LETTERS TO THE EDITOR

Drug treatment for depression in patients inside rehabilitation wards

EDITOR,—Shah and colleagues report on the prevalence of psychiatric disorders affecting elderly people institutionalised in a rehabilitation unit.[1] They conclude that depression is common among older adults, it is a treatable condition, and that in cases where there are cognitive impairments associated with concomitant depression, the cognitive impairments are worsened by the depressive disease. Although the high prevalence of cognitive impairments and depression in these patients is important and it is necessary to call attention to it, I believe that from the psychiatric perspective, the problem has been oversimplified. Therefore, we cannot accept the elementary proposition of Shah *et al* about a drug trial with selective serotonin reuptake inhibitors (SSRIs) to elucidate the correct diagnosis when diagnostic doubts remain.

Depressive states in older patient groups have a tremendous clinical heterogeneity. Beside the depressive elderly patient without complicated somatic pathology or psychiatric co-morbidity, that will respond adequately to antidepressant drugs, a significant proportion of other symptomatic depressed patients (whose cases are precisely studied by Shah and colleagues in their article) can be considered as follows.

(1) Elderly depressed patients who may show greater cognitive deficits compared with age similar normal subjects.[2] These patients also present (subcortical) dysfunction of learning and memory, comprising the so-called "depressive pseudodementia", and may show reversible cognitive deficits after successful somatic treatment of depression.

(2) Patients who display cognitive deficits characterised by severe prefrontal dysfunction, with perseveration, psychomotor retardation, and long P300 latency.[3]

(3) Patients who present features of depression that are related to underlying vascular disease and neurological lesions, corresponding to the hypothesised "vascular depression".[4]

(4) Patients with late life onset of cognitive deficits, psychomotor retardation and limited depressive ideation, many of whom will have the apathy syndrome[5] that frequently follows brain damage in caudate, putamen and thalamus, usually secondary to cerebrovascular heterogeneous diseases (and which may overlap with the previously mentioned third subgroup).

While SSRIs may be useful to the first subgroup, they are useless in the second and third groups, while in the fourth, dopamine agonists like bromocriptine are required. Hence, it is erroneous to overgeneralise that "depressed elderly respond well to SSRIs" like Shah *et al* suggest. Moreover, Shah and colleagues also state that patients with dementia may become depressed, particularly if they have insight into their condition. The frequent depressive symptoms founded in these patients may be early manifestations of Alzheimer's disease,[6] in which case, cholinesterase inhibitor drugs instead of antidepressants are indicated. And what about the caution needed in the SSRI prescription because of their significant drug interactions[7] resulting from interference with components of the hepatic "*P*-450 enzyme system"? (where Shah and colleagues state that these drugs are safe in the elderly, in spite of their frequent use of multiple medications). Drug treatments for every elderly disturbance, like for any other human complaint, must always be a careful skilled decision.

Incidentally, Shah and colleagues state that there are no biological diagnostic tests for depression. Beside the dexamethasone cortisol test, the high prevalence of brain dysfunction in the geriatric depressed and cognitive impaired patient suggests that the computer analysed, quantitative electroencephalographic record may help not only in the brain damage differential diagnosis but also in disclosing a depressive disease by showing the characteristic increased anterior alpha power and decreased coherence.[8] Also functional magnetic resonance imaging can yield valuable diagnostic information, showing abnormal activation in the left medial prefrontal cortex and in the right anterior cingulate gyrus[9] in depressive patients.

SALVADOR VALE
Unidad de Investigaciones Clinicas, Antiguo Hospital Concepción Beistegui, Regina 7, CP 06080, México D F, México
email: svale@dfl.telmex.net.mx

