PSSRU

Mental Health in Old Age Bulletin
Issue 4

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Layout by: Sue Martin

Discussion Paper M119
November 2003

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MENTAL HEALTH IN OLD AGE BULLETIN
ISSUE 4

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EDITORIAL

Vaccines and Alzheimer's disease

Nicoll *et al*. (2003) Neuropathology of human Alzheimer's disease following immunization with amyloid ß-peptide. *Nat. Med*., Apr 9 (4), pp.448-452.

To date, therapeutic options for the treatment of AD have focused on modifications of neurotransmitter systems to maximise the remaining activity in the neuronal circuits damaged by the disease. Such an approach can provide transient symptomatic benefits, and whilst it may ameliorate the course of the disease it does not address the underlying disease process and will not stop the progression of the disease itself. One suggestion that has stood the test of time is that Abeta amyloid protein (Ab), which forms deposits known as amyloid (Ab40-42) plaques, is central to the diagnosis of AD and may be a cause rather than result of the disease. All genetic variants of AD are linked to increased synthesis or deposition of Ab42 and its deposition precedes clinical symptoms of AD with brain levels correlating with disease severity. Abeta of course may have a necessary physiological role so one explanation for the accumulation in plaques is a breakdown in the transport system and indeed sporadic AD patients do have reduced clearance of Ab42 through the CSF.

Some exciting data on immunisation with Abeta 42 in a transgenic mouse model of AD showed that it reduced both age-related accumulation of Abeta in the brain and associated cognitive impairment (Schenk, Janus, Morgan) the crucial question with animal data is whether it translates to the clinic and whether AB immunotherapy could influence human AD pathology. Elan pharmaceuticals have developed a vaccine AN1792 (combined with the adjuvant QS-21), a preparation of aggregated human Ab42 which has been shown to markedly reduce the extent of the progression of AD like amyloid plaques in mouse models. A recent multi-centre study in the UK involving 80 patients with mild to moderate Alzheimer's disease, undertaken to test the safety, tolerability and immunigenicity of AN1792 (QS-21) has completed and the results which are in press will be presented in the Montreal Springfield symposium in April. This study, which was the first study in AD patients, has generated considerable interest in the research community already as a case report of the brain histology from one patient who died following a decline with features of viral encephalitis has been published. Post-mortem examination found remarkably similar reductions in, and distribution of, amyloid in the brain to those found in the mouse model, and this has now been confirmed from vaccination cases in the subsequent larger phase 3 international trial that had to be discontinued as a result of several non fatal cases of encephalitis. Despite this unfortunate side-effect it seems that modification of the vaccine may be possible which would retain similar effects on amyloid deposition without the inflammatory response. It is thought that the inflammatory response, predominantly a T-cell mediated meningoencephalitis, is triggered by an epitope near the N terminal of the Ab42 peptide that is not the same epitope as that necessary for the immune response. It may therefore be possible to immunoconjugate the desirable fragments of Ab42 to an egg albumin T-cell epitope in a multi-antigen peptide that may produce the desired immune response and not the inflammatory response and so far this has been successful *in vitro*. Another alternative is to use passive immunization with human monoclonal antibodies and

such a study is under way in the US but results in animal models whilst showing similar changes to the active immunization have not been as profound. It is crucially important to understand the effects of the vaccine in those patients exposed to it so far as they offer a unique opportunity to understand what the long-term effects will be. Interest in vaccination or passive immunisation as a method of preventing amyloid deposition or removing amyloid from the Alzheimer brain is still considerable and so is the need to understand whether there are any indications as to why some people develop an overwhelming immune response and others do not. This exciting area of research - stimulating an immune response, at first seemed counter-intuitive in view of the belief that inflammation *per se* may be a cause of deterioration in AD, but following the remarkable effect of Ab immunotherapy in animal models of AD and *in vivo* effects of AN 1792 on amyloid pathology it seems important to explore the hypothesis further.

The early preclinical observations with second-generation approaches are encouraging and there is no doubt that even if they do not produce a treatment Ab immunotherapy studies create an important platform for future evaluation of the amyloid hypothesis in the clinic.

