Recruitment and retention in psychiatry

We read with interest the literature review by Brockington & Mumford (2002) on recruitment into psychiatry. We agree it is possible that recruitment might be improved by influencing medical student intakes and having greater understanding of the pathways leading to a psychiatric career. However, we believe that the factors governing career choice at both undergraduate and postgraduate levels are uncomplicated. Students on clinical attachments in psychiatry are exposed to wards which are often dirty, unpleasant, frightening and understaffed. They see a service that is under-funded and, subsequently, staff with low morale and burnout. It is hardly surprising that many pursue alternative specialities.

Attempts to encourage potential and existing recruits by repackaging psychiatry at any nodal point in a medical career are likely to fail unless there is the financial investment to provide fully resourced working environments. Attractive conditions might also reduce stigma, contributing further to recruitment. The findings of a study being carried out by the Royal College of Psychiatrists looking at why psychiatrists leave the profession and retire early will be of interest to us all (Camm, 2002).


We were encouraged to read both a review article and an editorial on recruitment in psychiatry (Brockington & Mumford, 2002; Storer, 2002). Recruitment and retention is surely one of the most important challenges for British psychiatry today. It may be of interest to point out that not only have similar recruitment problems been identified in Wales but also that research into this is ongoing.

An initial qualitative study using grounded interviews with medical students, pre-registration house officers and psychiatrists of all grades across Wales has been completed. On the basis of this, a questionnaire was developed which has been distributed to all psychiatric senior house officers, specialist registrars and staff grade doctors in Wales. These questionnaire explore various aspects of psychiatric training experience and motivations behind career intentions. From the responses, we hope to gain a greater understanding of the reasons behind the crisis.

From the initial work, one theme that is emerging is the importance of a positive training experience, initially at undergraduate level but also at later stages in a doctor’s career. An enthusiastic teacher was particularly seen as a strong motivator to entering psychiatry. However, this was counterbalanced by the effect of the stigma of entering a specialty perceived as inferior. As well as problems with recruitment, there are increasing problems with retention of senior house officers, and subsequent lack of applicants for specialist registrar posts. Some disincentives to continue within training seem to be the perception of demoralised consultants not providing ideal role models for young aspiring psychiatrists. This is linked to the experience of a pressurised service that lacks resources. Both these factors appear to be an affliction affecting general psychiatry to a greater extent than the other specialities. Perceived stigma directed towards psychiatrists, mental health services and patients from our medical colleagues is a worryingly common observation, and is another potentially important finding in relation to the Royal College of Psychiatrists’ ‘Changing Minds’ campaign.

Improving the CHI

Professor Burns (2002) makes some good points in his article on the Commission for Health Improvement (CHI). The CHI is a relatively new organisation and is constantly learning. Already, many of the suggestions for change to our clinical governance review process made in his article have been identified and implemented through our own processes of self-review and improvement. Such improvements include shortened clinical governance reviews and shorter, more accessible reports.

However, Professor Burns unfairly doubts the experience of CHI reviewers who undergo a rigorous assessment and training programme. He also questions the consistency of clinical governance review reports. We have developed assessment frameworks to help review managers, and reviewers make reliable and consistent assessments transparent to both the organisation and its stakeholders. This framework underpins the entire process, driving the collection of data and information and all reporting arrangements.

Professor Burns also makes unhelpful comparisons between homicide inquiries and CHI reviews. Our role is not to identify individuals to whom blame can be attributed, but to help encourage improvement where improvement can be made. Many have found CHI’s reviews a positive experience enabling the organisation to recognise strengths as well as weaknesses. In the meantime, the CHI is committed to learning and improving our own systems through constant consultation. Feedback is always welcome; even better, why not become a CHI reviewer and make your own contribution?
Declaration of interest
L.P. is Medical Director and J.C. is Director of Policy and Development at the CHI.


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Author’s reply: I am delighted that the CHI has identified similar improvements through its internal monitoring as I suggested in my editorial. I was trying to be helpful.

