Fortnightly Review

Diagnosis and management of pituitary tumours

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Pituitary tumours are mostly benign epithelial neoplasms that result from mutation and subsequent clonal expansion of single adenohypophyseal parenchymal cells.[1] They account for 10-15% of intracranial neoplasms, and three quarters of them secrete inappropriate amounts of pituitary hormones. Although residual cells in parasellar structures may account for local recurrences that follow seemingly complete surgical clearances, metastatic spread and direct macroscopic invasion of surrounding structures is fortunately rare.[2]

Most patients with pituitary adenomas present with symptoms and signs of hormone hypersecretion, visual field defects, headaches, and hypopituitarism either alone or in combination (see box 1). Preoperative diabetes insipidus is extremely rare in primary pituitary disease and suggests involvement of the hypothalamus or pituitary infarction. Any visual disturbance can occur—from changes in the lateral fields that a patient typically finds hard to describe and which may go unrecognised for years to scotomas and complete blindness in one or both eyes. Many patients complain of vague peripheral shadows confined to the upper quadrants. Headaches are usually non-specific—annoying more than disabling and frequently helped by analgesia. In some patients they are relieved almost immediately by somatostatin or somatostatin analogues, only to recur as the peptide is cleared from the circulation. If panhypopituitarism is suspected (see box 2 for typical features) glucocorticoids should be replaced before thyroid hormones.

Diagnosis is usually clear after taking a patient's history and making an examination and can be confirmed by a scan of the pituitary and appropriate blood tests. Except for adrenocorticotrophic hormone, which must be collected on ice and separated as soon as possible, pituitary hormones are stable for several days in plasma at room temperature. Plasma samples can therefore be pooled before analysis to give mean hormone concentrations.

Box 1—Typical presenting features of pituitary adenomas

- Hormone hypersecretion
- Visual field defects
- Headaches
- Hypopituitarism
- Pituitary apoplexy
- Hydrocephalus
- Cranial nerve palsies
- Temporal lobe epilepsy

Summary points

- Pituitary tumours are mainly benign epithelial neoplasms; three quarters of them secrete inappropriate amounts of pituitary hormones
- Diagnosis is usually clear after examination and from patient's history and can be confirmed with scan of pituitary (by computed tomography or preferably magnetic resonance imaging) and suitable blood tests
- Except for prolactinomas, which are usually treated with dopamine agonist bromocriptine, first line treatment for adenomas that hypersecrete or cause mass effects is usually transsphenoidal surgery
- Standard radiotherapy halves rate of tumour recurrence but is now less usually given as adjunct to surgery because of reports of adverse effects
- Occult pituitary adenomas are found in up to 20% of random postmortem examinations

Visual field testing

The presence and rate of change of visual field defects profoundly affects the management of pituitary adenomas. Field examination by confrontation is insensitive and should be followed by formal analysis. Goldman perimetry has long been the standard method but is highly dependent on the operator. Computerised field testing with a field analyser (Allergan Humphrey) gives highly reproducible results that are virtually independent of the operator. The procedure takes about 15 minutes for each eye, and it can detect and measure subtle and surprisingly peripheral lesions when set to analyse visual threshold within a 60° cone around a central point of fixation (central 30-2 threshold test). A major advantage is that, with the same software, machines at different locations produce similar results. Multifixation campimetry (formerly opticokinetic perimetry), conceived and developed by B E Damato at Royal Liverpool University Hospital, is a rapid hand held test in which fixation is led outwards in a spiral of numbered points around a central static target. After fixating at each point the patient reports whether the central target remains visible. Preliminary findings suggest that the results correlate well with computerised field testing, and the test is ideal for use in general practice as well as hospital clinics.