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ARTICLE

The effects of improving hearing in dementia

Introduction

Impairment of hearing is a often overlooked and even accepted as being normal in the elderly population. It is a cause of significant morbidity and is often treatable. The prevalence varies from from 30-60% in community samples and up to 97% for residents of institutions (Herbst and Humphrey 1980). In a study of 30 chronically institutionalized residents of a veterans administration facility, only 17% had normal hearing (Weinstein and Amsel 1986).

Hearing impairment increases the effort required to recognise speech, leaving less reserve for rehearsal and later recall. It is reasonable to predict that, in dementia, that the cognitive effects of hearing loss are magnified. There are several possible links between dementia and hearing loss. Hearing loss can induce social isolation, which can lead to disorientation.

Depression is also associated both with poor hearing and with apparent cognitive impairment in the form of pseudodementia (Uhlmann *et al.,* 1989).

Neurodegeneration in Alzheimer's disease may cause specific damage to those parts of the cerebral cortex involved in auditory processing, but the effects of damage to higher cortical areas associated with language processing may be more profound (Kurylo *et al.,* 1993).

Aims of the study

This study monitored the effects of hearing aids to people with hearing loss and dementia.

Efficacy was measured in terms of cognitive function, non-cognitive symptoms and carer burden. The acceptability and compliance with hearing aids was measured, as was the improvement in hearing performance.

Methods

Patients were a drawn from 3 hospitals in the Greater Manchester area. Ethics committee approval was obtained prior to the study. All subjects had a diagnosis of primary dementia according to DSM IV criteria (American Psychiatric Association 1994). Subjects were only included if they had a pure tone average hearing loss in the speech frequencies of ≥40dBHL after removal of any occluding wax. Each subject had a carer able to give an account of the subjects' state during the course of the study, though not necessarily through living with the subject. Subjects were excluded if they were in possession of a functioning hearing aid.

Active ear disease was treated and patients reconsidered for the trial. An audiological relevant history was taken from each subject with the help of their carer. After removal of wax, pure tone thresholds (air and bone conduction) and uncomfortable loudness levels were ascertained for each ear using the British Society of Audiology (BSA) Recommended procedures. Middle ear function was measured using tympanometry Patients who had hearing thresholds of 40dBHL or worse, averaged at 0.5, 1.0, 2.0 and 4.0 kHz in the better hearing ear had an ear impression made according to BSA recommended procedures.

Patients were fitted with the optimum NHS post-aural hearing aid, set according to the National Acoustics Laboratory (revised) prescription formula (Byrne and Dillon 1986), using real-ear probe tube microphone measurements. The patient and the carer were instructed in the use and management of the hearing aid. Particularly close monitoring by the research staff followed the fitting of the aid, so that compliance was maximised. This study did not include a control group.

Outcome variables

The primary outcome measures were the Mini-Mental State Examination (MMSE), the Clinical Global Impression of change (CGI) (independently rated), and the Nursing Home Hearing Handicap Index for both patient (NHHHIP) and carer (NHHHIC). All ratings were carried out after 1, 3 and 6 months. Other measures were Euro-ADAS, the Instrumental Deterioration for Daily Living in Dementia scale (IDDD) MOUSEPAD, the Cornell scale for depression in dementia, the Carer Strain Scale, and the Carer Burden (visual analogue) scale. Subjects were visited every two weeks for the first twelve weeks and then every 4 weeks thereafter until the end of the study at 24 weeks. The NHHHIP and NHHHIC were completed at each visit. Hearing aid diaries were kept to record the acceptability of the hearing aids to the subjects.

Statistics

Data analysis was performed by the Medical Statistics department in South Manchester.

Baseline to end of study changes in outcome variables were compared using paired t-tests or Wilcoxon matched pairs tests as appropriate. The effect of compliance with hearing aid was assessed by repeated analyses of variance. Results are shown with 95% confidence intervals.

Results

35 patients entered the study. One withdrew consent after the baseline assessments, one after three months. One died after the first month, and one after three months. 31 subjects completed the study. 25 (74%) of the patients were female, and the mean age of participants was 84 years (range 67-96, standard deviation (s.d.) 6.6). The average unaided pure tone threshold of the better hearing ear was 59.32 dBHL (s.d. 9.55) indicating moderate to severe hearing loss.

