In consequence of the softness of the bones, fractures occur, and even in the scapulae. Cranio-tabes was present in two cases. The intensity of the disease was greatest in the forearm and leg-bones, then in the humeri, and the femora suffered least. There was no diarrhoea. The spleen was enlarged in three cases. Anæmia and emaciation were marked in all. No pyrexia was observed in uncomplicated cases. The urine did not show any increase of earthy phosphates.

The course of the disease was chronic, and lasted many months. Death occurred in two cases in consequence of diffuse bronchitis in one, and bronchopneumonia in the other. With regard to etiology, all the cases occurred during the first two years, and the disease began in the first year. They were all females. The food was, in all probability, poor in phosphoric acid and lime. No hereditary influence could be traced, and no evidence of hereditary syphilis. The disease is to be diagnosed, Dr. Rehn says, first, from congenital syphilis, secondly, from rickets; the softening of bone that sometimes occurs in syphilis being associated with other indications of the presence of the disease, is easily recognised. He finds the following differences from rickets:

1. The high degree of softening, and the slight epiphyseal swelling;
2. The thinness of the whole skeleton; in rickets, thickening of the bones;
3. The straightness of the lower extremities, which in rickets are usually curved;
4. Pain on movement much greater than in rickets.

It seems to me very difficult to see anything distinctive in the two last signs; and the diagnosis, if one is to be made, between osteomalacia and rickets surely depends upon what views we hold with regard to the importance of the relations between what Sir William Jenner calls "the increased preparation for ossification" in the epiphyseal cartilages and softening of bone-tissue. The bones that I have brought before you certainly seem to be more closely allied to the disease known as osteomalacia than to rickets. Dr. Rehn's case might be looked on as a sort of link between the two diseases; but without presuming to criticise the opinion of such a high authority as von Recklinghausen, perhaps I may venture to say that, if we are to hold on to anything as a sign of rickets, it is to the proliferation of cartilage-cells, to the hyperplasia that occurs in front of the irregular advancing line of ossification; and it must not be forgotten, too, to what a great degree erosion and softening of bone may take place in unmistakable rickets; this is well seen in sections across the middle of the shaft of this rickety radius kindly lent by Dr. Ashby.

There are three leading differences between the tibia in Dr. Rehn's case and that in mine. The former is straight, and thinner than natural; the latter is curved, and at least as broad as natural; the former has epiphyseal zones, measuring respectively five and four millimètres, the latter scarcely one millimètre; and, lastly, the structure of the shaft is different. A bone may be flexible from at least two causes: either the framework, though abundant, is deficient in lime; or there is no want of lime, but the trabecular framework is very scanty; the former holds in Dr. Rehn's case, the latter in mine. When the trabeculae in this specimen are touched with the point of a knife, they feel quite gritty, and the microscope verifies this. They have not been deprived of lime, but are being gradually eaten away, as a whole, and replaced by a soft cellular tissue; and you see that in places the process has led to the formation of cystic-like cavities. The defective calcification in Dr. Rehn's case may be explained in two different ways: either lime has been abstracted from previously healthy bone, or the latter has been gradually absorbed, and new bone, deficient in lime, laid down in its place. The latter view is strongly held by Kassowitz, in his recent monograph on rickets; and he dogmatically asserts that, both in osteomalacia and rickets, all the limeless parts of the bone are new formations, laid down in the place of absorbed calcified bone, or formed independently at the surface of bone; and that the process is never due to decalcification. Varying degrees of calcification are easily seen in carmine preparations of rickety and osteomalacic bone, not primarily softened by reagents; the bone deficient in lime takes a brilliant red colour, and contrasts with the silver-grey of the still calcified parts. I have a section showing this very well, and I pass round a rough painting of the appearances seen. One of the principal arguments of Kassowitz is, that the new uncalcified layers which stain bright red are never found to be continuous with the calcified lamellae; as would be the case if lime were simply abstracted from formerly existing calcified lamellae; and he says that those who, like Volkmann, Ribbert, etc., believe in an abstraction of lime from fully formed bone, have

never explained how it is that the limeless parts, which are concentric with the medullary spaces, are never continuous in series with the yet calcified portions. Kassowitz would, I suppose, regard the condition of these bones as a support to his theory. They illustrate his first stage, when the whole bone, not merely the lime, is removed; but it would be out of place here to enter into a histological discussion.