Pituitary imaging

Computed tomography and more recently magnetic resonance imaging have revolutionised pituitary imaging. Unlike computed tomography, magnetic resonance imaging can be performed repeatedly without risk of excessive exposure to x rays and shows surrounding soft tissue structures such as the pituitary stalk and optic chiasm (figure). However, some patients find the noise, confined space, and long duration of magnetic resonance imaging uncomfortable, and calcified lesions such as craniopharyngiomas may be more easily identified with computed tomography. Enhancement of pituitary magnetic resonance imaging with gadolinium and diethylenetriaminepentaacetic acid (DTPA) is helpful for small microadenomas, and

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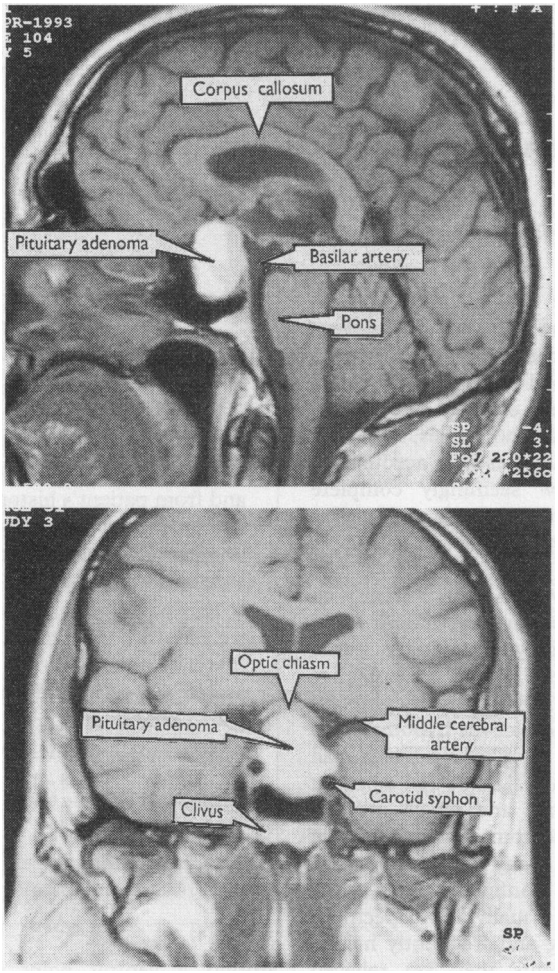
at high image capture rates the technique may in future be able to replace formal angiography. The sensitivity of detection of surgically proved microadenoma approaches 100% with magnetic resonance imaging compared with 50% with computed tomography, but 17% of pituitary lesions causing Cushing's disease are too small or diffuse to be reliably identified by either technique.



Sagittal (top) and coronal (bottom) views by magnetic resonance imaging of patient with large pituitary adenoma. Glairy appearance (without enhancement) because of recent bleed that acutely compressed optic chiasm; sella floor is at normal level, about parallel with junction of middle and upper third of pons. Pictures courtesy of Dr Julian Kabala

Surgical treatment

Except for prolactinomas, first line treatment for pituitary adenomas that hypersecrete or cause mass effects is usually transsphenoidal surgery.[3] This should always be preceded by measurement of prolactin and thyroxine concentrations at least and should be covered with either hydrocortisone or dexamethasone. With the most skilled operators abnormal visual fields improve in 80% of patients, and progression of visual deterioration is arrested in a further 16%. The introduction of endoscopic instruments and computerised real time, three dimensional image processing, which provides accurate positional information during the operation, is likely to increase the efficacy of surgery, particularly when local landmarks have been lost or disturbed by previous explorations. Transcranial surgery is reserved for tumours that cannot be resected transsphenoidally.

Radiotherapy

Standard fractionated supervoltage radiotherapy with 20-25 treatments over four to six weeks halves the rate of tumour recurrence and is often carried out as an adjunct to surgery.[4] This is becoming less usual because of anecdotal reports of low grade neuronal damage, radionecrosis, vaso-occlusive disease, and secondary tumours of the central nervous system such as anaplastic meningiomas. Furthermore, ready access to magnetic resonance imaging for regular post-operative scans allows tumour recurrence to be diagnosed early so that further surgery can be contemplated. The use of high energy particle beam radiotherapy has not yet been widely evaluated.

