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CLASSIFICATION AND CAUSES OF PERINATAL MORTALITY

BY

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This paper presents two sets of data. The first set concerns the pathological definitions of the conditions causing perinatal death. The definitions form the basis for the second set of data, which deals with the clinical associations, maternal and foetal, of these causes of death. The object of this method of classification is to correlate pathological and clinical findings so that a clearer picture of the causes of perinatal death will be seen, and from this to prevent such deaths.

Perinatal mortality is a term used for both stillbirths and neonatal deaths during the first week (Crosse and Mackintosh, 1954). Most published studies of perinatal mortality have been concerned with the causes of such deaths and with classifying them. The earliest classifications were clinical (Holt and Babbitt, 1915; McQuarrie, 1919; Holland and Lane-Claypon, 1926), the death of the baby being ascribed to maternal diseases or obstetric complications present. As routine post-mortem examinations came to be performed these classifications based on presumed aetiology were superseded by pathological or clinico-pathological systems based on the morbid anatomical findings (Browne, 1922; Palmer, 1928; Serbin, 1928; D'Esopo and Marchetti, 1942; MacGregor, 1946; Labate, 1947; Potter, 1952; Morison, 1952). Routine histological study of the post-mortem material extended the scope of the pathological contribution in several directions.

The present paper puts forward a pathological classification of stillbirths and neonatal deaths (perinatal mortality) based on the routine morbid anatomical and histological study of 337 consecutive necropsies performed on babies born at U.C.H. Obstetric Hospital between January, 1948, and September, 1955. The total number of births during this period was 10,028. In addition, the paper deals with a further 69 neonatal deaths in babies admitted to the U.C.H. Premature Baby Unit from outside sources.

This classification shows a correlation between some abnormalities of pregnancy and labour, and some maternal factors, and the cause of death of the infant. For this reason a part of the paper is devoted to a study of these obstetric factors. The intention, therefore, is to relate the stages of pregnancy and labour and the types of maternal complication to the perinatal mortality.

Finally, it was thought desirable to include a section on a clinical study of neonates prior to their death, so that their signs and symptoms could be compared with the post-mortem findings.

PART I. CLASSIFICATION

The conditions which cause perinatal death are set out in Table I.

TABLE I.—*Causes of Death in* 337 *Consecutive Necropsies Performed on Stillbirths and Neonatal Deaths Delivered at U.C.H. Between* 1948 *and* 1955

Cause of Death	Still-births	Prema-ture N.N.D.	Full-time N.N.D.	Total Cases	%
Ante-partum death with maceration only ..	61	—	—	61	18
Intra-partum asphyxia ..	44	5	6	55	16·3
Congenital malformations	24	11	17	52	15·5
Birth trauma	8	14	15	37	11
Pulmonary syndrome of the newborn	—	30	6	36	10·7
Ante-partum asphyxia ..	36	—	—	36	10·7
Pneumonia	—	8	11	19	5·6
Erythroblastosis foetalis ..	8	3	4	15	4·4
Intraventricular haemorrhage	—	10	1	11	3·3
Miscellaneous	4	1	3	8	2·4
Previability	—	7	—	7	2·1
Total	185	89	63	337	100

Ante-partum Death, with Maceration Only

Definition.—This group comprises those cases of stillbirth in which foetal death was known to have occurred before the onset of labour and which showed no pathological evidence of asphyxia or of other lesions sufficient to account for death.

Diagnosis.—The time of death was established by the record of the cessation of the foetal heart and by the extent to which maceration had progressed in relation to the time of onset of labour. A prolonged interval, up to four weeks in duration, between foetal death and delivery was characteristic of this group, and maceration was always present. Post-mortem examination revealed a foetus usually smaller than its expected size. The only naked-eye findings in the foetus were those of maceration. In order of occurrence after foetal death these were softening and peeling of the skin, blood-stained effusions into the serous cavities, separation of the dura from the skull, separation of the cranial bones, and

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mummification. Asphyxial haemorrhages were absent. The placenta was very often small (see Table II), sometimes weighing less than 100 g. at 32 weeks. Histologically, the organs showed only post-mortem change. The placenta was often characterized by ischaemia of the villi. In view, however, of possible involution following foetal death, the significance of these findings remains to be assessed.

