also complained of severe abdominal pain and as she was seven months advanced in pregnancy she feared labour had set in. I ordered her a mixture containing two drachms of Calvert's liquid carbolic acid in eight ounces of water, one tablespoonful to be taken every two hours. This is equal to six and one-third grains of pure phenol for a dose. She commenced to take this medicine at mid-day. I learned on the next morning that during the night she had poured out a small wineglassful of the mixture and had drunk that instead of a tablespoonful. Careful measurement of the glass which she had used showed that it held just two ounces. The patient declared that it was filled but I allowed only three-quarters as having been taken and even this would amount to 19 grains in the dose. The patient averred that the only apparent deleterious effect was feeling "a little warm" in the abdomen and that every other symptom had abated. The cephalalgia was reduced to a minimum, the myalgia ended in a little soreness of the limbs, the abdominal pain had ceased, and the vomiting had abated, and she was sitting up in bed laughing at her mistake. I had only to make one more visit when convalescence was established. As I had previously considered seven grains of phenol a maximum dose this experience was a revelation to me. The next case occurred a few days afterwards. I administered 12½ grains every two hours with the same gratifying results and I have done so in each case which I have had since. I can now assure my patients most confidently that after the first two doses relief will ensue. I desire, therefore, to recommend to my fellow practitioners this method of treatment, especially in the larger doses; feeling assured that they will find the same results to follow its adoption.

As I find that much misapprehension exists concerning, not only the dose of carbolic acid, but its toxic effects, I have endeavoured to ascertain what dose of carbolic acid has proved fatal but I cannot find any very definite records, as large and varying doses of undiluted crude carbolic acid have been taken. I believe there is a record of a teaspoonful having proved fatal, but as to the age of the patient or the attendant circumstances of the case I know nothing. In the general records of fatal cases I cannot find anything very definite concerning the therapeutical effects beyond those which accompany the administration of acute irritant poisons generally—viz., pain and nervous prostration—though in some few instances coma has, I believe, supervened. As to the effect, however, of the large doses of pure phenol to which I have referred there is neither sickness nor purging and not even nausea. In the special case referred to it will be seen that as much as 108 grains were taken in 18 hours, and I have under observation at present a case, not of influenza, however, in which I am administering 13½ grains of pure phenol every two hours during the waking hours of the patient and certainly no deleterious effects on the alimentary canal, the kidneys, the heart, or the nervous centres have ensued and this administration has now been going on for three weeks, so that there need be no apprehension of any unpleasant effects being produced by these large doses. I think if these facts were generally known carbolic acid would be much more extensively used, especially in cases of microbic origin, than hitherto has been the case. Moreover, when mixed with syrup of orange peel and water—say four drachms of syrup of orange peel and one and a half ounces of water for a 12-grain dose—it is by no means unpalatable and I have not found patients, whether old or young, object to it.

Liverpool.

NOTES OF A CASE OF ANEURYSM OF THE ASCENDING ARCH OF THE AORTA RUPTURING INTO THE PERICARDIUM; NECROPSY.

BY W. GRAY, M.D. EDIN.

ABOUT 5.30 P.M. on March 14th, 1899, I received an urgent message to "attend a railway shunter who had been injured" and immediately responded to the summons. The facts of the case are briefly as follows. The shunter, who was about 33 years of age and had since entering the service of the railway company nine years ago practically never been off duty through illness, was engaged with his mate in the performance of his usual duties. His mate left him standing on a wooden box (covering the cranks and apparatus for

moving railway points) about six or eight inches above the level of the railway sleepers and it was afterwards ascertained that he had moved the lever to shift the points. His mate on returning found him lying on his left side in a trench (which had been dug for a new siding) close by the side of the rails. The height from the bottom of the trench to the top of the wooden box was two and a half feet. On examination I found a small abrasion on the left cheek and a few scratches in the front of the left ear. He was unconscious, breathing very lightly, and no heart's action could be recognised. In spite of attempts at resuscitation he died about 5.50 P.M. on the same day.

In my evidence at the inquest I stated that with the exception of the abrasion mentioned I could not say what injuries the deceased had received or what was the cause of death, adding that from the examination which had been made I saw no evidence, apart from the history of the fall, of anything which would cause death. An adjournment took place for a post-mortem examination which was made in conjunction with Dr. F. H. Morison who acted on behalf of the relatives.

