Recruitment for post-natal depression studies

SIR: We wish to report a method of overcoming the common problem of identifying adequate numbers of patients in studies of post-natal depression. Non-psychotic depression is common in the puerperium but is often missed by clinical services, even when contact is made antenatally (Appleby *et al*, *Journal*, April 1989, **154,** 510–515). Similarly, in research a sufficient or representative subject sample is hard to recruit. One reason for this is that most women with post-natal depression are not in contact with general practitioners or psychiatrists who might refer them. In addition, they do not readily return postal questionnaires from unfamiliar researchers.

In preparation for a large therapeutic trial of fluoxetine and counselling in post-natal depression, we conducted a three-month pilot study of the following recruitment method. All women who successfully gave birth in one of two south Manchester maternity units were approached before discharge and asked to agree to an assessment at six weeks post-partum. A date and time were agreed, usually confirmed by a telephone call a week before the appointment. On the arranged date, each woman was visited at home and the Edinburgh Post-natal Depression Scale (EPDS; Cox *et al*, *Journal*, June 1987, **150,** 782–786) was completed.

Of 245 women delivering in hospital, 207 (84%) agreed to be visited. Thirty-eight refused or were ineligible for the study, usually because they did not live locally. Of those who agreed, 158 (76%) were available when visited. Thirty-two (20%) of those who completed the EPDS scored at or above 10, a threshold used in community surveys. Nineteen (12%) scored at or above 12, a threshold used in clinical studies. These figures are in keeping with previous estimates of prevalence.

The success of this recruitment appears to be the result of several factors. Firstly, the initial approach was made face-to-face while subjects were well rather than depressed. Secondly, the research was presented in an obstetric rather than a psychiatric setting. Thirdly, the women were visited by a person known to them. Fourthly, the visits took place in the patients' homes at a time convenient to them.

The only disadvantage of this method is the large number of people who must be screened but who score below threshold on the EPDS. However, 20% were found to be potential cases, all of whom were easily accessible within the catchment area of one district health authority.

The study sample resulting from this method of identification was large and likely to be representative of all cases of post-natal depression. We would recommend its use in other such studies.

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Tricyclic-induced seizures and absent ECT response

SIR: Further to previous correspondence on this subject (Silverstone & Fahy, *Journal*, September 1991, **159,** 446–447), we would like to outline details of a case in which tricyclic antidepressants produced seizure activity in a depressed patient, with a pronounced elevation of mood resulting. The subsequent use of electroconvulsive therapy (ECT) in this patient did not have a beneficial effect, instead producing paranoid state with irritability. The case details are outlined below.

Case report. C, a 37-year-old woman, was referred to our unit on a mental health certificate. She had a severe depressive illness of several years' duration, more pronounced in recent months. C had failed to respond to several courses of antidepressants as an out-patient, and had developed apathy, anorexia and a suicidal preoccupation. She was withdrawn, negative and suicidal at interview. C was commenced on amitriptyline in increasing doses and her mood and appetite gradually improved.

Following two weeks of therapy, C was much more positive and hopeful for the future, and she was allowed home for a visit. On her return, she developed a generalised tonic–clonic seizure which lasted one minute, ending spontaneously. This seizure was followed by a dramatic improvement in mood which lasted more than a week. C requested a change of therapy to avoid the possibility of further seizures, and was commenced on fluoxetine therapy. Discharge was effected five days later.

C was reviewed one week later and was demonstrably worse, again experiencing suicidal ideation. She agreed to a trial of ECT and was readmitted for this purpose. Following six treatments, C was actually worse, demonstrating features of depression, paranoia and irritability. ECT was discontinued and amitriptyline therapy was reinstituted with the addition of carbamazepine to limit seizure activity. Her mood quickly responded, and despite some myoclonic activity and hypnogogic experiences on three occasions, she has remained well on treatment and has resumed employment.

This case demonstrates that differences do exist, in terms of clinical response, between seizures induced by tricyclic agents and seizures induced by ECT. This phenomenon has been described in relation to spontaneous seizures, and it shows the complex relationship between seizure activity and mood elevation.

What can be said in relation to this case is that a tendency towards lowered seizure threshold with tricyclic antidepressants is not necessarily an indicator of ECT responsiveness.

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Alzheimer's disease and Lewy body dementia

SIR: The study by Förstl *et al* (*Journal*, March 1993, **162,** 385–392) highlights the current problems regarding the nosological status of cortical Lewy body pathology (CLBP) in people with co-existent Alzheimer-type pathology. They suggest that in a patient presenting with a combination of Alzheimer-type dementia together with severe Parkinsonian rigidity and a pattern of accentuated frontal lobe atrophy on computerised tomographic scan, the differential diagnosis should include a diagnosis of 'Lewy body dementia variant of Alzheimer's disease'. Thus, it is appropriate to review the evidence that examines whether Alzheimer's disease (AD) and Lewy body dementia (LBD) are indeed aetiologically distinct entities, or whether the variations in neuropathology seen represent a single disease process with a broad range of phenotypic expression.

Recent studies strongly support the aberrant processing of β-amyloid precursor protein (β-APP) and consequent deposition of β-amyloid protein (β-AP) as the primary pathological event in AD. If LBD and AD are different phenotypic presentations of a single nosological entity, it would be logical to surmise that β-AP deposition is the central feature in both conditions. One would therefore expect to find similar patterns of β-AP deposition in both AD and LBD.

In addressing this important point, Förstl and co-workers found that for patients who had CLBP, densities of cored (classic) and un-cored (diffuse) β-AP plaques in the cortex were slightly lower compared with cases who had AD pathology alone. However, the data supporting this statement is not presented. Moreover, the study examined only eight cases and used a silver staining technique to visualise the plaques, which often fails to show all the diffuse amyloid deposits present in the section.

In a larger fully quantitative immunohistochemical study, using a monoclonal antibody to β-AP, we found no significant difference between AD and LBD, in either the total β-AP load (Gentleman *et al*, 1992) or in the density or relative proportions of classic and diffuse plaques (McKenzie *et al*, 1993).

A further point is the finding of cortical Lewy bodies (LBs) in a familial case of AD which has a mis-sense mutation (Val-Ile) in codon 717 of APP and in the absence of genetic data would be classified as a form of LBD (Lantos *et al*, 1992). Other mutations at this codon (e.g. Val-Gly) have failed to result in Lewy body pathology (Mann *et al*, 1992). Mutations within a single gene are generally regarded as a single nosological entity despite variability in phenotypic presentation, as seen in prion disease (Roberts & Harrison, 1991).

Förstl *et al* noted that tangle densities in the frontal lobe and parahippocampal gyrus were significantly lower in the patients who had LBs; they also stated that these same two regions were the sites of great LB density. It has been proposed that the LBs themselves represent a form of modified neurofibrillary tangle (NFT) and in this context it is the total number of inclusion bodies (NFTs and LBs) that may be important. Therefore, an individual may exhibit a preponderance for either NFTs or for LBs, reflecting a common underlying aetiological process of neuronal damage due to β-AP deposition.

In summary, we suggest that the available evidence indicates that LBD represents part of the wide spectrum of clinico-pathological consequences of β-AP deposition. In such circumstances it seems inappropriate, at present, to propose a series of discrete subtypes of β-amyloid dementia.

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