Splenectomy and Susceptibility to Malaria and Babesia Infection

Sir,—The article by Dr. A. S. D. Spiers and others (25 January, p. 175) calls for special consideration concerning susceptibility to malaria and infection with some species of babesia in patients who have undergone splenectomy for haematological disorders.

Research work on apes and monkeys has shown that when these animals have acquired immunity to malaria they can be reinfected with the same strain of the same species of parasite if, before reinfestation, the spleen is removed. It has also been proved beyond all doubt that some species of apes (chimpanzees), which normally are resistant to Plasmodium vivax infections, develop severe parasitaemia if, before infection, the spleen is removed. Humans who have had their spleens removed should, if possible, avoid going to endemic malaria areas, especially areas where P. falciparum is prevalent. Few if any splenectomized Caucasians would survive for more than two weeks in a region with P. falciparum and other given antimalarial drugs within a few days of infection.

There have been fatal cases of babesia infection in nature among humans who have been travelling in both Europe and North America. As far as is known only one case (fatal) has occurred in the U.K. A few years ago an inshore fisherman who had lost his spleen developed fever while on holiday. On admission to hospital his blood was examined and numerous erythrocystic parasites were found which were diagnosed as P. falciparum. Antimalarial drugs were given but the patient did not respond and died. Blood films were sent to the Malaria Reference Laboratory, at Horton Hospital, Epsom, where it was recognized that the parasites were not malaria. If the films were shown to Professor P. C. C. Garnham he diagnosed Babesia divergens. 1 The parasites of this species of babesia so closely resemble P. falciparum that it takes an expert to distinguish them. Babesia infection (known to farmers as red water fever) among cattle is common in many parts of the U.K.; in one part of east Devon a farmer informed me that all his cattle had or later became infected. It therefore behoves farm workers, veterinary surgeons, and others who have had their spleens removed to avoid being bitten by ticks. It would certainly be advisable for people from the U.K. who have undergone splenectomy to go for holidays in tropical Africa.—I am, etc.,

P. G. SHUTE

Leatherhead, Surrey


Inhibition of Prostaglandin Biosynthesis by Analgesics in Relation to Asthma

Sir,—The demonstration by Dr. A. Szeczeklik and his colleagues of the in vitro inhibition of prostaglandin (PG) biosynthesis by aspirin (11 January, p. 67) is supported by our finding that during asthmatic attacks precipitated by aspirin only trace amounts of PGE and PGFα are present in the plasma. 1 However, the suggestion that the precipitation of asthmatic attacks in analgesic-sensitive patients is related to each analgesic's ability to inhibit PG synthesis should be accepted with reservation.

Though, in their experience, paracetamol did not inhibit PG synthesis it can be nevertheless of producing asthmatic attacks. Smith 2 observed marked falls in the FEV1 in five aspirin-sensitive subjects exposed to paracetamol. Similarly the increased airways obstruction observed in 13 aspirin-sensitive patients after I had challenged them with 1 g of paracetamol is shown in the table.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>FEV1% Before Challenge</th>
<th>FEV1% After Challenge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>49</td>
</tr>
<tr>
<td>2</td>
<td>84</td>
<td>69</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>52</td>
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<tr>
<td>4</td>
<td>71</td>
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<td>5</td>
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</tr>
<tr>
<td>8</td>
<td>91</td>
<td>77</td>
</tr>
<tr>
<td>9</td>
<td>81</td>
<td>64</td>
</tr>
</tbody>
</table>

It should be emphasized once more that patients with aspirin idiosyncrasy need not treat all analgesics with caution.—I am, etc.,

J. C. DELANEY

Wilton Hospital, Liverpool


Patient-satisfaction in General Practice

Sir,—The survey of patient-satisfaction carried out by Mr. P. F. R. Kaim-Caudle and Dr. G. N. Marsh in a Teesside practice of 3100 patients (1 February, p. 262) is a good example of how emphasizing certain aspects of statistical results can give rise to a misleading picture.

The authors are pleased at the high level of satisfaction shown in this practice, where half the visiting is done by the practice nurse. Approximately one-sixth of the adult practice population were interviewed by a non-medical research team and 92% were either "very satisfied" or "satisfied" with the treatment they received from their doctor. However, one respondent was "very dissatisfied," five were "dissatisfied," and 20 had "mixed feelings." If the sample is typical of the whole practice population this means that at least 7% of the respondents dissatisfied and between 100 and 150 more have mixed feelings about the treatment they receive from their doctor. Many general practitioners would be absolutely horrified if they knew that such a large number of their patients had such reservations about their treatment.