Table 1: Primary outcome variables

		Baseline (n=35)	Week 4 (n=29)	Week 12 (n=26)	Week 24 (n=27)	Statistics: mean difference, 95% CI, significance
MMSE		18.1			16.1	2.0, 0.51 – 3.2, p=0.008
CGI	Worse		13%	27%	28% (n=24)	
	Same		57%	35%	30%	
	Better		30%	38%	42%	
NHHHIP (reduction is improvement)		27.5			20.7	6.8, 1.9 – 10.7, p=0.007
NHHHIC (reduction is improvement)		34.3			21.7	12.6, 8.4 – 17.0, p=0.001

95% CI = 95% Confidence interval, ns = not significant

Table 1 shows outcome variables at baseline, and weeks 4, 12 and 24.

The MMSE scores show the expected decline for this subject group (Stern *et al.,* 1994).

However, the change score shows that over the 24 weeks of the study, less than 30% deteriorated, and more than 40% improved globally. Raskind (Raskind *et al.,* 2000) found that 87% of untreated patients with a similar degree of cognitive impairment to our subjects, remained unchanged or declined over a similar study duration. This compares to a total of 58% who have remained unchanged or declined in our study. (x^2 analysis, $p<0.001$). While comparisons between trials using different interventions have questionable validity, there is no reason to suggest that the deterioration in the untreated group would be any different to that expected in the present study.

Both measures of hearing handicap show improvements (reduction in scores) over the duration of the study, and the degree of improvement is greater in the carers' version of the scale, compared to the subjects' version.

A small number of subjects had their audiograms repeated. Test-retest differences between audiograms were found to be within normal variability, and there was no evidence to suggest unreliability of audiometric measures.

The hearing aid diary records showed that there was a decline in the use of the hearing aid over the 24 weeks. By the end of the study, only 56% were wearing the aid on every or most days. However, there was an association between compliance and reduction in social handicap. (mean decrease on NHHHIC = 17.4 compared to 8.1 for less compliant group, p=0.034). Other outcome measures were not affected by compliance with hearing aid use.

Discussion

This study has shown that:

- Patients with established dementia and hearing impairment benefit from the provision of a hearing aid.
- The improvements in the hearing can be measured using assessments previously validated in people without dementia.
- There was a correlation between carers' and patients' estimates of improvements of hearing.
- Simple removal of earwax can lead to significant hearing improvement in 10% of patients presenting with hearing loss.

This study has shown that patients with dementia can both tolerate the audiological testing procedure, and the wearing of hearing aids. This suggests that to confine hearing aid provision to those who are more mildly impaired may be to deny benefits to some patients who will be more tolerant of them. The study also serves as a reminder that otoscopic examination can lead to significant reduction in morbidity.

The measures chosen were those commonly used in such research. However, their relative insensitivity to change from such low baseline scores may have contributed to the negative findings. The annual rate of decline in MMSE from a baseline of 18

has been shown to be 3.6 (Morris *et al.,* 1993) and this is perhaps too great a rate of decline for the hearing aids to reverse. There was no particular pattern to the psychopathology as recorded by the MOUSEPAD. Although the sample was small, it might have been expected that delusions or over-valued ideas would be more frequent than the expected 30% (Allen and Burns 1995), but this was not the case in this patient group.

Conclusions

This study underlines the benefits of providing hearing aids to people with dementia. Future studies may consider including a comparison group – perhaps with a non-functioning hearing aid or with a randomised staggered start design or randomised withdrawal.

Acknowledgements

This study was supported by Defeating Deafness through a grant from Lloyds TSB. The study was carried out in three NHS facilities supported by Research and Development infrastructure.

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CURRENT KEY ISSUES

Change in rates of cerebral atrophy over time in early-onset Alzheimer's disease: longitudinal MRI study.

Chan, D. *et al*. (2003) Change in rates of cerebral atrophy over time in early-onset Alzheimer's disease: longitudinal MRI study. *Lancet*, 362 (9390), pp.1121-1122.

