STUDIES OF PNEUMONIA IN CHILDHOOD.

III. BRONCHO-PNEUMONIA.

BY

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Broncho-pneumonia, if we consider its frequency, its high mortality and its serious and lasting sequelæ, must be regarded as the most important type of pneumonia in early childhood. The recognition of the type and of its peculiar prevalence in the first years of life, began in the years following the clinical and pathological studies of Laennec. From this time, about 1820, until about the middle of the century, there was much discussion and controversy regarding this infantile type of pneumonia, based largely on post-mortem investigation. The factors of atelectasis, collapse, and inflammation, and the parts played by them in producing consolidation in the lungs of young infants, were much debated. In 1837, Seifert¹ introduced the term broncho-pneumonia.

About 1850 Barthez and Rilliet put the discussion on firmer ground. They adopted the term broncho-pneumonia (using other synonyms including lobular pneumonia); they declared that the essential and primary lesion in the condition was a severe inflammation of the finer bronchial tubes, the changes in the lungs being derived from this bronchitis; and they identified as being practically inseparable, capillary bronchitis and broncho-pneumonia. They considered broncho-pneumonia to be the characteristic and common type in young children; but they also pointed out that cases of croupous or lobar pneumonia were met with even at the earliest age. The final statement of their views can be read in the chapter on broncho-pneumonia in their book *Maladies des Enfants*² (1884), where they also give a full account of the long controversies of the preceding sixty years. This chapter remains still the classical presentation of the subject. There have been no substantial additions to our knowledge since it was written. There is still lacking a satisfactory explanation of the prevalence of this type of pneumonia in early childhood: on the one hand, a constitutional factor is alleged; on the other, some special bacterial cause.

West³ in this country, and Henoch⁴ in Germany, writing in the same decade, diverge little from the teaching of Barthez and Rilliet. They both emphasize the close, indeed inextricable, relation between bronchitis and broncho-pneumonia in young children.

It may be useful to set down together the numbers of our cases of bronchitis, broncho-pneumonia, and alveolar (lobar) pneumonia, already given in the statistical survey⁵, grouping them in age periods (Table I).

TABLE I.
AGE PERIODS IN AUTHORS' SERIES OF CASES.

Age period				Bronchitis	Broncho-pneumonia	Alveolar pneumonia
Under 2 years	...	...		155	110	143
2-5 years	...	...	...	55	31	131
5-12 years	...	...	...	21	3	112

Two points should be remembered in considering these figures. First, an attempt has been made to separate bronchitis from broncho-pneumonia in infants. Secondly, the group of broncho-pneumonia includes only a small number of cases of measles and whooping-cough. Both these facts have reduced the numbers of this group in comparison with the collected figures given by many other writers; and they give a relative prominence to the numbers of alveolar (lobar) pneumonia in the first age period.

Our figures justify the following general statement as to the relative incidence of the two types of pneumonia in childhood; whereas cases of alveolar (lobar) pneumonia occur throughout the whole period of childhood with fairly steady frequency, the great majority of cases of broncho-pneumonia are concentrated in the first two or three years of the period. This statement is in accord with the view prevalent to-day and for many years past.

PATHOLOGICAL STUDY.

Broncho-pneumonia is the form in which pneumonia is most often seen in the post-mortem room of a children's hospital. While differences of opinion may exist as to the relative frequency of broncho-pneumonia and alveolar pneumonia in cases which recover, there can be no doubt at all as to the preponderance of the former among those which are fatal.

In the first paper of this series⁵, it was stated that of 945 consecutive autopsies performed in the Royal Edinburgh Hospital for Sick Children, 140 (14·8%) were cases of broncho-pneumonia, and 23 (2·5%) of alveolar pneumonia. This figure (140) excludes a group of 29 cases of terminal hypostatic pneumonia which, for the purpose of the analysis, were placed in a class by themselves, although they might reasonably be regarded as a sub-group of broncho-pneumonia. Some facts of interest derived from a survey of the 140 cases may be given.

Age. The ages of the children ranged from 1 week to 7½ years. 70 were under one year old; 48 were between one and two years. Thus, about 84 per cent. of all the cases were in children under two years of age.

Sex. There were 78 boys and 62 girls, showing no significant difference in incidence between the sexes.

Complications. In 51 of the 140 cases the broncho-pneumonia was accompanied by some serious complication, which in some instances was the

actual cause of death. The nature and frequency of these complications were as follows :—empyema (34) ; suppuration in the lungs (27) ; pericarditis (10) ; peritonitis (8) ; meningitis (4) ; gangrene of lung (1) ; suppurative pyelonephritis (1) ; laryngeal diphtheria (2).

Morbid Anatomy.

The upper respiratory passages. There is usually an acute catarrhal inflammation of the upper air passages, which becomes more severe towards the lower end of the trachea and in the main bronchi. Often an immense amount of thick purulent secretion is found at autopsy in the main bronchi, trachea and larynx. As a rule there is a generalised acute bronchitis affecting bronchi of all sizes throughout both lungs. The walls are swollen, the mucous membrane intensely red, the lumina filled with muco-pus. This inflammation is often much more severe in the smaller than in the larger bronchi, and there may be some dilatation of the finer tubes.