At the necropsy the liver, spleen, and kidneys were found to be enlarged. There were old pleuritic adhesions over the whole surface of the right lung, also a few old pleuritic adhesions on the left side. The pericardial sac was thickened and contained large currant-jelly-like clots and some blood-stained fluid. The heart valves and muscular substance appeared to be normal. The ascending arch of the aorta was found to be the seat of a large sacculated aneurysm which had ruptured into the pericardial sac. The brain and other organs were normal. The jury returned a verdict that deceased died while in the execution of his usual duties from internal hæmorrhage as stated in the medical evidence.

Remarks.—The case is an excellent example of the great value (one might almost say urgent necessity) of a post-mortem examination where there is reasonable doubt as to the cause of death. As the Workmen's Compensation Act applied to this case, and as the relatives threatened an action against the railway company, by practically no other course could justice have been rendered to employers and relatives.

West Hartlepool.

NOTE ON A CASE OF ASPHYXIA FROM PRESSURE OF AN ABSCESS UNDER THE CHIN WHILST UNDER THE INFLUENCE OF NITROUS OXIDE.

BY HOWARD H. C. DENT, M.B. DURH., M.R.C.S. ENG., L.R.C.P. LOND.,

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A BOY, aged 12 years, was brought to the Queen's Hospital, Birmingham, on March 18th, 1899, with a large swelling containing pus between the chin and the hyoid bone. The lad was very ill and was evidently suffering from septic poisoning. The lower jaw was almost immovable, the mouth being slightly open. He had been ill for 10 days but had not complained of difficulty in breathing. Gas was administered in the recumbent position and the patient appeared to go under its influence more quickly than is usual and his breathing was not nearly so full as is generally the case. The face-piece was removed and an incision was made in the middle line over the swelling. The incision had only gone through the skin when the respiration became embarrassed and air ceased to enter the chest, although the movements of the thorax continued. At the same time there was a spasm of the jaw and slight convulsive movements of the limbs occurred. The tongue was with difficulty pulled forwards owing partly to the spasm and partly to the enormous amount of inflammatory thickening about its root. Artificial respiration was at once commenced, but no air entered the chest although the tongue was drawn well out of the mouth. Tracheotomy was performed and respiration was kept up for one hour, air entering freely through the wound, and ether was injected under the skin and directly into the heart but there were no signs of recovery. On opening the trachea a small quantity of pus was forced through the wound, and it was thought that the abscess had burst into the windpipe and caused suffocative spasm.

At the necropsy an abscess of the size of a small orange was found excavating the body of the tongue and sitting on

the top of the larynx. There was some pus in the trachea but no communication with the abscess could be found. The other organs were healthy. The usual signs of asphyxia were present. Death appears to have been due to pressure of the abscess on the glottis, acting either directly or by causing spasm of the sphincter laryngis. This pressure would be increased by (1) the recumbent position; (2) the extension of the head necessary to open the abscess; and (3) the pressure of the knife.

Birmingham.

A Mirror

OF

HOSPITAL PRACTICE, BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv. Proœmium.

PADDINGTON GREEN CHILDREN'S HOSPITAL.

A CASE OF TUBERCULOUS PERICARDITIS; NECROPSY.

(Under the care of Dr. G. A. SUTHERLAND.)

TUBERCULOUS disease of the pericardium is a by no means infrequent accompaniment of extensive tuberculosis elsewhere and Baginsky has recently shown¹ that in 9 per cent. (six cases out of 66) of a series of cases of pericarditis in children the disease was tuberculous. It is, however, in only a very small proportion of these cases of tuberculous pericarditis that the pericarditis gives rise to any important symptoms and in most instances the tuberculous disease is only discovered at the necropsy. The great distension of the pericardial cavity which—sometimes at least—occurs in these cases is well shown in the clinical history recorded below. A very similar instance in which the amount of fluid present was even greater has been described by Mussex of Philadelphia,² for the pericardial cavity contained 64 ounces of blood-stained serum.

A girl, eight years of age, was admitted into the Paddington Green Children's Hospital on Dec. 4th, 1896. No very precise account of her illness could be obtained as she had been living with friends in the country for five months and had returned home on the previous day. For six weeks she had been noticed to be "getting ill" and had complained of pains in her chest and tiredness. There had been shortness of breath and coughing and on the previous day she had fainted. There was a definite history of pulmonary phthisis in the family, her mother's father having died from that affection. The patient had always been a delicate child, subject to sore-throat, and had suffered from scarlet fever, measles, and whooping-cough.