TABLE II.—*Mean Foetal/Placental Ratio and Placental Weight at Different Periods of Gestation in Ante-partum Death with Maceration only and in Fatal Birth Trauma. (Twins Excluded from Both Groups)*

	Gestation in Weeks					
	28–31		32–35		Over 36	
	Foetal/Placental Ratio	Placental Weight	Foetal/Placental Ratio	Placental Weight	Foetal/Placental Ratio	Placental Weight
Ante-partum death with maceration only	5·1	171 g.	5·0	297 g.	4·7	502 g.
Birth trauma ..	4·1	356 ,,	4·3	443 ,,	5·2	674 ,,

Differential Diagnosis.—The condition is differentiated from other causes of ante-partum death—for example, asphyxia, erythroblastosis, congenital malformations, and syphilis—by the absence of the characteristic lesions associated with these diseases and pathological states (see below). In other words, ante-partum death with maceration only is diagnosed and classified partly by exclusion.

Clinical Associations.—These are summarized in Table III and set out in detail in Table V, which will appear in Part II of this paper. There was a high

TABLE III.—*Categories of Perinatal Mortality and Their Statistically Significant Maternal Associations*

Ante-partum death with maceration only	Toxaemia and hypertension, high maternal age, slight repeated ante-partum haemorrhage, diabetes, multiple pregnancy
Ante-partum asphyxia ..	Toxaemia and hypertension, high maternal age, severe ante-partum haemorrhage, diabetes, multiple pregnancy
Intra-partum ,, ..	Post-maturity, long labour, complicated vaginal delivery especially non-traumatic, multiple pregnancy, prematurity
Birth trauma	High maternal age, long labour, complicated vaginal delivery, especially traumatic, multiple pregnancy
Pulmonary syndrome of the newborn	Caesarean section, prematurity
Intraventricular haemorrhage	Prematurity
Pneumonia	Complicated vaginal delivery, caesarean section, prematurity
Haemolytic disease of the newborn	Blood-group incompatibility
Miscellaneous	Heterogeneous
Previability	Prematurity
Congenital malformations ..	Heterogeneous

proportion of mothers over 35 years of age (30%), a high incidence of toxaemia and hypertension (44%), of ante-partum haemorrhage (15%), of diabetes (6.6%), and of multiple pregnancy (11%). The ante-partum haemorrhages usually consisted of a small loss spread over several days or even weeks. Of the pregnancies in the group, 20% were free of obstetrically recognized complications. In addition, Table II shows that, on the average, babies in this group had smaller placentae than those stillbirths and neonates dying of birth trauma. The difference in average placental weight is also reflected in the ratios of placental and foetal weight in the two groups, the ratio being higher in the foetuses dying with maceration only.

Aetiology.—The most plausible hypothesis to explain the majority of deaths in this group, and one that is supported by the placental weights set out in Table II, is that of placental insufficiency. This implies inadequate supply of oxygen and nutriment to the foetus and an inadequate removal of metabolites, too gradual in its effect to cause sudden death with asphyxial haemorrhages, but sufficient to lead to impairment of growth and, as the foetal needs increase, to cause death. In the cases associated with repeated small ante-partum haemorrhages it may be that small areas of placental separation occur, leading again to a condition of placental insufficiency. If the hypothesis of placental insufficiency is valid it would seem that toxaemia of pregnancy is related to it in some way at present obscure, since toxaemia and small placentae often coexist.

Ante-partum Asphyxia

Definition.—This group comprises those cases of stillbirth in which foetal death occurred before the onset of labour, and in which evidence of asphyxia was found.

Diagnosis.—Time of death was established by the methods outlined in the previous section. Early maceration was usual in the ante-partum asphyxia group, but advanced maceration due to a long interval between death and delivery was the exception. Evidence of asphyxia has been summarized by Potter (1952). In practice it was found to take two main forms: (1) petechiae or ecchymoses on visceral surfaces and serous membranes, especially on the lungs; and (2) massive inhalation of amniotic contents, especially meconium. The most significant finding was the presence in large numbers of rounded ecchymoses, 1–3 mm. across, on the surface of the lungs and parietal pleura (Tardieu's spots). These were present in 83% of the ante-partum asphyxia group. The association of these ecchymoses with ante-partum haemorrhage and premature placental separation, described by Potter (1952), was borne out by our experience. Of cases with pleural ecchymoses, 50% had such a history.

In the remaining cases of ante-partum asphyxia the haemorrhages on the surface of the lungs consisted only of smaller petechiae. These were sometimes confluent and occasionally localized to one special part of the lung surface, such as the paravertebral region. In one or two cases ecchymoses were found in the serosal surface of the bowel. Ecchymoses on the cardiac surface were almost always present. In small numbers such cardiac ecchymoses are very common and are of doubtful significance. In large numbers, however, they have a correlation with other asphyxial lesions and with a history of foetal asphyxia or premature placental separation.