1 Shah DC, Evans M, King D. Prevalence of mental illness in a rehabilitation unit for older adults. *Postgrad Med J* 2000;**76**:153–6.
2 Kramer-Ginsberg E, Greenwald BS, Krishnan RR, *et al.* Neuropsychological functioning and MRI signal hyperintensities in geriatric depression. *Am J Psychiatry* 1999;**156**:438–44.
3 Kalayam B, Alexopoulos GS. Prefrontal dysfunction and treatment response in geriatric depression. *Arch Gen Psychiatry* 1999;**56**:713–18.
4 Alexopoulos GS, Meyers BS, Young RC, *et al.* Vascular depression hypothesis. *Arch Gen Psychiatry* 1997;**54**:915–22.
5 Marin RS. Apathy: a neuropsychiatric syndrome. *J Neuropsychiatry Clin Neurosci* 1991;**3**: 243–54.
6 Chen P, Ganguli M, Mulsant BH, *et al.* The temporal relationship between depressive symptoms and dementia. *Arch Gen Psychiatry* 1999; **56**:261–6.
7 Bruce P. The P-450 system: definition and relevance to the use of antidepressants in medical practice. *Arch Fam Med* 1996;**5**:406–12.
8 Hughes JR, John R. Conventional and quantitative electroencephalography in psychiatry. *J Neuropsychiatry Clin Neurosci* 1999;**11**:190–208.
9 Longworth C, Honey G, Sharma T. Functional magnetic resonance imaging in neuropsychiatry. *BMJ* 1999;**319**:1551–4.

The authors respond:

Depression in the elderly has enormous heterogeneity,[1] however it is very common, especially in those with concomitant physical illness. Its recognition in this area is important because of the effect it has on length of hospital stay and results of rehabilitation. Psychiatric intervention has been shown to increase recovery rate, reduce duration of stay, and reduce the need for residential care after discharge and therefore reduce costs.[2]

First line treatment of depression in the elderly with SSRIs has been repeatedly recommended in the literature, not least because they tend to be prescribed and tolerated in therapeutic dosages.[3] Any additional drug treatment in the elderly must be considered with due risk of interactions and side effects: given appropriate care, the SSRIs have been used safely in this population for many years.[4 5]

It is also accepted clinical practice to give a trial of antidepressants to patients with cognitive impairment if there is doubt as to whether the degree of depression may be causing/worsening the impairment. We know of no clinical service where functional magnetic resonance imaging or quantitative electroencephalography would be carried out as an alternative.

The dexamethasone suppression test is not accepted as a diagnostic tool in the elderly because of the high incidence of false positives.[6]

If depressive illness does not respond to treatment, liaison psychiatry services should be involved. However we would disagree strongly with the alternative suggestions given by Dr Vale.

Depression accompanying cerebrovascular changes can respond to antidepressants, and again a trial should be given.[7] Depression as a prodromal feature of Alzheimer's disease and multi-infarct dementia is well recognised as a *diagnosis made with hindsight*.

The cholinesterase inhibitors are not licensed as antidepressants, and given the difficulty in many areas to obtain funding for their use in established Alzheimer's disease are unlikely to become a treatment option for depression, nor is there any published evidence of efficacy in this area.

The dopamine agonist bromocriptine is again not licensed for use as an antidepressant and, other than a few anecdotal reports in the literature, is not a recognised treatment alternative. Given the relative frailty of this population and risk of side effects including precipitating psychotic features, we would not recommended its use outside specialised neuropsychiatric facilities.

In summary, our paper reported a study carried out in a district general hospital rehabilitation service; results and conclusions are applicable to everyday clinical practice in such areas.

1 Evans ME, Mottram P. Diagnosis of depression in elderly patients. *Advances in Psychiatric Treatment* 2000;**6**:49–56.
2 Levitan SJ, Kornfeld DS. Clinical and cost benefits of liaison psychiatry. *Am J Psychiatry* 1981; **138**:790–3.
3 Donoghue JM, Tylee A. The treatment of depression: prescribing patterns of antidepressants in primary care. *Br J Psychiatry* 1996;**168**: 164–8.
4 Evans ME. Depression in elderly physically ill inpatients: a 12 months prospective study. *International Journal of Geriatric Psychiatry* 1993;**8**: 587–92.
5 Abou-Saleh MT. Pharmacologic treatment of mood disorders in the elderly. *Current Opinion in Psychiatry* 1998;**11**:441–5.
6 Murphy E. Depression in the elderly. In: Herbst K, Paykel E, eds. *Depression, an integrative approach*. Oxford: Heinemann Medical, 1989.
7 Dam M, Tonin P, De Boni A, *et al.* Effects of fluoxetine and maprotiline on functional recovery in post-stroke hemiplegic patients undergoing rehabilitation therapy. *Stroke* 1996;**27**:1211–14.

Medical urology

EDITOR,—We describe the last two years' activity in our continence clinic as support for the thesis of Castleden and Duffin that a strong case can be made for the development of a specialty of medical urology.[1]

In our area many community nurses have undergone training by the continence advisers and are able to deal with simple lower urinary tract problems.

