Samples of several hundreds of pairs of twins are needed to detect even relatively large effects of cohabitation. Given the usual modelling strategy, finding no such effect when one actually exists will lead to an inflated estimate of the size of the genetic effect.[6] That is, usual application of the method is biased towards finding in favour of a genetic theory and has little power to deal with the alternative non-genetic explanation. If one only wears green glasses, one is bound to conclude that the world is green.

Spector *et al* have applied the method to osteoarthritis. From a sample of twins ascertained by advertisements and other means they found that monozygotic pairs were significantly more concordant for disease at one, possibly two, sites. For a score based on radiographic imaging, about half the variance was attributable to genetic factors under the classic twin method. Note that this does not mean that half of osteoarthritis is due to genes, a naive and incorrect interpretation sometimes made. Attempts were made to allow for non-genetic influences, but given the sample size and statistical power the above figure must be considered to be an upper bound of the genetic "influence."

As the authors indicate, it is now possible to test directly the genetic hypothesis by actually finding the genetic loci implicated and measuring the impact of variation at these putative loci. Replication of such findings is essential as the current controversy over the existence and size of the role of the vitamin D receptor locus on bone density is illustrating.[7,8] Understanding the biological mechanisms of the genes will confirm the truth of the genetic hypothesis and could have considerable clinical importance.

Such gene searches are very expensive and justified only if there is a priori evidence that genetic variation is important for a characteristic. Given the caveats above, the findings of classic twin studies can be informative. Moreover, particular twin pairs are the optimal design for some sib-pair methods of searching for disease genes.[9,10] Hundreds if not thousands of such pairs may be needed to ensure that important loci are not missed, however, and international collaborative efforts may be required. This may be difficult to achieve as there is a danger that the promise of huge commercial gains may overwhelm traditional scientific cooperation.

In summary, genetic research offers new insights into the aetiology, and hopefully the treatment, of diseases. Data from twin pairs will play a pivotal role in this development, but a good deal of circumspection is warranted in interpreting early findings, especially those from studies in which genes are not actually measured. False or inflated claims will be detrimental in the long term.

1 Doyle AC. A scandal in Bohemia. In: *The adventures of Sherlock Holmes*. London: George Newnes, 1892.
2 Hopper JL. The epidemiology of genetic epidemiology. *Acta Genet Gemellol (Roma)* 1992;41:261-73.
3 Hopper JL, Mathews JD. A multivariate normal model for pedigree and longitudinal data and the software "Fisher." *Australian Journal of Statistics* 1994;36:153-76.
4 Clifford CA, Hopper JL, Fulker DW, Murray RM. A genetic and environmental analysis of a twin study of alcohol use, anxiety and depression. *Genet Epidemiol* 1984;1:63-79.
5 Hopper JL, Mathews JD. Extensions to multivariate normal models for pedigree analysis. II. Modeling the effect of shared environment in the analysis of blood lead levels. *Am J Epidemiol* 1983;117:344-55.
6 Christian JC. Norton JA, Sorbel J, Williams CJ. Comparison of analysis of variance and maximum likelihood based path analysis of twin data: partitioning genetic and environmental sources of covariance. *Genet Epidemiol* 1995;12:27-35.
7 Eisman JA. Vitamin D receptor gene alleles and osteoporosis: an affirmative view. *J Bone Miner Res* 1995;10:1289-93.
8 Peacock M. Vitamin D receptor gene alleles and osteoporosis: a contrasting view. *J Bone Miner Res* 1995;10:1294-7.
9 Penrose LS. The general sib-pair linkage test. *Annals of Eugenics* 1953;18: 120-44.
10 Haseman JK, Elston RC. The investigation of linkage between a quantitative trait and a marker locus. *Behav Genet* 1972;2:3-19.

