not so. Regardless of the storage temperature, the addition of acetic acid (10 µl of a 20% aqueous solution per ml serum) or citric acid (4 mg per ml serum) is essential to stabilise activity. It can further be shown that while addition of either of these agents will reactivate the enzyme in sera previously stored at room temperature (18-25°C), or at 4°C for a week without them, no such reactivation is possible when sera are similarly stored at −20°C. Ignoring these facts has led and still leads to misleading results, which in turn devalue an otherwise excellent tumour marker.

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Handicapped children and the attendance allowance

SIR,—Our attention has been drawn to a recent decision on the part of the Department of Health and Social Security to withhold the attendance allowance from the parents of severely mentally and physically handicapped children once the number of weekend admissions to hospital has reached 28 days. In addition, the DHSS is reclaiming payment when this global figure has been exceeded during the past few years.

Apart from the apparent injustice in certain cases, this attempt to restrict short-term stay in hospital for these children for financial reasons will be self-defeating. We know that in many cases this short spell of rest and freedom from responsibility is the only way by which the family can continue to care for a handicapped child at home. Without it the consultant would be under considerable pressure to seek long-term, costly residential care for the child and in so doing separate him from his family, with all the attendant distress to those concerned.

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Drug-induced oesophageal ulceration

SIR,—The case of tetracycline-induced oesophageal ulceration reported by Drs K S Channer and D Hollanders (25 April, p 1359) highlights the continuing problem of direct chemical injury to the oesophagus by impacted tablets or capsules. Though a recent review[1] details a range of preparations implicated in causing oesophageal ulceration, this lesion has not been previously documented in association with naftidrofuryl (Praxilene).

A 21-year-old nursing auxiliary was prescribed naftidrofuryl capsules 100 mg thrice daily for pain in the legs of possible vascular origin. Two weeks after starting treatment she swallowed her usual evening capsule without water, and three hours later developed retrosternal pain radiating to the neck, back, and epigastrium. This persisted and was severely exacerbated by attempting to swallow solids or liquids. Physical examination was normal. Four days after the onset of her symptoms, fibreoptic oesophagoscopy revealed a 1 cm oval superficial haemorrhagic erosion 25 cm from the incisors. There were no other abnormalities in the upper gastrointestinal tract. Her pain and dysphagia resolved over the next week without specific treatment and had not recurred when she was last seen two months later.

The nocturnal onset of chest pain worsened by swallowing, and following closely the taking of a tablet or capsule without water, points towards drug-induced oesophageal injury.[2] In this instance endoscopy confirmed a mid-oesophageal lesion, a particularly vulnerable site anatomically, as pointed out by Drs Channer and Hollanders. Naftidrofuryl should be added to the growing list of tablets and capsules which patients should swallow with adequate amounts of fluid.

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[1] Anonymous. *Drug Ther Bull* 1981;**19**:33-4.
[2] Barrison IG, Trewby PN, Kane SP. *Endoscopy* 1980; **12**:197-9.

Antacids for duodenal ulcer

SIR,—Your leading article (9 May, p 1495) on the hazards of the longer-term use of antacids for duodenal ulcer is a timely warning against following the present trend in the United States. The reduction of gastric acidity brings its own set of problems from the loss of the normal acid gastric barrier and the encouragement of bacterial overgrowth in the stomach and in the intestine. The idea that it is necessary to reduce gastric acidity to achieve healing is untrue. Healing can be achieved with the risk of far fewer complications by aiding the normal defence mechanisms of the body. There are two main possibilities.

Firstly, we should not forget the benefit of old-fashioned bed rest. Both the horizontal position and the respite from environmental stresses and strains provide a powerful stimulus to ulcer healing. Its benefit in the healing of duodenal ulcers has been shown by Dölle *et al*[1] in West Germany, who have shown endoscopically that ulcers heal as well with bed rest as they do with cimetidine. Although economic forces may allow only a brief respite this may be of great benefit.