I did not question the assessment and training of CHI reviewers but their experience as reviewers. Obviously, CHI is new and so its current reviewers are new. A useful exercise at 2 years or so would be to report the average number of reviews conducted by members and to check the number where the second members are all first-timers. How comparable and consistent the reports are is also a judgement of outcome, not just of process. Stakeholders will make their own judgments – probably in the same way I did by reading a couple side by side and trying to compare and contrast. Time will tell but shorter reports will certainly help.

Linda Patterson’s and Jocelyn Cornwall’s comments on my ‘unhelpful comparisons’ with homicide inquiries do, however, warrant a reply. Homicide inquiry panels would also consider their aim to be ‘to help encourage improvement where improvement can be made’. The point I was trying to make is that there can be a gulf between this wholly admirable ambition and the impact of such reports (and that this impact is both direct and indirect through the media).

This point is being made infinitely more eloquently by the Cambridge University philosopher Onora O’Neill in the BBC Reith Lectures entitled ‘A Question of trust’ (O’Neill, 2002). In these she analyses with devastating precision how a pursuit of accountability and transparency at all costs can, and does, lead to the erosion of trust and, paradoxically, a reduction in disclosure and honest communication.

Having started my psychiatric training at a time when consultants really did seem free to do exactly what they wanted, I warmly welcome review and the establishment of consistent standards of clinical care. However, the age of innocence is surely passed. Professor O’Neill’s analysis is a call to more careful thought on how accountability and transparency can be achieved without damaging the process they are meant to foster. Hopefully, now it will be accepted that we can have a debate on these issues without it being seen simply as protectionism. I wish the CHI well.


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Estimating cognitive deterioration in schizophrenia

Two recent studies failed to establish a relationship between the duration of untreated psychosis (DUP) and cognitive deterioration in first-episode patients (Barnes et al, 2000; Norman et al, 2001). Both studies used the premorbid IQ (estimated using the National Adult Reading Test (NART)) minus the current full-scale IQ (measured using the Wechsler Adult Intelligence Scale (WAIS)) to measure cognitive deterioration. The validity of this approach to assessing cognitive deficit is open to question.

We examined DUP and cognitive deterioration in 42 individuals (mean age 22.3 years; s.d.=4.1) with first-episode schizophrenia (Amminger et al, 2002). The revised version of the NART and WAIS (WAIS–R) were administered at clinical stabilisation and we have since taken the opportunity to apply the NART IQ minus WAIS–R full-scale IQ approach. Current IQ was higher than the estimated premorbid IQ in 38.1% of cases, suggesting an IQ increase.

The NART has been validated in older samples. We were therefore interested in the relationship between age at admission and IQ measures. NART IQ, but not WAIS–R full-scale IQ, was positively correlated with age at admission in our sample, (r=0.331, P=0.032). The WAIS–R ‘vocabulary’ sub-test, suggested to be a better estimate of premorbid IQ than the NART (Russell et al, 2000), had also no relationship with age. It is possible that the NART underestimates premorbid IQ in young people with schizophrenia.

Age-standardised WAIS sub-tests are another method to estimate cognitive deterioration (Bilder et al, 1992). Performance on ‘information’ and ‘vocabulary’ sub-tests are relatively stable, whereas the ‘digit symbol’ sub-test is sensitive to brain insult. Bilder et al’s (1992) deterioration index (DI),

\[ DI = \frac{\text{Information} + \text{Vocabulary}}{2} - \text{Digit Symbol} \]

is based on the principle that a larger discrepancy between an individual’s best and poorest performance on cognitive functions suggests cognitive loss. We found longer DUP, male gender, higher NART IQ and younger age at depression to be independent significant predictors of the DI (Amminger et al, 2002).

A cross-sectional test score (e.g. low-average full-scale IQ) cannot indicate deterioration on its own. In the absence of longitudinal data, indices reflecting decline from premorbid levels of functioning are required. Limitations of proxy methods need to be considered and studies which aim to validate measures of cognitive deterioration should be pursued.


