

late to do anything, so I merely gave a small hypodermic injection of morphia. Death occurred at midnight.

At the post-mortem examination made 15 hours after death the body was found to be well nourished. The skin was pale, there was not much hypostatic congestion, and there were no lineæ albicantes. Rigor mortis was well marked in all the limbs. Froth issued from the nostrils. The face was pale and bloated. There was a thick layer of subcutaneous fat. On making the usual incisions dark fluid blood ran in a stream from the peritoneal cavity. About three or four pints were sponged up and removed and a good deal of black tenacious clot was taken in fragments from the pelvis, though at present nothing visibly wrong was observed. The heart was healthy but pale. The cavities were empty except for a very little clot in the right ventricle. There was a little emphysema in the lungs and the lower lobes were oedematous; otherwise they were normal. Examination of the abdomen revealed no peritonitis and no tumour or aneurysm could be discovered. The stomach was dilated and it contained several ounces of fluid food. The intestines were normal. The liver was of average size, pale and bloodless, and rather fatty. The spleen and pancreas were normal. The kidneys were of normal size and structure. After removing all the viscera and the remaining adherent blood-clots the uterus and its appendages were carefully examined. A lesion was then observed in the left Fallopian tube. About two inches from the uterus was a small, smooth, elongated swelling about three-quarters of an inch in length and of the size and shape of a small bean; in one spot on its upper surface it was rough, soft, and red, and blood exuded on gentle pressure. This was seen to be the chorion of a very early pregnancy exposed by rupture of the containing Fallopian tube. The ovum seemed partially moveable in the stretched tube and could be rolled about in it. There were no adhesions or any traces of former peritonitis or of any other disease about the Fallopian tube, except one or two very small cysts in the fimbriæ. The uterus was a very little enlarged and was lined by a thin, reddish, adherent membrane (decidua?). The substance of the uterus was normal. Each ovary was enlarged, about two inches in length, and full of cysts as big as peas. In the left ovary was a recent corpus luteum about one-third of an inch in diameter, with a corrugated yellow capsule and a red interior.

Remarks.—The points of interest about the case are as follows:—

1. It is uncommon for extra-uterine gestation to occur in a nullipara as in this case.

2. The chief feature of the case is the early period at which fatal hæmorrhage from rupture of the tube took place. It would be interesting to determine the precise age of the ovum. From the history above given it would appear probable that the woman conceived shortly after marriage and that this ended in an early miscarriage in July. It could not, however, have been this pregnancy, even if no actual abortion had taken place, which led to the final disaster, for an ovum must be still alive and growing to cause rupture of the containing tube and if that had been the case the ovum would have attained a much larger size. Probably another conception took place, dating soon after the catamenia ceased about the end of July or between that and the next menstrual period, which began about August 5th or 6th. At any rate, from the history the duration of the pregnancy at the time of death would appear to be between 10 and 20 days. Mr. T. W. P. Lawrence, the curator of University College Museum, has made a minute examination of the specimen and he reports that from the size of the chorionic vesicle and the development of its villi, which were of considerable length and decidedly branched, the age of the ovum must have been very nearly three weeks, so that conception must have taken place not much later than July 24th, when the protracted menstruation had hardly ceased. Mr. Lawrence's measurement of the distended part of the Fallopian tube is 20 millimetres, the size of the chorionic vesicle from 15 to 20 millimetres. These measurements probably indicate the extreme length and not the diameter of a sphere. But even allowing fully three weeks for the duration of pregnancy, it is very rare (and perhaps unique) for rupture with fatal hæmorrhage to take place as early as this. I have searched through many text-books and voluminous indices but have been unable to find another instance on record. Early rupture is of course not very uncommon but the hæmorrhage is usually slight

and only amounts to a hæmatocele and is practically never fatal. I need not quote all the opinions stated in the books about the period at which fatal rupture occurs. I will only mention one or two as examples. Thus Barnes, in his work on Diseases of Women, says that rupture occurs the more early the nearer the gestation sac is to the uterus. Thus an interstitial or tubal gestation usually ruptures from the sixth to the twelfth week though it may be postponed much longer. Six to eight weeks is the time given by most authors. Mr. J. B. Sutton, in his elaborate article on Extra-uterine Gestation in Allbutt and Playfair's "System of Gynæcology," says that primary rupture takes place in the majority of cases between the third week and the tenth week, but he does not say that *fatal* hæmorrhage may take place till after the first month.

3. The most interesting aspect of the case is the clinical one of diagnosis. I think that the difficulties in this case made certainty, when I saw it, out of the question. There was no physical, symptomatic, or historical evidence of pregnancy; the facts were all against it and I therefore excluded the possibility of this being the cause.

