CONGENITAL PNEUMONIA*

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The term congenital (or intrauterine) pneumonia is commonly used to describe an apparently inflammatory disease of the lungs found at autopsy in a small proportion of infants who are stillborn or who die in the first few days of life. There is general agreement that asphyxia is an important factor in the pathogenesis and that histological lesions common to the pneumonias of later life are usually absent. The purpose of this paper is to review these rather unusual features and to question the inflammatory nature of the disease.

Material and Methods

The cases were selected by examination of lung sections from infants who came to autopsy at the United Oxford Hospitals during a 15-year period from 1946 to 1961. In most, the typically diffuse changes of congenital pneumonia were present, but cases were also included in which patchy changes were found. It was hoped that the study of early as well as fully established cases would prove more valuable. In this way, 72 infants, liveborn and stillborn, were chosen. No infants dying later than the fourth day were included as we wished to discuss only those who acquired their lesions *in utero* or during their passage through the birth canal.

Clinical associations may be separated into maternal and infant factors. For comparison the corresponding data from a group of 3,930 women delivered at the Radcliffe Infirmary during 1960 and 1961 will, where available, be given in brackets. It should be emphasized that these cannot be considered as controls. The period covered is different and the incidence of prematurity (10%) is lower than in the cases under discussion. They should be taken merely as a set of averages for a particular Maternity Department admitting the obstetric abnormalities of its region.

A. Maternal Factors. *Complications of pregnancy and/or labour* were present in 70 of the 72

cases. The most common were infection in 36%, toxæmia in 26% and abnormalities of the cord, such as prolapse or compression, in 15%. Labour was induced surgically in 20% of cases. The diagnosis of maternal infection did not necessarily have bacteriological proof. However, women who had purulent vaginal discharge during labour, or who were draining foul-smelling liquor, or who were febrile during labour sometimes with rigors, were included.

The *maternal age* of 47% (34%) of the mothers was 30 years or over.

The *interval between membrane rupture and delivery* is shown in Fig. 1. The higher incidence of prolonged rupture in the cases under discussion should be noted.

Duration of labour is shown in Fig. 2.

The incidence of each *mode of delivery* is shown in Table 1.

B. Infant Factors. *Foetal distress* was present in 40% (24%) of the cases. Foetal distress has been taken as evident if there was meconium staining of the liquor, and/or a foetal heart rate of more than 160 or less than 120 noted on more than one occasion during labour.

Of the liveborn infants, 45% (17%) were asphyxiated at birth. Infants were considered asphyxiated if regular respirations were not established within two minutes of delivery.

The proportion of stillbirths, of premature babies, and the sex incidence is shown in Table 2. Of the premature infants, 11 were pre-viable. Ten infants were born after the 41st week of gestation.

Clinical Picture. Of the 57 liveborn babies there were adequate clinical details of 48. The majority were ill from birth and seven of them died almost immediately. Cerebral signs, such as increased limb tone, fisting, abnormal wakefulness and irritability were present in 23 babies and eight had frank convulsions. Nine infants showed signs of respiratory distress, but in every one a significant degree of secondary atelectasis was found at autopsy to

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FIG. 1.—Interval between rupture of membranes and delivery.

FIG. 2.—Duration of labour.

account for this. A small group of babies, one-fifth of the total, had a varying period of apparent well-being after birth from 18 hours to two days, ending in sudden collapse with death usually a few hours later. In the remainder, limpness, cyanotic attacks and a subnormal temperature were the most common findings. Four-fifths of the babies died within 48 hours of delivery, the majority within 24 hours.

Pathological Features. Amniotic aspiration was prominent in about one-half (Fig. 3); only rarely was there any evidence of fibrinous exudation or tissue destruction. In cases where the obstetric history suggested foetal asphyxia uncomplicated by obvious maternal infection there was a tendency to a focal aggregation of cells, often in relation to bronchioles, with amniotic squames predominating over polymorph leucocytes. Conversely, where there was maternal infection polymorphs were the more frequent and the distribution was more diffuse. Many of the cases in which squames could not be found were immature; it is likely that the amniotic fluid in these cases contained few.

The stromal tissue of immature lungs was searched for the presence of acute inflammatory cells because it seemed reasonable to suppose that a true exudate from vessels as yet separated from air spaces would

be found there in transit. No clear evidence of such exudation was found (Fig. 4). Occasionally, polymorphs were seen in bronchial lymph nodes, but they were usually in peripheral sinuses and appeared to be effete cells coming in from the alveoli. The spleen was examined in 26 cases for evidence of defensive reaction; an excess of polymorphs was found in five but the significance of this is uncertain. Sections of placenta were only occasionally available and therefore no attempt was made to estimate the frequency of chorioamnionitis.

One case differed sharply from all others in showing not only pulmonary lesions but a meningitis and marked fibrino-purulent pleurisy and a pericarditis as well. This infant was born to a woman who had contracted lobar pneumonia one day before the onset of labour. *Strep. pneumoniae* (Type 1) was isolated from the lung, pleural cavities, pericardium and meninges.

Pneumonia supervening on hyaline membrane disease was considered to be a different problem, and cases of this type were not included.