Survey of intensive care of severely head injured patients in the United Kingdom

D R Jeevaratnam, D K Menon

Abstract

***Objectives*—To study practice in intensive care of patients with severe head injury in neurosurgical referral centres in United Kingdom.**

***Design*—Structured telephone interview of senior nursing staff in intensive care unit of adult neurosurgical referral centre.**

***Setting*—39 intensive care units in hospitals that accepted acute head injuries for specialist neurosurgical management, identified from *Medical Directory* and information from professional bodies.**

***Main outcome measures*—Details of organisation and administration of intensive care and patterns of monitoring and treatment for patients admitted with severe head injury.**

***Results*—Patients were managed in specialist neurosurgical intensive care units in 21 of the centres and in general intensive care units in 18. Their intensive care was coordinated by an anaesthetist in 25 units and by a neurosurgeon in 12. Annual caseload varied between units: 20 received > 100 patients, 12 received 50-100, and seven received 25-49. Monitoring and treatment varied considerably between centres. Invasive arterial pressure monitoring was used routinely in 36 units, but central venous pressure monitoring was routinely used in 24 and intracranial pressure was routinely monitored in only 19. Corticosteroids were used to treat intracranial hypertension in 19 units. Seventeen units routinely aimed for arterial carbon dioxide pressure of 3·3-4·0 kPa, and one unit still used severe hyperventilation to a pressure of < 3·3 kPa.**

***Conclusion*—The intensive care of patients with acute head injuries varied widely between the centres surveyed. Rationalisation of the intensive care of severe head injury with the production of widely accepted guidelines ought to improve the quality of care.**

Department of Anaesthesia, University of Cambridge Clinical School, Addenbrooke's Hospital, Cambridge CB2 2QQ
D R Jeevaratnam, *senior registrar*
D K Menon, *lecturer and honorary consultant*

Correspondence to:
Dr Menon.

BMJ 1996;312:944-7

Introduction

Half a million patients with head injuries are seen by the health care system in the United Kingdom each year[1]; a fifth of these are admitted to hospital,[2] and 10% of admissions are for severe head injury (defined as a Glasgow coma score of less than 8[3]). Secondary physiological insults contribute to the extent of neurological injury,[4,5] and the quality of intensive care can be a major determinant of outcome. Recent research has re-evaluated some treatment methods that were commonly used in the past.[6,7] However, a recent paper has shown wide variations in the management of severe head injury in the United States, with some centres still using treatments that were not supported

by available findings from clinical research.[6] We report the results of a structured telephone survey of intensive care of severe head injury in the United Kingdom.

Materials and methods

SURVEY DESIGN AND DATA COLLECTION

We identified 39 neurosurgical units from data in the *Medical Directory* and from neurosurgical and anaesthetic professional bodies. We conducted our survey by telephone. Clinical nurse specialists or staff nurses working in the units were interviewed by a single interviewer, who asked for the senior nurse on duty. The survey was in the form of structured questions with a set of defined answers, from which the interviewee chose one, except where a specific volunteered response was clearly appropriate. We encouraged respondents to consult medical and nursing colleagues and offered to recontact them after a short period of consultation and data collection if they wished. Information on use of corticosteroids was accepted only after we had emphasised that our survey was specific to head injury and asked the respondent to exclude reference to patients with other diagnoses, including intracranial tumours.

All 39 centres participated in the survey. We assessed the reliability of the data by repeating our survey of 20 of the centres the following week, when a different nurse was asked the same questions.

Results

In 31 of the centres the intensive care unit and the neurosurgical referral unit were attached to a multidisciplinary hospital, while the remaining eight were either within free standing neurosurgical units or were in hospitals with one or two other specialised units (for example, plastic surgery). The respondent was a senior staff nurse in 20 units, a sister in 18, and a clinical nurse specialist in one. Our repeat survey of 20 intensive care units produced results that showed excellent concordance with those obtained in the first interview, with no changes in any of the questionnaire items except for a rebanding of the percentage of patients receiving corticosteroids (from >50% to 25-50% in two centres).

Tables 1 and 2 show the results of our survey. The use of specific monitoring procedures or treatments was unrelated to the type of intensive care unit, estimated annual case load, or speciality of the unit's director.

While we wished to determine whether all severely head injured patients were admitted to intensive care units, reliable information on this issue was difficult to obtain. We were unable to estimate the referral rate from peripheral hospitals, and the format of the survey did not allow us to investigate this issue further.