The case most nearly resembling mine in this respect that I have found reported is one by Dr. Burton in the Transactions of the Obstetrical Society of London, Feb. 3rd, 1881. In that case fatal hæmorrhage occurred in a woman who was still suckling a child seven months old and who had just menstruated again. The ovum was of the size of a Barcelona nut and was said to be of six weeks' duration. At the time I saw my patient first there was no indication of abundant hæmorrhage, there was no shock or collapse, there was no marked pallor of the lips, and there was no accumulation of blood in the peritoneum. There were, however, two points which I distinctly noticed—viz., coldness of the surface, and a peculiar, weak, compressible pulse. I find that I noted the same phenomena in another case of the kind which I met with several years ago. I suppose they denote hæmorrhage and I might perhaps have attached more importance to these symptoms, but, as I have already said, further evidence of hæmorrhage was wanting. The absence of uterine hæmorrhage was evidence against abortion in case pregnancy existed. I excluded peritonitis, rupture of the stomach, intestinal obstruction, and acute pancreatitis from the diagnosis and was forced to assume, at any rate provisionally, the presence of a bad attack of simple colic. When I saw her at the end the existence of intra-peritoneal hæmorrhage was plain, but she was then dying, and it is doubtful whether transfusion or intravenous injection of saline fluid preparatory to laparotomy would have saved her even if the means of carrying out the process had been at hand, which was certainly not the case.

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A CASE OF CHEYNE-STOKES RESPIRATION WITH OTHER RHYTHMICAL PHENOMENA.

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THE patient in this case, a child aged four years, was admitted to the Glasgow Royal Infirmary on Sept. 19th, 1898, in an unconscious condition, evidently the subject of some form of cerebral meningitis. The history obtained from the parents was to the effect that their child had been quite well till the month of March—i.e., six months before—when she contracted measles, and since when she had been ailing more or less constantly. During the summer the patient was in regular attendance at the outdoor department of the Children's Hospital where I saw her on several occasions and where she was diagnosed as having "some pulmonary trouble, probably tuberculous." Later a persistent diarrhoea set in and it was with difficulty that this could be controlled by means of drugs. A strabismus had been noticeable ever since the attack of measles but not before. The meningeal symptoms were first observed about the beginning of September, commencing with pain in the head and followed later by vomiting, listlessness, and drowsiness. From Sept. 15th the head had been much retracted and coma more or less complete was constantly present. On admission to hospital the child was

quite unconscious though the swallowing reflex was still present. The pupils were unequal, the left being dilated and the right medium. Ophthalmoscopic examination revealed a double optic atrophy but no tubercles were to be seen anywhere in the fundus. The head was retracted and the abdomen was slightly retracted but was quite soft and doughy to the touch. Tache cérébrale was well marked. The temperature was 98° F., the pulse was 133, and the respirations were 25. During the first few hours after admission twitching movements were at intervals noted in the right arm and leg, also more extensive movements in both arms and legs. The respirations all this time were sighing in character, but after midnight the breathing assumed the Cheyne-Stokes character, the respiratory cycle lasting 50 seconds and consisting of 80 separate respiratory movements. The pupils likewise showed rhythmical movements. During the pause they became contracted, while with the respiratory movements they began to dilate, till at the acme of respiration they were widely dilated. While thus dilated the pupils reacted slightly to light. Rhythmical movements corresponding in time to the above were also observed in the arms, especially in the right arm. The arms it is to be noted were at this time more or less rigid and they presented slight tremor. They were extended, with the thumbs adducted and the fingers flexed at the inter-phalangeal joints. During the advent of the Cheyne-Stokes respiration, then, this tremor exhibited a rhythmical activity of such a nature that during the pause the tremor was scarcely perceptible, increasing, however, with the breathing till at the height of the respiratory cycle the tremor presented movements of considerable amplitude. As the respiratory movements lessened so did the extent of the tremor movement. These various rhythmical phenomena were observed at frequent intervals until the time of death, about 30 hours after admission into hospital.

On post-mortem examination the convolutions of the brain were found to be much flattened and the soft membranes and ventricles contained a large quantity of clear serous fluid. Numerous tubercles were to be found scattered throughout the soft membranes. The brain substance generally was very soft, but otherwise it presented no abnormal appearances. Both lungs were studded with miliary tubercles of comparatively large size and there was extensive tuberculous consolidation and excavation of the upper lobe of the right lung. The right bronchial glands were enlarged and caseous, but the left seemed healthy. Miliary tubercles were to be seen in the liver, spleen, and kidneys, and some small and evidently recent tuberculous ulcers were found in the colon and ileum. The mesenteric glands seemed healthy. The medulla and pons were fixed in formol and stained with theonin according to Nissl's method. Numerous sections were examined at different levels, special attention being paid to the sections passing through the *alæ cinereæ*. In none of the sections, however, could any distinctive change be found in the ganglion cells of either the tenth, the twelfth, or of any of the other nerve nuclei. Many of the ganglion cells I found to be quite normal. Some showed what has been termed a "polar" or "apical" chromatolysis and some were very faintly stained like to the so-called "ghost-cells." But these changes were seen throughout the whole medulla and pons, not confined to any special level or nucleus, and I take it that they were due to the meningitis or the high temperature (the temperature reached 107·6° before death) and not to be specially associated with the Cheyne-Stokes respiration.