Another type of surface haemorrhage found in the ante-partum asphyxia group (26%) was the variety consisting of localized effusions of blood in the pia arachnoid of the cerebral hemispheres. Occasionally one or more of these effusions ruptured into the subdural space and led to the presence of a small quantity of free blood in the cranial cavity.

The placenta in the ante-partum asphyxia group often showed evidence of premature separation with adherent blood clot or crater formation (abruptio placentae). Small infarcted placentae with evidence of premature separation were also seen, especially in cases associated with toxaemia of pregnancy. In 25% of cases the placenta was apparently normal.

Histologically, most of the organs, especially the liver, kidneys, and lungs, were congested. The lungs in addition often showed massive inhalation of amniotic contents. The finding of meconium in the air spaces is especially significant, as meconium is often passed into the amniotic cavity when the foetus is short of oxygen. The placenta frequently showed villous ischaemia or excessive fibrin deposition or hyalinization, especially if toxaemia was present, but many were histologically normal.

Differential Diagnosis.—When confronted with a stillbirth showing signs of asphyxia, it may be difficult to decide whether death occurred before or during labour. If

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maceration is minimal or absent, it may be impossible to make this decision without a record of the foetal heart sounds.

Clinical Associations.—The clinical associations of ante-partum asphyxia are summarized in Table III. The most important was severe ante-partum haemorrhage occurring in 50% of the cases. The other significant maternal associations of ante-partum asphyxia were the same as in the previous group. Two other associations of ante-partum asphyxia, not shown in Table III, were external version under general anaesthesia and false labour. Each of these conditions occurred in 5.5% of cases of ante-partum asphyxia, which is probably significant in both instances.

Aetiology.—It seems very likely that deaths in this group are usually due to a sudden cessation of the supply of oxygen to the foetus. The most common cause of this catastrophe is premature placental separation with ante-partum haemorrhage. In cases not associated with this complication, and with the possible exception of two instances of false labour, the immediate cause of oxygen deprivation was usually obscure. Many may be due to placental separation that remains occult to the end. Cord obstruction seems unlikely *in utero* before labour, and in any case does not normally lead to large ecchymoses on the viscera but rather to fine petechiae (Potter, 1952).

Intra-partum Asphyxia

Definition.—This category comprises infants dying during labour or shortly after birth and showing evidence of asphyxia with no other pathological condition sufficient to account for death.

Diagnosis.—Time of death in stillbirths was established by the clinical record of the foetal heart sounds and by the degree of maceration, usually minimal or absent, depending on the duration of labour. Practically all the neonatal deaths in the group occurred within one hour of birth.

Of the cases of intra-partum asphyxia, 33% were premature. The changes of asphyxia were similar to those described in the previous section, but were often associated with petechiae of the skin of the limbs, face, and trunk, especially where difficult labour was present. The haemorrhages in the pleura took the form of rounded ecchymoses in 43% of cases and of small petechiae in the remainder. Localized ecchymoses in the pia arachnoid were more common than in ante-partum asphyxia, being present in 33% of the babies examined. The placenta was usually normal in size and appearance.

Histologically, the stillbirths revealed changes similar to those seen in the ante-partum asphyxia group, except that the placenta was usually normal. In those babies surviving for a short time the lungs showed areas of early resorption atelectasis with congestion, alveolar-wall cohesion, and overdistension of terminal air spaces.

It is babies of the intra-partum asphyxia group that most commonly require differentiation into those that have had a "separate existence" and those that have been born dead. With experience, histological study of the lungs, based on the above criteria of resorption atelectasis, makes this decision fairly easy except in cases living less than one or two minutes. The atelectasis seen in stillborn babies is characterized by separation of the alveolar walls to a uniform degree and by absence of vascular congestion.

Differential Diagnosis.—Stillborn cases of intra-partum asphyxia must be differentiated from the ante-partum asphyxia group by the history and by the degree of maceration, if any. Neonatal deaths due to intra-partum asphyxia can be distinguished from the "pulmonary syndrome" by histological examination of the lungs, which will also serve to exclude pneumonia in both stillbirths and neonatal deaths. If evidence of birth trauma is present, death would normally be attributed to this, unless it is slight in degree—for example, a small area of fraying in the tentorium with only traces of bleeding.