LARGE DORSAL TUMOUR REMOVED TO RELIEVE DYSPNŒA AND CARDIAC DISTRESS.

Read in the Section of Surgery.

By J. WARD COUSINS, M.D. Lond., F.R.C.S.,

Surgeon to the Royal Portsmouth Hospital.

MARY ANN N., widow, aged 65, came under treatment on March 1st, 1883. She had a large dorsal tumour, which had been growing fifteen years; and, for four years, it had been rapidly increasing in size. The circumference at the base was 28 inches. She stated that

she had suffered from cough and shortness of breath ten years, and occasionally from severe bronchitis. On admission into the Royal Portsmouth Hospital, she complained of the weight of the tumour, and great distress during movement. The chest-symptoms were very severe. At night, she slept in the sitting posture. Her face was livid, puffed, and marked with prominent venous markings. The act of expectorating was attended with prolonged distress. On examination, loud bronchial sounds were heard at all parts of the chest, with some crepitation in the dorsal regions. The cardiac impulse was weak, and the second sound loudly accentuated. The pulse 96, soft, compressible, and irregular. The urine was normal, and free from albumen. An attempt was made to sling the tumour, but it was abandoned, as it caused pain and respiratory trouble.

An operation was performed on March 15th. The tumour was placed on a wooden tray, and this was supported by ropes secured to the roof of the theatre. It was an ordinary fatty growth, intersected with very thick fibrous bands, and covered with dilated veins, which were very varicose about the base. The weight of the mass was nearly thirty pounds.

For some days after the operation, the temperature was high, and the chest-symptoms very urgent, but the patient gradually made a complete recovery, and the wound soundly healed.

A few days since, she came again to the hospital to report progress. Her appearance was much improved, and she could walk

several miles, and go upstairs without discomfort. The superficial vessels were much less distinct, and the skin looked healthy. The respiratory and cardiac sounds were normal, and the pulse was full and regular.

REMARKS.—The complete relief of all the chest-symptoms is the main point of interest in the case. The condition of the patient before the operation was certainly very critical. The weight of the tumour embarrassed the respiration, and prevented due expansion of the chest. Cardiac failure was, however, the great source of danger. The heart was greatly taxed in sustaining the circulation, and the serious retardation of the venous current was evident from the distension of the veins, and the chronic obstruction of the pulmonary system. The absence of marked valvular insufficiency and renal complication rendered the result of operative interference fairly favourable. Ether was carefully administered during the operation, and repeated hypodermic injections were employed to relieve the urgent symptoms of cardiac failure.

REMARKS ON THE TREATMENT OF PLACENTA PRÆVIA.

Read in the Section of Obstetric Medicine.

By JAMES MURPHY, M.D., B.A.,

Surgeon to the Hospital for Women and Children, Sunderland.

THE number of cases in the table which has been shown is fifteen; in all the mothers recovered completely, and, with one exception, rapidly; the exception being with case No. 4, who was attacked with phlegmasia alba dolens, which rendered her recovery very tedious.

Of the children, five were born alive; of these, two died within a few hours, the remaining three doing well. Thus, one in three was born with life; but only one in five ultimately survived. This is, no doubt, a high mortality, but several of the cases were not seen till after the death of the child. In case No. 10, the foetus was dead and putrid; in cases Nos. 2, 4, and 5, the foetus was found to be dead when the patient was first examined; and in cases Nos. 3 and 14, the early stage of pregnancy sufficiently accounts for the result. Thus, while all the deaths may fairly be said to be due to the placental presentation, where there was a chance of treatment only four children were lost of nine cases, and of these three survived, three out of a possible nine.