Box 2—Typical features of hypopituitarism

Hypogonadism

Women
- Oligomenorrhoea or amenorrhoea
- Reduced libido
- Dyspareunia

Men
- Reduced libido and potency
- Reduced facial and body hair
- Gonadal atrophy

Hypoprolactinaemia
- Failure to start and maintain lactation in women
- No defined clinical entity in men

Hypothyroidism
- Mild hypothyroidism with inappropriately low concentrations of thyroid stimulating hormone in otherwise well patient (should not be confused with sick euthyroid syndrome; depressed central thyroid axis drive in ill and catabolic patients)

Growth hormone deficiency
- Poorly defined clinical entity in adults
- Reduced body muscle:fat ratio

Hypoadrenalism
- Tiredness and malaise, especially in afternoon and evening
- Postural hypotension, pallor, anorexia, and nausea
- No substantial electrolyte changes as adrenal zona glomerulosa remains intact
- Loss of secondary sexual hair in women

Prolactinomas

Prolactinomas are the most common functional pituitary adenoma and are responsible for a quarter of the occult pituitary adenomas diagnosed at necropsy. All patients with hyperprolactinaemia (see box 3 for symptoms) that does not have an obvious cause (see

Box 3—Typical features of hyperprolactinaemia

Women
- Amenorrhoea, oligomenorrhoea, or normal periods with infertility
- Galactorrhoea
- Decreased libido
- Vaginal dryness and dyspareunia
- Delayed menarche

Men
- Decreased libido
- Impotence
- Galactorrhoea
- Reduced growth of facial and body hair
- Small, soft testes
- Apathy
- Weight gain

box 4) warrant further investigation by magnetic resonance imaging or computed tomographic scanning of the pituitary fossa. A prolactin concentration of over 5000 mU/l suggests a macroprolactinoma, while a macroadenoma with a prolactin concentration of less than 2500 mU/l is more likely to result from compression of the pituitary stalk by an endocrinologically inactive adenoma.

Box 4—Causes of hyperprolactinaemia

- Pregnancy and lactation
- Treatment with dopamine receptor antagonist
- Stress or anxiety
- Exogenous oestrogen treatment
- Breast manipulation
- Microprolactinomas
- Primary hypothyroidism
- Macroprolactinomas
- Compression of pituitary stalk
- Polycystic ovarian syndrome

TREATMENT

Bromocriptine (1·25-50 mg/day) reduces galactorrhoea, restores menstruation, and returns serum prolactin concentration to normal in most patients,[5] and diminished visual fields improve in at least 75% of affected patients. Unfortunately, nausea and vomiting, fatigue, mood changes, and other side effects can be dose limiting but may be minimised by starting with a low dose at bedtime or by taking tablets intravaginally. If intolerance persists useful alternatives are pergolide (50 μg-1 mg thrice daily), cabergoline[6] (125 μg-1 mg orally twice weekly), or quinagolide (formally CV205-502, 75-600 mg in divided doses daily; Sandoz, Camberley, Surrey). Although there is no evidence of teratogenicity, most doctors stop bromocriptine when pregnancy is diagnosed and monitor visual fields to identify the 16% of macroadenomas that grow substantially during this time. Those that increase in size rarely cause important problems, and bromocriptine can be reinstituted if required.

Microprolactinomas—Long term treatment with bromocriptine or an alternative dopamine agonist is the most common treatment for microprolactinomas. In some neurosurgical centres, however, transphenoidal surgery is the treatment of choice, although in most endocrine units it is generally reserved for patients who cannot tolerate dopamine agonists or whose microprolactinomas are unresponsive to them.[7] The advantage of microadenomectomy is that, even though it carries the risks of hypopituitarism and tumour recurrence, it is potentially curative unlike dopamine agonists.

