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 neoarsphenamine 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 neoarsphenamine.

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 folklore 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 HSII
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,

and is available from Down Brothers, Mayer and Phelps Ltd., Church Path, Mitcham, Surrey.—I am, etc.,

LAURENCE TINCKLER

Maelor General Hospital,
Wrexham

Treatment of Status Asthmaticus

SIR,—A noteworthy point on which I differ from the opinion expressed in your commendable leading article (9 December, p. 563) is in the statement that "physiotherapy has no place in the treatment of status asthmaticus." It has been our custom during the past three years in this hospital (which is part of Helsinki University Central Hospital), in addition to other routine treatment in cases of status asthmaticus, to facilitate the patient's breathing by manual pressure on the lower part of his chest during

This position is the most beneficial in status asthmaticus for movement of the diaphragm if the patient is still conscious or even semi-conscious.

every expiratory phase (see fig.). Measurements (performed with a Spirostat flow meter) show that this method expels 100-200 (or even 300) ml more air from the lungs than would happen spontaneously. In my opinion this respiratory help has often been decisive. It was used in 1972 in the management of 153 patients with status asthmaticus, none of whom died.

We have also diverged from the choice of drugs recommended in your article. For instance, we administer methylprednisolone intravenously instead of the hydrocortisone preparation suggested by you. This prevents retention of fluid and hypokalemia, while the antiallergic effect is at least equally good. I would like to point out, too, that ordinary adrenaline is often a more efficacious drug for asthma than beta-2-sympathomimetic preparations.—I am, etc.,

KLAUS A. J. JÄRVINEN

Hospital for Allergic Diseases
Helsinki, Finland

Viral Infection and Renal Transplant Rejection

SIR,—Dr. J. D. Briggs and others (2 December, p. 520) reported acute rejection of transplanted kidneys in three patients infected with influenza A virus and suggested that this might be due to a generalized stimulation of the host's immune response. I should like to suggest another mechanism

by which the virus may have caused rejection.

Influenza A virus has long been known to agglutinate erythrocytes.^{1,2} After a short time the erythrocytes separate and cannot be agglutinated a second time. This agglutination is due to the virus attaching itself to receptor sites on two different red cells and linking them together. The cell receptor sites are glycoproteins containing sialic acid, probably in a terminal position³ which is removed by a potent neuraminidase possessed by the virus, thus allowing the cells to separate once again.^{4,5} Neuraminidase-sensitive receptors have been found on other cells.⁶⁻⁸

When sialic acid is contained in an antigen it is very rarely antigenic itself. Indeed, it seems that sialic acid may serve to conceal more potent antigens and prevent them from stimulating an immune response, for treatment of many types of cell with neuraminidase has been shown to render them more highly antigenic.⁹⁻¹³ There is a possibility, therefore, that influenza virus may cause acute rejection of a renal transplant by removing terminal sialic acid groups from glycoprotein molecules in the renal cells and rendering them highly antigenic. If this hypothesis is correct there should be no danger of causing acute rejection by vaccinating against influenza so long as the virus is inactivated in such a way as to render it incapable of digesting sialic acid from renal cells.—I am, etc.,

W. T. MORRIS

Royal Postgraduate Medical School,
London W.12

- 1 Hirst, G. K., *Science* 1941, **94**, 22.
- 2 Hirst, G. K., *Journal of Experimental Medicine*, 1942, **76**, 195.
- 3 Wintzler, R. J., *International Review of Cytology*, 1970, **29**, 77.
- 4 Cohen, A., in *Mechanisms of Virus Infection*, W. Smith, London, Academic Press, 1963.
- 5 Gottschalk, A., *Physiological Reviews* 1957, **37**, 66.
- 6 O'Connell, C. J., and Milgrom, F., *Proceedings of the Society for Experimental Biology and Medicine*, 1966, **122**, 1255.
- 7 Peleg, B. A. and Iancu, M., *Nature*, 1966, **211**, 1211.
- 8 Pepper, D. S., and Jamieson, G. A., *Nature*, 1968, **219**, 1252.
- 9 Springer, G. F., *Bacteriological Reviews*, 1963, **27**, 191.
- 10 Currie, G. A., and Bagshawe, K. D., *British Journal of Cancer*, 1968, **22**, 843.
- 11 Kemp, R. B., *Nature*, 1968, **218**, 1255.
- 12 Bagshawe, K. D., and Currie, G. A., *Nature*, 1968, **218**, 1254.

