PNEUMONIA NEONATORUM.*

BY

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OUT of the 80 cases of infantile death there were 21, or 26.25 per cent., in which the cause of death was found to be pneumonia.

Eleven of these occurred in premature and 9 in full-time infants, the ages varying from 8 hours to 5 weeks. The very large proportion occurring in premature infants is to be noted, and illustrates the increased liability to infection on the part of the premature infant. Thus, if we take the proportion of premature to full-time infants as 1 to 10, we arrive at the conclusion that the premature infant is about fourteen times as liable to die from pneumonia as the infant born at full time.

Associated Conditions.—Five of the infants were syphilitic, another was probably so, while another had fracture of the supraorbital plates with extradural haemorrhage overlying them. One of the syphilitic cases showed an extradural haemorrhage in the spinal cord. In many of the cases, especially amongst the premature infants, there were atelectatic areas in the lungs, and in some cases these involved part of one lung and a large part of the other. It is, therefore, probable that atelectasis is a predisposing factor in the causation of pneumonia.

Premature Rupture of Membranes and Pneumonia.

A consideration of some of the cases would seem to show that premature rupture of the membranes can allow ante-natal infection to occur, and that a child may be born not only infected, but even suffering from pneumonia in an advanced stage.

In one case the mother was admitted to hospital after a long delay at the patient's home. The membranes had been ruptured for some time before admission. Forceps were applied in hospital and a blue baby, breathing badly, was born, which died eight hours afterwards.

At *post-mortem* examination the child was found to be at full time and 3,800 grams in weight. The lower lobes of both lungs were of solid consistence, and on squeezing the cut surfaces they exuded a yellowish frothy fluid. The lower lobe of the right lung sank in water, and each pleural cavity contained about two ounces of thin blood-stained serum. Microscopically the lungs showed an advanced degree of catarrhal pneumonia in the stage of grey hepatization. Granular debris was the main content of the alveoli, but some pigment-containing endothelial cells, a few polymorphs, lymphocytes, and an occasional disintegrating red cell with some fibrin could still be made out. At other parts no cells whatever could be found, and the whole appeared to be a necrotic mass, in which even the alveolar walls could scarcely be distinguished.

Cultures from the lung on blood agar yielded an organism of the pneumo-bacillus group. It is impossible that such an advanced degree of grey hepatization could have been reached in the eight hours during which the child lived, and its blueness and difficulty in breathing at birth are strong evidence in support of ante-natal pneumonia.

A second case was a four-para with contracted pelvis. The first two children had been born dead at full time (craniotomy). The third labour was induced, and the child is alive and well. The present labour was also induced by packing the lower uterine segment with gauze; the packing was removed in twenty-four hours, when the membranes were found ruptured. Labour pains did not start, however, for three days, during which the patient received hot douches. Labour was in the end rapid, the child being "shot out." It required resuscitation, and breathing was wheezy and rapid. It lived three days, and during this time it was blue, its breathing difficult, and the abdomen distended.

At *post-mortem* examination the weight was 2,690 grams, length 20 inches, and therefore it was very near, or actually at, full time. The cord was dry and healthy. In the right pleural cavity there was about an ounce of brownish-yellow purulent fluid containing flakes of lymph. The right lung was quite solid except for a small part at the apex. On section it resembled grey marble, greyish and reddish-brown areas alternating, and on squeezing it exuded a brownish purulent fluid. The left lung was crepitant and oedematous, and at its lower margin there were a few well-defined abscesses 1 mm. in diameter. The heart showed marked dilatation of the right auricle and ventricle. The tricuspid valves were puckered and covered with vegetations and dark-coloured blood clot. The mitral valve presented a similar appearance, but less marked, while the aortic and pulmonary valves seemed normal. Microscopically the lungs showed typical grey hepatization with large areas of necrosis, and the liver cloudy swelling and fatty change. Cultures from lung, heart, and pus in the pleural cavities gave a pure growth of *Bacillus coli*.

