patients with bony secondaries visible on x-ray were excluded.

Mr. M. J. Rees (April, p. 109) objects to the statement that the production of gastrin by pancreatic tumours cannot be called "ectopic." It is agreed that the non-aryrophilic α cells, or β cells are identical in nature with gastrin-producing cells of the fundus of the stomach, but have 8 cells been proved to secrete gastrin under normal circumstances?

I am fully in sympathy with Mr. Keynes's difficulty with nomenclature, particularly with the use of the term "ectopic," but no really appropriate adjective has yet been suggested; "para-endocrine" seems preferable to "ectopic." The term "Cushing's syndrome" is often employed—for example, by Dr. Azzopardi—to describe the consequences of production of corticotrophin by tumours, but its use in this context is incorrect as it is not the syndrome described by Harvey Cushing. The presence of "ectopic" production of corticotrophin by tumours is usually suspected initially by the presence of hypokalaemic alkalosis, which is very uncommon in classical Cushing's syndrome.10 Again, the majority of cancer patients with corticotrophin production, particularly when due to oat-cell carcinoma of the lungproduce very large amounts long enough to develop the physical features typical of classical Cushing's syndrome. The hormonal syndromes are best classified in terms of the hormones they secrete, rather than by eponymous diseases they resemble to a greater or lesser extent.—I am, etc.,

E. J. Ross

University College Hospital Medical School, London W C 1

Cytomegalovirus Oesophagitis

SIR,—Although the protein clinical manifestations of cytomegalovirus in the adult are now well recognized1 we should like to draw attention to severe ulcerative oesophagitis occurring as a presenting feature.

A 35-year-old fitter was admitted in a cachectic state with a month's history of progressive difficulty and pain on swallowing. A diagnosis of Hodgkin's disease had been established 10 years earlier on cervical lymph node biopsy and subsequently he had received treatment with radiotherapy, steroids, and cytotoxic agents. On examination he was deconditioned, wasted, and febrile. There was no oral moniliasis. He had bilateral pleural effusions and gross leg oedema. The liver and spleen were palpable.

There were crops of petechiae and subcutaneous nodules over the whole of the trunk. A barium swallow showed gross irregularity of the oesophageal mucosa with three prominent ulcer crater and numerous other tiny ulcerations. The appearances were considered to be highly suspicious of monilial oesophagitis, but there was no improvement with nystatin. His condition continued to deteriorate and he died three weeks later.

The main findings at necropsy included those due to invasive Hodgkin's disease and those attributable to disseminated cytomegalovirus infection. The distal third of the oesophagus and the fundus of the stomach showed confluent elevated white plaques up to 3 cm diameter. Microscopically, the epithelium was denuded and there was a non-specific mononuclear infiltrate in the lamina propria. Large numbers of degenerative cytomegalic cells were present (Fig.).

In seriously debilitated patients such as those with advanced malignant disease or those on immunosuppressive therapy opportunistic infection with cytomegalovirus is not uncommon and organs such as lungs, adrenals, spleen, pancreas, and kidneys are frequently involved.2 Lesions of the gastrointestinal tract, excluding the liver, are rare, and it is often difficult to define the specific role of the cytomegalovirus in their production.

Levine, Warner, and Johnson3 have described patients with cytomegalic inclusions in ulcers of jejenum, ileum, and colon, and a similar lesion in the anus and rectum has been reported in a woman dying from primary cytomegalovirus infection.4 Previous comment has been made of oesophagitis in cytomegalovirus infection5 and it seems likely that the gross ulcerative change in the oesophagus of our patient was due primarily to cytomegalovirus infection. It is possible that other forms of apparently non-specific ulceration of the gastrointestinal tract in debilitated patients might be related to cytomegalovirus infection.—We are, etc.,

P. J. Toghill

General Hospital, Nottingham

MAY McGAUGHEY

Department of Pathology, University of Nottingham

Tropical Splenomegaly, Sickle-cell Trait, and P. falciparum Infection

SIR,—The diagnosis of tropical splenomegaly (T.S.S.) is, as straightforward as Dr. Marianne Janosi (4 March, p. 628) implies. She points out a number of features, but there are recent series with a number of anomalies by her criteria. For instance, the size of the spleen may be very variable, and macroglobulinemia has not been a constant finding. In the series of Stuiver et al.,1 there are 7 out of 29 cases with spleens palpable less than 10 cm below the left costal margin including some with only 2 or 3 cm spleno-