Macroprolactinomas—Surgery is rarely curative for macroadenomas and often results in hypopituitarism. Dopamine agonists are the treatment of choice as they return serum prolactin to normal in over 75% of patients and result in long term shrinkage of most tumours.[5]

Box 5—Typical features of acromegaly

- Growth of hands and feet
- Coarse facial features
- Jaw growth and malocclusion
- Headache and arthralgia
- Hypertrichosis
- Excessive sweating
- Tiredness, weakness, and somnolence
- Impaired glucose tolerance
- Carpal tunnel syndrome
- Hypertension and cardiomegaly

Acromegaly

The diagnosis of active acromegaly is based on continuing somatic changes (see box 5) and is confirmed by measurement of raised basal growth hormone concentrations on two or more occasions, particularly with a raised concentration of insulin-like growth factor I (which in otherwise fit patients correlates reasonably well with mean secretion of growth hormone in 24 hours). Borderline cases require dynamic tests such as failure of a glucose load to suppress growth hormone secretion or a paradoxical secretory response of growth hormone to thyrotrophin releasing hormone. A comprehensive protocol is to measure growth hormone every 10 minutes for one hour, every 30 minutes for two hours after ingestion of 75 g of glucose, and every 30 minutes for a final hour after an intravenous bolus of 200 μg thyrotrophin releasing hormone. Acromegaly is indicated by a mean (pooled) basal concentration of growth hormone of 5 mU/l or more (certainly of >10 mU/l), failure to reduce growth hormone concentration to less than 2 mU/l after the glucose meal, or paradoxical growth hormone secretion (concentration increased by >6 μg/l or to $>150\%$ of basal) in response to thyrotrophin releasing hormone. Measurement of insulin-like growth factor binding protein I, which is low in acromegaly, and binding protein III, which is high, may become routine diagnostic procedure, but other tests for releasing hormone do not contribute to the diagnosis. As with all pituitary tumours, pituitary imaging—preferably magnetic resonance imaging—is needed to visualise the tumour and to assess its suitability for surgery.

TREATMENT

Transsphenoidal surgery reduces circulating growth hormone to less than 5 μg/ml in about 60% of patients. Normal pulsatile secretion of growth hormone does not always recur, however, and 20% of patients still secrete growth hormone in response to thyrotrophin releasing hormone.[8] A growth hormone concentration of 2-5 μg/l during a postoperative glucose load is associated with a 20% rate of recurrence. Repeat operations reduce growth hormone concentration to less than 5 μg/l in less than one third of cases.

Conventional radiotherapy alone produces a yearly fall in growth hormone concentration of 15-20%, improves headaches in 75% of patients, and reduces the risk of further visual loss related to the tumour. Treatment with α radiation is probably as good as surgery in some centres, but it takes two to four years for growth hormone concentration to fall to less than 2·5 μg/ml after a glucose load and concentrations are still high after 10 years in half of patients. Radiotherapy may also eventually result in hypopituitarism.[9]

Oral bromocriptine may reduce growth hormone secretion in some patients but alone rarely provides adequate control except in mild cases and in some tumours that cosecrete prolactin and growth hormone.[10]

Somatostatin analogues (such as octreotide 100-500 μg by subcutaneous injection thrice daily) have displaced dopamine agonists as first line medical therapy for somatotroph adenomas.[11] Formulations of more than one somatostatin analogue with longer action are now being evaluated. Initial claims of more than 20% reduction in tumour volume in 44% of patients may have been overoptimistic, but modest shrinkage is seen in some patients. Circulating growth hormone is reduced in more than 80% of patients with concurrent stimulation of insulin-like growth factor binding protein I, which may explain the dramatic clinical effects in cases where the decrease in circulating insulin-like growth factor I is marginal. Gall stones develop in up to half of patients on long term treatment but often resolve after stopping treatment, and preliminary evidence suggests that they may be prevented by concurrent treatment with ursodeoxycholic acid or with drug free interludes that allow normal gall bladder contraction.

Cushing's disease

Screening for Cushing's disease (see box 6 for symptoms) is most easily performed by measuring urinary cortisol concentration and with the dexamethasone

Box 6—Typical features of hypercortisolaemia (Cushing's disease)