During the years 1998 and 1999 we saw 141 patients in the clinic (125 female). The age range was 19 to 96 years with a median of 70 years. The main sources of referral were

Table 1 Diagnoses on 141 patients

Overactive bladder (unstable hyper-reflexia and low compliant bladder)	64	Impaction	6
Nocturnal enuresis	8	Catheter problems	8
Mixed (unstable and genuine stress incontinence (GSI))	26	Frequency and nocturia (no incontinence)	6
Voiding problem (mostly neurogenic)	20	Urinary infection	5
GSI	17	Postmicturition dribble	3
Excess fluid intake	10	Faecal incontinence	3
Nocturnal polyuria	9	Other	4

Table 2 Outcomes in 141 patients

Dry	37	Catheter advice	8
Much improved (wet <1/week)	20	Faecal incontinence better	1
Improved (on presenting symptom)	30	Containment	
		Catheter	5
		Sheath	1
		Pads	9
Frequency/nocturia improved	6	Referral	
		GSI	4
		BPH	1
		Physiotherapy	2
CIC	4	Lost to follow up	13

BPH = benign prostatic hypertrophy.

community based nurses: 56 referrals, general practitioners: 51, geriatricians: nine, consultant gynaecologists: eight, and consultant in rehabilitation medicine: seven.

All patients underwent a thorough clinical assessment including measurement of a flow rate and post-void residual (using bladder scanner) and the great majority of patients completed a frequency/volume chart. The average number of visits was 2.8 (range 1–8). The diagnoses made on these 141 patients are listed in table 1; a number of patients had more than one diagnosis.

Thirty five patients underwent filling and voiding cystometry, usually when treatment after a clinical diagnosis had failed and also in the few patients who were referred for a surgical opinion.

The treatment offered to patients with overactive bladders included habit retraining, regular and prompted voiding, and antimuscarinic drugs (oxybutynin, tolterodine, and imipramine most commonly). Those with genuine stress incontinence (GSI) were taught to do pelvic floor exercises using digital assessment and a perineometer. Vaginal cones were employed in a number of cases.

In those with voiding problems a few were treated with clean intermittent catheterisation (CIC) either done by the patient, a family member, or district nurse. Fluid intake was adjusted where appropriate. An afternoon diuretic was employed successfully in a number of patients with nocturnal polyuria. Constipation was treated with laxatives and/or enemas, urinary infections with antibiotics, and postmicturition dribble exercises were employed in the few with this problem. The outcomes in these patients are listed in table 2.

It was possible, therefore, to achieve relief of symptoms in the great majority of these patients. In those few in whom this was not possible appropriate advice about containment was given.

A very small number of these patients were referred for a surgical assessment, in fact a larger number was referred from the gynaecologists to this clinic.

Urinary incontinence remains one of the "giants of geriatrics"[2] and as a consequence a number of geriatricians run such clinics. The evidence base for useful conservative treatments for incontinence is growing,[3–5] and their effectiveness should continue to increase with time. The co-morbidity seen in old age (as stated by Castleden and Duffin) also makes the geriatrician an appropriate physician to run such clinics.

The medical specialty of urology (or urogynaecology) probably exists already but is not fully recognised. Our experience is that it has an important place in the management of lower urinary tract symptoms. Multidisciplinary working especially with continence advisers is imperative and also close links need to be maintained with surgical colleagues.

J SNAPE
R VIGNARAJA
F SAUNDERS
F SHAW
Department of Health Care of the Elderly, King's Mill Centre for Health Care Services, Mansfield Road, Sutton in Ashfield, Notts NG17 4JL, UK

1 Castleden CM, Duffin HM. Medical urology:a specialty long overdue. *Postgrad Med J* 2000;**76**:1–3.
2 Issacs B. *Recent advances in geriatric medicine 2*. Edinburgh: Churchill Livingstone, 1982: 1–4.
3 Burgio KL, Locher JL, Goode PS, *et al.* Behavioural vs drug treatment for urge urinary incontinence in old women. *JAMA* 1998:**280**;1995–2000.
4 Bo K, Talseth T, Holme I. Single blind randomised controlled trial of pelvic floor exercices, electrical stimulation, vaginal cones and no treatment in the management of GSI in women. *BMJ* 1999;**318**:487–93.
5 Whitelaw S, Hammonds JC, Tregellas R. Clean intermittent catheterisation in the elderly. *Br J Urol* 1987;**60**:125–7.