Table 1—*Characteristics of 39 intensive care units that accepted acute head injuries for specialist neurosurgical management*

Answers to survey questions	No (%) of units
Care of patients in acute coma from head injury:	
Yes	39 (100)
No	0
Nature of intensive care unit:	
Specialised neurosurgical or neurological	21 (54)
General	18 (46)
No of cases of severe head injury treated a year:	
>100	20 (51)
75-100	2 (5)
50-74	10 (26)
25-49	7 (18)
Director of unit:	
Anaesthetist	25 (64)
Neurosurgeon	12 (31)
Physician	1 (2·5)
Other	1 (2·5)

Table 2—*Care of patients with acute head injury in 39 intensive care units: monitoring of haemodynamics and intracranial pressure and treatment of intracranial hypertension*

Answers to survey questions	No (%) of units
Percentage of patients receiving monitoring of intracranial pressure:	
90-100	10 (26)
75-89	9 (23)
50-74	6 (15)
25-49	7 (18)
<25	4 (10)
0	3 (8)
Types of monitoring device used:	
Subdural	31 (79)
Ventriculostomy	5 (13)
Other	0
Treatments used for intracranial hypertension:	
Osmotic diuretics	39 (100)
Hyperventilation	39 (100)
Drainage of cerebrospinal fluid	37 (95)
Barbiturates	22 (56)
Corticosteroids	19 (49)
Percentage of patients receiving corticosteroids:	
>50	7 (18)
25-50	3 (8)
<25	9 (23)
0	20 (51)
Target arterial carbon dioxide pressure (kPa):	
>4	21 (54)
3·3-4	17 (44)
<3·3	1 (2·5)
Drugs used for sedation:	
Propofol	25 (64)
Midazolam	25 (64)
Other	1 (2·5)
Analgesic drugs used:	
Fentanyl	10 (26)
Alfentanil	11 (28)
Morphine	19 (49)
Other	1 (2·5)
Percentage of patients receiving neuromuscular blockade:	
100	26 (67)
75-99	3 (8)
50-74	3 (8)
25-49	4 (10)
<25	1 (2·5)
Neuromuscular blocking drug routinely used:	
Atracurium	36 (92)
Vecuronium	2 (5)
Pancuronium	1 (2·5)
Percentage of patients receiving invasive arterial monitoring:	
100	36 (92)
50-99	3 (8)
Percentage of patients receiving central venous pressure monitoring:	
100	24 (62)
50-99	9 (23)
<50	6 (15)

Discussion

METHODOLOGY

Telephone surveys are more effective than postal surveys in achieving complete participation since posted forms may not be received by the appropriate person or may be mislaid or ignored. While responses to written questionnaires are likely to be more considered, and hence more accurate, this cannot be guaranteed and there is no opportunity to discuss responses or allow interactive confirmation of the data obtained. However, telephone surveys suffer from several disadvantages. Responses are based on impressions rather than accumulated data, and the accuracy of the information obtained will vary with the training, seniority, and experience of the respondent. To

minimise these effects we spoke to the most senior nurse on duty and encouraged respondents to consult colleagues and offered to recontact them after a short period of consultation and data collection if they wished. This option was taken up by five centres. We also provided them with a range of specified responses rather than asking them to volunteer quantitative information. The excellent concordance that we obtained on the repeat survey of 20 of the units shows the reproducibility of our method of data collection. We chose to interview senior nursing staff because they probably provide the most objective source of information about actual (rather than planned) clinical practice.

FINDINGS IN THE CONTEXT OF BEST PRACTICE

The results of our survey highlight several important issues that are at odds with an emerging consensus about the management of patients with severe head injury.[2,3]

Clinical signs cannot be used to detect neurological deterioration in a sedated and paralysed patient, and isolated imaging studies cannot replace monitoring of intracranial pressure.[9] Intracranial and cerebral perfusion pressures have been shown to correlate strongly with outcome in several studies,[4,5,10-18] and many treatments are designed to optimise these variables. Clearly, in the absence of continuous monitoring these interventions may be underused or used blindly and, in some cases, inappropriately.

Induced hypocapnia can reduce cerebral blood volume and intracranial pressure. However, severe hypocapnia (< 3·3 kPa) can reduce cerebral blood flow to dangerous levels[19,20] and result in cerebral venous oxygen desaturation,[21,22] which is known to worsen outcome.[22] There is less information on the effects of moderate hyperventilation (arterial carbon dioxide pressure 3·3-4·0 kPa), but routine prolonged hyperventilation was shown to worsen outcome in one study.[6] These findings provide a rational basis for avoiding severe hypocapnia and using moderate hypocapnic ventilation with caution.

Corticosteroids are effective in reducing oedema in intracranial malignancies but are ineffective in head injury,[23-26] where they may worsen outcome[27,28] perhaps via metabolic effects. In several units corticosteroids were used by a single consultant, rather than as part of a unit's protocol.