* This paper formed part of a report on "Stillbirth, its Causes, Pathology, and Prevention" to the Medical Research Council. May, 1921.

The condition was therefore pneumonia and empyema with septic endocarditis, and the short duration of illness, the condition of the child at birth, and the finding of the *B. coli* as the infecting organism, seem to point to the infection being an ante-natal one, due to premature rupture of the membranes allowing infection of the liquor amnii. It should be stated that the mother's puerperium was perfectly normal, and there was not the slightest rise of temperature throughout. These two cases seem to prove the possibility of ante-natal infection and even of ante-natal pneumonia.

Condition of the Lungs.—The following tabular statement shows the condition of the lungs in the 21 cases, determined after naked-eye and microscopic examination:

Ordinary catarrhal pneumonia (1 with pleural effusion, 2 with empyema) ...	11
Interstitial pneumonia with catarrhal pneumonia superadded ...	2
Catarrhal pneumonia (patchy) plus haemorrhage into alveoli and bronchi ...	1
Acute congestion and oedema of the lungs with haemorrhage into alveoli and bronchi ...	5
Interstitial plus catarrhal pneumonia plus haemorrhage into alveoli and bronchi ...	1
Interstitial pneumonia plus congestion and haemorrhage into alveoli and bronchi ...	1
Total ...	21

All the catarrhal pneumonias were in a fairly advanced stage—red or grey hepatization. The alveoli contained chiefly large flattened endothelial cells, generally phagocytic and containing pigment, probably derived from red blood cells, red blood corpuscles, or even a complete polymorphonuclear leucocyte. There were generally exceedingly few polymorphs present, and this applied to all stages, though in the early cases they were slightly more plentiful. This absence of polymorphs shows that in the young infant there is little or no defence against the infecting agent. Red blood cells, generally with pigment, an occasional lymphocyte, a little fibrin and granular debris, were the only other contents. To the naked eye the lung in many of the cases of catarrhal pneumonia presents a somewhat deceptive appearance—namely, it may seem on removal from the chest to be well aerated, be crepitant, float buoyantly in water, and on section appear to be only slightly congested and oedematous. It is only on microscopic examination that the true state is discovered and a patchy catarrhal pneumonia revealed.

There were two cases of interstitial pneumonia with catarrhal pneumonia in patches superadded. The thickened alveolar walls of the former, with the poorly distended alveoli containing catarrhal cells, make it very difficult to distinguish, even microscopically, from the patches of catarrhal pneumonia, but in the former the cells in the alveoli are phagocytic endothelial only, derived from the lining of the alveoli, with the addition of a few red cells. The cells are comparatively few in number and are loosely arranged, while in the latter there are in addition to the endothelial cells many red blood corpuscles, fibrin, with a few polymorphs and lymphocytes, and the mass fills the alveolus more completely, giving to the part a much more solid appearance.

Signs and Symptoms.

In none of these cases of pneumonia had its existence been suspected before death and *post-mortem* examination. There was no rise of temperature, no notable degree of cyanosis, nor rapidity of breathing except in the case of No. 5, where blueness and rapidity and difficulty of breathing had been present from birth, and was naturally ascribed to atelectasis. In this case, too, the abdomen had been somewhat distended, though it is not clear if this sign was also present from birth. A history is generally obtained that the child seemed to be slightly blue and its extremities cold, and did not cry strongly or feed well. In one case where such symptoms were present I was able, on examining the bases of the lungs behind, to find well-marked tubular breathing, and thus a tentative diagnosis of pneumonia was made, though the child at that time was only 30 hours old. It died six hours later, and at *post-mortem* examination both lungs were found to be consolidated. This, however, was an unusually favourable case, and it is probable that in the usual type of patchy catarrhal pneumonia no definite physical signs could be made out even if most carefully looked for.

Acute Haemorrhagic Pneumonia of Infants.