The patient was a tall, well-built, fair child with a somewhat anæmic complexion and looked decidedly ill. The temperature was 100° F. and rose the same night to 104°. There was no eruption or flushing of the skin, but the superficial veins of the thorax and abdomen were distended. The breathing was rapid (46 to the minute) and somewhat laboured. The left side of the chest moved less than the right and after it in time. Percussion of the left side of the chest showed impaired resonance everywhere, there being dulness at the left apex anteriorly and at the back, while from the second rib anteriorly to the costal margin and from one inch to the right of the sternum to the posterior axillary line the note was absolutely dull. Over this latter area the breath sounds were scarcely audible and vocal fremitus and vocal resonance were absent. The breathing at the left apex was bronchial and over the back posteriorly it was very harsh vesicular, with some crepitations. The right lung was apparently normal. In the præcordial region there was distinct bulging from the third to the fifth intercostal spaces and also slight but definite œdema. The apex beat could not be localised and epigastric pulsation

was present. Firm pressure caused pain but no thrill or friction could be determined. As already stated, there was a large area of absolute dulness extending from the second rib to the costal margin and from the posterior axillary line to an inch beyond the right border of the sternum. The cardiac sounds were very weak and distant both at the base and the apex and were apparently unaccompanied by any murmur. The cardiac action was regular and rapid, being from 150 to 170 beats per minute. The pulse was running, weak, irregular, and of small volume. Examination of the abdomen showed nothing abnormal. The urine contained no albumin and there was no œdema of the extremities. On the same evening an exploring needle was introduced into the left axilla and some turbid fluid was drawn off. As a result of this fluid continued to ooze from the site of puncture until a dressing of considerable size was soaked with it and a bulging swelling formed in the axilla from the accumulation of fluid in the subcutaneous tissues. On again auscultating the heart distinct pericardial friction was heard over and to the right of the lower end of the sternum. The conclusion arrived at was that the patient was suffering from non-purulent pericardial effusion which had been at least temporarily relieved by the escape of the fluid. During the following week there was improvement. The temperature showed a diurnal range of from normal or 99° in the morning to 101° or 102° in the evening. The œdema of the præcordial region and the distension of the thoracic veins rapidly passed off, while more gradually the cardiac sounds became louder and more superficial and the area of pericardial friction became larger. The large area of dulness on the left side of the chest still persisting an exploring needle was introduced but with a negative result.

During the following month there was slow improvement in the patient's condition. Coughing was at times troublesome and sleep was rather broken, but she complained of no special pain or discomfort. The diurnal variations in the temperature continued, but the maximum at night was gradually becoming lower. The liver and spleen were enlarging. The condition of the left side of the chest showed little change. There was loud bronchial breathing at the left apex in front which became very faint and distant below the third rib. On the same side over the back the breath sounds were bronchial at the apex and along the spine, but were inaudible elsewhere, being replaced by coarse creaking sounds and râles. The enlarged area of præcordial dulness persisted with superficial friction and distant heart sounds. There were no evidences of valvular disease, no clubbing of the fingers, and no œdema of the extremities. A month later (Feb. 4th) it was noted that the face looked swollen and was of a waxy pallor with a pink flush on the malar regions. The temporal and the superficial abdominal veins were swollen and there was slight œdema of the feet and legs. The liver extended to an inch below the level of the umbilicus and the spleen to two inches below the costal margin. The cough was frequent and harsh. The physical signs in the left chest were practically unaltered and it was impossible to map out the cardiac area by auscultation or percussion. There was very slight movement of the left side of the chest during respiration, but distinct bulging was present in the præcordial region. The patient was transferred to the convalescent home on Feb. 27th. On March 9th it was noted that the patient had been very well since admission with the exception of persistent coughing at night. There was found in the upper part of the left axilla an elongated swelling which consisted of a number of small, hard nodules of about the size of split peas. They were not painful or tender, the skin was freely moveable over them, and they appeared to be attached to the deeper tissues. The temperature had been normal for some time but now began to show a slight elevation in the evening. On March 24th the patient complained of headache and there followed in rapid succession the ordinary signs of tuberculous meningitis. She was re-admitted to the hospital and died from that affection on April 6th. The total duration of the pericardial trouble cannot be definitely stated, but it was four and a half months from the time when the friends noticed any sign of disease to the day of her death.

Necropsy.—A post-mortem examination was made by Dr. Leonard Guthrie on the following day. On opening the thorax the pericardium was found to be firmly adherent to the inner surface of the sternum and also to the costal wall in the left axillary region. The pericardial sac occupied almost the whole of the left pleural cavity and extended two inches beyond the mid-line. On opening the pericardium a

¹ Berliner Klinische Wochenschrift, Nov. 28th, 1898, p. 1053.

² Transactions of the College of Medicine, Philadelphia, November, 1888.