I do not think that this survey has helped to promote the doubtful 5 mg + hydrochlorothiazide (2400 patients is too small, and I await with interest the results of a similar survey in a practice with a list of 2000 patients where the doctor does his own visiting.—I am, etc.,

DENIS CRADDOCK

South Croydon, Surrey

Lithium Poisoning Precipitated by Diuretics

Sir,—The toxic effects of high serum lithium levels are well known, but their precipitation by diuretic medication, while recognized, is probably not so widely appreciated. The following short story illustrates the dangers of prescribing diuretics to patients taking lithium without careful alteration of dosage requirements.

The patient was a 66-year-old man with a past history of severe manic-depressive psychosis. His condition had been under control since 1972 and the serum level had been steady on regular estimations. In August 1974 he was started on Moduretic (amiloride 2 mg + hydrochlorothiazide 50 mg) because of left ventricular failure associated with hypertension and E.C.G. evidence of myocardial ischaemia. One week later he was referred to this hospital urgently because of unsteadiness on his feet, slurred speech, and shaking of the upper limbs which had become progressively more severe since taking the diuretic. There was no verifiable diplopia or paresthesia but he complained of general malaise, anorexia, nausea, and diarrhoea over the previous 5 days. There was a complex combination of gross movement of the upper limbs, a mixed peripheral tremor involving the hands, feet, stomach, and occasional flapping movements. There were no other abnormal findings on further neurological examination. His serum lithium level was found to be 2.4 mEq/l.

All medication was discontinued but two days later there was no clinical change. Four days after that there was a considerable improvement, but return to normal function was slow, and seven weeks after his deterioration there was still slight evidence of slurred speech, tremor, and ataxia.

The major points which should perhaps be stressed in this history are the rapidity of onset of the symptoms, the slow return to normal function, and the present problem related to persuading the patient to recommence a drug which he rightly believes could have killed him.—I am, etc.,

A. C. MACFIE

Academic Department of Psychiatry, Middlesex Hospital Medical School, London W.1

Amitriptyline and Imipramine Poisoning in Children

Sir,—Dr. D. A. Price and R. J. Postlethwaite (23 March, p. 575; 1 June, p. 304) have recommended the use of parenteral diphenylhydantoin in preventing and con-
trolling convulsions due to tricyclic anti-depressant poisoning. This suggestion was made on the basis of the beneficial use of this drug "in two children and two young adults poisoned with amitriptyline." The effectiveness of diphenhydantoin in this situation has never been clearly substantiated either in the laboratory or in clinical practice.

Recently we have been investigating the effectiveness and safety of various anti-convulsants in the prophylaxis of imipramine-induced convulsions in rats. We found that diphenhydantoin does not prevent seizures if given from 5 to 200 mg/kg failed to prevent convulsions brought about by intraperitoneal administration of imiprane 112 mg/kg. This was in contrast to the high anti-convulsant effectiveness of diazepam or phenobarbital in similar experiments. This observed ineffectiveness of diphenhydantoin in controlling imipramine seizures in rats is not surprising, since an important factor in the use of this drug as an "anti-convulsant" is the aetiology of the convulsions. For example, diphenhydantoin does not control febrile convulsions in children under 3 years of age nor is it effective in the treatment of seizures associated with barbiturate withdrawal. Moreover, diphenhydantoin does not raise the threshold to minimal electroshock seizure or prevent pentylentetrazol-induced convulsions. Our experiments suggest that diphenhydantoin may not have any value as an anticonvulsant in the therapy of tricyclic anti-depressant poisoning. However, this does not exclude the possibility that diphenhydantoin may have a use in controlling the cardiac arrhythmias associated with tricyclic antidepressant overdose.—We are, etc.

D. C. CARPENTER
A. R. BEAUBIEN
L. F. MATRIEU
Health Protection Branch,
Department of National Health and Welfare,

Myocellular Pneumonia with Inappropriate Secretion of Antidiuretic Hormone

SIR,—We were interested to read the report by Drs. A. Spanos and C. J. Spyri (28 September, p. 785) of inappropriate secretion of antidiuretic hormone (ADH) in a patient with chronic bronchitis. We describe here the same phenomenon in a child with broncopneumonia due to Mycoplasma pneumoniae.

A 6-year-old boy was admitted with a five-day history of a febrile illness associated with headache, vomiting, and pain in the left elbow. He appeared to be a previously healthy child. He had attended at home with ampicillin for 24 h. On admission he had a temperature of 39.5°C and a rash. A few crepitations were heard at both lung bases and the chest X-ray showed consolidation in both lobes. The haemoglobin was 13 g/dl, W.B.C. 5.8 x 10^9/l (3800/mm^3), and E.S.R. 12 mm in 1 h. The creatinine clearance was estimated at 56 ml/min. A urine culture after mycoplasma was negative at first but the titre rose to 1/1024 after four weeks.