IMPLICATIONS

The variations in clinical practice that we observed, both between centres and between the quality of care seen in the survey and that which might be described as the best possible standard of care, have important implications. We do not think that our findings are the consequence of justifiable therapeutic nihilism. There is little doubt that the combination of early surgery and good intensive care can result in a 10-20% improvement in outcome in severe head injury.[29] Equally, we do not believe that many of these variations arose because of a lack of consensus among experts in the specialty. Many studies, only a small proportion of which are referenced in this paper, have demonstrated the need to monitor and control intracranial and cerebral perfusion pressures in patients with severe head injury. While costs and funding may be an important issue, a recently published survey in the United States, where spending on intensive care is higher, showed similar results.[8]

The findings of our survey provide a rational basis for a more detailed study, but there seems to be a strong case for producing nationally accepted guidelines on minimum standards of care for patients with severe head injury. Such guidelines would not only address the issues highlighted in this paper but could also provide guidance on the need for referral from receiving hospitals and the necessary levels of care in neurosurgical units for individual patients, depending on the severity of their head injury.

Key messages

- The quality of intensive care can be a major determinant of outcome in the management of patients with severe head injury
- We conducted a structured telephone survey of senior nursing staff in intensive care units in 39 neurosurgical referral centres
- The intensive care of patients varied widely, with only half the centres surveyed routinely monitoring intracranial pressure in comatose patients
- Moderate hyperventilation and treatment with corticosteroids were still used by several centres despite increasing evidence of their lack of efficacy and potential for causing harm
- There is a strong argument for establishing national minimum standards of care for the intensive care of patients with severe head injury

We thank Colette O'Kane and Liz Fahie, University Department of Neurosurgery, Addenbrooke's Hospital, for help with identifying neurosurgical centres.

Funding: DKM was supported by a grant from the Critical Care Trust, Leeds.

Conflict of interest: None.

1 Jennet B, Teasdale G. *Management of head injuries*. Philadelphia: FA Davies, 1981.
2 Pickard JD, Czosnyka M. Management of raised intracranial pressure. *J Neurol Neurosurg Psychiatry* 1993;56:845-58.
3 Miller JD. Head injury. *J Neurol Neurosurg Psychiatry* 1993;56:440-7.
4 Jones PA, Andrews PJD, Midgley S, Anderson SI, Piper IR, Tocher JL, *et al.* Measuring the burden of secondary insults in head injured patients during intensive care. *J Neurosurg Anesthesiol* 1994;6:4-14.
5 Chan KH, Dearden NM, Miller JD, Andrews PJ, Midgley S. Multimodality monitoring as a guide to treatment of intracranial hypertension after severe brain injury. *Neurosurgery* 1993;32:547-52.
6 Muizelaar JP, Marmarou A, Ward JD, Kontos HA, Choi SC, Becker DP, *et al.* Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg* 1991;75:731-9.
7 Braakman R, Schouten HJA, Dishoeck MB, Minderhoud JM. Megadose steroids in severe head injury: results of a prospective double-blind clinical trial. *J Neurosurg* 1983;58:326-30.
8 Ghajar J, Hariri RJ, Narayan RK, Iacono LA, Firlik K, Patterson RH. Survey of critical care management of comatose, head-injured patients in the United States. *Crit Care Med* 1995;23:560-7.
9 O'Sullivan MG, Statham PF, Jones PA, Miller JD, Dearden NM, Piper IR, *et al.* Role of intracranial pressure monitoring in severely head-injured patients without signs of intracranial hypertension on initial computerized tomography. *J Neurosurg* 1994;80:46-50.
10 Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg* 1991;75(suppl):S59-66.
11 Marshall LF, Smith RW, Shapiro HM. The outcome with aggressive treatment in severe head injuries. *J Neurosurg* 1979;50:20-5.
12 Narayan RK, Kishore PRS, Becker DP, Ward JD, Enas GG, Greenberg RP, *et al.* Intracranial pressure: to monitor or not to monitor. *J Neurosurg* 1982;56:650-9.
13 Miller JD, Becker DP, Ward JD, Sullivan HG, Adams WE, Rosner MJ. Significance of intracranial hypertension in severe head injury. *J Neurosurg* 1977;47:503-16.
14 Becker DP, Miller JD, Ward JD, Greenberg RP, Young HF. Outcome from severe head injury with early diagnosis and intense management. *J Neurosurg* 1977;47:491-502.
15 Changaris DG, McGraw CF, Richardson JD, Garretson HD, Arpin EJ, Shields EB. Correlation of cerebral perfusion and Glasgow coma scale to outcome. *J Trauma* 1987;27:1007-13.
16 Piek J, Chesnut RM, Marshall LF, van Berkum-Clark M, Klauber MR, Blunt BA, *et al.* Extracranial complications of severe head injury. *J Neurosurg* 1992;77:901-7.
17 Robertson CS, Contant CF, Gokaslan ZL, Narayan RK, Grossman RG. Cerebral blood flow, arteriovenous difference, and outcome in head injured patients. *J Neurol Neurosurg Psychiatry* 1992;55:594-603.
18 Rosner MJ, Daughton S. Cerebral perfusion pressure management in head injury. *J Trauma* 1990;30:933-40.
19 Cold GE. Does acute hyperventilation provoke cerebral oligaemia in comatose patients after acute injury? *Acta Neurochir (Wein)* 1989;96:100-6.
20 Obrist WD, Langfitt TW, Jaggi JL, Cruz J, Gennarelli TA. Cerebral blood flow and metabolism in comatose patients with acute head injury. *J Neurosurg* 1984;61:241-53.