Alzheimer's disease is characterised by an accumulation of amyloid plaques, neurofibrillary tangles and neuronal destruction. These characteristics are detected by magnetic resonance imaging (MRI) as cerebral atrophy in general and medial temporal lobe in particular. The rate of the progression of these atrophic changes with the progression of the disease is not known. Chan and his colleagues followed up 12 patients with early onset Alzheimer's disease from a pre-symptomatic stage through to moderately/severe dementia. All participants had serial volumetric MRI scans and MMSE scores were recorded at the time of each scanning. Hierarchical regression models with quadratic terms and time were used to calculate the yearly percentage losses in brain volume. The results showed that the mean yearly loss of brain volume was less (2.8%) in mild dementia and rose to a significantly higher level (0.32% per year) as the disease progressed. The acceleration of the brain atrophy during the progression of disease as shown in this study is in accord with other studies that have shown greater atrophy in patients with more severe disease (Murphy *et al* 1993 & O'Brien *et al*., 2001). This study also showed individual variation in the rates of atrophy between patients which accords with the clinical variability in functional decline reported by other studies (Fox *et al*., 1999). Findings of this study reinforce the need for early diagnosis and therapeutic intervention in Alzheimer's disease.

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Risk factors for mild cognitive impairment if the cardiovascular health cognitions study: part 2

Lopez, O.L *et al*. (2003) Risk factors for mild cognitive impairment if the cardiovascular health cognitions study: part 2. *Archives of Neurology,* 10 pp.1394-9.

Mild cognitive impairment is a clinical syndrome representing a stage between normal aging and dementia. It is characterised by subject memory complaints and object memory impairment for age and education but largely intact cognitive functions and preserved activities of daily living. (Patterson *et al*., 1999).

This study by Lopez *et al* was part of the Cardiovascular Health and Cognition Study.

The study was aimed at examining the factors that predicted the development of mild cognitive impairment. For the participants from the period 1991 to 1994 when MRI of the brain was done, a detailed neuropsychological, neurological and medical evaluation to identify the presence of mild cognitive impairment of dementia was done between 1998 and 1999. Risk factors of mild cognitive impairment at the time of MRI were identify by using logistic regression controlling for age, race, educational level, baseline Modified MMSE and a Digit Symbol test scores. All measurements of depression, MRI findings (atrophy, ventricular volume, white matter lesions and infarcts), the presence of apolipoprotein (APOE) E4 allele, hypertension, diabetes and heart disease were taken into account. The result indicated that out of 3608 participants, 577 developed mild cognitive impairment and it was found to be associated with race (African American), low educational level, low Modified MMSE and Digit Symbol tests scores, cortical atrophy, MRI identified infarcts and measurements of depression. The investigators further divided mild cognitive impairment into amnestic type and multiple cognitive deficit type based on the prominent area of cognitive deficit. The amnestic type of mild cognitive impairment was found to be associated with MRI identified infarcts, the presence of APOE 4 low Modified MMSE scores the multiple cognitive deficits type mild cognitive impairment was associated with low Modified MMSE and Digit Symbol tests scores. The study concluded that the development of mild cognitive impairment is associated with measurements of cognition and depression, racial and constitutional factors and cerebral vascular disease. Early cognitive deficits seem to be the common denominator for the two forms of mild cognitive impairment and the presence of cerebral vascular disease and APOE E4 is associated with amnestic type of mild cognitive impairment.

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Clinically significant drug interactions with cholinesterase inhibitors: a guide for neurologists

Bentue-Ferrer, D. *et al*. (2003) Clinically significant drug interactions with cholinesterase inhibitors: a guide for neurologists*. CNS Drugs* 17 (13), pp. 947-63.

In this review article, Bentue-Ferrer and his colleagues have presented the possible sources of pharmacokinetic and pharmacodynamics drug interactions involving cholinesterase inhibitors for the guidance of practitioners. They have emphasised the need for the awareness of these two drug interactions as cholinesterase inhibitors are being used increasingly for the treatment of dementia. Thus raising the possibility of their used in combination with other types of drugs in elderly patients with dementia. The four cholinesterase inhibitors that are currently used world-wide include tacrine, donepezil, rivastigmine and galantamine.

