organs and may affect any part of any organ of the body. Difficulty in diagnosis arises if the possibility of malaria is not thought of, and examination of the blood (if necessary, repeated examinations) is essential for a definite diagnosis.

Early detection and immediate specific treatment will save many lives: one cannot afford to wait long under such critical conditions.

Medical Memoranda

Vasa Praevia Simulating Ante-Partum Haemorrhage

The following case is recorded as a reminder of a rare cause of bleeding in the first stage of labour.

CASE REPORT

A primigravida aged 35 was admitted on April 24, 1949. Her last menstrual period started on July 25, 1948, and the pregnancy had been uneventful until two weeks before admission, when the blood pressure began rising and the urine contained a trace of albumin. On April 21 the membranes had ruptured spontaneously, and on the day of admission pains had started, followed two hours later by a sudden considerable loss of bright blood. From that time no foetal movements had been felt.

Photograph of the placenta, showing the torn umbilical vein. Black rods have been placed in the torn vessel.

On examination the patient's general condition was good. The pulse rate was 76, the blood pressure 130/85, and there was some oedema of the ankles. The uterus was at full term and the vertex presenting, deeply engaged; the foetal heart could not be heard. There was slight continuous vaginal bleeding. A diagnosis of toxic antepartum haemorrhage was made. Labour progressed rapidly, and five hours after admission the

os was fully dilated. After two hours in the second stage the infant was delivered by forceps—a male, 8½ lb. (3.85 kg.), still-born, fresh, and with marked pallor. The placenta and membranes were expelled intact within a few minutes, with very little blood clot. Examination of these showed that the insertion of the cord into the placenta was velamentous and bordered the hole in the membranes. At one point the membranes had torn into the umbilical vein as it lay separated from the arteries (see photograph). The placenta was ischaemic, but its maternal surface showed no sign of premature separation.

The sudden haemorrhage associated with cessation of foetal movements and absence of foetal heart sounds shortly after the onset of pains indicates that, as the cervix began to dilate, the opening in the membranes already present extended outwards and involved the more delicate vessel in the cord. Vasa praevia is an uncommon condition and provides an unexpected foetal risk.

My thanks are due to Mr. A. P. Bentall for permission to publish this case.

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Experimental Ascorbic Acid Deficiency

The article recently published in the *Journal* on the Falkland Islands Dependencies Survey (Roberts, 1949) prompts me to report an experiment in ascorbic acid deprivation carried out on a similar expedition to Hope Bay, Graham Land, in January, 1945. It was desired to observe how long an interval was necessary for the clinical manifestations of ascorbic acid deficiency to develop in an individual living on the normal diet at the base, and on the unsupplemented sledge ration. As the observations were made on myself alone, they can be regarded as indicating only the approximate period required.

During the journey to the Falkland Islands I had been taking the normal diet, which included large quantities of fresh oranges, orange juice, and fresh vegetables. It is therefore reasonable to assume that on arrival at Hope Bay a condition of near-saturation had been achieved. Thereafter I ate the normal unsupplemented diet, which included fresh penguin meat and fresh seal meat, when available, but I omitted the 50 mg. of ascorbic acid which was taken daily by all the other personnel.

During the following two months no abnormal symptoms or signs were observed. At the beginning of the third month, however, an increasing degree of hyperkeratosis was evident. This particularly affected the extensor and flexor surfaces of the thighs and the extensor surfaces of the arms and forearms, but was noticeable on the abdomen, chest, and back also. Some desquamation was apparent, scales of about 1 mm. square being lost. No petechiae were seen.

In the beginning of the fourth month I experienced lassitude and increasing fatigue, so that travelling became an effort. On May 7 I had a short attack of giddiness: at the same time irritability to an extent unwarranted by the provocative incidents was noted. There was no abnormality of the alimentary or urinary systems. Two ulcers developed in the mouth, one on the gums and the other on the hard palate, but no bleeding occurred. On April 16 and May 11 the tourniquet test for capillary resistance was performed. No capillary haemorrhages were observed on either occasion.

After May 20, I took 400 mg. of ascorbic acid daily for one week, then 100 mg. daily for a further week, after which time I consumed the normal dose of 50 mg. daily. During the following month the hyperkeratosis, ulceration of the mouth, and other signs gradually disappeared, and after two months no trace of the condition remained. One interesting feature was the appearance on June 5 of oedema of both ankles, for which no other cause was apparent. By the following month the oedema had disappeared, and has not recurred since.