only one survivor has been reported from haemorrhage into the peritoneal cavity.

Several surgical operations have been tried for this condition,³ such as excision of the aneurysm followed either by end-to-end suture of the hepatic artery or by anastomosis of the proximal end of the splenic artery (after splenectomy) with the distal end of the hepatic artery.

We are grateful to Mr. J. Withycombe for permission to publish this case.

—We are, etc.,

T. F. MORTIMER.
G. A. GRESHAM.

Department of Pathology,
University of Cambridge.

REFERENCES

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- ² Dwight, R. W., and Ratcliffe, J. W., *Surgery*, 1952, 31, 915.
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Atypical Responses to Nortriptyline

SIR,—We have now observed two patients suffering from anxiety-depressive reactions who, having been given nortriptyline medication, have rapidly developed acutely depressed and tense states with pressing ruminations on death in general and suicide in particular.

In the first case the nortriptyline was prescribed by the family doctor, with reassurance, and the patient was afterwards admitted to hospital. Cessation of nortriptyline medication produced rapid improvement. In the other case medication was changed from amitriptyline, and after an interval of five days nortriptyline (Aventyl) was prescribed, and a rapid onset of an intensely painful depressive swing with morbid preoccupation with death and suicide occurred. After her family doctor had returned her to amitriptyline medication these symptoms ceased abruptly.

The nortriptyline in both cases was prescribed with encouragement, and both doctors and patients expected a good outcome. It seems that these symptoms may well be atypical physiological responses to the drug.—We are, etc.,

JOHN C. N. TIBBITS.
Hollymoor Hospital,
Birmingham 31.

P. S. IRELAND.

Ampicillin for Typhoid Carriers

SIR,—Dr. A. B. Christie (3 October, p. 876) may be interested in our recent experiences with a typhoid carrier receiving ampicillin prior to cholecystectomy.

This lady, who had positive faecal cultures and a non-functioning gallbladder containing numerous calculi, received ampicillin 1 g. 6-hourly for three weeks before operation. During this period, her faecal cultures became negative and her serum ampicillin level was observed to be 5 µg./ml., 285 minutes after the last oral dose. A specimen of duodenal aspirate contained ampicillin in a concentration of 4 µg./ml. The minimal inhibitory concentration of ampicillin for the patient's strain of *Salmonella typhi* was 0.6 µg./ml. Bile from the excised gall-bladder contained ampicillin in a concentration of 16 µg./ml., and routine cultures of this and crushed calculi were negative. When these were repeated in the presence of 15,000 Levy units of penicillinase, *S. typhi* was recovered from all samples of bile and from numerous calculi. In an attempt to explore the effect of ampicillin on the patient's organism *in vitro*, 1.2×10^8 viable cells were added to sterile broth containing 20 µg./ml. ampicillin in a manner similar to that described

by Rolinson and Stevens.¹ At three hours the viable count was 7×10^4 , at seven hours 5×10^4 , and at twenty-four hours 3×10^4 .

This observation, together with the recovery of viable typhoid bacilli from bile containing high concentrations of ampicillin (and not merely from calculi), confirms the need for caution in assessing the value of this antibiotic in the treatment of chronic carriers.—I am, etc.,

K. F. ANDERSON.

Salmonella Reference Laboratory,
Adelaide,
South Australia.

REFERENCE

- ¹ Rolinson, G. N., and Stevens, S., *Brit. med. J.*, 1961, 2, 191.

Treatment of Acute Poisoning

SIR,—Your recent article on Treatment of Acute Poisoning (10 October, p. 927) considers various measures to remove poison which has gained access to the body. There is considerable experimental evidence to suggest that spontaneous emesis is more effective in the removal of poison than gastric lavage.¹ The considerable experience with poisoning at the Boston Poison Information Center as well as elsewhere has convinced us that Syrup of Ipecac. is the most effective means of producing spontaneous emesis.² We now recommend that when the substance ingested is not a corrosive, hydrocarbon, or if the patient is not unconscious or convulsing, he be given 15 ml. of Syrup of Ipecac. (one tablespoon). The patient then is given something to drink, and vomiting occurs in the vast majority of cases within 15 to 20 minutes. This treatment can be given in the home if the doctor has prescribed a small bottle of Syrup of Ipecac. for the medicine cabinet, and by its prescription, we hope, has practised accident prevention. It is also our experience, and that reported recently by Dr. William Robertson, that mechanical attempts to produce vomiting are generally unsuccessful.

