solid food because he was afraid to eat. When he was admitted he had typical symptoms and signs of a perforation, and from the history it was reasonable to assume it to be a perforated duodenal ulcer.

At operation there was a perforation in the anterior wall of either the first part of the duodenum or of the pyloric antrum of the stomach. As there was so much oedema it was difficult to say with certainty the exact site of the perforation. The perforation was dealt with and the patient made a rapid recovery. On the 14th day he was transferred to a medical ward, as was the usual practice in that hospital.

A fractional test meal on November 29 showed a normal free acid curve, but a trace of blood was found during the second and third hour. A chest radiograph taken about the same time was normal. On December 9 a barium meal showed a suspicious persistent filling defect in the pyloric antrum. Three days afterwards a gastroscope was passed, but for no obvious reason would not pass beyond the cardiac sphincter.

It was noted on December 16 that his appetite was very good, but on December 19 he developed a slight pyrexia which persisted. His general condition from now on began to deteriorate. His haemoglobin was found to be only 53%, but despite repeated blood transfusions there was little improvement in the anaemia. It was thought to be an aplastic type, possibly due to secondary growths in the bone marrow. Blood culture had been found to be negative. On December 20 a chest radiograph revealed an opacity in the left upper zone, and on January 6 early ascites was noted and this became worse in the next few days. His general condition deteriorated and he died on January 28—exactly three months after the operation.

At necropsy he was found to have a carcinoma of the pyloric antrum, with an unusual pattern of secondary growths in the lungs, left suprarenal, and in several ribs.

The interesting features about this case were the history, suggestive of a perforated duodenal ulcer; the fractional test meal —no achlorhydria; that his appetite was good, even up to six weeks after operation; and that the condition deteriorated so rapidly after operation that one would infer that secondaries were present at the time of the perforation, and a gastrectomy would not have prolonged life. This finding agrees well with the general poor ultimate prognosis in these cases.

—I am, etc.,

Swansea. W. O. WILLIAMS.

POINTS FROM LETTERS

Inflation of Lungs During Bronchoscopy

Dr. R. A. C. HERRON (Zürich) writes: I was most interested to read the article by Dr. R. Bryce-Smith (December 22, 1951, p. 1517) on inflation of the lungs during bronchoscopy. I was also gratified that this method should receive the wide publicity it deserves, as I have been using it for three years and know the obvious advantages, especially in safety for the patient (*Anaesthesia*, 1950, **5**, 40).

Invite Russian Doctors

Dr. JOHN PEMBERTON (Sheffield) writes: I would like to support Dr. R. L. Kitching's suggestion (December 1, 1951, p. 1331) that the British Medical Association should invite a group of Russian doctors to this country. In addition to leading to a valuable exchange of medical knowledge and experience, such a visit might contribute a little towards a better understanding between our two countries, especially if our Russian colleagues were willing to stay with us in our homes.

Tissue Reactions to Protein Sensitization

Dr. J. T. SHIRLAW (Wigan) writes: "The eosinophil cell is worthy of greater attention," says Professor J. R. Squire in his lecture (January 5, p. 1). So far, the following facts have been established: Allergy is accompanied by an eosinophilia; the administration of cortisone brings about an eosinopenia together with a dramatic improvement in allergic diseases; cortisone lessens cell growth activity. Surely these are points of great significance. I do not agree that the "eosinophil may be regarded as being concerned with 'mopping up' the products of tissue damage"; the eosinophil only appears on the scene when the mopping-up process has been finished by other leucocytes and should therefore be regarded more as a repair cell. . . .

Correction

In the paper on "Tuberculous Meningitis," by Dr. S. Russell Jamieson (January 12, p. 83), in Case 29 (p. 84, column 2) the dosage of vitamin B_{12} employed was given as 10 mg. This was a thousand times too high, and should read 10 μg.

Obituary

H. S. RAPER, C.B.E., D.Sc., M.B., F.R.C.P., F.R.S.

The death of Professor H. S. Raper on December 12 was recorded in the *Journal* of December 22 (p. 1527). We are indebted to Professor A. Wormall for the following appreciation.

The passing of Henry Stanley Raper removes from the ranks of British biochemists one of its most distinguished and revered leaders. His approach to medicine was basically scientific, for he was a member of that small but remarkable group of chemists who qualified in Leeds in the early 1900's and subsequently branched out and helped to develop the new chemical offspring, biochemistry. This group included Dakin, Dudley, Hartley, Raistrick, and Raper, and all have made contributions which will for ever keep their own names, and indirectly the name of their chemistry mentor, the late Professor J. B. Cohen, permanently recorded in the annals of biochemistry.

Raper's main interest was undoubtedly in medical science, but he was above all a magnificent chemist. His whole approach to physiology and medicine was through chemistry and biochemistry, and, although for the greater part of his career he was professor of physiology at Leeds or Manchester or Dean of the Faculty of Medicine at Manchester, he never lost his keen interest in all branches of biochemistry. His ability to keep up to date, and indeed to anticipate many new developments, made him the ideal adviser-in-chief. Chemists, biochemists, physiologists, bacteriologists, pathologists, and clinicians came to him with their problems, and rarely, if ever, did they go away without at least one and usually several valuable constructive ideas.

His biochemical interests were wide. With Leathes he wrote an excellent classical monograph on fats, and during the 1914–18 war he made valuable contributions in the field of chemical warfare defence. He was one of the first in this country to use (in 1936) deuterium in the study of the absorption and metabolism of fats, and in recent years, in collaboration with Harper, he made important advances in our knowledge of the hormone pancreozymin. It is probable, however, that he will best be remembered in biochemistry for his classical work on tyrosinase, an oxidizing enzyme responsible for producing much of the dark-brown and black pigmentation of many animals. It is to Raper and his colleagues that we owe our knowledge of the precise chemical changes involved in the enzymic conversion of the amino-acid tyrosine into the red pigment hallachrome which is the precursor of melanin.

His happiest working hours were spent in his laboratory. "How delightful it is," he wrote to me early in January, 1945, "to be able to use one's hands and do a few experiments. But alas, term begins on Tuesday with its lectures, committees, interviews, and what not, so it will only be in odd hours that I can escape to the laboratory bench." Many biochemists will regret that with his almost unrivalled skill as a biochemist he was given so little opportunity to develop the work he loved, but we may be happy in the knowledge of the major contribution he made in the wider field of medical education and in the organization of medical research. His friends, and they were truly legion, will treasure the memory of "a verray parfit gentil knight," in stature and in deed.

R. FOSTER KENNEDY, M.D., F.R.S.ED.

Dr. Robert Foster Kennedy, professor of neurology at Cornell University Medical College, and formerly president of the American Neurological Society, died in New York on January 7, aged 67.

Robert Foster Kennedy was born in Belfast. His grandfather was Professor R. F. Dill, and one of his cousins was the late Field-Marshal Sir John Dill. He