Ampicillin was continued intramuscularly but had to be withdrawn after two days when the patient developed a rash and a temperature of 39°C. Cephalixin was substituted but in spite of this his fever persisted for eight days after admission. On the fifth hospital day he was noted to be drowsy and irritable with a poor fluid intake. His serum electrolytes showed a marked rise in sodium and chloride levels (142 mmol/l, potassium 2.8 mmol/l, chloride 88 mmol/l, bicarbonate 28 mmol/l) and urea 3.7 mmol/l (22 mg/100 ml). The plasma osmolality was 265 mmol/kg and the urine osmolality 611 mmol/kg. Unfortunately ADH measurements were not available.

His fluid intake was restricted to 500 ml daily and on this his condition gradually improved. The serum electrolytes returned to normal within 72 h, as did the osmolality of his serum and urine. His cardiac arrhythmias subsided. His convulsions disappeared after 10 days and his chest X-ray was completely clear two weeks later.

Inappropriate secretion of ADH has been described in association with both acute and chronic chest infection in adults, but we believe this to be the first report of its occurrence in association with Myc. pneumoniae infection or in a child with acute pneumonia. The source of ADH in these patients is unknown; tumours of the lung may produce the hormone, as may tuberculous meningitis. It has also been suggested that hyponatraemia and low cardiac output may act as a stimulus to ADH production. It is also possible that excessive ADH secretion is a non-specific response to stress, and it would be of interest to investi- gate this possibility in patients subjected to other forms of stress such as major injury or surgery.—We are, etc.

T. M. LITTLE
R. H. DOWDLE
Department of Child Health, University Hospital of Wales, Cardiff


Fatality After Fine-needle Aspiration Biopsy of Liver

SIR,—Fine-needle aspiration biopsy of the liver is considered a safe procedure. Overt hemorrhage or unexpected death are considered only absolute contraindications.1 Open liver biopsy, on the other hand, is associated with a definite risk. We wish to report a case of death after a fine-needle aspiration biopsy.

A 62-year-old man, who had had a portacaval end-to-side shunt operation and splenectomy in 1973 for cryptogenic liver cirrhosis associated with bleeding oesophageal varices, splenomegaly, and thrombocytopenia, was admitted to hospital in September 1974 with impending hepatic coma. His blood ammonia was 165 μmol/l (321 μg/100 ml) (normal range 28-64 μmol/l) and total serum bilirubin 213 μmol/l (12.5 mg/100 ml) (normal range 5-20 μmol/l). His weight was 82 kg and his height 1.75 m, which gave a body mass index of 28.1. He was a moderately severe alcoholic who had not had any regular medical supervision for many years. His past history was notable for chronic liver disease and Raynaud's type symptoms in his hands.

He was admitted to hospital with features of hepatic encephalopathy, but he was not in hepatic coma. He had a history of 30 years of heavy alcohol abuse and had been noted to be drowsy and irritable with a poor fluid intake. His serum electrolytes showed a marked rise in sodium and chloride levels (142 mmol/l, potassium 2.8 mmol/l, chloride 88 mmol/l, bicarbonate 28 mmol/l) and urea 3.7 mmol/l (22 mg/100 ml). The plasma osmolality was 265 mmol/kg and the urine osmolality 611 mmol/kg. Unfortunately ADH measurements were not available.

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T. M. LITTLE
R. H. DOWDLE
Department of Child Health, University Hospital of Wales, Cardiff


Owen Working Party

SIR,—The B.M.A.'s account of the proceedings in the Owen Working Party (4 January, p. 48) and of subsequent events has not answered all the questions the thoughtful doctor would like to ask. I am therefore writing on behalf of the executive committee of the Association of University Clinical Academic Staff to clarify certain points. Our information on proceedings of the Owen Working Party is derived from discussions with eight of its members.

At the final meeting of the Owen Working Party on 20 December, our negotiators responded with heat to Mrs. Castle's chairmanship. She had introduced the Department of Health and Social Security's proposals by saying that they had been prepared at short notice, under pressure from the profession, and that she wanted our negotiators to take them as presented, with no further comment or discussion. Our negotiators became upset. At one stage they asked if the proposals represented a "take it or leave it" offer and were told that the principles were not negotiable, though the details were. Exchanges became heated.

HENRIK RISKA
CLAES FRIMAN
Department of Medicine, University of Helsinki, Helsinki, Finland

The liver was severely cirrhotic and contained many cavernous areas.

Fine-needle aspiration biopsy of the liver has been considered virtually risk-free. In this patient with liver cirrhosis associated with a primary hepatoma, however, the procedure, though correctly executed, led to gross intra-abdominal haemorrhage and, in conjunction with other factors, to the death of the patient.—We are, etc.


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