STUDIES OF PNEUMONIA IN CHILDHOOD.

II. ALVEOLAR (LOBAR) PNEUMONIA.

BY

CHARLES McNEIL, M.A., M.D., F.R.C.P., AGNES R. MACGREGOR, M.B., Ch.B., M.R.C.P., & W. ALISTER ALEXANDER, M.B., Ch.B., F.R.C.P.

(From the Royal Edinburgh Hospital for Sick Children, and the Laboratory of the Royal College of Physicians, Edinburgh).

In the first paper of the series, a statistical analysis of pneumonia and bronchitis in childhood was presented, based on two series of cases, clinical and post-mortem. These were reviewed and classified into various types. The present paper deals with that type of pneumonia usually called lobar, giving a general account of its morbid anatomy and histology, and a report of three cases. It is advisable to explain that we use the term 'alveolar' instead of 'lobar' to designate this type of pneumonia. The reasons for this preference have been given in the preceding paper and will again be alluded to in the course of this paper.

After Barthez and Rilliet had clearly differentiated the type broncho pneumonia in infants and young children, its great frequency was established, and the view was held by many that at this early period of life croupous or lobar pneumonia scarcely ever occurred. Thus Gairdner¹ in 1853 referred to this controversy on the relative frequency of lobular and lobar pneumonia in early childhood in the following passage :—

Some of the earliest writers on the subject distinctly state that of the two forms of pneumonia, the lobular and the lobar, the former *alone* is to be found in early infancy, the latter *alone* beyond that age. The latest authorities, in some instances at least, will be found to maintain the same opinion, and notwithstanding the distinct observations of Barthez and Rilliet, Legendre and Bailly, Friendleben and others as to the occurrence of *lobar* hepatization in young infants. . . .

In 1888 Henoch² summed up the prevailing contemporary view of the controversy as follows :—

Although catarrhal or broncho-pneumonia is the commonest inflammatory affection of the lung in childhood, yet the view which formerly obtained as to the rareness of the croupous form has long been done away with, and rightly too. Between the third and the twelfth years this disease is indeed very common, and also in the first two years of life it is by no means rare.

The weight of clinical opinion at the present day seems to take the same view. Table I shows the relative numbers of cases of alveolar (croupous, lobar) pneumonia occurring in childhood before and after two years as reported by various authors.

TABLE I.
ALVEOLAR (LOBAR) PNEUMONIA.

Author.	Date.	No. of cases.	No. under 2 yrs.	Percentage under 2 yrs.
Coutts ³	1902	86	43	50%
Dunlop ⁴	1907	147	45	30.5%
Hutchison ⁵	1926	70	35	50%
McNeil, Macgregor, and Alexander ⁶	1929	386	164	45.2%

These views, old and new, are, however, largely based on clinical diagnosis, corrected to some extent by post-mortem evidence. In a recent discussion on the subject, Findlay⁷ strongly reasserted the old doctrine. He maintained that practically all cases of primary pneumonia under the age of three years belonged to the type broncho-pneumonia and based this statement on the evidence of 65 consecutive autopsies of pneumonia in children. In a recent number of this journal, Olive Somerville⁸ has published a detailed analysis of this post-mortem material. Out of the total, only one typical lobar pneumonia was found and that in a child aged five years. There were two other cases, both in children under two years, which might be classed as lobar pneumonia but which presented atypical features. The principal criterion of diagnosis in this investigation was the microscopic examination of relatively small pieces of lung tissue.

Although the question in dispute cannot be completely settled by post-mortem evidence, Findlay's results, if confirmed, would go far to overthrow this long preponderant view that alveolar or lobar pneumonia occurs throughout childhood and is fairly common even in infancy. We therefore present a series of fatal cases of alveolar (lobar) pneumonia, the majority of them in children under two years, in which the post-mortem appearances are described, and in which we offer macroscopic and microscopic evidence in justification of the diagnosis of this type of pneumonia.

PATHOLOGICAL STUDY.