Secondly, bed rest can be combined with and followed by giving carbenoxolone (Duogastrone), which strengthens the defence mechanisms without affecting acid secretion. It does so by increasing the protective mucus secretion and also by a direct effect on the cellular repair mechanisms. Its one side effect, which may cause some electrolyte imbalance, is easily monitored and corrected and with medical supervision it can be safely used in older patients and in those with hypertension. With over 500 publications and 20 years' experience it offers an attractive alternative to antacids and to H_2-receptor blockers. It has the further advantage that once healing has taken place the risk of recurrence is less than after treatment with cimetidine, probably because the defence mechanisms have been positively strengthened and not weakened by disuse atrophy. There are other preparations which aim to help the defences; these include deglycyrrizinised liquorice (Caved S), which contains a considerable amount of antacid; and also tripotassium dicitrato bismuthate, which is thought to provide a protective coating to the ulcer. This needs further study in view of the known hazards of bismuth to which you refer.

I believe that the well-known Schwartz dictum "No acid—no ulcer" has been leading us in the wrong direction, trying to reduce aggressive forces when we should be trying to strengthen the defences of the stomach and so facilitate the normal repair mechanisms.

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[1] Dölle W, Malchow H, Sewing K-F, Albinus M, Schomerus H. *Acta Gastroenterol Belg* 1978;**41**: 424-6.

SIR,—I note in your leading article "Antacids for duodenal ulcer" (9 May, p 1495) the statement that "we do not know what causes ulcer pain." Confusion about the cause will persist until some notice is taken of the pain sensitivity of the patient with the ulcer.

The relevance of this factor has been demonstrated in relation to the syndromes of cardiac infarction[1] and rheumatoid arthritis.[2] No attempt, however, has been made to assess its importance in relation to the presence of pain in patients with duodenal ulcer. It may be of interest therefore that, with the pressure pain sensitivity test described in 1954,[3] no patient with a proved duodenal ulcer and a pressure pain sensitivity of 6·0 kg or more experienced any pain. They presented as painless cases of haematemesis, melaena, or perforation. The converse was not valid.

Four such cases occurred among 50 cases of duodenal ulcer, suggesting that the incidence of painless duodenal ulcer was at least about 8%. In these patient it would have caused no surprise to be informed of the "extraordinary fact" that "on endoscopic examination active ulcers may often be found in patients who suffer no pain at all."

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[1] Keele KD. *Br Med J* 1968;i:670-3.
[2] Huskisson EC, Hart F D. *Br Med J* 1972;iv:193-5.
[3] Keele KD. *Lancet* 1954;i:636-9.

Hydrallazine-induced necrotising vasculitis

SIR,—The paucity of reported cases of hydrallazine-induced cutaneous vasculitis commented on by Dr Andrew Peacock and Professor D Weatherall (4 April, p 1121) may not reflect the true incidence of cutaneous complications in the hydrallazine "lupus" syndrome. We have recently seen a patient on hydrallazine who presented with a widespread itchy maculopapular and purpuric eruption, which resolved on stopping the drug. Skin histology confirmed a vasculitis.

A 64-year-old gardener with hypertension had taken hydrallazine 50 mg thrice daily, with Slow Trasicor (sustained-release oxprenolol) 160 mg a day and two Navidrex K (cyclopenthiazide and potassium) tablets a day for several years. His skin rash had been present for two months and he gave a five-month history of widespread joint pains, pleuritic chest pain, and weight loss. When he was admitted in July 1980 hepatosplenomegaly was noted in addition to the rash affecting his trunk and limbs. Investigations revealed a pancytopenia, erythrocyte sedimentation rate (ESR) of 73 mm in one hour, antinuclear factor positive at 1/128 (homogenous pattern), and an anti-DNA (double-stranded) antibody level of 45 units/ml. Tests of acetylator status showed him to belong to the slow acetylator group. Skin biopsy showed a vasculitis of the dermal vessels and direct immunofluorescence showed IgM, fibrin, and C3 in and around vessels in the mid and lower dermis of affected skin. After he had stopped