Site and extent of pneumonic consolidation. In 20 cases in our series the right lung only was affected ; in 8 the left lung only ; in 112 both lungs were involved. In almost all instances when pneumonia was confined to one lung, bronchitis was present in the other. It is quite probable that, owing to the impossibility of examining microscopically every part of every lung which appeared to be free from consolidation, minute patches may have been overlooked. The number (28) of cases classed as unilateral may therefore be larger than it ought to be. In a certain number of bilateral cases, pneumonia was much more extensive in one lung than in the other (Plate II, Fig. 7 & 8), but it was more usual to find it similar in extent and apparent duration on the two sides (Plate I, Fig. 3 & 4 ; Plate II, Fig. 5 & 6).

In most cases pneumonic areas were present in all lobes. The lower lobes, however, especially the posterior and basal portions, were more constantly and usually more severely affected than other parts. If any confluence of broncho-pneumonic patches were present, it was most often at the base or along the posterior border. The parts most frequently found to be free from pneumonic patches were the anterior borders of the upper lobes. We have noticed, however, that a strip of consolidation at the antero-inferior angle of the left upper lobe, along the lower margin of the cardiac notch, is of remarkably frequent occurrence.

The size of individual areas of consolidation varies greatly in different cases. When there is no confluence, the patches are often exceedingly small, although they may be present throughout the whole of both lungs, with air-containing lung substance between them (Plate I, Fig. 1). Each of these small patches forms in relation to a bronchus or bronchiole, which occupies the centre of it. When there is copious exudation into the alveoli surrounding the central inflamed bronchiole and neighbouring patches become confluent over large areas, the resulting consolidation may affect a whole lobe or even almost a whole lung. In these cases a diagnosis of lobar pneumonia may suggest itself and differentiation may present some difficulty. It is quite

usual to find small discrete patches occupying the anterior parts of the lungs, with confluence in large areas at the bases and posterior borders (Plate II, Fig. 6).

The appearance of the pneumonic areas. This naturally varies greatly according to their size, their duration and the amount of confluence. When there is no confluence and the pneumonic patches are very small and occur in relation to practically every bronchiole, the cut surface of the lung shows innumerable sharply defined patches of pinhead size or even smaller, each with a tiny bronchiole in its centre. These minute patches project slightly above the general level of the cut surface, and the wall of the bronchiole is conspicuous owing to inflammatory thickening. At an early stage the patches are red ; later, grey ; still later, definitely yellow. The intervening lung substance is congested. The gross appearance of this form of broncho-pneumonia is sometimes remarkably like that of acute miliary tuberculosis, but close inspection reveals the presence of the central bronchiole in each patch and removes the likelihood of error. On slight pressure, quantities of purulent material exude from the bronchioles.

To the touch the lungs feel 'shotty,' the pneumonic areas being readily detected by palpation. Sometimes, indeed, their presence is more easily discerned by the fingers than by the eyes. In some cases, although the minute bronchioles are acutely inflamed, it is impossible to detect any consolidation by macroscopic examination, and a diagnosis of capillary bronchitis is apt to be made. But we have yet to find a case of this kind in which the microscope failed to reveal consolidation of tiny groups of alveoli surrounding the inflamed bronchioles.

When there is confluence so widespread that consolidation is almost or quite of lobar extent, certain features peculiar to broncho-pneumonia usually serve to distinguish it from alveolar pneumonia. The consolidated area is rarely as massively and completely consolidated as in alveolar pneumonia. There is almost always a certain amount of collapse associated with the consolidation. Because of this collapse, an area of confluent broncho-pneumonia is less voluminous than a corresponding area of alveolar pneumonia ; sometimes indeed it is even less voluminous than adjacent aerated lung substance (Plate III, Fig. 9). It has a rubber-like resilience, unlike the somewhat brittle firmness of alveolar pneumonia ; and an unequal or nodular consistence, readily appreciated on handling the organ. Where the area of confluent broncho-pneumonia abuts upon aerated lung substance, the margin is usually indefinite and irregular, and small pneumonic patches are present beyond it (Plate I, Fig. 3). The cut surface differs from that of an area of alveolar pneumonia. It is usually moist and shiny instead of dry, dull and granular, and there is conspicuous mottling corresponding to the distribution of small bronchi and bronchioles. Usually each bronchiole is surrounded by a small zone which is yellow or greyish yellow in colour and, being more completely consolidated, stands out slightly above the level of the adjacent lung substance. The walls of the bronchioles are swollen and stand out prominently ; the lumen is often a little dilated and contains pus. The intervening lung substance is

red or greyish red, and is in a condition of partial collapse and partial consolidation which renders it completely airless. This conspicuous peribronchial mottling is exceedingly characteristic of confluent broncho-pneumonia and points unmistakably to that diagnosis.

Suppuration and necrosis. In nearly 20 per cent. of the cases of broncho-pneumonia in our post-mortem series (27 out of 140) suppuration had occurred in some part of the lungs, associated in numerous instances with areas of necrosis, which had the characters of septic infarcts and were caused by thrombosis of blood vessels. These necrotic areas occurred in consolidated parts, and were sometimes red, but more often pale in colour. They had a great tendency to septic softening, and invariably showed suppuration at the edges, if not more extensively. Apart from these septic infarcts, when abscesses were present in the lungs they almost always originated in bronchi and were usually multiple and of small size.