Alveolar (lobar) pneumonia is comparatively seldom seen in the post-mortem room of a children's hospital. In uncomplicated cases the mortality is low. The cases which come to autopsy are, as a general rule, those of unusual extent or severity, those in which some dangerous complication has developed, or those in which some pre-existent abnormality has prejudiced the patient's chances of survival. Opportunities of studying the pathology of this disease in straightforward uncomplicated cases of the kind which usually recover are therefore relatively few. This fact occasions some difficulty in determining exactly what are the usual pathological characters of alveolar pneumonia in childhood and infancy, and is responsible for the uncertainty concerning the frequency of this type.

In our previous paper* it was stated that of a series of 945 autopsies in cases of death from all causes performed in the Royal Edinburgh Hospital for Sick Children during the period from July, 1922, to October, 1928, inclusive, 23 (2·5 per cent.) were cases of alveolar pneumonia, and 140 (14·8 per cent.) were cases of broncho-pneumonia. There were also examples of other types, the characters and number of which were stated. A short survey of these 23 cases of alveolar pneumonia may be of some interest as an introduction to a study of the morbid anatomy and histology of this type.

Age.—The ages of the children ranged from 4 months to 4 years and 9 months. Twelve were under one year old ; six were between one and two years. Thus more than three-quarters of all the cases were in children under two years of age.

Sex.—There were seventeen boys and six girls. Although the number of cases is far too small to warrant any drawing of conclusions with regard to sex incidence, the preponderance of males is striking, and is in agreement with the figures derived from a much larger series of clinical cases which was reviewed in our previous paper.

Complications.—The incidence of serious complications was very high in this series. In 15 of the 23 cases the pneumonia was accompanied by some complicating condition (either resulting directly from the pneumonic infection or pre-existent), which greatly increased the gravity of the illness, and was in some instances certainly the actual cause of death.

The nature and frequency of these complications were as follows :—empyema, 8 ; pericarditis, 3 ; meningitis, 3 ; abscess in lung, 3 ; peritonitis, 1 ; acute nephritis, 1 ; suppurative pyelonephritis, 1 ; congenital defect of the heart, 1.

Morbid Anatomy.

Site and extent of consolidation.—In 8 of the 23 cases, the right lung only was affected ; in 8, the left lung only ; in 7, both lungs were involved.

In 10 cases, the pneumonia was confined to one lobe. In 13 cases, more than one lobe was wholly or partly consolidated. The order of frequency with which the various lobes were affected was as follows :—left lower lobe, 13 ; right upper lobe, 9 ; right lower lobe, 8 ; left upper lobe, 5 ; right middle lobe, 4.

In ten cases the area of consolidation extended to one whole lobe or more. In eight cases the pneumonia was present in a single patch the extent of which was less than one lobe. In the remaining five cases, pneumonia was present in more than one lobe, but affected only a part of each. In no case was an entire lung consolidated, although there were instances in which the unaffected part was very small (Plate II, Fig. 5).

In those cases in which the pneumonic area was less than a whole lobe, it was, as a general rule, of considerable size, often about half a lobe (Plate II, Fig. 6) ; but in certain instances no more than a patch about an inch in diameter was consolidated (Plate II, Fig. 7).

When consolidation affected only a part of a lobe, its position in the lobe was very variable. It was more often posterior than anterior. It very seldom failed to reach the pleural surface at some point. In two cases it was situated deeply near the root, without having extended to the surface. Sometimes its position was peripheral, so that it did not approach the root (Plate II, Fig. 7.)

It is evident from these observations that, although this disease is commonly called lobar pneumonia, involvement of a whole lobe or nearly a whole lobe is not an essential character of alveolar pneumonia in children. In our experience, it is more usual to find consolidation of a part of a lobe or lobes. There is far more variation in the extent of consolidation in the alveolar pneumonia of childhood than in the corresponding disease (lobar pneumonia) of adult life. While it may be very extensive, the pneumonic area is often relatively smaller in children than is usual in adults. If this be true of fatal cases, it is likely to be even more often true of the slighter cases which recover : and this is confirmed by clinical experience. At the same time, it should be pointed out that a majority of our cases showed pneumonia in more than one lobe : but it does not follow, of course, that this is of equally frequent occurrence in non-fatal cases. It is apparently rather unusual for an entire lung to be completely consolidated.

