Letters to the Editor

Pituitary apoplexy after stimulation tests

Sir,

We read with interest the recent report by Vassallo, Rana, and Allen of a case of pituitary apoplexy following stimulation tests.¹ We report a similar case of pituitary apoplexy following medical intervention, although in somewhat different circumstances.

A 53-year-old woman presented with acute right iliac fossa pain associated with anorexia, nausea and vomiting. On examination, the patient was pyrexial (38°C) with right iliac fossa tenderness. At surgery, a right paratubal abscess was identified which involved the caecum and proximal ascending colon. A modified right hemicolectomy and right salpingectomy were performed. Suppurative paratubal abscess with culture of *Streptococcus faecalis* was confirmed by histopathological examination.

Thirty-six hours post-operatively the patient complained of sudden onset frontal headache and, on examination, right-sided ptosis and inability to laterally deviate the right eye was noted. Visual acuity was normal and no gross visual field defect was detected. Over the next day a complete right sixth cranial nerve palsy and partial right third cranial nerve palsy developed along with a minor deterioration in visual acuity and a temporal hemianopia. Computed tomographic (CT) scan of brain and pituitary fossa revealed a pituitary lesion of mixed density with some haematoma and zones of infarction in a probable pituitary adenoma. The patient was commenced on intravenous dexamethasone.

Two weeks later, a 75% improvement in the third cranial nerve function was recorded but the sixth nerve palsy was still complete. Over the next six months, the visual field defect and all cranial nerve palsies completely resolved. Investigations of pituitary endocrine function revealed normal levels of prolactin, thyroid stimulating hormone and adrenocorticotid stimulating hormone, with reduced levels of growth hormone, follicle-stimulating hormone and luteinising hormone. Hormone replacement therapy has not, as yet, been required.

Pituitary apoplexy has been reported on seven occasions following cardiopulmonary bypass surgery, but we have identified only one other case of pituitary apoplexy occurring after non-cardiac surgery which was in a Japanese man after cholecystectomy.³ The incidence of neurological dysfunction following extracorporeal cardiopulmonary bypass has been estimated to range from 7 to 44% for transient and 1.6 to 23% for persistent sequelae.⁴ Impairment of cerebral function following surgery may result from cerebrovascular damage, coagulation distur-

bances, thrombosis, and embolism of particulate matter. Per-operative hypotension has not been shown to correlate well with post-operative neurological dysfunction.⁴ However, in the case of pituitary apoplexy following non-cardiac surgery, catecholamine release secondary to intra-operative hypotension was suggested as a possible causative factor.³ Thus, techniques resulting in hypotension or increased cerebral venous pressure such as jugular venous or abdominal compression, should be avoided in patients with suspected pituitary adenoma. However, as is commonly the case, pituitary adenoma was not suspected in our patient prior to onset of symptoms.

This case demonstrates the necessity for thorough investigation of all patients who develop unusual neurological symptoms following general anaesthesia and serves to heighten clinical awareness of this potentially life-threatening complication of surgery.

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Guidelines for the clinical use and dispensing of thalidomide

Sir,

Formal guidelines for the use of thalidomide are welcome and long overdue. Thalidomide is rightfully regarded as a potentially dangerous drug but we recognise that it has a role in the treatment of certain rare and severe skin diseases that have failed to respond to all other available therapies.

In our Institute, over a three-year period, 28 patients were treated with thalidomide (box). Patients' ages ranged from 7–68 years; 21 were female and seven male. The dose range of thalidomide was wide, varying from 50 mg on alternate days to 300 mg daily. Duration of treatment ranged from three weeks to 66 months, but the patients with the photosensitivity disorder, actinic prurigo, usually had defined courses lasting 3–6 months in the spring and summer seasons.

Side-effects necessitating discontinuation of treatment occurred in 11 of the 28 patients. Of these, eight had symptoms of possible sensory neuropathy and three had abnormal electrophysiological studies (so-called reduced SNAP). Paraesthesiae persisted for some months after cessation of therapy. The

Thalidomide indications	
severe actinic prurigo	16
nodular prurigo	4
lupus erythematosus	4
Behçet's disease	1
major aphthosis	1
mucosal lichen planus	1
epidermolysis bullosa	1

occurrence of neuropathy was not related to the dose or duration of treatment and seemed less likely to occur in the actinic prurigo patients. One patient with discoid lupus erythematosus developed unequivocal neuropathy after three weeks (total dose = 2 g) while another with lupus erythematosus profundus had no evidence of it after 28 months (total dose = 174 g).

Of the 14 potentially fertile female patients, four were thought not to be at risk of pregnancy, because they were not sexually active or their partner was sterilised. The others were verbally counselled regarding the risks (six signed a consent form acknowledging their understanding of the risk of teratogenicity). There were no pregnancies during therapy or within the three-month period after its cessation. Apart from the drug dispensing details, the containers were labelled with a warning that it should only be taken by the named patient.

Following this review, we drew up guidelines and a consent form for the use of thalidomide in our department similar to those suggested by Powell and Gardner-Medwin,¹ and we congratulate them on drawing these matters to the attention of a wider audience.