- Weight gain and truncal obesity (in 95% of subjects)
- Round face (95%)
- Plethora (94%)
- Menstrual irregularity (84%)
- Hirsutism (81%)
- Thin skin and easy bruising (62%)
- Depression and mental changes (62%)
- Purple striae (56%)
- Muscle weakness (56%)
- Oedema (50%)

suppression test. Patients are asked to collect three sequential 24 hour urine samples for measurement of free cortisol concentration and to come to the clinic at 9 am for measurement of circulating cortisol after having ingested a single dose of 1 mg dexamethasone at 11 pm the previous evening. Values vary among laboratories, but urinary free cortisol concentrations exceeding about 275 nmol/24 h and failure to suppress circulating cortisol concentration to less than 138 ng/l require further investigation. In Cushing's disease feedback inhibition of release of adrenocorticotrophic hormone by circulating glucocorticoids remains intact but at a higher set point. The formal dexamethasone suppression test therefore remains the best means of diagnosis. For the formal test the patient is admitted to hospital, and a blood sample is taken at 11 pm on the day of admission for measurement of cortisol and adrenocorticotrophic hormone concentrations. This is repeated at 8 am daily for the next four days with sequential 24 hour collection of urine (8 am to 8 am) for measurement of free cortisol. Oral dexamethasone 0·5 mg is given every six hours for the first two days and increased to 4 mg for the last two days. In a normal response the circulating cortisol concentration falls below 138 ng/l and urinary free cortisol falls below 70 nmol/24 h. Once increased cortisol secretion has been confirmed, any consistent suppression of urinary cortisol concentration—no matter how small—by oral dexamethasone suggests pituitary disease,[12] while complete failure of suppression suggests primary adrenal disease or ectopic production.

The concentration of adrenocorticotrophic hormone is higher than normal (>5·5 pmol/l at 9 am and >2·2 pmol/l at midnight) in more than half of patients with Cushing's disease but is considerably lower than that found in classic ectopic production of adrenocorticotrophic hormone (>55 pmol/l). However, ectopic production of adrenocorticotrophic hormone from carcinoid tumours may be very difficult to distinguish from true Cushing's disease. In adrenal tumours, secretion of adrenocorticotrophic hormone is usually inhibited to undetectable levels. In patients with documented hypercortisolaemia increased secretion of adrenocorticotrophic hormone in response to a 100 μg bolus of corticotrophin releasing hormone suggests Cushing's disease. This test is particularly useful if performed at the time of bilaterally sampling the pituitary at its inferior petrosal aspect: an increase in petrosal adrenocorticotrophic hormone concentration occurs in Cushing's disease but not in ectopic adrenocorticotrophic hormone secretion, and the result may help to determine in which side of the pituitary the adenoma lies. Other tests such as insulin induced hypoglycaemia, which increases cortisol concentration in extreme obesity and endogenous depression but not in Cushing's disease, and the metyrapone test are rarely of any additional value.

TREATMENT

Transsphenoidal adenomectomy is the first line treatment for Cushing's disease caused by a microadenoma and is curative in up to 80% of patients.[13] The pituitary is irradiated in the remaining 20% to try to prevent Nelson's syndrome.

Bilateral adrenalectomy remains a useful treatment for patients who are not cured by surgery, although pituitary radiation must also be given to limit the development of Nelson's syndrome.

Radiotherapy alone has been shown to cure 40% of patients aged 18 or over and 80% of those aged under 18. In children pituitary radiotherapy and adrenalectomy is highly effective.[14]

Drug treatment only has a supportive role in Cushing's disease. The most commonly used drug, metyrapone, blocks 11-β-hydroxylation in the adrenal glands and can usually be given in increasing doses until cortisol hypersecretion is controlled. However, side effects may be troublesome and include nausea, oedema, somnolence, and hypertension. It is particularly useful to render patients euadrenal before surgery. Other drugs such as cyproheptadine, ketoconazole and mitotane, mifepristone, aminoglutethimide, and bromocriptine have only limited use.

Adenomas secreting glycopeptide hormones

SECRETING THYROTROPH ADENOMAS

These are rare, aggressive tumours that produce clinical thyrotoxicosis in about 1 in 500 pituitary tumours that require surgery. The diagnosis may be missed if it is not appreciated that a concentration of thyroid stimulating hormone in the normal range is inappropriately high if circulating thyroid hormones are consistently above the upper limit of normal concentrations.

Treatment—Somatostatin analogues are likely to usefully reduce hormone secretion from thyrotroph adenomas and may reduce tumour size. However, these adenomas often escape from the inhibitory effects of somatostatin analogues, although these drugs are dramatically effective in the short term, and tend to behave aggressively. Surgery therefore remains the first line treatment.

