A CASE OF PURULENT PERICARDITIS IN AN INFANT: OPERATION: DEATH.

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PURULENT pericarditis is seldom diagnosed in infancy, and the age of the present patient is, perhaps, the youngest at which an operation has been performed for its attempted relief. There are some points, too, in the method of operation that add to the interest of the case and to its value for being recorded. The notes have been compiled from the careful and elaborate ones taken by Dr. A. E. Hunter, formerly house-physician to the East London Hospital for Children, and for the sake of brevity only those of a more important and essential character have been dealt with.

R. W., a male infant, aged 1 year and eight months, was admitted into the East London Hospital for Children on January 22nd, 1904, under the care of Dr. Coutts.

History.

The history of the illness showed that the child had an attack of measles eight weeks before. Ever since the measles he has had a persistent cough which has got worse, and last night the mother thought the child was dying. He is sick after coughing but does not whoop. He sweats a great deal at nights and complains of pain in the abdomen. The last ten days he has been "off his food," and for the last two days has taken nothing. The father and mother and one brother are quite well. One child, however, died from "consumption," and the remaining member of the family, a sister of the patient, is in the same hospital, suffering from abscesses in the neck.

Condition on Admission.

The patient was a wasted infant with dry inelastic skin and distinctly rachitic. The fontanelle and cranial sutures were widely patent, the epiphyses of the long bones enlarged, and there was some beading of the ribs. The child did not seem very distressed, the alae nasi were not working, and there was an absence of cyanosis. The temperature, which on admission was normal, rose on the following day to slightly over 100° F., but for the next two days remained subnormal. There was absolute dullness and increased resistance at the right posterior thoracic base, extending from the seventh rib above and round to the nipple line in front. Over the dull area the breath sounds are feeble or abolished. The heart's apex beat was in the fourth left intercostal space, just internal to the nipple, and the sounds were clear and distinct. The left lung was resonant on percussion and healthy breathing was audible all over. Both liver and spleen seemed of natural size.

Treatment.

It being considered that the signs at the right base were due to fluid, an exploratory syringe was inserted into the dull area, but no fluid was withdrawn.

A week later the signs at the right base had much diminished. Dr. Graham, the Medical Officer to the Electrical Department, examined the child under the x rays, and reported that the diaphragm was immobile on the right side, and the shadow over the dull area at the base of the right lung very slightly defined. The dull area was much darker than the rest, but transmitted light. There were no indications of either cardiac enlargement or of fluid in the pericardium.

The child did not improve, and the temperature became very irregular, running up to 103° or 104° in the evenings, and dropping down to near the normal in the mornings. On the ninth day after admission a patch of dullness was found at the left posterior base, extending up to the angle of the scapula, and there was diminished or absent breathing over the dull area. An exploratory syringe was inserted, and about a drachm of clear greenish fluid, not pus, was drawn off. The fluid was examined by Dr. W. P. S. Branson, then Pathologist to the Hospital, who reported that it was opalescent in character, containing a small quantity of fibrin. The centrifugalate consisted of a small deposit of leucocytes. No organisms were seen or tubercle found. The fluid was not considered to be pus. Unfortunately no cultivations were made. A week later, as the signs in the left chest remained the same, it was aspirated, but only ½ oz. of the same fluid as before was drawn off.

On February 15th, about three weeks after admission, the dullness over the left base had diminished, and there was more air entering the lung. The infant, however, had not improved, and his breathing was unduly rapid. The precordial dullness was noticed for the first time to be increased, extending upwards to lower border of second rib, and from just outside the left nipple to the right border of the sternum. The heart sounds were very indistinct, but there was no pericardial friction.

A week later the precordial dullness had increased, and now reached to the first rib above, and from 1 in. outside the left nipple line to ½ in. beyond the right sternal border. The dull area was very resistant, and

suggestive of fluid. The heart sounds could not be heard below the third left costal space, and there very feebly. The pulse was very rapid, and could scarcely be felt, and there were orthopnoea, cyanosis, and other signs of cardiac distress. As a blood examination showed a leucocyte count of 32,000, it was decided that the fluid in the pericardium was probably pus, and that operation offered the only slight chance of saving the child. A consultation was accordingly held with Mr. Rowlands, who agreed to operate, provided the consent of the parents was obtained.

Operation.

Mr. N. R. Phillips, who was temporarily acting for the Resident Medical Officer, gave chloroform. When the compress was removed from the chest on the operating table purplish subcutaneous haemorrhages were noticed; these had been induced by the gentle preliminary scrubbing, and clearly indicated the serious and septic condition of the child.

An exploring needle was introduced into the left costo-xiphoid angle, and passed upwards, backwards, and inwards, but no pus was discovered, although the needle had been thrust far enough to reach the pericardium. The needle was then tried at the sternal edge in the fifth left intercostal space, but with a like negative result. Dr. Coutts, who was present, still adhered to his diagnosis, and the following operation was therefore carried out at once.

