a further two tablets three or four days after the initial dose to prevent a relapse, though this additional dose is not commonly needed.

The above doses of chloroquine and camoquin are adult doses, and children and infants are given proportionately less.

Some Interesting Cases

Most of these have been referred to above, but two cases of malarial pneumonia are here included.

Case 1.—An elderly man who had been ill for one week complained of slight headache, had pains all over his body, and felt tired. There was no shivering, no vomiting, and no feverishness. Physical examination, apart from revealing severe anaemia, was entirely negative. He was afebrile, and the spleen was not palpable. He was admitted for investigation, and to my surprise malaria parasites were found in his blood. He was given antimalarial therapy, and remained in hospital ten days. During this time his temperature remained subnormal, and the spleen did not become palpable; also there were no rigors. Quinine followed by pamaquin, and iron preparations for the anaemia, brought about a cure within three months.

Case 2.—A young woman complained on admission of headache, generalized body pains, and feverishness. There were no rigors and no other symptoms. Physical examination, apart from a pyrexia of 100.8° F. (38.2° C.), was entirely negative. Malaria parasites were present in the blood smear, and proguanil therapy was begun. On the fourth day her temperature was 101° F. (38.3° C.), and there was no subjective improvement. No further physical signs developed, and the blood smear was again positive. Examination of the urine and stools showed nothing abnormal. As the patient was not very ill, it was decided to continue the proguanil and await further developments. On the seventh day she was still pyrexial, and parasites, though scanty, were still present in the blood smear. Quinine was then given, since the proguanil had been discontinued after six days. Within 48 hours the temperature was down, and fell by crisis over two days, being normal on the ninth day. The further course was uneventful.

Case 3.—A boy aged 4½ years complained of severe diarrhoea. The stools were loose, though they contained no blood or mucus. The mother said there had been about 20 motions during the preceding 24 hours. The child had vomited once at the onset of the disease; there were no other symptoms. On examination his temperature was 105° F. (40.6° C.), but, apart from some abdominal tenderness, no abnormal physical signs were found. He had been receiving proguanil prophylactically twice a week. Examination of the urine and stools was repeatedly negative, and as a last resort a blood smear was taken. This showed a very heavy falciparum infection. The child responded well to quinine and symptomatic treatment. His spleen became palpable on the third day in hospital. At no time did he have a rigor.

Case 4.—A young woman was admitted as a case of lobar pneumonia, with typical signs of consolidation at the left base. There was no splenomegaly, and she had had only one rigor at the onset of the disease. She had a cough with blood-stained expectoration. She did not respond to sulphadiazine and penicillin, and a blood smear taken on the fourth day showed a falciparum infection. After quinine the pneumonia rapidly resolved.

Case 5.—This woman was a maternity patient, and on the fourth day of the puerperium developed a temperature of 104° F. (40° C.) and a severe dry cough. Consolidation was found in the right mid-zone, and this did not respond to antibiotics. On the sixth day of the puerperium the spleen became palpable, and a blood smear showed falciparum infection. She responded satisfactorily to antimalarial therapy.

Case 6.—A young female was admitted complaining of severe upper abdominal pain, incessant vomiting of blood-stained matter, and headache; there had been one rigor. She had become ill two days before admission. Examination showed a pyrexia of 101° F. (38.3° C.), which rose that evening to 104° F. (40° C.). There was rigidity of the left upper quadrant, with tenderness in the left hypochondrium. No other abnormality was found. The provisional diagnosis was splenitis of unknown aetiology. Examination of the urine and stools was negative, but a blood smear was positive for malaria. It was decided to treat expectantly, and quinine with symptomatic therapy produced a fall of temperature by lysis, beginning on the third day in hospital; she made a complete recovery in eight days.

Conclusions

These can best be summarized in tabular form. The figures in parentheses refer to the incidence of symptoms and signs in the 50 analysed cases.