Immunological Responses in Pregnancy

SIR,—The letter from Dr. P. Brain and others (25 November, p. 488) drew my attention to an earlier article by Dr. Ronald Finn and others (15 July, p. 150) reporting reduced T-cell activity during pregnancy. They found that the number of circulating T lymphocytes capable of responding to stimulation with phytohaemagglutinin was reduced in pregnant women by about 10-20% and that the incidence of negative tuberculin reactions was increased by about 64%.

Quite apart from the fact that some other authors have failed to find a deficiency in T-cells during pregnancy,^{1,2} the depression of tuberculin reaction could perhaps be attributed to hormonal changes in the course of pregnancy. In view of the most marked increase of oestrogen production it should be mentioned that although oestrogens have been shown to depress humoral antibody responses³ and cause involution of the thymus⁴ there is no conclusive evidence that

they affect peripheral immunologically competent cells,⁵ and their effects on various cell-mediated immune reactions are rather controversial.⁶ The possibility should be considered, however, that reduced tuberculin reactions during pregnancy could be the result of non-specific actions of oestrogens. There is evidence that these hormones alter the physicochemical properties of the ground substances of connective tissue⁷ and it has been reported that they restrict the spreading of materials and fluids in the skin.⁸ In fact, the reduced tuberculin reaction of oestrogen-treated animals has been attributed to this mechanism rather than to depression of immunological reactivity.⁹—I am, etc.,

V. S. ŠLJIVIĆ

St. Mary's Hospital Medical School,
London W.2

- 1 Brain, P., Marston, R. H., and Gordon, J., *British Medical Journal*, 1972, **4**, 488.
- 2 Watkins, S. M., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1972, **79**, 990.
- 3 Warr, G. W., and Šljivić, V. S., *Experimentia* 1972, **28**, 1356.
- 4 Dougherty, T. F., *Physiological Reviews*, 1952, **32**, 379.
- 5 Šljivić, V. S., and Warr, G. W., *British Journal of Experimental Pathology*, 1973. In press.
- 6 Šljivić, V. S., and Warr, G. W., *Periodicum Biologorum (Zagreb)*, 1973. In press.
- 7 Hytten, F. E., and Leitch, I., *The Physiology of Human Pregnancy*, 2nd edn. Oxford, Blackwell, 1971.
- 8 Sprunt, D. H., McDearman, S., and Raper, J., *Journal of Experimental Medicine*, 1938, **67**, 159.
- 9 Lurie, M. B., *Annals of the New York Academy of Sciences*, 1950, **52**, 1074.

Role of Pacing after Myocardial Infarction

SIR,—The mortality figures quoted by Dr. Helen H. Tucker and others in their study (6 January, p. 10) seem to provide some statistical evidence that electrical pacing of patients who develop complete heart block following a myocardial infarction may, in most instances, not be necessary. In view of their findings a controlled study of the problem might appear useful, but provided the necessary skills and facilities are available, few would dispute the advantages of pacing over an isoprenaline infusion in a patient who, at that very time, is at greatest risk of developing a dangerous arrhythmia. It would be of interest to know the reasons why the authors decided to adopt the non-pacing policy in their patients.

The role of pacing in preventing ventricular arrhythmias is certainly not confined to patients with heart block. Its value in the control of recurrent ventricular tachycardia and fibrillation not responding to drug therapy is not emphasized in most reports. Although these refractory dangerous arrhythmias often denote a poor prognosis because of underlying severe myocardial damage, a very gratifying response can sometimes be achieved with a combination of pacing and antiarrhythmic drugs, as two cases recently treated at this hospital demonstrate.

The first patient, a man of 56, suffered within a period of 24 hours from eight episodes of ventricular tachycardia, going on rapidly to ventricular fibrillation, two weeks after an extensive myocardial infarct. In spite of combined administration of all antiarrhythmic drugs available, the dangerous arrhythmias continued unabated until the intrinsic heart rate was over-paced to a rate of 120-130/min so that all ventricular ectopic beats could be suppressed. The pacemaker catheter was removed a week later, sinus rhythm has continued since discharge from hospital, and normal life has been resumed.