

doctors and social workers seem aware, as a positive and beneficent force for the good of our patients, living or dying.—I am, etc.,

W. R. MOORE

Enfield, Middlesex

Anthrax

SIR,—The article on anthrax (20 January, p. 157) by Dr. Robert Lamb was most disappointing. He refers frequently to the danger of infection from bone meal, but hardly mentions that hides, and hence tanneries, are an ever-present source of infection and patients. In spite of the increasing substitution of plastic materials, a large amount of leather is still used in this country, and since we are not self-sufficient in hides, many have to be imported. One tannery in Leeds when I was working there imported large numbers from Indonesia. The dust from these hides was always teeming with anthrax spores, for hides cannot be sterilized at the docks, like wool and hair. Dr. Lamb rightly stresses that in this country by law a dead infected animal has to be burnt in situ without postmortem examination. But in countries like Indonesia, even if such a law existed, a dead cow lying in the fields would be soon torn apart by vultures and the blood and tissue fluid would soak into the ground to form a long-lasting reservoir of contamination for the hides of other animals.

It is not true to say that there was no other treatment save excision and local phenol therapy before antibiotic days. The use of Sclavo's serum was well described in the surgical textbooks of the 1920s. The organic arsenicals such as nearsphenamine were specific and were a godsend in the tropics, where, in the absence of refrigeration, supplies of serum were non-existent; I know of one man in Nigeria who owes his life to nearsphenamine.

The important thing is to expect the disease; the golden rule should be that any worker in a tannery or a bone meal factory with a septic pimple has anthrax until proved otherwise. Any good laboratory can give an answer on a smear within half an hour and on a culture within 24. As soon as the bacteriologist has taken his specimens a massive dose of penicillin should be given, thereby preventing the rapid decline of a patient with an annoying septic pimple into the septicaemic patient in extremis so well described by Dr. Lamb.—I am, etc.,

M. ELLIS

Heysham, Lancs

Lead Poisoning from Contaminated Opium

SIR,—We wish to record two cases of chronic lead poisoning due to the ingestion of contaminated opium. So far as we know this is the first report of a most uncommon cause of lead poisoning.

The first case was that of a 40-year-old Chinese woman with a two-month history of frequent attacks of severe lower abdominal pain. Her haemoglobin concentration was 9.8 g/100 ml, her reticulocyte count was 5%, and her white blood cell count was normal. A peripheral blood film smear showed some punctate basophilia. The urine was positive for coproporphyrin and the 24-hour urinary lead excretion was 0.26

mg/l. After exhaustive questioning and a visit to the patient's home had failed to disclose a possible source of lead intoxication, she disclosed that she had been an opium addict for the past 10 years—firstly, as a smoker and then, for the past four years, as an eater of opium. She brewed raw opium, obtained illegally, for several hours in a large metal pot in preparation for consumption. Analysis of this prepared opium showed 33.8 mg of lead per 100 g of opium. Analysis of scrapings from the inner surface of the metal pot showed 154 parts of lead per million.

The patient was treated with intravenous 10% calcium gluconate injections and twice-daily intravenous infusions of 500 mg calcium disodium versenate for five days. After three days the 24-hour urinary lead excretion was 2.6 mg/l. She has been lost to follow-up after discharge from hospital.

One month after the above patient was admitted to hospital her father, a 63-year-old unemployed Chinese man, presented with a two-week history of abdominal pain. His haemoglobin was 8.9 g/100 ml, reticulocyte count 3%, and the peripheral film showed punctate basophilia. The urine was positive for coproporphyrin and the 24-hour urinary lead excretion was 1.5 mg/l. Like his daughter, the patient had been ingesting the same home-prepared opium for the past 4 years. He improved with intravenous calcium lactate and calcium disodium versenate treatment.