Emphysema. Acute emphysema is often a very conspicuous feature in broncho-pneumonic lungs. It affects those regions of the lungs where pneumonic patches are either absent or small and discrete, usually the anterior portions of the upper lobes. It may be merely of the vesicular variety, but interstitial emphysema also is often present and sometimes produces a most remarkable appearance (Plate II, Fig. 5 and 6). The air, escaping from ruptured alveoli into the stroma of the lungs, tracks along interlobular septa, distending them and separating their fibrous bundles, and forms bullæ under the pleura which may reach a large size, up to an inch or more in diameter. Rupture of these bullæ may occur and produce pneumothorax, but considering the frequency of their presence and the stretching of the pleura which they cause, it is perhaps surprising that that accident does not happen more often than it appears to do. Sometimes the air finds its way along the septa to the root and so to the mediastinum. It may then spread further and produce subcutaneous emphysema of the neck and chest wall.

This severe emphysema, which is characteristic of broncho-pneumonia and unusual in the alveolar type, may be explained as being in part 'complementary' in nature, intensified doubtless by the laboured respiration and severe cough usual in these cases. But it is probable that a more important factor in its production in this exaggerated form, is partial obstruction of the bronchi in emphysematous parts by the inflammatory exudate resulting from acute bronchitis, or the minute patches of pneumonia which are so often present (Plate IV, Fig. 17).

The pleura. Pleurisy is not constant as it is in alveolar pneumonia. It is indeed usually absent, unless consolidated areas of some size are present immediately beneath the pleura. Extensive confluent broncho-pneumonia usually shows fibrinous pleurisy, often with a very scanty exudate, affecting the pleura over the consolidated parts. As in alveolar pneumonia, it would seem that cases which have a copious pleural exudate tend to develop empyema. Subpleural petechiæ, sometimes very numerous and widespread, are extremely common in broncho-pneumonia.

Lymphatic glands. Intense congestion and very great swelling of the broncho-pulmonary and tracheo-bronchial glands are characteristic of broncho-pneumonia (Plate I, Fig. 1 ; Plate II, Fig. 6). The degree of enlargement is, indeed, sometimes very remarkable. Although these glands show evidence of intense acute inflammation, we have rarely found suppuration in them, even when it had occurred in the lungs.

Other internal organs. Toxic changes in the organs vary greatly in different cases of broncho-pneumonia. Sometimes they are remarkably slight in proportion to the severity of the local condition in the lungs. When they are really severe, as they are in a certain number of cases, the existence of septicæmia can usually be proved by bacteriological examination of the blood. Dilatation of the right side of the heart, with a pale soft myocardium, is usual.

Morbid Histology.

Broncho-pneumonia is an inflammatory condition affecting primarily the walls of bronchi and bronchioles and spreading to the alveoli immediately related to them. In the area of an early lesion (Plate III, Fig. 11) is a bronchiole with catarrhal contents in its lumen and inflammatory infiltration in its walls, polymorphonuclear leucocytes being numerous among the infiltrating cells. The condition affecting the bronchiole is much more than a superficial catarrh ; it is an intense inflammation of the substance of the wall throughout its whole thickness, indicating that the infection is not located merely in the lumen but penetrates the wall and invades the lymphatics (Plate III, Fig. 12 ; Plate IV., Fig. 13). This severe acute interstitial inflammation of the wall may affect bronchi of any size but is most severe in the smaller tubes. It may cause serious damage. Complete destruction of the epithelium is usual, and the infiltration of the wall by polymorphonuclears may be so dense that its structure becomes indistinguishable. Some dilatation of the smaller bronchi is of common occurrence, and a copious cellular exudate, usually devoid of fibrin, is present within them.

Around the central bronchiole is a group of consolidated alveoli, the walls of which are thickly infiltrated with inflammatory cells, so that they may be much obscured and difficult to distinguish. Often the infiltration of the walls seems to precede consolidation of the lumen and extends beyond the edge of the consolidation patch (Plate III, Fig. 11 ; Plate IV, Fig. 17).

In severe cases every branch of every bronchus down to its finest ramifications forms the centre of a pneumonic patch (Plate I, Fig. 1 and 2). The consolidated alveoli around a bronchus are not always those which are directly connected with it by continuity of lumen. Even tubes of some size, cut at a considerable distance from their termination, may be surrounded by a ring of consolidated alveoli. We are unable to accept the view that these alveoli are always the expansions of minute branches of the central bronchus. Their arrangement as a collar, often not more than one or two alveoli deep, around the whole circumference and along a considerable length of the bronchus, points to the conclusion that infection reaches these alveoli through the bronchial wall and the alveolar septa, and not along the lumen. Acute interstitial inflammation is an essential part of the process.

PLATE I.