Principally established and clinically relevant drug interactions include tacrine and the drugs metabolised by the cytochrom P450 (CYP) 1A2 enzyme as well as tacrine or donepezil and antipsychotics which results in the appearance of parkinsonian symptoms. Galantamine's bioavailability is increased with the coadministration of paroxetine, ketoconazole and erythromycin. Rivastigmine is metabolised by esterases rather than CYP enzymes, unlike other cholinesterase inhibitors and is less likely to be involved in pharmacokinetic drug interactions. Care must be taken to reduce the risk of inducing central (excitation, agitation) or peripheral (bradycardia, loss of consciousness, digestive disorders) hypercholinergic effects due drug interactions with cholinesterase inhibitors. This review of the literature does not reveal any alarming data about the drug interactions but does highlight the need for prudent prescription particularly when cholinesterase inhibitors are given in combination with psychotropic or antiarrhythmic drugs. Other possible interactions involving the coprescribed antidementia drugs like Memantine, antioxidants and cognitive enhancers remain an open area requiring prudent use.

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BOOK REVIEW

Alzheimer, the life of a physician and the career of a disease

Maurer, Konrad & Maurer, Ulrike (2003**)** *Alzheimer, the life of a physician and the career of a disease* translated by Neil Levi with Alistair Burns., New York, Columbia University Press.

This is a remarkable book which, as the title suggests, has dual aims: providing the reader with both a biography and an insight into the genesis of a disease. It thus combines a description of Alois Alzheimer's progress through childhood and adolescence, medical school and subsequent practice with an insight into the history of medical psychiatry at the end of the nineteenth century and the beginning of the twentieth. In brief it details the life of a person who was described by his

contemporaries as ‘the psychiatrist with the microscope’ yet remained committed to his family roots and offspring. The authors have given prominence in the text to both these aspects of Alzheimer’s life, painstakingly gathering information from his descendents.

As such it is compelling reading, a fact emphasised by the chapter structure. The first chapter is, in essence, the modern equivalent of an edited version of the case notes of the renowned patient, ‘Auguste D’ and the last chapter chronicles the developing knowledge base of Alzheimer’s disease following the death of the man who discovered it and the greater public recognition that has accompanied this. In contrast the intervening five chapters chronicle the life of Alzheimer from his birth in Marktbreit to his death in Breslau fifty two years later. These afford the reader a rare and valued insight into the life and career of physician who combined clinical practice and scientific research. It documents his student days and subsequent practice in Frankfurt, Heidelberg and Munich and Breslau, from the post of intern at the Mental Asylum at Frankfurt am Main to the chair of the medical faculty at the University of Breslau. However, even these chapters contain details of family life and in this way illustrate the importance of both work and kinsfolk to this indomitable man.

Reading this book one has a sense of taking a journey through time. The painstaking detail which characterises the way in which the material is presented leaves the reader with a sense of historical perspective in relation to both societal and family norms and practice within psychiatry at the end of the nineteenth century and the beginning of the next century. In this sense it can be deemed to fill a third perspective, additional to the two inherent in the title, that of a historical text. Thus in every sense this book can be described as ‘a good read’.

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WEBSITE REVIEW

Resource Discovery Network (RDN)

The RDN is an extremely valuable resource for anyone who needs accurate information at their fingertips. It is an ever-growing collection of websites that have been evaluated for quality and accuracy, produced by librarians who are well acquainted with information sources in a particular area; it is updated regularly and existing links are checked for currency. The purpose of the RDN is to support tutors, students and researchers in higher education by providing access to the best of the World Wide Web.

The part of the RDN of particular relevance to readers of the Bulletin is OMNI which we will look at in a future issue. But for now let’s turn our attention to their excellent tutorial which can help you to improve your Internet searching skills. The tutorial is available at http://www.vts.rdn.ac.uk and is actually a set of 61 subject-specific tutorials giving information about the best Internet resources. The tutorial of particular interest to Bulletin readers will most likely be Internet Medic and it would

probably repay any time investment you make in it, by alerting you to sites you can trust and turn to again and again in the future.
The following is reproduced from the VTS site and describes its features.

The Virtual Training Suite

What's in it?

Each tutorial has four main sections:

TOUR gives an orientation to the sorts of Internet resources available for your subject and presents a selection of "must-see" sites

DISCOVER introduces some ways to find resources on the Web, including subject gateways, search engines and Web directories. It discusses how to choose a Web search strategy.

REVIEW discusses some issues of information quality on the Internet, for instance how you can tell whether a document found on the Web is authoritative.

