answer his question because we did not feel justified in studying arterial gas tension and pH in healthy surgical patients.

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Bromocriptine as sole treatment for acromegaly

SIR,—There have been numerous reports of the use of bromocriptine in the treatment of acromegaly,¹⁻⁴ including a recent paper in the *BMJ* (22 September, p 700). Some of the patients have been treated with bromocriptine alone, and a reduction in size of the pituitary tumour has been demonstrated in one such patient by computed tomography⁴ and presumed in others following improvement in visual field defects.^{3,4} Changes in the size of the pituitary fossa on plain skull radiography have been reported only in patients who have received radiotherapy in addition to bromocriptine. We should therefore like to report a case of acromegaly treated with bromocriptine alone in which there has been evidence of a reduction in the size of the pituitary fossa on plain skull radiography.

The patient is a 58-year-old woman who initially presented in 1963 with symptoms of bilateral carpal tunnel compression. At this time she was noted to have features of acromegaly and gave a history of enlargement of her hands and feet over the previous seven years. On examination there was no evidence of optic nerve compression. Her skull radiographs showed unilateral expansion of the floor of the sella turcica on the left but carotid angiography and air encephalography were normal. The disease was thought to be inactive and she received no treatment but was seen regularly in the outpatient clinic until 1967, when she was lost to follow-up. During this time there was no progression in her symptoms or signs.

In September 1975, at the age of 53, she was referred back because of a history of headaches and excessive sweating for some months and a further increase in the size of her hands and feet. Again there was no clinical evidence of suprasella extension of the tumour. Her glucose tolerance test gave a normal result, but her fasting growth hormone concentration was raised at 53 µg/l. Skull radiographs showed enlargement of the pituitary fossa with thinning of the dorsum sella. Treatment was started with bromocriptine in a dosage of 10 mg a day; within a month her headaches and hyperhidrosis had improved and her serum growth hormone level had fallen to 13 µg/l. Bromocriptine treatment has been maintained ever since but no other treatment has been used.

Her skull radiograph was repeated in August 1976 and showed a reduction in the size of the pituitary fossa together with increased bone density and reossification of the dorsum sella (Dr B Worthington). Since starting treatment with bromocriptine there has been no further progression of her symptoms, although the dosage has been increased in stages to 20 mg a day because of occasional raised fasting growth hormone levels (8-43 µg/l). During a recent reassessment as an inpatient her visual fields were normal, her fasting growth hormone level was 5 µg/l, and skull radiographs confirmed reduction in size of the pituitary fossa and a normal dorsum sella. There was no evidence of suprasella extension on metrizamide cisternography.

Although a previous study⁴ has reported evidence of a reduction in the size of the pituitary fossa on plain skull radiography in acromegalic patients treated with bromocriptine, all these patients had been treated with radiotherapy in addition. Our patient

has received bromocriptine alone over a period of more than four years. The assessment of change in the size of the pituitary fossa on plain skull radiography is difficult because the views may not exactly coincide; however, in our patient there was both reduction in the size of the pituitary fossa and an increase in bone density with reossification of the dorsum sella.

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¹ Thorner MO, Chait A, Aitken M, et al. *Br Med J* 1975;i:299-303.

² Sachdev Y, Gomez-Pan A, Tunbridge WMG, Duns A, Weightman DR, Hall R. *Lancet* 1975;ii:1164-8.

³ Wass JAH, Thorner MO, Morris DV, et al. *Br Med J* 1977;i:875-8.

⁴ Wass JAH, Moutl PJA, Thorner MO, et al. *Lancet* 1979;ii:66-9.

Opiates in acute abdominal pain and head injury

SIR,—We fully agree with Mr D B Caro (6 October, p 863) that the provision of analgesia for patients with minor head injuries who have severe pain from other injuries is highly unsatisfactory. None the less, the dangers of the opiates in terms of changes in level of response, depression of pupillary reaction, and respiratory depression are real. It may be that small doses can be given intravenously by an experienced doctor, but only with highly intensive patient monitoring. Like Mr Caro, we wondered whether Entonox could be used as an analgesic in head injury, but found that it produced increases in intracranial pressure due, presumably, to the cerebral vasodilatory action of the drug which might be detrimental.¹ We would therefore caution against the use of Entonox in any patient who has suffered a head injury.

