antigens, believed by many to be viral, should receive considerable encouragement from the knowledge that immunity to them exists. In any case, the detailed characterization of these immunological factors themselves, and the possible isolation of specific immunoglobins and their use in future studies, may assist us in finally identifying the causative agent of the Burkitt tumour.

I should like to thank the patients for their willing co-operation in these studies, also the expatriate donors of blood. Dr. L. Luzzato assisted with the preparation of the plasma. I am grateful to the various members of the hospital staff whose assistance and co-operation were invaluable. Mr. M. O. Olumakin did useful secretarial work. This survey was supported in part by the Ella Lyman Cabot Trust and by a Senate Research Grant of the

University of Ibadan. Case 1 was reported at a Conference on the Chemotherapy of Burkitt Tumour under the auspices of U.I.C.C. in Kampala, Uganda.

V. ANOMAH NGU, M.S., F.R.C.S.,

Department of Surgery, University of Ibadan, Ibadan, Nigeria.

REFERENCES

- Burkitt, D. (1962). *Brit. med. J.*, **2**, 1019.
 — Hutt, M. S. R., and Wright, D. H. (1965). *Cancer (Philad.)*, **18**, 399.
 Chemotherapy of Burkitt Tumour (1966). Proceedings of a Conference held at Kampala, Uganda, under the auspices of the U.I.C.C., January 1966. To be published.
 Ngu, V. A. (1965). *Brit. J. Cancer*, **19**, 101.

Medical Memoranda

Peripheral Mercury Embolization Occurring during Arterial Blood Sampling

Brit. med. J., 1967, **1**, 347-348

The collection of peripheral arterial blood by an anaerobic technique is essential to many clinical procedures. Generally the blood is collected into heparinized syringes in which some inert fluid is placed to fill the dead space; mercury has been used as the inert fluid and cases of embolism of this mercury have been reported in America (Lathem *et al.*, 1954; Buxton *et al.*, 1965). We report here a further example of this misadventure occurring in Great Britain.

CASE REPORT

The patient, a 32-year-old physiologist, was taking part in an experiment which entailed collecting samples of arterial blood at

15-minute intervals over a two-hour period. In order to do this a Courmand needle was introduced into the left brachial artery in the antecubital fossa. Blood was collected in syringes with eccentric nozzles, the dead space in the syringe, about 0.3 ml., being filled with metallic mercury.

The needle was introduced without incident at 15.30 hours on 28 January 1966, and blood sampling began almost immediately. Throughout the experiment the subject was sitting upright with his left arm and the needle in it hanging by his side. During the experiment some "mechanical thrills" were felt in the left hand and fingers; these were presumed to be due to minor embolic episodes, and as this sort of phenomenon had been noticed in previous experiments no particular significance was attached to them.

After the experiment the subject noticed a rash developing on the dorsum of his hand, and at the same time the hand and the ulnar side three fingers became painful. The pain increased in intensity, and an x-ray film taken then revealed deposits of metallic mercury in the soft tissues of the middle, ring, and little fingers, the hypothenar muscles, and the medullary cavity of the proximal phalanx of the middle finger of the left hand, and in the soft tissues of the forearm (Fig. 1).

The patient was admitted to St. Thomas's Hospital on 29 January. He was then fully conscious but in considerable pain, and com-

FIG. 1.—X-ray film of left hand taken 20 hours after the accident. Metallic mercury is shown in the small blood-vessels.

plained of soreness and swelling in an area corresponding to the x-ray distribution of the mercury.

The skin of the hypothenar eminence and of the three affected fingers and the skin about the medial side of the left elbow showed a violet discoloration with some oedema and inflammation. All the pulses in the left arm and wrist were present and of normal volume. There was no evidence of any vascular or neurological abnormality found elsewhere on routine examination. X-ray examination revealed no mercury elsewhere in the body. Urinalysis revealed no proteinuria. Routine haematology showed a leucopenia (5,700/cu. mm.) and a thrombocytopenia (120,000/cu. mm.).

FIG. 2.—Mercury globules showing beneath the skin seven days after the accident.

FIG. 3.—Rise in platelet count 12 days after the accident. Blood mercury and urinary mercury values are also shown.

The case was treated as mercury poisoning, and accordingly dimercaprol was administered (2,875 mg. in six days). The dimercaprol caused much systemic upset, and was changed to D-penicillamine, 900 mg./daily for five days, and then N-acetyl-D-penicillamine, 900 mg./daily for five days. Analgesics, ampicillin, and tetanus toxoid were also given. The patient had a pyrexia up to 102.8° F. (39.3° C.) over the first 10 days in hospital.

The pain in the hand gradually lessened, and after six days globules of mercury became apparent beneath the epidermis in the affected fingers (Fig. 2); these globules subsequently ulcerated out. The discharge of metallic mercury has continued since this time.

Daily urinalysis showed microproteinuria on one day only, otherwise renal function remained normal throughout.

The level of mercury in the blood and in the urine was monitored from day to day (Fig. 3). The figures of the blood mercury ranged up to 12.5 µg./100 ml. on 29 January, but after this the level fell gradually, and on 18 February was 2 µg./100 ml. The quantity of mercury excreted daily in the urine ranged from 262 µg. on 31 January to 69 µg. on 18 February (Fig. 3).

Haematological investigations showed a thrombocytopenia and leucopenia, the platelet count being 120,000/cu. mm. on admission and gradually rising after the fifth day (Fig. 3). The leucopenia did not develop fully until 2 February, when the white blood count fell to 2,700/cu. mm.; it then gradually rose, reaching a normal level on 12 February.

COMMENT

This is a case of an avoidable misadventure. Previous similar accidents have been reported in the American literature, and this hazard has been mentioned in the *Lancet* (1965). A technique of arterial blood sampling which avoids this hazard of mercury embolization has been described by Buxton *et al.* (1965) and deserves to be more widely known.

The case described here showed systemic signs of mercury poisoning—namely, a thrombocytopenia and leucopenia, and a transient proteinuria. Fortunately there were no signs of permanent impairment of the haemopoietic or renal systems. Because of these transient signs of mercury poisoning we feel that treatment with chelating agents should be given in this type of case. The initial management with dimercaprol resulted in a high urinary excretion of mercury and a rapid fall in the blood mercury level.

We wish to thank Mr. R. W. Nevin, under whose care this patient was admitted, for permission to publish, and Professor W. I. Cranston, who advised us about treatment.

H. BRENDAN DEVLIN, M.A., M.D., M.CH., F.R.C.S., F.R.C.S.I.,
Senior Surgical Registrar.

MICHAEL SUDLOW, M.B., B.S.,
Lately Resident Pathologist.

St. Thomas's Hospital, London S.E.1.

REFERENCES

- Buxton, J. T., Hewitt, J. C., Gadsden, R. H., and Bradham, G. B. (1965). *J. Amer. med. Ass.*, **193**, 573.
Lancet, 1965, **2**, 1174.
Latham, W., Lesser, G. T., Messinger, W. J., and Galdston, M. (1954). *Arch. intern. Med.*, **93**, 550.