to foetal haemorrhage from placental incision during caesarean section for anterior placenta praevia (Butler and Martin, 1954). Correct diagnosis is important, because blood transfusion may be life-saving.

Finally, two rare conditions—bilateral renal agenesis and massive adrenal haemorrhage—have caused difficulty in diagnosis. Renal agenesis may be suggested by the characteristic facies (Potter, 1952), but adrenal haemorrhage is not usually suspected during life.

Prognosis

In view of the difficulty in distinguishing between the pulmonary syndrome of the newborn, intraventricular haemorrhage, pneumonia, and intracranial birth trauma in life, the prognosis of these conditions individually is uncertain. The problem in practice is to estimate the prognosis of a baby showing respiratory difficulty which may be due to one of these four conditions.

In premature infants the birth weight is an important factor in prognosis. Mortality in birth-weight groups in the present series is shown in Table IX and requires no further comment. The second factor is the time that the baby has survived. In general, prognosis is much improved when the fourth day of life is reached. Thus in fatal cases, 87% of 31 full-time babies and 96% of 113 premature infants died before the end of the third day. The next factor is the degree of inspiratory recession of the lower sternum. Severe recession, producing a funnel-shaped deformity, was usually unfavourable. Finally, the occurrence of convulsions was a bad prognostic sign, especially in premature infants, the mortality being 79% as opposed to 42% in full-time babies (Table X). The total death rate from convulsions in the neonatal period was 63%, which compares unfavourably with that of Burke (1954), whose rate was 37.5%, but the proportion of premature infants was two and a half times greater in our series.

TABLE X.—*Mortality in Babies With Convulsions*

	Full-time	Premature	Total
Total No. of cases	24	33	57
Died No.	10	26	36
Died Percentage	42	79	63

Conclusion

It has been shown that the four conditions (pulmonary syndrome of the newborn, intraventricular haemorrhage, pneumonia, and birth trauma) which caused two-thirds of all neonatal deaths in our series could not usually be distinguished in life. In a few cases consideration of the birth history and the signs shown by the baby, including their time of onset, strongly indicated the correct diagnosis. A vital task for the doctor confronted with a baby showing respiratory difficulty in the first hours of life is to exclude the presence of certain congenital malformations which may be amenable to surgical treatment.

It is important to note that cerebral irritation, even with convulsions and a tense anterior fontanelle, does not necessarily mean that intracranial trauma has occurred, and that such cases may not be hopeless.

Finally, it will be clear that, congenital malformations having been excluded, all babies showing respiratory difficulty, with or without cerebral irritation, in the first few days of life should receive any specific therapy that is known for the four conditions mentioned above. Thus, in addition to symptomatic treatment, all such infants should be given antibiotics in case pneumonia is present, and vitamin K (1 mg.) to try to prevent or diminish haemorrhage. Similarly, all will have to receive any treatment that may be discovered in the future for the pulmonary syndrome of the newborn.

SUMMARY

The post-mortem and histological examination of 406 stillbirths and neonatal deaths has enabled these deaths to be divided into 11 pathological categories forming a classification of perinatal mortality. The importance of necropsy is discussed. Each pathological group is defined and the diagnostic features are described.

The introduction of the term " pulmonary syndrome of the newborn " to include hyaline membrane, intra-alveolar haemorrhage, and pulmonary oedema is explained. Strict criteria for the diagnosis of intra-

cranial birth trauma have been used, and the value of distinguishing such cases from the intra-partum asphyxia group is stressed. A diagnosis of prematurity or previability is considered undesirable, and was made in only 2% of the present series.

The clinical associations of each category are enumerated, and the aetiology is discussed. In most cases there was a specific association between a given pathological group or groups and at least one feature of pregnancy or labour. Thus, the classification is not merely academic, but should also be of practical value to the obstetrician and paediatrician. It is felt that the general adoption of such a system throughout the country would assist the collation of valid statistics on the causes of perinatal mortality.

In addition to the classification of death, certain factors in pregnancy and labour have been re-examined in order to determine in what way and at which stage they cause foetal and neonatal loss.

Finally, a study has been made of some clinical aspects of babies dying in the first week of life. The pulmonary syndrome of the newborn, birth trauma, pneumonia, and intraventricular haemorrhage, which together caused two-thirds of the neonatal deaths, could not usually be distinguished during life. Successful prophylaxis of pneumonia during the latter part of the period covered by this study is described.

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"English is now more widely used and studied throughout the world than it, or any other language, has ever been before. It has been estimated that over half of the literate population of the world speak English as a first or second language, or use English as an indispensable instrument for vocational purposes, or are studying (or have studied) English. It is increasingly the international language of science and technology. In recent years about half of the world's output of literature on scientific research has been published in English. To a degree hitherto unknown it is recognized, and not only in the Western world, as the language of opportunity, of affairs, and of international communication."—Annual Report (1955–6) of the British Council.

RELIEF OF NEUROLOGICAL SYMPTOMS AND SIGNS BY RECONSTRUCTION OF A STENOSED INTERNAL CAROTID ARTERY

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In 1954 Eastcott, Pickering, and Rob reported the successful resection of an occluded segment of the internal carotid artery with reconstruction by a direct anastomosis between the common and internal carotid arteries. The patient, a woman now aged 69, is fit and well and has not suffered any further neurological symptoms. Since then the carotid arteries of 16 patients with various abnormalities have been operated on by the staff of the surgical unit at St. Mary's Hospital, London, with encouraging results. The object of this paper is to report the complete recovery of a 56-year-old man who before operation had definite pyramidal signs, was unable to write properly, and whose speech was slurred. The case reported in 1954 was, we believe, the first instance of successful reconstruction of an occluded internal carotid, and we also believe that the case recorded here is the first report of complete recovery of definite and persistent neurological signs due to internal carotid occlusion.

Clinical History

The patient, aged 56, was captain of a large ocean liner. In October, 1955, he felt a sudden lack of control of the right arm and leg, accompanied by tingling over the right side of his body which felt strange in a way he could not describe. He could walk during this episode and the symptoms cleared after about ten minutes. Two weeks later a second and similar attack occurred, lasting this time for less than five minutes, and two weeks after that his third attack occurred.

On January 23, 1956, five minutes after getting up from bed and while shaving, he suddenly lost control of his right arm and leg and found that his speech had become slurred. He could walk, but he staggered to the right; he was able to get back into bed. Neither in this nor in the three previous attacks did he suffer from impairment of consciousness or visual disturbance. He was transferred to the Royal Perth Hospital in Australia, where Dr. J. C. Anderson saw him and reported as follows: "On examination: slight cyanosis, pulse 100 a minute; right upper limb showed weakness of grip and of flexion but good power of extension. Right lower limb showed weakness of all movements with poor co-ordination in heel-to-toe test. Knee-jerks present, plantar response flexor. Tongue, no deviation. Eyes, pupils equal and react to light. Cardiovascular system, heart sounds muffled, blood pressure 150/80. Three hours later his speech was completely coherent and he was able to write clearly. All the cranial nerves appeared normal except for a dulling of sensation over the right cheek and forehead. Slight deviation of mouth to the right. Knee-jerks, right brisker than left. Plantar response flexor. Blood pressure 165/110."

Six hours later—that is, nine hours after his initial examination, Dr. Anderson reported: "Condition deteriorating.