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Author's reply: Amminger et al raise some interesting issues. I certainly agree that the estimation of premorbid IQ, particularly in patients with schizophrenia, is challenging and that further validation studies on methods for making such estimates should be pursued.

More specifically with reference to our earlier paper on the relationship of DUP to cognitive functioning (Norman et al., 2001), Amminger et al argue for the likely superiority of Bilder et al’s (1992) index as a measure of cognitive deterioration in contrast to estimates based on NART-estimated premorbid IQ minus current WAIS full-scale IQ. In this respect they note that 38.1% of patients in a recent study by their group showed higher current IQ than NART-estimated premorbid IQ. This would, of course, suggest an increase in IQ after illness onset—an unlikely occurrence. I have examined this issue in our data-set and found such a pattern in 17.8% of our sample, with the average discrepancy among these individuals being 8.4 points. I can also confirm that in our sample, as in Amminger et al’s sample, NART scores were correlated with age at admission ($r=0.24$, $P<0.05$), but WAIS-R full-scale scores were not.

The substantive question, of course, is whether DUP is related to cognitive deterioration. Amminger et al report that they have found DUP related to deterioration based on Bilder’s index. We had reported some results using Bilder’s index in our earlier paper. I will take this opportunity to report further that when we examined correlations between our two indices of DUP and Bilder’s deterioration index they were non-significant ($r=0.06$ and $r=0.04$). We are currently pursuing the issue of whether DUP may be related to recovery of cognitive functioning during the first year of treatment.

The discrepancy between our earlier findings and those of Dr Amminger and colleagues does not appear to be explained on the basis of use of the NART rather than Bilder index. Other variables related to sample composition may be relevant. Also of potential importance is the method of measuring DUP, which, as has been suggested elsewhere (Norman & Malla, 2001), also needs to be more carefully considered and standardised. In this, as in many areas of psychiatric research, cumulative progress is dependent on careful and comparable measurement across studies. I endorse Amminger et al’s comments in this respect.


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Depression: detection and diagnosis

The October 2001 issue of the Journal reports two prevalence studies of depressive disorders (Ayuso-Mateos et al., 2001; Thompson et al., 2001). Both studies used a self-report questionnaire as an initial screening device although both avoided the sometimes reported but unjustified assertion of diagnosis based upon scores of the scales. Such scales are widely used in the manner reported by these studies and a cautionary comment is in order. There is a widespread view that the selection of instrument is unimportant so long as it is designated as a ‘depression’ scale; this is not true. For instance, the scales used in the above-mentioned studies were the Beck Depression Inventory (BDI) (in Ayuso-Mateos et al., 2001) and the depression sub-scale of the Hospital Anxiety and Depression scale (HAD–D) (in Thompson et al., 2001). These two instruments highlight very different aspects of depressive disorders (Snait, 1993). The HAD–D has 86% of its variance directed to mood symptoms (depressed mood andanhedonia) but an absence of cognitive symptoms (hopelessness, low self-esteem and guilt ideation). With the BDI the reverse is the case, with 14% directed to mood and/or anhedonia but 33% focusing on the cognitive symptoms.

There is an unfortunate tendency to refute the importance of difference based upon predominant psychopathology and even, within the realm of depressive disorders, to deny the importance of diagnosis. Indeed, the first study uses the term ‘prejudice’ when referring to the separation of disorders and frankly advocates the conflation of disorders of major depression (for which one or other of the mood symptoms is prerequisite for diagnosis) and the other group of ‘dysthymia and adjustment disorders’, which are characterised by the cognitive distortion. Until diagnostic practice is based on exact psychopathology, research will remain in its present state of confusion. For instance, the oft-repeated statement that cognitive therapy and biological treatments are of equal worth in the treatment of ‘depression’ will continue to be made. The statement may be true if no distinction is made between different depressive disorders but non-responders to the one or other treatment will have different characteristics: the psychotherapeutic approach will be more successful in the disorders based on cognitive distortion whereas the biological treatments are likely to be more effective when major depressive disorder is present.