- Fig. 1. (Male, aged 3 years and 9 months. Case I.) Acute broncho-pneumonia following measles. Wide dissemination of small discrete pneumonic patches; little confluence. Enlarged bronchial glands.
- Fig. 2. (Female, aged 1 year. Case II.) Acute broncho-pneumonia, of 12 days' duration. Wide dissemination of minute pneumonic patches; confluence in middle lobe and medial border of upper lobe.
- Figs. 3 & 4. (Male, aged 1 year. Case III.) Acute broncho-pneumonia, of 4 weeks' duration. Extensive confluence, especially in right lung (Fig. 3.) Enlarged bronchial glands.

PLATE II.

- Figs. 5 & 6. (Female, aged 7 months. Case IV.) Acute broncho-pneumonia, of 8 days' duration. Extensive confluence at bases of lower lobes. Discrete patches and bronchitis elsewhere. Marked interstitial emphysema, best seen at apices. Enlarged bronchial glands.
- Figs. 7 & 8. (Male, aged 2 years and 6 months. Case V.) Acute pneumonia of mixed type, of 11 days' duration. Massive consolidation of whole lower lobe of right lung (Fig. 7), mainly of alveolar type. Irregular patches of broncho-pneumonia in upper and middle lobes of same lung. Very small early broncho-pneumonic areas in both lobes of left lung (Fig. 8).

PLATE III.

- Fig. 9. (Female, aged 2 years. Case VI.) Broncho-pneumonia following measles 6 weeks before death. Widespread consolidation of small patches with collapse of intervening lung tissue, rendering large areas airless. Severe purulent bronchitis. Dilatation of smaller bronchi.
- Fig. 10. (Male, aged 1 year and 3 months. Case VII.) Pneumonia of mixed type. Massive uniform consolidation of entire upper lobe, with characters of alveolar pneumonia. Capillary bronchitis and small broncho-pneumonic areas in lower lobe; slight confluence at periphery.
- Fig. 11. (Same case as Fig. 1.) Early broncho-pneumonic patch. Central bronchiole (B) with walls obscured by inflammatory cells; cellular exudate in adjacent alveoli; infiltration of alveolar septa.
- Fig. 12. (Same case as Figs. 5 & 6.) Small bronchus showing cellular exudate in lumen; destruction of epithelial lining; dense inflammatory infiltration of walls; spread of inflammation to adjacent alveoli.

PLATE IV.

- Fig. 13. Higher magnification of part of wall of bronchus shown in Fig. 12. A = bronchial wall infiltrated with cells, most of which are polymorphs; B = layer of muscle; C = remains of lining epithelium; D = exudate in lumen.
- Fig. 14. (Same case as Fig. 2.) Blood vessel (A) at bifurcation in broncho-pneumonic lung, showing dense infiltration of perivascular stroma by inflammatory cells. B = bronchiole with inflamed walls.
- Fig. 15. (Same case as Figs. 2 & 14.) Group of alveoli in area of confluent broncho-pneumonia, showing exudate of predominantly fibrinous nature.
- Fig. 16. Large lymphatic vessels (L) at root of lung from case of broncho-pneumonia, showing great dilatation, purulent content, and spread of inflammation to surrounding tissues. A = blood vessel.
- Fig. 17. (Same case as Figs. 5, 6, 12 & 13.) Acute vesicular emphysema in lung tissue surrounding a patch of early broncho-pneumonia. Central bronchiole (B) inflamed; bronchial and alveolar walls infiltrated; contiguous alveoli consolidated.
- Fig. 18. (Same case as Fig. 9.) Illustrating collapse of lung in broncho-pneumonia. Parts of two inflamed and dilated bronchioles (B) are shown, with exudate in alveoli immediately adjacent and collapse of intervening lung substance. An interlobular septum is seen passing obliquely across the middle of the field.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

PLATE II.

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

PLATE III.

Fig. 9.

Fig. 10.

Fig. 11

Fig. 12.

Fig. 13.

Fig. 14.

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 18.

There may be, in addition, consolidation in the terminal alveolar expansions of the central bronchiole. Where there is confluence this is always present and extensive, but the interstitial inflammation persists in the peribronchial zone.

In the acute interstitial inflammation, lymphangitis plays an important part. Beginning in the peribronchial lymphatics it soon spreads to the perivascular and septal lymphatics also. The perivascular connective tissue may be infiltrated almost as thickly as the bronchial walls (Plate IV, Fig. 14); the larger lymphatic vessels in the septa, under the pleura and at the root, are often greatly dilated and filled with contents of almost purulent character, the tissue in their neighbourhood participating in an acute inflammatory change (Plate IV, Fig. 16). The severe involvement of the lymphatic system of the lungs explains the inflammatory enlargement of the root glands which has already been mentioned.

This progressive lymphangitis, with associated acute interstitial inflammation, is an important feature of broncho-pneumonia. Its severity varies in different cases, but we have found no case in a large series examined with the aid of whole-lung sections, in which it was not present. Sometimes the interstitial inflammation is more conspicuous than the alveolar consolidation, of which there may be very little. Such cases have been described by some authors as a separate group under the name of 'acute interstitial broncho-pneumonia.' Influenzal and post-measles pneumonias are stated to be often of this type. But our studies have led us to believe that a progressive lymphangitis with acute interstitial inflammation of the bronchial walls, alveolar septa and stroma, is a constant feature of all forms of true broncho-pneumonia, and is indeed of the very essence of the pathological process.