Unusual and exotic causes of chronic lead poisoning in Singapore have been described. Hawes reported¹ lead poisoning in Chinese girls from face powders apparently made in China and containing lead carbonate. Danaraj reported² six cases of lead poisoning from Chinese folkore medicine adulterated with lead oxide. But so far as we know these two cases of chronic lead poisoning due to ingesting opium contaminated with lead are the first to be reported.—We are, etc.,

B. L. CHIA
CHUA KIT LENG
FENG PAO HSIEH
M. H. L. YAP
Y. K. LEE

Medical Unit,
Thomson Road General Hospital,
Singapore

¹ Hawes, R. B., *Malayan Medical Journal*, 1930, 5, 69.
² Danaraj, W., *Proceedings of the Alumni Association, Malaya*, 1954, 7, 261.

Hepatoma and Hepatitis-associated Antigen

SIR,—It was with great interest that we read the report by Dr. E. C. Campion and others (21 October, p. 149) of a case of hepatoma associated with hepatitis-associated (Australia) antigen in a white woman. Alpert and Isselbacher¹ failed to find Australia antigen in 31 cases of histologically proved hepatoma in whites born and raised in the U.S.A.

We have recently treated a 68-year-old white male who came in with a history of cirrhosis of the postnecrotic type and pain in the right upper quadrant. He was found to have Australia antigen and alpha-fetoprotein in his serum, a slight elevation of his serum alkaline phosphatase and aspartate aminotransferase (SGOT) levels, and a defect on liver scan. There was no history of hepatitis, exposure by any means to any hepatitis patient, or raw seafood ingestion

for several years. On biopsy of his liver mass he was found to have hepatocellular carcinoma, and he has responded nicely to 5-fluorouracil infusion.

We feel that this patient probably had anicteric hepatitis due to Australia antigen which progressed to a postnecrotic cirrhosis and eventually to the development of hepatoma. Unfortunately, we have no serum specimen from before his admission to our institution, but the postnecrotic cirrhosis had been documented at another hospital six months previously.

JAY F. ZIEGENFUSS
ARTHUR J. WEISS

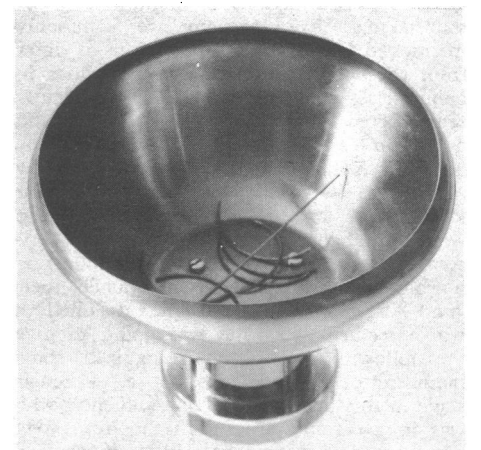
Department of Medicine,
Thomas Jefferson University,
Philadelphia,
Philadelphia, Pennsylvania, U.S.A.

¹ Alpert, E., and Isselbacher, K. J., *Lancet*, (1971), 2, 1087.

Magnetic Needle Dish

SIR,—Most operations involve some sort of suturing, with the consequence that surgeons have to dispose of the used needles as the operation proceeds. In so doing, needles are apt to go astray by becoming entangled in a swab or a pack or by bouncing off the table on to the floor; operating theatre staff are then diverted from their normal tasks to grovel about the floor seeking for the errant needles. At present there appears to be no organized drill for the collection and tally of needles during an operation, and this absence of system needlessly complicates accounting for the needles at the end of the procedure in the swab and instrument count. Again, needles discarded haphazardly by a surgeon may be difficult to distinguish among the instruments and other paraphernalia of an operation, making it tedious for the theatre sister to keep track of them.

These problems can be reduced by the use of a stainless steel dish 6 in (15 cm) in maximum diameter to the under surface of which a permanent magnet is attached (see fig.). In use the dish, sterilized by auto-



claving, is placed strategically in the operating field or on the Mayo instrument tray, conveniently available to both surgeon and theatre sister. Needles can then be discarded by being placed, dropped, or thrown on to the dish where they remain magnetically attached without the risk of bouncing off and being lost.

This instrument has been developed with the generous cooperation of Mr. R. Pickering of Polymatics Ltd., Tamworth, Staffs,