A vertical incision was made over the sternal end of the left seventh costal cartilage, half an inch of which was carefully removed by paring, so as to avoid the internal mammary vessels. The edge of the left pleura was now seen in the wound, therefore a portion of the cartilaginous gladiolus was removed to give more room. This was easily and rapidly performed with the knife. The thickened and vascular pericardium was thus displayed and then opened freely by a crucial incision, the vertical limb of which reached the diaphragm below. The edges of the pericardial wound were secured to the skin by three sutures. Between 1 oz. and 2 oz. of pus escaped, and some pieces of lymph were removed by the finger and forceps. A finger was passed upwards to the front of the base of the heart, and also into the oblique sinus of the pericardium behind the heart. Two soft rubber tubes were then introduced into these two localities, and fixed in position by safety pins.

The child stood the operation well; but, in striking contrast to our last case, the fluid was not at a great pressure, and consequently there was an absence of the immediate and marked relief noticed in that case. After the operation 2 minims of liquor strychninae were injected and the child replaced in bed.

The remaining history can be briefly summed up. In spite of occasional temporary improvement after injections of strychnine and saline solution with brandy the infant gradually sank, and died on the second day after the operation.

Post Mortem.

Extracts from the notes of the post-mortem examination made by Dr. W. P. S. Branson: The body is very wasted. The wound made to drain the pericardium lies immediately to the left of the ensiform cartilage. On either side of the oesophageal surface of the larynx is a grey membranous deposit. Cultures from this surface yielded the diphtheria bacillus. The tracheal bifurcation gland is large but not caseous. The sternum is densely adherent to the oedematous and fibro-gelatinous areolar tissue of the anterior mediastinum. The pericardium wound drains the sac thoroughly. The pericardium is very thick, the thickening being in a great part due to an organized addition to the inner surface. Both parietal and visceral layers are, on their opposed faces, covered with thick gelatinous pus, such as occurs in pneumococcal empyema, and the parietal layers are adherent to the lung on either side. The appearances point to a long existence of the pericarditis. The pus from the pericardium contains many capsulated diplococci which are morphologically typical pneumococci. Both lungs are universally adherent by adhesions of long standing. All the lobes are oedematous, and the lower ones, in addition, are collapsed. There is no sign of inflammatory consolidation. The peritoneal cavity contains a small quantity of turbid fluid, with flakes of fibrinous pus here and there, and there are a few recent adhesions between the viscera. The peritoneal fluid on examination contained organisms apparently belonging to the bacillus coli communis group. There was nothing noteworthy in the rest of the organs examined, except that the venous system was greatly congested, and the blood more fluid than usual.

REMARKS BY DR. COUTTS.

In the present case the diagnosis of purulent pericarditis was made with much less confidence than in any of the other instances I have ventured upon it. The reason of this was owing to the amount of fluid in the pericardium being small, between 1 and 2 oz., and there being an absence of any accessory aid to diagnosis in the presence of an accompanying empyema, or the history of a past pneumonia. The diagnosis was based upon the extension of the pericardial dullness and diminution of the cardiac sounds, along with the signs of cardiac distress, which were marked and quite disproportionate to the amount of fluid found in the pericardial sac. As usual in such cases, there was never any audible pericardial rub. The history of a recent attack of measles, the successive involvement of the three serous surfaces, the character of the fluid drawn from the left pleura, and the type of the pyrexia,

all led me to regard the case as one probably of tuberculosis. It was the high leucocyte count, furnished me at the last moment, that led me to suspect suppuration and to counsel operation. With so many factors suggesting tuberculosis, I even now think it just possible that the pneumococcal inflammation was secondary to a previous tuberculous affection of the pericardium, although I have to admit that there was nothing found *post mortem* to justify such a supposition.

The double failure to find pus with the exploratory needle, even with the child under full anaesthesia, is further proof of the unreliability of the procedure in numerous cases. The failure with the needle, too, may well tend to upset a diagnosis that was otherwise well established, and in the present instance made me momentarily hesitate about sanctioning further operative measures. The diphtheritic condition of the larynx, discovered *post mortem*, of course precluded any chance of success from the operation; but it may be said that the condition had not revealed itself by any signs, and could not have been diagnosed during life.

This is the fourth case of purulent pericarditis I have had operated on, all without success. Three of the patients died within a few days after operation. In a fourth case, however, recently reported by Mr. Rowlands and myself,² the child lived for four months after operation, and here the death was accelerated by the contraction of measles and bronchopneumonia at a time when recovery seemed hopeful. All my cases, too, it may be claimed, were much younger than the generality of those recorded. The results are certainly not encouraging so far as to operation in very young children. Still, as death is practically certain without it, I would not be deterred by past experience from giving such children the slight chance that operation affords in any future cases I may come across.

REMARKS BY MR. ROWLANDS.

The peculiar narrowness of the costo-xiphoid angles in this emaciated child, together with the absence of any fullness there, made me decide against adopting the epigastric operation in this case. I therefore removed the sternal end of the seventh left costal cartilage, but this gave very little room and exposed the edge of the left pleura. It was easy to remove a portion of the adjoining cartilaginous sternum; this gave ample room and a good view of the pericardium, and a safe and quick access to it. The sac was opened at the lowest extremity of its anterior surface, and thus at a good place for drainage.

