THE PRESSURE IN THE RENAL, PORTAL AND GLOMERULAR CAPILLARIES OF THE FROG'S KIDNEY.

BY
LEONARD HILL, M.B., F.R.S.

(From the National Institute for Medical Research.)

(Preliminary Note.)

By making an incision through the skin and abdominal wall in the lumbar region of a urethanized frog and removing the ileum on the same side the kidney is exposed. The cautery is used to stop all haemorrhage. A ligature is tied round the membrane attached to the outer edge of the kidney. By very gentle traction the kidney is then drawn on to the transparent peritoneal membrane which covers a small brass chamber with a glass floor, similar to that used by Roy and Graham Brown in their measurement of capillary pressure in the frog's web. A glass cover is then brought down on to the upper surface of the kidney so that it can be squeezed between the membrane and glass cover when air is blown into the chamber. A water manometer indicates the pressure used for compression. The kidney is examined under the microscope, using a magnification of one hundred times. The kidney appears closely covered with a network of branches of the renal portal vein, through which blood flows slowly in copious streams. On compressing with a pressure of about 2 centimetres of water these vessels are squeezed empty, and the tubules and, in successful preparations, the glomeruli then come into view; the arterial blood is seen rapidly circulating through the glomeruli. The Bowman capsules can be seen surrounding some of the glomeruli, and the glomeruli hanging in a clear space within these. In some glomeruli the afferent and efferent vessels are clearly seen, and the blood corpuscles can be watched racing through the afferent vessel, whirling round the twisted capillaries of the glomerulus and out through the efferent vessel. On the inner edge in one preparation a row of glomeruli were seen fed by the branches of one of the renal arteries.

The pressure required to stop the renal artery and the flow in the glomeruli is of the same order as that in the arteries of the web of the frog's foot—for example, when there has been little loss of blood and the circulation is vigorous, 30 to 40 cm. of water. A pressure of 7 to 9 cm. of water in this case sufficed to modify the flow in the glomerular capillaries, making it a little slower. By increasing the compression the corpuscles in the glomeruli could be driven out, the flow reversing through the afferent vessel. In a kidney in which the blood flow had stopped a pressure of 7 cm. of water sufficed to squeeze blood from the larger vessels through the glomeruli and produce a rapid flow. This preparation of the living frog's kidney seems likely to afford a valuable additional means of studying the renal secretion under various conditions.

A CASE OF COMPLETE PNEUMOTHORAX IN AN INFANT, DUE TO PULMONARY TUBERCULOSIS.

BY
GEOFFREY BOURNE, M.D.,

PHYSICIAN TO THE EAST LONDON HOSPITAL FOR CHILDREN,
SHADWELL.

This case is published for two reasons: First, the condition is extremely uncommon. Secondly, some light is shed as to the probable mode of transmission of the tubercle bacillus to the lungs in childhood in certain cases of pulmonary tuberculosis.

The patient, a boy aged 18 months, was admitted with cyanosis, cough, and dyspnoea, to the wards of the East London Hospital on May 11th, 1920. The history was that he had been in his usual health until three months before, when he contracted measles. Since then he had been in poor health, the chief symptoms being lack of appetite, constipation, wasting, and slight cough. For the last three weeks the cough was decidedly worse; food had frequently been vomited in association with it. The

acuteness of symptoms during this latter period had been distinctly accentuated. He had become bluer during the last few days.

Condition on Examination.

On admission the temperature was 102° F., the pulse rate 142, and the respiration rate 40. The child appeared to be extremely ill. Cyanosis was very marked, being especially obvious in the lips and finger-nails.

Examination of the chest gave the following physical signs: Respiratory movement was slightly diminished on the left side, the intercostal spaces on this side appeared to be fuller. Cardiac movements were visible in the fourth space to the right of the sternum. Cyrtometric examination showed the left side of the chest to be the larger by a quarter of an inch. By percussion the heart was found to be displaced two inches to the right. The whole of the left side was hyper-resonant. Over the right side the note was less resonant than normal, the impairment was particularly intense at the right base and less so in the right axilla. The respiratory murmur was very much diminished all over the left side, both back and front. Expiration was prolonged over the right lung in front; in the right axilla the breath sounds were bronchovesicular. Numerous fine râles were present over the right lung, back and front, especially at the base. No cardiac murmurs were heard.

The liver and spleen were both easily palpable, otherwise no abnormality was discovered in the abdomen. Clubbing of the fingers was not present.

A condition of complete pneumothorax of the left side, associated with bronchopneumonia of the right lung, was diagnosed.