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Changing clinical spectrum of liver abscess

Sir,

It is perhaps misleading to suggest, as Yinnon *et al* have done,¹ that ultrasonography and computed tomography (CT) possess comparable sensitivity in the diagnosis of liver abscess. The retrospective analysis by Rubinson *et al* suggests that CT scanning has a sensitivity of 95–100% *vs* a sensitivity of 85–95% for ultrasonography.² Furthermore, multiple liver abscesses are more easily detected by CT scanning than by ultrasonography.^{3,4} CT scanning has the additional advantage of depicting the anatomical relationships with other abdominal viscera more accurately.⁵ Because of the superior specificity of ultrasonography, however,⁵ the complementary nature of these two imaging

Post-operative pituitary apoplexy

- cardiopulmonary bypass surgery
- cholecystectomy
- salpingectomy

modalities can be exploited by utilising them in tandem.⁴

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Bone density measurement in osteogenesis imperfecta may well be important

Sir

We thank Drs Deodhar and Woolf¹ for their interest in our paper.² We agree that bone densitometry may contribute to the diagnosis of osteogenesis imperfecta, a low finding being supportive of the diagnosis in some cases. However, in practice this is not often the problem; in most patients with clinical features or family history the diagnosis is not difficult without densitometry.

The practical difficulty is often the expectation that patients with osteogenesis imperfecta should have a low density as part of the disorder. Some do but in others the bone fragility must be largely attributable to the collagen disorder. Our paper should not be taken to suggest that patients with osteogenesis imperfecta with a bone density within the reference range do not have bone fragility. We were merely anxious to demonstrate that, since many patients had normal values on densitometry, such findings cannot be used to 'exclude' a diagnosis of osteogenesis imperfecta. Since the assessment of bone density by ordinary radiology is much less precise than any form of densitometry,³⁻⁵ still less is it possible to exclude osteogenesis imperfecta with plain films.

We disagree with Drs Deodhar and Woolf on the appropriateness of our methods. The great majority of known fractures in osteogenesis imperfecta are fractures of the long bones. Single photon absorptiometry may therefore be particularly appropriate in this disorder.

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Ileal leiomyoma - cause of undiagnosed chronic iron deficiency anaemia

Sir,

Benign tumours of the small intestine are responsible for less than 1% of cases of gastrointestinal bleeding and hence present a daunting task in terms of diagnosis.¹ Even after a thorough search, using all available diagnostic modalities, it may still be impossible to identify the source of chronic blood loss.

A 55-year-old Caucasian man was admitted with a two-day history of fever and lower abdominal pain. On examination he was found to be pyrexial and tachycardic with a tender mass in the left lower quadrant. The haemoglobin was 9.1 g/dl with an iron deficiency picture and the white cell count was $13.3 \times 10^9/l$. An ultrasound scan revealed $11 \times 10 \times 12$ cm mass containing cystic elements arising posterior to the bladder. At operation a large pelvic mass arising from the ileum along with the enlarged mesenteric lymph nodes was found and these were excised *en bloc* with end-to-end anastomosis of the small bowel. The histology of the tumour revealed it to be a benign leiomyoma measuring $10 \times 11 \times 12$ cm, showing areas of acute inflammation and cystic degeneration. The enlarged mesenteric lymph nodes contain reactive changes only.

Looking back through the patient's case notes, we found that the patient had been thoroughly investigated 20 years ago for iron deficiency anaemia when no cause was found. He was again referred with the same problem three years ago when various investigations including gastroscopy, sigmoidoscopy, and large and small bowel enemas failed to reveal anything abnormal.

Despite the fact that the patient's haemoglobin had continued to fall over the past three years, he had refused further investigations.

The commonest benign tumours of the small intestine are adenomas, leiomyomas and lipomas. They may produce varying clinical pictures depending on the anatomical site, size and morphological characteristics (box).² The present case is unusual because the tumour remained undetected having produced symptoms for 20 years. Contrast barium studies³ miss about 50% of these tumours, but enteroclysis⁴ is more sensitive in detecting the tumour and associated abnormalities, eg, thickening or separation of the bowel loops. The technique tests the distensibility of all small bowel segments, allows increased separation of small bowel loops with fewer missed lesions due to overlapping bowel, and has a double-contrast see-through effect for evaluating small bowel folds. Visceral angiography⁵ can demonstrate active bleeding, abnormal vasculature or ischaemia in a tumour, but the positive yield is extremely low either as a single study or as a repeat procedure; hence this investigation is superseded by a small bowel enema which can even detect subtle mucosal abnormalities. The role of enteroscopy⁶ in chronic gastrointestinal bleeding of obscure origin is still a contentious issue because of the low diagnostic yield from 30 to 70% depending on the experience of the operator. CT scan⁷ is 97% sensitive in detecting small intestinal neoplasm. It can also detect adjacent and remote changes associated with the tumour, eg, enlarged mesenteric lymph nodes, thickened bowel wall, and liver metastases but it cannot

Benign tumours of the small intestine: clinical features

- intestinal obstruction (42%)
- haemorrhage (34%)
- pain (22%)
- asymptomatic

detect active bleeding from the tumour. The role of the abdominal ultrasound is similar though it is less sensitive than CT.

Therefore, in conclusion we recommend that male and postmenopausal female patients with iron deficiency anaemia, in whom upper and lower alimentary endoscopy and small bowel barium studies are normal, should be further investigated by an abdominal CT scan with contrast and if this is negative, referred to a centre experienced in enteroscopy.

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Gestational macromastia not responding to termination of pregnancy

Sir,

In a non-obese female, macromastia has been referred to as massive enlargement of breasts which cause discomfort to the patient because of their weight and is associated with crusting and ulcer formation on the breasts. Rarely, it can occur with pregnancy (gestational macromastia).

A 35-year-old married female during her second pregnancy noticed progressive enlargement of both breasts from the tenth week onwards, the breasts becoming massive in the next few weeks. Examination revealed an emaciated patient with mild hirsutism (familial) and pallor. Abdomen examination revealed a uterine height of 16 weeks. Breast examination revealed bilateral enlargement with diffuse tenderness, crusting, and superficial ulcers on the right. Investigations revealed haemoglobin of 6.9 g/dl, bone marrow aspirate revealed evidence of dimorphic anaemia (nutritional). Ultrasonography of