within a few weeks of the first symptoms, unless the attacks were relapses of an undetected primary infection months or years before. In neither history is there any suggestive record of undiagnosed fever. Case 1 had moved from Kent to London eight weeks before developing symptoms. The only known contact with potentially malarial subjects ended six months before that, when she left the neighbourhood of the American military camp; but no symptoms were noticed during or shortly after that period. It is most probable that she was bitten in Welling, Kent, by an anopheline mosquito infected by a Service case from overseas.

Case 2 must similarly have contracted the infection either in the Isle of Sheppey in August or in Chelsea thereafter. While in the Isle of Sheppey he was known to have been bitten by mosquitoes, which could have been anopheline. It is possible that his father's bouts of fever are malarial, although he was never overseas; and, while unlikely, it would also be possible during a month's holiday for infection to be transferred to the child. The mother was also bitten, but apparently was not infected. It is more probable that anophelines in the Isle of Sheppey acquired malaria parasites from a local resident infected overseas during the war. It is most unlikely that the infection in either case was contracted in London, where the density of population would lead to the recognition of more than one or two cases if infected mosquitoes existed in any area.

Although *Anopheles maculipennis* is widely distributed in Great Britain, indigenous malaria during this century has been confined almost entirely to the south-east seaboard of England and to the Thames Valley (Min. Hlth. Memo., 1943). Towards the end of the 1914-18 war numerous cases of malaria occurred among children and adults who had never left this country. In 1919, 300 cases of indigenous malaria were notified; since then the numbers have fallen fairly steadily (7 cases in 1945), the largest number occurring in north-east Kent. In addition to the two cases described above, Dr. P. G. Stock informs us that five other cases of indigenous malaria (all adults) were notified during 1946, including two from Oxford (Stevens and Blackman, 1946) and one from Kent. There is no record of primary malaria having been contracted in London for over 25 years.

REFERENCES

- Min. Hlth. Memo. (1943). *Measures for the Control of Mosquito Nuisances in Great Britain*. London.
Stevens, A. L. B., and Blackman, G. G. B. (1946). *British Medical Journal*, 2, 625.

Medical Memoranda

Peptic Ulceration in Identical Twins

The literature relating to peptic ulceration in identical twins was reviewed by Riecker (1946) when he reported the occurrence of duodenal ulceration in 20-year-old identical female twins. He found no reference to any such cases before 1935, but since then Schindler (1935) has recorded the occurrence of gastric ulcer in 39-year-old identical twin brothers; and von Mentzingen (1935) and McHardy and Browne (1944) have reported cases of duodenal ulceration in 20-year-old identical female twins and 28-year-old identical male twins. The family history was not included in McHardy and Browne's report, but in the other three peptic ulceration was present in one or other parent.

A fifth case of peptic ulceration, in 30-year-old identical male twins, can now be added to the series. These twins were born on Sept. 8, 1916. The onset of symptoms in Case 1 was followed within a month by the death of the other from repeated haematemesis. When they were first examined there was no history of any gastro-intestinal disorders in either

parent or in the remaining nine siblings (six females, aged 34, 33, 26, 24, 21, and 19 years; and three males, aged 28, 23, and 18 years), all of whom were alive and well. Since then the two youngest members of the family have both complained of indigestion, and at the time of writing there is radiological evidence of duodenal ulcer in the youngest male. The evidence that these twins were identical is based on the fact that they were identical in appearance and that there was only one placenta.

CASE 1

The patient, a leading aircraftman, aged 30, had been in the R.A.F. for 4½ years, and was in medical category A1. He was admitted to hospital in Antwerp under my care on Oct. 1, 1946, with a six-weeks history of abdominal pain. Following the death of his identical twin on Sept. 9, 1946, he became acutely depressed and anxious, and immediately experienced a pronounced intensification of his pre-existing symptoms. The pain was localized to the right upper quadrant of his abdomen, and did not radiate to any other site. It occurred at irregular intervals throughout each day, and not at any definite time after meals, and lasted on the average for one-half to one hour. Of late it had become worse when hungry, being then relieved by food, and had awakened him between 2 and 4 a.m. The pain bore no relation to the quality of food taken, and was not associated with either nausea or vomiting. His appetite was poor, but he was not afraid to eat. He did not think that he had lost weight. He had taken no alkalis. At the time of onset of symptoms he passed three or four fluid stools daily for three days, but he had no fever, and, so far as he knew, the stools contained neither blood nor mucus. He had had no serious illnesses in the past. His occupation entailed regular meal-times. He smoked 10 to 15 cigarettes a day, and during the last few weeks had consumed some 6 to 7 pints (3.4 to 4 litres) of beer daily.

On examination he was co-operative and intelligent, yet he appeared tired and apathetic. The only abnormalities present were that all his premolars and molars were deficient, though no denture was worn, and examination revealed some localized rigidity and tenderness below the right costal margin, extending 1½ to 5 in. (3.8 to 12.5 cm.) from the midline. The red cell count was 4,130,000 per c.mm.; haemoglobin, 92% (Haldane); and colour index, 1.11. The benzidine test for occult blood was positive on three consecutive examinations. A test meal showed a highly acid resting juice, with a rapid rise in the total and free acidity, and the presence of starch indicated a delay in emptying. A barium meal examination (Capt. W. N. Thompson) revealed that the stomach was orthotonic, with a moderate increase in the mucosal relief. The duodenum showed retraction (Åkerlund) deformity, with a small crater on the antero-lateral wall of the cap.

CASE 2

The twin brother of Case 1 was admitted to the Royal Salop Infirmary, Shrewsbury, on Sept. 6, 1946, having recently been released from the R.A.F. after 3½ years' service in the Middle East. For the last four or five years he had suffered from indigestion and attacks of mid-abdominal pain after meals. These attacks lasted on the average for six to seven weeks, and were then followed by long periods of complete freedom. During the 24 hours preceding admission he had complained of indigestion, had vomited food only on four occasions, and had developed diarrhoea, passing jet-black stools.

On admission he felt very weak and thirsty. His mucous membranes were pale and his tongue moderately dry. The pulse rate was 112, and the blood pressure 115/70. There was an apical systolic murmur. The liver and spleen were not palpable. On Sept. 9 he vomited 57 oz. (1.6 litre) of material which had the appearance of "coffee-grounds," and he also had a large melaena. In spite of transfusions he died the same day. The cause of death was given as "haematemesis and melaena from a duodenal ulcer." Consent was not given for a necropsy.

I wish to thank Dr. A. D. Rope for allowing me to use the notes of one of his cases (Case 2), and Col. W. I. FitzGerald-Powell, late R.A.M.C., for permission to publish this report.

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REFERENCES

- McHardy, G., and Browne, D. C. (1944). *J. Amer. med. Ass.*, 124, 503.
Riecker, H. H. (1946). *Ann. intern. Med.*, 24, 878.
Schindler, E. (1935). *Chirurg.*, 7, 327.
von Mentzingen, F. (1935). *Z. menschl. Vererb. Konst. Lehre.*, 19, 432.

The Australian Government has started a campaign against tuberculosis and appointed Dr. Harry Wenderly as Director of Tuberculosis. The Commonwealth Government has granted £250,000 for payment to sufferers from the disease and their dependants.