Correspondence

A Gift to the R.M.B.F.

SIR,—I am anxious to acknowledge through your columns the receipt of a donation of £647 18s. 10d. from Messrs. Burroughs Wellcome and Co. (the Wellcome Foundation Ltd.). This gift has been made by the very kind and generous thought of their directors in transferring the payments for the 1946 issue of the Wellcome Medical Diary to this fund. On behalf of my committee I desire to express our warmest thanks to Messrs. Burroughs Wellcome and Co. and also to assure all who made payments for the diary that this gift of £647 18s. 10d. will be the means of bringing some extra measure of comfort and ease to many of our beneficiaries.—I am, etc.,

London, S.W.15.

C. LUTHER BATTESON,
Hon. Treasurer.

The "Costoclavicular Syndrome"

SIR,—The instructive article by Prof. E. D. Telford and Mr. S. Mottershead (March 15, p. 325) on the "costoclavicular syndrome" prompts me to mention a cause of paraesthesia in the arms and hands of middle-aged men to which I have not seen any reference. Two cases have come to my notice in the last few years. The paraesthesia is caused by wearing a shirt of thick material with the sleeves rolled up to the axilla. From the causation it will be apparent that the symptoms are worse in cold weather and out of doors—i.e., when a thick overcoat is worn in addition. The mechanism is similar to that of the fakir's trick of stuffing a handkerchief into his armpit and stopping his radial pulse at will by pressing his arm closely to his side.

An interesting point in my two cases is that neither man would wear his shirt sleeves unrolled. When seen a year or so later they were free from symptoms, but each informed me that he could not bear long shirt sleeves and had cut them off.—I am, etc.,

Preston, Lancs.

R. SLATER.

SIR,—Amidst a mass of inconclusive literature on pain in the arm the article by Prof. E. D. Telford and Mr. S. Mottershead (March 15, p. 325) stands as a beacon of clear thinking founded on careful experiment and is only equalled by the summary of the literature in your editorial. It would appear from these articles that the key to the condition lies in the spasm of the scalene muscles, and it has long been held by the osteopaths that this spasm is initiated reflexly from a subluxed cervical joint. The strain of the cervical joint is maintained by, and is secondary to, a subluxation in the upper dorsal area. This may result from single or multiple trauma with the neck in side-bending rotation, such as may follow from a blow, or lifting bulky articles off a shelf above the head.

Manipulation of the cervical area frequently relieves the symptoms, but it is essential to obtain good movement of the upper dorsal subluxation in order to produce a permanent result. While a compression of the median nerve at the wrist may be a factor in certain cases, it is felt that most of these lesions are central in origin.—I am, etc.,

London, N.W.1.

W. HARGRAVE-WILSON.

Spirillum (Abacterial) Pyuria

SIR,—I was very interested to note in the article on "Abacterial Pyuria" by Drs. W. E. Coutts and R. Vargas-Zalazar (Dec. 28, 1946, p. 982) that a spirillum had been found in the centrifuged urinary deposit of the five cases presented.

During the last month I have had the opportunity of seeing a further case of abacterial pyuria. A centrifuged sample of the urine was examined under dark-ground illumination and was seen to contain numerous spirillum-like bodies, 5–10 per field, among the pus cells, length 5 μ , all of a similar shape, containing one or two regular curves along their length. This spirillum was also observed by a medical colleague and our sergeant special treatment orderly. The patient had no obvious dental sepsis or infected teeth. There was no radiological evidence of

a tuberculous focus in either lungs or renal tract. An intravenous pyelogram revealed no abnormality of the renal tract. Treatment was commenced with the following course of neosarsphenamine: on Feb. 7, 9, 11, 0.3 g. N.A.B., and on Feb. 19, 0.6 g. N.A.B. (total, 1.5 g.). There was an immediate diminution in the urinary frequency. After the second N.A.B. the urinary frequency was normal. On Feb. 17 the urine had almost cleared and on Feb. 19 the urine was perfectly clear.—I am, etc.,

A. B. FIELDSEND,
Major, R.A.M.C.

C.M.F.

Congenital Malaria

SIR,—With reference to the correspondence in the columns of the *British Medical Journal* during the early part of 1946 concerning congenital malaria, I should like to draw your attention to my article in the October–November, 1942, issue of the *East African Medical Journal*, 19, 223 and 247, in which I gave figures of infants who were found to be suffering from malaria at birth. The blood was taken from the baby's finger on three consecutive days following its birth, and out of 700 patients examined 40 mothers were found to have malaria parasites in the placental blood, and 2 infants.

These figures are by no means so impressive as those quoted by Dr. R. McKay in the *East African Medical Journal* in 1933, 10, 246, or in a communication published in 1936 entitled "An Account of an Investigation into Malaria at Dar-es-Salaam," p. 17.—I am, etc.,

Nairobi, Kenya.

P. G. PRESTON.

Haematological Heresy

SIR,—Unfortunately I have only recently been able to read Dr. R. R. Bomford's most stimulating article (Dec. 28, 1946, p. 996) and I wonder if I might be permitted to make a few inexperienced comments. Should the haemosiderosis, the hyperbilirubinaemia and urobilinuria, and the histological evidence of active phagocytosis be regarded as conclusive proof of haemolysis? The latter, surely, can be found in the normal subject if it is looked for. The others could, I think, be adequately explained by the excess of formed, and perhaps partially formed, haemoglobin now having "nowhere to go" because of the maturation arrest at the haemoglobinizing stage in haematopoiesis. There is seldom an iron lack in pernicious anaemia, as in normal erythropoiesis iron is neither excreted nor deposited but enters the haemoglobin molecule. In an erythropoietic upset, however, the iron cannot be utilized and it becomes deposited and increases the iron store. Hence the haemosiderosis.

Hyperbilirubinaemia and urobilinuria go hand in hand and are constant findings in the haemolytic anaemias. Their presence alone, however, does not, I think, justify the haemolytic theory. Again, bearing in mind the fact that there is no deficiency of iron and that there is every stimulus for the formation of haemoglobin from the tissue anoxaemia consequent upon the low red cell count, it is not difficult to imagine non-utilized haemoglobin being (a) broken down to haematin and hence to bilirubin, or (b) itself having a maturation arrest in its synthesis, and possibly the porphyrin III being diverted into bilirubin or some such similar pathological metabolism occurring. This would still explain the raised excretion of porphyrin I in pernicious anaemia, as the stimulus for haemoglobin formation is still there and the synthesis, or para-synthesis, still occurs—perhaps even in excess. Nevertheless the porphyrins are being utilized, and therefore the faecal coproporphyrin I would be raised if the call for haemoglobin is increased.

Another aspect of the hyperbilirubinaemia is the question of liver damage. I think Rich excised somewhere in the region of 90% of a normal dog's liver before he managed to produce jaundice. Reversing this statement it would mean that the destruction of red cells would need to be nine times greater than normal at least before jaundice developed. The life of a red cell must vary tremendously, and Lichtwitz states that it should not be measured in time but in actions—"oxygen carries" as he calls them. It has been stated that an average figure is a "life" of 50 days. Normal destruction would then be roughly a 1/50 cell per day, or in total 100,000 per day. To bring about a jaundiced condition in a haemolytic anaemia, therefore, would require the destruction of 1,000,000 cells per day. I do not know exact figures, but suggest that the blood level is not lowered so acutely as this even in P.A. relapse. The characteristic lack of symptoms in P.A. suggests an insidious lowering. To produce the biochemistry of the jaundice in P.A. it seems that liver damage then might conceivably also play