Appearance of the pneumonic area.—When pneumonia in a child is encountered at autopsy, the problem which confronts the pathologist is the diagnosis between alveolar pneumonia and confluent broncho-pneumonia. In typical cases the question is not a difficult one, for an area of alveolar pneumonia presents certain well-defined macroscopic characters by which it may be distinguished from confluent broncho-pneumonia.

The affected part, no matter of what extent, is massively and completely consolidated. As every alveolus is filled with exudate, no collapse is present within the area, which accordingly projects well above the level of neighbouring unconsolidated lung. Its consistence is solid, firm, and uniform. It lacks the nodular character and somewhat rubber-like resilience which in most broncho-pneumonic lungs, even of the most confluent type, are produced by an alternation of zones of complete consolidation with zones of partial collapse within the affected part. Where the pneumonic area abuts upon aerated lung substance, the margin is usually extremely definite (Plate II, Figs. 6 and 7). This also is a point of difference between alveolar pneumonia and confluent broncho-pneumonia.

Should the pneumonia extend to the surface of the lung, the pleura overlying it is invariably inflamed. Usually there is only a thin layer of dry fibrinous exudate, and this may or may not be present beyond the limits of the pneumonic area. Apart from the cases in which empyema had developed, we have rarely seen either a copious fibrinous exudate or a pleurisy with effusion. It is probably true to say that in childhood cases, in which the pleurisy is severe, usually go on to empyema.

On section, the appearance of the pneumonic area naturally depends upon the stage which has been reached. If resolution has not begun, the cut

surface has a dry, dull appearance and the projection of plugs of exudate from the alveoli often imparts to it a finely granular character. This differs from the appearance of a broncho-pneumonic lung, which on section usually presents a moist and shiny surface.

The colour varies from red to grey according to the stage of the disease. Most of our cases were at the stage of early grey hepatization at the time of death, but there were several examples of red hepatization. The colour of the cut surface is usually fairly uniform, but rarely perfectly so. Alternations of red and grey are often present throughout the patch due to differences in the composition of the exudate ; but these variations in colour are independent of the distribution of bronchi, and the appearance of mottling produced by them is quite different from the definite peri-bronchial mottling which is characteristic of broncho-pneumonia. In early cases there is often a zone of intense hæmorrhagic congestion at the spreading edge of the patch.

Bronchi and bronchioles within the pneumonic area are not conspicuous ; do not show inflammatory thickening of their walls ; are not noticeably dilated ; and do not contain large quantities of pus, although a certain amount of exudate is present in them. In unconsolidated parts of the lungs the bronchi are healthy. The absence of bronchitis, especially of the smaller tubes, in those parts where there is no consolidation is an important feature of typical alveolar pneumonia. Some hyperæmia of the mucous membrane of the large bronchi and of the trachea, and the presence therein of exudate derived from the pneumonic area, are usual.

Apart from the definite patch or patches of pneumonia, the lungs are free from consolidation. There is usually some general congestion, and a degree of acute vesicular emphysema may be present, but usually very much less than in broncho-pneumonia. Interstitial emphysema, which is of common occurrence in broncho-pneumonia, we have never seen in a case of alveolar pneumonia.

The tracheo-bronchial and root glands show swelling and active hyperæmia (Plate I, Fig. 3), but the very great enlargement and intense inflammation of these glands, so often associated with broncho-pneumonia (Plate II, Fig. 8), have not been found in any case in our series.

Morbid Histology.

In the pneumonic area complete consolidation of the whole part affected is a constant feature. Every alveolus is filled with exudate, and areas of collapsed or partially collapsed alveoli are not present.

