aggression (62% as defined in the study) should be on medication.

The evidence that drug treatments have major benefit in the management of such behaviour remains less than convincing (Schneider *et al* 1990, Schneider & Sobin, 1991) and side-effects of most drugs involved can be disabling. Effects such as lethargy, akathisia and akinesia can develop insidiously, and are often not recognised, being attributed to the disease process rather than to the drug. Treatment is often, perhaps usually, given without proper informed consent (Jackson & McGrath, 1996).

Physical aggression in people with dementia is not simply due to the disease process, but is affected by many factors including the expectations, training and tolerance of carers; the care environment; physical illness or discomfort; and the personality of the aggressor. These problems must be taken into account, and modified where possible.

A decision to manage physical aggression using regular drug treatment should not be taken lightly. Rather than "considering a trial withdrawal every 8 months", whether or not to continue use of medication should be continually reassessed. It is important to look at alternative managements at all times, and consider the risk: benefit ratio, that is, the not insignificant risk of side-effects compared with any perceived benefit. A Scottish guideline group (Scottish Intercollegiate Guidelines Network, 1998) suggests that "neuroleptics should only be considered for patients with serious problems, in particular psychotic symptoms, or in the presence of serious distress or danger from behaviour disturbance". This document also discusses other aspects of management.

Hope *et al*'s article is a welcome addition in its description of the behavioural and psychiatric problems associated with dementia, particularly in discussing its natural history. However, their "clinical implications" on medication use are not supported with their findings.

Hope, T., Keene, J., Fairburn, C. G., *et al* (1999) Natural history of behavioural changes and psychiatric symptoms in Alzheimer's disease. A longitudinal study. *British Journal of Psychiatry*, **174**, 39–44.

Jackson, G. A. & McGrath, A. M. (1996). Consent to treatment. *British Journal of Psychiatry*, **169**, 382–383.

Schneider, L. S., Pollock, V. E. & Lyness, S. A. (1990) A metanalysis of controlled trials of neuroleptic treatment in dementia. *Journal of the American Geriatric Society*, **38**, 553–563.

— & Sobin, P. B. (1991) Non-neuroleptic medication in the management of agitation in Alzheimer's disease and other dementia: a selective review. *International Journal of Geriatric Psychiatry*, **6**, 691–708.

Scottish Intercollegiate Guidelines Network (SIGN) (1998) *Interventions in the Management of Behavioural and Psychological Aspects of Dementia*. Edinburgh: SIGN.

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Author's reply: We welcome the letter from Dr Jackson. We did not intend in our paper to make any presumptions about the efficacy or otherwise of medical treatment for aggressive behaviour. This is an issue outside the remit of that paper. The point we wanted to stress is that since the natural history (untreated) of most behavioural problems in dementia is for them to remit spontaneously, it is important that patients who are given medication are not left on that medication without a trial of withdrawal. Our purpose, like that of Dr Jackson, in discussing the clinical implications of our findings was to stress the point that a patient should not be left on medication without review.

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Detecting vascular pathology in vascular dementia

Sir: In their study of older people on the Camberwell Dementia Case Register, Holmes *et al* (1999) highlight the influence of prevalence rates and mixed pathology on the validity of current diagnostic systems. However, it is possible that both clinical and neuropathological classificatory systems have underestimated the presence of vascular pathology.

It is unclear as to precisely how the location of infarcts influenced quantitative assessment of vascular pathology in the study, as it is known that strategic infarcts in frontal/subcortical structures may be associated with cognitive impairment, but are not traditionally seen as implicated in the development of dementia (Rao & Howard, 1998). It is also surprising that the neuropathological assessment of cerebrovascular disease was confined to infarcts, as Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINDS–AIREN) criteria for vascular dementia also allow the presence of extensive periventricular white matter lesions (Roman *et al*, 1993).

Although the presence of 'pure' vascular pathology comprised less than 10% of the total sample, it is striking that four people with infarcts alone at post-mortem were mis-classified as non-probable vascular dementia according to NINDS–AIREN criteria. Three of these had no clinical evidence of focal neurological signs; such 'silent' strokes are known to be associated with cognitive impairment (Price *et al*, 1997). Lastly, given the high prevalence of mixed pathology in the general population, it is suggested that the incorporation of mixed pathology into diagnostic criteria for both Alzheimer's disease and vascular dementia may be needed for research and treatment. The problem with mixed pathology is that it represents a nosologically heterogeneous group of disease processes, further compounded by the inextricable relationship between vascular risk factors and Alzheimer's disease (Prince *et al*, 1994). The way forward may be to improve the sensitivity of current diagnostic instruments by allowing a broader definition of both cerebrovascular disease and cognitive impairment, taking into account the wide range of aetiologies encountered in vascular dementia. This will not only encompass a larger group with 'pure' cerebrovascular lesions, but also serve to treat a distinct disorder that is still unduly influenced by the neuropsychological characterisation of Alzheimer's disease.

Holmes, C., Cairns, N., Lantos, P., *et al* (1999) Validity of current clinical criteria for Alzheimer's disease, vascular dementia and dementia with Lewy bodies. *British Journal of Psychiatry*, **174**, 45–50.

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