There were, it will be noticed, six cases in which there was congestion with more or less oedema of the lung associated

with haemorrhage into the alveoli and bronchi. Five of these occurred in previously healthy lungs, while in one there was a precedent interstitial pneumonia. The blood in these cases is invariably fresh, and has evidently been poured out just before death. Its amount varies, sometimes tightly filling the alveoli and bronchi over a large area of lung, in other cases filling the alveoli more loosely. There is always a corresponding amount of blood extravasation in the alveolar walls. There is at the same time a certain amount of catarrh of the endothelial cells lining the alveoli, so that the latter contain a few large flattened endothelial cells, generally containing pigment, and similar endothelial cells are found in the alveolar walls. Very rarely is there any actual catarrhal pneumonia in these cases. Sometimes the congestion is accompanied by intense oedema. In 4 of these cases death occurred suddenly: in 3 of these 4 it was preceded by epistaxis; the fourth occurred at the patient's home and no observation was made. The course of events seems to be as follows:

The first stage is that of lung congestion—general, or limited to certain areas. The fragile foetal vessels are unable to stand the strain of the sudden inrush of blood and consequently give way, and blood is poured out into the alveoli and bronchi. The child has generally up to this point appeared to be perfectly well, or at least is so till within an hour or less of death. Death occurs suddenly, probably due to the haemorrhage, and is immediately preceded by epistaxis. The child is blanched; there is no cyanosis, therefore the blood is of different origin from that found in cases of catarrhal pneumonia, where the blood comes from the congested nasal mucous membrane. In the haemorrhagic cases the blood comes from the lung itself, and traces of it may be found on examining the upper air passages. The presence of blood in the alveolar walls of the lung precludes the possibility of that in the alveoli and bronchi having been swallowed; besides, on careful examination one can find some capillaries that have been ruptured and from which blood had been flowing when death occurred.

Neither syphilis nor prematurity seems to be a necessary factor in the causation of these intrapulmonary haemorrhages. One of the six cases was syphilitic and four were premature; therefore it is probable that both of these conditions predispose to it, though in the case in which the haemorrhage was most marked the child was at full time and non-syphilitic.

Naked-eye Appearance of the Lung.—The lung appears congested and oedematous and feels rubbery and semi-solid. On squeezing the cut surface frothy bloody fluid exudes. It generally contains still enough air to enable it to float, though poorly. In the more severe cases, however, the lung or part of it (not necessarily the lower lobes, though these more frequently than the others) may be solid and of liver-like consistence, containing no air and consequently sinking in water if separated from the rest of the lung.

The Cause of the Congestion.—In only three of these cases were cultures made: in one there was a pure *Bacillus coli* infection, in one *Staphylococcus aureus*, while in the third there was no growth on blood agar, and Gram-stained sections of the lung failed to show any organism. In the last case the thymus was enlarged, and the cause of the acute congestion and oedema with resulting intrapulmonary haemorrhage may possibly be put down as anaphylactic. This case was the first which drew my attention to the condition, and, on account of its representing what appears to be a distinct type of pneumonia with certain unique clinical and pathological features, seems worthy of being reported in full. The case occurred in the practice of Dr. R. W. Johnstone, by whose permission I am able to report it. The clinical history in Dr. Johnstone's words was as follows:

"The child was the first of two healthy parents. The mother had a perfectly normal pregnancy and the infant was born exactly at full time. Labour was not difficult, and forceps were employed only to improve the flexion of the head and bring it on to the perineum. The actual delivery was spontaneous. The child, a male, was lusty at birth and weighed 8 lb. 3 oz. He made quite normal progress and put on weight satisfactorily on his mother's milk. At the end of three weeks he was circumcised. Dr. Torrance Thomson gave the anaesthetic and there were no signs of anything abnormal. At 4½ weeks he was vaccinated. The vaccination took mildly and satisfactorily, and the arm never gave any trouble. When he was exactly 5½ weeks old the night nurse found him lying dead in his cot with a little frothy blood exuding from his nose and staining the pillow. She had lifted him to change the napkin half an hour previously and had noticed nothing wrong, nor had he presented any symptoms during the preceding day or evening. During the half-hour since she had lifted him the

nurse was sitting within a few feet of his cot and he had made no sound. It was only accidentally that she discovered his condition when she rose to attend to another infant and saw that he was lying slightly more over on his face than was usual. She then drew aside the curtain of his cot to alter his position and found the condition described.