We also agree with your writer that emesis should be induced even if the substance was ingested as long as eight hours previously.—I am, etc.,

JOEL J. ALPERT.

Boston Poison Information Center,
Boston 15, Mass., U.S.A.

REFERENCES

- ¹ Arnold, F. J., Hodges, J. B., and Barta, R. A., *Pediatrics*, 1959, 23, 286.
- ² Robertson, W. O., *Amer. J. Dis. Child.*, 1962, 103, 136.

Safety with Parathion

SIR,—Parathion, an organophosphorus insecticide, has been used for the control of *Quelea quelea quelea* (the Sudan Dioch) for a number of years. It is important not only to have well-designed safety precautions but also to ensure that they are observed.

An anti-quelea campaign was started in the north-eastern area of Northern Nigeria using aerial spraying of parathion. The parathion used was 50% technical parathion diluted with diesel oil 1:1. Six weeks after the spraying started the aircraft crashed and the pilot was severely contaminated with parathion. The pilot was found within 20 minutes of the crash, and by that time he had stripped off his clothes and had washed himself in a small stream. One ampoule of atropine (1/100 gr.—0.65 mg.) was given

intramuscularly at the time he arrived at the base camp. The pulse rate at this time was 45 per minute. The pilot was apparently normal and made no complaint. He talked normally with the engineer about the crash and had coffee and his supper. Three hours after the crash he complained of pain in the eyes and some disturbance of vision and very soon after this began to perspire heavily and to vomit. Within 40 minutes the pilot began to have convulsions. His pulse was reported at this time as 30 per minute. His breathing stopped and artificial respiration was started and atropine (1/100 gr.) was given by intramuscular injection. The convulsions and paralysis of respiration continued and the pilot died five hours after the crash in spite of continuous artificial respiration. It was recorded that there was an excessive production of saliva and bronchial secretions at the time of the onset of the convulsions.

The safety regulations laid down that full protective clothing be worn by the pilot and by those handling insecticide and laid down the precautions to be taken in handling. The pilot was not allowed to fly through the insecticide spray mist on turning and was required to have eye protection (goggles or eye shield), and nose and throat protection from a mask made from several layers of gauze which were changed every second flying hour. It was further laid down that cholinesterase examinations for all staff handling or coming into contact in any way with parathion be made every two weeks, from four weeks before the start of the spraying operations to four weeks after the cessation of the spraying operations.

After the accident it was found that the standing orders had been followed with the exception of that part of the standing orders concerning the cholinesterase examinations. Only one cholinesterase examination was done and that four weeks after the start of the spraying and ten days before the fatal accident, when he was found to have a cholinesterase level of 50%, and 62.5% on retesting. If a level of between 75% and 50% was recorded and confirmed on retesting, the standing instructions laid down that overexposure was probable and suspension from work for two weeks should be made.

It seems likely that the crash was due to a minor acute intoxication with parathion superimposed on a chronic intoxication and that the resulting incoordination was responsible for the crash. The treatment given was not intensive enough and the dose of atropine was negligible. Organophosphorus insecticides are, like all pesticides, potentially dangerous if misused or handled carelessly. Some of this group, especially parathion, are extremely poisonous and are liable to be lethal if mishandled. Medical care should be immediately available—this is important in a field campaign in areas far away from a hospital or doctor. If poisoning occurs treatment must be prompt: the two specific antidotes, atropine and an oxime (such as pralidoxime) must be given and repeated until symptoms cease. The patient must be observed carefully for 48 hours. Evacuation by road is to be avoided until recovery is assured: this implies the ability for treatment to be completed on the spot. With the necessary drugs and a simple machine for achieving passive ventilation of the lungs and the requisite professional skill, this is not too difficult.

In this present case the degree of contamination was such as likely to be lethal in any case, except possibly if skilled professional attention had been available on the spot. However, inattention to standing orders with regard to the routine cholinesterase examinations occurred, and this was very probably a contributory factor. The practice of preventive medicine advances as

much by attention to the details of failure as to the details of success, and it is in an attempt to avoid a future tragedy from the same causes that this report is made.