This is in accordance with the experiences of most men, that the fatality of mothers and children in placenta prævia is not due so much to the impotence of our art, as it is to fatal hæmorrhage taking place before professional assistance has been obtained, or where that assistance has been of a helpless do-nothing character. Therefore, when we know that the placenta is presenting, in every case where the child is fairly viable (after the seventh month), the fatal hæmorrhage may ensue at any moment; and, by inducing labour, this can be prevented to a very great extent; and we have the opportunity of being present when our help is of vital importance to the patient and her offspring. The experience of those who advocate this mode of procedure, fully confirms its soundness. Dr. Gaillard Thomas reports eleven cases with one death from *post partum* hæmorrhage, and one from puerperal fever. Out of my fifteen cases, none of the mothers died. Hecker lost but three cases in forty; Hoffman, two cases in thirty; Barnes, up to 1864, had sixty-two cases with six deaths, and had a remarkable run of twenty-nine cases without a fatal issue; and Spiegelberg had four deaths in seventy-four cases, from hæmorrhage, and eight from other causes; while Lusk puts down the usual mortality, from hæmorrhage alone, at 25 per cent.; and Müller, of Stuttgart, who published, in 1877, an account of 1,574 cases collected from various sources, estimates the percentage of maternal deaths of all causes at about 40.

In the majority of cases, we have warning that the placenta is presenting, from the hæmorrhages that take place towards the end of pregnancy. These were formerly supposed to be due to the cervix casting off the placenta in the act of its being taken up and expanded during the last months of pregnancy. With a few exceptions, however, most writers have abandoned this theory, for the good and sufficient reasons, first, that the cervix does not expand so early as the floodings occur, or, indeed, until very near the end of pregnancy; and, secondly, that the placenta is never attached to the cervix. Dr. Barnes then suggested that the floodings occurred from detachment of the placenta, arising from an excess in rate of growth of the placenta over that of the cervical zone, a structure which was not designed for placental attachment, and which is not fitted to keep pace with the placenta. Hence, the placenta shoots beyond its site, and hæmorrhage results. Now, great as is my respect for Dr. Barnes, I very much doubt if hæmorrhage ever occurs from this cause. The idea that the placenta shoots beyond its site is purely hypothetical, and would apply to normally situated placenta as well; and, though I carefully looked for it, I never found atrophy, or other indication of its having occurred, in any of the cases recorded. Further, the bleeding would be more likely to occur at the circumference than at the centre, and would be placental, and not uterine, which Dr. Barnes alleges not to be the case. I have no doubt that bleeding occurs from the four causes stated by Dr. Matthews Duncan; namely, either by rupture of an utero-placental vessel, or marginal utero-placental sinus, by partial separation of the placenta from accidental causes, such as a jerk or fall, or in consequence of uterine pains from miscarriage commencing, but being arrested at an early stage. But, if we consider that contractions are frequently taking place in the uteri of pregnant women, and that these contractions have more or less of a shrinking effect on the normally placed placenta, and have a decidedly expanding effect on the placenta placed over the os, we readily perceive how liable an utero-placental vessel or sinus is to be opened, or even a portion of the placenta itself detached. Whatever be the cause, certain it is that such hæmorrhages occur with great frequency towards the end of pregnancy. When they occur early, they generally lead to abortion, which is generally not more serious than abortion from other causes, and thus supply their own treatment. Hæmorrhages occurring later, but previously to the seventh month, are, as a rule, not serious, as in Müller's cases no death occurred before the seventh month; and he finds that bleeding occurs in cases of complete placental presentation in the greatest frequency from the twenty-eighth to the thirty-sixth week, and, in cases of the incomplete form, more frequently after the thirty-second week. These bleedings should be taken as a serious warning; as, though occasionally they may be slight, still not unfrequently the next hæmorrhage may prove a fatal one; and hence, as I have before urged, when the child is fairly viable, and the diagnosis of placenta prævia established, the sooner labour is induced, the safer for both mother and child.

Then comes the question, How is labour to be induced? or, having commenced, how is it to be conducted? To save unnecessary repetition, I may here state that the operation is the same in principle, whether labour have already begun or not; and differs only in application in the manner suited to each stage. Now, with