Clinical Associations.—The incidence of the various complications of pregnancy in mothers whose babies were premature and died in intra-partum asphyxia was no greater than in all premature births. The full-time pregnancies similarly showed no significant association, except a raised rate of post-maturity—that is, a gestation of 42 weeks or more (27%). During parturition there was a significant association with a labour of 24 hours or more prior to foetal death (38%), with multiple pregnancy (11%), and with a history of complications of vaginal delivery, which were almost always those liable to deprive the foetus of oxygen—for example, prolapsed cord and intra-partum haemorrhage. Complications prone to cause foetal trauma were much less in evidence (see Part II).

Aetiology.—Various causes played their part. Post-maturity, long labour, and complications apt to cause asphyxia were the major factors. The common denominator is probably abnormal prolongation of the normal state of asphyxia present during labour, or interference with the supply of oxygen and removal of carbon dioxide and metabolites during parturition.

Birth Trauma

Definition.—A condition found in stillbirths and neonatal deaths in which there was haemorrhage within the cranial cavity, accompanied by evidence of damage to intracranial structures or to the skull, or, alternatively, similar haemorrhage and damage elsewhere in the body.

Diagnosis.—There were 48 cases in all, 37 born in U.C.H., of whom half were premature. In only two instances, both born prematurely outside U.C.H., was the trauma other than intracranial, involving the thorax and abdomen.

Within the cranium it was possible to diagnose the source of the bleeding with varying degrees of precision. The commonest variety was a tear of the tentorium cerebelli, present in 61% of the cases, and extending antero-medially to involve the straight sinus, or associated with rupture of the tributaries of the vein of Galen. Next in frequency came tearing of the falx cerebri, present in 15% of cases, and associated with damage to the inferior sagittal sinus or, less frequently, the superior end of the straight sinus. Less commonly the great vein of Galen itself was involved, together with its major tributaries. This type of lesion was found in 11% of the cases. Birth trauma produced subdural haemorrhage due to tearing of the meningeal veins in 4%, and a further 4% exhibited sub-tentorial haemorrhage alone, presumably due to damage to minor veins. One infant was found to have a fractured skull associated with subdural haemorrhage. Extracranial lesions found at two other post-mortem examinations were a haemopericardium and a haemoperitoneum associated with abrasions of the liver. There were three instances of suprarenal haemorrhage amongst our necropsies (6%), all showing tentorial tears and intracranial haemorrhage. Three of the group with tentorial tears were also found to have small intraventricular haemorrhages.

Minor injuries were sometimes present in babies dying of other causes, usually fraying of the fibres of the tentorium without much associated haemorrhage. In such circumstances birth trauma was sometimes put down as a secondary cause of death, but the significance of this kind of damage is doubtful.

In 45% of the trauma group evidence of asphyxia was found, usually in the form of surface haemorrhages on the lungs. In two cases, however, there was only massive inhalation of meconium. Every stillbirth due to birth trauma showed evidence of asphyxia, and the association of intra-partum asphyxia and damage to the cranial structures during delivery is too well known to require comment.

Of the 40 neonates dying of birth trauma, 35% showed evidence of the pulmonary syndrome of the newborn. All but one of these cases, an instance of pulmonary oedema, were premature and showed hyaline membrane or haemor-

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rhage singly or together. In addition, 10% of the 40 cases showed pneumonia (see below). Some degree of resorption atelectasis was found in all.

Incidence.—The incidence of fatal birth trauma in the babies delivered at U.C.H. (11%) is comparable to the corresponding figures published in recent times from other centres—for example, 11.6% given by D'Esopo and Marchetti (1942). Nevertheless, there is some evidence that birth trauma is declining in importance as a cause of stillbirth. Thus the percentage of our stillbirths caused by birth trauma (4.3%) contrasts with the corresponding figure of 24% given by MacGregor (1946).

Differential Diagnosis.—Two other forms of intracranial haemorrhage exist—asphyxial and intraventricular. The former occurs as localized effusions of blood into the pia arachnoid covering the surface of the cerebral hemispheres. These haemorrhages sometimes rupture into the cranial cavity, but the amount of blood is small and is not associated with damage to intracranial structures. Intraventricular haemorrhage without evidence of trauma is usually differentiated without difficulty. It is important to realize that it is a common cause of haemorrhage around the brain stem, the blood escaping from the fourth ventricle.

Clinical Associations.—During pregnancy the clinical associations were high maternal age and multiple pregnancy, both of premature and of full-time groups. Labour lasting more than 24 hours was a factor in full-time babies, as might be expected; so were those complications of vaginal delivery apt to produce trauma (see section on complications of vaginal delivery in Part II).