"I saw the body a quarter of an hour later. The infant was quite dead, and the body rather white; there were still traces of blood in the nostrils.

"As I could offer no explanation of the tragedy I asked Dr. John Thomson to see the body with me early next morning. He likewise could offer no explanation. The parents therefore willingly consented to a *post-mortem* examination, which was made by Dr. F. J. Browne about twelve hours after death."

The following was the *post-mortem* report:

The body was that of a well-nourished male child of average size and weight. There was one healthy and dry vaccination scab on the left arm, and the circumcision wound was quite healed. The body was rather white, and there were traces of blood about the nostrils.

The brain showed no injury or congestion, and the tentorium cerebelli was uninjured.

On opening the chest the large thymus at once attracted attention. Its increase in size was not from above downwards but *antero-posteriorly*, in which diameter it appeared to be almost twice the normal thickness. It was slightly congested, and weighed 17 grams. *Microscopically* no abnormality was found. The corpuscles of Hassall were few, reticulum scarce, and lymphoid tissue very pronounced. The thyroid was normal in size, and showed no abnormality on microscopic examination. The colloid seemed normal in amount, and there was no evidence of hyperplasia.

The lungs were well expanded and generally bright pink in colour; they floated buoyantly in water, with the heart attached. The right showed slight congestion of all its parts; it was pink in colour and was well aerated. The upper lobe of the left lung had a *rubbery feel* and was *dark red in colour*. On squeezing the cut surface frothy bloody fluid exuded. The rest of the lung was slightly congested, thus resembling the right. The upper lobe contained air and floated in water when separated from the rest of the lung. There was no subpleural haemorrhage and no pleural effusion.

Microscopically.—The upper lobe of the left lung showed intense congestion and oedema, so that the alveolar walls were much thickened. The alveoli and bronchioles contained fresh blood, in many cases completely filling them, in others leaving still a small round, air-containing space in the centre of the alveolus. In most of the alveoli and bronchioles there were a few large endothelial cells containing pigment. There were no other cells present except an occasional polymorph. Many of the alveolar walls also contained extravasated fresh blood, and here also there were numerous endothelial phagocytic cells. Sections from other parts of the lungs showed a fair amount of oedema and congestion, but far less intense; all sections examined showed well-marked *emphysema*, which was as noticeable in the less affected parts of the lung as in those parts where congestion and oedema were very marked.

The heart was normal; it showed no sub-epicardial haemorrhages, and *microscopically* showed nothing unusual.

The liver was slightly congested; cells were swollen, granular, and pale, but there was no evidence of cirrhosis.

The spleen was not enlarged, but was somewhat diffuent on section.

Microscopically.—The Malpighian bodies were very well marked, but could not be definitely said to be abnormal in extent. The pulp was very oedematous and in consequence details were difficult to make out.

All the other organs were normal and the epiphyseal lines were healthy. Levaditi stained sections of the organs failed to show spirochaetes. There was thus no evidence of syphilis, though the Wassermann test was not carried out upon either parent. Except for the thymus enlargement, which was quite definite, there was no other evidence of hyperplasia of the lymphoid tissue either in chest or abdomen, neither was there the least evidence of asphyxia.

Cultures from the left lung and from the liver on blood agar were sterile, and Gram-stained sections of the lungs failed to show any organism.