I am grateful to Dr. R. A. B. Dikko, Permanent Secretary, Ministry of Health, Northern Nigeria, for permission to publish this letter.

—I am, etc.,

Regional Malarial Unit,
F.M.B. 2106,
Kaduna, N. Nigeria.

J. S. DODGE.

Hookworm Anaemia

SIR,—I have read with great interest your leading article on hookworm anaemia (14 November, p. 1216).

I would like to make one comment about your statement, "These larvae develop only if the faeces of such persons contaminate warm moist earth, and normal sanitary arrangements—and often the weather—in Britain preclude this." In Oxford, Dr. S. C. Truelove and myself have carried out some experiments on faeces infected with hookworm ova.¹ In one of these experiments a part of the infected faeces was covered with moist earth in a flower-pot and left in the open (the garden of the Radcliffe Infirmary), under a classic English weather in June and July for six weeks. We were able to identify three stages of hookworm larvae, of which the third one was a typical filariform infective larva.

We have pointed out that such infection can occur in Great Britain if bad sanitary conditions are allowed to exist, particularly among immigrants. We have also drawn the attention of the public health authorities and the physicians in this country to be aware of the condition, emphasizing the point that the English weather is not a strong barrier against the spread of the disease.—I am, etc.,

S. N. SALEM.

Nuffield Department of Clinical
Medicine,
The Radcliffe Infirmary,
Oxford.

REFERENCE

- ¹ Salem, S. N., and Truelove, S. C., 1964, in press.

Hodgkin's Disease Limited to the Liver

SIR,—It is well established that the liver may be involved in Hodgkin's disease. However, Hodgkin's disease affecting the liver alone is very rare, and the following case was therefore thought worth while reporting.

The patient, a 15-year-old girl, was first seen at St. Thomas's Hospital in October 1963 with a two-week history of malaise, night sweats, and slight weight loss. On examination at that time there were no physical abnormalities; she was afebrile, and investigations showed: Hb 85%, E.S.R. 3, chest x-ray normal. She was not seen again until November 1963, when she was admitted to the ward with a three-week history of swelling of the legs and abdomen. On examination she was febrile and had tense ascites. No intra-abdominal masses were palpable, but there was considerable ankle and sacral oedema. Investigations at that time showed slight anaemia and thrombocytopenia.

At this stage investigations were unhelpful and laparotomy was performed. At operation the liver and spleen were seen to be enlarged, the former having a mottled greivish appearance. There were no enlarged glands. Attempt at portal venography on the table was unsuccessful owing to technical reasons. A splenunculus and liver biopsy were taken for histology.

Microscopically the splenunculus was normal, but the liver showed round-cell infiltration predominantly in the portal tracts, with liver-cell necrosis. The histological picture at this stage did not show conclusive evidence of lymphoma, and the diagnosis rested between this and an inflammatory process.

Post-operatively she was given antibiotics and transfused with intravenous albumin, which temporarily raised her serum-albumin level (which had been only 2.9 g./100 ml.) to 4.3 g./100 ml. However, she remained unwell with swinging fever, jaundice appeared, and the ascites reaccumulated. Further investigations in December 1963 showed among other things: bilirubin 1.9 mg./100 ml., and alkaline phosphatase 85 units.

As she remained so ill the diagnosis of malignant reticulosis became more likely, and she was treated with prednisone, 20 mg. four times a day, and vinblastine, which had to be discontinued after two weeks owing to progressive leucopenia. The steroids had no effect on either her fever or liver-function tests. She died on 12 January 1964.

At necropsy the liver was grossly enlarged, all the hepatic tissue being replaced by yellowish firm tissue. The portal vein, hepatic vein, and inferior vena cava were all fully patent. The spleen was grossly enlarged with a deep purple cut surface. There were enlarged lymph-nodes along the aorta and pancreas.

Post-mortem histology showed evidence of lymphocytic and histiocytic infiltration with irregular giant cells and increase of fibrous tissue in both the glands and liver. The diagnosis was that of a malignant reticulosis allied to Hodgkin's disease.

Liver involvement is quite common in Hodgkin's disease,^{1,2} and indeed liver biopsy may be used to make the diagnosis.³ However, only two cases have been reported of Hodgkin's disease affecting only the liver.^{4,5} Possibly the liver as a primary site is more common than is realized, as by the time that most patients come to necropsy the disease has become widespread.