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Factor structure of the Hospital Anxiety and Depression (HAD) scale

We would like to draw attention to the assertion by Mykletun et al (2001) that a two-factor structure best fits the Hospital Anxiety and Depression (HAD) scale, especially in individuals with mental problems. They stated that psychometric studies of this scale only involved small samples of non-psychiatric patients. However, we recently published the only factor analysis of the HAD scale based on a large population: 2669 ‘HAD completers’ from 3002 patients (89%) with major depression, DSM–IV criteria (Friedman et al., 2001).

Contrary to Mykletun et al, we found a three-factor solution using principal-components analysis with factors defined by eigenvalues $\geq 1$. One of Mykletun...
et al’ s reasons for rejecting the three-factor solution was that their third factor comprised heterogeneous items loading for both anxiety (items 7 and 11) and depression (item 14). Our three-factor structure discriminates the original depression factor and two separate constructs of anxiety: ‘psychic anxiety’ (items 3, 5, 9 and 13) and ‘psychomotor agitation’ (items 1, 7 and 11). This factor solution captured 48.6% of the variance and was relatively robust; it was not influenced by gender ratio and was also found in two random halves.

Two reasons may account for these discrepancies between our results. First, because of the high proportion of HAD scale non-completers (44%), Mykletun et al’s sample may have been biased. Patients with depression are probably not prone to answer such surveys and may therefore be underrepresented. Second, the factor structure of the HAD scale may not be stable across different categories of subjects: those with heterogeneous mental problems and those specifically suffering from major depression.

The HAD scale is not only useful for its initial screening purpose. It also showed potential ability in assessing change in specific symptoms of anxiety (‘psychic anxiety’ and ‘psychomotor agitation’ factors of the scale) during antidepressant treatment (Friedman et al, 2001). Moreover, recognition and monitoring of psychomotor agitation has several clinical implications: it is a potential side-effect of some antidepressants (Nutt, 1999), it may predict antidepressant response (Flament et al, 1999), it may predict adverse outcome and increase the risk of suicide (Schatzberg & DeBattista, 1999).

Declaration of interest
S.F. has formerly been CNS medical adviser for Pfizer France; J.C.S. has received fees from Pfizer France; J.D.G. has received fees from several pharmaceutical companies.


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Authors’ reply: Friedman et al raise doubts as to the two-factor structure of the HAD scale reported by us. The size of our sample (n=51,930) allowed us to test our finding in several sub-samples. Using principal-components analysis, the same two-factor solution was also found in all sub-samples. Reporting somatic and psychiatric problems, as well as in all age- and gender-groups from 20 to 89 years. This indicates that the two-factor structure of the HAD scale is robust and stable. Therefore, eventual minor biases due to response rates cannot account for the discrepancy between Friedman et al’s and our findings. Our third factor, which emerged only in sub-samples with low depression scores, always showed a low eigenvalue. Our results are in accordance with the conclusions of a recent literature review on the HAD scale (Bjelland et al, 2002) which concludes that a two-factor solution is most commonly found.

Friedman et al (2001) have a sample (n=2,669) characterised by major depression (DSM–IV), which corresponds to high depression and probably variable anxiety scores on the HAD scale. When performing factor analysis, composition of the sample is essential for the results. If an inclusion criterion restricts the variance and covariance of the variables entered in the factor analysis, this will influence the factor solution found. The results by Friedman et al can be interpreted as a consequence of their restriction of their sample to major depression only, as this restricts the covariance between items on the HAD scale. In our sub-sample with various mental problems (n=2,098) the two-factor solution is robust with high explained variance (82.1%).

Friedman et al’s findings are of interest, however, since they answer the question: What is the factor structure of the HAD scale when anxiety appears in major depression? Comparing the fit coefficients between two- and three-factor solutions using confirmatory factor analysis must show the advantage of a three-factor solution. Friedman et al seem to presume that the factor structure of anxiety found in major depression is identical to that found for anxiety in the general population.