Symptoms		Signs	
Rigors	Over half (60%)	Pyrexia	Usual (98%)
Headache	Usual (88%)	Splenomegaly	Over half (56%)
Vomiting	Not common (24%)	+Blood smear	Almost all (60%)
Feverishness	Usual (90%)	Herpes febrilis	Unusual (0%)
Generalized pains	Common (72%)		

The cases reviewed include patients of both sexes and all ages, and, though females preponderate, these patients form a fair cross-section of our hospital population.

Summary

The clinical picture of subtertian malaria is discussed. Cerebral malaria is not considered.

The variations in the clinical picture are discussed, and a series of 50 consecutive malaria patients admitted to hospital has been analysed.

Some illustrative cases are described.

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CONGENITAL DIAPHRAGMATIC HERNIA IN SIBLINGS

BY

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True congenital diaphragmatic hernia is a relatively uncommon anomaly, and its occurrence in siblings has been reported only once previously (Mäkelä, 1916). We record here the case of a woman who gave birth to two children, each of whom died from the condition shortly after birth.

Case History

A German woman, then aged 19, was admitted to hospital in 1948 for pre-eclamptic toxaemia and hydramnios necessitating induction of labour at 36 weeks. Forceps delivery was carried out because of maternal distress and the occipito-posterior position of the vertex. The infant, a boy weighing 6 lb. 14 oz. (3.1 kg.), gasped for about twenty minutes after birth, and then died. At necropsy there was a massive left-sided diaphragmatic hernia. The stomach and intestines, from the ileum to the transverse colon, were present in the thorax, together with the spleen and the left lobe of the liver. The left lung was collapsed and there was considerable displacement of the mediastinum to the right. Two-thirds of the liver, the duodenum, the pancreas, and kidneys remained in the abdominal cavity. There were no other congenital defects.

The patient became pregnant for the second time in 1951. She attended the antenatal clinic at 12 weeks, when her

condition was normal. At 25 weeks she was admitted for a few days with unexplained right-sided abdominal pain, which cleared up spontaneously. At 31 weeks she was complaining of vomiting and was mentally depressed, but there was no oedema, albuminuria, or hypertension. At 33 weeks she was admitted with severe vomiting and moderate oedema of the ankles; her blood pressure was 150/110. There was no further vomiting after admission. Her blood pressure at 36 weeks was 140/88; by 38 weeks it had risen to 150/100, and there was marked oedema of the ankles, face, and abdominal wall, but there was no albuminuria at any time.

After a medical induction at 39 weeks, she gave birth to a male child; its condition at first was good, but it rapidly deteriorated about ten minutes after birth, when it became cyanosed and limp. The air passages were cleared, oxygen was administered, and the child was put in an oxygen tent. Eight hours after birth only the feet remained slightly cyanosed; the breathing, however, was rapid and jerky, and there was diminished chest movement with absent air entry anteriorly and posteriorly at the right base, over the right mid-lobe, and in the right axilla. Bubbling rales were heard at both apices, louder on the right. Heart sounds were normal. A diagnosis of collapsed right lower and mid-lobes was made. His condition deteriorated suddenly and he died 19 hours after birth.

Necropsy showed that the body was well nourished. Cyanosis and engorgement of the face and neck and some oedema of the feet were present, but no external congenital anomaly. A massive right-sided diaphragmatic hernia through a large posterior defect in the diaphragm on the right side was found. The small intestine, spleen, ascending colon, right half of the transverse colon, and pancreas were present in the thorax. The greater part of the liver was in the abdominal cavity, but a large lobulated mass of liver tissue was present in the thorax, attached to the main mass by a narrow isthmus of fibrous tissue; the anterior edge of the diaphragmatic defect appeared to have cut into, and partly segregated, this lobule of liver from the remainder of the organ. The right lung was collapsed. The left pleural cavity was normal. The remainder of the liver, the spleen, the kidneys, the left half of the transverse colon, the descending and pelvic colons, and the rectum were in their normal situation. The brain appeared normal. Sections of liver, adrenal, pancreas, and myocardium were normal. Both lungs showed early expansion of the alveoli, especially in the left lung.