That, then, very briefly is the record of this case and it would seem to be of some interest in that (1) careful microscopic examination of sections of the medulla through the *alæ cinereæ*—the supposed rhythmical respiratory centre—showed no distinctive change, at least not more than in the rest of the medulla and pons; and (2) in that rhythmical movements were also present in the arms, showing the extent of the affection. Some time ago Dr. Terrien¹ reported a case of Cheyne-Stokes respiration where not only was there a rhythmical movement of the pupils, but also a rhythmical anæsthesia of the parts of the face supplied by the fifth nerve. During the pause this anæsthesia was present, passing off again as soon as the respiration began. In explanation of this Dr. Terrien points out that the cilio-spinal centre and the sensory nucleus of the fifth nerve lie close to the respiratory centre and that whatever was causing

the Cheyne-Stokes breathing had evidently extended its influence to these adjacent centres. In my case tactile sense over the area of the fifth nerve could not be tested as the child was unconscious all the time the Cheyne-Stokes breathing was present. But to it Dr. Terrien's explanation would not seem to be applicable, for the arm-centres are certainly in no way adjacent to those of the pupil and respiration. My case seems rather to favour the view advanced by Dr. G. A. Gibson² that Cheyne-Stokes breathing and its accompanying rhythmical phenomena "are instances, among many others, of the common tendency towards 'pulsatile or rhythmic activity' manifested by all living matter."

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THE DIASTOLIC MURMUR OF MITRAL STENOSIS.

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It is generally recognised that, as stated by Bristowe,¹ the murmur of mitral stenosis may be early, middle, or late diastolic; but the clinical significance of the variation in time of these murmurs is practically unknown; and it was the attempt to discover wherein cases with the early and middle diastolic murmur differed from those with the more ordinary presystolic murmur which led me to examine a series of cases of mitral stenosis. From clinical examination of a considerable number of cases I have arrived at the following conclusions, in support of which I am unable to give figures, but they will probably be found to agree with the experience of most.

1. The murmur in an early compensated case of mitral stenosis, such as is seen in the out-patient room and often discovered only on routine examination, is always presystolic. Early diastolic murmurs are practically never heard while compensation remains good.

2. On the other hand, when cardiac failure supervenes and the patient is troubled with dropsy, cyanosis, and other symptoms of mitral stenosis, the murmur is frequently heard in the first half of diastole, although even now it is not so common as the presystolic murmur. (A long diastolic murmur running right up to the thrust and increasing in loudness and roughness as it approaches the systole is included as a variety of the presystolic murmur.)

3. A presystolic murmur may or may not be accompanied by a systolic bruit, but an early diastolic murmur is almost always preceded by a systolic murmur. A single, early diastolic murmur at the apex is very rare in mitral stenosis and strongly suggests aortic regurgitation even if no murmur is heard at the base.

4. A presystolic murmur frequently changes into a diastolic murmur or *vice versa* in a short time and the period of diastole occupied by the murmur may even vary in the erect and recumbent postures. The difference in the cause of these murmurs is therefore not likely to be found in any structural condition of the affected valves, but rather in the way in which the heart is working at different times.

I have examined the records of University College Hospital for the past few years in order to see if in the fatal cases of mitral stenosis any post-mortem differences could be substantiated between the hearts of those in which during life the murmur was presystolic and those in which it was in the first part of the diastole. Such cases are not easy to collect, as it is frequently found in mitral stenosis that before death there may be no murmur or it may be confined to the systole, so that unless the case is under observation for some time it may be useless for this purpose. I have, however, collected 21 suitable cases of well-marked mitral stenosis with necropsies.

In 15 cases there was either during life a presystolic murmur or a very long murmur running up to the thrust. In 12 of these the left ventricle was either small, normal in size, or slightly hypertrophied without dilatation; in two the left ventricle was slightly dilated; and in one patient who died, not from the cardiac condition, but from septicæmia, it

¹ Le Progrès Médical, Jan. 8th, 1898.

² Cheyne-Stokes Respiration (Edinburgh, 1892).

¹ Proceedings of the Medical Society of London, 1888, p. 41.