advocate that ultrasound examination replaces these. The time factor prohibits the use of ultrasound for all patients with biliary symptoms.

MARGARET R JONES
DAVID WEBSTER

University Hospital of Wales,
Cardiff

¹ Gravelle, I H, Jones, M, and Roberts, E S, *British Journal of Radiology*, 1973, **46**, 568.
² Hublitz, U F, Kahn, P C, and Sell, L A, *Radiology*, 1972, **103**, 645.

Management of childhood epilepsy

SIR,—I hope that Dr H Wykeham Balme (14 May, p 1284) does not imply that all children with epilepsy should live in a home with protected staircases, without bathroom locks, and with guards around the fires and radiators; that they should bath in less than three inches of water, go to school accompanied, and not climb trees or go swimming or camping.

The management of the patient with epilepsy consists in not only trying to prevent attacks using anticonvulsants—not so ineffective or poisonous when handled correctly—but also attempting to reduce the social and psychological complications by talking to his family. This latter aspect is frequently forgotten. If parents understand what epilepsy is and adopt a sensible attitude, so will the child, whereas if they are ashamed of him or become over-anxious he will react accordingly. Too often parents are given a long list of restrictions irrespective of the frequency of their child's attacks and without explanation.

The goal should be for children with epilepsy to live a normal life without restriction. Perhaps we have forgotten that taking risks is part of growing up and that a child must experience failure as well as success. The important point is that patients must be treated as individuals and with common sense. There will be some who will be unable to take part in certain activities, but most children with epilepsy can live in normal homes, take part in the usual school activities, and go swimming and camping provided they are adequately supervised. Over-protectiveness, either by parents or school, should be corrected early, as this will reinforce the stigma of epilepsy. Restrictions should be as few as possible. Surely it is better to err in favour of under-restriction than over-restriction.

DAVID THRUSH

Department of Neurology,
Freedom Fields Hospital,
Plymouth

Acute renal failure associated with carbenoxolone treatment

SIR,—Following the report by Dr C Descamps and others on rhabdomyolysis and acute tubular necrosis associated with carbenoxolone and diuretic treatment (29 January, p 272) I wish to report another patient presenting with acute renal failure following administration of carbenoxolone.

The patient was a 38-year-old woman in whom gastric ulcer had been diagnosed five years previously. From this time onwards until her referral with renal failure she took carbenoxolone 200 mg daily. In addition, she was known to be hypertensive and had taken a proprietary compound analgesic preparation 2-3 times daily for 20 years.

Her present complaint to her own practitioner before referral was of progressive limb weakness for two weeks. She was found to have haematuria and proteinuria and was treated for pseudomonas urinary infection with nitrofurantoin. Over a period of days she became oliguric and was referred to this hospital for management of renal failure. She was hypertensive (blood pressure 190/110 mm Hg) and was found to be in renal failure despite adequate hydration (serum creatinine 640 μ mol/l (7.2 mg/100 ml), urea 21.5 mmol/l (130 mg/100 ml). Serum potassium was 1.6 mmol (mEq)/l, aspartate aminotransferase 1000 IU/l, and creatinine phosphokinase 2590 IU/l. Her renal failure progressed and she required peritoneal dialysis once and haemodialysis once. After a stormy clinical course marked by fever, severe hypertension, and pneumonia a diuresis ensued. Her treatment included ampicillin and gentamicin therapy. The serum creatinine ultimately fell to 150 μ mol/l (1.7 mg/100 ml), though she remained quite severely hypertensive and microscopic haematuria persisted after discharge from hospital. Attempts at intravenous pyelography were unsuccessful owing to a severe allergic reaction.

Multiple aetiology is presumed for this patient's renal disease. Pre-existing analgesic abuse, hypertension, urinary infection, and potentially nephrotoxic antibiotic therapy with gentamicin may have contributed to a greater or lesser degree. The striking feature of her presentation, however, was profound muscular weakness associated with severe hypokalaemia and oliguric renal failure. The high level of muscle enzymes approximates to those reported by Dr Descamps and his colleagues and it appears likely that carbenoxolone precipitated her acute renal decompensation.