Moreover, it was found possible to pass a finger into the various recesses of the cavity, and to introduce a tube behind the heart into the oblique sinus with comparative ease. The above points and especially the patency and directness of the aperture established strongly recommend the sternal route for young children. In our former case the fifth left costal cartilage and a piece of the sternum at that level were removed; but I am confident that the lower incision is a better one. Instructions were given to turn the child over on to the prone position several times a day for drainage, but unfortunately the child succumbed to his several diseases on the second day following the operation. Irrigation was not adopted at the operation, partly because Dr. Coutts reminded me of Mr. Parker's case, in which Mr. Parker believed irrigation to have proved fatal owing to pressure on the auricles due to failure to provide a good exit for the fluid; but the chief reason against irrigation was to avoid delay in the critical condition of the child.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, January 2nd, 1904. ² *Ibid.*

WATER SUPPLY AND SEWERAGE FOR MANILA.—When Manila passed into the control of the United States Government there were no water-pipe connexions in the houses. The residents were compelled to carry water from public fountains at the street corners. There was no connexion with sewers. The refuse was carried to inlets, which carried the sewage away only when flushed by rain. During the last two years the United States Government has ditched the sewers so as to flush them from the river, and it now proposes to spend £500,000 on the construction of a modern system of sewerage. The sum of £300,000 has been appropriated for a new water supply. The water, according to the new plans, will be taken from the Mariquina river, about fourteen miles above Manila.

AN "Osler" Club has been formed in New York, each member of which pledges himself to retire from business when the majority of his fellows declare that he should be "Oslerized."

THE HEPATIC FACTOR IN BILIOUSNESS.

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THE pathology of functional diseases—that is, diseases which have no morbid anatomy—is admittedly unsatisfactory. The reason seems clear. Modern medicine is in the main inductive; it relies largely on the study of effects—usually ultimate structural effects; and where such are absent or imperceptible, further progress is liable to be checked. There is only one obvious way out of the impasse: we should add to our accustomed means of investigation a review of functional diseases from the deductive standpoint; in other words, we should apply the known principles of physiological function to the elucidation of the meaning of pathological function. So viewed, many seemingly mysterious functional disorders appear simple. This is true at least of those commonest of functional disorders, bilious attacks and biliousness.

Clinically these affections have been ascribed to prolonged external heat, to deficiency of physical exercise, to excess of sugar and rich carbohydrate food stuffs, and to excess of meat and nitrogenous food generally. Now it will not be difficult to show that all these etiological factors tend to cause at least one effect in common, namely, accumulation of glycogen in the liver.

It has been proved experimentally that accumulation of glycogen in the mammalian liver is favoured by external heat and physical inactivity (Kulz¹). All physiologists are agreed that sugar and other carbohydrates are the chief food stuffs from which liver glycogen is derived. And it can be argued that, like the performance of function generally, the absorption of all food stuffs, and therefore of glycogen-forming material, is dependent upon the supply of proteid. Conformably Foster says that "the greatest accumulation of glycogen is effected, not by a pure carbohydrate diet, but by a mixed diet rich in carbohydrates."²

It is not difficult to form a definite conception of the mechanism whereby accumulation of glycogen to an ultra-physiological degree can give rise to all the clinical phenomena of a bilious attack. The liver cell which is rich in glycogen is large compared with the liver cell which, through previous starvation, contains no glycogen. This has been demonstrated by experiment. Enlargement of the liver cells involves enlargement of the liver lobules, and this of the whole organ, which is "as it were swollen" (Foster³). Now we have only to glance at the well-known representations of the histology of the liver in Quain's or Gray's *Anatomy* to see that enlargement of the liver cells will cause pressure on the intralobular capillaries, and that enlargement of the liver lobules will cause pressure on the interlobular veins. It seems physically impossible that these blood vessels can escape material pressure; and it is certainly impossible that such intrahepatic pressure can be exerted so as not to cause material retardation of the blood flow through the portal vein—a vein in which the blood pressure is extremely low (10 mm. Hg).

We may then assume that glycogenic distension of the liver causes mechanical congestion of the portal venous system behind the liver. Now "there are no valves in the portal vein or in its larger tributaries. . . . Valves are, however, present in the veins of the stomach and of the walls of the intestine; but, according to Hochstetter and Bryant, they early become insufficient, and in the adult to a great extent disappear."⁴ It follows that the mechanical congestion of the main branches of the portal vein immediately behind the liver will directly involve mechanical congestion, or tendency to stasis, in the whole of the alimentary mucosa from the stomach downwards to the junction of the skin and mucous membrane at the margin of the anus.

Such a vascular condition affecting the gastric mucosa will be opposed to that which obtains when food is introduced into the stomach of the hungry animal. Then not only do the arteries of the organ dilate and largely increase the supply of oxygenated blood, but, the passage through the liver being unobstructed by glycogen, the circulation is rapid, so rapid that the blood in the gastric veins continues to preserve its arterial hue (Foster).⁵ Manifestly for the rapid manufacture and discharge of the digestive juices, and for active digestion, a free escape for the blood is as essential as a plentiful supply.