Further, it is this feature of the disease that accounts for the fact that broncho-pneumonia is much more apt than alveolar pneumonia to be followed by undesirable results, such as suppuration, necrosis, bronchiectasis and fibrosis. That suppuration and necrosis in the lungs are not uncommon in fatal broncho-pneumonia has already been indicated. Both are to be attributed directly to interstitial inflammation and lymphangitis. When suppuration originates in the bronchial walls, which is often the case, it is merely a further development of the usual severe inflammation of those structures. The thrombosis of blood vessels which leads to the formation of areas of infarction is caused by involvement of the vessel walls in an acute inflammation spreading to them from the perivascular lymphatics. The association between acute broncho-pneumonia and development of bronchiectasis and fibrosis of the lungs will be dealt with in another paper.

The alveolar exudate. In very small and early broncho-pneumonic lesions the exudate in the tiny group of alveoli surrounding the central bronchiole is often composed entirely of polymorphonuclear leucocytes (Plate III, Fig. 11; Plate IV, Fig. 17). When the patch is somewhat larger, a greater amount of exudation into the alveoli having taken place, it usually contains fibrin, which may be in large amount. Often a patch of this kind shows a series of zones in which the composition of the exudate varies. In the lumen and

walls of the central bronchiole and the alveoli nearest to it, polymorphonuclear cells are present to the exclusion of everything else. Outside this purely cellular zone the alveoli contain fibrin and cells in varying proportions, and their walls show less infiltration than those of the alveoli nearer the bronchiole. At the periphery of the patch there may be a ring of alveoli whose contents almost consist entirely of catarrhal cells shed from the walls, together with a varying amount of œdematous fluid. Beyond this again is a ring of collapsed alveoli which contain little or no exudate. When the pneumonic patches are numerous, they may be separated from each other only by a narrow strip of collapsed alveoli, and the affected part may be completely airless, simulating a confluent broncho-pneumonia (Plate IV, Fig. 18).

Even in massively confluent broncho-pneumonia this 'zonal' character of the exudate is usually preserved. The peripheral alveoli of each area of pneumonia are rarely completely filled with exudate, but are partially collapsed, so that the fact that the consolidation is essentially patchy and peribronchial in distribution is evident. This, in conjunction with the presence of acute interstitial inflammation, excludes the diagnosis of alveolar pneumonia. The peribronchial mottling which is so distinctive a macroscopic character of confluent broncho-pneumonia is, of course, due to this feature of the process.

Fibrin is usually most plentiful when alveolar exudation is most abundant, and is therefore found chiefly in areas of massively confluent broncho-pneumonia. In such cases certain fields may present an appearance indistinguishable from alveolar (croupous) pneumonia, the exudate being as rich in fibrin as it ever is in that disease. The photograph shown in Plate IV, Fig. 15 (taken from one of the confluent areas in the lung shown in Plate I, Fig. 2,) illustrates this point. The case was a typical broncho-pneumonia, about which there could be no doubt, but the field shown might pass for one of alveolar pneumonia with a richly fibrinous exudate. It is not possible to distinguish between the two types of pneumonia by the characters of the alveolar contents in a limited field.

Atypical or mixed cases.

In any large series of cases of pneumonia there are likely to be some in which the pneumonic process appears to be of alveolar type in one part and broncho-pneumonic in another. This combination of the types in a single case is considered by some observers to occur fairly often. It has been indicated above that the presence of small areas which resemble alveolar pneumonia is not uncommon in confluent broncho-pneumonia, but we do not consider that all cases which show these ought to be classed as 'mixed' cases. Our series included only five to which this term seemed properly applicable. Three of them have been examined with whole lung sections. Two are illustrated (Plate II, Fig. 7 and 8; Plate III, Fig. 10). In these cases unmistakable broncho-pneumonic lesions were present in parts of one or both lungs. But each presented in addition a large area, usually extending to one whole lobe at least, the characters of which were, in most particulars, those of alveolar pneumonia rather than confluent broncho-pneumonia. Microscopic examination revealed that the appearances throughout the consolidated lobe were

in the main those of alveolar pneumonia, and parts might be perfectly typical, but in certain portions some interstitial inflammation was present in the bronchial walls. Although our first inclination was to regard these as examples of unusually massive confluent broncho-pneumonia, detailed study did not confirm that opinion. Both types of pneumonia were certainly present. The sequence of events in such cases is a difficult matter to determine. Either alveolar pneumonia must be superimposed upon an initial bronchitis, or the primary condition is alveolar pneumonia and broncho-pneumonia arises as a secondary development. We are of opinion that the latter event may occasionally occur.