Publishing case reports—a powerful tool for academic motivation

EDITOR,—The mission of academic medical centres typically includes three distinct goals: providing patient care, educating future doctors, and acquiring new medical knowledge.[1] Academic medical centres seek excellence in each of the three areas to distinguish themselves as outstanding in the local community, region, and nation.[2 3] Factors responsible for poor academic performance in less privileged centres include a failure in academic motivation, lack of pressure for publication, and limited research funding.[4] Ours is a tertiary neonatal care centre for North Queensland, Australia. Geographically, we are isolated from the well established academic centres in Australia. The nursery was almost closed down due to some unfortunate events in 1995 resulting in our appointment in 1996 as the new neonatologists. We were expected to get the nursery functioning to its full potential (eight ventilators, on site surgery, regional transport) and hopefully gain some academic status to justify being the regional teaching hospital. With lack of time, funding, academic support, and all registrars joining us from developing countries as new trainees, research as a goal seemed almost impossible to achieve. The easiest thing was to put more effort on clinical rounds ("patient care"), devote additional time towards rare, interesting, or clinically important cases ("clinical education"), and to publish these case reports as an academic exercise while "acquiring new knowledge".

Within a period of four years, 15 case reports have been published in peer reviewed journals by our team of five registrars working their usual 80–100 hours per fortnight. The quick rewards have motivated the team further, resulting in an additional 27 research projects being either published or presented at major international scientific meetings. The projects cover topics ranging from taping of intravenous cannulas, and adenosine infusion for pulmonary hypertension, to a randomised, controlled trial of erythromycin for facilitating feed tolerance in neonates. Motivated by our academic output (52 projects within four years), two students from the local university have completed the clinical components of their degrees (doctorate and master's) in our nursery.

We strongly believe that increased motivation of registrars due to quick publications serving as quick reward for their hard work is the reason for our academic output. We are aware that case reports may not be counted as "research", or serve as a marker of "academic quality". As long as they are not published in the "yellow pages", they do provide us a very strong academic tool for motivating our registrars towards research.[5]

SANJAY PATOLE
JOHN WHITEHALL
Department of Neonatology, Kirwan Hospital for Women, Townsville, Queensland, Australia
Correspondence to: Dr S Patole, Department of Neonatology, Kirwan Hospital for Women, PO Box 187, Thuringowa, Qld 4817, Australia
email: patoles@health.qld.gov.au

1 Carey RM, Wheby MS, Reynolds RF. Evaluating faculty clinical excellence in the academic health sciences center. *Acad Med* 1993;**68**:813–17.
2 Levinson W, Rubinstein A. Mission critical—integrating clinician-educators into academic medical centers. *N Engl J Med* 1999;**341**:840–3.
3 Bickel J. The changing faces of promotion and tenure at US medical schools. *Acad Med* 1991;**66**:249–56.
4 Weisinger JR, Bellorin-Font E. Latin American nephrology: scientific production and impact of the publications. *Kidney Int* 1999;**56**:1584–90.
5 Glaser RJ. Publish (in the yellow pages)—or perish? *Pharos* 1987;**50**:33.

Hyperthyroidism in an elderly patient

EDITOR,—We read with interest the case of hyperthyroidism in an elderly patient by Findlay and Seymour.[1] We have serious reservation in agreeing with the diagnosis as suggested by the authors. The patient was clinically euthyroid; free thyroxine was marginally raised with suppressed thyroid stimulating hormone (TSH). Thyroid scan was compatible with excessive iodine exposure. The crucial feature in making a diagnosis of amiodarone induced hyperthyroidism (AIH) is a raised triiodothyronine (the authors did mention this in the discussion), as thyroxine

is raised in patients who are on amiodarone and euthyroid. Therefore the triiodothyronine level is very much needed for making a diagnosis of AIH, which unfortunately has not been mentioned in the case. The question remains about the suppressed TSH. Is it surprising to have TSH suppressed in a 72 year old lady who is admitted to the hospital with a fall (and also who suffers from congestive cardiac failure)[2]?

The authors did not provide us with the follow up. Was the patient treated as having AIH? Was there any improvement in the clinical status? Lastly, there was no mention of two types of AIH in the discussion. It is important to subtype a patient with AIH, as the treatment is different in the two types of AIH. Incidentally Loh has reviewed this topic in the same issue of the journal.[3] In case the index patient had AIH, it would probably be type 2 where the main treatment option is corticosteroids and not antithyroid drugs.