Aetiology.—The effect of prolonged or complicated labour requires little comment, and the difficulty that multiple pregnancy adds to delivery is also self-evident. The influence of high maternal age will be discussed under that heading in Part II. It will be apparent, however, that there is an appreciable group, largest amongst premature babies, where labour was apparently normal.

Pulmonary Syndrome of the Newborn

Definition.—A condition associated with neonatal death in the first week of life and characterized by resorption atelectasis together with one or more of the following features: (1) hyaline membrane, (2) intra-alveolar haemorrhage, (3) pulmonary oedema (see Potter, 1952: Osborn, 1953; Claireaux, 1953).

It was the frequent coexistence of these abnormalities that led us to coin the term "pulmonary syndrome of the newborn" rather than to employ separate categories of hyaline membrane, pulmonary haemorrhage, and pulmonary oedema. As an example, of 30 cases in which death was attributed to the pulmonary syndrome of the newborn and which exhibited a hyaline membrane, 10 also had a significant degree of intra-alveolar haemorrhage. In many cases it would have been impossible to decide which was the more important of the two findings. Similarly, 5 of the 30 cases of hyaline membrane also showed pulmonary oedema. Our group probably has much in common with Potter's "abnormal pulmonary ventilation."

Diagnosis.—All but 6 of the 36 babies with the pulmonary syndrome born in U.C.H. were premature. Of these six, only two showed a hyaline membrane. At post-mortem examination of a typical case, the fontanelle was full and the mucosae and nail beds were cyanosed. The lungs were voluminous and usually dark purple in colour, depending on the degree of congestion. The cut surface of the lungs was dark and solid, resembling foetal liver, and sometimes had a shiny tense appearance, particularly if haemorrhage was present. The surface of the lungs showed petechiae or ecchymoses of the kind associated with asphyxia in 44% of cases. Pleural effusion of straw-coloured fluid was present in 15% of cases. Whether the lungs sank or floated in formol-saline was of little diagnostic significance.

Outside the lungs, the ventricles of the heart, especially the right, were usually dilated, as were the great veins. In 25% of cases the pia arachnoid of the cerebral hemispheres showed localized small haemorrhages. The haemorrhagic centrilobular necrosis of the liver described by Potter (1952) was seen twice. Three cases had a slight degree of intraventricular haemorrhage.

Histologically, all cases showed resorption atelectasis with cohesion of the walls of most alveoli and overdistension of the terminal air spaces. Congestion was invariable. A hyaline membrane was present in 73% of cases. The membrane varied from being slight and patchy to uniform and dense. The hyaline membrane lines the alveoli, especially the terminal alveoli, and stains pink with eosin, but otherwise there is little agreement regarding its nature. Intra-alveolar haemorrhage was seen in 35% of cases and also varied greatly in extent from patchy areas to massive involvement of the whole lung. The effused red cells were invariably well preserved, and were present in both alveoli and bronchioles. In two instances pulmonary oedema was the only lesion, but it was demonstrable in 20% of the total group. In two full-time babies resorption atelectasis and pulmonary congestion were the sole post-mortem discoveries. Only one case of pulmonary syndrome, an example of hyaline membrane, also had pneumonia.

In addition to the 59 babies of a total of 406, in whom it was judged to be the cause of death, the pulmonary syndrome was also present in 35 in whom death was attributed to some other cause. Thus 13 of 21 babies dying of intraventricular haemorrhage showed the condition as well as 14 out of 40 dying of birth trauma and 7 out of 33 congenital malformations. Certain differences were present between those babies dying of pulmonary syndrome and those in whom the condition was judged to be merely contributing to death. Thus in the first group 76% of the hyaline membranes were severe and generalized. In the second group, however, the corresponding figure was 33%. In the cases where pulmonary syndrome was merely contributory, pulmonary oedema and haemorrhage were relatively more common in relation to hyaline membrane than in the group where the syndrome was thought to be the cause of death.

Differential Diagnosis.—Provided all pulmonary lobes are examined histologically, little possibility of confusion exists. Babies who show no obvious cause of death outside the lungs and whose pulmonary organs exhibit atelectasis but none of the other features of the pulmonary syndrome constitute a problem. There are, however, few such cases. In the present state of knowledge it is hard to know whether to include them with the pulmonary syndrome group or not. Since there were only two instances in our group, they were included.

Clinical Associations.—Since 83% of babies with the pulmonary syndrome were premature, the incidence of maternal complications in this group must be compared not with all births but with all premature births. There was a significant association with caesarean section—a fact that is already accepted—and also an apparent (although not statistically significant) association with ante-partum haemorrhage. If, however, babies delivered by caesarean section for this complication are excluded from the group of pulmonary syndrome infants, the incidence of ante-partum haemorrhage in the remainder is no longer raised.