21 Sheinberg M, Kanter MJ, Robertson CS, Contant CF, Narayan RK, Grossman RG. Continuous monitoring of jugular venous oxygen saturation in head injured patients. *J Neurosurg* 1992;76:212-71.
22 Cruz J. On-line monitoring of global cerebral hypoxia in acute brain injury. *J Neurosurg* 1993;79:228-33.
23 Gianotta SL, Weiss MH, Apuzzo MLJ, Martin E. High dose glucocorticoids in the management of severe head injury. *Neurosurg* 1984;15:497-501.
24 Cooper PR, Moody S, Clark WK, Kirkpatrick J, Maravilla K, Gould AL, *et al.* Dexamethasone and severe head injury: a prospective double blind study. *J Neurosurg* 1979;51:307-16.
25 Saul TG, Ducker TB, Salcman M, Carro E. Steroids in severe head injury: a prospective randomized clinical trial. *J Neurosurg* 1981;54:596-600.
26 Gudeman SK, Miller JD, Becker DP. Failure of high-dose steroid therapy to influence intracranial pressure in patients with severe head injury. *J Neurosurg* 1979;51:301-6.
27 Robertson CS, Clifton GL, Goodman JC. Steroid administration and nitrogen excretion in the head injured patient. *J Neurosurg* 1985;63:714-8.
28 Young B, Ott L, Phillips R, McClain C. Metabolic management of the patient with head injury. *Neurosurg Clin N Am* 1991;2:301-28.
29 Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF. NINDS traumatic coma date bank: intracranial pressure monitoring methodology. *J Neurosurg* 1991;75(suppl);S21-7.

(Accepted 29 February 1996)

A follow up study of depression in the carers of dementia sufferers

C G Ballard, C Eastwood, M Gahir, G Wilcock

MRC Neurochemical Pathology Unit, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE
C G Ballard, *MRC clinical scientist*

Yardley Green Hospital, Birmingham B9 5SS
C Eastwood, *senior house officer in psychiatry*

Reaside Clinic, Birmingham B45 9BE
M Gahir, *senior registrar in psychiatry*

Frenchay Hospital, Bristol BS16 1LE
G Wilcock, *professor of healthcare for the elderly*

Correspondence to:
Dr Ballard.

BMJ 1996;312:947

Caring for people with dementia is stressful,[1] and depression occurs in 30-50% of carers.[2] Few data are available, however, about the course of depression or variables associated with the length of episodes, such as age, closeness of relationship, and non-cognitive symptoms among patients. We followed up a group of carers over a year to assess the length and determinants of depression.

Subjects, methods, and results

We recruited 124 patients with dementia as defined by DSMIIIR from consecutive referrals to psychiatric services in Birmingham (99) and a memory clinic in Bristol (25); 90% of those approached agreed to participate. One hundred and nine had informal carers, of whom 85 were followed up for one year.

