an attack of acute rheumatism which came on in the second week of her convalescence.

In going over the case, the interest to me is mainly in connexion with the symptoms of January 23rd and 30th, and their proper interpretation. Whether they were really attacks of biliary colic, or were associated in some way or other with the ruptured tube, it is difficult to say. If the latter, and a correct diagnosis had been possible at the time, the patient would have been in a much better condition to stand operation, and consequently the risk to life would have been less.

TWO CASES OF MITRAL STENOSIS COMPLICATED BY PREGNANCY.

By G. A. WILKES, M.D.BIRM., D.P.H.CONJ.
Birmingham.

THE following cases of mitral stenosis, ending fatally after the first parturition, illustrate the danger which attends this form of heart disease during gestation and labour:

Case 1.

Mrs. W., aged 23 years, first seen on April 6th, 1899, gave a history of rheumatism at 10 years of age. She had no knowledge of any subsequent heart disease, and enjoyed good health till a few days before I saw her. She was then in the seventh month of her first pregnancy, and was deeply cyanosed, suffering great dyspnoea, and expectorating blood-stained sputum. Moist rales were heard all over the chest, back and front. The cardiac impulse was in the fifth space, displaced a little horizontally, and accompanied by a presystolic thrill. There was epigastric pulsation. The superficial cardiac dullness began at the third rib. A presystolic murmur and slapping first sound were heard at the apex. The pulse was regular and fast (134 per minute). She was given a mixture of tr. digitalis m xv, and liq. strychn. mx, every four hours, and calomel gr. v.

I saw her again on the morning of April 7th. The bowels had acted freely. There was no improvement in her condition, and as labour pains had commenced in the night and the os was dilated I ruptured the membranes. The pains then became more vigorous, and in about an hour's time she was delivered of a seventh-month fetus. The second stage of labour was comparatively easy, but the bearing-down efforts caused the patient to become distressingly short of breath and increased the cyanosis and the pulse-rate. There was very little uterine haemorrhage during the third stage, and as the right heart was seriously embarrassed, venesection was suggested, but refused by the patient and her friends. Stimulants were freely exhibited, and gr. $\frac{1}{2}$ of strychnine injected under the skin, but without avail. The pulse became almost imperceptible, and she died two hours after the birth of the child.

Case 2.

Mrs. B., aged 29 years, first seen on April 10th, 1899, had had rheumatic fever at 15 years of age. Her subsequent history was good till she became pregnant. She was ignorant of her heart disease. During her pregnancy she suffered from cough and shortness of breath on exertion, symptoms which became more marked as she approached full term.

Labour commenced on April 9th, 1899. She was attended by a midwife. On April 10th she gave birth to a full time child after a long and exhausting labour, which greatly increased her dyspnoea. When I saw her some hours after parturition, she was in much the same condition as Case 1—cyanosed, breathless, and expectorating bloodstained sputum. There was a presystolic thrill and a presystolic murmur at the apex. The cardiac dullness was increased upwards. Epigastric pulsation was present. The pulse was small, regular, and 120 per minute. Moist rales were heard all over the chest. Although she had lost a considerable amount of blood *post partum*, and was still losing, it did not appear to have relieved her breathlessness and cyanosis in any way. Venesection, therefore, was recommended, but rejected in favour of six leeches over the liver area. Brandy was freely given, and gr. $\frac{1}{2}$ of strychnine injected. She was also given tr. digitalis mxv and liquor strychninae mx every four hours, and a calomel purge. I saw her again on April 11th. The bowels had acted freely, and there had been considerable oozing from the leech bites. Her condition, however, was hopeless. She had sunk down in the bed breathing easily, but greatly cyanosed. The extremities were cold and oedematous. The heart was very fast and feeble, and she died at 8 p.m. the same day, thirty hours after delivery.

The cases here recorded emphasize the liability to fatal loss of compensation in mitral stenosis when associated with pregnancy. During gestation the work of the heart is increased. The presence of the placental circulation increases the amount of blood and raises the blood pressure. The cavities of the heart become dilated, and the left ventricle is usually slightly hypertrophied. The right ventricle, being thinner and weaker, does not so readily undertake compensatory changes, and shortness of breath, pulmonary congestion, and other evidences of right ventricle embarrassment often occur in debilitated women during pregnancy in the absence of valvular disease.