GONADOTROPH ADENOMAS

These usually present as inactive adenomas but may very rarely present with premature puberty or the resumption of menstrual bleeding in postmenopausal women. However, free α and β subunits of follicle stimulating hormone are secreted by 7% and 15% of these adenomas respectively. Also, 40% of inactive adenomas give a paradoxical release of gonadotrophin subunits in response to thyrotrophin releasing hormone (measured at 0, 30, and 60 minutes after a 200 μg intravenous bolus of hormone), which may help to distinguish these tumours and may be useful when there is doubt about whether the lesion originated from the pituitary or from a suprasellar mass.[15] Mild hyperprolactinaemia secondary to compression of the pituitary stalk occurs in 80% of endocrinologically inactive adenomas, and secondary hypopituitarism is common by the time the tumour presents with signs of occupying space.

Treatment—Transsphenoidal hypophysectomy is first line treatment for endocrinologically inactive adenomas and gonadotrophinomas, although transfrontal surgery may be necessary for large tumours with lateral extension. Superagonists of luteinising hormone releasing hormone are not useful, but anecdotal reports of long acting somatostatin analogues dramatically reducing the volume of some endocrinologically inactive adenomas suggest that they may be worth trying. Dopamine agonists have also been

reported to decrease secretion of glycoprotein hormone subunits but rarely affect tumour size.

Incidentalomas

The sensitivity of computed tomography and magnetic resonance imaging has largely been responsible for establishing the "incidentaloma" as a new clinical entity. Occult microadenomas or macroadenomas have been found in as many as one in five of sequential necropsies of patients dying from unrelated causes. Incidental identification of a pituitary mass is said to occur in over 10% of patients undergoing cranial imaging for unrelated reasons (patients' mean age 69 years). Focal hypodensities are present in one third of normal women undergoing computed tomography, and a partially empty sella is present in 18%. The natural history of incidentalomas, particularly those less than 10 mm in diameter, is benign, but they do need occasional follow up.

Box 7—Unresolved issues in diagnosis and management of pituitary adenoma

- Cost effectiveness of surgery compared with long term bromocriptine treatment for microprolactinomas
- First line use of long lasting somatostatin analogues in acromegaly
- Role of growth hormone replacement in adults with panhypopituitarism
- Long term secondary effects of radiotherapy
- Use of α particle treatment instead of conventional radiotherapy
- Use of indium labelled somatostatin and somatostatin receptor analogues in diagnosis

Follow up

At one month or more after an operation, further glucocorticoid replacement is unnecessary in the short term if circulating cortisol concentration increases to over 495 nmol/l in response to an intravenous bolus of 250 μg tetracosactrin—given 24 hours after stopping hydrocortisone (typically 15 mg in the morning and 5-10 mg in the evening). If a patient has had radiotherapy the tetracosactrin test will need to be repeated every one to two years initially as a yearly reduction in hormone release of 15-20% can be expected.

A low concentration of free thyroxine indicates the need for thyroid hormone replacement. A low testosterone concentration associated with sexual dysfunction is treated with intramuscular testosterone (100 mg every two weeks or 250 mg every three to four weeks) as oral supplements are poorly absorbed and transdermal testosterone replacement is not yet available in Britain. Women are given conventional sex hormone replacement. When fertility is required both sexes require parenteral gonadotrophins.

Frequency of follow up depends on the size of the residual tumour, the presence or absence of residual visual field defects, and the possibility of further endocrine loss following radiotherapy. If there are no changes in pituitary imaging, hormonal status, or visual fields in the first two to three years after the operation magnetic resonance imaging can eventually be limited to once every three to five years.