The duration of carbenoxolone therapy (200 mg daily for five years) must surely be an all-time record. If she was taking her tablets as instructed it is remarkable that she did not present earlier with toxic effects from carbenoxolone.

BRIAN HURLEY

Renal Unit,
Canberra Hospital,
Acton, ACT,
Australia

Transient hypotension following intravenous ethamsylate (Dicynene)

SIR,—I am concerned at the recent introduction by Delandale Laboratories of their "theatre pack" of ethamsylate, an agent for capillary haemostasis. The new pack consists of 1 g of ethamsylate in 2 ml in a disposable dental-type syringe. The suggested dose is 1.5 ml (750 mg) but I am sure there will be a tendency to give the extra 0.5 ml and empty the syringe.

Slight transitory hypotension has been reported following this drug, but in a series of eight consecutive patients given 500-750 mg over 30 to 40 s intravenously an immediate fall of blood pressure was seen in all cases, the minimum fall being 25 mm Hg and the maximum 85 mm Hg—not "slight" by any standard. Fortunately the effect was short-lived and in all but one case the blood pressure had returned to within 10 mm Hg of the preinjection level within five minutes.

The fall was more marked in the elderly, is almost certainly dose-related, and probably could be avoided by slow intravenous injection. This, however, is not facilitated in the "theatre pack." All eight patients were under general anaesthesia but none was judged to be hypovolaemic at the time of injection.

I therefore write to suggest care in using the

"theatre pack" or possibly its avoidance when intravenous administration of ethamsylate is required.

L LANGDON

Lymington Hospital,
Lymington, Hants

Coronary artery spasm

SIR,—As emphasised in your leading article (7 May, p 1176) coronary artery disease is likely to have a variety of subtypes of which coronary artery spasm may be one. The onset of "variant angina" without provocation in patients with an arteriographically normal coronary arterial tree is quoted as a possible example of coronary artery spasm.

Is this not reminiscent of the time when "cerebral artery spasm" was thought to be responsible for transient ischaemic attacks now recognised to be due, in the main, to micro-embolisation from atheromatous plaques in the large extracranial vessels? It is recognised that these plaques may be quite small, producing neither stenosis nor bruit, and be demonstrable only by refined techniques such as "trickle angiography." I wonder whether information is available as to the presence (or absence) of ulcerated atheroma in the coronary arteries of patients with Prinzmetal's angina, plaques perhaps insufficient in extent to show on conventional arteriography?

M J BUTLER

Royal South Hants Hospital,
Southampton

¹ Hugh, A E, *British Medical Journal*, 1970, **2**, 574.

Effect of antihypertensive drugs on growth hormone secretion

SIR,—Lal *et al*¹ reported that clonidine, an antihypertensive drug with central α -adrenoceptor agonist properties, increased growth hormone (GH) secretion in normal volunteers in an acute trial. We have found the same effect in a study with young (20-33 years) normal male subjects after a single oral administration of 2 and 4 mg of BS 100-141,² a new α -adrenoceptor agonist.³ On the other hand no increases in plasma GH levels were found in 10 young (30-40 years) hypertensive subjects who had been under BS 100-141 treatment (3-6 mg/day) for 3-12 months. The question which then arose was whether treatment with BS 100-141 is able to stimulate GH secretion in older hypertensive patients, who more frequently require antihypertensive therapy.

Six male patients aged 44-60 years with mild hypertension (range 160/100 to 180/100 mm Hg) volunteered for the study. After an overnight fast and 30 minutes' bed rest an indwelling venous catheter was inserted. Blood for GH determination was drawn before and 30, 120, 150, and 180 min after oral administration of 2 mg of BS 100-141. The subjects remained in a recumbent position and ate nothing during the test. They also abstained from smoking. This test was followed by treatment with BS 100-141 2 mg thrice daily for 10 days, during which time no other medication was given. Every second day at 0900—that is, 2 h after the first daily dose of BS 100-141—blood was collected for GH determination. After 10 days of treatment the test carried out on the first day was repeated, blood samples being taken before and at intervals after the final 2-mg dose of BS 100-141. GH was assayed by double-antibody radioimmunoassay as previously reported.⁴ No significant increase in plasma GH concentration was noticed during the