Broncho-pneumonia usually develops from bronchitis. When the infection, originally located in the bronchial lumen, penetrates the wall and invades the peribronchial lymphatics, broncho-pneumonia results. If this view be correct, the critical step in the development of broncho-pneumonia is the invasion of the bronchial wall and lymphatics by the infecting agent. Should a similar invasion occur during the course of a primary alveolar pneumonia, the subsequent development of the case would be after the manner of broncho-pneumonia. In ordinary cases of alveolar pneumonia this does not occur. In our previous paper⁶ we emphasized the fact that in typical alveolar pneumonia the bronchial walls and the whole framework of the lungs are remarkably free from inflammatory infiltration. But in that paper we described certain cases which were atypical in that the bronchial walls in the consolidated parts did show inflammatory infiltration, that is to say, the beginnings of interstitial inflammation and peribronchial lymphangitis. Had those patients survived a little longer, we believe that the infection might have spread to other parts of the lungs, and that the lesions resulting from that spread would have taken the form of broncho-pneumonia. We suggest, therefore, that broncho-pneumonia may develop either from bronchitis or, as a rare event, from a patch of primary alveolar pneumonia in which a progressive lymphangitis takes origin.

DISCUSSION.

The conception of broncho-pneumonia set forth in the foregoing pathological study raises certain questions bearing upon the general problem of that disease, and offers an explanation of some of its clinical features. The greater severity of broncho-pneumonia compared with alveolar pneumonia is manifested clinically in the prolonged course, the tendency for undesirable consequences to follow, and the high mortality. It is manifested pathologically by the widespread distribution of the lesions in the lungs, and more particularly by the progressive involvement of the lung substance and lymphatics in the inflammatory process. An inflammation in which the infection invades the tissues in this way is obviously one which is likely to spread widely, almost certain to persist for a long time—if it be not quickly fatal—and liable to resolve imperfectly and to leave permanent effects. In view of the nature of the pathological change, it seems unlikely that true broncho-pneumonia

can ever end by crisis and rapid resolution, and it is not surprising that the usual course in cases which recover is a slow and irregular lysis, subject to many interruptions.

The course of a pneumonic infection in a child would seem to depend upon whether or not this persistent involvement of the lymphatics by the infection occurs. If it does not, the result is alveolar pneumonia, a disease which is attended by a relatively trivial mortality and usually ends in a perfect recovery. If, on the contrary, the infection does thus invade the lymphatics, the result is broncho-pneumonia, with its intimidating mortality and tendency to cause permanent disability in a proportion of the survivors. The important problem to be explained is why the broncho-pneumonic infection should result in this massive and progressive invasion of the lung substance and lymphatics.

It was pointed out in our previous paper⁶ that in alveolar pneumonia the infection is located chiefly in the alveoli, whose walls possess no lymphatics. Therefore, the development of lymphangitis is unlikely. In broncho-pneumonia the infection is primarily and chiefly in the bronchi, whose walls possess a rich supply of lymphatic vessels. Conditions are therefore more favourable for the development of lymphangitis. Yet in both diseases, it is generally agreed, the infection reaches the lungs by the air passages; in both it must pass through the bronchi. According to Blake and Cecil⁷ lobar (alveolar) pneumonia experimentally produced begins with lymphangitis in the bronchial walls. This, however, soon passes off with the onset of consolidation, when the infection passes out into the alveolar spaces. If this view be correct, it would seem that broncho-pneumonia results from the persistence and spread of this primary lymphangitis. Probably, then, the explanation of this important difference between the two types must be sought, not in any different mode of infection, but either in a special quality of the virus or in a condition of predisposition of the patient.

The association between broncho-pneumonia and other acute infective fevers, such as measles, whooping-cough and influenza, is well known. In these cases the antecedent fever acts in some way as a preparation for the subsequent in-road of the pneumonic infection. As already stated, it is when some definite predisposing condition of this kind has been present that the interstitial inflammation is commonly found in its most extreme form.

But broncho-pneumonia in children is often primary, without an antecedent infective fever. Some cases of apparently primary broncho-pneumonia may be instances of influenzal pneumonia, in which the nature of the initial illness is not recognised. But it can hardly be supposed that this explains all cases in which there is no obvious sequence of another infection. Nor do we think it likely that the explanation of these primary cases will be found in the constant presence of a virus of specific quality or special virulence. Rather is it to be sought in some condition of the patient, non-infective, yet predisposing in its effect.

Among predisposing conditions of this kind are age (under two years), bottle feeding, unhygienic surroundings, lack of fresh air and sunshine, rickets. That these conditions predispose a child to broncho-pneumonia is well known.

The comparative rarity of broncho-pneumonia, either primary or secondary, among better-class children, emphasizes the importance of home environment in preventing or promoting predisposition in a child.

It would follow from this, as a general conclusion, that the prevention of broncho-pneumonia in children depends to a corresponding extent upon the control of predisposing conditions.

ABSTRACTS OF CASES ILLUSTRATED.

Case I. (Plate I, Fig. 1, and Plate III, Fig. 11.) *Acute disseminated broncho-pneumonia following measles.* Male, aged 3 years and 7 months. Breast-fed for 1 year. Scarlet fever at 1 year. Pneumonia at 2 years. Fatal illness started with measles 3 weeks before admission. Admitted moribund and died almost at once.