The temperature fell to 98°, the pulse rate rose to 180, and the respiration rate to 70, within the next twelve hours, and the child died.

Post-mortem Examination.

The child appeared to be well nourished. Cyanosis was extreme; the nails of the hands and feet were light-blue in colour. Hypostasis was well marked. The brain was congested, and the subarachnoid in the basal region had a milky appearance; no gross tubercles, however, were seen.

Thorax.—The heart was normal. No pericardial tubercles were found. The left lung was collapsed down upon its root, except at the apex, where it remained adherent to the parietal pleura. The left parietal pleura was studded with miliary tubercles; none were seen on the right side. No excess of pleural fluid was found. The left lung was a solid mass of yellow tubercles. It sank in water. At the apex, where it was adherent to the pleura, a cavity the size of a hazel nut was present. The walls of this were formed by greyish ragged granulation tissue; it was surrounded by an area of extreme caseous change, and its interior communicated by means of a small rent with the left pleural cavity. The right lung was not solid, except in a few small areas. It contained many miliary tubercles. The glands at the hilum of both lungs were slightly enlarged, hyperaemic, but definitely not caseous anywhere.

Abdomen.—No free fluid was present. The liver was seen three inches below the costal margin, and the spleen was also visible. Both were studded with miliary tubercles. Section of the liver showed the presence in its substance of miliary tubercles, some of which were bile-stained. The spleen was adherent to the tender surface of the diaphragm; it, too, had many miliary tubercles within it. Both kidneys showed subcapsular tubercles. A few subperitoneal tubercles were found on the walls of the small intestine. The testes were normal. The parietal peritoneum had escaped except where it came into contact with the hepatic and splenic surfaces.

The right knee-joint was normal.

Bibliography.

Cruchet¹ states that of the causes of pneumothorax in children tuberculosis is the least common. He quotes, in addition to his own experience, Lenz, who found tuberculosis in 40 per cent. of his fourteen cases, and Chryanowska, whose figure is 38 per cent. of twelve cases. He states that in the tuberculous cases tuberculous bronchopneumonia was the commonest cause, but that the opening

into the pleural cavity was very rarely observed. Further that the base of the lung was the commonest site of the lesion.

Bovaird² collected 18 cases, 4 of which recovered. Particulars of 12 of these cases are available; 5 were associated with bronchopneumonia, and 4 of these with measles in addition; 2 with miliary tuberculosis; 2 with diphtheria and emphysema; 2 with pulmonary abscess; 1 with trauma.

I have found in the literature no other case of complete pneumothorax due to tuberculosis in a child of 18 months.

Pathology.

The probable course run by the disease in the case under survey is well marked out by the *post-mortem* findings. The oldest tuberculous focus found was the collection of very large mesenteric glands, the most recent was represented by the general miliary tuberculosis. Another point shedding great light on the direction of spread was that the left pleural cavity—the one corresponding to the lesion of the pneumothorax—was studded with miliary tubercles, whereas the right cavity was entirely free from them.

The tuberculous disease of the left apex, although fairly recent, was probably of considerably shorter duration than that present in the mesentery, for no caseation was found in the hilum glands of either lung, and the extent of the apical lesion was less than a square inch.

The following reconstruction of the spread of the disease is here tendered:

The exciting cause of active tuberculosis was the attack of measles, and the primary focus of disease was the mesenteric glands. From this origin tubercle bacilli reached the blood stream by way of the abdominal lymphatics, and were carried through the right side of the heart, eventually reaching among other places the apex of the left lung. Those that reached this position flourished and ultimately produced a rapidly caseating area whose territories spread till the left pleural surface was reached. Adhesions were formed here below the two layers of the pleura. During some violent effort of coughing the portion of diseased tissue between a bronchus and the left pleural cavity gave way. The history places this occurrence at three weeks before death. The pleura was severely infected with tuberculous material and became studded with miliary tubercles. As the direct result of this sudden massive infection of a serous surface generalized miliary tuberculosis arose and provided the final scene.

The absence of disease of the pulmonary hilum glands is interesting in view of Canti's observations³ upon the pulmonary lesions of tuberculosis in children, which suggest that the lung is frequently affected before the glands at its hilum. It seems clear, however, that in this case infection was alimentary and haematogenous, not inspiratory. Unfortunately cultures were not made to prove the class, whether human or bovine, to which the organism present belonged.

REFERENCES.