Aetiology.—The immediate cause and the pathogenesis of this condition remain obscure. There is little direct evidence

TABLE IV.—*Causes of Death in* 221 *Consecutive Necropsies on Neonates Performed at U.C.H. Between* 1948 *and* 1955 *on Cases Born Inside and Outside the Hospital*

Cause of Death	Premature	Full-time	Total	Percentage
Pulmonary syndrome of the newborn	53	6	59	26·6
Birth trauma	25	15	40	18·1
Congenital malformations	15	18	33	15·0
Pneumonia	16	11	27	12·2
Intraventricular haemorrhage	20	1	21	9·5
Miscellaneous	9	3	12	5·4
Previability	11	—	11	5·0
Intra-partum asphyxia	5	6	11	5·0
Erythroblastosis foetalis	3	4	7	3·2
Total	157	64	221	100

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that inhalation of material from the amniotic cavity is related to its development. Failure to establish respiration, for whatever reason, and cardiac failure may both be important factors. With regard to the influence of foetal distress in its widest sense, of all the 59 cases in Table IV, 31 were in poor condition at birth but the remainder were in good condition.

Intraventricular Haemorrhage

Definition.—A condition occurring almost always in live-born premature infants, characterized by massive haemorrhage into one or both lateral ventricles of the brain.

Diagnosis.—The haemorrhage is usually seen as a mass of blood clot, but if death occurs rapidly the blood will remain fluid. The source of the bleeding is either the choroid plexus or the veins that run across the floor of the lateral ventricle. The haemorrhage commonly extends throughout the ventricular system, escaping into the subarachnoid space through the roof of the fourth ventricle, where it forms a clot surrounding and compressing the brain stem. More rarely the blood ruptures into the cerebrum from the lateral ventricle and may excavate a hemisphere, resulting in a blood-filled cavity lined by an irregular jaundiced wall of nervous tissue.

Of the 21 cases of intraventricular haemorrhage (all but one being premature), 48% showed asphyxial petechiae over the lungs and 24% had pleural effusions. In two instances there were traces of birth trauma—that is, fraying of the tentorium—but the most striking feature was the high proportion of cases (62%) that also showed evidence of the pulmonary syndrome. Of these, two-thirds had a hyaline membrane, one-third pulmonary oedema, and a quarter intra-alveolar haemorrhage, singly or in combination. Of cases of intraventricular haemorrhage, 19% had a foetal type of lung structure and 14% had histological evidence of pneumonia.

Differential Diagnosis.—Intracranial birth trauma can be excluded by the absence of damage to the dural membranes and venous channels and the characteristic distribution of the haemorrhage. Massive intraventricular bleeding, obviously incompatible with life, was judged to be the cause of death even when any of the pulmonary complications mentioned were also present.

Clinical Associations.—The number of cases born in U.C.H. was too small for definite statements about clinical associations. There did appear to be a high incidence of caesarean section as compared with all premature births, and it may be of interest that all the babies born by caesarean section also showed the pulmonary syndrome of the newborn.

Aetiology.—Intraventricular haemorrhage seems to be one of the major hazards that befall small premature babies, but its pathogenesis is obscure. Other haemorrhagic phenomena are very rarely seen in the condition, and evidence of birth trauma is not usually present. Small petechiae are often seen on the viscera and may indicate that asphyxia is of aetiological importance. Whether the manifestations of pulmonary syndrome often associated with intraventricular haemorrhage come before or after bleeding into the lateral ventricles is another matter for speculation. Out of a total of 21 cases of intraventricular haemorrhage (see Table IV), nine were in poor condition at birth and four were in good condition, so that foetal distress does not appear to be an essential precursor.

Pneumonia

Definition.—A condition characterized by the presence of an inflammatory exudate in the air spaces and/or interstitial tissues of the lung.

Diagnosis.—Most cases were in neonates. The few stillbirths with pneumonia always had other pathological conditions and none were attributed to pneumonia alone, although this was probably a matter of chance. Of the 27 babies dying of pneumonia alone, 16 were premature.