REFLECT summarises practical ideas for efficient use of the Internet and gives some fictional scenarios illustrating advantages as well as pitfalls.

As well as being organised into sections, each tutorial has three strands:

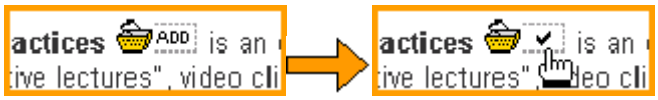
- for Teachers of the given subject
- for Students and
- for Researchers.

Use the navigation bar on the left of the screen to jump to a particular section, or simply use the "Next" buttons to read through the whole tutorial.

Additional Features

The Links Basket

This feature allows you to collect recommended links for later viewing. Clicking on the basket icon in the navigation bar brings up all your chosen links on one page. You can then visit and/or bookmark the sites.



Important: Links Basket contents are lost when you leave the tutorial – remember to print or save your list.

Interactive Quizzes

Each main section of a tutorial closes with a short test of your understanding. Your responses are only held in your Web browser and not passed to anyone else. Each quiz question gives interactive feedback, often with hints or further information.

Some of these use a split-screen to ask you a question about a specific site. When this happens, you may have to use the two scroll bars on the right of the screen to read through both the external site and the exercise.

Glossary of Internet Terms

Do you know your HTTP from your FTP? Your cookies from your spam? Internet jargon, services and formats are explained here. 

Printable Version

You can view a page, section, or entire tutorial in a format ideal for printing. This version shows the location of each site mentioned. 

Learn how to Cite Internet Resources

The variety of Internet resources, and their constant changing content, brings its own problems when we try to cite them in our work. Some advice and pointers on these issues are given at the end of each tutorial.

The Small Print

If this tutorial sounds like something you've needed for a long time, you're probably wondering what the catch is and secretly suspecting that some financial outlay is required. If so, here is more good news – it's free!

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YOUR PROBLEM ANSWERED

My elderly aunt who suffers from dementia rarely gets a good night's sleep. Is there anything that can be done about this?

Disorders of sleep and dreaming are unfortunately very common and troublesome in dementia. In addition as people age their sleep changes - they sleep less and wake up more frequently during the night and some take naps in the daytime. Dreams take place during the phase of sleep known as rapid eye movement sleep (REM). Some disorders of sleep have changes in REM and are associated with acting out vivid dreams. The best example of this type of disorder is rapid eye movement sleep

behaviour disorder (RBD) which is commonly found in Dementia with Lewy Bodies or in Parkinson's disease with dementia (which may be the same condition).
The commonest sleep disorders in Alzheimer's disease (AD) are an exaggeration of age changes ; disturbances of sleep rhythm (circadian rhythm, controlled by the "body clock") and excessive daytime sleepiness (EDS).

Our "body clock" regulates our sleep/wake cycle, our body temperature and some hormone levels. The clock is influenced by our environment and most especially by the level of light to which we are exposed during the daytime. In AD the sleep/wake cycle is often disturbed, at its most extreme sufferers reverse their sleep pattern-being awake at night and asleep in the daytime. There is some evidence that this is worse in the winter months, when daylight is of shorter duration than during the summer and that it is associated with abnormal behaviour, such as agitation.

EDS increases as we age but does correlate with poor memory.

Another common cause of sleep disorder associated with memory disorder is sleep apnoea-characterised by interruption of breathing (for seconds) and snoring.

What can be done for sleep problems in AD?

The first step is to try to establish the type of sleep problem. For some people simple measures - sleep hygiene - may suffice. Things like avoiding stimulation before bedtime, no coffee or cheese snacks in the evening, reducing noise and increasing comfort in the bedroom.

For RBD clonazepam in small doses can be effective. For circadian rhythm disturbances, increasing daytime light exposure or Light therapy (in the winter months) may be helpful. Medicines that are used to treat agitation in AD ,such as risperidone sometimes also help with sleep. The "anti-dementia" drugs-such as donepezil, are also quite effective in helping sleep disorder and dreaming in AD. Sleep apnoea can be treated by a special device which reduces snoring.

Poor sleep in AD sufferers also affects carers, it needs to be assessed and managed.

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