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¹ Moss E, McDowall DG. *Br J Anaesth* 1979;51:757-60.

Haemodialysis and transplantation in Wegener's granulomatosis

SIR,—We have read with interest the paper by Professor Charles van Ypersele De Strihou and others (14 July, p 93), in which two patients with Wegener's granulomatosis were reported as cured by the institution of dialysis followed by transplantation. They state that the complete remission of Wegener's disease in both cases clearly shows the value of dialysis and transplantation in such patients. Our experience with a patient with Wegener's granulomatosis is not in agreement with this.

A 31-year-old man presented at hospital in 1973 with deteriorating renal function. Chest radiography showed a small opacity in the right mid-zone and a renal biopsy was reported as showing acute glomerulonephritis. He was treated with a combination of cyclophosphamide, dipyrindimole, anticoagulants, which soon had to be abandoned because of side effects. Renal function did not improve, although the lung lesion disappeared. He was started on peritoneal dialysis and was soon afterwards established on home haemodialysis. Two years later he was readmitted to hospital with a cough and haemoptysis. Chest radiography revealed a large, rounded lesion containing a gas

fluid level in the right lung at the same site as the previous shadow. Tests for tuberculosis were negative and in August 1975 a right upper and middle lobectomy was performed. Histology confirmed the presence of Wegener's granulomatosis.

The patient was recommended on cyclophosphamide 100 mg/day and prednisone 10 mg/day. These drugs were continued until the following year, when he received a cadaveric renal transplant. He was then treated with azathioprine, prednisolone, and nifedazole. No rejection episodes were experienced and he was discharged home in August of that year taking azathioprine 100 mg/day and prednisolone 40 mg/day. For three years he remained in good health taking azathioprine 125 mg/day and prednisolone 15 mg/day. However, in November 1979 he developed a severe cough and breathlessness and was readmitted to hospital. Chest radiology demonstrated a very large lesion in the right lung with a cavity within it. This lesion has regressed once again following the reintroduction of cyclophosphamide and an increase in the steroid dosage.

The Wegener's granulomatosis which affected this man's lung recurred while he was on haemodialysis (but off treatment) and also after he had received a cadaveric renal transplant and was receiving routine immunosuppressive drugs. It cannot be said therefore that dialysis or transplantation were effective in preventing recurrent disease; rather it would appear that long-term cyclophosphamide therapy is required if disease recurrence is to be avoided. Certainly azathioprine was not an effective substitute in this case.

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Pathogenesis of acute renal failure

SIR,—Your leading article on acute renal failure (5 January, p 2) after major surgery may be factual but it surely conveys the impression that little can be done to prevent renal failure or to reduce the high mortality apart from care in prescribing nephrotoxic antibiotics. I differ from you in the emphasis that I would place on endotoxaemia as a causative factor.¹ My own observations suggest that it is common.² Indeed, I am surprised that so far no other group has published results on the incidence of endotoxaemia in a series of patients with acute renal failure. Endotoxaemia is logical because its experimental counterpart, the Schwartzman reaction, has been well studied and validated, and moreover we now know that many persons in the general population have only low anti-lipid-A (antitoxin) antibodies.³ The way should thus be open to new approaches to this refractory complication of surgery.

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¹ Wardle EN. *Scot Med J* 1976;21:83-91.

² Wardle EN. *Q J Med* 1975;44:389-98.

³ Wardle EN. *Br J Hosp Med* 1979;22:104.

Treatment of liposclerosis

SIR,—Mr K Burnand and colleagues (5 January, p 7) may be misinterpreting their data in attributing an increased rate of healing of liposclerosis to an effect of stanozolol. In comparing mean areas that were healed by the two forms of treatment, they assume that the rate of healing is in-