The presence of the large amount of oedema in this case was noteworthy; in no other case was it anything like so marked. It was a yellowish opaque fluid that could easily be seen in the stained section by the naked eye. The other cases showed none of this, but congestion and haemorrhage only. In none of the other cases was the thymus enlarged, nor was there evidence of hyperplasia of the lymphoid tissue elsewhere. In two, however, there were peculiar appearances in the thyroid. The child in the former case had been found dead in bed with the mother at the age of 10 days. It had been born at full time, had always seemed perfectly healthy, and had been in its usual good health the night previous to its death. Suspicion of overlaying naturally arose. Of this, however, there was no evidence, and the lung showed intense congestion and haemorrhage into the alveoli and bronchi. The thyroid contained no colloid, and the cells lining the acini were shed into the lumen. A similar appearance was found in another case, where there was also, however, fairly

well marked though patchy fibrosis of the thyroid, though there was no other reason to suspect the case as syphilitic, and the other organs showed no syphilitic change.

It would seem, therefore, that where—especially in a premature or syphilitic infant, in which the vessels are particularly apt to give way under strain—a fairly acute congestion of the lungs occurs, the capillaries in the alveolar walls may give way and suddenly flood the air passages (alveoli and bronchi) with blood, invariably causing sudden death, the only preceding physical sign being the blanching of the skin and epistaxis. The congestion may be due to an organism of normal or increased virulence, or it may occur in the absence of any organism and may then be possibly of the nature of an anaphylaxis. The condition is not limited to premature or syphilitic infants, but may also occur in full-time and previously healthy infants, though in these the congestion probably requires to be more acute and of greater degree. This "acute haemorrhagic pneumonia of infants" forms a distinct clinical and pathological entity and accounts for many of the sudden deaths occurring in infants previously apparently healthy.

Summary.

1. Pneumonia is a common cause of death amongst infants during the first week after birth, and accounted for 21 deaths out of 80, or 26 per cent.
2. It is sometimes due to ante-natal infection from premature rupture of the membranes, and the infant may be not only infected but also suffering from an advanced stage of pneumonia before its birth.
3. In the infant there is little defensive reaction as compared with the adult against the infecting agent.
4. Pneumonia in the first few days of life is an extremely insidious disease, generally presenting no characteristic symptoms which might lead to its presence being suspected and the carrying out of physical examination.
5. Even at *post mortem* examination the presence of the disease may be missed if microscopic examination of the lungs is not carried out.
6. "Acute haemorrhagic pneumonia of infants" forms a distinct clinical and pathological entity which gives rise to sudden death in children who may be apparently previously healthy, either full-time or premature. It follows acute congestion of the lungs, the fragile vessels in the alveolar walls rupturing and flooding the alveoli and bronchi with blood. The sudden death is preceded by epistaxis and blanching of the skin, and the etiological factor may be organismal or possibly of the nature of an immediate anaphylactic reaction.
7. The importance of avoiding premature rupture of the membranes during labour, and of taking every precaution to safeguard the newly born infant against infection, cannot be over-emphasized.

A CASE OF DISSECTING ANEURYSM OF THE AORTA.

BY

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AND

MABEL GATES, M.D. LOND.

[With Special Plate.]

THE patient, a man aged 55, had usually enjoyed good health. He was a married man with three grown-up healthy children, and had led a very active life as a master baker; he frequently lifted heavy weights (large baskets of loaves, etc.) in his business. Once a month he went to London and back in the day to attend a meeting as a director.

He first consulted Sir Henry Davy in June, 1914, for papular eczema on his right leg and left arm; the papules in many places formed small sores, but were easily cured with ordinary treatment. In 1915 and 1916 he was seen twice with two attacks of slight influenza. In 1919 he had a rather severe bleeding from the nose, and was attended by Dr. Andrew. A few months after this he had another attack, when he was sent to Mr. Worthington, who found and speedily cured an ulcer on one of the nostrils. He was seen twice in 1920 with a return of his rash, and then it was noted that he had a rather high blood pressure (180 and 183), but he quickly got rid of the rash and was under no medical attendance from December, 1920, until November, 1921.