megaly. Although Lowenthal et al.7 found only two cases of 19 in Zambia with normal IgM, a normal IgM was reported in four of eight cases from Uganda by Ziegler et al.9 Our patient was small. He weighed 45 lb (20.5 kg) and was 46 in (117 cm) tall. Consequently, his spleen, palpable 7.5 cm below the left costal margin, was proportionally large for his age. We excluded Hbs-β thalassaemia, as Ha’s was the major component, and HbA2 and HbF in the pro-

positus and his two siblings were normal. Dr. Janosi draws attention to some of the difficulties in diagnosing T.S.S. It is usually stated that T.S.S. is a diagnosis by exclusion.14 Sago4 suggested more rigid criteria for diagnosis. If Dr. Janosi and her colleagues also recognize a series of positive diagnostic features, they may invite her to report them.—We are, etc.,

D. I. K. EVANS P. M. REDDY B. WOLMAN

Department of Pathology, Booth Hall Children’s Hospital, Manchester


Duodenal Ulcer and Gastric Cancer

SIR,—Eight male cases of gastric cancer in men have been found in this average size practice since 1961. Four of these had a long-standing history of duodenal ulceration. All smoked heavily and developed chronic bronchitis and emphysema. Three were se-

men. In none of them was gastric cancer
suspected until a few months before death when increased pain necessitated surgery.

All these patients had severe anaemia (Hb 55-60%) at diagnosis. All had a long-standing history of duodenal ulcer and were then found to have gastric cancer. It seems, therefore, that such cases need early and careful examination if they come to operation for a duodenal ulcer. Tragically, all were late diagnoses chiefly because dyspeptic symptoms were wrongly interpreted in the 16-12 months before laparotomy.

It is said that duodenal ulcer and gastric cancer only rarely coexist,1 and a report from the Birmingham Regional Cancer Registry and the United Birmingham Hospitals stated that no association was found between cancer and peptic ulcer.2—I am, etc.,

M. MACLEOD

Wallasey, Cheshire

1 World Medicine: Review of the Year. 20 Feb.


Multiple Crashes on Motor Ways

SIR,—I would certainly endorse the remarks made by Dr. J. A. Frain (1 April, p. 49) regarding blood sugar levels in road traffic accident victims.

On several occasions, particularly in the last twelve months, we have had brought in to this department lorry drivers who, for no apparent reason, have crashed across the central reservation of the nearby motorway. Many of them have started driving in the early morning with a minimal breakfast, if any at all. We have made a point of carrying out blood sugar estimations and found them to be very low. In many cases this was found to be the extent that now we are instituting a system of checking blood sugar levels on every driver who has been involved in a road traffic accident.

I am in the process of doing a survey of 6,000 road traffic victims and one of the questions in the fairly lengthy survey is designed to establish the relationship between the site of the accident and their last meal.

—I am, etc.,

M. S. CHRISTIAN

Wexham Park Hospital, Slough, Bucks

Monitoring Heparin Infusions

SIR.—One is grateful to Dr. Judith M. Chesells and others for their work showing 5% dextrose to be a suitable vehicle for heparin infusions (8 April, p. 81). The practical need for a simple solution was driven by the unavailability of suitable heparin infusion solutions. There are, however, in their own words, ‘no convincing evidence’ to support using this vehicle nor is the argument that 5% dextrose is a physiological one convincing.

There was a significant difference in heparin clearance (95±8 vs 24±10 ml/min) between the two groups. The heparin clearance was lower in the 5% dextrose group. This was in keeping with the difference in rate of heparin excretion in the urine. However, the difference in heparin clearance was not statistically significant. The mean duration of heparin therapy was 14 days in the 5% dextrose group and 10 days in the normal saline group. The difference was not statistically significant. The mean volume of urine excreted was 500 ml in the 5% dextrose group and 300 ml in the normal saline group. The difference was statistically significant.

In any case under the circumstances any bleeding ought to be instantly detected and suitable treatment given. Compare this situation with that of outpatient oral anticoagulant therapy.—I am, etc.,

MICHAEL KLAEBER

London N.W.11

Need for Continued Oral Therapy in Diabetes

SIR.—Dr. J. E. M. Tomkins and Arnold Blockley say (11 April, p. 609) ‘there is now an onus on clinicians not to use this form of therapy (tolbutamide or phenformin) unless it can be shown that simple dietary restriction alone is unsuccessful in preventing hyperglycaemia.’ The great practical problem is to determine the right course of action when dietary restriction is ignored, not when it is ‘unsuccessful’.

