three weeks' history of pain and swelling in the epigastrium, dysphagia, anorexia, and loss of weight. His liver surface was palpable and unevenly nodular. Investigations showed serum bilirubin 43 mg/100 ml, alkaline phosphatase 1170 IU, aspartate transaminase 192 IU, total serum protein 6.3 g/100 ml, albumin 3.3 g/100 ml. A liver scan, using radioactive technetium (Tc), showed no lesion throughout the organ. The patient's serum gave a positive reaction for alpha-fetoprotein using crossed immunoelectrophoresis. This reaction was confirmed using three different commercial sources of antiserum. Postmortem examination showed a poorly-differentiated adenocarcinoma of the cardia of the stomach. Metastases were present in the lymph nodes along the lesser curvature of the stomach and in both left and right lobes of the grossly enlarged liver (4450 g). The hepatic tissue, though distorted by the metastases, retained its lobular architecture and gave no indication of a primary liver tumour. No multicentric or extragastrointestinal teratoma was found.

This case emphasizes that the presence of circulating alpha-fetoprotein can no longer be regarded as diagnostic of hepatocellular carcinoma. A recent report by Gitlin et al. indicates that the histogenetic connexion between tumours of the liver, gonads, and stomach. He found that synthesis of alpha-fetoprotein occurs not only in human fetal liver but also in human yolk sac and the human embryonic and fetal gastrointestinal tract. Clearly this raises the possibility that gastric tumours may share the responsiveness to embryonic potential that is shown by hepatocellular carcinoma. Furthermore, it is possible that the functional elements in teratomata may be hepatic, gastrointestinal, or possibly endodermal sinus tumour, which with yolk sac and the human embryonic and fetal gastrointestinal tract.

So far as we know there has been no study to show how common is the association between alpha-fetoprotein and carcinomas of the stomach. This merits further investigation.

We are grateful to Dr. D. J. Galton for permission to report the clinical details of this case and to Dr. C. E. Fenton for his help in the laboratory estimations.

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Footballer's Migraine

Sir,—We were interested in Professor W. B. Matthews's description of this syndrome (6 March, p. 687). We could hardly have expected similar features of the following case which we found puzzling when the patient consulted us in 1965.

A part-time professional footballer aged 22 complained of disturbances of vision, frequently precipitated by heading a ball. The first episode had occurred in 1959 when he went to head a ball but hit his head against an opponent. He fell to the ground, dazed but not knocked out, and was attended to by the trainer. Almost immediately he noticed flashes of light and speckles, shivering like the after-effects of looking at a bright light, affecting both eyes, and this lasted for about 30 minutes. During this time he could not focus properly or see to either side, and this was followed by bilateral headache lasting for 2-3 hours, without sickness. He played football again about two weeks later, headed the ball, and had no further attacks until some months later, when some visual disturbance, usually followed by headache, would then recur about twice a year, sometimes but not invariably related to heading a ball, especially if it was wet and heavy.

On examination he appeared healthy and there were no abnormal signs. Ophthalmic examination showed no appreciable refraction error or other ocular cause to account for his symptoms. X-rays of his skull, cervical spine, and chest were normal. It was considered that more elaborate investigations were not necessary, and that the best advice for him was to avoid heading a football.

When his condition was reviewed he was still playing football, but now as an amateur. He said that he was able to head the ball without any ill effects from a deliberate frontal impact, and although an accidental glancing blow could momentarily do this did not reproduce his former symptoms.

Professor Matthews wondered whether an isolated attack of classical migraine in young boys following a blow on the head at football was the forerunner of ordinary migraine or confined to minor injuries in football. In our case, the first attack was precipitated by his head injury, and subsequent attacks followed heading the ball although some occurred without such provocation, and he did not have to give up the game.—We are, etc.,

NOTTINGHAM

Michael L. E. Espir

LEICESTER

J. L. D. Hodget

P. H. N. MATTHEWS

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NITRACEPAM IN CHRONIC OBSTRUCTIVE BRONCHITIS

Sir,—I have found similar worsening of respiratory failure with nitracepam (17 June, p. 688) in five patients already in respiratory failure. Nitracepam was given as 5-10 mg by mouth at night as sedation. Three of the five patients were in coma next day and the remaining two were reaching that stage. The mean capillary Pco2 (Astrup) rose from 53 mm Hg to 79 mm Hg. All five were successfully resuscitated.

As Dr. J. Gaddie and others remark, no other sedative or tranquilizing drug is available which produced such marked effects. My own preference is for promethazine hydrochloride (Phenergan) 25-50 mg, which seems to cause less respiratory depression.—I am, etc.,

P. S. LONDON ACCIDENT HOSPITAL, BIRMINGHAM

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ANAESTHESIA BY ACUPUNCTURE

Sir,—We were members of a party of ten British doctors who visited the mainland of China at our own expense between 19 and 24 March, and marvilled at the hospitalisation of health centres, factories, and communes in Shanghai, Canton, Peking, Nanking, Wuhsi, and their neighbourhoods.

We were impressed with the standard of medical care, but above all we were astounded by the use of acupuncture anaesthesia in major surgery. As examples,