The exudate.—There is considerable variation in different cases in the composition of the exudate which fills the alveoli, depending partly, but not entirely, upon the stage of the disease. There would seem to be a tendency for the exudate to be of a more cellular and relatively less fibrinous character in children than in adults. This is true especially in very young children, in whom departure from the typical standard of the adult case is apt to be greater in this and in other respects. Nevertheless, this rule is not of universal

application, for even in infants the exudate may be composed mostly of fibrin. Fig. 9 (Plate III), which is taken from the lung of a male infant, aged 9 months, shows an exudate in which fibrin greatly preponderates.

There is always some variation in the composition of the exudate in different parts of the pneumonic area : and, as in adult cases also, the more cellular parts are often those nearest to the terminal bronchiole, the outlying alveoli of the lobule containing a more fibrinous exudate. In certain cases, which in other respects conform to the alveolar type, there is very little formation of fibrin, and the exudate is almost entirely composed of polymorphonuclear leucocytes. We are of opinion that the amount of fibrin in the exudate is not a reliable guide in differentiating alveolar pneumonia from broncho-pneumonia, especially when only small sections are used, and the microscopic examination is thus confined to a limited portion of the consolidated part. Most lungs with confluent broncho-pneumonia contain areas in which fibrin is as plentiful in the alveoli as in any case of croupous pneumonia in child or adult.

The alveolar walls.—It is characteristic of alveolar pneumonia that the products of inflammation are thrown out into the alveolar spaces and that the walls do not show any marked degree of infiltration with inflammatory cells (Plate III, Fig. 10). Congestion of capillaries in the early stages, and sometimes swelling of the lining epithelium before it is cast off, may cause the walls to look somewhat thicker than in health (Plate III, Fig. 9) ; but the dense infiltration with inflammatory cells, which so greatly obscures the alveolar walls in broncho-pneumonia, is characteristically absent in the alveolar type.

The bronchi.—Within the pneumonic area the bronchi always contain inflammatory exudate which resembles that present in the alveoli, although it is often relatively richer in leucocytes. When the alveolar exudate is fibrinous, the bronchi are often plugged with fibrino-cellular exudate, a condition rarely seen in broncho-pneumonia. When the exudate in the alveoli is of a very cellular type, in the bronchi it may have an almost purulent character. Evidence of severe inflammation of the bronchi themselves is, however, absent in typical cases. If it be present, the case is either atypical, or the diagnosis of alveolar pneumonia is mistaken. The blood vessels of the bronchial wall may share to some extent in the general hyperæmia of the part : but the wall is not significantly swollen, nor infiltrated with polymorphonuclear cells, and quite often even the epithelial lining remains surprisingly intact (Plate III, Fig. 11). The healthy condition of small bronchi in the midst of an area of consolidation is clearly shown in Fig. 12 (Plate III).

In some of our cases, especially those of young children, a certain amount of lymphocytic infiltration has been found in the bronchial walls in the pneumonic area. Some observers hold that this is characteristic of broncho-pneumonia, and would classify as such any case in which it was found. Our experience has been that in broncho-pneumonia the cellular infiltration of the bronchial walls is much more severe : that among the infiltrating cells polymorphonuclear leucocytes rather than lymphocytes predominate : that the walls are greatly swollen in addition to being infiltrated : and that the lining epithelium is usually destroyed. In the absence of other evidence of acute

inflammation of the bronchial wall, a certain amount of lymphocyte infiltration need not be regarded as inconsistent with a diagnosis of alveolar pneumonia. The lymphocytes are probably derived from the microscopic lymph nodes which occur normally in the walls of bronchi and readily undergo hyperplasia in the presence of any inflammation in their neighbourhood.