- ¹Cruchet, La pneumothorax tuberculeuse chez l'enfant. *Cong. intern. de la tuberc.*, Paris, 1906, ii, 238. ²Bovaird, Pneumothorax in Children. *Arch. Ped.*, New York, 1903. ³Canti, *Quart. Journ. Med.*, vol. xiii, Oct., 1919.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

MELAENA NEONATORUM TREATED BY BLOOD TRANSFUSION.

ON February 12th, 1921, I delivered a primipara of an apparently healthy female child weighing 6 lb. During the night the child vomited dark fluid and passed what I supposed to be meconium three times. On the following day the child was very restless and cried considerably. The bowels acted once, the motion still being like meconium. At 1.30 a.m., on February 14th, the baby vomited dark blood, and shortly afterwards passed a large quantity of black, foul-smelling blood from the bowel. Fluids given by the mouth were vomited immediately, and the child appeared to be very ill. At intervals of about two hours alarming quantities of blood were passed from the bowel, and at the same time the child had all the symptoms of a severe haemorrhage; the skin and lips were almost white,

the heart beats were too rapid and weak to count, the respirations were sighing, and the eyes half-closed. From the symptoms I concluded that the child would die shortly unless blood transfusion could be performed.

As I myself belong to Group IV (universal donors), I drew from a vein in my left arm 20 c.cm. of blood with an exploring syringe into which I had already drawn 5 grains of sodium citrate in sterile solution. I then exposed a vein in the baby's arm by an incision, and exchanging the large exploring needle of the syringe for a fine hypodermic needle injected the blood into the vein of the baby. It was a matter of extreme difficulty to strike the lumen of so small a vein in a collapsed condition, but I was fortunate in injecting the blood without wasting any. The improvement in the child was immediate and striking. The colour returned to the lips, the respirations slowed down, and the pulse at the groin became palpable. During the next twenty-four hours the bowels acted four times, dark blood being still passed, but in diminishing quantities. On the following day the baby took the breast well and the stools gradually became normal. Since then progress has been uninterrupted and the child is now healthy and with a good colour.

R. DOUGLAS LAURIE, O.B.E., M.B.,
Honorary Assistant Surgeon, Derbyshire
Royal Infirmary.

DIPHTHERIA AND FOLLICULAR TONSILLITIS.

DURING the last ten years in a general practice I have been accustomed to divide all cases of sore throat with exudates into three clinical groups: (1) Those which are plainly diphtheria, with an exudate present on other parts as well as on the tonsils. (2) Those which are plainly follicular tonsillitis. (3) Cases which are doubtful but are probably diphtheria; swabs from these cases yielded practically in all instances the Klebs-Loeffler bacillus.

The number of cases in the last group was small in proportion to the other two, about 5 per cent. of the total. I have observed normal convalescence in the follicular group; that there were only four "dissents" in ten years from the fever hospitals regarding the first group was reassuring. During the later period of the last epidemic, however, I was puzzled by two cases of definite follicular tonsillitis which did not lose the exudate after four or five days, and in each case a swab taken showed, on examination by a pathologist, the Klebs-Loeffler bacillus present. On account of this, swabs were taken from the next ten cases of exudative tonsillitis. In six cases, clinically follicular, the exudate was yellow, soft, pulsatious, in separate patches which did not coalesce and did not spread over the tonsillar boundary; there were no signs of haemorrhage on removing the exudate. In each case there was marked general malaise, a high temperature (102° or more), and pain on swallowing. In four of these six cases the Klebs-Loeffler bacillus was present, which to me was a startling result.

The other four cases were suspiciously like diphtheria; in them the patches of exudate coalesced, and the spread suggested that the exudate would pass beyond the tonsillary boundary. Of these cases, however, three were negative as regards the Klebs-Loeffler bacillus, and one, the worst, was a case of Vincent's angina.

These results raise in my mind one or two questions. Do epidemics of diphtheria differ as to their clinical manifestations? If not, why were the clinical tests I have been accustomed to apply in former years useless in these cases? Is it not extremely likely that the epidemics are caused by cases so plainly follicular, as far as clinical examination can go, that no swab is ever taken? In the face of the facts given above, is it safe for the general practitioner ever to rely on the clinical signs, however definite, in cases of sore throats with exudates, to give an answer to the question of whether a case is diphtheria or not? I for one, after this experience, will never feel that I can rely on the differential diagnosis by clinical signs.

London, S.W.

W. H. PALMER, M.B., B.S. Lond.

BRADYCARDIA.

THE following case may be of interest on account of its long duration. We first saw W. W., the patient, fifteen years ago, when he was 70 years of age and was employed as a blacksmith. He complained of attacks of unconsciousness suddenly coming on while at work. These