

tests has been called into question. In 105 patients with proved tumours we found tone decay in 25 (24%), abnormal loudness balance in 71 (68%), and abnormal speech audiometry in 49 (47%). Although better, the stapedial reflex threshold and decay parameters were normal in 17 (16%) of these patients. Even the auditory brainstem response has its limitations. In most series it has been found to be a sensitive test (only 2% of these patients had a normal auditory brainstem response), but Lai *et al* recently reported an incidence of 24% normal or equivocal auditory brainstem responses in patients with proved tumours.¹ Furthermore, the specificity of the test is very poor.

Radiological diagnosis is now highly reliable. Computed tomography with contrast will show most intracranial lesions. Magnetic resonance imaging enhanced with gadolinium diethylenetriaminepentaacetic acid (Gd-DTPA) will reveal all tumours regardless of size.⁴ We believe that there is an overwhelming case for all patients with unilateral audiostimulatory symptoms for which there is no other plausible explanation to be examined with computed tomography or magnetic resonance imaging. Computed tomography is now available in most parts of Britain, and the same will soon be true of magnetic resonance imaging, which is the optimal tool for neuro-otological diagnosis.

Our final point concerns cost. Financial audit in such matters is a firmament full of black holes. We accept the calculations of Drs Swann and Gatehouse, but there are certain omissions. Moffat *et al*⁵ made the point that the failure to diagnose a small tumour may make the difference between a patient who rapidly returns to work and one who is in need of state support for years. Lastly, we cannot ignore the medicolegal climate. An increasing number of suits are being filed for failed diagnosis of acoustic neuroma. Health authorities will have to consider this cost with as much concern as the cost of neuroradiological imaging techniques.

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Terodiline for treating detrusor instability in elderly people

SIR,—Terodiline has anticholinergic (selectively antimuscarinic) and calcium channel blocking activity and has been advocated for the treatment of urinary frequency and urge incontinence. Although it is widely prescribed, Dr Penelope Wiseman and colleagues could not show that it had any advantages over placebo in elderly patients with detrusor instability.¹

In the past 12 months we have treated two patients, aged 76 and 78, who were referred for consideration of permanent pacing; each was receiving terodiline 12.5 mg twice daily for urge incontinence. In each case 24 hour Holter monitoring showed prolonged episodes of atrioventricular dissociation with ventricular rates of 30-40 beats/minute. After terodiline was stopped repeat 24 hour recordings showed sinus rhythm through-

out with only marginal prolongation of the PR interval and left axis deviation in each case. The manufacturer's datasheet lists tachycardia but not bradycardia among the side effects,² although bradycardia and hypotension are described as features of overdosage, being attributed to pre-dominance of the drug's calcium antagonist effects over its anticholinergic actions.

It is important to appreciate that even standard doses of terodiline may precipitate atrioventricular dissociation in elderly patients who already have some features of conducting system disease. This may further alter the balance of possible risks to benefits to be considered when prescribing this drug.

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Referral for suspected glaucoma

SIR,—Messrs Maurice W Tuck and Ronald P Crick raise again the problem of direct referral of patients with suspected glaucoma from an optometrist to an ophthalmologist.¹ Many patients with raised intraocular pressures are referred initially by their optometrist.² The alarmed patient then has to trudge wearily and increasingly nervously to the general practitioner to obtain a further referral (often just a signature on the sight test form) to the local ophthalmologist. The system is clearly flawed, causing distress to the patient and wasting the general practitioner's time.

A sensible solution, hinted at by Messrs Tuck and Crick, would be to permit direct referral provided that the optometrist has performed all three main tests for glaucoma—tonometry, ophthalmoscopy, and perimetry—and that he or she notifies the general practitioner independently. The present system of referral through the general practitioner would otherwise apply. This would have the benefit of providing the ophthalmologist with an initial or "baseline" set of investigations, and would provide an incentive to optometrists to equip themselves adequately and to perform these tests routinely. With greater competition among high street optometrists, providing such services is likely to become increasingly important.

Close cooperation locally would allow quicker referral for the patient, enhanced professional responsibility for optometrists, better information for the ophthalmologist, and a reduction of the general practitioner's workload.

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No blood, no drug

SIR,—Dr Fred Charatan detailed the recent developments in the United States concerning the Clozaril Patient Management System (CPMS), and this news item was followed by a brief footnote on the situation in Britain.¹ The United Kingdom Clozaril Patient Monitoring Service is completely separate from the monitoring system in the United

States (though sharing the same safety objective of prompt detection of blood dyscrasia) and has been codeveloped by Sandoz Pharmaceuticals (UK) and leading representatives of the psychiatric and pharmacological professions in accordance with the requirements of health care services in the United Kingdom.

As a fully licensed medicine, Clozaril (clozapine) was first introduced into the United Kingdom in January 1990 for schizophrenia resistant to treatment, accompanied by the centralised safety service that individually registers all psychiatrists and pharmacists who prescribe clozapine and also each patient for whom treatment is intended. After an initial screening patients are formally monitored throughout the duration of their treatment with clozapine. The service provides the necessary materials to send regular blood samples (weekly at first and fortnightly after 18 weeks of treatment) to the CPMS laboratory for the standardised safety analysis (including white blood cell differential count). Where this screening detects an abnormal result the patient's care team is immediately notified to ensure that clozapine treatment stops and any necessary supportive measures are promptly instituted. A full 24 hour expert back up advisory service is provided. If a patient is withdrawn from clozapine the monitoring continues for a further four weeks. The monitoring service helps ensure that a patient is not inadvertently rechallenged with clozapine after withdrawal due to a blood abnormality.

In addition to the safety that this assurance provides for individual patients, the Clozaril Patient Monitoring Service enables the full safety profile of clozapine to be continuously observed by the appropriate professional bodies, including the drug safety regulatory authorities.

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What is a normal upper gastrointestinal tract?

SIR,—Drs D G Colin-Jones and P L Golding averred that "a normal upper gut does what is asked of it without complaint."¹ Were the behaviour of the lower gastrointestinal tract included, for continuity's sake, then the normal gastrointestinal tract could be regarded as that least susceptible, at the extreme, to gastric cancer and to chronic bowel diseases, including appendicitis, diverticular disease, and colorectal cancer. In this respect, rural Africans can claim merit. Until very recently gastric disease was rare and bowel diseases nearly absent. In 1986 at Murchison Hospital, Natal, of 136 patients with malignancies from a rural Zulu population of a quarter of a million there was one case of stomach cancer and were no cases of colorectal cancer.²

Even in cities, although dyspepsia and gastritis are becoming increasingly common, gastric cancer remains uncommon; moreover, the incidence of chronic bowel diseases has risen only slightly³—which is puzzling because the diet of urban black people now includes 25-35% of energy from fat, and fibre intake, now 10-15 g daily, has fallen considerably. What is inhibiting rises in gastrointestinal diseases? Regarding chronic bowel diseases, a large proportion of ingested starch from maize, the staple cereal, reaches the colon, ferments, and hence contributes to inhibit disease development.⁴ Faecal pH value has scarcely risen,⁵ thereby also conferring a measure of protection.⁶ Early local studies indicated absence of polyps in