The advantage of population samples is that selection bias is minimised. In several of our studies based on the unselected HUNT-II population (from the Nord-Trøndelag Health Study) we have found results at variance with those of clinical samples (Engum et al, 2002; Wenzel et al, 2002). This could also explain the discrepancy between Friedman et al’s and our results.


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Follow-up of childhood depression: historical factors
The study by Fombonne et al (2001), following adolescents with diagnoses of major depressive disorder into adulthood, raises some questions pertaining to the era when they were diagnosed (1970–1983).

First, it was only in the early 1980s that child abuse began to come into the awareness of professionals and, a few years later, the general public. Therefore, it is possible that some of the young people identified with depressive disorders may have had a history of sexual abuse which was not disclosed or enquired about. This raises the question of what would have been the outcome in those young people who had been sexually abused had they made disclosures and had appropriate therapeutic intervention for this. It is well known that
childhood sexual abuse is a significant factor in the histories of some adults presenting with depressive syndromes.

Second, this period was also a time when attention-deficit hyperactivity disorder (ADHD) was not recognised and hyperkinetic disorder was only rarely diagnosed. Some of the young people, especially those in the comorbid conduct disorder/major depressive disorder group, may have had undiagnosed and untreated ADHD. Certainly this was long before the use of psycho stimulants on a wider basis in the UK and it is possible that some of these young people untreated may have been more vulnerable to development of depressive syndromes because of untreated attentional and other behavioural problems impacting on their self-esteem.

Third, although antidepressants were in use by child and adolescent psychiatrists when the diagnosis was major depressive disorder, they may not always have been used in young people with major depressive disorder with comorbid conduct disorder because of the risks of overdose in such a population. Tricyclic antidepressants were the predominant antidepressants used at that time in this population. With the advent of selective serotonin reuptake inhibitors, child and adolescent psychiatrists probably began prescribing more antidepressants in the comorbid conduct disorder/major depressive group because of the lower risk of serious harm in overdose. This raises the possibility that more effective treatment of these young people might also have an impact on their outcomes in adult life.


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Author’s reply: The comments of Hynes & McCune raise pertinent questions. As they point out, it is possible that sexual abuse in childhood might have influenced the onset of juvenile depression, and also the likelihood of adult depression recurrence in our sample. In this study, we have collected data on sexual abuse, using both a review of medical charts at the time of Maudsley attendance and from adult interviews based on the Childhood Experience of Care and Abuse (CECA) measure. The effect of sexual abuse in childhood on patterns of adult depression recurrence will be investigated in the next analyses of this data-set, with particular attention given to differential risk processes according to childhood comorbidity.

Regarding comorbid ADHD as a risk factor for adolescent depression, particularly in the depression group with comorbid conduct disorder, we found a significantly increased rate of ADHD in the comorbid group, as we reported (Fombonne et al, 2001a, Table 2). Yet, it is plausible that the rate of ADHD in this sample was underestimated as many cases were ascertained before ADHD or hyperkinetic disorders were fully recognised as valid diagnostic entities. Nevertheless, our findings suggest that it is possible that ADHD might have been implicated in the development of conduct symptoms in the comorbid group although, because of the small sample size and likely understimation of ADHD in that group, we cannot test for the specific contribution of (untreated) ADHD in the onset and recurrence of depression.

We had provided explicit data on the use of tricyclic antidepressant drugs during childhood years and found that the rate of prescriptions of these drugs was significantly higher in the non-comorbid group than do the comorbid group (48.4% vs. 30.2%, P = 0.032; see Fombonne et al, 2001a). Most of these prescriptions were for amitriptyline and relied on dosages much lower than those considered appropriate by today’s standards. Although the rate of antidepressant use was lower in the comorbid group, antidepressants were nevertheless often prescribed in that group too. Obviously, we could not assess whether or not use of tricyclic medications in that sample influenced long-term outcomes, since our study relied on an observational design. The interesting aspect of these data was to point to the frequent use by practising child psychiatrists of antidepressant drugs (irrespective of their known efficacy) in this sample of youths with depression assessed in the 1970s at a time when child and adolescent depression was largely ignored in professional training and in the literature. Furthermore, the data indirectly validated our diagnostic procedures.