Carers were interviewed initially and every month with the geriatric mental state schedule and the Cornell depression scale, with additional questions about the duration and impact of symptoms. Depression was diagnosed according to research diagnostic criteria. In the patients the geriatric mental state schedule/history and aetiology schedule/secondary dementia schedule package was used to diagnose dementia; depression was assessed in the same way as for carers. Psychotic symptoms were evaluated with the Burns symptom check list. Cognitive function was assessed with the cognitive section of the Cambridge assessment of mental disorders in the elderly initially and after one year. Problem behaviours and social support were evaluated using the carers stress scale. References for the instruments are given elsewhere.[3]

Depression was defined as resolved if major or minor depression was absent for three consecutive months. Correlations between the number of months of depression and age, gender, whether the carer was living with the patient, patient depression, psychotic symptoms, severity of dementia, baseline cognitive function score, cognitive decline, social support, and being a first degree relative were calculated using Pearson's correlation coefficient. Significant associations were then tested with a logistic regression analysis comparing carers with and without at least three months of depression. A probability of 0·01 represented statistical significance.

Eighty five of the 109 (78%) carers were followed up for one year (table 1). Eighteen of the 26 cases of major (3/6) or minor (15/20) depression resolved during the follow up year but eight did not. Carers with depression had a mean of 5·27 (SD 4·54) months of depression and a mean Cornell depression score of 8·70 during depressed months. Fourteen (54%) experienced at least three months of depression and 10 (39%) at least six months. Twenty eight of the 59 (48%) carers without depression initially developed major (nine) or minor (19) depression during the follow up year, and 14 were depressed for at least three months.

Table 1—*Demographic characteristics of carers and patients. Results are numbers and percentages*

Characteristics	No (%)
Carers:	
No (%) men	46 (54·1)
Mean age (years)	64·8
No (%) living with patient	48 (56·5)
No (%) marital partners of patients	41 (48·2)
No (%) children of patients	30 (35·3)
No (%) siblings of patients	3 (3·5)
No distant relatives or friends of patients	11 (12·9)
No with minor depression at baseline	20 (23·5)
No with major depression at baseline	6 (7·1)
Patients	
No (%) women	65 (76·5)
Mean age (years)	79·36
Mean CAMCOG score at baseline	46·23
Clinical dementia rating scale	1-21, 2-53, 3-11

Only living with a dementia sufferer ($r=0·30$, $P=0·005$), depression in the patient ($r=0·37$, $P=0·001$), and problem behaviours ($r=0·39$, $P=0·007$) were significantly associated with the number of months of depression. In the logistic regression analysis only the overall level of problem behaviours was significantly associated (Wald 7·57, $P=0·006$).

Comment

Our sample was representative of dementia sufferers with mild to moderate impairment in contact with clinical services. Depression was diagnosed according to standardised criteria. Among these carers the annual incidence of depression lasting a month or more was almost 50% and of that lasting three months or more 25%. Thirty per cent of cases persisted for the whole follow up year, and carers with depression initially experienced on average over five months of depression. The incidence of depression was well above that reported in community studies, although the length of depressive episodes was similar.[4]

In our study both problem behaviours and depression were significantly associated with the number of months of depression, a feature not consistently shown in previous cross sectional studies.[5] This emphasises the importance of treating non-cognitive symptoms.

We thank Miss Lorraine Hood for her secretarial help.
Funding: MRC.
Conflict of interest: None.

1 Pearlin LI, Mullen JT, Semple SJ, Skaff MM. Caregiving and the stress process: An overview of concepts and their measures. *Gerontology* 1990;30:583-94.
2 Coope B, Ballard C, Saad K, Patel A, Bentham P, Bannister C, *et al.* The prevalence of depression in the carers of dementia sufferers. *International Journal of Geriatric Psychiatry* 1995;10:237-42.
3 Ballard CG, Saad K, Patel A, Gahir M, Solis M, Coope B, Wilcock G. The prevalence and phenomenology of psychotic symptoms in dementia sufferers. *International Journal of Geriatric Psychiatry* 1995;10:477-85.
4 Costello CG. The similarities and dissimilarities between community and clinic cases of depression. *Br J Psych* 1990;157:612-821.
5 Morris RS, Morris LW, Britton PG. Factors affecting the emotional wellbeing of the caregivers of dementia sufferers. *Br J Psychiatry* 1988;153:147-56.

(Accepted 5 December 1995)