Interlobular septa and perivascular stroma.—There are often large deposits of fibrin in the interlobular septa, causing them to be greatly broadened out so that they form a conspicuous feature of the section (Plate III, Fig. 13). A certain number of polymorphonuclear leucocytes may be entangled in the fibrin meshwork. Dense aggregations of leucocytes in the septa are unusual. We have found this fibrin deposit in the septa very frequently in cases of alveolar pneumonia, and comparatively seldom in broncho-pneumonia, even where there has been extensive confluence. The perivascular fibrous tissue may show the same condition, but often it is remarkably free from any inflammatory change (Plate III, Fig. 12). Sub-pleural and septal lymphatic vessels may be dilated and filled with a fibrinous coagulum. The pleura practically always shows an exudate, usually scanty and mostly fibrinous except when empyema is present.

The margin of the pneumonic area. When the area of pneumonia abuts upon unconsolidated lung, the edge is a fairly definite one (Plate II, Fig. 6). Sometimes the consolidated patch is bounded in part by interlobular septa, but a boundary of that kind is rarely complete along the whole extent of the patch. If the pneumonia be still spreading at the time of death, there may be a narrow zone of intense congestion and hæmorrhage at the edge of the consolidated area. Sometimes there is merely a zone in which the capillaries in the alveolar walls are congested and the spaces contain an œdematous material. In either case the appearances at the spreading edge suggest an extension of the inflammation by continuity from alveolus to alveolus in a more or less uniform manner all along the margin of the patch. This is quite different from the manner of spread in broncho-pneumonia, which is by way of the bronchi, so that beyond the edge of an area of confluent broncho-pneumonia outcrops of consolidation appear in small patches surrounding bronchioles (Plate II, Fig. 8).

In unconsolidated parts of the lungs the alveoli and bronchi alike are free from inflammatory exudate. Apart from some general hyperæmia and perhaps slight acute vesicular emphysema, the parts of the lungs which are not consolidated are healthy. This is exceedingly characteristic of typical alveolar pneumonia (Plate I, Figs. 2 and 4, and Plate II, Fig. 6), and is in sharp contrast to the state of affairs in confluent broncho-pneumonia (Plate II, Fig. 8).

DISCUSSION.

The features of alveolar pneumonia to which special importance is attached in distinguishing it from confluent broncho-pneumonia are the following:—the localization of consolidation to a certain definite area: the absence of generalized bronchitis; the absence of anything more than a very trivial

inflammation of the bronchi within the pneumonic area ; the freedom of the bronchial and alveolar walls and stroma generally from damage by infiltration with inflammatory cells.

The last of these deserves special emphasis. Alveolar pneumonia, in marked contrast to broncho-pneumonia, is a disease in which the inflammatory reaction results in the throwing out of the products of inflammation upon the surface (*i.e.*, the alveolar spaces), and in which the substance of the lung shows no severe or persistent involvement. A reason for this immunity of the interstitial frame work may be found in the fact that the infection is located in the alveoli, whose walls possess no lymphatics. Invasion of the lymphatics of the lungs by the infection, with consequent lymphangitis and acute interstitial inflammation, is therefore not liable to occur. In broncho-pneumonia, on the contrary, where the infection is centred chiefly in the bronchi whose walls possess a rich network of lymphatic vessels which are readily invaded, the occurrence of lymphangitis and acute interstitial inflammation is obviously a strong probability.

According to Blake and Cecil⁸, however, lobar pneumonia experimentally produced in monkeys begins as a lymphangitis in the walls of bronchi which is followed by alveolar inflammation ; and with the onset of consolidation of the alveoli, this lymphangitis passes off. The application of this result to human cases has yet to be demonstrated, but if it be correct, it is significant that the lymphangitis is not persistent or progressive, but a transient affair which disappears when the inflammatory reaction is fully established and the infection passes out into the alveolar spaces.

The absence of persistent lymphangitis and inflammatory involvement of the interstitial framework of the lungs in alveolar pneumonia is a significant pathological feature which has an important bearing upon certain clinical features of the disease. The relatively short course, the abrupt termination of serious symptoms, and the rapid return of the lung to a normal state, which are characteristic of alveolar pneumonia in cases which recover, would all be unlikely, if not impossible, in the presence of severe interstitial inflammation and lymphangitis. Further, because the alveolar septa and bronchial walls suffer no disorganization during the active stage of the inflammation, when recovery occurs the exudate is removed from the alveoli by resolution and there is no occasion for a process of reorganization. Therefore, the lung is able to recover perfectly and rapidly, without permanent change in structure which would inevitably cause disablement of function. Hence the comparative rarity of such conditions as fibrosis or bronchiectasis occurring as a sequel of alveolar pneumonia.