Most of the comments by Hynes & McCune raise questions about the mechanisms underlying recurrence of depression in adulthood following a first episode in childhood or adolescence. The findings of our study (Fombonne et al, 2001a,b) indicated that relapse rates were similar, irrespective of the presence of comorbid conduct disorder in childhood. This result is important in its own right as it refutes previous hypotheses that depression, when occurring in the context of conduct disturbances, reflected mostly local psychosocial circumstances and was not associated with long-term heightened risk of affective disorders in adulthood.

This study was designed to assess mechanisms underlying recurrence of depression in adult life and further reports will address the role of early childhood experiences (such as sexual abuse), life events, family history and individual psychological characteristics on the patterns of adult depressive recurrence. It could well be that, in spite of having similar rates of relapse in adulthood, the mechanisms of depressive recurrence differ for the two groups included in this study, according to childhood comorbidity.


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Genetics of Down’s syndrome and Alzheimer’s disease

In an extremely interesting article which touched upon early-onset dementia I feel that Dr Holmes (2002) failed to mention Down’s syndrome as being a particular risk factor for the development of early-onset Alzheimer’s disease because of triplcation of the amyloid precursor gene. It is well known that almost all adults over the age of 40 years with Down’s syndrome display Alzheimer’s neuropathology (Mann, 1988) and the prevalence of dementia in people with Down’s syndrome is 0–4% under the age of 30 years rising to 29–75% at 60–65 years of age, which falls under the category of early-onset Alzheimer’s disease (Zigman et al, 2001).
et al, 1997). Studies have shown that the prevalence of Alzheimer’s disease in those with learning disability, especially Down’s syndrome, is higher than in those with no learning disability (Patel et al, 1993).

The occurrence of Alzheimer-like neuropathology in Down's syndrome suggests that the genetic defect for familial Alzheimer’s disease might reside on chromosome 21, which was, therefore, the first of the 22 autosomes to be tested using a genetic linkage strategy (McGuffin et al, 1994).

Dr Holmes’s article was published among papers with the overall topic of old age psychiatry; early-onset Alzheimer’s disease tends to fall within the remit of old age psychiatry except in those with Down’s syndrome, who remain within learning disability services. From all the information I have gathered on Alzheimer’s disease I have assumed that the clearest evidence for a genetic contribution to the aetiology of Alzheimer’s disease is its association with Down’s syndrome, which surely deserves a mention when discussing this specific area.


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Authors’ reply: I thank Dr Shamas-Ud-Din for showing interest in my paper and would have to concur with the general criticism that Down’s syndrome should have been mentioned. I would like to say, in my defence, that the article was written within the remit of ‘advances in old age psychiatry’. A large number of old age psychiatric services see patients with Alzheimer’s disease regardless of their age of onset and hence there was a need to cover some of the aspects of the genetics of early-onset Alzheimer’s disease. However, I am unaware of any old age psychiatric service within the UK that routinely sees patients with Down’s syndrome and Alzheimer’s disease. This defence does not, however, excuse a restricted view that is damaging both to patient management and basic research.