I am indebted to Dr. H. K. Goadby for permission to publish this case and to Dr. R. Kittermaster for reporting on the sections.

—I am, etc.,

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

C. A. LOEHRY.

REFERENCES

- ¹ Levitan, R., Diamond, H. D., and Craver, L. F., *Amer. J. Med.*, 1961, 30, 99.
² ———, *Gut*, 1961, 2, 60.
³ MacLeod, M., and Stalker, A. L., *Brit. med. J.*, 1962, 1, 1449.
⁴ Goia, I., *Sang*, 1935, 9, 367.
⁵ Symmers, D., *Arch. intern. Med.*, 1944, 74, 163.

Inheritance in Gaucher's Disease

SIR,—We have recently described¹ typical Gaucher cells in the bone-marrow of both clinically normal parents and a normal sister of two children with overt Gaucher's disease. This finding had, to our knowledge, previously not been reported. We have regarded our findings as an indication of heterozygosity (incomplete recessivity).

Meanwhile we have been able to examine parents and siblings of another patient with Gaucher's disease. This 17-year-old patient suffers from clinically and histologically proved Gaucher's disease and underwent splenectomy eight years ago. Parents, two sisters (9 and 11 years old), and one 16-year-old brother are clinically normal. In the bone-marrow of both parents and sisters typical Gaucher cells could be demonstrated. The bone-marrow of the brother was normal.

We therefore differ from the views of Klein and Franceschetti² but agree with Hsia's³

opinion that the recessive mode of inheritance is the most frequent one in Gaucher's disease.—We are, etc.,

H. GERKEN.

E. GRAUCOB.

Paediatric Department,
Christian-Albrecht's University,
Kiel, Western Germany.

REFERENCES

- ¹ Gerken, H., and Wiedemann, H.-R., *Ann. paediat. (Basel)*, in press.
² Klein, D., and Franceschetti, A., in *Handbuch der Humangenetik*, 1964, vol. IV, edited by P. E. Becker, Thieme, Stuttgart.
³ Hsia, D. Y. Y., Naylor, J., and Bigler, J. A., *New Engl. J. Med.*, 1959, 261, 164.

Another Television Hazard?

SIR,—“Emergency—Ward 10” had just finished when my 50-year-old patient developed sudden giddiness, while sitting in his viewing-chair. An hour later he was still chair-bound and afraid to move his head, as this increased his vertigo.

He was found to have a normal heart and blood-pressure. Abnormal findings included a phasic nystagmus, unsustained to the right, but sustained with a rotary component to the left; a poorly moving palate; an equivocal right extensor plantar response; and minimal incoordination of his right leg. There was definite loss of appreciation of pin-prick over his left face, right limbs, and trunk. These findings were felt to be strongly suggestive of vertebro-basilar insufficiency. He made a good recovery over the succeeding week.

Reconstructing the events prior to the incident, it was found that his chair was positioned at an approximate angle of 60° to the plane of the screen. This meant that for comfortable viewing a compensatory head rotation of some 30° was required. There was an additional tendency to extend the head over the top of the back-rest for greater comfort. One or both these factors may well have jeopardized the vertebral arterial flow in the manner shown by Brain.¹ The post-prandial state, modified by the presence of an old partial gastrectomy, may have provided the backcloth of temporarily reduced arterial flow to the brain against which the above postural factors played a critical role.²

A repetition should be less likely if the patient in future pays special attention to his head posture, assuming, of course, that he still cares for television.—I am, etc.,

London W.11.

KLAUS HEYMANN.

REFERENCES

- ¹ Brain, Lord, *Brit. med. J.*, 1963, 1, 771.
² Williams, D., *ibid.*, 1964, 1, 84.

Professional Patients

SIR,—Dr. T. L. Dunn's suggestion (3 October, p. 879), that a study be made of those who choose to play the role of patient in order to avoid the responsibilities expected from healthy people, is timely. Our experience leads us to conclude that such people are only a few of those who are not prepared to accept the task of fulfilling an adult role in society. We are also of the opinion that doubt need not be cast upon the choice being conscious by the qualification “almost.” Such manipulative behaviour is often considered “neurotic” and regarded as sickness by the medical profession, whereas it is behaviour designed to lead unsuspecting