Atypical cases. It is clear that in typical cases it is no very difficult matter to distinguish between alveolar pneumonia and confluent broncho-pneumonia, for there are many points of difference sufficiently clearly defined. But from time to time cases are met with which are really difficult to place in either category, because of their atypical features. Among the cases which present this problem there are two groups :—(a) those which deviate in some important particular from the picture of alveolar pneumonia which has been

described: (b) those in which one part of the lung presents the typical picture of alveolar pneumonia, while obvious broncho-pneumonia is present in other parts.

For the purpose of classification in our series, all cases of the latter kind were excluded from the alveolar pneumonia group. With regard to the former, every case must be considered on its merits. In our series, a few cases which presented certain atypical features were classified as alveolar pneumonia, because, after due consideration, the weight of evidence was held to be in favour of that diagnosis. Atypical cases are most frequent in the earlier age periods. After the age of two years the cases tend more and more to approximate to the adult type of 'lobar' pneumonia.

The most important deviation is the involvement of the bronchi within the pneumonic area in active inflammation. No case in which this occurs can be regarded as typical alveolar pneumonia. But we have occasionally found it in cases which in other respects conformed strictly to the alveolar type. The bronchi may be denuded of epithelium, their walls invaded by inflammatory cells and much swollen: in one of our cases they had even suffered considerable disorganization and showed a degree of acute dilatation (Plate II, Fig. 5).

Some observers would undoubtedly class such a case as broncho-pneumonia. Yet, if in all other respects the pathological picture be typically that of alveolar pneumonia, it would appear to us to be more correctly placed in that group. Our experience has led us to believe that the inflammation of the bronchial walls in cases of this kind is probably secondary to a primary alveolar pneumonia, and is not the primary manifestation of infection as in broncho-pneumonia. In other words, it is possible that occasionally the infection, instead of remaining localized in the alveolar spaces, may spread to the bronchi and invade their walls as a secondary development. This is a serious occurrence which, it may be supposed, increases the gravity of the disease and prejudices the chances of recovery. It may be that it seldom, if ever, occurs in cases which recover: even in fatal cases it is rare.

Obviously in cases of this kind the pathological diagnosis must remain in doubt, and a matter of personal opinion. In the Glasgow series^a, all such cases were evidently classed as broncho-pneumonia. We have retained some of them in our alveolar pneumonia group. We would venture to express the opinion that in some of these doubtful cases it would be impossible to come to a reliable decision by a study of the limited field available in ordinary small microscopic sections, and in this respect the whole-lung sections which we have been able to use are of great importance.

ILLUSTRATIVE CASES.

CASE 1. *Double pneumonia of alveolar (lobar) type.* (Plate I, Figs. 1 and 2). Female, aged 3 years, seventh child. Mongolian imbecile. Breast-fed for 18 months. History of diphtheria, with good recovery, seven weeks before the fatal illness. Fat, flabby child. Died within 24 hours of admission after an illness of sudden onset and of six days' duration. Physical signs of consolidation in both lungs. Large heart and marked evidence of circulatory failure.

On post-mortem examination, the right upper lobe was partially and the left lower lobe more completely consolidated. The pneumonic areas were of a deep red colour and the overlying pleura was inflamed. The remaining lobes were free of pneumonia and showed some

emphysema. No general bronchitis was present. There was marked enlargement of the heart, especially on the right side, in association with a large defect in the membranous part of the septum.