History of Attack.

On October 31st, 1921, he felt quite well, and went to London, leaving Exeter by the 7 a.m. train, and returning at 9.30 p.m. He went to bed, slept well, and got up and went to business the next morning as usual. There is no clear history of what work he did that morning.

About 10 a.m. on November 1st he felt discomfort in his chest and stretched his arms backwards. Almost immediately he was seized with intense pain in the region of the heart and became very ill indeed. Dr. Mabel Gates, who attends his wife and daughter, saw him almost immediately. She found him "with intense pain in the region of the heart, collapsed and sweating, with a quick irregular pulse, and apparently dying." Sir H. Davy joined Dr. Gates just before 11 a.m., and found that the patient had somewhat recovered under her treatment with amyl nitrite. His pulse was very quick, but regular. He was pale and somewhat collapsed. He complained of a sharp intense pain, which he said started from the nipple in front and passed straight to almost the same level of the spine behind. This pain prevented his keeping still for a minute. He was constantly up and down in bed, and in and out of it, trying by change of position to get ease.

As the pain continued in spite of nitroglycerin and amyl nitrite, he was given an injection of morphine gr. 1/5 at about a quarter to 12, after which the restlessness nearly ceased, although he still said he was in pain. When we saw him again together at 9 p.m. he was quiet, his pulse had recovered considerably, and was 64 to the minute, and his temperature was subnormal, 97°. He still, however, complained of pain. Up to this time we could not examine him at all thoroughly: he was too restless and too ill. But on superficial examination of the heart and lungs there was nothing very definite to be found. We ordered another injection of morphine gr. 1/4, to be repeated after six hours if necessary.

Discussing the condition together, we could not arrive at any definite conclusion. The pain in the region of the heart, the quick irregular pulse, together with the collapse and sweating, might have gone very well with angina. But the restlessness was altogether unlike it. At our next visit the following morning we found the conditions entirely changed. He had passed a fairly good night, sleeping at intervals, and taking a fair amount of milk and water as nourishment. The pain had not been so severe and no second injection had been needed. Examination showed—

1. The area of cardiac dullness was much increased, extending upwards to the second rib on the left side, downwards about 2½ or 3 in. below the nipple line and outwards to about 3 in. outside the nipple line.
2. The heart was visibly displaced in accordance with these measurements, being pushed downwards and outwards. The area of pulsation corresponded with the areas described, and the apex beat was in a line with the axilla.
3. There were no adventitious heart sounds, and there was no bruit over the aorta, either back or front. The blood pressure was 180.
4. On turning the patient on his side a small area of dullness was found at the base of the lung; there was a want of entry of air all over the back of the chest, and an impaired note of resonance over a much greater area than the dullness.

It was evident that we had not to do with an ordinary case of angina, and, after discussing all the symptoms carefully, we came to the following conclusions:

1. That the heart had been displaced downwards and outwards by the formation of an aneurysm in the neighbourhood of the arch of the aorta.
  2. That this had developed more or less slowly with intense pain, and that therefore it was probably due to a dissecting aneurysm.
  3. That the dullness at the base of the left lung showed that there was a leakage of blood into the left pleura.
- The intense pain I have described led us to diagnose a dissecting aneurysm. Once before one of us had had an opportunity of observing the sudden formation of an aortic aneurysm.

A delicate lady with a very bad family history, who was accustomed to no muscular work, decided to roll her own lawn with a rather heavy new roller. After doing so for some little time she was suddenly seized with a pain in the back. Both she and her husband, who was a doctor, thought she had just strained her muscles or had rheumatism from the unusual work. I saw her ten days after, as the pain continued. Careful examination revealed a small pulsating tumour in the abdominal aorta, just above its bifurcation into the iliac arteries. In my opinion the pulsation was distinctly expansile, and I diagnosed an aneurysm which had suddenly developed during the rolling. The pain was