In mitral stenosis the compensation ultimately depends on the integrity of the right ventricle. In severe cases, apart from pregnancy, the right heart utilizes most of its reserve force. A breakdown of the compensation is almost inevitable when such cases are called upon to meet the greater demands of pregnancy and parturition. In a number of cases premature labour results, and the earlier in pregnancy the right ventricle becomes unable to cope with its difficulties the

sooner the pregnancy is liable to be brought to a close. A failing right ventricle leads to obstruction to the flow of blood in the great veins, to congestion of the placenta, and to the circulation of imperfectly oxygenated blood in that structure. Haemorrhage into the placental area may result, or the fetus may die from asphyxia, in either case initiating labour. It is during the second stage of labour, whether premature or not, that the compensatory mechanism is most severely taxed. In the effort of bearing down the glottis is closed, the diaphragm descends, and the abdominal muscles contract vigorously. The first effect of the contraction of the diaphragm and abdominal muscles is to drive the blood from the abdominal veins to the right auricle, assisting the filling of the ventricle in diastole. But as the pressure rises in the thorax, the blood is dammed back in the great veins, and the filling of the right heart opposed. At the same time the increased pressure of the air enclosed in the air passages tends to drive the blood from the lungs and raise the pressure in the pulmonary artery. The positive intrathoracic pressure of forced expiration is replaced by the negative pressure of a deep inspiration, and blood is aspirated from the venous system to the right side of the heart, overdistending the auricle and ventricle.

In the normal condition the expansion of the lungs in inspiration lessens the intrapulmonary pressure; the pulmonary vessels dilate and relieve the burden of the right heart by accommodating more blood. In mitral stenosis, however, the constant high pressure in the pulmonary vascular system counteracts to some extent the beneficial effects of inspiration. The right ventricle is thus persistently overloaded, and may become dilated to a point beyond recovery. Women with severe mitral stenosis should, if they seek an opinion on the subject, be advised not to marry. For, although such patients may survive the ordeal of parturition, a grave risk is incurred, and the danger becomes greater with succeeding pregnancies. In the case of patients who present symptoms of serious circulatory disturbance in the early months of pregnancy, or in the case of those who have with difficulty escaped a fatal issue from cardiac failure in a previous labour, the induction of abortion should be considered in preference to premature labour, as the latter, whether spontaneously or artificially induced, appears to be attended with as heavy a mortality as labour at term.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

MYIASIS INTESTINALIS.

IN connexion with the article in the *BRITISH MEDICAL JOURNAL* of March 15th, 1902 (*EPITOME*, par. 169), the following account of what I observed in North China appears to be interesting:

An officer in a regiment serving at Tongshau, North China, during the recent troubles in China, contracted dysentery. On examination of the stools, blood, mucus, and a number of small yellow-coloured worms, with a blackish head, resembling maggots, were found. Microscopic examination showed them to be dipterous maggots.

These were found in every stool passed by the patient. In consequence, calomel was given, but with no good result. Next santalin was given, but with no better result. The stools were then examined immediately after passing, and no larvae were found in them, although shortly afterwards numbers were detected. One day, whilst examining a stool, two large flies settled on it, and rapidly deposited the larvae upon it; in less than a minute the stool was swarming with them. After this I many times waited by freshly-passed stools in which were no larvae, and watched these flies deposit the larvae upon the stool. The larvae were deposited on the stools as larvae, and not as ova.

This led me to catch this species of fly and dissect it. Inside the fly were larvae in the same shape and form as when deposited on the stools. Some of the flies which were dissected were from those which settled on the stools, and the others were captured outside on the windows of houses; all had live larvae inside them. Finally, healthy stools, in which were no larvae, were found to be swarming with larvae very shortly after passing them, due to the flies depositing the larvae on them.

It appears to me, therefore, that these larvae are passed not