Microscopically, the pneumonia in both lungs was at much the same stage (red hepatization). There was no doubt as to its alveolar character. There was the usual exudate in and trivial inflammation of, the bronchi in the consolidated lobes. Both lungs showed evidence of previous damage—a slight fibrosis of unequal distribution, arterial changes, and lymphocytic accumulations in bronchial and other connective tissues. There was no history of a former illness, but this finding was not surprising in view of the susceptibility of mongols to respiratory infections.

CASE 2. *Pneumonia of alveolar type, with relapse.* (Plate I, Figs. 3 and 4). Male, aged 10 months. Second child of unemployed father; home conditions bad. Breast-fed. No previous illnesses. General condition good. Five teeth. Died after a three weeks' illness, in which there were three distinct pyrexial periods in close proximity, followed by low fever in the last few days (Chart I).

CHART I.

RELAPSING ALVEOLAR PNEUMONIA (CASE 2).

The child was not under observation during the first period. The second was associated with consolidation of the right lower, and the third with consolidation of the right upper lobe. During the latter there were signs of meningeal irritation. The left lung remained unaffected throughout.

Post mortem, the left lung and right middle lobe were healthy. The right lower lobe was the seat of an almost completely resolved pneumonia, with early organization of the overlying pleural exudate. The right upper lobe, with the exception of the apex and anterior border, was consolidated; the pneumonia recent; the colour and consistence fairly uniform; the margin sharp; the pleura acutely inflamed. The bronchi contained some pus. There was little to note elsewhere in the body.

On microscopic examination, the left lung showed merely superficial catarrh of the larger air-passages. In the right lower lobe, remnants of exudate in alveoli and the presence of large mononuclear cells provided ample evidence of a recent pneumonia in the final stages of resolution. The process was least advanced in the portion of the lobe adjacent to the root. There was catarrhal desquamation of the epithelium of bronchi, but no active inflammation. In the

PLATE I.

Figs. 1 and 2.—Double alveolar pneumonia in girl of 3 years (illustrative Case 1). Consolidation of right upper and left lower lobes.

Figs. 3 and 4.—Alveolar pneumonia in boy of 10 months (illustrative Case 2). Pneumonia resolving in right lower lobe. More recent consolidation of most of right upper lobe. Left lung unaffected. A=Lymphatic gland at root of right lung.

PLATE II.

Fig. 5.—Alveolar pneumonia in boy of 5 months (illustrative Case 3). Almost complete consolidation of right lung. Some dilatation of bronchi.

Fig. 6.—Alveolar pneumonia in boy of 14 months. Consolidation of half of left lower lobe.

Fig. 7.—Alveolar pneumonia in boy of 4 months. Consolidation of small area at base of right lung.

Fig. 8.—Confluent broncho-pneumonia in boy of 1 year, included for purposes of contrast with preceding illustrations of alveolar pneumonia.

PLATE III.

Fig. 9.— $\times 120$. Alveolar pneumonia in boy of 9 months showing uniform fibrino-cellular exudate in alveoli, with thickening of alveolar walls due to congestion of capillaries and swelling of endothelial and alveolar lining cells.

Fig. 10.— $\times 300$. Alveolar pneumonia in boy of 4 years and 9 months, showing fibrino-cellular exudate in alveoli. Rather later stage than above. No swelling or infiltration of alveolar walls.

Fig. 11.— $\times 150$. Alveolar pneumonia in girl of 3 years (same case as Figs. 1 and 2). Bronchus in pneumonic lobe, showing relative freedom from inflammation and intact epithelium.

Fig. 12.— $\times 100$. Alveolar pneumonia in boy of 9 months (same case as Fig. 9). Small bronchi (B) in consolidated area, with exudate in lumina but undamaged walls. Perivascular connective tissue also free from infiltration. A=Blood-vessels.

Fig. 13.— $\times 120$. Alveolar pneumonia in boy of 9 months (same case as Figs. 9 and 12). Interlobular septum broadened as result of deposit of fibrin.

PLATE I.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

PLATE II.

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 12.

