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 very 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 February 1968.

² *British Medical Journal*, 1965, 1, 1567.

Multiple Crashes on Motor Ways

SIR,—I would certainly endorse the remarks made by Dr. J. A. Fraiss (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 extremely low in many cases, to 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 time 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. Chessells and others for their work showing 5% dextrose to be a suitable vehicle for heparin infusions (8 April, p. 81). The situation was difficult, with dextrose allegedly unsuitable and the infusion of a litre of normal saline a day often contraindicated.

However, in their final paragraph they imply that all heparin infusions should be monitored by partial thromboplastin times with kaolin. On the one hand this would mean a great deal of extra work for haematology departments. On the other hand even on their own evidence laboratory control hardly seems necessary. The dose required varied only between 28,000 and 40,000 units per 24 hours; and there was no evidence of increasing sensitivity to heparin, so presumably the dose remained constant in each patient. Hence the practice of giving a set regimen of 30,000-40,000 units per day seems quite reasonable, and the theoretical risk of haemorrhage seems not to materialize.

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

MICHAEL KLABER

London N.W.11

Need for Continued Oral Therapy in Diabetes

SIR,—Drs. A. M. Tomkins and Arnold Bloom say (11 March, p. 649): "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 numerous obese maturity-onset diabetics who, in spite of all that I and the dietician say, refuse to restrict their diet indefinitely. If they are given sulphonylurea drugs or insulin they become more and more obese. Which is the less harmful situation—more 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.¹⁻⁴ While it is generally agreed that coma, pressure, and anoxia play important roles, there is no unanimity about the possible local effects of drugs on the skin. The following case provides further evidence of the prime importance of anoxia and pressure.

A 48-year-old woman, previously well, was admitted to hospital 24 hours after taking about 20 Tuinal capsules (quinabarbitone sodium and amylobarbitone sodium, 100 mg of each per capsule). 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 rucked 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 creatine phosphokinase. Electromyography revealed evidence of widespread denervation 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 continuing inten-

sive physiotherapy, Volkmann's ischaemic contracture has not developed four months later although there is considerable forearm 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.⁵ The association here with bullae strongly suggests that anoxia 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, as was demonstrated in a recent experimental study, albeit on monkeys.⁶ 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,
Archway Wing,
London N.19

¹ Ridley, C. M., *British Medical Journal*, 1971, 3, 28.

² Seddon, H. J., and Howse, A. J. D., *British Medical Journal*, 1971, 3, 371.

³ Berlyne, N., *British Medical Journal*, 1971, 3, 371.

⁴ Beveridge, G. W., *British Medical Journal*, 1971, 4, 116.

⁵ Howse, A. J. D., and Seddon, H. J., *British Medical Journal*, 1966, 1, 192.

⁶ Forsyth, R. P., and Hoffbrand, B. I., *American Journal of Physiology*, 1970, 218, 214.

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 Triplopen (benethamine, 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 that felt like a thick knitting needle 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

Edenbridge, 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 laparo-