I see no evidence of the necessity for ambulatory insulin therapy who, in spite of all that I and the dietician say, refuse to restrict their diet indefinitely. If they are given sulphonylureas or insulin they become more and more obese. While such a situation—morbid obesity without hyperglycaemia or less obesity with hyperglycaemia? This question is hardly ever posed—much less answered—in writings about diabetes.—I am, etc.,

JOHN W. TODD

Farnham, Surrey

Bullous Lesions in Poisoning

SIR,—There has been much discussion on the aetiology of the bullae seen in barbiturate poisoning and other states of unconsciousness.1 While it is generally agreed that pressure—and anaxia play an important part in the development of bullae, little is known of the mechanism of the bullae formation.2 The present study was undertaken to determine the role of ischaemia of the skin in the formation of bullae.

A 48-year-old woman, previously well, was admitted to hospital 24 hours after taking about 20 Tuinal capsules (quinvalbarbitone sodium—100 mg each of 4 capsules). She had been found at home semiconscious, lying on her right arm. On examination she was drowsy and complaining of pain in her right hand and forearm which were cold and oedematous. There were large tense bullae over the medial side of her forearm including a linear arrangement of small blisters where, it was thought, her night-clothes had rubbed up. There was global weakness and anaesthesia of the hand, the latter to about 10 cm above the wrist. The radial pulse was initially impalpable but readily felt when the oedema subsided. Her blood pressure was 130/85 mm Hg. Her urine was dark and gave a positive chemical test for blood.

Investigations showed considerably elevated levels of serum glutamic oxaloacetic transaminase, lactic dehydrogenase, and creatinine phosphokinase. Electrocardiography revealed evidence of widespread derangement of the small muscles of the hand. Further specimens of urine were normal on microscopy and chemical testing.

A diagnosis of brachial arterial and venous occlusion with ischaemic muscle necrosis, nerve damage, and myoglobinuria was made. With initial elevation and a continuing intense physiotherapy, Volkmann’s ischaemic contracture has not developed four months later although the ischaemia was considerable. Skinsparing muscle wasting. The area of anaesthesia is much less. The bullae healed rapidly without scarring.

The occurrence of ischaemic damage to muscle and nerves in barbiturate poisoning has been known for many years and undoubtedly occurs more frequently than reports suggest.3 The association here between bullae strongly suggests that anaemia and local pressure are important causative factors for these skin lesions. Local pressure is probably important, since skin blood flow is fairly well maintained in anaesthesia induced by long-acting barbiturates. This is demonstrated in a recent experimental study, albeit on monkeys.4 The marked progressive reduction in skeletal muscle blood flow found in this investigation may indicate a predisposition to muscle ischaemia and necrosis in barbiturate poisoning.—We are, etc.,

B. I. HOFFBRAND C. M. RIDLEY

Whittington Hospital, N. 19

1 Ridley, C. M., British Medical Journal, 1971, 1, 742.


Chinese Burn

SIR,—A girl of 14 presented with an area of cellulitis 4 in (10 cm) by 2 in (5 cm) on the extensor surface of the lower third of her left forearm. I could find no break in the skin, nor was there any local sepsis elsewhere on the hand or arm. I administered an injection of Triptopen (bene-thanine, procaine, and benzyl penicillin combined) and asked her to return in 48 hours. On her return the cellulitis was slightly more extensive and I therefore changed to a course of erythromycin. On further review three days later, the cellulitis had largely resolved and there was now palpable, something like a feeling of thickness about 4 in (10 cm) long in the subcutaneous tissues. Neither the patient nor her mother were able to recall any incident suggestive of penetration by a foreign body.

Four days later I undertook an exploration of the forearm under local anaesthetic and was surprised to find that the presumed foreign body in fact consisted of a linear streak of fat necrosis. This was confirmed by histology. The explanation lay in the fact that a few days before the onset of symptoms, the girl’s brother had subjected her to a so-called “Chinese torture,” consisting of gripping the wrist with both hands and twisting in opposite directions to produce a shearing stress which resulted in an acute fat necrosis.—I am, etc.,

B. S. MILNER

Edburgh, Kent

Cardiac Arrhythmias during Laparoscopy

SIR,—I read with interest the article by Drs. D. B. Scott and D. G. Julian on the occurrence of cardiac arrhythmias during, we—