Post-mortem, development and nutrition fairly good. Much thin muco-pus in upper respiratory passages. Scanty fibrinous pleural exudate on both sides. Lungs voluminous, and extensively and almost equally affected with small patches of pneumonia showing as tiny yellow spots round bronchi and bronchioles. Little tendency to confluence. Lung substance very moist, large amounts of frothy fluid exuding on pressure. Mediastinal glands swollen and of a deep red colour. Heart grossly dilated in all chambers and muscle pale and flabby. Moderate degree of toxic change in solid abdominal organs.

Case II. (Plate I, Fig. 2, and Plate IV, Fig. 14 and 15.) *Acute disseminated broncho-pneumonia with confluence in parts, of 12 days' duration.* Female, aged 1 year. Fourth child. One died at 3 months. Two basement rooms; gas burning all day. Breast-fed for 3 weeks. Thereafter on condensed milk. First tooth at 9 months. Chicken-pox at 7 months, with middle ear inflammation. At 9 months, in hospital for 2 weeks with sharp respiratory illness. Continued to cough. Readmitted 4 days before death. Had been acutely ill for 8 days, with severe cough and noisy breathing. Big pale flabby child. Four teeth. Temp. 102. Pulse 160. Respirations 52. Dyspnoeic and cyanosed. Lower chest drawn in on inspiration. Generalized bronchitis, with doubtful consolidation on both sides. Severe suffocative symptoms before death.

Post-mortem, both pleural sacs partially obliterated by loose organised adhesions. Both lungs widely involved. Broncho-pneumonia mostly discrete, but in right lung (Fig. 2) confluence in middle lobe, postero-medial part of upper lobe, and postero-inferior portion of lower. Microscopic evidence of chronic process in two areas in right lung, one in upper lobe near root and other in middle lobe (this chronic interstitial process, as well as organized pleural adhesions regarded as legacy of respiratory illness 3 months before death). Mediastinal glands large and inflamed. Heart dilated on right side. Early acute peritonitis in upper abdomen. Marked toxic changes in spleen, liver and kidneys. No evidence of rickets or lymphoid hyperplasia. B. Pfeiffer grown in pure culture from heart blood and in mixed culture from lung substance.

Case III. (Chart A, Plate I, Fig. 3 and 4.) *Acute broncho-pneumonia, with widespread confluence, of 4 weeks' duration.* Male, aged 1 year. Father unemployed. Three other children. Bad home. Artificially fed from birth on boiled cow's milk. Severe digestive disturbance at 2 months. Hernia operation at 4 months. At 10 months, in hospital for 3 weeks with dyspepsia. Subsequent progress unsatisfactory. Readmitted 3½ weeks before death, having been acutely ill for 3 days. Symptoms at outset mainly gastro-intestinal, but illness obviously respiratory. General state of nutrition poor. Acute bronchitis passed into typical broncho-pneumonia. Mouth and nasopharynx became septic and double otitis developed. Went steadily downhill. For temperature, etc., see chart.

Post-mortem, body extremely emaciated. Bilateral fibrinous pleural exudate. Very extensive broncho-pneumonia in both lungs (Figs. 3 and 4), especially right (Fig. 3). Posterior part of each base solid, and throughout rest of lungs large pneumonic patches of irregular shape and size, with intervening crepitant areas. Yellow mottling characteristic of confluent broncho-pneumonia prominent in solid areas. Smaller bronchi dilated. Mediastinal glands enlarged and red, and mediastinal tissues cedematous. No gross dilatation of heart. Very slight toxic changes in liver and kidneys. Spleen small.

Case IV. (Plate II, Fig. 5 and 6; Plate III, Fig. 12; Plate IV, Fig. 13 and 17.) *Acute broncho-pneumonia, with confluence at bases, of 8 days' duration.* Female, aged 7 months. Illegitimate child, adopted by deaf and dumb woman. Fed on cow's milk and oatflour. No previous illness. Admitted to hospital with 4 days' history of fever, cough and heavy breathing. Temp. 105. Pulse 176. Respirations 56. Indifferently nourished. Flushed and distressed, with indrawing of lower chest. Crepitations most numerous at bases. Cyanosis a conspicuous feature. Steam, oxygen and bleeding of no avail. Died 4 days after admission.

Post-mortem, much muco-pus in large air passages. Pleural surfaces sticky but without definite exudate. Numerous subpleural petechial hæmorrhages over pneumonic patches. Extensive pneumonia in lower lobes of both lungs (Fig. 5 and 6). Large areas in each lung relatively free from pneumonia, but no dubiety as to broncho-pneumonic nature of consolidation. Presence of air in connective tissue framework of lung (interstitial emphysema) a conspicuous feature at autopsy (shown very well in large sections). Heart muscle pale and soft, and chambers greatly dilated. Relatively slight toxic changes in organs.

CHART A.

TEMPERATURE AND WEIGHT CHART OF CASE III.

Case V. (Plate II, Fig. 7 and 8.) *Acute pneumonia of mixed type, of 2 weeks' duration.* Male, aged 2 years and a half. Home surroundings good. Bottle-fed (cow's milk). Much trouble with feeding in first year. Always delicate. Late in walking. No previous respiratory illness. Chickenpox at 1 year and measles at 2. Made good recovery from latter illness. Fatal illness started with series of fits a week before admission. Fevered. No cough noticed for first 3 days. No vomiting. Became delirious and developed squint. Thought to have meningitis. Small thin child. Semi-comatose on admission. Temperature 103. Pulse 160. Respirations 48. Breathing not embarrassed. Consolidation suspected in right upper lobe. Cerebro-spinal fluid not abnormal. Later pneumonia obvious in right lower lobe. Child very toxic. Died 6 days after admission, having been unconscious for some hours before death.