SHAILA BHATTACHARYYA
ARPANDEV BHATTACHARYYA
Flat 1, Hope Hospital,
Stott Lane, Salford M6 8HD, UK
email: Arpandev@aol.com

1 Findlay PF, Seymour DG. Hyperthyroidism in an elderly patient. *Postgrad Med J* 2000;**76**:173–5.
2 Parle JV, Franklyn JA, Cross KW, *et al.* Prevalence and follow up of abnormal thyrotrophin (TSH) concentrations in the elderly in United Kingdom. *Clin Endocrinol (Oxf)* 1991; **34**:77–83.
3 Loh KC. Amiodarone induced thyroid disorders: a clinical review. *Postgrad Med J* 2000;**76**:133–40.

The authors respond:

I would like to thank Drs S and A Bhattacharyya for their interest and comments on the above case.

It is quite correct to highlight the point that a suppressed TSH does not necessarily equate to a hyperthyroid state and indeed it is worth stressing that the causes of a suppressed TSH are legion. In our article we did not include the triiodothyronine which has caused my colleagues to question the diagnosis. I would, however, caution against overreliance on triiodothyronine values in this setting. Indeed, my colleagues also mention in their letter the excellent review article by Loh on amiodarone induced thyroid disorders,[1] which appears in the same journal. According to Loh the triiodothyronine in AIH is unreliable and may be either high or normal. For the record the value of free triiodothyronine in the case described was 12.6 pmol/l (normal range 3.0–7.0).

The other criticism was that no information was available on the patient's progress and follow up. After discussion with the endocrinologists this patient was treated with carbimazole starting at a dose of 20 mg daily for four weeks and then reduced to a maintenance dose of 10 mg daily. Amiodarone was discontinued. The patient continues to be reviewed on a regular basis and at present is on carbimazole 10 mg daily. She remains well and her last TSH was 2.83 mU/l.

1 Loh TT. Current concepts in medicine. Amiodarone-induced thyroid disorders: a clinical review. *Postgrad Med J* 2000;**76**:133–40.

DIARY

Falk Symposia

1–2 October 2000: Non-neoplastic diseases of the anorectum—an interdisciplinary approach (Freiburg, Germany)
3–4 October 2000: Immunosuppression in inflammatory bowel diseases—standards, news, and future trends (Freiburg, Germany)
12–13 October 2000: Biology of bile acids in health and disease (Den Haag, The Netherlands)
4 November 2000: Chronic inflammatory bowel diseases—progress and controversies at the turn of the century (Bucharest, Romania)
Details: Falk Foundation eV–Congress Division, Leinenweberstr 5, PO Box 6529, D-79041 Freiburg, Germany (tel: +49 (0) 761 130340, fax: +49 (0) 761 1303459, email: symposia@falkfoundation.de).

Royal College of Physicians of Edinburgh

7–8 October 2000: Stroke treatment and service delivery (consensus conference)
Details: Education, Audit, and Research Department, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK (tel: +44 (0) 131 225 7324, fax: +44 (0) 131 220 4393, web site: www.rcpe.ac.uk).

Royal College of Physicians of Edinburgh/Scottish Intercollegiate Guidelines Network

3 November 2000: Symposium on clinical effectiveness, clinical guidelines and clinical standards
Details: Mrs Anne Fairbairn, Coordinator for Research and EBM, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK (email: a.fairbairn@rcpe.ac.uk).

3rd Teupitz Colloquium

17–20 September 2000: Basic Research in Endocrine Dermatology
Details: Professor Dr Ch C Zouboulis, Department of Dermatology, University Medical Center Benjamin Franklin, Free University of Berlin, Hindenburgdamm 30, 12200 Berlin, Germany (tel: +49 30 84 45 28 08, fax: +49 30 84 45 42 62, email: zoubbere@zedat.fu-berlin.de).

St Mark's Hospital & Academic Institute

16–18 October 2000: Frontiers in colorectal disease (lecture course)
Details: The Administrator, St Mark's Academic Institute, St Mark's Hospital, Northwick Park, Harrow, Middlesex HA1 3UJ (tel: +44 (0) 20 8235 4046/8, fax: +44 (0) 20 8235 4039, email: e.power@ic.ac.uk; web site: www.stmarkshospital.org.uk).