Fig. 13.

right upper lobe, the pneumonia was of alveolar type. Alveoli were completely filled with fibrinous exudate containing polymorphonuclear and catarrhal cells; alveolar walls clearly distinguishable and not infiltrated with inflammatory cells; the exudate was of the same type throughout and not 'zonal' in distribution as in broncho-pneumonia. The bronchi showed up clearly owing to the presence of a cellular exudate, but the epithelium was intact and the substance of the walls not appreciably inflamed. The interlobular septa were cedematous. There was recent exudate on the pleural surface.

The diagnosis of alveolar pneumonia was clear on clinical and pathological grounds.

CASE 3. *Alveolar pneumonia involving most of one lung, with a patch in the lower lobe of the other lung.* (Plate II, Fig. 5.) Male, aged 5 months. Third child. Mother died of puerperal septicæmia. Fed on cow's milk mixtures. Fatal illness lasted two weeks (see Chart II.). Onset

CHART II.

ALVEOLAR PNEUMONIA (CASE 3).

of acute symptoms sudden but preceded by 'cold in head.' Pale (rather thin) infant, with signs of early rickets. Consolidation noted in right lower lobe on sixth day; spread to involve most of lung posteriorly. Effusion suspected but not confirmed by exploration. Cough short and obviously painful. Breathing rapid but not embarrassed. Some accompaniments in left lung throughout illness, but no pneumonia detected.

On post-mortem examination, the whole right lung, except for the middle lobe and the apical portion of the upper lobe, was solid. The colour was a more or less uniform greyish-red. There was well-marked bronchitis, but an absence of peri-bronchial mottling. A thick fibrinous deposit was present on the visceral pleura. The left lung contained an irregularly shaped but fairly well-defined patch of pneumonia in the substance of the lower lobe. It showed no other area of consolidation. The appearance of the pneumonic patch was not that of confluent broncho-pneumonia.

Microscopic examination confirmed the presence of rather severe bronchitis in the consolidated parts; some of the bronchi, in the right lung especially, were denuded of epithelium and appreciably dilated. Nevertheless, the pneumonia in its uniformity and other characters was essentially alveolar in type, not only in the extensively involved right lung but also in the partially consolidated left lower lobe, and no doubt was felt in so diagnosing the case,

SUMMARY.

1. An analysis of twenty-three autopsies in cases of alveolar (lobar) pneumonia in young children is given. Eighteen were under two years of age.
2. The study of this post-mortem material supports the view widely held on clinical grounds that this type of pneumonia is not uncommon in the first two years of life.
3. The morbid anatomy and histology of alveolar pneumonia in infancy and early childhood are described.
4. Atypical forms of alveolar pneumonia in children are briefly discussed.
5. Short clinical and post-mortem reports of three cases of alveolar pneumonia, two of them in infants under one year, are presented.
6. In this study, paraffin sections of entire lungs have been used. Illustrations of these are given.

The large sections illustrated in this and the succeeding papers are the work of Mr. T. D. Hamilton of the Royal College of Physicians' Laboratory, Edinburgh.

REFERENCES.

1. Gairdner, W. T., *Brit. & Foreign Med. Chir. Rev.*, Lond., 1853, XI, 462.
2. Henoch, E., *Lect. on Children's Diseases*, trans. by John Thomson, 1889, I, 399.
3. Coutts, J. A., *Edin. Med. J.*, Edin., 1902, XII, 209.
4. Dunlop, G. H. M., *Tr. Med. Chir. Soc. Edin.*, Edin., 1907-8, XXVII, 199.
5. Hutchison, R., *Lect. on Diseases of Children*, 1925, 210.
6. McNeil, C., Macgregor, A. R., Alexander, W. A., *Arch. Dis. Childhood*, Lond., 1929, IV, 12.
7. Findlay, L., *Brit. Med. J.*, Lond., 1927, ii, 587.
8. Somerville, O., *Arch. Dis. Childhood*, Lond., 1928, III, 194.
9. Blake, F. G., and Cecil, R. L., *J. Exp. Med.*, N.Y., 1920, XXXI, 403, 445.