Post-mortem, slight icteric tinge in skin and conjunctivæ. Brain and meninges normal except for hyperæmia. Very thick muco-pus in upper air passages. Fibrinous pleurisy on right side, with 1 ounce of thick pus (pneumococcal) at base posteriorly. Left pleural sac normal. Lungs afforded striking contrast. Left (Fig. 8) relatively healthy; some bronchitis and very early broncho-pneumonia. Right (Fig. 7) extensively consolidated. Lower lobe totally solid. Greyish red on section. Colour and consistence less uniform than in typical alveolar pneumonia. Right upper lobe more irregularly consolidated. One small area of pneumonia in middle lobe. Much thick pus in bronchi throughout lung. Condition

in lower lobe like lobar pneumonia, with atypical features of severe bronchitis and inflammatory infiltration of alveolar septa in certain areas. Condition in upper lobe more suggestive of broncho-pneumonia. Mediastinal glands moderately enlarged and pinkish-grey. Some pallor of myocardium but no dilatation of heart. Fairly well-marked toxic changes in organs.

It is suggested that in this case the first development was a perfectly typical alveolar (lobar) pneumonia, that the inflammatory process obtained a more than usually severe hold on the bronchial walls, and that the disease developed thereafter along the lines of broncho-pneumonia.

Case VI. (Plate III, Fig. 9, and Plate IV, Fig. 18.) *Broncho-pneumonia of several weeks' duration, following measles.* Female, aged 2 years. Father a hawker. Three other children alive, one suffering from abdominal tuberculosis. Another had died of tuberculosis. Breast-fed for 1 year. Very late in cutting teeth (16 months). Always delicate and subject to cough. Treated for rickets. Measles 5 weeks before admission. Details of subsequent progress uncertain, but evidently had severe cough and lost much weight. On admission, very ill and emaciated, with cyanosis, laboured breathing, and troublesome cough. Consolidation obvious in both lungs, especially right. Condition thought to be tuberculous, but no bacilli in sputum or fæces. Died 4 days after admission. Very little pyrexia while under observation.

Post-mortem, much very thick purulent secretion in air passages. Right pleural sac obliterated by recently organised adhesions. Left pleural sac healthy. Both lungs extensively involved. Right lung (Fig. 9) the more completely consolidated. On section, innumerable small sharply defined yellow patches of broncho-pneumonia, with much pus in, and usually dilation of, the central bronchiole. Intervening lung tissue collapsed. Features referred to well shown in large section (Fig. 9) and in detail in Fig. 18. Left lung very similar, but pneumonic patches more discrete, superficially resembling miliary tubercles. Mediastinal glands greatly swollen, especially on right side. Toxic changes in organs remarkably slight. No tuberculosis in body.

Case VII. (Plate III, Fig. 10.) *Pneumonia of mixed type.* Male, aged 1 year and 3 months. Artificially fed on cow's milk. First tooth at 6 months. At 2 months, had bronchitis, and at 8 months a rather severe respiratory illness which was probably broncho-pneumonia and from which recovery was incomplete. Fatal illness started suddenly 4 weeks before death and was regarded as severe bronchitis with the gradual supervention of pneumonia late in the illness.

Post-mortem, marked emaciation. Acute fibrinous pleurisy on right side. Massive consolidation of whole upper lobe of right lung (Fig. 10), which had naked-eye and microscopic characters of alveolar (lobar) pneumonia in an early stage. Degree of infiltration of bronchial walls in this lobe unusual in alveolar pneumonia, however. In middle and lower lobes, acute capillary bronchitis and typical broncho-pneumonia. Left lung much less affected, but the seat of bronchitis and a number of patches of combined collapse, œdema and pneumonia. Mediastinal glands very large. Heart greatly dilated. Toxic changes in organs not extreme. Pneumococci in culture from right upper lobe.

The clinical record indicates that pneumonia did not become obvious until the patient had been ill for some considerable time and the pathological findings are in keeping with this observation. It has been very difficult to determine the precise sequence of events in this case, but it looks as if a pneumonia of alveolar type had been superimposed on an initial bronchitis.

SUMMARY.

1. An analysis of 140 fatal cases of broncho-pneumonia in children is presented. The age incidence was:—In the first year, 70; in the second year, 48; from two to twelve years, 22 cases. Thus 84 per cent. of the total occurred in the first two years.

2. A description of the morbid anatomy and histology of broncho-pneumonia in children is given, based on the appearances of the lungs at autopsy and on microscopic study of large sections.

3. Of the total (140), 5 cases were atypical or mixed, showing the characters of alveolar (lobar) pneumonia in one part and of broncho-pneumonia in other parts of the lungs.

4. The essential pathological process in broncho-pneumonia, and its bearing on the clinical features and ætiology, are briefly discussed.

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