Clearly, no patient should be excluded from expert dementia services because of their learning disability. In addition, there is much to learn about the genetic influences on the development of Alzheimer’s disease in patients with Down’s syndrome and on its clinical phenotype. Thus, as well as the effects of triplexation of the amyloid precursor gene, the presence of the APOE e4 allele also appears to be associated with an increased risk of developing Alzheimer’s disease (Deb et al, 2000). The effect of the presence of the APOE e4 allele on age of onset is still unclear but, unlike in those with no learning disability, the presence of APOE e4 appears to be associated with an earlier age of death (Hardy et al, 1994). At post-mortem the brain lesions and cholinergic losses seen in individuals with Down’s syndrome are the same as those seen in both early- and late-onset Alzheimer’s disease. However, despite these neuropathological findings, the evidence for the beneficial effects of cholinesterase inhibitors in patients with Down’s syndrome and Alzheimer’s disease is still largely anecdotal (Kishani et al, 1999). It is clear that the two specialities, old age psychiatry and learning disabilities, have much to learn from each other.


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Clinical diagnosis of dementia with Lewy bodies

It is clinically important to differentiate dementia with Lewy bodies (DLB) from other types of neurodegenerative dementias because of the prospect of better response to drugs such as cholinesterase inhibitors as well as the risk of development of neuroleptic sensitivity. However, usefulness of the commonly used definition of DLB (McKeith et al, 1996) remains to be established in community and primary care settings. The findings of the Islington study (Stevens et al, 2002) suggest that DLB is a clinically significant type of dementia in the community.

We had an opportunity to look at the prevalence of DLB in a small but representative community sample of patients meeting DSM–IV criteria for dementia (American Psychiatric Association, 1994) in Kerala, a southern state of India. These cases were identified using a novel case-finding method (Shaji et al, 2002). We identified 33 cases of dementia in the study area with a population of 1979 aged >60 years. A psychiatrist assessed all the subjects at their residence and assigned a DSM–IV diagnosis. Seventeen people with dementia (52%) met criteria for Alzheimer’s disease, 12 (36%) were diagnosed as having vascular dementia, while in 4 (12%) the dementia was felt to be secondary to other conditions such as chronic obstructive pulmonary disease, cardiac failure, Parkinson’s disease and subdural haematoma. Three patients (9%) met criteria for probable DLB and one (3%) met criteria for possible DLB when the consensus criteria (McKeith et al, 1996) were applied. The combined prevalence of DLB was therefore 12%. All these patients had earlier met the criteria for Alzheimer’s disease as per DSM–IV.

On retrospective application of the separate ‘clinical’ diagnostic criteria used in the Islington study to our case records, only two of our patients met those criteria, reducing the proportion of patients with DLB to 6%. This is strikingly similar to the figure reported in the Islington study. One limitation of these ‘clinical’ criteria is that they are too restrictive a definition of DLB. If visual hallucinations are indeed such an important diagnostic marker, then the consensus criteria themselves could be modified by making visual hallucinations an essential symptom for the diagnosis of
One hundred years ago

**Verbal obsessions. By James Shaw, M.D., M.Ch.R.U.I.**

Magnan defines an obsession as being a mode of cerebral activity in which a word, thought, or image rises into consciousness — involuntarily and without discomfort when physiological, but forcibly and with painful persistence when pathological. Verbal obsessions are those in which isolated words—mostly obscene or blasphemous—constitute the morbid besetment. They should be distinguished from coprolalia, obscene speech; from the blasphematory mania of Verga, which he described as a special variety of obsession taking the form of oaths, blasphemies, or indecent utterances; and from the onomatomania of Charcot and Magnan, in which a word cannot be recollected without a painful searching of the memory, or a word or phrase is ever present in consciousness and must be emitted at intervals, its utterance being repressed for a short space only at the cost of extreme and constantly increasing mental pain. In my cases the besetting words were never uttered. The words differed from psychical or psychomotor hallucinations in that they were never spoken of as “voices in the head” nor as “voices” at all. Verbal obsessions may constitute the leading feature of a sort of obsessional aberration, as in the second case below, or an early symptom of a form of insanity, an obsessional melancholia, which appears to me to be as much an entity as hypochondriacal melancholia is. The first case exemplifies the induction of obsessional melancholia by verbal obsessions.

**REFERENCE**


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Recruitment and retention in psychiatry
C. O’Gara and J. Sauer

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