On naked-eye examination the lungs were usually dark and voluminous. Sometimes, however, they were light pink in colour and small in volume, especially in small premature babies. The exudate usually consisted of polymorph and mononuclear leucocytes in varying proportions with or without the addition of fibrin, red blood cells, and oedema fluid. Since pneumonia commonly complicates other conditions, such as congenital malformations, death was attributed to pneumonia only when no other abnormality sufficient to prove fatal was present. Among the babies dying of pneumonia there were two cases of mild birth trauma, two of slight intraventricular haemorrhage, one of melaena, one of hepatic cirrhosis, and one unilateral adrenal haemorrhage. Intra-alveolar haemorrhage and pulmonary oedema were common associates of pneumonia, and a scanty hyaline membrane was observed in one case. Signs of asphyxia in the form of pleural petechiae or ecchymoses were seen in 30% of the cases and pleural effusion in 22%.

Differential Diagnosis.—In lungs of premature infants, desquamated alveolar epithelial cells often occupy the air spaces and must not be confused with an inflammatory exudate. Similarly, alveolar phagocytes like the "heart failure cells" of passive venous congestion may occasionally cause difficulty. Other appearances to be differentiated from pneumonia are islands of haematopoiesis seen in erythroblastosis foetalis and white cells in vascular channels crowded together by atelectasis.

Clinical Associations.—Pregnancy in the pneumonia group was no more eventful than in the control group of all births, while in full-time babies there was a significant association with caesarean section and complicated vaginal delivery. Labour in premature cases of pneumonia showed no significant difference from the control group of premature babies.

Aetiology.—Of the 27 infants with pneumonia, born in U.C.H. or elsewhere, 15 died within 24 hours of birth and therefore were likely to have acquired their infection before or during labour. Seven died later than three days after delivery. Since five of these were full-time it may be that length of survival was determined as much by birth weight as by time of infection, and that some of the seven cases were not examples of post-natal infection. Of the 27 cases, 18 were in poor condition at birth and only six were said to be in good condition. Thus there seems to be a connexion between foetal distress, in its widest sense, and death from pneumonia. The association has, in fact, been put to therapeutic use, as will be shown in Part III. This connexion could account for the association of pneumonia with caesarean section and complicated vaginal delivery. It may be that the onset of foetal distress after the membranes have ruptured is particularly apt to cause pneumonia by leading to infection of the amniotic contents and their inhalation at a time when the resistance of the foetus to infection is lowered by anoxia. In support of this view 11 of the 15 babies dying in the first 24 hours were in poor condition at birth, but so were five of the seven dying after three days. In this latter group, intra-partum asphyxia or other stress may have lowered their resistance to post-natal infection.

Haemolytic Disease of the Newborn

This condition was present either in stillbirths as hydrops foetalis or in neonates as erythroblastosis foetalis, with or without kernicterus. Its pathological and clinical features are too well known to need reiteration.

Miscellaneous

This category was a small one, and included a variety of conditions in which the individual incidence was not great enough to justify separate groups for them. It comprised six cases of kernicterus of prematurity without demonstrable blood-group incompatibility, two cases of meconium peritonitis, one case of bilateral renal vein thrombosis, two cases of septicaemia, one upper respiratory tract infection and Ellis-van Crefeld syndrome, one acute pulmonary oedema at

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26 days, two cases of inhaled feeds in babies born outside the hospital, and one of massive inhalation of blood following ante-partum haemorrhage.

Previability

Deaths were placed under this heading when all the following criteria were fulfilled : (1) the baby weighed less than 1,000 g. at birth ; (2) histological examination of the lungs revealed a foetal structure ; and (3) no other pathological lesion sufficient to cause death was found. As can be seen from Table IV, only about 5% of the neonatal deaths had to be placed in this category.

A foetal lung structure consists of gland-like spaces lined by cuboidal epithelium replacing the alveoli of the mature lung and separated by a small quantity of stroma.

A diagnosis of prematurity or of previability is undesirable, as it might perpetuate ignorance about the lesions found in such babies—for example, intraventricular haemorrhage. In earlier surveys the diagnosis of prematurity was resorted to in a high proportion of cases—for example, 27.6% of all stillbirths and neonatal deaths (Labate, 1947). In our series of 337 consecutive necropsies (Table I) the corresponding figure was 2.1%.

Congenital Malformations

Of a total of 57 cases the major malformation was hydrocephalus and meningocele in 20 instances ; anencephaly in 9 ; urinary tract maldevelopment in 6 ; congenital heart disease in 6 ; gastro-intestinal malformation in 4 ; diaphragmatic hernia in 3, and tracheo-oesophageal fistula in 2. In the remaining seven cases the malformations were classed as multiple. Evidence of asphyxia was present in about 20% of the cases ; 15% showed evidence of pulmonary syndrome of the newborn, mainly in the form of intra-alveolar haemorrhage ; and about the same percentage had pneumonia. Signs of intracranial trauma and of intraventricular haemorrhage each occurred in about 4% of the cases.