Discussion

Severe congenital diaphragmatic hernia is calculated to occur once in 23,000 deliveries, but lesser defects of the diaphragm are more common. Mäkelä (1916) recorded the case of a woman who gave birth to four children, all of whom died during or shortly after delivery. Necropsies on the second and fourth children showed large left-sided diaphragmatic hernias through large defects in the diaphragm. In addition both children had abnormal genitalia, uterus duplex separatus and septate vagina. The latter observation is significant, as the mother also had a uterus unicollis with an extra vestigial horn, but did not have any diaphragmatic lesions. Necropsy was not performed on the first or third child, so that it is only surmised that they, too, may have suffered from congenital diaphragmatic defects because they died in a similar way. In our own case the mother had had no acute infectious disease during pregnancy, and neither she nor her husband had ever been subjected to irradiation. Although there is no known definite history of congenital defects, of this or any other type, in either the mother's or the father's family, it seems likely that these two identical uncommon congenital defects may be due to some inherited genetic trait. Mäkelä's cases seem to support such a possibility.

Andersen (1949) showed that a genetic tendency to diaphragmatic hernia which was present in a stock strain of laboratory rats could be increased by feeding them on a diet deficient in vitamin A, although the mother showed no evidence of vitamin deficiency. He suggested that these defects might be due to the rate of growth of the diaphragm being slowed. Normally the diaphragm closes before the intestines return to the abdominal cavity from the extra-embryonic coelom. If the diaphragm is late in closing, the abdominal contents can pass up into the thorax through the still patent pleuro-peritoneal canal, and the delayed diaphragm is ultimately unable to close. Such a mechanism might apply in the human lesions—a suggestion supported by the lobated appearance of the liver in the second case, as if it had been nipped by the edge of the closing diaphragm, which ultimately became unable to close completely. While alive to the errors inherent in pursuing too closely an analogy between animal results and human cases, we think it significant that, in experimental animals, tendencies towards inherited defects can be unmasked or accentuated by dietary deficiencies. How far such deficiencies could have played a part in our case is not known, though it is likely that the patient lived on a notably deficient diet during puberty and adolescence in Germany. The occurrence of toxaemia and hydramnios in both pregnancies may also be significant.

The true condition of the second child might have been diagnosed in view of the previous history of diaphragmatic hernia, but such an occurrence in successive pregnancies is obviously very rare. Had the condition been in fact diagnosed, it is clear that no surgical treatment, such as that of Belsey and Apley (1949) or of Gardiner (1950), could have been embarked upon. The chances of the patient having a third similarly affected child are unknown; they are probably high. A high-vitamin supplementary diet would be indicated in any subsequent pregnancy in view of Andersen's findings.

Summary

The case is reported of a woman who gave birth to two children each of whom died shortly after delivery with congenital diaphragmatic hernia.

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RECIPROCAL CHANGES IN BLOOD FLOW OF THE LIMBS AND RECTUM DURING GENERAL ANAESTHESIA

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It has been shown by plethysmography (Lynn and Shackman, 1951) that circulatory changes develop in the limbs of patients undergoing surgery under general anaesthesia. The changes begin as soon as anaesthesia is induced, and occur in the skin and muscle tissue of all four limbs. Further studies (Shackman, Graber, and Melrose) have led to an indirect deduction that coincidental and reciprocal changes take place in the splanchnic circulation.

The experiments of Grayson (1951) in unanaesthetized human subjects, showing that cutaneous blood-flow changes are accompanied by "directionally opposed bowel blood-flow changes," has lent emphasis to our observations. We have repeated Grayson's studies, with slight modification, on several patients undergoing operation, and have confirmed that the coincidental and reciprocal change in blood flow that he noted in the limbs and the lower bowel, after reflex heating, occur also during general anaesthesia.

We have recorded the rectal temperature by a copper-constantan thermocouple inserted into the rectum, the skin