Discussion

In congenital pneumonia many authors have already pointed to the complicated course that we

TABLE 1
INCIDENCE OF EACH MODE OF DELIVERY

Delivery	Congenital Pneumonia (%)	1960 and 1961 (%)
Sp. vertex	55.6	62.6
Forceps	22.2	24.4
Breech	12.5	4.8
Caesarean	9.7	8.2

TABLE 2
CONGENITAL PNEUMONIA
(72 Cases)

Condition, Weight and Sex	Numbers
Liveborn	57
Stillborn	15
< 2,500 g.	40
> 2,500 g.	32
Males	35
Females	37

FIG. 3.—Section of lung from liveborn infant at term, showing inflammatory cells and amniotic material. (H. and E. $\times 300$.)

have described in our cases. Prolonged rupture of membranes (Johnson and Meyer, 1925), prolonged and sometimes inert labour, a high proportion of abnormal deliveries, increased maternal age (Lang-

ley and Smith, 1959), maternal pyrexia (McCredie Smith, Jennison and Langley, 1956), foetal distress and prematurity (Bound, Butler and Spector, 1956) have all been stressed. These can be reduced to

FIG. 4.—Section of lung from immature liveborn infant, showing inflammatory cells in air spaces, but no stromal infiltration. (H. and E. $\times 150$.)

factors causing or indicating foetal hypoxia and infection of the uterine foetal environment.

Much less attention has been given to the clinical course of the liveborn infants and there are few clinical descriptions in the literature. Schaffer, Markowitz and Perlman (1955) stress delayed onset of respiration after delivery. Like Branton (1959) and Bound *et al.* (1956), they report clinical signs suggesting central nervous system involvement. We believe this is the most important feature. Only one of the babies who had convulsions was found to have an intracranial lesion at autopsy. The classic signs of respiratory distress such as grunting respirations with sternal retraction which are present in most babies with secondary atelectasis were not a conspicuous feature here, except for a small group who were in fact shown to have atelectasis in addition to their other lesions.

Histological signs of amniotic aspiration are found very commonly in congenital pneumonia. It has been supposed that organisms suspended in amniotic fluid may thus be drawn into the lungs and evoke a diffuse inflammatory response. No doubt this may happen. But as a general theory it raises certain difficulties. For in congenital pneumonia there is usually no infiltration or destruction of bronchopulmonary tissue, though the amnion exposed to the same noxious agent may be severely damaged; and there is usually no fibrinous exudation into alveoli or over the pleura, though cases such as our pneumococcal septicaemia show that this should be possible. Moreover, the majority of infants emerge unharmed from an infected uterine environment, or at autopsy show no sign of pneumonia. And even when inflammatory cells are present in the lungs at autopsy organisms cannot always be recovered.

These difficulties can be resolved by supposing that the polymorphs in typical congenital pneumonia have, like the squames which usually accompany them, been inhaled from the amniotic sac where they have been taking part in an ascending chorioamnionitis. Osborn (1958) and Macgregor (1960) have pointed out that such 'foreign' leucocytes may sometimes be recognized morphologically as amniotic pus cells—effete and degenerate, with hypersegmented pyknotic nuclei. Further evidence of their extrinsic origin might come, in male babies, from nuclear sexing; our material did not permit this investigation.

In this view congenital pneumonia, so called, is a passive condition which the foetus acquires by asphyxial gasping in the presence of exudate from the placenta and membranes. The equal sex incidence too makes it less likely that infection plays

a dominant role. For there is evidence (Nyhan and Fousek, 1958) that newborn boys are much more susceptible to infection than are girls. This is not to say that true pneumonia due to prenatal infection does not occur. Cases may certainly be found in which evidence of destructive bacterial aggression and local diapedesis of leucocytes (Sorba, 1948) may be seen. The crucial difficulty is to decide whether aspiration or infection accounts for the majority of cases. We do not feel that we can draw dogmatic conclusions from our series because of the inherent defects of a retrospective study. Nevertheless, we would agree with Osborn (1962) that the majority of congenital pneumonias are most fittingly interpreted as 'drowning in pus', and the clinical analysis is compatible with this view.

Clinical diagnosis should be possible in many cases if full maternal details are available. This may be supported by the demonstration of polymorphs in whole amounts of amnion (Blanc, 1961), frozen sections of umbilical cord (Benirschke and Clifford, 1959) or smears from cut surfaces of the cord (Aherne and Davies, 1962). In these tests a positive result indicates merely the presence of chorioamnionitis and not necessarily pneumonia as well. Nevertheless, it narrows the diagnostic field considerably. For there is now much evidence (reviewed by Blanc, 1961, and Bourne, 1962) that congenital pneumonia typically occurs as a sequel to chorioamnionitis.

The controversial matter of treatment is beyond the scope of this paper. If we have interpreted our findings correctly, metabolic consequences of asphyxia may be more important than infection, and it is possible that the correction of these may have an important part in treatment.

Summary

A review of the clinical and pathological features of congenital pneumonia is presented in a series of 72 cases.

In 70 cases complications of pregnancy or labour occurred, which in the main were those leading to foetal hypoxia and infection of the uterine foetal environment.

The commonest clinical signs in the 57 liveborn infants were neurological; they were not attributable to structural lesions. Signs of respiratory distress occurred only in infants with superadded secondary atelectasis.

Clear histological evidence of pulmonary inflammation was lacking in most cases. It is suggested that congenital pneumonia is usually a passive condition due to asphyxial aspiration of maternal inflammatory cells.

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