The overall incidence of congenital malformations in our stillbirths and neonatal deaths (14.7%) is similar to that of many other surveys—for example, 14.1% found by D'Esopo and Marchetti (1942).

Discussion

Importance of Necropsy

The value of post-mortem examination in cases of stillbirth and neonatal death has recently been challenged by Baird and his colleagues (1954). Most people would agree that the basic causes of perinatal mortality lie in pregnancy and labour, yet there appear to be at least four good reasons for performing necropsies in all cases.

Firstly, necropsy is necessary to reveal the presence of occult congenital malformations, such as diaphragmatic hernia. Death in such a case might be erroneously attributed to faulty management. Secondly, it is important for the obstetrician to know whether birth trauma has played a part in the death of a baby, so that he can decide on the management of similar cases in the future and on the value and hazards of standard obstetric procedures. Even a brief experience of foetal pathology makes it clear that diagnosis of birth trauma on the history alone without necropsy is highly unreliable.

Thirdly, post-mortem examination is necessary to determine the cause of death of neonates, especially when premature, because a clinical syndrome is often common to several pathological conditions. For example, without necropsy, a case of pneumonia could easily pass as "atelectasis." In addition, until premature labour can be prevented it is important to know the immediate cause of death in premature neonates, so that the paediatrician may apply or devise appropriate therapeutic measures. Finally, a classification based on pathological evidence is less susceptible to differences of opinion than one derived from clinical data —for example, the presence of toxaemia of pregnancy. In stating the cause of death, both kinds of evidence could be used—for example, intra-partum asphyxia due to prolapsed cord.

Desirability of Uniform Classification

In face of the ignorance that now prevails about the true causes of foetal and neonatal mortality, a classification based on narrowly defined pathological criteria may seem undesirable, by appearing to conceal this ignorance behind a series of labels. Provided, however, that a classification is seen as a means rather than an end, it should speed progress by making deficiencies in our knowledge more obvious, by acting as a framework on which knowledge can advance, and, if widely accepted, by standardizing mortality returns all over the country. The classification presented in this paper does not differ widely from many in practical use all over the world.

Possibility of Occult Birth Trauma

It is the practice of many pathologists and of ourselves to confine the diagnosis of birth trauma to cases in which certain definite pathological lesions exist. It is suspected by some, however, that lethal trauma may occur to the brain without anatomical evidence of damage to intracranial structures. Thus trauma has in the past been diagnosed without such evidence when the foetus dies during labour, particularly if parturition has been prolonged or difficult. Most of our cases of this kind are classified as intra-partum asphyxia. The great difference between this group and unchallenged examples of birth trauma is that 80% of cases of intra-partum asphyxia are stillborn, whereas 79% of birth trauma infants are born alive. It seems, in fact, that cerebral trauma by itself is not an important cause of death *in utero*.

If this is true the only groups in which occult birth trauma would play a major part are the pulmonary syndrome of the newborn and intraventricular haemorrhage. Trauma is acknowledged to be a possible factor in the aetiology of the latter condition. In the pulmonary syndrome, even if the leading role of cerebral dysfunction is accepted, there seems little point in postulating direct violence to the nervous system when there is nothing to suggest it and when its presence would do no more to explain the condition of the lungs than the cerebral anoxia for which evidence often exists.

Ante-partum Stillbirth

We have separated our ante-partum deaths into "asphyxia" and "maceration only," according to whether or not haemorrhages are present on the viscera. This apparently arbitrary division is justified by other differences between the two groups described earlier.

Nobody would suggest classifying together a macerated foetus born prematurely in an apparently normal pregnancy and a baby born dead shortly before term as a result of an accidental haemorrhage. It may be, however, that in some cases the distinction is unjustified—for example, in diabetes, where the baby may fall into either category. Even here, however, the direct cause of death might be different.

Intra-partum Asphyxia

In the case of asphyxia before labour, the clear-cut association between placental separation and pleural ecchymoses justifies the diagnosis. In intra-partum asphyxia, the haemorrhages seen are not usually so large or numerous and the cause of the oxygen deprivation is usually less obvious and dramatic. As a result it has been suggested that such babies die of some kind of unsuccessful alarm reaction or adaptation to stress. This hypothesis may prove to be fruitful, but at the moment there is little evidence in its favour, and, since clinical and pathological signs of asphyxia are often demonstrable, we have preferred to retain this term.

[*The conclusion, with a list of references, will be published in our next issue.*]

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