1 Levy A, Lightman SL. The pathogenesis of pituitary adenomas. *Clin Endocrinol* 1993;38:559-70.
2 Mountcastle RB, Roof BS, Mayfield RK, Mordes DB, Sagel J, Biggs PJ, *et al.* Pituitary adenocarcinoma in an acromegalic patient: response to bromocriptine and pituitary testing: a review of the literature on 36 cases of pituitary carcinoma. *Am J Med Sci* 1989;298:109-18.
3 Wilson CB. Role of surgery in the management of pituitary tumors. *Neurosurg Clin N Am* 1990;1:139-59.
4 Tran LM, Blount L, Horton D, Sadeghi A, Parker RG. Radiation therapy of pituitary tumors: results in 95 cases. *Am J Clin Oncol* 1991;14:25-9.
5 Bevan JS, Webster J, Burke CW, Scanlon MF. Dopamine agonists and pituitary tumor shrinkage. *Endocr Rev* 1992;13:220-40.
6 Webster J, Piscitelli G, Polli A, D'Alberton A, Falsetti L, Ferrari C, *et al.* Dose-dependent suppression of serum prolactin by cabergoline in hyperprolinaemia: A placebo controlled, double blind, multicentre study. *Clin Endocrinol* 1992;37:534-41.
7 Molich ME. Management of prolactinomas. *Annu Rev Med* 1989;40:225-32.
8 Losa M, Oeckler R, Schopohl J, Albrecht Muller I, Alba-Lopez J, von Werder K. Evaluation of selective transsphenoidal adenomectomy by endocrinological testing and somatomedin-C measurement in acromegaly. *J Neurosurg* 1989;70:561-7.
9 Littley MD, Shalet SM, Beardwell CG. Radiation and hypothalamic-pituitary function. *Baillieres Clin Endocrinol Metab* 1990;4:147-75.
10 Jaffe CA, Barkan AL. Treatment of acromegaly with dopamine agonists. *Endocrinol Metab Clin North Am* 1992;21:713-35.
11 Lamberts SW, Hofland LJ, de-Herder WW, Kwekkeboom D J, Reubi JC, Krenning EP. Octreotide and related somatostatin analogs in the diagnosis and treatment of pituitary disease and somatostatin receptor scintigraphy. *Front Neuroendocrinol* 1993;14:27-55.
12 Orth DN, Kovacs WJ, DeBold CR. The adrenal cortex. In: Wilson JD, Foster DW, eds. *Williams textbook of endocrinology.* Philadelphia: Saunders, 1992:489-619.
13 Ludecke DK. Transnasal microsurgery of Cushing's disease 1990. Overview including personal experiences with 256 patients. *Pathol Res Pract* 1991;187:608-12.
14 Jones KL. The Cushing syndromes. *Pediatr Clin North Am* 1990;37:1313-32.
15 Snyder PJ. Clinically nonfunctioning pituitary adenomas. *Endocrinol Metab Clin North Am* 1993;22:163-75.

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Correction

Impotence: diagnosis and management of male erectile dysfunction

A printer's error occurred in this article by Roger S Kirby (9 April, pp 957-61). In the second paragraph on p 960 the dose of prostaglandin E1 should be 20-40 μg [not 20-40 mg).

A PATIENT WHO CHANGED MY PARISH

An instant conversion

The church to which I had been appointed to serve my title as a curate was an ancient parish church in a village which had been swallowed up by a large housing estate. Although most of the population lived on the estate, the congregation attracted several well to do people who remembered the village as it had been years ago. On the first Sunday after my ordination I was instructed by the vicar to stand by the main door at the end of the service and to introduce myself to the congregation. A tall elderly lady bore down on me. As I shook her hand I noticed that her fingers were covered with bandages. "Yes," she said, "My doctor says it's septic arthritis." I glanced at her face. Beneath the expensive hat and above the gold earring there was a tiny white nodule on the pinna of her ear. "I wonder," I remarked, "whether it could be gout?"

I realised immediately that this was injudicious. Clearly, gout for Mrs C implied a life of dissipation. She drew herself up to her full height and stalked off down the church path. On my first Sunday I had succeeded in alienating an influential parishioner. But her doctor must have thought enough of my diagnosis to arrange a blood test, for by the following Friday I received a telephone call from the lady herself: "I've got gout. I've got gout." With appropriate treatment her tophi vanished quickly. From that time on I had no warmer supporter in the parish and no more active unlocker of doors than Mrs C.—KEITH LEIPER *was a general practitioner and is now a clergyman in Lancashire*